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CHAPTER 4

The Peritoneum, Retroperitoneum, and Mesentery

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General Considerations

The peritoneal cavity is a single unit incompletely divided into compartments by the mesentery, omentum, and ligaments of double peritoneal membrane. This, with the assistance of fibrin in inflammatory reactions, can result in localization of lesions within the abdomen, although peritonitis can also be more or less generalized. Omental bursitis is an example of peritonitis localized within a compartment, and the peritonitis of traumatic reticulitis is frequently localized by fibrin and adhesions. Blood or fluid in the abdomen can occasionally be localized within a cystic structure but is usually generalized. The peritoneal membrane forms "ligaments" between organs surrounded by it, and in some cases these ligaments are involved in bowel entrapments, particularly the nephrosplenic ligament in the horse. Normal holes, such as the epiploic foramen, and congenital or acquired defects in the double layer of peritoneum making up the mesentery can also be involved in entrapments.

The retroperitoneum is the areolar and adipose tissue immediately outside the peritoneal lining of the abdominal cavity. Its volume is small except when adipose tissue accumulates, as it normally does in the dorsal retroperitoneum, pelvic cavity, and mesenteries.

The normal peritoneum is a smooth, shiny membrane with just enough fluid present to keep it moist. It becomes dry in animals that die of, or with, severe dehydration and is then clammy, sticky, and slightly opaque. It seems that a horse's peritoneum dries out shortly after death because if examination is postponed for a few hours, apposed serous surfaces stick to each other so that when separated the serosa is drawn into tags; this is especially evident on the diaphragm and viscera touching it. These changes should not be taken as residue of peritoneal inflammation or confused with the fibrous tags of scar tissue that occur on the diaphragmatic surface of livers that have been traversed by many larval nematodes. Distinction should also be made between antemortem and postmortem effusions and discolorations. Some lymph accumulates in the peritoneal cavity after death, and this becomes stained with hemoglobin as soon as erythrocytes in the serosal vessels begin to undergo lysis. Such fluid does not clot and is also present in other serous cavities. Its nature is usually evident because gaseous changes of putrefaction accompany the lysis of erythrocytes. Diffusion of bile pigments through the wall of the gallbladder, the biliary ducts, or the duodenum will stain adjacent viscera. Where hydrogen sulfide from the intestine meets hemoglobin from lysed erythrocytes, greenish black sulfmethemoglobin is produced to discolor the tissues.

Congenital Anomalies

Congenital anomalies of the peritoneal membranes are rare and when present are most frequently associated with embryonic remnants of vitelline structures. A **persistent vitelline duct** may form a fibrous ligament between the intestine and the umbilicus, or a Meckel's diverticulum and the umbilicus. The remnant may be partial, not reaching the umbilicus, and attached to the mesentery or a loop of intestine to form a band that could act as an internal hernial ring. Either form of band may become involved in strangulation of the intestine.

A **persistent vitelline artery** results in an anomaly, classified as a mesodiverticular band. This is a fold of mesentery, occasionally carrying a patent vitelline artery in its free edge, that extends from the cranial mesenteric artery to the antimesenteric side of the intestine (to the site of Meckel's diverticulum). The pocket formed between this fold and the normal mesentery may entrap intestine, or defects may develop in it that allow passage of intestinal loops and strangulation. Occasionally, double (both left and right) mesodiverticular bands are present. Occasionally, fibrous cords of mesenteric tissue may be found that do not appear to be part of embryonic remnants of vitelline structures.

Abnormal Contents of the Peritoneal Cavity

Foreign bodies occur commonly in the peritoneal cavity, and obviously, there is potentially a wide variety in both objects and circumstances. Foreign materials of endogenous origin are important because their presence requires an abnormality of another organ.

Ingesta is frequently found in the peritoneal cavity of horses, cattle, and swine, seldom in sheep and goats, and rarely in dogs and cats. The origin of the ingesta is the stomach or intestine, and the site of leakage is usually easy to find, especially when the animal dies before there is time for peritonitis to develop; such is the case, for instance, in gastric rupture in the horse. Once peritonitis has developed and matted the intestines and mesenteries, the site of perforation may be very difficult to find, especially as severe peritonitis devitalizes segments of the intestinal wall and, occasionally, causes perforation of the bowel from the serosal surface.

Digestion of the abomasal wall postmortem will release gastric contents in calves and lambs fed on milk-replacement diets, especially if the amounts ingested are excessive. The margins of the abomasal defect and the peritoneum do not show evidence of reactionary changes. In horses, cecal rupture following impaction usually occurs on the medial side of the dorsal cecum following ischemic pressure necrosis. Perforation of the colon or rectum following impaction probably has a similar pathogenesis. Rectal perforation may be secondary to accidental injury, or occasionally in foals, to focal ischemic necrosis from adherent meconium.

Hemoperitoneum is the presence of blood in the peritoneal cavity. The amount present at death is not an indication of the volume of bleeding during life because the blood is removed quite rapidly by diaphragmatic lymphatics. The blood in the cavity may be fluid or partially clotted. Animals occasionally die from bleeding into the peritoneal cavity, but the outcome will depend on the rate and volume of bleeding, the site of hemorrhage, the cause, and the initial health of the animal.

Hemoperitoneum is seen most commonly in the dog and cat as a result of traumatic injury to the liver, spleen, and kidney. Rupture of the liver and hemorrhage occur in infectious canine hepatitis. Manual efforts at artificial resuscitation, especially if vigorous, will rupture the liver, particularly in anesthetized dogs, if the anesthetic is one causing hepatic congestion and turgidity. Repeated splenic ruptures at the site of hemangiomas are well known in dogs past middle age. Spleen and liver enlarged and tensed by infiltrating leukemic cells, fat, or amyloid are predisposed to rupture; the volume of hemorrhage in these cases may be very small.

Warfarin poisoning in several species may result in nonclotting blood in the abdomen. Manual squashing of a corpus luteum is a source of hemorrhage in cattle. In cattle and horses, laceration of the uterus or rupture of a uterine artery can result in a sometimes fatal hemorrhage. Calves born of cows fed moldy sweet-clover hay bleed from the umbilical vessels into the peritoneal cavity (as well as elsewhere).

Hemorrhage on or beneath the peritoneum without free blood in the cavity is commonly observed in many acute infectious toxemias and noninfectious conditions that interfere with vascular integrity or the hemostatic mechanisms. Peritoneal hemorrhage must be differentiated from hemorrhagic peritonitis, which is an important lesion in many diseases. Hemorrhage into an omental or mesenteric cyst can cause sudden abdominal enlargement without free blood in the abdomen.

Hydroperitoneum

Hydroperitoneum (ascites), is the accumulation of noninflammatory transudate in the peritoneal cavity. It is arguable that the term ascites should be reserved for peritoneal lymphedema of hepatic origin, whether the issue is primarily hepatic, as in hepatic fibrosis, or secondary to a sustained increase in central venous pressure with chronic hepatic congestion. The fluid of ascites is watery, clear, or straw colored and will contain few leukocytes but large numbers of desquamated mesothelial cells. The serosal lining is normal unless fluid has been present for some weeks, when the serosa acquires a flat, whitish semiopacity.

The principles governing the transfer of fluid (lymph) between blood vessels and lymphatics across the peritoneal membrane are, so far as known, basically the same as those governing fluid exchange in tissues, and there is normally a very rapid turnover of peritoneal fluid. In tissues generally, there is a small excess of filtration at the arteriolar end of the capillary bed over absorption at the venous end of the capillary bed; the excess of fluid is drained from the tissue spaces by lymphatics. These mechanisms are operative in abdominal viscera, but it is to be noted that free fluid in the peritoneal cavity drains through the ventral diaphragm and the sternal lymphatics. The accumulation of excess peritoneal fluid can be viewed most simply as the result of diminished removal or overproduction of fluid.

The relative importance of the various pathways for the **removal** of **fluid** from the peritoneal cavity has not been studied in detail for the various species, but the following schema is generally applicable. In spite of the large area of peritoneum, fluid absorption from the cavity is virtually limited to those diaphragmatic lymphatics that form an abundantly anastomotic plexus in the muscular portion of the right side of the diaphragm. Vessels penetrate the diaphragm to form a corresponding pleural plexus from which the lymph is conveyed in the sternal ducts to the anterior sternal node, and then via the right lymphatic duct to the vena cava. There are subsidiary pathways that will deliver some peritoneal lymph to the cisterna chyli or thoracic duct, but 80% or more of peritoneal lymph will follow sternal lymphatics. The smallness of the area of lymphatic absorption provides an explanation for the ease and rapidity with which peritoneal drainage can be obstructed.

Obstruction to **diaphragmatic lymphatics** leading to ascites is best exemplified in peritoneal carcinomatosis. Implantation metastasis of carcinomas usually and perhaps always, if given time, implant most extensively on the diaphragm in the region of the lymphatic exits, and there, permeation of the lymphatic vessels is easily demonstrated microscopically. It is probable that the neoplastic cells are carried to the anterior abdomen by the same forces that propel the lymph in that direction, namely, normal intestinal movements and normal respiratory excursions. The restriction of the area through which lymphatic absorption can take place is compensated by the high efficiency of the mechanism, a level of efficiency not satisfactorily explained.

Carcinomatous implants on the peritoneum, in addition to obstructing diaphragmatic lymphatics, may provoke fluid production. This has been argued for papillary adenocarcinoma of the ovary, which in the bitch provides the best example of implantation and ascites. It will be apparent that ascites may develop if there is obstruction to sternal pathways anterior to the diaphragm, and this condition seems often to be provided in lymphomatosis of adult cattle when there is massive neoplastic involvement of the anterior mediastinal lymph nodes.

The **overproduction** of **peritoneal lymph** is related to the mesenteric circulation and the hepatic circulation. This excludes the rare chylous ascites, which results when the cisterna chyli is ruptured, the chylous origin of the fluid indicated by the high content of chylomicron fat and the milkiness of the fluid.

The filtration pressures in mesenteries and omentum appear to differ quantitatively from those in other tissues, with the effective filtration occurring along the entire capillary bed. That all of the filtered fluid must return to the circulation via the lymphatics attests to the capacity for lymphatic drainage. Increased venous pressure in the portal venous system is not in itself expected to lead to ascites. Acute portal vein obstruction leads rapidly to death. Chronic obstruction to prehepatic portal flow leads quickly to the development of collateral venous circulation, with connections to the abdominal vena cava. The interstitial tissues of the mesentery and omentum are, as are interstitial tissues generally, rather noncompliant, and small increases in interstitial fluid volume produce marked increases of tissue hydrostatic pressure, which limit transudation.

The balance of oncotic and hydraulic pressure that regulates fluid exchange between blood vessels and interstitial tissue depends on the integrity of vascular structures and on the maintenance of their permeability characteristics. Vascular injury that allows increased permeability to plasma protein substantially alters the balance of forces and favors transudation. Small amounts of ascitic fluid may be generated under these circumstances in a variety of systemic illnesses, such as the clostridial intoxications, acute uremic syndromes in ruminants and pigs, and exudative diathesis of pigs deficient in vitamin E.

Ascites resulting from overproduction of fluid is usually an expression of hepatic lymphedema. It appears that the *sine qua non* of hepatic ascites is that there should be obstruction to the intrahepatic veins or the suprahepatic veins with congestion and edema of the liver; obstruction to the portal veins with increased portal venous pressure will not do, except perhaps if there is concurrently a critical degree of hypoproteinemia. The usual conditions providing obstruction to the efferent hepatic vessels are fibrosis of the liver and congestive heart failure. There is, of course, a variety of additional causes, including primary neo-

plasms of the liver, principally those of cholangiocellular type, which tend to be diffuse; secondary tumors, especially those of lymphocytic type; extensive infestation with hydatid cysts; chronic fascioliasis; and so on.

When the liver is congested, there is, as in any acutely congested tissue, an increased turnover of protein and fluid and an increased flow of hepatic lymph with a high concentration of protein. The bulk of hepatic lymph comes from the space of Disse, which is separated from the capillary lumen by a fenestrated endothelium that is freely permeable to plasma constituents, including protein of high molecular weight. Oncotic pressures, normally dependent on proteins of low molecular weight, are not as important in the liver as they are elsewhere in the regulation of fluid exchange. Instead, the formation of hepatic lymph is sensitive to small changes in hydraulic pressure in the sinusoids, which accounts for the frequency with which ascites is associated with those diseases leading to increased central and hepatic venous pressure.

If the ordinary capacity of the hepatic lymphatics is not equal to the challenge provided in hepatic venous stasis, then lymph high in protein oozes from the hepatic capsule, presumably from the rich lymphatic plexus there, and spills from the efferent lymphatics that pass from the porta hepatis to the cisterna chyli; in hepatic ascites, these efferent lymphatics become very large, numerous, and thick walled, and presumably, the same changes take place in all efferent hepatic lymphatics, some of which follow different routes, for example, through the suspensory ligaments.

The fluid that accumulates in the peritoneal cavity is not static but dynamic, continually produced and removed. The efficiency of normal removal has already been indicated, and it will be evident therefrom that it is only in instances of severe increased pressure in the hepatic vessels that gross ascites will develop. In all considerations of hepatic ascites, the thoracic duct, which is the main channel for removal of hepatic lymph, is assumed to be completely permissive. The conducting capacity of the duct is, however, probably quite limited, even though the duct is quite distensible. Concurrent increase of venous pressure at the thoracic inlet may impair lymph drainage; of greater importance may be altered configuration, when the duct is distended, of the opening between duct and vein.

In experimental ascites, the flow of lymph in the hepatic lymphatics can be increased in rate to five or more times the normal, and since only a part of this flow oozes into the peritoneal cavity, some additional factors would seem to be necessary before the drainage capacity is overwhelmed, if indeed it is overwhelmed, an event that seems unlikely in view of calculations purporting to show a complete turnover of the albumin in the ascitic fluid in ~2 days and a turnover of ~80% of the volume of ascitic fluid each hour. Thus it seems clear that although severe obstruction to the intrahepatic circulation is necessary to initiate hepatic ascites, the gross and persistent accumulation of fluid depends on additional factors. Chief among these is the retention, by diminished urinary excretion, of salt and therefore also of water.

There is much evidence connecting the retention of sodium and water, and the resultant expansion of plasma volume, with the development of edema and ascites in congestive heart failure and hepatic venous obstruction. The mechanisms remain obscure but may involve receptor sites for sensing volume–pressure changes in the systemic circulation. The effector mechanisms are renal and probably regulated by a variety of neural and humoral pathways that influence renal hemodynamics, the composition of tubular fluid, and ionic gradients.

The principal diseases of animals in which ascites occurs have been indicated in the foregoing discussion, but there are some others that ought to be mentioned. The ascites of congestive heart failure is presumably of the same pathogenesis as in humans, whatever that might be, and as indicated above, it may be related in part to the hepatic congestion that is part of the syndrome of cardiac failure. There is the additional possibility of retarded lymphatic flow, and although there appear to be no measurements of this, the tortuous dilatation of lymphatics in some cases of congestive heart failure are quite suggestive. Ascites occurs in congestive heart failure without edematous transudations elsewhere, especially in dogs and cats, but it should be pointed out that only a minority of cases of congestive failure in dogs and cats is associated with ascites. Excess peritoneal fluid is also part of a generalized dropsical condition in cachectic diseases, anemia, and starvation. Although the mechanisms have not been intensively studied, hypoproteinemia has been given consideration as one of the principal factors. Very probably it is also in those diseases that are known, or presumed, to result in chronic protein leakage and loss, such as gastrointestinal trichostrongylosis and Johne's disease. Hypoproteinemia appears not, however, to be of initiating significance in the edema and ascites of chronic starvation without other accompanying disease; it is suggested in such cases that the fluid-retention serves largely to replace wasted tissues, especially adipose tissue.

Effusion of fluid, sometimes massive, occurs in the peritoneal cavity, thorax, and ventral body wall in some instances of uremia in sheep and cattle. It is part of the postmortem picture in sheep and cattle dying of urethral obstruction by calculus, and the fluid may have a distinct uriniferous odor. The pathogenesis of this fluid accumulation has not been examined, and although in a few cases there is a rupture of the lower urinary tract (which might provide the fluid by leakage), in many cases a rupture cannot be demonstrated. Renal uremia also is associated with similar fluid accumulations in cattle; this is evident in some dying with renal amyloidosis, and the contributing factor here is probably hypoproteinemia consequent on prolonged massive proteinuria. Acute toxic nephrosis, of which certain plant poisonings and mycotoxicoses provide the best examples in ruminants and pigs, may also be accompanied by massive effusions, and as is usually the case with ascites of urinary tract disease, the mesenteries and retroperitoneum are also saturated.

Degenerative Conditions of the Peritoneum, Retroperitoneum, and Mesentery

Abdominal Fat Necrosis

Necrosis of the omental or other abdominal or retroperitoneal fat is a frequent finding at autopsy. The pathogenesis is poorly understood, but there appear to be a number of causes. In pancreatic necrosis, **enzymatic necrosis** of **fat** occurs constantly, and indeed, peripancreatic necrosis of fat may be the initial

morphologic change. Enzymatic necrosis of fat may extend throughout the abdomen. The acute lesions are discrete foci or confluent masses of yellow, necrotic adipose tissue surrounded by a zone of intense hyperemia with fibrin deposited on the surface. Free droplets of fat can be found in the peritoneal fluid.

The lesion has been attributed to lipolytic enzymes from the acinar pancreas, but there is no agreement on which enzymes initiate the reaction. Pancreatic amylase, elastase, lipase, trypsin, and phospholipase A are examples of enzymes that may be involved, but some of these would have to overcome local enzyme inhibitors before becoming activated. Kinins, Hageman factor, and complement have been suggested as possible causes of fat necrosis once the process has been initiated. Neutrophils respond in large numbers, and their degeneration may aid the ongoing process. Lipase released from degenerating fat cells also contributes to fat necrosis.

Microscopically, the acute lesion is made up of necrotic fat cells containing acidophilic, opaque, amorphous or lacy substance or basophilic fibrillar or granular mineralized material. Masses of degenerating neutrophils and necrotic debris are present. Fibroplasia and vacuolated macrophages are features of the chronic lesion, along with necrotic fat and, occasionally, dystrophic mineralization.

Widespread or isolated **focal necrosis** of abdominal and retroperitoneal fat is frequently found in sheep and sometimes in horses, pigs, and other animals. This necrotic fat is usually seen only at the chronic stage in the form of small, dry, firm or gritty plaques. A flat white color gives the plaques clear distinction from surrounding normal fat. There is no grossly apparent inflammatory reaction, and histologically the lesions resemble the chronic lesion described above, with occasional large lipid vacuoles containing necrotic debris encapsulated by connective tissue. The pathogenesis of this form of fat necrosis has not been explained. The focal lesion may be due to avascular necrosis of fat from pressure ischemia, perhaps due to differences in the texture or composition of the fat, or some other circulatory deficit in the small capillaries that nourish the large masses of fat, since the lesion does appear more frequently in excessively fat animals. It is also possible that the lesions are initiated by intracellular lipolytic disturbances in circumstances in which there is accelerated mobilization of fat to meet acute metabolic demands.

The third form, not uncommon and perhaps the most curious, is **massive fat necrosis** in **cattle**. Although this condition has been reported to occur more frequently in Channel Island breeds, there is more evidence for herd than breed predisposition. It does appear to be associated with excessively fat and fattening cattle. Both sexes are affected, but probably not clinically before the second year of life. The disease has two significant features. First, although it is seen more often in the slaughterhouse than in the postmortem room, it is frequently fatal, perhaps always progressive and potentially fatal, and second, the hard masses of necrotic fat have been, and will be, mistaken for fetal prominences in the diagnosis of pregnancy by perrectal palpation. The fatal outcome in this disease is usually by intestinal obstruction, the intestine compressed by the expanding lumps of necrotic fat (Fig. 4.1B), but other complications also occur, such as compression and stenosis of the ureters. Affected animals may exhibit a variety of clinical signs such as anorexia, diarrhea, constipation, colic, or bloat. They may become emaciated prior to death.

The pathologic process occurs in any portion or all of the omental, mesenteric, and retroperitoneal fat. In the early stage of development there is evidence of acute inflammation, and the hard necrotic masses are surrounded by a zone of hyperemia, the overlying peritoneum may be necrotic, and the necrotic margins umbilicate. The masses may vary from small nodules to large solid masses encapsulated by fibrous tissue and that on cut section are firm, dry and caseous, or moist, and deep yellow. The inner surfaces of such masses are firmly molded to the contours of enclosed organs. Because of the unusual bulk of the necrotic tissue, and because fat is sometimes found in unusual locations, such as under the serosa of the intestine, the condition has been called lipomatosis, but there is no indication that the lesions are neoplastic.

On microscopic examination the tissue resembles a mixture of acute and chronic enzymatic fat necrosis with fewer neutrophils, some lymphocytes, plasma cells, and eosinophils, and more macrophages and giant cells. Crystal-shaped clefts are present in fat cells and in macrophages and giant cells.

The pathogenisis of this diffuse lipogranulomatosis in cattle is not clear, but there is accumulating evidence of a dietary cause somewhat similar to that of steatitis as it occurs in horses, pigs, and other species. The different lesions in cattle may be due to the different chemical composition of fat or to a different host response to material recognized as "foreign" (which might also allow for breed predisposition, as might breed differences in texture and amount of abdominal fat). The lesion is possibly related to ingestion, or production in the rumen, of high levels of saturated fatty acids, which form long-chain compounds that are solid at normal body temperature. Chemical change or avascular necrosis may initiate a reaction that results in the formation of insoluble salts, cholesterol, or other material recognized by the body as foreign. This material provokes the inflammatory response, with fibrosis, collagen formation, and the accumulation of materials in fat cells, giant cells, and macrophages.

Steatitis (yellow-fat disease) occurs in many species of animals except ruminants, affecting the abdominal and retroperitoneal fat along with other body fat. The condition is described (as panniculitis) with the Skin and Appendages (Volume 1) and is caused by a diet high in polyunsaturated fat and low in tocopherols, allowing oxidation of body fatty acids. Peroxidation creates free radicals, which damage tissue and provoke the characteristic inflammatory response in the adipose tissue. Several types of steatitis, all vitamin E responsive, occur in newborn or young, as well as in mature animals. Lipofuscin, which is not present in all forms of steatitis, is responsible for the yellow color and often fills the macrophages, which are a feature of this form of steatitis.

Inflammation of the Peritoneum: Peritonitis

Peritonitis is very common in the large domestic animals but uncommon in dogs and cats. It may be serofibrinous, fibrinopurulent, purulent, or hemorrhagic, and whatever the type, it may be localized or more or less generalized. Differences in type and distribution can be more or less anticipated from the origin and the causes; the latter are numerous and

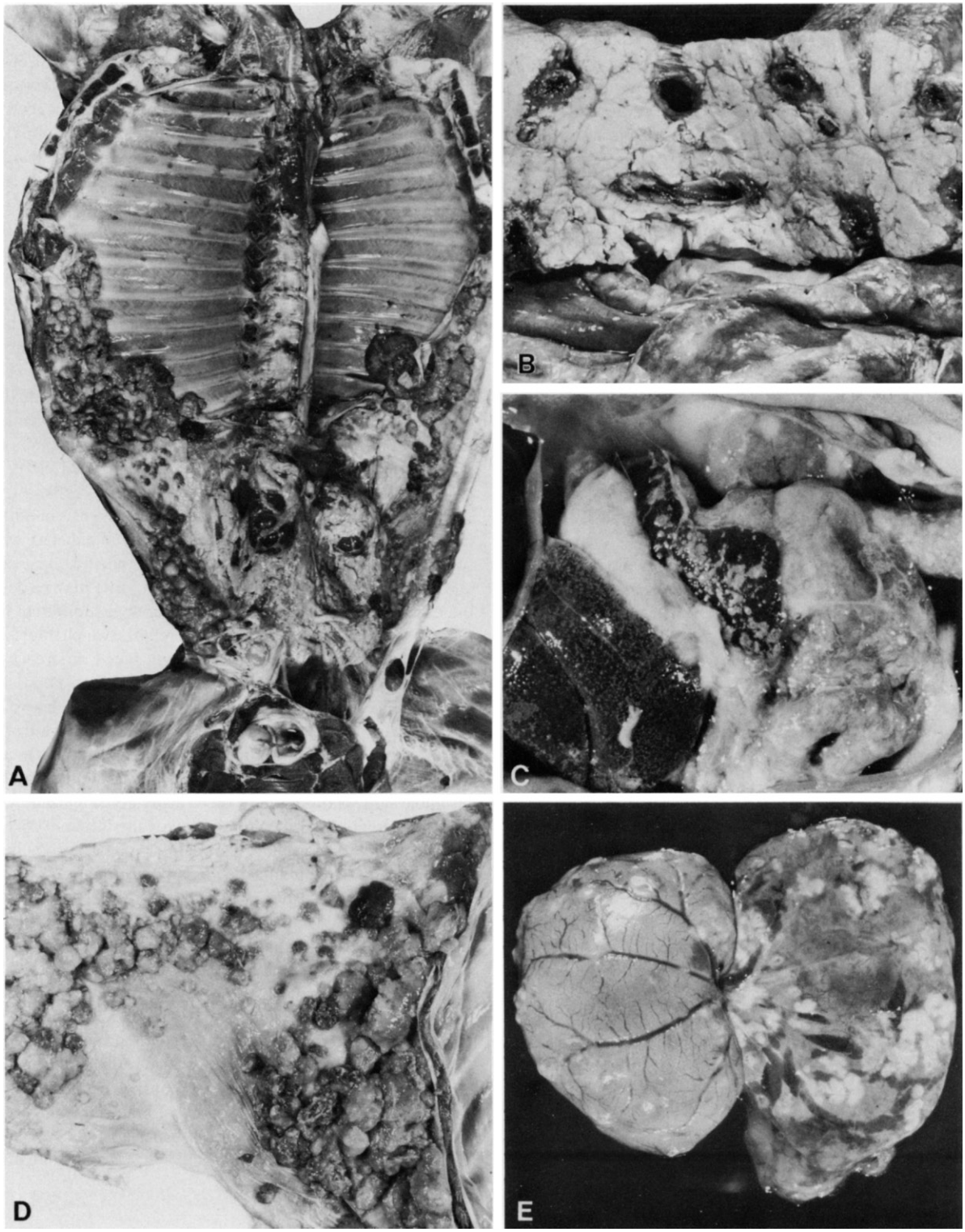

Fig. 4.1. (**A**) Congenital mesothelioma. Calf. Tumor nodules are confined to peritoneal cavity. (**B**) Mass of fat enclosing intestinal loops in abdominal fat necrosis. Ox. (**C**) Feline infectious peritonitis. Exudative lesion with fibrin on mesentery and viscera, and granulomas in liver, spleen, kidney, and wall of large and small intestine. (**D**) Close-up of (**A**), showing nodules of mesothelioma on peritoneum. (**E**) Feline infectious peritonitis. Focal pyogranulomatous lesions involving renal cortex and reflected capsule.

varied, so only some of the more common and important of them will be considered.

Most cases of peritonitis are caused by bacteria and their toxins, some by helminth infestations, a few are viral, and a few chemical. The intraperitoneal injection of a number of therapeutic agents causes a mild, and usually inconsequential, peritonitis. The most devastating forms of chemical peritonitis are endogenous and are caused by bile and pancreatic enzymes. The peritonitis caused by bile is intense, shock inducing, and may be rapidly fatal. There is very little exudate unless the leakage is minor and infected, and then it may become purulent. Biliary peritonitis is readily recognized by the typical staining. The peritonitis of pancreatic necrosis is also acute and is rather common in dogs, rare in horses, and virtually never occurs in other species. The reaction about the pancreas, particularly its head, is liquefactive and purulent, and the exudate mats the lesser omentum to the pancreas and the adjacent liver and other organs. This local peritoneal reaction resolves completely if the animal survives, and only very minor adhesions or slight puckering of the mesentery persist. The peritoneal exudate is usually scant but is distinctive because, as well as pus, it contains white droplets of fats and soaps released from the surrounding adipose tissues by the pancreatic enzymes.

Bacterial peritonitis may be regarded as primary in the sense that the bacteria are implanted directly on the peritoneum by perforating lesions from a contaminated surface, either a hollow viscus or the skin. It is secondary when it reaches the peritoneum from an adjacent focus by direct extension, or when the organisms are carried there in the blood or lymph streams.

Primary peritonitis need not be discussed further; it is most commonly associated with the lesions described above as being the source of peritoneal contamination by ingesta but may also rise from parasites penetrating the intestinal wall in some species. In females, the abdominal cavity is open to the exterior through the reproductive tract, and infection may enter the abdomen through the oviduct as well as by rupture of the uterus or laceration of the vagina.

Secondary bacterial peritonitis is also common either as an extension from localized inflammation in an abdominal viscus or as a typical part of the morbid picture in a number of specific diseases. Acute serofibrinous peritonitis occurs by extension through the wall of a gangrenous intestine or uterus prior to rupture, or death from toxemia may intervene before rupture occurs. Secondary peritonitis occasionally results by extension from retroperitoneal infection or, in ruminants, from omental bursitis. In those cases in which the peritonitis develops by direct extension, there is little difficulty in ascertaining its origin, even when the process becomes diffuse. The sources of secondary peritonitis are so varied that detailed consideration cannot be given to them here. Instead, some of the features of peritonitis in the different species are given with an indication of the specific diseases in which peritonitis is a feature, although not necessarily a consistent one. The incidence and nature of peritonitis in specific infections are discussed more fully with the specific diseases.

Horses

Diffuse peritonitis is usually fatal in horses, chronic diffuse peritonitis virtually never occurring. In almost all cases it is caused by rupture or perforation of the stomach or intestine. Acute or chronic local peritonitis does occur occasionally from castration wounds or other penetration from the skin, or streptococcal abscess in the mesentery. It may be due to local verminous lesions, suppurative gastritis in habronemiasis, or from perforating *Gasterophilus*. Abscesses or ulcerations attributable to *Strongylus* are more common in the colon than elsewhere. Migrating *S. equinus* or *S. edentatus* larvae cause retroperitoneal lesions in the flank, perirenal fat, and diaphragm, perihepatitis with fibrin tags on the liver capsule, and a chronic diffuse thickening and inflammation in the mesentery, omentum, and hepatorenal ligament, with occasional caseous nodules. Copious purulent peritonitis is often seen in infections of foals by *Corynebacterium equi*. Intestinal infection of foals by *Actinobacillus equuli* causes fibrinous mesenteric lymphadenitis and peritonitis.

Cattle

Acute diffuse purulent peritonitis in cattle is common and usually the result of perforation of a viscus, especially the reticulum or uterus. Both may also result in local acute, and then chronic, peritonitis. Perforation of the abomasum or intestine is more likely to give fibrinohemorrhagic peritonitis. A serofibrinous peritonitis, sometimes with very copious exudate (and with similar lesions on other serous membranes), is typical of sporadic bovine encephalomyelitis. A diffuse fibrinohemorrhagic peritonitis occurs in most cases of clostridial hemoglobinuria and in some cases of blackleg and septicemic pasteurellosis; a more localized peritonitis of this type occurs in some cases of clostridial enterotoxemia caused by *Clostridium perfringens* types B and C. Tuberculosis causes white, nodular granulomas (pearls), and actinobacillosis, although rather rare, produces the usual heavily scarified granulomas, especially about the peritoneum of the forestomachs. Extension of infection from the umbilicus of the newborn produces a fibrinopurulent peritonitis not localized but most severe along the ventral abdominal wall, or up the urachus to the bladder.

Sheep

Peritonitis of specific cause is uncommon in sheep except for the very local variety that accompanies penetration of the intestine by the larvae of *Oesophagostomum columbianum*. The uterus is probably the usual site in adults from which local spread occurs to the peritoneum, the antecedent lesion in most cases either a postpartum septic metritis or so-called blackleg of the fetus; in either event the peritonitis is fibrinosuppurative and hemorrhagic. A serofibrinous peritonitis is a feature of contagious agalactia, or any variant of it, caused by *Mycoplasma*.

Goats

Mycoplasma mycoides may cause an acute fibrinous peritonitis in goats, although acute death from septicemia or joint and mammary gland disease is more common. Paratuberculosis (Johne's disease) frequently produces a nodular granulomatous lymphangitis in the mesentery, and sometimes caseous or calcified lymphadenitis.

Swine

A few filmy strands of fibrin frequently overlie the intestine and the borders of the mesentery in many acute infectious diseases of swine, and in conditions that result in vascular damage such as edema disease and vitamin E/selenium-responsive conditions; this does not qualify as peritonitis. A diffuse suppurative and adhesive peritonitis is common in pigs; in these cases the intestines are so matted that they cannot be dissected. *Corynebacterium pyogenes, Escherichia coli,* or a miscellany of organisms are frequently present in these, and in some the extension of the inflammatory process can be traced up the inguinal canals from castration wounds; in other cases of similar type, the peritonitis is localized to the inguinal and pelvic regions, is adhesive, and causes death from intestinal obstruction. Occasionally, *C. pyogenes* produces discrete encapsulated abscesses as profuse implants on both visceral and parietal peritoneum. A serofibrinous peritonitis, more fibrinous than serous, and with similar lesions on other serous membranes, is almost pathognomonic for Glasser's disease caused by *Haemophilus suis. Mycoplasma hyorhinis* and possibly other mycoplasmas may produce a serofibrinous peritonitis that becomes fibrous, with adhesions to a thickened serous membrane; this disease is to be distinguished from Glasser's disease. Small, firm, lemon yellow nodules and flattened disks of inspissated fibrin often are found free in the peritoneal cavity in chronic *Mycoplasma* infections. Rectal strictures in swine cause a very dilated colon and cecum full of ingesta. The serosa is frequently thickened, white, and covered with fibrin tags, resembling infectious serositis. The thickening is probably caused by subserosal edema and fibrosis but may be due to passage of bacteria or by-products through the intestinal wall. *Stephanurus dentatus* larvae cause subserosal focal hepatitis and a mild reaction with edema in the perirenal fat and retroperitoneal tissue, and sometimes in the mesentery and local lymph nodes as they move to the kidney. In intestinal anthrax in swine there is an acute gelatinous hemorrhagic peritonitis very typically localized to the mesentery between the intestine and the mesenteric node. The distribution of the lesion is due to the lymphatic spread of the infection from the intestine. Tuberculous peritonitis in swine is localized and characterized by adhesions to the spleen.

Dogs

A fibrinohemorrhagic peritonitis, slight in degree and easily overlooked, is common in infectious canine hepatitis. The peritoneum, especially that covering the intestine, is gray and granular like ground glass, and there are a few reddish strands of fibrin, most of them about the liver. There is edema of the intestinal subserosa and, frequently, petechiae or larger hemorrhages. In parvovirus infection, similar lesions may be present on the serosa of the affected portion of intestine. Putrid peritonitis occurs when the uterus ruptures either as a result of pyometra or septic metritis with fetal putrefaction. Nocardiosis, which may be infection by bacteria of the genera *Nocardia* or *Actinomyces,* produces very characteristic lesions on the peritoneum (Fig. 4.2B). It is, however, more common on the pleura. Sometimes there are purulent granulomas, but most frequently there is a profuse, pink mush. The color is from admixture of copiously exuded cells and blood; the blood is derived from a tremendous proliferation of thin-walled capillaries from serous surfaces. Purulent peritonitis in the dog is rare; it has been observed as an extension from umbilical and hepatic abscesses in puppies and is caused by *Streptococcus canis.*

Cats

Putrid peritonitis occurs when the uterus ruptures in consequence of pyometra or fetal putrefaction. Peritonitis also occurs from penetrating wounds or by extension from retroperitoneal tissues. Nocardiosis similar in appearance to the disease in dogs frequently complicates myelodysplastic disease in cats.

FELINE INFECTIOUS PERITONITIS. Feline infectious peritonitis is a relatively recently recognized coronavirus infection of Felidae, particularly domestic cats. It has a worldwide distribution, but the incidence of disease is low and sporadic even in the countries where it is most common. The disease is in part immune mediated, and the lesions are for the most part the result of deposition of immune complexes leading to Arthus-type reactions. The naturally occurring disease tends to be chronic and results in death after one to several months, although there may be nonclinical carriers. Most cases have a relatively easily recognized clinical course with typical gross and histologic findings. Close contact appears to be necessary for the spread of the disease and is likely to occur by way of ingestion or inhalation of infective material from body secretions and excretions. Cats in catteries, pet shops, and households with multiple cats are at greatest risk. Cats of all ages can be affected, but there may be a higher incidence in two groups; young, entire adults and aged cats.

Feline infectious peritonitis has an insidious onset, and since cats have a propensity to mask serious illness, the disease may be well advanced before clinical signs are recognized. The early signs are nonspecific, consisting of decreased activity, depression, anorexia, or diarrhea. The increasing abdominal distension present in the effusive form of the disease may hide the progressive weight loss. A variety of mild to severe neurologic signs develop in up to 30% of affected cats, and it is these signs, and panophthalmitis (which is also relatively common), that are likely to attract attention. Affected cats are usually pyrexic and often hypergammaglobulinemic, with total serum protein increased and the albumin/globulin ratio lowered. Feline infectious peritonitis is unresponsive to antibiotics but may show a temporary response to corticosteroids. Occasionally, respiratory signs with cough or dyspnea are present, and in atypical or noneffusive forms, neurologic or ophthalmic signs may be the first noted. A variety of serologic tests are available to detect feline infectious peritonitis antibody. Antibody levels are usually high (titer $>$ 1:400) in cats with clinical infectious peritonitis, but interpretation is complicated because current tests do not distinguish between antibodies formed in response to the virus and those stimulated by the feline enteric coronavirus.

For descriptive purposes, the disease has been divided into effusive "wet" and noneffusive "dry" forms, but since the only distinction between these forms is the extent of fibrinous peritonitis and exudate, the division serves little useful purpose. All serous surfaces throughout the body may be involved in the inflammatory response, and pleurisy with pleural effusion is

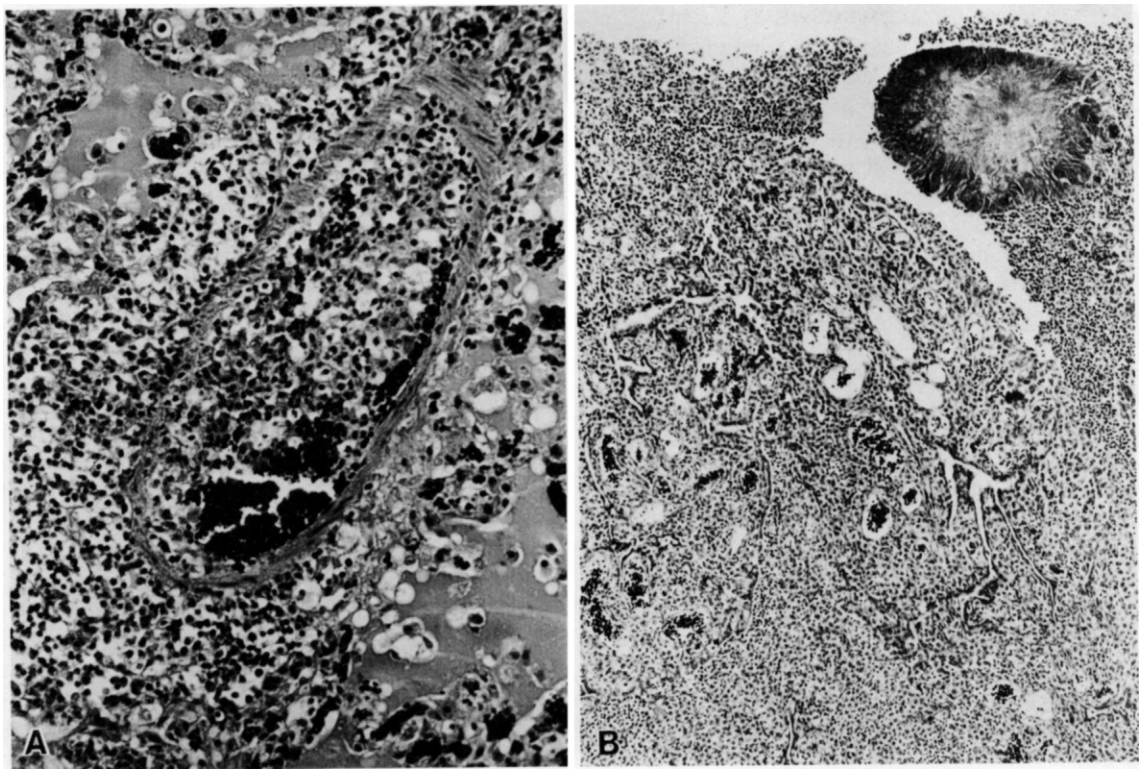

Fig. 4.2. (**A**) Feline infectious peritonitis. Vasculitis and perivasculitis in the lung. There is severe pulmonary edema. (**B**) Proliferative peritonitis in nocardial infection. Dog. Bacterial colony (top right) is attracting a stream of leukocytes.

present in ~40% of cases. Peritonitis is present in most natural cases, although this may be obvious grossly in only 60 to 70%. The tunica vaginalis is affected, resulting in periorchitis in entire males. Up to 1 liter of abdominal exudate may be present.

The fluid is usually clear and pale to deep yellow, although it may be flocculent and contain strands of fibrin. The serosal surfaces may be covered with fibrin exudate, giving them a granular appearance. Fibrin is frequently prominent over the visceral peritoneum (Fig. 4.1C), and fragile adhesions may be present. There are white foci of necrosis or raised granulomatous cellular infiltrations on the serosa and extending into the organs or wall of the intestine from the serosa. These vary in size from a few millimeters to a centimeter in diameter. The kidneys may be enlarged and nodular with single or multiple, small to large, white, granulomatous nodules protruding from the cortex (Fig. 4.1E). Severe or mild granulomatous hepatitis and pancreatitis may also be present. Small white foci of inflammation may be found in the organs. Fibrin is usually less prominent in the thorax, but white foci may be visible under the pleura, and the lungs may be dark, firm, and rubbery. Hydropericardium and fibrinous epicarditis occur less frequently but are similar in type to other serosal reactions. Abdominal and thoracic lymph nodes are enlarged and have a lobulated pattern. In the effusive form of the disease, visceral lesions are sometimes minimal, and in the noneffusive form, peritonitis may be mild or not obvious on gross examination, and in these cats the lesions can be in the abdominal or thoracic organs, as described above, or in the eyes or nervous system. The lesions in the eyes begin as a diffuse uveitis progressing to a panophthalmitis, with fibrin usually seen in the anterior chamber. Lesions in the central nervous system can involve the leptomeninges, spinal cord, or brain but usually are visible grossly only on the leptomeninges as thickenings or white streaks.

A subacute form of feline infectious peritonitis with a shorter clinical course is seen occasionally, primarily in weaned kittens. It occurs as a generalized systemic infection, and the lesions are more fibrinonecrotic than granulomatous. An acute hepatic form of the disease resembling suckling mouse hepatitis (coronavirus infection of mice) has been described in experimentally infected kittens.

The basic histopathological lesion of the disease is a generalized vasculitis and perivasculitis (Fig. 4.2A) and focal pyogranulomatous reaction that occurs in the serous membranes, the meninges, and in the connective tissue of the parenchymatous organs. This results in the serofibrinous and cellular exudate on the visceral and parietal serosal surfaces of the body cavities, and the fibrinonecrotic and pyogranulomatous reaction around affected vessels. The small, random, necrotic foci in organ parenchyma may be part of the same process and due to thrombophlebitis, or they may develop as a result of a separate mechanism, such as disseminated intravascular coagulation, or as the direct effect of the virus.

The vascular lesion begins as a proliferation and desquamation of vessel endothelium followed by medial necrosis, narrowed vascular lumina, and thrombophlebitis. There are accumulations of neutrophils, lymphocytes, plasma cells, and

macrophages in and around the affected vessels. Occasionally, adventitial fibrosis occurs with little cellular infiltrate. The changes in the omentum, mesentery, and serosal tissues may be mild or severe. The mild changes are proliferation of mesothelial cells, slight fibrin exudate with fibroblast proliferation, and scattered neutrophils and mononuclear cells. The severe changes result in a thick layer of fibrin adherent to the serosa, with necrosis and cuboidal metaplasia with syncytial cell formation of the serosa. Large numbers of neutrophils and mononuclear cells and necrotic debris are embedded in the fibrin. The focal necrosis and pyogranulomatous reaction may be present in the parenchyma of the organs and extend into the intestine, affecting the muscularis, peripheral nerve ganglia, submucosa, and mucosa.

Lesions in the various organs are caused by the vascular damage that occurs in the capsule and connective-tissue stroma. They may be found throughout the body. Subcapsular infiltrations occur particularly in the liver, lung, and pancreas, and the pyogranulomatous reaction can develop deep in the parenchyma, particularly of the kidney. In the spleen and lymph nodes there are histiocytic and fibroblastic proliferation and either depletion or hyperplasia of lymphoid follicles. In addition to focal lung lesions, there may be a diffuse interstitial pneumonia, sometimes most severe close to the visceral pleura. Similarly, a severe focal or generalized lymphocytic and plasmacytic interstitial nephritis may develop. Cellular infiltrations in the spinal or cerebral meninges and perivascular spaces tend to be more mononuclear and diffuse, with only occasional focal pyogranulomatous lesions. Degenerative and necrotic lesions in the parenchyma of the central nervous system appear to be related to the prominent vasculitis.

Experimental studies have clarified the pathogenesis of the disease and the role that the immunopathologic mechanisms play in the production of the lesion. The sequence of events proposed is as follows. The virus is phagocytized after introduction and transported to the regional node, where the primary viral replication takes place, with the generation of a primary viremia. This results in generalized infection of the monocyte–macrophage system and a secondary cell-associated viremia. The development of nonneutralizing antibody results in deposition of antigen–antibody complexes that fix complement and trigger the vascular and pyogranulomatous changes.

Complications of Peritonitis

Acute generalized peritonitis is a catastrophic development in many local diseases of the abdominal cavity, but it may be an inconsequential feature of the generalized infections because, obviously, peritonitis does not significantly affect the outcome of a strangulated loop of intestine or clostridial infections such as blackleg, hemoglobinuria, or braxy. Toxins from peritonitis may be absorbed directly through the peritoneum into retroperitoneal venules and lymphatics or may be carried from the peritoneal cavity via the diaphragmatic lymphatics. Death from toxemia may result before obvious peritonitis develops. The peculiarly efficient diaphragmatic lymphatics remove not only fluid but also particulate matter, including bacteria, and in this way the inflammation may spread via the lymphatics to the pleura and the mediastinal nodes. Of all the abdominal viscera, the intestine is most affected by inflammation of its serosa.

The typical clinical syndrome includes, within the first few hours of generalized peritonitis, intestinal hypermotility with diarrhea. By 24 hr there is absence of bowel movement and, on auscultation, complete abdominal silence; by this time the serosal irritation has caused reflex paralysis of the gut, so-called ileus. The development of ileus has the advantage that exudates are no longer distributed by intestinal movements, but there is also the disadvantage that fibrinous adhesions develop between loops of intestine, and if the animal recovers, they scarify and produce fibrous adhesions that may cause intestinal stenosis. Assessing the age of peritonitis may have significance, such as in iatrogenic rectal perforation, and requires careful gross and microscopic examination of the serosal surface and the adherent fibrin. The proliferation and age of fibroblastic components in the fibrin, on the serosa, and in the edge of the wound will give some indication of the age of the lesion.

Not all cases of generalized peritonitis are immediately fatal; depending on the nature of the exudate, the lesions may resolve completely, be converted to diffuse adhesions, or persist in some localized areas as active or adhesive peritonitis. It is remarkable how adhesions may become attentuated and even removed under the stress of continual tension.

The main defenses in the usual sorts of peritonitis are cellular, and this seems to be equally true whether or not the animal has an acquired immunity to the bacteria present. The removal and destruction of any particulate matter, including bacteria, from the abdominal cavity is one function of the lowly omentum; it is so generously endowed with phagocytes that their clusters are often visible to the naked eye as small milky spots. These fixed histiocytes are very efficient at ingesting bacteria and, if exposed to opsonins, of destroying them. In the case of Johne's disease, the organisms may be ingested by the macrophages but protected from destruction. When the bacteria are sufficiently virulent, they may proliferate in and destroy the phagocytes. This, no doubt, is why in many instances of acute peritonitis the omentum is first and most severely involved in the inflammatory reaction. It is also a "purpose" of the omentum to seal off foci of inflammation, and this it does with exceptional efficiency. The omentum is continually moved about the abdominal cavity by bodily and intestinal movements, and it sticks to areas of inflamed peritoneum. Such local omental adhesions may persist indefinitely.

Parasitic Diseases of the Peritoneum

Most of the parasites found in the peritoneal cavity have their final habitat elsewhere, and entry on to the peritoneum occurs in the normal course of migrations or as an accident. Thus various cysticerci may be found on the peritoneum during their normal development, *Dioctophyma renale,* the young *Fasciola hepatica,* and a variety of nematode larvae pass this way, and an example of accidental entry is that of *Ascaris equorum* through an intestinal perforation. Their significance and the lesions they produce are described in other chapters in these volumes with the parasite in question, and for the most part they are readily recognized at autopsy. Filariae, which are occasionally found as

young or adult worms in the abdomen of dogs, may be assumed to be *Dirofilaria immitis*.

Two parasites are unusual and deserve special attention. Young liver flukes, ***Fasciola hepatica,*** can cause acute and chronic peritonitis in cattle and sheep, and the inflammation involves the parietal peritoneum and sometimes the visceral peritoneum, especially that of liver, spleen, and omentum. The changes may consist of many tags of fibrin or a more diffuse, crusty thickening, and the young flukes may be found in the inflammatory lesions both on and beneath the peritoneum. *Stephanurus dentatus* larvae may produce severe peritonitis in the course of their migration across the peritoneal cavity of swine (see the Urinary System, this volume).

Plerocercoids, larval forms of *Mesocestoides* (dithyridium–tetrathyridium) or *Spirometra* (sparganum), occur as bladder worms or solid, straplike bodies in the peritoneal cavity of some carnivores, rodents, and reptiles in some countries. A local reaction may occur around these intermediate forms in tissues and in the peritoneal cavity, and ascites may be present. Some of these larvae can multiply by transverse division or budding, so very large numbers may be present.

Some parasites use the peritoneal cavity as their final habitat. Parasites of the subfamily Setariinae, family Onchocercidae, superfamily Filarioidea inhabit the peritoneal cavity of many domestic and wild ungulates, such as horses, cattle, buffalo, camels, sheep, goats, swine, deer, and antelope. They are commonly found at autopsy, particularly in cattle. There are only two genera, *Papillosetaria,* a monotypic genus found in *Thagulas,* which will not be referred to again, and *Setaria* (= *Hyraconema* = *Artionema*), in which there are many species.

Some species of *Setaria* have a cosmopolitan distribution and may be found in several species of wild and domestic ungulates within the same family (*S. equina* in Equidae, and *S. labiatopapillosa* in cattle, buffalo, and perhaps deer and antelope), while others are restricted geographically (*S. digitata,* Asia) perhaps by the distribution of intermediate hosts. All members of the genus live as well-adjusted symbionts as adults in their normal host and do not cause peritoneal lesions. The larval form of *S. digitata* can produce a mild fibrous peritonitis and granulomas in the retroperitoneum and bladder of cattle, and the larvae of *S. equina* and perhaps others that may normally spend part of their time in the central nervous system may occasionally penetrate the parenchyma and cause lesions.

Adults in the peritoneal cavity are oviparous, and the microfilariae can be found in the blood. The intermediate host may be one of a variety of genera of mosquitoes, or for some *Setaria* species, biting flies (*Hemotobia* spp.). The microfilariae develop into infective larvae in 2 to 3 weeks, depending on ambient temperatures, and may survive another 3 to 6 weeks in the intermediate host. They are released from the mosquito and penetrate the final host.

Setaria digitata is normally found as an adult in the peritoneal cavity of cattle and buffalo in Asia. The migration of immature forms in aberrant hosts, such as horses, camels, sheep, and goats, is an important cause of neurologic disease called ***kumri*** in Asia. The migratory pattern of the larvae is not known, but sensitive organs receive little or no damage in the natural host. In some aberrant hosts, however, infective larvae may invade the brain and spinal cord, usually about 2–6 weeks after infection. The sites of penetration and subsequent cerebral migrations are variable, as are the clinical signs produced. Characteristically, neurologic signs are of ataxia, weakness, or paralysis. The severity of the clinical signs varies from slight weakness to quadriplegia, depending on the number and location of the wandering parasites; however, affected animals may remain bright and alert. The lesions produced are fundamentally traumatic; the inflammatory component is less conspicuous. A careful gross examination of brain and spinal cord may show the areas of damage as brown foci or streaks. These are more prominent as black spots in formalin-fixed tissue. The lesions are apt to be confined to relatively small foci, so it is important that the examination be made carefully and the material for microscopic examination taken from suspicious areas. The clinical signs may suggest which parts of the brain and cord should receive the most attention. Apart from the foci of malacia, the remainder of the nervous system may be normal.

At low power, the most obvious feature of the lesions is the microcavitation that is caused mechanically by the migration of the larvae; hemorrhage is variable. Surrounding the areas of cavitation, there is loss of myelin, *Gitter*-cell formation, and fragmentation and beading of axons. In cross section this beading may appear as swollen basophilic masses, or alternatively as loss of axis cylinders. Gemastocytic astrocytes are present in older lesions. Occasionally the parasites can be found in section and made available for proper identification, but the cerebral lesions produced by aberrant parasitic migration, irrespective of the parasite, are distinctive and suggest the diagnosis. They differ from more conventional lesions in that all neural structures (myelin, axis cylinders, nerve cells, and glia) are involved, the patterns of damage are completely random, and frequently, eosinophils are the most common inflammatory cell. Neutrophils and macrophages are also frequently present, along with a mild meningitis and vascular cuffing.

The term **cerebrospinal nematodiasis** has been applied to the group of nervous diseases resulting from aberrant larval migrations (see the Nervous System, Volume 1); many parasites such as *Strongylus vulgaris* may be occasional culprits, but only *Setaria digitata* and a few other nematodes, such as *Pneumostrongylus tenuis* in moose, *Elaphostrongylus cervi* in deer, and *Baylissascaris procyonis* in rodents, have tropism for the nervous system and produce the syndrome in epizootic proportions. *Setaria cervi,* which may occur normally in deer and a variety of other cloven-hoofed animals, has been reported as causing cerebrospinal nematodiasis in deer, although *Elaphostrongylus* larvae produce similar signs and lesions, and differentiation may be difficult.

Setaria digitata larvae frequently invade the eyes of horses, as do the microfilariae of *S. equina.*

Neoplastic Disease of the Peritoneum

Primary tumors of the peritoneum may arise from the serosa itself, from the subserous connective tissues, and from the various differentiated special tissues, such as nerve sheaths. Tumors arising from the serosa are called **mesotheliomas**, sometimes

qualified as malignant. The qualification is unnecessary as virtually all are malignant, although the malignant capability is nearly always exhibited as implantation rather than metastasis.

Mesotheliomas are not common. They occur with greatest frequency in cattle and dogs but occasionally in other species. Interest in mesotheliomas has increased since the association between asbestos fiber and mesothelioma was discovered in humans. This association has not been made in animals, in which the tumor has the distinction of occurring most frequently as a congenital neoplasm in fetal or young cattle (Fig. 4.1A,D).

Mesotheliomas arise from the cells of the serous linings of pericardial, pleural, and peritoneal cavities, frequently involving all three locations. They appear as multiple small firm nodules or villous projections on a thickened mesentery or serosal surface, although fibrous or sclerosing forms have occasionally been reported. The tumor frequently is associated with a milky effusion, and in sclerosing tumors in which adhesions occasionally occur, the lesion might resemble chronic granulomatous peritonitis. Ascites as the result of effusion and blocked lymphatics is nearly always present with peritoneal tumors.

Mesotheliomas of the pleura, pericardium, or peritoneum may assume a variety of histologic patterns. This is not surprising given the diverse potentialities of the tissue that lines the embryonic celomic cavity. The tumors usually take either of two histologic forms, the one predominantly fibrous and resembling fibrosarcoma, and the other papillary and resembling adenocarcinoma. The most common tumor is a solid mass made up of single layers of dark, plump, cuboidal or columnar, neoplastic mesothelial cells with a distinct border and abundant pink cytoplasm, over a proliferating fibrocellular stroma. The mesothelial cells form loops and festoons in a papillary pattern, or line cystic spaces and tubular structures. There may be a mucinous matrix in this acinar pattern. Such malignant mesotheliomas, that is, those resembling adenocarcinoma, can mimic implantation of a true carcinoma so completely that an adequate differentiation may rest on very careful examination and exclusion of some primary focus of carcinoma. Special stains or electron microscopic examination may be required to identify the cell type.

In mesotheliomas that are predominantly fibrous, the cells may be spindle-shaped and resemble a fibrosarcoma. In the sclerosing forms there may be a thick, fibrous serosa with adhesions, and large anaplastic cuboidal cells may be found in clusters or lining cystic spaces within the fibrous tissue.

Of the retroperitoneal tumors, the **lipoma** is most frequent. These benign tumors are well known in horses, in which they originate usually in the mesenteries. They may reach enormous size, but their special significance is that they tend to become pedunculated and occasionally cause acute intestinal obstruction when the pedicle winds about a loop of intestine. In the dog, these tumors arise in the omentum rather than the mesenteries and settle on the abdominal floor. They may attain a very large size but tend not to be pedunculated. They do not, therefore, cause acute distress, and although they may be histologically malignant, metastases are unusual. They develop a pseudocapsule and central areas of necrosis. Tumors of the subserosal connective tissues, myxomas, fibromas, and their malignant counterparts, are rare, although fibrosarcomas are observed in dogs. Tumors of differentiated retroperitoneal tissues are also uncommon. Involvement of the abdominal nerves and plexuses occurs in **neurofibromatosis** of cattle, and ganglioneuromas have also been observed in this species. Extramedullary pheochromocytomas have been observed in cattle and dogs, and nonchromaffin paragangliomas occur in dogs, the latter usually in association with similar tumors elsewhere. Occasional adenocarcinomas of high malignancy, but of obscure histogenesis and origin, have been discovered in the dorsal retroperitoneum in dogs.

Secondary tumors of the peritoneum are not common but are to be expected in any abdominal neoplasia. These arise as direct implantations or as lymphogenous or hematogenous metastases. They are more usually carcinomas than sarcomas. Secondary carcinomas may be very scirrhous and, when accompanied by ascites, may closely resemble chronic peritonitis, although a relative or complete absence of adhesions is often a helpful distinguishing feature at autopsy. There are obviously many possibilities for the origin of secondary tumors; several common types are listed below. The cholangiocellular and ovarian carcinomas have already been mentioned. Those of bile duct tend to be scirrhous, as do intestinal adenocarcinomas in cattle and sheep. Squamous-cell carcinomas of the equine stomach form rather discrete implants, which may be partially caseated, but they are likely to be differentiated enough to be recognizably keratinized on gross inspection. Implants of ovarian carcinoma tend to be papillary. Vesical tumors developing in cattle in enzootic hematuria implant locally on pelvic epithelium, and rectal adenocarcinoma in dogs tends to confine its implants to the pelvic peritoneum. Malignant melanomas of perineal origin in horses produce flattish, black smudges on the peritoneum.

Miscellaneous Lesions

Cysts of the peritoneum are rather common but insignificant. Those associated with genital adnexa are described with those systems, and those associated with intermediate-stage tapeworms have been mentioned, except for *Echinococcus*, which, following the rupture of a mature hydatid into the abdomen, may develop cysts on the peritoneum. Small cysts, sometimes multiple, which are observed in the omentum, may be either inclusion cysts or local lymphatic ectasias. They are inert.

The normal flat, pavement-type cells of the serosa may undergo **metaplasia** to a cuboidal or columnar type resembling epithelium. Such metaplasia is probably the mildest response of the peritoneum to irritation but may also be a response to estrogen. Inflammatory metaplasia leading to ossification can occur in peritoneal scars, especially in swine, but may also be found in the mesenteries and the dorsal retroperitoneum without obvious antecedent change, although ossification may occur following fat necrosis as well. The newly formed bones are flat and of variable size and shape and are usually found in adipose tissue.

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