CONGENITAL CARDIOVACULAR ANOMALIES

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Formation of the heart is complete for the most part in the first trimester. Causative agents of defects can only act during this time. The cause of most defects is generally unknown. Fetal hypoxia, vitamin A deficiency, viral infections, and heredity may play a role in some cases. Congenital heart defects are the most common cause of clinical heart disease in young animals. Clinically they may be characterized by cardiac murmurs because of abnormal flow patterns, congestive heart failure, polycythemia (an increase in the number of red blood cells secondary to tissue hypoxia), and / or cyanosis (purple or blue discoloration of mucous membranes due to reduced hemoglobin in capillaries). Cyanosis is common in right to left shunts, that is when venous blood in the right heart or pulmonary trunk passes directly into the systemic circulation and bypasses the lungs. In the transition from fetal to neonatal life, substantial adjustments occur within the cardiovascular system.

The etiologies of congenital cardiovascular anomalies are varied. Most animal species have a low background frequency of spontaneous cardiac malformations. In many species, especially in dogs, these defects are heritable and may be attributed to either single or multiple gene effects. Under experimental conditions, at least, cardiovascular congenital defects may be elicited by exposure of pregnant dams to various chemicals and drugs, physical agents, or nutritional deficiencies and toxicities. Implicated chemical compounds include thalidomide, ethanol, salicylates, griseofulvin, and cortisone. Prenatal exposure to x-irradiation or fetal hypoxia may induce defects. Maternal nutritional deficiencies of vitamin A, riboflavin, or zinc and feeding excesses of vitamin A, or copper may result in newborn with cardiovascular anomalies.

The most common congenital heart defects may be classified in the following categories: (1) failure of closure of fetal or neonatal arteriovenous communication, (2) conotruncal abnormalities, (3) failure of adequate development of the semilunar or atrioventricular valves, (4) incomplete separation of, or abnormally positioned vessels, and (5) miscellaneous cardiac anomalies.

1) Failure of closure of fetal or neonatal arteriovenous communication

In the development of the heart there are three major arteriovenous communications: between the atria, the ventricles and the great vessels. Failure of closure results in several possible defects patent ductus arteriosus (PDA), atrial septal defect (ASD) and ventricular septal defect (VSD).

PDA – failure of the ductus arteriosis to close after birth. The ductus develops from the sixth left branchial arch, and functions in the fetus to divert a major portion of blood from the pulmonary artery to the aorta. In PDA, pulmonary hypertension occurs, with consequent shunting from right to left with right and left ventricular hypertrophy and cyanosis.

ASD – failure of closure of the interatrial communication. The atrial septal defect may result from two phenomena, failure of growth of the septum

secundum and defects of the septum between the right upper pulmonary veins and the cranial vena cava. The consequence is excessive flow from the left to the right atrium resulting in a volume overload on the right ventrical and elevated central venous pressure. In some cases following the development of pulmonary hypertension the flow through the defect is reversed leading to cyanosis.

VSD – insufficient closure of the interventriclar septum. This is one of the most common defects in domestic animals. The defect is usually single and may be small and without functional significance, or quite large. Because of higher pressure in the left ventricle the shunt is usually left to right causing a systolic murmur. Large defects produce severe cardiac dysfunction and death is due to left heart failure or chronic heart failure. The left and right ventricles are dilated and hypertrophied.

2) Conotruncal abnormalities

Tetrology of Fallot – a rare defect that is a combination of 4 components including VSD, PS, transposition (overriding aorta) and right ventricular hypertrophy.

3) Failure of adequate development of semilunar or atrioventricular valves.

Pulmonary stenosis – most common in dogs. The stenosis may be valvular, supravalvular or subvalvular. It is associated with right ventricular hypertrophy and often with post stenotic dilatation of the pulmonary trunk.

Aortic or subaortic stenosis – most common in dogs. Caused by a ring of fibrous tissue in the outflow tract of the left ventricle below the aortic valve. As a result there is a left ventricular hypertrophy and post stenotic dilatation.

Dysplasia or aplasia of the tricuspid valve – one of the more common defects observed in cats. The anatomic characteristics of tricuspid dysplasia are focal or diffuse thickening of the leaflets, some of which may be absent, or short chordae tendineae and papillary muscles, and direct fusion of portions of the affected valve with the ventricular wall. This defect of the valve may lead to right atrial enlargement and right ventricular hypertrophy.

Mitral valvular insufficiency – the most common congenital heart anomaly in cats. Anatomically there is an enlarged annulus, short thick leaflets, short thickened chordae tendineae, atrophic or hypertrophic papillary muscle, and enlargement of the left atrium and left ventricle with diffuse endocardial fibrosis.

Congenital hematomas (hematocyst) – common in calves. This lesion is on the margins of the atrioventricular valves These are blood filled cysts lined by an endothelial membrane that originate in the clefts normally present in the substance of the valves in intrauterine life. These cysts, which may measure up to 1.0 cm in diameter do not usually persist for more than a few months.

4) Incomplete separation or abnormal position of vessels

The most important defects in this group are persistent right aortic arch, malposition or transposition of the great vessels and persistent truncus arteriosus.

Persistent right aortic arch – is the small blood vessel (ductus arteriosus or ligamentum arteriosum), "vascular ring" passing from the aorta to the pulmonary artery that encloses the esophagus and compresses it against the trachea. This occurs when the adult aorta is derived from the right rather than the left fourth aortic arch.

Transposition of the great vessels - the aorta exits the right heart and the pulmonary artery the left heart. Other conditions may be present such as an overriding aorta, an overriding pulmonary artery, or partial transposition in which case both vessels leave the right ventricle.

5) Miscellaneous cardiac anomalies

Ectopia cordis
Two hearts
Pericardial anomalies
Endocardial fibroelastosis

PATHOLOGY OF THE HEART MUSCLE - outline

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The heart is formed of the:

Pericardium
Endocardium
Myocardium
Epicardium

1) Pericardium

A. Noninflammatory lesions
- hydropericardium
- hemopericardium
- serous atrophy of pericardial fat

B. Inflammatory lesions = pericarditis
- fibrinous pericarditis
- purulent pericarditis

2) Endocardium

A. Degenerative lesions
- subendocardial fibrosis
- subendocardial mineralization
- endocardiosis in dogs

B. Inflammatory lesions = endocarditis
- valvular endocarditis
- mural endocarditis

3) Myocardium

Myocardial degeneration
- hydrophic degeneration
- fatty degeneration
- atrophy of the heart
- mineralization in the myocardium

Myocardial necrosis
- focal myocardial necrosis / ischemic

- nutritional – vitamin E / Se deficiency
- feed additives - monensin, doxycycline
- cardiotoxic plants

Myocarditis

- bacterial
- viral
- parasitic

4) Cardiomyopathies

- hypertrophic
- dilated
- restrictive

5) Hemorrhage of the heart and its membranes

6) Diseases of the conduction system

7) Neoplasms of the heart

Epicardium ???????

PATHOLOGY OF THE RESPIRATORY SYSTEM - outline

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Functional anatomy

Nasal cavity and turbinates
Paranasal sinuses
Nasopharynx
Larynx
Trachea
Bronchi
Bronchioles
Pulmonary acinus

Pulmonary functions

Particle removal system
Gas exchange system

Defense mechanism

Mucociliary apparatus
Alveolar macrophages
Cough and sneeze reflex
Alveolar clearance by inactivation or mechanical clearance

Mechanisms of lung infection

Airborne
Hematogenous
Direct invasion

Pathological aspects of the nasal cavity, turbinates sinuses and larinx

Epistaxis
Rhinitis
Guttural pouch mycosis
Necrotizing laryngitis
Laryngeal hemiplegia

Pathological aspects of the trachea and bronchi

Congenital malformation of the trachea
Tracheitis
Bronchitis
Parasitic bronchitis
Bronchiectasis
Bronchiolitis

Pathological aspects of the lung

A. Congenital malformation
   Lung hypoplasia
B. Atelectasis
   Neonatorum
   Pulmonary collapse
C. Pulmonary edema and hyaline membranes
D. Emphysema
   Interstitial emphysema
   Destructive emphysema
E. Pneumonia

1. Classification according to gross distribution
   Bronchopneumonia
   Hematogenous pneumonia
   Lobar pneumonia
   Lobular pneumonia
   Diffuse, focal or multifocal pneumonia

2. Classification according to histologic criteria
   Interstitial pneumonia
   Alveolar pneumonia
   Bronchopneumonia
      Stage of congestion
      Stage of red hepatization (consolidation)
      Stage of gray hepatization (consolidation)
   Cuffing pneumonia
   Epithelializing pneumonia
   Granulomatous pneumomia
   Proliferative pneumonia
   Lymphoid interstitial pneumonia

3. Special types of pneumonia
   Aspiration pneumonias
   Embolic pneumonia
   Pulmonary abscesses
   Hypostatic pneumonia
   Dietary pneumonia (bovines)

Diseases of the pleura

Hydrothorax
Chylothorax
Hemothorax
Pneumothorax
Diaphragmatic hernia
Pyothorax
Pleuritis

Neoplasms of the nasal cavity

Adenocarcinoma
Chondrosarcoma
Osteosarcoma
Fibrosarcoma

Neoplasms of the lung

Bronchogenic carcinoma
Adenocarcinoma (pulmonary adenomatosis in sheep)

Neoplasms of the pleura

Mesothelioma
Metastatic thyroid tumor
Implant of thymic tumors

Suggested reading

1. Thomson's Special Veterinary Pathology – second edition
Edited by: William W. Carlton and M. Donald McGavin
Respiratory system – chapter 3, pp. 116-174.

2. Pathology of domestic animals – fourth edition, volume2
Edited by: K.V.F. Jubb, Peter C. Kennedy and Nigel Palmer
The respiratory system – chapter 6, pp. 539-698.

PATHOLOGY OF BLOOD VESSELS – Outline

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1. General remarks

Vascular system is subdivided into:

Arterial system

Capillary system

Venous system

Lymphatic system

Features of the vascular system:
histological structures of arteries, veins, capillaries

Post mortem changes: imbibition of hemoglobin, post mortem clots, post mortem contractions

2. Arterial diseases

Arterial aneurism and rupture

Arterial hypertrophy

Arteriosclerosis

Atherosclerosis

Arterial medial calcification

Hyaline degeneration, fibrinoid necrosis and amyloidosis

Thrombosis and embolism

Arterial inflammations: viral, bacterial, mycotic, parasitic, immune related

Neoplastic diseases:
- hemangiomas
- hemangiosarcomas
- hemangiopericytoma

3. Venous diseases

a. Congenital anomalies

- portocaval shunts

b. Dilations

- varices or phlebectasia

c. Inflammations

- phlebitis
- omphalophlebitis

4. Lymph vessel diseases

a. Hereditary lymphedema

b. Dilatation and rupture

- lymphangiectasis
- rupture of thoracic duct

c. Inflammation

- lymphangitis (bacterial, mycotic, parasitic)

5. Neoplastic diseases

- lymphangioma
- lymphangiosarcoma
- metastasis of carcinomas to the lymphatics