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DYNAMIC COMPRESSION OF THE CERVICAL SPINAL CORD

A MYELOGRAPHIC AND PATHOLOGIC INVESTIGATION IN GREAT DANE DOGS

By

Sten-Erik Olsson, Monica Stavenborn and *Fredrik Hoppe*

OLSSON, STEN-ERIK, MONICA STAVENBORN and FREDRIK HOPPE: *Dynamic compression of the cervical spinal cord. A myelographic and pathologic investigation in Great Dane dogs.* Acta vet. scand. 1982, *23*, 65—78. — The authors report the radiographic and pathologic findings in 10 Great Dane dogs with the wobbler syndrome. In all 10 dogs it was possible to demonstrate myelographically that there was cervical spinal cord compression at 1 or 2 sites. The spinal cord compression was mainly dynamic in nature, as degree of compression increased in extension and decreased in flexion of the neck in 8 dogs. In 1 dog with deformed vertebral bodies (C6 and C7), compression increased slightly in flexion of the neck. In another dog, compression was lateral and could only be seen in the ventrodorsal view.

The macroscopic findings substantiated the radiologic findings. The cause of the spinal cord compression was in 8 dogs a decrease in the dorsoventral diameter of the orifice of the vertebral canal of 1 or 2 vertebrae in combination with deformation and elongation of 1 or several vertebral arches. In extension of the neck, the cervical spinal cord was squeezed between the anterior tip of the elongated vertebral arch and the caudodorsal rim of the body of the adjacent cranial vertebra.

Histologic examination was made of the spinal cord in 5 dogs and the compressive lesions that were found could explain the neurologic signs.

In the discussion, the question is raised as to why pain is not a prominent sign in dogs with the wobbler syndrome in contrast to in dogs with cervical disc protrusion. It is believed that the inflammatory foreign body reaction, triggered by the protruded calcified nucleus pulposus is the main cause of pain in the disc protrusion syndrome. In the wobbler syndrome there is no obvious inflammatory reaction in the epidural space.

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Finally, the possible etiologic factors of importance for the deformation of the cervical vertebrae in wobblers are discussed. There are indications that both overnutrition and a genetic trait for rapid growth are of importance.

wobbler syndrome; cervical spinal cord compression; extension of cervical spinal; malformation of vertebrae.

Congenital, developmental and degenerative stenosis of the cervical and lumbar vertebral canal and the intervertebral foramina has long been known to exist in man. However, the clinical importance of these lesions has only been widely recognized during the last 15 years (*Hinck & Sachdev* 1966, *Adams & Logue* 1971, *Arnoldi et al.* 1976, *McIvor & Kirkaldy-Willis* 1976, *Paine* 1976, *Cauchoix et al.* 1976, and *Verbiest* 1977). *Naylor* (1979) made the observation that stenosis of the vertebral canal or of an intervertebral foramen may be clinically silent as long as it is not complicated by other lesions such as spinal instability or the bulging of a disc. Hence, in some cases of stenosis, the addition of a dynamic factor seems to be necessary for the occurrence of clinical signs.

In the dog, a similar observation has been made (*Olsson* 1958) in cases with compression of the spinal cord by disc protrusions. Chronic (stable) protrusions, often of considerable size, compressing the spinal cord are as a rule clinically silent, while small acute protrusions, bulging or increasing in size when intradiscal pressure is increased, i.e. being dynamic in nature, usually cause severe pain and neurologic deficits.

Cervical spinal cord compression in the dog caused by other lesions than disc protrusions, tumors and trauma was first reported in 1967 and a number of publications have followed. A syndrome in the dog, widely known as the wobbler syndrome, has been described as the result of vertebral canal stenosis or vertebral instability or a combination of both.

The wobbler syndrome in the dog is characterized by mild to severe ataxia and spastic paresis of the legs, primarily of the hind legs. The dogs with the syndrome have little control of the position of their limbs. This leads to an abducted and sometimes hypermetric gait with stumbling, particularly when turning, and occasional flexion of the digits with the dorsal surface of the paw placed on the ground. The condition is usually not painful. The spastic paresis is reported to be due to damage to the descending upper motor tracts and the ataxia to the lesion in the

ascending proprioceptive tracts, both lesions being caused by compression of the cervical spinal cord.

A number of Great Dane dogs with the above mentioned clinical signs have been examined by the present authors. The main reason for reporting the findings is that these dogs with the wobbler syndrome constitute a rather unique model for studying the dynamic factor in cervical spinal cord compression.

REVIEW OF THE LITERATURE

The first to describe vertebral canal stenosis as the cause of spinal cord compression in the dog were *Palmer & Wallace* (1967). They considered deformation of the vertebral bodies to be the cause of stenosis. A similar concept was held by *Geary* (1969), *Wright et al.* (1973), *Wolvekamp & Wentink* (1975), *Trotter et al.* (1976), *Denny et al.* (1977), *Chambers & Betts* (1977), and *Mason* (1979).

Deformation of the vertebral arches, including the articular processes and the intervertebral joints, was reported to be another cause of stenosis of the spinal canal (*Wright et al., Selcer & Oliver* 1975, *Trotter et al., Lord & Olsson* 1976, *Denny et al., Chambers & Betts, Wright* 1979, and *Rendano & Smith* 1981). Other changes reported as contributory to stenosis were hypertrophy of the ligamentum flavum (*Selcer & Oliver, Trotter et al.*), hypertrophy of the dorsal longitudinal ligament or dorsal annulus (*Selcer & Oliver*), and malarticulation (*Trotter et al.*).

Vertebral instability, also called vertebral subluxation or spondylolisthesis, was found to be another cause of compression of the cervical spinal cord, either alone or in combination with malformation of one or several vertebrae (*Geary, Gage & Hall* 1972, *Gage & Hoerlein* 1973, *Parker et al.* 1975, *Selcer & Oliver, Denny et al., Chambers & Betts,* and *Mason*). The diagnosis of instability of the cervical vertebrae was usually made on radiographs of the dog's neck in flexed position. Instability (subluxation, spondylolisthesis) was considered to be present when an increased degree of flexion was found between 2 vertebrae.

The validity of the diagnosis of instability has been questioned. *Wright* (1977) stated that an accurate diagnosis of stenosis of the cervical vertebral canal can not be made only on observations of abnormal angulation or "slipping". She found angulation or "slipping" on dogs without any clinical sign of cervical spinal cord lesion.

Most investigators seem to be of the opinion that it is sufficient to make the radiographic examination in lateral projection with the dog's neck in neutral position (corresponding to normal position when standing) and in flexion. Radiographs in extension have been reported to be of no or little diagnostic value as subluxation is considered to occur in flexion. Only one case in the dog has apparently been published (*Lord & Olsson*) in which it was demonstrated that a compression which was slight in neutral position of the neck increased in extension and disappeared in flexion. It should be mentioned that *Rendano & Smith* have emphasized the usefulness of the ventrodorsal view for myelographic demonstration of lateral compression of the cervical spinal cord.

MATERIAL AND METHODS

Twelve Great Dane dogs, 10 with the wobbler syndrome and 2 which were neurologically normal, were examined. All the dogs with the wobbler syndrome and 1 neurologically normal dog underwent radiographic examination, which included myelography. Nine of the 10 dogs with the wobbler syndrome and 1 of the normal dogs (not radiographed) were necropsied (Table 1).

Of the 10 dogs with the wobbler syndrome, 6 were males and 4 females, and their age ranged from 4½ months to 2 years. The 2 neurologically normal dogs (1 male and 1 female) were 14 months and 12 years old, respectively (Table 1).

After careful clinical examination, the dogs, with 1 exception (see above), were anesthetized and the vertebral column was radiographed. After the plain films had been taken, myelographic examination, using the suboccipital route, was made with Metrizamide (Amipaque, Nyegaard & Co AB, Oslo, Norway) as contrast medium. Films were taken in lateral projection of the recumbent dogs with the neck in neutral, flexed and extended position (Fig. 1). In 5 of the dogs, the ventrodorsal view was included in the examination. After completion of the radiographic examination, the dogs were killed with an i.v. overdose of nembutal.

In 8 of the 10 dogs, which were necropsied, the cervical vertebral canal was opened from the ventral aspect by sawing and cutting away the vertebral bodies. Thereafter, the spinal cord was removed and inspected. In 5 dogs, which were necropsied

within 1 h after death, the spinal cord was fixed in formalin. By opening the vertebral canal ventrally instead of dorsally, most of the walls and the entire roof of the canal were left intact and could be examined. Hence, any deviation of the lateral wall of the vertebral canal caused by hypertrophic or asymmetrically positioned intervertebral joints was easily detected. The next step in the macroscopic examination was to saw the vertebral arches along the median plane. The intervertebral joints were then opened. Specimens from the formalin fixed spinal cords were taken at the site of compression and at several segments cranially and caudally. The specimens were embedded in paraffin and cut in 6 μm thick sections. The following stains were used: H & E, van Gieson's stain and Weil's stain for myelin sheaths.

The necropsy procedure was different in 2 dogs, 1 with the wobbler syndrome and 1, which was neurologically normal. In these dogs, the cervical vertebral column was stripped of muscles and then boiled for about 8 h. After removal of all soft tissues, the intact vertebrae were then dried and inspected and some were sawed along the median plane.

RESULTS

Radiologic examination

The clinically normal dog (11), which was radiographed, had a normal radiographic and myelographic appearance (Fig. 1). As seen in many Great Dane dogs, the vertebral canal had a relatively smaller ventrodorsal diameter and the intervertebral joints were larger and more prominent than in many dogs of breeds with individuals of a similar or slightly smaller size. In this dog (11) there was an increased degree of flexion between C3 and C4 in flexed position of the neck, but the myelogram indicated that this did not cause any impingement on the spinal cord (Fig. 1).

On plane radiographs of the neck, conspicuous abnormalities were only seen in 3 dogs (6, 9 and 10). In Dog 6, the vertebral arches of C6 and C7 were elongated and deformed and the ventrodorsal diameter of the orifice of the vertebral canal of these 2 vertebrae was decreased (Fig. 2). In Dog 9, the vertebral bodies of C6 and C7 had a slightly abnormal shape (Fig. 3). In Dog 10, there was excessive bone formation on the vertebral arch both

Table 1. Details of the 12 dogs included in the study.

Dog No.	Age	Sex	Duration of clin. signs in months	Myelographic findings	Macroscopic findings	Histologic findings
1	18 mo	Male	5	Slight impingement on the spinal cord by the left intervertebral joint at C6—C7. The spinal cord deviated slightly to the right.	The compression was caused by the asymmetrically positioned left intervertebral joint at C6—C7.	No histology
2 (Fig. 4)	11 mo	Female	4	Compression at C5—C6 from the dorsal side. Increases in extension and disappears in flexion.	Deformation of vertebral arches. Compression of the cervical spinal cord at C5—C6.	No histology
3	8 mo	Female	2	Compression at C3—C4 from the dorsal side. Decreased diameter of the cranial orifice of spinal canal of C4. Compression increases in extension, decreases in flexion.	Deformation of vertebral arches. Deformation of several intervertebral joints. Compression of the cervical spinal cord at C3—C4.	No histology
4 (Fig. 6)	4½ mo	Female	1	Compression at C4—C5 from the dorsal side. Decreased diameter of the cranial orifice of spinal canal of C5. Compression increases in extension, decreases in flexion.	Deformation of vertebral arches. Compression of the spinal cord at C4—C5—C6. Moderate hydrocephalus.	Decreased myelin staining of the dorsal columns. Wallerian-like degeneration.
5	5 mo	Male	1	Compression at C4—C5 and C5—C6 from the dorsal side. Slight decrease in diameter of spinal canal. Compression increases in extension and decreases in flexion.	Deformation of vertebral arches. Compression of the spinal cord at C4—C5—C6.	Decreased myelin staining of mainly the dorsal columns. Wallerian-like degeneration.
6 (Fig. 2)	2 yrs	Male	4	Compression at C5—C6 and C6—C7 from the dorsal side. Narrowing of the spinal canal. Compression increases in extension and decreases in flexion.	Deformation of vertebral arches. Compression of the spinal cord C4—C5—C6.	Widespread loss of myelin staining. Myelomalacia. Wallerian-like degeneration.

Table 1 (continued).

Dog No.	Age	Sex	Duration of clin. signs in months	Myelographic findings	Macroscopic findings	Histologic findings
7	9 mo	Male	3	Compression at C5—C6 from the dorsal side. Increases in extension, decreases in flexion. Slight impingement on ventral contrast line by the caudal rim of vertebral body C2.	No necropsy	No histology
8 (Fig. 5)	7 mo	Male	1	Slight compression at C6—C7 from the dorsal side in extension. Increased degree of flexion C3—C4 (so-called subluxation).	Very slight compression of spinal cord at C6—C7. No compression at C3—C4. Deformation of intervertebral joints.	Neuronal fiber degeneration and necrosis, mainly in the dorsal column. Wallerian-like degeneration. No lesion at C3—C4.
9 (Fig. 3)	10 mo	Male	1	Deformation of vertebral bodies C6—C7. Compression at C6—C7 from the ventral side. Slight increase of compression in flexion.	Very slight compression of spinal cord at C6—C7. Abnormal shape of vertebral bodies C6 and C7.	Slight loss of myelin staining in the lateral and ventral columns. Wallerian-like degeneration.
10 (Figs. 7, 8, 9)	2 yrs	Female	6	Osseous proliferations and deformation of vertebral arches C5 and C6. Narrowing of the cranial orifice C6. Compression at C5—C6 from the dorsal side in extension. Disappears in flexion.	Radiographic findings verified on macerated spine. Crevices in intervertebral joints and periarticular osteophytes.	No histology
11 (Fig. 1)	14 mo	Male	Normal. No neurological signs.	Normal myelogram.	No necropsy	No histology
12 (Fig. 8)	12 yrs	Female	Normal. No neurological signs.	No radiologic examination.	Macerated cervical spine completely normal.	No histology

of C5 and C6 (Fig. 7). However, this did not seem to interfere with the size and shape of the vertebral canal.

In 2 of the dogs with the wobbler syndrome (2 and 8), there was an increased degree of flexion between 2 vertebrae when the neck was held in flexed position. The degree of flexion was slightly to moderately increased in Dog 2 (Fig. 4) and markedly increased in Dog 8 (Fig. 5). It is noteworthy that the increase in degree of flexion in these 2 dogs did not give rise to any spinal cord compression demonstrable on the myelogram (Figs. 4 and 5).

Myelography revealed that the 10 dogs with the wobbler syndrome (1—10) had 1 or 2, more or less obvious compressions of the spinal cord. Of these dogs, only 2 (6 and 9) had changes on the plain radiographs, that indicated the presence of spinal cord compression.

In all dogs with spinal cord compression except 2 (1 and 9) it was demonstrated on the myelogram that compression was more severe when the neck was in extended position than when it was kept in neutral or flexed position (Figs. 4 and 6). One of the dogs (1) did not have any compression of the spinal cord demonstrable in any position in the lateral view. However, in the ventrodorsal view it was seen that there was a slight deviation of the spinal cord to the right side, apparently caused by the left intervertebral joint between C6 and C7.

Macroscopic examination

In all the dogs, the myelographic findings of spinal cord compression were verified on macroscopic examination. This is illustrated in Figs. 2, 6 and 7 (Dogs 6, 4 and 10). The increase in compression in extension was demonstrated particularly well in these dogs (6, 4 and 10). By holding the specimen from Dog 4 (Fig. 6) in neutral, flexed and extended position it was well demonstrated what effect position had on the space available to the spinal cord in the vertebral canal. It was seen that the cranial part of the vertebral arch of C5 protruded slightly into the vertebral canal, when the neck was in neutral position. It protruded more when the neck was in extension and not at all when the neck was in flexion. Hence, spinal cord compression was most severe in extension. With the neck in this position, the spinal cord was squeezed between the cranial rim of the vertebral arch of C5 and the caudodorsal rim of the body of C4.

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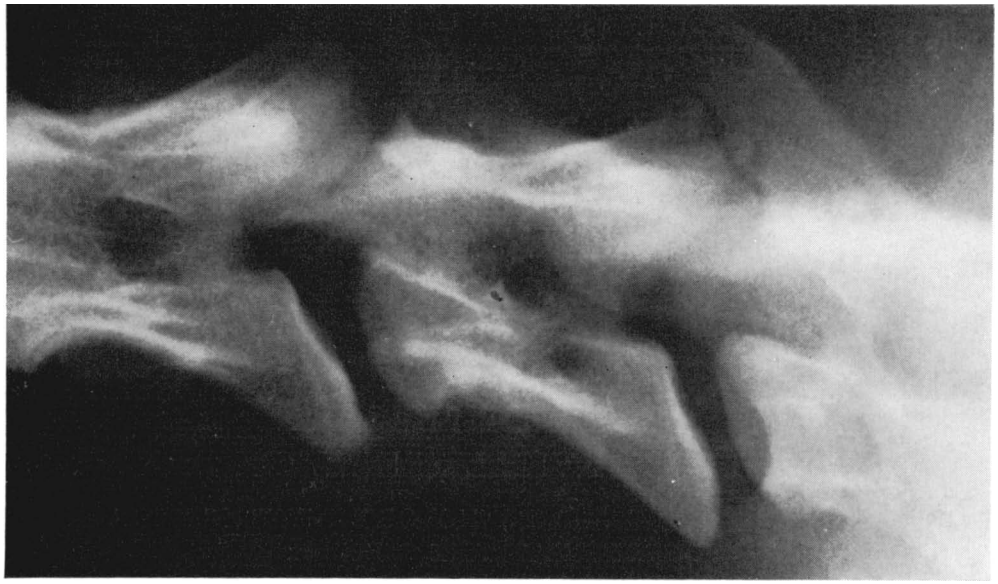
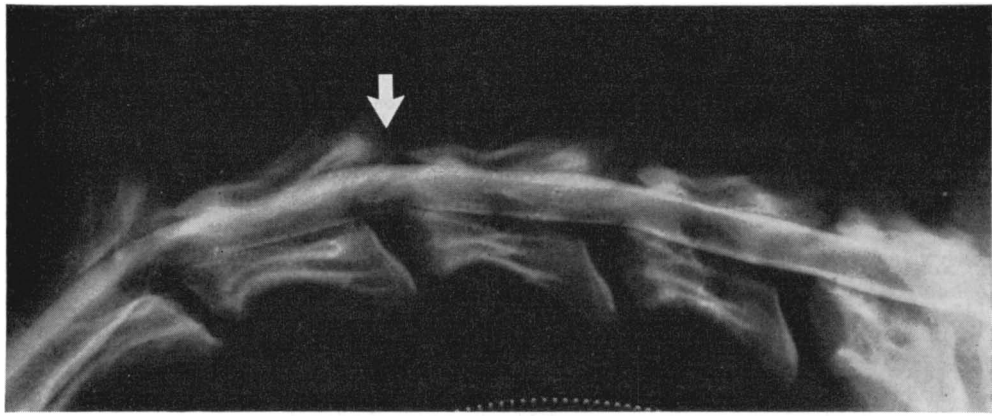
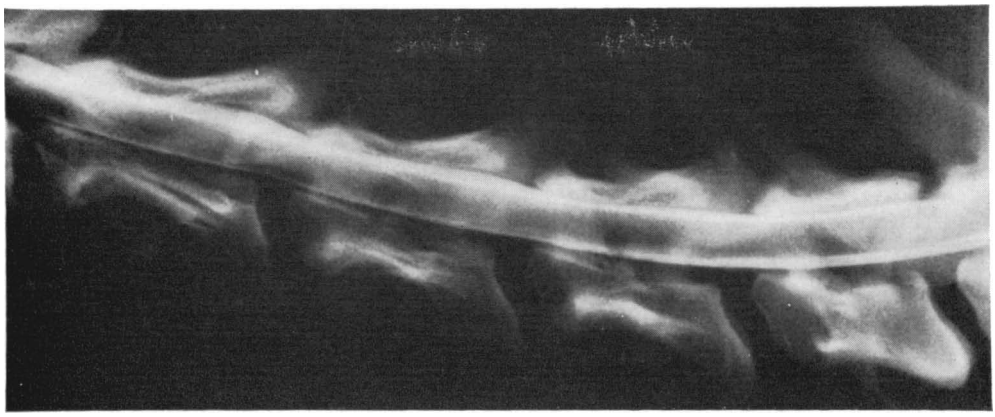


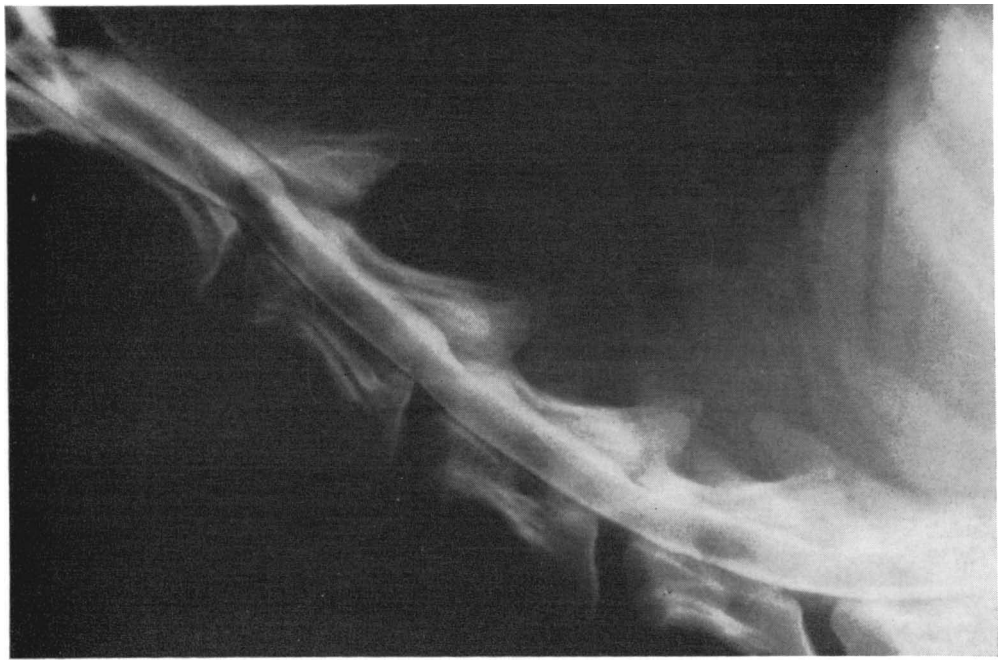
Figure 1. *Dog No. 11 (neurologically normal)*
a) Radiograph of C3, C4 and C5 with the neck in flexion. There is slightly increased degree of flexion between C3 and C4.



b) Myelogram with the neck in flexion. The slight increase in flexion between C3 and C4 (arrow) does not cause any compression of the spinal cord.



c) Myelogram with the neck in neutral position. No compression of the spinal cord.



d) Myelogram with the neck in extension. The cranial part of the vertebral arches of C4, C5 and C6 are impingeing sligthly on the subarachnoidal space dorsally, but there is no compression of the spinal cord.

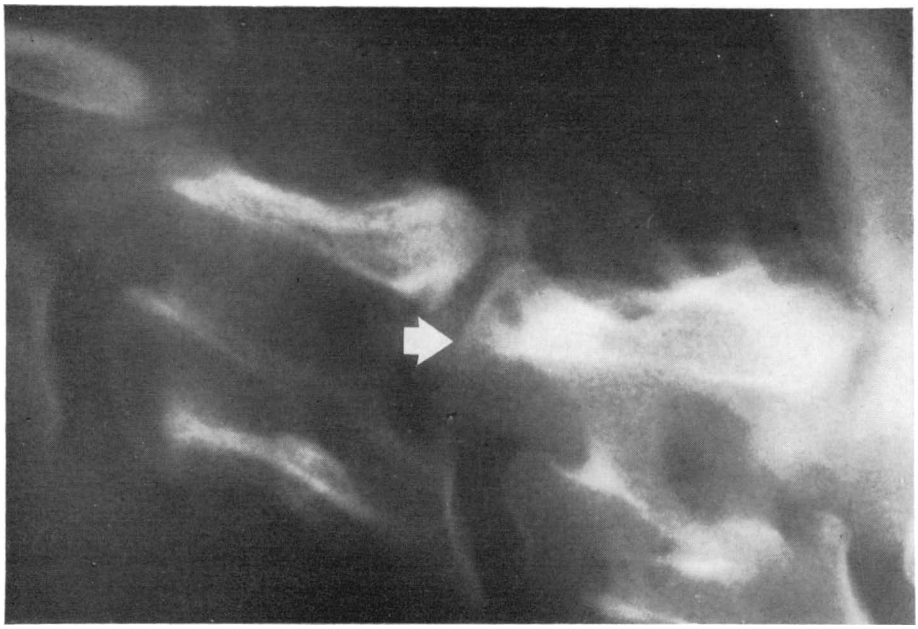
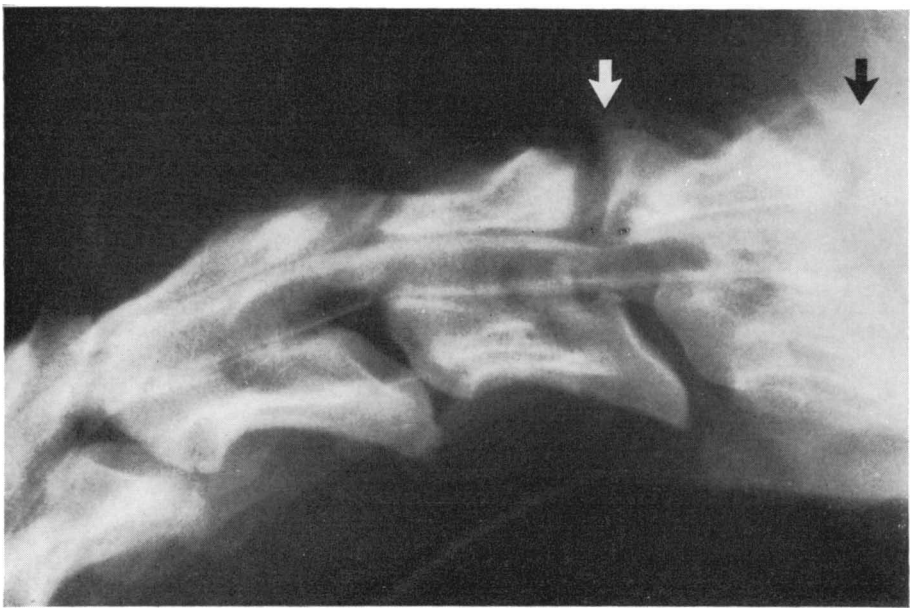
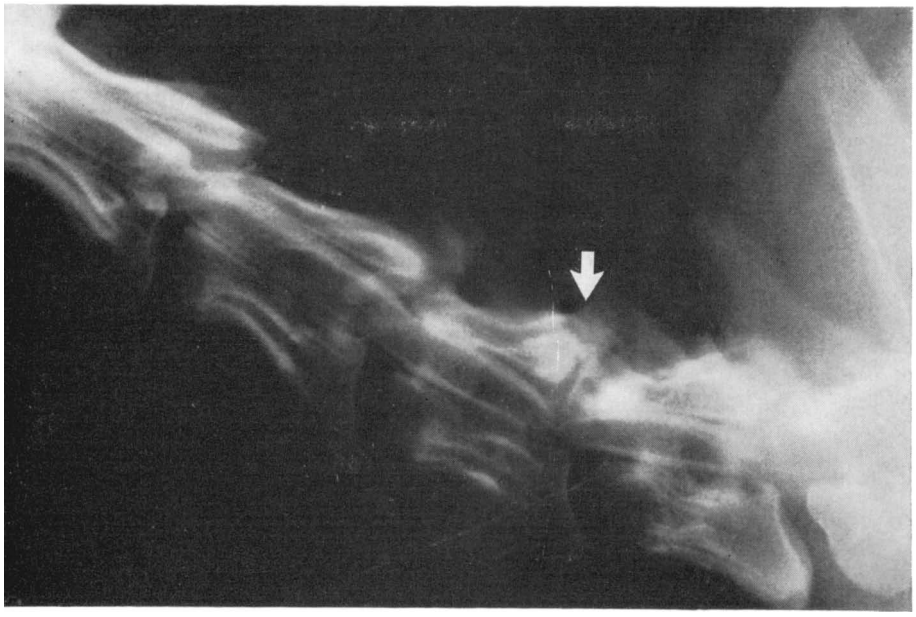


Figure 2. *Dog No. 6*

a) Radiograph of C5 and C6 taken with the neck in neutral position. There is cranial elongation (arrow) and deformation of the vertebral arch of C6, leading to malarticulation with the vertebral arch of C5. The ventro-dorsal diameter of the cranial orifice of the vertebral canal of C6 is decreased. The articulation between C4—C5 is normal, but the one between C6—C7 is abnormal.

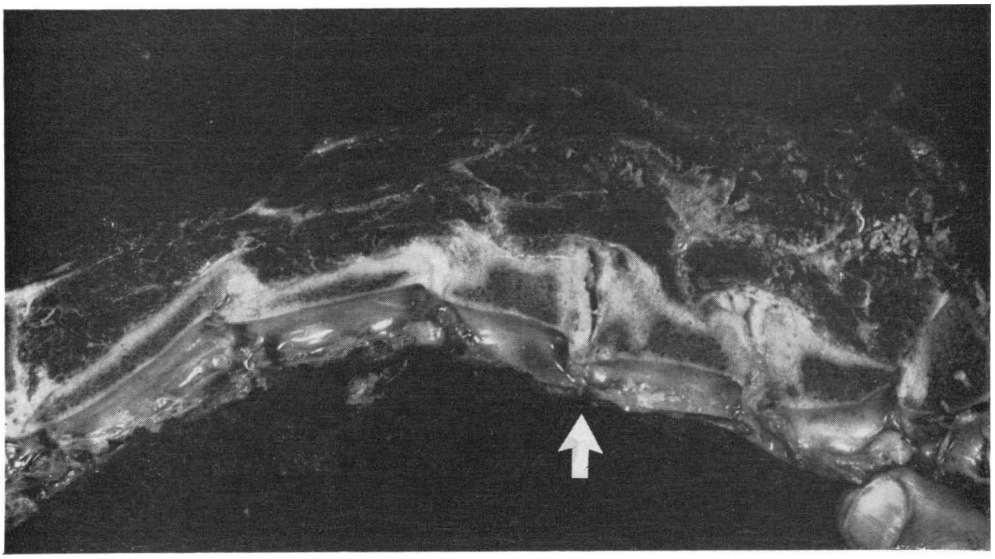


b) Myelogram with the neck in flexion. Impingement on the subarachnoidal space dorsally by the elongated and deformed vertebral arch of C6 (at the level of the white arrow). There is only mild compression of the spinal cord, which is pushed sligthly in a ventral direction. A similar lesion is seen at C6—C7 (at the level of the black arrow).

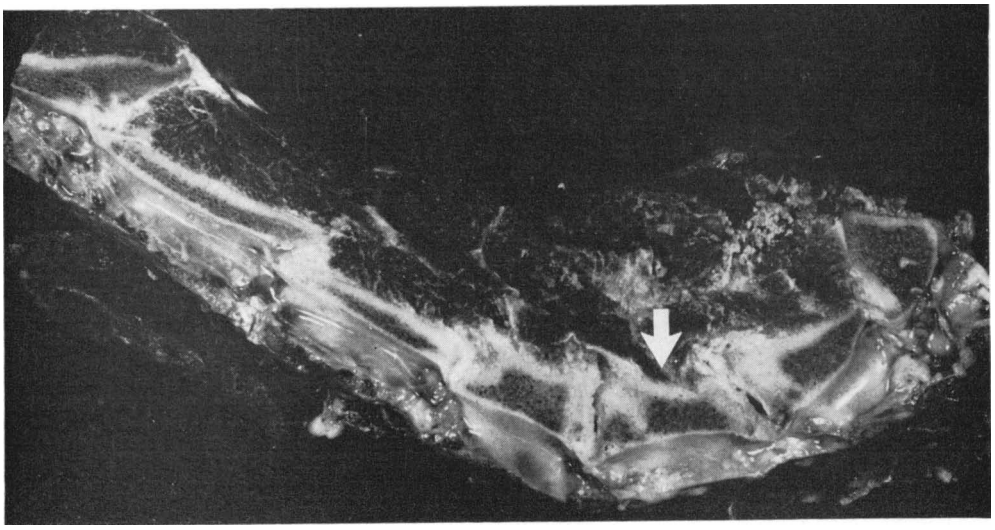


c) Myelogram with the neck in extension. The spinal cord is compressed at two sites (C5—C6 and C6—C7). As is best seen at the C5—C6 level (arrow), the compression is caused by the cranial tip of the elongated and deformed vertebral arch of C6 being brought closer to the dorsocaudal rim of the body of the adjacent cranial vertebra.

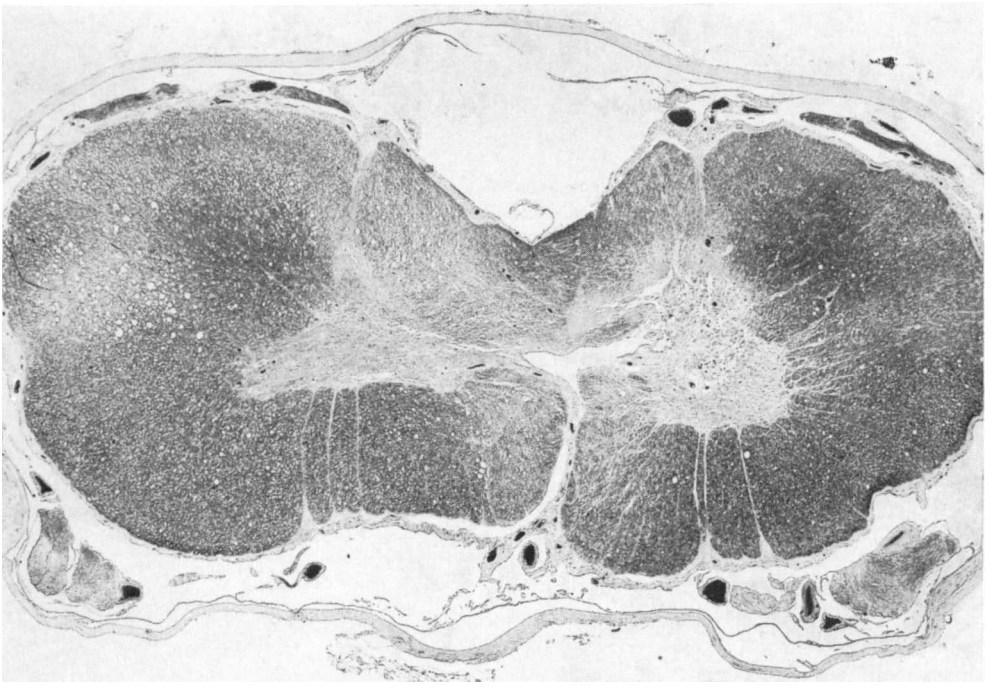
d) Ventral view of the necropsy specimen of the vertebral arches of C6 and C7. The cranial tip of the vertebral arch of C7 is seen as a bulge (arrow). The cranial tip of C6 is seen at the top of the figure.



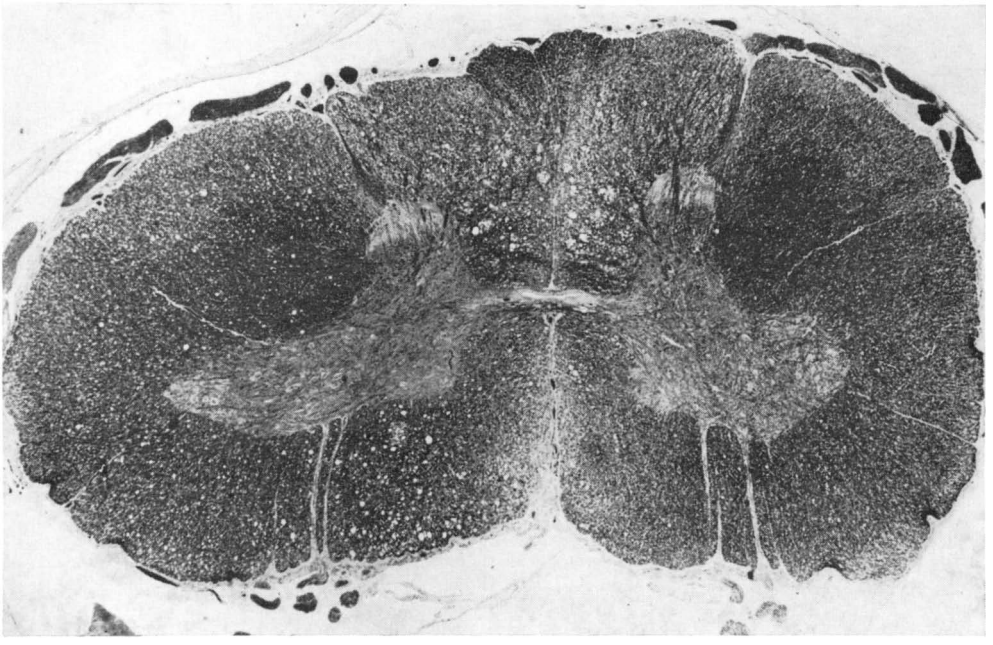
e) Necropsy specimen consisting of the right side of the vertebral arches of the cervical vertebral column kept in flexed position. There is obvious malarticulation between C5—C6 (arrow) and between C6—C7 because of deformation of the vertebral arches.



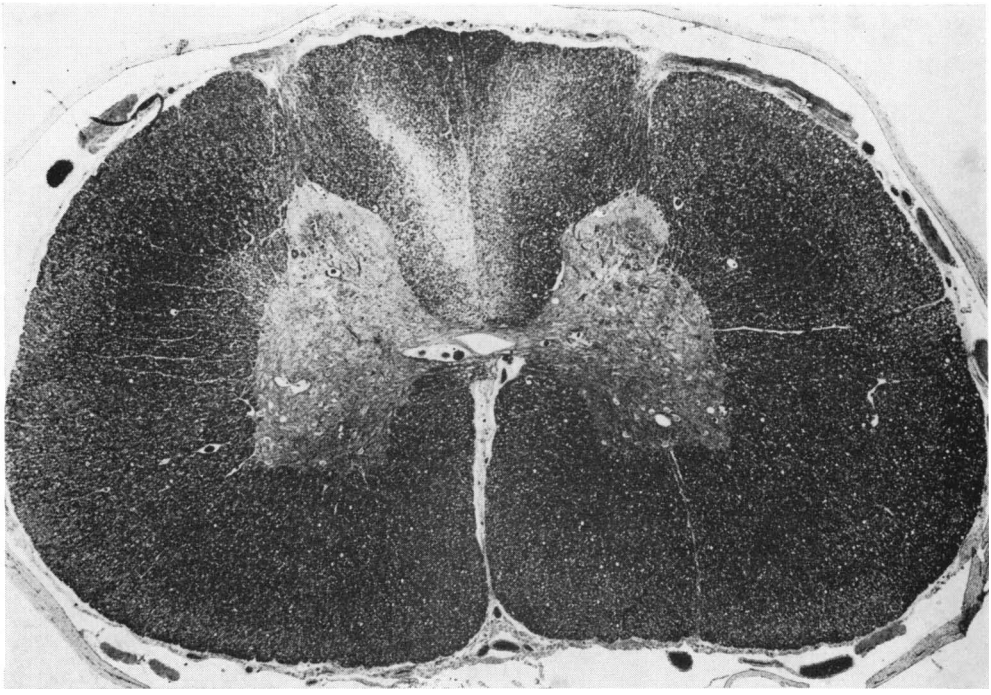
f) The right side of the vertebral arches of the cervical vertebral column kept in extended position. The malarticulating vertebral arches are protruding more into the spinal canal than when the specimen was held in flexion (C6 at arrow).



g) Histologic section of the spinal cord at the level of C5—C6. There is severe compression with loss of myelin staining, neuronal fiber degeneration and necrosis of the dorsal columns. Similar changes are seen in the lateral columns. The grey matter is partly disintegrating. Weil's method for myelin sheaths × 20.



h) Histologic section of the spinal cord at the level of C6—C7. There is compression with loss of myelin staining and neuronal fiber degeneration of the dorsal and ventral columns. The grey matter is spared. Weil's method for myelin sheats × 20.



i) Histologic section of the spinal cord at the level of C4—C5. There is obvious loss of myelin staining in the dorsal columns, sparing the dorsomedian part (fasciculus gracilis) and the area closest to the grey matter (fasciculi proprii). The lesion is a sign of Wallerian-like degeneration in the ascending tracts, which are believed to carry impulses associated with discrete touch, pressure and kinesthetic senses. Weil's method for myelin sheaths × 20.

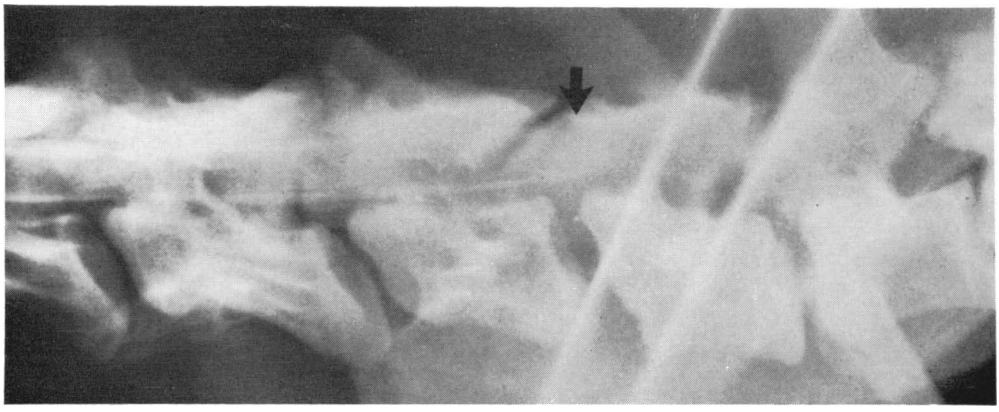


Figure 3. *Dog No. 9*
Myelogram with the neck in slight flexion demonstrating temporary arrest of the contrast medium at C6—C7 (arrow). The vertebral bodies of C6 and C7 are misshapen (on the necropsy table it was seen that compression was caused by the caudodorsal rim of the body of C6 and the craniodorsal rim of the body of C7).

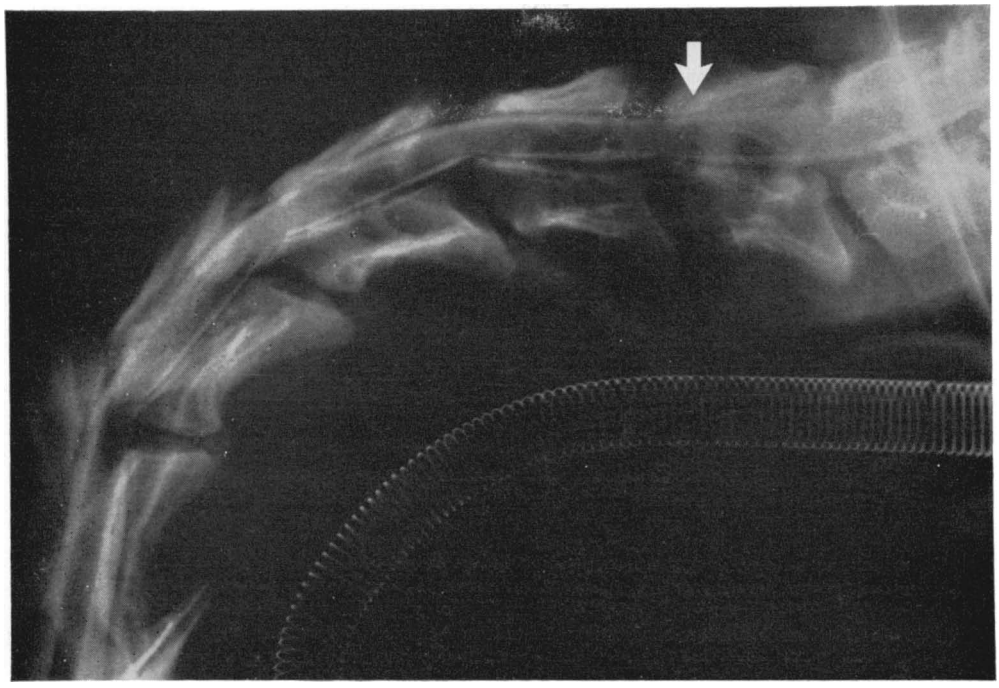
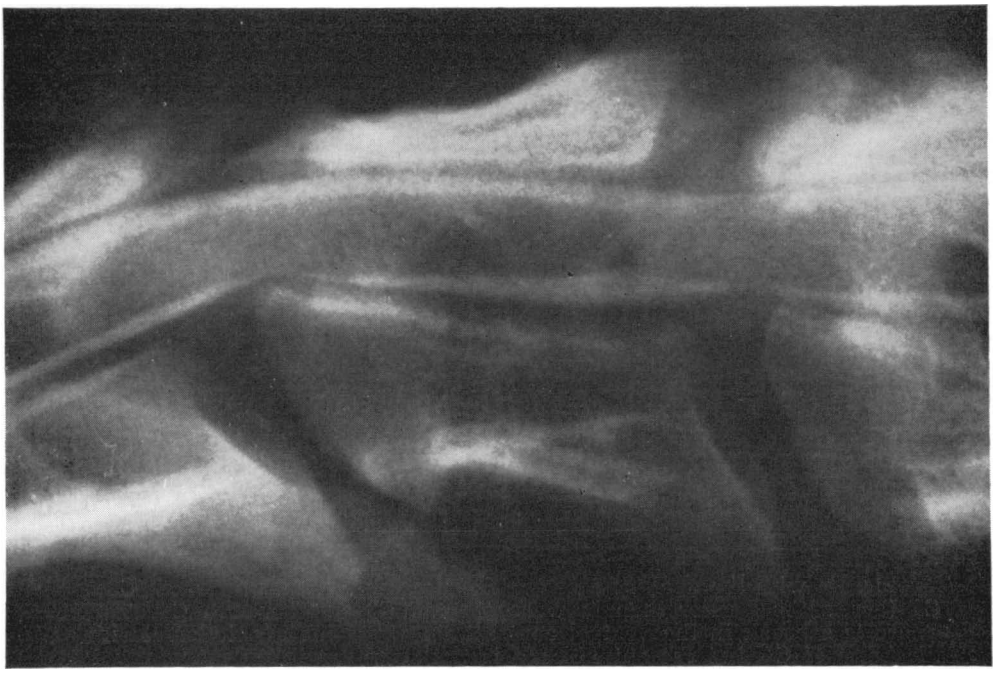
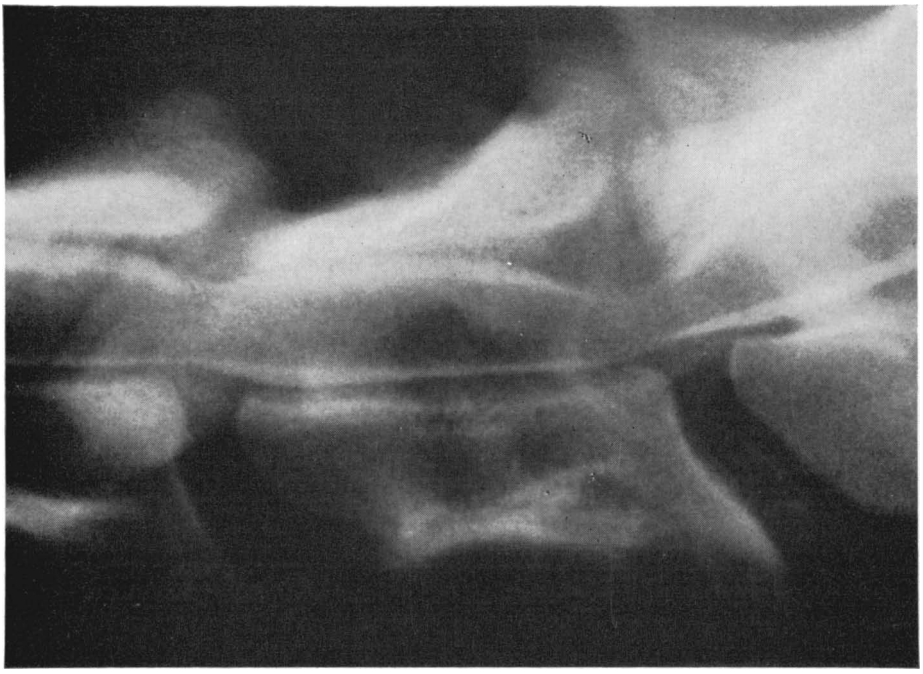


Figure 4. *Dog No. 2*

a) Myelogram with the neck in flexion. There is a slight narrowing of the cranial orifice of the vertebral canal of C6 (arrow) and increased degree of flexion between C4 and C5. However, no compression of the spinal cord is seen at these sites.



b) Detail of myelogram in a) with C5 in the center. The increased degree of flexion at C4 and C5 and the decrease in dorsoventral diameter of the cranial orifice of the vertebral canal of C6 are well demonstrated.



c) The same area as in b) is seen with the neck in extended position. The myelogram demonstrates that there is an obvious compression of the spinal cord from the dorsal side at C5—C6. (At necropsy it was demonstrated that compression was caused by malarticulation between the vertebral arches of C5 and C6, giving rise to a jointlike formation that bulged into the vertebral canal on extension).

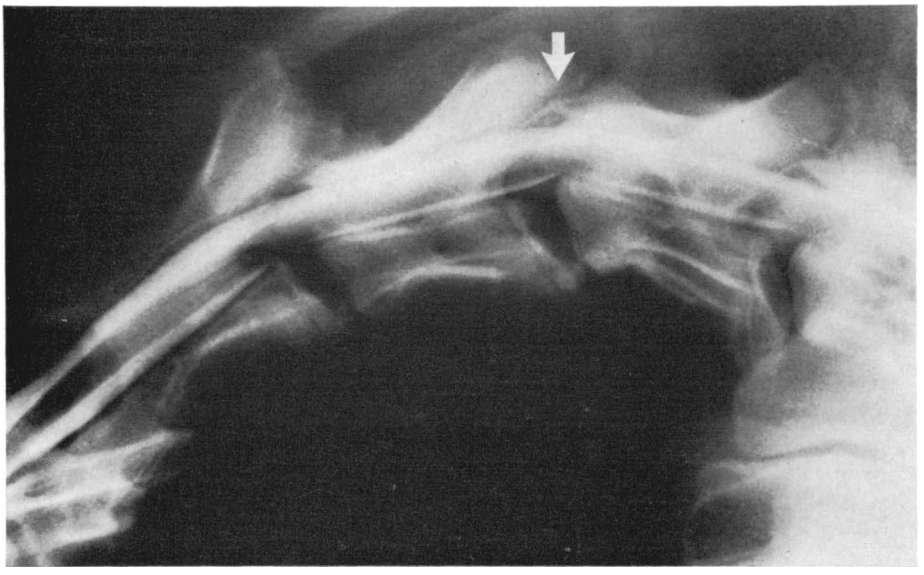


Figure 5. *Dog No. 8*

Myelogram with the neck in flexion. The degree of flexion between C3 and C4 (arrow) is greater than between C2 and C3 and between C4 and C5. There is a break in the ventral contrast line over the craniodorsal rim of the body of C4, but the spinal cord is not compressed. In many previous reports, such an increased degree of flexion has been considered as a sign of instability. It is therefore noteworthy that on histologic examination there were no lesions in the spinal cord at the level of C3—C4. A severe lesion was instead found at the level of C6—C7, where a slight compression from the dorsal side in extension was demonstrated on the myelogram.

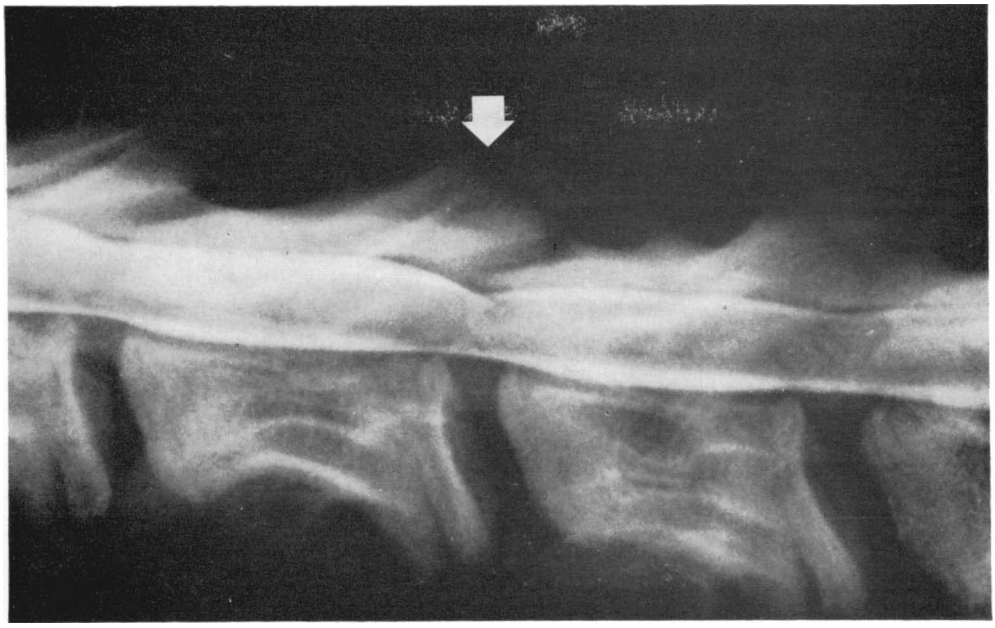
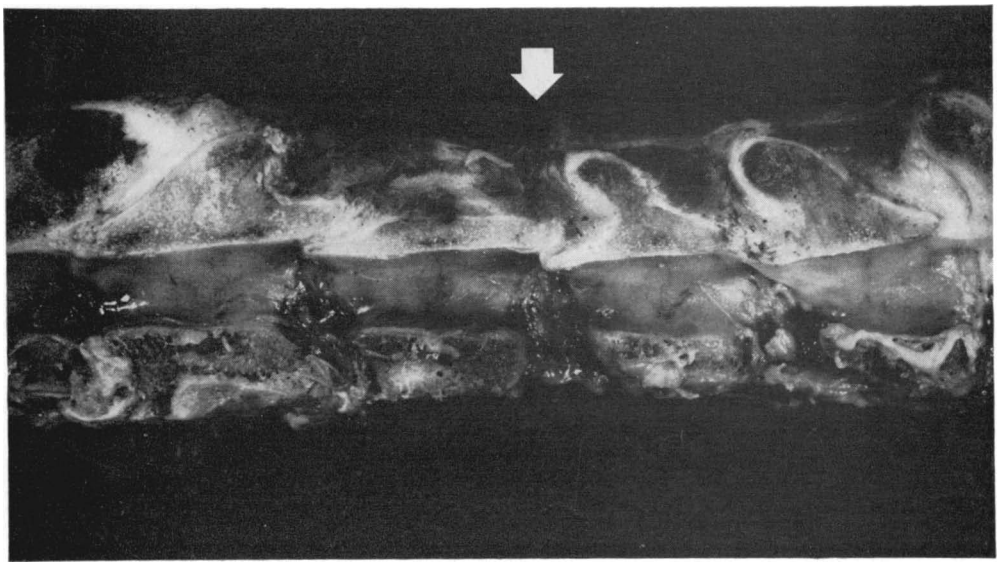
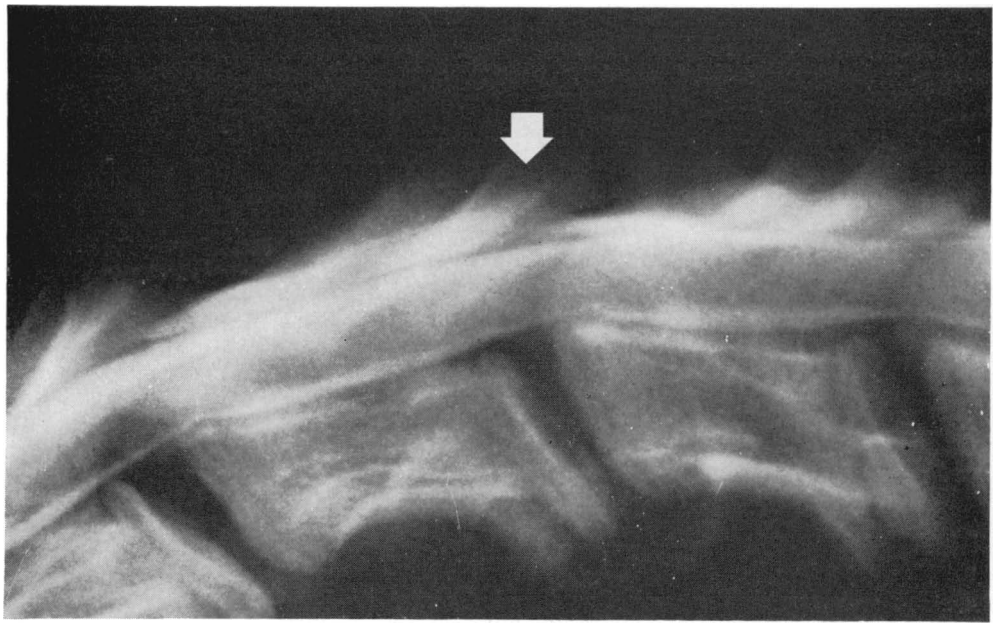


Figure 6. *Dog No. 4*

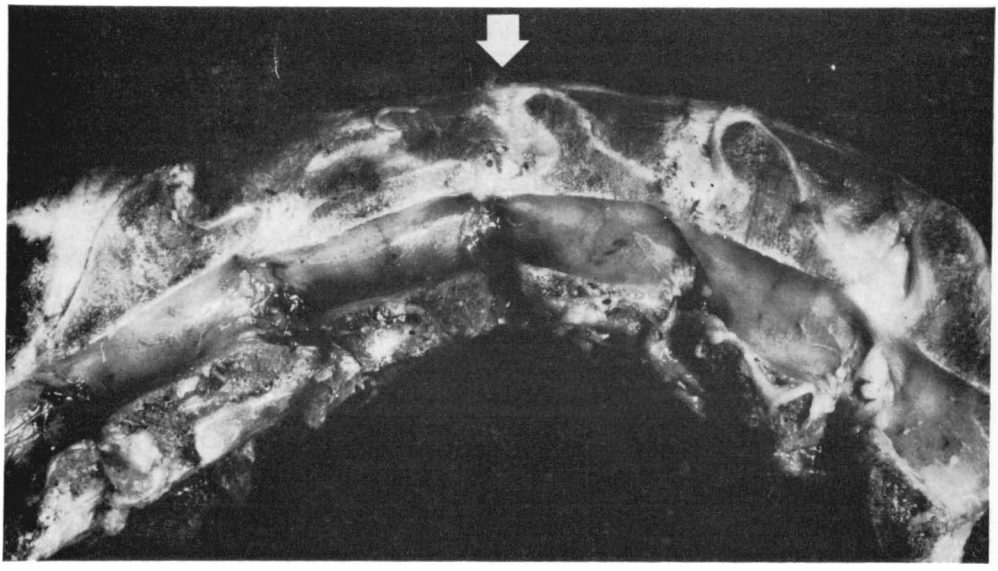
a) Myelogram with the neck in neutral position. There is compression of the spinal cord from the dorsal side at the level of C4—C5 (arrow). The compression seems to be caused by the elongated cranial part of the vertebral arch of C5. The ventrodorsal diameter of the cranial orifice of the vertebral canal of C5 is slightly more narrow than normal.



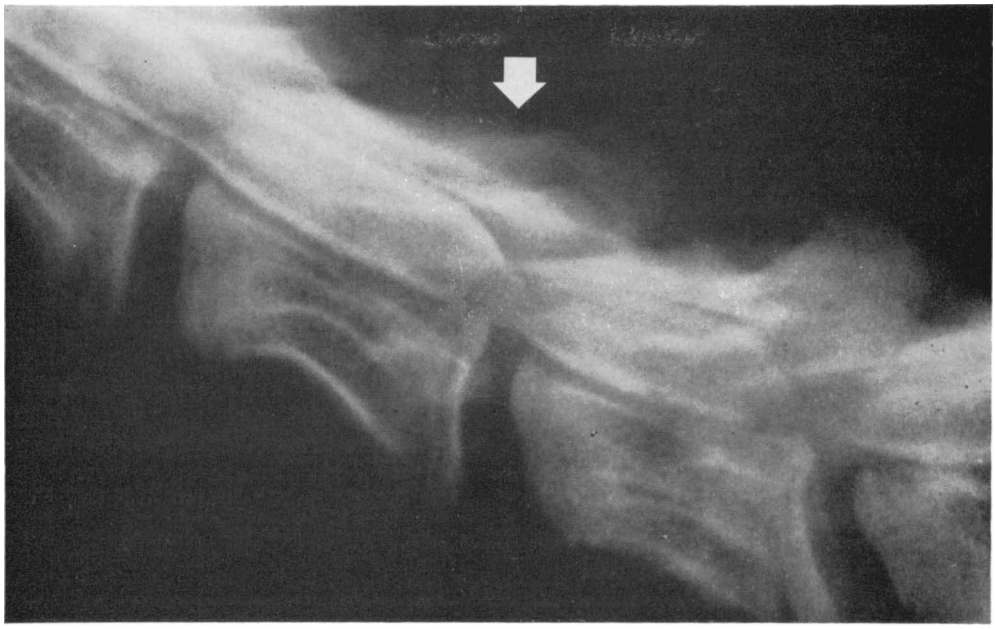
b) Necropsy specimen consisting of the right half of the vertebral arches and a part of the vertebral bodies. The specimen is kept in the same position as the neck was in the myelogram in a). It is seen that the cranial part of the vertebral arch of C5 at the transition between C4 and C5 (arrow) is protruding into the vertebral canal.



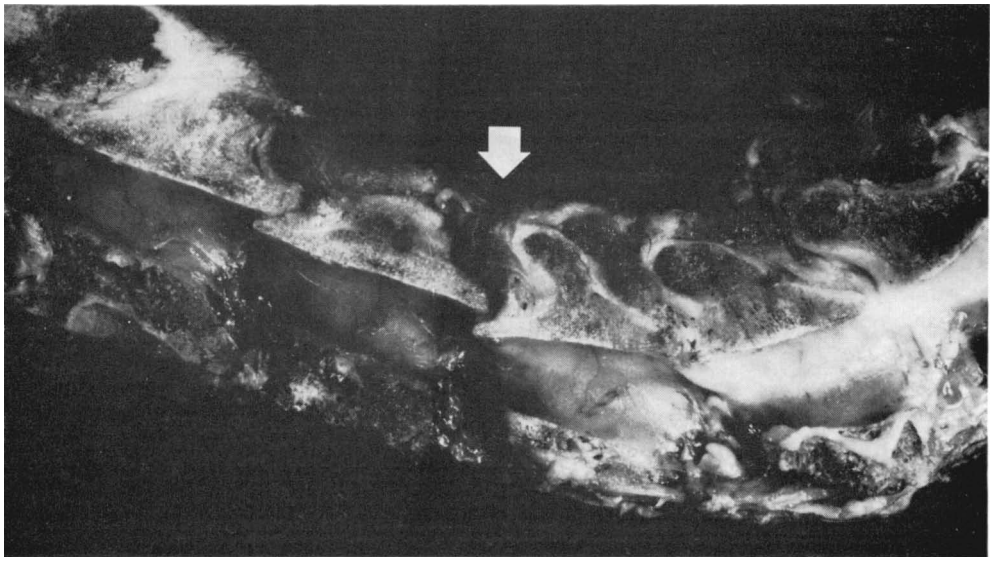
c) Myelogram with the neck in flexion. In this position there is no compression of the spinal cord at C4—C5 (arrow).



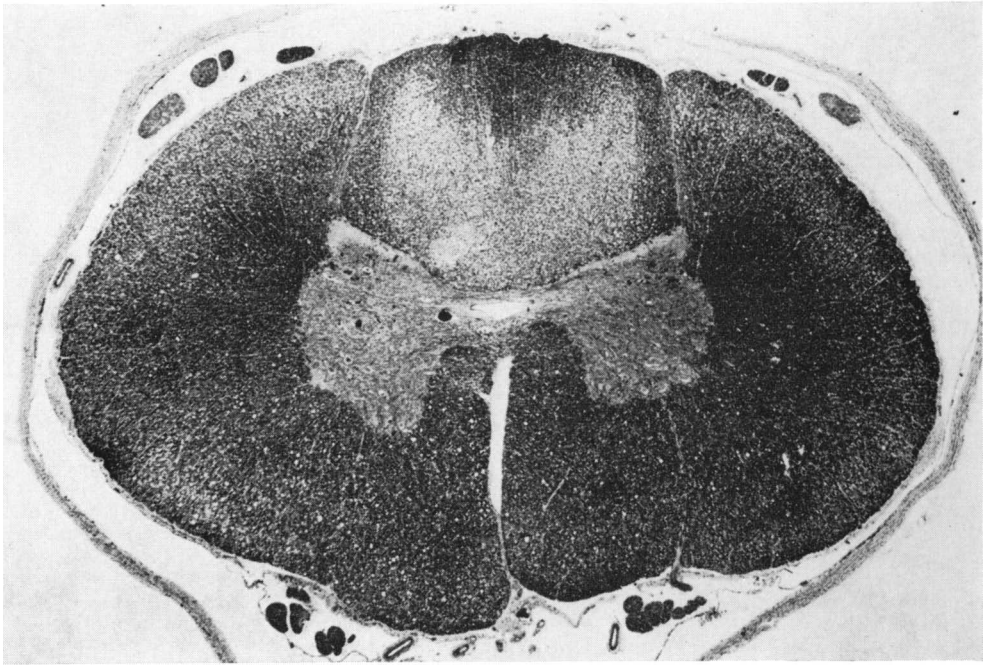
d) The necropsy specimen kept in flexed position as the neck was in the myelogram in c). The cranial part of the vertebral arch of C5 is now retracted, leaving more space in the vertebral canal.



e) Myelogram with the neck in extension. In this position there is an obvious compression of the spinal cord at C5—C6 (arrow) and also some compression at C6—C7. The spinal cord is compressed between the cranial part of the vertebral arch and the dorsocaudal rim of the body of the adjacent cranial vertebra.



f) The necropsy specimen kept in extended position as the neck was in the myelogram in e). The cranial part of the vertebral arch of C5 is now encroaching more upon the space in the vertebral canal.



g) Histologic section of the spinal cord at the level of C4—C5. There is considerable loss of myelin staining of the dorsal column with the exception of the dorsomedian part (fasciculus gracilis). A round area of necrosis is seen slightly to the left in the dorsal column. There is also slight loss of myelin staining in the dorsal part of the lateral columns. Weil's method for myelin sheaths × 20.

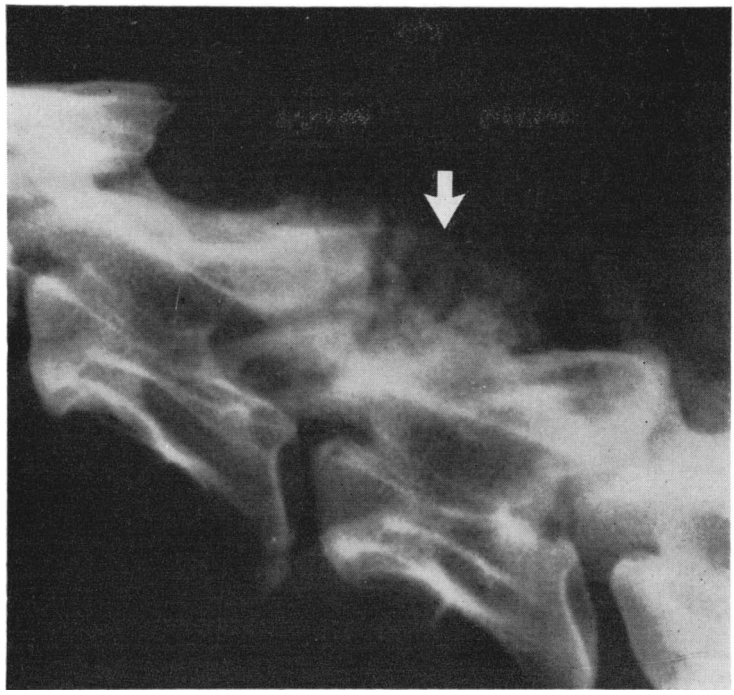
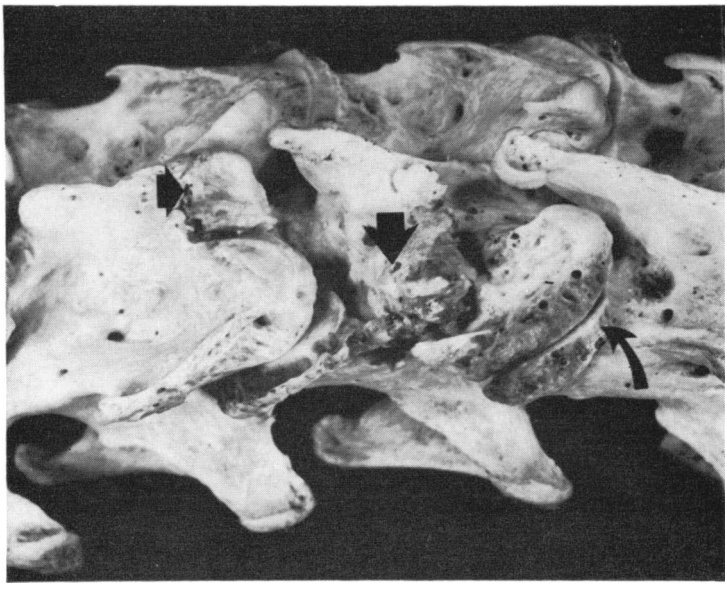
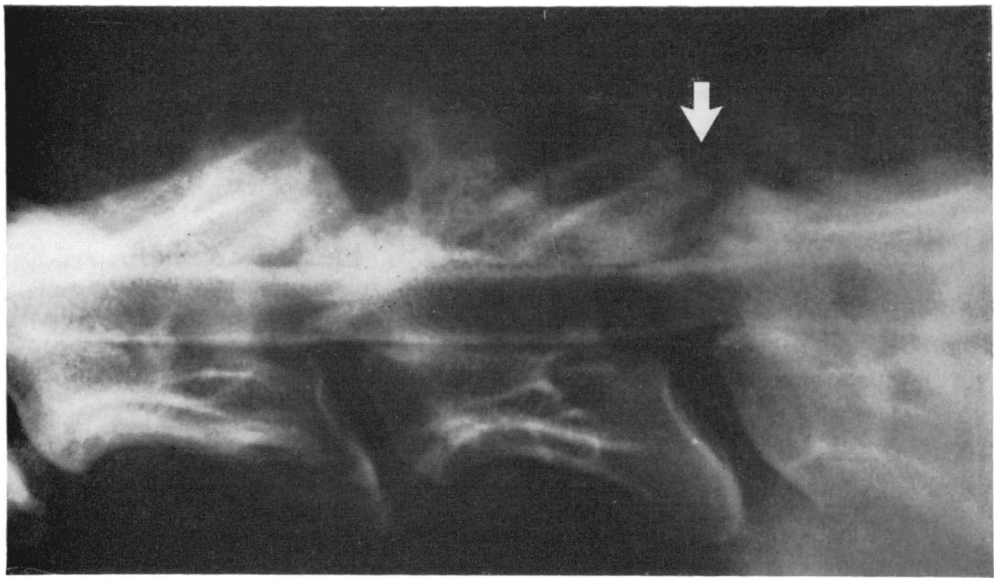


Figure 7. *Dog No. 10*

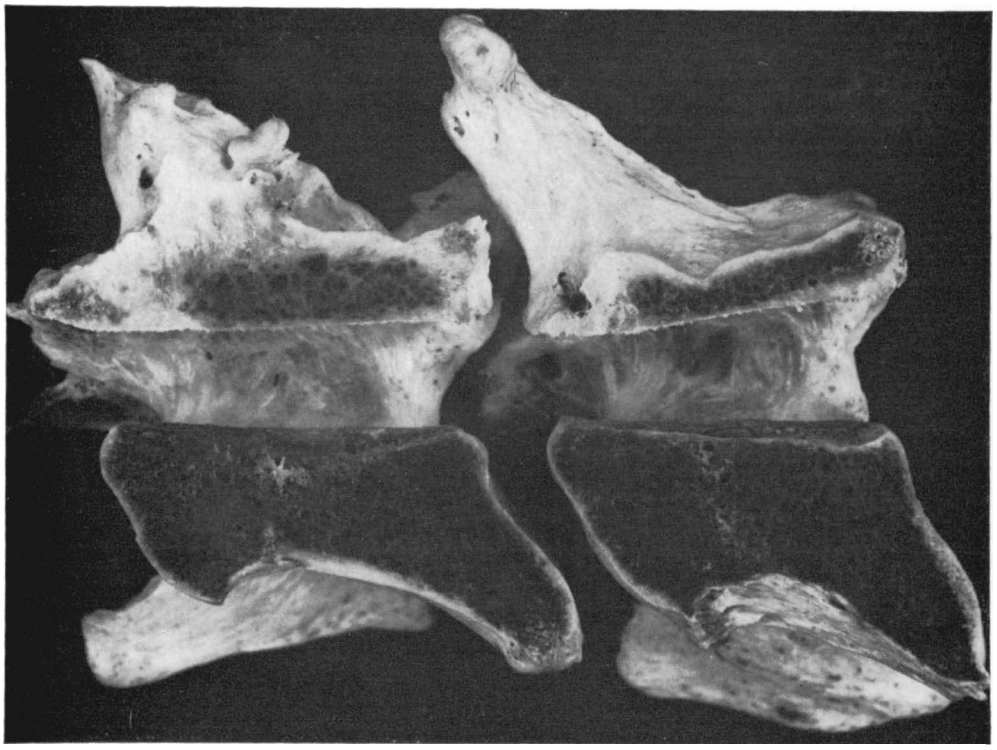
a) Lateral radiograph of the cervical vertebral column. On the caudal end of the vertebral arch of C4 and on the vertebral arch of C5 (arrow) are massive bone proliferations. The intervertebral joints have an increased density.



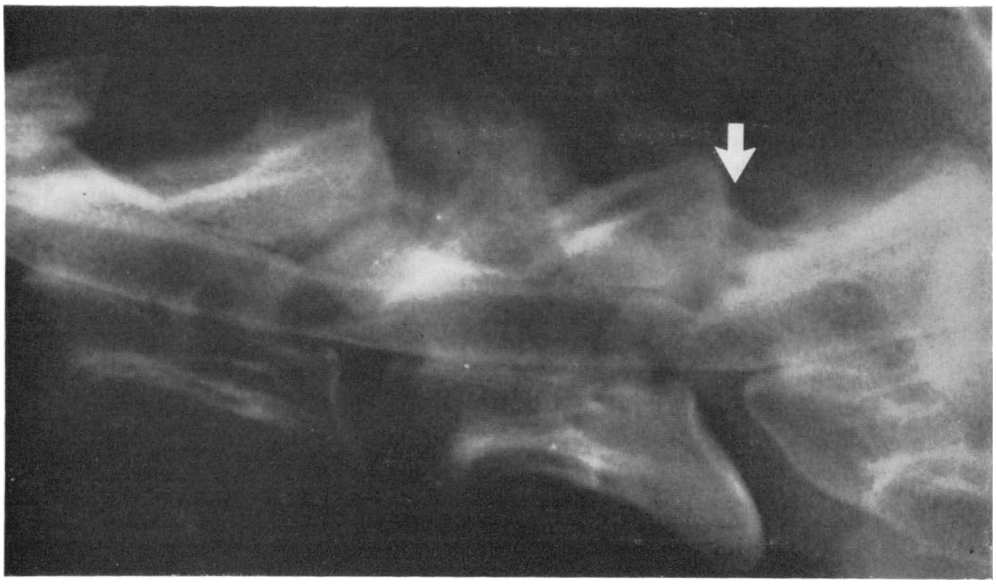
b) Macerated specimen. The osseous proliferations on C4 (small straight arrow) and on C5 (larger straight arrow) are well visible. The intervertebral joints, particularly those at the C5—C6 level, are thick and plump (curved arrow).



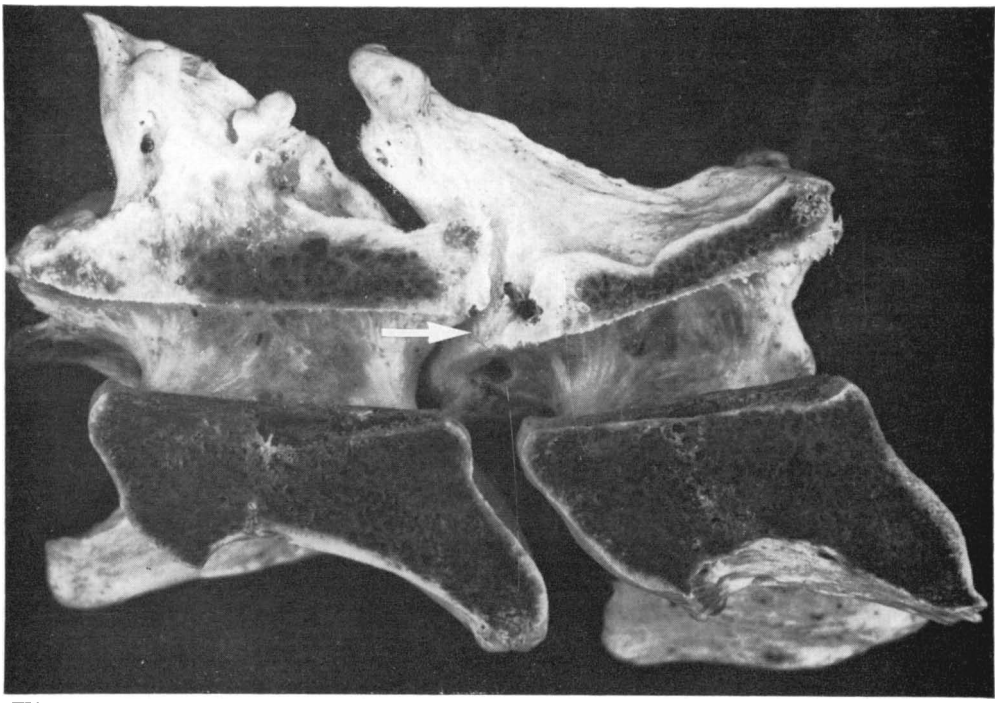
c) Myelogram (C4, C5, C6) with the neck in flexed position. There is no spinal cord compression, but it is seen that the spinal canal is slightly narrow at the cranial orifice of C6 (arrow).



d) Macerated specimen. Right half of C5 and C6 in a position similar to that in c). It is seen that the ventrodorsal diameter of the spinal canal is decreased at the cranial orifice of C6.



e) Myelogram (C4, C5, C6) with the neck in extension. In this position it is seen that there is compression of the spinal cord at the level of C5—C6 (arrow). The cranial tip of the vertebral arch of C6 is impingeing on the spinal cord.



f) Macerated specimen of the right half of C5 and C6 in a position similar to that in e). The cranial tip of the vertebral arch of C6 (arrow) is now closer to the caudodorsal rim of the body of C5 and to the disc space C5—C6.

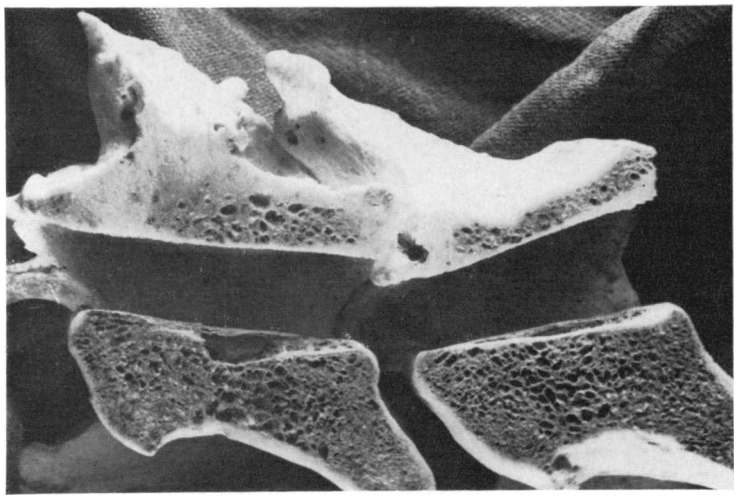
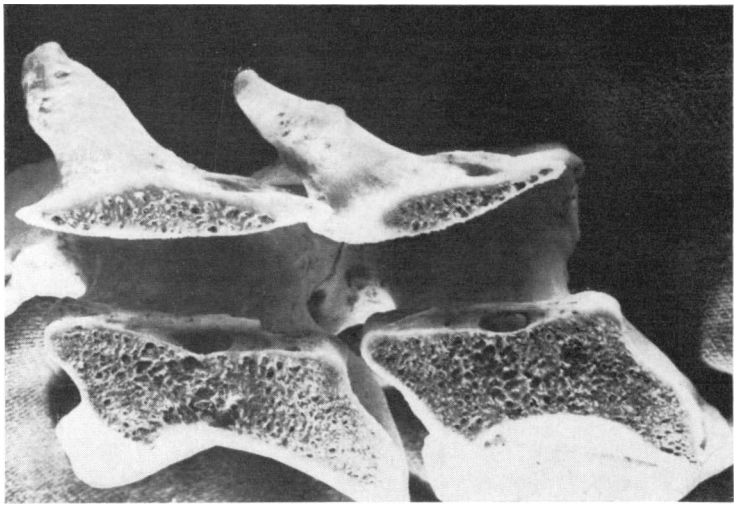


Figure 8. *Dog No. 10 (wobbler) a) and Dog No. 12 (normal) b)*

a) The same specimen as in Fig. 7 in extension, demonstrating how short the distance is between the cranial tip of the vertebral arch of C6 and the caudodorsal rim of the body of C5.



b) The macerated right half of C5—C6 for comparison with the C5 and C6 of Dog No. 10 in a). The 2 vertebrae are kept in extension. It is seen that the normal dog (12) has a larger ventrodorsal diameter of the cranial orifice of the vertebral canal of C6. The cranial part of the vertebral arch of C6 is rounded and there is no pointed tip of the vertebral arch that can give rise to compression of the spinal cord.

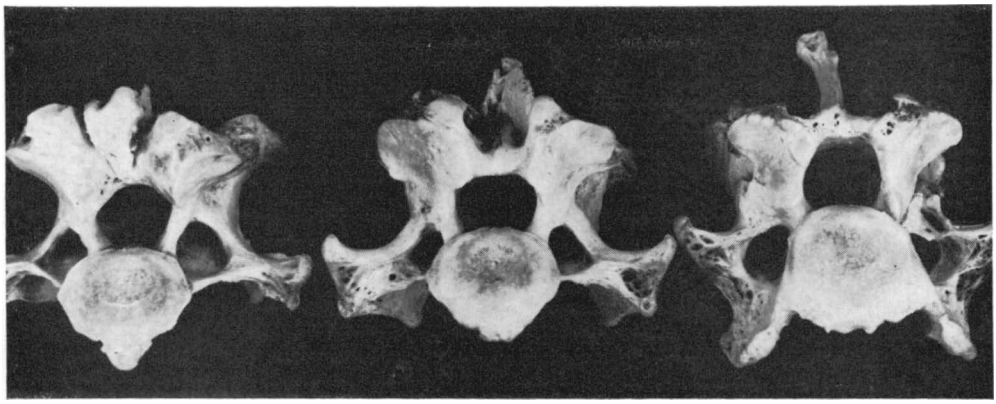
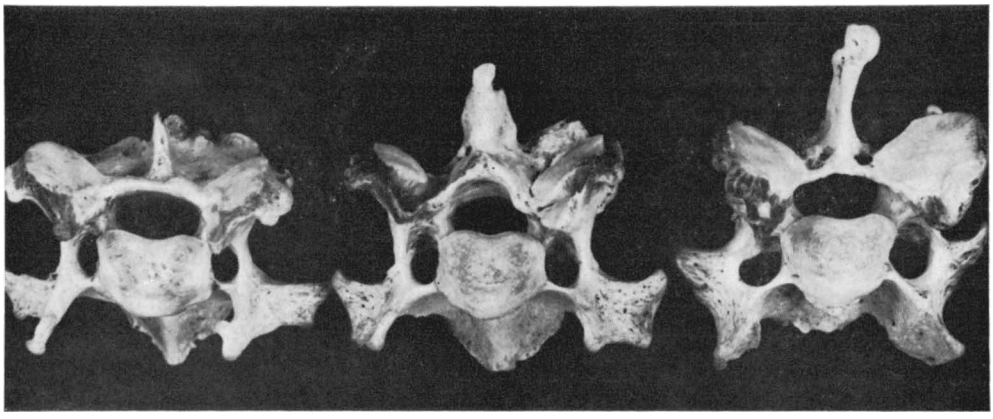
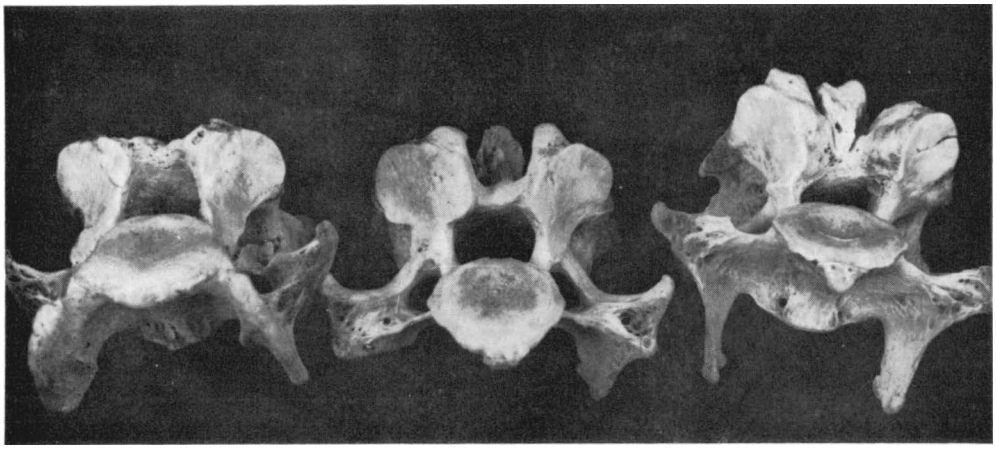


Figure 9. *Dog No. 10*

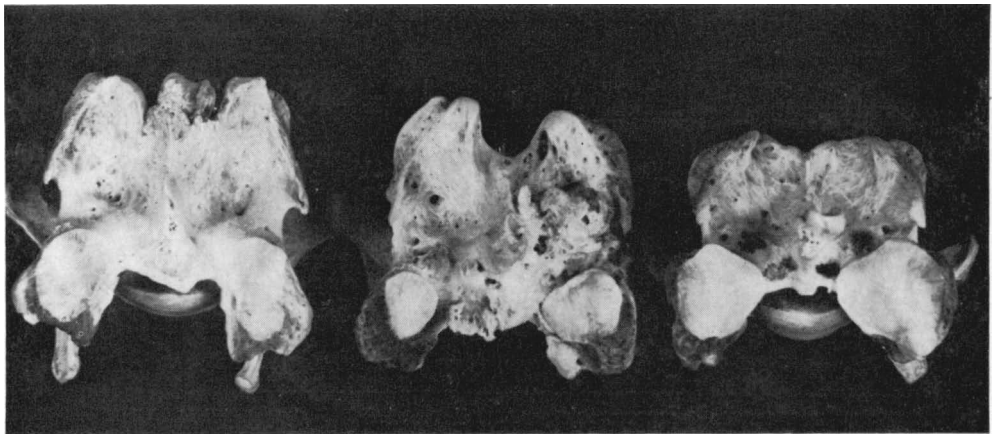
a) From left to right are the caudal aspects of the vertebrae C4, C5 and C6. The irregular shape and asymmetry of the vertebrae are well demonstrated.



b) The cranial aspect of the vertebrae C4, C5 and C6 (left to right). The cranial orifice of the vertebral canal of these vertebrae has a much smaller ventrodorsal diameter but a slightly larger lateral diameter than has the corresponding caudal orifice.



c) Ventrocaudal view of the vertebrae C4, C5 and C6 (left to right). The intervertebral joints are irregular and there are crevices in the bony surface of the left joint of C4 and the right of C6.



d) Craniodorsal view of the vertebrae C4, C5 and C6 (left to right). The intervertebral joints are very irregular in size and shape and there are large periarticular osteophytes.

A similar change in space available to the spinal cord was seen to occur in Dog 10 (Fig. 7). In this dog it was also found that the cranial orifice of the spinal canal of C6 was more narrow than normal with the ventrodorsal diameter particularly decreased (Fig. 8). In addition, excessive bone formation, irregular shape and position of the intervertebral joints with periarticular osseous proliferations, and clefts in the subchondral bone were seen (Fig. 9). In Dog 1, the hypertrophic and abnormally positioned left intervertebral joint of C6—C7 was the cause of a slight impingement of the cervical spinal cord. In Dog 8 (Fig. 5) there was compression of the spinal cord at the level of C6—C7. Hence, the myelographic observation was verified that there was no spinal cord compression at the level of C3—C4 in spite of the increased degree of flexion seen between these two vertebrae.

Histologic examination

On histologic examination of the spinal cord it was found, that most of the pathologic lesions were seen in the white matter of the dorsal columns. Here the fasciculus cuneatus seemed to be more affected than the fasciculus gracilis. The changes at the compression site varied from loss of myelin staining to neuronal fiber degeneration and necrosis (Figs. 2 and 6). Wallerian-like degeneration was usually most obvious in the ascending tracts of the dorsal columns (Fig. 2). In the dog with very severe compression (6), the gray matter was damaged also (Fig. 2). There was not always a good correlation between the degree of the compression seen on the myelogram and the severity of the compressive lesion.

DISCUSSION

In all the 10 Great Dane dogs with the wobbler syndrome, cervical spinal cord compressions of varying degree were seen myelographically. In 9 of these dogs, necropsy was performed, and the macroscopic findings substantiated the myelographic findings. Histologic examination of the spinal cord was made in 5 of the dogs, and compressive lesions that could explain the clinical signs were seen.

In 8 of the above mentioned 10 dogs with the wobbler syndrome there was encroachment of the vertebral canal due to deformation of the vertebral arch of 1 or several cervical verte-

brae. The vertebral arch was often elongated and plump, not seldom in combination with plump, deformed and asymmetric articular processes and joints. A decreased ventrodorsal diameter of the cranial orifice of the spinal canal of 1 or several vertebrae was not uncommon. The various abnormalities in combination with extension of the cervical vertebral column was the main cause of spinal cord compression. Hence, compression was to some extent static but more dynamic in nature, varying in degree depending on the position of the dog's neck. In contrast to what was published in earlier reports on the wobbler syndrome in the dog, compression was found to be most severe in extension and less severe or not present at all in flexion of the neck. It seems likely that no clinical problem had been caused, if compression had been only static without a dynamic compound, i.e. if movements of the cervical vertebral column had not increased and decreased the degree of compression.

The spinal cord compression was due to deformation of vertebral bodies in only 1 of the dogs. In this dog, in contrast to the other dogs, a slight increase in compression was seen, when the dog's neck was kept in flexed position.

The mechanism by which compression is increased in extension is the following. In extension, the cranial tip of the elongated and often plump vertebral arch of one vertebra is brought closer to the caudodorsal rim of the body of the adjacent cranial vertebra. The more narrow the cranial orifice of the vertebral canal is, the closer the cranial tip gets to the vertebral body of the adjacent cranial vertebra, when the neck is extended. When the neck is kept in flexion, the cranial tip of the elongated vertebral arch is retracted, thereby leaving more space for the spinal cord.

Instability (subluxation, spondylolisthesis), which so often has been reported as the cause of cervical spinal cord compression, could not be documented in the present investigation. In 2 dogs, an increased degree of flexion between 2 vertebrae was found, but this did not cause compression of the spinal cord. Compression was instead seen at another site and was not the result of instability.

It is obvious from the present investigation that location and degree of the cervical spinal cord compression cannot be demonstrated in dogs with the wobbler syndrome without myelography. In addition, myelography must be made with the dog's neck in

different positions. Compression may be overlooked if the dog's neck is radiographed only in flexed position. Even in neutral position of the neck, a compression may be difficult to diagnose.

It is difficult to explain why the result of the present investigation differs from the results of most previous studies. Instability and vertebral body deformation, 2 factors reported to be of great importance for the occurrence of spinal cord compression in the wobbler syndrome, were — with 1 exception of a case with slight deformation of 2 vertebral bodies — not found in the present investigation. It is possible that the stenosis causing the wobbler syndrome has a different morphologic background in different breeds of dogs. The present investigation only included Great Dane dogs, while most previous studies included Dobermann Pinschers, Basset Hounds and some other breeds. There seems to be at least one obvious difference between the wobbler syndrome in Great Dane dogs and in Dobermann Pinschers. In the first mentioned breed, the clinical signs appear in young dogs, while in the latter they occur in older dogs (*Trotter et al.* 1976, *Raffe & Knecht* 1980).

An interesting question is why pain was not an important sign in the dogs with the wobbler syndrome in the present study. In dogs with cervical disc protrusion with spinal cord compression of comparable or lesser degree, pain is almost always a very prominent feature.

The reason for this difference in clinical signs between dogs with the wobbler syndrome and dogs with the cervical disc protrusion syndrome may be the difference in degree of inflammatory reaction in the epidural space. In most cases of disc protrusion in the dog, the nucleus pulposus tissue has undergone calcification and when the calcified material protrudes and becomes lodged epidurally, it gives rise to a marked inflammation and foreign body reaction in the epidural space. It is apparently the inflammation more than the compression as such, that gives rise to the severe pain and hypersensitivity, that is so characteristic of the cervical disc protrusion syndrome in the dog. In this respect it is interesting to note that in the dog, noncalcified discs that protrude seem to give rise to much less pain than those that are calcified. In the wobbler syndrome, the inflammatory component in the epidural space is negligible.

The etiology of the deformation of the cervical vertebrae underlying the wobbler syndrome is still an open question. There

is, however, experimental evidence (*Hedhammar et al.* 1974) that the deformation of the vertebral bodies is caused by osteochondrosis induced by overnutrition and rapid growth. Some of the changes seen in the articular cartilage of the intervertebral joints in the present investigation have a certain similarity with osteochondrotic lesions occurring in other joints of the dog. It should also be mentioned that *Mayhew et al.* (1978) have found that osteochondrosis seems to be responsible for the cervical vertebral stenotic myelopathy that according to these authors is responsible for about 12 % of the wobbler syndrome in the horse. It is likely that also genetic factors play a role for the occurrence of the wobbler syndrome in the dog. This is indicated by the study of *Mason* (1979) and also by observations made by one of the present authors (*Stavenborn* 1980).

ACKNOWLEDGMENTS

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REFERENCES

Adams, C. B. T. & V. Logue: Studies in cervical spondylotic myelopathy, parts I, II and III. Brain 1971, *94*, 557—594.

Arnoldi, C. C., A. E. Brodsky, J. Cauchoix, H. V. Crock, G. F. Domisse, M. A. Edgar, F. P. Gargano, R. E. Jacobson, W. H. Kirkaldy-Willis, A. Kurihara, A. Langenskiöld, I. Macnab, G. W. D. McIvor, P. H. Newman, K. W. E. Paine, L. A. Russin, J. Sheldon, M. Tile, M. R. Urist, W. E. Wilson & L. L. Wiltse: Lumbar spinal stenosis and nerve root entrapment syndromes. Definition and classification. Clin. Orthop. Rel. Res. 1976, *115*, 4—5.

Cauchoix, J., M. Benoist & V. Chassaing: Degenerative spondylolisthesis. Clin. Orthop. Rel. Res. 1976, *115*, 122—129.

Chambers, J. N. & C. W. Betts: Caudal cervical spondylopathy in the dog: A review of 20 clinical cases and the literature. J. Amer. Anim. Hosp. Ass. 1977, *13*, 571—576.

Denny, H. R., C. Gibbs & C. J. Gaskell: Cervical spondylopathy in the dog — A review of thirty-five cases. J. small Anim. Pract. 1977, *18*. 117—132.

Dueland, R., R. W. Furneaux & M. M. Kaye: Spinal fusion and dorsal laminectomy for midcervical spondylolisthesis in a dog. J. Amer. vet. med. Ass. 1973, *162*, 366—369.

Gage, E. D. & C. L. Hall: Surgical repair of caudal cervical subluxation in a dog. J. Amer. vet. med. Ass. 1972, *160*, 424—426.

Gage, E. D. & B. F. Hoerlein: Surgical repair of cervical subluxation and spondylolisthesis in the dog. J. Amer. Anim. Hosp. Ass. 1973, *9*, 385—390.

Geary, J. C.: Canine spinal lesions not involving discs. J. Amer. vet. med. Ass. 1969, *155*, 2038—2044.

Hedhammar, Å., F. Wu, L. Krook, H. F. Schryver, A. de Lahunta, J. P. Whalen, F. A. Kallfelz, E. A. Nunez, H. F. Hintz, B. E. Sheffy & G. D. Ryan: Overnutrition and skeletal disease. An experimental study in growing Great Dane dogs. Cornell Vet. 1974, *64*, Suppl. 5.

Hinck, V. C. & N. S. Sachdev: Developmental stenosis of the cervical spinal canal. Brain 1966, *89*, 27—41.

Lord, P. F. & S-E. Olsson: Myelography with metrizamide in the dog: A clinical study on its use for the demonstration of spinal cord lesions other than those caused by intervertebral disk protrusions. J. Amer. vet. radiol. Soc. 1976, *17*, 42—50.

Mason, T. A.: Cervical vertebral instability (wobbler syndrome) in the dog. Vet. Rec. 1979, *104*, 142—145.

Mayhew, I. G., A. deLahunta, R. H. Whitlock, L. Krook & J. Tasker: Spinal cord disease in the horse. Cornell Vet. 1978, *68*, 1—174.

McIvor, G. W. D. & W. H. Kirkaldy-Willis: Pathological and myelographic changes in the major types of lumbar spinal stenosis. Clin. Orthop. Rel. Res. 1976, *115*, 72—76.

Naylor, A.: Factors in the development of the spinal stenosis syndrome. J. Bone Jt. Surg. 1979, *61-B*, 306—308.

Olsson, S-E.: The dynamic factor in spinal cord compression. J. Neurosurg. 1958, *15*, 308—321.

Paine, K. W. E.: Clinical features of lumbar spinal stenosis. Clin. Orthop. Rel. Res. 1976, *115*, 77—82.

Palmer, A. C. & M. E. Wallace: Deformation of cervical vertebrae in Basset hounds. Vet. Rec. 1967, *80*, 430—433.

Parker, A. J., R. D. Park, P. K. Cusick, E. Small & C. B. Jeffers: Cervical vertebral instability in the dog. J. Amer. vet. med. Ass. 1973, *163*, 71—74.

Raffe, M. R. & C. D. Knecht: Cervical vertebral malformation — A review of 36 cases. J. Amer. Anim. Hosp. Ass. 1980, *16*, 881—883.

Rendano, V. T. & L. L. Smith: Cervical vertebral malformation — malarticulation (wobbler syndrome) — the value of the ventrodorsal view in defining lateral spinal cord compression in the dog. J. Amer. Anim. Hosp. Ass. 1981, *17*, 627—634.

Selcer, R. R. & J. E. Oliver Jr.: Cervical spondylopathy — wobbler syndrome in dogs. J. Amer. Anim. Hosp. Ass. 1975, *11*, 175—179.

Stavenborn, M.: Personal communication 1980.

Trotter, E. J., A. de Lahunta, J. C. Geary & T. H. Brasmer: Caudal cervical vertebral malformation-malarticulation in Great Danes and Dobermann Pinschers. J. Amer. vet. med. Ass. 1976, *168*, 917—930.

Verbiest, H.: Results of surgical treatment of idiopathic developmental stenosis of the lumbar vertebral canal. J. Bone Jt. Surg. 1977, *59-B*, 181—188.

Wolvekamp, W. Th. & G. H. Wentink: Vertebral body deformation causing wobbler syndrome in a Great Dane. Tijdschr. Diergeneesk. 1975, *100*, 775—780.

Wright, E., J. R. Rest & A. C. Palmer: Ataxia of the Great Dane caused by stenosis of the cervical vertebral canal: Comparison with similar conditions in the Basset Hound, Dobermann Pinscher, Ridgeback and the thoroughbred horse. Vet. Rec. 1973, *92*, 1—6.

Wright, J. A.: A study of the radiographic anatomy of the cervical spine in the dog. J. small Anim. Pract. 1977, *18*, 341—357.

Wright, J. A.: The use of sagittal diameter measurement in the diagnosis of cervical spinal stenosis. J. small Anim. Pract. 1979, *20*, 331—344.

SAMMANFATTNING

Dynamisk kompression av halsryggmärgen hos Grand Danois.

Författarna redogör för röntgenologiska och patologiska fynd som gjorts på 10 Grand Danois med det s.k. wobbler-syndromet. Med hjälp av myelografi kunde hos samtliga 10 hundar påvisas att en cervikal ryggmärgskompression förelåg på ett eller två ställen. Ryggmärgskompressionen var huvudsakligen av dynamisk natur. Hos 8 hundar ökade kompressionsgraden när halsen hölls i extension och den minskade vid flexion. Hos en hund med deformerade kotkroppar (C6—C7) ökade kompressionen något då halsen flekterades. Hos en annan hund var kompressionen lateral och kunde endast ses på röntgenbilder tagna i ventrodorsal projektion.

De makroskopiska fynden bekräftade de radiologiska fynden. Orsaken till ryggmärgskompressionen var i 8 fall en minskning av den dorsoventrala diametern av främre öppningen av kotkanalen på en eller flera kotor. Dessutom var dessa kotbågar ofta deformerade och förlängda. När hundens hals extenderades kom ryggmärgen i kläm mellan den främre kanten av den förlängda och klumpiga kotbågen och den kaudodorsala kanten av framförliggande kotkropp. I 5 fall kunde ryggmärgen undersökas histologiskt, och den kompressionsskada som hittades kunde förklara de neurologiska symptomen.

Författarna diskuterar varför smärta inte är ett framträdande symptom hos hundar med wobbler-syndromet, när smärta dominerar bilden vid cervikal diskprotrusion. Man antar att det finns goda skäl att antaga att det är den inflammatoriska främmande-kroppsreaktionen, orsakad av den förkalkade nucleus pulposusvävnaden, som utlöser smärtan hos diskbråckshundarna.

Som avslutning diskuterar författarna vilka etiologiska faktorer som kan vara av betydelse för deformeringen av halskotorna och som är den yttersta orsaken till wobbler-syndromet. Det finns indicier som talar för att både övernutrition under tillväxten och genetiska faktorer är av betydelse för uppkomsten av "the wobbler syndrome".

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