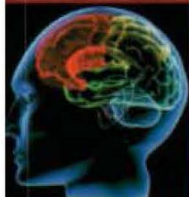
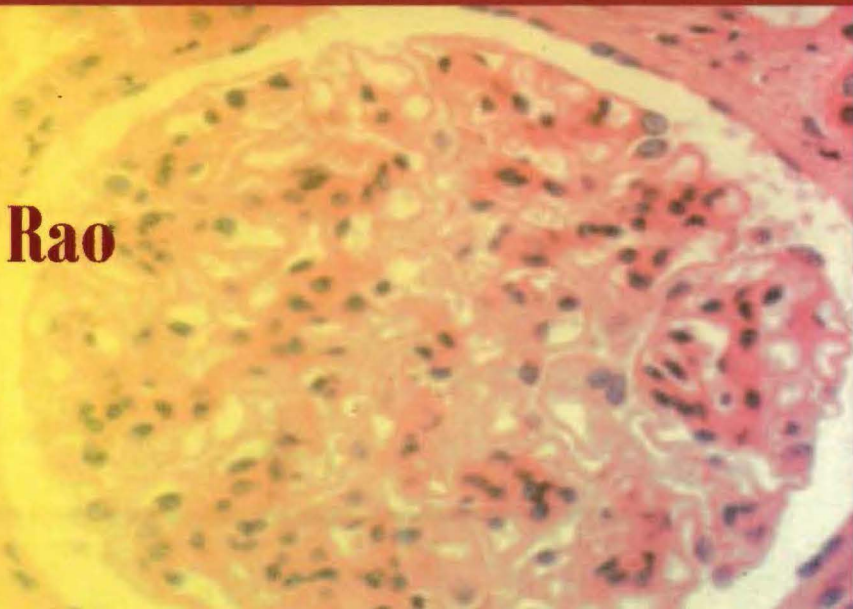


**A Text Book on
Systemic Pathology
of Domestic Animals**



D. Gopalakrishna Rao



A Text Book on Systemic Pathology of Domestic Animals

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&

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Preface

It gives me great pleasure in writing this text book on systemic pathology of domestic animals for veterinarians. Many observations done by me regarding the pathogenesis of lesions occur in different organs of domestic animals with variety of diseases have been incorporated in this book.

This book covers the syllabus outlined by Veterinary Council of India, 1994 and 2008 regulations for II year B.V.Sc. & A.H. students. Constructive criticism is welcome about the style of book and about lacunae in describing the pathogenesis of lesions in different diseases of animals.

D. Gopala Krishna Rao,
Retired Professor and Head
Department of Veterinary Pathology

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Diseases of Cardiovascular System

Summary

Postmortem examination. Heart failure causes congenital anomalies of heart- fetal circulation- diseases of pericardium, endocardium, myocardium Endothelial cell properties and functions: Elaboration of prothrombotic molecules: Modulation of blood flow and vascular reactivity: Regulation of cell growth: Growth stimulators: Growth inhibitors: Pericardium: Hydro pericardium: vegetative endocarditis- Atherosclerosis hypertension. Aneurysms—vasculitis: Causes of vasculitis: Infections-Rickettsial infections: Mycotic infections. Non-immune mediated infections. Diseases of veins— Sundry disease conditions of veins: Varicose veins, Diseases of lymph vessels: Lymph-Lymphangitis-lymphadenitis: Tumors; Hemangiosarcomas-Lymphangiosarcoma

Postmortem examination

The aim of a gross postmortem examination of the heart is to examine four major areas, the pericardium, the myocardium, the mural and valvular endocardium and the great vessels. It is preferable to commence by examining the heart and blood vessels in situ for abnormalities of size and position. The pericardial sac should also be incised and its contents examined before the thoracic contents are removed. Once removed, the external surface of the heart should be examined, including the pericardium, epicardium and major vessels.

The right ventricle bears responsibility for systemic circulation in the fetus, and in neonatal hearts, Thickness of the wall left and right chambers is about equal; it is not until several months after birth that the mature proportions are attained.

Rigor mortis begins rather earlier in myocardial than in skeletal musculature and reaches its greater development in the powerful left ventricle. Rigor should completely express the blood from the left ventricle; rigor of the right ventricle is less efficient and emptying it's incomplete. The presence of some clotted blood in the right ventricle is normal, whereas if present in the left ventricle after a reasonable postmortem interval, it is indicative of incomplete rigor and therefore of severe myocardial degeneration. Unclothed blood in ventricle is an indication of death due to hypoxia.

Blood usually clots slowly after death and permits RBCs to sediment, where blood is present in volume as in the heart and arterial trunk, this process of sedimentation and subsequent clotting leads to the formation of currant jelly and

chicken fat clots. Currant jelly clot is rich in RBCs hence red in color; chicken fat clot contains WBCs platelets and less RBCs. Chicken fat clot indicates slow death of animal; currant jelly clot indicates rapid death of animal. Chicken fat clot are to be expected in horses, which have rapid ESR. These postmortem clots are to be distinguished from thrombi; postmortem clot if one pulls from the vessels comes away easily, and are not attached to the endocardium or endothelium of blood vessels.

Pericardium: The pericardium is a thin layer that covers the heart. It is double layered pleura. Outer coat is the pericardium and the inner sac completely encircles the pericardium. In between outer and inner coats a potential space with fluid exists that is called pericardial sac. Primary disease confined to the pericardium is rare, but the loose anatomic relationship of the pericardium to the heart, lungs, and pleura sometimes results in the extension of disease processes from the latter organs to the pericardium. The pericardial cavity may communicate with the peritoneal cavity through clefts in diaphragm; the pericardial sac may be absent congenitally without clinical effect. The function of pericardium is to provide automatic compensation and ensures the end diastole trans-mural pressure is the same for all hydrostatic levels of the ventricles. The role of epicardium includes prevention of sudden cardiac dilatation, maintenance of flow of intramural pressures; limitation of right ventricular stroke work, hydrostatic compensation for gravitational or inertia forces; and maintenance of cardiac alignment and streamlined intra-cardiac flow. The heart is attached to the sternum by sterno-pericardial attachment. In that the movements of the heart will be limited to the physiological levels.

Hydro pericardium: The pericardial sac normally contains a very small quantity of clear, serous fluid. Any excess in the volume of fluid is referred to as hydro pericardium. The pericardial fluid is clear or light yellow without floccules, and with low content of protein. Sero-sanguinous fluid is often observed in variety of toxemic and septicaemic diseases. Inflammatory exudates can be differentiated from transudates on the basis of the higher content of protein and cells in exudates and histological evidence of inflammation involving the pericardium and epicardium. Hydro pericardium is often part of generalized anasarca and is seen in most of the cachetic diseases and congestive heart failure cases.

Myocardial necrosis: Saccharated iron compounds by virtue of the capacity of iron to generate free radicals, in ferric\ferrous translations causes' fatal myocardial necrosis in piglets. Ruminants of early age that is before the rumen is developed and pigs are susceptible to the cardio toxic effects of cotton seed poisoning and the active principles of gossypol. Monensin, ionophore coccidio-stat is toxic in horses and pigs. Myocardial necrosis occurs in dogs ingesting rodenticides that contain thallium. Anichkov cells also known as caterpillar cells have been observed in myocarditis. These cells appear as large mononuclear cells in which the nuclear chromatin is present in and undulating wavy ribbon with slender processes radiating from it. The origin of these cells is disputed. Suggestions include a fibroblastic, pericyte, endothelial, or myocytic origin.

Cardiovascular Pathology

Four sounds of heart are audible in different diseases. First sound ventricular systole occurs -probably due to the closure of auricular-ventricular valves. Second sound is due to the closure of semi lunar valves of aorta and starts with diastole. Third sound is used by rapid filling of ventricle and just occurs after the second sound. Fourth sound is caused by atrial systole and occurs just prior to the standard first heart sound.

The functions of the heart are to maintain sufficient supply of blood to meet the needs of the body tissues. For this, the heart should have sufficient cardiac reserve. Cardiac reserve is defined as the ability of the heart to meet increasing demands that are required under stress.

There are usually three catchwords that are required while reading the heart, viz., cardiac reserve, compensation, decompensation and heart failure. The term heart failure denotes a situation in which the heart is diseased, all compensatory mechanisms have been exhausted and characteristic clinical and pathological signs are exhibited.

The function of the heart is to maintain sufficient supply of blood to meet the needs of body tissues. The cardiac reserve represents the ability of the heart to meet increased demands that arose during physiological conditions like exercise, pregnancy and pathological conditions like anemia, fever etc. The heart is able to adapt itself to varying physiological needs and to pathological abnormalities. This ability is known as compensation. Compensation mechanisms include the cardiac response, namely, dilatation and hypertrophy. Consequent to this, there is systemic response, which includes an increase in heart rate, peripheral resistance, a redistribution of blood flow, vascular contraction and an increase in blood volume. In each case, the compensatory mechanisms are at least temporarily beneficial and are directed toward increasing cardiac output to meet the metabolic needs of the animal.

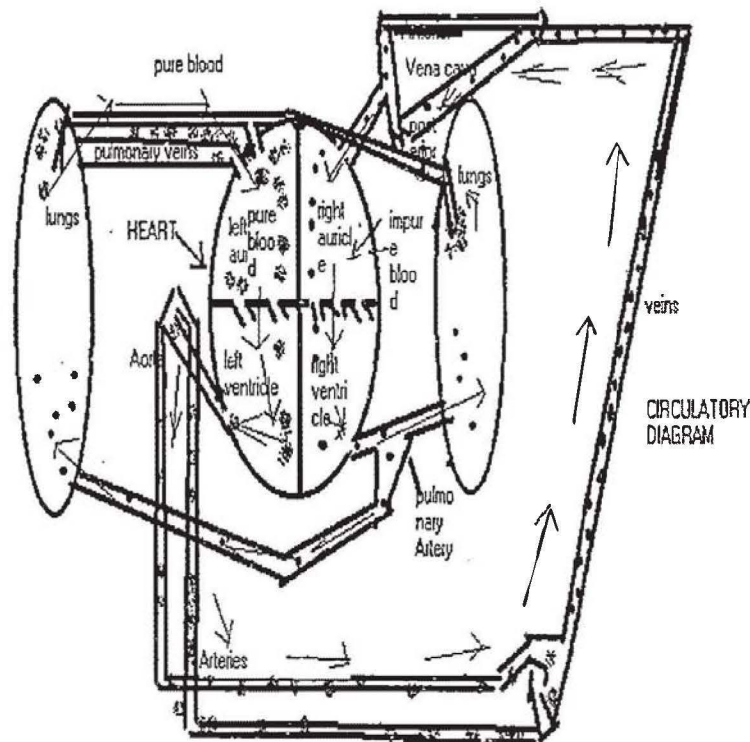
However, in the face of an ever-increasing peripheral resistance together with the increased workload put on the heart. The heart is not able to cope up with the demands and so become fatigued and fails. This state in which the heart is no longer able to compensate is called decompensation. Decompensation is gradual and results in dilatation of the ventricle.

The heart failure stems from two basic pathophysiologic changes, the accumulation of fluid, and tissue or organ ischemia. Fluid accumulation results from the retention of sodium and water. One of the earliest changes following a drop in cardiac output is the redistribution of blood flow within the kidney. The alterations in renal blood flow in heart failure also increase the activity of the renin-angiotensin-aldosterone system, producing more sodium reabsorption from the distal convoluted tubules. There is an increased activity of antidiuretic hormone also.

The fundamental differences between shock and congestive heart failure are that the total blood volume in heart failure is already more than adequate, but the effective blood volume is much diminished because of the poor cardiac output. The factors that lead to cardiac failure are three fold.

1. Alteration in the return of venous blood.
2. Increased resistance to outflow.
3. Impaired cardiac contraction.

Circulatory diagram in an adult mammalian system



If the venous return is inadequate, the heart is not capable of compensation to meet the situation and finally fails. Increased resistance results due to hypertension, narrowing or dilatation of valvular orifices, thrombosis and arteriosclerosis. The heart compensates for the increased resistance to outflow. This is evidence in the form of hypertrophy of heart musculature. However, in time the reserve power of the heart is exhausted and so fails. Any disease that injures the myocardium reduces the contractile power of the heart and so the compensatory mechanism cannot work.

The circulatory failure falls into three general types' namely cardiac syncope, peripheral circulatory failure and congestive heart failure.

The heart is usually attached to the sternum by the sternopericardial attachment. Any heart with pericardium is to be detached first means; the sternopericardial attachment is to be cut and to be separated. Primary diseases confined to the pericardium are rare, but the close anatomic relationship of the

pericardium to heart, lungs and pleura sometimes results in the extension of disease processes from the latter organ to the pericardium. The pericardium is filled with little fluid. The role of pericardium includes prevention of sudden cardiac dilatation, maintenance of low transmural pressure, limitation of light ventricular stroke work, hydrostatic compensation of gravitational forces, maintenance of cardiac alignments and streamlined intracardiac flow.

Three golden rules obtained for the heart necropsy are 1) always weight the heart after removing the post-mortem clots, 2) always inspects before cutting any structure 3) try to preserve any stenotic valve or vessel.

A few special points which may be noted on external examination while doing autopsy suspected for a case of cardiac failure are 1) the extent of any edema as elicited by palpation, 2) any jaundice of skin or conjunctiva any cyanosis and digital clubbing in humans, 4) and any congenital anomalies such as accessory nipple.

Embryology of the heart

Adult heart is a modified blood vessel. Its tunica media is primarily cardiac muscle. The primordial of heart first appears as clusters of angioblast cells between the mesoderm of common-pleural-pericardial cavity and the underlying mesoderm. These primordial referred to as cardiogenic mesoderm. The first change involves the formation of bulbo-ventricular loop. It is formed in that the part of the endothelial tube that comprises the bulbous and the primitive ventricle. While the looping occurs, there is localised expansion of the bulbous and ventricular areas by means of diverticulation. The external sulcus demarcating the constriction of bulbo-ventricular loop is referred as inter-ventricular sulcus. After S-shaped loop is formed, the heart undergoes further change in the position. The bulbous shifts to a more ventral and caudal portion while the atrium and sinus venous move cranial. Internal partitioning occurs.

Foetal circulation

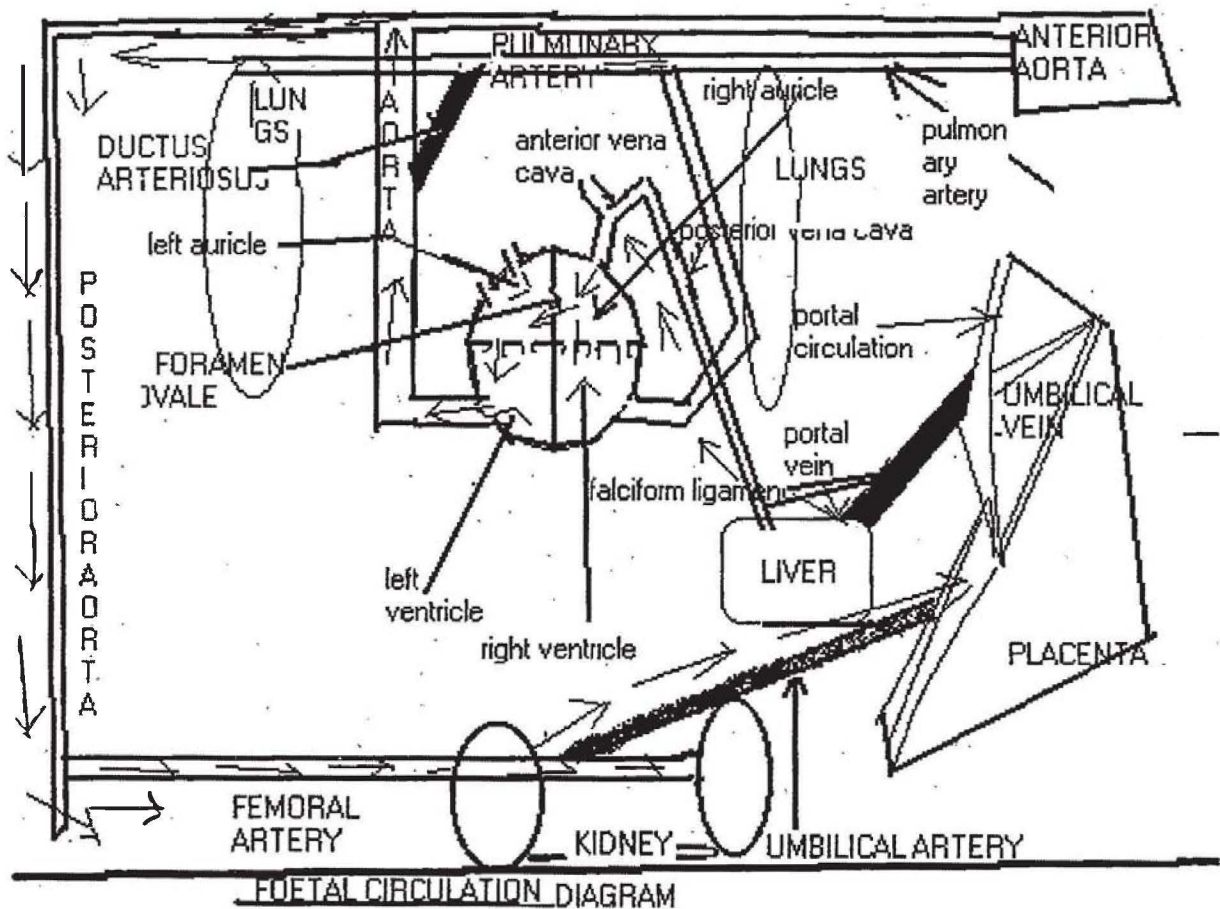
Before discussing the anomalies of heart, it is better to differentiate the changes that occur in foetal circulation and the disappearance of different structures and functions in the adult animal or the prenatal animal.

In the foetus, lungs are not functional. The foetus is a parasite staying in the womb. It is not excreting its secretions to the outside. The mother takes care of the excretions, nutrition and oxygenation of tissues, through the placenta. Placenta is a structure that develops between developing foetus and the uterus of the mother.

Thus, the umbilical arteries of the foetus, the right and left are large vessels, which arise from the internal iliac arteries and pass downward and forward in the umbilical folds of peritoneum on either side of bladder to umbilicus. Here

they are incorporated with the umbilical vein and the urachus in the umbilical cord, ramify in the allantois and end as the capillaries of the foetal placenta. They conduct the impure blood to the placenta. After birth, they are retracted and termed as round ligaments of the bladder.

Umbilical veins, receive the oxygenated blood from the placenta. These enter the falciform ligament of the liver and joins in the portal circulation, so that the blood passes through the capillaries of the liver before entering the posterior vena cava.



Ductus venosus is the vessel that is given off within the liver from a venous sinus formed by the confluence of portal and umbilical veins and passes directly to the posterior vena cava. Posterior vena cava and anterior vena cava open into the right auricle.

Foramen ovale is a septum that lies between the left auricle and right auricle. This is patent or in open condition is the foetal life. In the fully matured and born foetus, the foramen ovale is closed and its place there is a deep fossa ovalis in the right artial side.

The right auricle thus contains both pure blood that comes from posterior vena cava and impure blood that comes from the anterior venacava. Thus, right auricle contains mixed blood (99% pure blood). The pure blood from right auricle enters into the right ventricle and is pumped into the arterial system through pulmonary arteries, by passing lungs for oxygenation. The lungs are devoid of oxygen at this stage, as the foetus is in utero and is sealed from the external environment. Thus, nature has developed a system the pure blood that is in the pulmonary artery is connected to the aorta through a duct known as ductus arteriosus. When the foetus starts breathing, the duct is being closed and forms as ligamentum arteriosum.

The impure blood that comes from right ventricle may have access to the aorta as pulmonary artery is connected with aorta through ductus arteriosus. The heart develops from a S-shaped tube, with various bends. The adult aorta arises out of five pairs of left and right aortic arches, which are present at the beginning. The final aorta arises from the left ventricle. The foetal heart can function without oxygen for longer periods than adult heart because myocardial cells contain relatively large amounts of glycogen throughout-gestation.

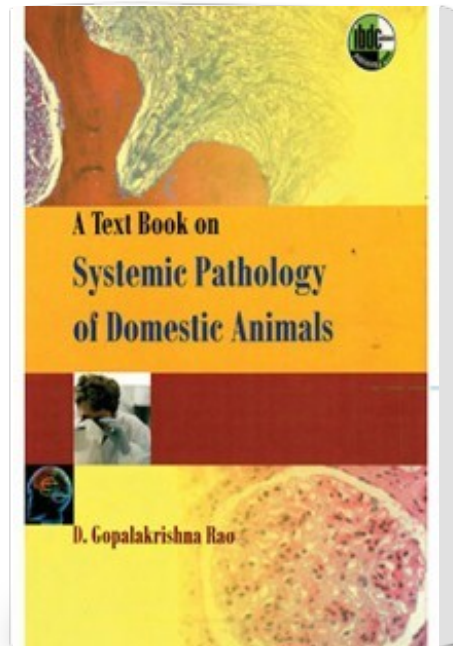
Developmental anomalies

These following conditions are recorded in animals and human. Patency of foramen ovale, persistent interventricular foramina, patent ductus arteriosus, persistence of the right aortic arch, coarctation of the aorta, transposition of the aorta, congenital aneurysm of the aorta or pulmonary artery, failure of adequate development of semi lunar valves, valvular stenosis, dysplasia of the tricuspid valve, mitral valvular insufficiency, congenital haematomas, tetralogy of Fallot and endocardial fibroelastosis.

Patency of foramen ovale: Very soon after birth, the foramen ovale, a communication between the right and left auricle, will be closed and the venous blood will be coming into the right ventricle and will be diverted to the pulmonary circulation.

The foramen if it is persisting in the life is called persistent foramen ovale, resulting in mixing of arterial blood and venous blood. Due to circulation of venous blood to arterial side, the efficiency of the individual or animal will be decreased and is exhibited by tiredness and other symptoms of fatigue. In humans, the skin appears to be blue in colour due to the circulation of venous blood to the arterial side, resulting in blue baby condition. This is also known as atrial septal defect. This is compatible with life. Atrial septal defects are tolerated well if they are less than 1 cm in diameter. Even larger defects do not constitute serious problems during the first years of life, when the low-pressure flow is from left to right. Persistency in the adult life creates overload in the right auricle, leading to dilatation and failure of heart in due course.

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