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THORACO-LUMBAR DISK PROTRUSION WITH SEVERE CORD COMPRESSION IN THE DOG

I. CLINICAL AND PATHO-ANATOMIC OBSERVATIONS WITH SPECIAL REFERENCE TO THE RATE OF DEVELOPMENT OF THE SYMPTOMS OF MOTOR LOSS

By
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Thoracic disk protrusions with compression of the spinal cord are considered in human medicine as a rule, to be an indication for immediate decompression by laminectomy (*Schneider* 1949, *Love and Kiefer* 1950, *Logue* 1952, *Epstein* 1954, *Abbott and Retter* 1956, *Tovi and Strang* 1960, *Arseni and Nash* 1960, *Austin and Roth* 1961). The decompression used to be accompanied by removal of the disk protrusion when, with consideration of the consistency and situation of the protruded disk substance, this could be done without risk of damaging the spinal cord (*Love and Kiefer* 1950, *Logue* 1952, *Abbott and Retter* 1956, *Tovi and Strang* 1960, *Arseni and Nash* 1960, *Austin and Roth* 1961). In those cases where the disk protrusion is very hard (*Müller* 1951, *Logue* 1952, *Austin and Roth* 1961), or where signs of necrosis in the spinal cord are observed (*Epstein* 1954), the disk protrusion is left, and the operation may be limited to a decompressive laminectomy.

To judge from the available literature, it appears that those authors who carried out laminectomy for disk protrusion in the dog, regarded the procedure principally as a means to remove

the prolapsed disk substance, while on the other hand, they gave less consideration to the immediate decompressive effects of the laminectomy (*Greene* 1951, *Redding* 1951, *Olsson* 1951, *Hoerlein* 1952, 1956 and 1960, *Vaughan* 1958). As a rule, the operations in these cases has been performed as hemilaminectomy. Some of the above authors (*Olsson* 1951, *Vaughan* 1958), regard both hemilaminectomy and complete laminectomy as too hazardous to be recommended as a treatment of disk protrusion in the dog. Furthermore, the risk is stressed, of a reversible spinal cord damage being converted into an irreversible condition by the trauma of the operation itself. This idea is shared by *Frauchiger* and *Fankhauser* (1957) who state, without however referring to any of their own investigations, that laminectomy in the dog, because of the anatomical circumstances, is a "recht heiklen Angelegenheit". *Hoerlein* (1956 and 1960) however, has a more positive attitude to the decompressive technique. Thus he has used hemilaminectomy combined with disk fenestration, as a method of treating thoracic and lumbar disk protrusion with acute paresis or paralysis in the dog.

Not even if the trauma from the operation is ignored, has agreement been reached on the therapeutic value of decompressive surgical interference for serious, acute, spinal cord compression. Thus it has been maintained, that the damage to the nervous element of the spinal cord would occur so quickly, that there would be nothing to gain by decompression (*Olsson* 1958 and 1960). *Tarlov*'s and coworkers (1953 and 1954) experimental investigations in the dog have however contributed greatly to the elucidation of this question, and thereby to the creation of a sound basis for the treatment in cases of severe spinal cord compression. Tarlov introduced into the posterior part of the thoracic epidural space, a small rubber balloon, which he filled with liquid at varying speeds, either to a determined volume, or until the animal showed complete motor and sensory paralysis of the hind quarters. The fluid in the balloon was removed after varying intervals, and the animals were studied for several months afterwards for the return of the function of the spinal cord. From Tarlov's results, the following appear to be of great importance in this present problem:

1. If the spinal cord is compressed so that complete motor and sensory paralysis occurs, the length of time available for a successful decompression is very much dependent on the speed of

development of the compression. If, on compression, symptoms are developed in the course of one or a few minutes, the decompression must be performed within two hours, if there is to be any prospect for the restitution of the function of the spinal cord. If the development time of the compression is lengthened, the time available is increased according to the data below:

The relationship between the speed of development of the compression, and the time available for successful decompression, (according to Tarlov and co-workers).

Time of development of paralysis (From the first signs of pain to the onset of complete sensory and motor paralysis).	*The period,* (calculated from the time of the onset of complete paralysis) *within which decompression must be carried out if complete restitution is to be assured.*
"Acute compression"[1])	2 hours
"Gradual compression"	
75 minutes	9 hours
20 hours	84 hours
48 hours	1 week

2. If the degree of compression is such that motor paralysis occurs, but sensory paralysis is not complete, there is some prospect of restitution after prolonged compression (1—3 weeks), and also in those cases where compression symptoms are developed quickly, (within a few minutes).

3. When the compression time is lengthened, but the experimental conditions not changed in other respects, and provided that the damage to the spinal cord does not become irreversible, as a rule, the first signs of improvement, as well as the possible restitution take place later. In those animals where a complete restitution of the motor function of the spinal cord occurred, the first signs of an improvement in the function of the spinal cord have been in accordance with the table below:

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[1]) The time of development of the compression is not given exactly in these cases, but can, taking into account the apparatus used, be estimated at a maximum of a few minutes.

The time of the appearance of the first signs of improvement, in animals which recovered from a spinal cord compression involving complete motor and sensory paralysis, (according to Tarlov and co-workers).

Time of development of paralysis.	*Duration of the compression.*	*Interval between the decompression and the first signs of improvement.*
"Acute compression"	10 minutes	2—3 days
„ „	30 „	15—20 days
„ „	50—120 minutes	20—30 days
"Gradual compression"		
75 minutes	max. 9 hours	1—13 days
20 hours	max. 84 hours	4—32 days
48 hours	max. 1 week	4—10 days

It appears to me, that points 1 and 2 in the above review of Tarlov's research results justifies a more optimistic attitude to surgical decompression as a method of treating intervertebral disk protrusion with severe symptoms of compression. The investigations reported below have aimed firstly at analysing the speed of development of the compression symptoms in a large disk protrusion material, and thereby, against the background of Tarlov's experiment, at judging the theoretical chances for effective decompressive treatment (part I). Secondly the aim has been to test in the clinic, decompressive operations of different types and to compare the results of this treatment with results from conservative treatment (part II and III).

MATERIAL AND METHODS

In most cases of disk protrusion included in this present investigation, the opinion concerning the first phase in the course of the disease had to be based principally on the information given by the animals' owners. In judging the speed of development of the symptoms of motor loss, I have therefore, in most cases, had to restrict myself to taking into account those symptoms which are easily observed, even by the layman (e. g. pain in moving, unsteadiness in the hind legs, inability to use the hind legs for walking).

Of those dogs which were clinically diagnosed as having

thoracic or lumbar disk protrusion and were treated at the Surgical Clinic of the Veterinary College, Stockholm, during the period 1959—1961, 230 cases, in some phase of the disease, showed paralysis or paresis to such a degree that they were unable to use the hind legs for walking. The material comprised 129 bitches and 101 males. The age distribution is recorded in Fig. 1. The breed distribution was as follows: Dachshund 210, Dachshund crosses 2, Pekingese 8, Cocker spaniel 4, French

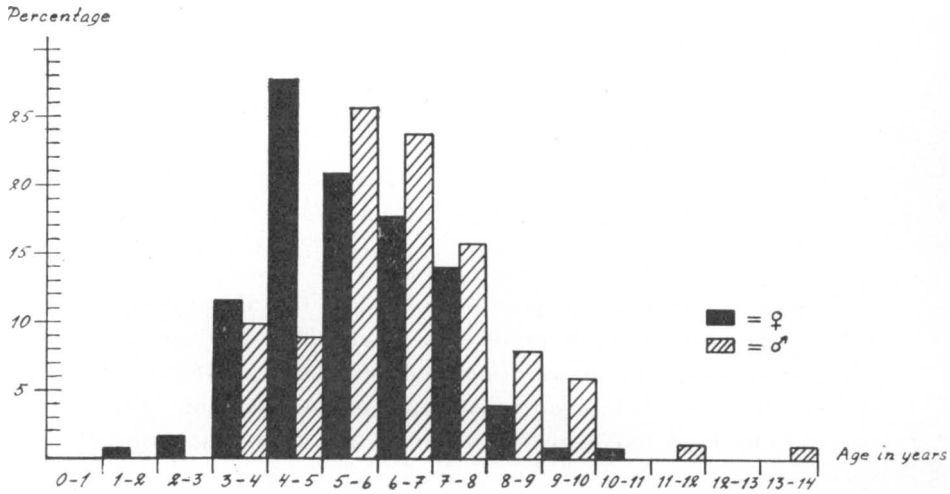

Fig. 1. Percent age distribution for male dogs and bitches. The percentage is calculated on the total number of animals of the respective sexes.

bulldog 1, Pug 1, "Dachsbrache" 1, Sealyham terrier 1, Fox-terrier 1, Boxer 1. In 205 of these 230 cases, it has been possible to obtain such information about the course of the disease to make it possible to determine approximately the interval between the time of onset of the symptoms of motor loss (unsteadiness observed by the owner of the animal) and the time at which the animal was no longer able to walk. In all these 205 cases, information has been obtained about the possible onset of prodromal symptoms in the form of signs of pain or unwillingness to move, in the days or weeks immediately before the onset of paralysis. The collected data for this material is given in Table 1 sections A and B.

Ten of those animals which on admission were certainly unable to walk, but which showed a certain degree of voluntary

Table 1. Time for development of the symptoms of motor loss to the stage of "inability to walk" from the onset of the first motor loss symptoms.

	Number of animals in the various groups	Development time							
		Unknown, but < 12 hours	Instantaneous or < 15 min.	90 min.–6 hours	6–12 hours	About 24 hours	About 48 hours	3–6 days	1–8 weeks
A. The total material.									
Number	205	64	31	25	12	33	12	14	14
Percentage		31.2 %	15.1 %	12.3 %	5.9 %	16.1 %	5.9 %	6.8 %	6.8 %
B. Animals under A with prodromal symptoms.									
Number	113	52	12	13	4	18	4	5	5
C. Animals under A which after conservative treatment either died or were destroyed.[1])									
Number	72	28	13	8	4	7	4	4	4
Percentage of the animals in group C		38.9 %	18.1 %	11.1 %	5.6 %	9.7 %	5.6 %	5.6 %	5.6 %
D. Animals under A, which after conservative treatment recovered.									
Number	48	13	10	4	5	9	4	2	1
Percentage of the animals in group D		27.1 %	20.8 %	8.3 %	10.4 %	18.8 %	8.3 %	4.2 %	2.1 %

[1]) Due to the prognosis being judged unfavourable.

movement, developed a complete motor, or motor and sensory paralysis during their stay at the clinic. In these animals, it has thus been possible to study in more detail, the continued development of the symptoms.

The 205 animals in the above material, have shown great variation in the matter of symptoms and patho-anatomic changes. Furthermore they have been treated by inter alia, varying surgical methods, which could be considered to have influenced the course of the disease in a positive or negative direction. In order to get an idea of the development of the symptoms in those cases where the damage to the spinal cord has definitely been very serious, and where the course of the disease can not have been greatly influenced by the treatment undertaken, I have from the total material, taken out and separately reported those cases (72 altogether) where, after conservative treatment, the animal either died (3 cases), from causes directly connected with spinal cord damage, (myelomalacia), or were destroyed, when the prognosis was deemed unfavourable. These animals (Table 1, section C), have — with reference to their distribution among the different degrees of development speed — been compared with those 48 animals within the total material which, with conservative treatment, recovered to such a degree that they were able to walk on their hind legs (Table 1, section D).

Of the animals in section C in Table 1, 67 have been subjected to patho-anatomic investigations by the method described in another paper (*Funkquist* 1962). In this material, it has been possible to classify the nature of the patho-anatomic changes in the epidural space, and to relate them to the speed of development of the symptoms and to the occurrence of certain spinal cord changes.

In the reporting of the patho-anatomic changes in the tables, I have used the following classification of the macroscopic appearance of the disk protrusions (*Funkquist* 1962). (All the protrusions have been of type I, according to the nomenclature used by *Hansen* 1952).

Type 1: Sharply defined "button-shaped" protrusion over one disk and parts of adjacent vertebrae.

Type 2: Protrusion as in 1, combined with extension of disk-substance like a carpet over adjacent vertebrae (vertebra).

Type 3: Extension of disk-substance like a carpet over several vertebrae.

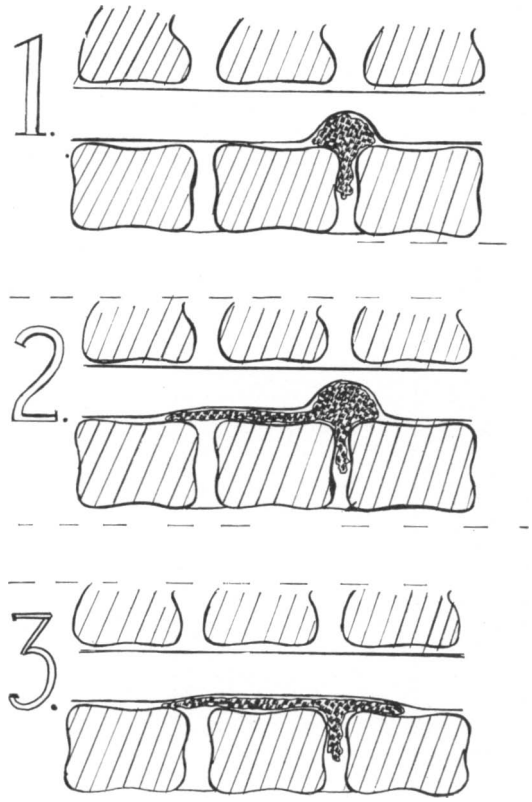

Fig. 2. Schematic presentation of the three principle types of disk protrusion as observed at patho-anatomic examination. See text page 262 concerning the classification. All types belong to Type I of Hansen's (1952) terminology.

The different types of disk protrusion described above are shown in Fig. 2.

The material subjected to patho-anatomic investigation (presented in Table 2) included 21 animals which either died or were destroyed within 1—7 days owing to ascending paralysis caused by a macroscopically observable myelomalacia which in most cases extended far beyond the limits of the area of the prolapsed disk-substance. In the remaining 46 cases, the symptoms were limited to paralysis of the hind legs. The macroscopically observable spinal cord changes in this part of the material, have been principally limited to the region of macroscopic changes in the epidural space. The observation time for the latter 46 animals

Table 2. The time for development of the symptoms of motor loss (to the stage of "inability to walk on the hind legs") in 67 dogs which were destroyed owing to thoracic or lumbar disk protrusion. The development time has been placed in the table in relation to the clinical picture and to the macroscopic patho-anatomic changes respectively.

Clinical symptoms and patho-anatomic changes [1]	Number of animals examined	Development time: Unknown, but < 12 hours	Instantaneous, or < 15 min.	90 min.–6 hours	6–12 hours	About 24 hours	About 48 hours	3–6 days	1–8 weeks
A. Paraplegia, complicated by ascending paralysis									
Disk protrusion type 1.									
„ „ „ 2.	1		1						
„ „ „ 3.	20	11	3	4	2				
Sum under A	21	11	4	4	2				
B. Paraplegia without the above complication.									
Disk protrusion type 1.	19	4	4		1	1	2	3	4
„ „ „ 2.	4	2				1		1	
„ „ „ 3.	23	10	3	4	1	3	2		
Sum under B	46	16	7	4	2	5	4	4	4

[1]) For terminology, see text page 262.

has been less than or equal to one week for 10 animals, 1—2 weeks for 10 animals, 2—4 weeks for 21 animals, 1—2 months for 4 animals and finally, 3 years for 1 animal.

RESULTS

As is shown in Table 1, in 64 (31,2 %) of all the 205 assessable cases, the animal has been found (by the owner) to be unable to walk with the hind legs after not having been observed during the 6—12 hours immediately before. As a rule (48 cases), these dogs have been found paralysed in the morning after having been completely free of symptoms the evening before (5 cases), or after showing slight prodromal symptoms without, however, symptoms of motor loss (43 cases). Of the 64 dogs in the group, it was possible to examine 21 on the same day paralysis was discovered, and of these, 6 cases showed certain voluntary movements in the hind legs at the first investigation. In 31 (15,1 %) of the 205 animals, the symptoms of motor loss while the animal was under observation (by the owner), almost instantaneously reached such a degree, that the animal could no longer walk. Only in 4 of these 31 animals did the examination take place on the same day as the paralysis occurred, at which time 2 of these showed certain remaining voluntary movements in the hind legs. In 37 cases (18,0 %), the development of the symptoms to "inability to walk", occurred in the course of 6—12 hours. In this group, 8 were examined the same day as the paralysis occurred, and in 6 of these certain residual voluntary movements existed. Corresponding development of symptoms in 33 animals (16,1 %) occurred in the course of 24 hours. Sixteen of these animals were examined the same day, and 10 of them showed certain voluntary movements. In 40 animals (19,5 %), the paralysis developed during 48 hours or more — this group including 26 cases within one week, 12 cases during 1—2 weeks, and 2 cases during 1—2 months. In this group of 40 animals, of the 17 which were examined on the same day as they lost their ability to walk, 9 showed residual voluntary movements.

Of the total material of 205 animals, 113 have shown, during varying periods (24 hours—3 weeks) before the appearance of the symptoms of motor loss, pain at lifting or unwillingness to move. This period with prodromal symptoms, was sometimes interrupted by a few days of freedom from symptoms, before

the condition grew worse and symptoms of motor loss began to appear. The distribution of these prodromal symptoms over the different groups is given in Table 1B.

The interval between the stages of walking inability and complete motor paralysis of the hind legs, has been, in all the 10 animals which could be examined in this respect, 1 and 5 hours respectively in 2 animals, and 1—4 days in 8 animals. In 4 of these animals, sensory paralysis did not occur. In 4 animals, sensory paralysis occurred about the same time as motor paralysis, while in 2 remaining animals, the interval between the time of complete motor paralysis and the time of sensory paralysis was 3 hours and 24 hours respectively.

The conservatively treated animals which either died or were destroyed in connection with the condition of disk protrusion (Table 1C) showed, in comparison with those animals, which after conservative treatment regained the ability to walk with the hind legs (Table 1D), a slight tendency towards the groups with a quicker development of symptoms of motor loss. Thus, in 73,6 % (53/72) of the former animals, the development to the stage of inability to walk on the hind legs occurred within 12 hours, while the corresponding percentage for the latter animals is 66,7 % (32/48).

In a closer analysis of the 67 animals in section C in Table 1, which were subjected to patho-anatomic investigation (Table 2), the following was found: The 21 animals which died or were destroyed owing to *ascending paralysis* (Table 2 A), differed from the remainder of the material as regards the distribution in the different groups, in that all the animals showed a development time for the symptoms of motor loss (to "inability to walk") which was equal to or less than 12 hours. Furthermore, all the animals, with respect to the macroscopic appearance of the disk protrusion, have belonged to groups 2 (1 case) and 3 (20 cases), meaning that in all the cases, there was disk-substance diffusely spread out over one or more vertebrae. Five of the 21 animals were examined the same day as the paralysis set in or was discovered, and in none of these was there any voluntary movement present.

Within the group of animals which either were destroyed or died (totally 46 animals), where paraplegia existed *without signs of ascending paralysis* (Table 2 B), the distribution over the different degrees of development speed agreed more closely with

Table 3. The type of the disk protrusion in relation to the case history (with special reference to the existence of protracted prodromal symptoms and previous attacks of disk protrusion which could be assigned to the current disk[1]).

Case history	Total number of assessable cases	Type 1	Type 2	Type 3	Percentage occurrence of types 1 + 2
a. Acute development of the paralysis (< 4d). No previous attacks. Prodromal symptoms < 1 week.	40	5	1	34	15.0 % (6/40)
b. Acute development of the paralysis (< 4d). No previous attacks definitely assigned to the current disk. Prodromal symptoms > 1 week.	11	5	3	3	
c. Acute development of the paralysis (< 4d). Previous attacks assigned to the current disk (irrespective of the possible existence of prodromal symptoms in the last attack).	7	3	1	3	72.7 % (16/22)
d. Subacute development of the paralysis (during a longer period than 1 week) irrespective of the occurrence of previous attacks or prodromal symptoms.	4	4			

[1]) Those animals being excluded where patho-anatomic changes at other disks could be responsible for prodromal symptoms or previous attacks.

the total material (Table 1 A) and with those animals respectively, which after conservative treatment regained the ability to walk (Table 1 D). The form of disk protrusion which is characterized by disk-substances spread out over one or more vertebrae (type 3), also dominates this material as it exists in 23 cases (50 %). The restricted "button shaped" disk prolapse (type 1) was next in frequency as it existed in 19 cases (41,3 %), while the transition form (type 2), only included 4 cases (8,7 %). The development of symptoms of motor loss in disk protrusion of type 1,

has been principally along two lines: Inability to walk has thus occurred in 9 cases within 12 hours and in 9 cases after more than two days; in 4 of the lastmentioned 9 cases, the time of development was more than one week. The development of symptoms of loss in type 3 on the other hand, similarly to those cases with ascending paralysis, mainly occurred within 12 hours (in 18 cases of 23). The transition form type 2, shows a quick development of the compression symptoms in 2 cases, but is also found in the groups with slower development (2 cases). Only in 4 of the 46 cases in Table 2 B has it been possible to ascertain the neurological status the same day as the inability to walk occurred. Of these 4, two have shown a certain capacity for voluntary movement.

The observed types of disk protrusion in the patho-anatomically examined material, have in Table 3 been set out in relation to the occurrence of such earlier clinical symptoms as could be assigned to the current disk by the method of elimination. The table shows that the disk protrusions arising from those disks with a history of disease (including earlier attacks) which is longer than 1 week (b, c, d taken together) were in 72,7 % of types 1 or 2, while the corresponding percentage for disks with a shorter history of disease than 1 week (group a in the table) was 15,0 %.

DISCUSSION

Tarlov's experiments described above makes, as stated previously, the most valuable contribution up to the present to the question of the therapeutic value of surgical procedures for space occupying disease processes in the epidural space with spinal cord compression. Tarlov's experimental conditions differ in the following respects from the conditions which apply in disk protrusion in the dog.

1. The balloons used in the experiments were situated dorsally or dorso-laterally in the vertebral canal, while in those forms of disk protrusion, where the prolapsed disk substance forms a compact protrusion, the spinal cord compression occurs as a rule, from the ventral side.

2. Those forms of disk protrusion which are characterized by disk-substances spread out over large parts of the epidural space, have no equivalent in Tarlov's experimental arrangements.

3. In those experiments where conditions are in best agreement with the situation in disk protrusion ("gradual compression"), the expansion of the balloon was stopped when complete motor or motor and sensory paralysis occurred. In an attack of disk protrusion, one must of course reckon with the fact that the degree of compression can also increase after complete paralysis occurs, through more disk substance being forced out and/or by means of the inflammatory reaction in the surrounding of the disk substance.

The differences enumerated above, however, do not appear to decrease the fundamental importance of Tarlov's work, for the judgement of the theoretical possibilities of there being time to carry out a surgical decompressive operation in an attack of disk protrusion with severe symptoms of motor loss, before the spinal cord damage becomes irreversible.

If the results of the reported anamnestic and clinical studies are placed in relation to Tarlov's experimental investigations, one finds (with reservations concerning the above differences in the mechanism initiating the injuries), that in 53,7 % (110/205) of the cases, the time available for a surgical decompression is more than 9 hours, and in 35,6 % (73/205) more than 84 hours, even if one regards the development time for the symptoms, only as the interval between the onset of the symptoms of motor loss and the time at which the animal is no longer able to walk with the hind legs, and if one assumes that the paralysis at this latter time is complete. In the few cases where the development time of the compression symptoms was able to be followed in more detail, the interval between the stages "inability to walk" and the onset of complete motor paralysis, was however, several hours up to several days. In reality, the time available to the surgeon will presumably in many cases be considerably longer than that given above. It should be stated furthermore, that most of those animals which are not included in the above group with 9 hours available for decompression, are animals of whom the exact development time is not known, and thus the 9 hours group may include a substantially larger part of the material.

One can of course object to the above reasoning on the grounds that those cases, where the compression is greatest (and where a decompressive operation is more indicated), could possibly belong to the group where the development is most rapid,

and where the possibilities of there being time to perform a decompression are consequently least. The material reported in Table 1C and Table 2 included animals which, during conservative treatment died or were destroyed (because the prognosis was deemed unfavourable), and this indicates that the objection is justified to a certain degree, as this material concerning the distribution among the different degrees of development speed (of the symptoms of motor loss), shows a slight bias towards the groups with rapid development of symptoms, so that less than half the animals fall with certainty within the "9 hours group". On the other hand it should be stated that those animals, of whom the development time is not known with certainty, make up a very large proportion (about 40 %) of the cases in Table 1C, and so it is not inconceivable that also in this selected material with severe spinal cord compression, in the majority of cases, there should be time to perform a surgical decompression, before the damage to the spinal cord could become irreversible.

Studies of the relationship between the case history and the appearance of the disk protrusion seem to justify the following comments: In experimental transvertebral injection into the epidural space of a substance which has a consistency comparable to that of degenerated disk substance, e. g. paraffin wax, heated to a salve-like consistency, the substance injected shows a marked tendency to spread along the length of the epidural space in the same way as the disk substance in disk prolapse type 3 (own unpublished investigations). In using injections of paraffin wax in order to bring about a *localized* compression of the spinal cord, it is necessary for this reason, to limit the spread of the paraffin in the epidural space, e. g. by injecting into a bladder of rubber (*Funkquist* 1961), or polythene (*Funkquist and Schantz* 1962). Table 3 on the other hand shows, that those cases of disk protrusion, where there is a tendency for the disk substance to be concentrated in the area immediately surrounding the disk (types 1 and 2), have as a rule, longer lasting prodromal symptoms (including earlier attacks which could not be referred to lesions of other disks), than those cases of disk protrusion, where the prolapsed disk substance is spread over large areas of the epidural space. With the background of these anamnestic studies and the above experimental observations, it should be possible to explain the variations in the appearance of the disk protrusion thus: disk prolapses of types 1 or 2, occur when disk substance is forced

into an area of the epidural space where there already exist older (= proliferative) pathological changes which prevent the lengthwise spreading of the substance, while disk protrusion of type 3 with a tendency towards the spreading of the disk substance over large areas, occur in previously intact parts of the epidural space.

Regarding the relationship between the appearance of the disk protrusion and the tendency to extensive myelomalacia, the following is relevant: The frequency of myelomalacia was 20/43 in protrusions of type 3 and 1/5 in protrusions of type 2 while this type of cord changes were not observed in the 19 cases with disk protrusion of type 1. In disk protrusion of type 3 the deformation of the cross-section of the spinal cord is, as a rule, nonexistent or unimportant, while on the other hand, the pronounced inflammatory reaction in the epidural space which usually occurs around the prolapsed disk substance (*Hansen* 1952) can be supposed to change the contents of the affected part of the epidural space into a "pressure cuff", which concentrically surrounds a relatively long part of the spinal cord. In such concentric compression of a longer part of the spinal cord, there is reason to suppose that the disturbances of circulation play a larger role than in compressions caused by a sharply delimited disk protrusion, since in the former type of compression there may be fewer possibilities for collateral circulation. It seems reasonable to assume that the tendency to myelomalacia in disk herniation of type 3 depends on this supposed disturbance of circulation (c. f. *Olsson* 1960). This assumption is in good agreement too with the results of experiments performed by *Woodard and Freeman* (1956) who observed similar changes when producing ischemia by ligation and section of 6 adjacent pairs of thoracic radicular blood vessels together with the nerve roots. Tarlov's statement (*Tarlov and co-worker* 1954) that mechanical deformation and not circulatory disturbances play the most important role in the pathogenesis of the damage of the spinal cord is not in disagreement with my assumption, as Tarlov used the sharply delimited form of compression in his experiments.

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SUMMARY

The development speed of symptoms of motor loss in 205 dogs with thoracic or lumbar disk protrusion with severe spinal cord compression has been studied. The results have been considered in relation to Tarlov's experimental investigations into the prospects of restoring the function of the spinal cord after a temporary spinal cord compression of varying degree, development speed, and duration. The results indicate that in most cases of disk protrusion there should be sufficient time available to perform a surgical decompression before the damage to the spinal cord becomes irreversible.

ZUSAMMENFASSUNG

Thorako-lumbale Bandscheibenbrüche mit schwerer Rückenmarkskompression beim Hund.

I. *Klinische und pathologisch-anatomische Beobachtungen mit besonderer Berücksichtigung der Entwicklungsgeschwindigkeit der motorischen Wegfallssymptome.*

Die Verf. hat die Entwicklungsgeschwindigkeit der motorischen Wegfallssymptome bei 205 Hunden studiert, die an thorakaler oder lumbaler Bandscheibenprolaps mit starken Wegfallssymptomen erkrankt waren. Die Ergebnisse sind mit den experimentellen Befunden von Tarlov verglichen, der die Möglichkeiten für Restitution der Rückenmarksfunktion nach übergehender Kompression von variierender Stärke, Entwicklungsgeschwindigkeit und Dauer studiert hat.

Die Ergebnisse dieser Vergleichung deuten darauf, dass es möglich wäre eine chirurgische Dekompression auszuführen, bevor die Rückenmarksschädigung irreversibel geworden ist.

SAMMANFATTNING

Thorako-lumbala diskbråck med svår ryggmärgskompression hos hund.

I. *Kliniska och patolog-anatomiska observationer med särskild hänsyntagen till utvecklingshastigheten för de spinale bortfallssymtomen.*

Utvecklingshastigheten för de motoriska bortfallssymtomen har studerats på 205 hundar med thorakalt eller lumbalt diskbråck med grav ryggmärgskompression. Resultaten ha ställts i relation till Tarlovs experimentella undersökningar över utsikterna till restitution av ryggmärgsfunktionen efter en temporär ryggmärgskompression av varierande grad, utvecklingshastighet och duration. Resultaten tala för att det i flertalet fall av diskbråck bör finnas möjlighet att hinna utföra en kirurgisk dekompression innan ryggmärgsskadan blivit irreversibel.

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