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From the Small Animal Clinic, Royal Veterinary and Agricultural University, Copenhagen, and the Department of Orthopaedic Surgery, University Hospital of Copenhagen, Denmark.

DYSOSTOSIS ENCHONDRALIS OF THE ULNAR BONE IN THE BASSET HOUND

By

P. G. Rasmussen and *Inge Reimann*

RASMUSSEN, P. G. and INGE REIMANN: *Dysostosis enchondralis of the ulnar bone in the Basset Hound.* Acta vet. scand. 1977, *18*, 31—39. — Ninety-four Basset Hounds were examined for the clinical features, the radiographical changes and the nutritional and hereditary possibilities connected with the development of decreased longitudinal growth of the ulnar bone. In six puppies the possibility was examined of the development of the disease being due to overloading of the foreleg. Ten of the dogs were examined for gross pathology and 4 by histology post mortem.

Clinical symptoms and radiographical changes were not seen until the age of 3½ months. In all cases there were broadening of the joint space in the elbow joint and degeneration of the articular cartilage at the semilunar notch. An abnormally broad and irregular growth plate was seen in the affected young dogs, and histology revealed obvious abnormalities in the growth plate located in the zone of resting cartilage indicating degenerative disturbances in the cartilage. There were no signs of rickets, and it was not possible to demonstrate that the disease was caused by misloading.

It is concluded that the disease corresponds to similar anomalies found in the other chondrodystrophic breeds of dogs, and it is suggested that the decreased growth of the ulnar bone may be hereditary.

bone disease; developmental; dog.

The cause of a limp in the forelegs of young dogs may often be found in the elbow joint. In addition to dysplasia of the elbow joint, the abnormalities are usually displacement of the articular surfaces of the radial and ulnar bones. Such displacements may be caused by a decrease in the longitudinal growth of these bones (*O'Brien et al.* 1971). As regards the radial bone, the growth disturbances are generally due to changes secondary to trauma, whereas this is rarely the case in the ulnar bone. Among the large breeds (Grand Danois, Labrador, Retriever), growth dis-

turbances of the ulnar bone have been observed as a consequence of hypertrophic osteodystrophy or rickets. Among the small breeds a decrease in the growth of the ulnar bone has been found particularly among the chondrodystrophic breeds (Basset Hound, Dachshound, Skye Terrier, Pekingese) for reasons still unknown (*Rasmussen* 1974). The secondary changes of the disease seen in the elbow joint, called distractio cubiti, have been investigated in the Basset Hound clinically, radiographically and by dissection (*Grüll & Henschel* 1973, *Hitz* 1974), and methods for surgical correction of the anomalies have been described (*Henschel & Grüll* 1975).

The present study is an attempt to elucidate the aetiopathogenesis of the disturbance in the longitudinal growth of the ulnar bone in the Basset Hound by investigation of the pathological and histological changes compared with the alterations seen by radiography.

MATERIAL AND METHODS

The forelegs of 94 Basset Hounds, aged 1½ to 11 months, were investigated clinically and by radiography. Four of these dogs, aged 7 to 11 months, were euthanized for study of gross pathology of the elbow joint and the bones of the forearm. A litter of 6 puppies, with an affected father and grandfather but a healthy mother, was investigated in the following way: Two by two at the age of 2, 3 and 4 months, 1 of the forelegs of the puppies was bandaged for a period of 3 weeks, so that 1 foreleg was non weight-bearing and the other was overloaded. Five of these puppies were euthanized at the age of 3½, 4½ (two), 6 and 8 months, and the forelegs were examined post mortem.

The ulnar bones from 4 of the 5 puppies, aged 3½, 4½, 4½ and 6 months, were prepared for histological examination. A 3 cm long piece, including the distal growth plate, was taken from the distal part of the ulnar bones from both sides and divided into 2 equal parts in the frontal plane. One part was embedded in paraffin after decalcification in 10 % formic acid; the other part, which was not decalcified, was embedded in methyl methacrylate. Sections 6 µ thick were stained with haematoxylin-eosin, toluidine blue and safranin O. The non-decalcified sections were also stained a.m. Goldner. Finally, decalcified sections from the elbow joints were prepared and stained as described above.

RESULTS

Clinical symptoms such as limp and radiographical changes in the elbow joints were not found until the Basset Hounds were 3½ to 4 months old. In the slight cases, the dogs showed a limp only intermittently, and radiography revealed broadening of the joint space between the distal part of the semilunar notch and the humeral condyle.

In the severe cases, the limp could be permanently present, together with an abnormal configuration and a paddle-like movement of the foreleg. In such cases, radiography showed also dislocation of the styloideal process in proximal direction or a bowing of the shaft of the radial bone anteriorly. In the extreme

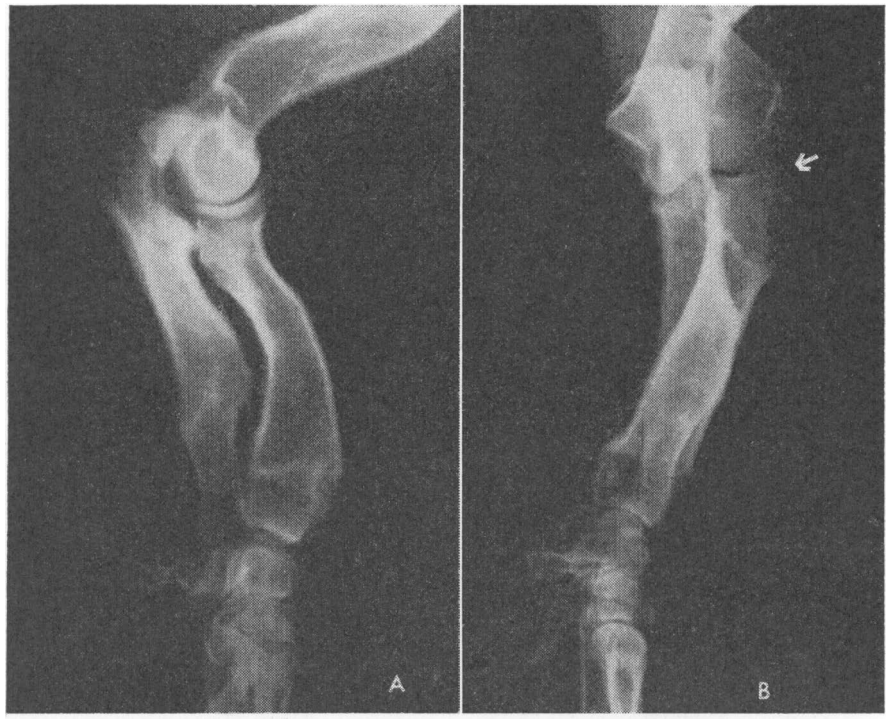

Figure 1. A severe case in a 9-months old dog.

A: A wide space is seen in the elbow joint between the humeral condyle and semilunar notch. Furthermore there is curving of the shaft of the radial bone anteriorly.

B: The head of the radial bone is subluxated laterally and a sesamoid bone is formed lateral to the head (→). There is a torsion in the elbow joint.

cases, the head of the radial bone was found to be subluxated laterally (Fig. 1).

By gross pathology degeneration of the articular cartilage in the semilunar notch was always present, and in most cases the cartilage of the ridge of the lateral humeral condyle also showed degeneration. In severe cases there was a torsion of the radial and ulnar bones in the elbow joint, and furthermore denuded bone was seen in the above mentioned areas (Fig. 2). The development of a sesamoid bone in the lateral ligament was a characteristic finding (Fig. 1).

Of the 94 dogs investigated 37 had 1 or more affected ancestors in a 3-generation pedigree. Thirty of these dogs had the anomaly clinically as well as radiographically, and 7 were normal. The pedigrees of the other dogs in the material were not available.

Five out of the 6 test puppies were affected, and degeneration of the articular cartilage was found at the semilunar notch and

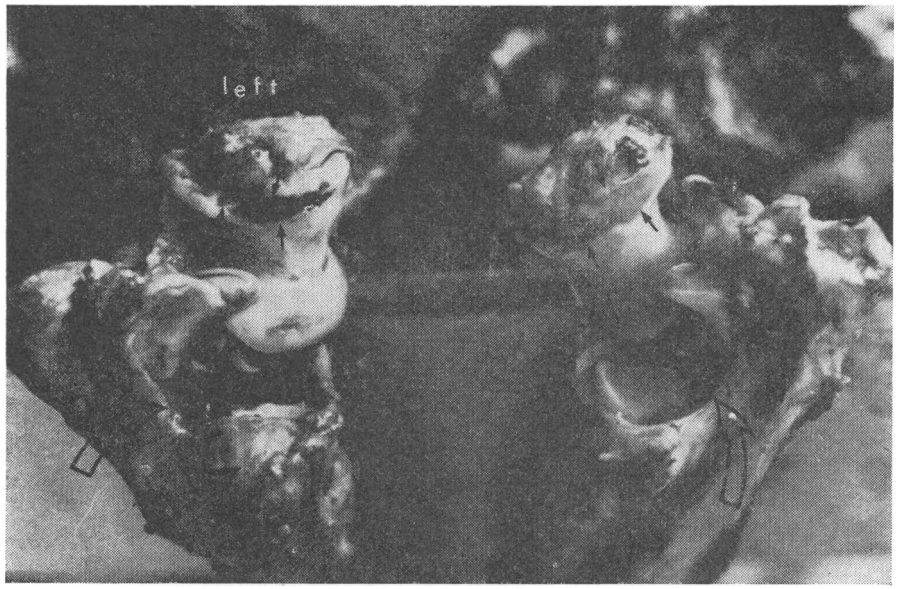

Figure 2. From the same dog as in Fig. 1. At the ridge of the lateral humeral condyle there is no cartilage left and the denuded subchondral bone can be seen (→). Neither is there any cartilage left at the semilunar notch of both ulnar bones (=▷). There is a prominence of the radial bone in the left elbow subluxated laterally, this prominence containing a sesamoid bone (□—▷).

lateral humeral condyle. This was most pronounced in the 6- and 8-months old puppies, in which there were areas without cartilage left and denudation of the subchondral bone. Comparison of the forelegs which had been bandaged for 3 weeks with the contralateral forelegs showed no difference. Radiography showed that the growth plate of the ulnar bone was normal in the puppies up to the age of 6 weeks. After the age of 2 months, the growth plate was seen to be abnormally broad and irregular (Fig. 3) in the puppies which later showed changes in the elbow joint (Fig. 1).

Histological examination of the distal growth plate of the

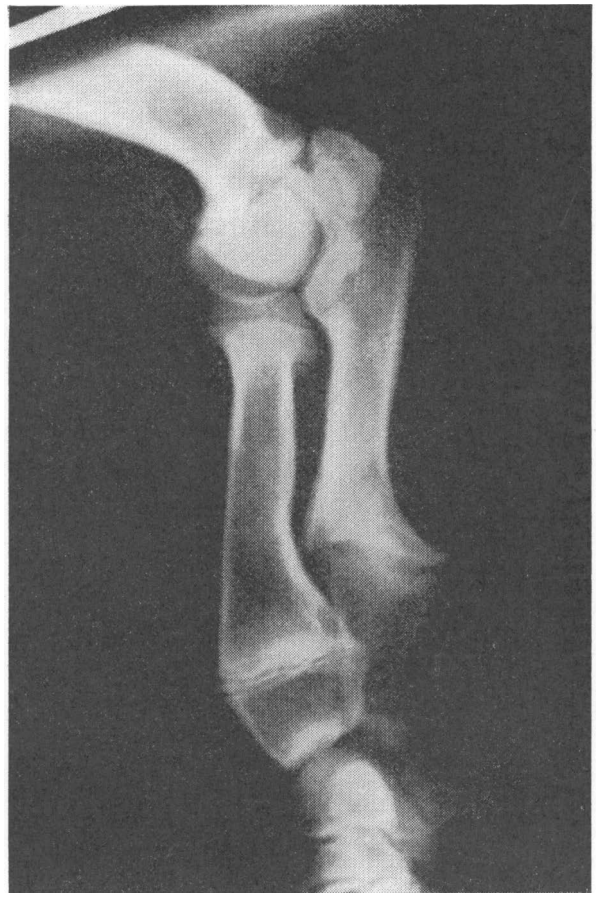

Figure 3. Puppy, 2½ months old. The distal growth plate in the ulnar bone is abnormally wide and irregular.

ulnar bone showed obvious abnormalities in the cartilage of the growth plate in the 4 puppies investigated, this being most pronounced in the 4½-months old puppies. The abnormalities consisted of varying thickness of the zone of resting cartilage and this was demarcated with peaks and deep grooves. Furthermore, there was disorganization, cloning and cluster formations of the chondrocytes. Safranin O staining (a special staining for mucopolysaccharides) showed that the intercellular matrix had decreased and heterogenous stainability (Fig. 4). The proliferative, hypertrophic and calcifying zones showed no demonstrable abnormalities. In the non-decalcified sections, stained by the method of Goldner (a special staining for osteoid tissue) there was no abnormal amount of osteoid tissue. In the histological sections from the elbow joints, pronounced degenerative changes of the articular cartilage were seen, with flaking, clefts, disorganization and decreased stainability with safranin O, and there were some areas with no cartilage left.

DISCUSSION

Although the majority of the Basset Hounds in the present material had lameness of the forelegs when examined at the Small Animal Clinic, the material does not statistically give an exact impression of the frequency of the disease. However, the large number of affected cases compared to the relative occurrence of the Basset Hound would indicate that the disease is relatively common. The many affected and the few unaffected dogs with ancestors with the disease also show that the disease probably has a hereditary disposition, which is not dominant, since the disease was not present in all the offspring of affected parents.

The immobilization and overloading, respectively, of the fore-

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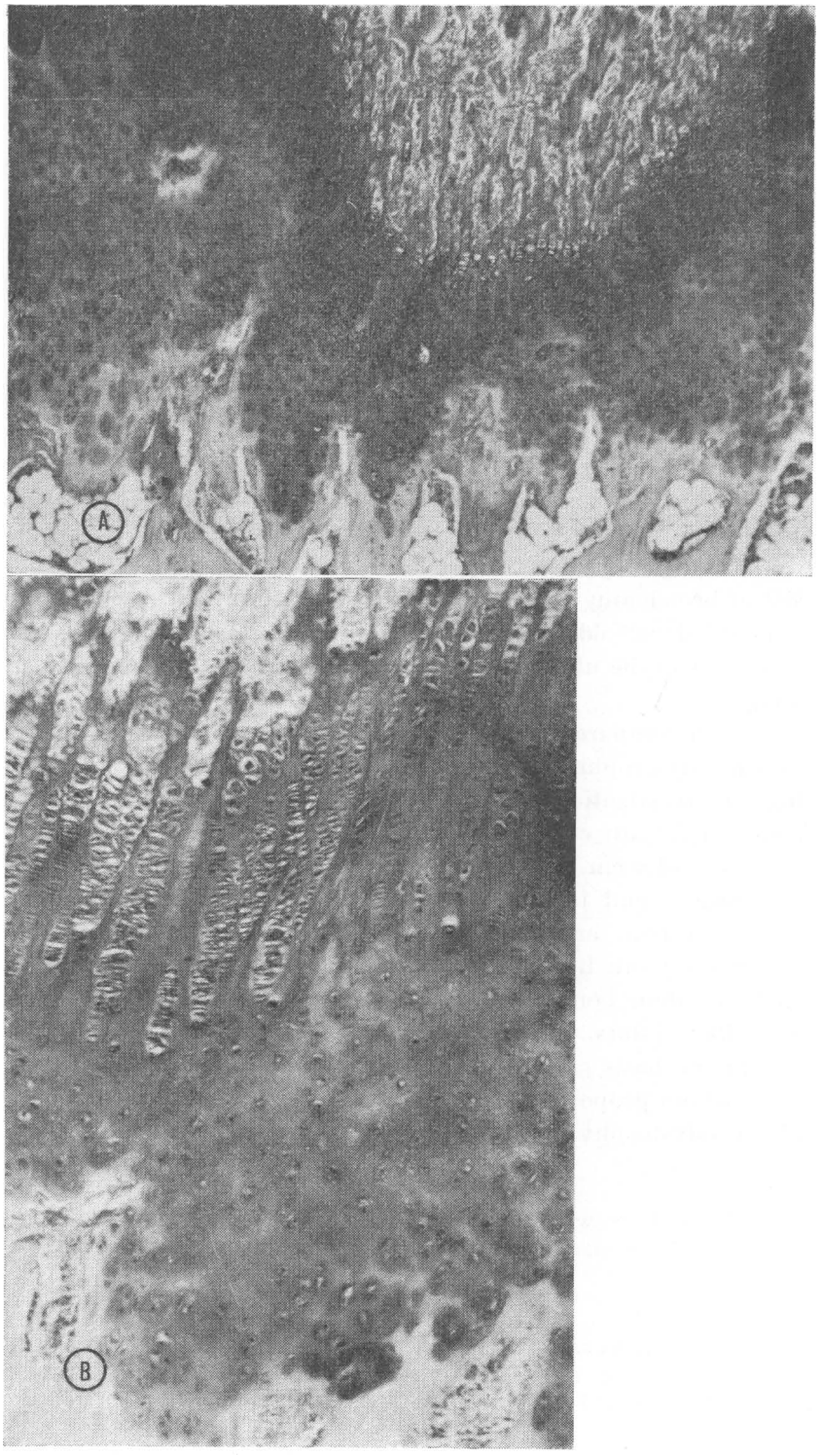
Figure 4.

A: Distal growth plate of the ulnar bone from a 3½ months old dog. There are pronounced changes in the growth plate with varied thickness of the zone of the resting cartilage, the irregularities being demarcated with peaks and grooves. There is also disorganization and cloning of the chondrocytes, and decreased stainability of the intercellular matrix. The proliferative, hypertrophic and calcifying zones seem normal.
Safranin O, magnification × 30.

B: Same as A, magnification × 175.


A
B

legs of the test puppies did not demonstrate that development of the disease could be caused by misloading.

As apparently all the dogs were given an adequate diet, there was no indication of a nutritive defect, although radiography showed a broad irregular growth plate in the young puppies similar to the findings in rickets. Furthermore the histological findings disproved the possibility of rickets. Histology showed an obvious irregular and broad growth plate in the distal part of the ulna, corresponding to the radiographical findings. Histologically, the abnormalities were located in the resting zone, with disorganization, clusters and a decreased staining with safranin O. This would indicate a decrease in the content of mucopolysaccharides in the matrix, similar to the findings in the articular cartilage in cases of degenerative joint disease (*Rosenberg* 1971, *Mankin et al.* 1971).

Histologically the radiographical changes seen in rickets consist of broadening of the proliferative cartilage and an increased amount of osteoid tissue. Thus there were no histological signs of rickets in the ulnar bone of the affected puppies in the present study.

When compared with the demonstration of the same disease in other dystrophic breeds (*Rasmussen* 1974), the present histological investigations indicate that the disease may have some connection with chondrodystrophy.

It would seem that the Basset Hound family has a hereditary disposition, and that the disease is abnormally accentuated in the ulnar bone and may produce severe secondary changes in the elbow joint. It is suggested that this may be due to the fact that the ulnar bone has only 1 growth plate between the carpal and elbow joints.

On the basis of the results of the histological investigations, the authors propose to designate the disease: Accentuated ulnar chondrodystrophy.

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REFERENCES

Grüll, F. & E. Henschel: Distractio cubiti beim Basset Hound. (Dislocation of the elbow in the Basset Hound). Kleintier-Prax. 1973, *18*, 217—223.

Henschel, E. & F. Grüll: Zur Therapie der Distractio cubiti beim Basset Hound. (Concerning the treatment of distractio cubiti in the Basset Hound). Kleintier-Prax. 1975, *20*, 267—271.

Hitz, D.: Ulnadysplasie beim Basset Hound. (Dysplasia of the ulna in the Basset Hound). Schweiz. Arch. Tierheilk. 1974, *116*, 285—294.

Mankin, H. J., H. Dorfman, L. Lipiello & A. Zarins: Biochemical and metabolic abnormalities in articular cartilage from osteoarthritic human hips. J. Bone Jt Surg. 1971, *53-A*, 523—537.

O'Brien, T. R., J. P. Morgan & P. F. Suter: Epiphyseal plate injury in the dog: A radiographic study of growth disturbance in the forelimb. J. small Anim. Pract. 1971, *12*, 19—36.

Rasmussen, P. G.: Præmatur epiphyselukning hos hund. Sequelae, diagnose og ætiologi. (Premature closure of the epiphyseal plate in the dog. Sequelae, diagnosis and aetiology). Proc. 12th Nord. Vet. Congr. Reykjavik 1974, 32—33.

Rosenberg, L.: Chemical basis for the histological use of Safranin O in the study of articular cartilage. J. Bone Jt Surg. 1971, *53-A*, 69—82.

SAMMENDRAG

Dysostosis enchondralis ulnae hos Basset Hound.

Fireoghalvfems Basset hunde blev undersøgt for det kliniske billede, de røntgenologiske forandringer og de ernæringsmæssige og heriditære muligheder ved udviklingen af en nedsat længdevækst af ulna. Seks hvalpe blev anvendt til undersøgelse af, om sygdommen kunne udvikle sig på grund af overbelastning af forbenene. Ti af de undersøgte hunde blev obduceret og heraf 4 histologisk undersøgt post mortem.

Hverken kliniske eller røntgenologiske forandringer kunne påvises før 3 måneders alderen. Der var i alle tilfældene breddeforøgelse af ledspalten i albueleddet og degeneration af ledbrusken i incisura semilunaris. Hos de afficerede unge hunde sås en abnorm bred og uregelmæssig epiphyselinie, og ved den histologiske undersøgelse fandtes degenerative forandringer i epiphyselinien lokaliseret til zonen med hvilende brusk. Der var ingen tegn til rachitis eller andre ernæringsforstyrrelser, og hos hvalpene i testgruppen var der intet der tydede på, at sygdommen skulle kunne fremkaldes på grund af fejlbelastning.

Det konkluderes, at sygdommen svarer til lignende anomalier hos andre chondrodystrofiske hunderacer (gravhund, sky terrier, pekingeser), og det antages, at den nedsatte vækst af ulna er heriditær.

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Reprints may be requested from: P. G. Rasmussen, The Small Animal Clinic, Royal Veterinary and Agricultural University, Bülowsvej 13, DK-1870 Copenhagen V, Denmark.