CERVICAL SPINAL CORD INJURY WITHOUT BONY INJURY

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Abstract. Cervical spinal cord injury without demonstrable bony injury occurred in 141 (37 per cent) of 384 successive neck injuries admitted to the Sheffield Spinal Injuries Unit. The condition was common in patients in the older age-groups and there were 70 per cent of the cases who were over the age of 50 years. The commonest cause was a fall. The patients presented with varying degrees of motor paralysis with the greatest deficits in the upper limbs and lesser deficits in the lower limbs. There was a dissociation of impairment of the sensory modalities and a loss of bladder and bowel control.

Key words: Cervical spinal cord injury; Central haemorrhagic necrosis; Tetraplegia; No bony injury.

Introduction

TWENTY-TWO years ago a 63-year-old miner fell down in the street whilst under the influence of alcohol. He was admitted to hospital conscious but incoherent and very difficult to examine. He claimed that there was something the matter with his arms and legs. Clinical examination revealed a laceration on the back of the scalp, a weakness of the upper arms and a total paralysis of all hand and finger movement and a total paralysis of both legs. He claimed some vague sensation to pressure on the legs and buttocks but detailed neurological examination was quite impossible at that stage. Radiographs of the cervical spine showing all 7 cervical vertebrae did not demonstrate any evidence of bony injury or displacement. In the next 3 days there was considerable respiratory difficulty and death occurred on the 4th day.

A post-mortem examination was carried out by the neuropathologist (the late Dr Lionel Wolman) and myself. There was no evidence of muscle bruising or rupture of the long or short interspinous, interarticular or interbody ligaments and no evidence of disc protrusion or any intervertebral displacement. There were some degenerative changes at the intervertebral margins but these were not gross.

The spinal cord showed some swelling and discoloration in the region of C4/5 (Fig. 1). Microscopically the maximal damage to the cord was at the C5/6 level where there was extensive necrosis of the ventral part of the dorsal columns and the central grey matter with extensions into both anterior horns (Fig. 2). The anterior horn cells had almost disappeared and there was severe demyelination of the ventral half of the posterior columns and in the lateral columns. The softened areas had gone on to form cavitation with masses of necrotic tissue.

Dr Lionel Wolman concluded his Coroner's report with the words, 'an unusual feature in this case is the very extensive spinal cord pathology in the absence of any bony or ligamentous damage'. I also shared that opinion and little did I think that that type of spinal cord injury was soon to become the most common type of cervical spinal cord injury subsequently due to be admitted to the Sheffield Spinal Injuries Unit. However, the collection of large numbers of cases of spinal cord injury in special centres has provided the opportunity to correlate a vast amount of information on this and other aspects. Statistics about incidence and aetiology are readily available and for the purpose of this presentation I have reviewed the notes on my personal series of 384 cases of cervical spinal cord injury

FIG. 1

Dorsal aspect of cervical spinal cord showing swelling of its lower segments.

FIG. 2

Transverse section at C6 showing haemorrhages predominantly in the grey matter.

which have been taken from the records of over 1000 cases of spinal cord injury who had immediate or early admission to the Sheffield Unit in the past years.

Types and Causes of Injury

An analysis of the types of bony injury (Table I) has shown the usual collection of fracture dislocations, dislocations and subluxations and comminutions arising

TABLE I
Type of bony injury

Fracture dislocations	101	(26 %)
Dislocations	42	(11 %)
Comminutions	100	(26 %)
No bony injury	141	(37 %)
Total	384	(100 %)

from a variety of accidents. Table I also shows those cases in which no bony injury or displacement could be identified. There were 141 cases in this latter group and a large proportion of them have occurred in the past 10 years. The causes of the accidents in this group of 141 cases could be identified under three main headings which are shown in Table II. Half the accidents occurred in the home and could be considered under the general heading of *Falls*. These included falls downstairs as well as simple falls from standing or walking positions. Many patients were found hours or days after the incident and were believed to have suffered from strokes or heart attacks. Many were unaware of how they came to fall but some gave a history of getting out of bed in the night and falling down on their way to the toilet. The history when available provided an important clue to the nature of the injury but certain predisposing factors were also apparent.

Predisposing Factors

The ages range from 14 to 80 years and the breakdown into 10-year age-groups is shown in Table III. The significant feature is that 70 per cent of the cases were over 50 years of age.

Spondylotic changes were radiologically identified in 107 of the 141 cases.

Congenital malformations occurred in four cases and two examples are shown in Figure 3.

TABLE II
Causes of injury for the no bony injury group (141 cases)

Road traffic	43
Falls	79
Falling objects	19

TABLE III
10-year age-groups

Years:	10	20	30	40	50	60	70	80
Cases:	3	8	10	26	46	37	20	1

FIG. 3, a & b

Lateral radiographs showing congenital malformations in cases of traumatic quadriplegia following minor injury.

The suspicion or actual evidence of alcoholic intoxication has sometimes provided problems. In some instances it has not been possible to identify the symptoms and signs of neurological disturbance in a confused and semi-conscious patient. In other instances the accident and inability to move have been attributed to the inebriated state. Six patients were actually taken to hospital and then sent home to sleep off the effects of their alcoholic excess. They were re-admitted hours or days later when it was obvious that they had a paralysis of the arms and legs and loss of bladder and bowel control.

Diagnosis

In a Conscious Patient

In these circumstances the history, clinical and radiological examinations will usually suffice to establish the correct diagnosis. The classical neurological features of central cord disturbance can be readily identified.

In an Unconscious Patient

In these circumstances evaluation can be very difficult and it is essential to have an awareness of the possibility of spinal cord injury in patients found in certain circumstances. The elderly or the inebriated with a history of fall are the classic examples but the combination of head and neck injuries in road traffic accidents also demands special consideration. The paradoxical movement of the chest and abdominal walls is a valuable sign of tetraplegia in an unconscious patient.

Radiology

The radiographs are only static recordings of a dynamic situation. They are the appearances of the cervical spine in the resting phase after injury and do not necessarily indicate the situation at the time of injury. Standard AP and lateral films may show no abnormality but carefully controlled flexion and extension views in the lateral position will identify or exclude the hidden subluxations or retroflexion injuries.

The effects of the so-called whiplash injury can rarely be identified by radiography.

Neuropathology of the Injury

In the normal subject the central grey matter is composed of loose glial tissue which is highly vascular (Fig. 4). The surrounding white matter is firm and so is the pia mater. The arrangement of the long tracts is such that the nerve fibres serving the most distal segments of the body are mainly located in the periphery of the spinal cord. This is one of the reasons why sacral sensory sparing is so common a feature in cervical spinal cord injuries with limited central necrosis.

The spinothalamic tracts are relatively close to the soft grey matter of the anterior horns and involvement of these and a lesser involvement of the posterior columns provide the reason for the dissociated anaesthesia found in these cases. These features are shown diagrammatically in the diagram (Fig. 4).

Eleven patients who died provided the material for the detailed histological examination of sections of the injured spinal cord. These all showed features which could be directly related to the neurological symptoms and signs. The features included an area of central necrosis and an asymmetry of overall neuropathological lesion with loss of nerve cells and demyelination of both local and long tract fibres (Fig. 5).

Clinical Features

The degree of paralysis was complete and remained complete in five cases but there was a substantial degree of recovery of power of movement in 121 cases. The paralysis was always most marked in the hands and less so in the legs. One side was almost invariably worse than the other. Sensory disturbance was remarkably constant with the preservation of touch, posture and deep pressure and the severe loss of the feeling of pain and temperature. The preservation of the sensory modalities of the sacral segments was also a common feature. Reflex activity was variable and in the initial instance almost always diminished in the arms and present or increased in the legs. Bladder and bowel actions were disturbed and retention of urine occurred for at least a few days but sometimes lasting for weeks and months.

Early Mortality

There were 20 deaths in the first year of injury and 13 of these occurred in the first week. The early mortality is shown in Table IV.

Treatment

No special treatment was provided for the neck apart from the comfort of a supporting collar or neck pillow. None of the cases had exploratory surgery. The

FIG. 4

(a) Diagram of section of cervical spinal cord showing anatomical and functional arrangement of long tracts. (b) Diagram of section of spinal cord showing central cord destruction and spread to adjacent long tracts (Hardy, A. G. & Rossier, A. B., 1975).

FIG. 5

(a) Transverse section of cervical spinal cord showing haemorrhages, central necrosis and demyelination of nerve fibres. (b) Transverse section of cervical spinal cord showing a small asymmetrical lesion with haemorrhage and necrosis with cavitation.

TABLE IV

Early mortality (141 cases)

1 week	13	6 months	3
4 weeks	2	1 year	2

TABLE V

Final mobility (126 cases)

Full recovery	7
Poor hands. Good legs	94
Bad hands. Poor legs	20
Complete paralysis	5

overall management of the paralytic state was considered to be the major factor in the treatment regime. This commenced with the usual bed care followed by the progressive programme of rehabilitation provided by a spinal cord injury service.

Final Mobility

One hundred and twenty-six cases survived for 6 months or longer and a general indication of their final mobility is shown in Table V. The seven cases who were regarded as having achieved a full recovery did so rapidly in a matter of weeks but almost all the others required long and intensive periods of rehabilitation.

It is essential to know that this condition exists and to recognise it when it occurs. The response to a conservative regime provides encouragement to both patients and staff alike.

SUMMARY

Cervical spinal cord injury without demonstrable bony injury occurred in 141 (37 per cent) of 384 successive neck injuries admitted to the Sheffield Spinal Injuries Unit. The condition was common in patients in the older age-groups and there were 70 per cent of the cases who were over the age of 50 years. The commonest cause was a fall. The patients presented with varying degrees of motor paralysis with the greatest deficits in the upper limbs and lesser deficits in the lower limbs. There was a dissociation of impairment of the sensory modalities and a loss of bladder and bowel control. An example of neuropathology revealed a condition of haemorrhagic necrosis involving the central grey matter with variable extensions into the adjacent white matter, particularly in relation to the anterior horns. Vertebral displacements were excluded by radiographs taken in the neutral flexed and retroflexed positions. No special treatment was required for the neck apart from considerations for the comfort of the patient. The overall management of the paralytic state was considered to be of paramount importance.

RÉSUMÉ

Sur 384 cas consécutifs de traumatisme cervical admis au centre de Paraplégiques de Sheffield, 141 (37%) avaient une atteinte de la moelle cervicale sans lésion osseuse visible, et ce particulièrement dans les groupes de patients âgés dont 70% avaient plus de 50 ans. Le facteur étiologique traumatique le plus fréquemment impliqué était une chute. Les patients présentaient divers degrés de paralysie avec déficit prédominant aux membres supérieurs et, à un moindre degré, aux membres inférieurs. Il existait une dissociation entre l'atteinte des déficits sensitifs et la perte du contrôle vésical et intestinal. L'examen neuropathologique d'un des cas révéla une nécrose hémorragique de la substance grise centrale, s'étendant en plusieurs endroits jusque dans la substance blanche avoisinante, particulièrement au niveau des cornes antérieures. La prise de radiographies en position neutre, en flexion et en rétroflexion permit d'exclure un déplacement vertébral. Hormis la prise en considération du confort du patient, il n'y eut aucun traitement particulier du traumatisme cervical. Il a été reconnu que priorité devait être donnée au traitement de l'état paralytique considéré dans son ensemble.

ZUSAMMENFASSUNG

Verletzung des Halsrückenmarks ohne nachweisbare Kochenschäden kam in 141 (37%) von 384 aufeinander folgenden Traumata der Halsgegend vor, die in unser Krankenhaus eingeliefert wurden. Ein solcher Befund war besonders häufig in älteren Patienten und 70% waren älter als 50 Jahre. Ein Sturz war die häufigste Ursache. Es zeigten sich verschiedene Grade motorischer Paralyse mit größerem Defizit in den oberen als den unteren Extremitäten. Dissoziationen des Defizits in den sensorischen Modalitäten und Störungen der Blasen- und Darmfunktion wurde beobachtet. Eine neuropathologische Untersuchung zeigte hämorrhagische Nekrose in der grauen Substanz mit variabler Ausbreitung in die weiße Substanz hinein, besonders in der Nähe der Vorderhörner. Röntgenuntersuchung in neutraler, gebeugter und überstreckter Halsstellung zeigte keine Wirbelluxationen. Abgesehen davon daß man sich um den Komfort des Patienten kümmerte, wurde keine Lokalbehandlung verschrieben. Die Paralyse in all ihren Aspekten wird als überragend wichtig angesehen.

REFERENCE

HARDY, A. G. & ROSSIER, A. B. (1975). *Spinal Cord Injuries*. Georg Thieme, Stuttgart, p. 30.

Verbal Discussion

DR F. W. MEINECKE (*Germany*). I am rather surprised about the 37 per cent without bony injury, I think that's a very high rate and my question is, did you see in later stage probably some kind of instability or some kind of reaction to ligamentous lesions or anything like this, where you came to the conclusion that there had been probably some minor injury which had not been seen in the first X-rays.

DR HARDY. I left out the page on diagnosis and radiology in order to keep within the green light, but we did of course think about subluxations, we did think about hidden hyperextension injury mechanisms and although we did not do it on the cases of the late 1950s we have taken special lateral radiographs with halter traction in control flexion and extension positions to see whether or not we had displacements which could be demonstrated initially. Of course I accept that some of them are disguised and hidden by muscle spasms, some of these were painful, but we did not find and have not so far found late subluxations or late instability in this particular group, and that's why I've put it down as no demonstrable bony injury and I am aware that it may be hidden initially and found later but these were deliberately put like this and the answer is no.

DR G. BEDBROOK (*Australia*). I think you've probably answered my question, but the question I wanted to ask is whether you have found tomography and some of the more sophisticated methods of radiology of use. I also noticed that the percentage you give of 37 per cent is a little but not much greater than the figure which we have come to for the hyperextension injuries causing incomplete tetraparesis. The other interesting feature was that most of the cases in our series that were incomplete from hyperextension injuries

are exactly comparable to the clinical features which you've described. My feeling is that a lot of these cases probably were hyperextension; the pathology is extremely difficult to demonstrate but in our post-mortem series we have been able to demonstrate this quite a number of times, and even the fact that the anterior longitudinal ligament is not necessarily ruptured you'll find that the ligament flavum at the back is the factor which will cause the pathology. I wonder if you've made these observations.

DR HARDY. The answer is yes, in part. In this series in which most of them have survived we've only done 11 more autopsies in the early stages, remember there were 13, and again we did not find any evidence of bony injury or ligamentous damage, but I think that the mechanism is one of hyperextension and I think that it simply shows that violence can be transmitted by an excessive movement without necessarily having caused rupture or ligamentous damage. And the other thing is that this group is the older age-group that have rigid necks; they are suspect when they have these injuries.

DR R. W. HUSSEY (*U.S.A.*). Two questions. Considering the fact that they have no demonstrable bony injuries how long and how are they immobilised and secondly in the incomplete patient how many have hyperactive spastic reflexes in the lower extremities at the time of initial presentation?

DR HARDY. Yes, I missed out one answer to George Bedbrook. We are doing zonal tomography now—as we get more sophisticated so we apply it, but I couldn't say we'd done it on them all—but the answer is, yes we are. The answer is yes to the second part, hyper-reflex activity was common but I can't give you an exact figure.

DR HUSSEY. That was at the time of initial evaluation?

DR HARDY. No, that's something I would look up. I did say that it was variable but I can't give you a figure, but there were some who had hyperactive spastic reflexes at a very early stage. Now what was the first question?

DR HUSSEY. How long and how do you immobilise the patients?

DR HARDY. Here again it is very difficult to generalise but 3 weeks was the period of bed rest and one really was more concerned with the general state of the patient, his blood pressure and so on, and more of the soft tissue effects of spinal cord injuries. Three weeks.

DR G. WHARTON (*U.S.A.*). The question that I have first of all is that in your epidemiological review you did not separate the male from the female patients. Did you in fact have enough female patients to draw any conclusion about the incidence of this injury and the magnitude of its severity in females compared with males.

DR HARDY. The answer is yes. There was a rising incidence of female patients in England in my part of the world.

DR WHARTON. The second part of the question was in regard to your admonition to stay the surgical hand in intervening in these particular cases. While I don't disagree with that, specifically I would raise the question do you have statistics from your series to prove that in essence surgical intervention was harmful or was not helpful.

DR HARDY. That's a very good question. What I ask myself is how many would I have made worse if I had wanted to do, shall I say, explorations or procedures which might be designed to relieve pathophysiological mechanisms which I couldn't relieve by any other means. All I'm saying is that in this series we had enough successes, shall we say improvements, to feel that a conservative regime was justified. No, I cannot do it statistically.

DR J. YEO (*Australia*). Could I just ask Dr Hardy, following on that, is this a vascular lesion and if that is true as I suspect it is, then do you agree that oxygen in good quantities obviously at atmospheric pressure might be helpful. We've noted clinically that in this group of patients if you give them intra-nasal oxygen for 2 or 3 days and then stop it there is often a dropping off of the clinical recovery that you might have seen earlier and perhaps you might like to comment on the earlier treatment of this particular lesion.

DR HARDY. I'm sure it is a haemorrhagic necrosis and various permutations of vascular abnormality. I'm afraid I don't know enough about it to comment on the question of hyperbaric oxygen. The answer is, I don't know.

CHAIRMAN. Thank you Dr Hardy.