Vasogenic edema following acute and chronic spinal cord compression in the dog

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The T-13 spinal cord segment of dogs was compressed both acutely and chronically by means of a balloon catheter. The vascular permeability to protein was assessed using Evans blue albumin (EBA), and the dorsal column evoked potential recorded to monitor conduction failure. With acute compression sufficient to cause conduction failure there was a marked leakage of EBA from the intermediate gray matter, which spread into the dorsolateral white matter. The degree of edema was similar whether the compression was maintained or released. Chronic compression maintained over 4 to 5 hours did not increase vascular permeability, but following release of compression leakage of EBA occurred in the same cord locations observed with leakage from acute compressions. This increased permeability following release of chronic compression may result from reactive hyperemia. Dorsal column conduction returned after the release of both acute and chronic compression. The extravasated EBA was present both in the extracellular space and within cells. The results and their clinical application are discussed.

KEY WORDS: spinal cord injury, vascular permeability, vasogenic edema, conduction failure, reactive hyperemia

Acute compression of the canine spinal cord often causes more severe neurological dysfunction than chronic compression, even when the degree of compression appears similar. This is particularly evident in protrusions of the cervical disc with which Olsson has introduced the term “dynamic factor” in spinal cord compression to account for greater susceptibility to sudden rather than static compression. Tarlov produced experimental compression of the canine cord by inflation of extradural balloons. He demonstrated that with a slowly developing compression the duration of compression compatible with functional recovery was longer than with acute compression. Griffiths has drawn attention to the severe cord damage that occurs with traumatic “explosions” of the cervical intervertebral discs in dogs, even though the degree of actual compression is minimal. However, clinical and experimental data also show that a slowly developing compression of sufficient magnitude can cause severe functional disturbance. The reasons for these discrepancies and the actual mechanisms by which compression
causes cord dysfunction are not fully known. Mechanical deformation, edema, and ischemia have all been suggested as causative factors. This study was designed to evaluate one particular aspect of the pathogenesis, namely, vascular permeability and the spread of protein in acute and gradual compressions of the canine spinal cord.

**Methods**

**Preparation and Instrumentation**

Twenty unselected dogs were used. They were anesthetised with pentobarbitone (25 mg/kg) and maintained on a 70% N₂O/O₂ mixture delivered through a semiclosed system by a Palmer pump.* Relaxation was produced with succinylcholine to allow control of blood gases which were maintained at a PaCO₂ of 38 to 42 mm Hg and a PaO₂ of 110 to 140 mm Hg. A femoral artery was cannulated to allow sampling for blood gas tensions and pH, and the arterial blood pressure was continuously recorded. In all dogs the mean arterial pressure was between 120 and 150 mm Hg. A femoral vein was cannulated to allow injection of the indicators. The pharyngeal temperature was measured with a mercury thermometer and maintained between 37 ° and 38 ° with heating lamps. Any base deficit was corrected with 8.4% sodium bicarbonate. The dog was supported by means of a head holder and steel pins in the pelvis. A laminectomy was performed to expose the T-13 segment with the dura intact. A Fogarty No. 3 or 4 charier gauge embolec-tomy catheter† was passed through the paravertebral muscles in a 14-gauge needle and introduced into the vertebral canal through the T-13 intervertebral foramen. The catheter was then positioned ventral to the cord in the epidural space and passed rostrally until it lay under the portion of the segment not exposed at laminectomy. The bone defect was then replaced by a prosthetic epon lamina held in position by dental cement. In later dogs this procedure was altered, and a small portion of the pedicle was removed with a micrometer trephine.‡ The catheter was then introduced through this opening and cemented in position as before. In the majority of dogs this was achieved without any damage to the intervertebral vein or nerve root.

The evoked dorsal column potential (DCEP) was recorded two segments rostral to the compression site. A silver/silver chloride, ball-point electrode, insulated except at the tip, was introduced into the vertebral canal through a trephine hole. The electrode was held on the dura mater with sufficient pressure to displace the cerebrospinal fluid (CSF), and maintained in position by dental cement. A reference electrode was placed on adjacent bone.

The right sciatic nerve was exposed at the level of the greater trochanter and sectioned. The central portion was placed on silver electrodes in a pool of liquid paraffin maintained at 37 °C by heating lamps. The nerve branch to the caudal thigh muscles was also sectioned. The sciatic nerve was stimulated at supramaximal intensity by stimuli of 0.2 msec duration at a frequency of 1/sec. The DCEP was amplified and displayed on a DISA 2 channel EMG unit (14.A.21); 32 successive sweeps were averaged with a DISA 14G01 digital averager.§ The latency to the onset and peak of the initial negative deflection, duration and amplitude of the DCEP were recorded at 15- or 30-minute intervals as appropriate. Conduction failure was defined as the absence of the negative potential.

The main indicator used to assess vascular permeability to protein was Evans blue albumin (EBA). Its preparation has been described previously.† In the dogs of Groups A, B, and C (see below), the EBA was injected intravenously 10 to 15 minutes before cord compression. In three dogs of Group D, another indicator, RISA, was also given intravenously. The time sequence of the injections in Group D is outlined below. In addition, some dogs of

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*Palmer pump made by C. F. Palmer (London) Ltd., P.O. Box 1, Romford, RM1 1HA, England.
†Fogarty 3 or 4 charier gauge embolectomy catheter made by Down Brothers and Mayer & Phelps Ltd., Church Path, Mitcham, Surrey, England.
‡Micrometer trephine manufactured by California Surgical Design Corporation, Gardena, California 90249.
§DISA 2 channel EMG unit and DISA 14G01 digital averager manufactured by DISA, Techno House, Redcliff House, Bristol, BS1 6NV, England.
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Groups A, B, and C were also injected intravenously with Thioflavine S* immediately before death to demonstrate vascular perfusion. It was not possible to isolate the cord segments within 15 seconds of injection, but cardiac arrest was produced within this time by a large dose of pentobarbitone.

Experiments

Group A. Group A consisted of four dogs with acute maintained compression. The cord was compressed rapidly (30 seconds) by injection of saline into the Fogarty catheter. Injection was continued to an amount just in excess of that required to produce conduction failure. This was approximately 0.3 to 0.4 ml. The compression was then maintained until death. The dogs were destroyed between 15 minutes and 5½ hours after compression.

Group B. Group B consisted of four dogs with acute released compression. Compression was produced in a manner identical to that of Group A. In two dogs the compression was maintained for 5 minutes and then released. In the other two the duration of the compression was about 30 seconds, i.e., long enough to establish that conduction failure was present. The dogs were destroyed at intervals similar to those of Group A.

Group C. Group C consisted of three dogs with chronic maintained compression. A more chronic compression was produced by the injection of saline using a slow infusion pump delivering approximately 0.001 ml/min. Inflation was continued until conduction failure occurred and the balloon was maintained at that volume. One dog was destroyed at 10 minutes and the other two at 1 hour after conduction failure.

Group D. Group D consisted of five dogs with chronic released compression. A chronic compression similar to that used in Group C was produced and continued until conduction failure. The compression was then immediately reduced. In one dog the compression was accidently reduced before total conduction failure had occurred. One protein indicator (either EBA or RISA) was given before the onset of compression and the other injected immediately before the release of compression. The sequence of indicator used varied in different dogs. After release of compression the indicators were allowed to circulate for 15 minutes to 2 hours.

Group E. Group E consisted of three dogs used as controls. In these dogs the catheter was inserted as before but not inflated. The dogs were killed after the EBA had circulated for 1, 4, and 5 hours. One dog was discarded because the balloon ruptured during the experiment.

Histological Preparations

At the completion of the experiment all dogs, except those injected with Thioflavine S, were fixed by perfusion of 10% formal saline into the aorta after flushing with saline. Blocks were taken from the spinal cord at the level of compression and at half a segment rostral and caudal to this level. One portion from each block was frozen in liquid nitrogen and the other further fixed in formal saline. Cryostat sections were cut at 10 μm for demonstration of EBA and at 100 and 200 μm for demonstration of Thioflavine S. These sections were mounted in 50% glycerol and examined with a Leitz Orthoplan microscope using a Ploem vertical illuminator. Blocks embedded in paraffin wax were sectioned at 8 μm and stained with hematoxylin and eosin (H & E), luxol fast blue and cresyl violet and the Martius scarlet blue method for fibrin. Sections cut at 8 μm were dewaxed, coated with Kodak AR10 stripping film and exposed for approximately 8 weeks.

Results

In the majority of dogs the compression was ventrolateral and the resulting edema was ipsilateral. In the few cases where a ventral midline compression occurred the lesion was symmetrical (bilateral).

A sudden increase in blood pressure similar to that described by Alexander and Kerr,†

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*Thioflavine S supplied by E. Gurr Ltd., Michrome Labs, Coronation Road, Cressex Industrial Estate, High Wycombe, Bucks, England.


‡ Kodak AR10 stripping film manufactured by Eastman Kodak, P.O. Box 66, Hemel Hempstead, Herts, HP1 1JU, England.
was seen in dogs of Groups A and B. This decreased over the next few minutes regardless of whether the compression was maintained. In Groups C and D this response was not seen.

**Group A (Acute Maintained Compression)**

**Histology.** Three of the four dogs had perivascular hemorrhages in the gray matter, predominantly in the intermediate and ventral areas. The vessels involved were thin-walled, probably small veins. These vessels were congested in spite of the perfusion fixation that had emptied all other vessels. There was disruption and general loosening of the neuropil in the gray matter. Degenerative changes in the neurons, mainly pallor of staining with basic dyes and loss of the Nissl granules appeared in all dogs. In one dog there was microvacuolation of the cytoplasm in some ventral horn cells. The degree of white matter involvement increased with the duration of survival time. The areas principally involved were the middle of the lateral funiculus and the base of the dorsal columns. The main abnormality in the white matter was a separation of myelinated fibers and disruption of normal architecture. In general these areas coincided with those stained by EBA but in one dog changes in the ventral funiculus were unstained by EBA. No obvious deposits of fibrin appeared around the vessels and no cellular reaction was detectable.

**Electrophysiology.** An abrupt loss of the DCEP occurred within 30 seconds of the onset of compression. Conduction remained blocked until death (Fig. 1).

**Evans Blue Studies.** Macroscopically, extensive leakage and spread of EBA occurred 15 minutes after the onset of compression. The intensity of staining was greatest in the intermediate gray matter and to a lesser degree around the central canal and dorsal horns. In the white matter staining of the
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FIG. 2. Photomicrographs of damaged vessels in the intermediate gray matter showing EBA both within the vessel walls and in the surrounding neuropil. Left: A large fluorescent neuron is present to the right of the vessel. Evans blue, × 400. Right: Damaged vessel shown at lower magnification. Evans blue, × 250.

lateral funiculus occurred adjacent and also ventral to the intermediolateral gray matter. This only involved the more central white matter. In the dorsal columns a thin strip in the basal midline was stained; the ventral and ventrolateral columns were not involved. When more time elapsed before sacrifice, a greater area of white matter was stained, the dye reaching the pia in approximately 5 hours. Some variation in degree of involvement existed but in all cases the dorsal portions of the lateral funiculi were predominantly involved. Staining of the dorsal columns was variable mainly in the basal area. The intensity of staining was less than that found in concussive injuries to the cord. In one dog there was no involvement of the dorsal columns and in no instance was there staining of the ventral and ventrolateral funiculi.

Intense EBA fluorescence was present in vessel walls and in the immediate perivascular area in the intermediate gray matter and to a lesser extent around the central canal (Fig. 2). This localization is probably analogous to the areas of perivascular hemorrhage found in paraffin sections. The neuropil of the gray matter was also fluorescent, as were numerous cells, and the neurons of the ventral horn and intermediate gray matter sometimes showed intense fluorescence in the absence of any obvious involvement of the neuropil. At short durations of compression some of the ventral horn cells were nonfluorescent; the white matter had the typical reticular appearance with the EBA surrounding the fibers (Fig. 3). Small focal deposits of EBA were present in some areas; this may represent protein uptake by astrocytes (Fig. 4). In areas where dye was present in the neuropil some vessel walls contained EBA, but the majority of vessels were normal in the nonedematous tissue.

Only one dog, with a compression of 15 minutes’ duration, was given Thioflavine S. This was present in pial and intramedullary vessels; however, a reduction in the number of perfused vessels in the edematous areas was apparent when compared to nonedematous control segments.
White matter from the lateral funiculus showing the reticular pattern of EBA. A vessel with EBA in the wall is in the center of the field. Evans blue, x 250.

Half a segment rostral or caudal to the point of compression EBA was present in the gray matter in similar locations. The amount and degree of spread into the white matter were less than at the compression site.

**Group B (Acute Released Compression)**

**Histology.** The changes were similar to those in Group A but with usually greater perivascular hemorrhage in the intermediate gray matter. The hemorrhages again surrounded thin-walled vessels. Loosening and disruption of the neuropil was evident in the lateral columns and the base of the dorsal columns, and the ventral horn cells showed loss of Nissl granules and pallor of staining with cresyl violet.

**Electrophysiology.** Abrupt loss of the DCEP occurred as in Group A. However, in all four dogs the potentials reappeared 5 to 15 minutes (mean 9.7 minutes) after the release of compression. There was no essential difference between the 1-minute and 5-minute compressions. In three of four dogs the amplitude was markedly reduced after compression and in all cases the latency was increased (Fig. 1).

**Evans Blue Studies.** Cords compressed for 1 minute and those compressed for 5 minutes were essentially the same. The cords from all four dogs were stained blue at the site of compression; the localization of the dye was similar to those dogs undergoing acute maintained compression (Group A) but the intensity of staining was greater. At 30 minutes after compression EBA was present in the gray matter, particularly in the intermediate and ventral areas; several vessels in these areas showed EBA in their walls and surrounding neuropil. The blue staining involved the white matter adjacent to the intermediolateral gray matter. The dorsal columns were also stained particularly at their base. With the longer compression time, more white matter became involved in the lateral funiculus and to a lesser degree in the dorsal column.
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columns. The ventral and ventrolateral funiculi were not involved except around the ventral white commissure (Fig. 5). The histological distribution of EBA was very similar to that seen in Group A (Figs. 2, 3, and 4). Very few vessels in nonedematous areas of white matter contained EBA in their vessel walls.

The results of Thioflavine S perfusion resembled results with the dog in Group A; perfusion was reduced in the edematous areas of white and gray matter.

Minimal changes were usually present at half a segment rostral or caudal to the point of compression, with EBA in the intermediate gray matter and occasionally in the adjacent white matter. Usually a few neurons contained dye in their nuclei and cytoplasm.

Group C (Chronic Maintained Compression)

Histology. The cord was flattened dorsoventrally. No intrinsic histological abnormalities were observed.

Electrophysiology. Throughout most of the compressive period the latency and amplitude remained unaffected. At 15 to 20 minutes before conduction failure, amplitude fell noticeably and latency increased. Conduction then failed. One observation not studied in detail was that the second negative component of the DCEP failed before the first. Failure of conduction continued until the dogs were destroyed (Fig. 1).

Evans Blue Studies. Evidence of EBA leakage was minimal in these dogs (Fig. 6). A very small amount of dye was occasionally present in a vessel wall in the central gray matter and one or two neurons sometimes contained EBA.

Group D (Chronic Released Compression)

Histology. Abnormalities were minimal. Some ventral horn neurons were pale-staining with loss of Nissl granules; no perivascular hemorrhages were noticeable. There was occasional slight loosening of the myelinated fibers in the lateral columns but this finding was not constant.

Electrophysiology. The manner of conduction failure was similar to that in Group C dogs. In the one dog where compression was maintained for 1 hour before release there was no return of the DCEP. In another dog the balloon was deflated before conduction failure. In the remaining three dogs that were decompressed immediately, potentials returned approximately 15 minutes after release (Fig. 1).

Evans Blue Studies. Four of the five dogs had macroscopic staining of the spinal cord. After 15 minutes of circulation following release of the balloon, EBA was present in the intermediate and ventral gray matter and extended slightly into the adjacent white matter of the lateral column. Some vessels in the unstained white matter between this area and the pia mater contained dye in their walls. As the durations of time after release of the compression increased, the dye spread more toward the pia, but still confined to the lateral columns (Fig. 7). The dorsal and ventral columns were seldom stained and then only very slightly. The histological localization of the EBA was similar to Groups A and B. In the gray matter many neurons were strongly fluorescent, while the white matter showed typical reticular fluorescence. In four dogs at both the level of compression and at half a segment rostral and caudal to this point, the intramedullary portions of the central artery contained EBA within the vessel walls (Fig. 8).

 Autoradiographic Studies. The distribution of the RISA was similar to that of the EBA in the three dogs studied.

Group E (Control)

Blue discoloration of the cord did not appear in any dog in this group, and fluorescent microscopy showed EBA to be absent in both neuropil and vessels.
I. R. Griffiths

Discussion

The permeability changes in Groups A (acute maintained compression) and B (acute released compression) were very similar but slightly more marked in Group B. The results from Group C (slow maintained compression) suggest that slow maintained compression does not cause marked changes in the vascular permeability to protein even when severe enough to cause conduction failure, a situation markedly different from that of Groups A and B. This suggests that the acuteness of compression of the cord is the important factor contributing to the abnormal vascular permeability. Indeed some of the differences in the degree of staining probably result from variations in the acuteness of the compression. Very rapid compression has some features in common with the acute concussive injuries produced by the weight-dropping technique. In both forms of injury perivascular hemorrhages and the initial protein leaks occur in the gray matter. This study did not examine the time course of the permeability changes in detail. Initial leakage appears to occur chiefly in the intermediate and ventral gray matter and to a lesser degree around the central canal. The protein then spreads peripherally toward the pia and reaches it in about 5 to 6 hours. This spread was limited mainly to the lateral column, particularly in Group A, in contrast to the concussive injury experiments in which the ventral and dorsal columns were severely involved. Although not proved conclusively in our experiments, it seems likely that the leakage occurred immediately on compression; this would account for the amount of spread at 15 minutes. Extravasated protein spreads through the neuropil mainly in the extracellular space; however, it does enter neurons and some accumulates in astrocytes by pinocytotic activity. This histological localization in the cord is very similar to that seen in concussive injuries.

In Group D when the EBA was circulated for 15 minutes after decompression, the walls of some vessels outside the edematous area of the lateral column contained dye. This probably represented protein confined to the basement membrane and these vessels are unlikely to be a major source of leakage into the neuropil. In Group D dogs increased permeability followed release of a slowly applied compression. The resulting leakage of EBA was not quite so marked as in Groups A and B but followed a similar pattern in that extravasation occurred in the intermediate gray matter and spread toward the pia. This increased permeability following release of compression occurred even in the one dog where compression was not sufficient to cause conduction failure. The combined use of RISA and EBA in Group D confirms that the increased permeability occurred only after the release of the compression. The areas of spread of albumin shown by both tracers were

FIG. 7. Cord from Group D dog. Chronic compression was released and dye circulated for 30 minutes. EBA is present in the lateral column. The ventral horn of gray matter is outlined (arrows).

FIG. 8. Photomicrograph of a central artery adjacent to the central canal. The vessel wall and surrounding neuropil contains EBA. Evans blue, × 400.
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very similar; however, if extravasation had occurred during compression, the area of the indicator injected initially would be much greater than the one injected prior to decompression. The spread of various tracers injected simultaneously has been proven similar in cerebral edema caused by cold injury.18

Although limited, the results of the Thioflavine S perfusions suggest that the compressions used did not cause complete ischemia of the cord in any of the groups, although obvious reduction of perfused vessels occurred in edematous areas. Even if complete cessation of spinal cord blood flow (SCBF) is assumed to occur during the chronic compression, ischemia per se is unlikely to be the cause of the increased permeability in Group D. Long, et al.,20 have shown that after the aorta was clamped for 6 to 48 hours no EBA leaked from the vessels. Olsson, et al.,24 produced complete ischemia in the distribution of the middle cerebral artery in the rhesus monkey by clipping it at the base of the brain. They found that prolonged ischemia of 40 hours duration did not cause increased permeability to EBA; however, following temporary ischemia of 4 hours, five out of eight animals showed extravasation of EBA 2 hours after release of the clip. The permeability returned to normal within 24 hours.

Reactive hyperemia probably plays an important role in the increased permeability following release of compression. This phenomenon has been observed around infarcts in the brain in experimental13,33 and clinical studies,10 and after hypotension.18 Loss of autoregulation and a variable response to CO2 often occurs in these areas. Palleske, et al.,28 have demonstrated reactive hyperemia in the thoracic and lumbar spinal cord, following release of minor and strong compression. The SCBF rose above its pre-compression level and remained higher for 3 to 4 minutes. Similar changes were sometimes observed in adjacent segments. Reactive hyperemia is possibly implicated in disturbed permeability in the studies of Olsson et al.,24 and the dogs in our Group D.

One feature of the present experiments was the localization of dye in the central artery walls and its immediate branches in the central gray matter. This occurred at the level of compression and both rostral and caudal to this point, and in these latter areas was occasionally the only evidence of disturbed permeability. This restricted penetration of protein is an early change in cerebral trauma29 and chemical injury.31 Previous angiographic studies on cord compression have demonstrated that central arteries are affected at an early stage of ventral cord compression.4,17 Kinking and obstruction occurred in these vessels, while the ventral spinal artery was displaced but occluded only with application of very severe compression. The prevalence of these changes in the central artery may explain the disturbed permeability.

The mechanism of vascular endothelial damage is not clear. In the chronic maintained compressions this form of mechanical deformity is evidently not in itself sufficient to cause extravasation. Where increased permeability followed release of chronic compression the hyperemia was possibly in itself sufficient to cause breakdown of the endothelial barrier to protein. Although Group D had no systemic hypertension, local mechanical effects of a sudden increase in SCBF were possibly sufficient to increase permeability. Certainly evidence shows that rapid hypertension will impair the blood-brain barrier.15,16 Further studies of the ultrastructure of blood vessels are required to ascertain the state of the endothelium in these dogs.

The origin of the extravasated EBA in the intermedial and ventral gray matter in Groups A, B, and D was probably from capillaries and venules. No evidence of arterial damage appeared in paraffin-embedded material, while focal perivascular hemorrhages surrounded thin-walled vessels. The type of damaged vessel in the fluorescent preparations was difficult to identify when located in an area of massive EBA extravasation and hemorrhage. However, no thick-walled vessels were observed; the leaky vessels most closely resembled veins and venules. These have been proven more susceptible in concussive cord injuries7 and are the source of the protein leak in these cases.14 Further work is needed to ascertain the cause of the protein leak in Groups A and B, and to determine whether the damage is purely mechanical or if there is a possibility of a biochemical basis involving vasoactive amines, as suggested in some reports of concussive injuries.26,27
The normal DCEP recordings were very similar to those described by Deecke and Tator in the monkey. This potential is probably due to conduction in fibers of the dorsal column. The dorsal columns were not undercut in the present series and fibers in the dorsolateral funiculus may have contributed to the potential. Gelfan and Tarlov have studied in detail the potentials recorded from the dorsal cord in the caudal lumbar region after dorsal root or peripheral nerve stimulation. They concluded that conduction failure was primarily due to mechanical deformation rather than ischemia. We did not record this more complex potential, but found that following acute compression, the potential could be blocked within 30 seconds, too short a time for ischemia to be the main cause. It is not necessary to adopt an either-or attitude to this problem. Although Thioflavine S studies did not show complete ischemia, the blood flow was almost certainly reduced in the cord. However, the effects of hypotensive ischemia and pressure are additive and both factors probably apply in the abolition of the DCEP in chronic compression; if compression was released, the potentials reappeared. A similar finding was reported with cerebral evoked potentials in spinal cord compression.

The possibility also exists that conduction failure was due to edema, but the rapid loss of conduction in the acute compressions makes this unlikely. No group had marked proteinaceous edema of the dorsal columns, apart from at their base. Gledhill, et al., reported an expanded extracellular space in the dorsal columns 21 hours after compression. A nonproteinaceous edema fluid may be present; this feature is well known in the brain where swelling can occur following ischemia and trauma in the absence of EBA extravasation. Present experiments would not detect this type of edema; further ultrastructural and biochemical examinations are therefore indicated. Changes similar to those reported in peripheral nerves during tourniquet compression form an additional consideration in conduction failure. These changes in cord compression have not been described, but experimental compression has produced a demyelinating lesion.

This study has several clinical applications. When the cord is compressed acutely, rapid leakage of protein occurs mainly in the intermediate gray matter. Therapy is therefore unlikely to prevent increased permeability and edema; however, chronic compression probably will not lead to a marked increase in permeability. Of course, the 4- to 5-hour compression used in this study is rapid compared to compression by an extradural tumor; therefore, the probability of a protein leak would be less in the clinical case. This study concerns fairly localized compression and the situation might be different if the extradural compressing mass encircled the cord. The demonstration of a protein leak after removal of a chronic compression offers at least a partial explanation for cases that become worse or fail to improve after surgical decompression. Experimental decompression entailed less trauma than decompression in many clinical situations; clinically, edema would probably be more likely. Preoperative use of steroids combined with anesthetic techniques may be worth considering to reduce edema in such cases; certainly these factors deserve further laboratory studies.

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References

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