THE AVIAN CARDIOVASCULAR SYSTEM

Slide Study Set # 25

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Cardiovascular disease is an important cause of death in commercial turkeys and meat-type chickens. Spontaneous turkey cardiomyopathy (STC; roundheart), ruptured aorta and sudden death account for over 50% of the "normal" mortality in tom turkeys. Flip-over (sudden death syndrome) and pulmonary hypertension, leading to right ventricular failure, cause high losses in broiler and roaster chickens. In both chickens and turkeys these conditions are related directly to growth rate. The diagnosis is usually based on history and gross examination.

Other cardiovascular diseases are rare in poultry, waterfowl, pet and wild birds, but occur occasionally because of infectious, nutritional, toxic or unknown insults. In these cases histology or other tests may be required for diagnosis.

NOTE: Reference #4 is the chapter on the cardiovascular system in the AAAP **Avian Histopathology** manual. Numerous figures from that chapter are cited (4: Fig.#) throughout this study set rather than being included as slides. You are encouraged to review the figures in the manual as a supplement to this slide study set.

Abbreviations Used

AV:	atrioventricular	RA:	right atrium
BW:	body weight	RV:	right ventricle
LV:	left ventricle	RVF:	right ventricular failure
PH:	pulmonary hypertension	STC:	spontaneous turkey cardiomyopathy
PHS:	pulmonary hypertension syndrome	TV:	total ventricle weight

Normal. The avian heart differs from the mammalian heart in that it is cone-shaped, has a thin right ventricle (RV) and thick left ventricle (LV) wall (1:4). A muscular flap rather than mammalian type valves separates the right atrium and ventricle (7). The muscle flap is a continuation of muscle from the right ventricle wall. Hypertrophy of the RV wall results in thickening of the valve and leads to valvular insufficiency.

Other valves are similar to the mammalian heart. A cartilaginous plaque is found in the wall of the aorta where the major vessels leave the heart (4: Fig. 5.1). Lymphoid foci and foci of extramedullary granulopoiesis are common in the myocardium of broilers (4: Fig. 5.2).

Birds have a renal portal system (7) and the kidney has no capsule. In turkeys, if there is sudden failure of forward flow through the kidney, blood returning from the legs may pool around the lobules and on the ventral surface of the kidney (2).

Necropsy of the heart. Examine the heart and vessels, serous membranes and coelomic cavities *in situ* for changes in size, shape, color, fluid, fibrin, and urate crystals. Dilation and congestion of the atria and veins, particularly the sinus venosus and vena cava, suggest right valvular insufficiency. Remove the heart and transect it at the midsection (at the level of the free edge of the right AV valve). Look for hypertrophy, dilation or other abnormalities. Open the heart with scissors by following the path of blood flow and examine the valves, endo-, epi- and myocardium. The heart can be separated into its various parts for heart, and heart-to-body weight ratios. The normal heart-to-body weight ratio in broilers decreases with age from 0.0082 at day 9 to 0.0047 at day 42 (heart 0.47% of BW at day 42). The normal right ventricle-to-total ventricle ratio is 0.20 ± 0.03 (RV 20% of TV).

Routine histologic examination is frequently limited to a mid section across both ventricles, but should also include a longitudinal section of the R-V valve and atria and two sections of the LV through the chordae tendineae; one from the free wall including the atria and one from the opposite side. Gross lesions in the heart, pericardium and vessels should always be examined.

Congenital defects. A variety of anomalies have been described in the cardiovascular system in poultry, but only interventricular and interatrial septal defects occur

with any frequency. Most congenital defects result in ascites and death from right ventricular failure (RVF) in the first 2-3 weeks (3,5,6).

RESPONSE TO VOLUME, PRESSURE AND INJURY

Circulatory problems or injury causing edema in the myocardium or under the epi- or endocardium will result in fibroplasia and fibrosis in those tissues, and if there is hydropericardium, in pericarditis as well (4: Figs. 5.2, 5.3, 5.4). Cardiac lipidosis is seen occasionally (4: Fig. 5.6).

Hypertrophic cardiomyopathy. Response to volume and pressure. The heart muscle responds to an increased workload as all muscle does, by hypertrophy. A volume increase, which occurs with increased oxygen requirement, valvular insufficiency, septal defects, anemia, sodium toxicity, etc., causes hypertrophy in which the heart enlarges because of increased chamber volume (eccentric hypertrophy). In broiler chickens, a volume overload may quickly lead to an increased pressure load on the RV because of the restricted space for blood flow in the lungs. In a volume overload the ventricular wall does not become thicker but the mass of the ventricle does increase. In this form of hypertrophy, sarcomeres are added in series and fibers do not become thicker.

A pressure increase occurs most frequently because of increased blood flow, but also because of increased resistance to flow as the result of constriction, stenosis or obstruction of arteries, arterioles or capillaries, or because of increased blood viscosity. Hypertrophy as the result of a pressure overload causes thickening of the ventricle wall. In pressure-induced hypertrophy of the LV (concentric hypertrophy) the chamber becomes smaller. Stroke volume may become so small that the heart rate increases to the point where the LV no longer has time to fill, and the heart is unable to supply the blood flow required by the body. Concentric hypertrophy may be the cause of sudden death syndrome in turkeys.

In the ascites syndrome, or pulmonary hypertension syndrome (PHS) in broilers, both a pressure and a volume overload are present in the right ventricle until moderate valvular insufficiency occurs; then the pressure is reduced, but the volume is increased. Ascites secondary to right AV valvular insufficiency resulting in increased portal pressure is the

most frequent cause of ascites in poultry. PHS causes ascites because of increased portal pressure following valvular insufficiency and RVF, secondary to pulmonary hypertension (PH). PH caused by increased blood flow or increased resistance to flow in the lung results in right ventricular hypertrophy (RVH) as measured by the RV: TV ratio, and hypertrophy of the right AV valve (3,6,10). (See AAAP slide set "Ascites Syndrome in Meat-Type Chickens")

Sodium intoxication results in PHS very rapidly in young chicks. The pathogenesis of the heart lesion in sodium toxicity in turkey poults is not clear and is said to be similar to DTC. It is likely a volume induced dilatory cardiomyopathy. In both chicks and poults, subcutaneous edema may occur, likely secondary to RVF. Additionally, testicular edema has been reported in chicks.

Degenerative changes. Dilatory cardiomyopathy describes a condition in which the ventricular chamber is enlarged and the ventricular wall is thinned. Because the heart is larger, ventricle mass is usually increased. Dilatory cardiomyopathy is a degenerative condition in which myocytes are lost because of hypoxic, inflammatory, autoimmune or other insults. As myocytes die the ventricle dilates and loses its ability to contract to empty completely on systole. This results in a volume overload. The heart responds as to a volume overload by adding sarcomeres in series. In young poults with dilatory cardiomyopathy, fibers may appear thin and long with many mitotic figures. Heart muscle mass increases as a % of body weight as the heart continues to dilate. The heart muscle becomes stiff with connective tissue and collagen. Consequently, it loses its ability to dilate and contract (both of which require muscle energy) and becomes ineffective, resulting in heart failure. Frequently the dilation results in valvular insufficiency, particularly of the right AV valve, causing increased portal pressure and ascites. Hydropericardium and fibrous epicarditis may be present. Dilatory cardiomyopathy is common in turkeys, but is rare in chickens, where it is seen occasionally in male breeders.

In both hypertrophic and dilatory cardiomyopathies interference with blood supply may result in myocardial degeneration, focal necrosis, edema, fibrosis, valvular insufficiency, decompensation, muscle atrophy and terminal heart failure. Degenerative changes and scarring result in cardiosclerosis, which is the stage that is most frequently

described in the literature. The histologic lesions are caused by anoxia, myocardial cell death, edema and fibrosis (4: Fig. 5.5, 5.7).

Endocardiosis. Nodules on the endocardium of chickens are common. They are most common on the chamber side of the right AV valve about 3 mm from the free edge and on the left AV valve. The nodules consist of loose connective tissue and amorphous ground material and are similar to myxoid degeneration of the valve in humans (4: Fig. 5.8). They have been associated with right heart failure but are probably stretch-induced and are the result of valvular insufficiency rather than the cause of the insufficiency. The incidence is increased in chickens with PHS.

Roundheart disease in chickens. The name roundheart has been used to describe distinct entities in chickens and turkeys. In turkeys it refers to STC. It has also been used to describe RVF, which is discussed under ascites.

In chickens, roundheart disease affects birds older than 4 months and is characterized by sudden death. The heart of affected chickens is pale and enlarged, with hypertrophy confined to the left ventricle. The etiology is unknown. The fibers throughout the myocardium are swollen and granular and contain fine vacuoles (4: Fig. 5.9). In severe lesions, the vacuoles may coalesce, producing an apparent empty space around the nucleus and leaving a prominent cell membrane around the periphery of the myocyte. The vacuoles and resulting empty spaces represent fat.

Other causes of cardiomyopathy. Excessive amounts of furazolidone in the diet will produce cardiac dilation and ascites in chickens, ducks and turkeys. Histologic lesions reported are similar to STC and are probably secondary to tissue hypoxia caused by dilation. Myocytolysis occurs as an early lesion prior to dilation in the myocardium of furazolidone-poisoned ducks (9) and STC (4: Fig. 5.10). This loss of myocytes results in dilatory cardiomyopathy.

Cardiomyopathy and ascites have been produced by feeding poultry high levels of rapeseed oil containing erucic acid. Erucic acid accumulates in the heart of birds, resulting in fatty degeneration of the myofibers, necrosis and fibrosis. The associated edema is

presumably the result of heart failure, but it may also be due to hypoproteinemia secondary to liver damage.

A cardiomyopathy has been associated with potassium deficiency causing sudden death in hens near point of lay (8). The hearts are enlarged with both dilation and hypertrophy as occurs in PHS.

Ascites and hydropericardium have been described as prominent features in the toxic fat syndrome (dioxin toxicity) and chlorinated biphenyl toxicoses. Carbolinium toxicity also causes hydropericardium.

In selenium/vitamin E-deficient birds, myopathy in skeletal, gizzard or intestinal muscle is generally more remarkable than that in the myocardium. However there may be heart lesions recognized as white streaks or patches and associated with hydropericardium. Metals (e.g., cobalt, lead), chemicals, ionophore toxicity (1), and poisonous plants (Cassia, Crotolaria etc.) may cause myocardial damage.

Flip-over; sudden (acute) death syndrome; dead in good condition. The term sudden death syndrome has been used to describe well-fleshed broiler chickens that die suddenly with food in their gastrointestinal tract. There are no diagnostic lesions, but generalized congestion of the lung and dilation of the atria with constriction of the ventricles are significant. Death has been attributed to "heart attack" but is probably the result of ventricular fibrillation (4,5).

Inflammation of the heart. Pericarditis is frequently found in birds that have generalized bacterial infection. In commercial poultry it is particularly common in *E. coli* infection secondary to respiratory disease. In early lesions there is fibrin and many heterophils are present; later lymphocytes and macrophages predominate and, if the chicken survives, fibrous tissue and adhesions result.

Non-septic epicarditis and pericarditis occurs in birds with hydropericardium (see slide #3 and Ref. 4: Figs. 5.2, 5.3, 5.4). In broilers and heavy turkeys, focal fibrinous epicarditis, occasionally with pericardial adhesion, is a frequent lesion near the tip on the anterioventral side of the LV where it is close to the sternum. This white area of fibrosis on the epicardium is made up of proliferating and mature fibroblasts with few inflammatory

cells (4: Fig. 5.14). It likely occurs secondary to localized subepicardial edema as the result of trauma when the beating heart contacts the sternum.

Visceral urate deposits (visceral gout) on serous membranes may be confused with inflammatory exudate. They are most prominent on the heart and kidney. They also occur in the tissue causing microscopic focal areas of necrosis. Urate crystals are also visible in the lesion.

Myocarditis accompanies many viral and bacterial infections of birds. Focal necrosis of myofibers and infiltration of mononuclear cells occur when chickens are infected with virulent strains of Newcastle disease and influenza viruses (4: Fig. 5.15). Similar microscopic lesions associated with intranuclear inclusion bodies have been described as a prominent feature of Derzsy's disease in goslings. Myocarditis is a prominent feature of the hydropericardium syndrome caused by adenovirus infection in broilers in Pakistan and other countries. There are also isolated reports of myocarditis caused by adenovirus, parvovirus, avian leukosis virus, and other viral agents.

Granulomatous myocarditis occurs in chickens with *Staphylococcus* sp. and *Salmonella pullorum* infection (4: Figs. 5.17, 5.18) and in various birds infected with miscellaneous protozoan species. Fungal and tubercular granulomas also occur. Although the gross appearance of granulomas and granulomatous inflammation in the heart may resemble neoplasia (see slide #27), the microscopic appearance is usually quite diagnostic.

Endocarditis is usually seen in individual birds but may occur as a flock problem. It is most frequently caused by *Streptococcus* spp. but it may also be due to *Staphylococcus* spp., *Pasteurella* spp., *Mannheimia* spp., *Erysipelothrix rhusiopathiae* or other bacteria. The lesions occur most commonly on the left AV and aortic valves. Endocarditis is commonly associated with infarcts in liver, spleen, heart, and brain, or with ascites if on the right AV valve.

PATHOLOGY OF THE BLOOD VESSELS

Arteriosclerosis. Arteriosclerosis is common in many different species. The early lesions consist mainly of collagen and connective tissue. In older birds foamy cells, extracellular lipid, cholesterol and calcium become incorporated into the plaques, which then

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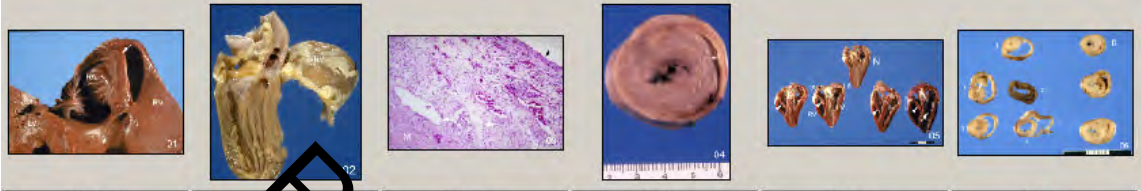

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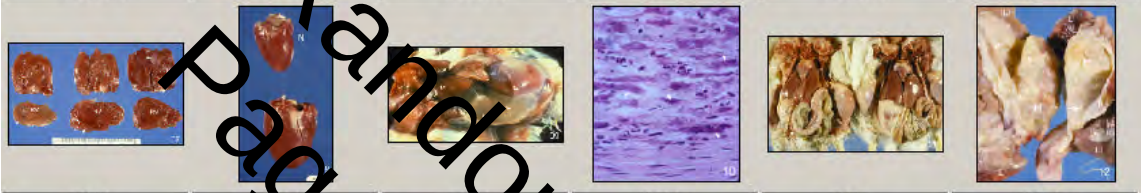

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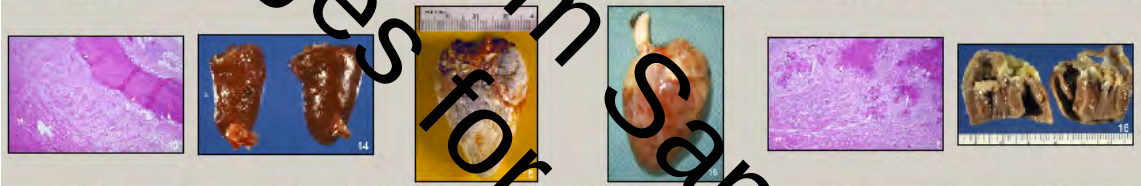

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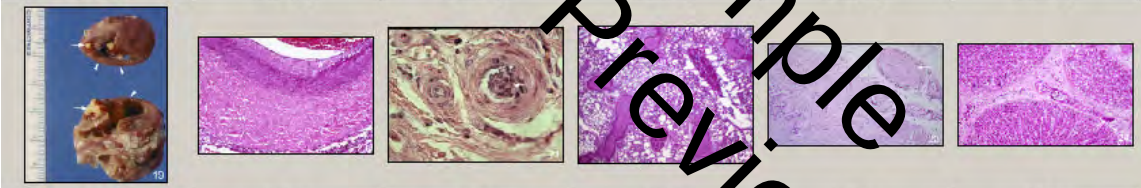

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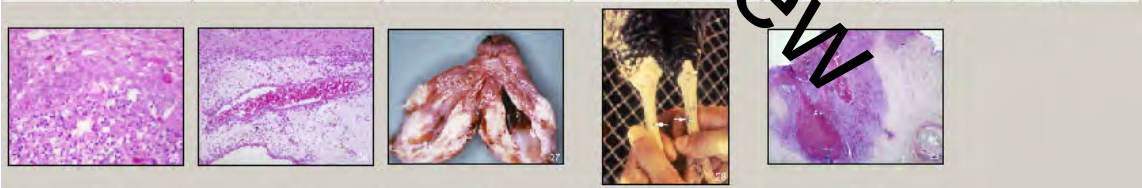

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