PATHOPHYSIOLOGICAL MECHANISM OF TRAUMATIC CAVITATION OF THE SPINAL CORD

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Summary. The gross anatomy and the histological appearances of the spinal cord were studied in 120 patients who died in the Metropolitan Rehabilitation Centre at Konstancin as a result of a spinal injury. Three varieties of lesion were found:

1. Total disorganisation of the spinal cord at the site of trauma.
2. Total disorganisation of the cord at the site of trauma associated with pipelike necrosis extending up and down from the main injury. This longitudinal cylindrical-pipelike necrosis is described in this paper as 'traumatic haemorrhagic cavitation'.
3. Partial spinal cord necrosis localised to the area of supply of intraspinal arteries.

The possible vascular mechanisms of traumatic spinal cord injury, resembling the changes observed in experimental animals, are discussed.

Key words: Spinal cord trauma; Haemorrhagic cavitation of the spinal cord; Post-traumatic syringomyelia; Autopsy findings.

Introduction

THE SPINAL CORD was examined at autopsy in patients with traumatic tetraplegia or paraplegia to determine the frequency with which haemorrhagic cavitation occurred in the cord and extended above and below the site of vertebral injury.

In man, two patterns of traumatic necrosis may be seen in the spinal cord. One type occurs at the level of the vertebral injury and is usually ill-defined. The other is sharply outlined and extends for some distance from the main lesion to involve multiple segments both above and below the level of complete destruction of the cord. This lesion has been variously called longitudinal polysegmental 'pipelike' necrosis, cystic degeneration, pencil-like central necrosis or post-traumatic syringomyelia. The latter is the better term as it has already been identified with a characteristic clinical entity described as a late effect of incomplete or complete spinal cord injury (Barnett and Jousse, 1976).

It is uncertain whether these two types of injury result from different mechanisms or whether they differ only in degree. A recent suggestion is that the pattern of traumatic necrosis which results in post-traumatic syringomyelia is a result of intraspinal vascular injury (Balentine, 1978), a view which is supported by work on experimental animals and by the findings in the human autopsy material described in this paper.

Material and Methods

The spinal cord was examined grossly by dissection and microscopically by histological sectioning in 120 cases with traumatic tetraplegia or paraplegia. Survival had ranged from 3 hours to 12 years. Particular attention was paid to the relationship of the level of the cord lesion to the vertebral level, and to the extent of the cord lesion. In each case the distribution and condition of the thoracic aorta and vertebral arteries, the bony lesion displacement and rupture of ligaments and the presence of epidural or of subdural haemorrhage were recorded in detail. In 24 cases the cervical vertebral column and the spinal cord were removed *en bloc*, fixed, decalcified and sectioned longitudinally in the sagittal plane. The diameter of the spinal cord could then be compared with that of the spinal canal and the meningeal space. In the remaining 96 cases the entire cord was removed, fixed in formalin and divided into segments. These were then embedded in paraffin wax for sectioning. Histological analysis was carried out on sections stained with haematoxylin and eosin.

Results

Three types of lesion were found (Tables I and II):

1. Total disorganisation of the spinal cord at the site of trauma.
2. Total disorganisation of the cord associated with a central core of necrosis extending up and down for a greater or less distance from the level of total cord disorganisation (Fig. 1).
3. Partial disorganisation and necrosis of the spinal cord which was usually localised either in an arterial distribution territory or in a watershed zone between territories, i.e. the anterior or posterior

TABLE I
Spinal cord autopsy changes observed in 96 cases of complete tetraplegia or paraplegia

Severity of spinal cord injury	Total	Cervical	Dorsal	Lumbar
1. Crush Transection	30	26	3	1
2. Crush Transection with 'Cavitation'	18	16	1	1
total	48	42	4	2
1. Localised necrosis at: PSA	26	19	3	4
PSA + CSA	22	19	2	1
CSA	0	0	0	0
2. Localised necrosis with 'Cavitation'	0	0	0	0
total	48	38	5	5
General	96	80	9	7

PSA = Posterior Spinal Arteries

CSA = Central Spinal Artery

TABLE II
Co-existence of traumatic cavitation with crush transection of
the spinal cord

	Level of trauma	Severity of lesion	Extent of the syrinx		Survival time
1	C7-8	crash	C2-6	D1-2	13 days
2	C6-7	crash	C3-5	C8-D2	30 days
3	C7	crash	C2-6	C8-D1	11 days
4	C6	crash	C2-5	C7-D4	10 days
5	C6	crash	C2-5	C7-D4	20 days
6	C5	crash	—	D1-5	4 days
7	C8	crash	C2-7	D1	12 days
8	C7-D1	crash	M-C6	D2-4	13 days
9	L1-3	crash	C8-D12	L4-S3	8 months
10	C6	crash	C3-5	C7-D4	28 days
11	C7	crash		C8-D4	12 days
12	C5	crash		C6-D3	36 days
13	C6	crash	C2-5	C7-D3	18 days
14	C7	crash	C3-6	C8-D3	16 days
15	C6-7	crash		C8-D6	2 months
16	C6-7	crash		C8-D2	12 days
17	C5	crash	C2-4	C6-8	30 days
18	C7	crash	C4-6	C8-D5	3 days

FIG. 1
Transverse sections of the spinal cord showing 'total disorganisation'.

spinal artery distribution or the boundary zone between them (Fig. 2).
Total transection was noted in 50 per cent of cases and depending on

FIG. 2

Transverse sections of the spinal cord showing 'partial disorganisation'.

the duration of survival the lesions ranged from haemorrhagic to fibrous. The severity of the spinal cord injury bore no relationship to the occurrence of epidural or to subdural haemorrhage. Thrombosis was not observed in either intraspinal or extraspinal arteries or veins. In patients over 40 years of age, 90 per cent showed some degree of atherosclerosis of the thoracic aorta and the vertebral arteries.

One in five of the cord lesions was complicated by an extension of the lesion in the central part of the spinal cord in the form of sharply outlined tapering cylinders, located both above and below the main site of the spinal cord injury. Occasionally, it was seen below the level of the vertebral trauma. In one case of cervical vertebral injury two discrete areas of necrosis were present, one at the level of C5 and the other at the level of D1-D5, the intervening C6-C8 segments being almost normal. This pattern of tapering cylindrical necrosis was seen in 18 out of the 48 cases with severe trauma. In these the continuity of the dura, arachnoid and pia was maintained. Of these 18 patients, 16 had a cervical injury, one cervico-dorsal and one lumbar. The length of the lesion varied (Fig. 3), the longest being seen in the patient with a lumbar lesion who survived for 8 months following injury. In one case with a C7/D1 fracture traumatic cavitation extended to the caudal level of the medulla oblongata. Histological examination only showed the outline of blood vessels in the necrotic areas.

In partial traumatic necrosis the lesions in the cord at the level of trauma were minor; in the majority vertebral damage was not severe and the patients were of a young age group. Quite often the spinal canal was normal, there was no bony displacement and the cord was not significantly compressed. Despite the apparent continuity of the spinal cord in these cases all of the patients had had complete paralysis. The lesion was confined to the site of trauma. In three cases a cyst, a few millimeters in diameter and filled with translucent material was found in the region of the posterior horn. Lesions tended to be located in the grey matter or at the interface

FIG. 3
Level of trauma and vertical extent of necrosis beyond it.

between the white and grey matter, that is the boundary zone between the central spinal and posterior spinal arteries. Selective necrosis was not seen in the distribution of the central spinal artery.

Discussion

The pathomorphology of post-traumatic myelopathy has been thoroughly described (Barnett and Jousse, 1976; Wozniewicz, 1977; a and b), and experimental work is essentially in agreement (Balentine, 1978; Kao and Chang, 1977; Kao *et al.*, 1977).

The fact that two types of necrosis were found suggests that two mechanisms may be operative. Haemorrhagic transection of the spinal cord at the level of fracture is interpreted as crush necrosis and requires no comment. Extension of spinal cord necrosis over consecutive segments above and below the level of injury, described by other authors (Barnett and Jousse, 1976), is more difficult to explain and has been little studied. Post-traumatic syringomyelia generally occurs in patients with partial paralysis who have survived for many years following the injury. Only then do the clinical symptoms of deterioration appear which indicate that segments of the spinal cord above the primary lesion are affected. We have been able to demonstrate that morphological changes analogous to those described in post-traumatic syringomyelia occur very much earlier, in fact in the immediate post-traumatic period.

When the distribution of traumatic lesions of the spinal cord in man is compared with that occurring in the experimental animal the similarity suggests a similar pathogenesis. It must be remembered that traumatic cavitation occurred only in cases where there was transection. Various authors (Barnett and Jousse, 1976; Kao and Chang, 1977) have sought to explain the occurrence of post-traumatic syringomyelia as a result of disturbances in venous pressure.

In the Allen experimental model using a 400 g/cm² force applied to the exposed cord there is a latent period of about 1 hour before the onset of paralysis. During this time, oedema and ischaemic changes are observed which are considered to be a consequence of vaso-constriction. This is followed by a period of vaso-dilatation in which necrosis of nervous tissue and haemorrhage occurs. These changes occur principally in grey matter.

The prevention of necrosis in the early post-traumatic period is a major therapeutic problem, especially in patients without a crush transection of the spinal cord and therefore with a potentially recoverable lesion.

RÉSUMÉ

On a étudié l'anatomie macroscopique et les apparences histologiques du cordon médullaire chez 120 souffrants qui sont morts au Centre Métropolitain de Réhabilitation à Konstancin à la suite d'une blessure spinale. On a trouvé trois variétés de lésion:

1. Désorganisation totale du cordon médullaire aux lieux de traumatisme.
2. Désorganisation totale du cordon aux lieux de traumatisme en conjonction avec une nécrose en tube qui s'étend en haut et en bas de la blessure principale. Cette nécrose longitudinale cylindrique en tube est décrite dans ce papier comme une formation de cavernes traumatique et hémorragique.
3. Nécrose partielle du cordon médullaire localisée dans la région de fourniture d'artères intraspinales.

On discute les mécanismes vasculaires possibles d'une blessure traumatique du cordon médullaire, qui se ressemblent aux changements observés chez les animaux expérimentaux.

ZUSAMMENFASSUNG

Man hat die grobe Anatomie und die histologischen Aussehen des Rückenmarks bei 120 Patienten, die im Hauptstädtischen Rehabilitationszentrum in Konstancin infolge einer Rückgratswunde gestorben sind, studiert. Man hat drei Läsionssorten gefunden:

1. Völlige Zerstörung des Rückenmarks an den Traumatismusstellen.
2. Völlige Zerstörung des Rückenmarks an den Traumatismusstellen im Zusammenhang mit rohrachtiger Nekrose, die sich aufwärts und abwärts von der Hauptwunde ausdehnt. Diese zylindrisch-rohrartige Längennekrose wird in diesem Papier als traumatische Blutungskavernenbildung beschrieben.
3. Teilweise Rückenmarksnarkose, die in der Zuflusszone der intraspinalen Arterien lokalisiert ist.

Man diskutiert die möglichen Gefäßmechanismen der traumatischen Rückenmarksverletzung, die den bei Versuchstieren beobachteten Veränderungen ähnlich sind.

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