Some aspects of the pathology and pathogenesis of the myelopathy caused by disc protrusions in the dog

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SUMMARY The pathological changes in 23 cord lesions caused by disc protrusion are described. It is suggested that three broad classifications of damage can be found: (1) compressive change; (2) malacia; and (3) diffuse demyelination. The distribution of damage, within the segment, was different in each type of change as were the glial and vascular reactions. It is suggested that vascular factors play a major part in the pathology of the malacic and demyelinating lesions and may be of secondary importance in compressive lesions.

Several authors have described pathological aspects of the myelopathy caused by intervertebral disc protrusion in dogs affecting both the cervical and thoracolumbar areas (Hoerlein, 1953; Olsson, 1960; Funkquist, 1962; Lindblad, Ljunggren, and Olsson, 1962; Palmer, 1964, 1970; Wright and Palmer, 1970). It is well known that clinically and pathologically dogs may exhibit either a localized myelopathy or signs of more extensive involvement. This latter condition is often called the ascending syndrome (Hoerlein, 1953; Funkquist, 1962) and is characterized by extensive myelomalacia and meningeal haemorrhage. The purpose of this report is to describe the pathology of the localized form of myelopathy and no cases of the ascending syndrome are included. The maximal number of segments involved in any dog was four, and all protrusions were in the thoracolumbar area.

METHODS

The material for this investigation was obtained from 21 dogs with myelopathy due to disc protrusions. Two dogs had been previously treated successfully for protrusions of another disc and, therefore, the number of lesions studied was 23.

Sixteen dogs were destroyed after conservative treatment and one died after a urinary tract infection. Four dogs were treated surgically, one dying under anaesthesia and three being destroyed subsequently after failing to recover.

Most cords were removed from the vertebral canal for fixation, although in a few cases the entire vertebral column was removed and fixed *in situ* to

study the compressive changes resulting from the protrusion. Samples from three cords were fixed in formol ammonium bromide for seven days when frozen sections were cut for staining with Cajal's gold sublimate. The remainder were fixed in 10% formol saline for two to three weeks and were sectioned and embedded in paraffin wax. The stains employed were haematoxylin and eosin (H and E), van Gieson, Mallory's phosphotungstic acid haematoxylin, cresyl violet, Holzer, Martius scarlet blue, Marchi, Weigart Pal, and Glees silver impregnation.

RESULTS

The examination of these cords suggested that three broad classifications of lesion could be made.

1. Compressive change: this was estimated at surgery or at necropsy, after fixation of the cord *in situ* or after removal for fixation, and was recognized by indentations or flattening of the cord (Fig. 1). These distortions of cord shape could then be assessed microscopically.

2. Malacic areas: these were localized areas of necrosis of the nervous tissue often with preservation of the vasculature and the mesenchymal elements (Fig. 2). In lesions of short duration, phagocytic cells were present but cystic spaces were the eventual result.

3. A diffuse demyelination of the white matter which varied in severity from area to area. The term demyelination is used to indicate a myelinoclastic process as defined and illustrated by Wright and Palmer (1970). The degeneration involved both the axons and myelin sheaths and was accompanied by a glial reaction (Fig. 3).

FIG. 1. *Compressed cord, junction T9/10 segments. Note radial fissures in white matter, and anchoring effect of denticulate ligament on left. Period of survival three weeks. H and E, $\times 9$.*

FIG. 2. *L5 segment showing characteristic areas of malacia. Note trabeculae of mesenchymal tissue crossing these areas and lack of cord compression. Seven weeks' survival. H and E, $\times 9$.*

TABLE	
FREQUENCY OF OCCURRENCE OF LESIONS	
Process	Cord lesion (no.)
Malacia alone	1
Demyelination alone	3
Compression alone	8
Malacia and compression	2
Malacia and demyelination	5
Demyelination and compression	1
Demyelination, compression, and malacia	3
Total	23

It was found that one cord might show mainly one type of appearance, or a combination of all three. The three processes might be present at the same segment or in different segments. The frequency of each process is shown in the Table.

COMPRESSIVE CHANGE As would be expected, maximal compression occurred at the level of the protrusion. It was most marked in those 'button like' protrusions classified as type I by Funkquist (1962). Where the protrusion had spread out along the epidural space, as in Funkquist's type III, the compression was minimal or could not be demonstrated. When the protrusion remained in the mid-line the compression was usually bilateral, whereas a dorsolateral protrusion produced mainly unilateral compression. The compression caused dorsoventral flattening of the cord with an increase in the transverse distance of the lateral columns. In four specimens it appeared as if the cord were anchored laterally to the dura by the denticulate ligaments which prevented any displacement of the cord (Fig. 1). This anchoring effect was either unilateral or bilateral, depending on the position of the protrusion. This feature was not found in all cords with obvious compression. In conjunction with this anchoring effect, small radial fissures could sometimes be found running from the grey matter towards the pial attachment of the ligament (Fig. 1). Whether these are true pathological 'lines of stress' or an artefact produced by section of previously damaged tissue is not certain, but they were present when the dura mater was removed before section and lipid phagocytes were found along these fissures.

In the majority of compressed cords the anatomical divisions of grey and white matter could be distinguished but, in a few, total disruption of structure had occurred. The white matter was usually more severely affected than the grey. The lateral columns showed more damage than the ventral with the dorsal columns usually being least affected.

In transverse section many of the axons were swollen, staining pink in H and E sections with occasional axons showing basophilic stippling. In longitudinal sections, some axons showed irregular swellings along their length and ended in large retraction balls, a feature best demonstrated in silver impregnations. The number of retraction balls decreased as the period of survival increased.

FIG. 3. T13 segment showing diffuse demyelination. Note lack of compression and small irregular malacic areas in both lateral columns and base of dorsal column. Six weeks' survival. H and E, $\times 9$.

FIG. 4. Two blood vessels at site of compression. Note increased connective tissue of adventitia. Twelve weeks' survival. MSB, $\times 400$.

Some of the myelin sheaths were swollen and contained myeloclasts. These cells contained little cytoplasm and the mature lipid phagocytes, with swollen foamy cytoplasm, were seldom found. The microglia appeared to be the earliest cells to react and their response was limited mainly to the perivascular areas where they were found in small clumps around the intramedullary vessels. Very little evidence of phagocytosis could be seen in these cells. In the areas between the vessels only an occasional myeloclastic cell could be found. The perivascular accumulations of microglia were still present one year after onset.

Irregular patchy demyelination could be demonstrated by myelin stains and appeared to proceed slowly. This was compatible with the presence of a few actively phagocytic microglia. With the swelling of the myelin sheaths and removal of degenerate myelin, spaces and fissures of various sizes developed giving the white matter a slightly 'moth-eaten' appearance. The astrocytic response in the white matter varied but in most cords a general increase in astrocytic nuclei could be seen in H and E sections. The nuclei appeared slightly swollen and often small groups of astrocytes could be found, suggesting proliferation. Slight swelling of the cell body was also seen. Sections stained for astrocytic fibres failed to demonstrate any degree of neuroglial sclerosis.

A striking feature encountered in all but one of the cords showing compression was a marked increase in blood vessels in the white matter and this was greater in the areas showing the most damage. It was, therefore, usually most marked in the lateral columns and least in the dorsal. The increase could be marked at two and a half weeks and was still present at one year after onset.

The new vessels were mainly precapillary arterioles. Many of these arterioles, together with the veins, had thickened adventitia due to an increase in collagen. The earliest increase in adventitial collagen appeared at about two weeks with a very thin layer around some vessels. The amounts increased until, in long-standing lesions, the adventitia became markedly thicker (Fig. 4). In some instances thickened vessels could be seen invading the cord from the meninges. It is interesting to note that in the compressed cords with these vascular changes the compression was gradually increasing as judged from the clinical history and signs, while in one case where the compression was acute the increase in vessels was minimal with very little increase in the adventitia.

MALACIA Most of the cords with malacia had little evidence of compression. The malacic areas were not always maximal at the level of the protrusion and in some cords were not even present at the site of the protrusion. The malacia was mainly in the white matter with only the periphery of the grey matter being affected.

The position of the malacic areas varied from cord to cord and between segments in a cord. The characteristic sites were in the dorsolateral

FIG. 5. Upper: section from posterior of T10 segment. Lower: section from middle of T11 segment. Four weeks' survival. H and E, $\times 9$.

FIG. 6. T13 segment. Note malacia in base of dorsal columns, right lateral and ventral column, and grey matter. The peripheral white matter is spared. One year survival. H and E, $\times 9$.

funiculus, the lateral funiculus, the base and central area of the dorsal funiculus, and the ventral funiculus (Fig. 2). Figure 5 shows how the position of the malacia may vary within one cord. The shape varied from circular to triangular with the base of the triangle being to the periphery. There was, in many instances, a strip of nonmalacic white matter between the malacic area and the pia mater, while in other sections the malacic area extended to the meninges causing outward bulging of the pia mater. Adjacent areas of malacia could fuse to cause large crescentic shaped areas of necrosis which might involve the tips of the dorsal horns.

In two cords the area of malacia varied from this. In these cords there was involvement of the whole lateral and ventral column with the exception of a thin strip of white matter adjacent to the pia mater. The base of the dorsal column was involved together with the grey matter (Fig. 6). One case was of a few days' duration, whereas the other had survived for over a year. The more recent case showed necrosis of the areas mentioned with numerous lipid phagocytes. There was also marked proliferation of astrocytes, especially in the grey matter with some swelling of their cell bodies. There was a marked increase in blood vessels throughout the malacic area in both cords.

The character of the border with the non-malacic tissue varied with the stage. In the case of shortest duration (eight days) there was a well established triangular area of malacia in one dorsolateral funiculus. The borders were ragged and ill defined with capillary proliferation, whereas with longer duration the borders were clear cut and well defined and the vessels were surrounded by a thin sheath of collagen.

Within the malacic areas, necrosis of all neural elements had occurred with phagocytosis of breakdown products by numerous lipid phagocytes. Lesions of longer standing contained eosinophilic fluid. Trabeculae of blood vessels and connective tissue crossed the malacic areas, often surrounded by lipid phagocytes. An increase in microglia in the bordering areas was noted. Astrocytes were increased in the border zone and showed slight swelling. Stains for neuroglial fibres demonstrated a fine sclerosis around the malacic areas.

DIFFUSE DEMYELINATION Cords showing diffuse demyelination often had no evidence of compression but demyelination was frequently

present at the same level as malacic areas. The demyelination in the white matter might affect all columns or only small areas of one column. There appeared to be no obvious preferred site. In two cords small, irregular malacic areas were present in the centre of the vacuolated white matter. These areas did not resemble the previously described malacia in appearance, being smaller, slowly developing, and with poorly defined borders (Fig. 3). The demyelinated areas contained numerous swollen myelin sheaths, some of which contained swollen or fragmented axons. In lesions of short duration,

FIG. 7. L2 segment. Note neuroglial fibres in demyelinated area. Six weeks' survival. Holzer, $\times 160$.

retraction balls were present but their numbers decreased with longer periods of survival. There was considerable variation in the size of the swollen myelin sheaths, and lipid phagocytes in various stages of development were present. There was an increase in microglia in the white matter, with small clumps either in the vacuolated area or surrounding vessels. Holzer's stain demonstrated a neuroglial sclerosis, the fibres being displaced by the swollen myelin sheaths. The appearance was, therefore, of swollen degenerating fibres being supported on a fine network of neuroglial fibres (Fig. 7).

There was a moderate increase in small vessels, especially in the demyelinated areas. These vessels, which were mainly veins, were also surrounded by a sheath of collagen of varying thickness. The increase in vessels, how-

ever, was not usually as marked as in the compressed cords nor were the adventitial sheaths so thickened. No fibrin was demonstrated in or around the vessel walls in any cord.

CHANGES IN GREY MATTER Malacia was present only occasionally when the periphery of a dorsal horn was involved. Loss of neurones was common to all cords. The severity varied but dorsal, intermediate, and ventral areas of grey matter were involved. The cell loss was often marked and in longer standing lesions the grey matter had a fenestrated appearance due to loss of neurones and fibres. This fenestrated appearance was most marked in those cords with malacic areas in the white matter. Degeneration of the neurones was found in every case, the commonest change being chromatolysis. Both central and peripheral chromatolysis was present, often in the same area of grey matter. Some cells showed a diffuse loss of Nissl's granules throughout the cytoplasm. In many instances the type of degeneration could not be categorized. Many cells were shrunken and distorted and a few showed eosinophilia of the cytoplasm, although they did not resemble the type seen in acute ischaemic necrosis. In all cases the severity of neuronal degeneration was irregular and markedly shrunken cells could be present with those showing minor degrees of chromatolysis (Fig. 8).

The glial response varied markedly but both microglial and astrocytic proliferation occurred. Microgliosis was never as marked as in the white matter and apart from the two cases showing necrosis of the grey matter, lipid phagocytes were very infrequent. Astrocytic proliferation was common and was seen in H and E and Cajal sections. Astrocytic gliosis could lead to quite marked sclerosis of the grey matter.

CHANGES IN NERVE ROOTS AND MENINGES These tended to occur late and progress slowly. There was an extremely variable rate of onset and development. There could be remarkably little change at two months or marked change at three weeks. Degeneration of the fibres was patchy, normal and degenerating fibres being present together in the same root. In general, the ventral root fibres were affected before the dorsal root but this was not constant. Swelling of axons and myelin sheaths was the initial change, followed by an increase in Schwann cells and macrophages which could be demonstrated in

the swollen myelin sheaths. In cords with malacic areas the changes in nerve roots occurred early and progressed rapidly. The affected fibres showed pallor of staining, with marked breakdown and phagocytosis of myelin.

In older lesions fibrosis occurred. This could be seen occasionally as early as one month after onset and the amount of collagen increased with the duration of the lesion. It appeared initially as an increase in the endoneurial connective

tissue. Concurrent with the fibroplasia was a loss of nerve fibres and at a year there was a marked loss of fibres (Fig. 9). There was an increase of collagen in the pia and arachnoid maters and also thickening of the adventitia of many of the arteries and veins supplying the cord.

CHANGES AT SEGMENTS NOT ASSOCIATED WITH THE PROTRUSION Lesions were present in several cords in the dorsal funiculi caudal to the main

FIG. 8. Examples of neuronal changes taken from various cords. Note shrinkage and sclerosis of cells with ? vacuolation; also examples of central and peripheral chromatolysis. H and E, $\times 500$.

FIG. 9. Dorsal nerve root to show loss of nerve fibres and fibrosis. From cord showing compression. One year survival. Van Gieson, $\times 150$.

FIG. 10. L2 segment from cord with protrusion compressing junction of T12/13 segments. Note degeneration in lateral and ventral columns and also centre of dorsal columns. There is a malacic area in the left ventral column. One year survival. Van Gieson, $\times 9$.

lesion. These consisted of small areas of demyelination or malacia in the base of the funiculi which sometimes coalesced to form a single lesion.

In the base of the ventral funiculi, small malacic foci were sometimes found, separated from the main lesion by one or even two non-malacic segments (Fig. 10).

In the lateral, dorsolateral, and ventrolateral funiculi, scattered areas of degenerating fibres were present, with an accompanying microglial reaction. Blood vessels with thickened adventitia

were present in these areas. The swelling of the myelin sheaths was greater than that usually associated with Wallerian type degeneration but these degenerating fibres usually extended only one or two segments cranial and caudal to the main lesion.

Wallerian degeneration was best demonstrated in the spinocerebellar tracts and the dorsal column fibres which eventually form the fasciculus gracilis. With appropriate stains, the degenerating fibres could be traced into the caudal medulla oblongata but no attempt was made to localize their terminations.

Cords and meninges were examined for evidence of haemorrhage, either recent or longstanding, as indicated by the presence of macrophages containing haemosiderin. Extradural haemorrhage was present in eight cases, subarachnoid haemorrhage in one case, and intramedullary haemorrhage in five cases. The intramedullary haemorrhage was always slight and usually present in the grey matter around congested blood vessels.

DISCUSSION

The pathogenesis of this type of myelopathy does not appear to have been investigated in detail by many workers. It has often been assumed to be due to compression of the cord with resultant traumatic damage. While this may be of importance in some cases, it is by no means the only cause. Many cords show no evidence of compression by the protrusion and those with severe compression often have less severe lesions than those with no compression. The site of maximal cord damage is not always opposite the protrusion and many cords show lesser changes several segments from the site of maximal damage. These latter findings suggest a probable vascular factor in the pathogenesis. Wright and Palmer (1970) have suggested the probability of vascular factors in cases of disc protrusions in the dog. This suggestion has been made previously in cases of cervical spondylosis in the human. Mair and Druckman (1953) suggested that reduction of blood supply in the distal distribution of the ventral spinal artery due to compression of that vessel and its branches caused the lesions in their four cases. Taylor (1964) has summarized the role of vascular factors in this disorder and has demonstrated that the radicular arteries become surrounded by thickened hyalinized fibrous tissue in the intervertebral foramen.

Based on the pathological findings presented in this paper, it is suggested that vascular factors play an important role. A large number of cords have malacic areas which show a consistency in both distribution and appearance. The typical areas are in the white matter in the dorsolateral, lateral, and ventral funiculi and in the base of the dorsal columns. The appearance is often wedge shaped and in many cases there is an area of relatively normal white matter between the malacic area and the pia mater. In the older lesions the border is well defined. It is suggested that these are infarcts of the white matter occurring in the area supplied by distal ramifications of the ventral spinal artery, probably in the border zone with vessels penetrating from the pia mater. These infarcts correspond in shape to those described by Wolman and Bradshaw (1967) in their cases of spinal cord embolism. A crescentic infarct found in one case in this series is also described by these authors and attributed by them to ischaemia of the watershed area between the territory of the ventral spinal artery and pial branches. The cavities in the base of the dorsal columns resemble those produced experimentally by Woodard and Freeman (1956) after ligation of the nerve roots and blood vessels from T6 to T11 segments in the dog. It is interesting to note that the grey matter is spared from these infarcts with only the periphery of one horn being involved at the most. The two cases which showed necrosis of the grey matter and surrounding white matter probably represent another form of ischaemic damage. The area of the lesion did not include the dorsal portions of the dorsal columns or the periphery of the lateral and ventral columns and, therefore, corresponds to what is almost certainly the territory of the ventral spinal artery in the dog.

The origin of these infarcts is most probably a reduced blood flow through the intramedullary branches of the ventral spinal artery and possibly those derived from the pial vessels. The cause of the reduced flow is not certain in every case but there are three possibilities:

1. Compression by the disc material of the branches of the ventral spinal artery in the cord tissue: Breig, Turnbull, and Hassler (1966) suggested from a study of human necropsy material, that in spondylosis, during flexion of the neck, there is a decreased blood flow through the vessels in the lateral columns due to the mechanical stresses placed upon them.

2. Compression of the ventral spinal artery itself.

3. Compression of a major medullary artery in the intervertebral foramen before it can supply the ventral spinal artery.

Obviously a combination of all three factors could occur.

Whether a localized peripheral infarct or destruction of the whole territory of the ventral spinal artery is produced probably depends on the degree of reduction in flow, being far more severe in the latter. A diminution in flow will first affect the areas supplied by the terminal branches of the system. The fact that no marked pathological changes have been found in the majority of these vessels suggests that compression of the vessels rather than obstruction by thrombi etc., is the major factor and that the infarcts are usually a result of chronic, rather than acute, ischaemia. The suggestion of a decreased blood flow rather than complete ischaemia is also supported by the differential vulnerability of the tissues, in that the mesenchymal components survived while the neural elements became necrotic.

The rostrocaudal extent of the malacic lesions and the foci of malacia which occurred one or two segments from the primary lesion are probably also governed by similar factors. The ventral spinal artery does not receive a supply from every radicular vessel but is reinforced by a number of medullary arteries at various levels. It is known in man (Schneider and Crosby, 1959; Garland, Greenberg, and Harriman, 1966) that damage to the ventral spinal artery or its contributory branches at one level can cause ischaemia at a different level. These secondary levels are in the intermediate areas between the reinforcing branches of the ventral spinal artery and are, therefore, governed by the position of these reinforcing arteries and the direction of flow in the artery. These anatomical details have not been investigated in the dog but the variation in the rostrocaudal extent of the malacic areas in some of these cases is probably also determined by a decreased longitudinal flow in the ventral spinal artery causing ischaemia in the watershed areas. In the other cases the malacic areas are related to those segments immediately overlying the protrusion.

The histology of these malacic areas is almost identical with those described by Woodard and Freeman (1956) in their experimental cord ischaemia and by Garland *et al.* (1966) in the

human. The only variation appeared to be the lesser degree of astrocytic response in the present cases. Fine neuroglial sclerosis occurred around the border of the infarct but was not marked and progressed only slowly. Any swelling of the cell body was slight and only a few astrocytes were involved.

Some cords showed neither malacic areas nor compressive changes and in these the major damage was in the white matter. This has been described morphologically as a vacuolated appearance with degeneration of both axons and myelin sheaths.

Wright and Palmer (1970) have suggested that this non-malacic lesion, which primarily involves the white matter, is possibly due to venous obstruction. In man, Brain *et al.* (1948) have suggested that impairment of venous drainage was a factor in the pathogenesis of cervical spondylosis.

Barron, Hirano, Araki, and Terry (1959) in a series of extramedullary spinal cord tumours demonstrated that changes occurred mainly in the white matter and that there was no correlation between cord compression and histological damage. They suggested that the myelopathy was due to venous obstruction caused by the tumour. The small irregular malacic areas found occasionally in the vacuolated white matter in this series, are probably also infarcts, and appear similar to those described by Barron *et al.*

Woolf (1954) has shown that in experimental obstruction of the sagittal vein in cats the major changes occurred in the white matter. He further showed that the histological changes consisted of beading and swelling of the myelin sheaths progressing to formation of globules which became phagocytosed by myeloclasts. There was degeneration and loss of axons and a resultant diffuse spongy gliosis. The astrocytes in these areas were 'bloated'. This histological description resembles that seen in these cords with the exception of the 'bloated' astrocytes. The astrocytes in these cords did not tend to show marked hypertrophy.

Lindblad *et al.* (1962) have demonstrated that in disc protrusions in dogs the vertebral venous sinuses may become obstructed due to compression. The present author would agree with this statement on the basis of a small series of spinal venous angiograms performed on clinical cases.

Gillilan (1970), in a study of spinal veins in the human, has suggested an anatomical basis for the localization of the lesions in the white matter of the cord in cases of disc protrusion. This

theory is based on obstruction of venous drainage from the affected areas of the lateral and dorsolateral funiculi.

In the cords with the diffusely demyelinated white matter the pathology differed from both the compressed and malacic cords and it would appear, therefore, that in these cords showing minimal signs or no signs of compression, and with the demyelination of the white matter there is a reasonable possibility that venous obstruction may play a part in the pathogenesis.

In cords where marked compression was evident, it is more difficult to decide on the pathogenesis. As the majority of cases were not operated upon, compression was judged on the appearance at necropsy, either fresh, or after fixation *in situ*. This latter procedure may give erroneous results, as Payne and Spillane (1957) have shown that during fixation the cord adapts to the surface upon which it is lying. If lying on a protrusion the cord will, therefore, show an impression which they consider to be fixation artefact. The present author was satisfied that the majority of cords which showed indentations after fixation *in situ* were, in fact, compressed *in vivo* as in most cases the protruded mass occupied over half the diameter of the vertebral canal. In addition, similar but smaller indentations could be found after the cord was removed for fixation with the dura mater intact.

Besides any compressing effects of the protruded disc, other factors have been incriminated in aggravating the degree of compression. Stoltmann and Blackwood (1964) have suggested that inward bulging of the ligamentum flavum during extension was a major factor in cervical spondylosis. We have seen slight thickening of this ligament in the thoracolumbar area at surgery but it has never appeared to play any part in compression of the cord.

Kahn (1947) has suggested that the denticulate ligaments anchor the cord and prevent dorsal displacement during compression. He suggests that the primary pressure is on the ventral funiculus and a secondary force via the denticulate ligaments is exerted on the dorsolateral funiculus. This theory seems to have fallen into disrepute but in four cords I have noted that the compressed cord appeared to be anchored by the ligaments. The cords showing this feature were all severely flattened. It is hard to determine the exact role that they were playing as, in other cords with similar degrees of compression, the ligaments could be seen to be not anchoring the

cord. It is possible that in certain cases the ligaments accentuate the damage caused by compression.

The vascular changes, consisting of hyperplasia and adventitial fibrosis, are of interest. Hughes and Brownell (1966) describe similar thickenings around capillaries and small arteries in their cases of spinal cord ischaemia. They state, however, that the spinal veins were normal, whereas in this series veins also showed thickening. Garland *et al.* (1966) mention the thickening of intraspinal vessels in cases of venous obstruction. Mair and Druckman (1953) and Wilkinson (1960) both mention the fibrous thickening of blood vessel walls in cervical spondylosis. This fibrous thickening of the vessels is identical to the 'hyalinisation of small vessels' mentioned by Wright and Palmer, who found that the veins and capillaries were mainly affected. The present author would agree with their statement that this change is more common in the white matter and is closely correlated with the occurrence of demyelination. These latter authors suggest that the hyalinization may be a result of an increased vascular permeability to plasmatic constituents. In this present series no evidence of a fibrin 'leak' from the vessels was found. The adventitial fibrosis may be a result of a chronically decreased blood flow as sub-endothelial proliferation of connective tissue has been found in cerebral arteries after ischaemic episodes (Romanul and Abramowicz, 1964) and the changes seen in the much smaller cord vessels may be of a similar nature. The vascular permeability in the acute stage of the myelopathy has yet to be established. Whatever the origin of this vascular thickening, it will decrease diffusion rates across the vessel walls.

It is well known that if spinal cord ischaemia is produced experimentally by occlusion of the aorta, the principal histological changes are found in the grey matter, particularly the intermediate and ventral areas (Tureen, 1936; van Harreveld and Marmont, 1939). This effect may also be seen after aortic surgery in man (Adams and van Geertruyden, 1956) and aortic aneurysm (Thompson, 1956).

Tarlov (1957) has suggested on this and other evidence, that the clinical signs of cord compression are not due to ischaemia but to mechanical deformation. The present author suggests that the two situations cannot be compared, as in cord compression only a small part of the spinal circulation (ventral spinal artery, its

branches or a medullary artery) is being occluded, so decreasing the flow in certain areas of the cord, mainly the white matter. In aortic occlusion there is a sudden decrease in flow to all parts of the cord and, therefore, those areas with the highest blood flow and highest oxygen demand—that is, the grey matter—will be affected first. (In Tarlov's publication there are illustrations of cords with malacic areas in positions corresponding to those described in this publication.) Infarcts in the white matter have been well documented, both clinically (Hughes and Brownell, 1966; Wolman and Bradshaw, 1967) and experimentally (Woodard and Freeman, 1956) after atherosclerosis, arterial embolism, or ligature.

Although this publication has dealt with the role of vascular factors in the pathogenesis of the myelopathy of disc protrusions, it is not suggested that mechanical deformation plays no part. It would appear likely that compression of nerve fibres plays a major role in those cords showing marked evidence of compressive change, although the thickened blood vessels suggest that a chronic reduction in blood flow may follow.

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