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CASE REPORT GEVALVERSLAG

AORTIC STENOSIS IN A DOG

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SUMMARY

A case of valvular aortic stenosis in a dog is described. The presenting sign was syncope during exercise. An unusual feature was a right bundle branch block electrocardiographic pattern. During syncope electrocardiograms usually indicated atrial standstill and always had sever ST segment changes indicative of myocardial ischaemia. Pathological findings included chronic valcular changes which did not appear to be the result of a infectious inflammatory condition.

HISTORY

The subject, an 8 month old male Alaskan Malamute, had been successfully treated for biliary fever (*Babesia canis* infection) at the age of three months.

At 6 months the dog began to faint during exercise. Preliminary examination revealed a systolic murmur and a bizarre QRS electrocardiographic pattern. The dog was treated with a digitalis preparation and rested. As there was no improvement and the dog was having up to 8 syncopial episodes daily, it was then referred to the Department of Medicine.

On enquiry the breeder of the dog reported that, to the best of his knowledge, neither parent of the patient nor any sibling nor ancestor had suffered from heart desease.

CLINICAL EXAMINATION

The patient was very lively and hyperexcitable. He was well grown but poorly muscled and weighed only 27kg. Temperature was normal throughout. The resting pulse rate varied from 80 to 120 beats per minute. The pulse was "small" (of poor volume). The dog's excessive panting, possibly caused in part by hyperexcitability, rendered auscultation so difficult that detailed examination and phonocardiography had to be performed under general anaesthesia. The murmur was systolic, grade III/V (Detweiler and Patterson[4]), and was best heard over the left anterior thorax in the 2nd and 3rd intercostal spaces at the level of the costo-chondral junction. The murmur radiated widely and could be heard over the right anterior thorax and over the carotid arteries. The first heart sound, auscultated over the mitral area, was normal. There was no palpable precordial thrill, nor was there a ventricular heave. Mucous membranes were a normal pink colour. Appetite, water intake, urine and faeces were normal.

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ELECTROCARDIOGRAPHIC EXAMINATION.

ECG's were recorded with the dog in right lateral recumbency. P Waves varied slightly in conformation, were slightly prolonged (0,05 sec) and often notched. The PR interval was 0,13 sec.

The QRS complex was prolonged (0,06 to 0,07 sec) and bizarre. Initial vectors were normal, but later vectors were aberrant; the QRS complex was represented by QRSR'S'R" on lead I. The amplitude of the waves was not exceptional (Fig. 1).

The QT interval was 0,12 to 0,13 sec. The ST segment showed elevation of up to 0,2mv on lead II. The T wave varied in amplitude from day to day, occasionally reaching +0,6mv on lead II.

On several occasions the dog was exercised until he fainted. He ran keenly with a handler; between one and 10 minutes of running precipitated syncope, except on one occasion, when sustained running did not result in syncope. Syncope was preceded by a sudden slowing down, a few staggering steps and a very relaxed fall. The dog's pupils dilated and its mucous membranes became moderately cyanotic. There was profuse salivation and breathing was deep but regular. Syncope lasted one to two minutes and recovery was preceded by panting or howling. The dog usually regained his feet within two and a half minutes of falling.

Occasionally the dog was lifted and connected to the ECG recorder within 30 seconds of falling. On the syncopial ECG the P wave was usually absent, there was a slight variation in the RR interval, and the heart rate was usually about 100 beats a minute. There was severe depression of the ST segment on leads I, II and III and aVF (maximum depression was 0,9mv on lead II) and elevation on leads aVR and aVL (Fig. 2). The ST changes gradually disappeared and P waves reappeared 60 to 90 seconds after recordings commenced.

PHONOCARDIOGRAPHIC EXAMINATION

Phonocardiography was performed under general anaesthesia using a shortacting intravenous barbiturate*. The chest piece was placed over the point of maximum intensity of the murmur. The dog's nose

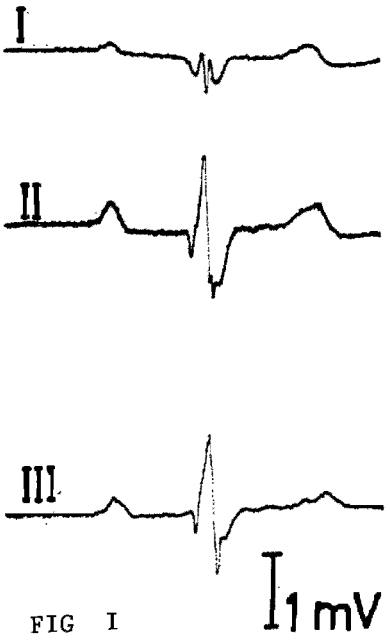

FIG 1

ECG leads I, II and III. Paper speed 100mm/sec.

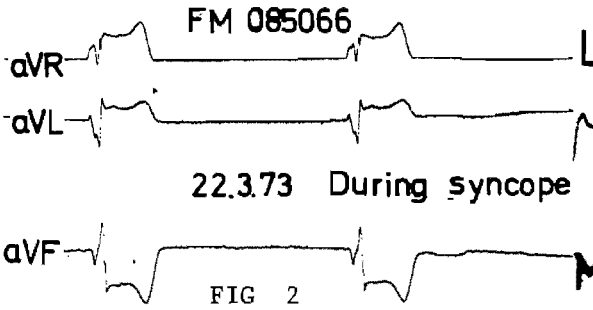

FIG 2

ECG leads aVR, aVL and aVF during syncope. ST segment changes are marked. Paper speed 100mm/sec.

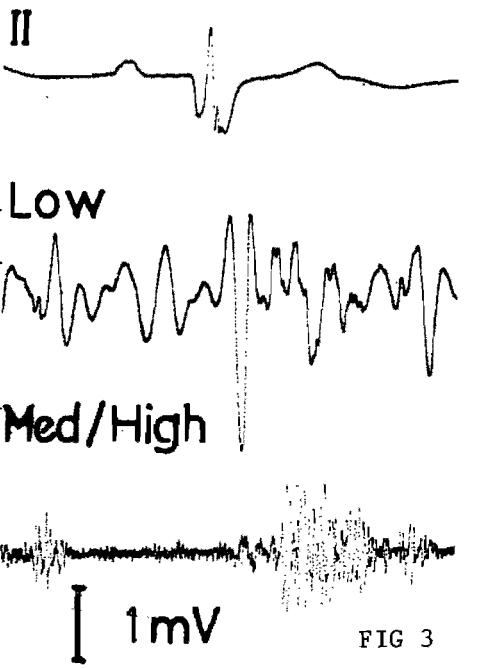

FIG 3

ECG lead II, low and medium/high frequency phonocardiogram. Paper speed 100mm/sec.

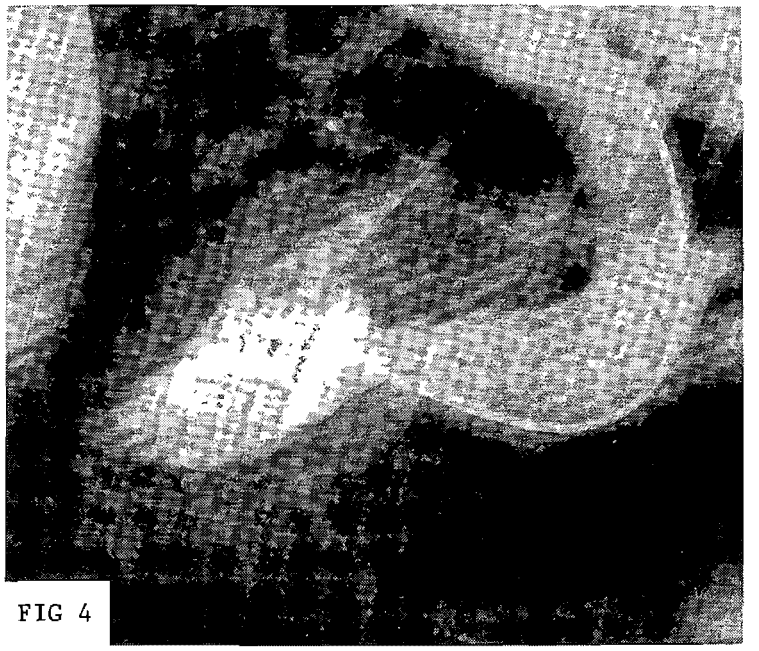

FIG 4

Angiogram showing the catheter in place in the left ventricle. The area of valvular stenosis and the post stenotic dilation of the aorta are easily seen.

FIG 5

Aortic semilunar valves. There is obvious thickening and loss of elasticity.

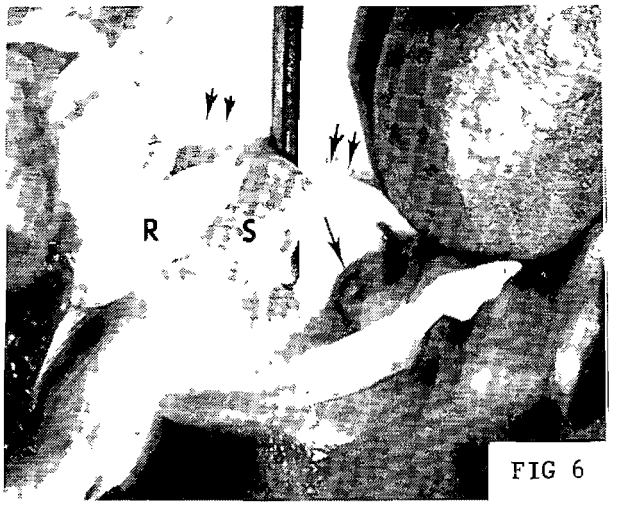

FIG 6

Right (R) and septal (S) semilunar valves. Note umbilicated nodule (arrow) on the wall of the sinus of Valsalva, myxomatous thickening of the leaflet (in front of probe) and projecting ring of fibrous tissue in the aorta (double arrows).

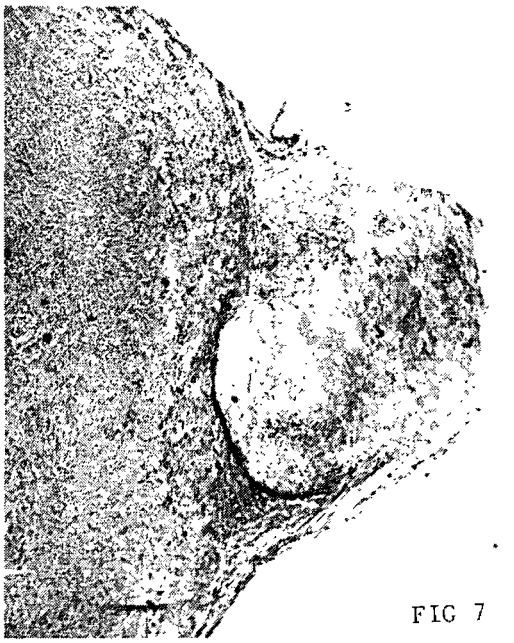

FIG 7

Fibrous ring in the aorta at the level of the top of the sinuses of Valsalva. H E x 30.

Fig. 8
Fibroelastic cartilagenous metaplasia of connective tissue in aortic and valvular lesions. Verhoeff's elastic stain x 500.

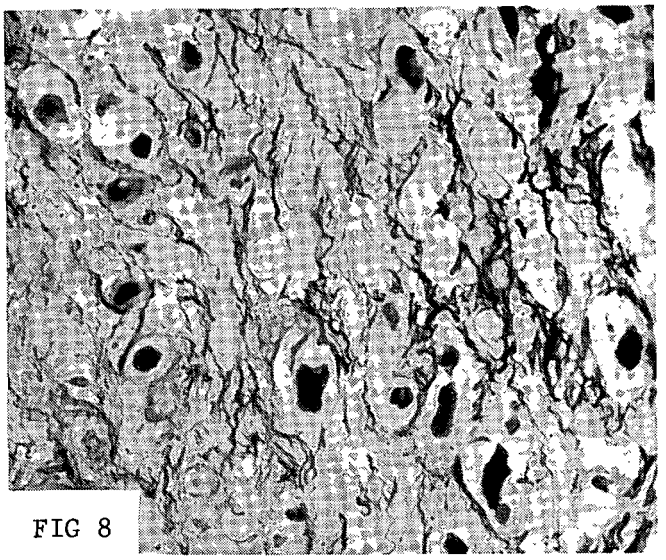

FIG 8

Fig. 9
Nodule on aortic semilunar valve. Composed of collagenous connective tissue undergoing metaplasia to fibroelastic cartilage H E x 75.

FIG 9

was held closed for periods of up to 15 seconds to eliminate respiratory interference. On the phonocardiogram the first sound was visible as a separate entity, and was followed 0,03 seconds later by a "diamond shaped" (crescendo - decrescendo) systolic murmur lasting 0,15 seconds. The second sound was visible as a separate entity (Fig. 3). The murmur was also recorded over the carotid arteries.

RADIOGRAPHY

Radiographs were exposed with the dog in the dorsoventral and lateral positions as described by Ettinger and Suter[4].

On the lateral view a grossly dilated aortic arch, slight elevation of the trachea and slight enlargment of the left ventricle were visible. The cranial waist of the heart shadow was absent.

On the dorsoventral view the cardiac silhouette was elongated by a very prominent and bulging aortic arch.

PROVISIONAL DIAGNOSIS

On the basis of the typical murmur, the severely dilated aortic arch and the syncopial episodes, a provisional diagnosis of aortic stenosis was made. The ECG was confusing and was ignored at this stage.

CARDIAC CATHETERIZATION AND ANGIOGRAPHY

To make a final diagnosis, anaesthesia was induced using a short acting barbiturate, the dog was intubated and anaesthesia was maintained using a gaseous mixture of oxygen and Fluothane**.

Following skin preparation, the right femoral artery was exposed surgically. A No. 6 gauge catheter was passed via the artery into the left ventricle. Considerable difficulty was experienced in passing the catheter between the aortic valves, and during this manipulation a test radiograph was exposed after injection of 10ml of Urografin 60***. On the radiograph the catheter was seen to have entered the left coronary artery. This had no apparent ill effect on the dog. When the catheter tip had entered the left ventricle, as ascertained by fluoroscopic examination, 10ml quantities of Urografin were injected using a syringe and maximum manual pressure. Radiographs were exposed as the injection was completed. (Neither pressure injector nor rapid plate changer was available.) Angiograms so obtained (Fig. 4) showed narrowing in the region of the aortic valves and gross post stenotic dilation of the aorta and the left subclavian and brachiocephalic arteries. Systolic pressure measurements were recorded as the catheter was withdrawn. These were 240mm Hg for ventricular systole and 140mm for aortic systole.

The pressure gradient and the radiographic picture confirmed the diagnosis of aortic stenosis.

PROGNOSIS

The prognosis without surgical interference was poor. Since surgery required extracorporeal circulation and the necessary equipment was not available, correction could not be undertaken. The dog would probably have died of congestive heart failure or ventricular fibrillation[4]. For these reasons the owner requested euthanasia.

"Intraval Sodium" (Thiopentone sodium) Maybaker.
**Halothane, I.C.I.
***Amidotrizoate 76%, Schering.

NECROPSY FINDINGS:

(a) *Gross*

The heart weighed 194g. There was a severe concentric left ventricular hypertrophy, the heart wall measuring 23mm in thickness at the level of the mitral valve and 10mm at the apex. Aortic cusps were thickened, wrinkled and curled (Fig. 5), and little elasticity could be demonstrated. Inside the aorta, at the level of the top of the sinuses of Valsalva, a dense ring of tissue projected into the lumen of the aorta (Fig. 6). The right semilunar valve had a hard nodule on the ventral surface which was 2mm in diameter and there was a similar nodule on the aortic wall in the septal sinus of Valsalva (Fig. 7). The nodule on the right semilunar valve was not related to the lunula. No jet lesions were seen on the endocardium.

(b) *Microscopic*

The projecting ring and nodule in the aorta were confined to the intima and were well demarcated from smooth muscle fibres of the media (Fig. 7). They consisted of collagenous connective tissue rich in a ground substance which was basophilic when stained with haemotoxylin eosin and positive for acid muco-polysaccharide when stained with alcian blue. Distributed throughout the ring and nodule were large cells partially surrounded by clear spaces, giving the appearance of the lacunae of cartilage (Fig. 8). Elastic fibres were demonstrated in association with these large cells by use of Verhoeff's elastic stain(Fig. 8).

The nodules on the semilunar valves were morphologically similar to the aortic lesion but contained less ground substance (Fig 9).

The cusps were diffusely thickened, the dorsal fibrous portions by increased collagen and the ventral spongy portions by myxomatous tissue and collagen. Elastic fibres extending from the endocardium of the ventricle into the semilunar valves were separated and fragmented.

DISCUSSION

Aortic stenosis is a relatively common cardiac defect amongst dogs[4]. It is usually congenital and subvalvular[5]. The Boxer, German Shepherd[4 6] and Newfoundland[4] breeds are predisposed to congenital subaortic stenosis. Valvular and supravalvular lesions have also been described[4]. In this case the lesions were valvular.

The cause of the lesions could not be determined. Microscopic changes were end-stage in nature, consisting of scar tissue undergoing fibrocartilagenous metaplasia. There was none of the inflammatory infiltrate or neocapillary proliferation which would be expected if an infectious agent had been involved. Considering the degree of change present and the youth of the dog, a congenital condition possibly related to improper, or absent, differentiation of mesenchymal tissue making up the valves seemed likely.

Many cases of aortic stenosis are asymptomatic[4]. Where present, symptoms usually include syncope, excessive panting, coughing and pulmonary congestion[4]. This dog had syncope and panted excessively. Syncope in such cases is presumed to be due to lack of left ventricular reserve[1].

The slightly prolonged P wave was considered indicative of mild left atrial dilation. This case was unusual in that the ECG included bizarre QRS complexes which were interpreted as a right bundle branch block pattern. This may have been the result of left ventricular hypertrophy of such severity that the electrical axis swung past -90°resulting in a false right bundle branch block pattern. The slight ST segment changes and the large but variable T wave may have been indicative of a slight degree of myocardial ischaemia at rest.

During syncope, the absence of P waves with a fairly regular and relatively slow heart rate was interpreted as atrial standstill [2]. The radical ST segment changes were presumably due to myocardial ischaemia.

The phonocardiographic findings were typical of aortic stenosis [4]. Translesional pressure gradients recorded in other cases have varied between 30 and 200mm Hg[4]. This case had a gradient of 100mm Hg.

Post stenotic dilation of the aorta in such cases is variable and may be impossible to ascertain on plain radiographs[4] . In such cases measurement of the "A/S ratio" on angiograms can be helpful. The "A/S ratio" is the maximum diameter of the aorta after the stenosis over the diameter of the aorta at the sinuses of Valsalva. In aortic stenosis the A/S ratio is more than I [3]. In this case it was 1,6.

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