A report on cardiac examinations performed at the Saluki or Gazelle Hound Club Championship show on 1/11/2009

S.E. Brownlie PhD BVM&S MRCVS Cert SAC
Kileeekie, Crosshill, Maybole, Ayrshire KA19 7PY

My first experience of examining Salukis was in October 1996 and it was interesting to see if there were any changes in Saluki hearts thirteen years down the line.

I was originally asked to conduct a study in the breed because of several instances of sudden death. However it appears that problems are now rarer than before, possibly because breeders have bred away from suspect families. For readers new to the breed, I have rewritten the original introduction which I wrote in 1996 to set the scene:

Sudden death of an apparently healthy animal is usually assumed to be heart-related, but there are many other possible causes, such as respiratory obstruction, internal haemorrhage, electrolyte or calcium imbalance, poisoning and peracute viral infections. A post-mortem study by Mary Dee Sist (2008) in USA has shown that a surprising number of sudden deaths in Salukis were actually due to haemorrhage from tumours, often haemangiosarcoma, which may originate in the right atrium of the heart and cause pericardial effusion.

However it is probably true that many sudden deaths are related to cardiac disease and rhythm disturbances are likely to be present before the heart actually stops. The underlying pathology in dogs differs from that in humans in that myocardial infarction is considered to be very rare. Heart disease in dogs is often associated with a murmur – a noise in addition to the heart sounds signifying blood turbulence due to leaking valves, blood vessel narrowing or holes in the septum. Mitral valve disease, a slowly progressive thickening and curling of the main valve between the left atrium and the left ventricle, is common in all sizes of dogs in old age, and heart muscle disease (idiopathic cardiomyopathy) is also common, particularly in large and giant breed dogs. The most common congenital defect is now considered to be aortic stenosis (a narrowing of the main blood vessel leaving the left side of the heart). This defect is associated with a heart murmur, which is typically audible on both sides of the chest at the heart base and at the thoracic inlet. I did not identify any cases of this in my previous Saluki studies, despite many dogs having classic murmurs. However there are many other kinds of heart defects and there have been occasional reports of various types in the Saluki.
Auscultation screening (listening with a stethoscope) at shows for murmurs which could be associated with aortic stenosis has been underway for some years in boxers and Newfoundlands. Animals with loud murmurs may then be sent to a cardiologist for further investigation. Murmurs are graded according to loudness from 1 (very quiet) to 6 (very loud). There have been problems with these schemes but the general opinion is that they are the best way of excluding affected animals from breeding, because heart conditions are generally accepted to be inherited, even if the exact mode of inheritance is still poorly understood. However I concluded previously that this would not be useful in Salukis because of the large number of dogs with murmurs.

Cardiac rhythm disturbances are often associated with heart disease, particularly cardiomyopathy and aortic stenosis. An electrocardiographic (ECG) survey has been carried out in Irish wolfhounds, a breed which is well-known to be predisposed to myocardial disease, which has demonstrated a high prevalence of dysrhythmias and conduction disturbances in apparently normal animals (Brownlie, 1991). Until other breeds have been examined in a similar way, we do not know whether or not the wolfhound situation is unique. Although signs of heart failure may be sudden in onset, affected animals may have had detectable rhythm disturbances for months or years before heart failure ensues, and some cases die suddenly with no previous indication of illness. The electrophysiological mechanisms of dysrhythmias are variable. Some are thought to be triggered by increased sympathetic nervous system activity during excitement or exercise, but this is not always the case, as has been demonstrated by Moise, (1995), in her studies of an inherited problem associated with sudden death in German shepherd dogs, in which the dysrhythmia tends to occur at low heart rates particularly during sleep. However not all rhythm disturbances are associated with myocardial disease, and some may be benign and of no consequence to the animal.

A routine static electrocardiograph (ECG) is the easiest and most cost-effective method of screening dogs for rhythm disturbances. However it is a “snapshot in time”, and when an intermittent rhythm abnormality is demonstrated in a symptomless animal, there is difficulty in deciding whether or not this represents a threat to the animal’s life and whether or not therapy should be recommended. Other methods of investigation such as Holter monitoring (24 hour ECG) and ultrasound scanning (echocardiography) are therefore required.

In the sighthounds there is another factor which may be involved with sudden death and that is hypertension (high blood pressure). In the young dog this is probably a physiological adaptation to an athletic lifestyle, but in the older animal it may be a significant cause of heart disease and/or
cerebrovascular accidents (strokes). Angela Bodey carried out a small BP study in Salukis in 1998 which showed a range from low to moderately high but none of the dogs she examined had significant hypertension.

**Methods**

In order to gain the most information from a limited number of dogs in one day, it was decided that echocardiography and ECGs would only be carried out on older dogs, preferably those over the age of 5 years. Listening to hearts with a stethoscope only (auscultation) was carried out in the younger dogs.

A quiet room was provided, but some of the dogs were rather anxious as it was a rather “clinical” setting resembling a veterinary practice. As a result heart rates were variable, depending on the dogs’ temperaments. Any murmurs and rhythm disturbances were noted. The site of maximum loudness of the murmur was also noted eg left or right side, heart base or apex, or thoracic inlet. The heart rate was also recorded (beats per minute).

ECG examinations were carried out with the dogs lying on their right side and routine 6 lead traces were obtained using a Hellige Marquette three channel machine at 5, 25 and 50mm/s. Criteria for interpretation of the ECGs were as described in standard veterinary textbooks.(eg Tilley, 1992) Ultrasound examination was carried out, using a Sonosite Titan machine, mainly from the right side with the animals in a standing position. Both short and long axis views and M-mode measurements were recorded on to a digital flash card. Colour flow Doppler examination was also carried out from the right and left sides, and in one dog spectral Doppler measurement of the aortic and pulmonary valve velocity was carried out because of a typical aortic stenosis-type murmur. However results from this dog were within normal limits, demonstrating as we did previously that auscultation findings may be misleading in Salukis!

Standard measurements were obtained using the criteria of the American Society of Echocardiographers with the exception of maximum left atrial diameter because of the difference between canine and human anatomy in this area.

**Results**

The total number of animals examined was 27, 12 males and 15 females. Auscultation only was carried out in 20 dogs. Auscultation results were as follows:
Heart rates varied from 48 to 140 / minute. Only 6 dogs had heart rates over 100 / minute and these were all quite anxious.

No murmur on sides of chest: 11
Grade 1: 9
Grade 2: 7
Grade 3 - 6: 0
Irregularity only: 1

Most of the murmurs were louder on the left, although one dog had a murmur louder on the right. In 4 dogs, which were older animals, the murmurs were suggestive of mitral valve regurgitation, as they were loudest more caudally near the heart apex. However in most of the dogs, murmurs were heard at the heart base, and in three on both sides and at the thoracic inlet, which would normally be considered to be aortic in origin.

**ECG results:**
ECGS were obtained from 6 dogs.
Normal: 5
Conduction abnormalities: 1

**Ultrasound results:**
On this occasion only older dogs were examined as it was felt that they would be more likely to show abnormalities (3 males and 4 females). Only one of these did not have a murmur, 1 had a grade 1 and 5 had grade 2 murmurs. Results from the first examinations back in 1996 are shown in brackets for comparison.
Mean heart rate: 94 (86)
Mean left ventricular internal diameter in systole (LVIDS) (mm): 31 (33)
Mean left ventricular internal diameter in diastole (LVIDD) (mm): 42 (46)
Mean fractional shortening (%): 29 (27)
Mean ejection fraction: 50.6 (not done last time)
Interventricular septal thickness in systole (IVSS) (mm): 13 (12)
Interventricular septal thickness in diastole (IVSD) (mm): 10.1 (9)
Left ventricular free wall thickness in systole (LVFWS) (mm): 14.2 (12)
Left ventricular free wall thickness in diastole (LVFWD) (mm): 9.7 (8)
E point to septal separation (EPSS) (mm): 6.6 (7)
Maximum left atrial diameter (LA) (mm): 43 (42)
Mean aortic diameter (AO) (mm): 22 (24)
Mean left atrial : aortic ratio 1.28 (nd)

One dog showed fractional shortening and ejection fraction measurements which were lower than the range of the majority ie reduced left ventricular contractility, and therefore could have occult dilated cardiomyopathy (DCM). However its atrial and ventricular measurements were not outside normal limits. (The measurements given above include measurements from this dog.) Six dogs had mitral regurgitation, two with obvious mitral valve disease, and three had mild tricuspid regurgitation.

Discussion
This was a much smaller sample of dogs than took part in my previous study – perhaps because sudden death is now rare, owners are more complacent and less interested in having their dogs tested. Nevertheless I am most grateful to the owners who brought their dogs for testing. Evidence for the existence of mitral valve disease was seen in the older dogs and there was one with possible occult DCM. Although the differences in the ultrasound measurements were slight, and the sample not large enough for statistical analysis, the hearts of the dogs scanned were slightly smaller. Some of the dogs examined had murmurs, but compared with the numbers found in 1996 when over 10% of the dogs examined had grade 3 or louder murmurs, the situation now is quite different. Some of those dogs in 1996 clearly had heart disease but many did not. It is generally considered by veterinary cardiologists that flow murmurs (ie murmurs which indicate only blood turbulence without the presence of structural heart disease) are rare in dogs over one year of age but from our previous studies and from those carried out in USA that is certainly not the case in Salukis. One factor which may be important is the effect of exercise. In 1996 some of the dogs (unfortunately I did not note how many) were coursing regularly, which is not the case today. Like racehorses and humans, athletic working dogs have been shown to develop heart enlargement and murmurs with exercise training.

Salukis are still very fascinating to examine but it seems that the need to heart test is much less now than it was thirteen years ago. However breeders must beware of complacency and it is still good practice to examine potential breeding stock for heart problems.
I apologise for the length of time that it has taken to produce this report, but I hope Saluki owners will still find it interesting.

**References**

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