***Edema***

**Etiology:**

Edema results from four causes:

* Increased hydrostatic pressure in capillaries and veins due to chronic (congestive) heart failure; or obstruction to venous return.
* Decreased plasma oncotic pressure.
* Increased capillary permeability in endotoxemia, part of the allergic response vasculitis and damage to the vascular endothelium.
* Or Obstruction to lymphatic flow.

**Increased hydrostatic pressure:**

* Symmetric ventral edema in chronic (congestive) heart failure, symmetric pulmonary edema in acute heart failure.
* Generalized edema in enzootic calcinosis of cattle.
* Local symmetric ventral edema in udder edema in late pregnancy from compression of veins and lymphatics by the developing mammary gland (and possibly the enlarging fetuses and uterus) causing mammary or ventral edema in cows (particularly heifers), mares and occasionally ewes.

Edema resolves 5- 10 days following parturition.

* Local edema by compressive lesions on veins (as in thymic lymphosarcoma with compression of the cranial vena cava) draining other anatomic locations.
* Local edema in portal hypertension due to hepatic fibrosis causing ascites.

**Decreased plasma oncotic pressure:**

Decreased total protein concentration in plasma, and particularly decreased plasma albumin concentration, will result in symmetric ventral edema.

Hypoalbuminemia is more important than hypoglobulinemia in inducing edema formation because albumin provides the largest contribution to plasma oncotic pressure.

Hypoalbuminemia can result from increased loss (due to blood sucking parasites or a cross the gastrointestinal tract, kidneys, or into a large third space such as the pleural or peritoneal cavities).

Decreased production (as in chronic hepatic failure) or decreased intake.

Chronic blood loss, especially in heavy infestation with blood sucking parasites such as strongylus sp. In the horse, fasciola sp. In ruminants, Hemonchus sp. In ruminant of all ages, especially goats and Bunostomum sp. In calves.

Protein losing gastroenteropathies as in Johne 's disease and amyloidosis in adult cattle , right dorsal colitis in horse , heavy infestations with nematode parasites in ruminants , particularly Ostertagia sp. In young cattle and Cyathostomiasis in horses.

Glomerulonephropathies , such as amyloidosis in adult cattle .

Chronic liver damage causing failure of plasma protein synthesis (rare and terminal in large animals).

terminally in prolonged malnutrition with low dietary protein intakes e.g. ruminants at range in drought time.

**Increased capillary permeability**

* Due to endotoxins
* Allergic edema as in urticaria and angioneurotic edema caused by local liberation of vasodilators.
* Toxic damage to vascular endothelium or vasculitis – in anthrax , gas gangrene , and malignant edema in ruminants , equine viral arteritis , equine infectious anemia , purpura hemorrhagica in horses , and heartwater ( cowdriosis ) in ruminants .

**Obstruction to lymphatic flow:**

* Part of the edema caused by tumors or inflammatory swellings is lymphatic obstruction. Excessive fluid loss also originates from granulomatous lesions on serous surfaces .Ascites or hydrothorax may results.
* Congenital in inherited lymphatic obstruction edema of Ayrshire and Herford calves.
* Sporadic lmphangitis (big leg) of horses.
* Edema of the lower limbs of horses immobilized because of injury or illness.

**Pathogenesis:**

Edema is the excessive accumulation of fluid in the interstitial space of tissue caused by a disturbance in the mechanism of fluid interchange between capillaries, the interstitial spaces and the lymphatic vessels.

At the arteriolar end of the capillaries the hydrostatic pressure of the blood is sufficient to overcome its oncotic pressure and fluid tends to pass into the interstitial space.

At the venous ends of the capillaries the position is reversed and fluid tends to return to the vascular system.

A small decrease in oncotic pressure or small increase in hydrostatic pressure leads to failure of the fluid to return to the capillaries.

Increased fluid passage into the interstitial space can also occur where there is increased vascular permeability due to vascular damage.

Under these circumstances, fluid accumulates in the interstitial space when the fluid flux across the endothelium is greater than the ability of the lymphatic system to drain it.

**Clinical findings**

Accumulation of edematous transudation in subcutaneous tissues is referred to as anasarca .in the peritoneal cavity as ascites. In the pleural cavities as hydrothorax. And in the pericardial sac as hydro pericardium

Anasarca in large animals is usually confined to the ventral wall of the abdomen, and thorax, the brisket and if the animal is gazing, in the intermandibular space because of the large hydrostatic pressure gradient between the submandibular space and heart.

Edema of the limbs uncommon in cattle, sheep, but occurs in horses quite commonly when the venous return is obstructed or there is lack of muscular movements.

Local edema of the head in the horse is common lesion in African horse sickness and purpura hemorrhagica.

Edematous swellings are soft, painless and cool to tough, and pit n pressure.

In ascites there is distention of the abdomen and the fluid can be detected by a fluid thrill on tactile percussion, fluid sounds on succession and by paracentesis.

A level of top line of fluid may be detectable by any of these means.

In the pleural cavities and pericardial sac the clinical signs produced by the fluid accumulation include restriction of cardiac movements, embarrassment of respiration and collapse of the ventral parts of the lungs.

 The heart sounds and respiratory sounds are muffled.

**Clinical pathology**

Cytological examination of a sample of fluid reveals an absence of inflammatory cells where edema is the result of hypoproteinemia or due to obstruction.

**Differential diagnosis**

* Rupture of urethra or bladder for differentiation of ascites.
* Peritonitis or pleuritis for accumulation of fluid in abdominal or pleural cavities.
* Cellulitis for local edema.

**Treatment**

* The treatment of edema should be aimed at correcting the primary disease.
* Congestive heart failure may need to be treated with digoxin.
* Pericarditis by drainage of the sac.
* Parasitic gastroenteritis by administration of the appropriate anthelmintics.
* Obstructive edema by removal of the cause and hypoprtoteinemia by the administration of plasma or plasma substitutes and the feeding of high quality protein.
* Ancillary measures include restriction of the amount of salt in the diet, the use of diuretic and aspiration of fluid.

***Anemia***

**Etiology**

Anemia can be classified as hemorrhagic or hemolytic anemia, or anemia due to decreased production of erythrocytes.

**Hemorrhagic anemia**

* Spontaneous rupture or traumatic injuries to large blood vessels are the common reasons for acute severe hemorrhage.
* Any sort of minor surgical wound e.g. castration, dehorning may lead to excess hemorrhage where there is hemorrhagic tendency due to defects of clotting.
* Severe parasitic infestations as can occur in massive infestation with strongyles, hoockworm, and haemonchus sp. Infestation, fasciola hepatica and coccidian c.
* Heavy infestations with ticks and sucking lice.
* Clotting defect.

**Hemolytic anemia**

**Cattle and sheep**

* Babesiosis , anaplasmosis , eperythrozonosis , trypanosomiasis , nagana , theileriasis
* Bacillary hemoglobinuria.
* Leptospirosis (L. interrogans sero var Pomona).
* Post parturient hemoglobinuria.
* Associated with grazing Brassica spp. Rape, kale, chon moellier, turnips, and cabbages.
* Associated with the excessive feeding of called onion, the feeding of cannery offal, especially tomatoes and onion.
* Poisoning by miscellaneous agents including phenothiazine.
* Poisoning – chronic copper poisoning.
* Treatment with long acting oxytetracycline.
* Water intoxication and drinking cold water in calves.
* Part of transfusion reaction.

Rare cases of isoerythrolysis in calves, from vaccination of the dam with blood derived vaccine such as anaplasma vaccine.

Autoimmune hemolytic anemia is recorded in calves but is rare.

* Immune- mediated anemia can occur in lambs that are fed cow colostrums as a source of immunoglobulin. Occurs only with colostrums from certain cows.
* Anemia is evident at 7-20 days of age but jaundice and hemoglobinuria are not usually present.
* The syndrome must be differentiated from immune – mediated thrombocytopenia which occurs at a younger age in some lambs fed bovine colostrums.

**Horses**

* Equine infectious anemia
* Babesiosis
* Phenothiazine poisoning
* Red maple leaf toxicity
* Isoerythrolysis in foals
* Autoimmune hemolytic anemia
* Immune- mediated hemolytic anemia and thrombocytopenia (Evan's syndrome).
* Penicillin – induced hemolytic anemia. This is a rare event but can occur when horses develop IgG antipenicillin – antibodies. These antibodies bind to penicillin on erythrocytes agglutinate with patient serum.
* Immune – mediated hemolytic anemia associated with trimethoprim – sulfa methoxazole.
* Some snake envenomation cause intravascular hemolysis in dogs and cats' .and hemnolytic anemia can occur in snake bite in horses and calves.

**Anemia due to decreased production of erythrocytes or hemoglobin**

The diseases in this group tend to affect all species equally so that they are divided up according to cause rather than according to animal; species.

**Nutritional deficiency:**

* Cobalt and copper: these elements are necessary for all animals, but clinically occurring anemia is observed only in ruminants.
* Iron , but as a clinical occurrence this is limited to baby pigs and possibly to young calves designated for the white veal marker.
* Potassium deficiency is implicated in causing anemia in calves.
* Pyridoxine deficiency.
* Folic acid deficiency.

Anemia in stabled horses, especially pregnant mares who have no access to pasture has been reported to respond well to folic acid.

**Chronic diseases**

* Chronic suppurative processes can cause severe anemia by depression of erythyropoiesis.
* Radiation injury.
* Poisoning by bracken, trichloroethylene – extracted soybean meal, arsenic, furazolidone, phenylbutazone, cause depression of bone marrow activity.
* Intestinal parasitism e.g. ostertagiasis, trichostrongylos in calves and sheep, have this effect.
* Temporarily for several weeks after sudden movement to high altitude.

**Myelophthistic anemia**

Myeophthistic anemia, in which the bone marrow cavities are occupied by other, usually neoplastic tissues, is rare in farm animals.

Clinical signs, other than of the anemia which is macrocytic and normochromic, includes skeletal pain, pathological fractures and pasresis due to the osteolytic lesions produced by the invading neoplasm.cavitation of the bone may be detected on radiographic examination.

 Lyposarcoma is seen in calves

Plasma cell myelomatosis has been observed as a cause of such anemia in pigs, calves, and horses.

Myelophthitic anemia is also reported in a 10 – year old pony associated with myelofibrosis.

**Pathogenesis**

Irrespective of the cause of anemia the primary abnormality of function is the anemic anoxia which follows.

Hemolytic anemia is often sufficient severe to cause hemogloibiunuria and may result in hemoglobinuric nephrosis and depression of renal function.

Apolastic anemia caused by toxins from a suppurative process is a secondary manifestation and is relieved by removal of the cause, and anemia due to nutritional deficiency is similarly reversible.

In the situation where erythrolysis occurs immediately after drinking a large volume of cold water, the hemolysis occurs in the capillaries in the intestinal wall.

The plasma osmotic pressure decreases as a result of the fluid intake, and hemolysis occurs because this is below the minimum osmotic tolerance of the erythrocytes.

The primary responses to tissue anoxia caused by anemia are an increase in cardiac output due to increases in stroke volume and heart rate, and a decrease in circulation time.

A diversion of blood from the peripheral to the splanchnic circulation also occurs.

In the terminal stages when the tissue anoxia is sufficiently severe there may be a moderate increase in respiratory activity.

Provided the activity of the bone marrow is not reduced, erythropoiesis is stimulated by lowering of the tissue oxygen tension.

**Autoimmune hemolytic anemia**

The disease is believed to result from an aberrant production of antibodies targeted against surface antigens of the erythrocytes as a result of an alteration in the erythrocyte membrane from systemic bacterial, viral, or neoplastic disease.

An alternate hypothesis is the development of immune competent clones that direct antibody at the red cell membrane.

Red cells are lost by intravascular hemolysis or removal by macrophages of the reticuloendothelial system and anemia occurs when the capacity of the bone marrow to compensate for increased red cell destruction is exceeded.

Autoimmune hemolytic anemia is cxonside3red idiopathic if it cannot be associated with an underlying disease and is considered secondary if associated with another condition.

Often this is neoplastic disease.

The antibodies are of the IgG or IgG class, may be agglutinating or non – agglutinating.

**Clinical findings**

* Pallor of the mucosae.

In clinical cases of anemia there are signs of pallor, muscular weakness, depression and anorexia.

The heart rate is increased. The pulse has large amplitude and the absolute intensity of the heart sounds is markedly increased.

Terminally the moderate tachycardia of the compensatory phase is replaced by a severe tachycardia, a decrease in the intensity of the heart sounds and a weak pulse.

Anemia , particularly chronic anemia result in cardiac dilatation and dilatation of the annulus of the right atrio ventricular orifices causing a hemic murmur which is systolic in timing and waxes and wanes with each respiratory cycle , reaching its maximum at the peak of inspiration .

This type of murmur is not pathognomonic of anemia and may be present in any form of myocardial asthenia or when the viscosity of the blood is reduced for any reason,

The most severe degree of respiratory distress appearing as an increase in depth of respiration without much increase in rate. Labored breathing occurs only in the terminal stages.

**Clinical pathology**

PCV, erythrocyte count number and size, hemoglobin content and erythrocyte morphology.

In hemorrhagic and hemolytic anemia there is an increase in the number of immature red cells in the blood .Examination of bone marrow aspirates and biopsies can determine if the anemia is regenerative and the nature of degenerative anemia .

Ameyloid to erythroid ratio less than 0.5 and a reticulocyte counts greater than 5% are indicative of erythrocyte regeneration and ratios > 0.93 in combination with normal peripheral white cell counts\ , indicative of non regenerative or hypoplastic anemia ,

**Hemolytic anemia**

Are characterized by a normal total protein, bilirubinemia and bilirubinuria.

In the acute stage the serum or plasma may be discolored due to presence of hemoglobin.

In cases caused by oxidative agents the erythrocytes show morphologic changes and Heinz bodies.

Immune – mediated hemolytic anemias: show increased erythrocyte fragility, erythrocyte agglutination and a positive antiglobulin (Coomb's test).

**Throbocytopenia**

Causing hemorrhage produces low platelets counts (< 20000\ µl) but normal coagulation profiles.

**Deficiency anemia**

Mast shows hypochromasia.

Aplastic anemia

Caused by toxins is manifested by normocytic, normochromic, non – regenerative anemia.

Bleeding time and coagulation test platelets function, and the function of the blood vessels in hemostasis can be evaluated by bleeding time tests.

The site is usually the inside of the lip or a clipped side of the neck.

Normal bleeding times for the horse are 2-6 minutes.

**Differential diagnosis**

Red cell morphology as an aid to differentiation.

The classic approach initially divides anemias into regenerative anemias, and degenerative anemias.

Regenerative anemias are caused by hemorrhage or hemolysis in the presence of normal bone marrow function and are identified by the regenerative response as determined by erythrocyte morphology and reticulocyte counts.

]degenerative anemia are caused by primary or secondary bone marrow disorders in reed cell production and are identified by a lack of regenerative response in peripheral blood .

Plasma total protein as an aid to differentiation

Low plasma total protein is indicative of acute or chronic hemorrhage

Clinical examination, including paracentesis, fecal occult blood, determines the site and coagulation profiles, platelets counts and fecal occult blood help established the etiology.

**Treatment**

Treat primary cause

Blood transfusion ,.

***Water intoxication***

The ingestion of large amounts of water by young calves may result in hemoglobinuria, or hemoglobinuria and signs of cerebral edema.

A similar syndrome, manifest only hemoglobinuria, occurs when calves in winter ingest cold water.

**Etiology**

The ingestion o9f excessive quantities of water when animals are very thirsty may results in water intoxication.

**Epidemiology**

It occurs in calves in normal husbandry systems, when animals that have3 had limited access to water are suddenly gives free access.

**Clinical findings**

Hemoglobinuria as a result of intravascular hemolysis is prominent in most reports. And there may be a moderate to severe hemolytic anemia.

Dark red urine is passed shortly following access to water.

Hypothermia, tachycardia followed by bradycardia, and salivation.

Nervous signs include muscle weakness and tremor, restlessness, hyperesthesia, ataxia, tonic and clonic convulsions and terminal coma occur in some outbreaks and dead calves may be the first indication of the disease in a group.

**Clinical pathology**

Hemoglobinuria is evident and there is hyponatremia and hypochloremia and elevated serum aspartate aminotransferase.

**Treatment**

Sedation if necessary.

An administration of diuretic (Furosemide 1 mg \ kg body weight).

In severe cases the intravenous injection of hypertonic solutions v such as 5% saline solution.

***Venous thrombosis***

The development of thrombi in veins may results in local obstruction to venous drainage, in liberation of emboli which lodge n the lungs, liver or other organs and in the development of septicemia, or of endocarditis.

 **Phlebitis**

It is the common origin of thrombi and may be caused by localization of a blood – borne infection, by extension of infection from surrounding diseased tissues, by infection of the umbilical veins in the newborn, and by irritant injections into the major veins.

**Thrombophlebitis of the jugular vein**

Is a complication of injections or catheterization in some animals and occurs in all species.

It can result from damage to the vascular endothelium by canula or indwelling intravenous catheters, inflammation caused by chemical irritation ort bacterial invasion from contamination during insertion of the needle or catheter or migration along the catheter from the skin.

Phlebitis develops and can be detected clinically 24- 72 hours after catheter insertion,

In horses a study indicated that ongoing infectious disease was a risk factor for the development of catheter associated thrombophlebitis and thrombophlebitis is especially common in horses with severe gastrointestinal disease that are accompanied by endotoxemia.

Horses are also at higher risk following surgery.

Severely ill cows are more likely to develop jugular vein thrombo phlebitis than healthy cows.

Intravenous injections of irritating materials.

Materials, such as tetracycline, phenylbutazone, or solutions of calcium chloride or hypertonic saline, may cause endothelial damage followed by ci8catrical contraction, with or without thrombus formation.

Jugular phlebitis with thrombosis is not uncommon in feedlot cattle that have received repeated intravenous antibiotic medication and may lead to thrombo9ewmbolic respiratory disease.

Accidental injection of irritating materials around the vein usually causes marked local swellings, sometimes with necrosis and local sloughing of tissue which may be followed by cicatrical contraction of local tissues.

Phenylbutazone commonly used as a non – steroidal anti inflammatory drug. Its use in horse may be associated with toxicity which is manifested with oral and gastrointestinal ulceration and renal medullary crest necrosis.

Affected horses show depression, anorexia, and neutropenia with ulcers in the mouth. Especially on the ventral aspect of the tongue.

Ulcers in the fundic and pyloric portion of the stomach also develop, but are usually sub clinical although they may be evident on gastroscopc examination or at necropsy.

More severe cases show signs of colic and diarrhea in association with intestinal ulceration and duodenitis and show evidence of renal

Toxicity may develop following either intravenous or oral administration of the drug.

Intravenous administration is frequently associated with the development of phlebitis and jugular thrombosis at the site of injection.

Phlebitis may also develop in those animals at sites of venepuncture performed for purpose other than phenylbutazone administration.

Venous thrombi are relatively common in strangles in the horse, and may affect the jugular veins or the caudal vena cava.

Thrombosis of the caudal vena cava due to hepatic abscessation and resulting in embolic pneumonias and pulmonary arterial lesions occurs in cows and is described together with cranial vena caval thrombosis.

Less common examples of venous thrombosis are those occurring in the cerebral sinuses either by drainage of an infection from the face or those caused by the migration of parasitic larvae.

Purpling and later sloughing of the ears which occur in many septicemias in pigs also caused by phlebitis and venous thrombosis.

Thrombosis of the tarsal in is a complication of infections in the claw of cattle,

**Clinical signs of venous thrombosis**

 Engorgement of the vein, pain on palpation sand local edema.

In unsupported tissues rupture may occur and lead to fatal internal or external hemorrhage.

Ultrasonographic examination assists the diagnosis and the detection of a cavitating area within the thrombous supports a diagnosis of septic thrombophlebitis.

Angiography can also assist in diagnosis

Bacteriological culture should be attempt, preferably from the tip of the removed catheter.

At necropsy the obstructed vessels and thrombus are usually easily located by the situation of the edema and local hemorrhage.

**Diagnosis**

Depend on the presence of signs of local venous obstruction in the absence of obvious external pressure by tumors, enlarged lymph nodes, hematomas or fibrous tissue constriction.

Pressure of a fetus may cause edema of the perineum, udder and ventral abdominal wall during late pregnancy.

Local edema due to infective processes such as black leg, malignant edema and per acute staphylococcal mastitis, severe toxemia, acute local inflammation and necrosis.

**Parenteral treatment**

With antibacterial drugs and hot fomentations to external veins, or treatment with a topical anti inflammatory agent such as 50% dimethyl sulfoxide are usually instituted to remove the obstruction or allay the swelling.

If a catheter is being used it should be removed.

***Cor pulmonale***

Cor pulmonale is the syndrome of right heart failure resulting from an increase in right heart workload secondary to increased pulmonary vascular resistance and pulmonary hypertension.

The most documented cause of pulmonary hypertension in livestock is alveolar hypoxia.

Acute alveolar hypoxia (lowered alveolar po2) is a potent cause of pulmonary hypertension in several species, but cattle are especially reactive and this is the cause of the syndrome of cor pulmonale in cattle at high altitudes, bovine brisket disease.

An outbreak of cor pulmonale with pulmonary vascular lesions similar to those seen with mountain disease but occurring in calves not at altitude is recorded.

Pulmonary hypertension can also result from partial destruction of the pulmonary vascular bed and a reduction in it total cross- sectional area.

Pulmonary thromboembolic disease can produce right heart failure by this mechanism.

Chronic interstitial pneumonia and emphysema may also induce cor pulmonale by the same mechanism.

Chronic obstructive pneumonia, where there is airway constriction and accumulation of fluid in distal ways, may induce pulmonary hypertension by a combination of chronic hypoxia and reduction of the pulmonary vascular bed.

Although pulmonary hypertension and right heart hyper trophy may occur in livestock with primary pulmonary disease, clinical cardiac insufficiency is usually minor and right heart failure rare.

Nevertheless, it can occur and is a cause of congestive heart failure in cattle.

In goats, cor pulmonale, with right ventricular and right atrial hypertrophy secondary to interstitial pneumonia, may lead to atrial fibrillation, and cor pulmonale leading to atrial fibrilation is also recorded in horses.

In highly conditioned feedlot cattle, increased intra- abdominal pressure resulting from excessive abdominal fat, fore stomach engorgement and recumbency can lead to pulmonary hypoventilation with decreased alveolar po2 and subsequent right heart failure, a syndrome analogous to the Pickwickian syndrome in man.

Chronic severe elevation in pulmonary venous pressure can lead to constriction and hypertrophy of the vascular smooth muscle of precapillary vessels with resultant pulmonary hypertensio9n .an elevated left ventricular filling pressure is perhaps the more common cause and can set the stage of right heart failure in the left heart failure situations.

**Cor pulmonale**

**Etiology**

Conditions of right heart dilatation, hypertrophy, and subsequent failure caused by pulmonary hypertension and increased pulmonary vascular resistance often is referred to collectively as cor pulmonale.

This condition is uncommon and sporadic in dairy cattle.

Most cases of cor pulmonale occur in cows known to have chronic pneumonia , bronchiectesis , and pulmonary abscesses , secondary to bacterial bronchopneumonia , consolidated anteroventral lung lobes from previous pneumonia , or chronic , lungworms .

Severe chronic interstitial pulmonary disease, although rare, may also result in cor pulmonale in mature cattle with diffuse pulmonary fibrosis .in these instances pulmonary hypertension initially may result from alveolar hypoxia and subsequent percapillary vasoconstriction.

Chronic hypoxia and pulmonary hypertension in cattle may provoke hypertrophy of medial smooth musculature within pulmonary arteriers and arterioles, causing further work for the right ventricle.

The most common example of cor pulmonale is brisket edema or mountain sickness of beef cattle.

Pulmonary hypertension secondary to pulmonary and bronchial arteritis recently was observed as an endemic problem in a group of dairy calves.

**Signs**

Dyspnea, tachycardia, ventral edema, and venous distention and pulsation characterize cor pulmonale. Murmur or a gallop rhythm may be auscultated, depending on vulvular function, the degree of myocardial hypertrophy, or cardiac chamber dilation.

Heart sounds have normal or increased intensity.

**Diagnosis**

History of chronic pulmonary disease (or exposure to high altitude), ruling out other cardiac diseases, and findings signs consistent with right heart failure provide suggestive evidence of cor pulmonale.

**Treatment**

Treatment of the primary lung disease coupled with furosemide therapy may be beneficial.

Furosemide is administered at 0.5 to 1.0 mg \ kg twice daily as a diuretic.

**Pericarditis**

**Etiology**

Pericarditis is not common.

Traumatic pericarditis, perforation of the pericardial sac by an infected foreign body, occurs commonly only in cattle.

Traumatic pericarditis is also recorded in the horse, and in a lamb.

Localization of blood borne infection occurs sporadically in many diseases.

Direct extension of infection from pleurisy or myocarditis may also occur in all animals, but the clinical signs of pericarditis in such cases are usually dominated by those of the primary disease.

In most cases of pericarditis in horses no causative agent is isolated.

There is commonly a history of upper or lower respiratory tract disease.

Most cases are fibrinous or sepsis. But an effusive non septic form is also described and has been called idiopathic effusive pericarditis.

Pericarditis in horses occurs predominantly in adults.

**Cattle:**

Pasteurellosis

Black disease – if patients survive more than 24 hours.

Sporadic bovine encephalomyelitis

Haemophilus spp., including haemosomnus.

Tuberculosis

Pseudomonas aeriginosa

Mycoplasma spp.

Actinobacillus Sui.

**Horses**

Streptococcus spp., including: str. equi, stre. Zooepidemicus and str. Faecalis.

Tuberculosis

Actinobacillus equuli

In association with EHV.1 infection

Idiopathic effusive (non – septic) pericarditis.

**Sheep and goats**

Pasteurellosis

Staphylococcus aureus

Mycoplasma spp.,

Pathogenesis

In the early stages inflammation of the pericardium is accompanied by hyperemia and the deposition of fibrinous exudates which producers a friction sound when the pericardium and epicardium rub together during cardiac movement.

As effusion develops the inflamed surfaces are separated, the friction sound is rep-0laced by muffling of the heart sounds, and the accumulated fluid compresses the atria and right ventricle, preventing their complete filling.

Congestive heart failure follows.

A severe toxemia is usually present in suppurative pericarditis because of the toxins produced by the bacteria in the pericardial sac.

Gas will occur along with fluid in the sac, if gas producing bacteria are present.

I8n the recovery stage of non suppurative pericarditis the fluid is reabsorbed, and adhesions form between the pericardium and epicardium to cause van adhesive pericarditis, but the adhesions are usually not sufficiently strong to impair cardiac movement.

In suppurative pericarditis the adhesions which form become organized and may cause complete attachment of the pericardium to the epicardium, or this may occur only in patches to leave some loculi which are filled with serous fluid.

In either case restriction of cardiac movement will probably be followed by the appearance of congestive heart failure.

**Clinical findings**

In the early stages there is pain, avoidance of movement, abduction of the elbows, arching of the back and shallow, abdominal respiration.

Pain is evidenced on percussion or firm palpation over the cardiac area of the chest wall, and the animal lies down carefully.

A pericardial friction sound is detectable on auscultation of the cardiac area.

The temperature is elevated to 39.5 – 41 oC, and the pulse rate is increased.

Associated signs of pleurisy, pneumonia and peritonitis may be present.

In most cases of pericarditis caused by traumatic reticuloperitonitis , hematogenous infection , or spread from pleurisy , the secondary stage of effusion is manifested by muffling of the heart sounds , decreased palpability of the a pex beat and an increase in the area of cardiac dullness with decreased amplitude of the peripheral pulse .

If gas is\present in the pericardial sac each cardiac cycle may also accompanied by splashing sounds.

Signs of congestive heart failure become evidence.

Fever is present.

The heart rate is markedly increased and toxemia is severe.

This is the most dangerous period and affected animals usually die of congestive heart failure or of toxemia in 1-3 weeks.

In the stage of slow recovery of chronic pericardits additional signs of myocarditis, particularly irregularity, may appear.

The heart sounds becomes less muffled and fluid sounds disappear altogether or persist in restricted areas.

In the horse, both the idiiopathic effusive and the septic forms of pericarditis present with marked muffling of the heart sounds. Tachycardia, distension of the jugular veins and subcutaneous edema of the ventral body wall.

**Clinical pathology**

A marked leukocytosis and shift to the left is detectable on hematological; examination in traumatic pericarditis.

In the other forms of pericarditis changes in the blood depend upon the other lesions present and on the causative agent.

In the stage in which effusion occurs a sample of fluid may be aspirated from the pericardial sac and submitted for bacteriological examination.

**Necropsy findings**

In the early stages, there is hyperemia of the pericardial lining and a deposit of fibrin.

When effusion occurs there is an accumulation of turbid fluid and tags of fibrin are present in their greatly thickened epicardium and pericardium.

 Gas may also be present and the fluid may have a putrid odor.

When the pericarditis has reached a chronic stage, the pericardium is adherent to the epicardium over a greater or lesser part of the cardiac surface.

**Differential diagnosis**

Pleuritis Mediastinal abscess Cardiac valvular disease .Hydro pericardium occurs in congestive heart failure, in mulberry heart disease of pigs.

Clostridial intoxication of sheep and in lymphomatosis

**Treatment**

Antibacterial treatment of the specific infection.

A combination of penicillin and gentamicin is common and provides cover of the likely organisms associated with this infection.

Pericardiocentesis and drainage should be conducted as required to relieve the fluid pressure in the pericartdial sac.

**The prognosis**

Varies with the etiology. But it is generally grave in cases of septic pericarditis in horses.

***Traumatic pericarditis***

Perforation of the pericardial sac by a sharp foreign body originating in the reticulum causes pericarditis with the development of toxemia and congestive heart failure.

Tachycardia, fever, engorgement of the jugular veins, anasarca, hydrothorax, and ascites, and abnormalities of the heart sounds are the diagnostic features of the disease.

**Etiology**

Traumatic pericarditis is caused by penetration of the pericardial sac by in migrating metal foreign body from the reticulum.

The incidence is greater during the last 2- 3 months of pregnancy and at parturition than at other times.

Approximately 8% of all cases of traumatic reticuloperitonitis will develop pericarditis.

Most affected animals die or suffer from chronic pericarditis and do not return to completely normal healthy.

**Pathogenesis**

The penetration of the pericardial sac may occur with the initial perforation of the reticular wall.

The animal may have had a history of traumatic reticuloperitonitis some times previously, followed by pericarditis, usually during later pregnancy or at parturition.

Introduction of a mixed bacterial infection from the reticulum causes a severe local inflammation, and persistence of the foreign body in the tissue is not essential for the further progress of the disease.

The first effect of the inflammation is hyperemia of the pericardial surfaces and the production of friction sounds synchronous with the heart beat.

Two mechanisms then operate to produce signs:

The toxemia due to the infection and the pressure on the heart from the fluid which accumulates in the sac and produce congestive heart failure.

Depression is characteristic of the first and edemas of the second.

Thus an affected animal may be severely ill for several weeks with edema developing only gradually, or extreme edema may develop within 2-3 days.

The rapid development of edema usually indicates early death. If chronic pericarditis persists there is restriction of the heart action due to adhesion of the pericardium to the heart.

Congestive heart failure results in most cases but some animals may recover.

Uncommon sequel after perforation of the pericardial sac by a foreign body is rupture of a coronary artery or the ventricular wall.

Death usually occurs suddenly due to acute, congestive heart failure from compression of the heart by the hemopeticardium.

**Clinical findings**

Depression, anorexia, habitual recumbenmcy, and rapid weight loss are common.

Diarrhea or scant feces may be present. Grinding of teeth. Salivation. Nasal discharge is occasionally observed. Arching back. Abducted elbows. Respiratory movements are more obvious, abdominal respiration, shallow, increased in rate to 40- 50 \ minute, and often accompanied by grunting.

Engorgement of the jugular veins, edema of the brisket and ventral abdominal wall are common.

And in severe cases there may be even edema of the conjunctive with grape – like masses of edematous conjunctive hanging over the eyelids.

A prominent jugular venous pulse is usually visible and extends proximally up the neck.

Pyrexia 40- 41 oC in early stages. Increased heart rate to 100 \ min. diminution of pulse amplitude. Rumen movement depressed. Painful grunt.

In early stages normal heart sounds accompanied by pericardial friction sounds, which may wax and wane with respiratory movements.

Several days later there is effusion heart sounds are muffled. Gurgling, splashing, or tinkling sounds.

Most animals die with 1-2 weeks.

In terminal stages

Gross edema, dyspnea, severe watery diarrhea, depression, recumbency, complete anorexia, enlargement of the liver.

**Clinical pathology**

Hemogram, leukocytosis – TLC 16000 – 30000 \ ml, neutrophilia, eosinopenia, percardiocentesis, foul – smelling and turbid which is diagnostic for pericarditis.

**Differential diagnosis**

The typical clinical findings in pereicarditis are:'

Chronic illness, toxemia, fever, congestive heart failure, muffled heart sounds.

The major causes of congestive heart failure in cattle are pericarditis, endocardial disease, myocardiopathy, and cor pulmonale (pulmonary hypertension due to chronic pulmonary disease)

Endocarditis, lymphomatosis. Congenital cardiac defects

**Treatment**

Unsatisfactory

***Hemorrhagic disease***

Petechial and ecchymotic hemorrhage, spontaneous hemorrhage or excessive bleeding after minor injury may result from increased capillary fragility, disorders in platelet function or from defects in the coagulation mechanism of the blood.

**Examples of diseases with hemorrhage**

**Vasculitis**

**Septicemic and viremic diseases**

The vasculitis is associated with endothelial damage from the direct inflammatory or degenerative effects of the infection.'

It may be complicated by defects in blood coagulation and platelets disorders depending upon the infection.

In many instances coagulation defects are a manifestation of early disseminated intravascular coagulation.

Clinical, petechial and ecchymotic hemorrhsge associated with septicemia are most obvious in the mucous membranes of the mouth, vulva, and conjunctiva or in the sclera but they are widely distributed throughout the body on postmortem examination.

***Purpura hemorrhagica***

This is a hemorrhagic disease of horses associated with aleukocyclasdtic vasculitis.'

The majority of cases occur as a sequel to strangles.

Cases also occur following immunization against streptococcus equi and as sequel to infection with other streptococci.

The disease appears to be an immune complex – mediated disease with deposition of IgA containing immune complex or vessels walls.

Hemorrhagic tendencies in the disease include petechial and ecchymotic hemorrhages but also may results in large extravasations of blood and serum into tissues.

The hemorrhage and exudation of serum may result in anemia Sand depression in the circulating blood volume

**Treatment**

Blood transfusion and corticosteroids

***Disseminated intravascular coagulopathy (DIC)***

Disseminated intravascular coagulation can be initiated by a variety of different mechanisms:

* Extensive tissue necrosis such as occurs in trauma , rapidly growing neoplasm , acute intravascular hemolysis and infective diseases such as black leg , can cause extensive release of tissue thromboplastin and initiate exuberant coagulation via the extrinsic coagulation .
* Exuberant activation of the intrinsic pathway can occur when there is activation of the Hageman factor by extensive contact with vascular collagen, as occurs in disease with vasculitis, or those associated with poor tissue perfusion and tissue hypoxia with resultant endothelial damage.
* Factors that initiate platelet aggregation such as endotoxin , that cause reticulo- endothelial blockage such as excessive iron administration to piglets or that cause hepatic damage to interfere with clearance of activated clotting factors .

***Hemorrhage***

**Etiology**

Spontaneous rupture or traumatic injury to large blood vessels is the common reason for acute severe hemorrhage.

Any sort of minor surgical wound e.g. castration, dehorning, may lead to excess hemorrhage where there is hemorrhagic tendency due to defects of clotting.

Severe parasitic infestation can be accompanied by severe loss of blood as can occur in massive infestations with strongyles, hookworm, and haemonchus sp. Infestation, fasciola hepatica and coccidian.

Heavy infestation with ticks and sucking lice are quoted as causes, but would have to be extremely heavy.

**Clotting defects**

That led to hemorrhage (coagulation defects that causes of acute hemolytic anemia). Some of the more common causes of primary hemorrhage include the following:

**Cattle and sheep**

* Spontaneous pulmonary hemorrhage associated with the caudal vena caval syndrome.
* Abomasal ulcer, sometimes originating from a bovine viral leukosis lesion (cattle).
* Enzootic hematuria with bleeding from a bladder lesion (cattle).
* Pyelonephritis with bleeding from a renal lesion ( cattle)
* Intra- abdominal hemorrhage as a result of arterial aneurysm possibly associated with copper deficiency (cattle).
* Ruptured middle uterine artery during prolapsed or torsion of uterus.
* Cardiac tamponade due to rupture of coronary artery or ventricular chamber, rupture of the aorta.
* Rupture of liver associated with dystocia in lambs and in older lambs possibly associate with vitamin E deficiency.

**Horses**

* Ethemoidal hematoma
* Exercise – induced pulmonary hemorrhage
* Rupture of the middle uterine, uteri – ovarian artery (especially right side) or iliac artery associated with parturition, more commonly in aged mares.
* Nasal bleeding from hemorrhage into the guttural pouch. From carotid or maxillary arteries with guttural pouch mycosis or associated with rupture of the longus capitis muscle following trauma.
* Rupture of mesenteric arteries secondary to strongyle larval migration.
* Splenic hematoma
* Splenic rupture following blunt trauma
* Rupture of liver with hyper lipemia
* Hemangioma and hemangiosarcoma and other neoplasia
* Persistent bleeding from the vulva in association with ulcerated vericase veins on the dorsal wall of vagina.

**Pathogenesis**

The major effects of hemorrhage are loss of blood volume, loss of plasma protein, or loss of erythrocytes.

With acute and severe hemorrhage the rapid loss of blood volume results in peripheral circulatory failure, and loss of erythrocytes in anemic anoxia.

The combination of these two factors is often fatal.

With less severe hemorrhage, the normal compensatory mechanisms, including release of blood stored in the spleen and liver, and the with drawl of fluid from the tissue spaces.

May maintain a sufficient circulating blood volume, but the anemia is not relieved and the osmotic pressure of the blood is reduced by dilution of residual plasma protein.

The resulting anemia and edema are repaired with time provided the blood loss is halted.

**Clinical findings**

Pallor of the mucosae is the outstanding sign.

In addition, weakness, staggering, and recumbency, a rapid heart rate, cold extremities and subnormal temperature.

The respiration is deep, but is not dyspnic .there is listlessness and dullness. And in fatal cases the animals dies in a coma in lateral recumbency.

The animals that live longer may develop dependent edema.

**Clinical pathology**

Examination of the blood for hemoglobin and hematocrit levels and the erythrocyte count are of value in indicating the severity of the blood loss and provide an index to the progress of the disease.

Total protein and PCV fall to their lowest levels 12- 24 hours following the hemorrhage.

**Differential diagnosis**

There is usually a history of bleeding or evidence of it on physical examination.

Other forms of peripheral circulatory failure include shock and dehydration but they can usually be differentiated on history alone.

Aneamia due to other causes is not accompanied by signs of peripheral circulatory failure.

**Treatment**

 The source of the hemorrhage should be determined and the cause corrected

Blood or fluid supply

A decision for blood transfusion should not be made lightly as the procedure is time consuming, costly ad has some risk.

Fluids

Hypovolemic shock can be effectively treated by the prompt administration of fluid and the use of crystalloid and colloid solution.

Hypertonic saline solution is recommended in hemorrhagic shock.