Congenital cardiac defects

The defects often produce clinical signs at birth and cause severe illness or death in the first week of life but in a number of cases adequate compensation occurs and the defect may be not observed until a comparatively late age.

The important factor in the pathogenesis of most congenital cardiac defects is :

1- The mixing of oxygenated and reduced blood through an anastomosis between the pulmonary and systemic circuits.
2- The blood leaving the left ventricle or oarta therefore includes some oxygenated and some reduced blood.
3- The resulting anoxic anoxia causes severe dyspnea and cyanosis may be marked if the proportion of unsaturated blood is high. This in most likely to occur when there is obstruction of the pulmonary artery.

In animals which survives to maturity, sudden death due to acute heart failure or congestive heart failure are likely to occur when the animals are subjected to a physical stress such as the first pregnancy.

The more common defects are set out below.

1- Ectopia cardis : An abnormal position of the heart outside the thoracic cavity is most common in cattle. Affected animals may survive for period of years but those with a displacement through a defective sternum or ribs rarely survive for more than a few days.

2- Patent interventricular septum (subaortal septal defect) : This defects occur in all species and may be manifested by clinical sings in the first few days of life or by failure to grow well when young or permit an apparently normal existence until maturity. There is a loud, blowing murmur audible over the base of the heart on both sides of the chest.

3- Patent foramen ovale: This defect of the atrial septum usually causes on clinical sings and is detected only at necropsy.

4- Patent ductus arteriosus : The clinical signs are comparable to those of insufficiency of the aortic semilunar valves.

5- Coarctation of the aorta : Constriction of the aorta at the site of entrance of the ductus arteriosus causes a syndrome similar to that of stenosis of the aortic semilunar valve . There is a systolic murmur and a slow – rising pulse of small amplitude.

6- Persistence of the right aortic arch : Persistence of the right aortic arch causes construction of the esophagus, dysplagia and regurgitation. Clinical signs are evident soon after birth but survival until five years of age has been recorded. In a bull which manifested chronic bloat and visible esophageal dilatation.

7- Fibro – elastosis : This anomaly has been observed in calves and pigs. The endocardium is converted into a thick fibro elastic coat, and although the wall of the left ventricle is hypertrophied the capacity of the ventricle is reduced. The aortic valves may be thickened and irregular and obviously stenosed. A similar condition occur in lumens but the cause in all

species in unknown. The syndrome is one congestive heart failure but there are no signs which indicates the pressure of specific lesions of the myocardium,endocardium or pericardium.

8- Subvulvular aortic stenosis: Stenosis of the aorta at or just below the point of attachment of the aortic semilunar valves has been recorded as common defect in pigs.

a- It's differentiation from other causes of heart of heart failure is difficult and the significant of the stenosis in open to doubt.

b- Clinically affected animals may die suddenly with asphyxia, dyspnea, and foaming at the mouth and nostrils, or after a long period of ill with recurrent attacks of dyspnea .

c- In acute form death may occur after exercise.

9- Anomalus origin of carotid arteries: Either or both carotid arteries may originates form the pulmonary artery instead of the aorta. The resulting anoxia causes myocardial weakness in the ventricle of the affected side, congestive heart failure usually follows. The defect has been recoded in cattle and dogs.