

# THE NECROPSY BOOK

## A Guide for Veterinary Students, Residents, Clinicians, Pathologists, and Biological Researchers

Revised January 2013

by

**John M. King**

**Lois Roth-Johnson**

**David C. Dodd**

**Marion E. Newsom**

### PROFESSORIAL POST SCRIPTS

*Cornell faculty often have manuscripts in various states of completion when they reach emeritus status. This archival repository is a place where these manuscripts, some previously published but out of print, and some that have not been subjected to peer review, may be valuable for their content, but sometimes also for their historical value. This collection is a place for these legacy works to become accessible online.*

Cornell University

Released: October 2014

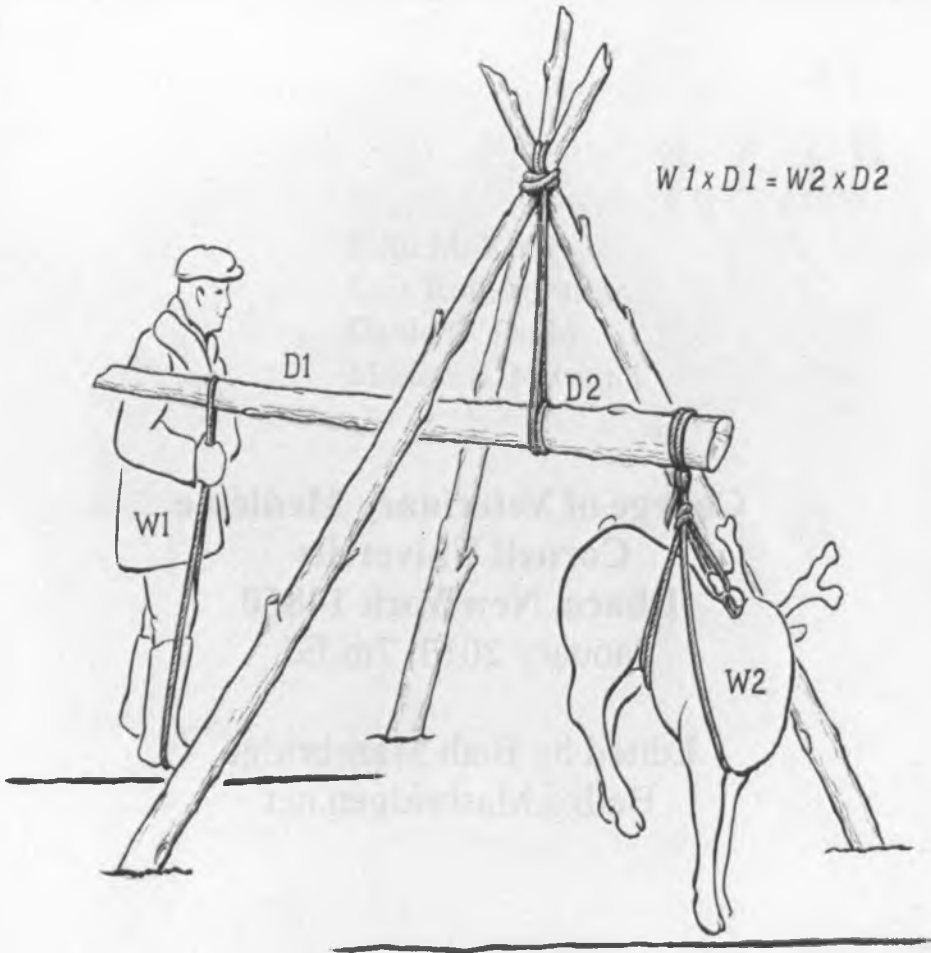
This book – <http://hdl.handle.net/1813/37948>

Professorial Post Scripts Collection – <http://ecommons.library.cornell.edu/handle/1813/37929>

The Internet-First University Press – <http://ecommons.library.cornell.edu/handle/1813/62>

# The Necropsy Book

King  
Roth-Johnson  
Dodd  
Newsom



**THE  
NECROPSY  
BOOK**

**A Guide for Veterinary Students,  
Residents, Clinicians, Pathologists,  
and Biological Researchers**

**College of Veterinary Medicine  
Cornell University  
Ithaca, New York 14850  
January 2013, 7th Ed.**

**Edited by Beth Mansbridge  
[beth@mansbridge.net](mailto:beth@mansbridge.net)**

**Charles Louis Davis DVM Foundation  
Publisher**

**THE  
NECROPSY  
BOOK**

**A Guide for Veterinary Students,  
Residents, Clinicians, Pathologists,  
and Biological Researchers**

---

*Revised January 2013*

---

John M. King  
Lois Roth-Johnson  
David C. Dodd  
Marion E. Newsom



**John M. King**  
N.Y.S. College of Veterinary Medicine  
Cornell University  
Ithaca, NY 14853  
jmking31@aol.com

**Lois Roth-Johnson**  
2955 Herring Brook Rd.  
Eastham, MA 02642

**David C. Dodd**  
11506 Oakmont Ct.  
Fort Myers, FL 33908

**Marion E. Newsom**  
304-1234 West 14th Avenue  
Vancouver, BC V6J 2J4  
Canada

*Dedicated to  
Marie King  
and  
Jon David King*

**This booklet could not have been written  
without the teaching by great teachers:**

**Dr. John Bentinck-Smith**

**Dr. Lennart Krook**

**Dr. Wendell Krull**

**Dr. Kenneth McEntee**

**Dr. Peter Olafson**

**Dr. Roger Panciera**

**Dr. Charles Rickard**

<b>Reportable Features of Most Lesions</b>	
<b>Morphological Descriptions</b>	
<b>Dedication</b>	
<b>Introduction</b>	<b>1</b>
<b>History (Anamnesis)</b>	<b>4</b>
<b>Necropsy Request Form</b>	<b>5</b>
<b>Necropsy Report</b>	<b>6</b>
<b>The Gross Necropsy Description and Report</b>	<b>8</b>
<b>Main Features to Include for Each Lesion</b>	<b>10</b>
<b>Sample Necropsy Report</b>	<b>12</b>
<b>General Necropsy Technique</b>	<b>14</b>
<b>External Examination Subcutis and Umbilical Area</b>	<b>17</b>
<b>Body Cavity Opening and Evaluation</b>	<b>19</b>
Gastrointestinal Tract Removal	22
Abdominal Visceral Removal	24
Tongue, Thyroids, Esophagus, Trachea, Lung, and Heart Removal	29
Opening the Heart	33
Joint Examination	36
Eye and Brain Removal	37
Pituitary Removal and Bone Marrow Examination	42
Ligaments, Tendons, and Joints	43
To Remove Mandible	47
Guttural Pouch Examination	49



<b>Anatomic Diagrams for Necropsy Findings</b>	<b>50</b>
Horse, Cow, Pig, Sheep and Goat, Dog, Cat	
<b>Common Findings of Little or No Diagnostic Importance</b>	<b>56</b>
<b>General and External Findings</b>	<b>56</b>
<b>Spleen</b>	<b>60</b>
<b>Liver and Gall Bladder</b>	<b>62</b>
<b>Endocrine</b>	<b>64</b>
<b>Renal</b>	<b>65</b>
<b>Respiratory</b>	<b>66</b>
<b>Cardiovascular</b>	<b>69</b>
<b>Bone</b>	<b>72</b>
<b>Central Nervous System</b>	<b>73</b>
<b>Fetal and Neonate</b>	<b>73</b>
<b>Gastrointestinal</b>	<b>74</b>
<b>Addenda Notes</b>	<b>81</b>

## Table of Contents

Page

<b>Odors Encountered at Necropsy</b>	<b>81</b>
<b>Brief Comments on Neoplasia</b>	<b>84</b>
<b>The Time of Death</b>	<b>85</b>
<b>Decomposition</b>	<b>88</b>
<b>Miscellaneous Comments</b>	<b>89</b>
<b>Terminology</b>	<b>91</b>
<b>Fatal Diseases or Conditions with No Lesions</b>	<b>92</b>
Allergy and Allergy-Like Deaths	93
Fetal Deaths	95
Poisonings and Toxins	95
Deficiency Diseases	98
Metabolic-Related Diseases	100
Miscellaneous	101
<b>Representative Stages of Postmortem Lung Inflation</b>	<b>103</b>
<b>The Pneumonias</b>	<b>104</b>
<b>Rough Sketches of Selected Items</b>	<b>106</b>
Lung	107
Liver	154
Kidney	176
Heart	193
<b>Types of Fat Necrosis</b>	<b>222</b>
<b>Klotz Solution Formulation</b>	<b>223</b>
<b>Greek and Latin Prefix and Suffix List</b>	<b>224</b>

<b>Ancillary Testing from the Necropsy Table</b>	<b>232</b>
Field Weighing of Large Animals	232
Pandy's Reagent and CSF	234
Grease-Free Skeleton Preparation	235
Muscle Weights and Hypertrophy	236
The Collection of Heavy Tiny Particles	237
Bone Breaking (Shear Force) Strengths	238
Clearing Small Animal Bones	240
Hoof Examination by Wall Removal	241
Large Animal Euthanasia	242
Intestinal Villi Movement	243
Lymphosarcoma and Adipose Tissue	243
Better Lung Tissue Preservation	243
Glass Slide Smears	243
Short-Term Burial for Carcass Preservation	244
Parasite Demonstration and Collection	245
Simple Feed Testing in Rats	246
<b>Reportable Features</b>	<b>247</b>

## INTRODUCTION

A necropsy is an autopsy performed on an animal. A method for doing a necropsy, using the horse as a model, is described. Since the use of the horse may be questioned, we could merely reply that we had to use an animal and the horse is ubiquitous. Another reason, not usually expressed, is that the horse not only has a single stomach as does the pig, dog, and cat, but also has large fermentation chambers equivalent in function to the rumen of the ox and sheep.

The actual procedure of doing the necropsy is one of many used by veterinary pathologists and its practicality and adaptability have been demonstrated. We make no claim for the superiority of this method over others, but from considerable experience with it, we know it can be used with facility on any species under consideration; by adhering to it, no organ will be and no lesion should be overlooked. No special equipment or instruments are usually required. Any phase of the procedure can easily be modified to suit the prosector's special needs or interests. We stress, however, that having acquired facility and speed with one procedure, it is unwise to change it capriciously; a different method usually takes more time and may cause one to forget a necessary part of the procedure. A change in technique may also preclude instant recognition of an absent or displaced organ or otherwise familiar abnormality. For instance, the pylorus and first part of the duodenum are always close to view when the abdomen is opened by this technique and, if not directly in view, one should immediately consider a twist or displacement of the stomach. The first kidney observed is the right kidney, and if it is the only one with the cranial pole infarct, this can be remembered more easily if one necropsies all animals with the right side up. Except for the GI tract, all tissues and organs are examined as they are removed to prevent gross contamination of the carcass.

We have not gone into many areas, such as organ weights as often judging the amount of blood still present even by weight can cause significant misjudgments, because we as general pathologists are more than likely to recognize organs as being too small or too large or too thick or too thin. Average weights are given in many texts or reference books. However, several weights have been given. For instance, the cat's heart is normally under 17 grams, and knowing this helps make one specific disease more recognizable.

Although it takes time, a thorough necropsy is probably the best use of time overall. The vast majority of lesions, especially significant ones, will be uncovered. At a later date, the pathologist will know that he/she looked at all of the significant tissues and organs without thinking, "Did I or didn't I?"

In the same vein (no pun intended), the worst disservice a teacher can do to a student is to say: "Go ahead and do the necropsy and if you run into a lesion or find that you have a problem with one, call me and I'll be right there to help you out." How about the seventeen great or even insignificant lesions or non-lesions causing interpretation problems that the student did not recognize as being abnormal? The least the instructor must do for a trainee in any area of diagnostics is to review every carcass, tissue, and organ before it is discarded or destroyed.

This technique intends that all major organs and tissues are examined, and others, especially of clinical interest, will be opened in due course of the necropsy. Tissues considered normal grossly may not be described in favor of brevity, but tissues suspected clinically must be described and so stated to not err by omission.

This booklet is intended to be used at least in part with the pictures of actual tissues to be found on the Cornell Web page:

**<http://w3.vet.cornell.edu/nst/>**. From this home page, click on Advance Search and type the number of the slide (which follows the WP in this text) in the Image ID box.

## **THE HISTORY (ANAMNESIS)**

Ideally, a complete history should be available prior to the necropsy, as it is for the clinician doing his own necropsies. Sometimes it is not available, and to some pathologists it is important to do a necropsy without being prejudiced by an erroneous, misleading, or incomplete history; however, never discard the carcass without reading the history. Often the individual doing the necropsy will erroneously not finish doing a complete necropsy when some obvious, but not necessarily important or fatal lesion, is found. For instance, a brain lesion may be the cause for inhalation pneumonia in an adult animal, but if the pneumonia is severe enough, the pathologist may quit the necropsy and not take out the brain. Take the history in all cases by first filling out the request form. A model of the request form is given in this text. Many specialty items must be checked, as the history may suggest, but to include all in a general diagnostic technique book would make it too cumbersome.

# NECROPSY REQUEST FORM

Category C N O U

Date Rcvd. \_\_\_\_\_

Diag. Vet Acct # \_\_\_\_\_ Clinic # \_\_\_\_\_ Postmortem # \_\_\_\_\_

Animal's ID \_\_\_\_\_ Species \_\_\_\_\_ Breed \_\_\_\_\_ Sex \_\_\_\_\_ Age \_\_\_\_\_ Wt. \_\_\_\_\_

Died/Killed/Unknown \_\_\_\_\_ Method of Euthanasia \_\_\_\_\_ Date & Time of Death \_\_\_\_\_

Number of animals on premises: Adult \_\_\_\_\_ Young \_\_\_\_\_ Duration of Illness \_\_\_\_\_

Number affected: Adult \_\_\_\_\_ Young \_\_\_\_\_ Number Dead \_\_\_\_\_ Seen by Vet.: yes \_\_\_\_\_ no \_\_\_\_\_

Antemortem Diagnosis: \_\_\_\_\_

Veterinarian: \_\_\_\_\_

Client: \_\_\_\_\_

Address: \_\_\_\_\_

Address: \_\_\_\_\_

City \_\_\_\_\_ State \_\_\_\_\_ ZIP \_\_\_\_\_

City \_\_\_\_\_ State \_\_\_\_\_ ZIP \_\_\_\_\_

Telephone # (\_\_\_\_) \_\_\_\_\_

Telephone # (\_\_\_\_) \_\_\_\_\_

**HISTORY: Please include a complete history to include feed and husbandry changes, new animals, treatments, date of onset of illness, etc. Use reverse side if necessary.**

Fee Required \_\_\_\_\_

Clinician/Owner Signature \_\_\_\_\_

NOTE: Reports/results/bills are sent directly to clinicians who are responsible for notifying owners of necropsy and other results.

Additional copies to be sent to: \_\_\_\_\_

Provisional Gross Diagnosis: \_\_\_\_\_

Koda \_\_\_\_\_ Histo \_\_\_\_\_ Bac \_\_\_\_\_ Virol \_\_\_\_\_ Parasit \_\_\_\_\_ Toxicol \_\_\_\_\_ Freezer/Storage \_\_\_\_\_

List tissues as needed:

Student \_\_\_\_\_ Intern/Resident \_\_\_\_\_ Pathologist \_\_\_\_\_

Initial report via phone by \_\_\_\_\_ on \_\_\_\_\_ to \_\_\_\_\_



## THE NECROPSY REPORT

Fill out the Necropsy Request Form as requested. The date is often a legal requirement. You would be lost in a court of law without it.

**Animal's ID:** Usually refers to its given name, ear tag, or tattoo number. Possibly find and record the now common implanted animal's ID chip.

**Species, Breed, Sex, and Age:** Are most helpful and required.

**Died/Killed/Unknown:** Indicate the correct one.

**Method of Euthanasia:** Tells how this was done and with what, i.e., intracardiac barbiturate, halothane, gunshot, etc.

**Date and Time of Death:** Do not make the common error of accepting the time when the animal was found dead as the actual time of death. Establish the time, if possible.

**Total Number Affected:** The obvious is necessary.

**Duration of Illness:** Of great importance in history taking.

**Antemortem Diagnosis:** Give several, if necessary.

**Body Weight:** Helpful and necessary.

**Veterinarian:** Absolutely necessary, as we report to the veterinarian in all cases. Include the phone number, fax or email, if possible.

**Client:** Absolutely necessary, including the owner's signature for legal permission to do the necropsy. Point out the fee requirement to the owner.

**History:** Fill in all pertinent history. Attach accompanying history sheets if available.

**Additional copies:** To be sent to clinicians and those listed.

**Provisional Gross Diagnosis:** Fill this in after the necropsy to help the ancillary labs to determine their testing procedures as needed.

**Photographs, Histology, etc.:** Should be checked off if used and note each tissue selected.

**Student, etc.:** Fill these in.

**Initial Report By:** The necropsy room pathologist in charge usually calls the referring clinician by the next morning. If anyone contacts the involved clinician before this time, it should be noted on the sheet so the referring clinician is not called twice.

All of the requested facts above may be of great legal value and should be recorded.

**Special note:** A definite statement should be made about any tissue or condition called into question by the referring clinician or owner to clarify that the condition or organ was specifically examined.

Of course the necropsy must come before the descriptive write-up, but in this book we've given the write-up technique first to make the prosector aware of what should be looked for before doing the necropsy.

## THE GROSS NECROPSY DESCRIPTION AND REPORT

An important part of the necropsy is the description of lesions. Prosectors, especially neophytes, should realize that all findings may not be lesions; that is, they may not be abnormal changes in structure (or function). In fact, it is more often the case that the neophyte will consider lesions such things as: **(1)** physiological changes, gastrointestinal congestion, or postmortem hypostasis in the lung and liver as antemortem congestion or even hemorrhage; **(2)** normal features such as the torus pyloricus in the pig stomach, the normal duodenal papillae in the horse and dog, which are often mistakenly called tumor nodules or ulcers. Prominent lymphoid foci in the colon or even Peyer's patches are misdiagnosed as ulcers; **(3)** artifacts, which are changes caused at death, either physically or chemically, are not antemortem changes. Barbiturate salt deposition on the pleura is a good example and so are changes associated with rat or other wild animals feeding from the dead carcass; **(4)** postmortem changes in all species associated with decomposition are common misdiagnosed changes. They range from nasal froth to mucosal sloughing of the forestomachs in ruminants.

We often tell the novice that most things seen during a necropsy fall into several categories, which include: **normal, artifact, postmortem change** (autolysis), **parasite lesion**, and last but not least, **lesions of significance**. Thus, with three out of five changes being of no great significance, it is no wonder that the neophyte may be in error.

**The first sentence should be an accurate description of the carcass and its condition.** In the actual write-up (in the present tense) of a necropsy report of an individual animal, it is recommended that the initial sentence identify the carcass to avoid any mistake in identification, e.g., “The carcass is that of an estimated/given age 4-year-old, 8.5 kg., intact female, tri-color Beagle dog, ear tattoo N312, in good nutritional condition.” The remainder of the necropsy report may be done in many ways. Often, the order of the necropsy is followed in the report as it may bring to mind more easily the lesions seen, or it may be dictated directly at the necropsy table. Lesions are described in the main text, but they are **not** interpreted. Following the written report, it is often advisable to list the interpretations of the gross findings next to the organs involved. This is best done by an experienced pathologist who should correctly interpret the lesions and give the morphologic diagnoses to the lesions found. Then the pathologist’s summation of all findings in the case should be given as the gross diagnosis. A comment may follow to help clarify the case to the clinician. All lesions should be described in regards to: location, color, size, shape, consistency, and number or percent of involvement of a specific organ. The following relatively standard set of features should be noted to describe the changes seen at necropsy. It should be noted here that the freezing of a carcass does not usually destroy critical diagnostic lesions, provided the carcass was not decomposed before or after freezing.

## MAIN FEATURES TO INCLUDE FOR EACH LESION

1. **Location:** Where on the skin, what part of the lung, etc. The anatomical position, and its relationship to other organs and tissues (cranial, caudal, dorsal, ventral, left side, stomach, right adrenal) is given. Even a poor hand-drawn picture may be of more value here than the word description.

2. **Color:** Use of the primary colors is best, with shades and degrees as needed. Some people have never seen a green apple, or any apple at all. All oranges are not orange. Mauve and pastels are difficult to interpret. Dark, brilliant, light, mottled, streaked, or stippled may apply.

3. **Size:** Only metric units should be used. Your necropsy knife handle should be marked every 1/2 cm so that you have an instantly available measuring device. Be objective and never use common objects, including fruits and vegetables, to indicate size. Centimeter notches on the knife handle are very useful here.

4. **Shape:** Use descriptive terms such as: ovoid, round, conical, flat, nodular, lobular, tortuous, discoid, punctate, bulbous, wedge-shaped, fusiform, laminated, clustered, lace-like, straight-edged, etc.

5. **Consistency and Texture:** A most important feature of lungs; palpation is the key. Sometimes even physical manipulation, such as actual bone breaking at the necropsy table, is helpful. Soft (lips), firm (nose), and hard (forehead); as well as fluctuant, gas-filled, friable, viscous, mucoid, gelatinous, stringy, turgid, dry, inspissated, caseous, crepitant, adhesive, gritty, granular, pliable, homogenous, etc., may be used.

**6. Number and Extent (%):** Give a count whenever possible. Do not use the words “few” or “several.” Use dozens or hundreds in cases that apply. In cases of pneumonia, liver disease, or where portions of a large organ are affected, the extent of involvement given in percent is of great judicial significance. Many animals may have one whole lung (50%) involved and still be clinically normal.

**7. Content:** Quantity and nature of content in any cavity, natural or pathologic, is described in volumetric terms as well as the weight, color, odor, consistency, and shape of the content itself. The content description of the GI tract or uterus is of value and especially so for medicolegal cases. Stricture or collapse of these hollow organs may require such terms as: patent, dilated, partially obstructed, obliterated, narrowed (including degree of narrowing), branched, communicating, tortuous, etc., with respect to their lumen.

In addition to the above standard set of features to be noted about each lesion, the necropsy report should include, when applicable, comments on:

(1) **Odor:** This is one of the hardest features to evaluate, but it is often quite diagnostic. By necessity, it is sometimes described in relation to well-known odors. For example, “similar to rancid butter,” cider, onions, etc.

(2) **Distribution and Surface Appearance:** “Focal” or “multifocal,” “locally extensive,” or “diffuse” covers many conditions while “total,” “scattered,” “streaked,” and “laminated” are useful. “Ulcerated,” “hairy,” “smooth,” “depressed,” “irregular,” “eroded,” “pitted,” “elevated,” “glistening,” “dull,” “scaly,” and “membranous” are of much value for surface descriptions.

## SAMPLE NECROPSY REPORT

P.M. # N88-36

This is the carcass of a 41 kg., 1–2 year-old, Suffolk ewe in poor nutritional condition with no appreciable autolysis and minimal body fat stores. A yellow, plastic ear tag #57 is in the left ear. The oral and nasal mucosa are pale gray, and the conjunctiva and vaginal mucosa are pale white. Only a small amount of fecal staining is in the wool of the perineum and es-cutcheon.

A moderate amount of clear, gelatinous tissue in the ventral cervical subcutis and about 150 cc of clear, light-yellow watery fluid in the peritoneal cavity. 30 cc of similar fluid with 4 fibrin clumps are in the pericardial sac.

The lungs are diffusely wet and mottled bright red. Seven, 0.2 cm spherical nodules and six, 0.2 x 0.5 cm, elongate, dark-gray nodules scattered in the dorsal caudal right lung lobe.

The liver is diffusely pale. The kidneys have about 50, 1 x 1 mm white foci scattered in their outer cortices. Four tape-worms, 30–50 cm x 10 x 0.2 cm, are in the ileum. The abomasum has only two thin 1 x 10 mm worms present. The forestomachs are normal as to content. No appreciable content is in the entire intestinal tract. Numerous, 50–100, gray-white, circumscribed, 0.25–0.5 cm, irregular, hard, gritty nodules are scattered in the cecal wall and serosa.

The animal is not pregnant (when clinically questioned).

No gross lesions are in the brain or spinal cord.

### **GROSS FINDINGS:**

Body as a whole – Emaciation; anemia

Abomasum – Endoparasites, Hemonchus sp.

Ileum – Endoparasites, Monezia sp.

Cecum – Chronic endoparasitism, Esophagostomum sp.

Lung – Terminal edema; chronic multifocal pneumonia,  
Protostrongylus sp., presumptive

Pericardial sac – Hydropericardium

Peritoneal cavity – Ascites

Subcutis, ventral neck – Edema

### **GROSS DIAGNOSIS:**

Emaciation

Chronic Endoparasitism

Note: Basic husbandry problems with nutrition and parasites are suspected.

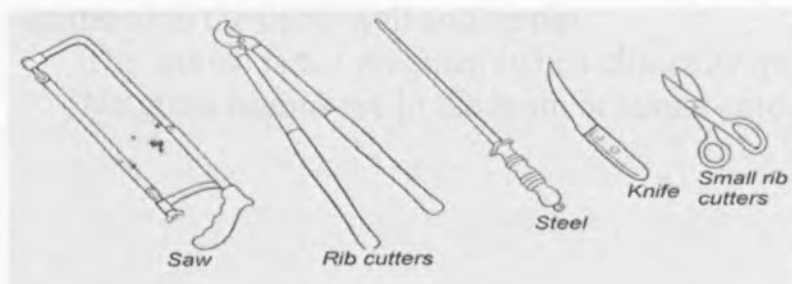


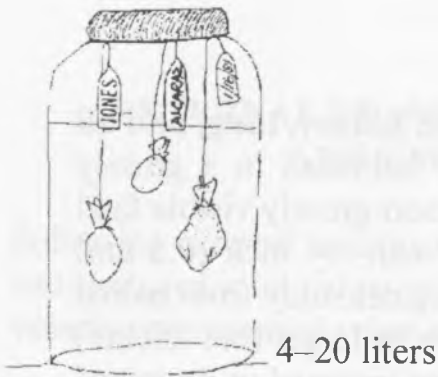
## GENERAL NECROPSY TECHNIQUE

All animals are placed on their left side with the dissector facing the animal's abdomen. As this technique is demonstrated with a normal animal, it is expected that common-sense variations will have to be used when one encounters abnormalities (lesions) or various physiologic states such as pregnancy. Although the horse is the model in this text, the major procedures are applicable to most species. Variations in techniques are noted where necessary.

Wet the necropsy table surface to prevent adhesion of blood and other fluids for easier clean-up. Read the history for indications of special techniques and care to be taken during the necropsy. Rubber gloves are required for all necropsies. Use at least ten times the volume of 10 percent neutral buffered formalin to the volume of tissue taken for histopathology. Label the tissue bottle or other containers properly. Some other instruments may be needed as occasion dictates (bone chisel, Stryker saw, scissors, syringe, etc.).

Make notches one centimeter apart on knife handle to prevent hand slippage and to have an instantly available measuring device.





Samples from several animals are being fixed at the same time to conserve solution. This solution can be used over and over with good results.

Individual, properly labeled (name and date) tissue samples to be sent to a laboratory after proper fixation in a large volume of fixative for fast, good fixation. It may now be sent with minimal fluid or just formalin-soaked cotton to keep it moist if sent to a diagnostic facility.

A large 50 x 30 cm white-bottomed tray is very useful for examining portions of intestinal content and looking for parasites.

From years of use and observation, a properly sharpened knife needing only a few swipes on a sharpening steel during the necropsy of an adult horse or even a small animal is the most important instrument. Surprisingly a good sharpened knife is not easily found throughout the world but can be made sharp with a 24-inch belt sander belt of 100 or finer grit stretched over a hard wood sanding block. A sharp knife is understood to be able to shave hair from one's arm with care.

**Routinely take tissue samples of liver, kidney, lung, and all lesions.** Brain and heart sections are indicated in a grossly negative necropsy as they may harbor non-grossly visible fatal lesions. Sections should be no more than 1/4 inch (0.5 cm) thick. When taking sections for histological study from paired organs, make the left side pieces longer or larger (not thicker) for easier identification later when being trimmed or described to pathologists. As in any technique description, experience is needed to increase proficiency.

Use of the carcass itself as a cutting board is recommended to prevent dulling the knife. To prevent cutting hair and thereby dulling the knife, the one stab wound in the axilla is the only time the knife cuts hair because the skin is reflected by cutting the subcutaneous tissue with the back of the blade towards the carcass.

During the dissection of a carcass it is often of benefit to save wet tissues so that they retain some of their color and softness. Quick rinsing in water and putting them in a holding solution, such as cold Klotz Solution\*, can be used to keep the tissues for a week or two in order to show others, including students, the almost natural appearance of findings which otherwise would be just firm, even hard, and with a tan uniform color if stored in formalin solution alone.

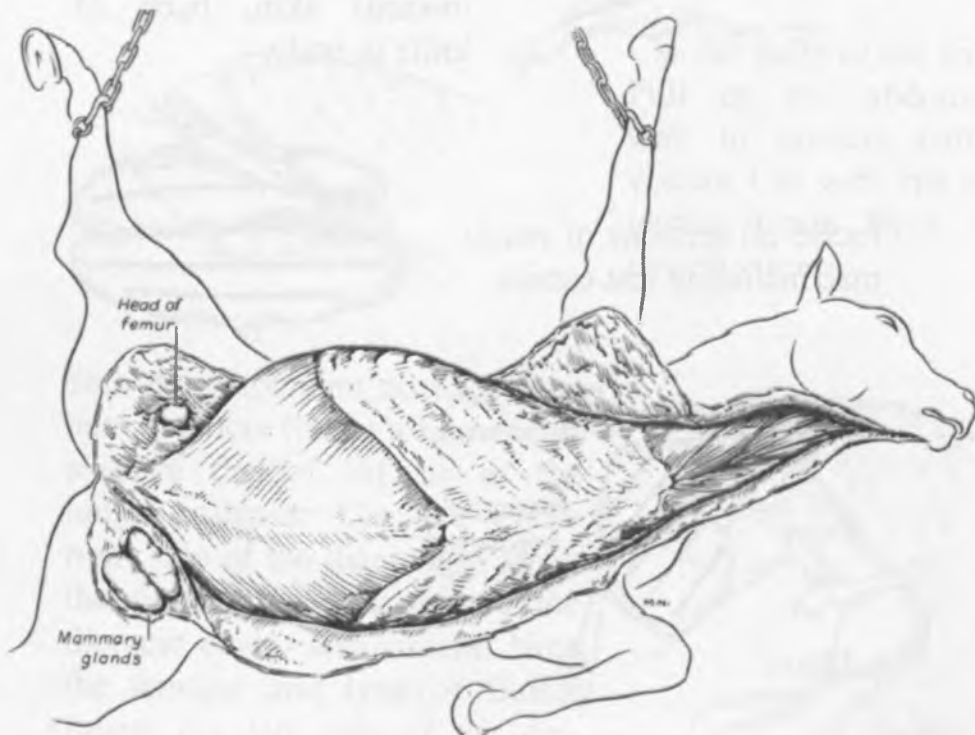
\*See page 23 for Klotz formula.

Certainly, other tissues than those listed are to be examined if lesions are suspected in them clinically.

## EXTERNAL EXAMINATION AND SUBCUTIS AND UMBILICAL AREA

Following external examination, including natural orifices, eyes, and limb and joint palpation, the lymph nodes, nerves, and most vessels are examined when exposed.

After an initial stab incision into the right axilla, extend skin incision cranially, just to right of midline, to chin and caudally to perineum, just above the genitalia. Examine jugular veins at this time.

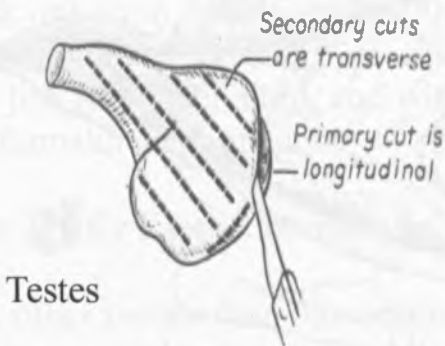
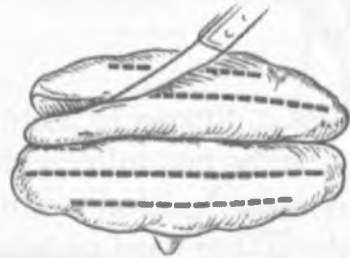


Reflect skin on right side and completely abduct right limbs by cutting muscular attachments of scapula and freeing femoral head. Reflect mammae or free each testicle separately and make sure to examine the right mamma or right testicle or any right-sided organ of paired organs before the left to help make recollection of which was the affected organ when time for write-up comes. Cut open sheath. Cut back skin for short distance to left of midline.

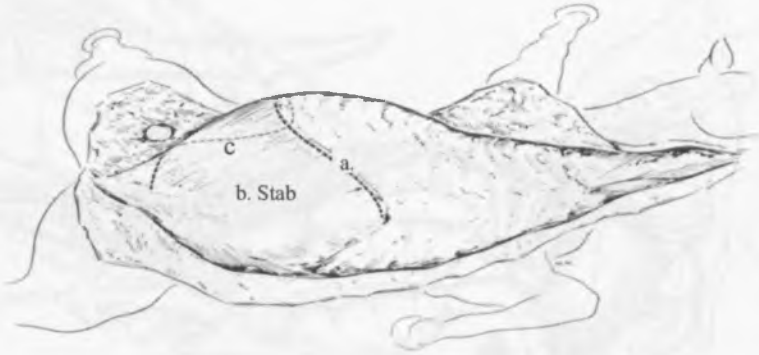


When cutting back skin, maintain belly of knife towards skin, back of knife to body.

Incise all sections of mammae including teat canals.



## BODY CAVITY OPENING AND EVALUATION



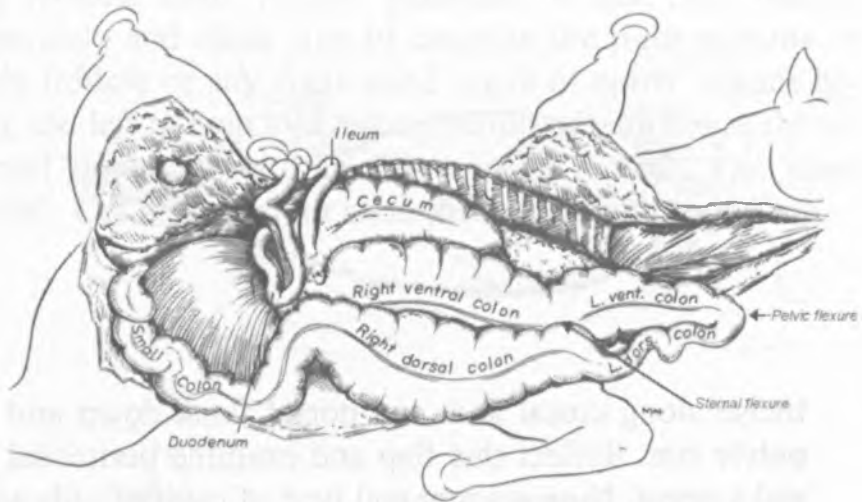
Incise along costal arch and dorsal flank down and across pelvic rim. Reflect this flap and examine peritoneal cavity and viscera. Note amount and type of cavity fluids and other contents as the body cavities are opened.



Use the belly of the knife. Pull up the abdominal wall to prevent cutting viscera. Cut with fist protecting blade from distended viscera.

Stab the diaphragm near the sternum and note (listen to) inrush, or absence thereof, of air as the lungs collapse. Cut the entire right side of the diaphragm along the costal arch and observe the thoracic cavity and viscera. Note the amount and type of fluids. Leave the left side of the diaphragm attached to the wall when removing the thoracic and abdominal viscera.

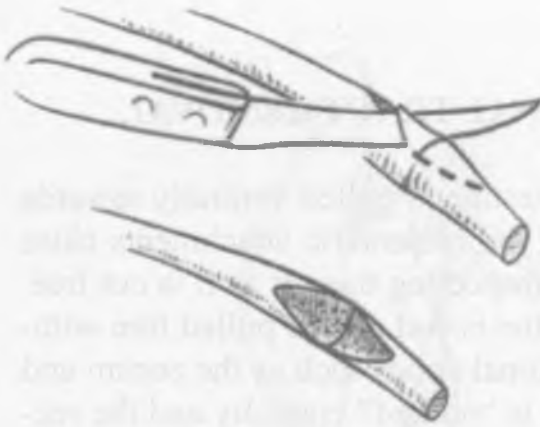




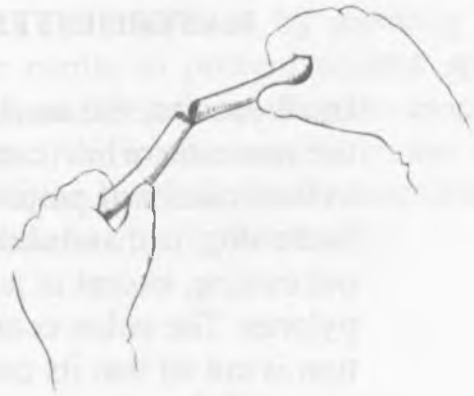
Remove the ribs by cutting with rib cutters or saw, first close to the sternum, then several inches from the vertebrae. Check the presence and position of all organs. Arrange the GI tract to display all of the parts before removal. In the **horse**, place cecum dorsocranially, small colon on left thigh, large colon cranially, and small intestine over right flank. In **cattle** and **sheep**, place the small intestine and colon over the right lumbar area, leaving forestomachs and abomasum in place. Examine but leave the pancreas attached to the duodenum or the root of mesentery. Before any visceral organs are removed from the peritoneal cavity, look for abnormal vessels (shunts) to or from the liver or intestine and especially leading to the caudal vena cava.



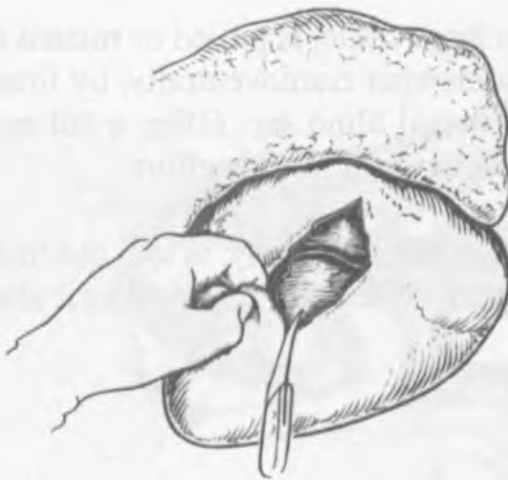
Free a central rib by cutting off adjacent soft tissue close to bone.



Check the costochondral junction of young animals by cutting along the thin edge cranially or caudally and **not** the flat medial (pleural) or flat lateral surface.



Break or attempt to break rib against curvature for test of general bone strength. See page 247.



*In situ*, open the pericardium and examine the pericardial contents as well as the external surface of the heart itself. Note the amount and type of fluids.



## GASTROINTESTINAL TRACT REMOVAL

In all species, the small intestine is pulled ventrally towards the prosector while cutting the mesenteric attachments close to the bowel and properly inspecting the gut as it is cut free. In the dog, cat, and sheep, the bowel can be pulled free without cutting, except at junctional zones such as the cecum and pylorus. The colon content is “milked” cranially and the rectum is cut so that its content is not spilled into the peritoneal cavity if, for some reason, the entire rectum and anus are not removed.

The large bowel of the horse is removed by pulling it further over the back while using blunt finger dissection to release it from its dorsal attachments and by finger stripping the major mesenteric vessels free while doing so.

The rumen, even in large cattle, is pulled or rotated out of the abdominal cavity somewhat cranioventrally, by firmly grasping the wall of the dorsal blind sac. Often a cut into the rumen itself allows a better grip for this effort.

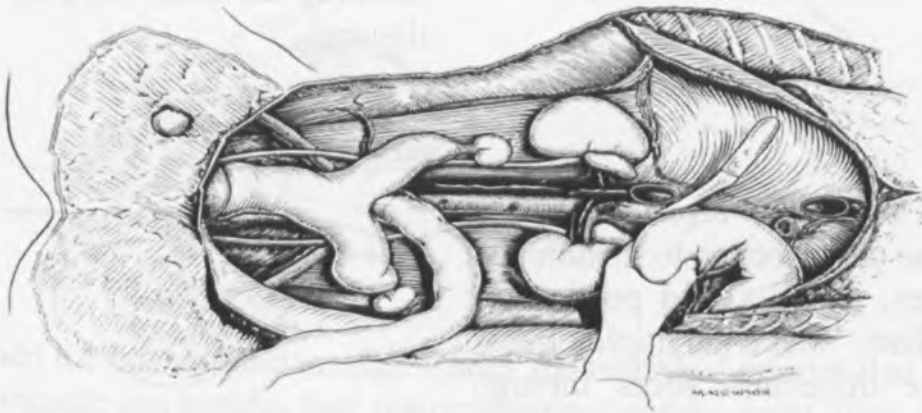
The GI tract, although removed early, is laid out in sequence, but only after all other viscera are removed is it also opened in sequence.





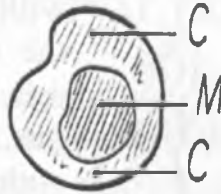
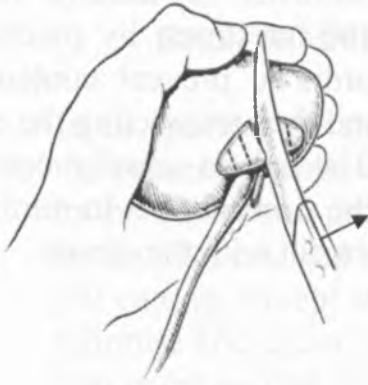
The stomach is usually removed with the intestines, by pinching off the cardia to prevent content spillage, and then transecting the esophagus. The spleen usually comes out with the stomach, but in ruminants it is taken out with the rumen.

Make several inspection slices into spleen.



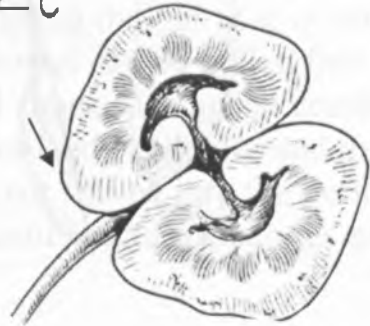
Remove the liver, leaving the diaphragm in place. Incise and inspect the gall bladder, in appropriate species, by opening onto a clean surface. Make multiple inspection slices into the liver and incise major vessels.

## ABDOMINAL VISCERAL REMOVAL



The ratio is roughly 1:2:1 in this example.

Do not hold the adrenal itself. Incise the adrenal. Note the cortex-medulla-cortex (CMC) ratio.



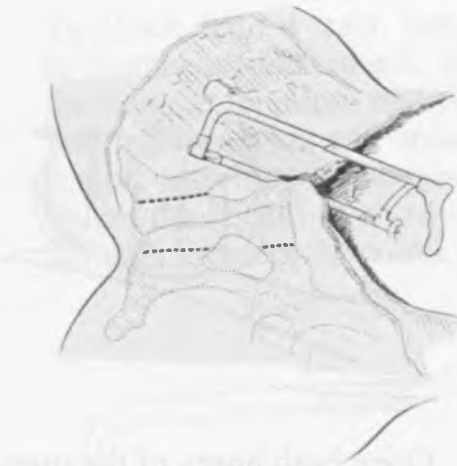
Cut each kidney longitudinally to pelvis. Leave each kidney attached to the bladder to allow the ureter to act as a third hand when handling the small slippery tissue.



Peel away the capsule. Take a tissue cross section to include the cortex, medulla, and pelvic epithelium. Make multiple transverse inspection slices through the organ.

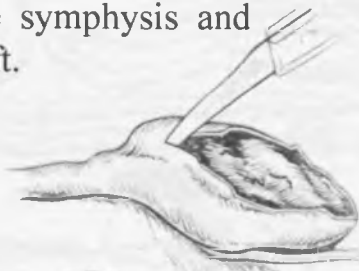


Cut down ureter by pulling slightly on the kidney to straighten the ureter and to allow a sharp pointed knife to cut it open to the bladder.



Open the pelvic cavity by sawing (or using rib cutters) through the pubis to the obturator foramen, then through the ischium, both sides. Remove the symphysis. Some suggest a single cut through both the symphysis and ilial shaft.

Cutting close to the bone, remove the pelvic viscera, kidneys with ureters, and bladder, genitalia, and rectum. Incise and inspect the lumen of the abdominal aorta and the vena cava.



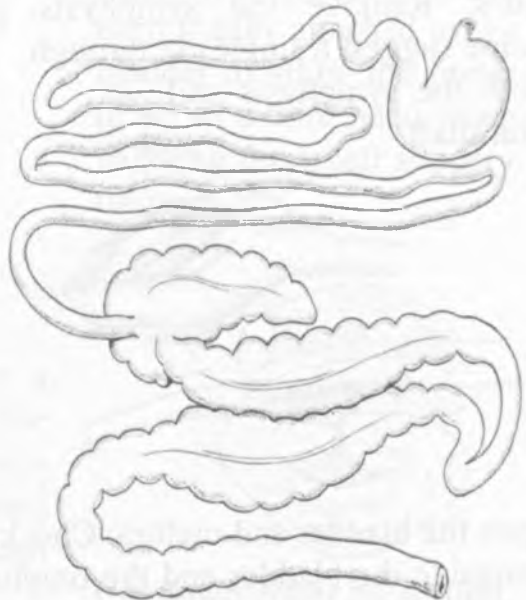
Open the bladder and urethra. Check the umbilical arteries that lie alongside the bladder and the omphalomesenteric veins from the umbilicus to the liver in newborn animals.

Cut the ovaries first longitudinally, then transversely.

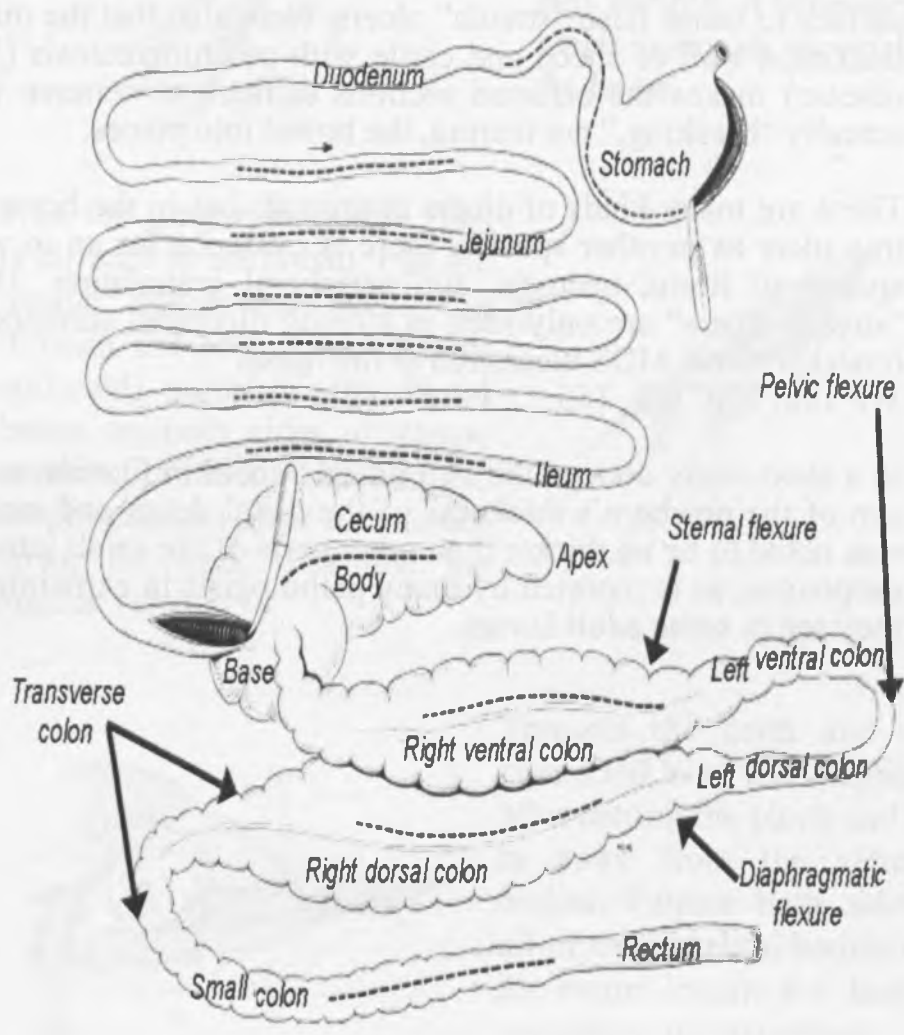


Open both horns of the uterus, then the cervix and vagina from the dorsal surface.

Lay GI tract in relative order, to be opened later as the last major procedure of the necropsy to prevent fecal contamination of the tissues and instruments.



To check the GI tract, cut along the greater curvature of stomach, forestomach, and representative lengths of duodenum, jejunum, and ileum. Open ileocecal orifice and cecum, large and small colon, and rectum. Incise major vessels when exposed. The ileum of the horse enters the cecum as shown here, but in other domestic animals it enters the colon.



**Note:** The small intestinal wall thickness is normally the same throughout its entire length as is the thickness of the large intestinal wall. Whenever a thickened bowel wall is suspected, a measured square of suspected thickened gut wall can be removed and weighed to be compared to a similar measured section from a normal bowel.

Decomposed tissues, including bowel, are more fragile than normal bowel. Increased care is needed in their removal. Edematous bowel, especially the colon of horses, tears easily on its mucosal surface to cause false “stretch” ulcers. Note also that the markedly thickened wall of sheep and cattle with paratuberculosis (Johne’s disease) makes the affected sections difficult to remove without actually “breaking,” not tearing, the bowel into pieces.

There are many kinds of ulcers in animals but in the horse with a true ulcer as in other species, there is evidence for an in vivo response of fibrin, necrosis, and peripheral granulation. The true “stretch ulcers” are only seen in already ulcerated surfaces of the bowel with NSAIDS ulceration in the horse.

WP 146, 150, 153, 155

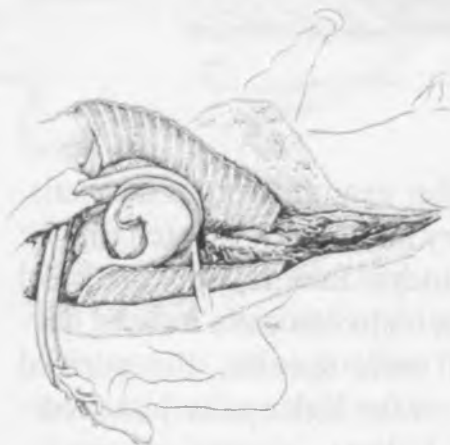
In a short study done at the Veterinary School in Florida, comparison of the newborn’s thickness of the distal ileum and esophagus was noted to be no thicker than other parts of the small intestine or esophagus, as is reported by many pathologists in explaining what they see in some adult horses.

## TONGUE, THYROIDS, ESOPHAGUS, TRACHEA, LUNG, AND HEART REMOVAL



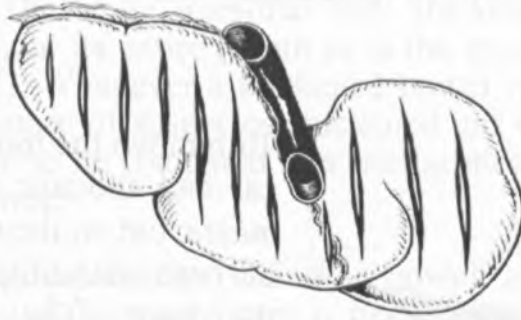
To remove the tongue, cervical and thoracic viscera *en masse*, cut on the medial side of both mandibles close to the bone. The symphysis may be split, if necessary, for easier removal, especially in the horse.

Free the tongue manually (this is difficult in the horse). Pull the tongue down and back. Cut through the prominent (kerato-epihyoid) joint of the hyoid bones on both sides. Continue traction, removing the trachea, esophagus, and other soft tissues down the neck. Examine the jugular veins.

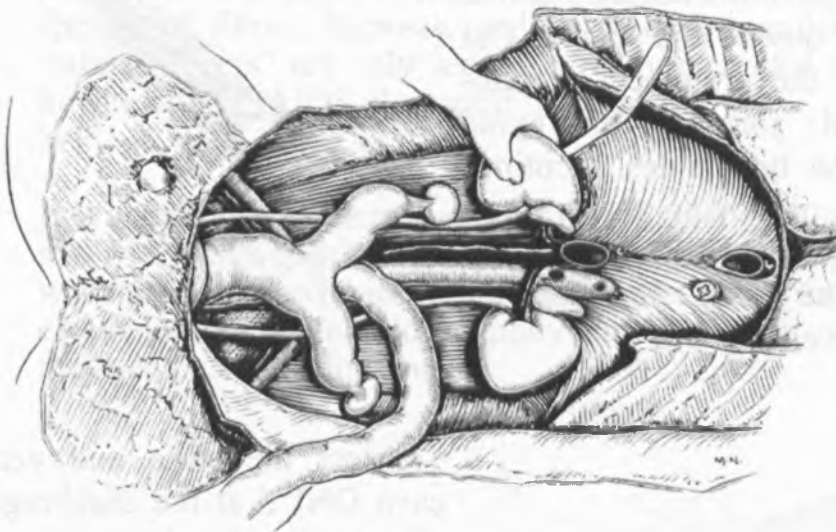


Transect the aorta and vena cava **ONLY** at the diaphragm after cutting the pluck and aorta away from the vertebral bodies. Palpate both sides of all of the vertebral bodies over its entire length for lack of symmetry and exostoses.





**Remember:** All sections for fixation should be less than 1/4" (0.5 cm) thick. Caution: Do not scrape or squeeze sections to be taken for histological examination. Always take sections of all tissues with a sharp knife, never with a pair of scissors.

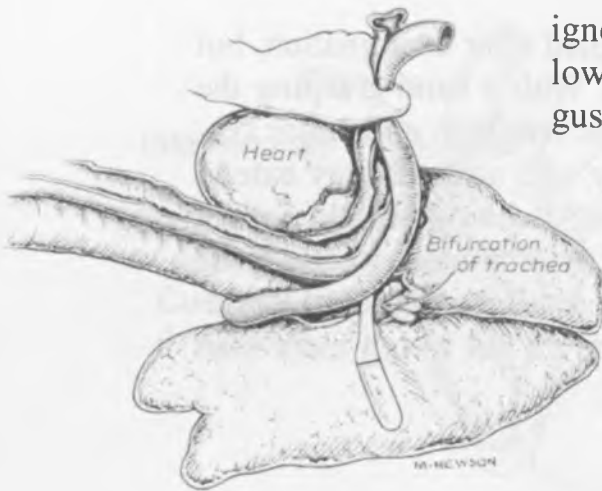


Remove kidneys separately after examination of adrenals. Leave ureter attached to kidney and bladder. Care must be taken when removing the kidneys, ureters, adrenals, and uterus, not to damage the aorta and vena cava located dorsally and close to them. In most species, the adrenal glands are found just in front of the kidneys or just medially to the cranial pole of each kidney.

Examine the tongue by transverse sections. Observe and incise the thyroids, remembering that the pig has only a single thyroid located midline near the thoracic inlet. Observe parathyroids. These are difficult to find in some species. Cut down the full length of the esophagus.



Free the esophagus and the aorta from the dorsal caudal mediastinum to allow access to both bronchi. This is a commonly ignored step in the procedure, which allows unnecessary damage to the esophagus and aorta if not done.



Leave the heart attached to the lung for best evaluation of the vessels involved and leave the lungs attached to the diaphragm to act as another third hand in pulling the esophagus and trachea tight when cutting down each of them, and down the bronchi.

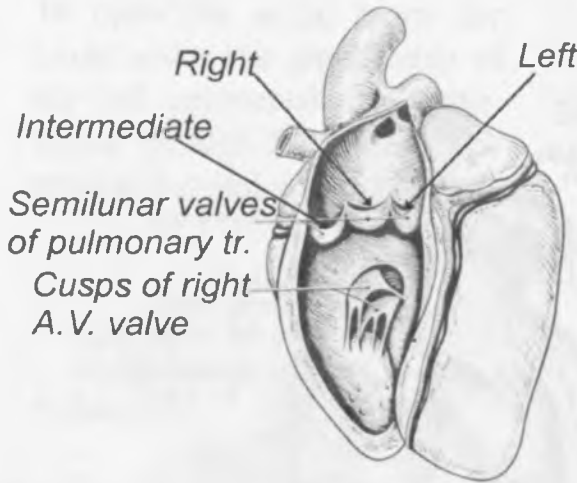
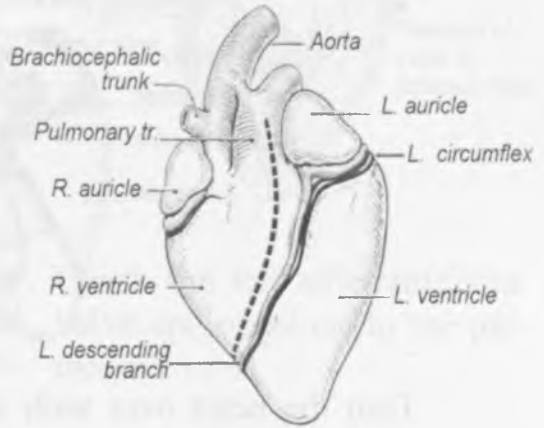
Palpate the lungs gently. Cut down the trachea and major bronchi and observe the cut ends of the pulmonary arteries for emboli. Incise the tracheobronchial lymph nodes. To cut down and examine the pulmonary arteries, it is best to turn the lungs over and cut from their ventral surfaces.

**Note:** Although shown isolated for better depiction in these drawings, it is wise to leave the heart attached to the lungs for the best orientation of major vessels and associated structures including malformations.

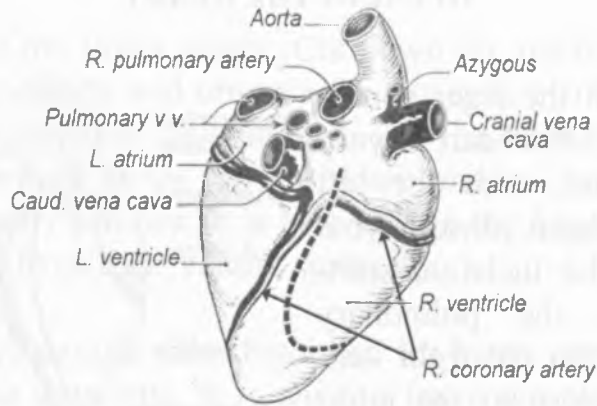
The heart should be weighed after examination, but before sections are taken. With a hand grasping the base of the heart, cut the pericardium and major vessels, the pulmonary artery, and aorta as they extend through the pericardium. All blood should be removed before weighing. One important weight to be mentioned here is that normal cat, raccoon, woodchuck, and even fox hearts do not weigh more than 17 grams.

## OPENING THE HEART

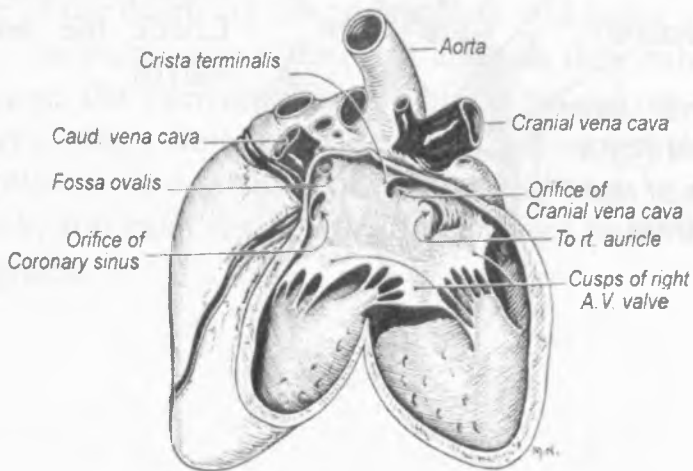
To open the right ventricle, hold the heart in your left hand, with the left side of heart towards you. Make the incision, starting at the pulmonary trunk, into the right ventricle, close to the inter-ventricular septum.



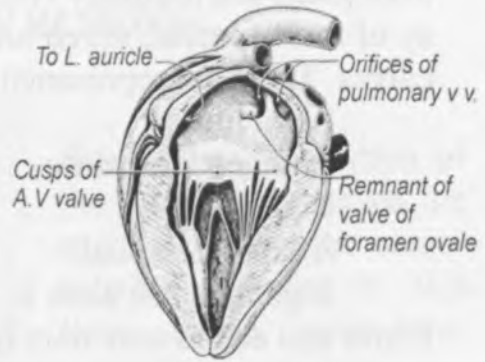
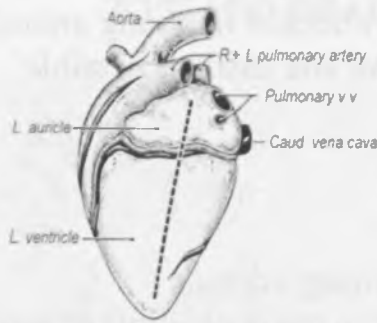
Open the pulmonary trunk past bifurcation. Check the semilunar valves.



Turn the heart over with its right side towards you. Continue the incision, following the interventricular septum, into the right atrium.



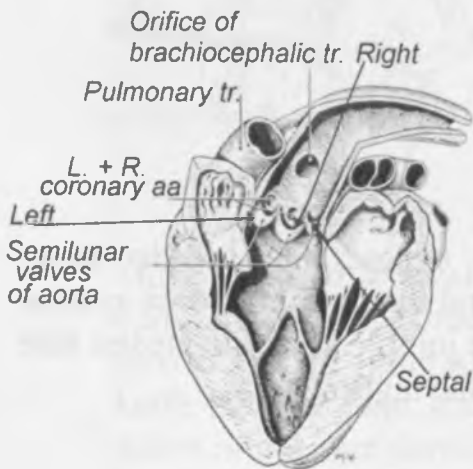
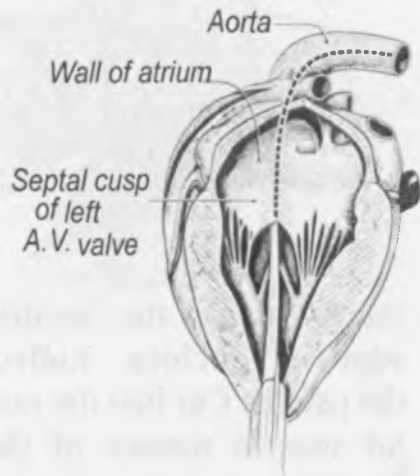
Open the right ventricle and atrium. Check the right atrioventricular valve, the orifices of the cranial vena cava, the caudal vena cava, the fossa ovalis, and the coronary sinus.



Open the left atrium and ventricle with a straight incision. Incise through the parietal cusp of the left atrioventricular valve.

Check the left atrioventricular valve and openings to the pulmonary veins.

To open the aorta, insert the knife under the septal cusp of the left atrioventricular valve. Incise through the wall of the atrium, out and down the aorta.



Check the semilunar valves of the aorta, orifices, and right and left coronary arteries, orifice of the brachiocephalic trunk. Make multiple inspection slices through the organ. Open the abdominal aorta and its major branches (mesenterics, iliacs, etc.).

## JOINT EXAMINATION

Five joints are routinely checked, in the order given, in the necropsy of most species; seven are checked in young animals, especially calves. These are representative and easily accessible.

Right hip

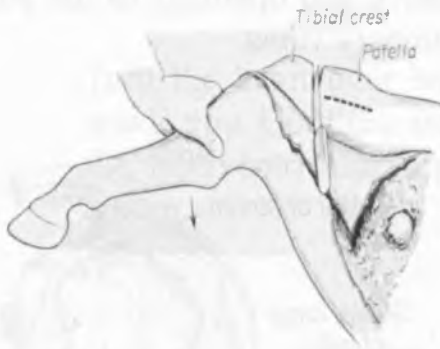
Right and left stifle

Right shoulder

Atlanto-occipital

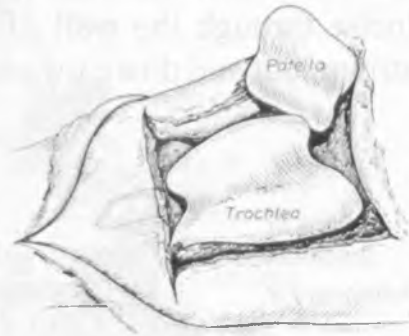
Right and left hock in young animals

Fibrin and debris may only be in the hock joints of septicemic animals. Tiny bits of fibrin may also be seen normally in the hock joints of newborn calves, causing some interpretation problem.



To expose the stifle joints, reflect the skin, bend the joint, and cut the patellar ligament 1/3 of the way up from the tibial tuberosity.

Incise along the medial edge of trochlea. Reflect the patella. Cut into the major muscle masses of the limbs.



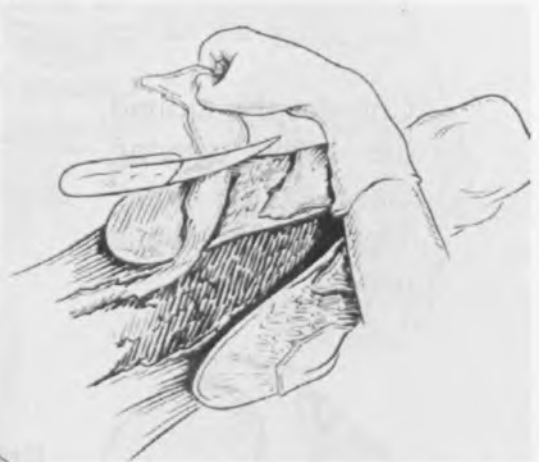
To expose the shoulder joint, bend the forelimb down to raise the joint. Cut on the medial side at the highest point.

## EYE AND BRAIN REMOVAL



Skin the major portion of the head and remove the ears. Examine the major salivary glands at this time. Leave some skin around the eyes.

Prop the head up (hoist, knee, or assistant) to skin the left (down) side of the head.



Move the head to locate the atlanto-occipital joint. Obtain CSF at this time, if required, from a dorsal or ventral approach. Cut all of the soft tissues around the joint. Insert the knife into the joint and transect the spinal cord and ligaments of the joint dorsally and ventrally. Do not direct knife into brain proper. Remove the head.



**Note:** Due to the apparent rapid autolysis of the retina, it is often recommended that eyeballs be removed and fixed before starting the rest of the necropsy.



To remove an eye:

Grasp, with minimum traction, the skin which has been left around the eye. With the belly of the knife, cut the soft but tough tissues around the orbit.

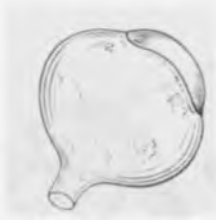
Cut deeply around the orbit, staying close to the bone. Transect the optic nerve.



Remove the eye, leaving the optic nerve longer on the left eye for easy identification.

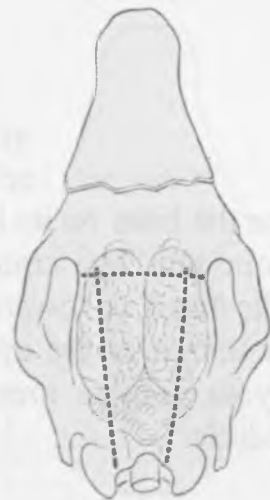
Pull the soft tissue back and forth under the knife blade while holding the back of the knife gently against the eyeball proper. Use the carcass as a cutting board.





For proper fixation, fix the globe, free of surrounding tissue, *in toto*. Some ocular pathologists prefer Bouin's or Zenker's, but due to their mercury content and biohazard activity, formalin is satisfactory if the eyes are fixed early.

To the right is a diagram showing the location of the brain in a dorsal view of the skull. Dotted lines represent the lines of incision. Remove the major muscle masses from inside the area of dotted lines. Look into the foramen magnum to note the normal absence of the cerebellar vermis. Suspect a brain lesion if seen (prolapsed).



WP 20190, 20412, 20414

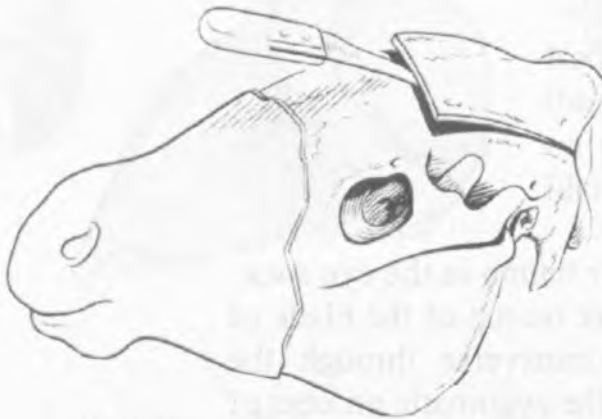


Hold the head with your thumb in the eye socket, and your index finger on top of the blade of the saw. One cut is transverse through the frontal bone, caudal to the zygomatic process of frontal bone.

Place the head on its right side. Another cut is sagittal, just medial to left occipital condyle.



Place the head on its left side for one cut. The cranial part of the head is towards you, your thumb in the eye socket, your fingers around the mandible.



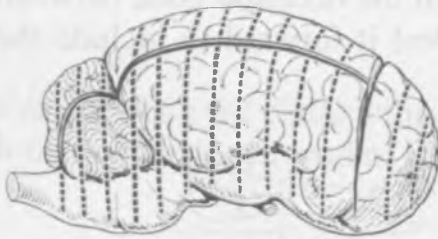
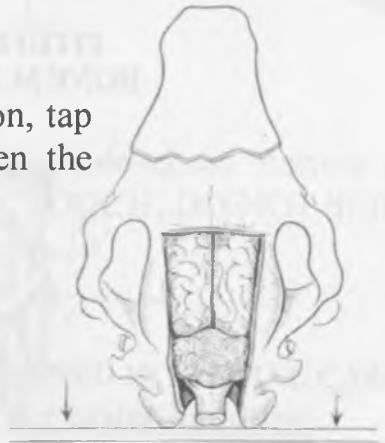
Pry up the skull cap. Use a stronger prying instrument than a knife if the bone was not sawn free. Remove. Sinuses, nasal cavities, and cranial vault should be examined now.



Check to see that the tentorium cerebelli is removed as well as other limiting dura. The arrow points to its location.

With the head in upright position, tap it lightly on the table to loosen the brain.

Cut the olfactory peduncles, internal carotid arteries, and cranial nerves as the brain is removed. Tilt the head so that the brain will rest on the table.



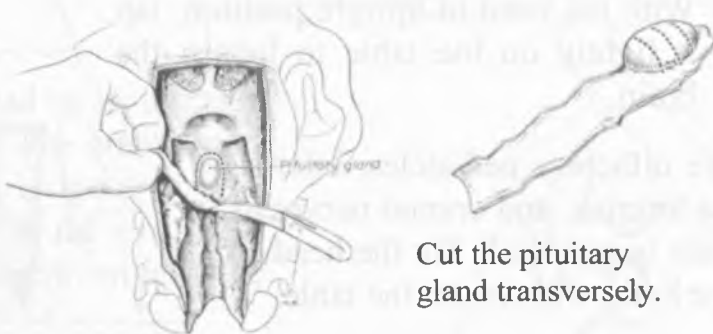
If the brain is not to be kept, cut 1 cm transverse sections for inspection.

**Note:** It is normal to find a single white firm  $\frac{1}{2}$ –1 cm white tapered conical mass hanging from the skull cap midline attached to meninges. This is the pineal gland. In many animals, two reddish soft masses may be found hanging in a similar position or slightly forward, and careful manipulation will show them to be the normal roughly triangular choroid plexi from the lateral ventricles. WP 11086, 20423

A transverse saw cut through the skull at the level of the middle of the eye sockets will cut through the ethmoid turbinates to check for progressive ethmoid hematoma in the horse and parasites in many species.

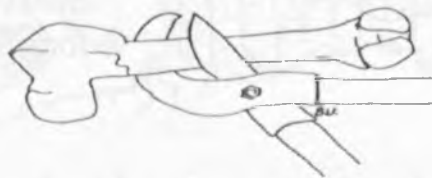
A transverse saw cut through the snout at the level of the commissures of the lips is needed to check for atrophic rhinitis in pigs.

### PITUITARY REMOVAL AND BONE MARROW EXAMINATION



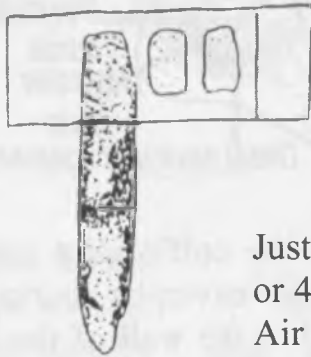
Cut the pituitary gland transversely.

To remove the pituitary gland: Pick up the dura from the basilar part of the occipital bone between the sawn condyles. Peel it forward to include the pituitary.



To make a bone marrow impression smear or obtain a section of marrow, crack open almost any large bone of young or small animals using the rib cutters to **obliquely** crack the bone. In large animals the bones must be sawn to collect marrow.

If the area of bone marrow or other tissues used for touch preparation are too bloody, then touch the surfaces first with absorbent paper towel and then make the touch preparation. The remaining core can be put in formalin for fixation.

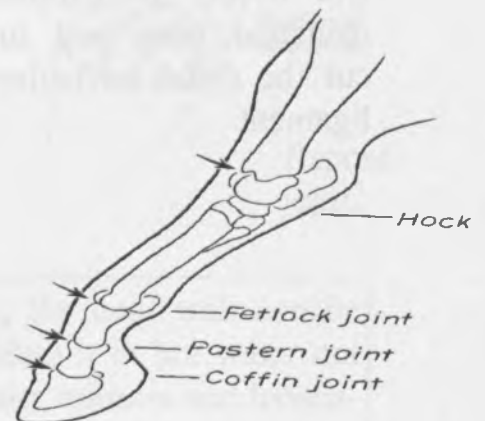


Just touch the glass slide to red marrow at 3 or 4 contact areas. **TOUCH, DO NOT SLIDE.** Air dry the slide.

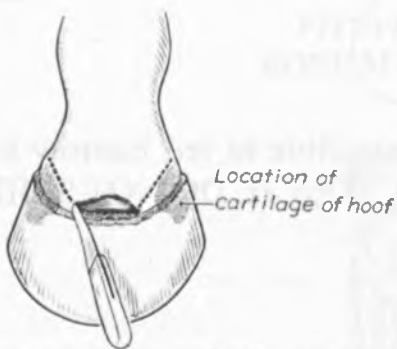
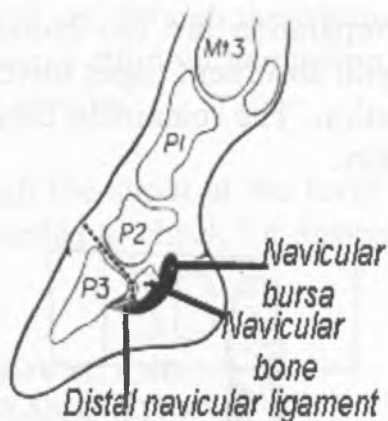
**LIGAMENTS, TENDONS, JOINTS EXAMINED IN SPECIAL CASES**  
**Leave the leg attached to the carcass for easier handling**  
**(another third hand technique suggestion and very useful).**

Prior to checking the distal joints on the limb, skin the limb to include removal of the coronary band.

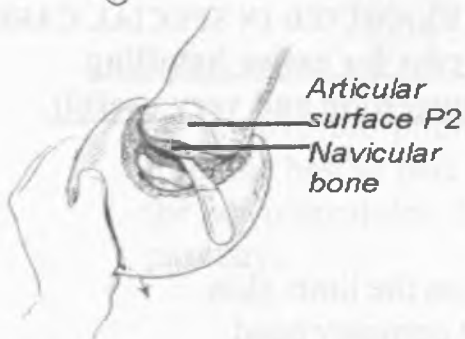
To check the joints on the hind limb distal to stifle:  
 For reasons of leverage, start distally at the coffin joint, then pastern, fetlock, and tibiotarsal articulation of the hock.



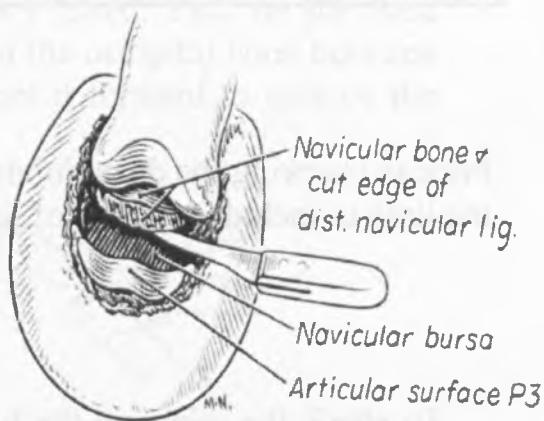
To the right is a diagram of a cross section showing the depth of the incision at the coffin joint.



To open the coffin joint and examine the navicular bursa, cut proximal to the wall of the hoof, first dorsally, then on the medial and lateral sides, deep to the cartilage of the hoof.



Cut deeply to expose navicular bone and to cut the distal navicular ligament.



Raise navicular bone to expose the bursa.

Open the pastern joint with incision dorsally.



Expose joint.



Open the fetlock with incision dorsally and up one side.



Expose joint.

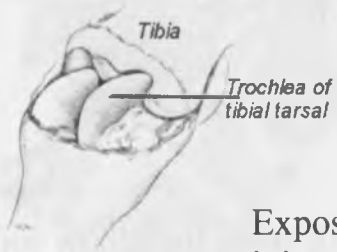


Proximal sesamoids

Expose joint.



To expose the tibiotarsal articulation of hock, cut dorsally into the joint at distal end of the tibia.



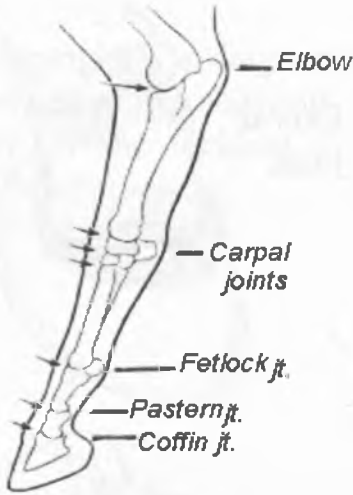
Tibia

Trochlea of tibial tarsal

Expose joint.

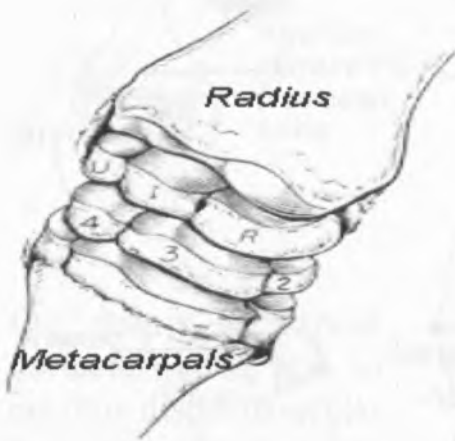
**Note:** In the horse and cow, the hoof wall can be more easily removed by holding it in hot water not more than 65 degrees for twenty minutes and loosening it with a rod and prying off the wall.



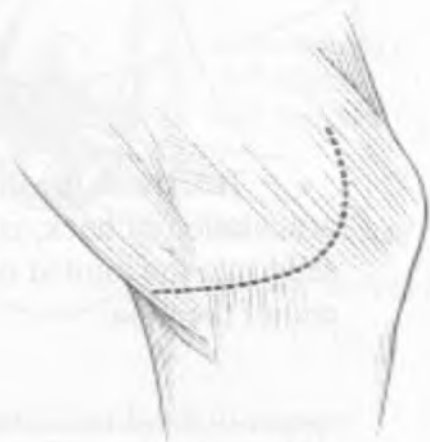


To check the joints of the forelimb distal to the shoulder, start at the most distal joint. Skin the limb. For necessary leverage, start at the coffin joint. Procedures for opening the coffin, pastern, and fetlock joints are the same as described for the hind limb.

To open the carpal joints, make three incisions: at the distal end of radius, between the rows of carpal bones, and at the proximal end of the metacarpals.

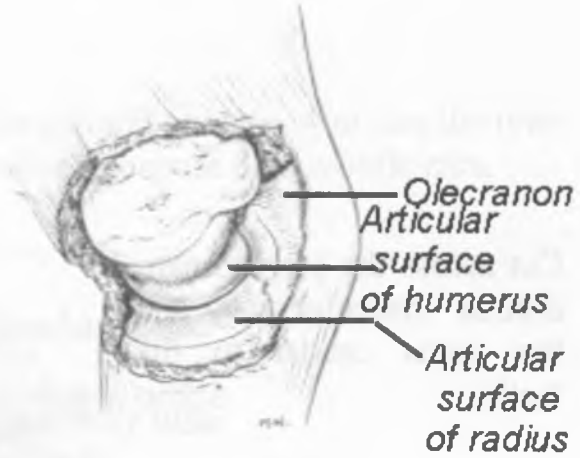


Expose joint.



To open the elbow joint, go in from the medial side, and incise around the distal end of the humerus.

Expose joint.



### TO REMOVE MANDIBLE

With the dorsal surface of the head on the table, cut all of the muscles on the medial and lateral sides of both mandibles from the cranial or caudal (as shown) position.

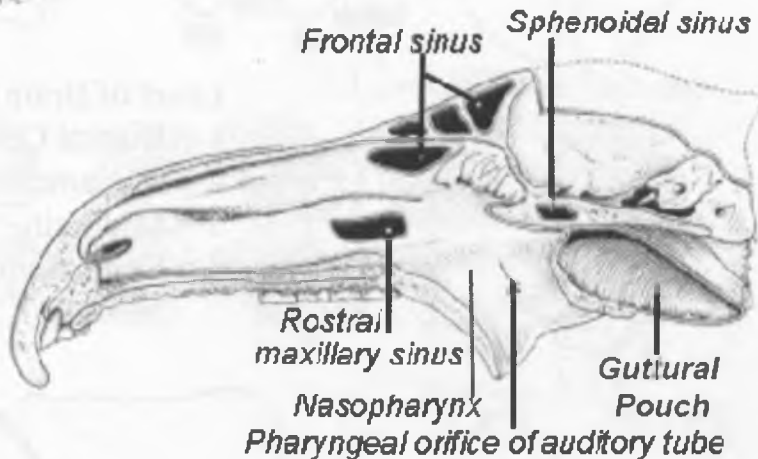
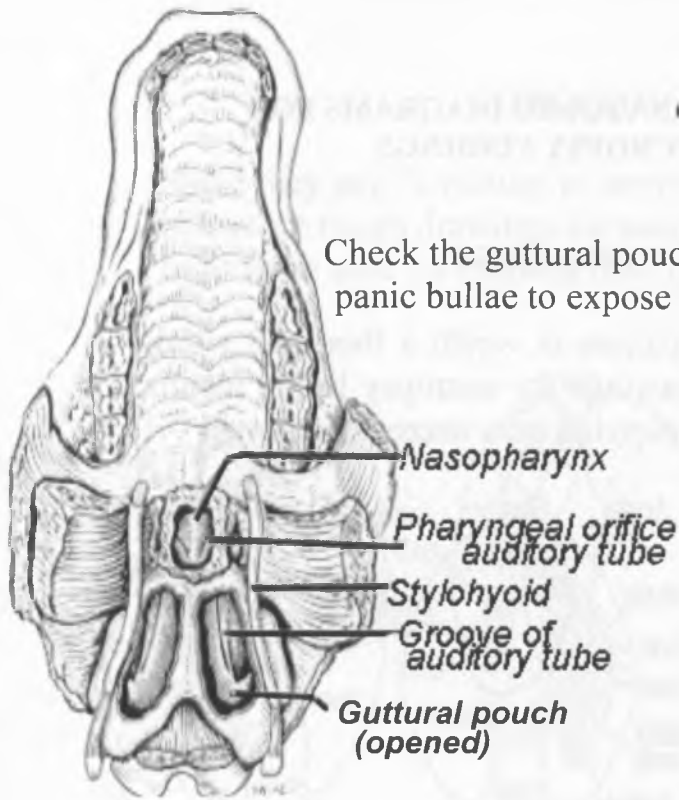


Cut across the pterygomandibular muscular fold and the gums caudal to the teeth.



Pull back and free the lower jaw. This is very difficult in large animals if ALL soft tissues are not cut first.

# GUTTURAL POUCH EXAMINATION

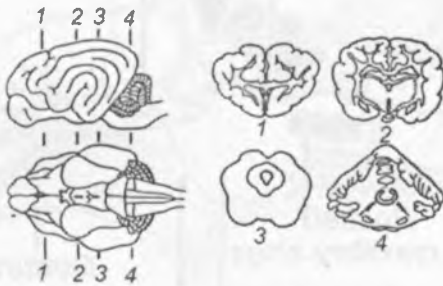
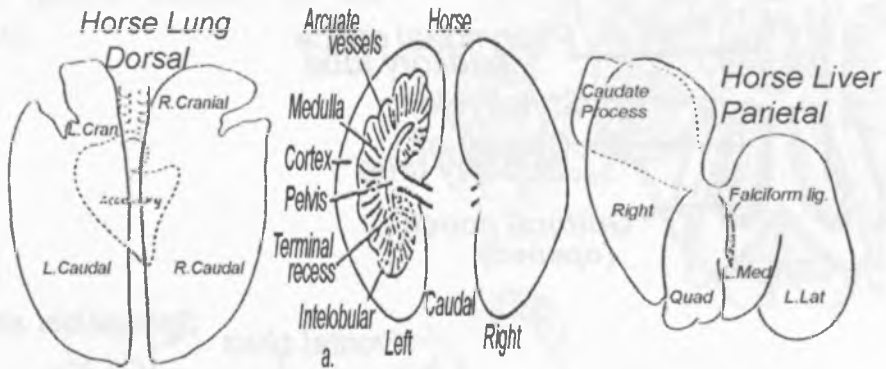


Saw the head in two sagittally. Avoid the front teeth (they are hard on saw blades). Remove the nasal septum to check the nasal sinuses. Another transverse saw cut, cranial to the premolars, will eliminate cutting any teeth. The stylohyoid bone articulates dorsally at the tympanohyoid articulation, and must be examined in horses for the initiation lesion of arthritis-associated guttural pouch mycosis.

# NORMAL ANATOMIC DIAGRAMS FOR NECROPSY FINDINGS

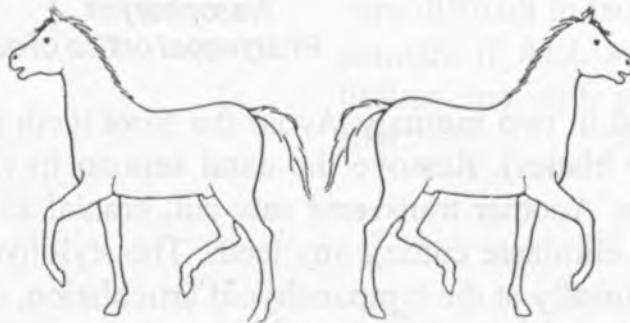
## THE HORSE

Since they say "a picture is worth a thousand words," below are rough drawings for necropsy lesion localizations to be used for copying onto necropsy reports.



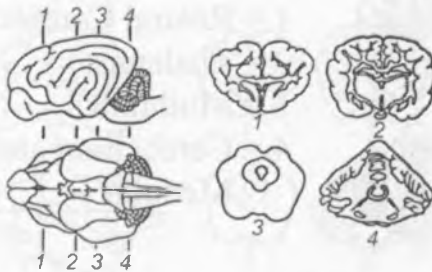
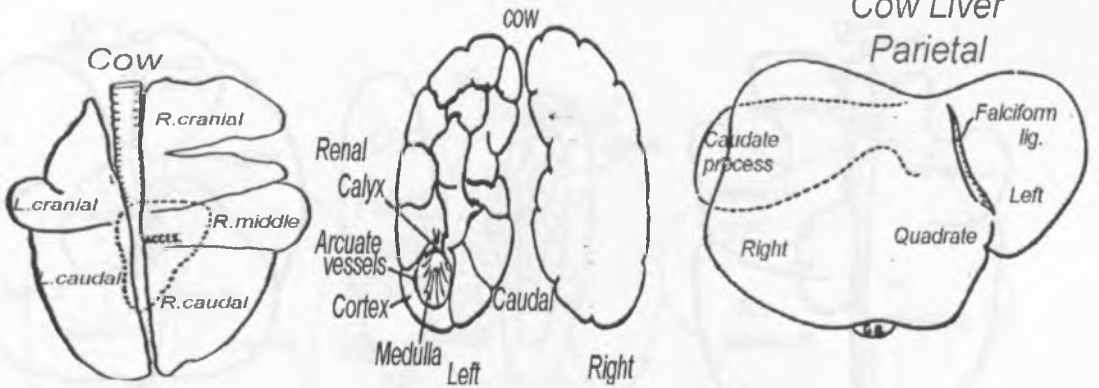
### Level of Brain Sections

- 1 = Rostral Cerebrum
- 2 = Thalamus
- 3 = Midbrain
- 4 = Cerebellum and Medulla



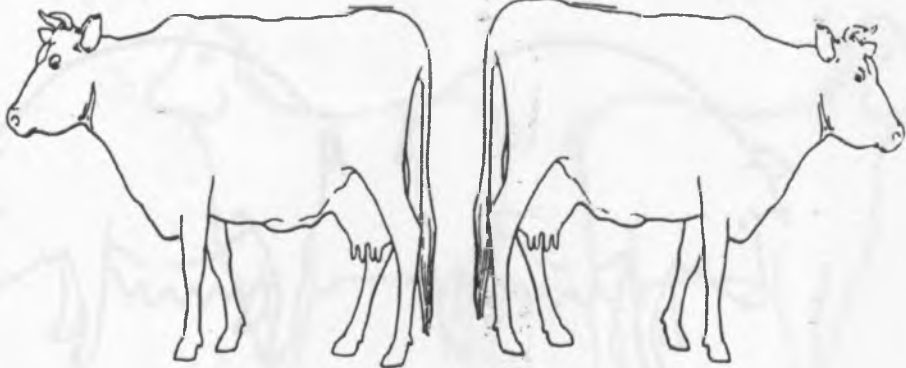
# THE COW

Since they say “a picture is worth a thousand words,” below are rough drawings for necropsy lesion localizations to be used for copying onto necropsy reports.



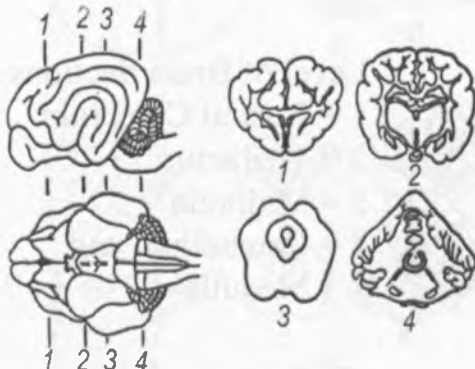
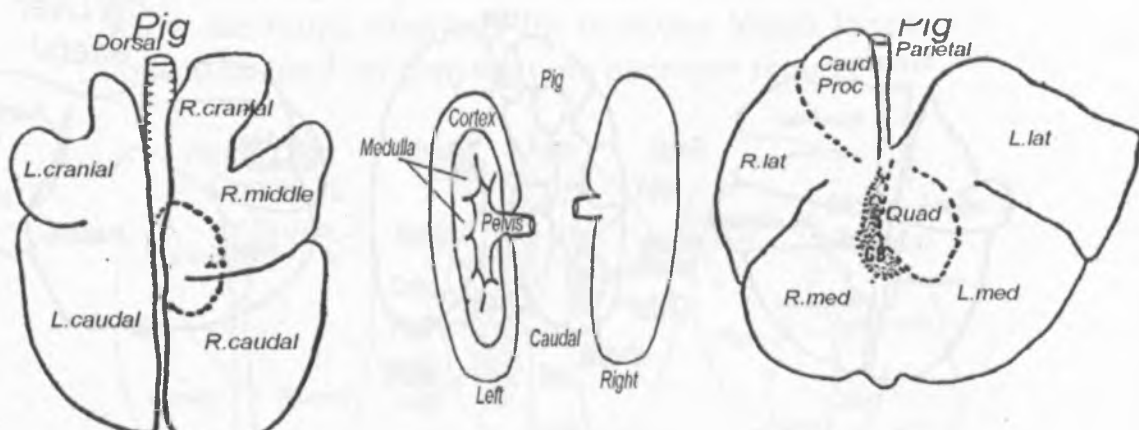
## Level of Brain Sections

- 1 = Rostral Cerebrum
- 2 = Thalamus
- 3 = Midbrain
- 4 = Cerebellum and Medulla



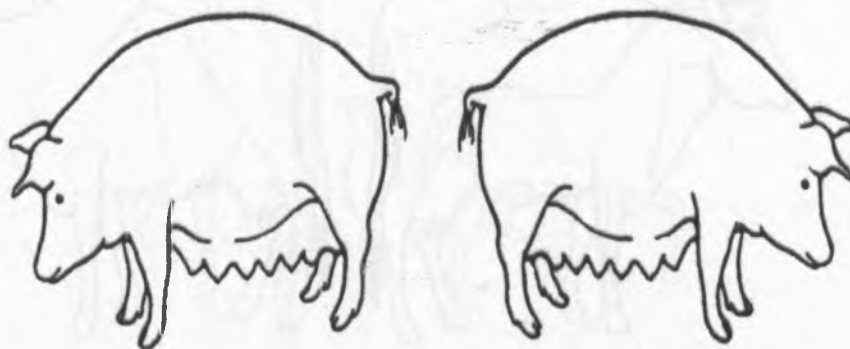
## THE PIG

Since they say "a picture is worth a thousand words," below are rough drawings for necropsy lesion localizations to be used for copying onto necropsy reports.



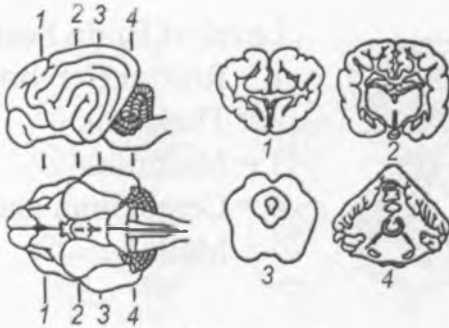
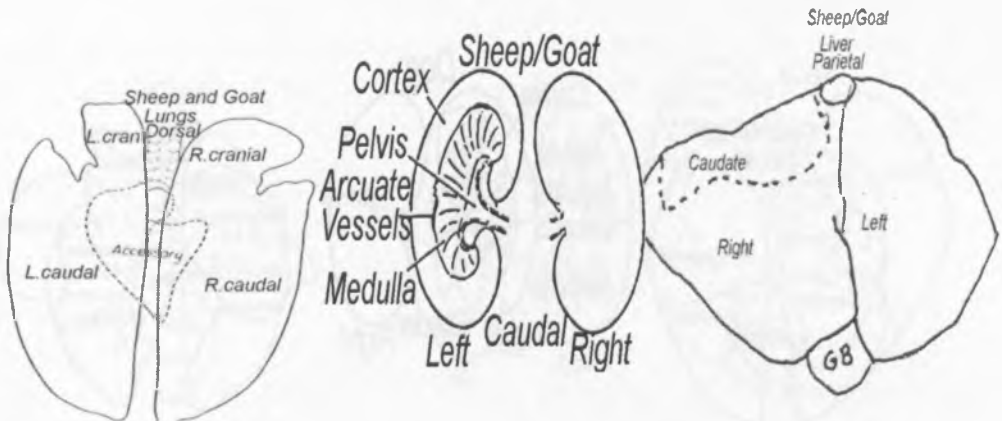
### Level of Brain Sections

- 1 = Rostral Cerebrum
- 2 = Thalamus
- 3 = Midbrain
- 4 = Cerebellum and Medulla



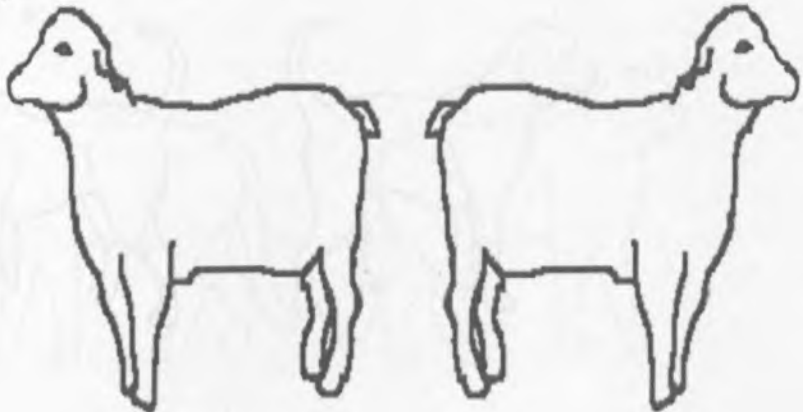
## THE SHEEP AND GOAT

Since they say “a picture is worth a thousand words,” below are rough drawings for necropsy lesion localizations to be used for copying onto necropsy reports.



### Level of Brain Sections

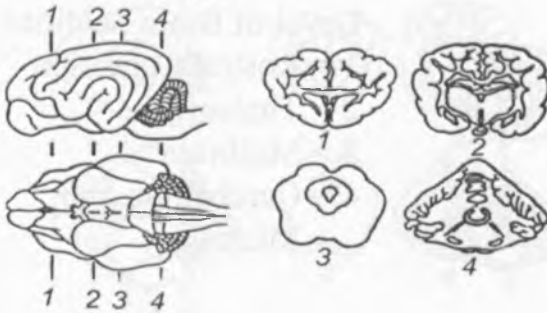
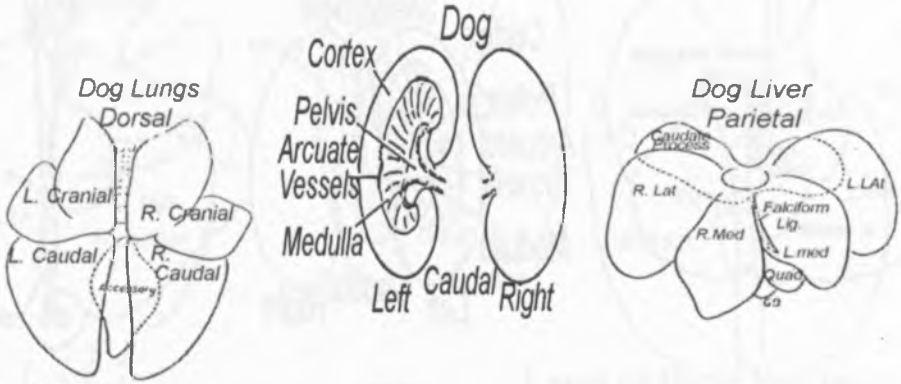
- 1 = Rostral Cerebrum
- 2 = Thalamus
- 3 = Midbrain
- 4 = Cerebellum and Medulla





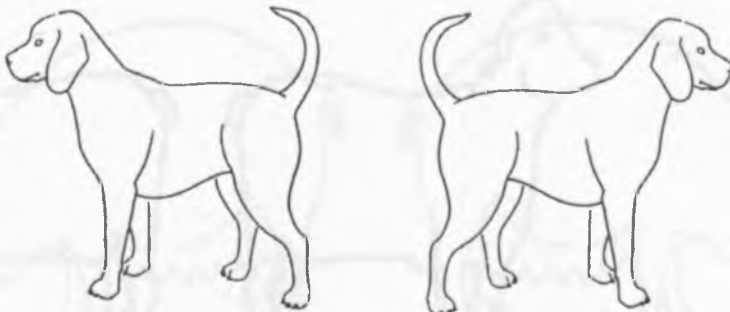
## THE DOG

Since they say "a picture is worth a thousand words," below are rough drawings for necropsy lesion localizations to be used for copying onto necropsy reports.



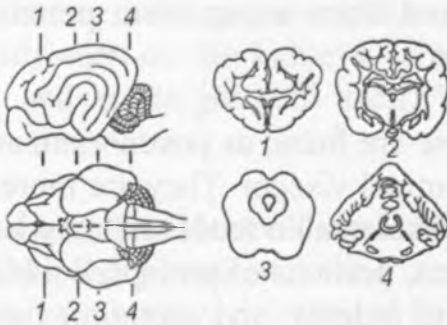
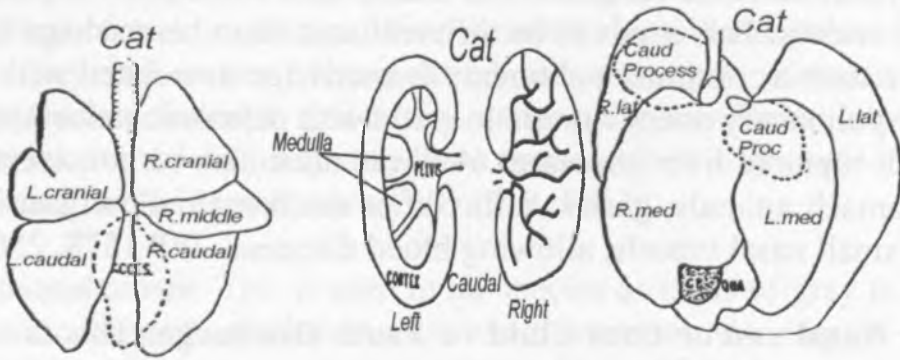
### Level of Brain Sections

- 1 = Rostral Cerebrum
- 2 = Thalamus
- 3 = Midbrain
- 4 = Cerebellum and Medulla



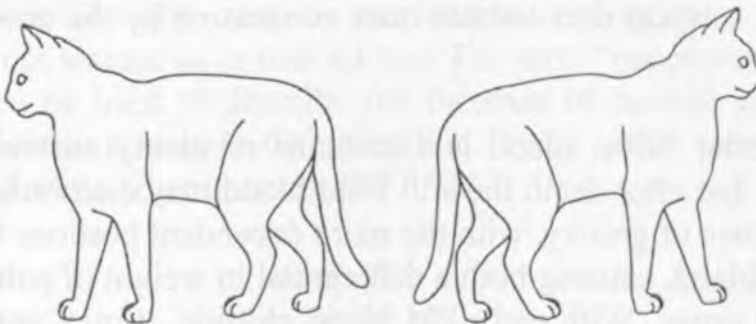
## THE CAT

Since they say “a picture is worth a thousand words,” below are rough drawings for necropsy lesion localizations to be used for copying onto necropsy reports.



### Level of Brain Sections

- 1 = Rostral Cerebrum
- 2 = Thalamus
- 3 = Midbrain
- 4 = Cerebellum and Medulla



## COMMON FINDINGS OF LITTLE OR NO DIAGNOSTIC IMPORTANCE

This section is to introduce a small but quite important part of changes of little or no diagnostic importance, but with their common occurrence and easily misinterpreted significance. They should be recognized.

### General and External Findings

**Bloody Nasal Discharge:** This is a common postmortem (PM) artifact, usually due to nasal congestion at death, with subsequent rupture of congested vessels. This needs to be differentiated from hemorrhage from nasal lesions such as: tumors, pulmonary hemorrhage associated with pneumonia, or pulmonary artery rupture in cattle with pulmonic abscesses associated with ruptured liver abscesses. Anthrax must also be considered. In single stomach animals, gastric reflux after death may allow gastric fluid to erode small nasal vessels, allowing blood dispersal. WP 1578, 2507

**Clear Nasal and/or Oral Fluid or Froth Discharge:** This is a common finding, even early after death. This may be seen as a common terminal finding, and must be differentiated from antemortem pulmonary edema from pathologic causes. WP 16106

**Rectal or Vaginal Prolapse:** These are found as postmortem artifacts, usually due to gas distention of abdominal viscera. They are more likely to be seen earlier in animals on highly fermentable feeds and may be easily confused with antemortem bloat. Often, portions of prolapsed tissues are eaten by scavenger rats, dogs, birds, and rodents; and sometimes so cleanly removed that surgical removal has been considered by the unwary prosector. WP 8078

**Livor Mortis:** Alive, blood is distributed relatively normally throughout our bodies; but after death the still fluid blood may distribute itself according to the laws of gravity, with the more dependent portions being suffused with more blood, causing both a differential in weight of paired organs and a more red organ. With early PM blood clotting, it may not be so readily apparent, if present at all, as the clots prevent redistribution. Some external

tissues, such as the pale skin of pigs, show distinct patterns on the surface, mimicking the variations of the surface on which it died. The same variations may be seen on the liver, lungs, heart, and kidneys where ribs or adjacent organs may be pressed against them, causing the area to be paler than surrounding areas. WP 16870

**Melanosis:** This is dark-gray to black normal melanotic pigmentation of various tissues. It is commonly seen in the pulmonary artery and aorta of sheep; and brain, meninges, adrenals, uterus, trachea, kidneys, oral cavity, and esophagus of most other species. It may diffusely discolor a tissue, which may cause it to be confused with melanoma. WP 2719, 16135

**Pseudomelanosis:** This is seen in all species as areas of gray to black discoloration of the tissues due to postmortem decomposition of blood by bacterial action forming iron sulfide. It can occur soon after death in some tissues, such as the gut. It is most commonly seen on the kidneys, liver, or spleen in contact with the gut, and in the gut wall itself. WP 11330, 840, 867, 2816, 2902

**Pseudoicterus of Horses:** Pseudoicterus is a relative increase of normal blood pigments including carotene. The tissues of young horse, and almost any dehydrated horse, tend to be more yellow than normal. The skin, mucous membranes, and articular cartilages are not stained as in true icterus. The term "pseudoicterus" could even be used to describe the increase of normal yellow color of certain tissues of Guernsey and Jersey cattle, most primates, and some other animals. WP 1377

**Normal Lymph Nodes of Young Animals:** An inexperienced prosector often falsely considers the large normal nodes to be enlarged; this applies especially to the nodes of the GI tract of calves. WP 13106

**Hemal Nodes:** These are normal structures, somewhat similar to lymph nodes, found only in ruminants. They are dark red, as they consist of lymphatic tissue with blood-filled sinuses. They are scattered in the peritoneal and thoracic cavities, mainly along the dorsal mediastinum and dorsal abdominal mesenteric fat. WP 16105

**Injection Sites:** Tan, dry-appearing material with fluid and fibrin in the pleura or heart sac having a medicinal (alcohol) odor maybe from a barbiturate or other euthanasia solution. A greenish or tan watery focus surrounded by dry-appearing heart muscle may indicate an intracardiac injection. Again, the odor may help here. The heart blood itself may be granular, clumped, and often brownish with other euthanasia chemicals also. WP 6162

**Thoracic Inlet Pseudomalignant Edema:** Most common in cattle but to a lesser degree in pigs, sheep, and horses, one may find moderate to marked edema and emphysema at the thoracic inlet, neck, and cranial shoulder region in moderately to severe decomposing carcasses. One may find on smear and culture, many *Clostridia* sp. and the tissue may have a slightly sweet odor of blackleg or malignant edema. This is almost always a postmortem change. It is often misdiagnosed as one of the clostridial diseases.

**Juvenile Udder of Calves:** Often, newborn or young calves have a very discrete mass of red mottled tissue in the region of the udder that would have become the gland proper. It may be well-formed and 10 centimeters or more in length. This juvenile precocious udder development manifests in animals under hormonal control *in utero*. WP 12574

**Focal or Multifocal Pericardial or Peritoneal Loose Fibrovascular Tissue:** These areas of red to tan, round or partially linear islands of connective tissue are very common, scattered on the epicardium of the atria and the pericardium at the base of the heart, in the horse and the ox. Similar lesions are common on the mesentery and omentum. The foci consist almost entirely of loose, highly vascular connective tissue without inflammatory cells.

Although a very common finding in large animals, the underlying cause is unknown. Some believe friction is the cause. It is almost never significant, but in some animals this fibrovascular granulation is the suspected source of blood, causing some cases of cardiac tamponade. WP 19892

**Central Nervous Tissue Venous Embolization:** These white pieces or elongated, 10–30 cm strips of central nervous tissue mixed with blood can be found in many major veins of the heart, lung, kidney, and liver as the result of high-pressure air injection used at the time of captive bolt pistol use in slaughterhouses. The air injection at low pressure is not prone to cause this, but higher pressure may. It allows CNS tissue to get into the food chain and should be considered a possible mode of transmission of BSE to humans. WP 3366, 3367, 3420

**Blood-Filled Organs:** At death many body organs may appear to have more blood in them than expected, especially the lung and the liver. In fact, however, it is often the result of muscle contraction of rigor pushing blood centrally to the body from the more peripheral tissues such as muscles. It is a very common postmortem finding when no other pathological or physiological cause is found.

## Spleen

**Splenic Capsule Defects:** These are seen mainly in the horse and appear as deep depressions, slits, or folds in the parietal surface of the spleen—primarily in its middle third. The deeper involved surfaces of the defect are similar to the remaining normal splenic surface. These may also be seen in pigs, cats, mink, and others. These splenic fold defects are often mistaken for healed traumatic lesions. WP 12905

**Extruded Red Pulp:** A few to numerous red 1–3 mm nodules of extruded red pulp through the capsules are commonly found in the foal and pig. They are often on the edge of the spleen but may be on any or all surfaces and even through the wall of the splenic vein. They are covered by the overlying serosa or endothelium. This is an anatomical variation of no significance. WP 13075

**Anesthetic Spleen:** These are moderately (2x to 20x) or more to enormously enlarged spleens which are very soft; when cut, blood flows freely from them. Anesthetic agents, especially barbiturates in euthanasia solutions, cause this condition. It would be hard to differentiate this from acute torsion, but the easily flowing blood differentiates it from the splenomegaly of chronic bacterial infection, protozoal infection, passive congestion from vascular impairment, and diffuse splenic neoplasia. The anesthetic-enlarged spleens can be seen with other chemical agents like chloroform. WP 13136

**Pseudoinfarcts of the Spleen (Unequal Expulsion of Blood):** These are irregular focal areas of swollen spleen, often with a straight edge; usually along the free edges of the spleen, and are usually in the ventral half of the spleen. They are red or dark red, being filled with blood, and most are soft. They result from blood being trapped in these sites while the blood from the rest of the spleen was differentially expelled by splenic contractions. They are commonly seen in animals, mostly dogs, when euthanized. Most of these splenic pseudoinfarcts are 2–10 mm, but they may involve almost half the spleen. These comprise almost 50% of irregular masses found in the dog's spleen. Most other splenic masses would have a more or less rounded outline and not be found so commonly on the edges. Real infarcts can be confusing, but a source for them should be found, as infarcts don't arise *de novo*. WP 505



## LIVER AND GALL BLADDER

**Tension Lipidosis:** These are usually yellow or pale foci in the liver near the edges, or any part of the liver in which there is connective tissue attachment. They have straight, well-defined edges, and extend as deep into the parenchyma as they are wide. They are common in horses, less so in the ox, and rare in other species. They have been called pseudoinfarcts and are thought to occur when the liver gets enlarged or when for some other reason there is tension on the capsule and stroma, including associated vessels causing vascular compromise and anoxia with subsequent fatty change. Another form of this lesion commonly seen in cattle are multiple yellow or pale irregular liver foci, 1–3 cm, mostly on the ventral visceral surface, with each focus having a thin-walled vessel opening to it from the liver itself. These are called “vascular-related hepatic lipidosis,” and are suspected of being due to a localized reversal of blood flow to the surface connective tissue. WP 296, 300

**Capsular Fibrosis:** Perihepatitis filamentosa. This occurs in two forms in the horse: a large plaque of connective tissue up to 20 cm or more on the diaphragmatic surface of the liver; or as a few large numbers of 1 mm by 5 mm long, fine connective tissue tags attached also to the parietal surface of the liver. In neither case do these go deeper than the capsule. The large plaque-like capsular fibrosis often has a corresponding similar lesion on the adjacent diaphragmatic serosa, but not always. Often, both the multiple single tags

and plaques may be found in one horse. They are also seen in newborn foals. They may be related to strongyle larval migration, but this is not proven nor is its supposed inflammatory nature seen.

Similar lesions can occasionally be seen in the cow. Another suspected cause is thought to be chronic peritonitis with capillary action adhesion and subsequent resolution with fibrosis. WP 83

**Gall Bladder Anomalies:** Anomalies such as: bi- or tri-lobed gall bladders, buried bladders, extremely tortuous, or other such findings are considered to be within the limits of normal variation for cats. WP 574

**Gall Bladder Enlargement:** Variation in gall bladder size due to the amount of bile present is considered normal in most species but extreme enlargement up to the size of half the liver itself or larger may indicate biliary obstruction. Without a cause for obstruction, a physiologic failure to empty is most often associated with inanition for whatever reason such as: dental problems, starvation, malnutrition, etc. WP 10101

As the condition for not eating becomes more chronic, the content often becomes more watery, and thus less viscid. Massive distension is most common in calves without obstruction.

**Prominent Liver Patterns:** Often seen in the cat and horse, the normal pattern, slightly accentuated, is commonly mistaken for chronic passive congestion (nutmeg liver). WP 1523

## ENDOCRINE

**Pseudocystic Adrenal Glands:** Most species often seem to have a central cavity, even containing blood, at necropsy. Almost without exception it is an artifact caused by rough pulling on the surrounding soft tissues during the necropsy.

**Adrenal Hemorrhage and Congestion:** This is a common finding mostly in large animals such as the horse and cow, and likewise in the young of those species. It is considered an artifact most of the time due to the terminal increased peripheral resistance to blood flow that corresponds with dying, so that central pressure increases, causing such hemorrhage agonally in this organ. It can also be evidence of septicemia, in which case there would be other lesions found. WP 11041

**Extra-Cortical Adrenal Nodules:** Mainly in the horse, but also in the dog and other species, one to many pale-yellow to tan, round, 1–5 mm nodules may be found bulging through the adrenal capsule. On section, they are similar to the adrenal cortex itself. They are often found in foals. There is no known cause, but they may represent developmental variation; no clinical disease is related to them.

WP 11138

**Thyroid Cysts:** These cysts, up to 5 mm in diameter, are filled with gray to yellow greasy material in one or both lobes of the thyroid of sheep and goats. They are thyroglossal duct cysts lined with respiratory epithelium or cuboidal cells. WP 11047

## RENAL

**Pale Kidneys in the Cat:** Cat and rabbit kidneys may be pale tan or even light yellow rather than the usual tan to red-brown as in most species. The cortical vessels are prominent and lie in grooves in the subcapsular surface. These two conditions are normal. Oddly enough, the hyena has a similar external renal appearance normally. WP 560

**Renal Pelvic Mucus in the Horse and Pig:** The horse and pig have mucous glands in the renal pelvis, hence it is usual to find thick tenacious yellow mucus here. WP 11752, 129, 130

**Pulpy Kidneys:** In this autolytic change seen mostly in sheep, the cortices of both kidneys are soft and mushy while the medullary tissue is much less affected and may look normal grossly. The same change can be seen in the horse and sometimes in the ox given glucose. It is probably due to high glucose and glycogen levels in the cortical tubules supplying the substrate for enhanced autolytic change. It is a well accepted procedure to test the urine for sugar in sheep, and it is often positive in cases of enterotoxemia. WP 971, 11786

**Renal Capsule Adhesions:** In spite of the many described instances in the literature and in textbooks, rarely is the renal capsule truly adherent. In most cases, failure to remove the capsule cleanly is the fault of the prosector. In a dehydrated animal, a thin layer of the cortex may remain attached to the capsule when it is stripped from the kidney. Normal vessels, which extend from the cortex to the capsule, can cause some difficulty in capsule removal, especially in the horse. Nephrologists may consider these normal vessels as parasites when first questioned. WP 16256

**Umbilical Arteries:** These are two thick vessels that extend from the abdominal aorta alongside the urinary bladder to the umbilicus. Normally, in newborns these vessels have varying degrees of hemorrhage of their walls and adventitia following rupture at birth. These vessels constrict at birth and the trapped blood then clots. Postnatally they become the round ligaments of the bladder. WP 225

**Thickened Urinary Bladder Wall:** The thickness of the bladder wall is difficult to interpret due to normal physiologic variation. In normal animals with an empty contracted bladder, the wall is frequently startlingly thick. It may be physiologically hypertrophied in cases of prolonged incontinence. The fully contracted bladder might be misinterpreted as a tumor because it is a firm rounded mass, as one usually thinks of a bladder as an organ distended with urine. WP 7553

**Urinary Sediment:** A relatively large quantity of yellow-orange, dry, putty-like material in the urinary bladder of normal horses is often erroneously considered to be pus. Inflammation is not associated with it, and by careful observation it can be determined to be in the gravitational bottom of the urinary bladder. It is a normal finding, especially in horses with limited access to water. It is made up of urinary waste, salts, and mucus from the renal pelvic glands. Similar material is commonly seen in rabbits and guinea pigs. It is seen in both sexes, but is more common in males. WP 129

## **RESPIRATORY**

**Gastric Contents in Nasal Cavity:** Most of the time, finding ingesta in the nasal cavity, pharynx, or larynx probably indicates a terminal or postmortem event. It is difficult to evaluate unless the animal was under observation at death. A ruminant can partially eructate a bolus of food at death with subsequent propulsion of it up to the pharynx and nasal cavities by the pressure of rumen gas after death. WP 2529

**Pharyngeal Lymphoid Hyperplasia:** This is a common finding in young adult horses and recognized by the irregularity of the pharyngeal mucosa, caused by numerous 2–3 mm, smooth nodules on the caudal dorsal surface of the tongue and the pharynx around the epiglottis. WP 12947

**Gastric Contents in Trachea and Lungs:** This almost always is a post-mortem change, especially in single stomach animals, and may be due to some abdominal gas pushing contents up the esophagus. Some cases may be due to postmortem handling of the carcass. It is necessary to see a tissue reaction to make this finding an antemortem lesion. If aspiration occurred as a terminal event, evaluation is more difficult. WP 11278

**Tracheal Froth:** Certainly this may be a lesion in some cases, but marked tracheal froth production, even with froth flowing from the nostrils, may be seen in euthanized animals that don't have a history of dyspnea or respiratory distress. In these cases and others in which there is no apparent physiologic explanation for froth formation, its significance is suspect. In intentionally electrocuted animals, the electric shock is given for several minutes. During the electrocution, the body is in extensor rigidity with stopped cardiorespiratory movement. The elastic recoil of the vessels continues and pushes serum from the pulmonary capillary bed into the alveoli. Apparently on cessation of the electric shock, the carcass relaxes, and the inflated lungs deflate, pushing pulmonary air and serum out mixing it to a froth as it escapes. This results in the tracheobronchial froth which, with time, can extend out the nostrils. We can reproduce this in many euthanasia animals and is not to be confused with the clinically significant interstitial pulmonary edema. WP 16106

**Pleural Fibrosis:** This is the presence of large areas of pale connective tissue on the surface of the dorsal part of the caudal lobes of the lungs in most species. It is most prominent in the cow and sheep; and it is especially pronounced in uninflated fetal lungs. It is a normal anatomical feature in these animals. WP 20969

**Pulmonary Emphysema:** One of the most commonly misunderstood findings in animals is that of pulmonary alveolar and interstitial emphysema. It is rarely of any significance in any species unless supported by clinical signs of dyspnea. It is a very common finding in cows that die for almost any reason, so its significance has to be critically evaluated. This change is seen in cats, and less often in horses. It is best to ignore this change unless presented with a history of dyspnea. Pulmonary emphysema is recognized as more air in the lung than normal, and cattle may have large bullae up to 10–15 cm in diameter as a result of terminal gasping. WP 72

**So-Called Pulmonary Congestion and Edema:** This is recognized as a mottled reddened lung which is heavier than normal. Unless the lung is firm, pneumonia is not present. On section, the lung is very bloody and much blood can escape into the airways. Without a specific cause, this is almost always an artifact of blood pooling in the lungs after being pushed out of muscles by rigor mortis and out of the liver and vena cava by gastrointestinal gas production. This is another non-lesion that is best to ignore. In any animals not bled out, blood should be present—even histologically. WP 3302

## CARDIOVASCULAR

Goat and horse hearts generally have a very pointed apex. The coronary groove fat, along with most body fat, is very white in sheep and goats.

**Lack of Apparent Rigor Mortis:** A common finding in many animals is an extremely flaccid heart for a variable period of time after death. In most instances this is not a significant finding. It usually is seen in both the left and right side of the heart, with the left sometimes being affected earlier than the right, and is evident when the heart is examined soon after death, before rigor mortis has set in. In the horse and ox, it is surprising how long (several hours) it takes for rigor to set in, but it usually will. In cases of anthrax, it is probable that the failure to see rigor in the heart is really because it has already occurred, but too rapidly and too incompletely to be recognized.



**Pericardial, Endocardial, and Myocardial Hemorrhage:** Petechiae and ecchymoses, especially in large animals, are terminal findings as the result of increased peripheral resistance in the limbs with an intermittent, forceful cardiac contraction associated with dying for almost any reason. The heart then pumps against this increased resistance and causes these more centrally located hemorrhages throughout the various parts of the heart, mediastinum, and costal pleura. Some may be seen on the diaphragm, in the adrenals, and scattered other tissues. These are usually only petechiae or ecchymoses, but if they are more serious, such as suffusions or even closer to hematomas in these areas, then they may be more significant and represent a bleeding or clotting problem. They can sometimes be seen in septicemias and toxemias, but as they can so often be seen in non-septic and nontoxic conditions, their significance should be questioned, and you should look for other diagnostic features of septicemias. WP 1631

**Edema of the Heart Valves:** All four major valves, including all of their cusps, may be markedly thickened up to 5 mm with a clear, watery fluid giving the valve a swollen, shiny, appearance. Usually most cusps are involved, but some or all cusps, and even portions of cusps, can be affected. The base of the cusps may be similarly swollen. This is a common finding and of no diagnostic value, but it must be differentiated from the firm, irregular nodules on the free edges of the A-V valves, valvular endocardiosis, commonly best seen in older dogs. One suspected cause of valvular edema is the slowing of blood flow in the terminal stage of life.

**Endocardial Fat:** In most species, there may be small pale foci of fat 1–2 mm up to several centimeters in diameter and under the endocardium of both ventricles. They do not extend deeper than the endocardium unless they follow the natural clefts in the endocardium and often are more extensive around the attachment areas of the moderator bands. This deposition of normal fat is usually more extensive in excessively fat cattle or horses.

**Nodules of Arantius:** These small, 1–2 mm, firm, pale foci in the middle of the free edge of the cusps of the aortic semilunar valve are normal structures, most obvious in pigs, but may be seen or felt in other animals. They are to be differentiated from strongyle granulomas on the aortic cusps of horses which are irregular in location and size. They must also be differentiated from the common friction rub lesions seen on the aortic cusps of mature horses which are opposition lesions and thus paired where the cusps rub together near the cusps' free edges. WP 42

**Ossa Cordis:** These are two or three thin, curved, irregular three-pointed normal bones found at the base of the heart in the ox. They are often mistaken for abnormal ossification of the fibrous ring at the base of the heart in most species as they age. WP 1016

**Venous Emboli:** White, soft, elongated pieces of brain tissue, up to forty or so cms. long can be found in the major veins of the liver, lung and kidneys, among others, in cattle killed with the captive bolt pistol in conjunction with high-pressure air at the time of slaughter. This can allow brain tissue to get into the food chain of man. WP 3366, 3367, 3376, 21248

## BONE

**Synovial Fossae:** These are nonarticulating, normal shallow to relatively deep, irregular depressions in the articular surfaces of some joints, particularly in horses. These developmental structures may not be seen at birth but develop with age. They are thought to help in the distribution of synovial fluid. WP 16495

**Bones:** Pig bones have significantly thicker cortices than bones in other domestic animals. WP 16763

**Core Temperature Gangrene:** The foal or young horse with this entity may have lameness in one or more feet or even sloughed hooves. Palpation of all feet may reveal decreased temperature in each. A common history is that the animal has had a very high fever in the recent past from an infection somewhere in the body, usually the lungs. As a result of the high fever, the body may respond with peripheral vasoconstriction, causing the peripheral gangrene to the limbs. In different animals such as rats, entire litters may lose one or all of their feet and parts of their legs or lose their ears and tail. It must be differentiated from frostbite and ergot poisoning by the reasons above. Stagnation thrombi may be seen in affected areas but should not be considered necessarily embolic in nature, but only stagnant blood.

## CENTRAL NERVOUS SYSTEM

**Pia-arachnoid Fibrosis:** This is the common opaque white deposit of soft fibrous connective tissue present in the meninges of old animals, especially dogs. It is concentrated deep in the sulci, primarily over the cerebral cortex and the ventral surface of the brain. Nodules of connective tissue may be found in the cerebellar meninges of horses. The cause is considered to be the normal wear and tear of the CNS. WP 3277

**Meningeal Congestion:** It is common to see congested meninges due to body position and gravity after death. It is rarely diagnostic, unless quite distinctly localized.

**Prolapsed Cerebellum:** This is a common finding at the foramen magnum in many CNS disease cases, even in chickens.

## FETAL AND NEONATE

**Umbilical Cord Twisting:** There may be up to fourteen twists in the umbilical cord of the normal placenta in foals. If there is excessive edema, tearing, fibrosis of the cord, emaciation of the foal, or other evidence of vascular embarrassment including urachal and bladder distension, the twists, often greater than seven, may be of importance. WP 6020

**Placental Deposits:** Scattered, opaque, white, calcific deposits, 1–5 mm in diameter, are often seen on the amnion of cows, horses, sheep, and pigs. These are considered normal and occur only during a part of pregnancy. Another common finding are the rounded yellowish-green to white plaques of

hyperkeratotic epithelium often in linear arrangement on the amnion of horses, sheep, and cattle. Many are rounded and have a depressed circular-ringed center. These are again common findings but without a known cause or effect. Their surface layers peel off easily, often leaving a rough surface. Brown-tan, flat, rubbery masses, hippomanes, from 2–10 or more cm in diameter, may be found floating free in the allantoic fluid of horses and cows. Usually one or two are found and when cut in half, many, not all, have a central laminated appearance suggesting that they may have once been spherical but are now collapsed. Their source is still in question. WP 12731

**Fetal Hooves:** Slough early, often with attached ragged eponychium; especially in camelids.

**Umbilicus:** Some hemorrhage around the umbilicus is very common and usually not significant.

## GASTROINTESTINAL

**Glossal Fimbriae:** These are fleshy, usually flat, papilliferous 5–10 mm epithelial projections from the edge of the tongue in normal newborn piglets. Their cause and use is not known, but they usually disappear shortly after birth. Smaller ones may be seen in puppies. WP 2923

**Idiopathic Hypertrophy of the Distal Esophagus:** This is recognized mostly in horses and pigs as the pale, thickened muscle in the entire distal half, usually distal third or less of the esophagus just as it enters the stomach. In very few cases, the muscle hypertrophy can extend into the muscle of the stomach. Clinical problems are seen in only 1–2% of horses, so some consider it almost normal. WP 615, 244

**Unguiculate Papillae:** These may be 1–2 mm, or up to several centimeters, firm, curved, or straight, keratinized papillae found at the distal end of the esophageal groove and omasum in ruminants. These are normal structures and in young milk-fed animals, they are often opaque white, but in older animals they get darkly stained with ingested food pigments. Unguiculate refers to claw-like and ungulate refers to hoof. WP 707

**Rumen Mucosal Sloughing:** As early as twenty minutes after death, the lining of the rumen may peel off in large patches, leaving a pale or intensely reddened submucosa, depending upon whether or not the animal was bled out at the time of death. This is an artifact mistakenly called a hemorrhagic inflammation, but no exudate, edema, or hemorrhage is present to make it a lesion. WP 18064

**Torus Pyloricus:** This is a discrete swelling of fat connective tissue and smooth muscle in the gastric wall at the junction of the pyloric portion of the stomach and the duodenum. In the pig, it is large (3–5 cm), with a central groove. It is a smooth lump in the cow and less prominent in most other species. It is also one of the most commonly misdiagnosed normal anatomic features in animals, often called tumors. WP 611

**Gastrointestinal Mucus:** This appears as the thick, usually yellowish brown or pale white, mucoid material loosely adherent to the stomach and small intestine mucosa. It can be easily scraped off with no underlying ulceration or inflammation and, if the surface is kept moist, more of the same mucoid material may form in the same area by apparent extrusion from the underlying mucous glands. This normal material has often erroneously been called a catarrhal exudate. This finding of mucus is seen in most species, but it is especially prominent in the stomach of horses and pigs and the large intestine of many species. Intestinal dehydration helps make it appear more prominent and even string-like.

**Gastric Hyperemia:** The more or less intensive reddening of the glandular mucosa of the stomach is a common physiological finding in many animal stomachs, especially in the horse and pig. The lack of free hemorrhage, exudate, ulcers, or edema precludes gastritis. WP 3064

**Postmortem Gastric Ruptures:** These are usually large eroded areas of thin gastric wall with one or several present, allowing content to escape. The edges are thin and discrete. Postmortem digestion by the acidic gastric juice is the usual cause. Postmortem gastric ruptures are quite common in the dog, calf, and rabbit, less so in the horse, and difficult to differentiate from true ulcers. WP 2944

**Duodenal Papillae:** These are two mucosal nodular structures, one larger than the other, seen in the beginning of the duodenum of most species. They are normal openings of the bile and pancreatic ducts. They are erroneously considered to be polyps or neoplasms. They may be craterform. WP 17207

**Postmortem Bile Dilatation of the Bowel:** Often one sees discrete segments of small intestine, usually the duodenum, dilated and thin walled, to two or three times normal, which have a distinctly green color under the serosa. Bile is extremely caustic and may be propelled in small bolus amounts by continued postmortem peristalsis to cause this artifact in many species, especially the dog and cat.

**Segmental Intestinal Hyperemia with and without Diapedesis:** Commonly in all species, segmental areas of congestion of the small intestine are found. With time after death the vessels break down, resulting hemorrhage-like blood in the lumen. They should not be considered of evidence of enteritis without edema, necrosis, ulceration, or fibrin. Large segments or only small segments may be involved and should be considered postmortem artifacts. WP 6258

**Dystocia-Related Small Intestine Blowout:** All cases of this entity, with septic peritonitis mainly in the pelvic cavity, occur following mechanical pulling of a calf by the owner with a loop of small intestine of the cow in the pelvic cavity as the calf is pulled during delivery. The cow's intestine being compressed causes the rupture. The cow usually dies hours or a day later with severe fibrinopurulent peritonitis especially in this area or in the abdominal cavity generally, which may hide the cause if not looked for diligently. There is usually no uterine trauma.

**Abomasal Impaction:** Cattle can die with a severely swollen abomasum filled with ingesta without a specific cause, but often vagal nerves are considered to be involved as they course through the pleura as with chronic pneumonia..

This booklet is intended to be used at least in part with the pictures of actual tissues to be found on the Cornell Web page:

**<http://w3.vet.cornell.edu/nst/>**. From the home page, click on Advance Search and type the number of the slide (which follows the WP in this text) in the Image ID box.



**Duodenal Papillae:** These are two mucosal nodular structures, one larger than the other, seen in the beginning of the duodenum of most species. They are normal openings of the bile and pancreatic ducts. They are erroneously considered to be polyps or neoplasms, especially in the horse in which the major papilla is very prominent. In the horse, they are often normally craterform. WP17207

**Postmortem Bile Dilatation of the Bowel:** Often one sees discrete segments of small intestine, usually the duodenum, dilated and thin walled, to two or three times the diameter of the rest of the bowel, which have a distinctly green color under the serosa. Bile is extremely caustic and when released into the duodenum at death, it may be propelled in small bolus amounts by continued postmortem peristalsis to cause this relatively common postmortem artifact in many species, but especially in the dog and the cat.

**Segmental Intestinal Hyperemia with and without Diapedesis:** Commonly in all species, segmental areas of congestion of the small intestine are found. These are areas of physiologic hyperemia interspersed with non-congested segments. With time after death, the mucosal vessels may break down with resulting hemorrhage-like blood in the lumen. They should not be considered as evidence of hemorrhagic enteritis without evidence of edema, necrosis, ulceration, or fibrin. Sometimes large segments and at other times only small segments may be involved, but all should be considered postmortem artifacts. WP6258

**Postmortem Intussusception:** This occurs after death when a section of intestine (the intussusceptum) has invaginated a portion of bowel (the intussusciens) cranial or caudal to it. There is no vascular response such as edema, hemorrhage, or fibrin accumulation, although the intussusception may be slightly more congested than the rest of the bowel. This is considered a terminal or postmortem event in all species, as peristalsis continues even after death. Antemortem intussusception can certainly occur, but shows inflammation and even necrosis.

**Intestinal Lymphoid Follicles:** These are most commonly seen as opaque, pale, round, several millimeters in diameter foci, often with a dark center scattered throughout the cecum and colon. These are most prominent in the dog and pig. Other aggregates of lymphoid tissue, oval or linear, plaques, or even areas encompassing the entire circumference may be the Peyer's patches seen in the distal ileum, jejunum, and sometimes surprisingly in the duodenum. In the ox a large patch (colonic tonsil) is commonly seen just a short distance distal to the ileal opening. It is also seen in many normal pigs and debris can be squeezed from the follicles. WP3221

**Mucosal Linear Reddening (Tiger Striping):** It is not unusual to find linear patches of reddening of the colonic mucosa, often in the rectum proper and sometimes also in the urinary bladder. Blood is trapped and subsequently clots in these superficial areas of the mucosal folds as the animal terminally strains to urinate or defecate. Rarely is it of diagnostic significance although some erroneously think it is diagnostic for some diarrheal diseases. WP3214

**Distal Ileum Thickening (Idiopathic Hypertrophy of the Distal Ileum):** Several references say this hypertrophy is normal in the horse, but it is not because the distal ileum is just as thin in normal animals as in the rest of the small bowel. There is not complete agreement on this statement. The cause for the hypertrophy when seen is not known, but some functional disturbance is suspect. Usually the idiopathic hypertrophy of the distal ileum does have some clinical importance, as it may be a cause of colic. WP 75

**Pacinian Corpuscles:** These are about 5 mm, white, oval, slightly gray and shiny, flat discs best seen in the mesocolon of cats. They are normal pressure-sensitive structures. Histologically, they are also seen more commonly in the cat pancreas. WP 575

**Firm to Hard Enlarged Omasum:** This is a common finding in normal ruminants' omasum. It has very dry, flat flakes of ingesta between the folds. WP 17882

**Horse Stomach Enlarged and Firm:** This is a normal condition with relatively dry ingesta, often erroneously considered as impacted. WP 3086

## ADDENDA NOTES

---

### ODORS ENCOUNTERED AT NECROPSY

One of the most important senses that we possess is often neglected at the necropsy table: the use of the sense of smell. Thus odors, or a few of them, will be discussed here. The actual description of an odor is difficult, or often impossible, and so must be identified by comparison. For example, try to tell someone how an orange smells without using the word "orange."

**Rancid Butter:** The disease blackleg, caused by *Clostridium chauvoei*, has the distinct odor of goat acids (caprillic, capric, and caproic) or rancid butter. This odor has also been noticed in some cases of *Cl. septicum* infection in pigs and horses. WP 20290

**Septic Tank Odor:** In all species with Salmonellosis, the affected, non-treated intestine has this distinct odor. This does not mean the fecal odor of decay. A septic tank odor is the best description and very diagnostic. WP 2994

**Odd Sweet Odor:** In most species, large round worms, ascarids, give off this odor. This is especially true when many sexually mature female worms are present. WP 3053

**Fermentation:** While often overlooked initially, the dark, firm mammary gland tissue with septic mastitis has a definite sour odor of fermentation at necropsy. The odor becomes more prominent as the gland lies open exposed to the air, and the color also darkens. Often an infected udder may appear normal at first. Even with a normal color, flies will often be attracted to the infected quarter early. WP 12517

**Ammonia:** In some cases of urea poisoning, a sharp ammoniacal odor may be noted when the rumen is opened.

**Myiasis:** In sheep's wool, dog's hair, or anywhere, maggots have a sour odor of their own. WP 13577

**Mange:** Mange mites of several genera give off a definite odor peculiar to that species. The smell on dogs and foxes is especially noticeable when they are affected with sarcoptic mange. WP 9326

**Onions:** This obvious odor is no problem except that glycerol guaiacolate (guaifenesin) with propylene glycol, a commonly used pre-surgical drug, will give off a similar odor. Propylene glycol in the rumen content gives off this odor in normal cattle and sheep. Garlic is also possible with this odor, as is organic phosphates poisoning.

**Used Motor oil:** Everyone knows this odor. Its importance in pathology is related to the toxic component, lead, that is a serious contaminant of used motor oil and from the leaded gasoline used in the past.

**Chemically Sweet:** As with many drugs in solution, nembutal has a background alcoholic odor; but in addition, a sweet odor is present. This is most easily noticed in the lungs of animals killed with this drug. WP 3317, 13615.

***Arcanobacterium pyogenes*:** A very characteristic odor is often associated with large accumulations of fluid pus, as in traumatic pericarditis or peritonitis. WP 22132

***Fusobacterium necrophorum*:** The odor of foot rot is one of the best examples.

**Pine tars:** Turpentine, Cresol, Turcapsol – These pine-related odors are most distinct and are most often related to treatment.

**Apple cider:** Many times, predominantly large quantities of blood in the stomach of pigs or cattle caused by bleeding ulcers gives the opened stomach, and indeed the entire necropsy area, the smell of slightly fermented apple juice. WP 17073

**Salmon poisoning disease** in dogs, and probably foxes or other affected canids, gives off its own distinct odor.

**Garlic:** Often, in cases of poisoning with organic phosphates, the rumen content and the insecticide material itself has a garlic-like odor and must be distinguished from the odor of onions. Propylene glycol in the rumen also gives off the same odor. WP 623, 624

**Fish:** In most cases of nutritional panniculitis, also called nutritional steatitis in cats, horses, and pigs, the affected fat has the odor of fish because of the association with fish diets in cats and pigs or the administration of cod liver oil. WP 595

**Alcohol:** This is a common diluent with many parenteral uses and should be easily recognized.

## BRIEF COMMENTS ON NEOPLASIA

A lump is a lump no more and no less, until it is identified either by histologic study or other means. Being able to identify the lump is a good reason for the invention of the microscope.

Certain tumors can be presumptively diagnosed grossly because of the animal's history or the prosector's experience, but a mass by itself could be one of several conditions.

Lymphosarcoma can mimic almost any condition grossly, and can occur in any age animal, even aborted fetuses. When found in tissues such as the heart or even the lung, instead of the expected lymphoid tissues, the malignant cells tend to stimulate fibrosis, which they usually do not do when in normal lymphoid areas.

In some species, such as the dog and cat, there are certainly specific tumor ages (about seven years in these animals) and conversely masses seen in the young may not be neoplastic. This is variable among different species and with different tumors.

Without evidence of prior damage in an organ, such as the liver or thyroid, multiple lumps probably represent neoplasia and not compensatory hyperplasia or regeneration. Thus the commonly seen mass or masses of liver-like tissue in old dogs' livers are probably benign hepatomas. The spleen is an exception to this rule, as it has hyperplastic lymphoid nodules without any apparent splenic damage to cause a compensatory need and is common in the dog and, less so, in the cow.

It has been noted in many animals that congenitally misplaced tissues (ectopic tissues) are thought to be 20 to 40 times more prone to neoplasia than normally placed tissues, such as Sertoli cell tumors occurring in cryptorchid testicles, and heart-based tumors often being thyroid adenocarcinomas instead of the expected aortic body tumors.

## THE TIME OF DEATH

Death, as we know, is the culmination of dying; but to establish the exact time is often difficult. A few generalities can be mentioned.

**Rigor mortis**, the stiffening after death, is best related to the body temperature and the metabolic activity at the time of death. If death occurs during a high fever disease such as porcine stress syndrome, heat stroke, or anthrax, rigor can occur almost simultaneously with death. Rigor mortis also occurs rapidly in animals that are excited or severely stressed just before death. The opposite is true for animals that are moribund or cachectic for a long period before death; rigor may not occur for hours, if it occurs at all, and when it does, it may not be easily noticeable. The length of time a body is in rigor is directly related to the onset, as is the post rigor relaxation time. An animal that takes minutes to go into rigor will only have a similar short period of rigor. WP 3311

The jaw muscles of dogs and most animals are the first to set up in rigor, followed by the eyelids, tail, digits, distal limb muscles, and finally the larger limb muscles. They relax in a similar sequence. Once rigor has been broken by moving the body or limb, the rigor will not return.

**Algor mortis**, the cooling of the carcass with death, is another useful but not absolute parameter to be noted. Oral, axillary, or even deep muscle, abdominal, or heart blood temperature are at best only suggestive of algor mortis, as they vary so widely after death.



Ambient temperature is important in its interpretation. In one horse experiment at Cornell, the best location to take temperature was through a hole in the skull. An extra-long thermometer was pushed through the brain to the floor of the skull. The temperature here decreased in a relatively straight line to ambient temperature about 18 hours after death. In twelve other sites, the temperature varied too much for evaluation of time of death. In an obese, heavily-haired, or wool-covered carcass, the values have to be suspect. When a heavily woolled sheep is put in the necropsy refrigerator or left out in freezing weather, its body temperature will increase for hours because of insulation afforded by the wool and the heat produced by continued fermentation in the gastrointestinal tract to greatly enhance the severity of decomposition.

**Corneal clouding**, at least in dogs, begins not sooner than twenty-five hours after death. Closed eyelids help this evaluation by preventing corneal dehydration, and temperature does not seem to affect this change. WP 1360, 20160

**Clouding of the lens** of most species is quite variable and the most apropos comment to make here is that the lens clouds easily when cold. Such cloudiness is often mistaken for cataracts. To differentiate from true cataracts, one has only to warm the head and eyes, and these “cold cataracts” will disappear. WP 20166, 21510

**Rumen mucosal sloughing** is of little value as it can begin within twenty minutes in the “normal cow.” However, this usually takes several hours. WP 18064

Failure to slough after many hours (6–12) may suggest an underlying lesion is causing the adhesion, such as mycotic infection or even a toxic problem as arsenic ingestion or even formaldehyde ingestion.

**Blood glucose** drops rapidly about 20 minutes after death while some other biochemical values, such as calcium or other minerals, may not decrease until much later. Comparison with C.S.F. glucose values may be helpful for some. On the other hand, the continued plateau of glucose levels may indicate a poisoning by sodium fluoroacetate which prevents the enzymatic breakdown of glucose. Blood urea nitrogen (BUN) does not increase continuously after death, as many believe, but instead plateaus for up to 4 hours before gradually decreasing.

**Food digestion:** Animals removed from their normal habitat just after eating may not have any appreciable digestion noted of their stomach contents if they are severely stressed for 24 to 48 hours or more after being moved to new housing, for instance, just prior to death.

**Related facts:** Other related aspects can be used to determine time of death. For instance, the consistent developmental stages of identifiable eggs or maggots on a flyblown carcass may indicate when the wound or carcass became flyblown and may thus be an indication of time of death. Although not related to time of death, I'd like to suggest that one rather unique way to slow down decomposition is to bury the carcass in the cool earth. This will often delay autolysis considerably, if no other means of preservation is available. A fresh dead cat buried for a week like this is difficult to tell from one just cooled out for several hours.

## DECOMPOSITION

**Autolysis** of animals is a problem which we, luckily in veterinary medicine, can often ignore except probably in medico-legal cases as with human cadavers.

Certainly one can make a good or even great diagnosis from a dog buried 8 months with the radiographic evidence of lead pellets associated with a fractured bone, but this is an exception. An accurate diagnosis is rarely made in a sheep found dead on pasture with its wool falling off its green body. It would be far better to tell the owner or clinician that it was too decomposed for diagnostic purposes and hint that a more expedient necropsy would have been a better sample selection. Likewise, a frozen dog can allow a decent diagnosis even if frozen for an extended period, providing the animal had not been allowed to decompose for 5 days before it was frozen and then thawed quickly for the necropsy.

Fat animals, or fully woolled or haired animals, actually increase in body temperature when placed in a refrigerator after death, and this often causes some increase of heated dialogue between pathologists and owners concerning the supposedly immediate refrigeration of the body after death. A blanket, like wool or long hair, is a great insulator keeping **cold out** and **heat in**. Even paper or cardboard is not a friend to the pathologist's duties, like that afforded to the sleeping human.

## MISCELLANEOUS COMMENTS

Many very small animals (mice, finches, etc.) come into a busy diagnostic service, and pathologists or students to do a routine necropsy on them. It is far better to open the major cavities and skull, mouth down to the stomach, chest and abdominal cavity, and evaluate all **organs in place** and then fix the whole carcass. When the time comes for the histologic examination, the prosector can then be sure to trim the proper tissues.

One of the more common dilemmas noted in veterinary pathology is the finding of a no-fat, dark tissues, starved lamb or other animal that came into the necropsy room, but its abomasum was over half filled with milk. So how could it be starved? The clinician involved told the owners it was hungry, so they force-fed it lots of milk—a bit too late. The lacteals of most may be full of chyle if just recently force-fed.

Do not tie the “lead” rope of a horse’s (especially) halter at ground or floor level as when the neck is extremely flexed, fractures of the neck or floor of skull may be caused

It is interesting to note that just as it is widely accepted that breast milk is probably the best milk for human babies, in veterinary medicine the same is true. When a nest of baby owls or hawks, etc., is found, the human Samaritans realize they need food (and water), and the baby birds will readily eat or drink whatever they are offered with wide-opened mouths as a very early reflex. If too much liquid is given, one will have several dead birds hours later, probably from electrolyte imbalance.

It is similar for calves. Dairy cows' milk has become more dilute (in comparison to the milk of beef cows) because dairy cattle have been bred for milk quantity, not quality. So we should not consider all milks as equal. The raising of calves in cold climates is also a severe problem, as calves (or other animals) need more calories. As the weather gets colder or wetter, they need more calories. If they are cold and wet (as many calves are), they need even more. This is almost never a problem in beef calves nursing their dams. Beef calves raised on their dam's milk consume "multiple meals" each day, while dairy calves are usually fed twice. With less concentrated dairy cow milk, or milk replacer, leading to a caloric deficiency, or if the quantity is increased, the volume may induce diarrhea by itself. **Cold, wet calves in the winter need about 4 times the calories required in summer.**

## Terminology

Congestion is an antemortem increase of blood to a part either pathologically or physiologically and should not be used for blood pooling (hypostasis) in the body after death.

Common is not a synonym for normal. The words torsion, twist, and volvulus are synonyms.

Starvation is a willful or ignorant cause for the lack of caloric intake, whereas malnutrition refers to an imbalance of one or more nutrients as the lack of one nutrient or excess of another causes the imbalance. Inanition is considered the inability to eat as from diseased teeth or pharyngeal damage, etc.

Prolapse is the significant bulging of an organ such as the anus or the vulva, whereas eversion is the bulging from turning inside out of the organ.

A common mistake by neophytes in pathology is to equate emaciation with acute infectious processes, whereas the opposite is more likely true.

As mentioned in the first paragraph of this booklet, the use of the word necropsy is restricted to the gross detailed study of a dead animal. It may or may not include histological examination.

Skull fractures in horses with severe flexure of the head may be caused by the horse falling backwards onto its head or being tied low to the ground and abruptly stopped at the end of the rope. The ventrally located portions of occipital, sphenoid, or temporal bones are the ones usually injured.

## **Fatal Diseases or Conditions with No Lesions**

With few exceptions, good postmortem diagnosis is based on the presence of key lesions and a corroborative or at least compatible signalment and clinical data. There are, however, disease entities in which gross lesions are sparse, absent, unrecognizable, or which fail to be recognized. A list of such entities follows.

A rough attempt to put these entities into strict categories has been given here. The experience of one individual is not likely to include all the entities to be listed, thus several individuals are to be thanked for their contribution to the book and to this section specifically.

Dr. Claus Buergelt, Gainesville, Florida  
Dr. John Edwards, College Station, Texas  
Dr. Francis Fox, Ithaca, New York  
Dr. Robert Hillman, Ithaca, New York  
Dr. Linda Munson, Davis, California  
Dr. Roger Panciera, Stillwater, Oklahoma  
Dr. Linda Parrish, Kendall, Wisconsin  
Dr. Gustavo Perkins, Tandil, Argentina  
Dr. Mary Smith, Ithaca, New York  
and the authors of this book.

## **Allergy or Allergy-Like Deaths**

### **Milk Allergy:**

Well written up by Dr. Gordon Campbell, these deaths usually occur in Jersey and Guernsey cattle which die suddenly in association with milk let-down. Severe head, neck, and lung edema may be noted clinically. It kills many, but the edema disappears shortly after death even though it was seen clinically just before death. WP 255

### **Postvaccinal Reactions:**

Animals die after vaccinations with no, or only slight, pulmonary edema. Some of the older anaplasmosis vaccines caused death with or without acute hemoglobinemia (peracute isohemolytic disease).

WP 2680

### **Intravenous Therapy:**

Dairy cattle are occasionally victims of electrolyte therapy, especially those treated for milk fever that die during treatment. Death is usually considered to be caused by the rapid streaming effect of the calcium-containing solution in contact with the endocardium, leading to heart block. Jugular needle punctures may be noted.

### **Anesthetic Deaths:**

During or after surgery, animals may die for no apparent reason and without any lesions. The lungs may be darker than normal, with a sweet odor of barbiturates or alcohol in the lungs or heart blood. Copious amounts of blood are found in the organs, but this should be expected in animals not bled out.



**Botulism:**

Most species exhibit signs. Head, limb, and neck paralysis, along with dysphagia, are significant. Decomposing animal parts mixed into feed should be checked for, as well as ingesta examined for any chicken litter that may have been mixed into feed for cattle.

WP 8239, 16272

**Taxus:**

The evergreen needles should be found in the gastrointestinal tract of horses, cows, sheep, and goats. Animals usually die within minutes or hours after ingestion. Some animals may die from only a mouthful of the needles, but other animals may be far more resistant, even to needles from the same tree. WP 190

**Sodium Fluoroacetate (1080) Poisoning:**

These rodenticides are extremely toxic to most species, and the animals die starting about 20 minutes after ingestion. A kennel of 24 dogs was poisoned by an accidental exposure after liquid 1080 soaked into a dry kibble bag; 23 died within hours, the 24th dog did not eat any of the feed and lived. None had lesions. Some suggest heart lesions may be found in herbivores. Vomitus may be fed to experimental rats as an aid in diagnosis.

**Cyanide Poisoning**

Cyanide poisoning is sometimes diagnosed when cyanogenic glycoside-containing plants are known to be in the environment and parts of the plants are found in the gastrointestinal tracts of the dead animals. Blood and body tissues may be various shades of pink, but this is easily overlooked. WP 11638

**Shock, Heart Attack, or Stroke:**

These three entities are often given as the cause of death in both man and animals. However, they are very rare in animals and unless one finds respectively: brain hemorrhage, heart lesions, or extreme traumatic or related lesions, the three terms should not be used. Certainly they can occur, but very rarely.

**Fetal Death:**

The presence of twin foals, often found dead singly and of different gestational age, is a common cause of abortion. When each fetus utilizes the maternal endometrium there may not be enough placentation for fetal development and one twin may abort first, still not leaving enough for the second twin to develop and survive. Diagnostic lesions may not be found in the fetus itself, except for smaller than normal size and emaciation. Care should be made to also rule out the umbilical problems of wrapping around the fetus and umbilical torsion.

**Poisonings:**

Generally speaking, poisonous agents require a keen knowledge of signs to be detected definitively. Chemical analysis may be helpful. Portions of liver, kidney, gastrointestinal content, and even depot fat should be submitted for screening. Urine, blood, and CSF may be useful if freshly sampled. Clinical histories are of major importance for most poisonings. Signs are often diagnostic in themselves. Also, it is often necessary and very beneficial to make a farm or home visit with a different set of eyes.

**Carbon Monoxide Poisoning:**

Some dead animals may have very pink blood and tissues. Several great cases have been associated with inadequately vented charcoal fires in enclosed spaces. WP 596

**Mushroom Poisoning:**

Sudden death with pieces of identifiable mushroom in the gastrointestinal tract is highly suggestive. In a few cases, periportal liver degeneration may be found histologically. WP 875

**Strychnine:**

Signs include fast, strong muscle contractions in response to loud noises, or even slight body trauma when alive. Often, meat of scraps containing colored seeds are found in the stomach. These mercury or strychnine treated seeds are used for rodent control. WP 1070, 18701

**Urea Toxicity:**

The odor of ammonia can sometimes be noticed in the rumen and sometimes only in the colon contents. Hard granules of urea may be found in feed. The percentages of ingredients in a formulated grain mixture are not always correct as stated by the manufacturer.

**Monensin (Ionophores):**

This common feed additive, used as a growth promoter, can certainly kill sheep, cattle, and horses acutely, with no specific lesions. In more chronic exposure, pale, even opaque white streaks of muscle degeneration may be seen scattered in myocardial and other muscles.

**Lead Toxicity:**

Animals are often clinically blind, without other lesions grossly. Some cases of acute poisoning in calves may show marked subperiosteal hemorrhages of the rib cage with distinctly pale muscles. WP 798, 1050, 10561

**Organophosphate Poisoning (Insecticides):**

A garlic or onion-like odor to the gut content is often noted in any animal poisoned with organophosphates. Suspect feed or gastrointestinal content smeared on an inside wall of a jar will often kill freshly caught flies.

**Propylene Glycol Toxicity:**

Usually given as a source of energy, too much can kill, without lesions. A characteristic odor, similar to onions or garlic, may be noted in rumen content; it is a similar scent to that of organophosphates.

**Tetanus:**

This is usually related to deep anaerobic infections with bacterial release of toxins. The deep lesions may be hard to find and are usually in the feet. The severe spasms of tetany will often result in the animal dying with stiff extended limbs if the necropsy is done soon after death. WP 10794, 13546

## Deficiency Diseases

### **Grass Tetany (Transport Tetany):**

This is seen usually in milking or nursing sheep and cattle on lush pastures in the spring. Many of the dead animals have higher magnesium blood levels than the unaffected, living animals, suggesting that magnesium deficiency is not the entire problem. One hypothesis is that the green grass with its high vitamin A (carotene) content is antivitamin D; decreasing the active absorption of calcium from content interferes with vitamin D's absorption of calcium from the bowel, which at the same time prevents the passive absorption of magnesium.

### **Winter Tetany:**

This is primarily seen in lactating beef cattle on well-to-do farms where high-quality alfalfa is being fed. Good alfalfa is known to have higher levels of vitamin A than other grasses. The same hypothesis holds as for grass tetany. One can treat with magnesium to overcome the apparent vitamin A's antivitamin D effect. With some tongue-in-cheek, it is not a deficiency of antibiotics that allows bacterial disease to occur, even though when given they can cure the infection.

### **Nutritional Muscular Dystrophy (White Muscle Disease):**

Calves, lambs, foals, and most other herbivores, even newborn ones, may have the classic gross lesion of opaque, white patches of muscle degeneration. However, there are many instances, up to 50%, in which gross lesions are not found. Histologic changes are usually in the more active muscles. One strong hypothesis for this disease is that it is not a primary deficiency of vitamin E/selenium except at the cellular level, but is a vitamin E/selenium responsive disease. A biologic factor in spoiled or moldy feed makes the vitamin E/selenium unavailable at the cellular level. This functional deficiency can be overwhelmed by the relatively large doses of vitamin E/selenium therapy. WP 324, 885, 3265

### **Subacute Emaciation**

This is seen in almost any young animal with no specific lesions except minimal body fat stores. Ingesta may or may not be present in the bowel. Possible slight edema and hemorrhage may be found in the distal subcutaneous soft tissues of the limbs. Cold, wet, even foggy conditions, inadequate shelter and insufficient nutrition are usually found. These specific conditions are additive to effectively cause these deaths. This is a common entity throughout the world, even in more affluent areas. WP1792, WP3017

## **Metabolic-Related Deaths**

### **Acute Lactic Acidosis:**

Usually found in ruminants, and associated with sudden access to highly fermentable feed, an excess of which will be found in the rumen. The rumen pH will be much lower than normal, and blood lactate levels will be elevated. In chronic cases, other lesions will have time to develop and be found, such as bloat, laminitis, rumenitis, and others.

### **Milk Fever (Hypocalcemia):**

In spite of the name, neither fever nor hypocalcemia are usually found in these cases. Lesions are also not found in the very few that die in uncomplicated cases.

### **Hypoglycemia:**

Often newborn animals, especially piglets and toy breed puppies, will die with no milk in their stomachs. The dams must be checked for their state of lactation.

### **Heat Stroke (Hyperthermia):**

Findings include a distinctly elevated body temperature and a history of being in conditions conducive to overheating, such as a dog left in a closed vehicle in hot weather. Small adrenals are easily overlooked in some species such as pigs. The carcasses may be extremely pale, suggestive of a cooked appearance. The temperature of a fat, or heavily hair-coated, otherwise normal animal may actually rise after death, even under refrigeration, and thus must be considered in such cases, as well as in animals that can die with this condition while under a blanket during anesthesia. WP 604

## Miscellaneous

### **Bloat (Tympany):**

Bloat can be a lesion if noted in the live animal, but if the bloat is noted only after death it is often difficult to differentiate antemortem from postmortem bloat. Hindquarter paleness and some fascial edema may be noted in true tympany, while not likely in postmortem bloat. A “bloat line” with dark esophageal mucosa cranial to the thoracic inlet is suggestive of true bloat, but a similar demarcation may be seen in animals that die and bloat quickly before the blood coagulates. WP 2067, 3084

### **Vagal Death:**

In a very few proven cases, animals have died suddenly, sometimes noted as catastrophically, from minute trauma to the pleura, pericardium, or peritoneum (as minimal as a needle puncture). In the human field this is considered to be a fatal vagal reflex death and is assumed to be the same in animals when no other lesion or cause of death is found. See *Autopsy Pathology*, Rezek and Millard, p. 14, Charles Thomas, publisher, 1963.

### **Central Nervous System Disease:**

While there are often no gross lesions to be seen in fatal viral CNS diseases, most cases will have histologic changes in the tissues that are definitive. This almost mandates that CNS tissue be taken for evaluation. **Prosector note:** Because of the potential for aerosol contamination, mechanical devices such as electric saws should not be used for CNS tissue harvesting without proper protective gear. WP 1265, 2243, 20216

### **Anemia:**

A very pale carcass is often diagnostic for heavy parasitism, for example, sheep with Haemonchosis. However, such animals may not have many or any parasites in the abomasum. While they may have received treatment which removed the parasites, but it was given too late to save the animal. It is also true that even without treatment, worms, like rats, will “leave the sinking ship.” WP 1280, 3089



### **Electrocution:**

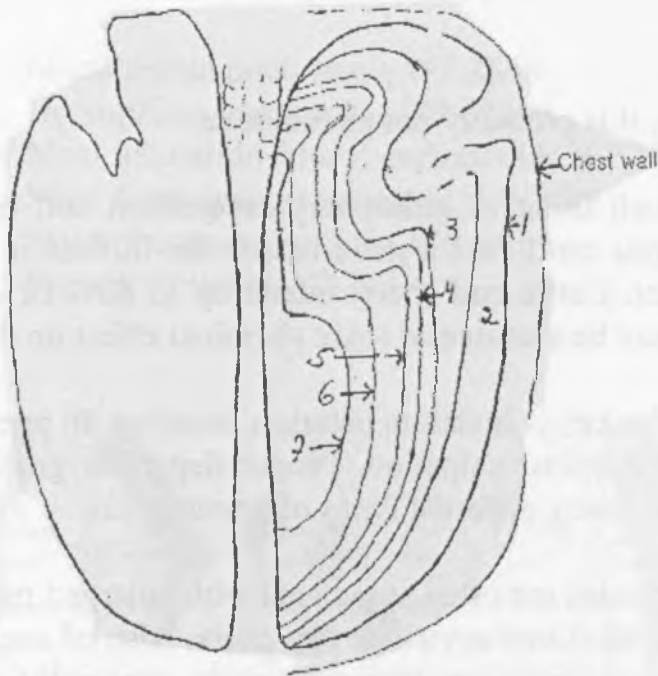
Lightning strike may cause a fractured vertebral column from the physical force involved. Grounding burns in the hair and skin may or may not be present. Often no lesions can be found. The same can be said of animals electrocuted because of faulty wiring of machines or buildings. Some electrocutions are malicious. Clamp marks have been found on tongues, lips, anus, or vulva. A vertebral compression fracture would provide more evidence as well as psoas muscle tearing. WP 14, 10634, 10633

Examples of many of the actual tissue and organ items discussed in this book and identified by the Web Page (WP) number for each are available free on the Cornell website <http://w3.vet.cornell.edu/nst/>.

From the home page, click on **Advanced Search** box and type in the number given.

A broad identification of subjects can be found by clicking on the **Table of Contents** also in the home page.

## REPRESENTATIVE STAGES OF POSTMORTEM LUNG INFLATION



1. Parietal pleura (costal pleura).
2. Inspiration lung expansion.
3. Dead 6 hours from choke (neck torsion, tracheal obstruction, etc.). Lungs still inflated from subsequent loss of elastic recoil.
4. Dead from choke, but obstruction cause removed immediately. Immediate postmortem expiration.
5. Dead for a few minutes with O<sub>2</sub> (not N<sub>2</sub>) metabolized.
6. Atelectasis, fetal or acquired.
7. Anesthetic gas machine lung (prolonged). Gas anesthetic totally metabolized after having replaced the inert N<sub>2</sub> during surgery, and not having been replaced at the termination of surgery. Hypoplastic lung may be seen in the fetus due to failure to develop because of CNS dysplasia.

## THE PNEUMONIAS

### Comments:

If it is not firm, it is probably not pneumonia.

There is no such thing as pulmonary congestion and edema without a cause, i.e., if you can't find a good reason for it, then it is probably just terminal artifact. Cattle and sheep inhale up to 80% of eructated rumen gas, and this may be a source of toxic chemical effect on the lung itself.

In real estate, the key is location, location, location. In pneumonia, the key is palpation, palpation, palpation. Proper depiction graphically helps to differentiate the many different types of pneumonias.

Chronic pneumonias are often associated with enlarged regional nodes, just as chronic infections anywhere can cause. Also of some note is a fact that cases of severe acute pneumonia in cattle, especially, may be associated with severe diarrhea without a known reason.

**Distribution of Lesions:** There are three major distribution types that may overlap or even coexist. Gravity is often important here.

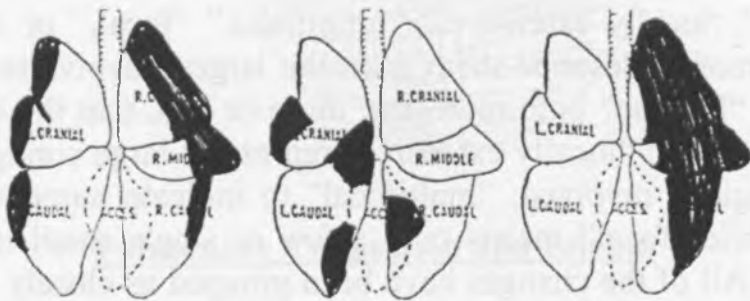
**Diffuse:** Affects the entire lung or a scattered major share of it (80–90 %).



Examples: Proliferative pneumonia WP 3566  
Toxoplasmosis WP 13559  
Heart Failure Lung WP 255  
Anaphylaxis (bovine) WP 6146

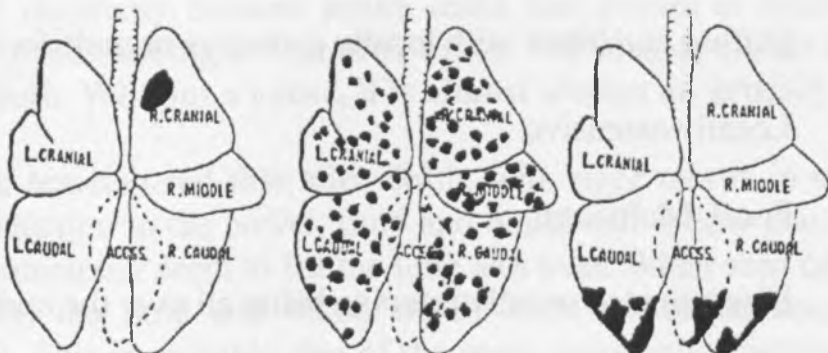
**Locally Extensive:** A large confluent area, or several large confluent areas, are affected and usually involve several to many adjacent lobules.

- Examples: Mannheimia pneumonia WP 2648  
Hemophilus pneumonia WP 675  
Most inhalation (fluid) pneumonias WP 12  
Most distemper pneumonias WP 2699  
Mycoplasma pneumonia WP 670  
Pneumocystosis WP 213



**Focal or Multifocal:** Scattered individual or many small, usually discrete, foci throughout the lung; or as demonstrated by a vascular or airway spread. These are often found only by palpation.

- Examples: Embolic abscess shower WP 2662  
Dust inhalation WP 2662  
Most lungworms WP 925  
Single foreign body WP 113



## Rough Sketches of Selected Findings

As there are so many possible variations of different findings, lesions, artifacts, and other tissue changes for the four organ systems partially covered in this publication, we have to admit that only a few have been included, but we hope that they cover the majority of common ones.

We have taken great leeway in the various depictions, and have described most in a simple fashion as to location and extent, using “diffuse,” “locally extensive,” “multifocal,” “focal,” or combinations thereof to describe them from the largest involvement to the smallest. “Diffuse” is to represent, more or less, that the entire organ is involved; “locally extensive” represents large contiguous areas or regions involved; “multifocal” to indicate numerous small areas; while “focal” means only a few or single small lesions or changes. All of the changes have been grouped as closely as possible:

### Diffuse

All Animals

Horse

Cow

Pig

Sheep/Goat

Dog

Cat

Diffuse combined with locally extensive or multifocal.

Locally extensive.

Focal, Multifocal.

Disseminated usually refers to being all over the body.

## ROUGH SKETCHES OF LUNG LESIONS

### DIFFUSE \* All

1. Enlarged,  
red, wet  
lung.



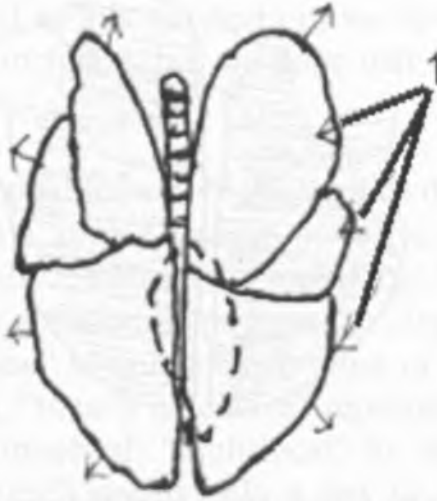
### Pulmonary Congestion and Edema

Almost any aged animal lung is enlarged and wet, slightly firm, unilaterally, bilaterally, or scattered anywhere in lung. May be more in one area than another. Froth may be prominent feature in trachea, bronchi, or nasally. Emphysema may be marked or absent, euthanasia solutions often noted by smell. Alcohol solutions, if given slowly, can cause more congestion and edema than other types when given I.V., and the odor is often diagnostic for the alcohol diluent.

Excessive froth, even to exiting the nostrils, may be seen in animals euthanized by electricity because serum leaks into alveoli at death, with elastic lung recoil forcing air out after death, and beating the air and serum into a froth. **Without a cause, it is almost always an artifact.**

It should be remembered that after death, with rigor mortis in muscles and gas production in the bowel, fluid and blood will follow gravity and space and commonly seem to fill the lung and liver. When seen on histological study, the fluid and blood should NOT be called congestion (hyperemia). This is probably one of the most commonly misinterpreted lesion of the lungs. WP 256

## DIFFUSE \* All

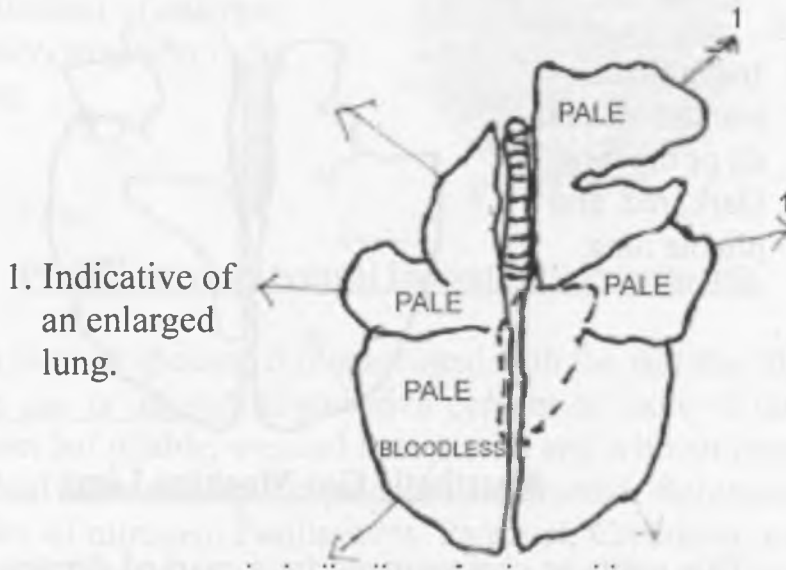


1. Enlarged, mottled, wet, red, yellow, tan lung.

### Enlarged, Tan Lung (Heart Failure Lung)

The lung is diffusely affected with various shades of red. It is wet and enlarged when it is acute. It is various shades of light yellow to tan and even brown when chronic, in mottled patterns scattered in the lung. Of course, a heart lesion should be found either grossly or histologically. Thrombi may be found in the scattered vessels. WP 2714

## DIFFUSE \* All



## Anemia

In the lung, as in other body tissues, this is represented by a diffusely pale lung organ.

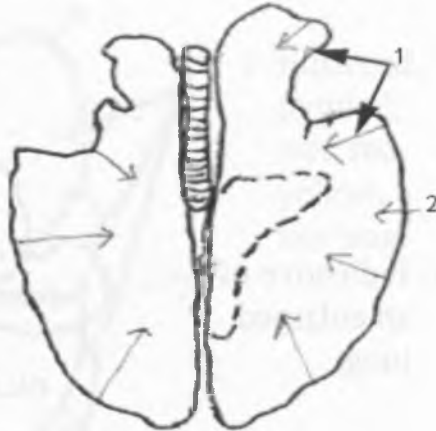
Edema may be present if the condition is at least several hours old and time has allowed revolumization of the remaining blood to occur.

Gastrointestinal exsanguination by parasites should be suspected (*H. contortus*, etc.), or bleeding ulcers if no other apparent cause is seen; remembering that in sheep, like rats deserting a sinking ship, the nematodes may not be present in the abomasums at the actual time of necropsy. WP 2720



## DIFFUSE \* All

1. Indication for marked atelectasis of the lung.
2. Dark, red, and pliable lung.

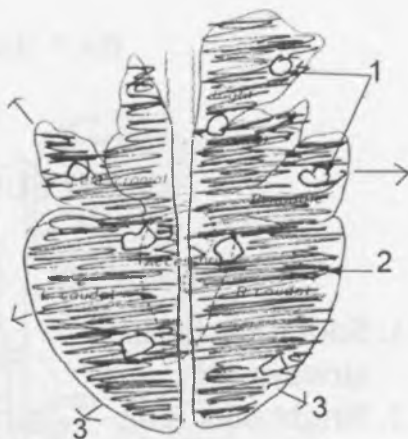


### Anesthetic Gas Machine Lung

This entity is characterized by a marked decrease in lung size overall, with the lung being pliable and dark red. It may fail to float or just barely float. It occurs in all species and all cases have a history of the animal being on a gas anesthetic machine in which the residual  $N_2$  in the lung has been replaced by metabolizable anesthetic gas. Subsequently, with any hindrance of respiratory movement by drug or tight bandaging after removal from the machine, the gas will be absorbed with subsequent collapse of the lung. This has not been seen in the ox. WP 419

## DIFFUSE \* All

1. Scattered unaffected lobules.
2. Diffuse alveolar lining cell proliferative pneumonia.
3. Indication of enlarged, meaty, wet, heavy lung.



### Proliferative (Atypical Interstitial) Pneumonia

This is seen in most species. It is associated with the fact that 80% of eructated rumen gas is inhaled. Eighty-five percent or more of the lungs are enlarged, firm but pliable, wet and heavy, with and without emphysema. A few individual lobules may be pale and unaffected. Substances such as higher oxides of nitrogen, Perilla mint, Paraquat, Crotalaria, and Ipomeanol from moldy sweet potatoes, can all damage the alveolar lining cells, to be replaced by the proliferative type II pneumocytes (proliferative pneumonia). Similar disease occurs in non-ruminants, thought to be caused by the excretion of the toxic materials through the lung. Atropine in high doses (1 gm/cow) can be curative if given early. This proliferation is noted as a diffuse alveolar lining cell proliferation and is differentiated from the adenomatoid reaction, which is a very focal proliferation of alveolar lining cells in association with any chronic lesions, parasites, and foreign bodies in the lung. Adeomatosis is a specific neoplastic disease of sheep. WP 345

**Horse:** The cause in young horses is not explained, but access to *Crotalaria* sp. plant toxins can cause this lesion in adult horses.

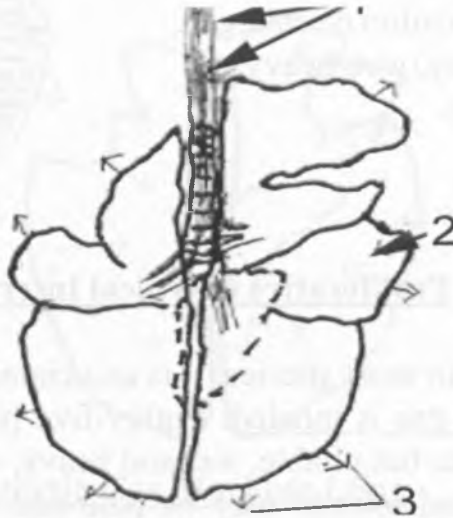
**Ox:** A change in pasture from poor to good quality is often in the history.

**Goat:** A common historic feature in affected goats is the availability of lime for ingestion.

**Pig:** Ipomeanol from moldy sweet potatoes and *Crotalaria* seeds are a common cause in pigs.

## **DIFFUSE \* All**

1. Soot particles in airways.
2. Bright pink lung.
3. Indication for enlarged lung.



## **Smoke Inhalation**

The lungs may be emphysematous or not, with black soot particles anywhere along the entire tracheobronchial tree—or even deeper in the airways. A definite odor of fire-damaged building material is present, and the entire carcass may demonstrate a bright pink discoloration similar to most light-colored tissues due to CO (carbon monoxide) poisoning. WP 11508

## DIFFUSE \* All



### Acute Pulmonary Edema

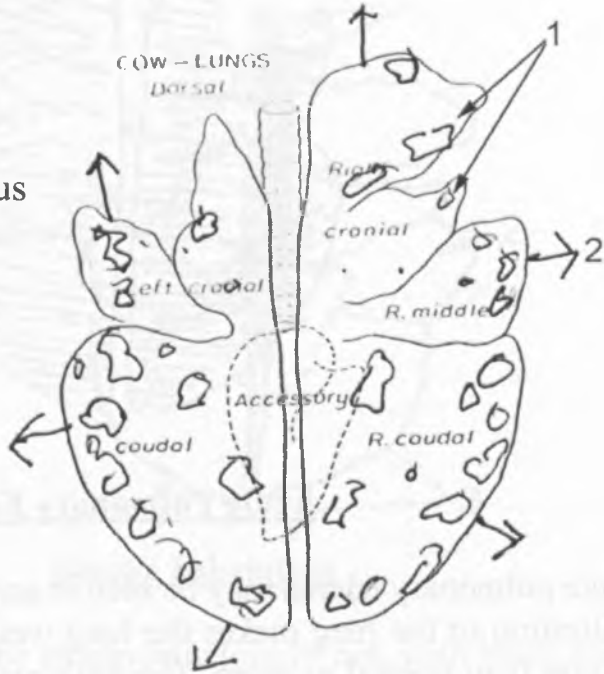
Acute pulmonary edema may be seen in any age of animal. The diffuse infiltration of the lung makes the lung wet and heavy, even 3–5 times heavier than normal or more. The lung may be only slightly enlarged. Edema is much more prominent than congestion. WP 1398

**Ox:** Heart failure is caused by white muscle disease (vitamin E/selenium responsive disease); heart anomalies and vegetative endocarditis are also common causes. Hypersensitivity associated with some vaccines, and even their own milk let-down in susceptible Jerseys and Guernseys, are commonly seen.

A note of caution is that many animals and other species, especially cattle, may die slowly and develop remarkably dark and wet lungs as a terminal artifact which must be ruled out.

## DIFFUSE \* All

1. Emphysematous bullae.
2. Indication of emphysematous enlargement.

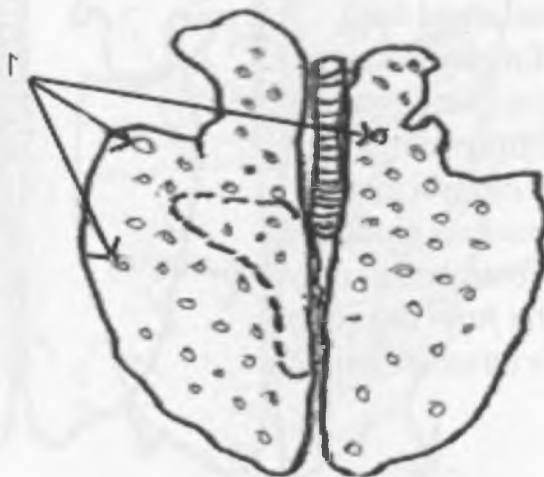


### Pulmonary Emphysema

Pulmonary emphysema occurs in almost any age animal. It appears as air-filled bubbles (pockets) of air scattered in various lobes, and septae with adjacent collapsed red-purple parenchyma. Without a history of dyspnea (breathing difficulty), it should just be considered a terminal artifact because it is so common in most species, especially the ox, but rare in the sheep and goat. WP 10148

## DIFFUSE \* HORSE

1. Multifocal firm, gray, glistening foci on cut section of mucoid bronchiolitis.



### Mucoid Bronchiolitis: Heaves (Acute Heaves)

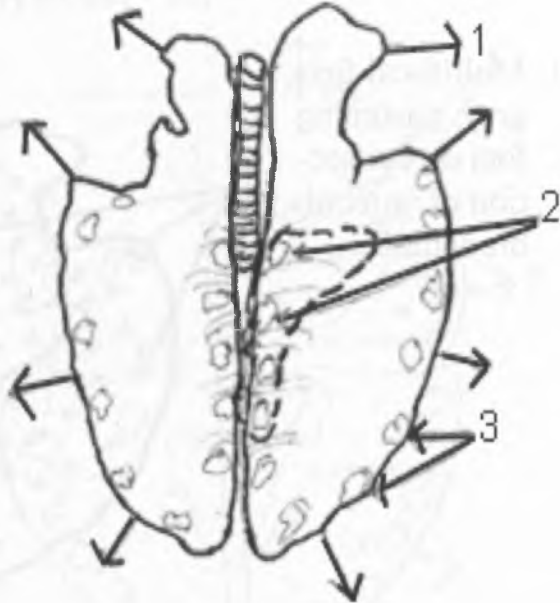
Heaves, acute or chronic, affects adult horses and is considered a chronic problem associated with sensitivity to feedstuffs. Alveolar emphysema, or chronic heaves, is the end stage and the most often recognized form.

Lung can be smaller than normal, normal size, or enlarged and spongy, diffusely.

The cut sections in acute heaves (mucoid bronchiolitis) show multifocal, gray, glistening prominent airways, mucus-filled acutely, and chronic inflammatory cells, and smooth muscle cell hypertrophy/hyperplasia when chronic. Emphysema results when inspired air cannot be pushed out past the mucus via elastic lung recoil, or the elastic recoil itself is defective. Both forms of heaves are usually considered to be chronic clinically.  
WP 163, 164

## DIFFUSE \* HORSE

1. Indication for an enlarged lung.
2. Emphysematous foci between rib impressions.
3. Emphysematous pockets along the ventral edge may be from heaves or a terminal artifact.

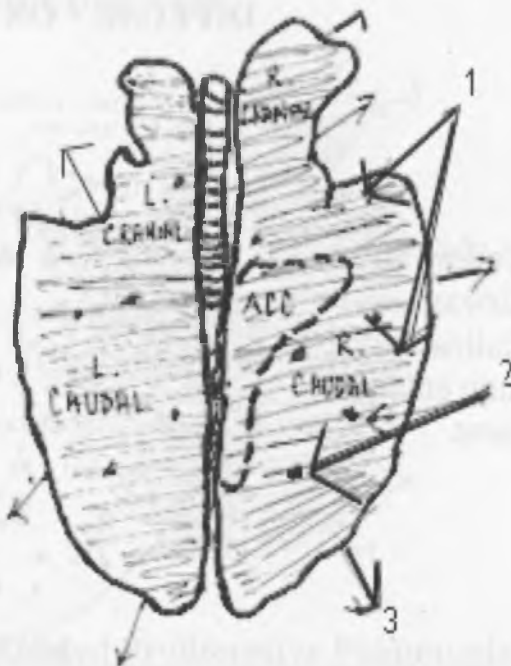


### Chronic Alveolar Emphysema

Chronic alveolar emphysema occurs mostly in adult and older horses. Clinically, they often have a double expiratory grunt. The lung is characteristically enlarged throughout, often having large air-filled bullae along the ventral and dorsal borders of the diaphragmatic lobes. Bullae may be scattered in the lung. This is considered to be the more chronic form of heaves, and by the time this occurs the mucus and chronic inflammatory cell response have usually disappeared, leaving only the inflated air spaces. Some call this an example of chronic obstructive pulmonary disease (COPD). WP 1995

## DIFFUSE \* HORSE

1. Diffusely dark red, swollen firm lung.
2. Scattered focal necrosis.
3. Indication for a diffusely enlarged lung.



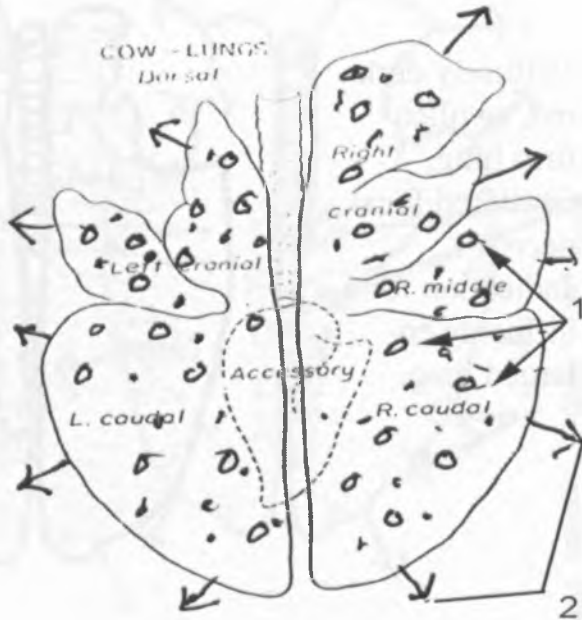
### Equine Herpes I Viral Pneumonia of Foals

This occurs mostly in foals aborted in the last trimester of pregnancy. The lungs are diffusely dark red, enlarged, and actually firmer than the slight firmness of the normal fetal lung. Often the lungs may show rib impressions due to their increased size from the pneumonia. Few or many white, 1–2 mm foci of necrosis may be scattered in the lung and liver. Intranuclear inclusions are common in areas of necrosis, especially in the adrenal gland. Equine herpes 5, multinodular pulmonary fibrosis virus causes large 1–4 cm opaque, white, uniquely soft masses and streaks of fibrosis in some horses with intranuclear inclusions. WP 6105



## DIFFUSE \* OX

1. Yellow affected airways.
2. Indication of lung enlargement.

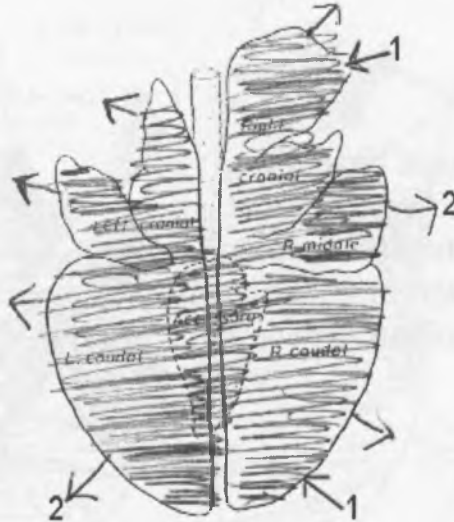


## Bovine Asthma

Adult Guernsey and Jerseys, less in Holsteins, may show an enlarged soft lung with prominent, yellow discoloring of edematous large and small airways and tracheal mucosa. Eosinophils predominate when acute, and more smooth muscle hypertrophy when chronic. Animals cough excessively, with a drop in milk production, usually in autumn and early winter. Bovine asthma is usually caused by moldy red clover, green chop, or silage being fed. Exposed humans also tend to cough. WP 2684, 344

## DIFFUSE \* OX

1. Diffuse alveolar lining cell proliferation.
2. Indication for enlarged lung.

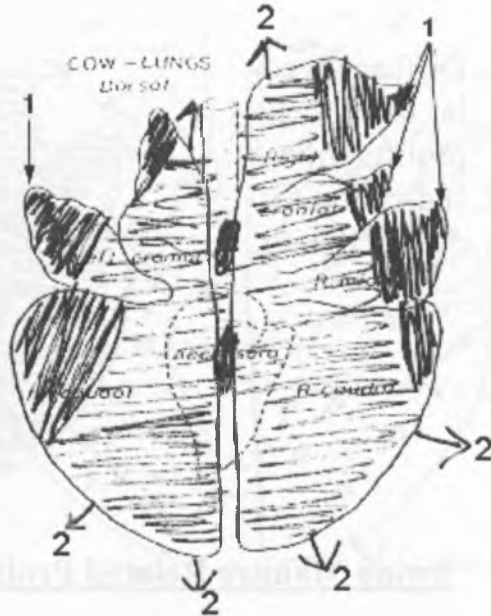


### Swine Manure Related Proliferative Pneumonia

This pneumonia occurs in young adult cattle with access to dried swine manure. It causes a diffuse, wet, heavy, slightly firm lung, with or without emphysema. Histologically, alveolar lining cells proliferate 24–48 hours after exposure. Many larval forms of incidental swine ascarids are scattered. The ascarid larvae are evidence of the young animals having eaten the high nitrogen-containing feces. This allows the rumen production of the higher oxides of nitrogen to be part of the 80% of eructated rumen gas which is inhaled normally by ruminants. Atropine in high doses is therapeutic, if timely. The alveolar lining cell proliferation (proliferative pneumonia) is of course diffuse and due to the higher oxides of nitrogen inhaled. It is not evidence for humeral response to the swine ascarid larvae. WP 2555, 2556, 353

## DIFFUSE & LOCALLY EXTENSIVE \* OX

1. Chronic enzootic bronchopneumonia.
2. Acute diffuse secondary alveolar lining cell proliferative pneumonia.



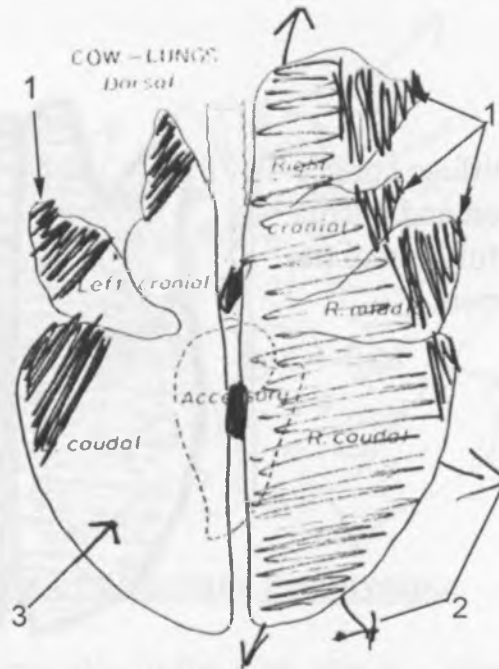
### Secondary Proliferative Pneumonia of Calves

This almost always occurs 21 days after an outbreak of enzootic pneumonia in calves. Severe dyspnea is characteristic clinically. The primary lesion is in the cranioventral lobes as expected, but the secondary proliferative pneumonia affects the entire remaining lobes as a diffuse, firm, meaty, heavy, wet lung, often slightly tan and enlarged, due to the alveolar lining cell proliferation of most alveoli if they live 2–3 days. The cranioventral lobes are not affected with the proliferation, as they have been preempted with the enzootic pneumonia. There is no explanation for this entity, and atropine is necessary for treatment.

WP 358, 356

## LOCALLY EXTENSIVE & DIFFUSE \* OX

1. Chronic enzootic bronchopneumonia.
2. Secondary unilateral proliferative pneumonia of alveolar lining cells.
3. Absolutely normal lung.

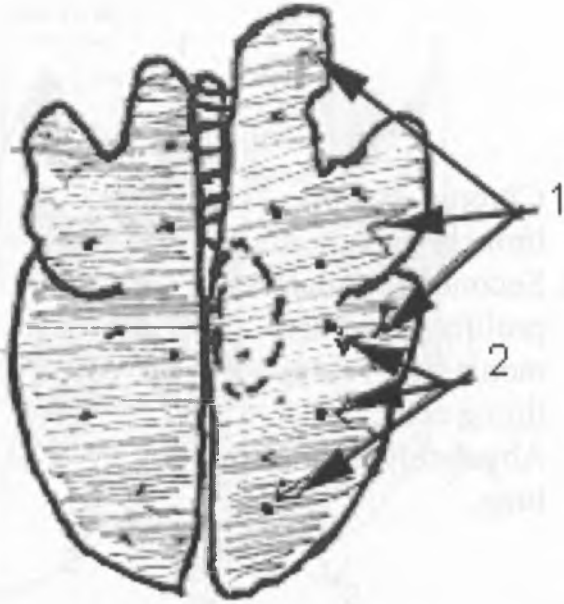


### Unilateral Secondary Proliferative Pneumonia of Calves

This is similar to the secondary, proliferative pneumonia of calves, but it is even more bizarre, as it is only unilateral, with proliferation secondary to the chronic enzootic pneumonia of calves. Again, no explanation is forthcoming as to why it occurs at all and why only on one side. WP 361, 2645

## DIFFUSE & MULTIFOCAL\* PIG, DOG, CAT

1. Diffuse congestion and edema.
2. Multimodal necrosis.

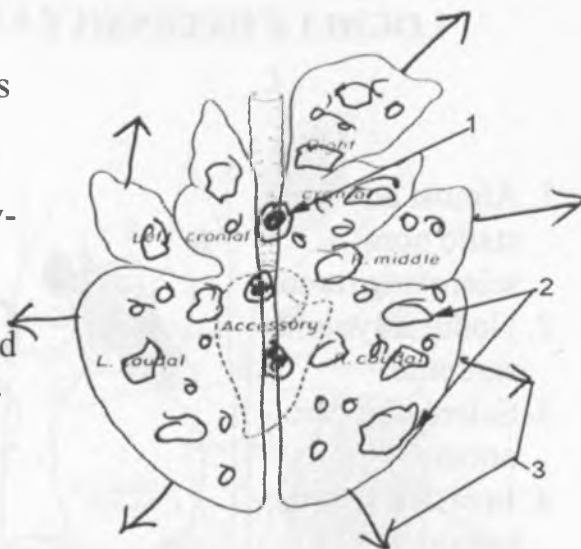


### Acute Toxoplasmosis

An enlarged, uncollapsed, edematous, congested lung, with many 1–2 mm pale foci of focal necrosis is quite characteristic for acute toxoplasmosis in the pig, and sometimes in the dog and cat. Impression smears can be diagnostic. WP 2703

## LOCALLY EXTENSIVE \* ALL

1. Emphysematous lymph nodes.
2. Large, air-filled bullae of emphysema.
3. Indications for greatly distended emphysematous lung.



### Terminal Pulmonary Emphysema

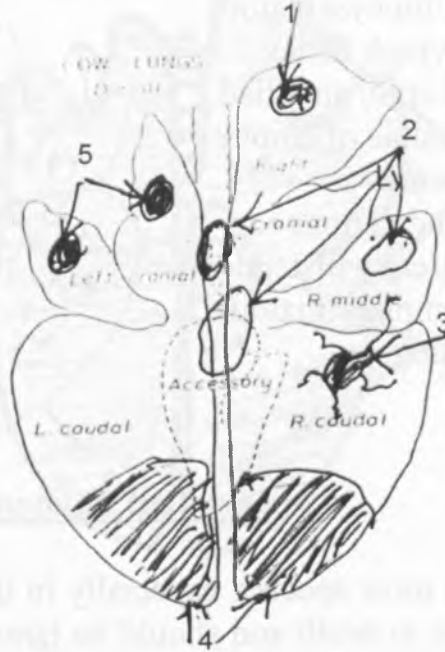
In most species, especially in the cow, this is a common finding at death and should be ignored when a cause is not found, suspected, or has no antemortem history of dyspnea. Lungs are enlarged and soft with air throughout, within large pockets (bullae), or diffusely without bullae. The bullae, when cut, collapse and leave the immediate adjacent parenchyma compressed and dark. Local nodes may have air pockets in them if the cow dies slowly. Air pockets can also be found under the visceral and mediastinal pleura and even under the skin dorsally.

**Ox:** This is most common and extensive in the ox.

**Dog and Cat:** It is common in the dog and cat, more so than in the **horse**, but rare in the **sheep, goat, and pig**. WP 340

## LOCALLY EXTENSIVE \* ALL

1. Almost any meta-  
static non-  
sclerosing tumor.
2. Nodular lympho-  
sarcoma.
3. Sclerosing carci-  
noma.
4. Invasive lympho-  
sarcoma.
5. Metastatic mela-  
noma.

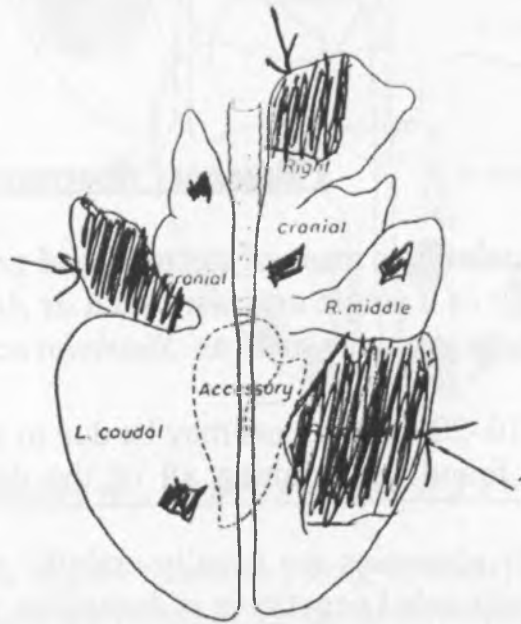


### Primary or Metastatic Tumors

Not too many of these metastatic tumors can be diagnosed grossly. Of course, melanomas are the major exception, since they are black. Nodes can be involved with any tumor. Lymphosarcoma may nodularly or locally extensively affect the lung in the dog and cow, Adenocarcinomas are often sclerosing. Connective tissue fingers may extend irregularly around adenocarcinomas, especially in the cow with genital adenocarcinoma. Hemangiosarcomas should also be considered. WP 2107, 2779

## LOCALLY EXTENSIVE \* ALL

1. Indication for collapsed lung or area of lung.

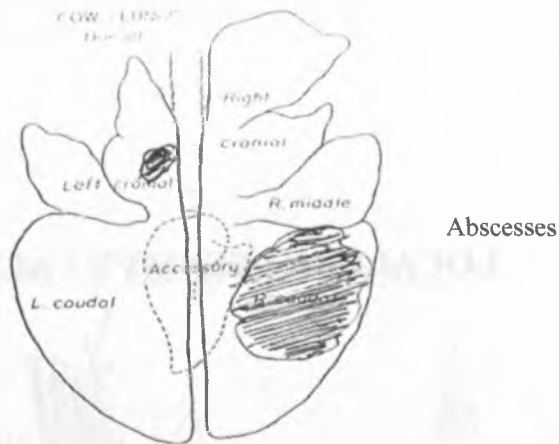


### Fetal Atelectasis

Atelectasis mainly affects newborn animals. Small, scattered, discrete lobular patterned foci, or even large parts of entire lobes, may be pliable, red, dark, and depressed but not elevated nor firm. These may regress within hours, days, or weeks. Blocking mucus may often be found in the airways, but not always. Small, multifocal, slightly raised, firm, pneumonic foci with a central area of necrosis or pus may be easily confused with this lesion. In adult animals this is usually seen associated with pneumonia. WP 1607



## MULTIFOCAL – LOCALLY EXTENSIVE \*



### Pulmonary Abscesses

Considered a coalescing mass of necrotic and purulent debris. One or many may be the result of a single organism such as *Arcanobacterium pyogenes* or abscess-forming organisms such as *Staphylococcus* spp. and *Streptococcus* spp.

A single large (10–20 cm) abscess may be due to a single inhaled foreign body, which may be found by straining all of the debris through a kitchen-type strainer.

Multiple smaller abscesses are usually embolic and may be from a ruptured liver abscess, right-sided vegetative endocarditis, or from mammary or jugular veins. If severely septic and foul smelling, black or dark, septic joints or ligaments should be looked for as to source. Often, pulmonary abscesses associated with foreign bodies are the major causes for empyema (pyothorax) when they rupture into the pleural cavity.

*Arcanobacterium* was formerly referred to as *Actinomyces*, and even earlier, *Corynebacterium pyogenes*. In many species, firm, yellow, caseous, sometimes mineralized abscesses often with similar abscessed local nodes, should be considered tuberculosis until proven otherwise. WP 19118

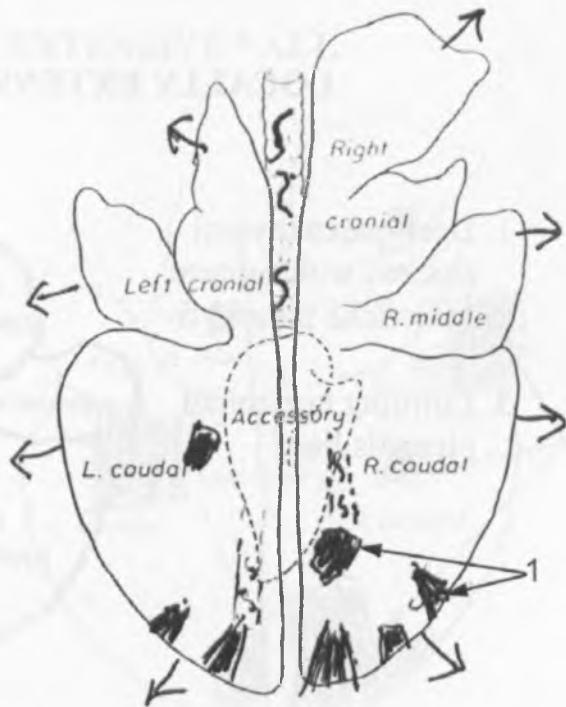
**Horse:** *Rhodococcus* sp. WP 212, 213, 214, 12933, 13606, 21347

**Ox:** Abscesses are commonly seen in the ox.

**Sheep:** Almost as common as in the ox.

Associated with *Rhodococcus* abscess in young horses in a lung or elsewhere, a well-marked firm lung of *Pneumocystis* infection is thought to be secondary to immune exhaustion and is seen in other species. The lung abscess of *Rhodococcus* in the horse will usually be obvious, but the patches of firm lung around the *Pneumocystis* are usually overlooked. Silver stains are needed for diagnosis. Normal horses may have a few organisms of *Pneumocystis* scattered in the lungs. WP 13606

1. Parasitic pneumonia with nematodes in airways.



**Lungworm (*Dictyocaulus* sp., *Metastrongylus* sp.) Pneumonia**

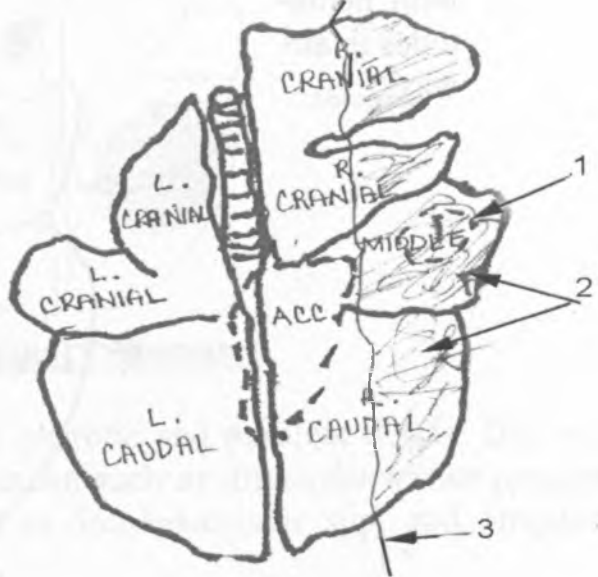
Lungworm pneumonia is mainly found in young animals, but is sometimes found in adults. Most are found in the distal airways of diaphragmatic lobes. After death, many parasites migrate to other airways and trachea, which often causes diagnostic problems for the unwary. White nematodes may practically fill airways and cause pale, discrete, firm, white, square or triangular foci at the caudal edge or near the surface of slightly swollen emphysematous parenchyma. The entire lung may or may not be emphysematous. Scattered areas of pneumonia caused by *Muellarius* spp. may also be found in the parenchyma, which can cause confusion with these two parasites when seen in sheep, but the *Muellarius* spp. are microscopic. WP 925

**Horse:** Many cases of *D. arnfeldi* are found in horses housed with donkeys.

**Cats:** *Aleurostrongylus abstrusus* are usually seen grossly just under the pleura and are usually associated with hypertrophy arterioles histologically.

## LOCALLY EXTENSIVE \* ALL

1. Deep parenchymal abscess with rupture.
2. Infectious pleural fibrin.
3. Limiting horizontal pleuritis line.

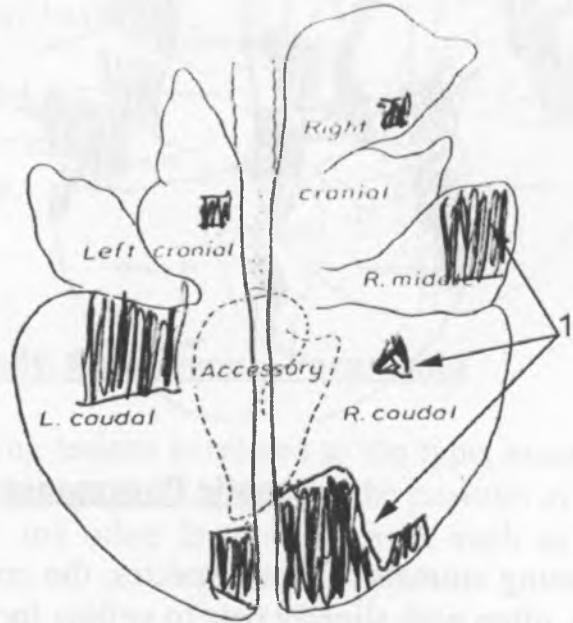


### Purulent Pleuritis

The slight to severe fibrinopurulent infection of the pleural cavity in the ox, or any other animal, should make one consider first a ruptured lung abscess from inhalation pneumonia, as they are the most likely cause in 85% or better of cases. Certainly, other causes such as septic extension down the neck, as from esophageal punctures, may be causative. The pleural involvement may be limited to one side only and have a dorsal-horizontal fluid line of involvement especially in the horse that is constantly standing. The abscess may have been quite small and could have healed over, if time permitted, so such a lesion is often overlooked. Many organisms may be found, including *Nocardia* sp., but they are usually not the primary cause. This is also known as pyothorax, suppurative pleuritis, or empyema. WP 112, 115, 576

## LOCALLY EXTENSIVE \* ALL

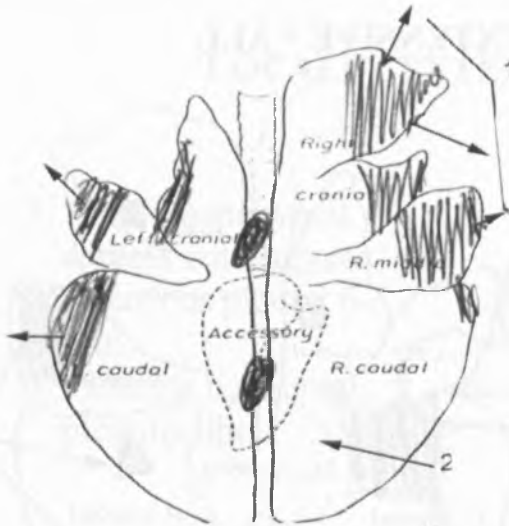
1. Discrete locally extensive gritty parenchyma.



### Pulmonary Mineralization

Almost any age can be affected by pulmonary mineralization, depending on the cause. Large portions of entire lobes or small 1 cm or less (or any size in between) of firm, gritty lung, when cut, have a dry, mineralized, gritty cut surface scattered in the lung, often in dorsal diaphragmatic lobes. Most cases are associated with excessive vitamin D use, but a vitamin A deficiency can also cause this, as well as uremia is able to. Affected areas in most instances have a straight border suggesting its vascular relationship. In most species, including cattle, the hypercalcemia of malignancy can be causative and is sometimes seen in X disease in cattle caused by chlorinated naphthelene with its vitamin A deficiency component. WP 201, 467

## LOCALLY EXTENSIVE \* ALL



1. Indicator of slight pneumonic enlargement.
2. Emphysema may or may not be present.

### Enzootic Pneumonia of the Young

In young animals of many species, the cranioventral lobes may be red and firm, often with slightly pale to yellow foci of necrosis and suppurative surface fibrin or fibrinous adhesions to the rib cage. With chronicity, the affected areas are paler, less firm, and more pliable. The fibrin deposits may be replaced by connective tissue, and local nodes may be enlarged but not always as expected. Affected lobules, even as they respond to therapy, may be atelectatic and remain nonfunctional for months or years. Many cases are often difficult to differentiate from mild inhalation pneumonia.

**Horse:** *Rhodococcus* spp. abscesses and secondary *Pneumocystis carini* can be enzootic and recognized by palpating firm areas of connective tissue around the abscesses. WP 212, 213, 214, 12933, 13606, 21347

**Ox:** In the young ox, atelectasis may be prominent, with dilated bronchioles filled with mucoid or purulent debris, purulent bronchiectasis, or just appear dilated because of its relative degree of atelectasis. *Mannheimia* and *Pasteurella* are common isolates, but heavy antibiotic use often prevents any agent from being isolated on culture. Viruses are commonly considered a predisposing factor, but viruses are rarely isolated by cultures, FA techniques, presence of inclusion bodies, or immunohistochemistry. WP 357

**Pig:** *Bordetella* sp. and *Mycoplasma* sp. are likely candidates of enzootic pneumonia for pigs.

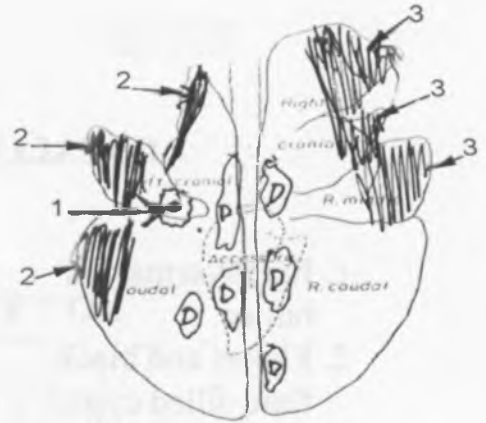
**Sheep and Ox:** *Pasteurella* sp. is a common isolate.

**Dog:** *Bordetella* sp. and distemper virus are commonly found.

**Cat:** Feline herpes virus infection with necrosis and inclusions are found.

## LOCALLY EXTENSIVE \* ALL

1. A single oat hull may be found anywhere in the lung.
  2. A cow down on one side may only have the lesion in the down lung.
  3. A standing calf with laryngeal white muscle disease damage may have milk inhalation lesions cranioventrally.
- D. Sheep on back when drenched.



### Inhalation (Foreign Body, Gangrenous) Pneumonia

The type and distribution of the lung lesions is related to the type, amount, virulence of the organisms, of the foreign body involved, the position of the animal when inhalation occurred, and other factors involved, such as the resistance status of the host.

**Horse:** Shipping fever (from head held high by the halter) is a common cause, but improper drenching technique can also be involved. Mineral oil inhalation is a special category in the horse, associated usually with mineral oil drenching for colic, and has scattered, large pale areas of parenchyma with a hemorrhagic core. Emphysema is common. Oil droplets may be seen floating in the formalin or oil smears on brown (not white) paper towels as an aid to diagnosis. Unless expectorant was given in the oil inhalation cases, the clinician may not be aware of the iatrogenic error because oil stops the gag and coughing reflexes. WP 2568, 168

**Ox:** Dorsal decumbency may cause the lesion primarily in the dorsal portions of any or all lobes. Most inhalation (gangrenous) pneumonias are very septic. Misdirected drenching is a common cause. In beef calves, white muscle disease is seen because muscles of deglutition are often affected.

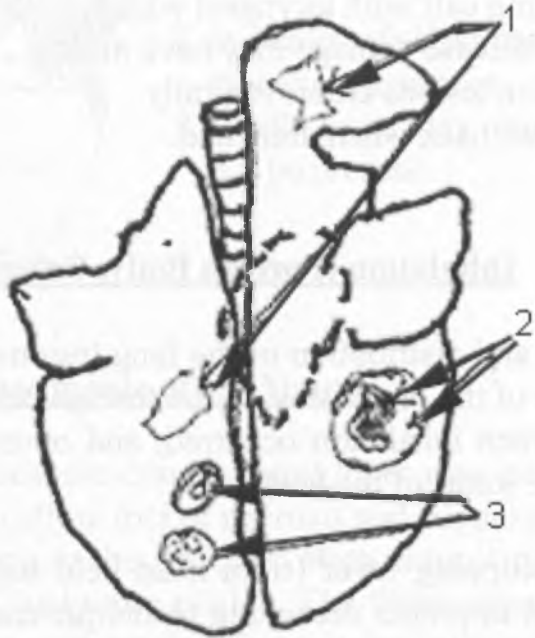
**Sheep/Goat:** Improper drenching is a common cause, and white muscle disease of the muscles of deglutition should be considered early because it commonly affects the muscles of deglutition.

**Cat:** Like the horse, and for the same reason, mineral oil is a common cause of inhalant foreign body.

**Note:** Mineral oil and such products decrease or even prevent the gag reflexes which stops coughing as a defense mechanism.

## LOCALLY EXTENSIVE \* ALL

1. Emphysematous bullae.
2. Flukes and black fluid-filled cysts.
3. Intermediate tape-worm cysts.

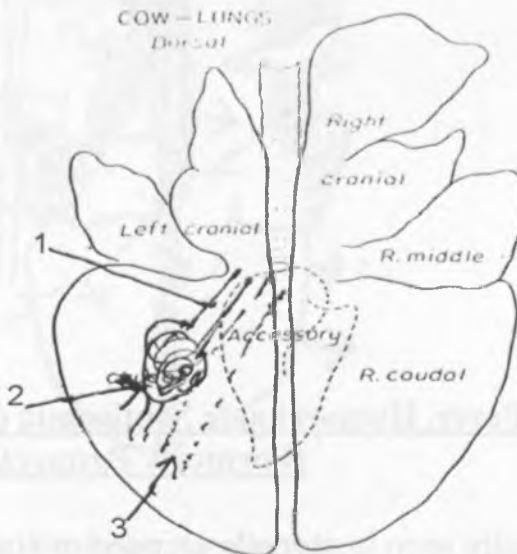


### Pulmonary Cysts

Various sized and shaped air pockets, without their own structured wall, are usually emphysematous bullae most commonly seen in the ox and less so in the horse and cat. They are considered rare in the sheep because of their relatively tough lungs compared to other domestic species. These are usually terminal artifacts. More regular, rounded, one centimeter or more, clear or cloudy, fluid-filled cysts with their own distinct thin wall are usually intermediate tapeworm cysts with intact or degenerate parasites present. In the horse and the cow, similar cysts may represent echinococcosis. The fluke *Paragonimus kellicoti* makes black fluid and debris and parasite-containing cysts somewhat similar to aberrant liver flukes in the sheep, dogs, and others. A few or many cysts may be seen in any one host. Congenital bronchial cysts are also seen in some dogs. WP 13568

## LOCALLY EXTENSIVE \* OX

1. Pulmonary artery.
2. Septic thrombus.
3. Airway.



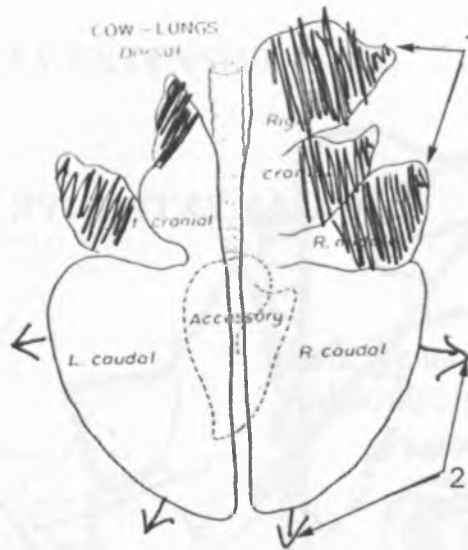
### Respiratory Tract Exsanguination Hemorrhage

This is associated with cattle from lung hemorrhage, usually with epistaxis. Blood noted in airways and associated alveoli and, if swallowed, even in the esophagus, abomasum, and forestomachs. A large septic thrombus in one branch of the pulmonary artery is usually embolic from the vena cava over a liver abscess. Entrance of blood to the airway is usually direct from the septic thrombus eroding through the blocked branch of the pulmonary artery, then through the bronchial walls into the bronchial lumen. Surprisingly, the thrombosed vessel may be palpated more readily than it can be seen. Infarcts are not common in the lung due to its dual blood supply. WP 3110, 313



## LOCALLY EXTENSIVE \* OX

1. Fibrinopurulent pneumonia.
2. Emphysematous enlargement.



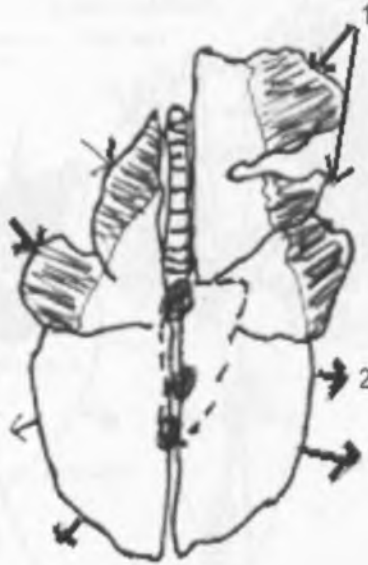
### Shipping Fever, Hemorrhagic Septicemia (*Mannheimia*) Pneumonia (Formerly *Pasturella*)

This is usually seen in recently shipped or stressed adults. It may affect a single lobe or, more usually, several lobes bilaterally and cranioventrally, with extension to the pleura and even the rib cage. Affected areas are locally extensive, firm, but friable mostly because of released inflammatory cell enzymes, with surface fibrin, sometimes hemorrhage, and edema. The septae are thickened at times with opaque, pale fibrin and clotted serum, giving the septae a marbled appearance. Note that resident animals that have not been shipped can also be affected by contact with affected shipped cattle, probably because of the increased bacterial virulence associated with animal passage in the bacteria themselves. *Pasteurella multocida* and *Mannheimia hemolytica* are common isolates, with more hemorrhage seen with *M. hemolytica*. Diarrhea is a common feature with some pneumonias, causing some diagnostic problems clinically. Emphysema may be mild to severe.

*Actinobacillus* sp. (*Hemophilus*) pneumonia is similar but usually more fibrinous, hemorrhagic and scattered, and not usually as cranioventral as *Mannheimia* pneumonia. WP 2659, 11

## LOCALLY EXTENSIVE \* OX

1. Dark pliable resolving bronchopneumonia.
2. Enlargement.



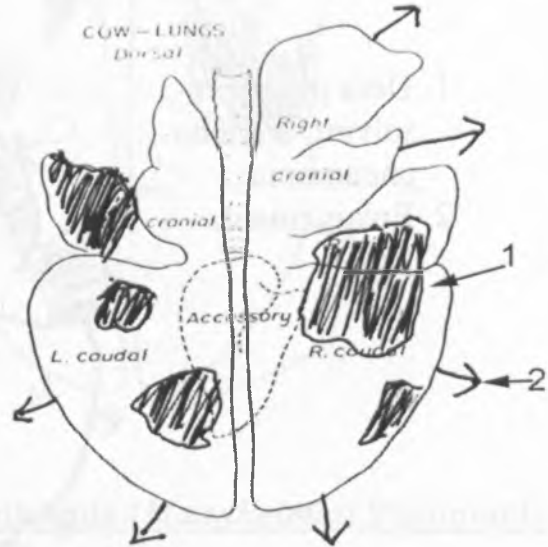
### Resolving Bronchopneumonia

This is usually seen in older calves or young adults, and affected lobes are usually collapsed, slightly firm, and pliable. Fibrous adhesions are common to adjacent lobes and rib cage. Local nodes are enlarged, suggesting chronicity.

Scattered thick-wall abscesses are sometimes present. Emphysema is usually severe. Cultures are often sterile or nonspecific. Acute changes such as edema, congestion, hemorrhage, or necrotic debris and fibrin may not be present. As all enzootic pneumonias are not fatal, this entity must exist but is rarely recognized. These lesions may persist for several years and erroneously be considered an integral part of other pulmonary diseases which occur at a later times, for instance, as with bovine respiratory syncytial (BRSV) virus pneumonia. Enlarged nodes are expected. WP 2624

## LOCALLY EXTENSIVE \* OX, PIG

1. Locally extensive areas of fibrino-hemorrhagic pneumonia.
2. Indication of enlarged lung with emphysema.



### **Hemophilus spp. (Actinobacillus) Pneumonia**

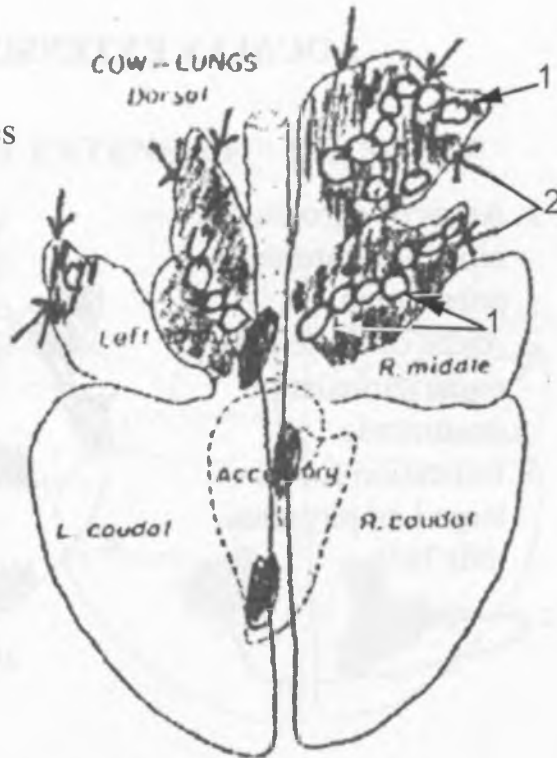
This is usually seen in young adults. It is scattered, locally extensive, in areas of acute fibrinohemorrhagic pneumonia, not limited to the cranioventral lobes as is more characteristic of shipping fever pneumonia. The lesions in this disease are also more hemorrhagic than in shipping fever pneumonia. Emphysema is usually considered severe and is associated with the severe clinical dyspnea.

**Ox:** This is the characteristic description for the ox with *Mannheimia* spp.

**Pig:** This is the characteristic description for the pig with *Actinobacillus pleuropneumonia*. WP 671, 672

## LOCALLY EXTENSIVE \* OX

1. Dilated bronchioles of bronchiectasis.
2. Chronic enzootic pneumonia.

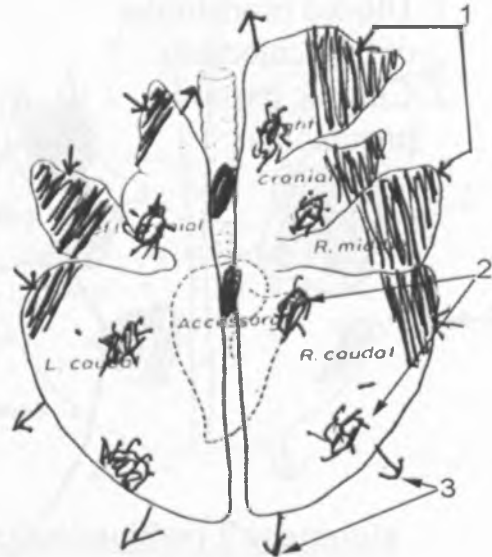


### Suppurative (Mucopurulent) Bronchiectasis

Suppurative (mucopurulent) bronchiectasis most commonly occurs in calves that are months to several years old, or are young adults. Cranioventral lobes usually have rounded, cystic fluid or more solid abscess pockets in a crooked line following airways. Surrounding parenchyma often collapsed from pressure and disuse, giving airways a relative increase in size. This is a common sequel to enzootic pneumonia and even mild shipping fever. Cultures may be sterile due to antibiotic use. WP 1429, 19874

## LOCALLY EXTENSIVE \* OX

1. Areas of chronic cranioventral broncho-pneumonia.
2. Areas of acute secondary inhalation pneumonia.
3. Indication for enlarged emphysematous lung.

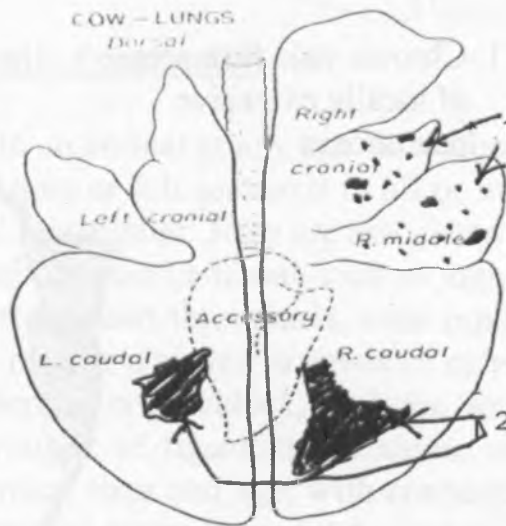


### Secondary Inhalation Pneumonia

This is mostly seen in adult cattle with residual, chronic, enzootic pneumonia, and the animal has been placed down or upside-down for displaced abomasal surgery or other procedures. The chronic pneumonias are usually cranioventral and firm but pliable, with purulent debris in airways. Scattered areas of a more acute nature (from the debris which fell gravitationally when the animal was upside down) are found in the upper lung. Emphysema is common and extensive. Enlarged draining nodes are noted because of the chronic resolving cranioventral lobes. WP 375

## LOCALLY EXTENSIVE \* OX, SHEEP

1. Melanosis: small.
2. Melanosis: large.

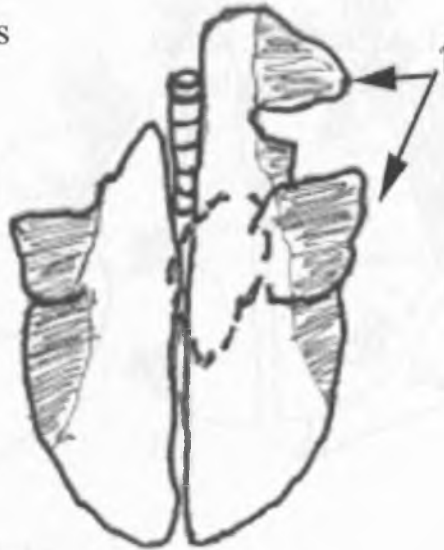


### Melanosis

Melanosis may also be multifocal. It is seen mainly in young ox and sheep, as it is a congenital lesion especially in black-faced sheep which often have the pigment in the heart and meninges. It appears as small black spots (multifocal) of pigmented parenchyma in the airway, from 1 mm to patches of 4–5 cm or larger (locally extensive), anywhere in the lung. They are not space-occupying masses and should not be called tumors. WP 2719

## **LOCALLY EXTENSIVE \* SHEEP, GOAT**

1. Chronic pale firm areas of locally extensive pneumonia.

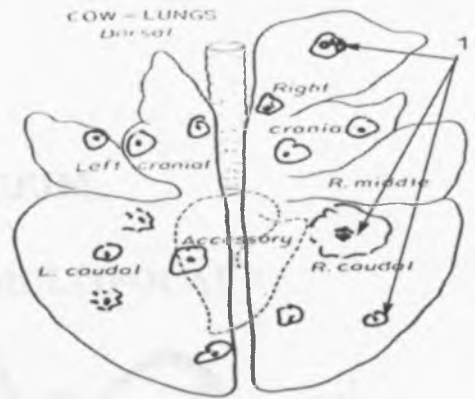


### **CAE Virus Pneumonia (Maedi, Marsh's Progressive Pneumonia) (Chronic Obstructive Pulmonary Disease)**

This is a large, locally extensive pneumonia in older sheep. It is usually cranioventral, pale, and very firm. Histologically, chronic inflammatory cells, such as mononuclear phagocytes and lymphocytes, predominate, with the formation of many lymphoid follicles. Some alveolar lining cells may also be proliferated. Local nodes are enlarged. Chronic inflammatory cell foci may be scattered in the bone marrow. Some cases of chronic enzootic pneumonia may be similar grossly and histologically. Initially jaagsiekte (adenomatosis) was confused with this entity, but jaagsiekte is a neoplastic disease. WP 2588, 16349

## MULTIFOCAL \* ALL

### 1. Embolic abscesses.



### Embolic Pneumonia

Embolic pneumonia can occur in an animal at any age. Multifocal hemorrhagic and/or purulent foci are seen or felt scattered in all or most lobes. These are usually round and not rectangular. They are associated with vessels. Sources for these septic emboli must be found, such as jugular, mammary veins, caudal vena cava, right heart thrombosis, even ruptured liver abscesses. Umbilical infections may be a source in newborn calves via the ductus venosus. Excessively septic, brown/black, embolic foci have a most likely source in open wounds of ligaments, tendons, and joints. Many septic lung lesions of various sizes and age, with connective build-up, suggests a prolonged shower, but many foci of the same size and development suggests a liver abscess rupture. In most species, embolic material from the right heart does not cause infarctions unless extensively distributed, as the lung has a dual blood supply with the bronchial artery supplying the oxygen to help prevent the infarction. Initially, while the acute liver rupture may kill quickly, the lung may not have a septic odor, but if the necropsy is delayed the lung may have a definite septic odor as embolic organisms may be rapidly multiplying. WP 257, 2660

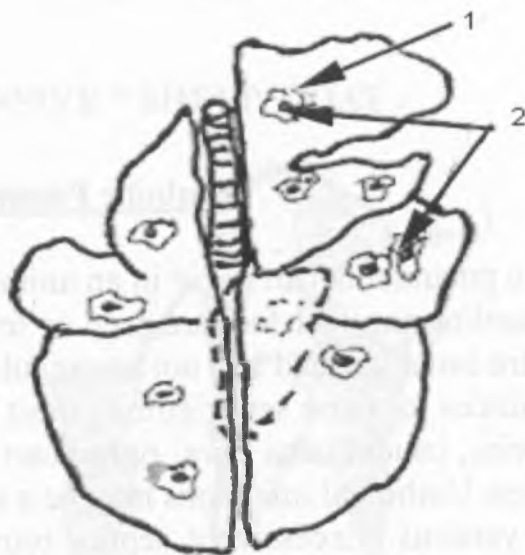
**Ox:** While seen in most species, it is most common in the ox, with jugular vein, mammary veins, and especially vena caval thrombi, as well as liver abscess ruptures into the vena cava and chronic joint disease, as mentioned above.

**Swine:** Vegetative endocarditis in the pig is a common cause. In most species, embolic material from the right heart does not cause lung infarcts unless they are quite extensive and block many vessels. The dual blood supply via the bronchial artery with oxygenated blood helps prevent the infarctions.



## MULTIFOCAL \* ALL

1. Peripheral red zone.
2. Center of necrotic debris.



### Mycotic Infection

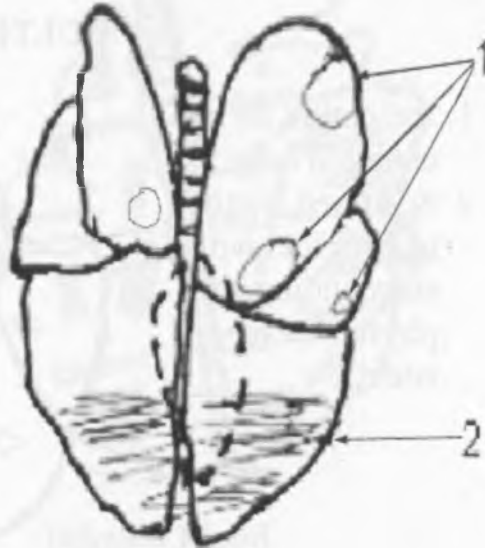
Few to many, dark, firm foci of necrosis and debris, with a target-like outline having a definite red peripheral zone of congestion, are highly suggestive of mycotic infection. Often, surface fibrin over the lesion is unique.

**Ox:** These occur with some regularity in ruptured liver abscesses.

**Equine:** These can occur in horses with intestinal salmonellosis. The large, granulomatous mycotic lung lesions with a target-like appearance are to be differentiated from the tiny, just palpable lesions of the mold *Polymicrosporone faeni*. *Polymicrosporone faeni* is the cause of farmer's lung in man and a similar disease in Guernsey and Jersey cattle, mainly. WP 332

## LOCALLY EXTENSIVE & MULTIFOCAL \*

1. Almost any possibility exists.
2. Probable lymphosarcoma.

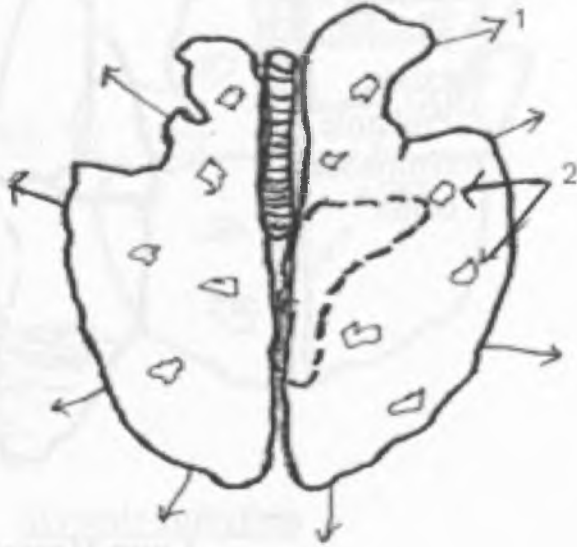


### Lung Masses

Masses scattered in the lung can be of many different causes, but in the young, infections, granulomas, or abscesses should first be considered. In older animals, tumors should first be considered. A third quality for masses is that almost any lump should be considered possibly lymphosarcoma, no matter what the animal's age. Lymphosarcoma may be locally extensive in the caudal diaphragmatic lobes. All masses must be examined grossly, culturally as needed, and histologically to be definitive. WP 271, 221, 321

## MULTIFOCAL \* HORSE

1. Indication for an enlarged lung.
2. Scattered, slightly firm foci with surrounding emphysema and edema.



### Equine Herpes Virus 5 Pneumonia

This pneumonia mostly occurs in foals and young horses. It consists of multifocally scattered, 3–5 cm, pale, slightly dark, irregular, slightly firm, foci in all lobes, with moderate edema and emphysema around these pale foci. When seen chronically, multiple foci of fibrosis have formed into streaks that continue from one surface of a lobe completely through the lobe to form nodules of connective tissue on each surface affected. It is presently determined to be a gamma herpes virus. The question often asked by the prosector is, “What kind of pneumonia is this? It’s different!” Some even consider it unique. WP 2723

## MULTIFOCAL \* ALL

1. Inhaled  
blood.

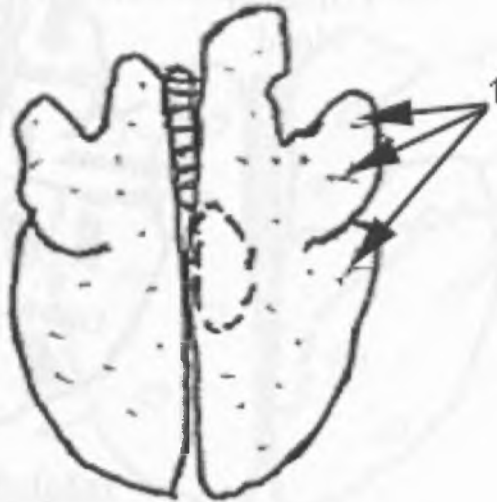


### Inhaled Blood

Inhaled blood is a common lesion, usually associated with trauma-induced frank hemorrhage of the upper respiratory tract. The trachea may not have blood present. The affected lobes have linear areas of blood, as parts of total lobules may be filled with blood. It is described as a common actual lesion in the racing horse, but this pathologist has never seen one to his knowledge. WP 19703

## MULTIFOCAL \* ALL

1. Multiple  
petechia.

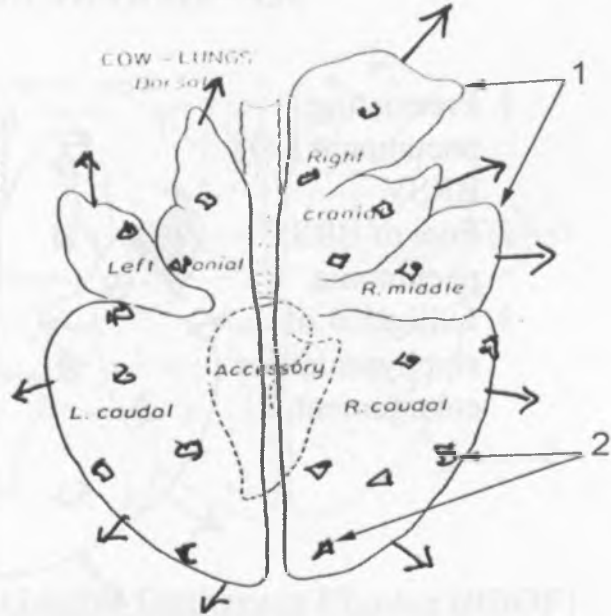


### Multiple Pulmonary Petechiation

Multiple pulmonary petechiation involves multiple scattered foci of several millimeter hemorrhages throughout the lungs. Histological sections of parasites are usually found near these hemorrhagic foci. In piglets, these are usually due to ascarid larvae migration associated with their clinical "thumps." Similar findings in puppies may be due to larval hookworms or larval ascarids. Septicemia in most species can also cause petechia with and without DIC (disseminated intravascular coagulation).  
WP 6275

## MULTIFOCAL \* OX

1. Diffuse emphysema.
2. Green, yellow foci of dead lung-worms.



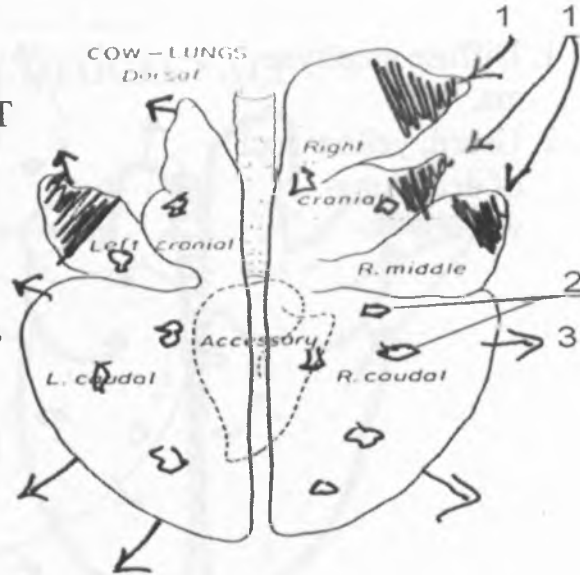
### Post Fogging Pneumonitis

Post fogging pneumonitis is seen in housed cattle that are face-fogged with insecticide to kill face and horn flies, etc., prior to allowing them to pasture for the day. Insecticide is inhaled and kills the lungworm parasites *in situ*. Farmers notice coughing and marked drop in milk production, mostly in the Guernseys and Jerseys. Lungs are enlarged with emphysema and many irregular 3–10 mm yellow to greenish nodules throughout the lung grossly, with parasite sections, with many eosinophiles histologically associated with the parasites.

WP 2563, 2564

## MULTIFOCAL \* OX

1. Preexisting pneumonia NOT BRSV.
2. Foci of BRSV pneumonia.
3. Indication of emphysematous enlargement.

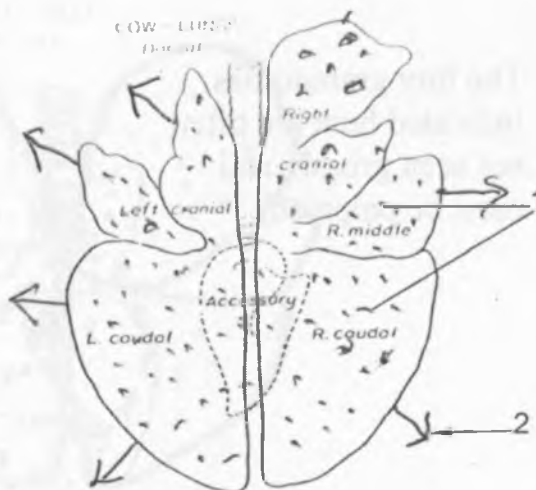


### Bovine Respiratory Syncytial Virus Pneumonia (BRSV)

BRSV pneumonia is seen in young adult and older cows usually as an acute, high fever outbreak. Multifocal areas of scattered pneumonias are the primary feature of this disease, with some emphysema 1–5 cm in the lung. Emphysema, even to bullae formation, may be common. Epithelial syncytia (epithelial multinucleated giant cells) are seen histologically in airways and alveoli. However, a very common error made by many is to consider the chronic enzootic pneumonia lesions found in the cranioventral lobes as caused by the virus. These enzootic pneumonia lesions are sometimes seen with this viral infection, but in fact the chronic cranioventral pneumonic foci are only residual lesions of a preexisting bronchopneumonia and not related to this acute viral pneumonia. It has been reported in sheep. WP 2623, 338

## MULTIFOCAL \* OX

1. Enlarged prominent bronchioles.
2. Indicative of lung enlargement.



### Bovine Bronchiolitis Obliterans Fibrosa (BBOF) (Silo Filler's Disease in Man)

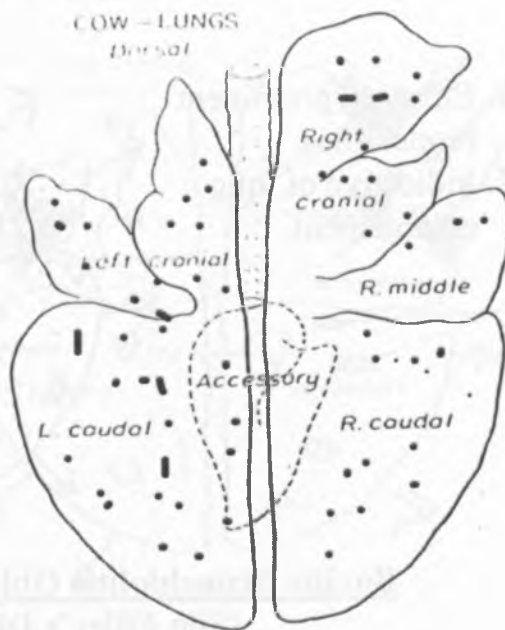
Adult cattle with BBOF usually have diffusely pale, enlarged, light weight lung, with hundreds of pale gray 2–5 mm foci scattered throughout the entire lung.

Histologically, the gray foci are bronchioles which are blocked by in-growth of connective tissue polyp formation. This is associated with chronically inhaled higher oxides of nitrogen which react with the deep lung moisture, forming weak nitric acid which burns the type I pneumocytes and bronchiolar lining cells lining the airways. As in man, this is the chronic lesion of the inhaled fumes and is seen in late winter when the animals are on silage from the bottom of the silo or old hay. Ruminants inhale up to 80% of eructated rumen gas and this may be the primary cause of this disease. Again, as in man, in this chronic form, corticosteroids are necessary to prevent the connective tissue polyp development into this chronic form as they use it in man, and atropine is used in cattle for the acute proliferative form of the acute toxicity. WP 273, 2559



## MULTIFOCAL \* OX

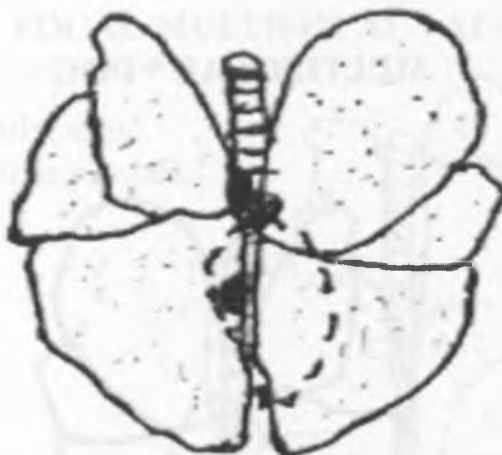
The tiny granulomas indicated here are often not seen grossly and must be palpated.



### Multifocal Granulomatous Pneumonia of Cattle (Farmer's Lung of Man)

This multifocal granulomatous disease is associated with spores or portions of *Polymicrosporon faeni* seen in their centers. Histologically, it is seen mostly in adult Guernsey and Jersey cows but also in Holsteins. Grossly, the lung may appear normal, but on palpation hundreds or more 2–5 mm nodules can be felt throughout the lung. The high morbidity is associated with dyspnea and drop in milk production. The moldy silage or hay must be eliminated. Humans can have the same disease, often mistaken for tuberculosis on radiographs. They are quite different from the more common, larger, classical, target-like lesions of *Aspergillosis* and similar organisms. WP 1990, 1992

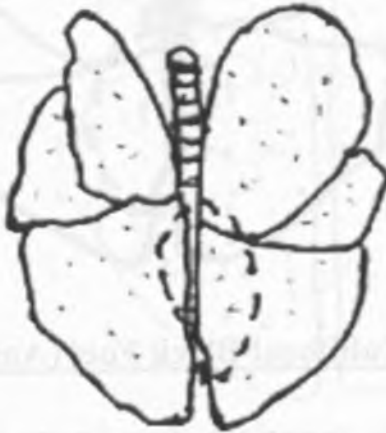
## MULTIFOCAL \* DOG



### Multifocal Black Foci (Anthracosis)

Many animals, especially dogs living in urban areas, have a very fine black dust-like appearance to the costal pleura, with more concentrated black pigment in the regional lymph nodes. Sometimes it is more concentrated along the sharper edges of the lobes and may not be seen on adjacent visceral pleura. This dust accumulation, called anthracosis (pneumoconiosis), is much less visible on lobes that are emphysematous at death, although it is still present. It is usually not a significant finding. Local lymph nodes are usually black. Chronic cases may show distinct black lines of apparent reorganization of the pigment, with attempts of elimination by migration to local nodes. WP 2717, 15079

## MULTIFOCAL \* DOG

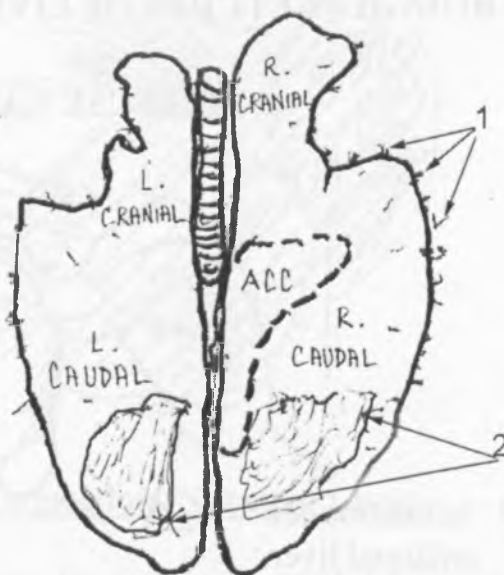


### Multifocal Pulmonary Mineralization (Ossification) in Dogs

Multifocal, hard, 1–3 mm nodules scattered in the dog's lung are pieces of bone. Histologically, it is without cellular reaction and considered to be an incidental finding without a known cause. Although once reported as being inhaled, this is not likely without a foreign body or other reaction associated with them.

## FOCAL/MULTIFOCAL \* ALL

1. Fibrovascular tags.
2. Fibrovascular plaques.

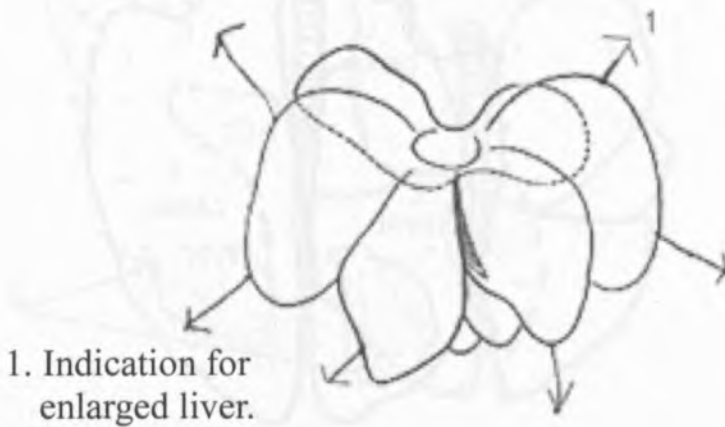


### Pleural Fibrovascular Tags and Plaques

Small 1–5 mm, with some larger pale to red, fibrillary, tissue tags on the ventral edges of the various lung lobes as well as 1–2 mm or slightly thicker, connective tissue, irregular plaques up to 20–30 cm on the pleural surface of the diaphragmatic lobes are common findings in animals, especially the ox and horse. Histologically, they consist of fibrovascular tissue without appreciable inflammatory cells. Their early presence can be seen often on the dark surface of aborted calves. WP 11513

## ROUGH SKETCHES OF LIVER LESIONS

### DIFFUSE \* ALL

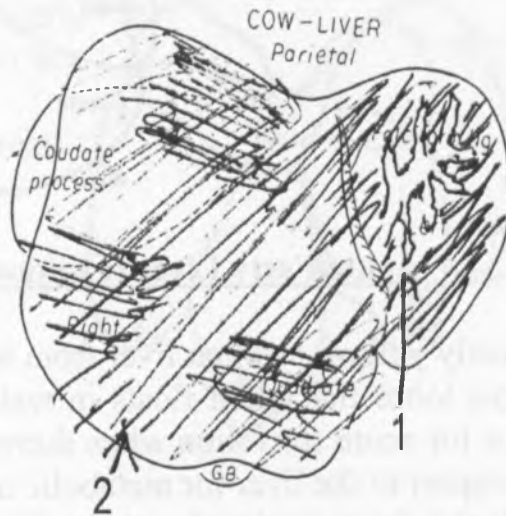


### Diffusely Enlarged Liver (Hepatomegaly)

A bloody, slightly enlarged liver should make one consider terminal hypostasis with rigor mortis and intestinal gas pushing mobile blood to the liver from muscle and bowel, respectively. This should not be called congestion or hyperemia, as it is also found in the lung, kidney, and other tissues, as it is only a terminal or postmortem change. An enlarged firm liver with round edges may be pale or congested with diffuse neoplastic infiltration by myeloid or lymphoid neoplasia, even mastocytosis. A large firm liver with a shiny mottled cut surface can be hepatic amyloidosis, especially in the horse, with the distinction being the marked firmness of amyloidosis. One instance of **soft** amyloidosis has been seen, but in no way explained. WP 7006, 6771

## DIFFUSE \* All

1. Arborization.
2. Mottled by chronic congestion.



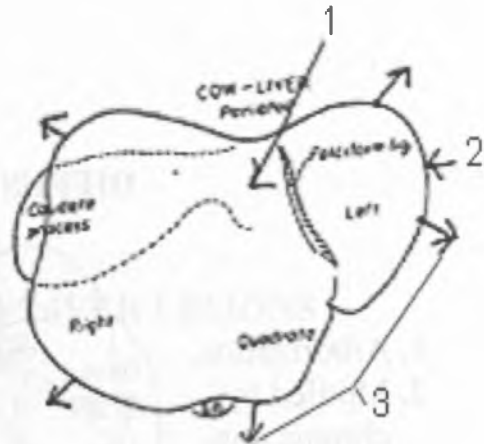
### Chronic Passive Congestion (Nutmeg) Liver

With nutmeg liver, the entire liver may be slightly enlarged, with a definite mottled dark and light parenchyma. Large areas of dark may be mixed with light areas of parenchyma. With time, the color change may be more uniform, with pale areas appearing arborized (tree form) in a darker background. The cause must be looked for in the form of any cause of resistance to forward blood flow such as: severe lung disease, heart anomalies, chronic heart disease, or caudal vena cava thrombosis. This can be seen in any age animal, including the fetus. It has often been noticed that even with aortic stenosis or other left heart lesions, this hepatic passive congestion may be found without any evidence of expected lung involvement. One explanation is that the liver is more susceptible to passive congestion, but that, with time, the lung would also be involved in such delayed cases.

WP 383

## DIFFUSE \* All

1. Diffuse fatty liver.
2. Rounded edges.
3. Indication of enlargement.



### Acute Fatty Liver (Lipidosis)

A diffuse, distinctly yellow, swollen liver from any species, with rounded edges of most lobes that often floats in water or formalin fixative, may be evidence for acute starvation when there is body fat available to mobilize for transport to the liver for metabolic use, but not chronic starvation in which the fat is depleted and the liver shrinks and darkens. Some metabolic diseases, such as diabetes mellitus and pregnancy diseases, may be a cause as well as many toxins such as aflatoxin and phosphorus poisoning, and an upset in dietary fat metabolism in some animals. These livers are usually yellow and not just pale gray as in acute anemia.

**Horse:** With an unknown cause, this is very common in ponies.

**Ox:** Most of these are seen associated with ketosis, but the cause is often not known in others. A mineral imbalance is often suspect.

**Sheep:** Pregnancy disease is a common cause when multiple fetuses are present. The rumen may be reduced in size and function because of the enlarged uterus.

**Dog:** Diabetes mellitus is a common cause, as well as some toxins such as aflatoxin and phosphorus poisoning.

**Cat:** Diabetes, toxic, and metabolic problems are commonly seen.

WP 6122, 7006, 7008, 7572, 7580

## DIFFUSE \* All

1. Indication for slightly smaller liver than normal.
2. Very pale liver, relatively bloodless.

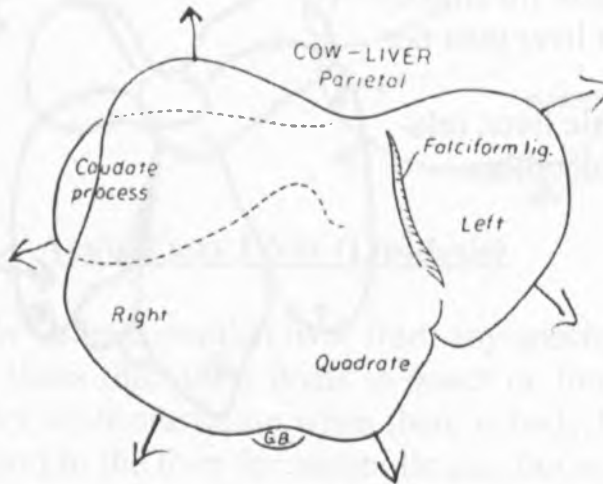


## Anemia

A diffuse pale liver from any species, often with a slight grayish tinge to the capsular surface, or a liver that sinks in water or formalin fixative, may be evidence for anemia. This evidence exists with or without an anemic carcass, as the animal dies too quickly to allow revolumizing of the blood and thus will not cause an anemic (pale) carcass, organs, tissues, or blood. WP 3089



## DIFFUSE \* ALL

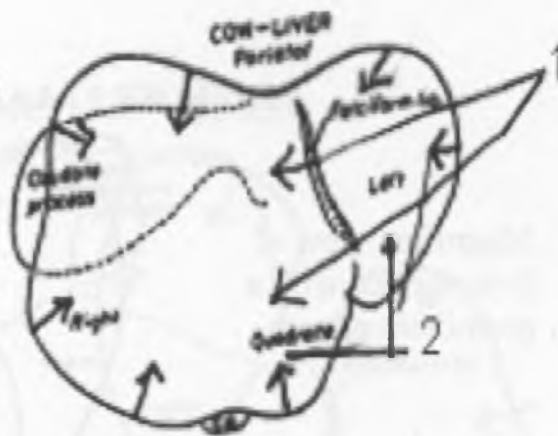


### Hepatic Hypostasis (Diffuse Bloody Liver)

At necropsy, a diffusely bloody liver is often an artifact or normal finding due to hypostasis because when the animal dies the blood has nowhere else to go. In a fresh necropsy, the amount of blood may even seem excessive to the neophyte pathologist. This also occurs in the lung, kidneys, and other tissues. Even histologically, observers may erroneously call it congestion. Of course, other lesions could cause these pathologic or physiologic changes and should be ruled out.

## DIFFUSE \* All

1. Small dark liver.
2. Indication of small liver.



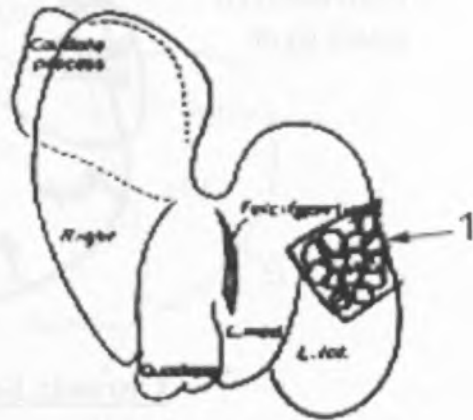
### Chronic Emaciation

A smaller and darker than normal liver in most species is evidence for chronic starvation, in which all body fat and liver fat has been utilized completely. If the liver is half of its normal size, then each cell and each lobule will be approximately half of its normal size histologically. The portal triad bile ducts in many animals may also be increased in number. The reduction in cell volume may suggest an increase in stroma, which should not be considered as absolute fibrosis.

**Horse:** By all means, check the horse's teeth for inability to chew properly and the colon wall for small strongyles (easily overlooked) if no other apparent reason is found to account for emaciation. The reduction in cell volume may suggest an increase of stroma and should not be considered as absolute fibrosis. WP 7008, 108, 109

## DIFFUSE \* ALL

1. Magnified view of liver surface with a prominent pattern.

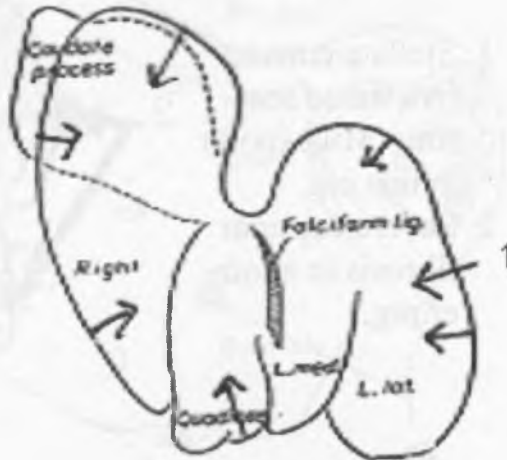


### Prominent Lobular Pattern

In the entire surface of the liver, each and every lobule appears distinctly separate, with a paler zone of liver lobule cord cells around a darker central vein area. There is also a slightly darker peripheral biliary triad area around the entire lobule. Although seen in many species sporadically, it is especially prominent in the **cat** and the **horse** and is considered normal. WP 1523

## DIFFUSE \* HORSE

1. Small, dark, flabby liver.



### Theiler's Disease (Serum Sickness)

A small, dark, sometimes pale, mottled yellow and dark liver which is often described as being very flabby, with a “wet dishrag” consistency, may be seen in jaundiced horses with nervous signs, even mania. Most, not all, have a history of a parenteral horse product injection within the last 30–90 days and is characteristic of “serum sickness” or Theiler's disease of horses. Histologically, the liver lesion is a massive liver necrosis. A cansatin virus has recently, in 2013, been isolated in this disease and is designated as TDAV, Theiler's Disease Associated Virus, a Flavivirus . WP 7103

## DIFFUSE \* PIG

1. Stellate connective tissue scarring (Milk spots) in one pig.
2. Severe capsular fibrosis in another pig.

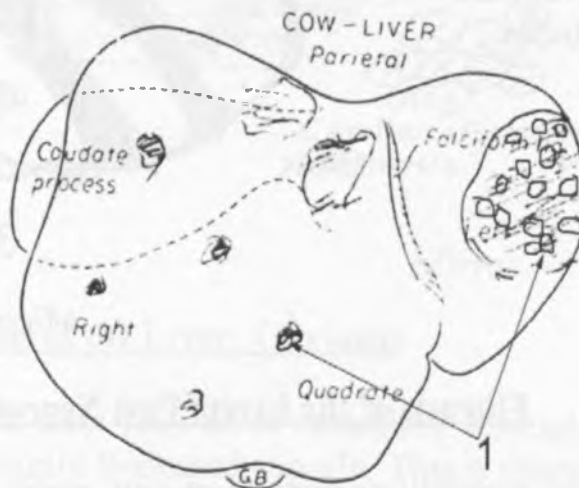


### Scattered Multifocal to Diffuse Hepatic Scarring (Ascarid-Induced Milk Spotted Liver)

In the pig, multiple pale-yellow spots of fine, wavy connective tissue lines radiating from the center are characteristic for *Ascaris lumbricoides* or *Stephanurus* spp. larval migration scarring throughout the liver, but best seen on the capsule. In some cases the capsule may be so severely involved that it is almost a completely white cover. It is surprising, even in severe cases, how few larvae may be found histologically. The scarring is often more severe with *Stephanurus*. WP 7303, 1364

## LOCALLY EXTENSIVE \* ALL

1. Hepatic autolysis with and without gas formation.



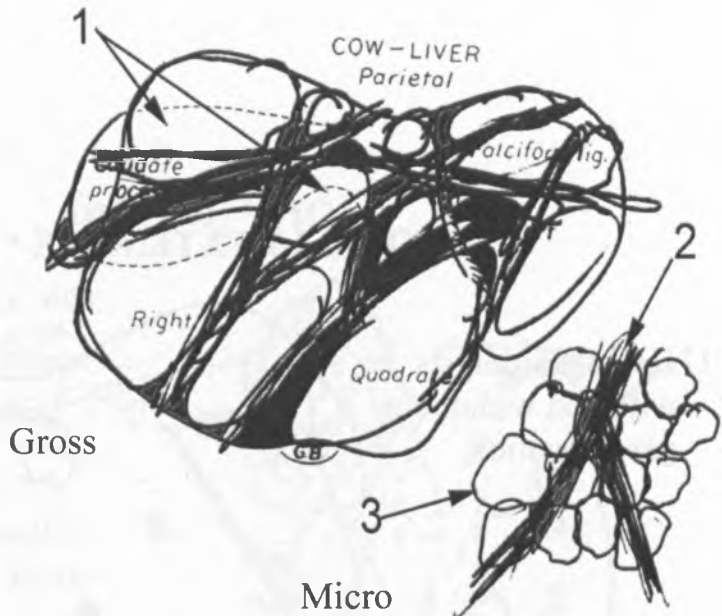
### Postmortem Decomposition

Pale, discrete areas of liver, often with many tiny gas bubbles or a few larger bubbles scattered in the pale areas, are usually the result of decomposition. Many bubbles closely involve the hilar area but can be located anywhere in the liver in ruminants and pigs. No inflammatory reaction around them is seen in these cases of postmortem decomposition. Most float in water. Some livers may be diffusely enlarged with decomposition, and very soft.

**Dog:** Somewhat similar pale distinct areas of liver, again near the hilus areas, are common in the dog and other species killed with alcoholic euthanasia solutions. These chemicals may back up into the liver, giving these areas a definite medicinal odor. WP 870, 7768

## LOCALLY EXTENSIVE \*

1. Unaffected parenchyma.
2. Replacement scarring.
3. Liver cord lobules.

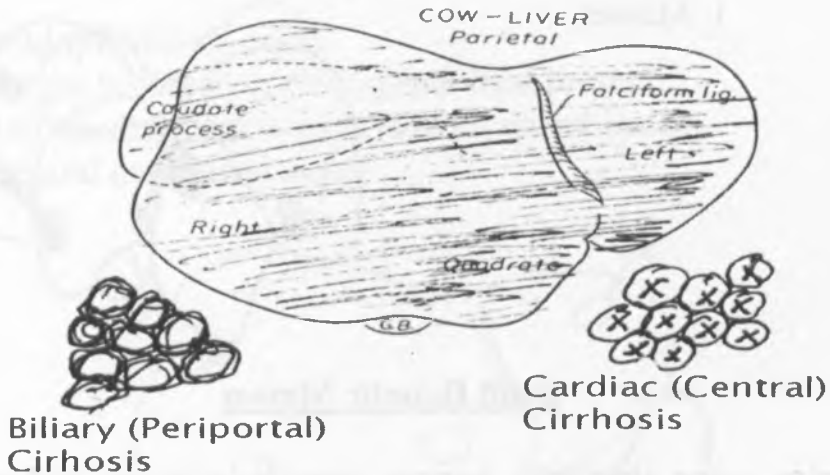


### Fibrosis of the Liver: Post Necrotic Scarring

A large, coarsely nodular liver with marked irregular bands or stellate scars of connective tissue scattered among various areas of regenerating liver lobules. The connective tissue is due to the repair of acute massive damage. Toxic effect of parenchyma by trauma or single-dose toxic effect affects only those areas supplied for the short period by the afferent vessels bringing the toxin to the liver. The long-term toxic effect associated with chronic exposure to a liver toxin causes cirrhosis, affecting the entire liver more uniformly. The regenerating lobular nodules usually have normal-type architecture, but greatly distorted. WP 3185, 108, 109

**NOTE: Regeneration is a function of amount of damage, about 15–20% at one time, and thus may or may not be present with either form of fibrosis.**

## DIFFUSE \* All



Micro

Micro

### Fibrosis of the Liver: Cirrhosis

A diffusely, uniformly firm liver may be of essentially normal size, or slightly enlarged with a slightly thickened capsule. This is characteristic for cirrhosis, which is defined as an increase of connective tissue in the same location to the relative same degree of each and every lobule from chronic repetitive damage. With heart disease or obstructive blood flow, the central vein area is involved and is called cardiac cirrhosis. With chronic biliary disease, and many different types of plant poisonings over time, the lesion is peripheral around the lobules and is called biliary cirrhosis. Livers with chronic biliary cirrhosis often give the liver a green tinge. WP 3185, 7044, 109

**Horse, Ox, Sheep, Goat:** Heart anomalies are a common cause, as well as vitamin E/Se responsive disease (white muscle disease), for cardiac cirrhosis and chronic plant poisonings such as pyrrolizidine for biliary cirrhosis.

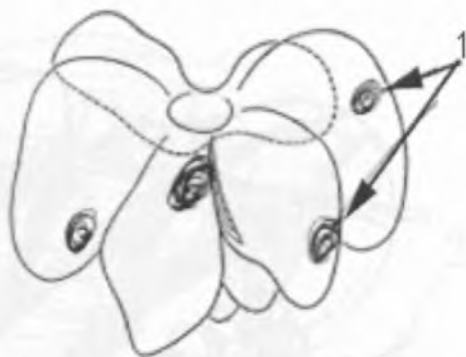
**Dog:** Heart anomalies in young dogs and chronic verrucous endocardiosis are common causes in older dogs.

**Note:** Regeneration is not necessarily involved unless enough liver is damaged at one time to reach the 15–20% threshold to stimulate regeneration.



## MULTIFOCAL TO EXTENSIVE \* ALL

### 1. Masses.



### Solid Hepatic Masses

Granulomas, some abscesses, tumors, parasitic nodules, and cysts tend to look alike in the gross and must be dissected, cultured, and histologically examined in order to be definitive. Many malignant tumors should suggest diligent search of the bowel, pancreas, and elsewhere to find the primary. Lymphosarcomas, in most species, often have yellow-green dry necrotic centers. Blood-filled, small 1–2 mm up to large 10–20 cm masses in most species are hemangiosarcomas and may be seen in the spleen, right atrium, and lungs. In young animals, masses should be close to the bottom of a differential list, with abscesses near the top. In older (tumor-aged) animals, tumor masses should be near the top of the differential list. A special note is that for many solid masses, lymphosarcoma should be on the list.

**Dog:** In the dog, and sometimes other animals, one or several solid pale, yellow cellular mass or masses, or even a liver-colored mass with no other lesion present in the liver, should be considered the common benign hepatoma. It is usually seen in tumor-aged dogs, usually does not have a capsule, and there is no compensatory (regenerative) need for it. It should not be called nodular hyperplasia. In performing many necropsies during a sabbatical leave in South America (Brazil and Argentina), very few cases of hepatoma were found in those animals compared to the number seen in the USA.

**Note for clarification:** The surgical removal of one thyroid lobe results in diffuse, not nodular, regeneration of the remaining lobe. In the woodchuck (easily done anatomically), and theoretically any other animal, if one half of the liver is removed surgically, the other half regenerates diffusely, not nodularly, which helps differentiate hyperplasia from neoplasia. WP 7267, 543

## LOCALLY EXTENSIVE \* ALL

1. Multiple biliary cysts.
2. Single biliary cyst, often green fluid present.
3. Intermediate tapeworm cyst with scolex inside.
4. Ventral mesenteric cysts.



### Hepatic Cysts

Hepatic cysts are clear, fluid-filled cysts, from a few millimeters to many centimeters, in or on the liver. In tumor-aged animals it should make one consider biliary tumors, especially if the fluid is green. Parasite cysts, such as intermediate tapeworm cysts, are quite common in many species, but usually in young animals.

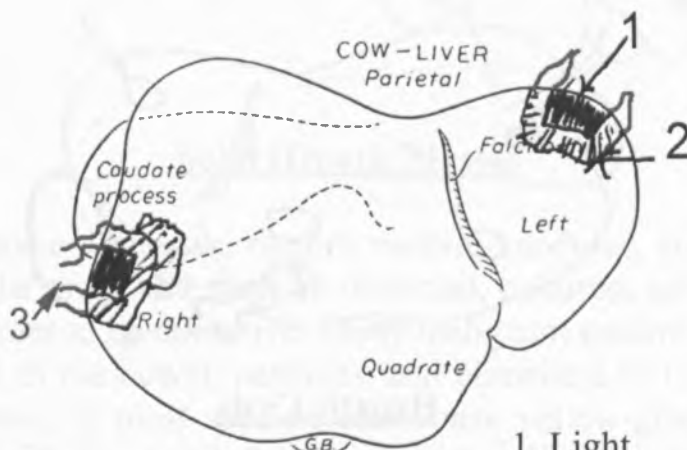
**Horse:** Sterile echinococcus, 1–3 cm cysts are common. WP 7131, 7219, 13569, 18244, 7395

**Ox:** A common finding, mainly in aborted calves, is large, 5–50 cm, clear or blood tinged, fluid-filled cysts apparently arising from the left visceral surfaces along the liver edges. Their exact origin is in dispute as to ventral mesenteric remnants or even the liver capsule. They are not biliary cysts.

**Sheep/Goat/Dog:** Black, fluid filled, 1–4 cm cysts with pieces of flukes (*P. kellicoti*).

**Cat:** Very common in the cat, but classification between tumors and possible simple obstruction is in question.

## LOCALLY EXTENSIVE \* OX



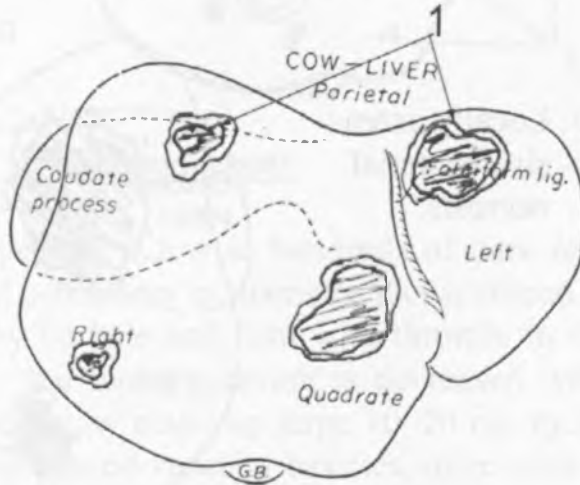
1. Light.
2. Dark yellow.
3. Fibrous adhesions.

### Tension Lipidosis

Often one to several discrete straight-edged, pale, fatty foci along the side or dorsal edges of the liver represent this lesion. It is usually seen in adults with an enlarged and hanging liver edge held by a slight amount of connective tissue that compromises the intrahepatic vessels to the area, causing the hypoxic change focally. Various color changes may be noted in one focus associated with the time affected. These have been called pseudoinfarcts. Most are seen in the ox but may be seen, rarely, in other species. Another form is associated with small vessels in the capsule, also seen in cattle. WP 296, 7024, 301, 299

## LOCALLY EXTENSIVE \* OX

### 1. Sequestered liver.



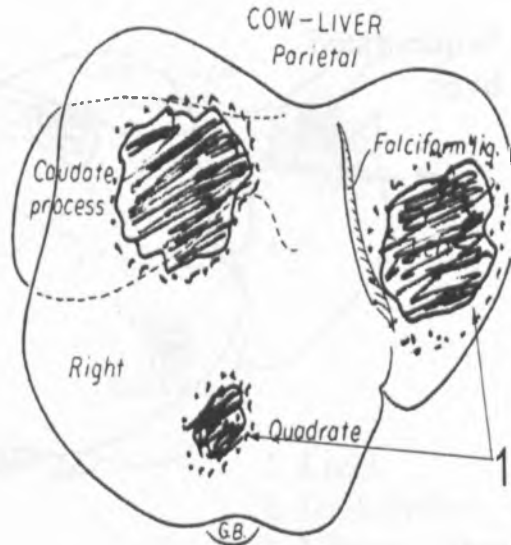
### Necrobacillosis (*Fusobacterium necrophorum*)

Necrobacillosis is seen at almost any age of the ox, scattered in the liver as 0.5 cm–10 cm or more of pale-yellow, dead, sequestered liver with a red border of inflammation and, if seen on the surface, they may have fibrin attached. They are sometimes difficult to differentiate grossly from fungal infection.

WP 1860

## LOCALLY EXTENSIVE \* ALL

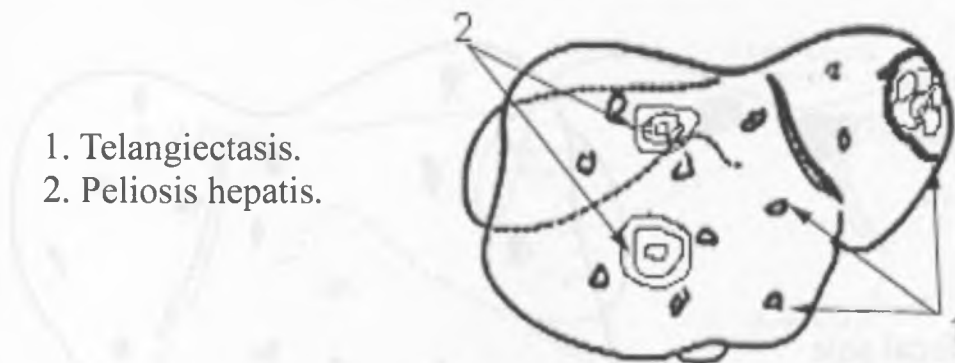
1. Locally extensive bacterial necrosis.



### Bacillary Hemoglobinuria (*Clostridium hemolyticum*)

Bacillary hemoglobinuria is most common in the adult ox; the large areas of necrotic liver stand out from the rest of the liver as mottled, discolored, discrete necrosis. A zone of inflammation may be present around them. Flukes are commonly associated and are thought to play a causative role, but are not always found. Similar large lesions without flukes involved can be iatrogenically produced with large doses of corticosteroids because this anti-inflammatory drug depresses the tissue response and the normally present anaerobes then duplicate. WP 7232, 7234

## MULTIFOCAL \* ALL



1. Telangiectasis.
2. Peliosis hepatis.

### Telangiectasis

Most telangiectasis appear as a few to hundreds of dark red or just dark 2–3 mm to several centimeter in diameter foci scattered throughout the liver. Some may be pale and firm with thrombi in them, but most are depressed, as the blood pressure is decreased with death. Conglomerations of these may make up large 10–20 cm foci. Exceptionally large, 10–30 cm dilated vascular cavities, often with laminated clotted blood, are called “peliosis hepatis” and are usually associated with food-derived estrogens.

A few to many, small to large, 2 mm to several centimeters, dark-red foci, some with straight edges, may be scattered in all of the lobes of the liver in many species. When cut, they ooze free blood when freshly dead. Most will be depressed in or on the liver as the blood pressure has gone, but some may be slightly swollen, with a pale stagnation thrombus within. These are rare in the horse.

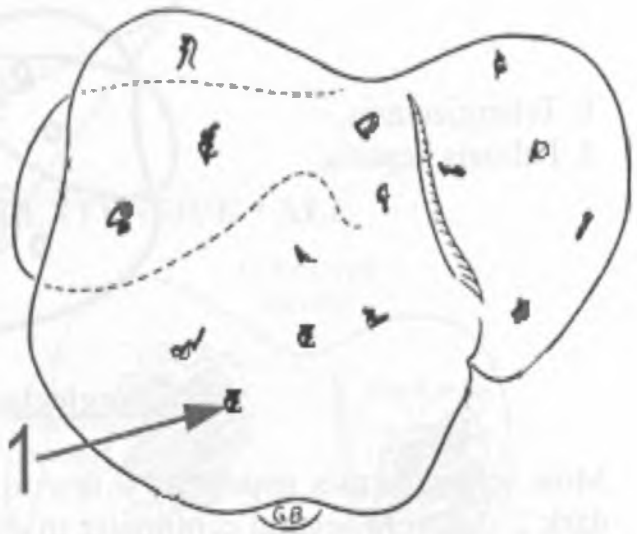
**Ox:** These are found mainly in older cows or bulls, but are common in steers and are a common cause of liver condemnation at slaughter.

**Dog:** Dogs fed soybean can have marked peliosis hepatis from the estrogen as gleiden and genistein in the soybeans.

**Cat:** Old cats as well as steers are the most affected domestic animals with telangiectasis; but captive large wild cats such as lions and tigers commonly have peliosis hepatis mostly thought to be related to plant estrogens, gleiden and genistein. WP 7040, 7195, 9145

## MULTIFOCAL \* ALL

1. Multifocal pale foci.



### Multifocal Pale Foci

A few to many, usually small 1–5 mm, pale-red, yellow, or variously colored foci in the liver of any animal should be initially cultured for fungi, bacteria, and viruses. They should be examined histologically for bacteria and viruses with inclusion bodies, fungi, protozoal agents, and migrating parasites as they, like tumors, need microscopic evaluation to be definitive. WP 1862, 7250

**Horse:** In the fetus, focal necrosis like this is often due to the herpes virus.

**Ox:** IBR virus is a common cause in the bovine fetus. Severe cases, or even mild cases in the young ox, are often associated with *Salmonella* sp. and called “sawdust livers.” These areas of focal necrosis are often called “typhoid nodules.”

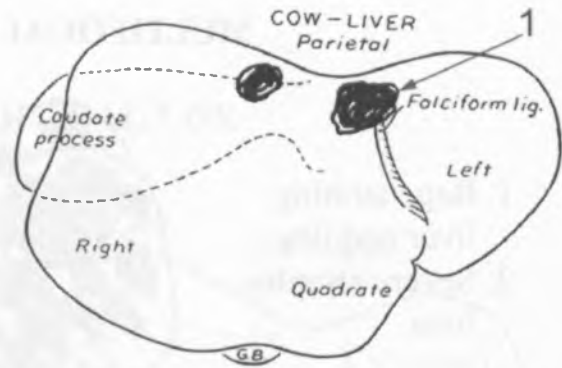
**Pig:** Pseudorabies in piglets should be suspected. *Ascaris* and *Stephanuris* sp. parasitic migration can also cause these.

**Sheep/Goat:** *Listeria* is a common agent found.

**Dog:** Both ascarid and hookworm larvae can cause these during migration in young dogs without immunity, and again in aged dogs which have lost their immunity.

## MULTIFOCAL \* ALL

### 1. Abscess.



### Acute and Chronic Abscesses

Small, .5 cm to over 8 to 10 cm areas, often having a red peripheral zone of inflammation, are usually indicative of infection. With time, many will have an observable connective tissue capsule of a mature abscess with thick or thin liquefied pus in their center. All must be cultured to be definitive. Many, especially in the ox, are near the hilar area, especially those originating from the bowel. Most abscesses have a distinct connective tissue capsule making differentiation easy. WP 309, 1511, 7245

**Horse:** *Rhodococcus* abscesses are common in young horses.

**Ox:** Several large areas of the acute necrosis caused by *Pasteurella* sp. may be in cattle given large doses of corticosteroids, as it is thought that ruminant livers are usually not sterile and anti-inflammatory drugs may predispose for these acute necrotic areas. Abscesses can be found anywhere in the liver, but if only one is present, it is usually near the hilum and vena cava. Flukes such as *Fasciola* often are associated with the acute lesions in Bacillary hemoglobinuria caused by *Clostridium hemolyticum*. Similar lesions that have dry necrotic centers of infarcted (sequestered) liver parenchyma are usually associated with *Fusobacterium necrophorum* (necrobacillosis). WP 309, 7243

**Dog:** A few to many, .5–4 cm, gray, mucoid-filled, multiloculated abscess pockets may be found in the major lobes around the hilum, with or without local peritonitis. These are considered classical for *Nocardia* spp. and a culture is necessary. Most are in older dogs and some may be associated with local tumors. WP 536,537

**Sheep/Goat:** Concentric-layered green-tinged pus in these abscess pockets with a 1–3 mm capsule are characteristically due to caseous lymphadenitis (*C. pseudotuberculosis*) organisms.



## MULTIFOCAL \* ALL

1. Regenerating liver nodules.
2. Severe atrophic liver.

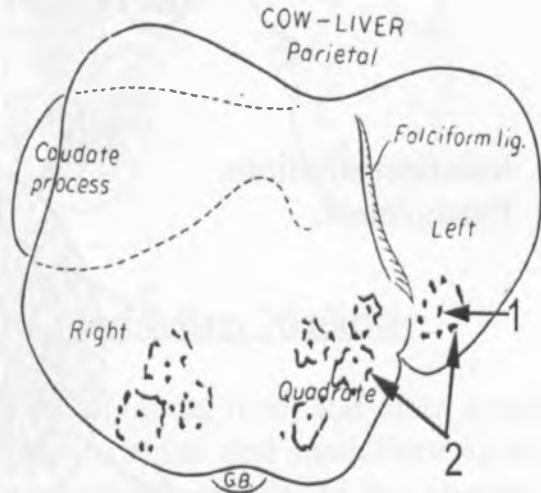


### Hepatic Atrophy and Regeneration

Numerous multifocal, several millimeter to several centimeter nodules make up almost the entire liver, with bands of soft, pliable stroma separating the nodules. The nodules are regenerating liver. The interlacing soft pliable bands are atrophic residual stroma and biliary tree as the result of the chronic low-level toxicity of Dilantin or other chemicals causing atrophy and dilation. Histologically, the interlacing bands are indeed connective tissue, but for proper identification the connective tissue is mainly only a condensation of residual connective tissue called relative fibrosis, and not the production of new fibrous tissue called absolute fibrosis. This can be seen in any species, but is common in the Doberman pinscher dog. WP 170, 7013

## MULTIFOCAL \* OX

1. Tiny 1–2 mm thin walled vessel.
2. Irregular connective tissue adhesions.



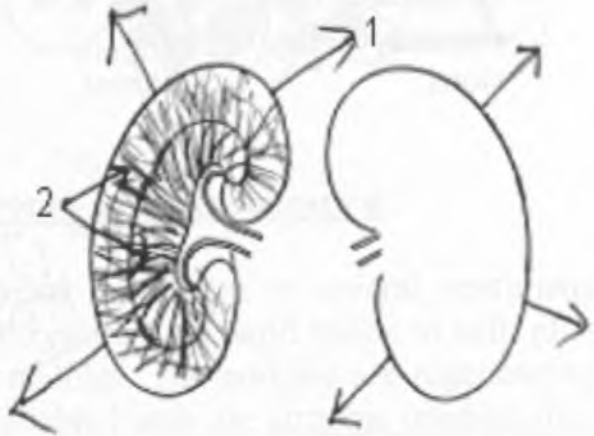
### Vascular-Related Lipidosis

Vascular-related lipidosis occurs mainly in adult cattle with visceral surface usually having round, 1–5 cm areas of yellow, irregular patches with a slight covering of irregular connective tissue. One to a dozen such yellow patches each present with a centrally located 1 mm thin vessel exiting the area into the surface connective tissue. These are thought to be areas of compromised fatty changed liver associated with the reverse flow of blood from these affected areas. There is no known cause except their relationship to the surface fibrosis. WP 300, 301

## ROUGH SKETCHES OF KIDNEY LESIONS

### DIFFUSE \* All

1. Indistinct striations.
2. Enlargement.



### Renomegaly (Enlarged Kidneys)

Massive renal enlargement, up to 3–4 times normal size and even heavier, should be considered amyloidosis, or possibly lymphosarcoma involvement when pale and firm. Moderately enlarged kidneys may be similar but also include glomerulonephritis or toxic nephrosis from oxalates and other toxins as sulfonamides, even arsenic. The iodine-staining of large pale kidneys may be positive 50% of the time grossly for amyloid, but chemical analyses and histological evaluation are needed in most cases. Many of these enlarged kidneys are firm, will bulge from the cut surface when the capsule is cut, and have a tendency to lose their distinct cortical and medullary radiating striations. Some of the chemical poisonings, such as the oxalate crystals, with antifreeze poisoning, and the sulfa drugs, may be noticed by the presence of renomegaly in the tubules themselves and easily identified on smears under a microscope.

WP 366, 11792, 863

1. Scarring.
2. Indication of enlargement.



### Interstitial Corticomedullary Nephritis

A kidney that is diffusely and, usually pale, firm, and often smaller than normal kidney, with loss of clear cortical and medullary striations, is suggestive grossly of chronic interstitial nephritis in any species. Histologically the lesion is characterized by diffusely interstitial fibrosis with many chronic inflammatory cells. WP 12156, 12110

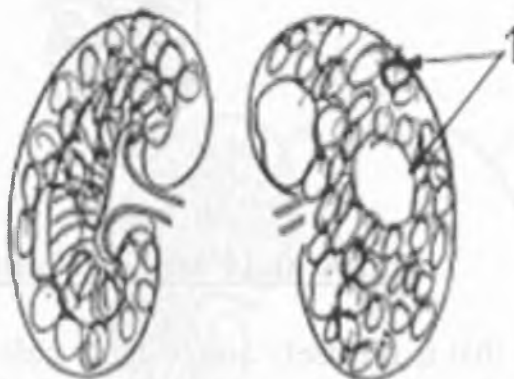
**Dog:** It is most common in dogs and usually caused by chronic leptospirosis.

It should be noted here that while it is commonly stated by many pathologists that capsular adhesions are common in dogs and other animals, it is quite rare. Capsular adhesions are usually associated with dehydration and some postmortem change, when the outer portion of the cortex is easily peeled off with the capsule as an artifact, if capsular removal is done without care.

**Pig:** In the pig, a very chronic form with severe fibrosis, even to the extent of almost masking the normal architecture of the kidney, is recognized and is caused by a specific fungal toxin, ochratoxin.

## DIFFUSE \* All

1. Congenital cysts.



## Polycystic Kidneys

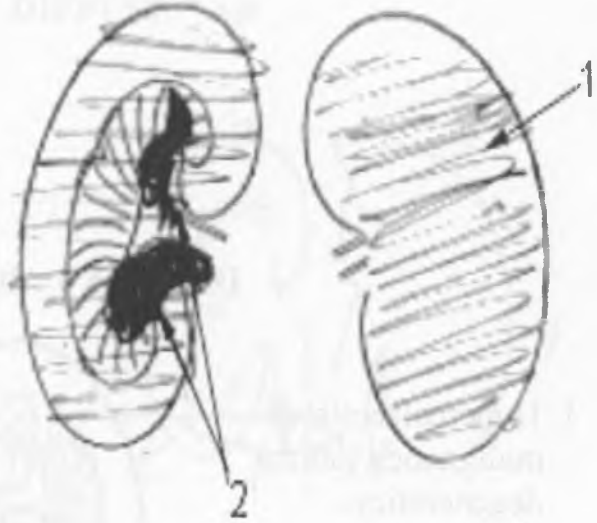
The multiple, clear, fluid-filled cystic tubules seen in the young and newborn may number only a few or in the thousands, to completely replace the normal parenchyma. An animal can live with a few and even a great many. It is sometimes surprising to see an older animal with a severe case of this congenital entity. The cysts are usually found in the cortex and medulla. Sometimes it is difficult to differentiate them from acquired cysts which have usually fewer cysts in a more fibrotic kidney. WP 12014, 19021, 15411

**Ox:** A few cases have been found with these cysts only in medullary tissue of calves. Also, many cases of one or only a few large, clear cysts may be found in one or two of the multi-lobulated kidney of the ox, again considered congenital as no other cause can be ascertained.

**Pig/Cat:** Polycystic kidneys are common congenitally.

## DIFFUSE \* All

1. Dark, pigmented.
2. Pigment calculi.



### Pigmentary Nephrosis

Normal or dark scarred, firm kidneys, or small, chronically damaged fibrotic kidneys with a very dark discoloration, are almost always associated with a pigmentary nephrosis. In many, one will find firm but fragile clumps of fairly dry brown to black calculi possibly large enough to deform the renal pelvis in the scarred kidneys. Histologically, the renal tubules are damaged and the lesion is usually classed as a chronic pigmentary nephrosis. WP 15, 564, 8293

**Ox:** In the female ox, 30–60 days after calving, some animals have very dark, almost black pigmented kidneys. The cause is suspected to be associated with low liver phosphorus and possibly other metabolic imbalances (postparturient hemoglobinuria, hemosiderosis, lipofuscinosis.)

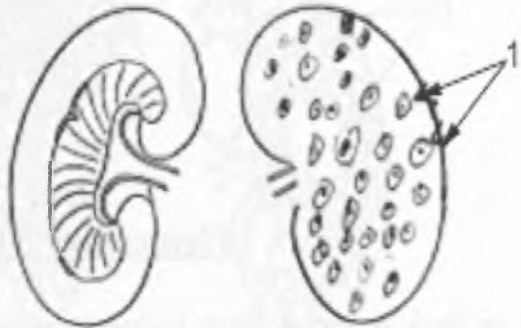
**Sheep/Goat:** Very dark kidneys are sometimes called gun-metal colored and are quite diagnostic for chronic copper poisoning. Dark kidneys are a feature of the cloisonne kidney in the Angora goat.

**Pig:** Dark kidneys in this species, especially when found with dark bones, should suggest congenital porphyrinosis.

**Dog/Cat:** Small scarred kidneys with clumps of brittle pigment in the pelvis are considered to be pigmentary casts thought to be due to either bile obstruction or hemolytic problems. This is a lesion most common in the cat.

## DIFFUSE \* All

1. Hypercalcemia of malignancy tubular degeneration.

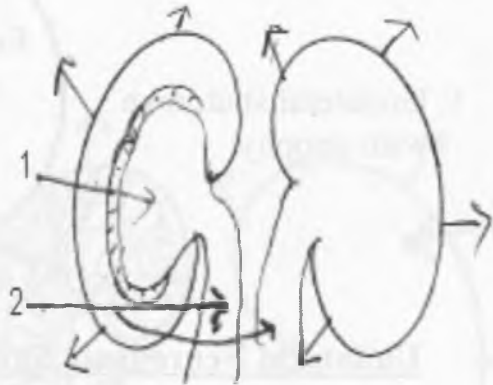


### Hypercalcemia of Malignancy Tubular Nephrosis

Hypercalcemia of malignancy tubular nephrosis is a relatively newly recognized lesion in the dog kidney. They are multifocal, round, opaque, white 3–4 mm circles seen on the cortical surface with gray central 1 mm areas. These represent a dilated tubule surrounded by some mineralized debris and few cells, called the tubular nephrosis of hypercalcemia of malignancy, because it is only seen mostly in those cases. The most common malignant tumor associated with this entity is the malignant adenocarcinoma of the anal sac glands. The renal lesion has been seen with other malignancies including lymphosarcoma. The central gray areas are often slightly depressed. WP 492, 494

## DIFFUSE \* All

1. Dilated pelvis.
2. Dilated ureters.



### Hydronephrosis

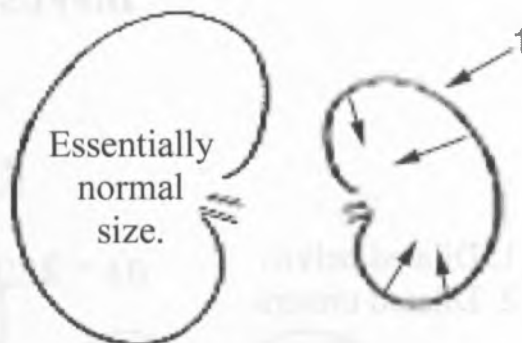
Any dilation of the renal pelvis, with or without ureteral dilation, is the characteristic feature of hydronephrosis. Most are associated with blockage of urinary outflow for almost any reason. The degree of dilation is dependent on the length of time and completeness of obstruction. Minimal dilation may be noted with acute obstruction. Chronic obstruction may cause almost complete destruction of parenchyma in both kidneys, making one wonder how the animal lived so long. Urethral obstruction can cause both kidneys to be dilated, while a blocked single ureter may affect one side only. Tumors, calculi, chronic infections, and anomalies can all be suspected. In all species with failure of bladder sphincter control from CNS disease, for instance, a degree of urinary bladder dilation is common, along with some hydronephrosis. WP 11877, 11894, 12021

**Sheep:** In orphaned lambs especially, as well as in orphaned deer and other animals, it is common to see a degree of hydronephrosis, as the mother is not present to lick the genitalia to stimulate urination. This dilation will disappear with time in the young.



## DIFFUSE \* All

1. Unilateral shutdown with atrophy.



### Unilateral Neurogenic Shutdown with Atrophy

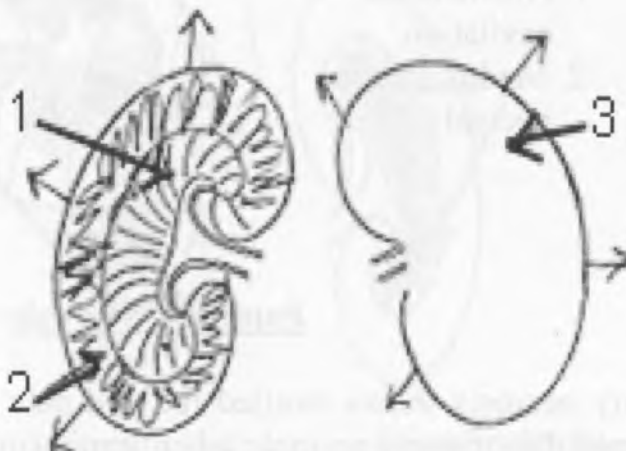
In most species, the left-side organ of a pair is usually slightly larger than the right-side organ normally. At birth, when one is noted to be smaller it is often considered hypoplastic or aplastic, but this hypoplasia is not a common finding. In some young adults and older animals of all species, it is common to see a major difference in size of either side over the other. In the adult, when the kidneys are examined, either grossly or histologically, and one often finds evidence that both kidneys were insulted at the same time with the result that neurogenically one kidney shuts down and subsequently atrophies. The larger kidney is usually of normal size comparatively, as they usually increase in efficient function but do not enlarge appreciably. Many toxic agents and disease can apparently do this, providing of course the insult was not acutely fatal. An underlying cause must be looked for historically and histologically. WP 11894, 559, 564

**Sheep:** Sheep with Dubin-Johnson syndrome often have unilateral neurogenic shutdown with atrophy in cases of pigmentary nephrosis. Lambs commonly have this entity in Finn sheep, for instance, when affected with the Finn sheep immunomembranous glomerulopathy. The urine apparently has an abnormal taste to the dam and she may not lick the young animal's genitalia to stimulate excretion.

**Cat:** This entity of neurogenic renal shutdown with atrophy is seen more in cats than other species.

## LOCALLY EXTENSIVE \* ALL

1. Essentially normal medulla.
2. Soft, mushy cortex.
3. Pale, enlarged, soft, mushy cortical parenchyma.

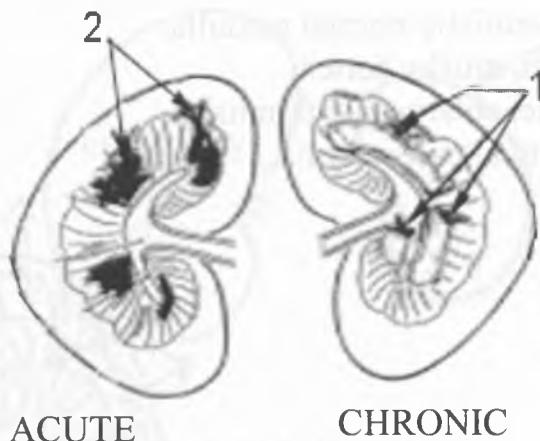


### Glucose-Related Rapid Autolysis (Pulpy Kidneys)

A diffusely enlarged, extremely soft and mushy cortex, usually pale but sometimes mottled, is usually the result of autolysis in association with excess glucose in the kidney. It is associated for reasons such as enterotoxemia and the gluconeogenesis associated with that disease in cattle and sheep, or in any animal with diabetes and glucosuria. It is seen most commonly in animals given glucose intravenously just prior to death, especially in the foal. With substrate glucose available, autolysis can be rapid and severe. In some of these cases the cortex may be so reduced that the kidney may actually be reduced in size due to the autolysis of the renal cortex. WP 866, 871

## LOCALLY EXTENSIVE \* ALL

1. Post-necrotic cavitation.
2. Medullary necrosis.



### Papillary Necrosis

Papillary necrosis shows mottled red and pale tissue of part or all of the renal medulla of many animals when seen acutely. It shows yellow-green and dull when more chronic, with urine effect on the dead tissue. Finally, when extremely chronic it leaves an irregular cavity just under the cribriform plate area of the renal crest, where the collecting tubules empty into the renal pelvis, or in even more extensive cases leaving essentially no medullary tissue. The cavitation left behind is called post-necrotic cavitation. WP 1587, 1457, 12178

**Horse:** In the horse, this is usually associated with the use of NSAIDS (nonsteroidal anti-inflammatory drugs) in a dehydrated adult animal. It could also be seen in right dorsal ulcerative colitis cases, but in dehydrated foals the NSAID usually causes perforating gastroduodenal ulcers, not renal lesions, and is thought to be due to the level of prostaglandin in these areas.

**Ox:** Normally, calves are born with cystic structures on the tips of the medulla, which disappear early.

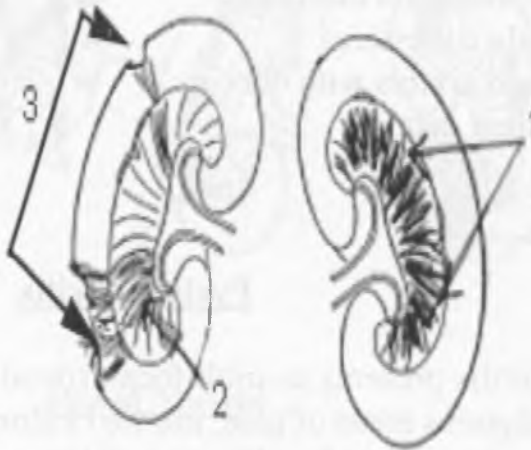
**Sheep/Goat:** Sheep and goats usually develop this lesion when given a prolonged course of nonsteroidal drugs.

**Dog:** Arsenic toxicity, amyloidosis, and diabetes cause similar lesions.

**Cat:** Cats are often affected by the use of NSAIDS such as aspirin.

**LOCALLY EXTENSIVE \* ALL**

1. Dehydration salts.
2. Dehydration salts.
3. Chronic dimpled infarcts.



Infarction with underlying dehydration salts.

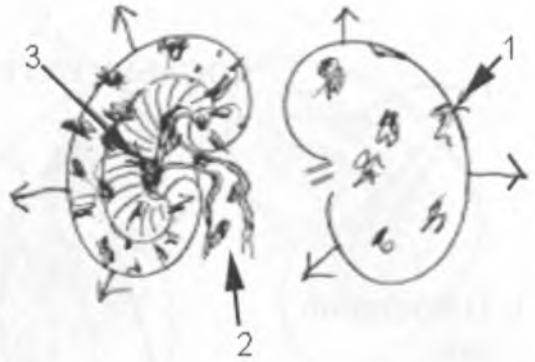
Severe dehydration with dehydration salts.

**Dehydration Salts**

In most species with impediment to normal fluid flow distal to the cortical infarct and under the infarct, dehydration allows urinary salts to precipitate, leaving distinct white lines in the medulla. These will dissolve out during histology preparation and not be seen on glass slides. In severely dehydrated young animals, dehydration salts may involve almost the entire medulla, especially in piglets. These are not clinically significant. WP 11912, 12151

## LOCALLY EXTENSIVE \* ALL

1. Purulent debris often with capsule adhesions.
2. Dilated ureters with debris.
3. Purulent debris.



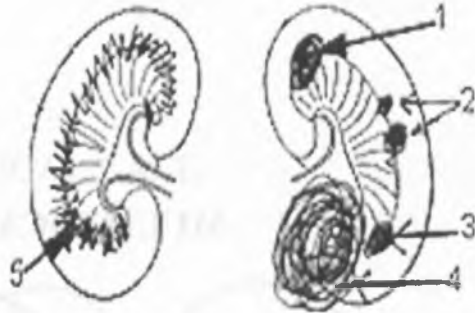
### Pyelonephritis

Pyelonephritis presents as multifocal, round, and irregular, .5–1 cm to large contiguous areas of pale, mottled kidney parenchyma scattered in the kidneys, some with yellow or white purulent debris in the center. These represent bacterial infection, usually considered ascending from a lower urinary tract infection, because they are to be seen in both medulla and cortical areas. The renal pelvis likewise has purulent debris or eroded surfaces of the pelvis and medulla. It should be noted that some cases may have both pelvis and cortex involved without grossly observable medullary lesions.

**Ox:** In the ox, a very strong odor of ammonia is usually noted in the affected kidney as the usual agent, *Arcanobacterium* (*Corynebacterium renale*), has urease capable of breaking down nitrogenous wastes to ammonia while many other organisms do not. The ox may have severe ureteritis with swelling and necrotic debris in one or both ureters. The urinary tract may be extraordinarily enlarged 2–3 times (hydronephrosis) with pus. In the normal multiple lobular kidney of the cow, many lobules, or parts thereof, may not be involved, again suggesting an ascending infection, not an embolic (descending) infection which would usually shower all lobules. WP 231, 1406, 1482, 8356, 11837, 12076, 12210

## LOCALLY EXTENSIVE \* ALL

1. Nodular lymphosarcoma.
2. Cortical fibromas.
3. Any metastatic tumor.
4. Nephroblastoma.
5. Corticomedullary locally extensive lymphosarcoma.



### Renal Masses

Masses from tiny (1 mm) up to massive (10–20 cm), or even replacing the entire kidney, may be caused by almost any metastatic tumor from the body; or even ill-defined other types of masses as parasitic or infectious foci. Microscopy is needed. In several species, lymphosarcoma, for instance, may be nodular, but it may also be a diffuse tumor and even of the corticomedullary zone only. With few exceptions, renal tumors themselves exist but are not that common in animals. WP 498, 6514

**Horse:** Renal tumors are rare.

**Ox:** Lymphosarcoma may be nodular around the renal pelvis and ureter or diffuse in the corticomedullary areas alone.

**Pig:** Nephroblastomas, usually malignant, are found under 6 months in pigs. This is similar to man.

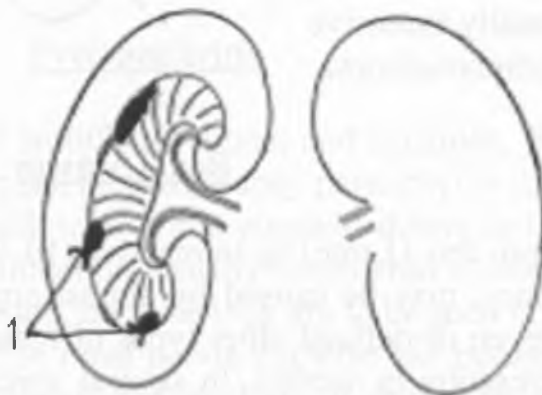
**Sheep/Goat:** Lymphosarcoma also.

**Dog:** 1–5 or more multiple, firm, white masses at the corticomedullary junction are usually so-called cortical fibromas.

**Cat:** Lymphosarcoma is common.

## MULTIFOCAL \* ALL

1. Renal vein thrombi.



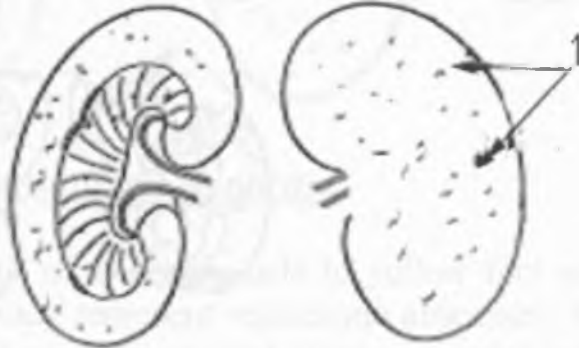
### Renal Vein Thrombi

As in man, grossly, large or small renal vein thrombi are a common finding in animals that have been heavily treated with electrolytes or that have severe diarrhea. They are probably best seen because of size in the hilar and corticomedullary junction veins. Probably less than 50% are associated with renal infarction, but when infarcts are found with them, they are probably the effect of the infarct, not the cause, as would be expected with arterial thrombosis. Usually no gross lesions are seen on the external surfaces. Most often seen in calves.

Slaughtered cattle may have CNS tissues as renal emboli appearing like these thrombi from the compressed air use associated with the captive bolt method of slaughter. WP 293, 12023

## MULTIFOCAL \* ALL

1. Pinpoint hemorrhages.



### Multifocal Petechial Hemorrhages

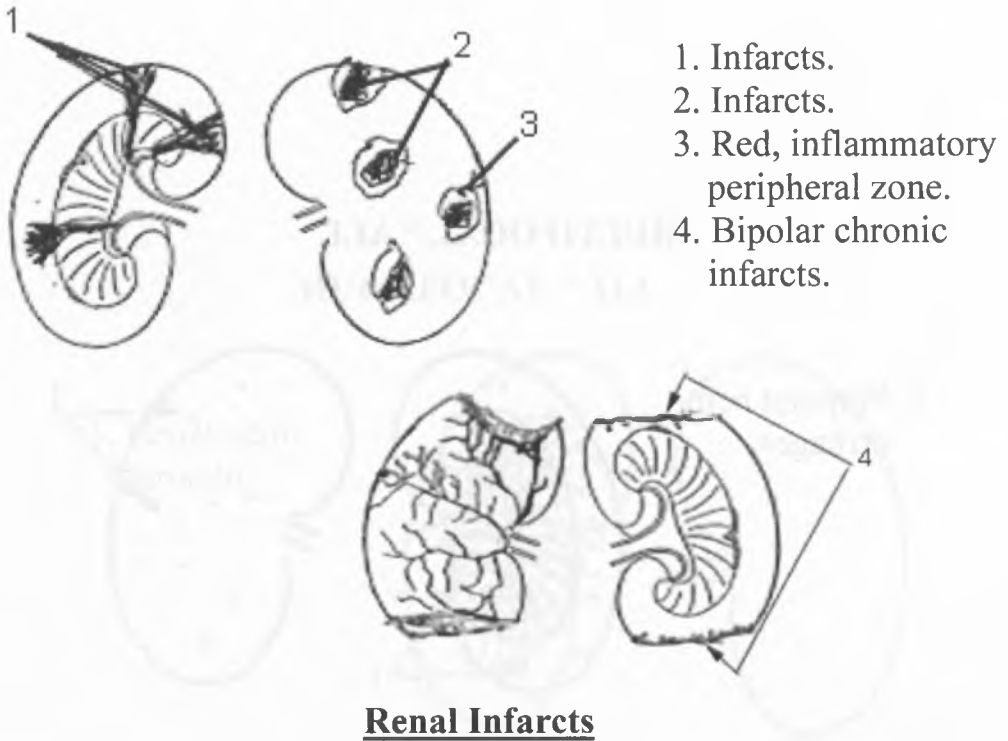
Multiple pinpoint hemorrhages in the kidney are commonly seen in all species with septicemia and some toxemias. Disseminated intravascular coagulation can appear in the same fashion. In some bleeding disorders, disseminated intravascular coagulation may be seen. Prominent glomeruli in some animals may be congested and appear somewhat similar. WP 8818, 6195

**Pig:** In susceptible pigs, such hemorrhages are often diagnostic for hog cholera, but baby pigs often have many hemorrhages when they die from many different causes.

**Dog:** Multiple pinpoint, or slightly larger hemorrhages are often diagnostic for herpes virus in young puppies that get the disease before 10 days of age. WP 1517, 1531, 12101



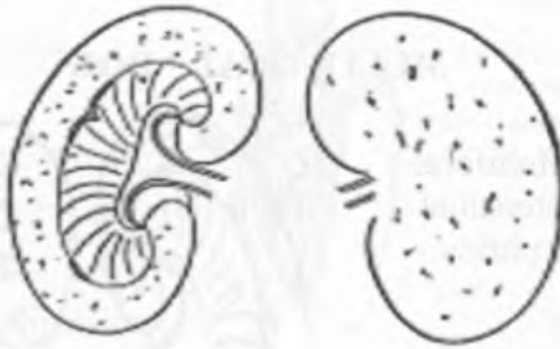
## MULTIFOCAL \* ALL



Cone or roughly triangular-shaped, pale or mottled, red lesions with their base towards the capsule on cut sections; often with red periphery when acute or pale, and indented on the cortical surface when chronic, are probably the result of infarction. Those with yellow specks of pus are usually embolic from vegetative endocarditis. Common in all species, often the source of emboli may not be found. The size varies considerably from pinpoint to several centimeters. Massive infarction of the cortex, primarily in the outer cortex, should make one consider part of the Schwartzman reaction, as in rabbits mainly.

**Dog/Cat:** A special form of renal infarction seen, especially in the dog and cat, is the single polar infarct in which large pale areas of kidney are missing, with subsequent dimpling of the poles. Some suggest an increased renal pelvis pressure, reflux nephropathy, with acute angle kinking of the arcuate vessels to the poles being involved, but this is still in debate. WP 480, 481, 568, 1463, 12041

## MULTIFOCAL \* ALL



### Multifocal Nephritis

Multiple, even hundreds, of 1–4 mm, pale to yellow foci scattered throughout the kidney may represent septicemic abscesses, embolic bacterial infection, focal necrosis, or viral disease, parasite migration scars, fungal infection, and even metastatic tumors. All of those causes must be differentiated by cultural, histological, or other means, as many cannot be diagnosed grossly. WP 10270, 1452, 12156

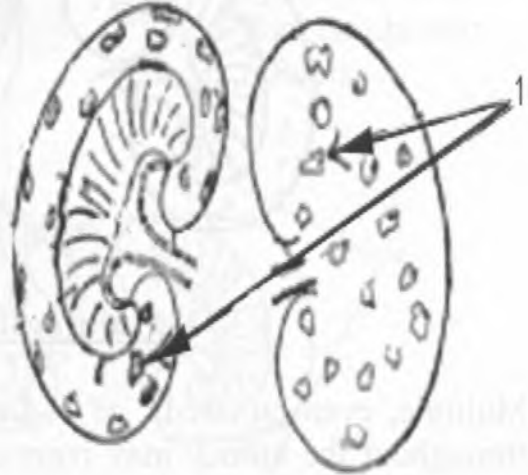
**Horse:** Yellow discrete foci in the foal certainly can be due to shigellosis (*Actinobacillus equuli*). Pale white foci may be viral in the fetus due to equine herpes virus I.

**Dog/Cat:** Pale foci in the dog may suggest ascarid or hookworm larval migration scars in young and old dogs without immunity, as the old dogs usually lose their earlier immunity with time.

**Ox:** Slightly larger foci, from 3–6 mm, scattered in the calf kidney are quite specific for white spotted kidney, *E. coli* infection. Malignant catarrhal fever can also be causative.

## MULTIFOCAL \* DOG

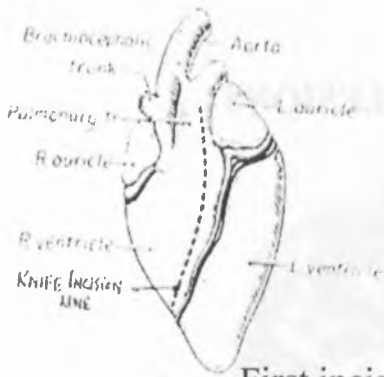
1. Multifocal interstitial nephritis.



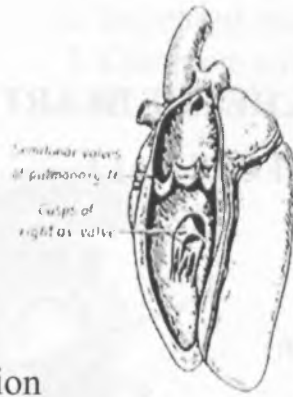
### Multifocal Interstitial Nephritis

There is no known cause for this tiny, relatively uniform, multifocal interstitial nephritis. They are multifocal, usually dozens of irregular 3–4 mm depressions scattered on the cortical surface, hidden by the renal capsule, and histologically consisting of shallow depressions with underlying connective tissue and chronic inflammatory cells limited to these areas. These are usually limited to older dogs. WP 12156, 11861

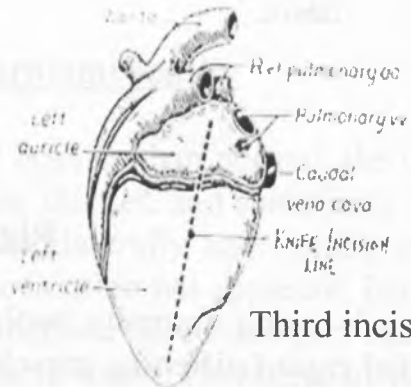
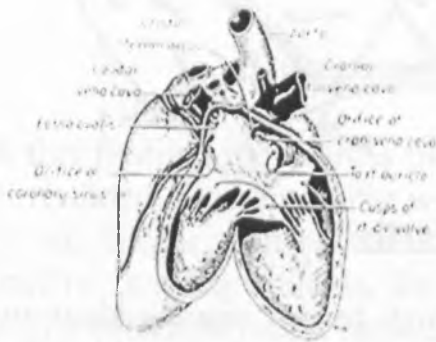
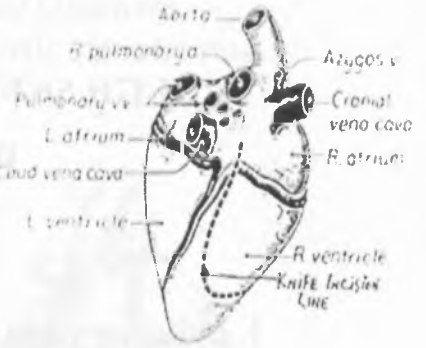
# Heart Incisions and Examination



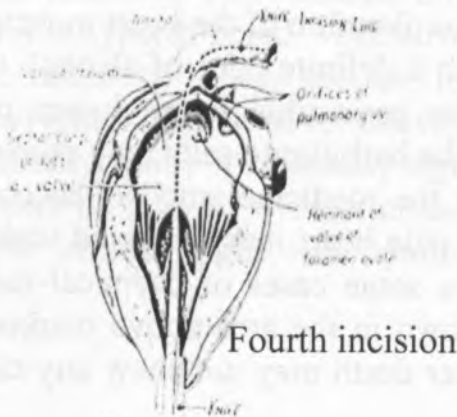
First incision



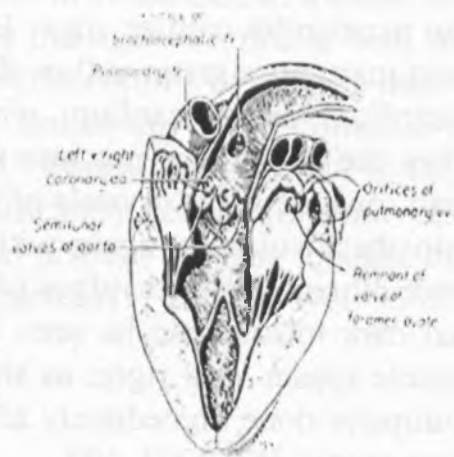
Second incision



Third incision



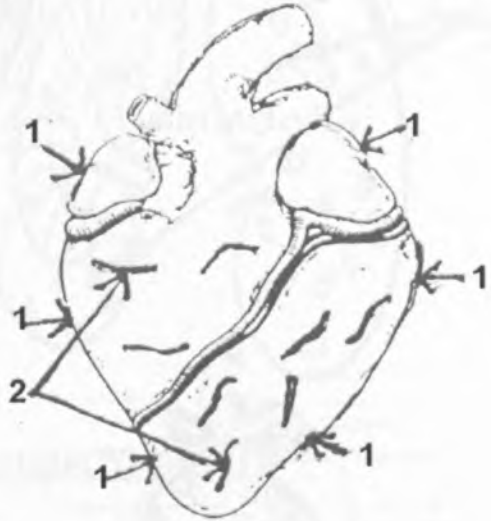
Fourth incision



## ROUGH SKETCHES OF HEART LESIONS

### DIFFUSE \* ALL

1. Indicates rigor contraction.
2. Individual accentuated contraction bands of muscle associated with chemical euthanasia.

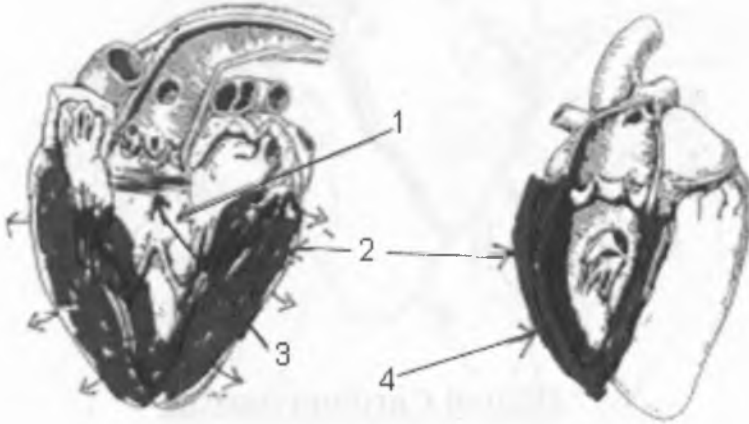


### Rigor Mortis

Animals dying normally, without high fevers, usually show myocardial rigor (stiffening muscles) in an hour or so, but high fevers may cause such rapid and incomplete rigor that often it is said that they don't go into rigor at all. Severe exercise shortly before death also accelerates cardiac rigor. Euthanasia solution directly into the heart may cause green-yellow discoloration of the heart muscle, endocardium, and epicardium, with a definite odor of alcohol. Often when the solution is put into the pericardial sac or lumen of the heart, opaque white crystals of the barbiturate salts may precipitate onto these surfaces, again with the medicine smell of alcohol or other diluent. Distinct ridges of pale heart muscle mixed with normal dark muscle can be seen in some cases of chemical-induced muscle spasm with rigor, as shown in the area above marked (2). Autopsies done immediately after death may not show any cardiac rigor mortis. WP 2203, 603

## DIFFUSE \* ALL

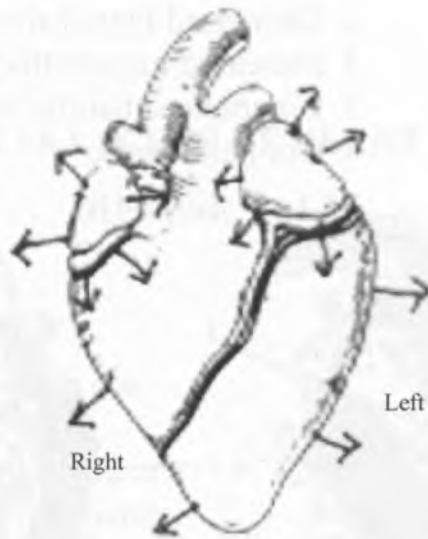
1. Decreased lumen size.
2. Increased muscle thickness.
3. Complete subaortic stenotic ring.
4. Cor pulmonale.



### Hypertrophic Cardiomyopathy

With this finding, the heart is usually heavier than normal, the outline is more rounded, the ventricular walls are thicker, and sometimes the atrial walls are thicker than normal—even unilaterally and usually related to chronicity. In many animals, the cause is often not apparent, but in some there is a complete rough band of connective tissue that may encircle the aortic outflow just under the aortic valve itself. This band of tissue is a congenital anomaly called a complete subaortic stenotic ring. In some animals with lung disease, intrapulmonic, diffuse vascular disease, stenosis, or other pulmonic valve disease only the right ventricular wall (cor pulmonale) is hypertrophied. Vegetative endocarditis of specific heart valves can be causative in any species. In almost any sized domestic cat, the heart weight is almost never above 17 grams normally, but in this entity of cardiomyopathy it is usually significantly heavier. In some cases, the heart may be heavier without clinical disease but it is thought that with time the clinical signs will commence. WP 1567, 2041, 9330, 9334, 16193

## DIFFUSE \* ALL



### Dilated Cardiomyopathy

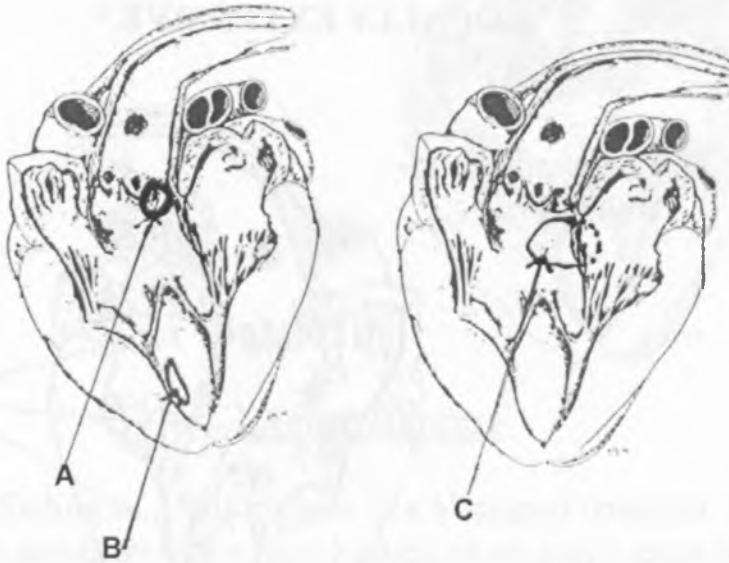
In dilated cardiomyopathy, the heart is markedly dilated with thinner walls than normal. The lumen of both atria and auricles may be up to 2 to 4 times normal size, with the ventricles less dilated. When dilation exceeds the ability of the atrioventricular valves to close completely, regurgitation occurs with subsequent heart failure. In many cases, compensatory myocardial hypertrophy may occur bilaterally, or even unilaterally. Several breeds of dogs such as the Doberman pinscher and water dogs are more affected than other breeds. WP 569, 570, 657

**Ox/Swine:** This is usually considered genetic in origin.

**Cat:** Taurine deficiency is causative in cats. The normal heart weight of most domestic cats is under 17 grams but in this disease the heart weight may be doubled or more.

**Fox:** The silver fox is very prone to this disease.

## LOCALLY EXTENSIVE \* ALL



- A. Common subaortic septal defect.
- B. Low septal defect (lambs).
- C. Large subaortic septal defect.

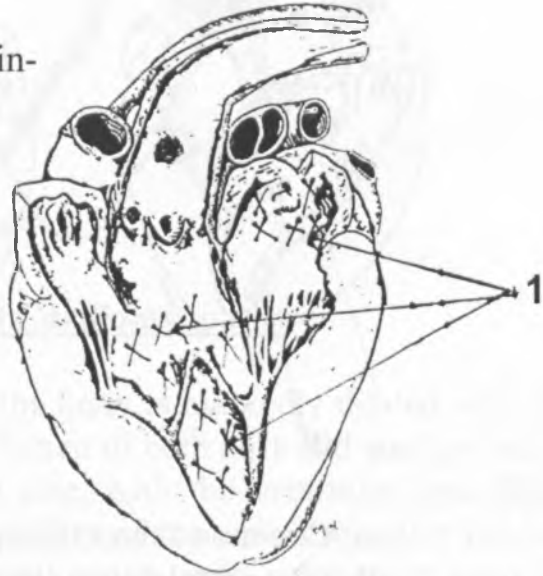
### Septal Heart Defects

In the position (A) above, small openings may be found at birth that will often close spontaneously with age. Actually, some massive openings may let animals survive to old age, but it is rare. Low septal defects between the ventricles (near the apex) are more common in lambs than in other species. Atrial septal defects of several types also occur between the atria. WP 1274, 1700, 690



## LOCALLY EXTENSIVE \*

1. Endocardial mineralization.



### Endocardial Mineralization

Endocardial mineralization occurs primarily as white, irregular mineral deposits on endocardial surfaces of the right atrium. Scattered mineralization on other endocardial surfaces and sometimes on the endothelium of major vessels are diagnostic for uremia as the result of renal failure. More extensive, similar mineral deposits may be seen with vitamin D poisoning and other plant-related poisonings, and some therapeutic drugs. A relatively new recognition of an extensive mineralization can be seen in some cases of hypercalcemia of malignancy. These are associated with various malignancies, especially adenocarcinomas of anal sac glands in dogs and lymphosarcoma in many species. WP 3318, 1663, 204

## LOCALLY EXTENSIVE \*

1. Common aortic rupture site.

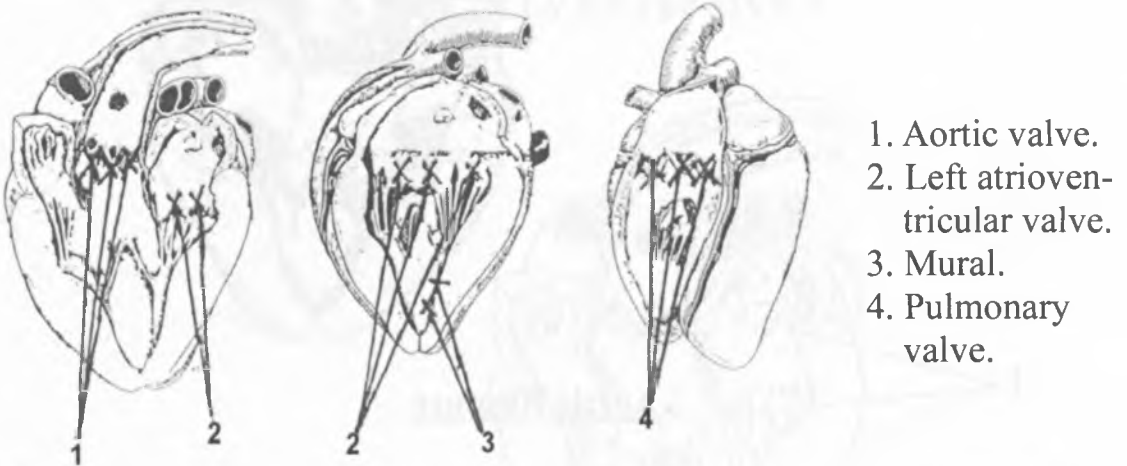


### Aortic Rupture

The usual finding in aortic rupture is a V-shaped irregular rupture at the base of the aorta, with the apex located at an aortic cusp leaflet attachment site. The rupture occurs with hemorrhage through this tear into the septal wall itself, the periaortic soft tissues, or even into the pericardial sac. Care must be taken during the dissection, as it may occur at any site around the aortic ring and be destroyed during the necropsy.

**Horse:** Most cases that occur in horses have a common finding of trauma to the chest as in: stud horses falling onto the rump of the mares during service; racers tripping during a race and falling on their chest; or horses being shot in the head for euthanasia purposes and collapsing rapidly, to fall on their chest. Each of these cases cause kinking of the heart and aorta—probably at high systole, when all valves were closed and the weak link of that system being the aortic valve attachment site. Aortic ruptures in young animals, including the horse, can be associated with trauma and also copper deficiency. Commonly, with trauma they will be found almost anywhere along the aorta or other large vessels, however; and, not so specifically, at the aortic cusp attachments site. WP 41, 42, 45

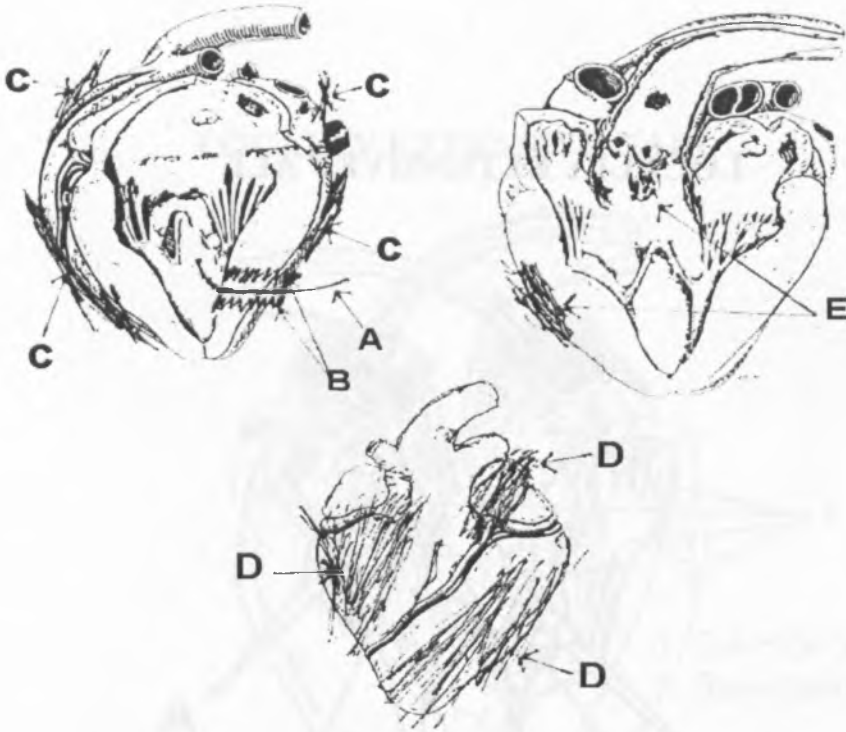
## LOCALLY EXTENSIVE \* ALL



### Locations of Common Vegetative Endocarditis Lesions

From tiny to extremely large, friable, often dull, irregular masses are attached to any valve and even the endocardial walls. The masses contain some heavy, pale, yellow, green, or even black foul-smelling bacterial material in their centers of necrotic, purulent debris. These are evidence of vegetative endocarditis, being the result of a bacterial, rarely mycotic, inflammation of the endocardium of the walls or heart valves proper. Most, by far in any species, are associated with heart anomalies, chronic joint disease, or chronic infection anywhere in the body. Each of these account for about 30% of such cases, leaving approximately 10% to be caused by a specific organism such as *Erysipelas rhusiopathii* in pigs, *Streptococcus viridens* in the captured opossum, and Aspergillosis in dogs. In many instances, with septic thrombi as these in the left heart near the coronary artery openings, they may embolize to the heart proper. If in the right heart, the thrombi embolize to the lung. WP 281, 516, 1627

## LOCALLY EXTENSIVE \*

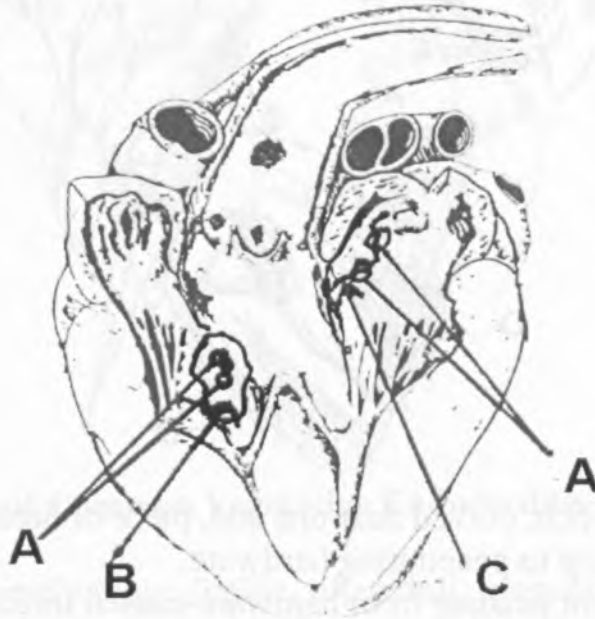


- A. Typical, 4–5 inch, curved near one end, piece of hard wire.
- B. Wound reaction to penetrating hardware.
- C. Fibrinopurulent exudate from hardware-caused infection.
- D. Classical sheets and strands of fibrinous exudate of pericarditis.
- E. Focal fibrinonecrotic and emphysematous myositis (blackleg) with associated odor of sour milk.

### Other Heart-Associated Infections

Hardware-caused myocarditis, or endocarditis, or both can be associated with many different organisms in cattle. *Corynebacterium* spp. and *Escherichia coli* are common causes for infectious pericarditis in many species. It is difficult to explain the pathogenesis of blackleg in the myocardial form of the disease as many consider blackleg as starting *de novo* from the bacterial spores present in the muscle cells before they are damaged. It is of some interest that the hardware metal, usually wire, found in the majority of hardware penetrations to the heart from the reticulum, are about 6–12 cm long, with a curve towards one end. WP 1363, 1714, 21137

## LOCALLY EXTENSIVE \* ALL

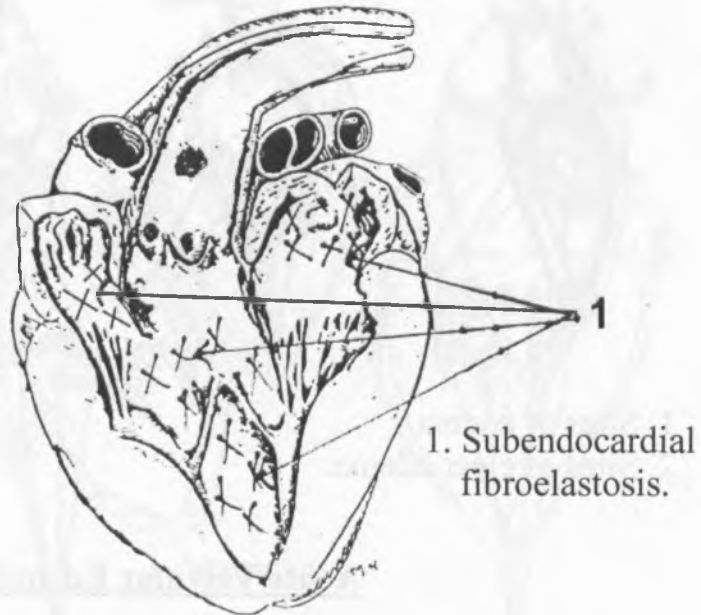


- A. Nonseptic fibrin thrombi.
- B. Subendocardial suffusion hemorrhage.
- C. Endocardial tear.

### Nonseptic Fibrin Thrombosis

Nonseptic fibrin thrombi are the response to endocardial damage, which is different from the more common bacterial vegetative endocardial thrombosis. Likely causes include terminal endocardial suffusion hemorrhages and endocardial rupture from intraluminal pressure associated with incompetent valves, either from anomalies at birth or acquired at a later age. WP 125

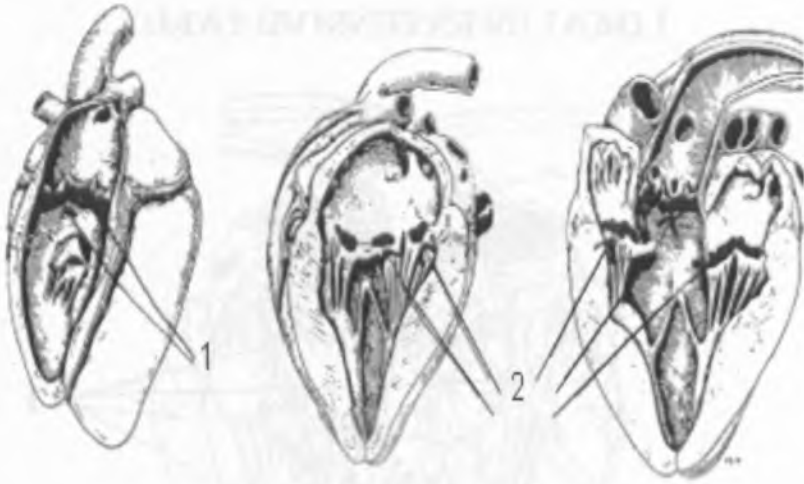
## LOCALLY EXTENSIVE \* ALL



### Subendocardial Fibroelastosis

In many species, subendocardial fibroelastosis is very common in the newborn with heart anomalies. Several or all chambers may be dilated with a definite pale to gray, glistening, slightly thickened endocardium. The term subendocardial fibroelastosis describes the histological features well. The cause is thought to be a function of anoxia and turbulent flow. It is also associated with incompetent valves in the atria in older animals. These affected atria may rupture, at least superficially, with crooked linear stretch tears developing. These irregular endocardial stretch tears may develop nonseptic fibrin thrombi along the tear itself.  
WP 545, 1755, 6957

## LOCALLY EXTENSIVE \* ALL

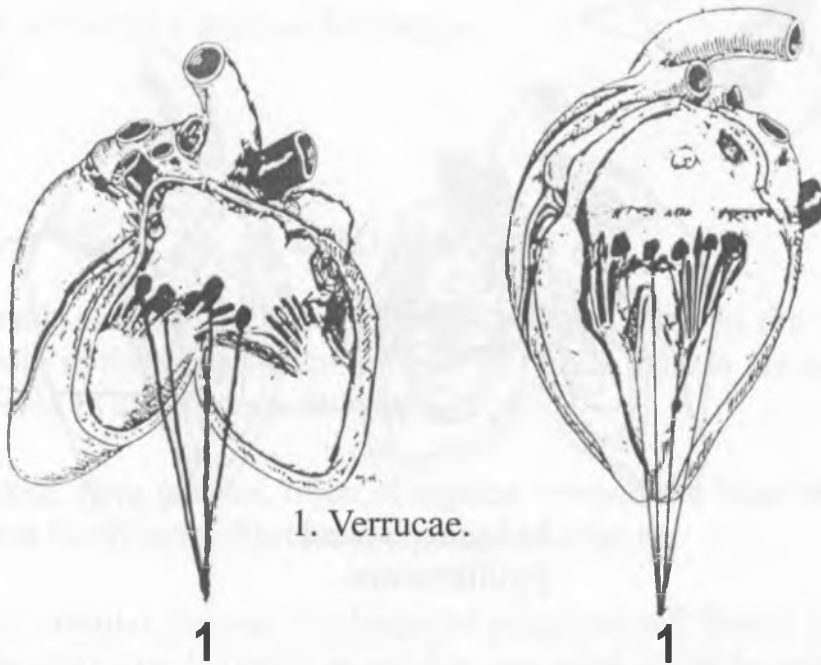


1. Sites of edema.
2. Sites of clear edema.

### Acute Valvular Edema

In all species, it is very common to find slightly elevated blebs of clear edema at the base of most heart valves. Most occur in the aortic valve cusps, less in the pulmonary valve cusps, and less near the free edges of the AV valves. Most can be considered a functional change, with a slow death as with some euthanasias. Most would not be significant and certainly should not be considered verrucous endocardiosis (chronic valvular fibrosis), which are at the free edge of the valve cusps, nor vegetative endocarditis (irregular, fragile).  
WP 16122

## LOCALLY EXTENSIVE\* DOG

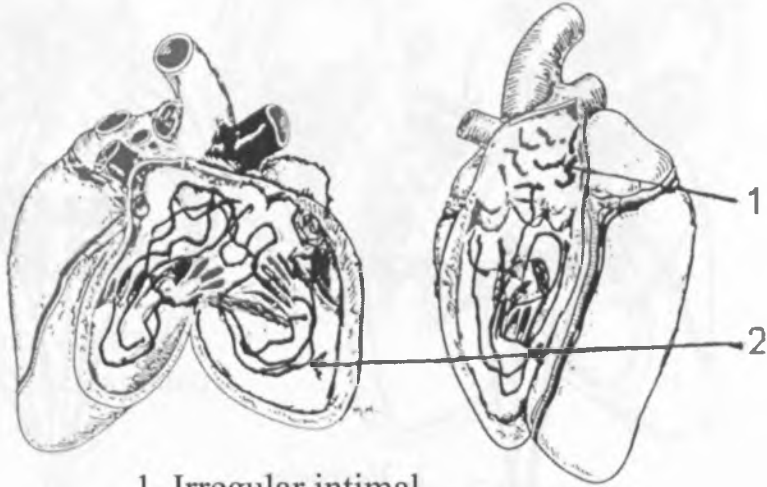


### Chronic Valvular Fibrosis (Verrucous Endocarditis)

Chronic valvular fibrosis presents as multiple, firm, glistening, flesh-colored 1–3 mm, wart-like nodules (verrucae) formed mainly along the free edges of left atrioventricular valve, and in some cases, along the free edges of the right atrioventricular valve. The nodules consist of connective tissue, myxoid degeneration, edema, and intimal proliferation. These are degenerate changes and, often, as the animals age they become more prominent and deform the valve to compromise its function enough to be the most common form of heart disease in older dogs. Sometimes the AV valves may prolapse (bulge) into the atria and is called the “parachute reaction.” These verrucae are seen only sporadically in other animals. WP 544, 1300, 1251, 1753



## LOCALLY EXTENSIVE \* DOG, CAT



1. Irregular intimal proliferations.

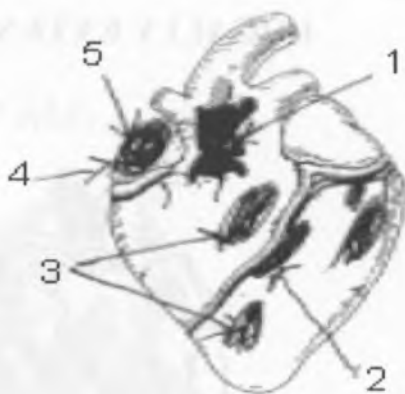
### Dirofilariosis

*D. immitus* are long, 10–20 cm, thin, 1–2 mm-wide, white worms found in the right atrium. They extend into the pulmonary artery and backward into the posterior vena cava. Every now and then, a single worm may be found in the left side of the heart. These parasites usually do not cause lesions in the heart proper, but multiple irregular intimal thickenings to the pulmonary trunk and its branches may be seen. Dead or degenerate adult worms may embolize to the lung and cause some vascular compromise to the lung. They usually do not cause true infarcts in the lungs because the lung has a dual blood supply. WP 1396, 1770

**Dog/Cat:** *Dirofilaria immitus* are more common in some geographic areas than in others.

## LOCALLY EXTENSIVE – MULTIFOCAL \* ALL

1. Aortic body tumors.
2. Firm, elongated neurofibromas.
3. Pale, solid lymphosarcoma, especially in R.A., but anywhere in myocardium.
4. Pale, solid lymphosarcoma, especially in R.A., but anywhere in myocardium.
5. Highly vascularized mass of hemangiosarcoma.



### Heart Masses

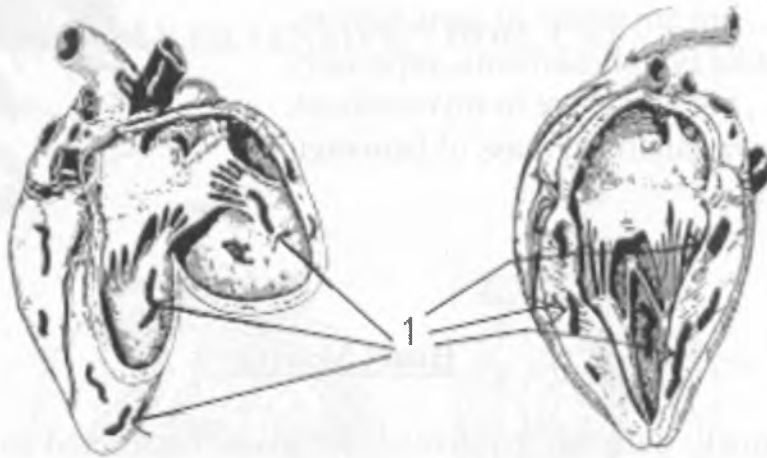
Large or small, pale tan, relatively soft masses scattered in the myocardium. Especially in the right atrium and auricle, heart masses are most likely lymphosarcoma in any species. WP 1678, 1744

**Ox:** Pale white, firm masses, often elongated, around the base or endocardium are most likely neurofibromas, especially in the ox.

**Dog:** Highly vascular masses, or elongated polypoid soft fleshy masses, attached to the right auricle inside or outside, are most likely hemangiosarcomas. Firm masses at the base of the heart and between the aorta and pulmonary artery, often with local spread in the pericardial cavity, are aortic body tumors (chemodectomas). These also are most common in the dog. Metastases are common in the lung. Such highly vascular masses may be seen in the liver and spleen of these cases, but it may still be difficult to accurately determine the primary tumor of origin of the metastases. Often, associated vessels rupture and cause fatal tamponade (bleed-out into the heart sac, heart muscle, or mediastinum).

**Note:** In some species, tumors at the base of the heart may also be thyroid tumors. These occur as a result of vestigial remnants being ectopic and thus more likely than normal tissues to become neoplastic. This is similar to retained testicles developing Sertoli cell tumors in dogs. One suspects that the anoxia common to the right auricle may have some relationship to the high incidence of certain tumors, as lymphosarcoma and hemangiosarcoma developing in such a low metabolic tissue.

## LOCALLY EXTENSIVE – MULTIFOCAL \* ALL

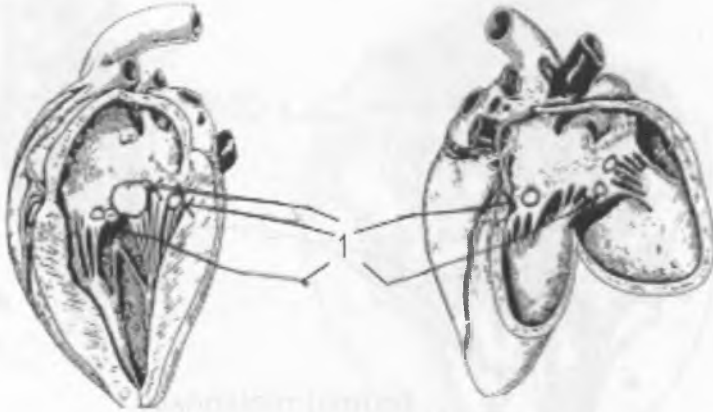


1. Pale, opaque, irregular patches of myocardial degeneration.

### Locally Extensive Degeneration

In most young domestic farm animals, opaque white linear patches of muscle in any part of the heart, often with a sharp line of demarcation, can be diagnostic for nutritional muscular dystrophy (white muscle disease) as a result of this vitamin E/Se responsive disease. The lesion may be a single discrete streak or involve a major portion of the heart, usually with pulmonary edema or a nutmeg liver indicative of heart failure. Of course, other active muscle groups in the nursing young and muscles of deglutition are commonly involved. In older animals, capture myopathy (exhaustion myopathy) may look similar. In many species, and at any age, less obvious patches of pale muscle, mostly in the ventricles, should make one consider the likelihood of neurogenic cardiomyopathy (brain-heart syndrome) in animals with brain, spinal cord, renal failure, or damage to larger nerve plexi. Pale areas without distinct borders probably represent artifacts. These latter findings should be smelled to rule out intracardiac injection sites. WP 228, 514, 323, 886, 1413

## MULTIFOCAL \* ALL



1. Hematocysts and Lymphatic Cysts.

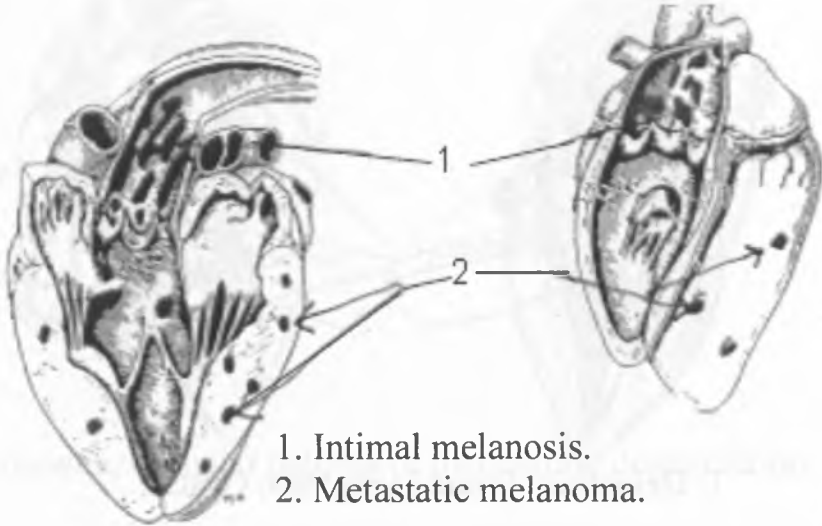
### Valvular Hematocysts or Lymphatic Cysts

Valves may be found with clear yellow fluid cysts or actually blood-filled cysts from 1–10 mm or even larger, especially anywhere on the valve leaflets of the atrioventricular valves (AV). These are considered malformations of the associated lymphatic and blood vessels and are called lymphatic and hematocysts. When small, the trapped fluid may desiccate and disappear with time. Some very large cysts may distort the valve leaflets and be cause for some valvular incompetence. WP 390, 1698

**Ox:** These lymphatic and hematocysts are common.

**Pig:** These are common in the pig, but usually are smaller than those of the cow.

## MULTIFOCAL \* ALL



### Cardiac Pigmentation

Large irregular patches of black pigment in the wall of the pulmonary artery and aorta (melanosis) are very characteristic of normal cardiac pigmentation in black-faced sheep and goats and much less so in other species. In the myocardium it can be seen in many species, especially the ox. Smaller, round, discrete, pigmented lesions in any species should be considered metastatic lesions of a melanosarcoma. Diffuse brown discoloration (brown atrophy) throughout the heart in extremely old animals is suggestive of wear-and-tear pigmentation (lipofuscin). WP 1633, 1741

## MULTIFOCAL \* ALL

1. Incomplete subaortic stenotic rings.



### Subendocardial Fibrous Bands (Incomplete Subaortic Stenotic Rings)

On the septal wall, just under the aortic valve cusps, may be irregular horizontal bands of connective tissue in the two distinct locations shown most commonly in affected animals. In any one animal, these may be only one band at either side, or several incompletely across, attached to the septal wall in this location. In all animals, these congenital bands make up the single most common heart anomaly called "incomplete subaortic stenotic rings" (ISSR). Incomplete, they are almost never of clinical significance. If the band extends across the entire wall they usually result in a complete subaortic stenotic ring, which is usually fatal early in life. WP 279, 280, 1541

## MULTIFOCAL-HORSE

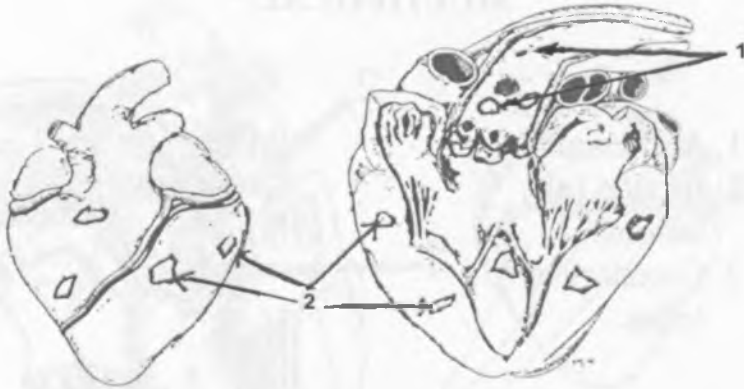
1. Ventricular endocardial plaques.



### Ventricular Endocardial Plaques

Limited almost entirely to the left ventricular endocardium are a few to many (dozens) of discrete opaque, white, discoid, 5–10 mm plaques of endothelial proliferation histologically. Their cause is unknown, but one suggestion is that they are the result of constant friction effected during systole between the septal endocardium and the opposing wall endocardium. It is very common in the adult horse. WP 48, 1764

## MULTIFOCAL \* HORSE



1. Parasite (*S. vulgaris*) endothelial granulomas.
2. Embolic related scarring.

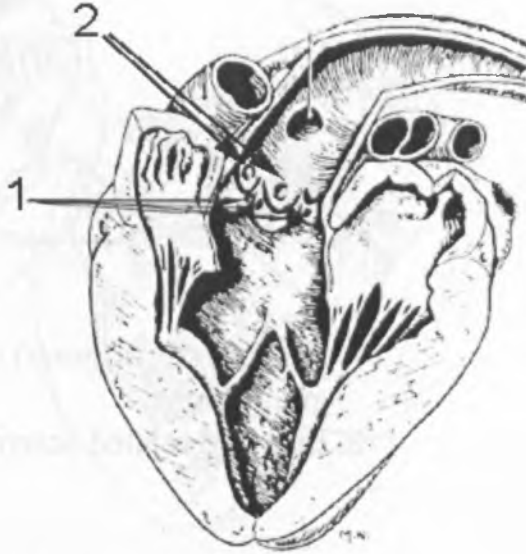
### ***Strongylus vulgaris* Scarring**

In the horse, 1–2 cm vermiform plaques, and irregular vermiform masses on the intima of the aortic arch and/or the aortic valve cusps, as well as in the cavity of the aortic valve cusps themselves (sinuses of Valsalva) may be single or multiple (2–6). When sectioned histologically, they are often mineralized and pieces of the causative agent, immature *Strongylus vulgaris*, may be found. Elsewhere in the heart, irregular pale scars of connective tissue may be found if the animal is aged, attesting to the chronicity of the lesions in the aorta. Many of these scars can be found in both atria. In young animals, viable or at least recently living larvae may be found anywhere in the entire aorta, from the cranial or caudal mesenteric arteries back to the arch of the aorta, even right into the ventricular endocardium. This indicates larval migrations from the bowel, along with minimal to marked thickening and thrombosis with larvae in the lumen, called verminous endarteritis. The newer worm medicines have all but eradicated this lesion. WP 31, 32, 1765



## MULTIFOCAL \*

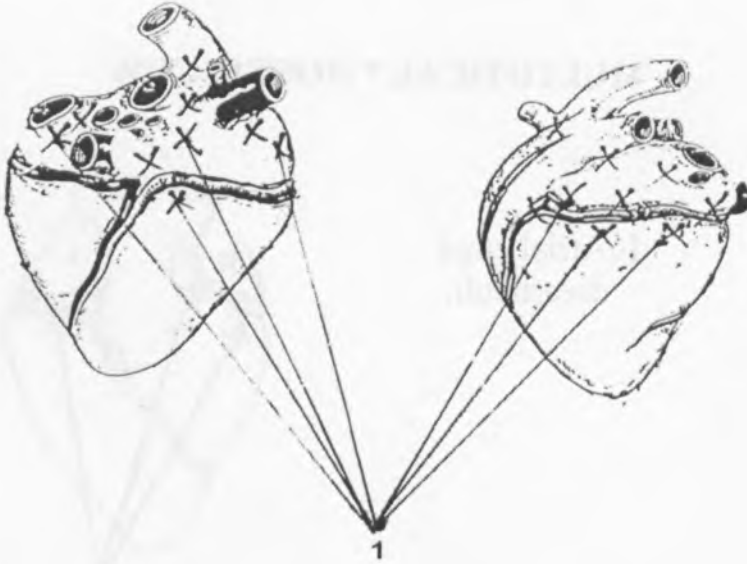
1. Aortic valve friction rub lesions.
2. Coronary arteries.



### Aortic Valve Friction Lesions

In the horse, aortic valve friction rubs are a very common lesion on the aortic valve cusps near the free edges and are found as paired 2–3 mm nodules of scar tissue. They occur about equally distant from the valve attachment site and are apparently caused at the sites of the valves' rubbing together at closure. They are called “aortic valve friction lesions” or “kissing lesions.” Sometimes there are two pairs on one side of the valve, but usually only one. All three cusps may have an opposing pair. These are to be differentiated from nodules of Arantius, which are single nodules in the center of each aortic cusp edge that are normal structures. The kissing lesion, while commonly mistaken for strongyle larval granulomas, are not related to parasites. WP 84, 85

## MULTIFOCAL \* HORSE, COW



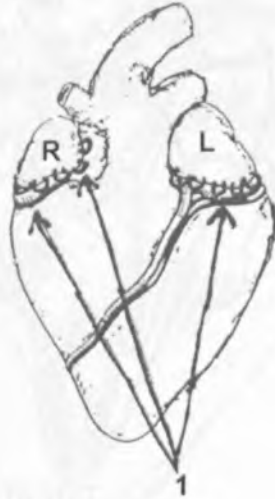
1. Fibrillar fibrovascular proliferation.

### Pericardial Fibrovascular Proliferation

Red, soft, fibrillar, highly vascular tissue in strands and plaques, attached to the tissues of the base of the heart and nearby major vessels within the pericardial sac, are very common in the horse and cow and are called fibrovascular proliferations. The cause is suspected to be minimal friction reaction, as almost no inflammatory cells are present histologically. Most cases are not clinically important, but some may become more extensive and even be a source of severe hemorrhage pericardially, with fatal tamponade resulting. Some cases may result with a severe lymphocytic pericarditis.

## MULTIFOCAL \* HORSE, COW

1. Atrial edge diverticuli.



### Atrial Diverticuli

Multiple (5–20) pale to red, round, 1–5 mm blebs of atrial tissue, lined up along the free edges of both left and right atria are called atrial diverticuli and are common findings in the horse and ox. They consist of thin walls of atrial muscle and connective tissue filled with blood in the red ones, and clotted blood in the paler ones. They may be found collapsed and slightly inverted when the blood pressure drops at death. WP 1688

## MULTIFOCAL \* DOG, CAT

1. Surface vessels thickened with pale-yellow lipid.



### Lipid Deposition, Coronary Arteries (Atheromatosis)

Various vessels of the heart, especially on the epicardium, are swollen with a definite white-yellow fat-like deposit in their walls of cholesterol. In a recent retrospective study, 19 of 21 cases in dogs that had these atheromatous deposits in their vessels also had diabetes mellitus. This lesion may be found in just about any species with diabetes and is very common in man.

WP 12135

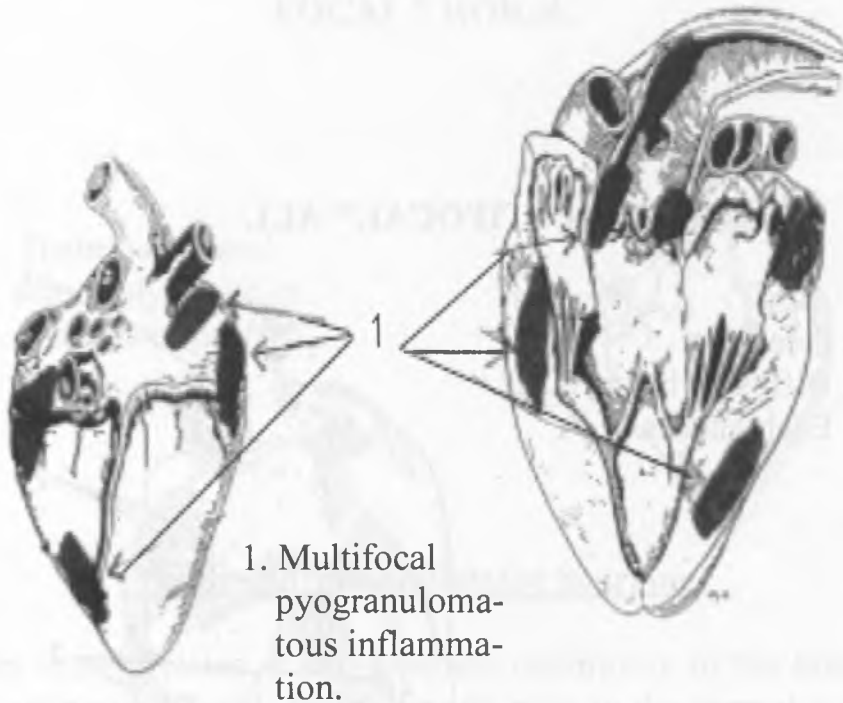
## MULTIFOCAL \* DOG

1. Intimal proliferative plaques.



### Aortic Intimal Proliferation

Aortic intimal proliferation is primarily seen in older dogs. They are opaque, pale, white plaques or strands of very thin (less than 1 mm) tissue on the intima of the arch of the aorta, but also the thoracic aorta. On histological evaluation the tissue appears to be only intimal proliferation. Grossly, the material can be elevated by knife point and peeled off with no underlying lesion to be noted. This is not fatty material of any type, and is considered only an aging change in older dogs. WP 3263, 3297



### Pyogranulomatous Myocarditis and Pericarditis

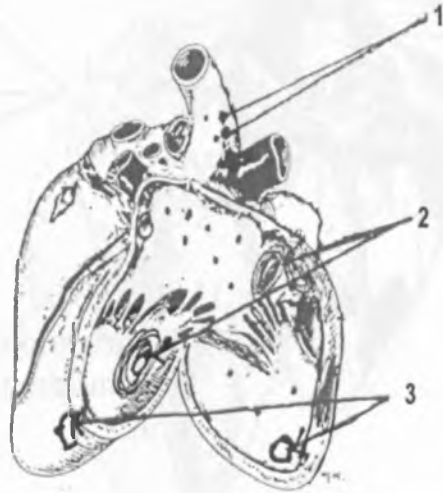
Multiple, various-sized areas of purulent inflammation may be found in and around the heart and inside the pericardium. Some may have pus, and there may even be some with multiple, fibrinous adhesions of the heart to the sac. Many granulomas may also be found scattered in the muscle throughout the heart, from the purulent thrombi on or near the aortic cusps and adjacent coronary arteries. In any species, many different agents are potentially capable of causing these septic emboli to the myocardium. WP 1636, 1645, 1719

**Ox/Cow:** Such lesions are common with traumatic pericarditis. *Actinomyces pyogenes* is commonly isolated.

**Dog:** The fungal agent *Aspergillus terreus* is a common cause of this entity in German shepherd dogs, specifically.

## FOCAL/MULTIFOCAL \* ALL

1. Petechiae.
2. Diffusion hemorrhage.
3. Ecchymoses.



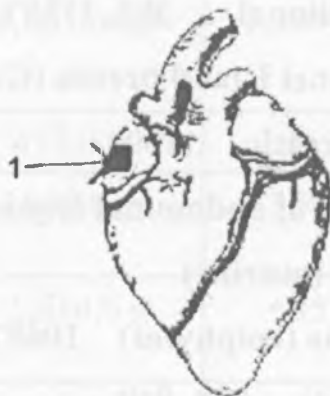
### Hemorrhages of the Heart

Small (pinpoint) to larger (ecchymotic, or even diffuse, hemorrhages scattered on the epicardium, endocardium, or even within the heart muscle, are usually only an artifact seen with dying, probably in part from terminal anoxia. Almost without exception they should be ignored. Pools of blood may suggest a bleeding problem in any species, but again caution is advised in their interpretation. In a busy necropsy room over the years, maybe only one or two cases of significance have been seen. WP 1631

**Fig:** Streaked hemorrhages and pale heart muscles together may be helpful in the diagnosis of mulberry heart disease associated with vitamin E/Se responsive disease.

## FOCAL \* HORSE

1. Thebesian vessel scar (only found in this area of heart).



### Thebesian Vessel-Related Scarring

Thebesian vessel-related scarring occurs commonly in the horse only, and is characterized by: **1.** being located only in the central portion of the right atrial wall; **2.** being usually square or roughly rectangular shaped; **3.** having only a single lesion present in most instances; **4.** parasites are usually not found in aorta or coronary vessels. It is to be differentiated from the scarred lesions of the *S. vulgaris* parasite lesions which are: **1)** usually round in outline; **2)** often have multiple lesions; **3)** they are found scattered in the wall of all chambers; and **4)** parasites themselves are usually found in the aorta or near the coronary artery ostia, allowing for thrombi formation and embolic shower source to the heart proper.

The pathogenesis of these Thebesian lesions have been suggested associated with its name, that the right atrium has no major nutrient artery, only the tiny vessels, the Thebesian arterioles, coming from the lumen directly. These tiny vessels may not be able to supply enough oxygen and nutrition under certain cardiac muscle conditions such as a heavy workload, and the fibers degenerate and are replaced by fat or connective tissue. This has not been proven. WP 37, 38, 39



## **Types of Fat Necrosis**

- 1. Nutritional 595, 11587, 11593, 19812**
- 2. Channel Island breeds (Granulomatous) 6211, 11289, 19826**
- 3. Pancreatic 11594**
- 4. Bilateral abdominal (equine) 186, 187, 188, 189**
- 5. Viral (murine)**
- 6. Fescue (epiphytes) 10687, 17453**
- 7. Ketotic 840, 949**
- 8. Freezing (ovine laying down)**
- 9. Perirenal (deer) 19289, 19290, 19327**
- 10. Ischemic 3093, 19013, 12964**
- 11. Traumatic**
- 12. Mycotic (bovine)**
- 13. Idiopathic**
- 14. Subcutaneous (cats) 11589, 11590, 19816**
- 15. Spontaneous (rats)**
- 16. Pedunculated fat**

## KLOTZ SOLUTION FORMULATION

Water	50 gallons 18,925 liters	25 gallons 9,463 liters	10 gallons 3,785 liters
40% Formalin	946.0 ml	473.0 ml	189.2 ml
Chloral hydrate $C_2H_3Cl_3O_2$	1,892.0 g	946.0 g	378.4 g
Sodium sulfate $Na_2SO_4 \cdot 10H_2O$	3,027.0 g	1,514.0 g	605.4 g
Potassium sulfate $K_2SO_4$	47.0 g	24.0 g	9.4 g
Sodium chloride NaCl	56.8 g	28.0 g	11.36 g
Sodium bicarbonate $NaHCO_2$	113.65 g	57.0 g	22.73 g
Ascorbic acid $C_6H_8O_6$	100.0 g	50.0 g	20.0 g

Tissues placed in routine formalin solutions rapidly lose their color and become very stiff, but they keep very well and for a long time. The use of Klotz solution is recommended for short-term storage for demonstration purposes. Chloral hydrate is a controlled substance, expensive, and harder to obtain, so citric acid may be used (less satisfactorily) instead. Too much formalin will darken and stiffen the tissues one is trying to save for demonstration.

## LIST OF SOME OF THE MORE COMMON GREEK AND LATIN PREFIXES AND SUFFIXES

**Note:** This list, if learned, is almost guaranteed to increase the vocabulary of almost anyone. It's a necessity for anyone in the biological sciences.

L – Latin derivation

Gr – Greek derivation

-an	L-without, not	arthro-	Gr-joint
ab-	L-from	-ase	L-enzyme
acro-	Gr-extremity	auto-	Gr-self
ad	L-to	bi-	L-two
adeno-	Gr-gland	bio-	Gr-life
adipo-	L-fat	blast-	Gr-germ, bud
ala-i	L-wing	bothri-	Gr-pit
alb-i	L-white	brachi-	Gr-arm
-algia	Gr-pain	brady-	Gr-slow
ambi--	L-both	brevis-	L-short
amphi-	Gr-both	caec-	L-blind
amyl-	L-starch	capit-	L-head
an-	Gr-without, not	card-	Gr-heart
ana-	Gr-up	cata-	Gr-down
ankylo-	Gr-bent	cer-	Gr-horn
ante-	L-before	cervix-	L-neck
anti-	Gr-against	chlor-	Gr-green
aqua-	L-water	choan	Gr-funnel
archi-	Gr-first	chrom-	Gr-color

chyl-	Gr-juice	dys-	Gr-bad, ill
chym-	Gr-juice	ectasis-	Gr-dilated
clast-	Gr-to break	ecto-	gr-outside
coel-	Gr-hollow	ella,us	L-diminutive
collum-	L-neck	-emia	Gr-blood
conch-	Gr-a shell	endo-	Gr-within
corn-	L-horn	ento-	Gr-within
cortico-	L-bark	enero-	Gr-intestine
cruci-	L-cross	erythro-	Gr-red
crypt-	Gr-hidden	ex-	L-out
cten-	Gr-comb	extra-	L-beyond
cyan-	Gr-dark blue	fenestra-	L-window
cyn-	Gr-dog	fer-	L-to carry, bear
cyt-	Gr-cell, hollow	flav-	L-yellow
de-	L-down, from	fug(s)-	L-flee
dent-	L-tooth	gamo-	Gr-marriage
derma-	Gr-skin	gastro-	Gr-stomach
di-	Gr-double	-gen	Gr-to produce
dia-	Gr-through	-glia	L-glue
diplo	Gr-double	glossi-	Gr-tongue
dis-	L-down, from	glyco-	Gr-sweet, sugar
duo-	L-two	gnath-	Gr-jaw
dura-	L-hard	-gogue	Gr-leading
duct-	L-convey	gon-	Gr-angle-seed

graph-	Gr-to write	lepto-	Gr-thin
haemo-	Gr-blood	leuco-	Gr-white
hemi-	Gr-half	lemma-	Gr-skin
hepta-	Gr-liver	lingua-	L-tongue
hetero	Gr-different	lipo-	Gr-fat
hex-	Gr-six	lith-	Gr-stone
hippo-	Gr-horse	-logy	Gr-discourse
histo-	Gr-tissue	-lysis	Gr-divide, destroy
homo-	Gr-same	macro-	Gr-large
horm-	Gr-to excite	macula-	L-spot
hyalo-	Gr-glassy, clear	mal-	L-bad, ill
hydro-	Gr-water	mast	Gr-breast
hymen-	Gr-membrane	medi-	L-middle
hypo-	Gr-under	mega-	Gr-large
in-	L-not, without	mes-	Gr-middle
in-en	L-into	meta-	Gr-change, beyond
inter-	L-between	-meter	L-measure
intra-	L-within	micro-	Gr-small
iso-	Gr-equal	mono-	Gr-single
-itis	L-inflammation	morph-	Gr-form
lact-	L-milk	morti-	L-death
lacun-	L-pit, lake	myi-	Gr-fly
lamin-	L-layer, plate	myo-	Gr-muscle
leio-	Gr-smooth	myelo-	Gr-marrow

myxo-	Gr-mucus, slime	opisth-	Gr-behind
necro-	Gr-dead	or-, os-	L-mouth
nemo-	Gr-thread	orchi-	Gr-testicle
neo-	Gr-new	ortho-	Gr-straight
nephro-	Gr-kidney	-ose	L-sugar
neuro-	Gr-nerve	ossi-	L-bone
nid-	L-nest	osteo-	Gr-bone
nigr-	L-black	osti-	L-door
noct-	Gr-night	ot(o)	Gr-ear
noto-	Gr-back	oxy	Gr-sharp
nucleus	L-nut	pachy-	Gr-thick
nud-	L-naked	palaeo-	Gr-ancient
nyssus-	Gr-to prick	pan-	Gr-all
oct-	L-eight	par-	L-to beget
odont-	Gr-tooth	para-	Gr-beside
oid-	Gr-like	pariet-	L-wall
olig-	Gr-few	ped-	L-foot
-oma	Gr-tumor	penta-	Gr-five
-omma	Gr-eye	peri-	Gr-around
ophalo-	Gr-navel	petro-	Gr-stone
oneh-	Gr-barb	phago-	Gr-to eat
oo-	Gr-egg	phil-	Gr-loving
opercul-	L-a cover	phlebo-	Gr-vein
ophthalm-	Gr-eye	phon-	Gr-voice, sound

phot-	Gr-light	ptero-	Gr-wing
phyll-	Gr-leaf	ptyl-	Gr-saliva
phyto-	Gr-plant	pulic-	L-flea
pia-	L-tender	pulmo-	L-lung
plasm-	Gr-formed	pupa-	L-baby
platy-	Gr-flat	pyl-	Gr-gate
pleo-	Gr-more	pyo-	Gr-pus
pleuro-	Gr-side	pyri-	L-pear
pneumon-	Gr-lung	quadr-	L-four
pneumo	Gr-air	rachi-	Gr-spine
pod(i-o)	Gr-foot	ram(i)	L-branch
poly-	Gr-many	re-	L-again
pons-	L-bridge	rect-	L-straight
porta-	L-gate	ren-(i)	L-kidney
post-	L-after	ret-(a-i)	L-net
pre-	L-before	rhabdo-	Gr-a rod
pro-	Gr-before	rheo-	Gr-to flow
proct-	Gr-anus	rhino	Gr-nose
proto-	Gr-first	rhizo	Gr-root
psalter-	Gr-book	ryncho-	Gr-snout
pseudo-	Gr-false	rostri-	L-beak
psor-	Gr-itch	rumin-	L-throat
psych-	Gr-breath, soul	sagitta-	L-arrow
psyll-	Gr-flea	salpi-	Gr-trumpet

sarco-	Gr-flesh	squam-	L-scale
sapro-	Gr-putrid	stat-	Gr-standing
scalar-	L-ladder	stell-	L-star
schizo-	Gr-cleft	steno-	Gr-narrow
sclera-	Gr-hard	stereo-	Gr-solid
scoli-	Gr-bend	sterco-	Gr-dung, feces
-scope	Gr-see	stetho-	Gr-breast
scut-	L-a shield	stigmo-	Gr-point
seb-	L-tallow	stoma-	Gr-mouth
sect-	L-to cut	strati-	Gr-layered
sella-	L-saddle	strepto-	Gr-turned
semi-	L-half	stria-	L-furrowed
sept-	L-a wall	strongylo-	Gr-round
septic	L-putrid	stylo-	L-column
serra	L-a saw	sub-	L-under
seti-	L-bristle	super-	L-over
sialo-	Gr-saliva	supra-	L-above
siphon-	Gr-tube	sym-	Gr-together
siphuncal-	L-small tube	syn-	Gr-together
soma-	Gr-body	syringo-	Gr-pipe
somn-	L-sleep	tachy	Gr-rapid
sperm-	Gr-seed	tact-	L-touch
spheno-	Gr-wedge	taen-	Gr-ribbon
splanchno-	Gr-viscera	ov(i)	L-egg



tapet-	L-carpet	trop-	Gr-turning
tel-	Gr-far, end	trophy-	Gr-nutrition
tenui-	L-thin	trypano-	Gr-an auger
terato-	Gr-monster	tumor	L-swelling
tetra-	Gr-four	tunic-	L-a garment
thalam-	Gr-chamber	tympano-	Gr-a drum
theco-	Gr-case, covering	tyro-	Gr-cheese
theli-	Gr-nipple	ultra-	L-beyond
therio-	Gr-beast	unci-	L-hook
thermo-	Gr-heat	-uncula	L-little
thromb-	Gr-clot	unguli-	L-hoof
thyro-	Gr-door, shield	uni-	L-one
thysan-	Gr-fringe	uro-	Gr-urine
tok-	Gr-birth	vaso-	L-vessel
tomo-	Gr-to cut	ven-	L-vein
tox-	Gr-poison	ventr-	L-belly
trabecul-	L-a small bean	vermi-	L-worm
tracheo-	Gr-windpipe	vesicul-	L-blister
trachy-	Gr-rough	via-	L-way
tremat-	Gr-hole	villi-	L-shaggy
tri-	Gr-three	vita-	L-life
tricho-	Gr-hair	vit-	L-glassy
troch-	Gr-pulley	vivi-	L-alive
trombid-	Gr-timid	vora-	L-to devour

# Affixes: Prefixes from the Language Table

xantho	Gr-yellow	zoo-	Gr-life, animal
xero-	Gr-dry	zygo-	Gr-yoke
xylon	Gr-wood	zym-	Gr-ferment

L – Latin derivation  
Gr – Greek derivation

## Ancillary Testing from the Necropsy Table

These testing techniques are intended to be simple and relatively easy procedures to follow or even precede the actual necropsy at the necropsy table. They have been used over the last 50 years by the authors and have proven useful for gaining additional information to aid or complement the diagnoses.

Certainly not all necropsy facilities will have the equipment discussed in these procedures, but maybe because of their proven value they may be obtained in each laboratory with time.

### Field Weighing of Large Animals

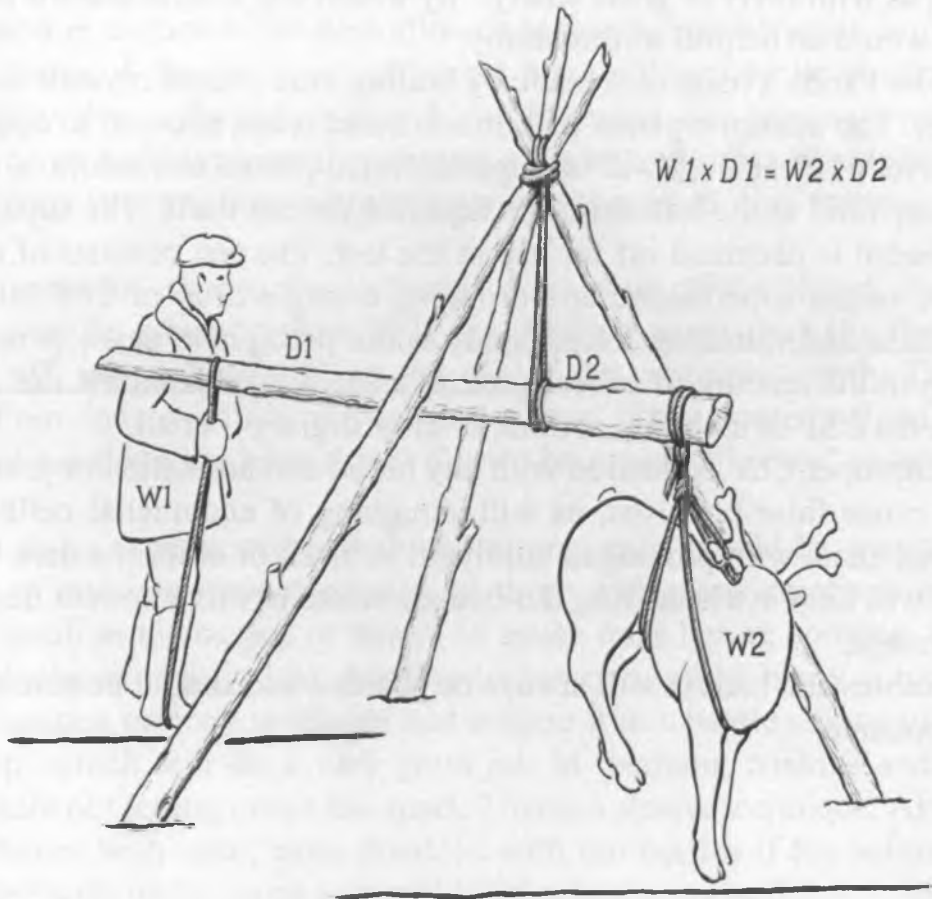
Dr. Alan Woolf, Southern Illinois University, Carbondale, Illinois

Weighing of the carcass: Scales are available most everywhere except not in less accessible locations including jungles, forests, etc. Even at less well-equipped necropsy facilities this technique can be modified for use as needed. Our initial use has been to weigh adult moose, bears and caribou in a research project in Newfoundland under the auspices of Dr. Stuart Peters, the Director of Wildlife, Newfoundland, in determining the cause of severe die-offs of caribou calves there.

The pine forests there supplied the 9–10 foot straight poles, three of which, cleared of branches, were used to construct a tripod tied at the top with very strong nylon parachute cord. A double piece of cord hung down from the apex about 12 inches as an open loop through which a fourth cleared pole was inserted into its approximate equilibrium position. A notch was made on the pole to hold it roughly in place and another notch made at the thick end, to which the large animal's body was closely attached, again with the lightweight but extremely strong parachute cord (easier to carry than thick rope). The long, thin end of the pole also has a larger loop of the same cord loosely hung over this pole in order to pull the longer lever arm portion down, allowing a known weight, usually the pathologist or biologist, to step into the loop. This allowed the loop to be moved closer to or further from the fulcrum to achieve balance. This he does until the lever arm length, easily measured with a pocket tape, times the known researcher's weight (with clothes, as measured by scales at

camp) would “balance” the shorter lever arm length and animal’s carcass weight using the simple equation of known body weight times lever arm length equals the unknown animal’s body weight times its lever arm length when the pole is balanced. Almost any tall enough structure could be used as the fulcrum, even a stout branch of a standing tree. The human’s weight was always checked at the camp or laboratory with the relative same gear on each day.

Even weighing each other researcher with this technique was never more than 3–5 pounds off by actual scale comparison. Of course a longer lever arm would be used for the heavier animals.



Marion E. Newson

## Pandy's Reagent and CNS Disease

Dr. John Bentinck-Smith, Cornell University, Ithaca, NY

The normal cerebrospinal fluid has only a small amount of protein present but with most CNS infections and some brain destructive processes, as parasite migration or trauma, the protein levels can increase significantly.

In the postmortem room, it is often wanted to have a rapid answer to the question, "Were the nervous signs seen in this animal the result of an infection, or possibly due to the often common effect of a metabolic disease, as with fever or grass tetany," by which this simple test for excess protein would be helpful differentially.

The Pandy's reagent is made by boiling pure phenol crystals in sterile water. The phenol crystals will dissolve and when allowed to cool, the upper layer of clear fluid will be supersaturated phenol test solution, while the darker fluid at the bottom is the liquified phenol itself. The supersaturated phenol is decanted off for use in the test. The test consists of a half test tube of the supernatant, and dropping a single drop of CSF into the supernatant and watching for a cloudy white precipitate develop in relationship to the amount of excess protein present, which should not cloud at all in the CSF of a normal animal, or only slightly if at all.

Improper CSF collection with any blood contamination or joint fluid will cause false positives, as will sloughing of endothelial cells from the spinal canal with prolonged autolysis. A black or at least a dark background will help in visualizing the opaque white positive protein denaturation change.

Rabies and listeria will always be positive and should be considered if it is positive.

## Clean, Grease-Free Skeleton Preparation for Examination

Often we are asked to get or give a fractured limb or other bone for the clinician, owner, researcher, etc., from an animal we have necropsied. They may want it in a hurry for whatever reason and we the pathologists often want it clean for photography, comparisons, lesion observations, etc.

We take off as much skin and muscle mass as is easily done and we let the autoclave or even pressure cooker do the rest. However, one must be careful to not make the bones too soft or they will crumble too easily. We use a small quantity of non-sudsing detergent in the water, being even more careful, as it will also remove mineral rapidly from the bone. The bones being prepared must be well under the top of the water when in the pressure cookers or boiling containers. The amount of soap used must be titrated with experience, as very thin or small animal bones are extremely tender to this technique.

Another caution is the time allowed to “cook,” which again is guided by experience. A small, thin, and young bone will surely be cleaned far more easily than a larger, thicker bone. Stopping the process every 15 minutes or so and checking the amount of meat and other tissues still attached firmly is better than ruining the whole specimen. Use the soap judiciously.

A common error in the technique is to pour off the liquid, floating bone marrow fat when finished. **It is an absolute must that the fluid be flooded off, not poured off, at the top** of the container, gently. Do not shake. If not done carefully, the buoyant grease, if just poured off, will immediately penetrate the bone that will then be greasy “forever” in any display setup.

Even an experienced pathologist or anatomist would be wise to put each set of multiple bone structures, as those with many sesamoid bones, large or small as the carpus or tarsus, in gauge bags before cooking. Some of the fabellae are easily lost. Another technique used by many is to dry a skinned carcass or bony specimen and subject it to a beetle colony of *Der-mestes* sp. which will do a very good job in retaining tendons and ligaments while not letting joints fall apart. This is a slower technique. Also, do not put bones with meat, even dried, in with the beetles if the animal has been killed with barbiturates as it will kill the beetles as well.

The bone-cooking technique can give clean, greaseless specimens in hours when combined with a high-pressure water hose to force off all the muscle and soft tissue still loosely attached.

## Muscle Weights and Hypertrophy

It is often asked about or noticed at necropsy that some smooth muscle, but mostly some skeletal muscles, are thicker (bigger) or larger than normal. Such examples of this enlargement is called hypertrophy and is sometimes caused by an increased workload as in physical training, but also by related disease in other organs as with chronic lung disease, as pulmonary emphysema causing marked hypertrophy of the diaphragm. Many instances of bowel obstruction may cause preceding segments, or rarely, more caudal sections of bowel, to overwork, trying to push content past the obstruction. It is also seen in the distal ileum and distal esophagus in the horse and pig, as entities called idiopathic hypertrophy. It is also present in other examples of obstruction.

However, in known causal cases it is difficult in some instances to give an objective evaluation other than thickness.

In these instances it is suggested to weigh a segment of bowel either as a segment of the gut itself or a square segment of the thickened diaphragm or gastric wall or a large dilated, very thin wall to get a specific weighed area of these tissues and compare them to a similar sized but normal area from a normal animal, for an objective value as weight per square unit for comparison. WP 73, 78, 244, 2434, 19914

## The Collection of Heavy Tiny Particles

One of the tedious things to collect at the necropsy table are the one or many pieces of lead used in a shooting that remain in a body. Radiographs can be useful in almost any animal shot with lead, as pieces associated with hitting bone and fragmenting the radiograph will be demonstrated easily. The collection of these heavy but smaller pieces is more difficult.

One proven method to find even the smallest pieces as fragments or particles, is to put all the cooked tissue and “soup” from an autoclaved or otherwise prepared “soup” of the bird, such as an eagle, for example, into a deep (tall) three-foot container.

Then, using a high-pressure water hose, break up the cooked tissues. This will allow the autoclaved soup materials such as feathers and fat to float and be flooded off, leaving behind only the heavy lead and stony materials. These heavy materials can then be washed through graded sieves for collection or just examination in the white enameled trays.

Even rumen content that may have suspected small lead pellets or the like can easily be treated in this manner, without cooking, to find the heavy particles. The gastrointestinal content can also be treated in the same way to find pieces of ingested lead in ducks, for instance.



## **Bone Breaking (Shear Force) Strengths**

During the necropsy procedure, the prosector, usually a student, is advised to isolate a central rib and bend it against its curvature to get an idea of its resistance. The comment usually from them is how are they supposed to judge with only their limited experience? They are told that by the time they have done this to a dozen bones from different animals, they will have an idea that it is easier to break the bones in some animals than others, indicating a difference they can judge.

A more objective “bone breaking” technique can be used with many smaller bones of large animals or even the larger bones of smaller animals. It is almost impossible to use with really strong bones of larger animals or even some of the leg bones of dogs and cats. It is extremely useful to use with smaller mammals, using the femur of rats, squirrels, etc. See also page 30.

It is a general aid in giving an objective evaluation of bone strength that can be related to age, disuse, nutrition, or disease, etc.

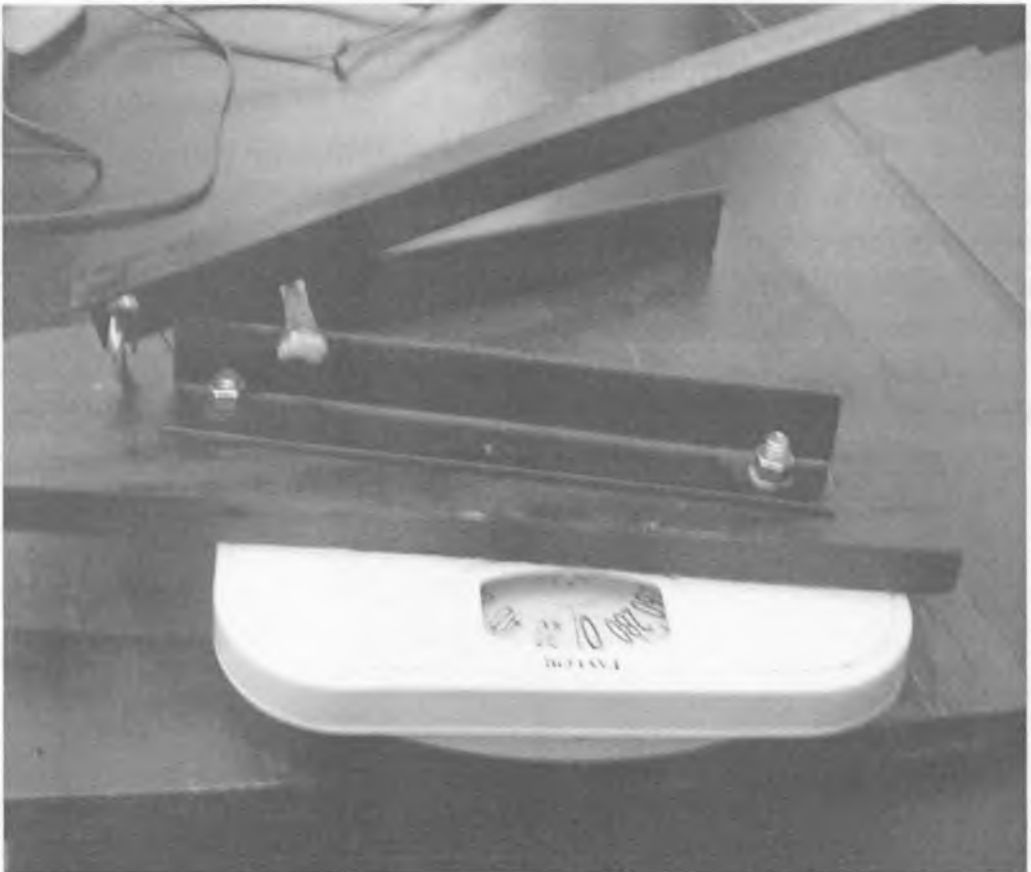
The bone supports used are 1/8-inch angle irons, 6 inches long, attached in a V-shaped fashion to a 12-inch solid surface as a piece of 3/4-inch plastic. This is placed on the bathroom-type platform scales raised on a wooden platform, allowing an 1/8-inch iron bar attached to its fulcrum attachment site at the same level as the V-shaped bone-breaking supports. The bone chosen for breaking is the same one as used for the control bones, as the femur, etc. They are roughly cleaned of soft tissue, being careful not to nick the bone shaft and to place the bone on the bone support of angle iron, with each support point being 1/3 the distance from each end of the bone.

The lever attached on the wood frame extends over the approximate center point of the bone when on its supports, which are placed on the platform scales. This lever is the same thickness, 1/8 inch, as the supports and can be 18–30 inches long, but touches only the approximate center of the supported bone.

Slow downward pressure on the lever is then exerted, with the increasing pressure measured on the scales and noted when it breaks, usually quite suddenly, giving an objective strength for each bone, including bones of control animals for comparison purposes.

Of note is the need for a reference table of breaking strengths of known aged similar species being evaluated, to be used as controls in order to give an indication of the relationship between the individuals. The same type of controls can be used for any bone strong enough to be measured conveniently by this technique. The more uniform the bones used to establish the control levels, the more useful in giving the objective numbers needed to evaluate age or disease states for comparison of soft bones in nutritional imbalances or metabolic variation in disease states, such as rickets, renal failures, or increased density in hypercalcemias when male animals are fed lactating animal diets.

Of course the materials used can be enlarged and strengthened for use of this technique in larger-boned animals. It is also noted in many studies using these techniques that the bone strengths recorded increase, in rats, for instance, up to about 15 months of age. Then they plateau for several months and decrease gradually. Also it should be noted that control levels, for instance, in squirrels or fox in one area, may not be the same levels at known ages in other areas because of dietary differences. Although usually close, it may be necessary to establish the fact with another comparative study for each geographically different area.



## The Clearing of the Whole Body for Skeletal Evaluation

Dr. Howard Evans, Cornell University, Ithaca, NY

This technique is used for whole body preparation of the carcasses of very small animals such as snakes, birds, even embryos, in which the whole carcass is immersed in a 2% potassium or sodium hydroxide mixture until the entire body is relatively translucent. Feathers and some other external body coverings may have to be removed previously. After the body is transparent, it is immersed in an Alizarin Red S solution for a several-day soaking in which all tissues are red (purple) stained. The entire carcass is then placed in glycerol, at which time most of the dye stain is removed, leaving only the red-stained (purple) bones or mineralized soft tissues still avidly stained in the rest of the almost invisible body.

This will allow for better bone evaluation in cases of bone malformation examinations in toxicological studies and finding of early mineral depositions in pathological studies and normal early stages of bone development and other related studies involving bone development.

This technique works well for fetal studies or young animal studies that do not have a heavy hair coat that may have to be removed before clearing the skeleton. It is good for snakes and about any studies for bone evaluations and even for soft-tissue mineralization, such as the aorta or dura mater of the spinal cord.

Care must be used in handling these very fragile, cleared bodies.

## Hoof Examination by Wall Removal

Dr. Peter Ossent, Universitat Zurich, Switzerland

It has been a problem over the years to examine the inside bones and joints of feet of most large domestic animals because of the difficulty in removing the hoof wall from the feet in order to examine the inner structures. As most diagnostic facilities have hand-type saws or band saws to cut the wall as well as the bone structures for closer examination, it still leaves the attached wall to cover other possible lesions.

This technique involves the heating of the entire foot and distal bones in a container partly filled with water. An electric hot plate is used to heat the water to only 60–65°C.

The heating time for large animals, when the water reaches 60–65°C, is approximately 40–60 minutes for cattle and horses, and only 15–20 or fewer minutes for smaller ruminants and pigs. The time of immersion can be varied without damage to the foot, but do not allow the temperature to rise above 65°C.

The feet are cleaned first and examined for superficial lesions. Holding the hoof wall, after heating as above, in a vice, the metacarpals and metatarsals can be jerked suddenly to both sides to separate the horn from the underlying tissue and be removed. If some difficulty is encountered, the foot should be reimmersed in the 60–65°C water bath for a longer period. Higher temperatures should NOT be used, as they will cook and discolor the tissue, hiding the lesions. Also, the hooves should be examined as soon as possible to prevent discoloration even without higher temperature use.

Of course, continued examination of involved ligaments, tendons, bones, joint surfaces, etc., is necessary, but the hoof wall removal initially will make most of the remaining examinations easier.

## Humane Large Animal Euthanasia

Under certain conditions many animals must be killed rapidly, with minimal likelihood of further damage to oneself, caretakers, and others. A good example is an accident on the highway with both humans and animals at risk, with such injuries as partial evisceration of the horse. The police have often asked how to kill the animals in pain, etc. They often shoot them between the eyes, but in the long-nosed horses, dogs, and others, even apes in the zoo that must be killed, this location will often destroy the olfactory ability of the animal but will not result in the instant death which is desired. Multiple shots, in one case, involved 8 shots to the ape's head, and it still struggled even though it had its nasal passages absolutely destroyed.

We have subsequently tried to advise police officers, game wardens, and others an almost 100% sure way to kill rapidly in such cases. It is to draw a line between each eye and the opposite ear, forming a cross, and hit at the junction, aiming towards the spinal cord (foramen magnum).

Even then, some animals will respond to movement of the carcass minutes after shooting, but these actions are usually only spinal reflexes. Of course, no one should be behind or beside the head of any animal being shot.

The pig is one exception to this method of shooting, as large pigs have greatly enlarged diploe, sinus cavities in the skull that can deflect small-caliber ammunition. They can be more quickly killed if shot in the back of the skull, aiming towards the nose.

In the event of not having the chemical nor a gun for the euthanasia, a sharp necropsy knife with a 6-inch, slightly curved blade is a very efficient instrument used with a halter, to hold the head down towards the front feet, enlarging the dorsal joint space between the skull and the first vertebra. Holding the knife crosswise in the midline over this joint with one hand and hitting the hilt forcefully with the palm of the other will instantly transect the spinal cord. Caution is advised when the large animal falls.

The large pig can also be killed with a noose put on the upper snout, from which the pig will naturally pull backwards, exposing its entire throat and allowing a sudden stab into the thoracic inlet just above the bony sternum, directed caudally and to one side, cutting the anterior vena cava. The pig will immediately stop squealing and bleed out while standing. WP 9509, 20694

### **Intestinal Villi Movement**

To better assess the motility and appearance of intestinal villi, slowly flow on warm saline, and note also their presence and size.

### **Lymphosarcoma and Adipose Tissue**

Lymphomatous (tumor) tissue in very fatty locations such as the epidural space can be difficult to visually differentiate from adipose tissue depots. One simple way to check at the necropsy is to compare the suspected tissue's floatation rate in the fixative or water. WP 798, 16859, 20230, 20234

### **Better Lung Tissue Preservation**

To better define the histology of certain lung lesions is to use gentle bronchial perfusion with fixative of a lobe with a syringe and needle, depending on size, before immersion of the lung in fixative. A control section of lung for comparison, not treated by bronchial perfusion before final fixation, may be very enlightening.

### **Glass Slide Smears**

In preparation of imprints/smears of necropsy specimens, warm the unused slide surface first by placing the slide on the forearm for a few seconds. This hastens drying of cells on the preparation and reduces cell distortion. This procedure is particularly useful when searching for intracellular organisms, i.e., Toxoplasma, Histoplasma, etc.

## Short-Term Burial for Carcass Preservation

Probably not a recommended procedure to be suggested here, but it is one which has served to good effect when nothing else is available. Many carcasses have been refrigerated early after death, either natural or with euthanasia, only to be fairly decomposed at the time of necropsy because of the good insulation effect of fat condition, thick coat, or even wool as with sheep. Even in a good postmortem refrigerator in which the actual temperature of the carcass with a wool coat or much fat has been measured, it has been noted that the internal body of these fat or well-insulated animals will initially increase significantly while in the refrigerator.

The suggestion given here is that putting the body of an animal with minimal insulation (wool or excess fat) in the cool ground, will immediately allow cooling, as the earth will drain the heat from the carcass, giving appreciable cooling to the carcass and prevent rapid bacterial multiplication. Animals killed in the fall and buried quickly may even be of diagnostic value, carcass-wise, in the spring, 8–9 months later. Of course, common sense must be used, as an animal already starting to decompose when buried may not be suitable using this technique. WP 16933

## Parasite Demonstration and Collection

Often we are called upon as pathologists to collect and show gastrointestinal parasites to the owners and clinicians or even to collect them in a research endeavor.

At the necropsy table, one can often pour the abomasal content or part of it, through a series of graded wire mesh of known size from coarse to fine, put a small mass of content in the top coarse mesh and wash it softly with a small hose to get most of the finely ground-up dark ingesta through, into the next smaller size mesh to clear the water. The more coarse material trapped in the sieve can then be tipped into rectangular white enamel pans, again using a soft-flow hose to separate the visible parasites from the remaining debris. The parasites present may be removed with forceps to small saline-filled or other preservative fluid (not hypotonic tap water) vials for later identification from each sieve as they may appear.

The largest parasites will be found in this coarse sieve but smaller parasites will be washed progressively through to the next sieve. Again, finger collections of the debris may be washed in the white trays for collection. The sieve series material with finer mesh can be washed out of the sieve by tilting and into the white enamel trays for similar collection as above. A cascade of three sieves of coarse, medium, and fine will suffice for finding most of the parasites present.



Some caution is advised with leaving some species of parasites in water used for flushing, as it may cause some destruction of the parasites by swelling and rupturing caused by the hypotonic tap water.

The white enamel pans are among the best containers that can be used, as they clean easily and make visualization of the parasites easier. The gravity source seen here, with the on-the-job-constructed holder for the graded sieves, was very beneficial in Newfoundland, with a small hose from the drum to direct and control the water from the filtered river water.



## Simple Feed Testing in Rats

Dr. Henry Smythe  
and Dr. Charles Carpenter  
Carnegie Mellon University, Pittsburgh, PA

We are often presented with various species of young animals which fail to gain weight on certain commercial feeds, and which, at necropsy and after histological study, do not have diagnostic lesions except of emaciation.

An attempt is made to obtain from the owner enough sample of the suspect feed to feed 6–10 young rats for three weeks, at least. The suspect feeds have been, for example, milk replacer for calves, hog, mink, fish, dog feeds, or other species-specific commercial feeds.

Both the suspect feed and a well-recognized control feed for the animal species involved are finely ground and fed ad lib to 10 rats matched in age, sex, and weight, in separate but similar housing. Their individual weights are all recorded before and weekly for comparison evaluation. The final weighings should be similar, and if not, a toxic or other deleterious factor may be involved with the suspect feed. The feed company and owner do not always easily accept that feed is at fault until proven by this technique. Show them the growth curves for both rat groups and ask them why. It is now their problem.

This simple technique will often demonstrate that the suspect feed is faulty, but will not identify the toxic factor itself; it will tell the owner, the feed company, and the clinician where the fault lies, in the failure of the animals to gain weight normally.

The technique has been used successfully to show excess of vitamin D in dog feed from a damaged vitamin D chute into the company's mixer process. Another was the toxicity of arsenic in dog feed from the failure of the feed mixer to be thoroughly cleaned out of arsenic-treated chicken feed just before dog feed was made. Pig feed was shown to be accidentally mislabeled as growing ration instead of maintenance ration, and in a case of frog feed, the use of rancid ground fish caused severe losses from steatitis.

## **Reportable Features of Most Lesions**

Location

Color

Size/Weight

Shape

Consistency

Number (#) or Percent (%) Involved

Content

Odor

**There are many common lesions,  
but never any normal ones.**

---

### **Morphological descriptions and interpretations of lesions may include the following:**

Distribution: Organ(s) Unilateral – Bilateral

Focal – Multifocal (Disseminated)

Locally Extensive – Diffuse

Whole Body – Localized

Generalized

Time: Peracute – Acute – Subacute – Chronic – Chronic Active

Severity: Minimal Moderate Marked

Slight Severe

Cause: Verminous – Bacterial – Chemical – Viral –

Traumatic – Protozoal – Mycotic – Toxic – etc.

Type: Croupous – Hemorrhagic – Purulent – Fibrinous –

Fibrinopurulent – etc.

## About the Author

John M. King started life at Boston City Hospital in 1927 and followed up visiting many ports of call in and around Boston as his three older sisters and he were juggled among three aunts and grandparents. Finally, at seven he was deposited at a boys' farm home school in Marlboro, Massachusetts, for ten years. He got to follow the footsteps of a great local veterinarian who visited to treat any of the many animals needing his care. John learned about garget, foot rot in cattle, and all about iodine for everything. He even did his first necropsy at fourteen, on a cow, for meat for the eighty or so boys and staff.

The U.S. Army Paratroopers sent him to Germany but there was no shooting in 1945, thankfully, as both President Roosevelt died and the atom bomb went off while John was there. He was able to serve four Army years, thank goodness, which gave him and his wife forty-eight months of the fabulous GI education bill at the University of Delaware and Oklahoma Agricultural and Mechanical College in Stillwater, Oklahoma, with a DVM in 1955. His son, Jon David, went with them to Cornell University for five years and at Washington State University for two years, where John worked to finish his PhD from Cornell in 1963 and also became an ACVP member.

After six years at Mellon Institute in Pittsburgh, Pennsylvania, doing toxicology for Union Carbide Corporation, he returned to Cornell, Ithaca, New York, as a professor until he retired from the New York State Veterinary College. There he taught veterinary pathology, mostly in the Necropsy Room and at the microscope, with world travels for sabbaticals and other leaves of absence for periods of times totaling of over eight years, doing similar teaching at various facilities. Marie, John's wife, traveled with him on most of the trips.

In addition to *Fifty Years at the Necropsy Table, 1955–2005: True Stories of a Different Kind*, his other published works include: the two-volume set, *An Atlas of General Pathology*; over the course of his career of more than 50 years he has taken over 50,000 photo images of pathologic specimens, and 27,000 of these can be found on the "Dr. John M. King's Necropsy Show and Tell" website at <http://w3.vet.cornell.edu/nst/>; *Have Knife, Will Travel: Veterinary Pathology Sabbaticals and Leaves of Absence 1975–2005*; and *The Necropsy Book: A Guide for Veterinary Students, Residents, Clinicians, Pathologists, and Biological Researchers*.

Being the recipient of a 2008 Distinguished Alumnus Award from the Oklahoma State University College of Veterinary Medicine, of the Olafson Medal, and of the Pillar of Pathology Award have been highlights of Dr. King's career.