THE SALUKI HEART

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Eighty eight Salukis without symptoms of heart disease were evaluated from one to six times over a six year period. Studies were done by Dr. Cathy Gaber, a board certified veterinary cardiologist. Unsedated Salukis were studied in a quiet, non-hospital setting. The following non-invasive tests were done: physical examination, 6-lead electrocardiogram(ECG), M-mode and two dimensional echocardiography(ECHO). In an attempt to verify the non-invasive findings, heart autopsies were done on some dogs.

Physical examination revealed heart murmurs in over one third of the dogs. Most clinically important heart murmurs occur when blood flows through a leaking or obstructed heart valve, or through a congenital heart defect like a patent ductus arteriosus. The significance of a murmur may be partially evaluated if its location on the chest and sound intensity is known. The sound intensity ranges from I (very soft) to VI (audible without a stethoscope), and may be affected by variables such as amount of body fat, noise level during the examination, physiologic state of animal, and the experience of the examiner. Grade I-II systolic murmurs heard best at the left heart base are frequently not harmful to the health of the dog, but may be important to evaluate in breeding stock, especially those breeds with a recognized predilection for a specific type of heart disease. Murmurs of grade III or more in intensity are often associated with significant cardiac disease. The majority of Salukis studied had grade I-II systolic murmurs heard best at the left heart base, and Doppler echocardiograms were attempted on dogs with murmurs. These dogs were followed over time with repeat echocardiograms to see if heart disease developed. Other performance dog breeds as well as human athletes have been found to have soft, non pathologic flow murmurs.

Radiographs and ECG have historically been used to evaluate heart size. Although both are still very useful, echocardiography allows more complete evaluation of heart anatomy and function. When echocardiography includes a Doppler examination (non-invasive heart catheterization) information about the pattern of blood flow in the heart is obtained. All three tests are combined for a complete heart evaluation. The most common measurements made during an echocardiogram include left ventricular internal dimensions at contraction (LVIDs) and relaxation (LVIDd), % contraction of the left ventricle (%FS), interventricular septal thickness (ST), left ventricular wall thickness (LVWT), aortic diameter (AO), and left atrial size (LA).

Mixed breed populations have been used to establish weight referenced normal echocardiographic measurements. A recent publication by Morrison and co-workers supports the necessity for breed specific normal values. Eighteen of twenty Salukis in our pilot study had enlarged left ventricles using mixed breed normal values. These dogs were followed over time to determine if this enlargement was indicative of early heart disease, or simply normal for this athletic breed. Thirteen remained normal after approximately six years of follow up (age range 7-14 years), five died or were euthanized but had no signs of heart failure, and two developed dilated cardiomyopathy.

Symptomatic DCM is associated with an increases in LVIDd and LVIDs, with a decrease in %FS. To establish diagnostic criteria for early asymptomatic DCM Dr. Mike O'Grady defined a group of asymptomatic Dobermans that developed DCM. He compared echocardiographic measurements of

LVIDd, LVIDs, and %FS between Dobermans that developed symptomatic DCM and those that did not.. From this he defined criteria that predict which dogs will develop symptomatic DCM. Sixteen Salukis fit the Doberman left ventricular size criteria for DCM. Out of these Salukis with large left ventricles, nine have remained clinically normal to date (2.5-5.5 years later). Two Salukis have shown clinical symptoms consistent with DCM. The other five Salukis demonstrated a variety of acquired cardiac diseases or neoplasia.(see table: Salukis with large left ventricles on echo))

A decreased %FS is one criteria used to support the diagnosis of DCM. The lower limit of "normal " for %FS is 25-28% in dogs, although clinical signs of DCM are not usually seen until the %FS is 11-15 %. Our Salukis tended to have lower %FS than expected. Several dogs in our study had low %FS when calm, but increased to "normal" when their heart rates increased during excitement. This might lead to a false positive diagnosis of DCM. Since most Salukis will be studied in busy veterinary hospitals, where the dogs may be more excited during the ECHO, this may not be a major concern, but should be considered if the %FS is low.

As indicated in our pilot data, dogs in this larger study also had large aortas without evidence of subaortic stenosis. One reason that has been hypothesized for large aortas in athletic dogs is that they need large, extremely elastic aortas to allow large blood volumes to move quickly from the heart during exercise.

Finally, our study did not find a large number of Salukis with DCM (2/88). However a significant number of acquired cardiac conditions were identified, as well as a few congenital defects. Since we selected for dogs without signs of heart disease, it is not surprising that we found none of the congenital heart defects reported by Dr. Ogburn in Salukis. However, we found a surprisingly high incidence of cardiac abnormalities. It should be noted that a large number of these abnormalities did not cause cardiac signs, and some were unrelated to the death of the Saluki(see table, Salukis with "normal sized" left ventricles on echo).

In conclusion, our prospective study shows that the normal values for M-mode echocardiography derived from random breeds do not apply to the Saluki. The left ventricular size and aorta are larger, and in some cases the %FS is lower than 25 %. We were not able to adequately identify early onset of acquired heart disease. We did identify some Salukis with acquired heart disease, as well as a sporadic case of congenital heart disease.

Salukis used in breeding programs should have a careful cardiac examination done as puppies, and again prior to breeding. If a murmur is identified, it should be further evaluated using non-invasive tests including thoracic radiographs and ECG. Further testing including echocardiography with Doppler examination is frequently done by board certified cardiologists. A Saluki with no evidence of congenital heart disease after a through examination may be bred, but this animal may subsequently develop acquired heart disease. Since the common acquired heart diseases occur primarily in middle age(DCM) and old age (mitral regurgitation due to a thickened mitral valve), a single normal echocardiogram cannot render a dog "free and clear of heart disease".

Examination of the heart should be part of an annual physical for each dog. Any new murmur or an old murmur that has increased in intensity could herald early heart failure, and further evaluation is warranted.

1. Sist MD. Salukis with broken hearts. ASA Newsletter, Summer 1991.

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SALUKIS WITH "NORMAL SIZED" LEFT VENTRICLES ON ECHO

Murmur	Activity	Cardiac Symtoms	Outcome	Age-Yrs	Diagnosis
IV	↓ 8 yrs	Ascites / RHF 3½ yrs Wasting / Dyspnea	Died	13½	Tricuspid Dysplasia (Mod MI pleural & peritoneal effusion)
II	Normal	Coughing / Lethargic 1 wk	Died	9½	Hemangiosarcoma RRA Mets Lungs (Mod MI)
II	Normal	Abdominal Enlargement Lethargic / Not Eating 1 wk	Euth	11¾	Hemangiosarcoma RRA Mets Liver & Spleen (Mod MI)
No	Normal	Acute Collapse	Died	12½	Hemangiosarcoma RRA Hem into Pericardium & Chest Mets Lungs
II	↓ 2 yrs	Lethargic / Not Eating Limb Edema 1 mo	Euth	13½	Hemangiosarcoma of Spleen Mets Lungs (Mod MI)
III	↓ 3 mos	Lethargic / Wasting 1 mo	Euth	8½	Multicentric Lympho-Sarcoma (Mod MI)
No	Normal	None	Euth Trauma	11½	(Mild MI)

SALUKIS WITH LARGE LEFT VENTRICLES ON ECHO

Murmur	Activity	Cardiac Symtoms	Outcome	Age-Yrs	Diagnosis
No - 4 I - 2 II - 3	Normal	None	Alive	2½ - 5½	Normal - 9 dogs
III - IV	↓ 2 yrs	Collapse / Cough Congestive Heart Failure	Died	9½	DCM + Valvar Disease (Severe MI)
III - IV	↓ 2 yrs	Congestive Heart Failure	Died	9½	DCM + Valvar Disease (Mod MI + TI)
III	↓ 3 yrs	Exercise Intolerance Fainting	Died	5 3/4	Valvar Disease (Severs MI)
III	↓ 3 yrs	Irregular Heart Rate Acute Collapse	Died	13¼	Valvar Disease (Mod MI + Mild TI)
II	Normal	Irregular Heart Rate	Euth Trauma	8½	(Mod MI - Severe TI)
III	Normal	None	Euth Trauma	8½	(Mod MI)
III	Normal	Weakness / Wasting	Euth	14 3/4	Hemangiosarcoma RRA Mets Lungs,Liver (Pericardial Effusion)