Tetralogy of Fallot is a rare condition of the heart which can be a serious, life-threatening anatomical malformation of the heart, appearing as a congenital defect and in some cases considered to be a genetic defect. To my knowledge it has been reported in the Keeshond and Bulldog breeds but there may be others that have an experience of the condition.

As the name implies, Tetralogy of Fallot (pronounced fallow) has four components and each is a malformation of the normal heart structure. For the sake of interest, what follows is a description of the defects, hopefully not too technical!

The first is where both main chambers of the heart are connected, so the left and right ventricles are not divided by an intact septum as they normally would be (defect 1 – ventricular septal defect).

In the normal heart, the outflow from the right side of the heart (pulmonary artery) should go to the lungs, to allow blood to dump carbon dioxide and pick up oxygen. Whereas the outflow from the left side (the aorta) contains blood destined for the head and body, having replenished its oxygen supplies in the lungs. In this condition, the aorta is situated more to the right hand side of the heart (Defect 2 – dextrapositioning of the aorta)

so that blood pumping from both ventricles passes easily into the aorta instead of the pulmonary artery. Furthermore, blood intended for the lungs is restricted because the pulmonary outlet is narrowed (Defect 3 – pulmonary stenosis). So the net result is less blood passes to the lungs than normal and output to the body is a mixture of blood full of Carbon Dioxide from the right side of the heart, with oxygenated blood returning to the heart from the lungs.

The final defect is an increase in muscle thickness in the wall of the right ventricle to compensate for the fact that it is this ventricle, not the left side that is effectively pumping blood around the body (No. 4 – right sided hypertrophy).

The heart may appear to have a normal size on x-ray examination, but it is only when the internal structure and the blood flow patterns are examined with ultrasound scanners that the defects may be uncovered.

The genetics of the condition are interesting as it appears that a cascade of genes are responsible and as more of the cascade become involved, then the condition increasingly worsens in its degree and severity of symptoms. So it is possible in some affected dogs, to have only some of the defects, in some cases so mildly they show no symptoms of ill health at all; thus the reasons why the condition may never be spotted in a dog which appears to be normal.

Dogs with the full tetralogy of defects may be signalled by a heart murmur but even this is not guaranteed and the main symptom identifying severely affected dogs is cyanosis, where the dog goes blue and collapses, with sudden death a possibility. This is not surprising because the amount of oxygen in the blood is already compromised and at some point a growing, active dog may exceed the capabilities of the heart to supply sufficient oxygenated blood to sustain life.

What is amazing is how a dog can survive with reasonably serious defects with little in the way of obvious symptoms. So this is not a problem that is always picked up in young growing pups, despite being a defect that is present at birth.