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EXPERIMENTAL STUDY ON ANTERIOR SPINAL CORD COMPRESSION WITH SPECIAL EMPHASIS ON VASCULAR DISTURBANCE

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ABSTRACT

The present study concerns with the vascular changes of the spinal cord, when it is compressed anteriorly such as spondylotic myelopathy.

The method of spinal cord compression was explored to give the pressure only anteriorly without opening the spinal canal. The procedure is to approach anteriorly to the vertebral column and the intervertebral disc is drilled and the anterior aspect of the spinal cord is visualized without the laminectomy procedure which may act as a decompressive. Microangiographic study is made with extensive injection of aqueous suspension of micro-particle of barium sulfate into arterial system of the spinal cord via the vertebral arteries. Various sections of less than 5 mm in thickness are made of the spinal cord in which the arterial system has been filled with barium. Transverse section of the spinal cord readily shows the beautiful pattern of arterial supply on the microangiogram. Parasagittal section of the spinal cord, however, is found best to demonstrate the change of vascular pattern in the case of anterior cord compression.

From the present observations, the results are summarized as follows:

1. The anterior spinal artery is difficult to be occluded by anterior pressure, so that the theories of anterior spinal artery insufficiency in the case of spondylotic myelopathy seem equivocal.

2. Parasagittal sections show early and persistent change of the central arteries by anterior pressure instead.

3. Deformity of the spinal cord could be due to tight fixation by the dentate ligaments, and this deformity could be the main cause of the central artery insufficiency.

Vascular changes of the spinal cord by anterior pressure were discussed in detail in regard to the pathogenesis of spondylotic myelopathy.

INTRODUCTION

Recently much attention has been given to the problem of cervical spondylosis, and many authors have written on the pathogenesis of cervical myelopathy due to spondylosis. Yet, the multiplicity of clinical symptoms, courses and treatments presents much remaining to be discussed.

Many articles written on cervical spondylosis emphasize that when the

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spinal cord is exposed at operation the cervical cord is seldom found to be compressed. Even after operation there is little, if any, clinical improvement despite the release of a compressed spinal cord. This observation has been agreed upon by Brain, Northfield, and Wilkinson[1], O'Connell[2], Clarke and Robinson[3], Nugent[4], and Taylor[5].

Furthermore, it should be considered that the extent of the neurological deficit is seldom related to the magnitude of the radiological findings as seen on cervical X-ray films, and according to Bradshaw[6] and others, in less than half of cases with cervical spondylosis did the neurological level of dysfunction correspond to the level of the main bony lesions seen on X-ray films.

These observations would suggest that the entity of the cervical myelopathy is not a direct neuronal compression, but vascular insufficiency caused by arterial or venous obstruction, or both as Taylor[5] has emphasized. The etiology of the cord lesions in cervical spondylosis is still under discussion, but the hypothesis of vascular insufficiency suggested by Barré[7], Morton[8] and Allen[9] has been supported by many authors including Mair and Druckman[10], Greenfield[11], Girard, *et al.*[12], and Taylor[5], although they do not agree on the actual mechanism. However, study of this hypothesis has shown that there is lack of evidences, and there is need for more investigation.

On the other hand, many other authors believe that the spinal cord is damaged by compression between spondylotic bars and the lamina and ligamenta flava. At any rate, the pathogenesis of the myelopathy in cervical spondylosis is complex and a number of the factors are concerned. However, interference with the blood supply to the spinal cord must be an important factor as well as a mechanical compression of the spinal cord.

The primary purpose of this experiment is to elucidate this point, of which there are not sufficient experiments reported to confirm this from the purely experimental standpoint.

MATERIALS AND METHODS

As the experimental animals, dogs were chosen for the study because dogs were easily obtained. The primate has the habit of walking on foot, and the head-neck-body relationship resembles to the human. In order to make spinal cord compression, the anterior approach was made to avoid opening the spinal canal as in the case of the laminectomy approach which had been the usual technique of previous reporters such as Matsumoto[13], Amako[14], Tarlov[15], Izumida[16], Kaneko[17] and Kano[18].

A dog was anesthetized by the intraperitoneal injection of 30 mg/kg of thiobarbiturate, then an endotracheal tube was inserted to keep a free air way during the experimental procedure. The dog was placed on its back, and the extremities were tied to the table. The neck was extended by underlying

wooden pillow of 5 cm diameter.

The neck was shaved over wide area, including anterior aspect of the neck and upper portion of the chest, and prepared with regular surgical sterilization. A lineal skin incision was made of about 5 cm in length on the midline just above the sternum, the platysma and other neck muscles were divided and pushed laterally while the trachea and esophagus were pulled medially so that the anterior surfaces of the lower cervical spines were visualized. When the soft and rough connective tissues over the vertebral bodies were divided with electrocoagulation, soft grey and yellow appearance of intervertebral discs would be exposed. Intervertebral cartilage of dogs seem to be much thinner as compared to human's ones. Usual width of dog's intervertebral disc is about 3 mm, and this can be identified by soft consistency confirmed by puncture with hypodermic needle. The special fine drill with depth-proved perforator tip was designed for this purpose and a small hole of 6 mm in diameter was made onto the intervertebral body. Through this hole, the posterior longitudinal ligament could be visualized and after peeling off, the spinal dura mater with physiological pulsation could be readily observed. At this time, if the dura mater is opened, the anterior spinal artery can be seen through this hole occasionally.

The depth of the perforator must be carefully adjusted to avoid damage to the spinal cord. The depth of intervertebral disc was measured on X-ray film and the depth of perforator was settled by stopper adjusted on each individual animals. Then, pressing material will be inserted and pushed onto the spinal cord through this hole. For pressing material, bone-wax (W), dental resin material (R), metallic plug (M), rubber balloon with air inflation catheter (B), and laminaria (L) was investigated.

After insertion of the pressing material, intervertebral disc hole was filled up with bone-wax and dental cement. Anterior surface of the hole was sealed with surgical adhesive.

Wound of this experimental surgery was carefully closed with aseptic technique. Postoperatively, in some cases, antibiotics were given to prevent infection and postoperative fluid transfusion was given for additional care.

Postoperatively, neurological observation was made subsequently, mainly the spinal cord function, development, and recovery of spinal paralysis as follows:

1) The development of motor weakness of fore and hind limbs as well as the movement of tail, muscle tonus of extremities, gait disturbances, deep tendon reflexes, withdrawal movement of limbs to painful stimuli, and the disturbances of vesicorectal functions was observed. All these changes were carefully recorded to describe the development, course and recovery of the spinal paralysis.

2) X-ray examination was made of the cervical spine and myelographic examination was also made to observe existence of spinal subarachnoidal blockage in some cases.

After 3 to 110 days, usually 7 to 10 days following the spinal cord compression surgery, dogs were subjected to micro-angiographic study of vertebral-spinal cord preparation. Dogs were again anesthetized with intraperitoneal barbiturate, and then endotracheal tube was inserted. The previous surgical wound was re-opened and bilaterally the common carotid arteries, the external and internal jugular veins, and the vertebral arteries were exposed widely. Polyethylene catheters of about 1.5 mm internal diameter were inserted into the common carotid arteries of both sides towards distal direction, then they were ligated at the cardiac end of these arteries.

The jugular veins of both sides were catheterized for letting blood out, and the vertebral arteries of both sides were also catheterized and their cardiac sides were ligated to avoid the retrograde flow of injected material. Furthermore, the subclavian arteries were ligated near the vertebral origin to avoid the escape of injected material to the extremities.

Two mg/kg of heparin-solution was given intravenously to prevent intravascular coagulation of blood. After extravasation of blood through the carotid and jugular vessels, these vascular systems were irrigated with heparin added to saline of 36°C in temperature until returning saline was free from blood. During the experiment, the animals were usually irrigated with 1.5 to 2 liters of the fluid. Passing the latter half of the procedure, the carotid arteries were clamped. Then, 25 per cent aqueous suspension of micro-particle of barium sulfate (Micropaque) of 36°C in temperature was injected through the vertebral arteries very slowly. It was injected under low pressure of 50 cm of suspension. At the end of infusion, 10 per cent gelatin added Micropaque suspension was infused. At this time, the animal's tongue, sclera and retinal blood vessels would be stained the white color of barium sulfate. Then all catheters were withdrawn and the vessels were ligated at their openings, and muscular layers were approximated. Artificial respirator was used during the irrigation to keep the cardiac functioning as much as possible.

The cerebellomedullary cistern was punctured, and the lumbar subarachnoid space was catheterized with 0.5 mm diameter polyethylene catheter, and 10 per cent formalin solution was used for irrigation of subarchnoid space from above and below. After sealing of these puncture points, the animals were placed in ice box for next 24 hours.

The cervical spinal cord was taken out with the cervical spine and neck muscles, and the preparation was placed into the 10 per cent formalin solution for 10 days for fixation and later subjected to the microangiographic study. Super-soft X-ray apparatus, "Softex" was adapted. Some of the vertebral

spinal cord preparations were frozen to −20°C and sliced with a saw. The frozen sections were also subjected to the microangiographic examination. The cervical spinal cord was separated from the vertebra, and postero-anterior and lateral roentgenograms were made of all the preparations by laying them on the film. Konilitho contact film was used for the present study. After this roentgenography the spinal cord was cut and different slices of transverse, sagittal and coronal sections were made in 2 to 5 mm in thickness for further studies.

Conditions of the super-soft X-ray picture in the experiment are as follows:

Secondary voltage	20-30 kV
Electric current	2.5 mA
Exposure time	8-15 min
Focus-film distance	38 cm

After the X-ray picture was taken, these spinal cords were subjected to histological studies by means of haematoxylin and eosin, Masson's and Weigert's stains.

Observation period of the neurological changes ranges from 3 days to 6 months, usually 7 days for observation of acute changes.

RESULTS

It is rather difficult to pick the neurological changes of the experimental animals at early stage following the operation.

As to spinal cord dysfunction, the main signs were as follows:

1) Motor disturbances of limbs and tail.

2) Sensory disturbances by observing withdrawal of limbs by pinprick stimuli.

3) Vesico-rectal disturbances by observing way of urination and defecation.

As to the motor disturbances among 48 experimental animals, 39 dogs (81.3%) developed the disturbances to some extent. Among them, 19 dogs (48.7%) developed quadriplegic type paralysis, 17 dogs (43.6%) developed paralysis of hind limbs (paraplegic type) and 3 dogs (7.7%) developed motor disturbances of fore-limbs. Unilateral weakness developed in 13 dogs (33.3%) and fore-limbs paralysis were seen in 2 dogs.

There were some features of laterality of weakness, but this is more or less unilateral and the spinal cord compression was revealed sometimes unilaterally in retrospect.

Thirty-five dogs (89.7%) showed paralysis of flaccid type and 4 dogs (10.3%) showed the spasticity from early stage. Spastic quadriplegia was observed in only one dog. Paralysis, of course, was observed after complete arousal of anesthesia and usually it was the day following the experimental surgery.

Fourteen dogs of advanced paralysis showed the progressive paralysis gradually, some dogs such as No. 24 in the table showed temporal recovery of spinal paralysis which became completely paralyzed. Some dogs such as No. 38 developed delayed paralysis that occurred 3 days following the spinal cord compression.

No obvious muscular atrophy was observed in this series even in the cases of long term observation. As to deep tendon reflex, most dogs revealed diminished or absent reflex of the fore-limbs that was the result of spinal root compression locally, while hyperreflexia in hind-limbs was the result of spinal long tract compression.

As to response to pinprick stimuli, 10 dogs (25%) showed absent reaction, and it seemed that dogs of severe compression showed the tendency of less response in general.

However, the problem of sensory disturbances among dogs remains to discussed, since the withdrawal by painful stimuli could be caused on the basis of escape response as the reflex mechanism of the segmental spinal neuronal chains.

Recovery of spinal paralysis was observed in 13 dogs (33.3%), and if any, this occurred on the 3rd postoperative day or started at the latest on the 7th postoperative day. Among this recovered group, all dogs but one showed mild or moderate degree of paralysis to start with.

Observation at the time of spinal cord compression is also very interesting, at this moment, extensor response of the limbs was seen in 19 dogs and this response lasted from 5 to 15 seconds and this is, of course, spinal reflex and not paralysis.

Another observation was made on the respiratory change, accelerated respiration was seen in 4 dogs, and respiratory arrest in 14 dogs. Among the accelerated respiration group, one died within 2 hours and a half, 9 dogs which survived were kept in artificial respirator for 5 to 40 minutes.

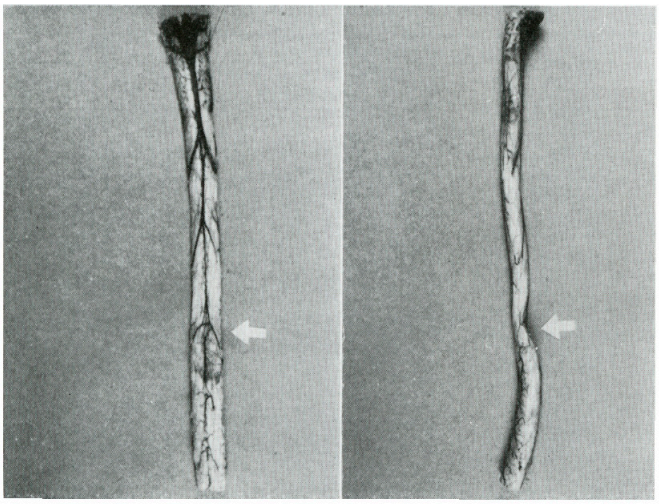
Anatomical studies of these animals revealed the deformity of the flattened spinal cord in antero-posterior diameter (Fig. 1). This strain is, of course, due to the anterior compression, but the fixation of the spinal cord by the dentate ligaments makes a special shape which has been studied and described by Kahn[19].

In the present study, special effort was made to observe the development of spinal cord edema, but none of them showed such findings macroscopically. Three dogs revealed extradural bleeding and one showed subdural bleeding, and of these, all died within a few postoperative days except one. In 3 dogs, myelographic studies were made which revealed remarkable or complete subarchnoid blockage at the level of compression (Fig. 2).

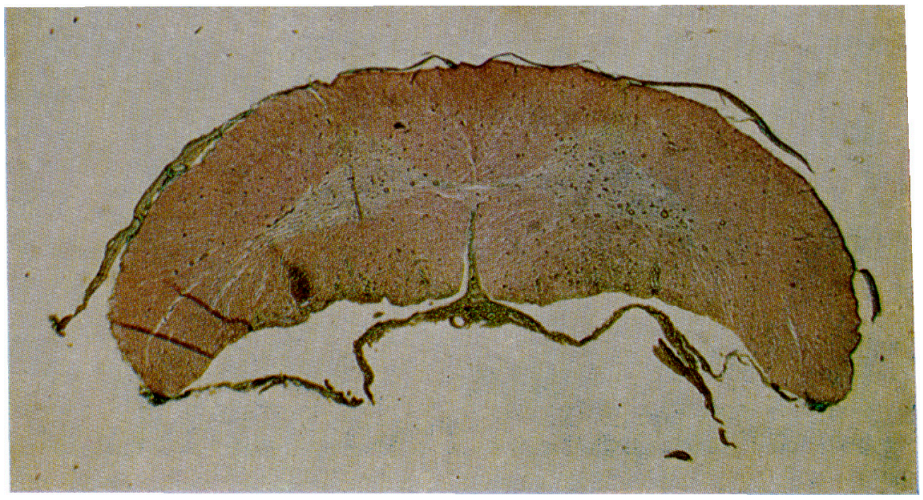
TABLE Experiments of anterior spinal cord compression of dogs

Animal No.	Material	Level C	Acute response		Paralysis			Observation period (days)
			Extension	Respiratory arrest	Development	Type	Recovery	
1	M	3~4	+	+				(2.5 hr)
2	〃	4~5	−	−	+	F. RFI	−	47
3	〃	5~6	+	−	+	F. Q	−	7
4	〃	4~5	−	+	+	F. Q	−	15
5	〃	5~6	+	+	+	F. RFI	+(3 d)	167
6	〃	3~4	−	+				(1.5 hr)
7	〃	4~5	−	A B				(1.5 hr)
8	〃	4~5	+	−	−			6
9	〃	4~5	+	−	+	F. HI	+(5 d)	5
10	〃	4~5	+	−	+	F. HI	−	3
11	〃	4~5	−	+	+	F. Q	−	4
12	〃	4~5	−	+	+	F. HI	+(5 d)	27
13	〃	4~5	+	+	+	F. Q	−	4
14	〃	3~4	−	+	+	S. Q	−	3
15	R	3~4	−	−	+	F. HI	−	7
16	〃	4~5	−	+				(1 hr)
17	〃	3~4	+	+				(1 hr)
18	〃	5~6	−	−				11
19	〃	4~5	+	−	+	F. Q	+(5 d)	213
20	〃	3~4	−	−	+	F. HI	+(7 d)	182
21	W	4~5	−	+	+	F. HI	−	3
22	〃	5~6	−	A B	+	F. HI	+(3 d)	4
23	B	4~5	−	−	−			176
24	L	3~4	−	−	+	F. HI	+(3 d)	5
25	〃	4~5	+	−	+	F. HI	−	8
26	〃	4~5	−	−	+	F. HI	−	5
27	〃	4~5	−	−	+	F. HI	+(7 d)	110
28	〃	5~6	−	−	+	F. Q	−	3
29	〃	4~5	+	+	+	F. Q	−	2
30	〃	4~5	−	−	+	F. Q	−	3
31	〃	3~4	+	+				(1 hr)
32	〃	3~4	−	−	+	F. Q	−	4
33	〃	4~5	−	−	+	F. HI	+(4 d)	87
34	〃	4~5	−	−	+	F. HI	+(7 d)	30
35	〃	4~5	−	−	+	S. Q	−	4
36	〃	4~5	−	−	+	F. HI	+(4 d)	50
37	〃	3~4	+	+	+	F. Q	−	2
38	〃	4~5	−	−	+	F. HI	−	4
39	〃	4~5	+	−	+	F. Q	−	1
40	〃	4~5	−	−	+	F. HI	+(4 d)	5
41	〃	4~5	−	−	+	F. FI	+(3 d)	5
42	〃	4~5	−	−	+	S. HI	−	3
43	〃	4~5	−	−	+	F. Q	−	5
44	〃	4~5	+	A B	+	E. Q	−	4
45	〃	3~4	+	A B	+	S. FI F. HI	−	2
46	〃	3~4	+	−	+	F. Q	−	4
47	〃	3~4	+	−	+	F. Q	−	4
48	〃	4~5	+	−	+	F. Q	−	3

AB=Accelerated breathing
F.=Flaccid, R=Right, FI=Fore-limb
S.=Spastic, Q=Quadriplegia
HI=Hind-limb



A B



C

FIG. 1. Cervical spinal cord with intra-arterial filling of Micropaque and India ink. Arrows indicate level of compression.

A: Anterior aspect. B: Lateral aspect. C: Transverse section of the spinal cord (C_4-D_5) anteriorly compressed. Masson's stain with intraarterial India ink injection. ×10.

Microangiographic findings. The arterial distribution pattern of lower portion of medulla oblongata and the cervical spinal cord is illustrated in Fig. 3. In this segment of the spinal cord, most of the blood supply comes from the vertebral arterial system, and this pattern in the canine cervical spinal

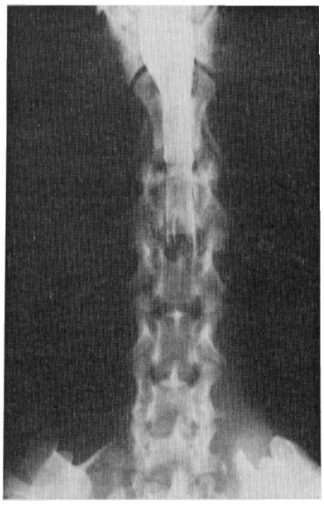
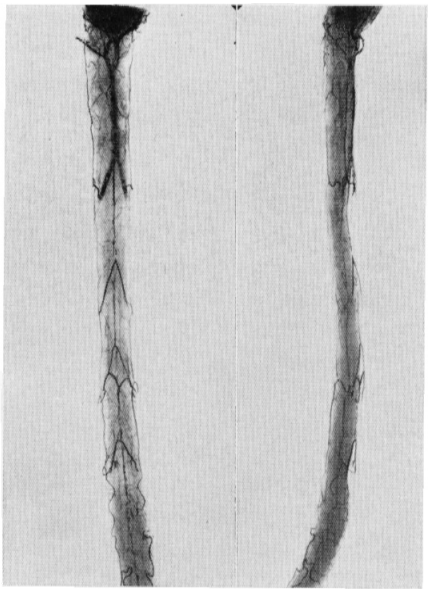


FIG. 2. Myelogram shows the obstruction of cerebrospinal fluid passage at the level of compression.



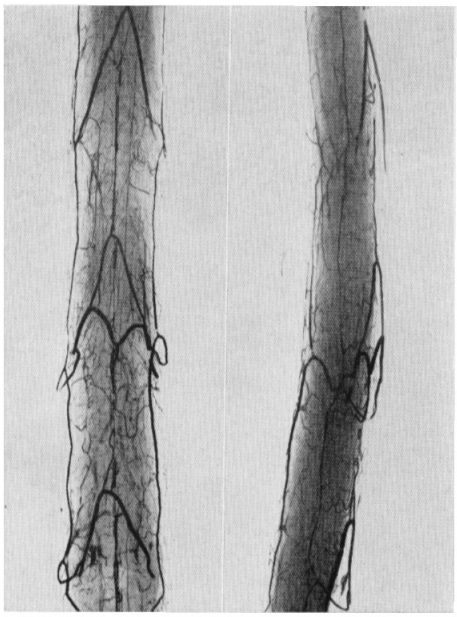
A B

FIG. 3. Angiogram of a normal cervical spinal cord.
A: A-P view. B: Lateral view.

cord is fairly constant in contrast with considerable variation reported in other animals and in man.

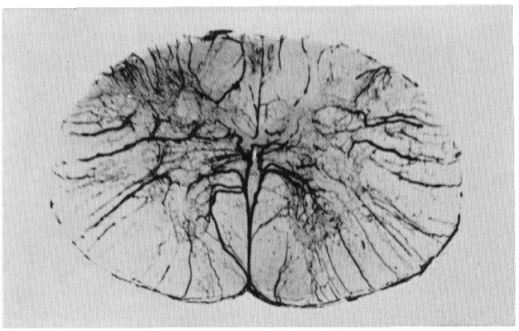
A radicular artery branches off from the vertebral artery and enters the spinal canal through each intervertebral foramen from the first cervical to the first thoracic vertebra and reaches into the subarachnoid space where it divides into anterior and posterior radicular arteries. At the level of cervical portion of the spinal cord, among them, the anterior radicular arteries distribute regularly segmentally while they are not so in more caudal segments of the spinal cord. The anterior radicular arteries are usually larger than the posterior radicular arteries and join the anterior spinal artery near the midline. The anterior radicular arteries passing through C_2 and C_3 intervertebral foramina on each side are the largest, and they make the ascending anterior spinal artery towards medulla oblongata in dogs. The anterior spinal artery, formed by the ascending and descending branches of the anterior radicular arteries, runs near the anterior midline all along the spinal cord.

The vertebral arteries in the dogs have an origin and a course similar to those in man. Distal to the origin of the C_3 radicular artery the vertebral artery becomes smaller and then anastomoses with a branch of the occipital artery at the level of superior border of the massive wing of the atlas. The vertebral artery, joined by the occipital artery and now termed the occipito-

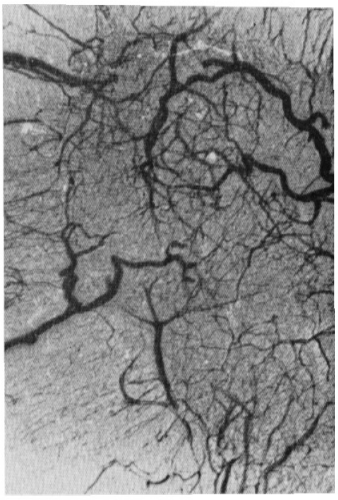


A B

FIG. 4



A



B

FIG. 5

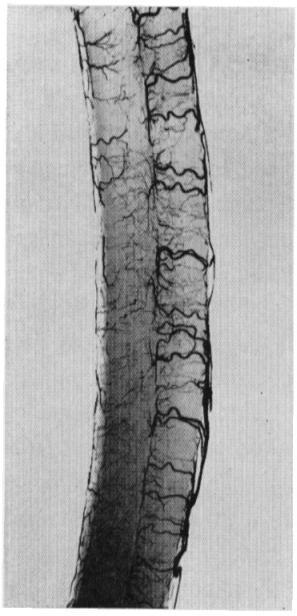


FIG. 6

FIG. 4. Angiogram of a normal cervical spinal cord (C_4-C_6).

A: A-P view. B: Lateral view. ×2.5.

FIG. 5. A: Angiogram of transverse section of a normal cervical spinal cord. B: Microangiogram of transverse section of a normal spinal cord. ×20.

FIG. 6. Parasagittal section of a normal cervical spinal cord.

vertebral artery, enters the oblique foramen of the atlas and forms an arterial diamond with the anterior spinal artery, the basilar artery and the occipito-vertebral artery from the opposite side. The unique arterial junction has been designated the cerebrospinal arterial anastomosis by de la Torre, *et al.*[20] In the study of normal spinal cord, vascular pattern is shown in Fig. 3, 4.

The anterior spinal artery gives the branches called the central arteries segmentally into the anterior spinal fissure; usually several central arteries run into one spinal cord segment. This artery divides to the left and the right just before the central canal of the spinal cord and these branches supply the anterior and lateral horns. The circumflexing branches run around the spinal cord, making so-called pial plexus and send perforating branches into the anterior and lateral fasciculi.

The postero-lateral spinal arteries which run on the posterior surface of the spinal cord send perforating branches into the posterior horn and posterior fasciculi of the spinal cord. These perforating branches also make anastomosis in the parenchyma usually in the grey matter (Fig. 5 a, b). Sagittal section of the normal spinal cord reveals the vascular pattern as Fig. 6.

The spinal central arteries run in the anterior spinal fissure nearly in right angle to the axis of the spinal cord and end just anterior to the spinal central canal, which divide off to both sides on the horizontal plane. There is fairly large anastomotic vessel along the spinal axis between these ends of the central arteries, but there is no anastomotic vessel in the course through the anterior spinal fissure.

From the posterior surface, the perforating branches are seen from the postero-lateral spinal arteries which appear poorer and smaller compared to the anterior spinal artery.

Now, the changes of pattern of these spinal arteries by experimental anterior compression of the spinal cord will be discussed (Fig. 7～22). They show complicated pattern and changes, depending upon the severity and the portion of actual compression of the spinal cord, and the common findings can be summarized as follows:

1) Arterial distribution patterns show circulatory insufficiency by the spinal cord compression on the morphological observation.

2) These changes are most sensitively seen of the central arteries, and not of the anterior spinal artery as some authors have described. The anterior spinal arteries never show mechanical occlusion by the mild or moderate compression in my studies.

3) The anterior spinal artery can be occluded by the extreme compression which is, however, just locally and not above and below the mechanical compression site.

4) The deformity of the spinal cord by anterior compression makes the

central arteries linked in their course. If the deformity of the spinal cord becomes severe, circulation of these arteries could be arrested (arterial obstruction). When the anterior spinal artery and the central arteries get involved in circulatory insufficiency, longitudial anastomotic vascular nets around the spinal central canal at the compression site dilate for compensation.

Therefore, the anterior spinal artery may not be considered as the main blood supply of the spinal cord, but the responsibility of the central arteries and the anterior spinal artery should be regarded as a longitudinal anastomotic chain. These features will be described in detail with actual demonstration of the microangiograms of the spinal cord compression.

DISCUSSION

As described in the introduction of the article, many theories have been stated as to the pathogenesis of the spinal cord compression especially on the problems of circulatory changes of the compressed spinal cord. And, as I have demonstrated with the micro-angiographic technique, the main and important changes were seen in the intramedullary arteries group such as the central arteries and not the extramedullary vascular system such as the anterior spinal artery of which many authors[10)11)21)] have emphasized.

Barré[7)] suggested that the myelopathy of cervical spondylosis was caused by ischemia, and he noted that compression of the cervical roots in the intervertebral foramina would also impair the blood supply of the cord. Bucy, *et al.*[21)] mentioned the anterior spinal artery as a possible etiologic point of interest. On the dissection study of forty cadavers' spinal columns, Morton[8)] described that the disc herniations may have occurred slowly and the condition of the collateral circulation in the cord could probably have kept up with the gradually increasing pressure. Allen[9)] has observed, while operating on cases with cervical spondylosis, that the spinal cord may become blanched when the neck is flexed. His observation supported that relative movements of the spine and cord caused intermittent interference of the blood supply from the anterior spinal artery and its branches which produced the symptoms.

Brain[22)] made several observations concerning the vascular involvement associated with disc disease that were pertinent to a discussion of cervical spondylosis: "No doubt the earliest changes produced by a protruded intervertebral disc are circulatory, and these must be important at all stages. The veins with their thin walls and low pressure would naturally be compressed first and since the flow in them is upwards an edema of the anterior or anterolateral region of the cord at and for some segments below the site of compression would cause symptoms related to these segments. Arterial blood supply is likely to suffer late, if at all, but obstruction of the lateral spinal artery at the foramen would tend to cause ischemia of the lateral region of the cord.

The anterior spinal artery might conceivably be compressed by a mid-line protrusion, and in severe cervical spondylitis the vertebral artery and vein may undergo compression in their foramen".

These hypotheses of vascular insufficiency have been given support by many authors including Mair and Druckman[10], and Girard, *et al.*[12], although they do not agree on the actual mechanism.

On the other hand, Kahn[19] stated a direct mechanical cause of the neurological symptoms and stressed the role of the dentate ligaments in spinal cord compression. Bedford, *et al.*[23] followed Kahn in their emphasis on the fixation of the cord by thickened dentate ligaments and noted that unusual tension on this structure and a tethering of this structure had played some role in the production of the cervical myelopathy.

Bucy, *et al.*[21] also supported the view that Kahn's mechanical explanation of the development of the symptoms is more applicable than is the vascular one. But according to Mair and Druckman's article, although Kahn's theory postulated that mid-line disc protrusions would cause maximal damage to the anterior columns of the cord, such a lesion was not formed in the 3 cases where the cord was examined in transverse section. Furthermore, Brain, *et al.*[22], and Clarke and Robinson[3] have stressed that operation does not often provide clinical improvement despite the release of a compressed spinal cord by sectioning the dentate ligaments.

Other authors studied the sagittal diameter of the spinal canal. Indeed, there is a considerable individual difference in the natural antero-posterior diameter of the cervical spinal canal. This was first demonstrated by Boijsen[24]. Wolf, *et al.*[25] found that the lower 5 cervical vertebrae had a sagittal diameter measuring 17 ± 5 mm and that where simple measurement of the narrowest distance from a spondylotic bar to the nearest point on the posterior wall of the canal was 10 mm or less, cord compression was likely, whereas at 13 mm or more, spondylotic myelopathy was unlikely. This measurement has been confirmed by Payne and Spillane[26]. They found that in cervical spondylotic spines, the average measurement of the cervical canal was smaller by about 3 mm (14 mm as opposed to 17 mm) than normal, and spondylotic changes reduced the size of the canal by myelopathy was more likely to occur when the original dimensions were smaller than the normal. Pallis, *et al.*[27] and Nugent[4] made similar observations.

Wilkinson[28] noted that in patients with spondylotic myelopathy, the smaller measurements were found not only at the levels of the decreased vertebral discs. but also throughout the cervical spine, and signs of cervical myelopathy were likely to develop if the antero-posterior diameter of the canal was reduced below 10 or 11 mm. These findings were agreed by McRac[29], Burrows[30], and Symon, *et al.*[31].

On the other hand, Breig, *et al.*[32] found on the cadavers' study that the cord was grossly distorted by spondylotic ridges when the neck was flexed, and cervical extension failed to produce any evidence of compression of the cord. Aboulker, *et al.*[33] and Reid[34] felt that the cord might be damaged by cervical flexion as well as by the compression that accompanied cervical extension.

In addition to the bony changes, some investigators have drawn attention to the importance of the ligamentum flavum as a cause of symptoms. By the radiological study on cadavers, Taylor[35] has emphasized that these ligaments, bulging anteriorly in cervical extension, cause recurring trauma to the cervical cord raised upon a disc protrusion or an osteophytic bar. X-ray-examination study in living subjects by Nugent[4] and macroscopic and microscopic studies by Stoltmann[36], Payne and Spillane[26] showed clearly that the ligamenta flava bulged anteriorly. But, according to Bradshaw's article, there was not much evidence that the mechanism played an important role in the production of the myelopathy of cervical spondylosis, although a post-mortem examination was described by Taylor[35] in which forward bulging of the ligamentum flavum had lacerated the posterior columns of the spinal cord.

On the other hand, some authors have described the adhesions that frequently are found at operation between the dura mater and arachnoid as well as the arachnoid and pia mater. Brain, *et al.*[22] found that the dura mater was thickened and adherent to the posterior longitudinal ligament, and emphasized that this formation of extradural adhesions limited movement of the spinal cord, and they mentioned the greater deformity to occur in extension. Frykholm[37] described epidural and arachnoidal adhesions restricting normal movement of the spinal cord and nerve roots. Allen[9] has observed at operation that adhesions around the protrusions and the nerve roots fix the cord preventing the gliding which normally takes place during cervical movement. But Bradshaw[6] has reported that in his series adhesions are never of a degree of great etiological importance.

As vascular factors causing cervical myelopathy have been suggested before, Mair and Druckman[10] described the histological changes in the cervical cord which resulted from compression of the anterior spinal artery and its branches by the protruded disc. Greenfield[11] believed that the areas of demyelination were too limited to be explained on the basis of direct compression of the cord. He agreed that interference with the blood flow through the anterior spinal artery was probably the important factor. Tureen[38] postulated compression of the anterior spinal artery or its central branches to explain some of the clinical features of disc herniation. Höök[39] also noted that the mechanism of producing damage to the spinal cord was probably due to successive and temporary occlusion of different arterial branches, mainly from the

anterior spinal artery, but also, in part, from the peripheral arteries. Bucy, *et al.*[21] and Knight[22] have mentioned the anterior spinal artery as a possible etiologic point of interest.

However, Wilkinson[38] stated that in many cases with flattened or destorted spinal cord corresponding to disc protrusions there was no evidence of gross change in the anterior spinal artery. Breig[32] has also commented that the anterior spinal artery is probably well protected as it would simply be pushed deeper into the anterior median fissure by any spondylotic ridges.

It seems possible that the main blood supply to the cervical spinal cord may come through the radicular arteries, and the anterior radicular arteries passing in the intervertebral foramina may well be compressed if there is gross narrowing of the foramina due to osteophytic bars or if there is adhesions with root-sleeve fibrosis. Many writers have emphasized this finding. For example, Bradshaw[6] noted that because of the blood supply of the cervical cord by only two or at most three unpaired radicular arteries, foraminal compression of these vessels by osteophytes or adhesions associated with root-sleeve fibrosis may severely impair the blood supply of the cord. Recently, Taylor[5] also described that finding at operation and histologic examination of cervical myelopathy showed that the radicular arteries could be compressed in the narrowed foramina and fibrous perineural root-sleeves. Breig, *et al.*[32] further stated that narrowing of extra-spinal arteries by contact with spondylotic ridges and other hypertrophic connective tissue contributed to the reduction of blood supply to the cervical cord and, especially, have emphasized the possible significance of variations in the number and location of the radicular arteries. But Payne[40] commented that on anatomicopathological study, compression and obliteration of the vessels traversing the constricted foramina have never been demonstrated. Breig, *et al.*[32] also stated that so far as they know, no occluded radicular artery has ever been demonstrated in post-mortem examination in a case of myelopathy due to cervical spondylosis.

With regards to the circulation through the vertebral arteries, both numerous anatomical studies and clinico-pathological observations of many different syndromes due to the tortuosity and compression of these arteries have been reported. Hadley[41] described that the vertebral arteries may be deflected and distorted by extrinsic pressure of bony spurs at the covertebral or apophyseal joints, and that the decreased blood flow may be a factor in producing neurological symptoms. Hutchinson[42] commented that cervical spondylosis capable of distorting the vertebral artery, may play a subsidiary role in obstructing one or another vertebral artery during movement of the cervical spine. Radner[43] has described the partial occlusion of the vertebral artery by cervical osteophytes.

Furthermore, narrowing of the vertebral artery by head and neck movement has been shown by some authors. Maslowski[44] suggested that the lumen of

the vertebral artery could be constricted by the interarticular joints and joints of Luschka in middle aged individuals when head and neck were rotated towards the side of the artery. At any rate, narrowing of the vertebral artery and a decrease in its flow were frequently observed only by various movements of the head and neck. But Mizuno, *et al.*[45] demonstrated experimentally that occlusion of the vertebral or the radicular arteries caused slight cord damage.

Furthermore, Girard, *et al.*[12] have described hyaline degeneration of the smaller intramedullary arteries as pathological change in the brain stem and spinal cord to be on a vascular basis. It is secondary to cervical spondylosis without evidence of compression of the cord, and it is further noted that myelopathy has not, as a rule, been restricted to an area supplied by a single vessel.

Nugent[4] also described the numerous aberrant blood vessels in the subarachnoid space that often were associated with an arachnoiditis. These vessels played some role in a significant proportion of the blood supply of the cord with some degree of vascular insufficiency.

On the other hand, Brain[22] mentioned, as described above, congestion of the veins in the anterior and antero-lateral regions of the cord below the spondylotic bars as one explanation of the early spinal cord symptoms. However, Breig, *et al.*[32] described that the venous drainage system was too extensive to be interfered significantly by cervical spondylosis, and there was no evidence to suggest that the lesion of the compressed segment was a result of venous stasis in their cadavers' studies.

Recently, Mizuno, *et al.*[45] emphasized that extramedullary vessels of the cervical spinal cord, including the anterior and posterior spinal arteries and pial plexus, were of considerable significance in producing spinal cord damage.

At any rate, mechanical compression of the spinal cord is the primary factor of this disease, and no doubt, the vascular factors play some role even if not the most important.

The microangiographic technique used in the presnt study was a modification of that previously described by Barclay[46], Tirman, *et al.*[47], Bellman and Engström[48], and Bellman[49]. The method is contact microradiography of fixed specimens in which the arterial system has been filled with contrast medium. The microangiograms were investigated microscopically or after magnification with conventional photographic methods.

By the microangiographic technique, one can demonstrate the small vessels such as arterioles, precapillary arteries and capillaries in the opaque tissue with a wider scope without damaging procedure to a piece of tissue. This, therefore, has the advantage of better visualization of various blood vessels in the tissue compared with the usual histological technique which has so many limitations to demonstrate the vascular system in a piece of tissue.

As to the study of circulatory system of organs, Barclay[50] stated the injection method of contrast medium, or there were reports of injection of plastic materials into vascular system of removed organs. These techniques are rather artificial because the organs are removed before the injection of contrast media. But I injected Micropaque while the animals were still living, in order to follow the physiological circulation.

Bellman[49] attemped the vital microangiography, but he could only demonstrate the vascular system of rabbit's ears. More investigation is needed before one can apply it to other organs. Therefore, the present study can be very useful to observe the circulatory conditions of organs if one consider various factors such as constriction of vessels, circulatory volume, vascular resistance, organ circulation, etc.

As to the experimental spinal cord compression, Kahler[51], Allen[52], Bornstein[53], Matsumoto[13], Amako[14], Tarlov[15], Izumida[16], Kaneko[17], Kano[18], and other authors have reported.

However, all of them reached the spinal cord by laminectomy which necessitated the opening the spinal canal. I attempted to approach to the spinal cord without laminectomy through the intervertebral space so that I did not have to open the spinal canal.

Some authors as Matsumoto[13] used glass ball to compress the spinal cord, or Kano[18] tied the spinal cord. In the present study, the laminaria was investigated to produce gradual and continuous spinal cord compression to imitate the clinical cord compression mechanism.

It was observed spinal paralysis or its recovery from not only the clinical or pathological follow up but with microangiographic technique to demonstrate the circulatory disturbances of the spinal cord by experimental compression.

SUMMARY AND CONCLUSION

Many theories of pathogenesis of the spinal cord compression especially in the case of cervical spondylosis are reviewed.

Experimental spinal cord compression was made through the anterior approach without laminectomy procedure in dogs, and neurological observations were made. Dogs were sacrificed and investigation of spinal cord arteries was made with the microangiographic technique.

It was confirmed that the vascular system played an important role on the spinal paralysis with experimental compression, and the early and maximum changes were seen in the intramedullary arteries such as the spinal central arteries. The anterior spinal arteries could be occluded, but only with extremely severe compression.

Attention should be made in treating these disorders with consideration of improvement of the circulatory disturbances, of the spinal cord.

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Spinal cord compression of mild degree. Mild degree of the cord compression means the reduction of the A-P diameter of the cord less than 1/4 of normal diameter. This group usually developed minimal change of motor function which recovered promptly.

FIG. 7. A-P view of gross specimen shows small arteries of the spinal cord to be poorly visualized and the perforating branches such as central arteries seems smaller but the anterior spinal artery showed no change. No essentially conclusive finding can be observed on this photograph. Spec. no. 40.

FIG. 8. Parasagittal section can reveal a little more findings. The spinal cord is seen strained smaller in A-P diameter but no changes by compression onto the anterior spinal artery. However, the central arteries reveal clearly their changes, that is, poor filling, being pushed antero-posteriorly, and being bent like an accordion. Spec. no. 9.

FIG. 9. Photograph of the accordion-like bending of the central arteries, ×20, Spec. no. 9.

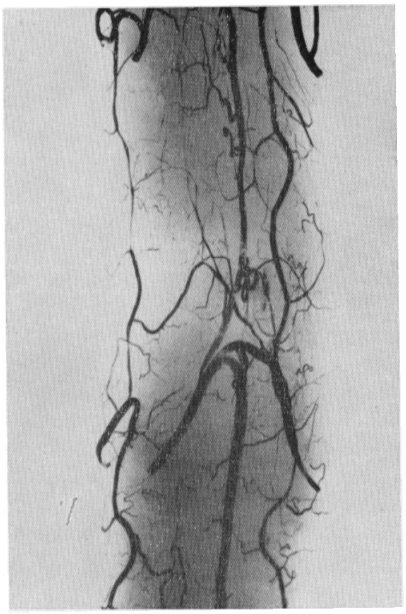


FIG. 7

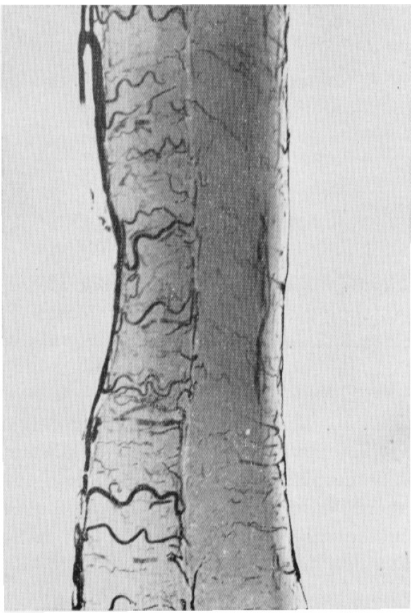


FIG. 8

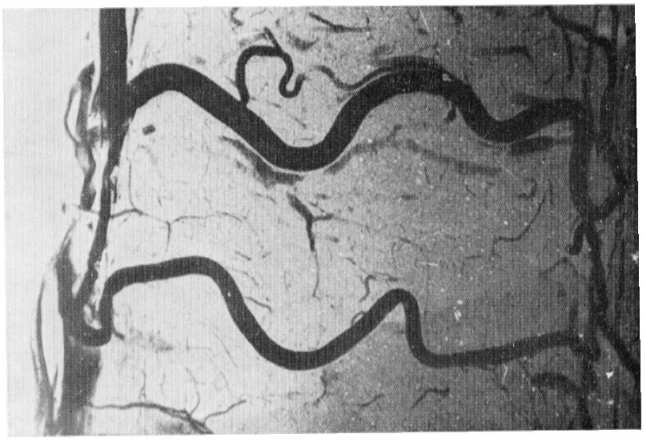
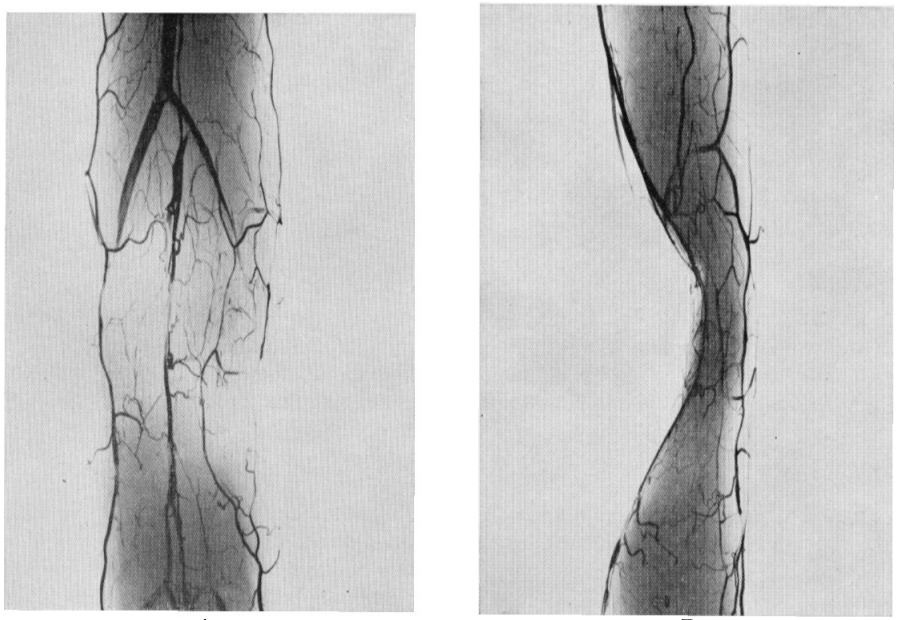


FIG. 9

Spinal cord compression of moderate degree. Moderate degree of the cord compression means the reduction of the A-P diameter of the cord more than 1/4 and less than 1/3 of normal size. Dogs of this group developed gait disturbances.

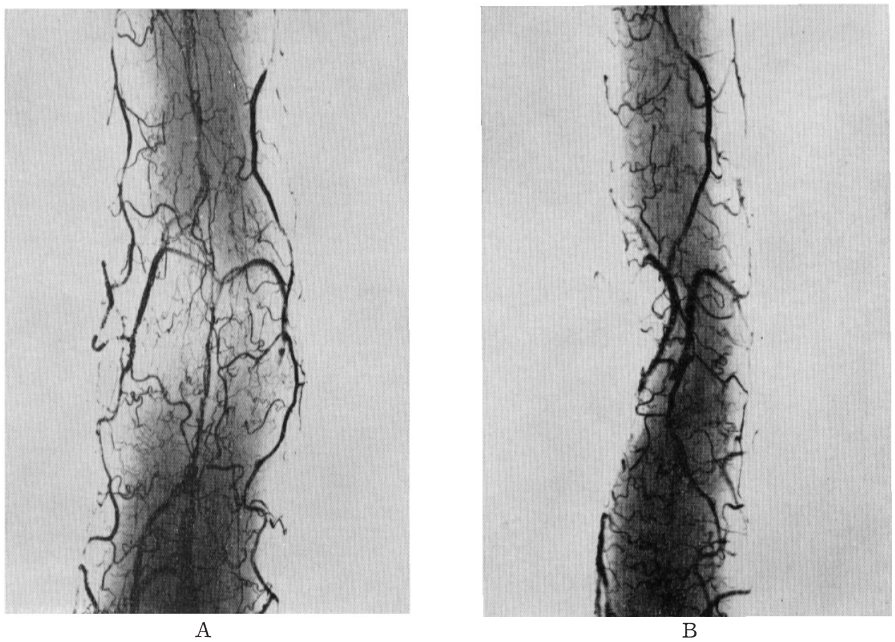
FIG. 10. A-P view shows no gross changes of vascular pattern of the spinal cord except the poor vascularization of parenchyma at the compression site, but the lateral view demonstrates the marked deformity of the spinal cord. At the compression site, the spinal cord is seen markedly small and strained in A-P diameter. However, in spite of this severity of compression, the anterior spinal artery is seen patent. A: A-P view. B: Lateral view. Spec. no. 32.

FIG. 11. In this dog spinal cord compression of moderate degree was made and developed complete paraplegia for 4 days and then recovered. This dog was sacrificed 50 days after the compression as a chronic experiment. In spite of severe deformity of the spinal cord, the anterior spinal artery is seen patent and increased vascularity is seen around the compression site which is assumed to have developed as collateral circulation for recovery. Spec. no. 36. A: A-P view. B: Lateral view.



A B

FIG. 10

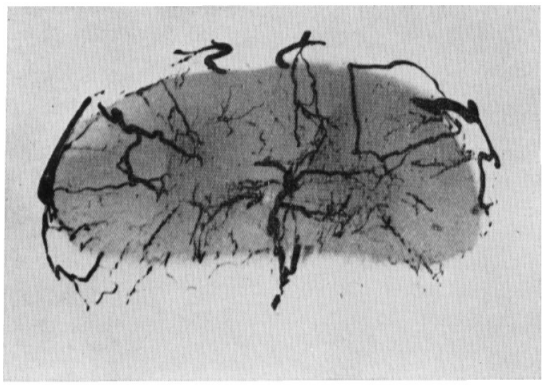


A B

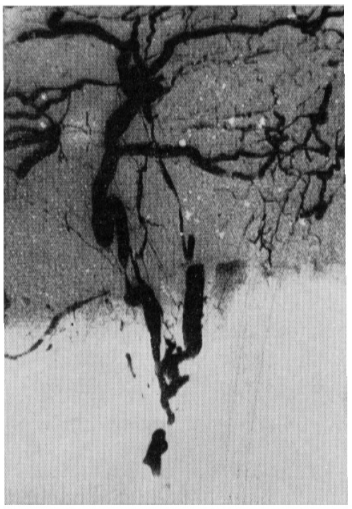
FIG. 11

FIG. 12. A: This shows the transverse section of chronic compression experiment with recovery. All arterial vessels seem engorged and kinking, including the central artery. B: The photograph of $\times 20$ magnification shows dilated and kinked central artery which might be one of the compensatory mechanism of recovery from paralysis. Spec. no. 36.

FIG. 13. Parasagittal section shows fairly marked deformity of the spinal cord, that is, the A-P diameter of the spinal cord was reduced to about one third of normal diameter. The anterior spinal artery can be observed patent with the diameter markedly reduced at the compression site. Essential change is poor filling of the central arteries at the compression site. Please notice that the increased vascularities along the central canal area may be interpretated as the anastomotic increase from superior or inferior segment of the spinal cord. The central arteries at other segments of the spinal cord than the compression site are fairly regularly visualized. Spec. no. 35.



A



B

FIG. 12

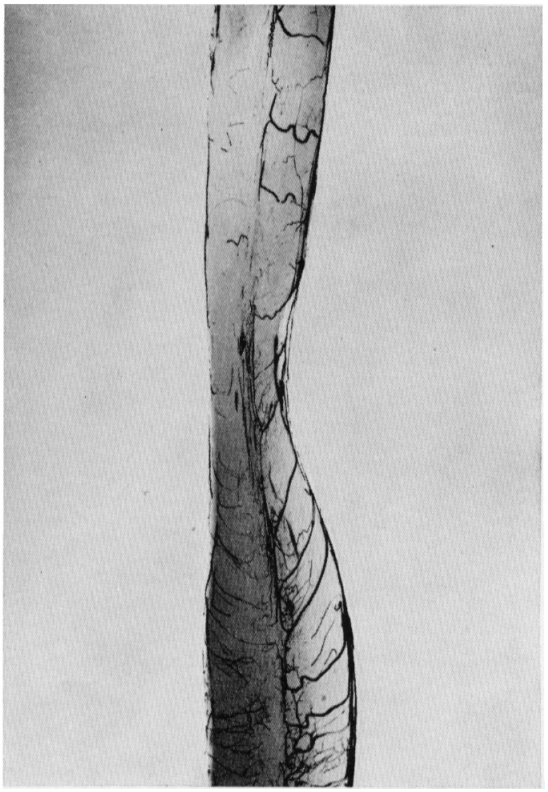


FIG. 13

FIG. 14. This photograph shows the marked accordion-like bending of the central arteries. Some appear like this and some of them seem obliterated and show no filling on the angiogram. There are increased vascularity seen along the central canal as shown at the right bottom corner of this picture. Spec. no. 26. ×20.

FIG. 15. This shows the bending of the central artery and the smaller anterior spinal artery under the ×20 magnification. In this photograph a few large arterial vessels are identified which run along the central canal. I believe this is the anastomotic blood suppply as collateral. Spec. no. 35. ×20.

FIG. 16. Transverse section of lateral compression of the spinal cord shows poor fillings of the perforating arteries at compression site and markedly dilatated vessels in posterior horn plexus of artery on opposite side. Spec. no. 27.

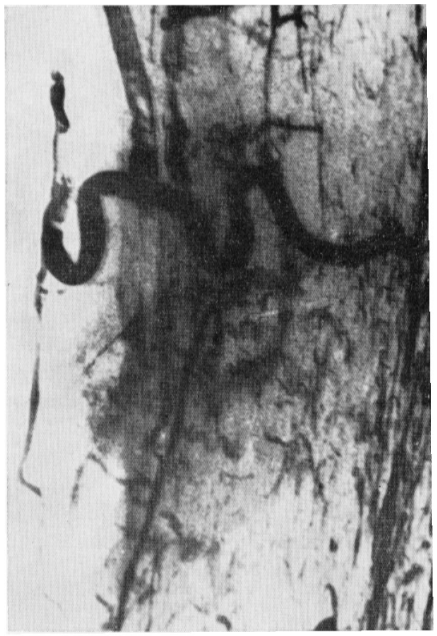


FIG. 14

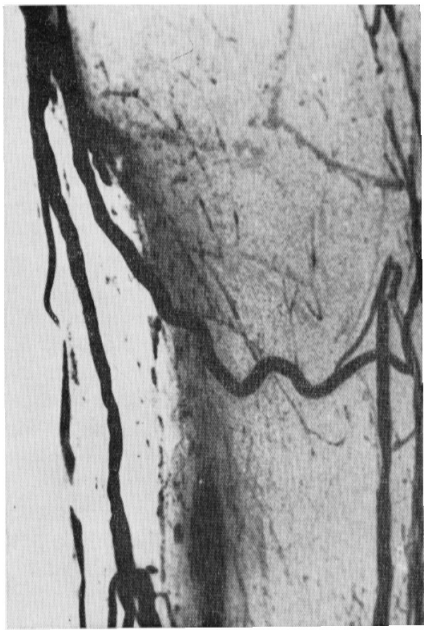


FIG. 15

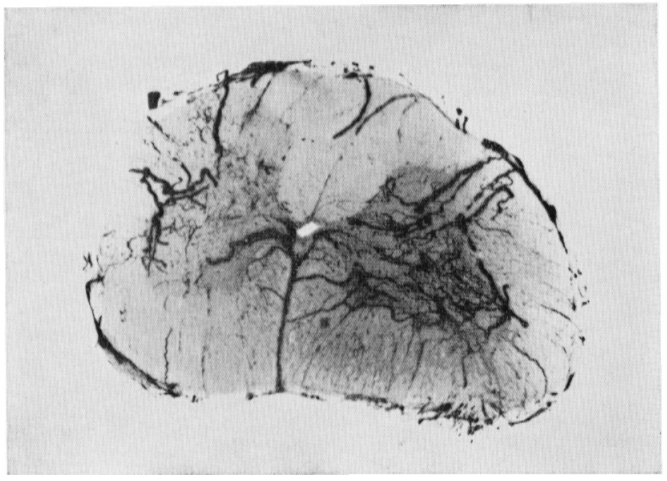


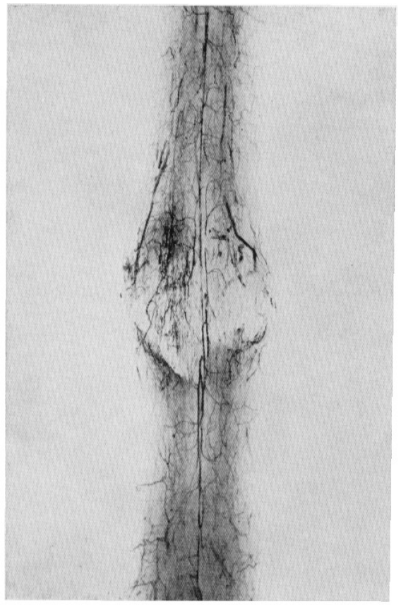
FIG. 16

Spinal cord compression of extreme degree. Extreme degree of the cord compression means the reduction of the A-P diameter of the cord more than 1/3 of normal size. Dogs of this group developed complete paralysis of the limbs and recovery was exceptional.

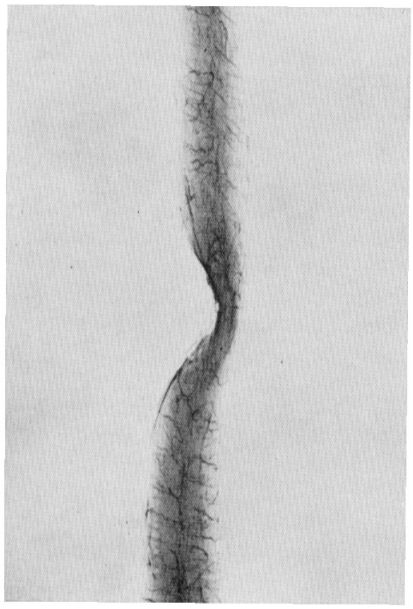
FiG. 17. Lateral view shows deformity of the spinal cord by marked compression. The A-P diameter of the spinal cord is reduced to nearly half of normal diameter. Similar changes are noted in moderate compression experiments. In some experiments, the anterior spinal artery is seen obliterated, but this photograph shows the fine but patent anterior spinal artery. A: A-P view. B: Lateral view. Spec. no. 43.

FIG. 18. This is the transverse section of the extreme compression. Please note the deformity of the spinal cord which is flattened antero-posteriorly while the lateral poles are well fixed by the dentate ligaments. Spec. no. 43.

FIG. 19. This specimen shows obliteration of the anterior spinal artery by extreme compression. Only two central arteries are seen to be running longitudinally at the compression site. Vessels along the central canal area show dilatation. Spec. no. 25. Parasagittal section.



A



B

FIG. 17

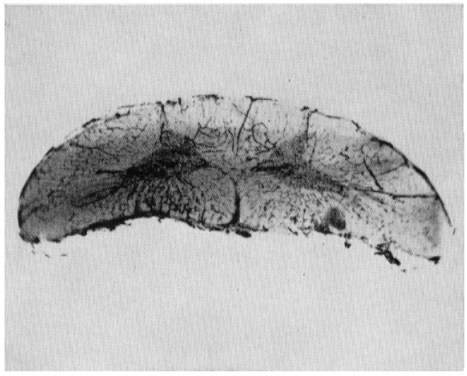


FIG. 18

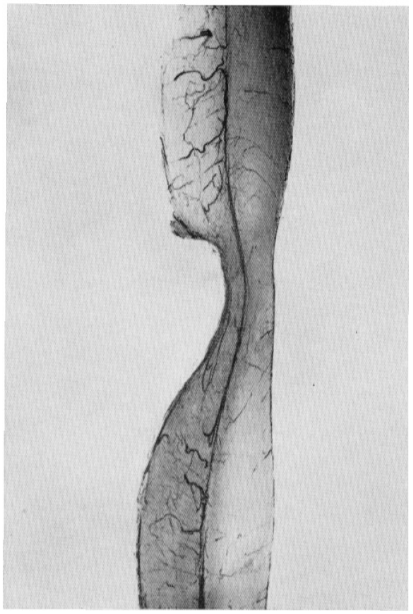
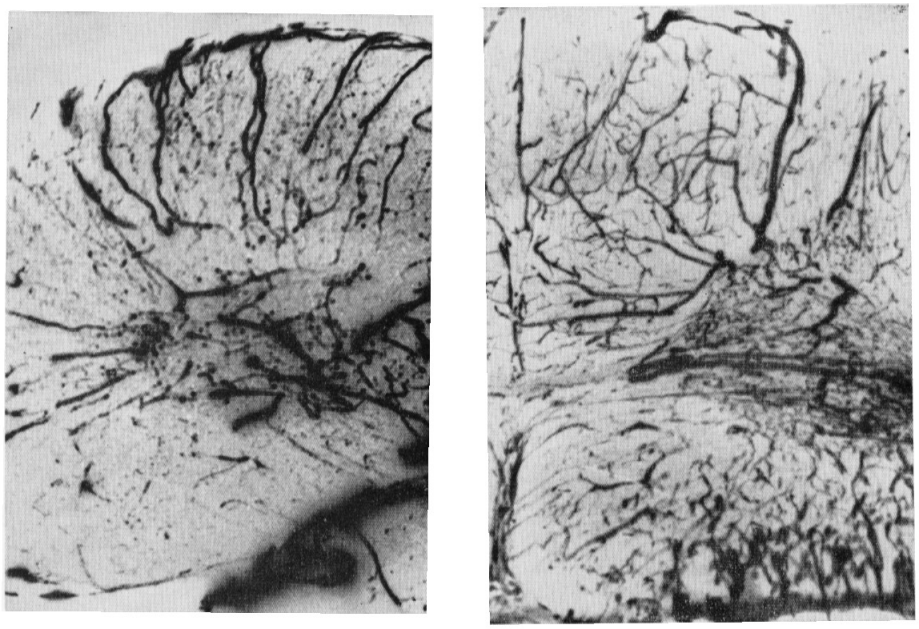


FIG. 19

FIG. 20. Transverse section under the ×20 magnification shows marked engorgement of posterior horn plexus of artery to supply the central structure of the spinal cord from the postero-lateral spinal arteries. A: Spec. no. 42. B: Spec. no. 43. ×20.

FIG. 21. Parasagittal section of extreme compression site under the ×20 magnification is shown. Neither anterior spinal artery nor any central arteries can be seen, but the vascular pattern along the axis of spinal cord can be observed to be remarkable. Spec. no. 25.

FIG. 22. This shows parasagittal section under ×20 magnification. Accordion-like bending of the central arteries is observed right next to the compression site. These arteries are seen larger than usual and branch off from the anterior spinal artery. Please notice filling defect of the anterior spinal artery in its lower half and, of course, no central arteries are visualized in this segment. Spec. no. 26.



A B

FIG. 20

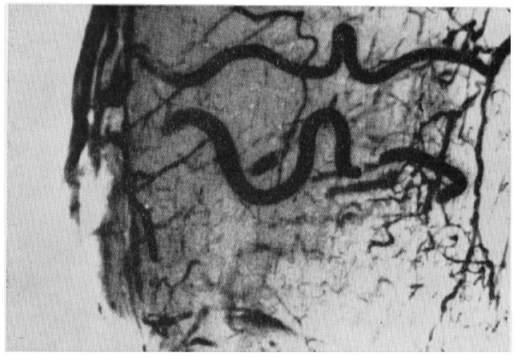


FIG. 21

FIG. 22