This is a section of liver. At lowest magnification, the lobules are "mottled" and some have dense red material in the central vein area.

At higher magnification, this red material is seen to be erythrocytes. Whether this is congestion (centrilobular) or hemorrhage is a difficult decision because blood is normally seen in sinusoids around hepatocytes. However, these areas have lost some hepatocytes and thus this is most likely hemorrhage due to necrosis. Did you spot the golden to brown pigmented deposits in the Kupffer cells? This is probably hemosiderin due to breakdown of erythrocytes in these areas.

A major change is seen in the hepatocytes. How would you describe them and what are your rule-outs? Many hepatocytes have variably sized, clear vacuoles in their cytoplasm. In some hepatocytes, the nuclei are displaced from their central location due to these vacuoles. The diagnosis is "hepatic vacuolar change" and the rule-outs would be glycogen deposition or fatty change. Special stains would be needed to confirm the vacuole content. Because the vacuoles are variably sized and occasional displace the cell nucleus, this is most likely fatty change.

It was confirmed clinically that this cat had diabetes mellitus. A probable mechanism for fatty change would include increased demand for body fat stores → increased mobilization of body fat stores → presentation of excess free fatty acids to the liver → inability to export all fatty acids → fatty liver.

This change is reversible. If the cause is corrected, the hepatocytes will return to normal.
This is a section of liver that stains "pink and poorly". The hepatocytes are often present as single cells rather than cords of cells. These changes (hepatocyte "individualization", uniform eosinophilic staining, and lack of basophilic staining) are compatible with AUTOLYSIS or POSTMORTEM CHANGE.

In addition to these changes, there are multiple, round foci which stain more intensely pink. These foci are randomly scattered throughout the liver and at higher magnification are seen to be NECROTIC.

Morphologic diagnosis: multifocal hepatic necrosis

These necrotic foci have cells with solid to granular pink cytoplasm and nuclei that are pyknotic, karyorrhectic or karyolytic. Some of these cells are inflammatory cells in addition to hepatocytes.

Do you see a cause? With diligent searching of the more normal hepatocytes at the margin
of the necrotic foci, you can find small pink bodies in the nuclei of some hepatocytes. These are the intranuclear inclusion bodies of infectious bovine rhinotracheitis (IBR) virus. This virus causes an upper respiratory tract disease in adult cattle and may cause abortion in pregnant animals.

Why is autolysis so prevalent in this liver? The calf died while still in the uterus and remained in the uterus for several days. The cow's body temperature promoted autolytic processes and thus postmortem change was advanced by the time the calf was expelled (aborted).
Find the section of liver. Look at the tissue against a white sheet of paper. You should be able to see areas that appear paler in color. With low power microscopic examination, there appears to be very little normal appearing hepatic parenchyma left. Portal areas can be detected based on the bile ducts found there. The rest of the liver contains hepatocytes that stain intensely pink. The nuclei of these hepatocytes are shrunken (pyknotic), fragmented (karyorrhectic) or missing (karyolytic).

Why do these hepatocytes stain more eosinophilic than usual? The eosin (acidic dye) has increased affinity for all the (basic) proteolytic enzymes that are breaking the cell down.

Morphologic diagnosis: massive centrlobular hepatic necrosis

What type of necrosis is this? This is coagulative necrosis - note you can still recognize the dead cells as having been hepatocytes.
What caused this necrosis? This puppy ate mushrooms probably *Amanita sp.* These mushrooms found all over the southern USA and can pop up suddenly especially after a rainstorm. They contain potent hepatotoxins (aminatoxins) that inhibit RNA polymerase B and hence protein synthesis. Usually within 24-48 hours after then the mushroom is ingested, sever liver damage and death results.
This is a section of lymph node, but it is difficult to ascertain that it is lymph node because the normal architecture has been all but obliterated by a central core of eosinophilic amorphous material, which is surrounded by a margin of fibrous connective tissues.

Can you recognize any cells in the debris? No – it is just caseous debris. Caseous is Greek for “cheesy” and it implies that the material is a homogeneous necrotic mass whose origin cannot be determined morphologically.

There are numerous macrophages at the interface of the fibrous connective tissue and necrotic debris. There are also occasional neutrophils. The outermost concentric ring is composed of fibroblasts and connective tissue. This process was due to infection with Corynebacterium pseudotuberculosis, the cause of “caseous lymphadenitis.”
This is a section of spinal cord. If you hold this section against a white background, you will notice there are areas of pallor (poor staining) in the ventral white matter and in the dorsal central segment of the cord. Look at the dorsal central region under the microscope. There is absence of neural tissue in this area. In fact this is a cystic area (microcavitation). Note there are macrophages (gitter cells) with foamy cytoplasm in this lesion. Gitter cells are actively phagocytosing neural material and thus have cytoplasm distended with foamy stuff.

Morphologic diagnosis: Myelomalacia

Malacia is the term for necrosis of nervous tissue. It is characterized as a “liquefactive” type of necrosis. The lipid material in the myelin is released and degraded. The gitter cells remove debris. A hole (microcavity) is left in the neural tissue.

This disease is hereditary myelopathy of the Afghan hound. It is inherited as an autosomal recessive disorder. Signs are first seen between 3 and 12 months, beginning with posterior ataxia and progressing to paraplegia and eventually phrenic paralysis and death. The exact mechanisms of disease are unknown.
This is a section of kidney. Most glomeruli contain eosinophilic hyaline material. Some tubules are dilated and contain eosinophilic hyaline material too.

The glomerular lesions are really spectacular. Many of the glomerular tufts are just chock full of a homogeneous eosinophilic material that is, in all probability, amyloid, making this a case of glomerular amyloidosis. This glomerular lesion has then led to the tubular lesion.
The tubular epithelium is "flattened" in some tubules. This is a response to injury, in this case, hypoxia. Some cells have been lost and the remaining cells "spread out" to cover the tubular lumen. The cells are lost due to ischemia and necrosis. Also, many of the tubules are filled with eosinophilic material – this is protein leaking through the damaged glomerular tufts.

Why would ischemia occur in the tubular epithelium? Remember the blood to the tubules must flow through the glomerular capillaries first. If the amyloid deposits hinder this flow and results in poor perfusion, tubular epithelial cells become ischemic.

What causes amyloid? Amyloidosis can be a primary disorder or it can be secondary due to chronic inflammation.
Find the pancreas on this slide. Within the pancreas there are focal areas of necrosis characterized by pyknotic nuclei and loss of acinar cells. Neutrophils are numerous in the pancreatic interstitium. There are marked numbers of neutrophils in the fat. Most of these neutrophils have pyknotic nuclei. Intermixed with the fat cells and neutrophils is “foamy” or vacuolated debris that has areas of both pink and blue staining.
The pink areas represent release of plasma proteins and perhaps some fibrin. The blue areas represent a mineralized focus – fatty acids released from the fat deposits combine with calcium to form a “soap” which stains blue.

Morphologic diagnosis: acute necrotizing pancreatitis with fat necrosis

The fat necrosis is secondary to the pancreatitis. Enzymes released from the necrotic pancreatic cells cause lysis of the fat cells and fat deposits. The fatty acids are then able to combine with calcium => fat necrosis and saponification.

There is another lesion in this slide. Note the large blood vessel near the area of fat necrosis? The vessel wall is thickened with eosinophilic fibrillar material. This is fibrin and the term is “fibrinoid necrosis.

8. 1649 5-year-old Irish Setter

The tissue on the slide is skeletal muscle. There are many normal appearing skeletal muscle fibers. However, some appear narrower and more lightly staining than normal.

Morphologic diagnosis: skeletal muscle atrophy
What happens to the cell in muscle atrophy? Remember, the cellular organelles become diminished in number due to a decreased workload.

This was a case of neurogenic atrophy. This dog had loss of innervation to his hind limbs and the muscle fibers atrophied. Note that there are also a few swollen, hypereosinophilic fibers in this slide too. Why do you think they are there?

Remember, the muscle fibers can only compensate for a certain time by atrophying. After a while, if the loss of innervation continues, the fibers will die and undergo coagulative necrosis.