FROM THE DEPARTMENT OF PATHOLOGY OF THE ROYAL VETERINARY COLLEGE IN STOCKHOLM. CHIEF: PROFESSOR SVEN RUBARTH

A PATHOLOGIC-ANATOMICAL INTERPRETATION OF DISC DEGENERATION IN DOGS

By

HANS-JÖRGEN HANSEN

Ever since Dexler's work in the 1890's, a certain disease in dogs has been known by the name of "enchondrosis intervertebralis". The term has referred to a disease that has most frequently affected middle-aged and older, small, short- and bandy-legged breeds and which shows clinically the great variation that can develop from any kind of spinal cord and/or nerve-root compression.

In minor cases the illness is apparent only from excessive sensitivity of the skin, diffuse pains in the back and general dorsal insufficiency which in more serious cases can lead to complete paraplegia, either suddenly or preceded by the above symptoms. Even in extreme cases, the disease has a tendency to disappear without treatment but shows a high frequency of relapse.

In accordance with its name, the disease has been considered as localized in the intervertebral discs and through regressive and progressive changes in these it results in encroachment on the space in the vertebral canal. Although the name "enchondrosis intervertebralis" as such indicates a clearly defined diagnosis, the uncertainty has been great concerning the actual nature of the changes. More ambitious systematic investigations of the pathological anatomy of the discs have not been presented in current literature and the knowledge we have been able to secure from it has instead been based on the observation of a few cases or of shorter series of serious cases. Interest has been shown more in the neuro-pathological aspects as the more immediate causes of the clinical symptoms rather than in changes in the discs.

In the Department of Pathology of the Royal Veterinary College in Stockholm, a topographic and pathologic-anatomical investigation of the intervertebral discs was conducted with special reference to the

conditions when so-called enchondrosis intervertebralis occurred. The point of departure for this investigation was the following:

In 1896 Dexler attributed chronic compressions of the spinal cord in dogs mainly to two causes; one—, chronic disseminated ossification of the dura is omitted. Dexler considered that the other cause was primary disease in one or more intervertebral joints with hyperplastic processes on the dorsal side of the discs and secondary to these, an inflammatory reaction in the vertebral periosteum, epidural space and dura. *Joest* (1921) and *Wiedemann* (1922) also observed primary regressive changes in the inner part of the disc and interpreted the formations in the epidural space as the result of a cartilage growth from the disc perichondrium. In 1939 *Tillmanns* showed that these formations could be protruded disc material and in 1948 *Fankhauser* reported identical observations in his works. Independent of the European conclusions *Riser* had in 1946 concluded that disc protrusion in dogs was the cause of posterior paralysis. Disregarding occasional doubts of the discogen nature of the disease (*Jacob* 1910, *Boddie* 1949), the general conclusion has been that diseased changes in the discs are of primary importance. On the other hand, interpretation of the nature of the formations bending toward the spinal canal has varied.

In the late 1920s *Schmorl* drew attention to the occurrence and to the frequency of pathological changes in the discs of human beings. With the work of *Alajouanine* and *Petit-Dutaillis* (1930), *Elsberg* (1931) and *Mixter* and *Barr* (1934), the extradural ventral chondromas that had been observed and described in many case reports could be identified as *Schmorl*'s "Knorpelknötchen". *Mixter* and *Barr* determined that disc prolapse was a common cause of sciatica. Since in that way attention was directed to the significance of the discs in lumbago-sciatica in human beings, other causes of these syndromes, such as intra-abdominal diseases and myogenic and neurogenic factors, all took subordinate place.

In our investigations of so-called enchondrosis intervertebralis in dogs, it has been evident that the ventral extradural formations are without exception signs of a disc prolapse, and the historical development of our interpretation of these intraspinal processes of disease in both human beings and dogs united after an approximately parallel development in the conceptions "disc degeneration" and "disc prolapse". Therefore, from now on, I wish to use this current orthopedic nomenclature. Prolapse and protrusion are used as synonymous terms in this paper.

Before a more detailed account is given of the discs' pathology, I wish to give some statistical data on the frequency of disc degenera-

tion for different breeds, sexes and ages. Disc degeneration in dogs is a disease characterized by definite breed disposition, a fact that should be of value in investigating the causal genesis. As we shall point out later, disc prolapse always postulates a disc degeneration, but the latter can of course occur without prolapse and may even express itself in other ways. A statistical investigation of 100 cases, in which a clinical suspicion of disc prolapse was verified by the post mortem examination gave the following results as to breed, sex and age.

The population from which this material was secured consists of the licensed dogs in Stockholm. Beginning with the O-hypothesis that no disposition due to breed exists, the probability that the different breeds would be represented in the diseased group has been compared with the probability that the same breeds would be represented in the mother population chosen at random. Thereby we have been able to show that the O-hypothesis cannot be rejected for most of the breeds, which may therefore be said to have a "normal" disposition for the disease. The three breeds, dachshund, French bulldog and pekinese are exposed to disc degeneration at an abnormally high rate; terriers and spitz dogs at an abnormally low rate. For all these breeds, the probability that the O-hypothesis is valid is less than one per cent.

The significance of this disposition in groups of breeds is of course connected with the extent to which a common denominator exists for the exposed breeds. In works of reference in this field, mention is often made of the dachshunds' long back or more properly described, its long rectangular shape. Since the French bulldog is one of our most quadratic dogs, shape can scarcely have any decisive significance. *Fankhauser* (1948) has surmised that the tendency to disc diseases may be referred to the same fundamental cause as that of dwarf growth in general. An important common denominator is undoubtedly certain traits of chondrodystrophy. It is apparent that such an abnormal factor exists in the normal skeletal development of these breeds from a number of coarser anatomical traits in their build (the extremities of the dachshund, and the extremities and receding nose root of the French bulldog and the pekinese), as well as from my own histological studies of ribs and of the epiphyses of the long bones in new-born pups of different breeds. These studies will be referred to in a later paper. Here I wish only to mention that these three breeds display, in contrast to many others, a tendency towards alteration of the endochondral ossification, of the same type as in chondrodystrophy. These three breeds will henceforth be referred to as "the chondrodystrophoid breed group".

The sex proportion for the same disease material lies as close to

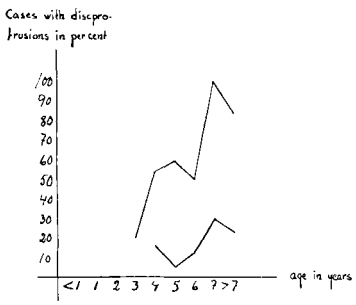

Fig. 1.
Age distribution of cases with disc protrusions. The upper curve concerns the chondrodystrophoid breeds; the lower concerns dogs of non-chondrodystrophoid type.

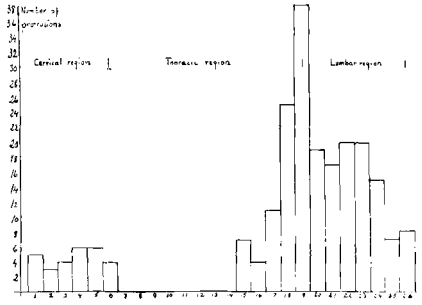

Fig. 2.
Distribution of disc protrusions within the vertebral column.

50 % as one can possibly expect if chance alone is the determining factor. The same percentage holds good for the mother population.

It is more difficult to judge age as a factor, since the post mortem material consists of animals that have died or been put to death in different stages of the disease. The curves in Fig. 1 demonstrate the percentage of prolapse cases for different ages taken from non-selective dog material. The upper curve concerns the chondrodystrophoid breeds; the lower concerns the breeds that can definitely be said to lack chondrodystrophoid traits. They support the conclusions on the disposition of breeds already demonstrated but are otherwise fairly parallel, with increasing frequency for increases in age. A few cases of disc prolapse have been observed in the chondrodystrophoid group as early as the age of three. Although the tendency is not statistically proved, we shall notice later that disc degeneration appears earlier with this breed group than with others.

The macroscopic investigation included the vertebral column as a whole after the vertebral arches and the spinal cord were taken off. After an inspection of the base of the vertebral canal, all the discs have been examined in vertical sections at right angles to the direction of the vertebral column. The protrusions discovered were solitary or multiple and were distributed within the vertebral column in the way shown in Fig. 2. This diagram shows how 217 protrusions from 88 cases are distributed between the different discs. The abscissa shows the vertebral column—1 is the disc between the second and the third cervical vertebra, 19 the thoracolumbar disc and 26 the lumbosacral. The diagram has three sections: a cervical with comparatively low frequency of protrusions; a thoracic section in which protrusions are

Fig. 3.
Disc from the thoracic region, showing the broad *lig. conjugale costarum*. Nucleus is the seat of a dystrophic calcification with tendency to expansion in dorsal direction.

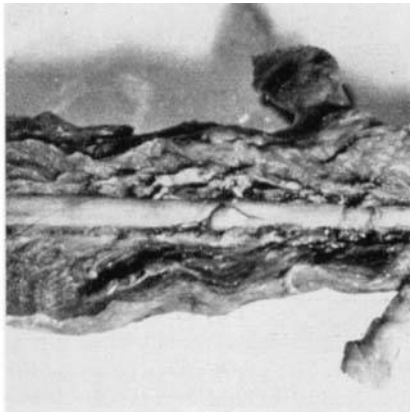

Fig. 4.
Chondrodystrophoid dog with a large protrusion of the disc between L. 2 and L. 3. The vertebral arches, but not the spinal cord, are taken off.

entirely absent; and finally, the thoracolumbar, where the frequency is greatest. The thoracic vertebral column in dogs consists of 13 thoracic vertebrae and contains discs 6-18. The 9 sternal ribs lead toward the intervertebral joints 6-14. With the exception of the first pair of ribs, the heads of the ribs on the right and on the left are connected by the so-called *ligamentum conjugale costarum* which extends as a broad cartilage-like band right under the *ligamentum longitudinale internum*, forming a roof over the discs. (See Fig. 3). The ligament corresponds to the human *ligamentum capituli costae interarticulare. The presence of this ligament is the only reasonable explanation for the absence of protrusions in this field, since the prerequisite conditions, i.e. nucleus- and annulus-degeneration and partial rupture are of common occurrence even in this section.*

The diagram in Fig. 2 shows the great frequency of protrusions in the thoracolumbar section with a very marked top level for disc No. 19, that is, the one found between Th 13 and L. 1. It may be as well to point out this difference in predilection for location of disc degeneration in human beings and in dogs. This fact determines what kind of clinical expression the disease assumes. In dogs, the disease is clinically especially marked by compression of the spinal cord. Initial symptoms and minor abortive cases show greater similarity to lumbago in human beings than the serious cases, where paraplegia, not sciatica, characterizes the disease. Prolapse within the vertebral

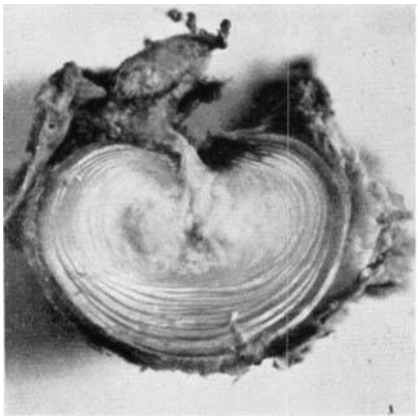

Fig. 5.
The disc between L. 2 and L. 3 from the dog mentioned above in Fig. 4. The picture shows the large proportions of the protrusion and demonstrates clearly its origin from nucleus.

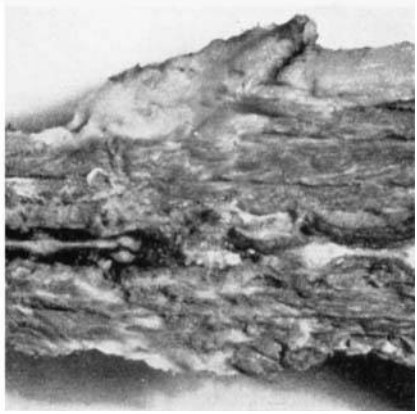

Fig. 6.
Chondrodystrophoid dog with protrusion from the disc between Th 13 and L. 1. The protrusion is long and broad, but rather low.

canal may be either solitary or multiple. They are more often multiple and in our material, the average number is about 2.5. In the case of multiple protrusions, they are often located in neighbouring discs. As I have mentioned before, they are an expression of the degeneration of the discs. The multiplicity of protrusions then becomes an expression of the multiplicity in disc degeneraton. The tendency shows up even more clearly if one takes into account other forms of expression. If this is done a vertebral column is practically never found where only one disc is diseased. *In other words, disc degeneration is a definite systemic disease in dogs.*

The macroscopic appearance of these dorsal protrusions in dogs varies for the most part along two lines—an observation which Tillmanns (1939) and Fankhauser (1948) made earlier. One type as a rule assumes rather large size—up to the main part of the vertebral canal's profile or, if the spread has occurred in a more horizontal direction, the length of a vertebra. It is characterized by an uneven, rough surface which as a rule is adherent to the dura in a fibrous or fibrinous way, of irregular form, of brittle granular consistency and greyish red to greyish yellow in color. In more chronic cases, they may become more stable with firmer consistency, but with ossification only in extreme cases. They are placed either as a median ridge-formed elevation of the *ligamentum longitudinale* or as bud-shaped protrusions on the side of this. This type of disc protrusion seems to be a

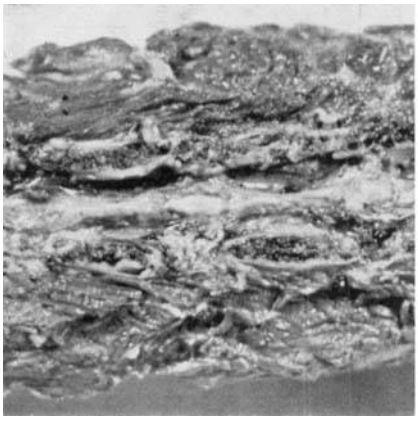

Fig. 7.
Protrusion from the disc between L. 2 and L. 3 forming a median ridge-formed elevation of *lig. longitudinale internum.*

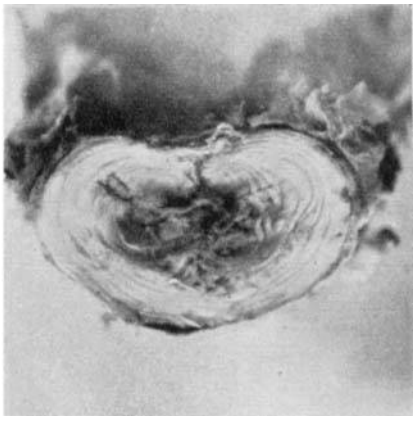

Fig. 8.
The disc between L. 3 and L. 4, seat of a severe degeneration of the nucleus and ruptures of the annulus. The dorsomedian rupture is T-formed, causing a slight protrusion on the right side of the median plane.

purely senile phenomenon without any greater clinical significance and may be found within all breeds. However, these can in some cases reach greater proportions with a compression of either the spinal cord and/or of the nerve root as a result, and it is in this form that clinical cases of prolapse may appear in other breeds than the chondrodystrophoid dogs. However, the former type is typical for the chondrodystrophoid breeds.

On the surface of the section of the involved discs, a prolapse route could be followed in all cases, sometimes at first only histologically, with the aid of a series of sections; in the latter cases the prolapse routes were often rather narrow and twisting. The annulus rupture is often located quite close to one of the two vertebrae and has a radial course in a dorsomedian or a faintly dorsolateral direction. The dorsomedian ones sometimes have a T-form. In some cases the prolapse has originally begun through a ventral rupture of the inner annulus layers and afterwards has dissected itself in a dorsal direction with a protrusion toward the vertebral canal as a result. As I have mentioned before, many more discs show degenerative pictures than those affected by prolapse. These take the form of calcification processes, splitting or break-down of the inner layers of the annulus and of the nucleus. Finally, many more than these show microscopic signs of degeneration, thus supporting the impression that here we have to do with a systemic disease and not with a limited and local process of disease.

The result of the histological investigations will be given here in brief.

It is well known that in human beings the disc undergoes certain changes from decade to decade. These changes have been called regressive and it has sometimes been difficult to determine how much change should be assumed to lie within the range of normal physiological processes. Friberg and Hirsch (1949) believed that definite degeneration must be presumed when ruptures are to be observed in the annulus. It is obvious that a pathologic-anatomical study of intervertebral discs should be an investigation of these from age-group to age-group, in order to be able to determine when definite pathologic changes appear. If one does this in dogs, it will be discovered that the metamorphosis of the nucleus from the gelatinous and strongly hydrous form of the infancy period to the more cartilaginous form of old age shows itself to be somewhat different in dogs with a tendency to chondrodystrophy than in others. In the former not only is there an increase of collagen fibers but also a powerful cell proliferation with the appearance of a non-gelatinous chondroid mass, and moreover this metamorphosis begins at a much earlier stage, often at the age of 3 to 4 months. In these dogs it is exceptional to find even a trace of the original nucleus tissue after the age of one year. The cells as well as the intercellular substance in this chondroid nucleus have structural and tinctorial resemblances to youthful hyaline cartilage. This chondroid nucleus and the inner layers of the annulus very soon become the seat of degenerative changes, resulting in larger and smaller areas of an amorphous, often completely uncellular necrosis, in most cases calcified in a diffuse or scaly fashion. This form of nucleus-metamorphosis *may* also be observed in dogs of other breeds, where the more apparent chondrodystrophoid traits are lacking. By far the most usual development of the nucleus in the latter group will most likely be comparable to the changes in a human being described as a successive dehydration and collagenisation and at advanced age a degenerative decline of the transformed nucleus and of the inner layers of the annulus. In both instances, through ruptures or the breaking away of or within the lamellae, degenerated material can thereafter make its way toward the periphery at varying distances. This happens sometimes without change in the contours, and at other times results in a bending outward of the outer lamellae and at last even in a complete perforation.

From the histological viewpoint, the prolapse in the latter case is developed in part by the amorphous and acellular nucleus- and annulus necrosis calcified either in a diffuse or scaly fashion; in part

by the still vital, even, proliferating chondroid tissue; and finally by a reactive inflammatory tissue of very imposing intensity. In cases which are not too chronic the latter is very rich in vessels and has a large content of fibroblasts, mononuclear cells, and in many cases polynuclear giant cells. The picture then exhibits the character of a foreign body reaction. Lindblom and Hultquist (1950) were not able in their cases to prove any foreign body reaction with giant cells. So far as observations of the dynamics of prolapse have been made, these have indicated that a decrease of volume can occur through a reabsorbing activity from the surrounding granulation tissue and reduction of the inflammatory reaction. The question whether a prolapse can increase in size is of extraordinary practical and clinical significance. Olsson (1950) has observed repeated relapses in cases with only one prolapse, and in my own material are many cases with more attacks of illness than number of protrusions. For a long time it has been suspected that progressive changes bear the responsibility for the growth of protrusions; this has been expressed in the view that they are of blastomatous nature and as far as dogs are concerned, in the fact that the disease has been named "enchondrosis intervertebralis". According to my view the proliferative processes played their role in most of the cases when the disease reached the prolapse stage. Their significance for minor increases in volume cannot be entirely neglected, however. In some cases it looks as if the prolapse has developed in stages, in such a way that a prolapse which has been bridged by connective tissue has in its turn been ruptured.

Disc degeneration in dogs is a systemic disease with high frequency even for the relatively early years. One cannot accept the disease as a necessary evil brought about by purely mechanical factors. Mechanical factors may to a great extent be contributary and releasing in effect and doubtless determine the distribution of the changes within the spinal column. The disease appears especially in three breeds (dachshund, French bulldog and pekinese), here grouped as "the chondrodystrophoid breed group" and within this group, expresses itself to some extent in characteristic forms. It seems reasonable to me that the disease in this group can be referred to a tendency that exists in the constitution itself. The metamorphosis of the nucleus which has been described in these animals is characterized by an early beginning and abundant cellular activity. The chondroid tissue that appears is similar to a youthful hyaline cartilage. Prader (1947) has pointed out that the perichordal tissue at a certain stage in the embryonic development of human beings is built up with hyaline cartilage which later partly changes to a mucoid tissue and partly to

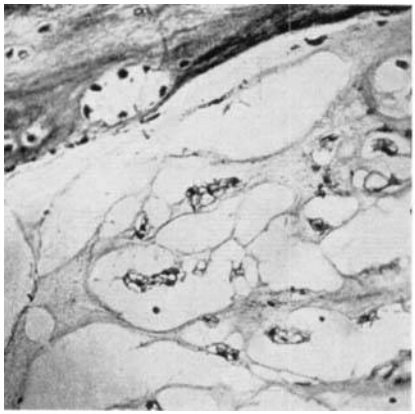

Fig. 9.
The nucleus, just on the border of the annulus, from a very young chondrodystrophoid dog. The picture shows the characteristic nucleus-cells with their vesicular cytoplasm and the mucoid intercellular substance. Azan stain (Heidenhain).

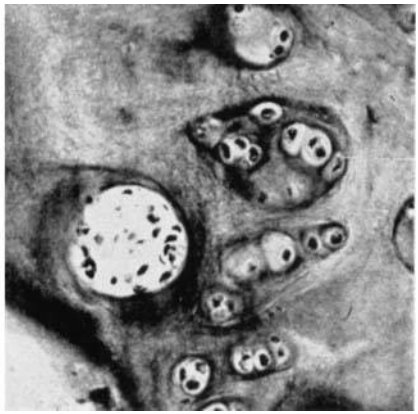

Fig. 10.
The nucleus from a chondrodystrophoid dog, 4 months of age. The picture shows a small cell group, that still has certain traits of original nuclear material. Except for this, however, a chondroid tissue is exhibited with pronounced cell progression. Hansen's htx—van Gieson pikrofuchsin.

fibrocartilage. The fact that one can find a tissue similar to hyaline cartilage within the nucleus- and the inner annulus region during postnatal life seems to me to mean that the development of discs in these individuals is delayed. The relatively high potential for growth may be explained by such a dedifferentiation as, according to Wilton (1933), characterizes the tissue-biological occurrence in rickets and chondrodystrophy. The early tendency to chondroid transformation of the nucleus causes this mesenchymal organ, so essential for the discs' normal functioning, to lose the capacity to fulfill its purpose. The disc can no longer function as a shock absorber, whereby many small stresses may have a traumatic effect on the disc and, as a result, lead to degeneration of the nucleus and annulus, and to ruptures and protrusions.

It is clear from this study that disc degeneration is a centrifugal process and that in chondrodystrophoid dogs it may very likely be referred to the early chondroid metamorphosis of the nucleus. Bradford and Spurling (1945) also hint in their book that the injuries to the annulus are to a great extent the result of the failure of the nucleus to function properly. The chondroid metamorphosis of the nucleus, which is characterized by a cell progression, has come to play a primary role in this attempt to formulate a hypothesis concerning

the pathogenesis of the most important disc injuries in dogs. It seems to me, therefore, as if the old name "enchondrosis intervertebralis" contains a good deal of truth.

SUMMARY

In his introduction the author discusses the disease, "enchondrosis intervertebralis" in dogs, long well-known to us.

In a very brief historical review the author is able to establish a very clear morphological correspondance between this affection and disc degeneration in man.

A statistical study is compiled of the frequency of disc degeneration in different breeds, with reference to different ages and sex. The most important result of this investigation proved the existence of a particularly pronounced disposition towards this affection in the breeds, dachshund, French bulldog, and pekinese. Certain characteristics of chondrodystrophy are found as common denominators of these breeds.

The pathological-anatomical examination of a large series of dogs, including 88 cases of disc prolapse amongst others, shows:—

that disc prolapse presupposes disc degeneration,

that disc degeneration exists in the spinal column much more generally than the number of prolapses allows us to suppose,

that the prolapses are distributed within the spinal column in accordance with the diagram in Fig. 2.

that the prolapses, as the most serious manifestations of disc degeneration, are due to mechanical factors as far as localisation within the spinal column is concerned, but—

that the multiple localisation argues in all other respects that disc degeneration in dogs is a definite systemic disease,

and also, that the tendency of the discs to disease is probably to be explained by the predisposition of the above breeds to chondrodystrophy.

In an attempt to formulate a hypothesis concerning the pathogenesis of disc degeneration, the author draws this last conclusion on the basis of his histological examinations of disc development from age group to age group.

RESUME

L'auteur présente à titre d'introduction l' «enchondrosis intervertebralis », maladie connue depuis longtemps chez les chiens. Dans un historique sommaire de cette maladie, l'auteur constate qu'il existe

un parallélisme frappant entre celle-ci et la dégénération discale chez l'homme.

Il fournit une étude statistique sur la fréquence de la dégénération discale dans les différentes races, par classe d'âge et de sexe. Le plus important résultat de cette enquête réside dans le fait qu'on a trouvé une disposition particulièrement prononcée à la maladie chez les bassets, les bouledogues et les pékinois. Certains traits de chondrodystrophie sont le dénominateur commun à ces races.

Une enquête anatomo-pathologique portant sur un grand nombre de chiens et qui compte entre autres 88 cas de prolapsus discal montre :

que le prolapsus discal suppose toujours l'existence d'une dégénération discale,

que la dégénération discale est beaucoup plus commune dans la colonne vertébrale que ne pourrait le faire supposer le nombre des prolapsus,

que les prolapsus se répartissent dans la colonne vertébrale conformément au diagramme de la fig. 2.,

que les prolapsus qui sont l'expression la plus grave de la dégénération discale sont probablement dus à des facteurs mécaniques lorsqu'ils sont localisés dans la colonne vertébrale,

que la forte extension des modifications semble toutefois indiquer que la dégénération discale chez les chiens est un système de maladie définitif

et que la disposition à la maladie doit être vraisemblablement imputée au caractère chondrodystrophoïde des races prédisposées.

C'est en essayant de formuler une hypothèse sur la pathogénie des dégénérations discales que l'auteur tire cette dernière conclusion sur la base de recherches histologiques sur le développement des disques d'un groupe d'âge à l'autre.

ZUSAMMENFASSUNG

In der Einleitung bespricht der Verfasser die bei Hunden schon seit langem bekannte Erkrankung der „Enchondrosis intervertebralis". In einem kurz gefassten historischen Rückblick kann der Verfasser einen ganz augenfälligen morphologischen Parallelismus zwischen diesem Leiden und der Bandscheibendegeneration beim Menschen feststellen.

Eine statistische Studie über die Häufigkeit der Scheibendegeneration bei verschiedenen Rassen, verschiedenem Alter und Geschlecht wird vorgelegt. Das wichtigste Ergebnis dieser Untersuchung ist der Nachweis einer besonders ausgesprochenen Disposition für das Leiden

bei den Rassegruppen der Dachshunde, französischer Bulldoggen und Pekineser. Der gemeinsame Nenner für diese Rassen ist ein gewisser chondrodystrophischer Einschlag.

Die pathologisch-anatomische Untersuchung eines grösseren Materiales von Hunden, das, unter anderem, 88 Fälle von Diskusprolaps umfasst, zeigt,

dass der Diskusprolaps immer eine Diskusdegeneration voraussetzt,

dass eine Diskusdegeneration viel häufiger vorliegt, als die Anzahl der Prolapse vermuten lässt,

dass die Prolapse sich in Übereinstimmung mit dem Diagramm in Fig. 2 über die Wirbelsäule verteilen,

dass die Lokalisation der Prolapse, ebenso wie der schwersten Erscheinungen von Diskusdegeneration, innerhalb der Wirbelsäule, wahrscheinlich durch mechanische Ursachen bestimmt wird, aber

dass die starke Ausbreitung der Veränderungen im übrigen dafür spricht, dass die Diskusdegeneration beim Hunde sicher eine Systemerkrankung ist, ferner

dass die Ursache der Krankheitsanfälligkeit der Zwischenwirbelscheiben wahrscheinlich in dem chondrodystrophoiden Gepräge der disponierten Rassen zu suchen ist.

In einem Versuch eine Hypothese über die Pathogenese der Diskusdegeneration aufzustellen, legt der Verfasser diese letzte Schlussfolgerung auf Grund histologischer Untersuchungen der Entwicklung der Bandscheiben von Altersgruppe zu Altersgruppe vor .

REFERENCES

Alajouanine, T. and *D. Petit-Dutaillis:* Presse Méd. 38: 1657, 1930.
Boddie, G. F.: The Vet. Rec. 61: 511, 1949.
Bradford, F. K. and *R. E. Spurling:* The Intervertebral Disc. Charles C. Thomas, Springfield, Illinois, 1945, Second Ed.
Dexler, H.: Zschr. f. Tierheilkunde 7: 1, 1896.
Elsberg, C. A.: Bull. Neurol. Inst. New York 1: 350, 1931.
Fankhauser, R.: Schweiz. Arch. f. Tierheilkunde 90: 143, 1948.
— Schweiz. Arch. f. Tierheilkunde 90: 494, 1948.
— Schweiz. Arch. f. Neurologie und Psychiatrie 62: 415, 1948.
Friberg, S. and *C. Hirsch:* Acta Orthopaed. Scand. 19: 222, 1949.
Jakob, H.: Münch. tierärztl. Wschr. 54: 305, 1910.
Joest, E.: Spezielle pathologische Anatomie der Haustiere. II Band, Berlin 1921.
Lindblom, K. and *G. Hultquist:* J. Bone and Joint Surg. 32 A: 557, 1950.
Mixter, W. J. and *J. S. Barr:* New England J. Med. 211: 210, 1934.
Olsson, S. E.: Nord. Vet. med. 2: 1034, 1950.
Prader, A.: Acta Anatomica 3: 115, 1947.
Riser, W. H.: The North Am. Vet. 27: 633, 1946.

Schmorl, G.: Verhandl. d. deutschen orthop. Gesellsch. 21: 3, 1927.
— Zentralblatt f. Chir. 55: 2305, 1928.
— Fortschr. a. d. Geb. d. Röntgenstrahlen 40: 629, 1929.
Tillmanns, S.: Beiträge zur Enchondrosis intervertebralis und den Wirbelkanaltumoren des Hundes. Diss. 1939, Giessen.
Wiedemann, R.: Ein Beitrag zur Enchondrosis intervertebralis und der dadurch bedingten Compression des Rückenmarks des Hundes. Diss. 1922, Hannover.
Wilton, Å.: Acta Path. et Microbiol. Scand. Suppl. XV, 1933.