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THE DEVELOPMENT OF INTRAMEDULLARY CAVITATION FOLLOWING SPINAL CORD INJURY: AN EXPERIMENTAL PATHOLOGICAL STUDY

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Abstract. It is concluded from this investigation that localised intramedullary cavitation will develop following non-disruptive spinal cord trauma if the magnitude of original trauma and resulting vascular damage is sufficient. Although an adhesive arachnoiditis also occurs with similar amounts of trauma, the initial vascular damage and subsequent reparative changes within the spinal cord appear to adequately explain the cavitation observed.

Key words: Paraplegia; Spinal cord injuries; Spinal cord; Arachnoiditis.

Introduction

AMONG the observations made in post-mortem material from patients with chronic traumatic spinal cord injuries has been intramedullary cavitation (Wolman, 1965). This finding has been attributed to earlier vascular damage, but the sequence of pathological events which result in its occurrence remains indefinite where post-mortem studies are concerned.

The present light microscopic study was undertaken to delineate the pathological changes occurring in the spinal cords of experimental animals from 4 hours to 4 months after graded trauma. Thereby an attempt to more fully explain the development of intramedullary cavitation noted in post-mortem material is made.

Materials and Methods

Forty-one cats varying in weight from 2.8 to 4.8 kg were used in this study. Each animal was anaesthetised initially with intraperitoneal sodium pentobarbital. Following intubation and the intravenous administration of Flaxedil respirations were controlled to maintain the end tidal $p\text{CO}_2$ between 2 and 4 per cent. The systemic blood pressure was monitored by a femoral arterial catheter connected to a pressure transducer and polygraph. The rectal temperature was maintained between 35.2°C and 39.7°C with a heating pad.

In each animal a two-level laminectomy was performed at T5-T6. Each animal was then subjected to transdural trauma of 100, 300, 500, or 700 g cm. The pre-drop potential energy was varied by changing the height from which the fixed weight (20 g) fell before striking a teflon impounder with a flat circular surface measuring 5 mm in diameter resting on the intact dura mater.

The animals were then maintained for either 4 hours, 4 weeks, or 4 months after which intracardiac perfusion with 2 per cent glutaraldehyde—3 per cent paraformaldehyde 0.2 M cacodylate buffered solution was carried out. The injured portion of the spinal cord was then excised and post-fixed by immersion in 10 per cent formalin. Paraffin-embedded specimens were then serially sectioned transversely and prepared with haematoxylin and eosin, Bodian, Nissl, and Kluver-Barrera stains.

Results

Four Hours Post-injury

The most striking feature of the sections from animals 4 hours after injury was the haemorrhage in the central grey matter. The haemorrhages observed varied directly with the amount of the original trauma. In those animals with trauma of 100 and 300 g cm, small, petechial haemorrhages were noted in the area of the central canal while in those animals with greater trauma of 500 and 700 g cm the haemorrhages were more extensive and widespread in the grey matter (Fig. 1).

Another area where haemorrhage was commonly observed was in the sub-arachnoid space especially surrounding the anterior sulcal vein.

The ependymal lining of the central canal was commonly discontinuous in sections from animals in whom trauma had been greater. A communication between the lumen of the central cavity and the grey matter was thereby established. Associated with this, a homogenous eosinophilic fluid was frequently observed in the surrounding interstitium of the grey matter.

Four Weeks Post-injury

At this interval after injury animals with trauma of either 100 or 300 g cm demonstrated an increase in cellularity in the grey matter. This appearance was due to an increase in the number of glial cells.

No aggregates of microglial cells were identified in either the grey or white matter in these same sections.

FIG. 1

Cross-section of spinal cord demonstrating grey matter and subarachnoid haemorrhage 4 hours after trauma. H & E, $\times 10$.

In sections from animals in which the magnitude of trauma had been greater, either 500 or 700 g cm, compound granular cells with refractile cytoplasm were seen. These cells were most readily observed in areas of cystic necrosis within the grey matter. They were absent from the white matter where an increase in astrocytes was noted (Fig. 2).

When cystic degeneration was observed it was noted to take a variety of forms which appeared to correspond to the extent of the original trauma. Small, non-communicating cysts were seen in the area of the intact central canal. In other sections the cavitation was more extensive with glial septal extending into it. Communication often existed with the central canal and less frequently with the subarachnoid space.

The lining of the cavities were most often composed of loose glial tissue. Where a communication with the leptomeninges existed that portion of the cavity was lined by fibroblasts and were connected to the central canal by the remaining ependyma.

Four Months Post-injury

Whereas haemorrhage in the grey matter was characteristic of the appearance of sections 4 hours after trauma, cavitation of the central portion of the cord was commonly observed at 4 months.

In addition, marked alterations in the dura mater and the leptomeninges were apparent in those sections from animals in whom trauma had been either 500 or 700 g cm. These changes consisted of a thickening of the dura mater by dense fibrous tissue and an increase in the cellularity of the leptomeninges. Often the cellular leptomeninges were adherent to the underlying spinal cord with the cells of these membranes merging with the substance of the cord (Fig. 3).

Discussion

The haemorrhages noted in the grey matter of the spinal cord 4 hours after injury in the present study have become a common observation in other investigations in which a similar model has been employed (Ducker *et al.*, 1971). Whether the vascular damage represented by the haemorrhages is primary to the parenchymal changes which occur or is a concomitant phenomenon has been uncertain (Wolman, 1964). The similar appearance of the spinal cord to what has been noted in cases of non-traumatic infarction elsewhere in the central nervous system and the lack of parenchymal changes except in proximity to areas of haemorrhage would seem to indicate the overall importance of the vascular damage.

As has been demonstrated by Hughes (1966) in post-mortem material, the occurrence of a reparative process was evident by 4 weeks after trauma. As with the other findings at 4 hours and at 4 months, the changes at this interval corresponded with the magnitude of the initial trauma. In the more severely traumatised animals compound granular cells were readily identified especially in areas of cystic necrosis.

The presence of cavitation at 4 months in similar areas to where compound granular cells had been noted at 4 weeks and in sections from animals in whom the magnitude of trauma had been the same indicate that these cavities resulted largely from the resorption of necrotic material. Such a process of removal followed by the replacement of the necrotic parenchyma by cystic spaces has been well described following infarction in the cerebrum.

FIG. 2

Compound granular cells in area of cavitation with gliosis in surrounding parenchyma 4 weeks after trauma. H & E, $\times 100$.

FIG. 3

Thickened leptomeninges adherent to cavitated spinal cord 4 months after trauma. Kluver-Barrera, $\times 100$.

The frequent marked reaction at 4 months in the dura mater and the leptomeninges in sections in which cavitation was observed corresponds to changes seen in arachnoiditis due to a variety of causes. The same reaction has been noted in spinal cord injured patients (Wolman, 1965) and has been produced experimentally by the injection of blood into the cerebrospinal fluid (Bagley, 1928). It is likely therefore that this response in the present study is due to the haemorrhage in the subarachnoid space noted 4 hours after injury.

Although adhesive arachnoiditis has been described as a cause of intramedullary cavitation through the mechanism of medullary ischemia resulting from the constriction of pial vessels (McLaurin *et al.*, 1954), the haemorrhagic infarction and subsequent reparative process would appear to be sufficient to explain the cavitation noted in the present study.

SUMMARY

The microscopic changes in the feline spinal cord were investigated from 4 hours to 4 months following a traumatic injury and were related to the intramedullary cavitation observed in late post-mortem material. Characteristic features were identified at each interval studied. These changes consisted of an initial haemorrhagic infarction followed by the removal of necrotic parenchyma and the development of an adhesive arachnoiditis and intramedullary cavitation in the more severely injured animals. It is concluded that the alterations observed correspond to the magnitude of the original trauma and that these changes are related to the degree of vascular damage sustained.

RÉSUMÉ

Les variations microscopiques dans la moëlle épinière feline furent examinées pendant la période de quatre heures jusqu'à quatre mois suivant une lésion traumatique; ces changements avaient rapport à la cavitation intramédullaire chez des organes observés dans des délais considérables après le décès. À chaque intervalle étudié, les traits caractéristiques furent signalés. Ces changements consistaient en un infarctus hémorragique initial suivi de l'éloignement du parenchyme nécrotique et, chez des animaux encore plus sévèrement blessés, en le développement d'une arachnoïdite adhésive et d'une cavitation intramédullaire. En guise de conclusion, on constate que les altérations observées sont conformes à la sévérité de la blessure originelle, et que ces changements ont un rapport direct à l'étendue de l'avarie vasculaire subie.

ZUSAMMENFASSUNG

Die mikroskopischen Veränderungen in Rückenmark der Katze wurden während eines Zeitabstandes von vier Stunden bezw vier Monaten untersucht in Relation zu medullären Höhlenbildungen, die später im post-mortem Material gefunden wurden. Charakteristische Merkmale für jedes Zwischenstadium wurden identifiziert. Diese Veränderungen bestanden in einem initialen Infarktbildung folgend bei Entfernung von nekrotischem Parenchym und Entwicklung von adhesiver Arachnoiditis und intramedullärer Höhlenbildung in den schwerer verletzten Tiere. Man kommt zum Schluss, dass die beobachteten Veränderungen dem Grade der anfänglichen Verletzungen entsprechen und dass sie in Beziehung zum Grad der vaskulären Schädigung stehen.

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General Discussion

DR C. H. TATOR (*Canada*). I'd like to ask Dr Wagner if he observed any ascending or descending cavitation. The experimental production of post-traumatic syringomyelia is really possible with some experimental model of spinal cord injury such as the extradural circumferential cuff technique but I haven't seen it described with the weight dropping model that Dr Wagner uses and I wonder if he had an opportunity to examine the cervical cord or the lumbar cord in any of his animals to see whether the cavities were propagated up or down the cord.

DR WAGNER. No, we didn't have an opportunity to examine the cervical or the lumbar cord. The reason these areas were not examined was we felt that the relative contributions to the radicular arteries to the intrinsic vasculature of the spinal cord at these particular levels might in some way affect our sections. Specimens were examined for a distance of approximately a centimetre cephalad and caudal to the area of maximum injury and as has been noted by others the cavities extended in a cone shape in both directions.

DR TATOR. Thank you. May I ask too if there was a fair bit of non-haemorrhagic necrotic material in the traumatic sections above and below or at the face where there wasn't very much haemorrhage but there was obviously necrosis of the tissues.

DR WAGNER. There was necrosis of tissue and it seemed to vary again with the amount of trauma in those animals in which trauma had either been 100 or 300 g. cm. The changes were characteristic of an astrocytic reaction or what one may expect to see with an ischaemic infarction as opposed to an haemorrhagic infarction which seemed to predominate in those sections from animals in which the trauma had been greater.

MR P. HARRIS (*G.B.*). Could I ask Dr Wagner about this whole problem. Does he feel that there is a true post-traumatic haematomyelia? Does this condition post-traumatic haematomyelia exist, or are these patients with haemorrhagic infarctions?

DR WAGNER. I think that, as Dr Barnett and as Dr Rossier have emphasised in their monograph and in their various papers, one has to be careful to separate the two syndromes. Had the spinal cord in the lumbar and the cervical regions been examined four months after trauma a syringomyelia may have been found. I think post-traumatic haematomyelia is very common and that the destruction of ganglion cells and axon cylinders shortly after a non-disruptive injury such as was used in this study is related to the vascular damage represented by the haemorrhage which is of significance.