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SUDDEN DEATH IN DOBERMAN PINSchERS

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Among serious admirers of the Doberman Pinscher it is well known that these dogs occasionally die suddenly and unexpectedly. Whether such deaths are more common among Dobermans than other breeds of dog is not known, but to see one of these handsome animals in apparently excellent health abruptly die is an unforgettable experience. Some of the deaths have been sudden collapse but the majority have been preceded by one or more convulsions, leading to a consideration of possible cerebral lesions; however, examinations of the brain have failed to demonstrate significant pathology there. Since a cardiac lesion could cause both convulsions and sudden death, this study was undertaken to see whether there was pathology in the heart, with particular attention to the conduction system.

Material

In the course of two years one of us (E. H. D.) who frequently attends shows and other meetings concerning the Doberman Pinscher has discovered two instances of sudden unexpected death in this breed of dog. Both dogs were male, one being 5 years old and the other 6. They were owned by different persons, and did not share kennel or other facilities. The two dogs did have a common ancestor, a magnificent red Doberman which was an international show champion. This common ancestor died suddenly and unexpectedly at the age of 10 years. Pertinent genealogical information which is available is as follows. The common ancestor (Red “B”) was mated with a dam which produced three pups, a female which died in an accident, a male which is living and well at present, and Dog #1 in the present study; this dam died at the age of 10 of “tetanus” which did not respond to appropriate treatment with tetanus antitoxin. Red “B” was mated with another dam, the health of which is unknown, producing a litter which included the mother of Dog #2 in the present study. The mother of Dog #2 is living and well, but the father died at the age of 5 years of unknown cause. Both dogs in the present study were black Dobermans.

The first dog in this report developed recurring convulsions one evening, with no apparent cause, and died during one of these the next morning. The second dog had been anorexic for several days and was in a veterinarian’s kennel because of this,
Three photomicrographs of AV bundle, comparing its appearance in the normal dog (A) with that of the first (B) and second (C) Doberman Pinscher. The tricuspid valve is in the lower left in A and to the right in B and C but otherwise orientation is the same in all three sections with the atrial septum above and the ventricular septum below. The open arrow indicates AV bundle in each, and the solid black arrow indicates the bone cyst in B and C. The bone was broken during preparation in C. Original magnification is X20 in A and X17 in B and C. The sections here and in the subsequent illustrations were all prepared with the Goldner trichrome stain.
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but had been eating normally at least two days prior to his being found dead in his cage one morning, with physical evidence suggesting the dog may have been violent during the preceding night. Because of the possibility of poisoning, stomach contents were examined toxicologically and gastrointestinal mucosa histologically, with negative results. Necropsy examination except for the heart was also negative.

In both dogs the heart was grossly normal in size, configuration and external appearance. The major coronary arteries distributed normally and there were no lesions within their walls or lumina on gross dissection. The aortic, mitral and pulmonary valves were grossly normal, but in both dogs the septal leaflet of the tricuspid valve was slightly thickened, particularly at its base. Subserial sections of the sinus node were prepared in manner described previously² and were found to be normal. Because a suddenly lethal cardiac lesion would logically be in the AV (atrioventricular) node or bundle, this region was sectioned serially at 6 micra with every 10th section saved and every 20th stained and examined. The block of tissue which was so examined extended from the posterior margin of the aorta anteriorly through the coronary sinus posteriorly, and included at least two centimeters of atrial septum above and ventricular septum below.

An identical lesion was present in the region of the AV bundle of both dogs. This consisted of a small bone cyst approximately a millimeter in diameter but of irregular contour, located in the central fibrous body directly above the undivided portion of the AV bundle (Figs. 1, 2). At this point the AV bundle exhibited extensive loss of substance, with only a few normal fibers remaining and the rest being replaced by fat. There was no evidence of old or recent inflammation around the bone. The six year old dog had no other recognizable intracardiac bone, but there were islands of cartilage approximately a millimeter in diameter in the aortic valve and annulus of the mitral valve, as well as a similar focus of cartilage several millimeters behind the bone cyst in the central fibrous body. The five year old dog had extensive cartilaginous thickening of the septal leaflet of the tricuspid valve (Fig. 1B) and two bone cysts within the valve similar in size to the bone in the central fibrous body. There were a number of smaller islands of cartilage adjacent to these three bone cysts.

Normally the blood supply of the canine AV node and bundle is dual,³ which is in contrast to man, in whom the same region is usually supplied predominantly by a single artery.⁴ A branch from the origin of the posterior descending artery penetrates the region of the canine AV node from behind, but a similar branch rises to that region from the septal artery, for which there is no normal counterpart in man. Although no single lesion could be found in the main trunk of either nutrient nodal artery in these two dogs, a number of small (100-300 micra diameter) coronary arteries in the region of the AV node and bundle were nearly occluded by intimal proliferation (Fig. 3).
Figure 2
Three photomicrographs demonstrating the bone in the wall of the two cysts. A shows spicules of bone (from the first dog) with ordinary light illumination and B the same area under polarized light, demonstrating the birefringent character of the bone. C shows a bone spicule (under polarized light) from the cyst of the second dog, with adjacent particles of marrow fat. Original magnification in all three sections is X192.
No electrocardiograms or examinations of pulse are available on the two dogs which died. To see whether apparently normal Doberman Pinschers had any evidence suggesting heart block or other cardiac electrical disturbance, electrocardiograms were made on 15 purebred Dobermans and were all normal. There was specifically no arrhythmia, no degree of PR interval prolongation, and no QRS configuration change. We have not had the opportunity to examine the heart of normal Doberman Pinschers dying of an accident, but in examinations of the conduction system from over 50 mongrel dogs a bone cyst has not been observed in the heart.
Two photomicrographs from different sites and at different magnifications to show the relation of the bovine os cordis to AV bundle. In each the atrial septum is above, ventricular septum below and right atrial cavity to the right. A (X3.2) is from the anterior end of the bundle, as it is dividing into right and left branches, while B (X17) is 4 mm posterior from A. In each the os cordis is indicated by a solid black arrow and the AV bundle by an open arrow; note the substance of the AV bundle is intact (compare to Figs. 1B and 1C).
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The beef heart (and that of closely related ungulates) is known to contain a bone (os cordis) normally. The os cordis of four beef hearts was examined grossly and microscopically (Fig. 4) for comparison to the lesion found in the two Dobermans. The os cordis extends from the right side of the aortic root almost to the coronary sinus, lying near the lower margin of the interatrial septum, thus being above the AV bundle and parallel to the AV node. It is densely encased in the collagen of the central fibrous body. From its anatomic relationships it seems unlikely that the os cordis could compress or otherwise impinge on the AV node or bundle, and in no instance was there focal fatty replacement of the AV bundle.

DISCUSSION

In view of an identical lesion (a small bone cyst) adjacent to the AV bundle being present in the hearts of two Doberman Pinschers which died suddenly and unexpectedly, it seems unlikely that this is an incidental finding without functional significance. Two critical questions are why bone should be located in the position it was found, and what the functional significance might be, particularly relative to AV conduction. Bone in the heart is not a new observation, but there has been no consistency in location within the heart, it most often being present in a site of chronic inflammation. It should be stressed that an important difference exists between simple calcification and bone, since the former is not uncommon in many locations in the heart while the latter is rare. Calcification in association with chronic aortic valvulitis, for example, is a frequent necropsy finding in patients with aortic valve disease who have complete heart block.

That the central fibrous body is either under unusual physical stress or some other influence (possibly a metabolic factor) is suggested by the normal presence of bone in this location in the beef heart. The length of the bovine os cordis may be expected to stabilize it so focal compression of adjacent structures (such as the AV bundle) would not occur. The smaller pieces of bone in the Doberman hearts on the other hand might move more freely and thus produce local trauma. There is a more plausible explanation, however, by which the bone cyst might be responsible for degeneration and fatty replacement of adjacent AV bundle, and that is metabolic competition. It is well known that cartilage and new bone have a high metabolic requirement, so that even with normal local arterial supply the adjacent AV bundle may receive inadequate nutrition to survive. Furthermore, in both these Doberman hearts some of the regional small arteries had significantly narrowed lumina, indicating that the local blood supply was less than normal. It has recently been demonstrated in chicken hearts that the combination of focal ischemia and an appropriate antigenic stimulus can consistently produce focal new growth of cartilage in myocardium.

Since the large os cordis and the AV bundle in the beef heart both receive their principal blood supply from the same artery, and since the AV bundle does not degenerate, the anatomic proximity of these two structures is not of itself an adequate explanation for the postulated metabolic competition for available blood supply. In both Dobermans, however, there was additionally a significant luminal narrowing of small arteries in the region, so that the new bone and AV bundle
were competing for an abnormally reduced local blood supply. Narrowing and occlusion of small intramural coronary arteries is one of the commonest forms of coronary pathology in many animals, including the dog. A similar coronary arteriopathy has recently been described in association with several different heritable neuromuscular diseases in man, and was suggested as a possible etiology in the obscure myocardio-pathy frequently associated with these and other heritable diseases. Since both dogs in this report had a common ancestor, it is possible that their coronary arteriopathy was an inherited fault.

For the present it is our belief that a bone cyst in the central fibrous body is responsible for focal degeneration in the adjacent AV bundle in the hearts of Doberman Pinschers which die suddenly and unexpectedly, and that the mode of death is most likely an inefficient cardiac electrical mechanism during transient complete heart block. To substantiate the hypothesis it will be necessary to confirm these observations in more such instances of unexpected sudden death, and ideally to have electrocardiographic documentation of heart block. We are aware of no special reason why this lesion should be unique in the Doberman Pinscher and anticipate that it will eventually be observed in other breeds of dog and possibly other animals.

**SUMMARY**

In two Doberman Pinschers which died suddenly and unexpectedly an identical lesion was found in the heart. This was a small bone cyst in the central fibrous body directly adjacent to the AV bundle, which was largely replaced by fat at that point. It is postulated that sudden unexpected death in these dogs may be due to a form of Stokes-Adams attack.

**REFERENCES**