

Case 1. Tissue from a cat.

**MICROSCOPIC DIAGNOSIS:** Lung: Scattered throughout the section are large areas of coagulative and lytic necrosis **(1pt.)** which are centered on airways **(1pt.)**, and affect approximately 33% of the section. Affected airways are filled with innumerable viable and degenerate neutrophils **(1pt.)**, necrotic epithelial cells, sloughed rafts of intact airway epithelium **(1pt.)**, admixed with abundant cellular debris and colonies **(1pt.)** of low to moderate numbers of 2x3 coccobacilli. **(1pt.)** Bacteria occasionally line the apical surface of columnar epithelium. This exudate often ruptures the wall of the airway and spills into the adjacent peribronchiolar fibrous connective tissue and adjacent alveoli. **(1pt.)** Surrounding affected airways, alveoli are expanded with a similar exudate with the addition of extensive polymerized fibrin **(1pt.)**, edema fluid **(1pt.)** and multifocal hemorrhage (most profoundly at the edges of the section), as well as alveolar macrophages and bacterial colonies. There is often septal necrosis **(1pt.)** and discontinuity (easily identified as areas of interruption of the markedly congested alveolar capillaries) as well as fibrin thrombi **(1pt.)** within alveolar capillaries and focal hemorrhage. There is a diffuse neutrophilia **(1pt.)** evident within intact alveolar capillaries as well as scattered megakaryocytes within septal capillaries. In areas of necrosis, low numbers of neutrophils infiltrate the tunica adventitia and media of medium-sized arterioles. **(1pt.)** There is edema of periarteriolar connective tissue and dilated lymphatics in these areas as well as within the pleura.

**MORPHOLOGIC DIAGNOSIS:** Lung: Bronchopneumonia **(1pt.)**, necrotizing **(1pt.)** and fibrinosuppurative **(1pt.)**, multifocal to coalescing, severe, with numerous bacterial colonies.

**CAUSE:** *Bordetella bronchiseptica* **(3pt.)**

**O/C:** **(1pt.)**

Case 2. Tissue from an ox.

**MICROSCOPIC DESCRIPTION:** Lymph node: Approximately 90% of the mildly hyperplastic node is effaced by several well-defined and occasionally coalescing granulomas **(2pt.)** which range up to 1cm in diameter **(1pt.)**. These granulomas are composed of a central, multifocally mineralized **(1pt.)** core of abundant amorphous eosinophilic cellular debris (and small amounts of basophilic nuclear debris) **(2pt.)** which contains few degenerate neutrophils. **(1pt.)** The core is surrounded by a thick layer of epithelioid macrophages **(2pt.)** enmeshed in loosely arranged collagen, which peripherally are interspersed with moderate numbers of lymphocytes, neutrophils **(1pt.)**, and plasma cells **(1pt.)** with scattered multinucleated giant cell macrophages of the Langhans **(1pt.)** and foreign body types (often in aggregates). The periphery of the granulomas are bounded by dense circumferential lamellations of collagen. **(1pt.)** Between granulomas, the normal follicular architecture of the node is effaced by a diffuse marked paracortical hyperplasia **(1pt.)** which markedly expands the remaining cortex and fills sinuses. Follicles are decreased in size and number and lack mantle zones. **(1pt.)**

**MORPHOLOGIC DIAGNOSIS:** Lymph node: Lymphadenitis, granulomatous **(2pt.)**, multifocal to coalescing, severe, moderate with diffuse moderate follicular and paracortical hyperplasia. **(1pt.)**

**CAUSE:** *Mycobacterium bovis* **(2pt.)**

**O/C:** **(1pt.)**

Case 3. Tissue from a dog.

**MICROSCOPIC DESCRIPTION:** Omentum: 80% of the omentum is replaced by dense bands of fibrous connective tissue **(1pt.)** and randomly pyogranulomas **(1pt.)** ranging up to 2mm in diameter. Foci of inflammation and are centered on colonies of large colonies of filamentous **(1pt.)** bacteria **(1pt.)** which range up to 1mm diameter. Bacterial colonies are surrounded by large numbers of viable and degenerate neutrophils **(1pt.)** and epithelioid macrophages **(1pt.)**, rare multinucleated giant cell macrophages admixed with cellular debris. More peripherally, these inflammatory cells are admixed with lymphocytes **(1pt.)** and plasma cells **(1pt.)** and are separated by lamellar bands of fibrous connective tissue **(1pt.)** ultimately coalesce to form dense bands of collagen which effaced normal tissue and subdivide remaining omental adipose tissue. **(1pt.)** Remaining adipocytes within the fibrous connective tissue bands between foci of inflammation variably-sized and are infiltrated by moderate numbers of macrophages, lymphocytes, and plasma cells (steatitis) **(1pt.)**. Proliferating fronds of fibrovascular tissue **(1pt.)** at the edge of the section exhibits marked congestion, mesothelial hyperplasia, and there are scattered aggregates of lymphoid tissues throughout. There are rare scattered bacterial colonies (granules) outside the tissue. **(1pt.)**

**MORPHOLOGIC DIAGNOSIS:** Omentum (mesentery ok): Steatitis **(1pt.)**, chronic-active **(1pt.)** and pyogranulomatous **(1pt.)**, multifocal to coalescing, severe, with large colonies of filamentous bacilli.

**CAUSE:** *Actinomyces sp.* **(3 pt)**

O/C: **(1pt)**

Case 4. Tissue from a dog.

MICROSCOPIC DESCRIPTION: Lung: Diffusely, the walls of small **(1pt.)** arterioles are diffusely and circumferentially expanded **(1pt.)** up to twice normal, often impinging upon the lumen **(1pt.)**. There is moderate, often assymmetric **(1pt.)** intimal hyperplasia **(1pt.)** as well as thickening of the media by smooth muscle hyperplasia **(1pt.)** (often disordered and lacking lamellar orientation) **(1pt.)** and increased amounts of medial fibrous connective tissue **(1pt.)** and extracellular matrix. Multifocally, there is extrusion of granular brightly eosinophilic protein within the wall of affected arterioles **(1pt.)** (fibrinoid necrosis) **(1pt.)**. Many of these vessels are accompanied by an asymmetrical proliferation of smaller branching thin-walled arterioles at their periphery **(1pt.)** (plexiform lesion), with slit-like lumens **(1pt.)** and sclerotic or necrotic changes to their walls as previously described. **(1pt.)** Some of these vessels contain fibrin thrombi. **(1pt.)** Within alveoli in proximity to these lesions, there is often aggregates of polymerized fibrin, **(1pt.)** hemorrhage, edema, and increased numbers of alveolar macrophages as well as patchy type II pneumocytes hyperplasia (exudative alveolitis). **(1pt.)** There is diffuse congestion of alveolar capillaries throughout the section and multifocal areas of alveolar emphysema.

MORPHOLOGIC DIAGNOSIS: Lung, small arterioles: Plexiform (plexogenic) arteritis **(1pt.)** with marked intimal and medial fibrosis **(1pt.)**, recanalization **(1pt.)**, fibrinoid necrosis **(1pt.)**, and exudative alveolitis.

O/C: **(1pt)**