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SPINAL CORD INJURIES. A CLINICAL, PATHOLOGICAL, AND EXPERIMENTAL STUDY.

VOLUME 1

by

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Thesis presented for the Degree of Master of Surgery in the University of Glasgow.

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INTRODUCTION.

When a junior member of the staff of the Professorial Surgical Unit in the Royal Infirmary, Glasgow in 1957, I was able to observe two cases of traumatic quadriplegia. Neither patient was completely paralysed. One was discharged after a little more than a year so incapacitated by spasms and pains that he remained almost completely bed-ridden thereafter. The other died after two years in the ward with severe pressure sores, urinary infection and advanced paraplegia-in-flexion. Both patients had been nursed conscientiously and competently, both had received physiotherapy, and neurosurgical, orthopaedic and urological advice had been sought and followed. I could not reconcile the outcome of these cases with references in publications and text-books to revolutionary changes which had taken place in the treatment of spinal cord injuries and decided to investigate

the matter. My original intention was to find out how cases of spinal cord injury fared in other hospital units in Glasgow. In the course of my investigations I became fascinated by the problems of the condition and the extent to which they transgressed nearly all the specialised compartments into which hospital practice is divided.

The first part of this thesis is a clinical review of fifty-two cases of spinal cord injury treated in the Glasgow area with several observations, some of which are original.

In my reading I found out that descriptions of the pathology of the injured cord tended to be derivative. In the course of a year spent in the Mayo Clinic, U.S.A. I studied microscopic specimens of over one hundred injured spinal cords, my attention being focused particularly on the appearances of axons. The second part of the thesis presents material from a small number of these, each one selected to show typical appearances of axons at different times after injury. In addition, histological material from two of the cases in the clinical review who came to post-mortem is presented.

The third and last part of the theses is the presentation of an experimental study upon the spinal cord made in the course of my stay at the Mayo Clinic.

PART ONE

A CLINICAL REVIEW OF 52 CASES OF CLOSED SPINAL CORD INJURY.

1. INTRODUCTION - APPLIED PHYSIOLOGY OF THE SPINAL CORD

a) Spinal Shock and Reflex Behaviour

When the spinal cord is transected, there is a total suppression of function in those segments of the cord below the level of transection. This state is known as spinal shock, a name originally coined by Marshall Hall in 1843. In lower animals the effect of spinal shock passes away quickly, the reflexes in the affected limbs reappear and become increased, and the paralysis which was flaccid during the shocked period becomes spastic and of the upper motor neurone type. Sherrington (1906) concluded that spinal shock was produced by the rupture of certain caudally running paths and the state of exaggerated reflex activity due to release from higher centres normally exerting an inhibitory effect on the spinal cord.

The 'release' theory forms the foundation of modern teaching (Liddell 1934, Fulton 1943, Magoun 1950). "Understanding of the significance of over-all spinal integrative patterns in governing posture has advanced very little since Sherrington" (Hunt and Perl, 1960).

At different times many conflicting views have been put forward to account for the neurological changes

following spinal cord injury. Ross (1881) attributed increased reflex activity to diminished cerebral influence. Bastian (1890) held that the chronic reflex state was one of lessened activity. Collier (1904) regarded the reappearance of deep reflexes as a sign of recovery or regeneration of conducting elements. Spinal shock is described in Oppenheim's Textbook of Neurology (1900) in terms which we would regard today as descriptive of surgical shock or peripheral circulatory failure. In more recent times Scarff and Poole (1946) and McCarty (1954) have attributed the increased reflex state to hyper-irritability of the distal cord stump.

The main stream of clinical work supports the release theory of Sherrington. One of the earliest corroborative studies in the human was the detailed report of their paraplegic cases by Head and Riddoch (1917, 1918) during the First World War. They found that spinal shock lasted several weeks and merged gradually into the state of increased reflex activity. The first reflexes to appear were primitive genital reflexes, namely the bulbo-cavernosus and anal reflexes; these reflexes could be elicited within a few hours of injury and in some cases were never lost. The found in association with increased reflex activity the occurrence of mass flexion movements

of an involuntary nature to which they gave the name 'mass reflexes.' The mass movements were elicited by very minor stimuli such as lightly brushing the bed-clothes against the paraplegic limb, or by autonomic stimuli such as distension of the bladder. Mass reflexes often included spontaneous autonomic activity such as evacuation of the bladder, sweating and rises in blood pressure. During the spasm the patient often complained of pain. Varying severity of mass movement occurred ranging from incapacitating spasm to occasional involuntary twitches of one or more muscles. In complete paraplegia spasms were flexor as a rule and in incomplete paraplegia, extensor. Head and Riddoch also made the observation that the occurrence of febrile illness in the patient, either from urinary sepsis or pressure sores, delayed the appearance of mass reflexes and caused them to revert towards the flaccid state of spinal shock.

Most of these observations have now been confirmed by many sources. Text-books of neurology by eminent authors (Fulton 1949, Walsho 1958 and Brain 1962) repeat the view that complete paraplegia results in a predominance of flexor spasm and ultimately the clinical picture of paraplegia-in-flexion and incomplete paraplegia extensor

spasm and paraplegia-in-extension. This pattern is explained by the fact that continuity with the higher centres results in dominence of the anti-gravity muscles while severance of the cord from all higher control produces a more primitive type of reflex behaviour which is characterised by its flexor nature. A second impetus to the study of the spinal cord injuries was provided by the Second World War (Davison, 1943, 1960, Munro, 1943, 1952, Grant 1945, Guttmann 1946, 1947, 1949, 1953, 1954, Freeman 1949, Bors 1951, Mayfield 1953, Scarff 1960) and by the Korean War (Vanmamaker 1954). One important study (Kuhn and Macht, 1949) confirmed Head and Riddoch's description, with one or two modifications. The type of reflex pattern, flexor or extensor, was not determined by the degree of completeness of the cord injury but by the length of time that had elapsed since injury. Following spinal shock, which these authors found lasted one to six weeks, there occurred a stage of minimal reflex activity lasting for several weeks or months; this was followed by a stage of alternating flexor and extensor spasm up to about a year after injury, and finally there emerged a stage when extensor spasm predominated which lasted indefinitely. Five cases were described in which the injury

was unquestionably sustained at cervical cord level, but the resultant paraplegia was of lower motor neurone type. They were unable to confirm that febrile illness in the patient suppressed the occurrence of mass reflex activity.

It is often assumed that if there is no recovery of voluntary movement in complete paraplegia within the first 24 hours of injury physiological transection of the cord has occurred and no recovery can be expected (Holdsworth 1954, Naffziger 1938). This view has been challenged, all cases of traumatic paraplegia being potentially recoverable for at least several months after injury. (Scarff 1960). It is rare for anatomical transection of the cord to occur in closed injuries (Jefferson 1927). A temporary suppression of spinal cord function may result from less severe injury to the cord which is followed within a few minutes or hours by a complete return to normal. This is known as spinal concussion (Davison 1943).

A component of spinal injury may be damage to the nerve root. This assumes significance at the level of the lumbodorsal junction where, owing to the obliquity of the roots only the sacral segments of the cord may be affected in the presence of a totally paraplegic lower

limb. The problem of differentiating root injury from cord injury in the early stages of spinal shock has been examined by Holdsworth (1954, 1956), the early emergence of ano-genital reflexes distinguishing a cord injury from a root injury in the absence of any recovery of motor or sensory function.

(b) Regeneration of the Spinal Cord.

It is widely accepted that regeneration in the human cord does not occur (Cajal, 1928, Cushing, 1905, Thompson, 1923, Clark, 1943, Naffziger, 1938). Regeneration has been shown to occur in fish (Koppanji, 1924, Sperry, 1948), amphibians (Spallanzani, 1768, Piatt, 1955) and reptiles, Kamrin and Singer, 1955). The failure to regenerate in higher species is attributed not to lack of intrinsic growth potential in nervous tissue, but to the absence of a favourable environment for growth (Cajal, 1928). Frazier (1918) suggested that the missing growth factor in the central nervous system was the absence of Schwann cells. It has been shown (Duel and Ballance, 1932) that growth in peripheral nerves was under the influence of neurotropic influences arising in the distal, degenerate portion of the nerve. Weiss (1934) presented evidence which strongly challenged the theory of neurotropic influences, and Bentley and Hill (1936) showed that degenerate peripheral nerve had no enhanced neurotropic

power. In an important contribution to the subject, Sugar and Gerard (1940) found anatomical and electrophysiological evidence of regeneration in the spinal cord of young rats. They showed that the presence of red cell extravasation and phagocytosis prevented regeneration, that transplanted muscle and peripheral nerve promoted regeneration by facilitating orientation, and that signs of grey matter regeneration were never seen. They challanged Cajal's conclusions about degeneration and regeneration in the nervous system on the grounds that most of his spinal cord transections were performed below the level of the large lumbar intersegmental artery, and were therefore prejudiced by ischaemia of the distal stump. The neurotropic theory was revived by the finding (Shapiro and Warren, 1949, Bucker, 1948) that axons within the central nervous system grew into transplanted tumours in rabbit eye. Levi-Montalcini (1953, 1956) demonstrated neuroregenerative properties for extract of mouse sarcoma and snake venom, and extracted an active principle which was a protein fraction derived from microsomes and behaved as a humeral agent. A feature of Levi-Montalcini's regeneration was hyperplasia, and increased differentiation of neurones. It was concluded that the appearances were not simply explicable by a hastening of normal growth, but

that "hitherto unknown mechanisms are in operation." (Levi-Montalcini, 1953). Windle (1950) presented experimental evidence that a pyrogen derived from bacterial polysaccharide possessed neuroregenerative properties. This substance, called 'piromen', caused young cellular fibrous tissue to be produced which, in turn, promoted the orientation and growth of sprouting axons. Scott and Clemente (1955) showed histological and electrophysiological evidence of regeneration in 'piromen' treated animals. It had previously been shown (Clark 1943) that peripheral nerve, implanted into the central nervous system, only grew in the presence of a cellular reaction, the presence of mature collagen having an inhibitory effect. Freeman (1959) showed a similar neuroregenerative action for the enzyme trypsin. Windle's work has been challanged. McCullough (1959) did not find piromen beneficial in peripheral nerve regeneration, Arteta (1956) found no real functional or anatomical benefit from piromen in regeneration studies on the central nervous system, and Davidoff (1948) found that the prevention of mature scar formation in cats did not promote regeneration within the central nervous system. It is now known that the spinal cord possesses remarkable powers of functional recovery if only a small part of it is spared in experimental studies, particularly if that part is an area

of the anteromedial portion of the ventral columns (Windle, 1955). Some interesting pictures of dense axon penetration in transection areas have been shown using a porous cuff to restore continuity of the cord (Campbell 1957). Freeman et al (1960) following up an observation that nerve fibres have no specificity, (Sperry 1947), has attempted to use segments of intercostal nerve as internuncial pathways by swinging them down and inserting them into the distal cord stump.

2. SCOPE OF THE STUDY

From the records of Glasgow hospitals fifty two cases of spinal cord injury were obtained. The location of cases was as follows:

Royal Infirmary	25
Western Infirmary	18
Victoria Infirmary	5
Law Hospital	3
Southern General Hospital	1
Total	52

The only criterion for inclusion of a case in the series was injury to the spinal cord or cauda equina. Cases of myelopathy due to herniation of the intervertebral disc were not included.

The administrative practice during the period covered by the study was to admit spinal injuries to the city hospital serving the area in which the accident occurred. In each hospital subsequent disposal varied. In the Royal Infirmary, all except two of the 25 cases admitted there were admitted to the Receiving General Surgical Unit of the day. The other two were admitted to the Orthopaedic Unit. In the Western Infirmary, all patients except one were admitted to the Orthopaedic Unit. The exception, occurring in the early part of the period, was admitted to the Receiving General Surgical Unit of the day.

Cases in the Victoria Infirmary and Law Hospital were admitted to the orthopaedic wards and the solitary case in the Southern General Hospital to a general surgical ward. In all hospitals a neurosurgical opinion was obtained if desired and patients deemed suitable by the neurosurgeon for a decompression operation, transferred to the Neurosurgical Unit at Killearn Hospital. In the course of treatment other patients might be referred to the urological, plastic and gastrointestinal units for various complications. Towards the end of the series a small number were transferred at an early stage to a Spinal Injuries centre in England. Except for this latter group, the original unit admitting the patient was responsible for long term care and rehabilitation, those cases being transferred to specialised units, being referred back when the specialised treatment was completed.

Abstracts were made from the case notes and studied. Where the Case Notes were deficient in respect of information on these aspects, the records were restudied to obtain fuller information. For instance, where the radiological data was incomplete, the original x-rays were sought; where the patient had been referred to other departments for definitive treatment of pressure sores, urinary complications, gastrointestinal complications or

neurosurgical decompression the Case Notes from these departments were sought.

The next step was to locate the survivors and visit them. In this connection the Miner's Rehabilitation Centre at Uddingston was most helpful. Twenty-four patients were questioned and examined personally; the remainder had either died or left the country or could not be traced. A tabular summary of all the information obtained was drawn up (Appendix).

The cases are not a true random selection. The number was limited to fifty-two because it was difficult to obtain any more reasonably well documented case notes. Many cases had to be discarded because of inadequate documentation. The period covered is 1945 to 1958. All the patients except one were injured during this period; the exception occurred in 1940. The review, therefore, covers spinal cord injuries treated under peace-time conditions in and around the city of Glasgow, in the decade or so following the Second World War.

3. CONSIDERATION OF VARIOUS ASPECTS OF THE CASES

a) Age and Sex Incidence

The series comprises fifty-two cases of whom 48 are male and 4 female. The greatest incidence occurred in adult men of working age (Figure 1). The youngest was 17 years and the oldest 73 years.

b) Aetiology

The type of injury was readily classified into one of the following groups: falls from a height, mining accidents, traffic accidents, falls when under the influence of alcohol, industrial accidents, and a miscellaneous group (Table 1). Traffic accidents affected predominantly the cervical spine; other accidents the dorsal and lumbar spine.

c) Early Mortality

11 of the 52 cases in the series died within the first few weeks after injury, most of them within the first few days, giving an early mortality of 21%. This figure was not reflected evenly over the different types of injury, mortality from traffic accidents being 66.7%, and from all other injuries 12.8%, a highly significant difference.

	Dead	Survivals	Totals
Traffic	6 (66.7%)	3	9
Others	5 (12.8%)	34	39
Totals	11	37	48

$\chi^2 = 9.15 \quad p < 0.01$

Seven of the eight fatal cases of cervical cord injury died from respiratory insufficiency which is precipitated by paralysis of intercostal muscles and is a direct consequence of the injury to the cord (Table 2). All three injuries of the dorsal and lumbar spine died from associated injuries, mostly to the chest and abdomen.

In addition to respiratory insufficiency, one patient in the cervical cord group sustained an acute failure of temperature regulating mechanism becoming irreversibly hypothermic. A complete neurological deficit in cervical injuries is associated with a 100% mortality (Table 2). A complete neurological deficit in dorso-lumbar injuries is associated with a higher mortality rate than in incomplete deficit but the numbers are too small to show a significant difference.

d) Radiological Appearances

In the group of injuries to the cervical spine eight were found to have radiological evidence of injury to the bone. (Table 3) Three sustained fracture-dislocations, three sustained dislocation and two sustained crush fracture of the vertebral body. The fifth or sixth cervical vertebra was involved in all but one case; in that case a fracture dislocation occurred between the seventh and eighth vertebrae.

Seven cervical cord patients had no radiological evidence of injury to the spinal column. Four of these exhibited radiological evidence of osteoarthritic change of the spine and three were in the over-60 age group. Both cervical cord injuries sustained when the patient was drunk belonged to this group. The high incidence of cord injury without bone injury in the cervical spine (47%) is different to a highly significant extent from the incidence in lumbo-dorsal injuries (8%) (Table 3).

	Positive X-rays	Negative X-rays	Totals
Cervical	8	7	15
Lumbodorsal	25	2	27
Totals	33	9	42

$X^2 = 11.35 \quad p < 0.01$

Two of the eight early cervical deaths belonged to the group showing no bone injury.

In the group of six dorsal spine injuries all were found to have radiological evidence of crush fracture of the vertebral body. In five of these, three or more adjoining vertebral bodies were crushed. All sustained violent injury of a direct nature; four fell from a height, one was crushed by a weight on his back and one was injured in a fall when drunk.

The third group comprises injury to the lumbar spine and lumbo-dorsal junction. Injury to the last two dorsal vertebrae are placed in this group. There were thirty-one in all: nineteen sustained fracture-dislocation, five sustained a crush fracture of the vertebral body, and one had evidence of an isolated transverse process fracture. Two showed no radiological evidence of bone injury, and the radiological picture of four is unknown.

Paraplegia of delayed onset occurred in two patients whose initial radiological picture revealed little or no damage to the spine.

Case 43. A 61 year old man fell from a scaffold and injured the cervical spine. There was no neurological deficit. Bone

injury was suspected but full radiological investigation of the spine revealed no bone lesion and he was discharged within a few days. He continued to suffer severe neck pain. Seven weeks after injury he quite suddenly lost the power in his limbs and fell. On re-admission he was found to have a complete quadriplegia. X-rays showed a forward dislocation of the sixth cervical vertebra upon the seventh. He died four days later of respiratory insufficiency and pulmonary oedema.

Case 23. A 56 year old man fell 40 feet, landing on his back. There was no neurological deficit. The only positive radiological finding was an isolated fracture of a lumbar transverse process. Ten years later he was re-admitted to a medical ward with a history of progressive spastic paraplegia. After intensive investigations for the presence of neurological disease, a diagnosis was reached

of chronic sequelae of trauma to the spinal cord. X-rays then showed advanced osteoarthritic change in the spine.

The first case is an example of paraplegia following an unstable cervical spine injury which was not immobilised at the time. The second case is an example of the chronic neurological sequelae of acute cord injury.

e) Surgical Treatment

Only the 41 patients surviving the initial few weeks after injury were included. With one exception, injuries to the cervical spinal column were treated by non-operative methods (Table 4). Of the injuries sustained by the dorsal and lumbar spine approximately half were subjected to operation. Various devices to facilitate turning of the patient were used. These included a water bed, air bed, meccano bed, plaster shells, and a turning frame. The turning frame was used with increasing frequency in the later years covered in the series. Of seven patients whose spine was fixed with a metal plate at operation six were subsequently nursed in a turning frame; five of these developed severe pressure sores.

The relationship of the various orthopaedic measures to the complications of pressure sores and residual stiff

back is shown in this table (Table 4). There is a tendency for patients nursed in an ordinary bed to fare better in respect of pressure sores than those nursed in other ways but the numbers are too small to show any significant difference. When the incidence of pressure sores is related to open versus closed methods of treatment no significant difference emerges and when related to the level of the spinal injury there is no significant difference. Patients are classified as having residual stiff back if either of these two criteria were present: complaint of pain and stiffness of the back, and greatly diminished voluntary movement in the back. Eight patients were found to have residual stiff back. They were evenly distributed between the group treated conservatively and the group treated by operative methods.

Patients were transferred from general surgical and orthopaedic wards to the neurosurgical unit at Killearn Hospital if there was evidence that there was continuing compression upon the cord. Compression upon the cord was thought to be present if there was progression of the neurological deficit in the first hours or days after injury, and the demonstration of a sub-arachnoid block by the Queckenstedt test or by myelography. Six patients fell into this group and were subjected to the operation of

decompression laminectomy within a few days or weeks after injury (Table 5). Four had lesions of the lumbo-dorsal spine, one a lesion of the mid-dorsal spine and one a cervical spine injury (Table 5). The cervical laminectomy benefited slightly from this operation, the remainder showing no neurological improvement. It was common to find extra-dural blood clot at operation or fragments of bone or disc lying against the cord. In the case which showed some improvement a large spur of bone compressing the cord was removed, the cord remaining tense and swollen. Although the dura was incised more than once in no case was a myelotomy performed to decompress the swollen cord.

Three cases came to laminectomy many months or years after injury, evidence being present that the cord was being subject to compression. In one a sub-arachnoid block alone was present, in another progression of the neurological deficit alone was present, and in the third progression of neurological deficit and a sub-arachnoid block were present. These late laminectomies did better than the early ones, all three benefiting from the operation. In each case a spur of bone was found impinging on the cord, and after its removal a spinal bone fusion performed. There was no evidence of chronic meningeal scarring in any of them.

(f) Neurological Prognosis.

Immediately following injury 28 patients had a complete neurological deficit, and 24 an incomplete one (Table 2). Eleven patients died within the first few weeks. Of the 41 survivors 13 were known to have remained complete, ten of whom exhibited paralysis of lower motor neurone type and three a mixture of lower and upper motor neurone paralysis (Table 6). No patient whose paraplegia was wholly of upper motor neurone type was found in the group whose neurological deficit had remained complete. A classification of improvement was made according to whether recovery from the neurological deficit was major or negligible. Comparing the improvement which occurred in upper motor neurone lesions with that in lower motor neurone lesions, no significant difference emerges in the incomplete paraplegia. Table 6 does illustrate a tendency for improvement to be better in upper motor neurone lesions but the numbers are too small for a significant difference to emerge. In order to show a statistical difference between the two groups the complete and incomplete lesions are taken together and the degree of recovery compared between upper and lower motor neurone lesions.

	Major Recovery	Negligible Recovery	Totals
U.M.N.	8 (57.1%)	6	14
L.M.N.	1 (5.6%)	17	18
Totals	9	23	32

$\chi^2 = 7.97 \quad p < 0.01$

It is apparent that there is a highly significant difference between the percentage of upper motor neurone lesions exhibiting a major recovery (57.1%) and the percentage of lower motor neurone lesions exhibiting a major recovery (5.6%).

The difference in prognosis for neurological recovery is reflected in the level of injury to the spine. All surviving cervical cord injuries were of upper motor neurone type, and all flaccid injuries were found in the lumbodorsal injuries. It is impossible on clinical grounds to distinguish between a lower motor neurone paralysis due to anterior horn injury and one due to root injury. Five of the survivors were thought to have pure cauda equina lesions on clinical and radiological grounds. Since these are root injuries any superiority for root injuries in respect of neurological recovery might be expected to bear on these statistics. That the prognosis

was so significantly superior in upper motor neurone lesions not withstanding the inclusion of these pure root lesions is adduced as further evidence that cord injuries fare better than root injuries.

Ten patients were known to be much troubled by involuntary movements, four most distressingly so. These spasms usually commenced in the first week or so after injury, increased in severity for a further period of weeks or months, and then gradually subsided over a longer period of months or years. Development of joint contractures seemed to diminish involuntary movements. There was no greater liability to spasms between high and low cord lesions, and no preponderence of flexor or extensor spasms in either type. One patient whose residual paraplegia was of lower motor neurone type swore he had been troubled by involuntary movements at an earlier stage. Flexor spasms were seen more often than extensor spasms. In five patients spasms were always flexor, in one always extensor, and in four both flexor and extensor at different times. The worst spasms tended to be flexor in type. Incomplete lesions were not more associated with extensor than flexor spasms, and complete lesions not more associated with flexor spasms. The case of exclusive extensor spasms was

an incomplete cervical lesion; the only complete paraplegia which exhibited paralysis of upper motor neurone type (a mixed lesion) was troubled with both flexor and extensor movements. The posture of the limb at the onset of a spasm seemed to affect the type of spasm; a patient would say that, curled up in bed his legs would shoot straight out while, standing his legs would buckle forcibly under him causing him to be almost thrown to the ground.

Involuntary movements were often associated with intractable pain. The pain was described variously but common to most descriptions of it was the adjective 'burning'. It was often located in the lower parts of the abdomen as well as in the paraplegic limbs themselves. Relief was only obtained with heavy sedation with morphine or pethidine. No record could be found of any patient suffering from painful spasms being treated by surgical measures such as rhizotomy, instillation of necrotising agents into the sub-arachnoid space, or peripheral neurectomy. Some cases were referred to Paraplegic Centres in other parts of the country for treatment of spasms and other late complications. One patient with an incomplete cervical cord injury who suffered from flexor spasms which prevented him standing had relief following the per-

-cutaneous infiltration of the sciatic nerve with local anaesthetic which lasted for about two weeks. Several patients with advanced paraplegia-in-flexion were seen. The problem of spasms was one of the most difficult and distressing encountered.

g) Skin Complications

Twenty-four of the forty-one survivors were known to suffer from severe pressure sores, an incidence of 66.7% since the state of the skin of five was unknown. Pressure sores were considered severe if they required surgical excision and grafting, if they persisted throughout the period of hospitalisation, if they were the cause of the patient being readmitted to hospital, or if they required arrangements for permanent dressings at home.

There was a particular liability to develop pressure sores during the phase of spinal shock. In the later stages pressure sores were more frequently encountered in complete paraplegias than incomplete lesions, (Table 7) as might be expected since loss of sensation is the prime cause of sores. In studying whether upper motor neurone lesions in which the neurological deficit was incomplete were more or less prone to pressure sores than lower motor neurone lesions in the same group, the numbers proved to be too small for any significant statistical difference to

be detected although there did appear to be a tendency for the former to be less afflicted. When the complete and incomplete lesions are considered together the incidence of pressure sores in upper motor neurone lesions is 33.3%, and in lower motor neurone lesions 87.5%, a highly significant difference.

	Severe Sores	Negligible Sores	Totals
U.M.N.	4 (33.3%)	8	12
L.M.N.	14 (87.5%)	2	16
	18	10	28

$\chi^2 = 6.6$ $p < 0.01$

Cord lesions, therefore, have a better prognosis than root lesions in respect of pressure sores.

h) Urinary Complications

Seven of the 41 survivors were known not to have required initial drainage of the bladder, and none of these developed a urinary infection which was not easily controlled. The initial bladder treatment of another seven could not be established. Of the remaining 27 in whom some form of bladder drainage was instituted, 20 developed severe urinary sepsis, and incidence of 74% (Table 8). The description 'severe urinary sepsis' indicates that the patient suffered recurrent febrile

illnesses and the continuing presence of resistant organisms in the urine. 17 of these patients never regained any useful bladder function and were permanently incontinent. Chronic renal sepsis accounted for the death of 8 patients, a late mortality of 19.5%.

The type of initial bladder drainage is shown in this table (Table 8). An in-dwelling urethral catheter of Foley type was most commonly used, being changed and washed out at intervals. A Gibbon catheter was employed in two cases, both of whom developed severe urinary sepsis. The system of automatic bladder wash-out known as tidal drainage was employed in six cases all of whom developed severe urinary sepsis.

Seven patients underwent trans-urethral resection of the bladder neck after assessment of bladder function by cystometry. Four of these remained on continuous catheter drainage and the ultimate status of the other three not known.

It was not possible to arrive at any estimation of the incidence of urinary lithiasis. The reason for this was that no arrangements were made for routine urological follow-up of patients after they were discharged from hospital unless the patient had been referred to a urological unit while he was still in hospital.

i) Gastro-intestinal Complications

Several patients were referred by their own doctors to general surgical and medical units with symptoms referable to the gastro-intestinal tract. Records of all such referrals are probably incomplete but two distinct groups emerged: a) a group with dyspeptic symptoms and b) a group with diarrhoea.

a) Dyspepsia. There are 11 patients in this group. All had severe symptoms suggesting the possibility of peptic ulceration of the stomach or duodenum. One was admitted with a haematemesis. Records of the radiological findings in seven was obtained. A diaphragmatic hernia was queried in one, duodenal spasm was noted in another, acute erosions were suspected in a third (the patient with haematemesis), and the remaining four showed no radiological abnormality. In no case was a peptic ulcer demonstrated. An emergency laparotomy for suspected perforation of a peptic ulcer was performed in one and two others came to laparotomy for suspected peptic ulceration. In none of these cases was any lesion of stomach or duodenum found. A cholecystectomy was performed in one of them.

b) Diarrhoea. There are 6 patients in this group. Common to all was a history of severe recurring attacks of

diarrhoea usually accompanied by severe lower abdominal colic. Bacteriological examination of the stool was carried out in five of these and was negative in every instance. An attempt was made to trace any record of barium enema investigation having been carried out but no record was found. Repeated unsuccessful sigmoidoscopy was performed in one in which a provisional diagnosis of ulcerative colitis was reached. In this case a colectomy was contemplated but not performed. Two patients passed blood in the stool during attacks of diarrhoea. Amyloid disease was confirmed in two patients who came to post-mortem, both of whom also had chronic renal sepsis.

j) Morbidity

Eight of the 41 patients surviving the initial injury ultimately died, all from renal sepsis, a mortality of 19.5%.

The average period of continuous hospitalisation was 13½ months (Table 9). Many of these were subsequently re-admitted for treatment of complications such as pressure sores, urinary sepsis, gastro-intestinal complications or stiff joints. Seven patients were hospitalised continuously for over two years, one of these in a Professorial Surgical Unit.

Few were rehabilitated to the stage of being gainfully employed (Table 10). 24 were never gainfully employed at

any time, 3 were gainfully employed sporadically, and three were gainfully employed for continuous periods. The employment status of the remainder is not known.

The sex life of married paraplegics was much impaired. Nine of the fifteen complete paraplegics were questioned as to their sex status. One was able to have a form of intercourse on occasions, two still had sexual desire, two were able to have emissions, and five could be manually stimulated to have erection of the penis. Chronic oedema of the penis was seen in several cases. Descriptions of the acute period immediately following injury often mentioned the occurrence of priapism.

Invalidism was caused by joint contracture in some cases. Spontaneous ossification in and around these joints, particularly the hip joint, was seen. Ossification in the belly of quadriceps muscle, well away from the joint, was seen in one case. An other patient had pronounced enlargement of the breasts. Laboratory studies of metabolic disturbances were not made.

4. DISCUSSION

a) The Organisation of Paraplegic Care

The results presented are at variance with other reports on spinal cord injuries. Discussing the vast improvement in the outlook for patients with traumatic paraplegia, Watson-Jones (1955) has written - "Until very recent years, only five or ten years, nearly every patient who sustained fracture-dislocation of the spine with permanent paralysis died very soon. They lay rotting in bed with large stinking bed-sores, lacking sensation, incapable of movement, often with distressing involuntary spasm and secondary contracture of joints, incontinent of urine and faeces, with infection of the urethra, bladder and renal tract." The recent development of which Watson-Jones speaks is the creation of special centres for paraplegics. He states - "The first important duty of a doctor called in to see a patient with fracture-dislocation of the spine and paraplegia is to arrange prompt admission to a centre where special arrangements exist, even if a hundred or a thousand miles away." Writing of the results from the centre at Sheffield, Holdsworth reported "We have, I think, clearly shown that by correct nursing and careful attention to simple bladder drainage, and by correct rehabilitation, all serious complications can be easily avoided, the general health of the patient maintained, and the stay in hospital

out down to nine or ten months. In the 71 patients treated from the outset here there has not been one serious bed-sore and no case of urinary infection which was not easily controlled." (Holdsworth 1954).

These reports are in sharp contrast to the material presented herein. The incidence of severe pressure sores in this series was 66.7% and the incidence of severe urinary infection, in those whose bladder required drainage initially, was 74%. Distressing involuntary movements were a common occurrence and severe joint contractures were seen. Seventeen patients were left totally incontinent of urine and eight died from chronic renal sepsis. The average period of hospitalisation was 13½ months, a figure which excludes all readmissions. Only 10% of patients were employed for any continuous length of time, in contrast to an incidence of 69% reported from the Spinal Injuries Unit at Stoke Mandeville (Guttmann 1954b). Patients still lacked sensation and movement but in this respect they were no different from patients in Paraplegic Centres, notwithstanding Watson-Jones' remarks.

"General hospitals, no matter how good, are not equipped to deal with this condition, and in particular lack the special nursing provision." (Ross 1957). The difficulty is evident when a paraplegic patient's medical

and nursing needs are fully met in a general ward some acutely ill patient might relapse for lack of attention. One of the most striking aspects of organisation seen on a visit to the Spinal Injuries Unit at Stoke Mandeville is the skilled nursing and medical attention which is available to the paraplegic at all times. If a paraplegic complains of a slight rigor it may mean his urinary catheter is not functioning properly. Immediate adjustment to the catheter may be all that is needed to restore drainage and prevent a renal infection supervening.

Mechanical aids are not a substitute for nursing and medical attention. Both patients in this series whose bladder was drained by the less irritating small bore polythene catheter (Gibbon, 1958) and all six patients having tidal drainage of the bladder (Munro 1947) developed severe urinary sepsis. The incidence of pressure sores in patients nursed in a turning frame was high, five out of six nursed in a turning frame after internal fixation of the spine developing sores. Nearly all the patients had intensive physiotherapy and many developed joint contracture, particularly in the small joints of the wrist and hand in cervical lesions. In some cases a slight but useful degree of active finger movement was lost through joint stiffness. The reason for the occurrence of joint stiffness is thought to be

that physiotherapy was not given sufficient emphasis and was not integrated into an intensive programme of rehabilitation. Another striking aspect of the organisation at Stoke Mandeville is that rehabilitation of the patient starts as soon as he is admitted to hospital. The same is true for the other Paraplegic Centres. If a patient has, for example, a complete quadriplegia with a level at the sixth cervical cord segment he will come to depend on shoulder movements and flexion of the elbow for all his useful function. These joints and the appropriate muscles will be developed to the maximum and a start made on providing suitable active splinting to give him some sort of grasp. The new flexor hinge splints with artificial muscles may be useful. Arrangements will be got under way with the Local Authorities to provide him with a suitable kitchen at home so that he can still, in spite of his grave incapacity, look after himself to some extent, and even have a means of gainful employment. In a spinal centre the patient is told of the disability which can be expected and encouraged to co-operate to the full to obtain the best advantage. Such a programme is in sharp contrast to the pathetic, vague encouragement given to

paraplegics in general hospital wards, and the gradual disillusionment of both patient and doctor as it becomes all too apparent what the end result is really going to be.

At the present time there are not enough paraplegic beds in the country. The new centre at Edenhall, Edinburgh already has a waiting list of several months. Specialised skill abounds in Glasgow but it is not being co-ordinated effectively because the paraplegics are scattered all over the city in general hospital wards. Responsibility is shared by various specialists who are brought into consultation and there is a tendency for no-one to assume ultimate responsibility for the patient. To provide all the specialist services needed in cases of traumatic paraplegia within a simple doctor-patient relationship the concept of a consultant-in-charge should be realised. There has been no change in the medical provisions for paraplegics in Glasgow five years after the period covered by this review. They are still shuttled backwards and forwards between surgical wards, orthopaedic wards, plastic wards, urological wards and neurosurgical wards. At the time of writing (May, 1969) there is an incomplete cervical quadriplegic patient in the general surgical ward in the Royal Infirmary, Glasgow. She was

admitted in December, 1962 and was transferred to the neurosurgical unit at Killearn where an early decompression laminectomy was performed without neurological improvement. Unsuccessful efforts have been made to have her admitted to the Spinal Injuries Centres at Edenhall and Stoke Mandeville for rehabilitation. She is troubled by painful flexor spasms of the legs and increasing flexion-adduction contracture at the hips. She has organic contractures of the fingers of both hands, and is totally incontinent. Although she has no pressure sores she is not rehabilitated, cannot be allowed home, and meanwhile must remain in a general surgical ward.

The highly significant increase in early mortality from traffic accidents when compared with all other injuries to the cord has not been previously reported as far as is known. This has been shown to be due to the high incidence of cervical cord injury in this group and respiratory insufficiency. With more use of the acute respiratory unit in hospitals it can be expected that more of these patients will be salvaged. As the rehabilitation of high cord lesions is the most demanding of all cord injuries any future planning for spinal injuries should take into account the fact that more cervical injuries are going to survive.

b) The Place of Surgery

In injuries of the cervical spine a peak incidence at the fifth and sixth cervical level (Jefferson 1928) was confirmed.

Cervical injuries are usually the result of indirect violence, associated injuries to the occiput and face suggesting flexion and extension cervical injury respectively (Jefferson 1927). When a head injury leads to unconsciousness and paralysis, a concomitant cervical cord injury may be missed. In these circumstances the occurrence of priapism, curved penile erection, points to associated cord injury (Jefferson 1927). This sign was observed frequently in cervical cord injuries in the series.

Cervical cord injuries are classified according to whether the neck is forcibly flexed or extended and whether there is or is not associated vertebral column damage. Barnes (1948) has classified them as follows:

1. Flexion injury	a) Dislocation
	b) Compression fracture
	c) Retropulsion of the disc
2. Hyperextension injury	a) Dislocation
	b) Associated spondylosis

The compression fracture is a fracture dislocation, the so-called tear-drop fracture, and is characterised radiologically by "downward and forward displacement of the

antero-inferior margin of the involved vertebral body with displacement of the same vertebral body into the spinal canal." (Schneider 1956). Dislocation and fracture dislocation occurred with almost the same frequency in the series.. Reduction is effected by skull traction (Crutchfield 1933), and weights of up to 30 lbs. applied over several days are sometimes necessary (Barnes 1948). One dislocation, apparently missed at the time due to spontaneous reduction having taken place, redislocated six weeks later. Instability of these injuries may be present after a full period of immobilisation and for that reason bone fusion advised by some, either at an early stage (Schneider 1956) or at the end of a full period of immobilisation (Guttmann 1963). Stabilisation of the cervical spine may be performed by anterior fusion (Bailey and Dodgley 1960).

The patients who did not exhibit radiological evidence of bone injury nearly all belonged to the group having pre-existing cervical spondylosis. In only one case was retropulsion of a disc a possibility and at operation in that case a prominent spur of bone found. The method of selection of cases may to some extent be responsible for the absence of disc retropulsions, as cases with pre-existing disc disease were excluded. This was done mainly to exclude pre-existing lumbar disc disease and it is

possible, in retrospective study, that protrusions of healthy cervical discs were discarded. There is no doubt that retropulsion of a healthy cervical disc can occur, the whiplash injury, and give rise to compression myelopathy (Cramer and McGowan 1944, Barnes 1948). It is not a common cause of traumatic paraplegia (Guttmann 1949). When the spine is injured neurological signs are caused by instantaneous damage to the structures within the cord and only very rarely by external compression upon the cord (Thorburn, Holmes 1915, Thompson 1923, Naffziger 1938, Jefferson 1927, Scarff 1960). When it is present the most important sign of compression is progression of the neurological deficit and decompression laminectomy should be performed (Jefferson 1927, Guttmann 1949, Naffziger 1938, Schneider 1951). Some writers (Davis 1942, Munro 1943) require in addition a manometric block before operating and others (Mixter 1934, Mock 1933, Coleman 1927, Semmes 1933, Gurdjian 1930) operate in the absence of clinical progression so long as a complete block can be demonstrated. Scarff (1960) has pointed out that evidence of compression upon the cord may be produced by swelling of the cord itself. Progression of neurological deficit and the demonstration of a block were present in one cervical cord lesion in this series. The block was found at

operation to have been caused by swelling of the cord, and although a prominent spur of bone was removed the neurological improvement was only slight. Where progression of neurological deficit and a block are present it is probably wise to perform a decompression operation as a disc retropulsion may be present. The expectation that large numbers of cervical cord injuries will benefit from surgical decompression (Wannamaker 1954, McGravey 1945) is not justified.

The frequency of cervical cord damage in spondylitic spines was confirmed in this series. This injury is commonly attributed to hyperextension of the neck, (Taylor and Blackwood 1948, Crooks and Birkett 1944, Barnes 1948). It is not always produced by extension (Symonds 1953). The spinal cord of spondylitic patients appears to be more vulnerable to injury. The risk is greatly increased under anaesthesia (Walshe 1958) and it is noteworthy that the two patients who sustained cervical cord injury when drunk had pre-existing cervical spondylosis. The cord is normally protected from vibration stresses by the roominess of the spinal canal and the tone of the spinal muscles. It has been suggested (Schneider 1959) that myelopathy in a healthy spine may be caused by recurrent episodes of acute trauma which pass unremarked by the patient. One case in

the series is possibly an example of this (Case 23). Surgical exploration is not indicated in spondylitic myelopathy (Barnes 1948). Stabilisation of the spine of patients with cervical spondylosis as a prophylactic against the occurrence of traumatic myelopathy might be worth considering in those cases who already exhibit minimal cord signs.

Dorsal spine injuries are usually the result of direct violence. The patients reported had either fallen on their backs or been crushed by a weight on their backs. Common to all was a crush fracture of the vertebral body and in five of the six dorsal injuries several adjoining vertebrae were involved. The solitary vertebral crush fracture where the body assumes the form of a wedge, the base being situated posteriorly, is a stable flexion injury in which the posterior longitudinal ligaments have remained intact and the cord is seldom involved (Holdsworth 1956) when several vertebral bodies are involved it must be considered an unstable injury and damage to the cord a frequent occurrence. It is commonly stated that the prognosis is particularly poor in dorsal cord injuries (Holdsworth 1954). The reasons put forward to account for this are that the spinal canal is narrowest in the dorsal spine (Scarff 1960, Guttmann 1954a).

and that the dorsal cord has the poorest blood supply. (Suh and Alexander 1939). It is thought from the small number of dorsal injuries in this series that the dorsal spine is less prone to injury. The fact that the incidence of complete paraplegia was no greater in mid-dorsal injuries than in lumbo-dorsal injuries does not substantiate the claim that the prognosis is worse in dorsal injuries.

Injuries of the lumbo-dorsal junction and lumbar spine are also due to direct violence. The former are extremely unstable, and any displacement correcting itself when the patient is placed supine, the radiological picture in consequence seldom showing the extent of the injury (Holdsworth 1956). The instability of multiple dorsal and lumbo-dorsal injuries requires that they be immobilised untill union of bone has occurred. This may be achieved by internal fixation with a metal plate (Holdsworth 1954, 1956) or simply nursing the patient in an ordinary bed with pillows suitably placed to keep the spine extended (Guttmann 1954). External fixation in plaster is absolutely contra-indicated in a paraplegic because of the danger of pressure sores. It is argued that the residual back function is better after conservative immobilisation than after internal fixation. The results presented here

showed no superiority for non-operative methods in this respect.

c) Prognosis in Upper and Lower Motor Neurone Paralysis

The finding that prognosis for neurological recovery was better in cord lesions with upper motor neurone paralysis, than segmental or root lesions with lower motor neurone paralysis, is at variance with most opinions. Holdsworth (1954) has written "The prognosis of root paralysis is better than that of cord paralysis or at any rate it cannot be worse." It is however, impossible to distinguish a segmental injury to the anterior horns from a root injury, and while roots may be theoretically capable of regeneration since they are peripheral nerves, the anterior horn cells are easily irreversibly injured.

Five cases were thought to be pure root lesions, the cauda equina alone being involved. They did not make a good recovery, two remaining totally paralysed, one showing a slight improvement and one reaching the stage of walking with a caliper and drop foot stop. The residual deficit of the fifth was not ascertained. The prognosis for a root lesion in a brachial plexus injury is very bad when the root is injured close to the cord (Barnes 1949, Bonney 1959). These injuries are nearly always caused by considerable violence being applied. (Barnes 1949). They must be very

similar in nature to the root injury in a lumbo-dorsal fracture dislocation, the ends being torn apart and tending to curl away from each other, and if so there is no reason for supposing that lumbar root injuries should regenerate any more than these cervical root injuries do.

In a statistical review of 1,000 paraplegics Guttmann (1954b) reported that in a group of 239 injuries of the cauda equina 199 were left with complete paraplegia, while out of 66 cervical cord injuries only 17 remained complete. These figures show that the cord lesion has a better chance of making some recovery than the root lesion which is highly significant. It is assumed too readily that the root component of traumatic paraplegia fares better than the cord component. Active measures to promote root regeneration, such as the use of nerve cross grafts, are worth the most thorough investigation.

Involuntary movements, often associated with pain, were a distressing problem. In complete paraplegics who have not achieved an automatic bladder, conversion to a lower motor neurone type paralysis is the treatment of choice (Munro 1947, Freeman and Heimburger 1948, Bors 1951). This may be achieved by the instillation of alcohol (Cooper and Hoen 1949, Sheldon and Bors 1948) or phenol (Nathan 1959) into the sub-arachnoid space, or by anterior

rhizotomy (Munro 1945, Freeman and Heimburger 1948). If it is shown by cystometry that the bladder is atonic with excessive residual urine or is hypertonic with small capacity and frequent voiding sub-arachnoid injection is indicated (Sheldon and Bors 1948). If the bladder has some sort of useful function an anterior rhizotomy carried out from the eleventh or twelfth roots through the first sacral root is preferable. The difficulty of identifying roots is facilitated by the observation of Freeman and Heimburger 1947 that the last firm dentate ligament is a relatively constant finding at the twelfth dorsal root. Dorsal rhizotomy is not favoured (Freeman and Heimburger 1948). McCarty (1954) believes that spasms are caused by irritability of the distal cord stump and for this reason advocates cordectomy. Scarff and Poole (1946) believe also that the distal cord stump has a lowered stimulus threshold with reversal of direction in the dorsal columns. The pain which often accompanied spasms is a problem. Phantom limb pain in an amputee is not abolished by subsequent traumatic severance of the cord (Cooke and Druckemiller 1952). Even the addition of anterior rhizotomy in two of their patients did not relieve the symptoms of phantom limb.

Pharmacological relief of painful spasms is seldom obtained without resort to heavy sedation. Curare is of no benefit (Cooper and Hoen 1948, James and Broden 1946, Kuhn and Bickers 1948). Continuous spinal and caudal anaesthesia introduces too great a risk of infection (Freeman and Heimburger 1948). Some experimental evidence has been presented (Brooks and Koizumi 1953) that some drugs of the tranquilliser group, and in particular 'mephanesin', are of benefit.

Treatment of spasms in patients whose paraplegia is incomplete presents a more difficult problem since it is not justifiable to convert an upper motor neurone lesion to a lower motor neurone lesion and thereby sacrifice what voluntary power the patient might have. Guttmann (1949) believes that early rehabilitation prevents spasms occurring. He writes - "if adequate care and appropriate preventive measures are instituted at an early date exaggerated reflex activity never becomes so severe." It is difficult to draw a line between adequate and inadequate early rehabilitation, and sometimes confusion arises between spasms and contractures. Joint contracture occurs with both upper and lower motor neurone paralysis and can be prevented by the appropriate physiotherapy. Spasms, or involuntary movements only occur with upper motor neurone

lesions and some of the patients who were afflicted with them in this series had active physiotherapy from the start.

Spasms usually commence in the first week and become increasingly more severe for a time thereafter. Incomplete paraplegics were not demonstrably more prone to extensor spasms than flexor spasms as is often stated (Head and Riddoch 1917; Fulton 1943, Walshe 1959, Brain 1955). The observation (Kuhn 1949) that the final stage of both complete and incomplete paraplegics who are afflicted is one of extensor spasms was not confirmed. This study is not sufficiently detailed in this respect to allow any pattern of involuntary movements to be detected. The inhibitory effect of inter-nuncial neurones (Eccles 1953) has prompted the observation (Denny-Brown 1960) that increased reflex activity is due to damage to inter-neurones in the cord. It has also been suggested (Ten Cate 1959) that the source of spinal shock lies in inter-neurones. It is now known (Lloyd 1941) that cat cortico-spinal axons end on inter-neurones. In cases with established spasms whose paraplegia is incomplete conservative measures such as adductor tenotomy and obturator neurectomy should be tried. (Guttmann 1949). Freeman (1948) advocates sciatic nerve section with careful resuture. Munro (1959) believes

fairly long lasting relief can be obtained by infiltration of the sciatic nerve with local anaesthetic. One patient in this series treated in this manner had benefit lasting for several weeks.

d) Skin Complications

Two out of three patients in the series were found to suffer from pressure sores of more than a transient nature. This is a high incidence which has been discussed in connection with the problem of nursing these patients in a general hospital. Only in special centres does it seem possible to have a low incidence of pressure sores. The most important prophylactic measure against sores is two-hourly turning of the patient starting immediately after injury. It is very important that it be started immediately because there is a particular liability to develop sores during the initial phase of spinal shock (Holdsworth 1956).

The prognosis in lower motor neurone lesions in respect of pressure sores in the Glasgow series was also shown to be worse than that in upper motor neurone lesions. This may simply reflect a greater frequency of sensory loss as recovery is poorer in lower motor neurone lesions. Cord lesions in the phase of spinal shock are prone to pressure sores. It is not known whether loss of sensation is the sole explanation for the occurrence of pressure sores in

paraplegics or whether trophic influences play a part. It is possible that continuity between the skin and the isolated spinal cord preserves a reflex arc with tone in small blood vessels and capillaries of skin (Crawford 1930) and thereby offers some protection against pressure sores which is absent in root lesions and in cord lesions during the initial stage of spinal shock.

When sores are present it may be possible to cure them by conservative measures including local antibiotic lotions. It is particularly important that high haemoglobin levels be maintained, by blood transfusion if necessary (Guttmann 1954). Many resist all local measures. For the surgery of skin reconstruction in paraplegic pressure sores reference is made to papers by Bors, 1948, Barker et al, 1946, Gibbon and Freeman, 1946 and Conway and Griffith, 1956, Walshe, 1954.

e) Urinary Complications

The incidence of resistent urinary infection was also high in the series, 74% of those whose bladders required drainage being affected. A liability to infection is caused by retention of urine. "In all cases of acute paraplegia vesical emptying contractions are abolished and there is acute retention of urine with overflow." (Ross et al 1957). The bladder is drained by indwelling

urethral catheter (Munro 1952, Scarff 1960), or by intermittent urethral catheterisation (Guttmann 1954), or high supra-pubic cystostomy (Bors 1951, Riches 1943). The introduction of the small bore urethral catheter made of polythene (Gibbon 1958) has been shown to reduce the incidence of urethritis in the paraplegic centre at Liverpool. Catheter management is the most important factor in reducing the incidence of infection. A typical regime is that described by Cottrell and Lloyd (1961) as follows: A No. 16 Foley catheter per-urethram is used, changed weekly, and the bladder irrigated through it daily with 'zephiron'; the catheter is removed when residual urine is less than 90 ml. and there is no serious bladder infection. Even when an automatic bladder has been established these writers have shown that 84% have a-symptomatic gram negative infection with frequent flare-ups. The observation (Stebbing 1927) that pyelonephritis is the commonest cause of death in those surviving the initial complications is confirmed It is important that renal function studies, urinalysis, plasma urea, and intravenous pyelography, be carried out at regular intervals in the paraplegic's life.

Emergence of the state of automatic voiding in cord bladders may be delayed by detrusor hypertrophy and stenosis of the internal sphincter. There are various

ways of improving bladder usefulness in these circumstances (Bors 1951). These are trans-urethral resection of the bladder neck in detrusor hypertrophy in association with hypertrophy of the internal sphincter, the bladder status having been determined by cystometry, cysto-urethroscopy, and cysto-urethrography. Pudendal nerve block is preferable for isolated hypertrophy of the sphincter (Bors et al, 1950). When useless bladder function is combined with troublesome spasms the introduction of alcohol into the sub-arachnoid space is indicated. Conversion of an irritable bladder to an isolated bladder permits evacuation of urine by abdominal pressure.

The incidence of urinary lithiasis was not determined in the series. In a review of 700 patients Freeman (1949) found an incidence of calculus of 23 to 35%, and attributed this to prolonged recumbancy. On the other hand Soule (1945) believes there is a calcium disturbance of neurogenic cause.

f) Abdominal Complications

Symptoms of dyspepsia and diarrhoea were present in several cases in the absence of any demonstrable organic disease of the gastro-intestinal tract. In three cases a negative laparotomy was carried out. Visceral pain in

paraplegics is well recognised (Davis and Martin 1947). This study shows how difficult it is to exclude organic disease of viscera in the presence of visceral pain. One patient with dyspeptic symptoms was admitted to hospital as an emergency with haematemesis, and two patients with diarrhoea passed melaena stools. These patients re-inforce the view (Guttmann 1963) that small ulcerative lesions or erosions of the gastro-intestinal tract occur in paraplegics. Gastro-intestinal ulceration following lesions in the base of the brain in experimental animals has been long known (Schiff 1867) and extensively investigated (Sheehan 1940). Evidence suggests that the gastro-intestinal phenomena are the indirect result of the many chemico-physical processes occasioned by hypothalamic ablation (Keller 1960). In spinal cord injuries abnormal chemico-physical processes are set up (Cooper and Koen 1952). In the early stages, there is catabolism of body proteins, creatinuria, impaired liver function, low basal metabolic rate and eosinopenia and in the later stages, testicular atrophy, gynaecomastia, infertility, prostatic atrophy and altered excretion of 17 keto-steroids.

Although biochemical changes in this series were not reported extensively such ouvert manifestations of metabolic disturbance as gynaecomastia, oedema, particularly

of the penis and lower limbs, and spontaneous calcification in soft tissues were observed. It may be that spinal cord injury interferes with hypothalamic impulses regulating the normal internal body environment which are mediated by spinal cord neurones. In this way it leads to abnormal physico-chemical processes which result in gastro-intestinal erosions. Spinal cord disease may have the same effect, and it has been shown (Brain 1955) that there is a greater incidence of gastro-duodenal ulceration in tabetics than in the population at large. There is a growing realisation of the importance of the central nervous system in metabolic disease (Walshe 1958). Spontaneous calcification in paraplegics is evidence of biochemical calcium imbalance, a condition called "neurogenic ossifying fibro-myopathy" (Soule 1945). There is some evidence (Bors et al 1950) that changes in sex organs in paraplegics, in particular tubular atrophy of the testis, are neurogenic in origin and not endocrine.

It will sometimes happen that co-incidental abdominal disease will give rise to a surgical emergency which, in a paraplegic patient, passes undetected. This did not happen in any of the cases reported the problem being the other way round, paraplegic pain simulating abdominal disease. Should perforation of a diseased viscus occur in a paraplegic, shoulder pain (Guttmann 1963) and

conversion of spasticity from extensor to flexor pattern (Bors 1963) are most useful guides.

5. CONCLUSION.

The results of treatment of spinal cord injuries in Glasgow, where there is no special centre, differ unfavourably from results reported from Spinal Injuries Centres in other parts of the country.

Surgical decompression of the cord is of limited value in acute cases.

Root lesions do not have a better prognosis than cord lesions as they are usually stated to have.

Gastro-intestinal symptoms in paraplegic patients may give rise to an erroneous diagnosis of intra-abdominal disease.

PART TWO

OBSERVATIONS ON THE PATHOLOGY OF THE INJURED SPINAL CORD.

1. INTRODUCTION

There is difference of opinion about the interpretation of some of the changes which take place in the spinal cord when it is injured. What is meant for instance, by concussion of the cord? Does oedema of the cord contribute to the paralysis? Does haematomyelia occur? What is the role of compression? Is there any attempt at regeneration?

(a) Concepts of Concussion.

By concussion of the cord Holmes (1915) meant the concussive effect of a bullet which set up asynchronous oscillations in the cord as it passed through nearby tissues and caused structural changes in the cord in the vicinity of its path and at a distance. Davison (1943, 1960) distinguished on pathological grounds the two entities, concussion and contusion. Thorburn (1919) and Thompson (1923) maintained that the structural changes in the cord were caused by a concussive effect described by the former as a "divulsive wave" and by the latter as a "vibratory Wave." Before structural changes in the cord were demonstrated, it was held that permanent functional deficit could be present in the absence of

pathological change, the molecular concussion of Erichsen (Davison 1960). In clinical usage concussion of the cord describes a transient state with widespread symptoms of a paralytic type which clear up quickly leaving no evidence of structural damage and is the counterpart of concussion in head injuries.

(b) Concepts of Oedema.

Acute traumatic swelling and softening of the cord is usually referred to as oedema and thought by some (Allen 1914, McVeigh 1923, Riddoch 1927, Taylor 1929, Freeman and Wright 1953, Schneider 1954, Scarff 1960), to contribute to the neurological deficit in the initial stages, Riddoch actually attributing spinal shock to oedema. Surgical decompression by making an incision into the cord, myelotomy, has been advocated (Allen 1914) but has never found favour in surgical practice, the contents of the swollen cord herniating through the incision (McVeigh 1923). Decompression by the operation of laminectomy within the first day or two has been advocated by some for an increasing number of cases (McGravey 1945, Wannamaker 1954, Schneider 1954), and condemned by others (Guttmann 1954).

Medical decompression by intravenous infusion of hypertonic solutions such as 50% glucose (Scarff 1960), and 30% urea (Rand and Crandall 1962), has been suggested.

(c) Concepts of Haematomyelia.

The central part of the cord is sometimes replaced by what appears to the naked eye to be haemorrhage, and the appearance described as haematomyelia of the cord (Riddoch 1927, Davison 1943, Schneider 1954, Brain 1955, Scarff 1960). Haematomyelia describes a condition of spontaneous haemorrhage into the central parts of the cord, whereas the soft haemorrhagic mass seen after injury to the cord is produced by extravasation of blood into softened necrotic cord tissue and is therefore incorrectly called haematomyelia (Holmes 1915, McVeigh 1923, Baldwin 1934, Blackwood 1958, Walshe 1958).

(d) Concepts of Compression.

Compression probably does not play an important role in acute spinal cord injuries (Thorburn 1919, Thompson 1923, Jefferson 1927, Scarff 1960). It may be responsible fof late progression of paraplegia (Riddoch 1927, Kuhn and Macht 1947, Scarff 1960), or for the

onset of paraplegia in patients whose initial trauma passed unremarked and left no neurological deficit (Schneider 1959).

(e) Concepts of Regeneration.

It has been written "we must accept as final the statement that regeneration of the human spinal cord does not occur" (Thompson 1923). Occasional surgical and pathalogical evidence of attempts at regeneration in the human cord has appeared in the literature (Cadwalder 1920, Davison 1943), and there is growing experimental interest in comparative regeneration studies (Windle 1956).

Histological sections from 8 spinal cords removed 1 hour, 2 days, 5 days, 7 weeks, 14 weeks, 2 years, 4 years and 5 years are presented. The principal features examined are changes in axis cylinders and neuroglial cells. The manner in which these changes bear on the theories of concussion, oedema, haematomyelia, compression and regeneration is discussed.

2. MATERIAL.

The material is in two parts. The first part comprises six cords from the Mayo Clinic, Rochester, U.S.A. The second part comprises two cords from patients in the clinical review who came to post-mortem in the Royal Infirmary, Glasgow.

(a) Mayo Clinic Specimens.

At post-mortem the spinal cord was removed by pulling it upwards through the foramen magnum, the roots having been severed in blind fashion by a long narrow knife passed down the canal from above. Histological specimens of approximately one hundred cords were examined. The sections of six of these are presented to show some pathological changes 1 hour, 2 days, 5 days, 7 weeks, 14 weeks and 5 years after injury. Staining is by the Bodian silver technique (Bodian 1936).

Case 1.

Female 61 years. Died within one hour of an automobile accident. Death was caused by head and visceral injuries. At post-mortem the odontoid process was fractured and there was some softening of the cord at this level. Sections through this area of softening show most of the

axis cylinders to be well preserved but one or two resemble burned string (Plate 1.) They have disintegrated, the normally smooth contour is replaced by a series of ragged saccular swellings, and the axon cytoplasm has lost its homogeneous avidity for silver stains, is attenuated and granular, and tends to be concentrated at the rim of the saccular swellings. There are one or two small haemorrhages and a microscopic fissure in this zone.

Case 2.

Male 14 years. Died two days after sustaining a fracture dislocation of the cervical spine.

At postmortem the cord was greatly softened for several centimetres at the level of the bone injury. In this zone there is extensive structural damage (Plate 2). Few recognisable axons can be made out, the majority having disintegrated and the cytoplasm extruded to give an amorphous background appearance of fine granular debris. Scattered throughout the field are cystic spaces which give the appearance of a sieve to the tissue. These spaces are formed by the remains of axon sheaths assuming a ring shape. There is a mild infiltration with small dark round cells and no

evidence of erythrocyte extravasation.

Case 3.

Male 50 years. Died 5 days after a hyperextension injury to the cervical spine which was the seat of pre-existing spondylosis. At postmortem the cord was pulped and blood-stained over several cervical segments. The junction between the pulped and adjoining cord is fairly abrupt (Plate 3). There is a fissure separating the two zones. On the pulped side little structural organisation can be made out and there is an extensive erythrocyte extravasation which is not shown in the figure. On the healthy side the first millimetre or so has been compressed and the axis cylinders are distorted and exhibit evidence of degeneration of axon stumps, namely, giant sterile globes, coarseness of axon cytoplasm and loss of homogeneous avidity for silver stains.

Case 4.

Male 18 years. Died 7 weeks after a fracture dislocation of the upper thoracic spine.

At post-mortem the cord had been transected and the severed ends were somewhat swollen. In these swollen stumps numerous terminal axon figures were seen and

there was an increase in the number of glial cells. In one stump, about one centimetre from the transection, a bundle of slender axons in relation to the pia-arachnoid is seen (Plate 4). These axons exhibit frequently occurring smooth fusiform swellings, and oval nuclei, the cells of Schwann, are disposed alongside them. Their cytoplasm is fine and the reticulum of neurofibrils can almost be made out. As soon as cord tissue is encountered these axons are arrested and several terminal axon figures are seen at the junctional zone. The cord has been moderately damaged at this level, axons being attenuated and ragged, and there being a moderate increase in the number of darkly staining neurolglial cells.

Case 5.

Male, 62 years. Died 14 weeks after a lumbo-dorsal fracture dislocation. At post-mortem the cord was slightly indented but otherwise appeared grossly normal. In the area of the lesion moderate axon destruction has taken place (Plate 5). Although a number of axons appear to have survived, there is a background of fine amorphous debris suggesting that extrusion of cytoplasm from a number of ruptured axons

has taken place. A few poorly delineated cystic spaces can still be made out. A prominent feature is the proliferation and alteration of glial cells. The cytoplasm of these neuroglial cells is enlarged and stains well with the silver stain and the nucleus is eccentrically placed and in some cases double. These appearances constitute active neuroglial scarring of the cord, the giant cell neuroglial types being known as gamestate cells.

Case 6.

Male, 27 years. Died 5 years after multiple fractures of the lower thoracic vertebrae.

At post-mortem the cord was shrunken, compressed, and reduced to a thin ribbon of white tissue surrounded by a dense connective tissue scar of the menninges. The cord remnant can still be made out and is the seat of advanced glial scarring (Plate 6). A few axons appear to have survived. The amorphous debris from ruptured axons has been incorporated into the neuroglial reticulum, and giant-cell neuroglial types are no longer seen. There is luxuriant deposition of collagen in menningeal connective tissue around the gliosed cord but this has not penetrated the cord, the demarcation

between cord and meningeal scar being abrupt.

(b) Glasgow Royal Infirmary Specimens.

The two cords in this group came to post-mortem 2 and 4 years after injury respectively. The manner of removing the cord was to lift it out of the spinal canal after sawing off all the lamina and dividing the roots.

Case 7. (No. 39 in the clinical series)

Male, 61 years. Died two years after a hyperextension injury to the cervical spine which was the seat of spondylosis. Incomplete quadriplegia with a level at C7-8.

At post-mortem no abnormality could be detected in the cord, naked eye. Just caudal to the level of the clinical lesion there is secondary demyelination of the lateral columns (Plate 7), and destruction of neurones with loss of definition of grey matter structure (Plate 8). In the demyelinated zone the axis cylinders are well preserved (Plate 9). In the myelinated dorsal columns and the deeper, myelinated parts of the lateral columns there are focal patches of light collagen deposition, and in their vicinity

clusters of short axis cylinder segments which seem to radiate from small cystic spaces (Plate 10). Cranial to the lesion, in the upper cervical cord, there is secondary demyelination in the dorsal columns and, to a lessor degree, in the lateral and antero-lateral columns (Plate 11). Neurones are in a fairly good state of preservation but there is loss of definition of supporting tissue (Plate 12). In the demyelinated dorsal columns some axons appear to have fallen out of their sheaths, which are themselves swollen, leaving cystic spaces but the cytoplasm of axons is still evident in many other sheaths (Plate 13). Neuroglial cells in this area are increased in number, and avidity for silver stain, and have protoplasmic processes or feet which make up a dense neuroglial reticulum.

The lumbar cord exhibits a small wedge of secondary demyelination in the lateral columns (Plate 14). Neurones are in a relatively good state of preservation but there is loss of definition of grey matter structure (Plate 15). Axis cylinders are preserved in the demyelinated area in the lateral columns where also can be seen many darkly staining neuroglial cells with protoplasmic feet (Plate 16).

Case 8 (No. 7 in the clinical series)

Male, 26 years. Died four years after sustaining a fracture dislocation at the lumbo-dorsal junction. Complete paraplegia.

The compression of the cord can be seen with a band of demyelination extending cranially from the median parts of the dorsal columns and some demyelination of the lateral columns (Plate 17). The cord was twisted upon itself immediately cranial to this and part of the longitudinal section does not pass through cord tissues. Immediately adjacent to the compression neurones exhibit degenerative changes while the reticulum of axis cylinders in the grey matter is well preserved (Plate 18). Axis cylinders are in a good state of preservation in the partially demyelinated lateral columns, and a few large argentophil neuroglial cells with protoplasmic feet can be seen (Plate 19). Further laterally several bands of wavy collagen have been deposited causing axis cylinders in their vicinity to assume a corresponding wavy form (Plate 20).

Extensive necrosis is seen in the cauda equina where only a few myelinated nerve bundles have survived (Plate 21). There is hypertrophy of endothelium in a

capillary, the swollen endothelial cells assuming a glassy appearance (Plate 22).

The first 6 cords exhibit varying degrees of axis cylinder damage and glial scarring. The process of disruption of axis cylinders and extrusion of axis cytoplasm is seen in the early stages. There is evidence of degeneration in axon stumps in the cord and failure of a growing root to penetrate the cord later on. Still later established glial scarring of a cord with abundant connective tissue round about it is seen. The remaining two cords show glial scarring, late appearances of grey matter and secondary degeneration of axis cylinders and myelin sheaths.

3. DISCUSSION.

(a) Concussion.

The appearances of some axis cylinders (Plate 1), which were described as resembling 'burned string', indicate irreversible damage to them (Cajal 1928). Their isolated occurrence shows a remarkably selective

action by the traumatising force. They have been singled out for destruction. It is impossible to explain these appearances as a secondary effect of compression, or ischaemia, or oedema, for they are present within one hour of injury. They can be explained by the conception of injury to the cord setting up vibration stresses (Holmes 1915, Thorburn 1919, Thompson 1923), and may be taken as pathological evidence of concussion of individual axons, (Holmes 1915, Brain 1955, Davison 1960). They are seen both locally and at a distance (Holmes 1915, Davison 1960). They re-inforce the view that neurological deficit is always accompanied by structural change, both in head injuries (Strich 1961) and cord injuries (Davison 1960, Bailey 1960). The older theory of 'molecular concussion' which implied permanent functional deficit in the absence of any structural change, is not entirely invalidated as it has been suggested that the basis of these structural changes may be a chemical disturbance of molecules (Hassin 1944, Davison 1960).

The same changes can be seen to a much more extensive degree in Plate 2. Throughout the field axis cylinders have broken up and appear to have been shaken out of their sheaths giving the appearance of a sieve

to the tissue. Even this more severe degree of concussion is not associated with much cellular reaction, although there is some increase in the number of small dark round cells as compared to Plate 1.

(b) Haematomyelia.

When the cord is crushed by direct violence the pathological changes seen are those of contusion. The affected area of the cord is pulped, the consistency being that of custard (Holmes 1915), and there are no recognisable neural elements. An acute cellular reaction occurs comprising polymorphs, round cells, and a large phagocytic cell known as a compound granular corpuscle or 'gitter cell' which is probably derived from microglia (Bailey 1961). The pulped area also contains numerous erythrocytes and tends to be located in the central parts of the cord because, as McVeigh(1923) showed experimentally, the vascular and neural framework supporting the cord is weakest in this area. Because of its central situation and superficial resemblance to a haemorrhage, this blood stained soft necrotic tissue has been called haematomyelia by some writers (Riddoch 1927, Davison 1943, Schneider 1954, Brain 1955, Scarff 1960), an error which has been exposed by others (Holmes 1915,

McVeigh 1923, Baldwin 1934, Blackwood 1958, Walshe 1958). It expands longitudinally as a 'flask shaped' area of softening (Holmes 1915) pushing normal tissue before it, the demarcation between pulped and healthy cord being quite abrupt (Davison 1960). A peculiar feature of the acute cellular reaction, namely that it does not progress to healing by neuroglial or connective tissue scarring, has earned it the name "sympathetic inflammatory reaction of Spielmyer" (Baldwin 1934, Davison 1960). Instead, the contents are absorbed to leave a fluid cyst which resembles the central cavitation of syringomyelia (McVeigh 1923, Thompson 1923, Schneider 1959, Scarff 1960). The edge of a contused area can be seen in Plate 3 with a sharp demarcation from compressed healthy cord.

(c) Oedema.

Concussion and contusion lead to extrusion of axon cytoplasm as the axis cylinders are ruptured. The cytoplasm of axons is unusually fluid, in some invertebrates being so fluid as to be extruded whenever the axon membrane is traumatised (Young 1934). Normally axons are bathed in hypertonic fluid (Porter 1959). In consequence

concussion and contusion are accompanied by swelling and softening of the cord. This is facilitated by the occurence of scattered small haemorrhages, which are frequently seen in cord injuries (Holmes 1915), Riddoch 1927, Taylor 1929, Scarff 1960), and microscopic fissuring which is also characteristic of trauma (Cajal 1928). It is this swelling and softening which is often referred to as oedema, a term which should be reserved in the central nervous system for a "diffuse yellowish discolouration of the white matter in fresh post-mortem specimens" (Greenfield 1942). Although many authors (Allen 1914, McVeigh 1923, Riddoch 1927, Taylor 1929, Freeman and Wright 1953, Schneider 1954, Scarff 1960) hold the view that oedema is a secondary event and is responsible for part of the neurological deficit in traumatic paraplegia, it is really only the outward sign of damage to axis cylinders and should be regarded as a primary event as Holmes (1915) emphasised. An analogy may be drawn between oedema in cord injuries and oedema in burns. Extrusion of fluid into the tissue spaces is a feature of burns but the residual skin defect is determined by the severity of the damage sustained by the skin at the instant of burning and not by the extent

of oedema, and is not lessened by therapeutic measures to relieve oedema. The exception to this analogy are those rare cases of traumatic softening of the cord caused by the ischaemic effect of compression.

(d) Compression.

Compression of the cord in acute traumatic paraplegia is far less common than often supposed (Scarff 1960). Thorburn (1919) pointed out that the damage to the cord was of a nature that could not be explained by compression and this is supported by the material presented in this study. Thompson (1923) and Jefferson (1927) stated that compression never occurred, the former pointing out that the spinal canal was a very roomy compartment. Various authorities describe a delayed ischaemic softening which occurs one or two days after injury and is due to compression upon the cord (Brain 1955, Walshe 1958, Davison 1960). It is widely accepted that an acute disc protrusion compresses the anterior spinal artery and produces the syndrome of acute anterior spinal cord injury (Schneider 1955), pathological change in anterior cord compression being found in the lateral columns due to the tethering effect

of thedentate ligament (Kahn 1947). Brain and Walshe and Davison have said that there is an element of ischaemic softening in many other cord injuries. The production of compression myelopathy by bleeding into the extra-dural space, haematorachis, can be ruled out (Thompson 1923, Riddoch 1927, Davison 1960). Softening in the hyperextension injury to a spondylotic spine was produced by gross necrosis and not compression as illustrated by Case 3. Vascular changes in the injured cord are minimal (Holmes 1915, Baldwin 1934, Hassin 1944), and Holmes stressed that he never encountered an instance of thrombosis of the anterior spinal artery. The spinal arteries are not prone to thrombosis either as a result of trauma or disease (Blackwood 1948). They are not affected by atherosclerosis or hypertensive disease (Staemb1 1939). The main blood supply to the cord is via segmental arteries each one of which supplies two or three adjacent segments (Wollam and Millen 1958). There are two especially large such arteries supplying the cervical and lumbar enlargements of the cord. It has been suggested (Suh and Alexander 1939) that as a consequence of this arrangement the thoracic cord has a

relatively poor supply and is more prone to softening in acute injuries of the thoracic spine but it also happens that the spinal canal is narrowest in this region and the cord more cramped and therefore liable to damage. Acute spontaneous myelitis has been ascribed to ischaemia arising from anterior spinal artery thrombosis (Penny-backer 1958) but the rapidity with which these lesions sometimes clear up does not support this aetiology. Ischaemic damage to the cord has been reported following clamping of the abdominal aorta at operation (Hara and Lipin 1960), but considering the number of times this procedure is carried out without causing paraplegia it must be a very rare occurrence, DeBakey and Cooley (1960) never having encountered a case. Myelopathy following aortography has also been attributed to ischaemia but when all the reports of this happening were investigated (Killen and Foster 1960), in no single instance was there evidence of anterior spinal artery thrombosis or damage or thrombosis of segmental arteries to suggest a vascular cause for the appearances of softening which were a characteristic finding at post-mortem. From the point of view of treatment it is important to establish for a fact whether ischaemia contributes to the picture in

acute traumatic paraplegia for modern therapy can do something in this field by, for example, the exhibition of hyperbaric oxygen (Illingworth 1962).

The appearances shown in Case 8, where the cord was actually indented, suggest that the underlying axis cylinders are not sensitive to compression while the underlying neurones are.

Prolonged compression upon the cord may lead to pathological changes and paraplegia. Injury may bring this about in three ways. In the first place some unremarked trauma or occupational trauma may lead to degeneration of the intervertebral disc and compression of the anterior parts of the cord by it, the syndrome of chronic anterior cord compression (Schneider 1959). This was not encountered. Secondly, the injury may lead to compression by the development of bone deformity (Scarff 1960). Distortion of the cord was produced in this way in Plate 17. Thirdly, compression may be produced by meningeal scarring variously termed meningitis circumspecta serosa, post traumatic arachnoiditis, adhesive arachnoiditis. Meningeal scarring may follow severe trauma or arise in consequence of compression of spinal arteries (Davison 1960). Surgical removal of this

scar has found favour with some authors (Riddoch 1927, Taylor 1929, Freeman and Heimburger 1948) and been condemned by others (Kuhn and Macht 1949, Guttmann 1954). Although it may be produced by ischaemia it is difficult to imagine these meningeal changes themselves leading to ischaemic damage of the cord and they do not penetrate the cord.

(c) Regeneration.

It has been suggested (Sugar and Gerard 1940) that ischaemia may be a factor preventing regeneration of nerve fibres in the cord, many of Cajal's regeneration studies having been performed in the caudal part of the cord after transections below the large lumbar segmental artery which would prejudice its blood supply. There was no evidence of regeneration in the material examined for this study. The characteristic appearances of unsuccessful regeneration of axon stumps are spheres and giant sterile axon balls, coarse axon cytoplasm and loss of homogeneous avidity for silver stains, and the absence of cones of growth or bifurcation of axons (Cajal 1928). Some of these appearances are illustrated in Plate 3 at the boundary zone between pulped and healthy cord.

The essential features of a regenerating axon are the fine neuroplasm with its reticulum of neurofibrils, the presence of a cone of growth at the tip of the axon, and bifurcation of the axon (Cajal 1928). It is not always easy to distinguish between degeneration and regeneration, particularly when there is no obvious loss of cord continuity. Davison (1943), who claims that newly formed axons and signs of regeneration are seen, bases his claim on pictures which are compatible with his own description of degeneration.

There is evidence of the failure of a root to penetrate the cord substance in Plate 4. The young slender argentophil axons of the root, with the oval cells of Schwann disposed alongside them, are arrested as soon as the meninges have been penetrated and there are one or two terminal axon figures at the junctional zone. The cord is the seat of some glial change but there is no evidence of an acute cellular reaction. Experimental evidence has suggested that the presence of an inflammatory reaction favours regeneration of axons (Clark 1943) and the presence of astrocytes inhibits it (Windle 1950). Is the presence of gliosis and the absence of inflammatory reaction linked in some

way with the arrest of growing root fibres in this case?

Proliferation of glial cells is a response to injury of the cord, astrocytes increasing in number, their cytoplasm becoming abundant, and more avid for silver stains and more than one nucleus sometimes being seen (Cajal 1928). These changes may be well advanced by two weeks (Davison 1943) but generally take several weeks to be well developed, and exemplified in Plate 5, the cord removed at 14 weeks. It is noticeable that glial changes or gliosis as it is called, is associated with an intermediate degree of axon damage and especially, as Holmes pointed out, where the axon sheaths are swollen, as in Plate 13. Gliosis does not occur where severe contusion of the cord with pulping has occurred, these areas becoming a fluid cavity. Gliosis has been described following a period of ischaemia (Scarff 1960) but it is of limited occurence in compression myelopathy (Davison 1960). Glial changes were prominent in the areas where secondary demyelination had occurred in Plate 13. The location of secondary demyelination in this cord clearly indicates ascending and descending long tract degeneration. It has been said (Walshe 1958) that secondary demyelination only takes place when axons have been

covered, and it is not known whether normally myelinated axons can conduct when demyelinated (Waelsch 1959). The occurrence together of argentophil axis cylinders and demyelination of the sheaths suggests either that the axons are degenerate but have not undergone Wallerian degeneration or phagocytosis, or that they are viable in spite of having no myelin sheath. The former explanation is considered more likely. It seems inconceivable that the scarred cord in Plate 6 for example should still contain conducting fibres and this patient was completely paraplegic. Is it possible that the glial cell reaction in the cord has something to do with the histological preservation of non-viable axons as well as the failure of peripheral nerve fibres to penetrate it? It is also noteworthy in Plate 6 that the exuberant connective tissue scarring of the meninges has failed to penetrate the gliosed cord remnant. It is almost as if gliosis puts the cord into cold storage, dead axons being preserved and penetration by connective tissue and nerve tissue blocked. There is no progressive neuroglial reaction in the cauda equina (Haasin 1944) and regeneration can be anticipated here as in a peripheral nerve (Holdsworth 1956). Cajal (1928) has said that neurones

within the central nervous system have regeneration potential but are prevented from doing so by some factor. Could the factor be the reaction of neuroglial cells? It is a coincidence that the time when growth of axon stumps might be expected to be seen, around 14 days, is also the time when neuroglial cell reaction is getting under way.

Deposition of collagen was mostly confined to mesenchymal tissue either in the meninges or around pial tissue in the cord substance. Some collagen deposition was seen in Plate 10 where it had some effect on the orientation of axis cylinders, and sub-pial collagen was seen in Case 8 where it caused adjacent axons to assume a wavy form. It is assumed that the contractile nature of collagen is responsible for these changes in nearby axis cylinders.

4. CONCLUSION.

Concussion is caused by the axon sheath being traumatised.

Oedema is produced as the axon cytoplasm extrudes through the damaged sheath.

Haematomyelia does not occur, and compression

plays an insignificant role after the initial traumatising force has passed.

There is no regeneration of the cord and this may be linked in some way to the occurence of changes in neuroglial cells.

PART THREE.

BLOOD PRESSURE RESPONSES EVOKED BY EXPERIMENTAL COMPRESSION OF THE SPINAL CORD IN CATS.

1. INTRODUCTION.

The Relevance of the Experiment to Spinal Cord Injury in Human Beings.

Transection of the cord at any cervical level causes an immediate and profound fall in arterial blood pressure which is later maintained at normal levels (Bernard 1863). If the isolated thoracolumbar cord is destroyed, arterial pressure falls permanently to spinal shock levels (Goltz 1874). The isolated thoracolumbar spinal segments are therefore capable of initiating a tonic vasoconstrictor activity. Patients who have sustained physiological transection of the cord are able to maintain their blood pressure at normal levels for this reason. They are not always completely normal in this respect. Some are subject to postural hypotension (Guttmann 1946, 1953, 1963: Koster and Bethlem, 1961), others to episodes of autonomic hyperreflexia (Head and Riddoch 1917, Learmonth 1931, Guttmann and Whitteridge 1947, Thompson and Witham 1948, Pollock 1951, Bors and French 1952, Schiebert1955, Mannion, Cottrell and Lloyd 1959), the full picture of which comprises dyspnoea,

sweating, palpitation, headache, a slowing of the pulse and rising of arterial blood pressure.

A relationship was observed between the spinal cord and blood pressure during some studies of spinal cord compression in cats. Some experiments to elucidate this relationship were performed and are reported.

2. MATERIALS AND METHODS.

(a) The Experimental Preparation.

Thirty-six adult cats were studied. Material from eighteen is not included as the data obtained from them was considered unreliable. Anaesthesia was by intraperitoneal pentobarbital sodium (30 mg. per kilo). The animals were intubated endotracheally or by tracheostomy and ventilated with air by a small pump when necessary. Succinylcholine (anectine 20 mg. per ml.) was given intravenously to rule out any responses which might be associated with movement at the time of cord compression. Arterial blood pressure was recorded from a No. 19 polyethylene catheter placed in the abdominal aorta via the femoral artery and connected to a Statham Pressure Transducer (0-15 psig) which in turn was connec-

connected to a Fisher Galvinometric Recorder. A number of laminectomies were performed, at all levels and of varying lengths so that tunnels of the spinal canal containing single and contiguous segments of cord were created. A slender elongated balloon made of latex and containing air was linked by a three-way tap to a mercury manometer and a syringe containing air (Kerr 1963).

(b) Procedures Carried Out.

The balloon was placed in these tunnels, both intradurally and extradurally, between the cord and the bone and compression of the cord obtained by raising the pressure in the balloon to levels of 50 mm.Hg, 100 mm.Hg, 200 mm.Hg, and higher. Periods of balloon inflation ranged from 5 to 25 seconds and, on occasion, up to two minutes. Responses from different levels were noted.

Various procedures were carried out in an effort to determine the factors responsible for the pressor activity which was elicited and the role played by certain vasomotor reflexes. These procedures included bilateral denervation of the carotid and aortic sinuses, bilateral division of the vagus nerve high in the neck,

bilateral adrenalectomy, decerebration at the level of the superior colliculus under ether anaesthesia following which the anaesthetic was discontinued, transection of the cord rostral to the area to be compressed, injection of the cord with 2% Novocaine, perfusion of upper thoracic segments of the cord with autologous blood or saline under pressure via a corresponding intercostal artery, and deafferentiation of the upper thoracic cord by bilateral division of the upper three dorsal roots four days prior to the experiment.

3. RESULTS.

(a) Spinal Cord Presser Responses.

Compression of the cord evoked a transient rise in arterial blood pressure the principal characteristic of which was an abrupt rise in mean systemic arterial pressure sometimes by as much as 100 mg.Hg., after a latent period of some five or six seconds (Figure 1). After a short period, usually less than a minute, blood pressure quickly fell to previous levels, sometimes interrupted by a momentary rebound or a short plateau. Bradycardic pulses of large amplitude occurred during

the response from upper thoracic levels of the cord. Maximum response lasted approximately 45 seconds regardless of whether the cord was compressed for a short or long period. No difference was observed between intradural and extradural compressions. As the animal underwent a generalised spasm when the cord was compressed, succinylcholine was administered beforehand and some few minutes allowed to elapse to allow a transient rise in arterial pressure which this drug produced in the doses used, to settle. Abolition of muscle tone in this way did not affect the blood pressure response to cord compression in any observable way. Intravenous injection of a small quantity (approximately 0.2 ml) of a 1:1000 solution of adrenaline chloride evoked changes in blood pressure which were very similar to maximum responses obtained by cord compression (Figure 2.)

(b) The Effect of Varying Degrees of Compression.

Rises in mean blood pressure tended to be proportional to the degree of cord compression within limits (Figures 3 and 4). Above compressions of 200 mm.Hg., or 250 mm.Hg. no greater rise in blood pressure was obtained.

(c) The Effect of Varying Levels of Compression.

Differences in the magnitude of the response were observed when different segments of the cord were compressed (Gifure 5). The greatest rises were obtained from the upper thoracic cord and the occurrence of bradycardic pulses of large amplitude only observed from compression of the upper five thoracic segments. The cervical cord evoked a lesser response, the thoracolumbar junction a still smaller response, and the lower thoracic area the least response. The response was not affected by the number of cord segments being compressed at any one time, a maximum response being obtained from compression of single segments.

(d) The Effect of Fatigue.

After a small number of compressions the preparation deteriorated, further compressions requiring to be of greater magnitude until a response could only be obtained by squeezing the cord with forceps. At the same time base levels of blood pressure fell to very low levels. This condition proved to be reversible in some cases by leaving the animal undisturbed for a while.

A similar degree of unresponsiveness was observed when the animal was deeply anaesthetised.

(e) The Source of the Response.

Compression of the cord after deafferentiation in the manner described did not affect the magnitude of the response but return of blood pressure to previous levels took much longer (Figures 6a and b). Bradycardic pulses of large amplitude were still present although they occurred earlier in a second compression without any material change in the preparation being made. Mechanical stimulation by compression of two dorsal roots bilaterally in another intact animal evoked a rise in blood pressure of much smaller magnitude, about 5 or 10 mm.Hg.

Perfusion of the cord with blood or saline at a high pressure (724 mm.Hg.) in the manner described resulted in small rises in blood pressure which were similar to those obtained by perfusing the same quantities at normal pressures into the femoral vein.

Bilateral sinu-aortic denervation was itself responsible for an elevation of blood pressure. When the

spinal cord of an animal subjected to this procedure was compressed, a rise in blood pressure was evoked, during which bradycardic pulses of large amplitude were no longer observed (Figure 6c).

Division of the vagus nerve high in the neck diminished those bradycardic pulses but only abolished them in one instance.

Transection of the cord at any level resulted in a sharp rise in arterial pressure similar to that described as characteristic of the response evoked by compression but the consequent fall in blood pressure was much greater, sinking to levels of 40 or 50 mm.Hg. At this point the animal was in the state of spinal shock. When transection was performed above thoracic levels, subsequent compression of the thoracic cord resulted in blood pressure rises from these low levels to as much as 180 mm.Hg. and bradycardic pulses of large amplitude no longer observed.

Bilateral adrenalectomy had no effect on the pressor response evoked by compressing the cord.

The response from a decerebrate animal was similar to that from a lightly anaesthetised animal.

Injection of 2% Novocaine resulted in a transient rise in blood pressure followed by a steady fall to very low levels. When this was repeated with saline a transient rise was noted followed by a return to previous levels.

It was observed during compression of the upper two or three thoracic segments that the animal's pupils dilated widely. This response was bilateral so long as the compression was produced in the midline of the dorsum of the cord, and was ipsilateral when the compression was placed to one side of the cord.

4. DISCUSSION.

Marked rises in blood pressure resulting from rises in intracranial tension were first described by Duret (1878), although the discovery of this important relationship is usually attributed to Cushing (1901). It was postulated by Cushing that the mechanism involved was anoxia of the brain, the resultant rise in blood pressure being due to reflex sympathetic discharge.

Blood pressure responses due to anoxia of the

spinal cord were described by Kaya and Starling (1909) by making the spinal animal breathe pure nitrogen. This was confirmed by Alexander (1945) who found that alteration in blood flow in a completely de-afferentiated segment of cord (low cervical and mid thoracic transections, sections of all intervening dorsal roots, bilateral section of sympathetic chains above and below the ganglia at level of lower cord section, and bilateral section of vago-sympathetic trunks) evoked changes in sympathetic discharge, as measured by cardio-accelerator tone. That similar changes in blood pressure may result from increase in pressure involving spinal intradural structures is not well recognised. In a review of the literature it was found that Great and Peele (1945) had been the first to report the phenomenon, confirmed by Bhargava and Kulkreshtha (1959) and Borrison and White (1955). In these reports the method used consisted of injection of saline into the spinal subarachnoid space which was isolated from the cranial compartment by a constricting ligature at the cervico-medullary junction. Increasing spinal fluid pressure by 50 to 100 mm. mercury resulted in blood pressure rises of up to 90 mm. mercury, and a proportionality was obtained between the two up to

blood pressure increases of 150 mm. mercury beyond which further rises could not be obtained and the preparation deteriorated. It was also stated that the duration of applied cerebrospinal pressure was proportional to the rise in blood pressure obtained, and a latency of 5 to 20 seconds (average 10 to 12 seconds) between application of pressure and rise in blood pressure was reported.

Most of these observations have been confirmed in this study, but a relationship between duration of cord compression and blood pressure response could not be demonstrated: in no instance could the maximum pressor effect be maintained for longer than one minute by sustained cord compression. A considerable degree of variability in response was found between different animals and in the same animal under different procedures and different levels of anaesthesia. Although the balloon fitted snugly into the spinal canal between cord and bone, and therefore allowed a measure of the relative pressure on the cord, it did not indicate the true pressure on the cord, a roomier spinal canal requiring greater degrees of inflation to evoke a pressor response.

There has been some controversy over the

mechanism by which the response is produced. Although Groat and Poole (1945) themselves believed that it was due to anoxia of the cord, Dorrison and White (1955) subscribed to the view that it was mediated by barore-ceptors in the cord. Assuming that such receptors would be stimulated by stretch or collapse of the walls of blood vessels in the cord, it would be expected that external pressure on the cord would collapse these vessels and trigger the response as in the sinu-aortic reflex and, conversely, that distension of these vessels would lead to a drop in systemic pressure. Since perfusion of the spinal branch of an intercostal artery at a pressure well in excess of normal led to a slight rise in blood pressure, this mechanism would appear quite unlikely.

Another possibility, that the response is triggered by stimulation of peripheral afferent fibres in the dorsal roots, must be considered, since it is well known that stimulation of pain afferents produces a marked pressor effect. Since 25% of severed nerve fibres have an almost normal electrical response after 72 hours (Erlanger and Schoepfle, 1946), cord compression after acute rhizotomy, as in the experiments of Dorrison and

White (1955), does not rule out a dorsal pressor effect. Preservation of the complete response in a chronically deafferentiated segment of the cord as described herein, would therefore appear to prove conclusively that the response arises from compression of the cord itself. Small rises in blood pressure of the order of 5 or 10 mm. mercury were produced by compression of the upper thoracic roots alone, indicating that peripheral nerve afferent stimulation contributed a small fraction to the pressor response.

The blood pressure rise following rise in intracranial pressure has been attributed by Rodbard and Stone (1955) to liberation of vasopressor substances into the blood stream. This conclusion is based on the latency of 10 to 12 seconds, on the demonstration that hypothermia to 25 C prolongs this latency by as much as 26 seconds, and on the fact that the rise in blood pressure associated with increased intracranial pressure has a short latency whereas a long latency is associated with the increase in blood pressure following breathing of nitrogen instead of air.

As reported here, removal of the most probable source of circulating vaso-active substances, namely the

adrenal glands, did not affect the response. Celander (1954) has shown that normal sympathetic vaso-motor tone is mediated by nervous channels, hormonal influence on blood pressure being a response to local metabolic needs of tissue. If spinal hypertension were due to circulating vaso-active substances the presence of catechol amines in the urine should be detected. Hodgson and Wood (1958) examined this hypothesis in human cases who exhibited autonomic hyperreflexia and found that the urinary excretion of catechol amines was unchanged during episodes of hypertension and also that the hypertension was reversed by ganglion blocking agents but not by adrenolytic agents. The fact that hypothermia, in the hands of these other workers, increased the latency does not necessarily support a chemical mechanism since neural conduction is considerably slowed at lower temperatures, and comparisons with the longer latency of the presser response to respiratory (nitrogen) anoxia can be interpreted quite readily by considering that the anoxic stimulus in that case is not applied instantly, as is the case with compression of the cord, but requires the passing of a number of seconds for gaseous exchange

between alveolar air and blood, circulating time and depletion of existing oxygen levels in the central nervous system. One other related finding, namely an ipsilateral pupillary response on compressing the pupillo-dilator centre in the first two or three segments of the thoracic cord to one side of the midline, implicates a nervous mechanism rather than a humeral one; a neurone pool responsible for mydriasis is situated in the intermedio-lateral column along with other sympathetic neurones (Simeone, Smithwick and White, 1952).

The latency of the response of 5 or 6 seconds suggests activation of multisynaptic pathways. The finding that localised compression of a small area consisting of a single cord segment in continuity with the rest of the cord evoked maximum responses also points to recruitment of multisynaptic pathways.

The finding that blood pressure responses were greatest when compression was performed above the mid-thoracic cord has a parallel in the findings of Guttmann and Whitteridge (1947), Thompsom and Witham (1948), Guttmann (1953), that blood pressure rises are greater

in paraplegics when cord transection lies above the mid thoracic region. This may be due to the addition of a component of sympathetic cardiac stimulation to the vasomotor response when the sympathetic outflow to the heart from the upper thoracic cord lies with the isolated segment of cord. The abolition of bradycardic pulses of large amplitude when the sinu-aortic mechanism was denervated or rostral transection of the cord performed prior to compression, points to the fact that they are part of the compensatory vasodepressor mechanism which presumably is evoked by the initial rise in blood pressure. They must be mainly sympathetic in origin since vagal section abolished them only in one instance. It would appear likely, therefore, that when the sensitive upper thoracic region of the cord is compressed there is an initial sharp rise in blood pressure which is partly due to a vasopressor effect and partly to increased action of the heart; this is quickly followed by a fall in blood pressure which is partly effected by a vasodepressor effect and partly by the reduced action of the heart, the latter being mainly a reduction of sympathetic cardiac tone and partly a vagal stimulation effect.

In paraplegics no difference has been observed between cervical and upper thoracic cord lesions in respect of blood pressure and pulse changes (Guttmann and Whitteridge, (1947) and Thompson and Witham (1948). The experiments reported here localise the sensitive area of the cord in the upper thoracic segments.

5. CONCLUSION.

There is a central regulating vasomotor mechanism within the spinal cord.

This mechanism responds to anoxia and is mediated by nervous pathways involving polysynaptic chains.

The upper thoracic region of the cord is the most responsive area, cardiac effects being obtained from this region alone.

ACKNOWLEDGMENTS

The work on this thesis was performed at the Royal Infirmary, Glasgow, and the Mayo Clinic, Rochester, Minnesota, U.S.A. I wish to thank Professor W.A. Mackoy for his stimulating interest throughout this period.

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The experimental study was performed under the guidance of Dr. F.W.L. Kerr, Mayo Clinic, to whom I am most indebted.

I wish to thank Mr. Ian Murray, Department of Medical Illustrations, Royal Infirmary, for taking the prints and mounting them, Mr. Towler; photographer, and Miss M. Smith; artist, Department of Surgery, Royal Infirmary, for assistance.

SPINAL CORD INJURIES. A CLINICAL, PATHOLOGICAL, AND EXPERIMENTAL STUDY.

VOLUME II.

by

Shedden Alexander.

Thesis presented for the Degree of Master of Surgery in the University of Glasgow.

September, 1963.

PART ONE.

A CLINICAL REVIEW OF 52 CASES OF CLOSED SPINAL CORD INJURY.

LEGENDS AND FIGURES.

FIGURE 1.

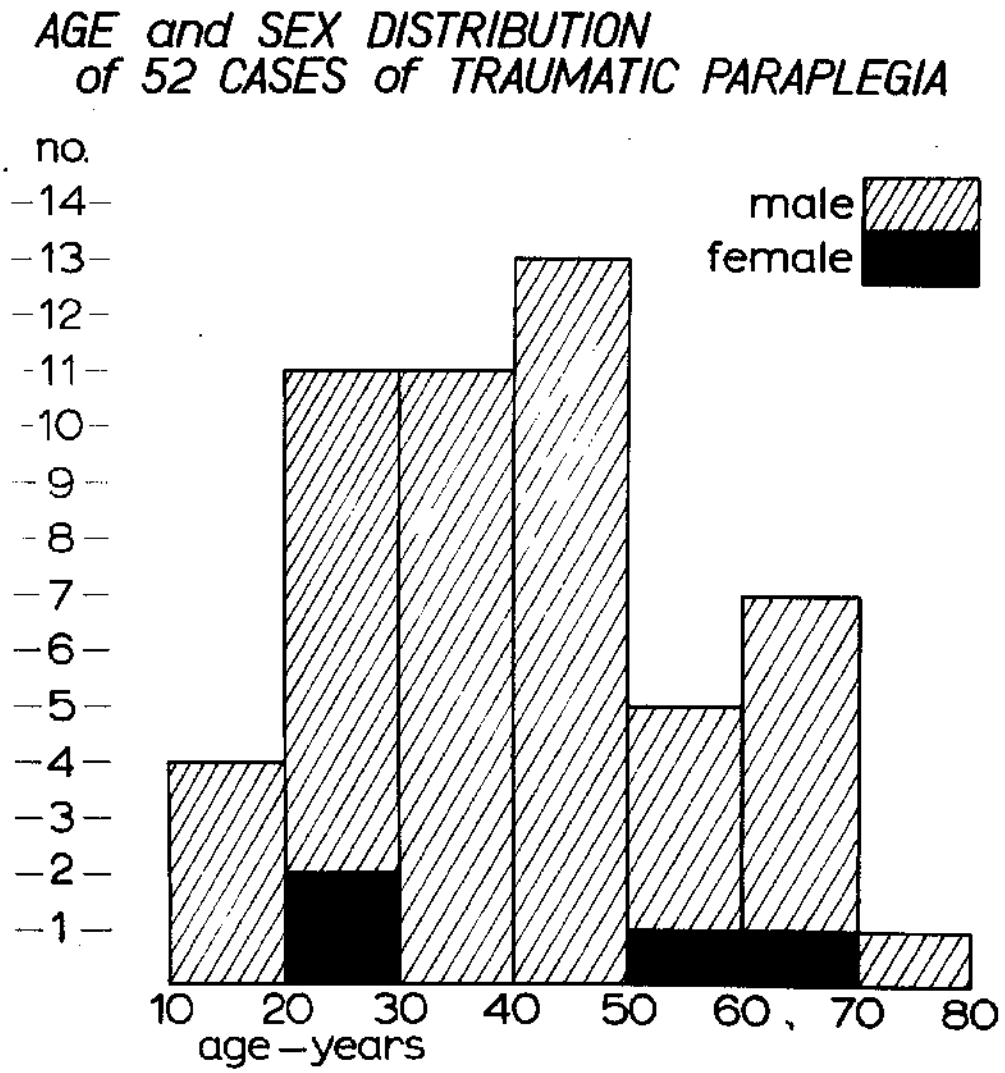

AGE and SEX DISTRIBUTION
of 52 CASES of TRAUMATIC PARAPLEGIA
no.
14
13
12
11
10
9
8
7
6
5
4
3
2
1
male
female
10
20
30
40
50
60
70
80
age—years

TABLE 1

Effect of the type of injury on the level of the spinal injury and on the early mortality in injuries of the spinal cord.

Type of Injury	Level of Spinal Injury				
	Cervical	Dorso-lumbar	Totals	Early Mortality	
Falls from a height	3	13	16	3	19%
Mining Accidents	1	12	13	0	0%
Traffic Accidents	6	3	9	6	66%
Falls when drunk	2	3	5	0	0%
Industrial Accidents	2	3	5	2	40%
Unknown	-	-	4	-	-
			52	11	21%

TABLE 2.

Relationship between the level of spinal injury, the severity of the initial neurological deficit and early mortality in injuries of the spinal cord.

Severity of Initial Neurological Deficit.		Level of Spinal Injury			Total
		Cervical	Dorsal	Lumbo-dorsal	
		15	6	31	52
Complete	Total	7	3	18	28
	Survivors	0	2	16	18
	Early Mortality	7	1	2	10
Incomplete.	Total	8	3	13	24
	Survivors	7	3	13	23
	Early Mortality	1	0	0	1
Died.	Primarily due to C.N.S. injury.	7	0	0	
	Primarily due to concomitant injury.	1	1	2	

TABLE 3

Relationship between the level of spinal injury and radiological evidence of associated injury to the vertebral column in injuries of the spinal cord.

Level of Injury to Spine		Radiological evidence of bone injury	No radiological evidence of bone injury	Initial radiology unknown
Cervical	15	8	7	
Dorsal	6	6	0	
Lumbodorsal	31	25	2	4
Total	52	39	9	4

TABLE 4

Relationship between open and closed methods of treating the spinal injury and the incidence of pressure sores and residual stiff back in injuries of the spinal cord.

Treatment of Injured spine	Cervical Spine		Dorsal and lumbar spine		
CLOSED METHODS	Survivors	Having severe pressure sores	Survivors	Having severe pressure sores	Having residual stiff back
Ordinary Bed	4	1	6	2	
Air Bed			1	1	
Water Bed			1	1	
Meccano Bed			1	1	
Complete plaster	1	1	2	2	
Plaster Shells			2	1	
Turning frame			3	3	
Closed reduction and P.O.P.			1	1	
Skull traction	1	0			
Total: 23	6	2	17	12	4 (24%)
OPEN METHODS					
Laminectomy	1	0	3	1	
Laminectomy with fusion			4	2	
Open reduction			1	1	
Open reduction and plating			7	5	
Total: 16	1	0	15	9	4 (27%)
Methods unknown: 2			2	1	
GRAND TOTALS: 41	7	2	34	22	8 (25%)

TABLE 5

Effect of laminectomy performed in the early and late stages on neurological deficit in injuries of the spinal cord.

		Improvement in neurological deficit	No improvement in neurological deficit
Early Laminectomy	6	1 (Cervical)	5 (1 dorsal, 4 lumbo-dorsal)
Late Laminectomy	3	3 (All lumbo-dorsal)	0

TABLE 6

Relationship between the degree of neurological recovery and the residual reflex pattern in injuries of the spinal cord

		RESIDUAL NEUROLOGICAL DEFICIT				
		COMPLETE		INCOMPLETE		
		Degree of Recovery		Degree of Recovery		
Residual Reflex Pattern		Major	Nil or Negligible	Major	Nil or Negligible	Unknown
Normal	1	Nil	0	1		
Upper motor neurone	14	Nil	0	8	6	
Lower motor neurone	19	Nil	10	1	7	1
Mixed	4	Nil	3		1	
Unknown	3		0			3
	41		13			

TABLE 7

Relationship of pressure sores to the residual neurological deficit and reflex pattern in injuries of the spinal cord

Ultimate Reflex Pattern	Totals	COMPLETE DEFICIT		INCOMPLETE DEFICIT		
		Pressure Sores		Pressure Sores		
		Severe	Negligible	Severe	Negligible	Unknown
Normal	1	-	-		1	
Upper motor neurone	14	-	-	4	8	2
Lower motor neurone	19	10	Nil	4	2	3
Mixed	4	2	1	1	-	
Unknown	3	3				
Totals	41	15	1	9	11	5

TABLE 8

Effect of initial treatment of the bladder on the incidence of urinary sepsis in injuries of the spinal cord.

Initial bladder treatment	Number	Developing Severe Urinary Sepsis	Permanently Incontinent	Dying from renal sepsis
Bladder drained per urethram - Indwelling catheter - Foley	17	10	9	3
-Indwelling catheter - Gibbon	2	2	2	1
Tidal drainage	6	6	4	2
Intermittent catheterisation	0	0		
Suprapubic catheterisation	1	1	1	1
Manual compression	1	1	1	1
Total	27	20(74%)	17	8
Bladder not drained-Total	7	0	0	0
Bladder status unknown - Total	7	2	2	0

TABLE 9.

Duration of continuous hospitalisation of those surviving the acute phase of injuries of the spinal cord.

	No.	Less than 1 Year	Between 1 and 2 Years	Over 2 Years	Undetermined Period	Average Stay	Died
Survivors	41	20	11	7	3	13½ months	8

TABLE 10

Employment record of those surviving the acute phase in injuries of the spinal cord.

	Number	%
Never gainfully employed	24	80
Sporadically gainfully employed	3	10
Continuously gainfully employed	3	10
Employment record unknown	11	-
	41	

PART TWO.

OBSERVATIONS ON THE PATHOLOGY OF THE INJURED SPINAL CORD.

LEGENDS AND FIGURES

PLATE 1.

Photomicrograph of upper cervical cord in Case 1. Cord examined 1 hour after injury.

The upper plate shows the cytoplasm of one or two axis cylinders, indicated by an arrow, to have been replaced by a series of ragged, saccular swellings which give the axon the appearance of a piece of burned string.

The lower plate shows two small haemorrhages and a fissure; at the margin of the latter is a saccular axon swelling, indicated by an arrow.

(Bodian X 210)

PLATE I.

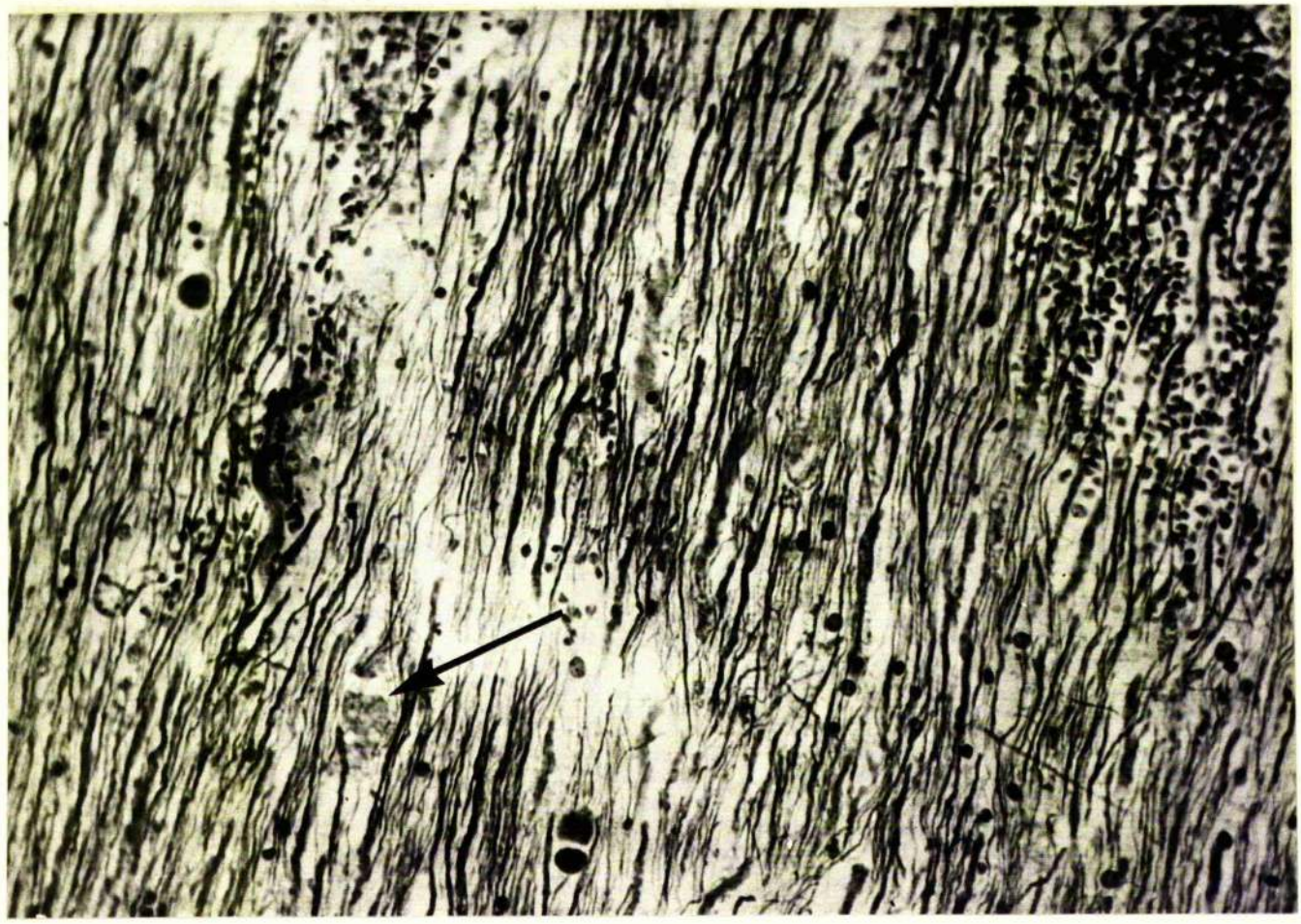


PLATE 2.

Photomicrograph of servical cord in Case 2. Cord examined 2 days after injury.

There is extensive structural damage and few axis cylinders are recognisable. The cytoplasm of damaged axons has extruded to give an amorphous background of fine granular debris. There are numerous cystic spaces which give the appearance of a sieve to the tissue. These spaces appear to have been formed by precipitation of axon cytoplasm round the rim. There is a mild infiltration with small dark round cells.

(Bodian X 210)

PLATE 2.

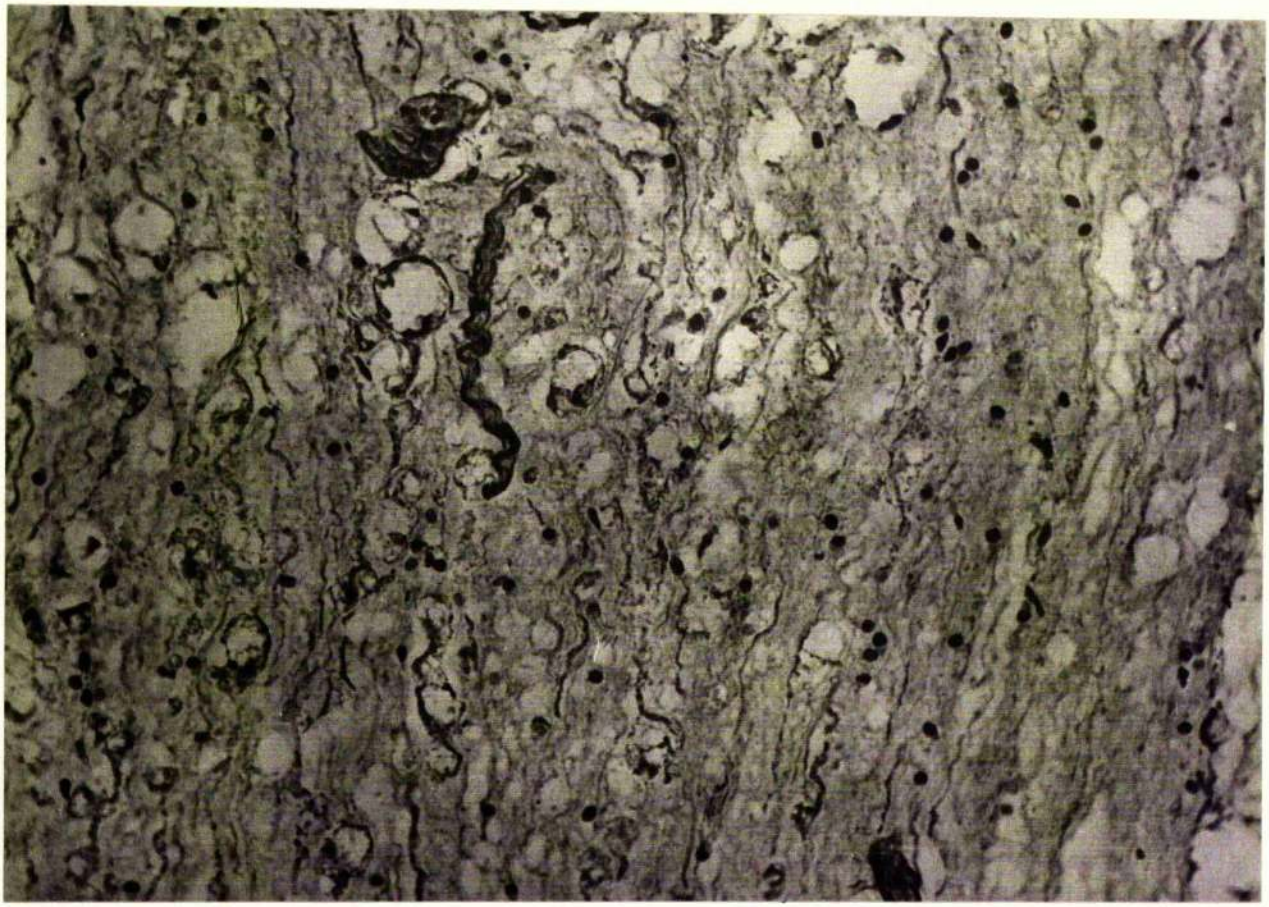


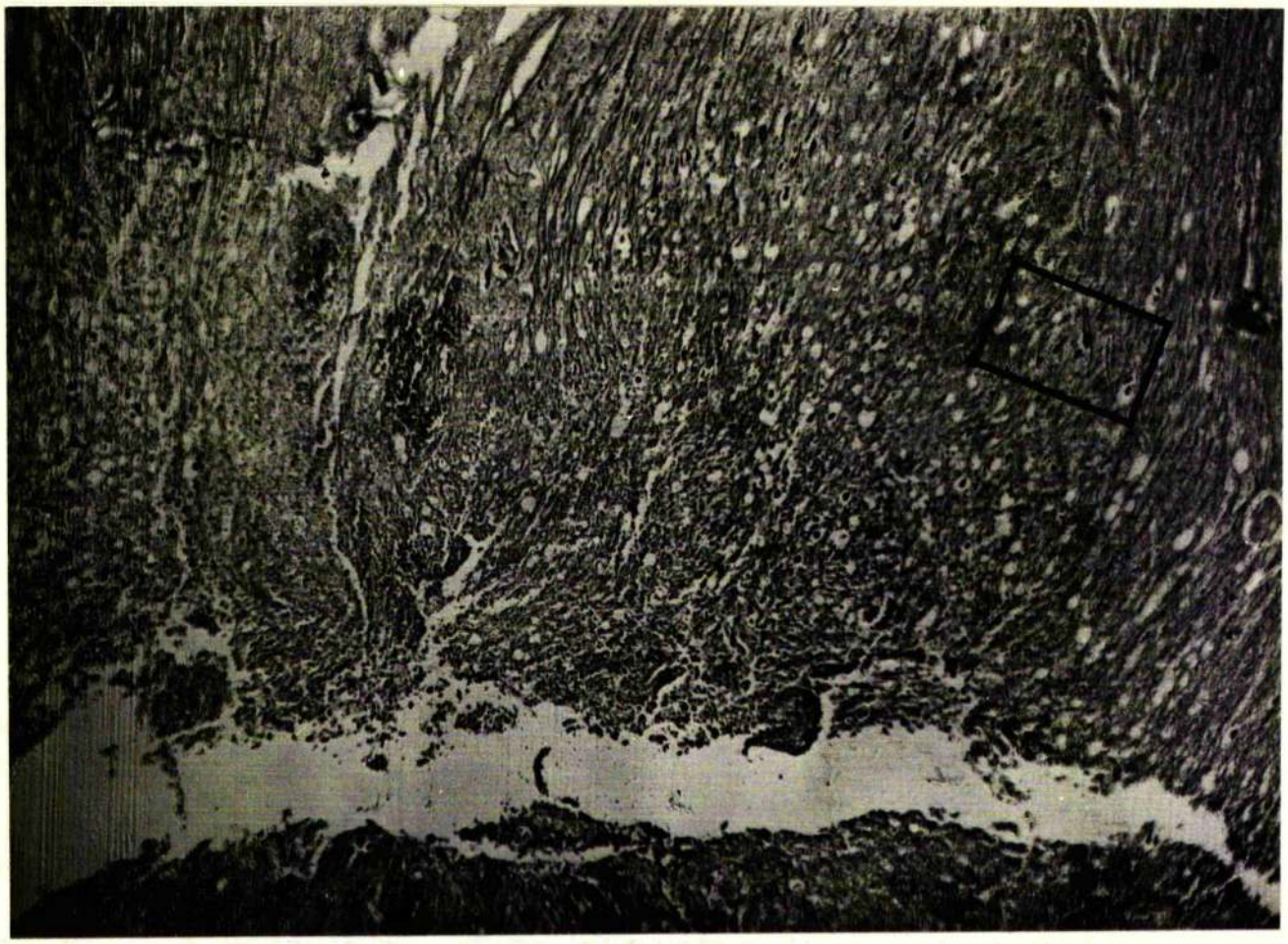
PLATE 3.

Photomicrograph of cervical cord in Case 3. Cord examined 5 days after injury.

The upper plate shows the edge of a contused area at the bottom of the field, sharply demarcated from the rest of the cord. Numerous terminal axon figures are seen at the junctional zone of neighbouring cord. (X 24)

The lower plate shows a detail of the above. There are many giant sterile axon globes and the cytoplasm is coarse and lacks homogeneous avidity for the silver stain

(Bodian X 210)



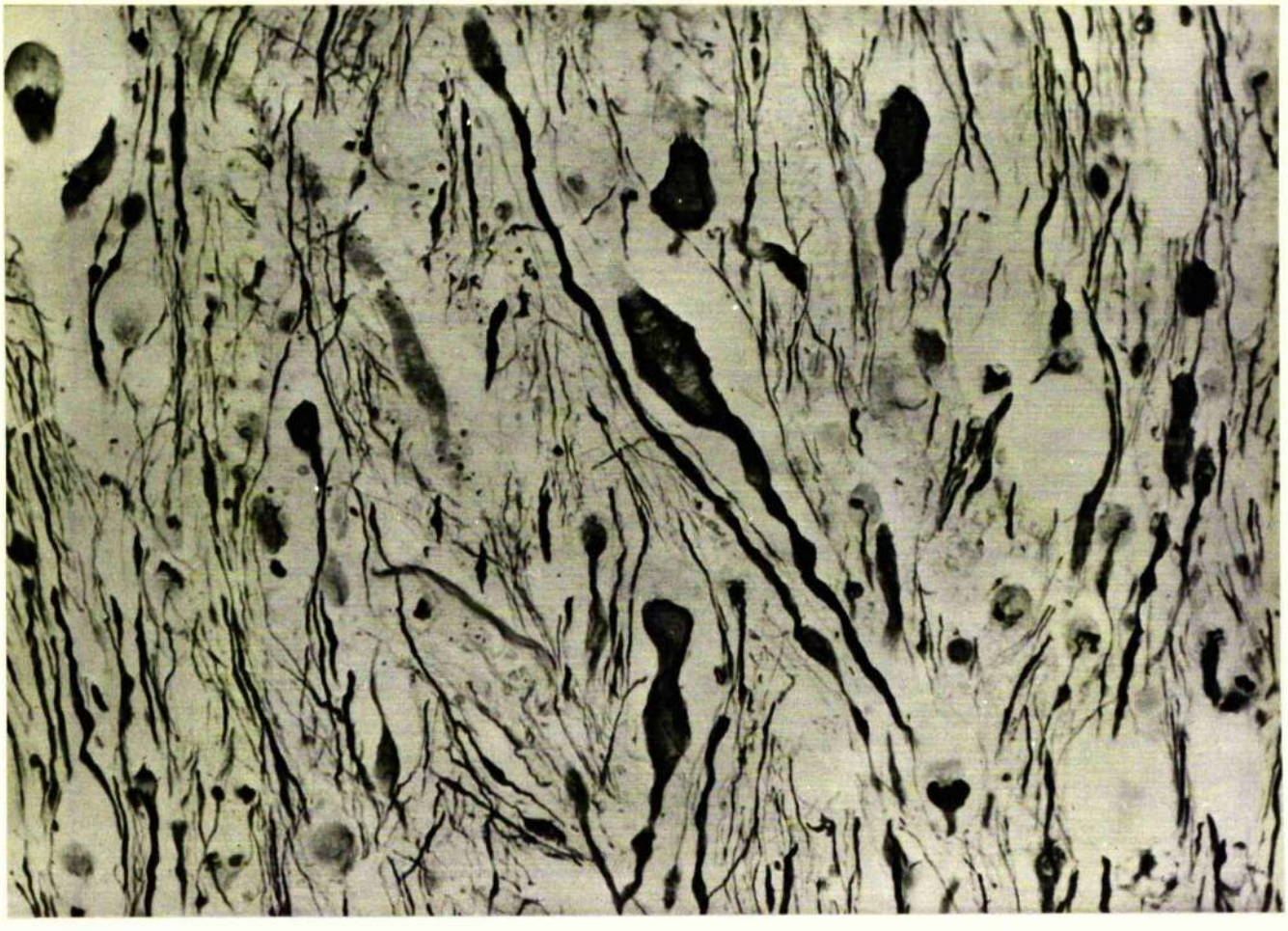


PLATE 4.

Photomicrograph of upper dorsal cord in Case 4. Cord examined 7 weeks after injury.

Young root fibres are seen streaming in towards the cord where they appear to be arrested. Several terminal axon figures, indicated by an arrow, are situated at the junctional zone. Deeper to this, in the cord substance, axis cylinders are attenuated and ragged, and there is a moderate increase in number of darkly staining neuroglial cells.

(Bodian X 210)

PLATE 4.

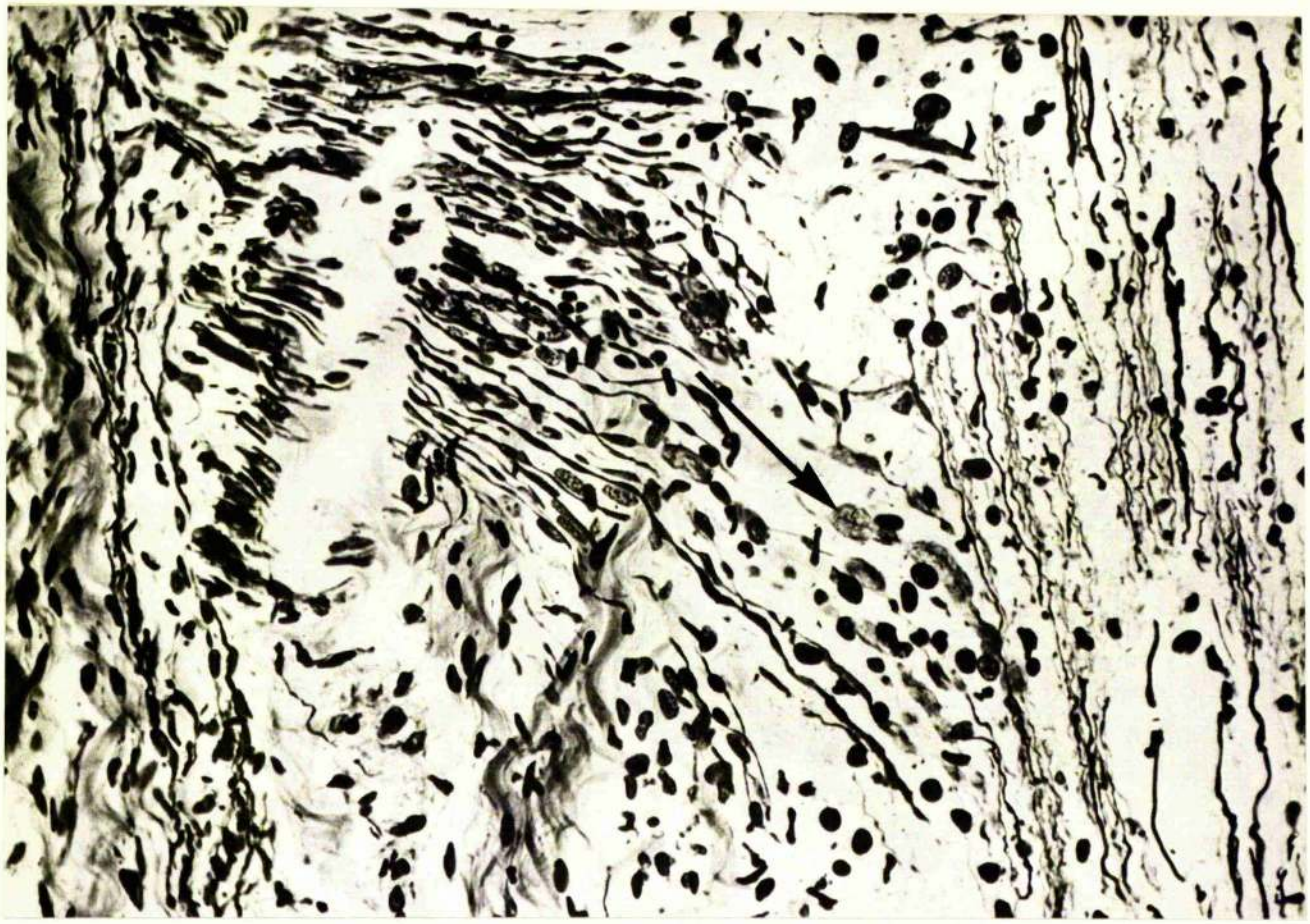


PLATE 5.

Photomicrograph of cord at lumbo-dorsal junction in Case 5.

Axis cylinders are reduced in number, and cytoplasm, extruded from damaged axons, forms a background of fine amorphous debris. Neuroglial cells are numerous. Their cytoplasm is enlarged and possesses greater avidity for silver stain than usual. The nucleus of these cells is often eccentrically placed and one or two are multinucleated.

(Bodian X 210)

PLATE 2.

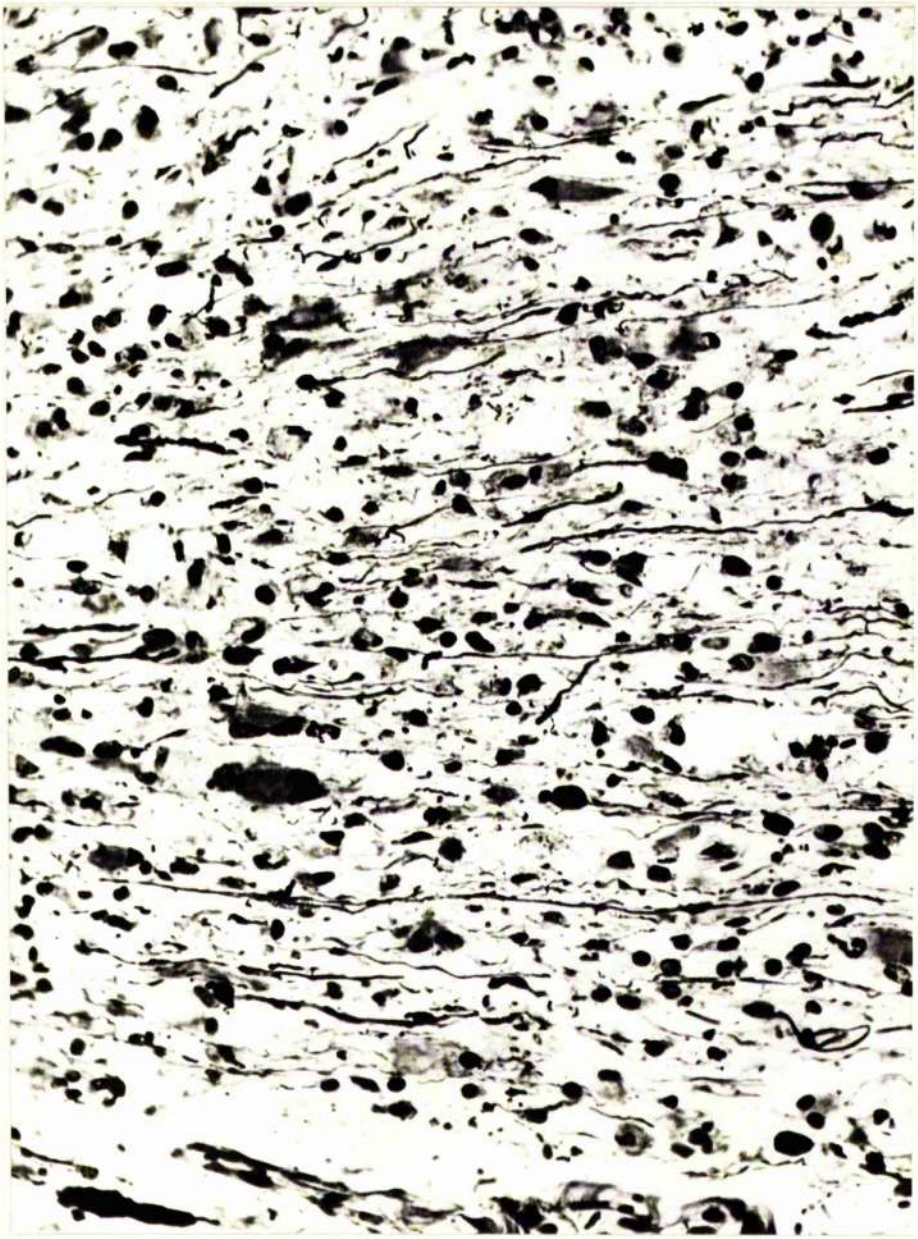


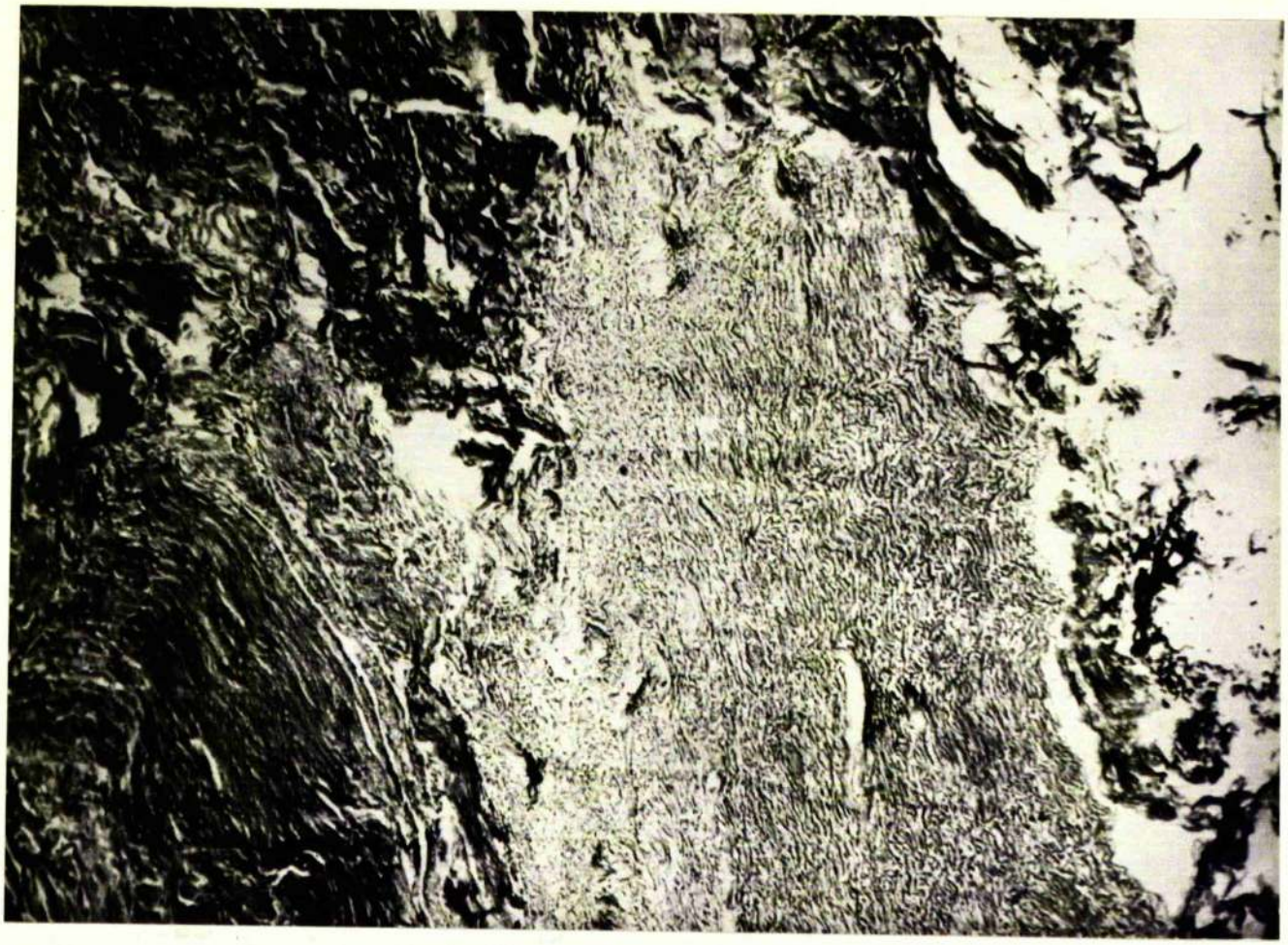
PLATE 6.

Photomicrograph of lower dorsal cord in Case 6. Cord examined 5 years after injury.

The upper plate shows the gliosed cord remnant framed by luxuriant scarring of the menninges. (x 40)

The lower plate shows a number of, apparently surviving axons. A moderate increase in neuroglial cells, without evidence of large argentophil forms, comprises the mature glial scar.

(Bodian X 210)



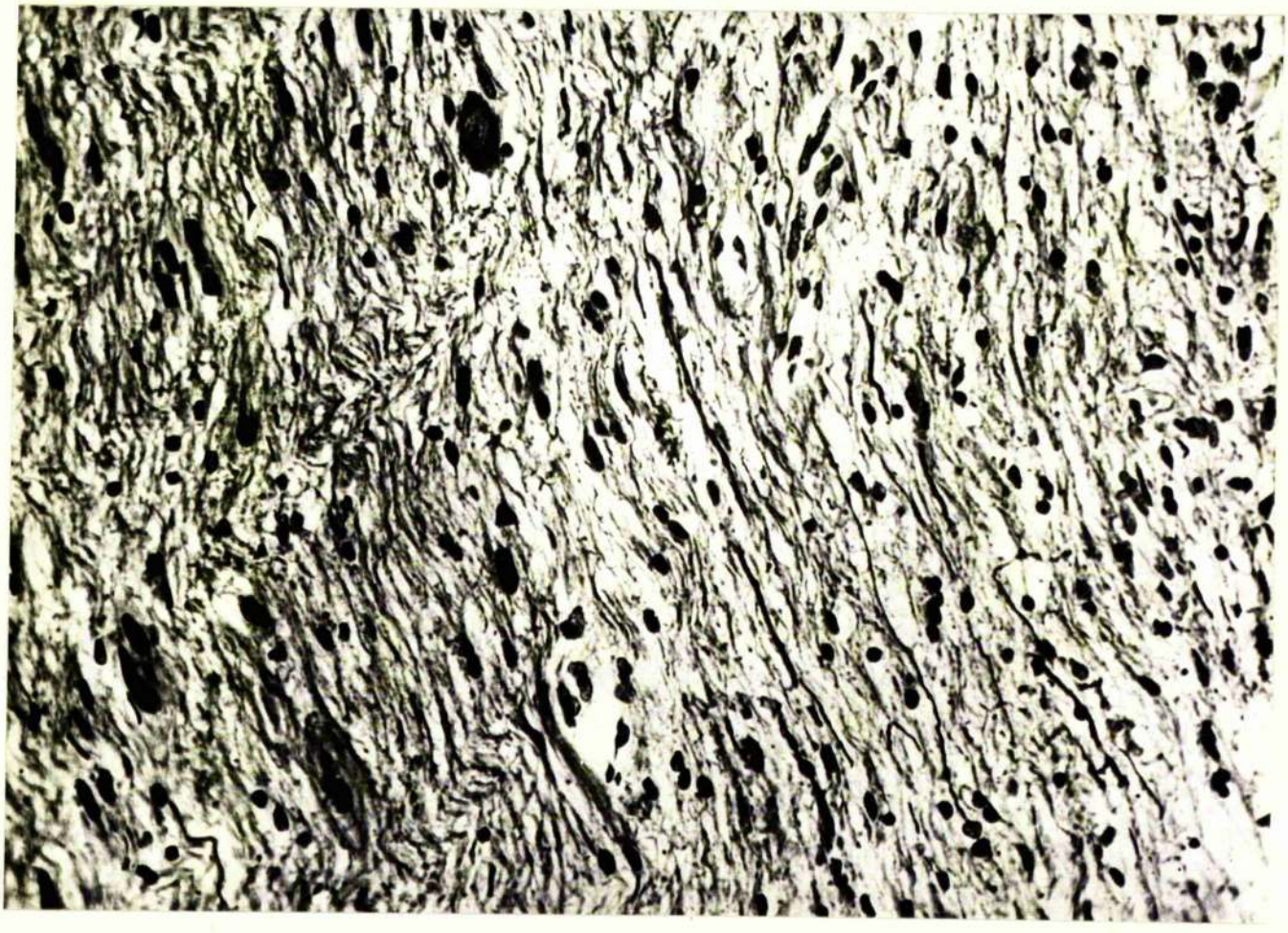


PLATE 7.

Photomicrograph of cord at the cervico-dorsal junction in Case 7. Cord examined 2 years after injury.

There is a wedge of demyelination in both lateral columns.

(Weigert x 16)

PLATE 7.

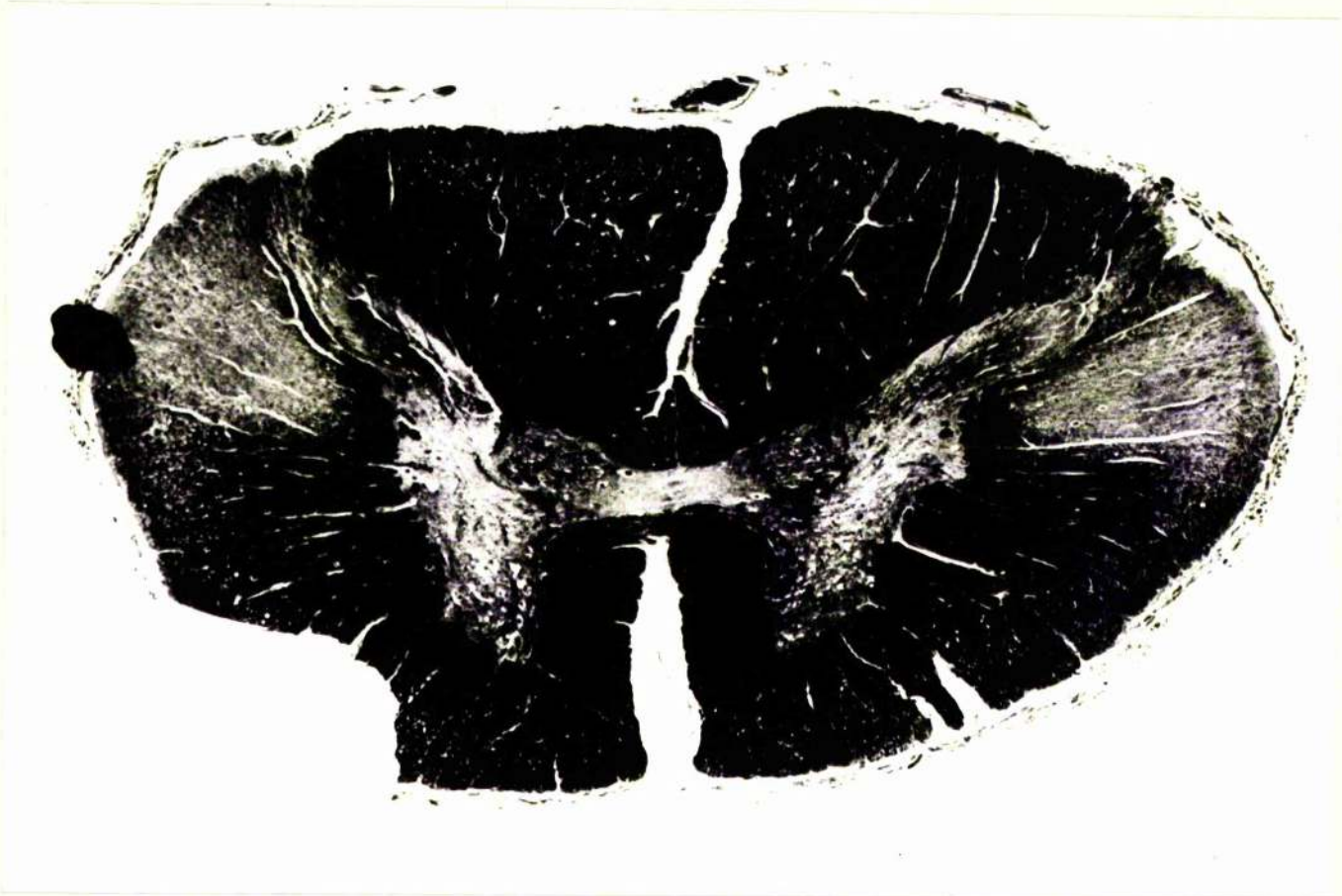


PLATE 8.

Photomicrograph of the anterior horn of cord at the cervico-dorsal junction in Case 7. Cord examined 2 years after injury.

There is pyknosis and shrinkage indicating destruction of anterior horn cells. The rest of the grey matter structure is poorly defined.

(unna Pap X 135)

PLATE 8.

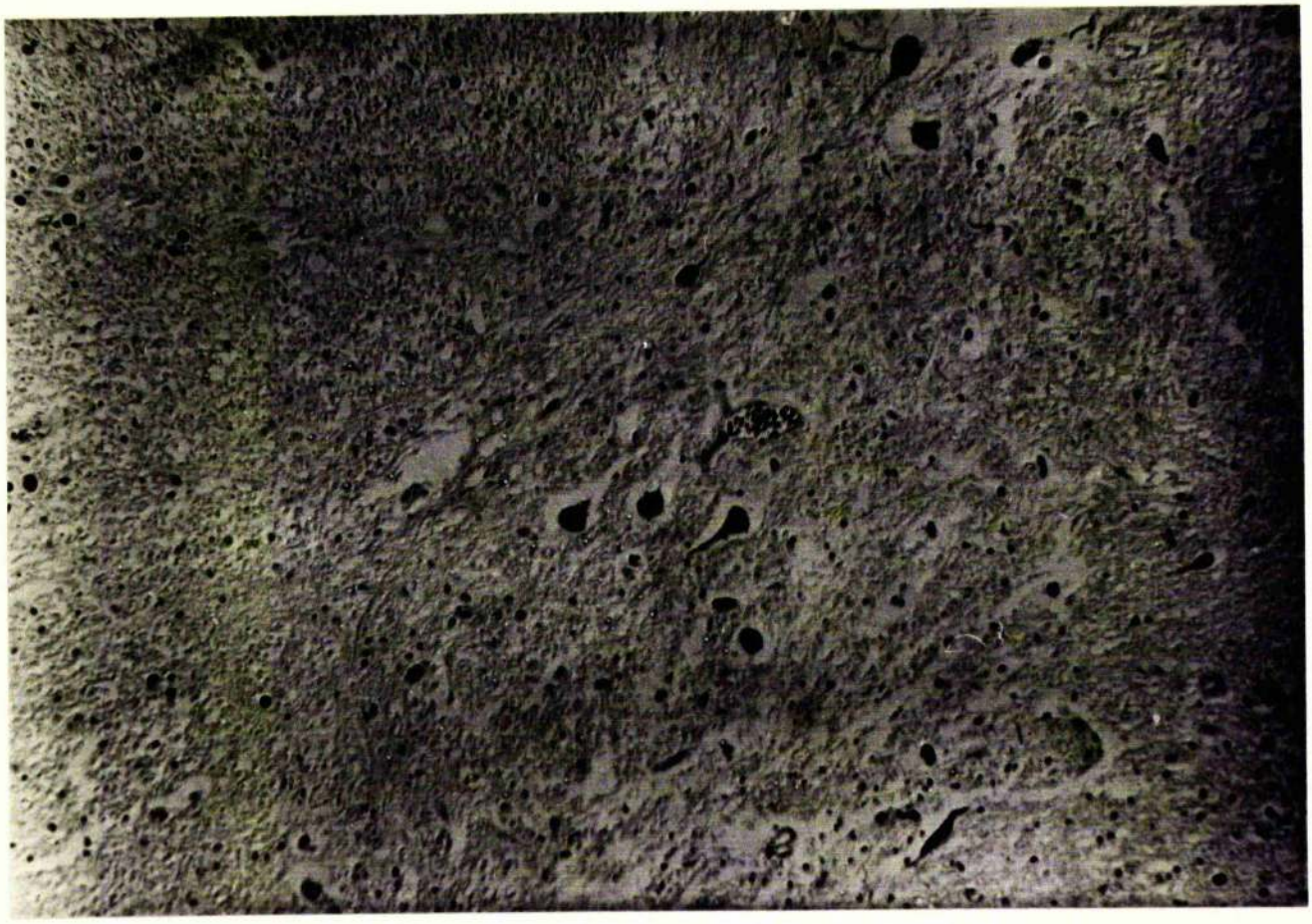


PLATE 9.

Photomicrograph of the demyelinated lateral columns of cord at the cervico-dorsal junction in Case 7. Cord examined 2 years after injury.

Axis cylinders are well preserved. Note the presence of numerous corpora amylacea.

(Bielschowsky X 225)

PLATE 9.

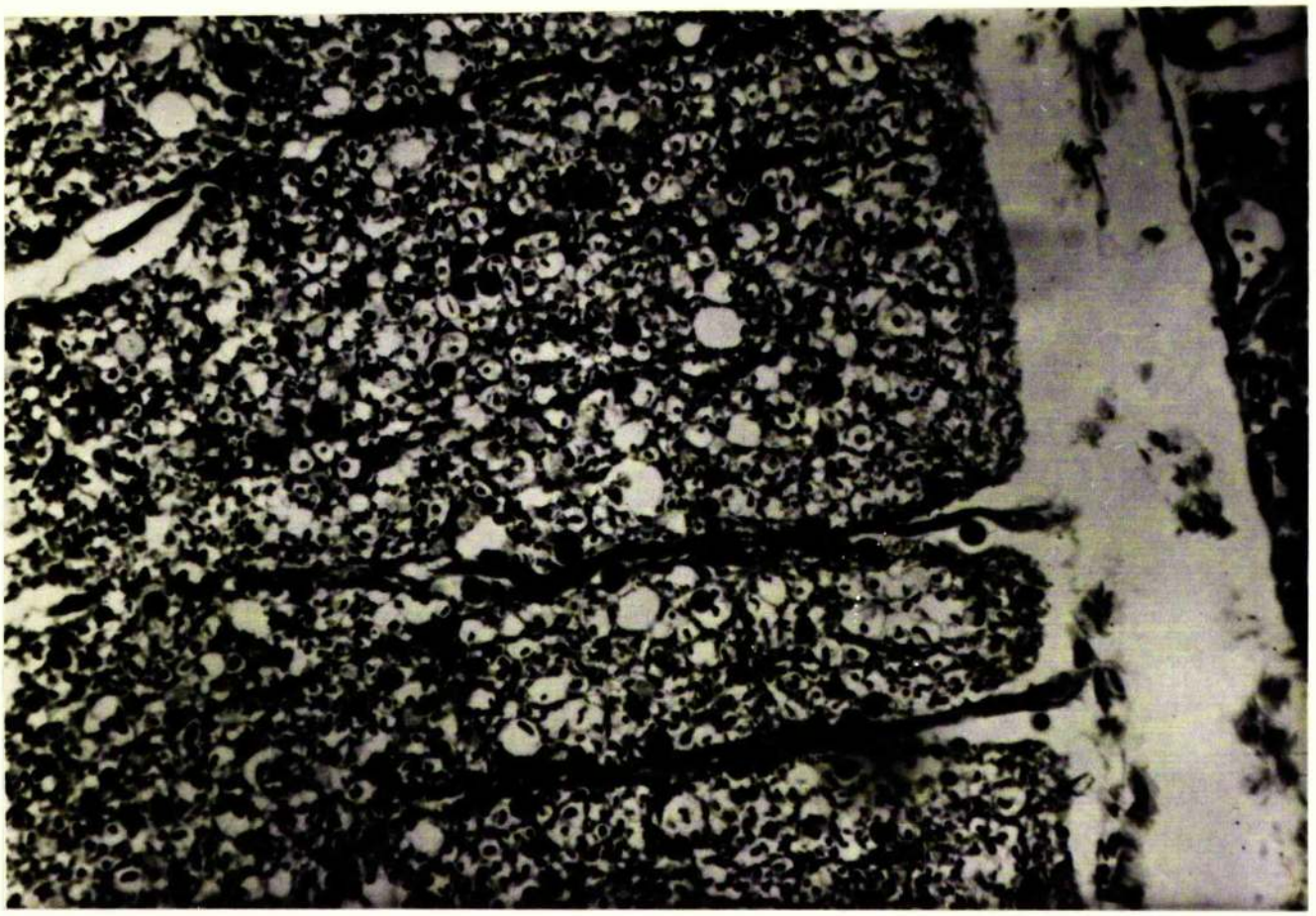


PLATE 10.

Photomicrograph of dorsal column at the cervico-dorsal junction in Case 7. Cord examined 2 years after injury.

The upper plate shows focal accumulations of axons radiating from small cystic spaces. (Bielschowsky X

The lower plate shows a detail of this. Smudges are present in these areas, indicating collagen deposition.

(Bielschowsky X 225)

PLATE 10.

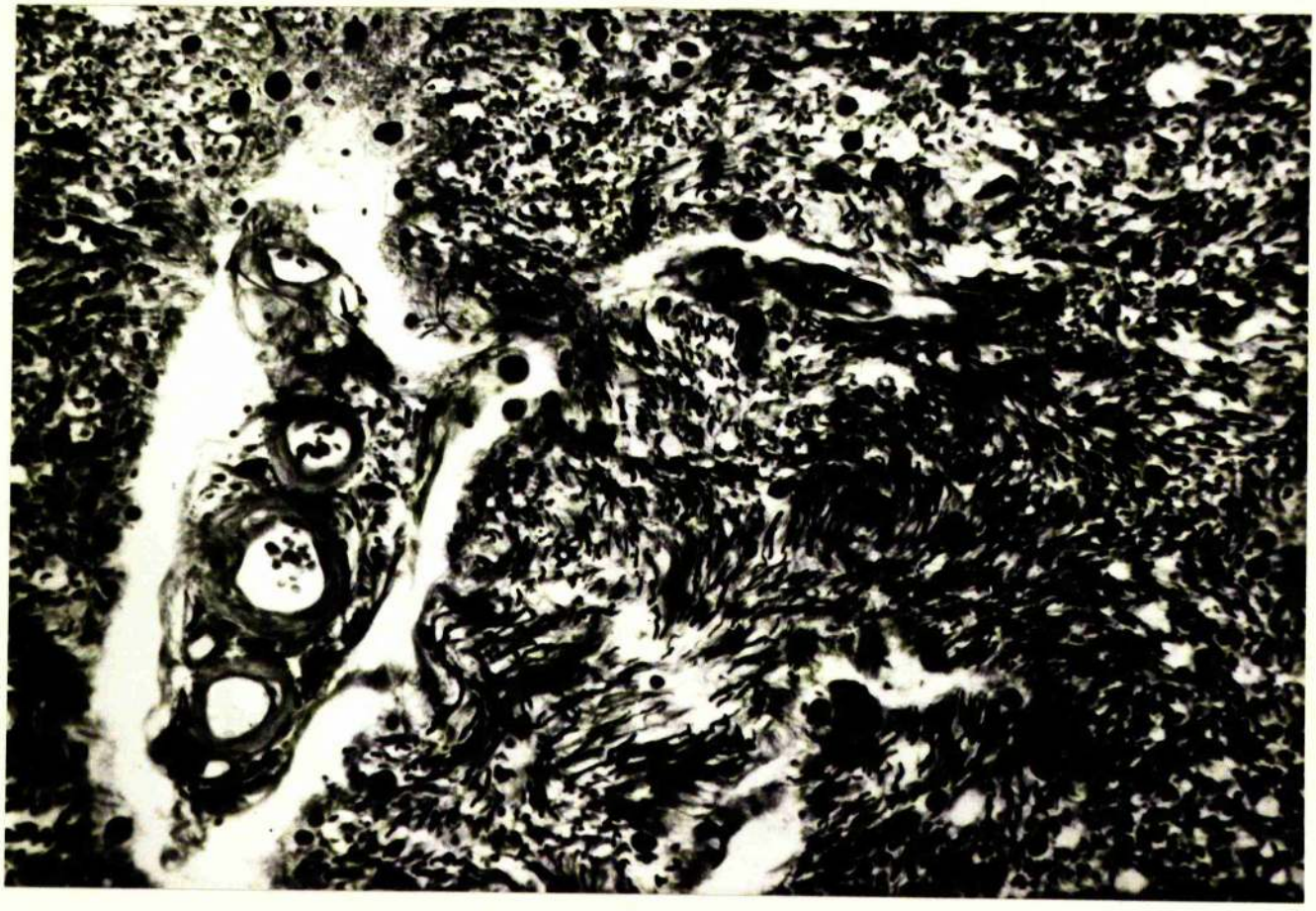


PLATE 11.

Photomicrograph of upper cervical cord in Case 7. Cord examined 2 years after injury.

There is extensive demyelination of the dorsal columns and some demyelination of lateral and antero-lateral columns.

(Weigert X 12)

PLATE 11.

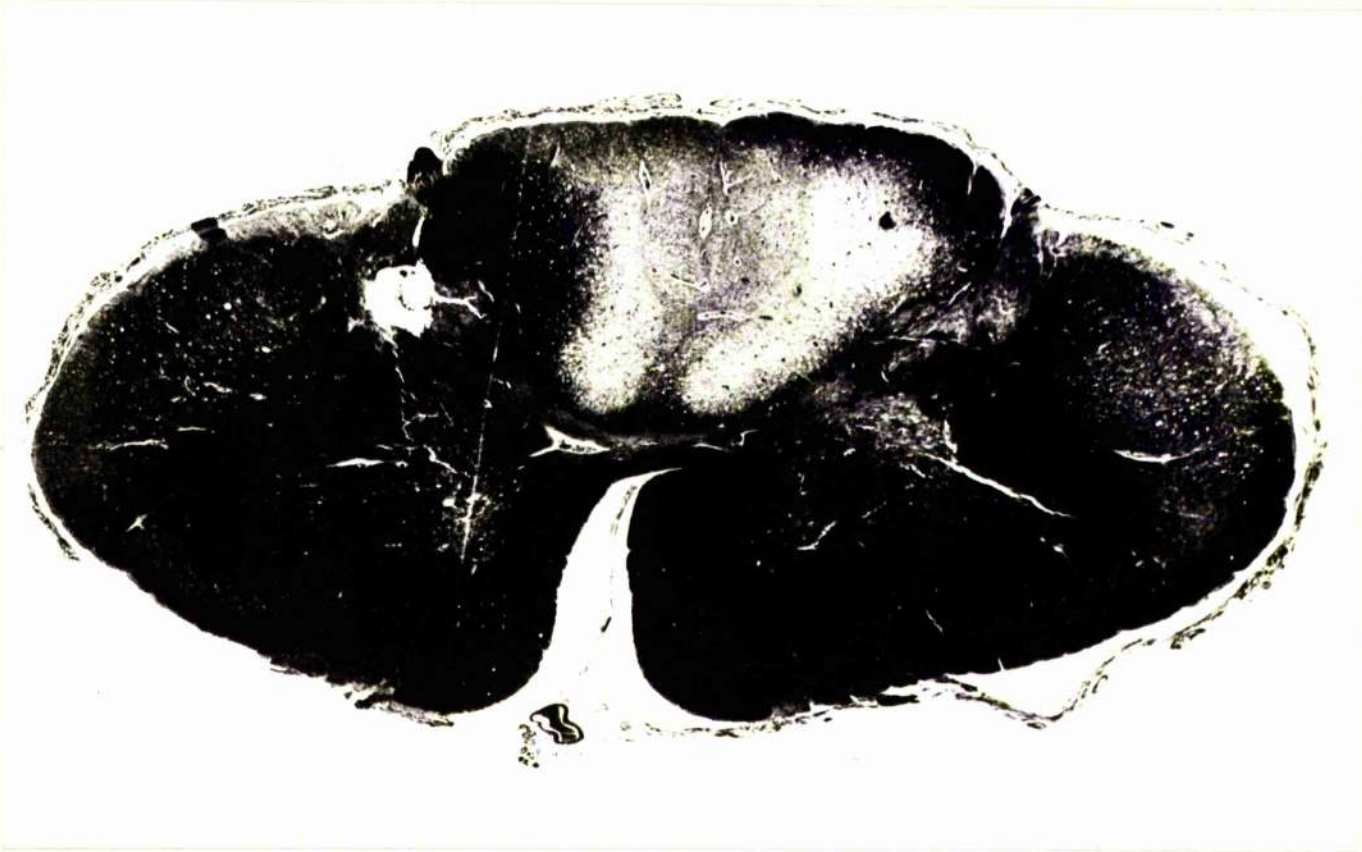


PLATE 12.

Photomicrograph of anterior horn of upper cervical cord in Case 7. Cord examined 2 years after injury.

Anterior horn cells are relatively well preserved. There is some loss of definition of supporting grey matter structure.

(Unna Pap X 135)

PLATE 12.

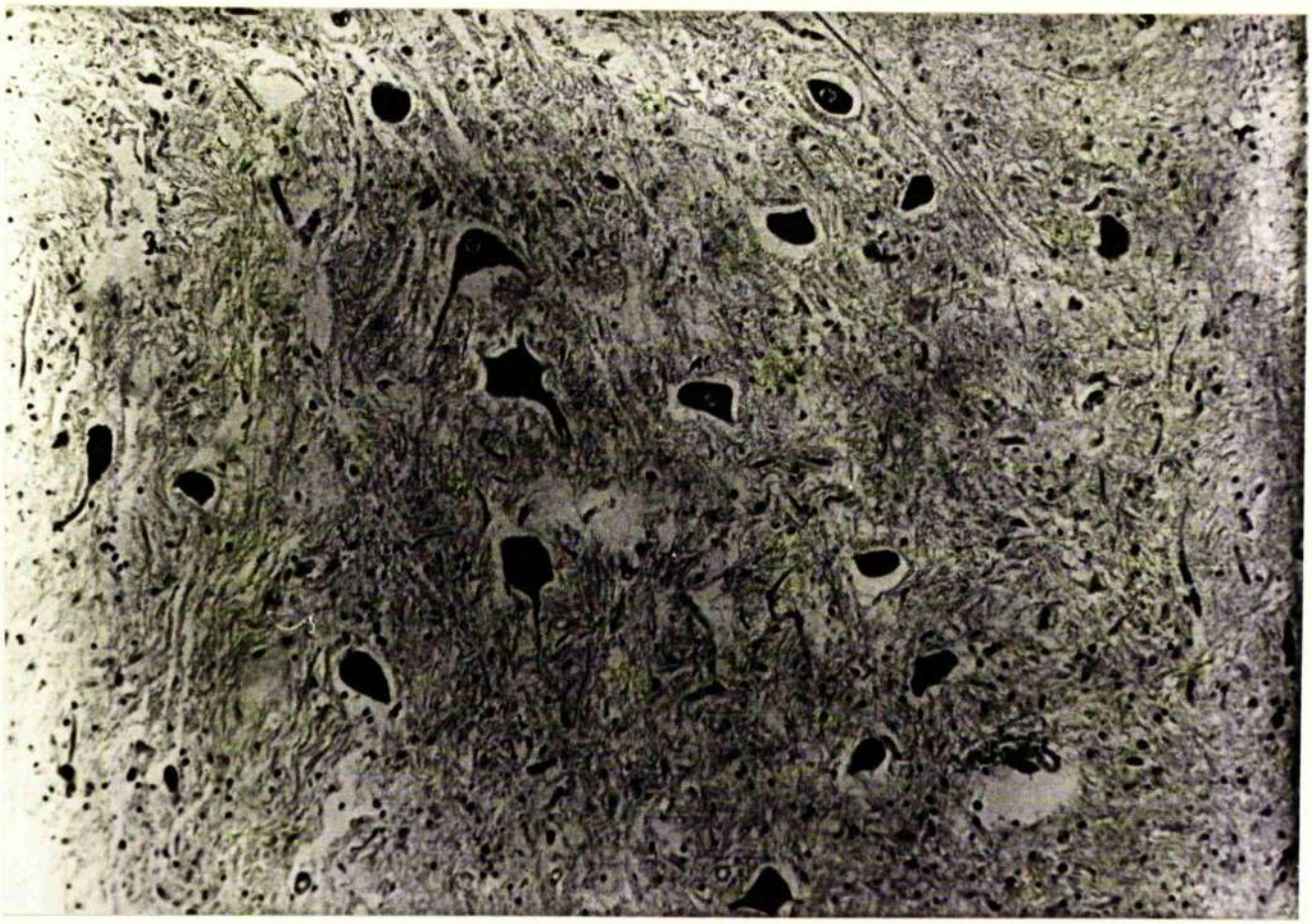


PLATE 13.

Photomicrograph of demyelinated dorsal columns of upper cervical cord in Case 7. Cord examined 2 years after injury.

Many axon sheaths are swollen and some axons appear to have fallen out of their sheaths. Neuroglial cells are increased in number and avidity for silver stain, and possess coarse protoplasmic feet.

(Bielschowsky X 90)

PLATE 13.

PLATE 14.

Photomicrograph of lumbar cord in Case 7. Cord removed 2 years after injury.

There is a small wedge of demyelination in the dorsi-lateral columns.

(Weigert X 15)

PLATE 14.

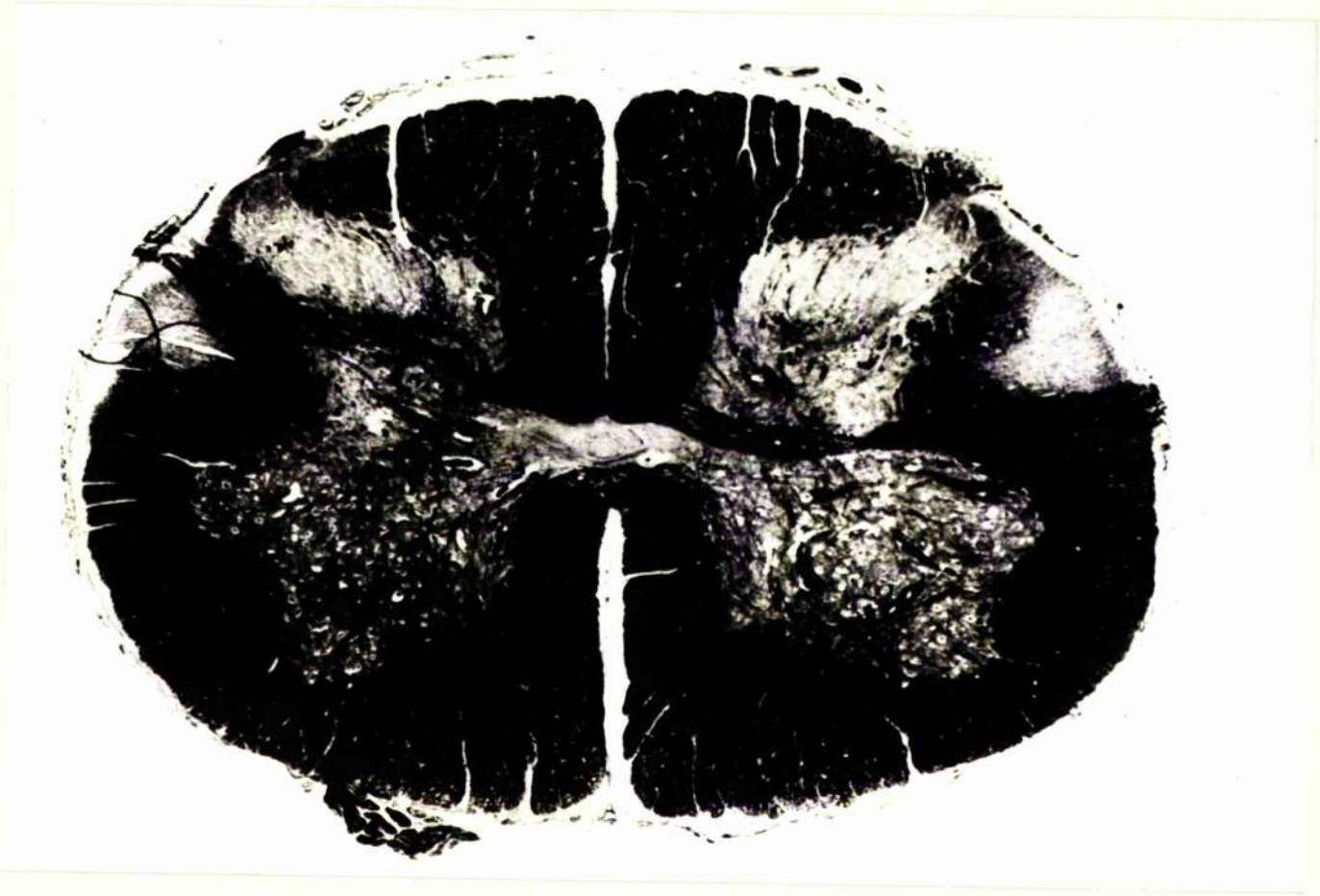


PLATE 15.

Photomicrograph of anterior horn of lumbar cord in Case 7. Cord examined 2 years after injury.

Anterior horn cells are relatively well preserved. There is some loss of definition of the supporting grey matter structure.

(Unna Pap X 135)

PLATE 15.

PLATE 16.

Photomicrograph of demyelinated dorsi-lateral column of lumbar cord in Case 7. Cord examined 2 years after injury.

Many axis cylinders have been preserved. Neuroglial cells are increased in number and avidity for silver, and exhibit coarse protoplasmic feet.

(Bielschowsky X 90)

PLATE 16.

PLATE 17.

Photomicrograph of segment of cord at lumbo-dorsal junction in Case 8. Cord examined 4 years after injury.

Compression upon the cord has left an impression on it, indicated by an arrow. There is a band of demyelination extending cranially from the median parts of the dorsal columns and some demyelination of the lateral columns. The apparent gap in the cord is caused by its being twisted upon itself.

(Weigert X 4)

PLATE 17.

PLATE 18.

Photomicrograph of anterior horn of segment of cord adjoining the compressed area shown in Plate 17. Cord examined 4 years after injury.

Anterior horn cells exhibit some pyknosis. Otherwise grey matter structure is preserved.

(Bielschowsky X 200)

PLATE 18.

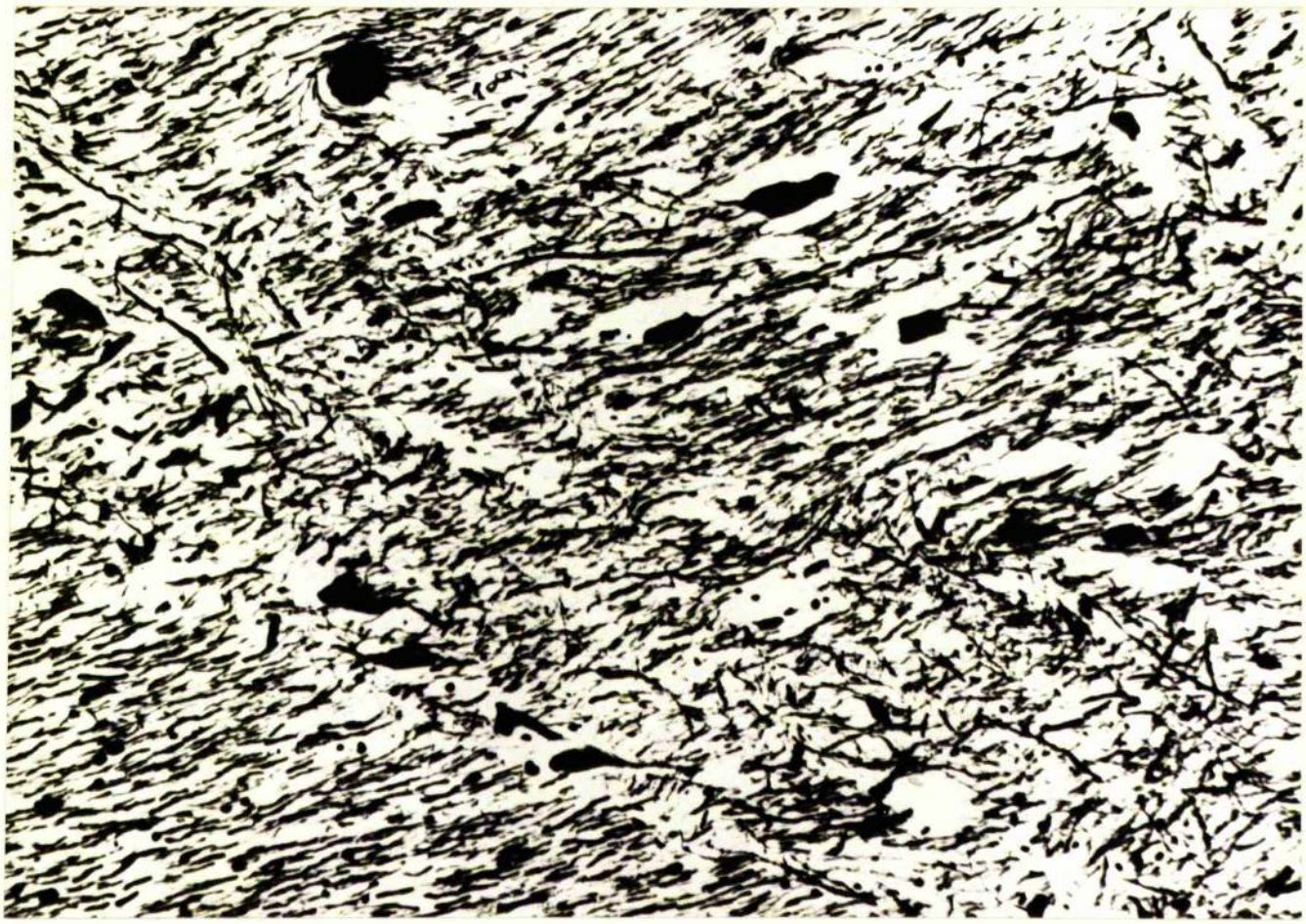


PLATE 19.

Photomicrograph of partially delyelinated lateral columns adjoining the compressed area shown in Plate 17 Cord removed 4 years after injury.

Axis cylinders are preserved. There are one or two large argentophil neuroglial cells with protoplasmic feet, indicated by an arrow.

(Bielschowsky X 90)

PLATE 19.

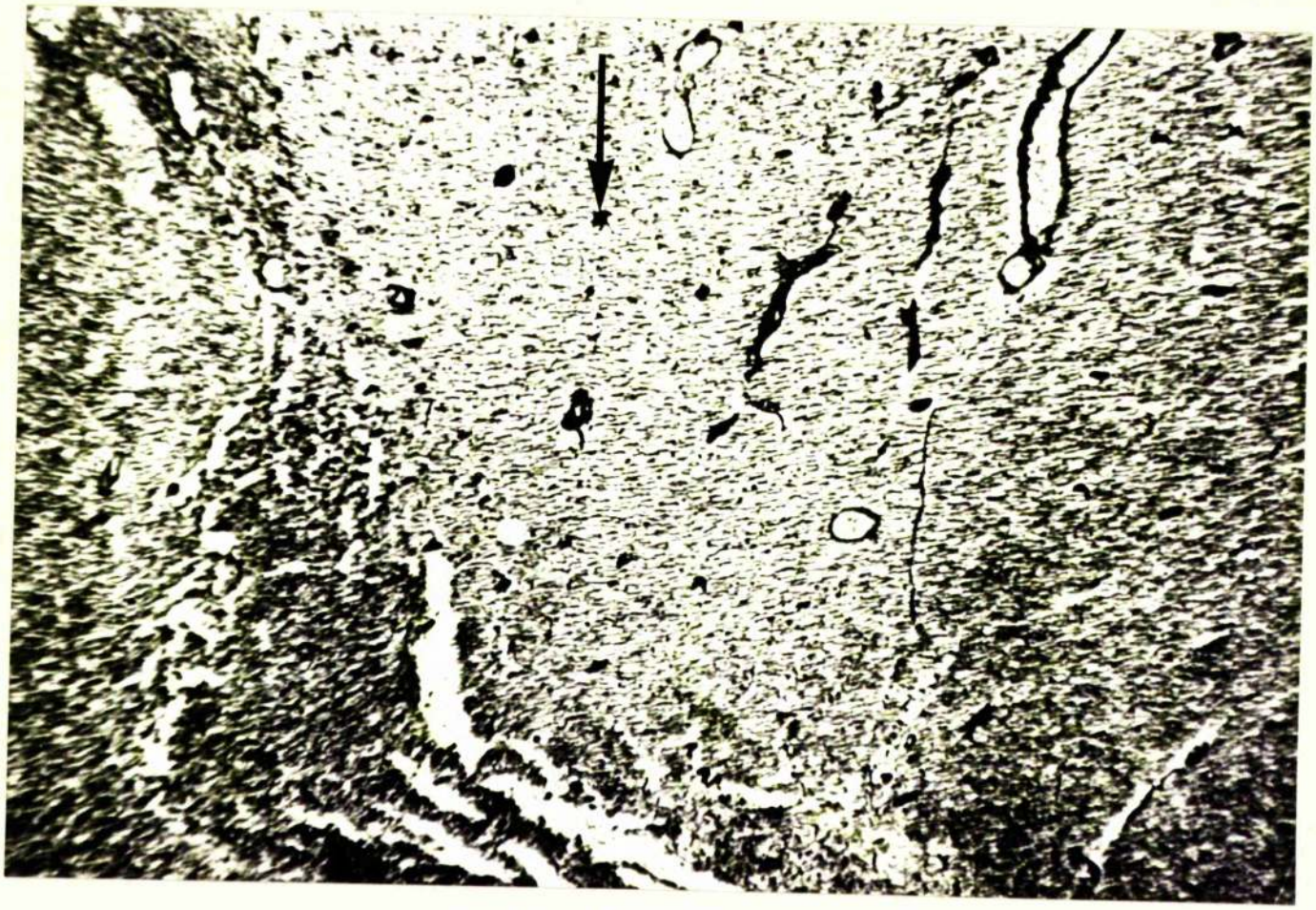


PLATE 20.

Photomicrograph of periphery of lateral columns adjoining the compressed area shown in Plate 17. Cord examined 4 years after injury.

Several wavy bands of collagen have been laid down. These bands have caused some neighbouring axis cylinders to assume a corresponding wavy form.

(Bielschowsky X 90)

PLATE 20.

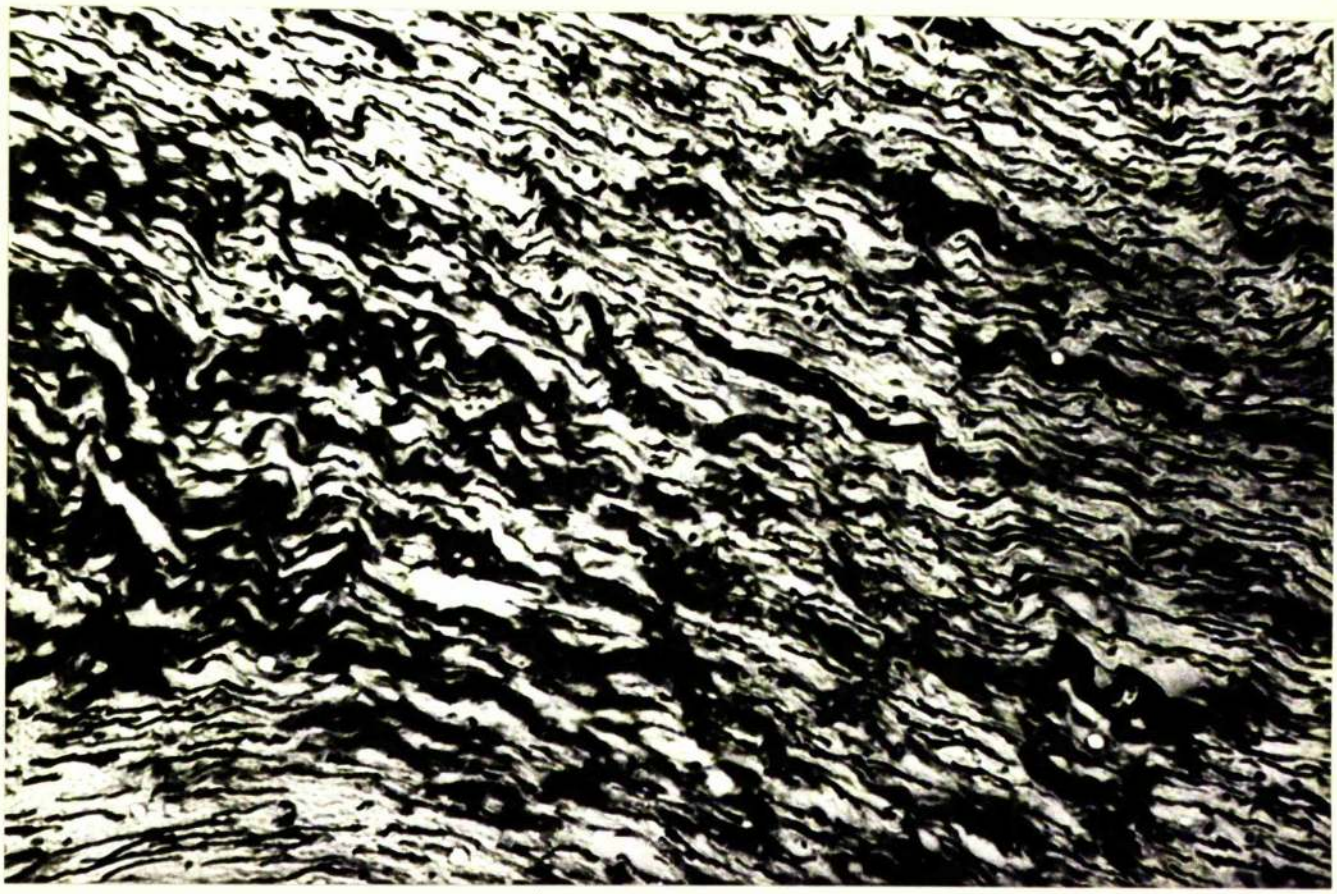


PLATE 21.

Photomicrograph of cauda equina in Case 8. Cord examined 4 years after injury.

There is extensive loss of definition, only a few myelinated nerve bundles surviving.

(Weigert X 19)

PLATE 21.

PLATE 22.

Photomicrograph of a cauda equina rootlet in Case 8. Cord examined 4 years after injury.

There is hypertrophy of the endothelium in a capillary, the swollen cells having a glassy appearance.

(H & E X 790)

PLATE 22.

PART THREE.

BLOOD PRESSURE RESPONSES EVOKED BY EXPERIMENTAL COMPRESSION OF THE SPINAL CORD IN CATS.

LEGENDS AND FIGURES

FIGURE 1.

Blood pressure response evoked by momentary compression of the third thoracic segment of the spinal cord. Compression applied at the arrow D.

FIGURE 1.

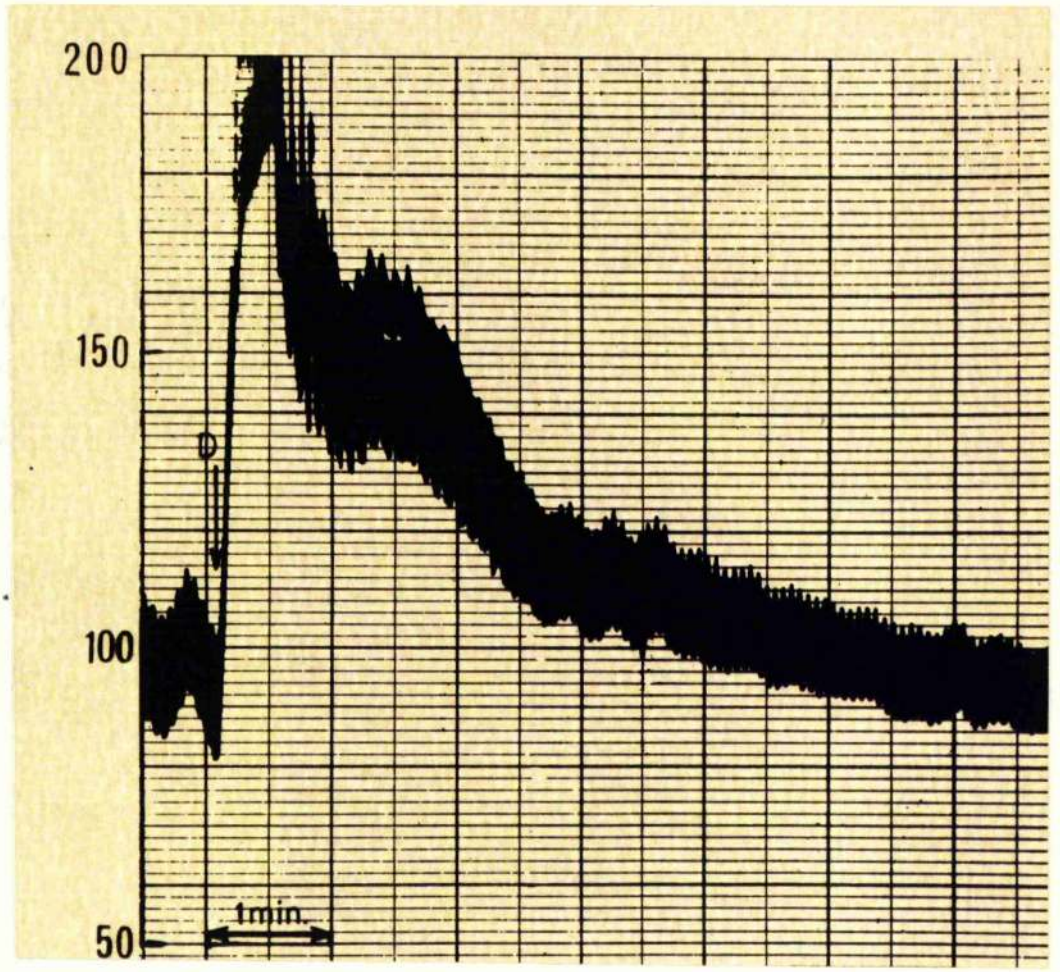


FIGURE 2.

Blood pressure responses evoked by compression of the spinal cord and intra-arterial administration of adrenaline. A'B' represents a two minute period of compression at the second thoracic segment at 200 mm. mercury balloon pressure. At C 0.2 ml. of 1:1000 adrenaline chloride was administered.

FIGURE 2.

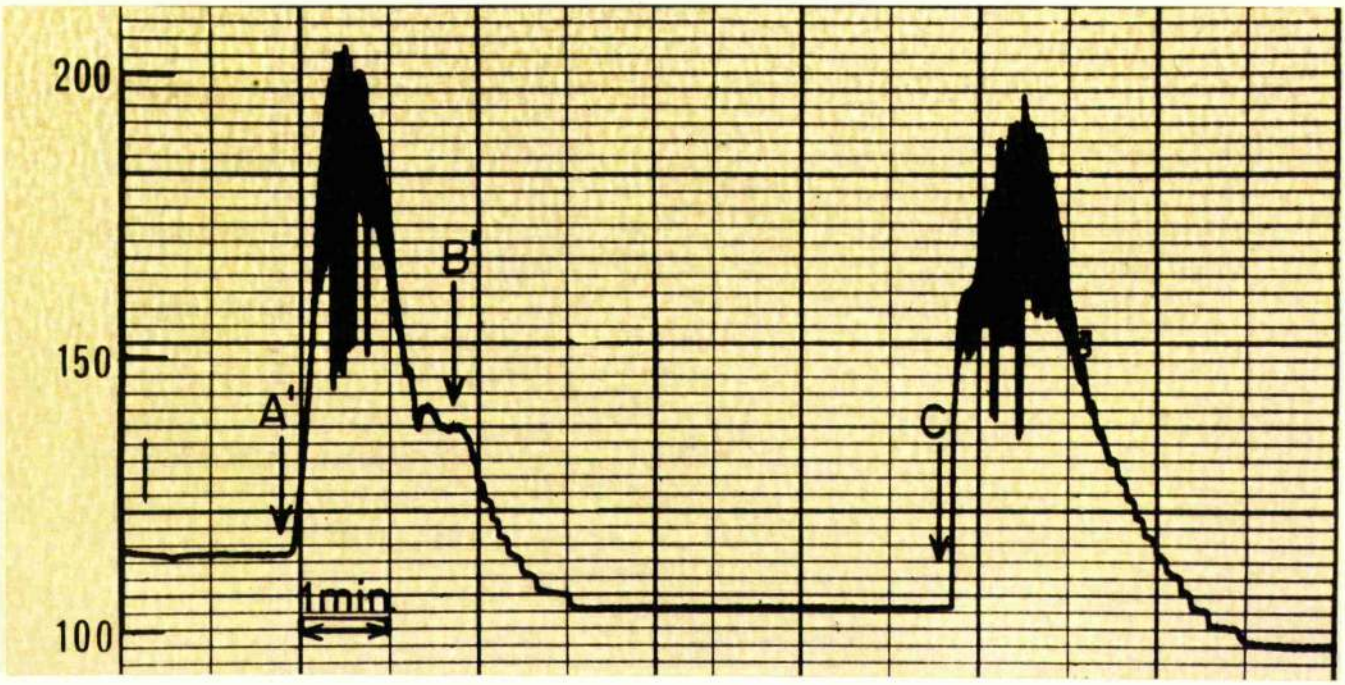


FIGURE 3.

Blood pressure responses evoked by different degrees of spinal cord compression at the fifth thoracic segment. AB represents a period of compression at 100 mm. mercury and A'B' at 200 mm. mercury, balloon pressure.

FIGURE 3.

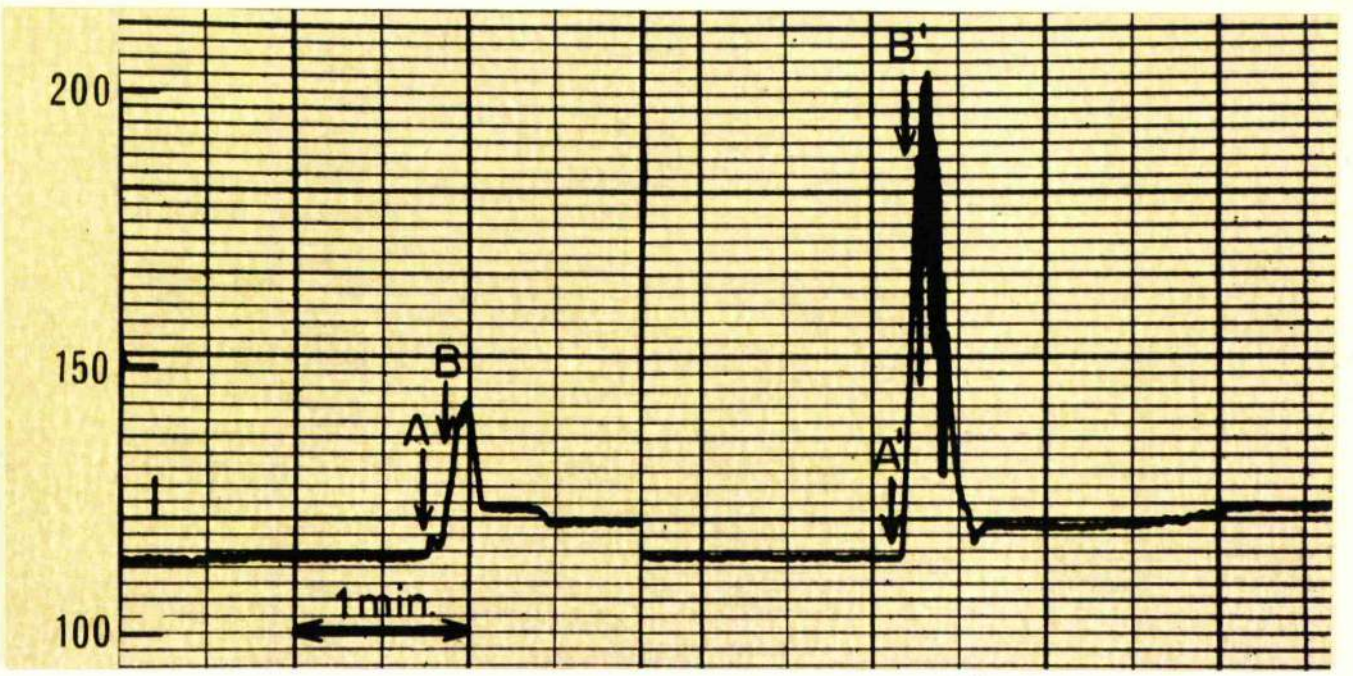


FIGURE 4.

Blood pressure responses evoked by different degrees of spinal cord compression at the seventh thoracic segment. $A^{\circ}B^{\circ}$ represents a period of compression at 50 mm. mercury, AB at 100 mm. mercury and A'B' at 200 mm. mercury, balloon pressure.

FIGURE 4.

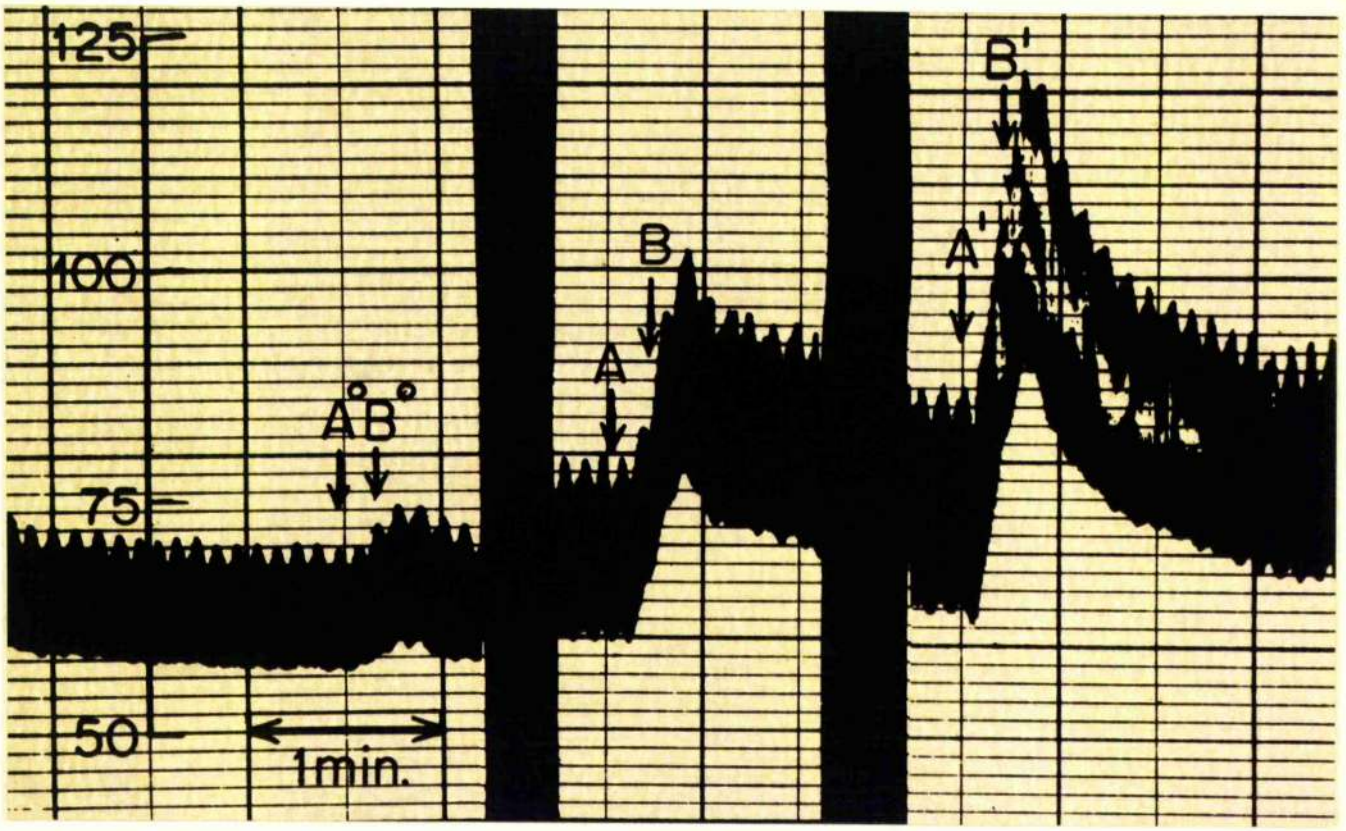


FIGURE 5.

Rises in mean blood pressure in nine cats following compression of different segments of spinal cord at 200 mm. mercury balloon pressure.

FIGURE 5.

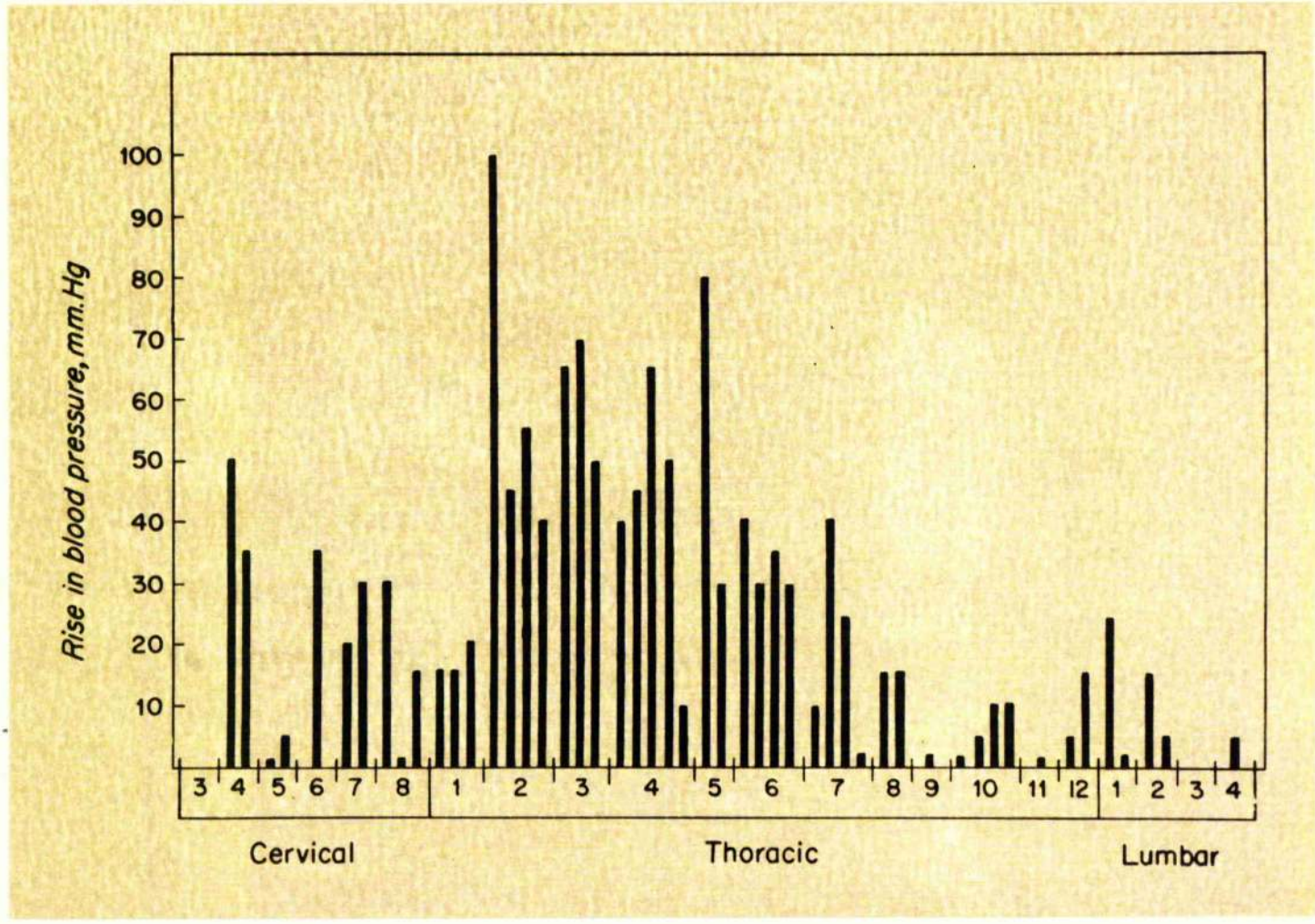
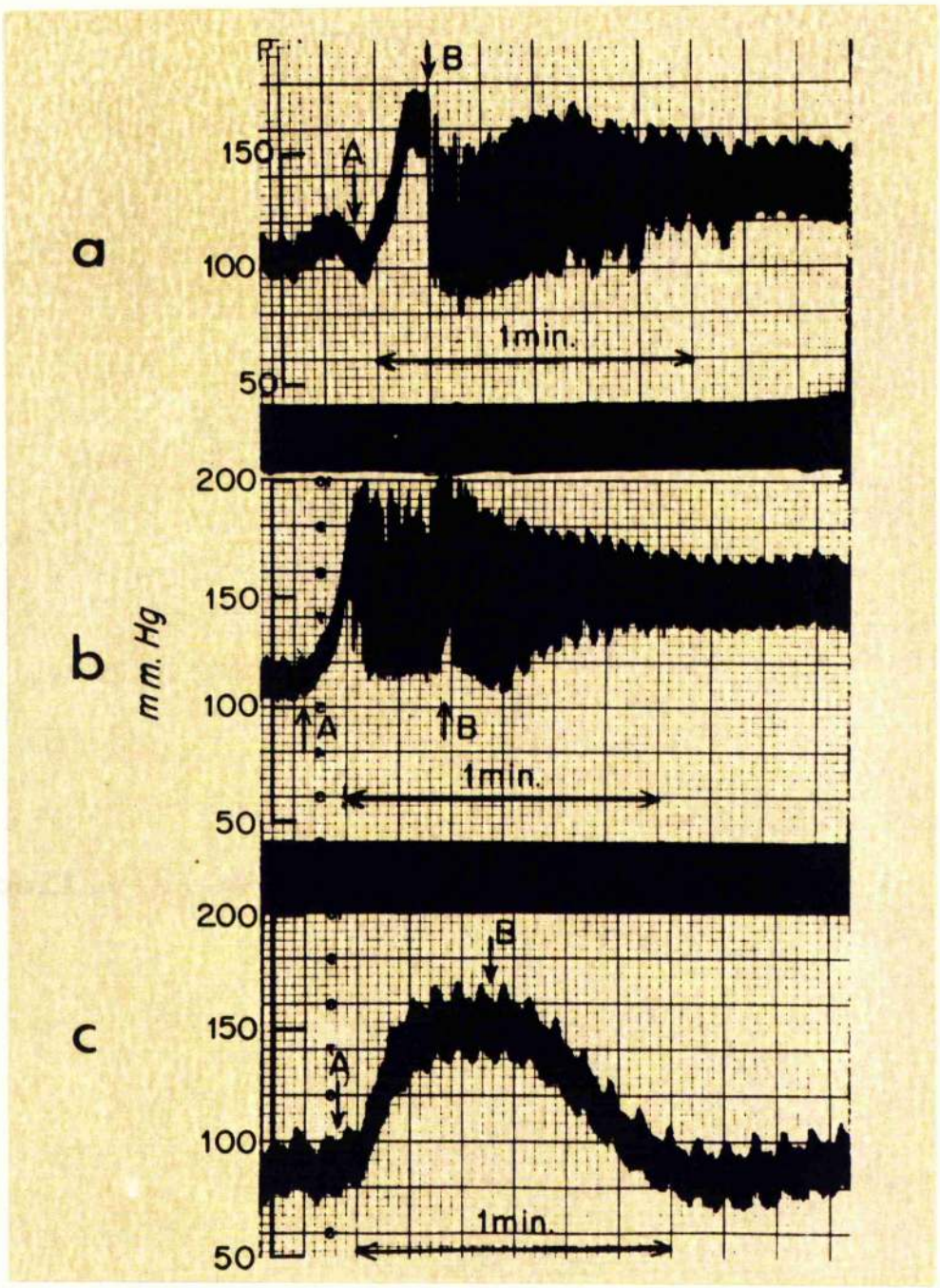

Rise in blood pressure, mm.Hg
100
90
80
70
60
50
40
30
20
10
3 4 5 6 7 8 1 2 3 4 5 6 7 8 9 10 11 12 1 2 3 4
Cervical
Thoracic
Lumbar

FIGURE 6.

Blood pressure responses from second thoracic segment after de-afferentiation (a & b). Upper three dorsal roots have been severed four days prior to compression. (c) sinu-aortic denervation has been performed. AB represents a period of compression at 100 mm. mercury, balloon pressure.

FIGURE 6.


a
b
c
mm. Hg
150
100
50
200
150
100
50
200
150
100
50
A
B
1min.
A
B
1min.
B
A
1min.

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APPENDIX

TABULAR SUMMARY OF THE 52 CASES OF CLOSED SPINAL CORD INJURY

	Age	Sex	Date of Injury	Cause of Accident	Neurological Level	Defect	Early Mortality Due to Spinal Injury	Early Mortality Due to Other Injury	X-ray
1	73	M	20-7-53	TRAFFIC.	C 5	COMPLETE	ASPHYXIA		NBI. O.A
2	28	M	18-2-53	MACHINERY	L-D	COMPLETE			NAI
3	53	F	2-12-57	UNKNOWN	C 5	INCOMPLETE			NBI
4	63	M	27-6-57	WT. ON BACK INDUSTRIAL	T10	COMPLETE	PULM. OEDEMA	DIAPH. HERNIA	CRUSH # D. 9.10.11
5	61	M	3-2-55	TRAFFIC	C 5	COMPLETE	HYPOTHERMIA		# DIS. C.V. 4.5
6	68	F	28-8-55	TRAFFIC	C 6	INCOMPLETE		RUPTURED VISCUS	CRUSH # C.V 6
7	31	M	4-4-49	WT. ON BACK MINER	L-D	COMPLETE			# DIS L.V 1-2
8	32	M	18-6-51	WT ON BACK MINER	L-D	COMPLETE			CRUSH # L.V 1
9	42	M	18-5-49	WT. ON BACK MINER	T 11	COMPLETE			# DIS LV. 2-3
10	46	M	13-12-54	WT. ON BACK MINER	L-D	INCOMPLETE			# DIS DV. 12 LV 1.
11	37	M	20-4-54	WT. ON BACK MINER	L-D	COMPLETE			# DIS L.V 1-2
12	25	M	18-8-48	WT ON BACK MINER	L-D	COMPLETE			# DIS L.V. 4-5.
13	45	M	16-8-56	FELL 30'	C 5	COMPLETE	PULM. OEDEMA		# DIS C.V. 5-6
14	23	M	24-1-56	FELL OFF ROOF	DORSAL	INCOMPLETE			CRUSH # D.V. 9 10 11 12
15	17	M	8-9-54	FELL 3 STORIES	L-D	COMPLETE		RUPTURED VISCUS	UNKNOWN
16									
17	48	M	13-12-57	TRAFFIC	C 7	COMPLETE	BRONCHO PNEUMONIA		# DIS C.V. 7-8
18	40	M	18-8-48	FELL 30'	L-D	COMPLETE			# DIS D.V. 11-12
19	32	M	29-5-54	FELL 50'	L-D	INCOMPLETE			# DIS L.V. 1-2
20	54	M	19 5 45	FELL 30'	DORSAL	INCOMPLETE			CRUSH # D.V. 8
21	21	F	1940	UNKNOWN	L-D	INCOMPLETE			CRUSH # L.V. 1.
22	26	M	17 11 49	UNKNOWN	L-D	COMPLETE			# DIS L.V. 1
23	56	M	9-6-47	FELL 40'	L-D	INCOMPLETE			# T.P
24	?24	F	14-8-54	TRAFFIC	L-D	COMPLETE		ABDOM. BLEEDING	UNKNOWN

C = CERVICAL
L-D = LUMBO-DORSAL

N.B.I. = NO BONE INJURY
O.A. = OSTEO ARTHRITIS

	Age	Sex	Date of Injury	Cause of Accident	Neurological Level	Defect	Early Mortality Due to Spinal Injury	Due to Other Injury	X-ray
25	20	M	18·5·54	FELL OFF ROOF	L-D	INCOMPLETE			CRUSH # L.V.1
26	29	M	14·8·54	TRAFFIC	L-D	COMPLETE			# DIS L.V.1.
27	19	M	27·12·45	TRAFFIC	L-D	INCOMPLETE			# DIS L.V.2-3
28	18	M	24·8·53	FELL 20'	C7	INCOMPLETE			CRUSH # C.V.5
29	46	M	27·8·57	MACHINERY	C6	INCOMPLETE			DIS C.V.5-6
30	36	M	19·8·56	FELL DRUNK	C5-6	INCOMPLETE			N.B.I.
31	48	M	14·10·56	WT. ON BACK MINER	C7	INCOMPLETE			N.B.I. O.A.
32	61	M	26·5·57	WT. ON BACK	C5-6	COMPLETE	PULM. OEDEMA		DIS C.V.5-6 # SKULL
33	41	M	21·8·55	TRAFFIC	C5	INCOMPLETE			N.B.I.
34	24	M	3·10·55	FELL 4 STORIES	L-D	INCOMPLETE			CRUSH # D.V.12
35	21	M	26·11·49	FELL DRUNK	L-D	INCOMPLETE			CRUSH # L.V.4.
36	51	M	18·9·48	WT. ON BACK MINER	L-D	COMPLETE			# DIS D.V.11.
37	25	M	20·10·51	FELL 4 STORIES	L-D	INCOMPLETE			# DIS L.V.2-3
38	50	M	26·11·51	FELL DRUNK	L-D	INCOMPLETE			NIL O.A
39	61	M	7·1·58	FELL DRUNK	C7-8	INCOMPLETE			NIL O.A
40	53	M	30·9·58	FELL DRUNK	DORSAL	INCOMPLETE			CRUSH # D.V.7-10
41	62	M	19·10·58	TRAFFIC	C6	COMPLETE	PULM COLLAPSE		NIL O.A
42	37	M	19·7·56	WT. ON BACK	L-D	COMPLETE			# DIS L.V.1-2.
43	61	M	22·7·55	FELL SCAFFOLD	C6-7	COMPLETE	PULM. OEDEMA		DIS C.V.6-7
44	44	M	7·12·50	WT. ON BACK MINER	L-D.	COMPLETE			# DIS L.V.
44a	42	M	22·10·52	WT. ON BACK MINER	L-D	COMPLETE			UNKNOWN
45	48	M	18·11·56	WT. ON BACK MINER	L-D	COMPLETE			# DIS D.V.12.
45a	31	M	23·2·51	WT. ON BACK MINER	L-D	INCOMPLETE			# DIS D.V.11-12
46	35	M	27·3·46	WT. ON BACK MINER	L-D	INCOMPLETE			# DIS L.V.1-2
46a	44	M	25·9·51	UNKNOWN	L-D	INCOMPLETE			UNKNOWN
47	31	M	21·4·53	FELL SHAFT	DORSAL	COMPLETE			CRUSH # D.V.10.11.12
47a	38	M	17·2·55	FELL 20'	L-D	COMPLETE			# DIS L.V.1.
48	34	M	23·3·58	FELL 35'	D6	COMPLETE			CRUSH # D.V.4-6
49	29	M	26·2·56	FELL LADDER	L-D	COMPLETE			# DIS L.V.1

	ORTHOPAEDIC TREATMENT			TROPHIC SKIN ULCERATION			
	Conservative	Closed Reduction	Operative	Residual Spinal Movement	Incidence	Grafted	Necessitated Readmission
1	ORD BED						
2	ORD BED			GOOD	SEVERE TRANSIENT	YES	NO
3	ORD BED + COLLAR			UNKNOWN	UNKNOWN		
4	ORD. BED						
5	ORD. BED						
6	ORD. BED						
7		MANIPULATION & COMPLETE PLASTER		RESTRICTED	SEVERE PERMANENT	YES	YES
8	ORD. BED 6/52 PLASTER SHELL			GOOD	SEVERE PERMANENT	YES	YES
9	HIR BED			BAD	SEVERE PERMANENT	NO	NO
10			ORD. BED PLATED	GOOD	MINOR TRANSIENT	NO	NO
11			PLATED TURNING FRAME	UNKNOWN	SEVERE UNKNOWN	UNKNOWN	UNKNOWN
12			REDUCTION COMPLETE PLASTER	RESTRICTED	SEVERE PERMANENT	YES	YES
13		SKULL TRACTION ORD. BED					
14			LAMINECTOMY TURNING FRAME	GOOD	NONE	NO	NO
15	ORD. BED						
16							
17		SKULL TRACTION ORD. BED					
18	COMPLETE PLASTER TURNING FRAME 4/52			RESTRICTED	SEVERE PERMANENT	YES	YES
19	MECCANO BED			GOOD	SEVERE TRANSIENT	NO	NO
20	ORD. BED 7/12		LAMINECTOMY 7/12 BONE FUSION TURNING FRAME	RESTRICTED	MINOR TRANSIENT	NO	NO
21	COMPLETE PLASTER			UNKNOWN	SEVERE PERMANENT	UNKNOWN	UNKNOWN
22			PLATED ORD. BED	RESTRICTED	SEVERE PERMANENT	NO	YES
23	ORD. BED			GOOD	UNKNOWN		
24	ORD. BED						

ORD. = ORDINARY

	ORTHOPAEDIC TREATMENT			TROPHIC SKIN ULCERATION			
	Conservative	Closed Reduction	Operative	Residual Spinal Movement	Incidence	Grafted	Necessitated Readmission
25			LAMINECTOMY BONE FUSION TURNING FRAME		SEVERE PERMANENT	NO	NO
26			PLATED TURNING FRAME	GOOD	SEVERE PERMANENT	YES	YES
27			BONE FUSION TURNING FRAME	GOOD	SEVERE UNKNOWN	UNKNOWN	UNKNOWN
28			LAMINECTOMY COMPLETE P.O.P	GOOD	NONE	NO	NO
29		SKULL TRACTION COMPLETE P.O.P		GOOD	MINOR TRANSIENT	NO	NO
30	ORD. BED			GOOD	NONE	NO	NO
31	ORD. BED			GOOD	NONE	NO	NO
32		SKULL TRACTION ORD. BED					
33	ORD. BED			GOOD	SEVERE TRANSIENT		
34	ORD. BED			GOOD	NONE	NO	NO
35	PLASTER SHELL			UNKNOWN	UNKNOWN		
36			LAMINECTOMY TURNING FRAME	UNKNOWN	SEVERE PERMANENT	NO	YES
37			PLATED TURNING FRAME	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN
38			LAMINECTOMY ORD BED	GOOD	NIL	NO	NO
39	COMPLETE PLASTER +TURNING FRAME			RESTRICTED	SEVERE PERMANENT	NO	
40	ORD BED		G.	GOOD	NIL		
41	ORD BED						
42			PLATED TURNING FRAME	UNKNOWN	SEVERE TRANSIENT	NO	YES
43	ORD. BED						
44	WATER BED			RESTRICTED	SEVERE PERMANENT	NO	NO
44a	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN	SEVERE PERMANENT	NO	NO
45	ORD. BED			GOOD	MINOR TRANSIENT	NO	YES
45a	ORD. BED			GOOD	SEVERE TRANSIENT	NO	NO
46			UNSUCCESSFUL WIRING BONE GRAFT	GOOD	MINOR TRANSIENT	NO	NO

P.O.P = PLASTER OF PARIS

46a	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN
47	TURNING FRAME			GOOD	SEVERE TRANSIENT	YES	NO
47a	TURNING FRAME			GOOD	SEVERE PERMANENT	NO	NO
48	TURNING FRAME			GOOD	SEVERE TRANSIENT	NO	NO
49			PLATED TURNING FRAME	GOOD	SEVERE PERMANENT	NO	NO

NEUROSURGICAL PROBLEMS

	Clinically progressive Initially	Later	L.P. Block	Operations	Outcome
1	NIL	NIL			
2	NIL	NIL			
3	NIL	NIL			
4	NIL	NIL			
5	NIL	NIL			
6	NIL	NIL			
7	NIL	NIL			
8	NIL	NIL			
9	YES	NIL			
10	NIL	NIL			
11	UNKNOWN	UNKNOWN			
12	UNKNOWN	NIL			
13	NIL	NIL			
14	NIL	NIL	UNKNOWN	EARLY LAMINECTOMY EXTRA DURA CLOT	UNAFFECTED
15	NIL	NIL			
16					
17	NIL	NIL			
18	NIL	NIL			
19	NIL	NIL			
20	NIL	YES 5/12	COMPLETE BLOCK	LATE LAMINECTOMY BONE RIDGE	IMPROVED
21	UNKNOWN	UNKNOWN			
22	NIL	NIL			
23	NIL	YES 10/YEARS	NO BLOCK		

NEUROSURGICAL PROBLEMS

	Climically progressive Initially	Later	L.P. Block	Operations	Outcome
24	NIL	NIL			
25	YES	NIL	UNKNOWN	EARLY LAMINECTOMY	UNAFFECTED
26	YES	NIL	COMPLETE BLOCK	EARLY LAMINECTOMY ANT. COMPRESSION	UNAFFECTED
27	NIL	NIL	UNKNOWN		IMPROVED
28	YES	NIL	COMPLETE BLOCK	EARLY LAMINECTOMY COMPRESSION	IMPROVED
29	NIL	NIL			
30	NIL	NIL	NO BLOCK		
31	NIL	NIL			
32	NIL	NIL			
33	YES	NIL	NO BLOCK		
34	NIL	NIL	NO BLOCK		
35	NIL	NIL			
36	NIL	NIL	UNKNOWN	EARLY LAMINECTOMY DISC FRAGMENT	UNAFFECTED
37	YES	UNKNOWN	COMPLETE BLOCK	NIL	UNKNOWN
38	NIL	NIL	COMPLETE BLOCK	EARLY LAMINECTOMY	UNAFFECTED
39	NIL	NIL	NIL	NIL	
40	NIL	NIL	NO BLOCK	NIL	
41					
42	NIL	NIL			
43					
44	NIL	NIL	UNKNOWN		
44a	UNKNOWN	UNKNOWN		UNKNOWN	
45	NIL	NIL		NIL	
45a	NIL	YES	NO BLOCK	LATE LAMINECTOMY NO BLOCK	IMPROVED
46	NIL	NIL	COMPLETE BLOCK	LATE LAMINECTOMY CONSTRICTING LAMINA	IMPROVED
46a	UNKNOWN				
47	NIL	NIL	NO BLOCK	NIL	
47a	NIL	NIL		NIL	
48	NIL	NIL		NIL	
49	NIL	NIL		NIL	

URINARY PROBLEMS

	Initial	Sepsis	Stones	Residual State	Readmission for Urinary Sepsis
1					
2	NIL	NIL		CONTINENT	
3	NIL	NIL		CONTINENT	
4					
5					
6					
7	TIDAL DRAINAGE	SEVERE		INCONTINENT DIED OF PYELONEPH.	YES
8	TIDAL DRAINAGE	SEVERE		AUTONOMOUS BLADDER	NO
9	UNKNOWN	SEVERE		INCONTINENT	NO
10	INDWELLING CATH.	MINOR		INCONTINENT	NO
11	UNKNOWN	SEVERE		UNKNOWN	UNKNOWN
12	UNKNOWN	SEVERE		AUTOMATIC BLADDER	YES
13					
14	INDWELLING CATH.	MINOR		NORMAL	NO
15					
16					
17					
18	TIDAL DRAINAGE	SEVERE		DIED SUPRA PUBIC CATH.	YES
19	INDWELLING CATH.	SEVERE		INCONTINENT	YES
20	NIL	NIL		CONTINENT	NO
21	SUPRA PUBIC CATH.	UNKNOWN		DIED AUTOMATIC BLADDER	UNKNOWN
22	MANUAL COMPRESSION	SEVERE		DIED SUPRA PUBIC CATH.	YES
23	UNKNOWN	UNKNOWN		INCONTINENT	NO

CATH. = CATHETER

URINARY PROBLEMS

	Initial	Sepsis	Stones	Residual State	Readmission for Urinary Sepsis
24					
25	INDWELLING CATH.	SEVERE		INCONTINENT DIED OF SUPPURATION	NO
26	INDWELLING CATH.	SEVERE		INCONTINENT	NO
27	UNKNOWN	MINOR		CONTINENT	NO
28	NIL	NIL		CONTINENT	NO
29	INDWELLING CATH.	SEVERE		INCONTINENT	YES
30	NIL	NIL		NORMAL	NO
31	NIL	NIL		NORMAL	NO
32					
33	INDWELLING CATH.	SEVERE		CONTINENT	NO
34	INDWELLING CATH.	MINOR		AUTOMATIC BLADDER	NO
35	UNKNOWN	UNKNOWN		UNKNOWN	UNKNOWN
36	INDWELLING CATH.	SEVERE		INCONTINENT DIED OF AMYLOID	
37	INDWELLING CATH.	MINOR		UNKNOWN	
38	INDWELLING CATH.	MINOR		NORMAL	
39	GIBBON CATH.	SEVERE		DIED INCONTINENT	
40	NIL	NIL		NORMAL	
41					
42	FOLEY CATH.	MINOR		AUTOMATIC	
43					
44	INDWELLING CATH.	SEVERE		INCONTINENT	
44a	INDWELLING CATH.	SEVERE		INCONTINENT DIED PYELONEPH.	
45	INDWELLING CATH.	MINOR		AUTOMATIC WITH PRECIPITENCE	
45a	INDWELLING CATH.	SEVERE		CONTINENT WITH PRECIPITENCE	YES
46	TIDAL DRAINAGE	SEVERE		CONTINENT	YES
46a	UNKNOWN	UNKNOWN		UNKNOWN	YES
47	INDWELLING CATH.	SEVERE		AUTOMATIC	NIL
47a	TIDAL DRAINAGE	SEVERE		INCONTINENT	NIL
48	GIBBON CATH	SEVERE		INCONTINENT	NIL
49	TIDAL DRAINAGE	SEVERE		INCONTINENT	NIL

ABDOMINAL SYMPTOMS

	Dyspepsia	Diarrhoea	Abdominal pain	Investigations (X-rays)	Abdominal Operations
1					
2	NIL	NIL	NIL	NIL	NIL
3	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN
4					
5					
6					
7	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN
8	NIL	SEVERE BOUTS	SEVERE	NEGATIVE STOOL CULTURE	NIL
9	SEVERE	NIL	MILD	?DIAPHRAGMATIC HERNIA	NIL
10	NIL	NIL	NIL	NIL	NIL
11	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN
12	NIL	NIL	NIL	NIL	NIL
13					
14	SEVERE	NIL	NIL	NIL	NIL
15					
16					
17					
18	SEVERE	SEVERE BLOOD & MUCUS	UNKNOWN	NEGATIVE STOOL CULTURE ?AMYLOID DISEASE ?ULCERATIVE COLITIS	NONE
19	NIL	NIL	NIL	NIL	NIL
20	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN
21	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN
22	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN
23	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN
24					

ABDOMINAL SYMPTOMS

	Dyspepsia	Diarrhoea	Abdominal pain	Investigations (X-rays)	Abdominal Operations
25					
26	SEVERE COLD WEATHER	NIL	NIL	DUODENAL SPASM	NEGATIVE LAPAROTOMY FOR SUSPECTED PERFORATION
27	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN
28	SEVERE	NIL	NIL	NEGATIVE BARIUM MEAL	NIL
29	SEVERE WITH HAEMATEMESIS	SEVERE BOUTS	SEVERE	NEGATIVE BARIUM MEAL ? ACUTE EROSIONS NEGATIVE STOOL CULTURE	NIL
30	SEVERE	NIL	MILD	NIL	NIL
31	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN
32					
33	NIL	NIL	NIL	NIL	NIL
34	NIL	NIL	NIL	NIL	NIL
35	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN
36	NIL	SEVERE BOUTS	MILD	AMYLOID DISEASE NEGATIVE STOOL CULTURE	NIL
37	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN
38	MILD				
39		SEVERE BOUTS	SEVERE	NIL NEGATIVE STOOL CULTURE	NIL
40	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN
41					
42	SEVERE			NEGATIVE BARIUM MEAL	NIL
43					
44	NIL	NIL	NIL	NIL	NIL
44a	UNKNOWN				
45	MILD	NIL		NIL	NIL
45a	SEVERE	NIL	NIL		NEGATIVE LAPARCTOMY FOR PYLORIC STENOSIS
46	SEVERE	SEVERE BOUTS		NEGATIVE BARIUM MEAL	NEGATIVE LAPAROTOMY CHOLECYSTECTOMY
46a	UNKNOWN				
47	SEVERE HAEMATEMESIS	NIL	SEVERE	NEGATIVE BARIUM MEAL	NIL
47a	NIL	NIL	MILD	NIL	NIL
48	NIL	NIL	NIL	NIL	NIL
49	NIL	NIL	SEVERE	NIL	NIL

NEUROLOGICAL FINDINGS

	Initial Severity	Late Reflex Pattern	Recovery	Involuntary Movements Extent Early	Extent Late	Type
1	COMPLETE					
2	COMPLETE	U.M.N.	FULL MOBILE	MODERATE	MILD	FLEXOR
3	INCOMPLETE	U M.N.	FULL MOBILE	MODERATE	UNKNOWN	FLEXOR
4	COMPLETE					
5	COMPLETE	HYPOTHERMIA				
6	INCOMPLETE					
7	COMPLETE	L.M.N.	NIL	NIL	NIL	NIL
8	COMPLETE	L.M N.	NIL		MILD	EXTENSOR
9	COMPLETE	L.M.N.	NIL	NIL	NIL	NIL
10	INCOMPLETE	U.M.N.	SLIGHT	NIL	NIL	NIL
11	COMPLETE	UNKNOWN	UNKNOWN	UNKNOWN		UNKNOWN
12	COMPLETE	L.M.N.	NIL	MODERATE	MILD	FLEXOR
13	COMPLETE					
14	INCOMPLETE	U.M.N.	ALMOST FULL STIFF	MILD	MILDER	EXTENSOR
15	COMPLETE					
16						
17	COMPLETE					
18	COMPLETE	L.M N.	NIL	NIL		NIL
19	INCOMPLETE	L.M.N.	NIL	NIL		NIL
20	INCOMPLETE	U.M N.	ALMOST FULL STIFF	UNKNOWN		UNKNOWN
21	INCOMPLETE	L M.N.	NIL	UNKNOWN		UNKNOWN
22	COMPLETE	L.M N.	NIL	UNKNOWN		UNKNOWN
23	INCOMPLETE	U.M.N.	NIL	UNKNOWN	MODERATE	FLEXOR

U.M.N. = UPPER MOTOR NEURON
L.M.N. = LOWER MOTOR NEURON

NEUROLOGICAL FINDINGS

	Initial Severity	Late Reflex Pattern	Recovery	Involuntary Movements Extent Early Late	Involuntary Movements Type
24	COMPLETE				
25	INCOMPLETE	L.M.N.	NIL		
26	COMPLETE	L.M.N.	NIL	NIL	NIL
27	INCOMPLETE	L.M.N.	SLIGHT	UNKNOWN	UNKNOWN
28	INCOMPLETE	U.M.N.	ALMOST FULL STIFF	NIL	NIL
29	INCOMPLETE	U.M.N.	SLIGHT	SEVERE MODERATE	FLEXOR + EXTENSOR
30	INCOMPLETE	U.M.N.	ALMOST FULL STIFF	MODERATE	EXTENSOR
31	INCOMPLETE	U.M.N.	FULL MOBILE	NIL	NIL
32	COMPLETE				
33	INCOMPLETE	U.M.N.	SLIGHT	SEVERE	FLEXOR
34	INCOMPLETE	L.M.N.	FULL	NIL	NIL
35	INCOMPLETE	L.M.N.	SLIGHT	UNKNOWN	UNKNOWN
36	COMPLETE	UNKNOWN	NIL	UNKNOWN	UNKNOWN
37	INCOMPLETE	L.M.N.	UNKNOWN	UNKNOWN	UNKNOWN
38	INCOMPLETE	U.M.N.	ALMOST FULL STIFF	MILD	INDETERMINATE
39	INCOMPLETE	U.M.N.	SLIGHT	MILD	INDETERMINATE
40	INCOMPLETE	NORMAL	FULL NORMAL	NIL	NIL
41	COMPLETE				
42	COMPLETE	L.M.N.	NIL	UNKNOWN	UNKNOWN
43	COMPLETE				
44	COMPLETE	L.M.N.	NIL	NIL	NIL
44a	COMPLETE	UNKNOWN	UNKNOWN	UNKNOWN	
45	COMPLETE	MIXED	NIL	VERY MILD	? FLEXOR
45a	INCOMPLETE	U.M.N.	SLIGHT STIFF	SEVERE MODERATE	EXTENSOR + FLEXOR
46	INCOMPLETE	L.M.N.	SLIGHT	VERY MILD	? EXTENSOR
46a	INCOMPLETE	L.M.N.	SLIGHT	UNKNOWN	UNKNOWN
47	COMPLETE	L.M.N.	NIL	VERY FAINT	NIL
47a	COMPLETE	MIXED	NIL	MODERATE MILD	EXTENSOR + FLEXOR
48	COMPLETE	MIXED	NIL	SEVERE MODERATE	EXTENSOR + FLEXOR
49	COMPLETE	MIXED	SLIGHT	MILD	EXTENSOR

U.M.N. = UPPER MOTOR NEURON

L.M.N = LOWER MOTOR NEURON

	Pain Type	Late Sex State
1		
2	NIL	NORMAL - VIABLE CONCEPTION 7YRS
3	UNKNOWN	UNKNOWN
4		
5		
6		
7	VISCERAL	UNKNOWN
8	ROOT AND VISCERAL	DESIRE NORMAL NO SENSATION HAS ERECTIONS + EMISSIONS
9	VISCERAL	NO ERECTIONS, EMISSIONS, SENSATION OR DESIRE
10	NIL	UNKNOWN
11	UNKNOWN	UNKNOWN
12	BURNING DIFFUSE	UNKNOWN
13		
14	VISCERAL	HAS ERECTIONS, EMISSIONS LOST DESIRE
15		
16		
17		
18	UNKNOWN	UNKNOWN
19	NIL	2 VIABLE CONCEPTIONS
20	UNKNOWN	UNKNOWN
21	UNKNOWN	UNKNOWN
22	BURNING DIFFUSE	UNKNOWN
23	UNKNOWN	UNKNOWN
24		

	Pain Type	Late Sex State
25		
26	BURNING DIFFUSE, VISCERAL	HAS EMISSIONS, DESIRE NO ERECTIONS
27	UNKNOWN	UNKNOWN
28	VISCERAL	NORMAL 2 VIABLE CHILDREN
29	BURNING DIFFUSE	HAS EMISSIONS NO DESIRE OR ERECTIONS
30	VISCERAL	NORMAL
31	UNKNOWN	UNKNOWN
32		
33	NIL	NORMAL
34	NIL	HAS ERECTION, DESIRE, PREMATURE EMISSION, NO INTERCOURSE
35	UNKNOWN	UNKNOWN
36	UNKNOWN	SPONTANEOUS ERECTIONS
37	UNKNOWN	UNKNOWN
38	BURNING DIFFUSE PAIN BACK AND HIPS	HAS ERECTIONS, EMISSIONS LIBIDO REDUCED
39	VISCERAL	UNKNOWN
40	UNKNOWN	UNKNOWN
41		
42	UNKNOWN	UNKNOWN
43		
44	SEVERE LOW SPINAL PAIN	NO ERECTIONS, EMISSIONS OR REFLEXES
44a	UNKNOWN	UNKNOWN
45	ROOT	NO DESIRE, ERECTIONS! OR EMISSIONS
45a	VISCERAL	NORMAL - VIABLE CHILD
46	ROOT	HAS DESIRE, SPONTANEOUS EMISSIONS. NO ERECTIONS. INTERCOURSE
46a	BURNING DIFFUSE BILATERAL CORDOTOMY	UNKNOWN
47	ROOT BURNING DIFFUSE	HAS ERECTIONS DESIRE REDUCED
47a	VISCERAL	NO EMISSIONS, INTERCOURSE HAS ERECTIONS - REDUCED DESIRE
48	ROOT	HAS ERECTIONS NO EMISSIONS - SUBDUED DESIRE
49	VISCERAL	HAS ERECTIONS LATE EMISSION - SUBDUED DESIRE

MORBIDITY

	Initial Hospitalisation	Ultimate State	Gainful Employment	Late Oedema	Late Ossification
1					
2	18 MONTHS	FULLY ACTIVE	FULL	NIL	NIL
3	5 MONTHS	FULLY ACTIVE	UNKNOWN	UNKNOWN	UNKNOWN
4					
5					
6					
7	8 MONTHS	DIED 10 YEARS CHRONIC SEPSIS PYELONEPHRITIS	NIL	NIL	NIL
8	24 MONTHS	CHAIR RIDDEN	NIL	NIL	NIL
9	16 MONTHS	SEMI BEDRIDDEN	NIL	SEVERE	SEVERE - HIPS
10	18 MONTHS	CHAIR RIDDEN	NIL	NIL	NIL
11	16 MONTHS	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN
12	34 MONTHS	SEMI BEDRIDDEN	NIL	NIL	NIL
13					
14	5 MONTHS	FULLY ACTIVE	NIL	NIL	NIL
15					
16					
17					
18	33 MONTHS	DIED 10 YEARS CHRONIC SEPSIS	NIL	NIL	NIL
19	7 MONTHS	FULLY WT. BEARING	NIL	TRANSIENT	NIL
20	13 MONTHS	FULLY WT. BEARING	UNKNOWN	NIL	NIL
21	14 MONTHS	DIED 13 YEARS CHRONIC SEPSIS	1 YEAR IN 12	UNKNOWN	UNKNOWN
22	48 MONTHS	DIED 4 YEARS CHRONIC SEPSIS	NIL	NIL	NIL
23	1 MONTH	FULLY WT. BEARING	UNKNOWN	NIL	NIL
24					
25	3 MONTHS	DIED 3 MONTHS ACUTE SEPSIS	NIL	NIL	NIL

MORBIDITY

	Initial Hospitalisation	Ultimate State	Gainful Employment	Late Oedema	Late Ossification
26	17 MONTHS	CHAIR RIDDEN	NIL	NIL	NIL
27	8 MONTHS	FULLY WT. BEARING	UNKNOWN	NIL	AROUND KNEE
28	5 MONTHS	FULLY ACTIVE	FULL	NIL	NIL
29	18 MONTHS	SEMI BEDRIDDEN	NIL	NIL	NIL
30	3 MONTHS	FULLY WT. BEARING	SPORADIC	NIL	NIL
31	10 DAYS	FULLY ACTIVE	UNKNOWN	NIL	NIL
32					
33	22 MONTHS	CHAIR RIDDEN	NIL	NIL	NIL
34	5 MONTHS	FULLY ACTIVE	SPORADIC	NIL	NIL
35	5 MONTHS	FULLY WT. BEARING	UNKNOWN	NIL	NIL
36	29 MONTHS	DIED 4 YEARS CHRONIC SEPSIS	UNKNOWN	SEVERE	NIL
37	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN
38	4 MONTHS	FULLY ACTIVE	FULL	NIL	NIL
39	24 MONTHS	DIED 2 YEARS CHRONIC SEPSIS BED RIDDEN	NIL		
40	3 WEEKS	FULLY ACTIVE	UNKNOWN	NIL	NIL
41					
42	5 MONTHS	SEMI BEDRIDDEN	NIL	NIL	NIL
43					
44	29 MONTHS	CHAIR RIDDEN	NIL	NIL	NIL
44a	14 MONTHS	DIED 7 YEARS CHRONIC SEPSIS BED RIDDEN	NIL	UNKNOWN	UNKNOWN
45	UNKNOWN	JUST WT. BEARING	NIL	NIL	NIL
45a	10 MONTHS	FULLY ACTIVE	NIL	NIL	NIL
46	20 MONTHS	WT. BEARING CALLIPER	NIL	SEVERE	NIL
46a	UNKNOWN	ACTIVE	UNKNOWN	UNKNOWN	UNKNOWN
47	5 MONTHS	CHAIR RIDDEN	NIL	NIL	NIL
47a	10 MONTHS	CHAIR RIDDEN	NIL	NIL	NIL
48	5 MONTHS	CHAIR RIDDEN	NIL	NIL	NIL
49	10 MONTHS	CHAIR RIDDEN	NIL	MODERATE	NIL

SPINAL CORD INJURIES. A CLINICAL, PATHOLOGICAL, AND EXPERIMENTAL STUDY.

VOLUME 11.

by

Shedden Alexander.

Thesis presented for the Degree of Master of Surgery in the University of Glasgow.

September, 1963.

PART ONE.

A CLINICAL REVIEW OF 52 CASES OF CLOSED SPINAL CORD INJURY.

LEGENDS AND FIGURES.

FIGURE 1.

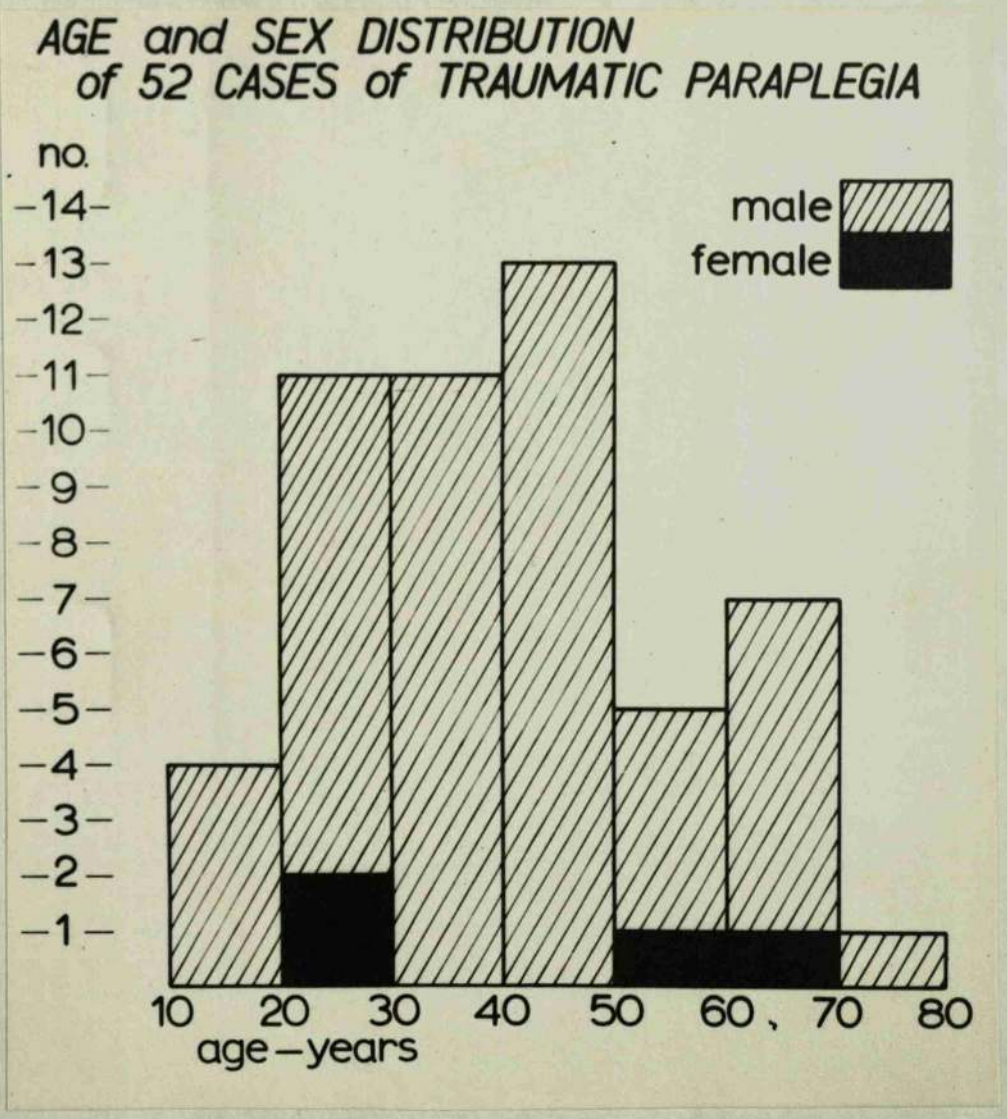

AGE and SEX DISTRIBUTION
of 52 CASES of TRAUMATIC PARAPLEGIA
no.
14
13
12
11
10
9
8
7
6
5
4
3
2
1
male
female
10
20
30
40
50
60
70
80
age—years

TABLE 1

Effect of the type of injury on the level of the spinal injury and on the early mortality in injuries of the spinal cord.

Type of Injury	Level of Spinal Injury				
	Cervical	Dorso-lumbar	Totals	Early Mortality	
Falls from a height	3	13	16	3	19%
Mining Accidents	1	12	13	0	0%
Traffic Accidents	6	3	9	6	66%
Falls when drunk	2	3	5	0	0%
Industrial Accidents	2	3	5	2	40%
Unknown	-	-	4	-	-
			52	11	21%

TABLE 2.

Relationship between the level of spinal injury, the severity of the initial neurological deficit and early mortality in injuries of the spinal cord.

Severity of Initial Neurological Deficit.		Level of Spinal Injury			Total
		Cervical	Dorsal	Lumbo-dorsal	
		15	6	31	52
Complete	Total	7	3	18	28
	Survivors	0	2	16	18
	Early Mortality	7	1	2	10
Incomplete.	Total	8	3	13	24
	Survivors	7	3	13	23
	Early Mortality	1	0	0	1
Died.	Primarily due to C.N.S. injury.	7	0	0	
	Primarily due to concomitant injury.	1	1	2	

TABLE 3

Relationship between the level of spinal injury and radiological evidence of associated injury to the vertebral column in injuries of the spinal cord.

Level of Injury to Spine		Radiological evidence of bone injury	No radiological evidence of bone injury	Initial radiology unknown
Cervical	15	8	7	
Dorsal	6	6	0	
Lumbodorsal	31	25	2	4
Total	52	39	9	4

TABLE 4

Relationship between open and closed methods of treating the spinal injury and the incidence of pressure sores and residual stiff back in injuries of the spinal cord.

Treatment of Injured spine	Cervical Spine		Dorsal and lumbar spine		
CLOSED METHODS	Survivors	Having severe pressure sores	Survivors	Having severe pressure sores	Having residual stiff back
Ordinary Bed	4	1	6	2	
Air Bed			1	1	
Water Bed			1	1	
Meccano Bed			1	1	
Complete plaster	1	1	2	2	
Plaster Shells			2	1	
Turning frame			3	3	
Closed reduction and P.O.P.			1	1	
Skull traction	1	0			
Total: 23	6	2	17	12	4 (24%)
OPEN METHODS					
Laminectomy	1	0	3	1	
Laminectomy with fusion			4	2	
Open reduction			1	1	
Open reduction and plating			7	5	
Total: 16	1	0	15	9	4 (27%)
Methods unknown: 2			2	1	
GRAND TOTALS: 41	7	2	34	22	8 (25%)

TABLE 5

Effect of laminectomy performed in the early and late stages on neurological deficit in injuries of the spinal cord.

		Improvement in neurological deficit	No improvement in neurological deficit
Early Laminectomy	6	1 (Cervical)	5 (1 dorsal, 4 lumbo-dorsal)
Late Laminectomy	3	3 (All lumbo-dorsal)	0

TABLE 6

Relationship between the degree of neurological recovery and the residual reflex pattern in injuries of the spinal cord

	RESIDUAL NEUROLOGICAL DEFICIT				
	COMPLETE		INCOMPLETE		
Residual Reflex Pattern	Degree of Recovery: Nil	Degree of Recovery: Major or Negligible	Degree of Recovery: Nil	Degree of Recovery: Major or Negligible	Unknown
Normal 1	Nil	0	1		
Upper motor neurone 14	Nil	0	8	6	
Lower motor neurone 19	Nil	10	1	7	1
Mixed 4	Nil	3		1	
Unknown 3		0			3
41		13			

TABLE 7

Relationship of pressure sores to the residual neurological deficit and reflex pattern in injuries of the spinal cord

Ultimate Reflex Pattern	Totals	COMPLETE DEFICIT		INCOMPLETE DEFICIT		
		Pressure Sores		Pressure Sores		
		Severe	Negligible	Severe	Negligible	Unknown
Normal	1	-	-		1	
Upper motor neurone	14	-	-	4	8	2
Lower motor neurone	19	10	Nil	4	2	3
Mixed	4	2	1	1	-	
Unknown	3	3				
Totals	41	15	1	9	11	5

TABLE 8

Effect of initial treatment of the bladder on the incidence of urinary sepsis in injuries of the spinal cord.

Initial bladder treatment	Number	Developing Severe Urinary Sepsis	Permanently Incontinent	Dying from renal sepsis
Bladder drained per urethram - Indwelling catheter - Foley	17	10	9	3
-Indwelling catheter - Gibbon	2	2	2	1
Tidal drainage	6	6	4	2
Intermittent catheterisation	0	0		
Suprapubic catheterisation	1	1	1	1
Manual compression	1	1	1	1
Total	27	20(74%)	17	8
Bladder not drained-Total	7	0	0	0
Bladder status unknown - Total	7	2	2	0

TABLE 9.

Duration of continuous hospitalisation of those surviving the acute phase of injuries of the spinal cord.

	No,	Less than 1 Year	Between 1 and 2 Years	Over 2 Years	Undetermined Period	Average Stay	Died
Survivors	41	20	11	7	3	13½ months	8

TABLE 10

Employment record of those surviving the acute phase in injuries of the spinal cord.

	Number	%
Never gainfully employed	24	80
Sporadically gainfully employed	3	10
Continuously gainfully employed	3	10
Employment record unknown	11	-
	41	

PART TWO.

OBSERVATIONS ON THE PATHOLOGY OF THE INJURED SPINAL CORD.

LEGENDS AND FIGURES

PLATE 1.

Photomicrograph of upper cervical cord in Case 1. Cord examined 1 hour after injury.

The upper plate shows the cytoplasm of one or two axis cylinders, indicated by an arrow, to have been replaced by a series of ragged, saccular swellings which give the axon the appearance of a piece of burned string.

The lower plate shows two small haemorrhages and a fissure; at the margin of the latter is a saccular axon swelling, indicated by an arrow.

(Bodian X 210)

PLATE 1.

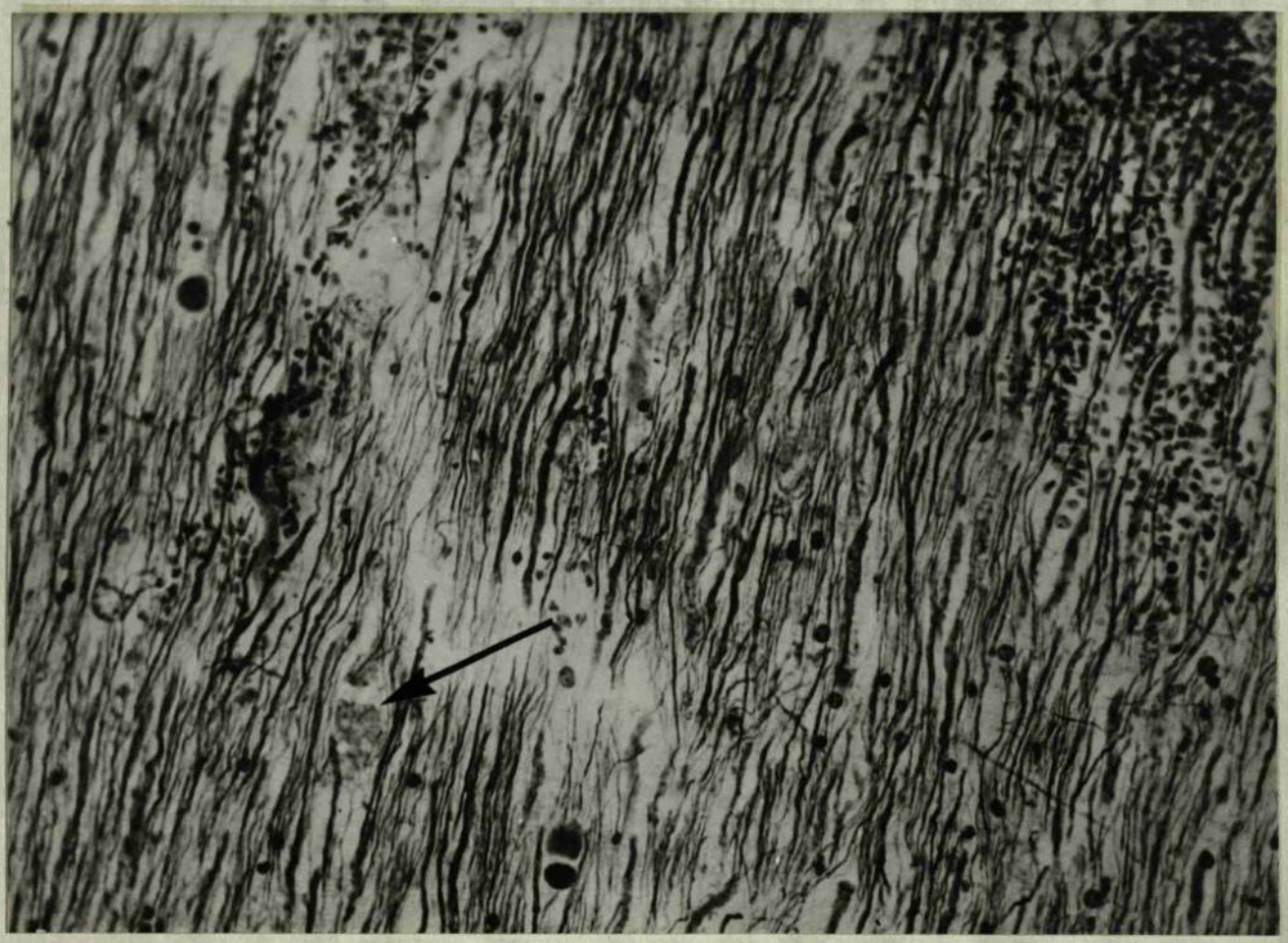


PLATE 2.

Photomicrograph of servical cord in Case 2. Cord examined 2 days after injury.

There is extensive structural damage and few axis cylinders are recognisable. The cytoplasm of damaged axons has extruded to give an amorphous background of fine granular debris. There are numerous cystic spaces which give the appearance of a sieve to the tissue. These spaces appear to have been formed by precipitation of axon cytoplasm round the rim. There is a mild infiltration with small dark round cells.

(Bodian X 210)

PLATE 2.

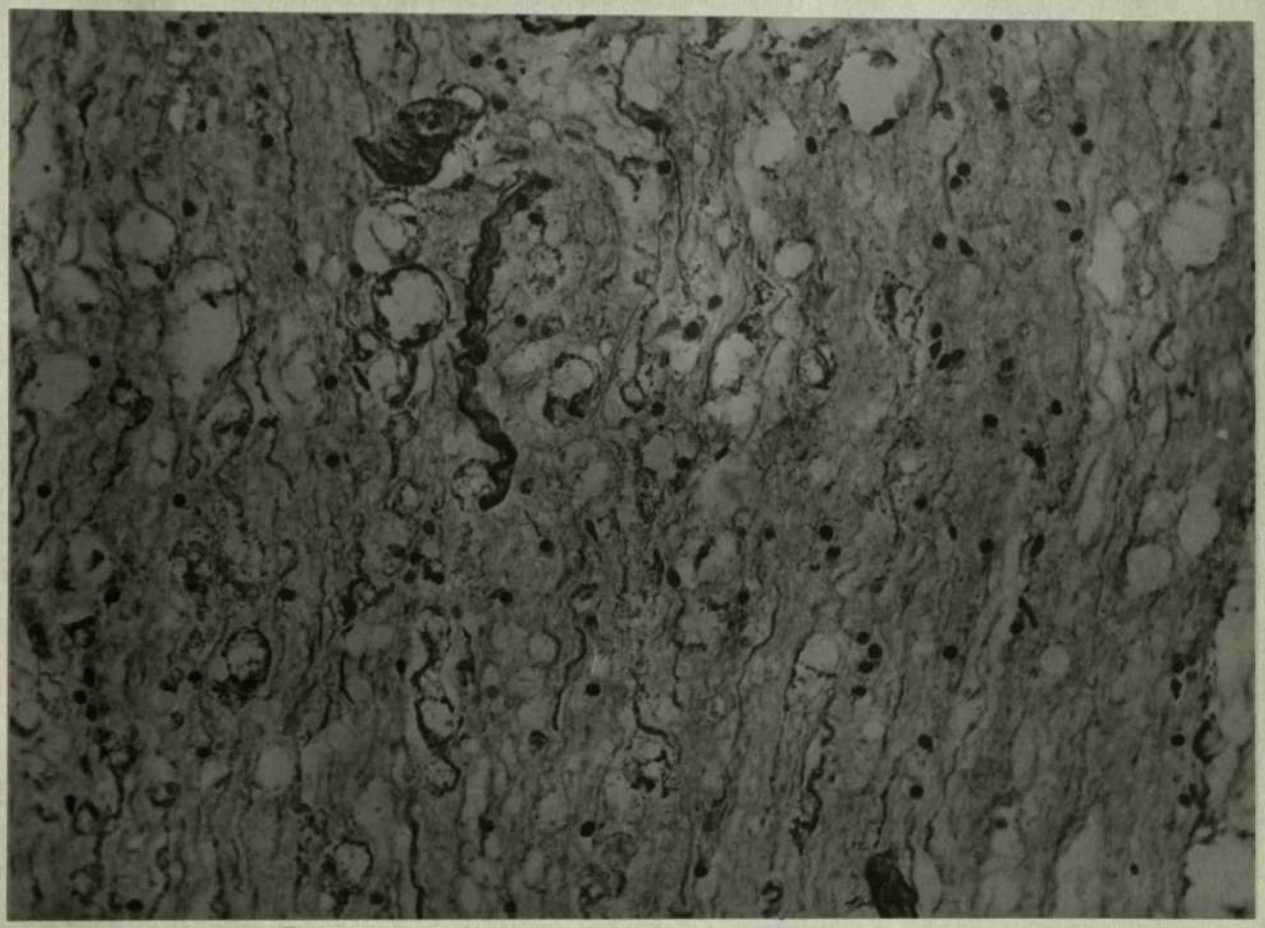


PLATE 3.

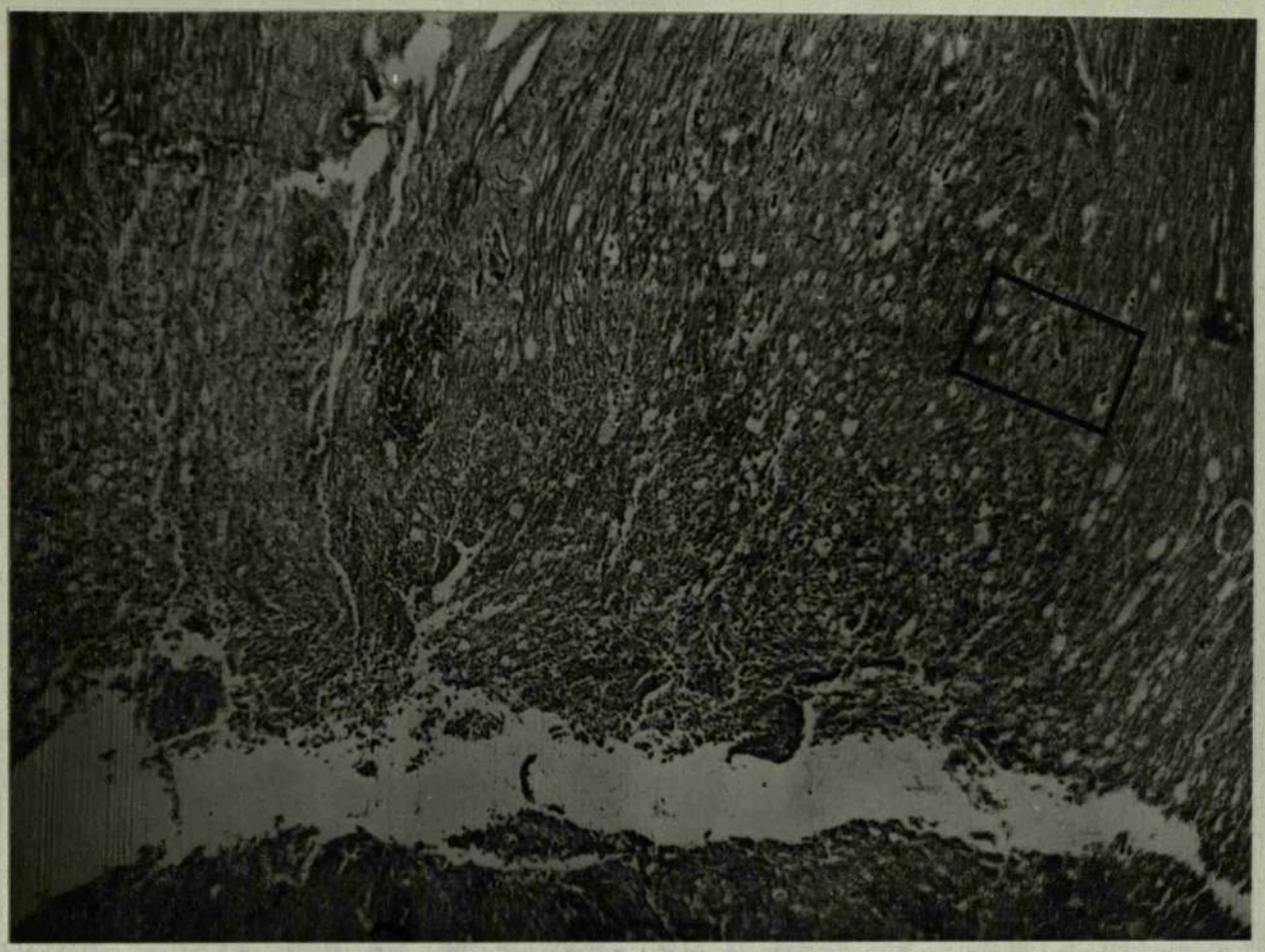
Photomicrograph of cervical cord in Case 3. Cord examined 5 days after injury.

The upper plate shows the edge of a contused area at the bottom of the field, sharply demarcated from the rest of the cord. Numerous terminal axon figures are seen at the junctional zone of neighbouring cord. (X 24).

The lower plate shows a detail of the above. There are many giant sterile axon globes and the cytoplasm is coarse and lacks homogeneous avidity for the silver stain.

(Bodian X 210)

PLATE 3.



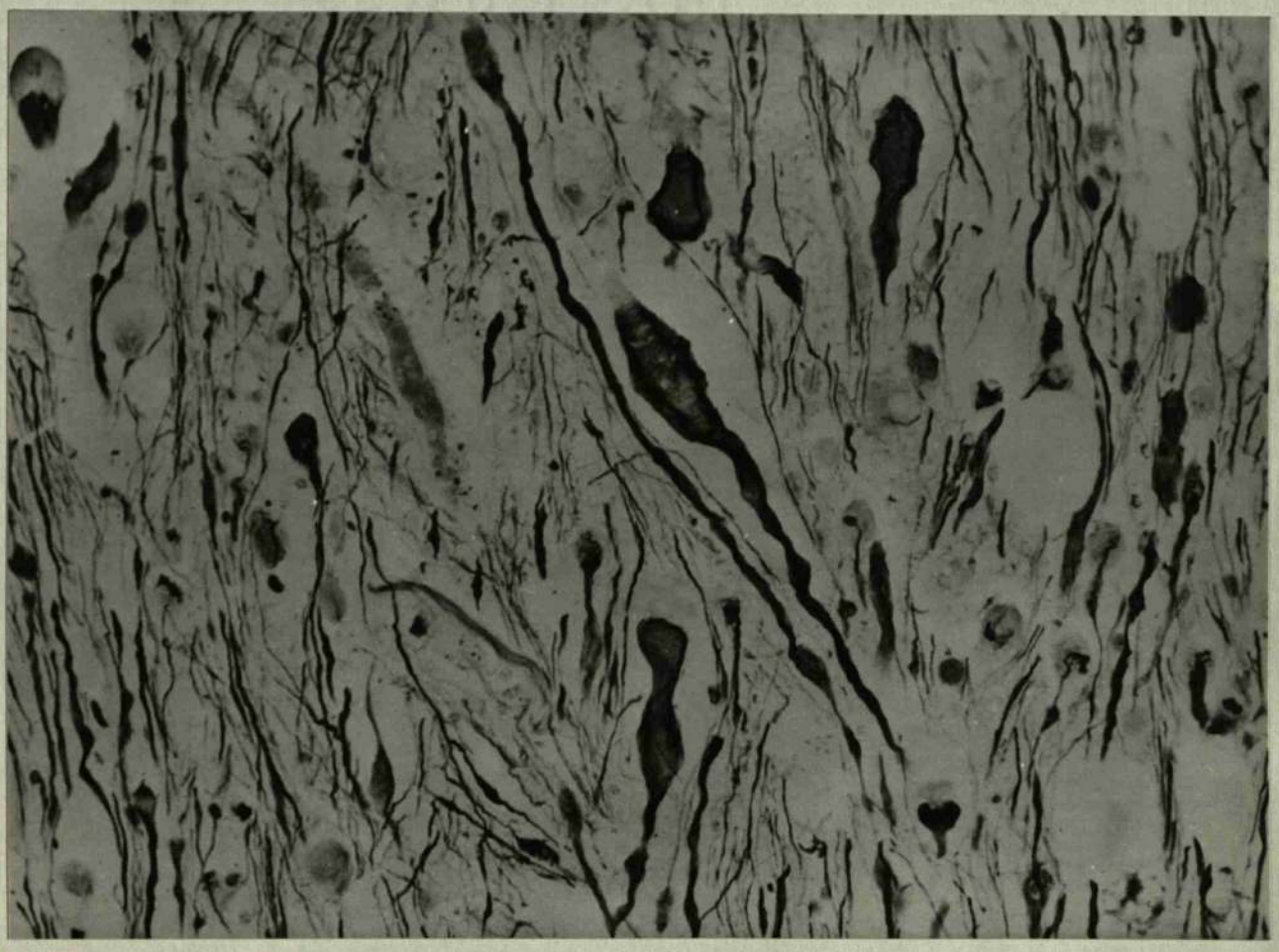


PLATE 4.

Photomicrograph of upper dorsal cord in Case 4. Cord examined 7 weeks after injury.

Young root fibres are seen streaming in towards the cord where they appear to be arrested. Several terminal axon figures, indicated by an arrow, are situated at the junctional zone. Deeper to this, in the cord substance, axis cylinders are attenuated and ragged, and there is a moderate increase in number of darkly staining neuroglial cells.

(Bodian X 210)

PLATE 4.

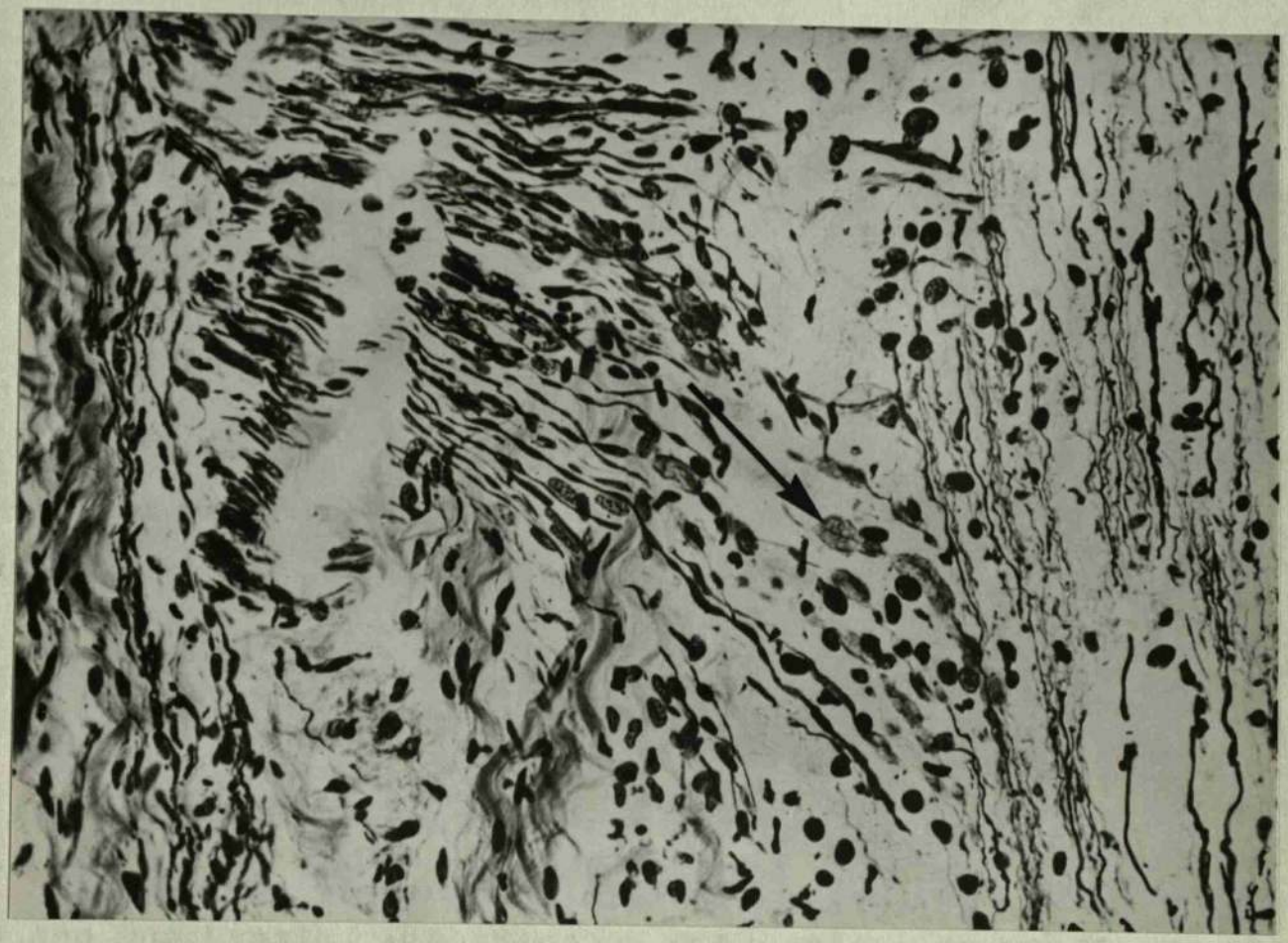


PLATE 5.

Photomicrograph of cord at lumbo-dorsal junction in Case 5.

Axis cylinders are reduced in number, and cytoplasm, extruded from damaged axons, forms a background of fine amorphous debris. Neuroglial cells are numerous. Their cytoplasm is enlarged and possesses greater avidity for silver stain than usual. The nucleus of these cells is often eccentrically placed and one or two are multinucleated.

(Bodian X 210)

PLATE 5.

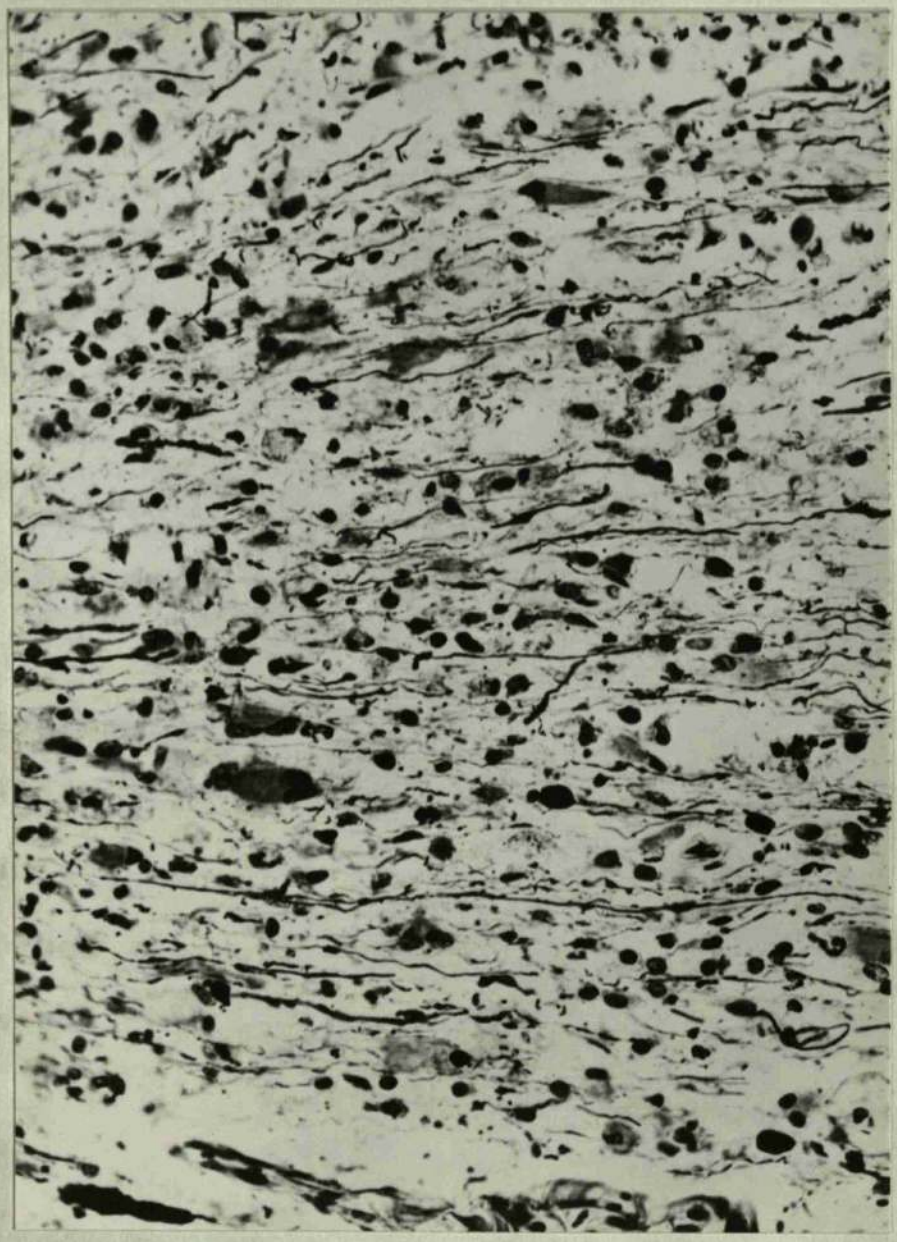


PLATE 6.

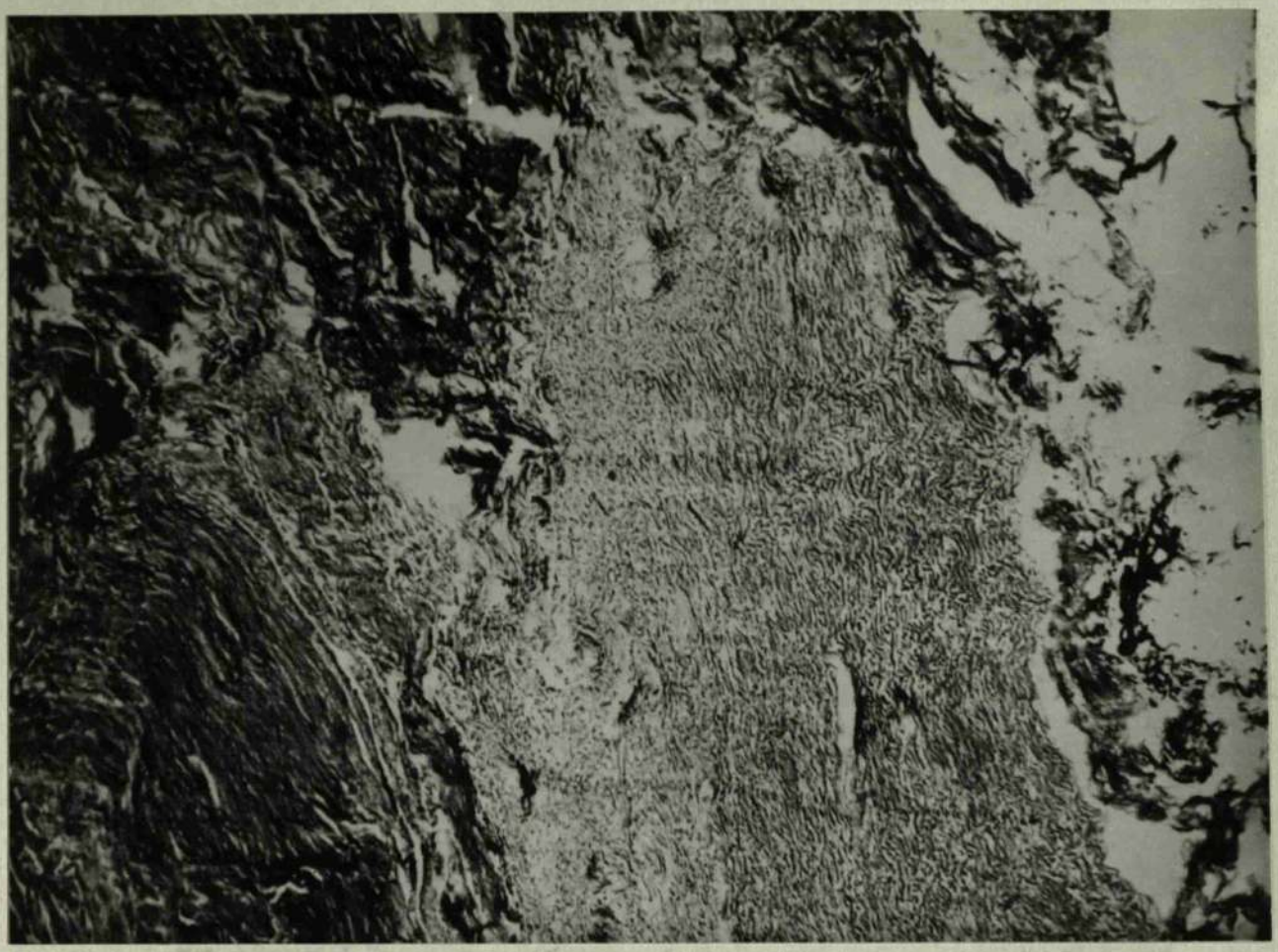
Photomicrograph of lower dorsal cord in Case 6. Cord examined 5 years after injury.

The upper plate shows the gliosed cord remnant framed by luxuriant scarring of the menninges. (X 40)

The lower plate shows a number of, apparently surviving axons. A moderate increase in neuroglial cells, without evidence of large argentophil forms, comprises the mature glial scar.

(Bodian X 210)

PLATE 6.



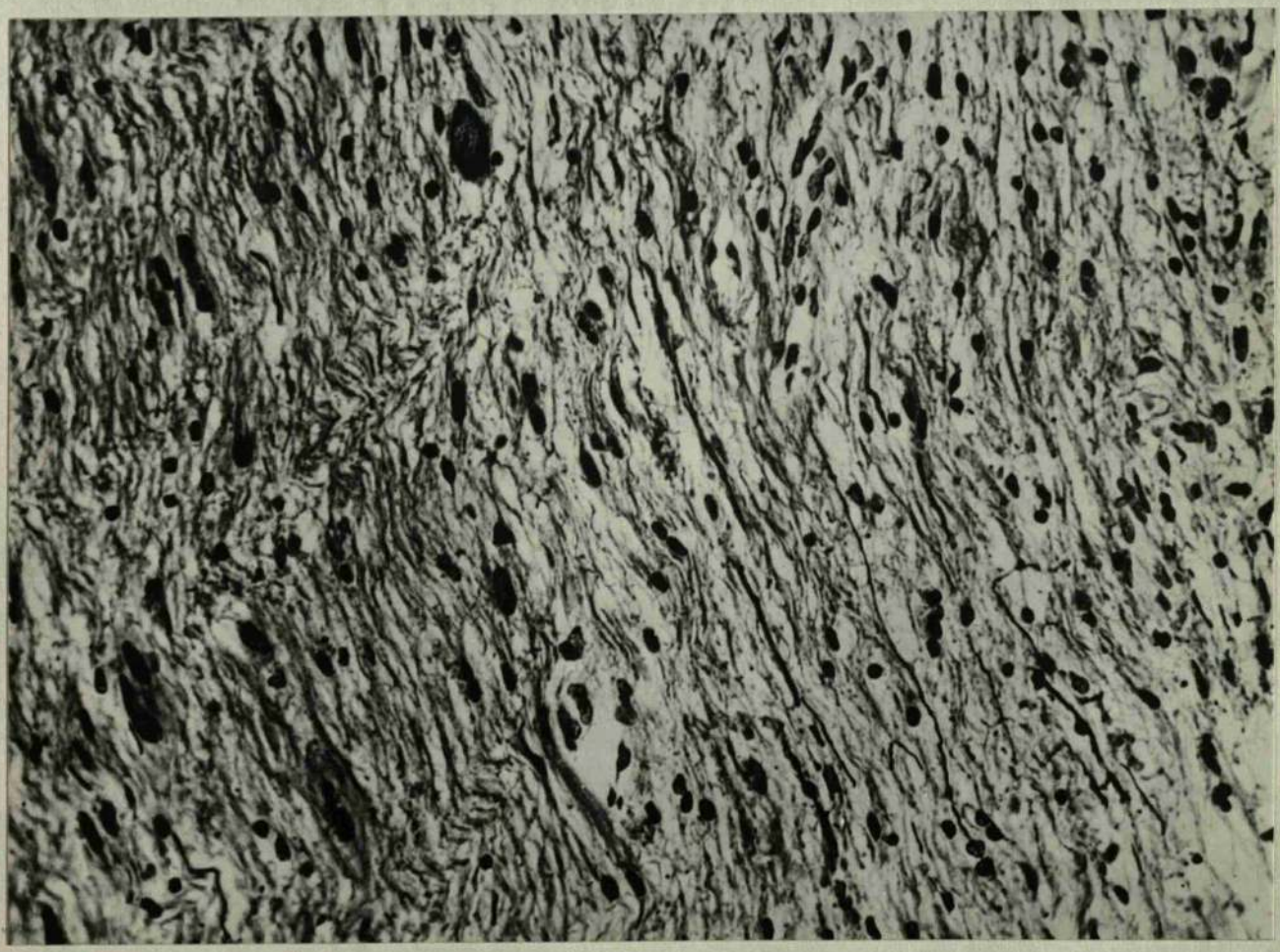


PLATE 7.

Photomicrograph of cord at the cervico-dorsal junction in Case 7. Cord examined 2 years after injury.

There is a wedge of demyelination in both lateral columns.

(Weigert X 16)

PLATE 7.

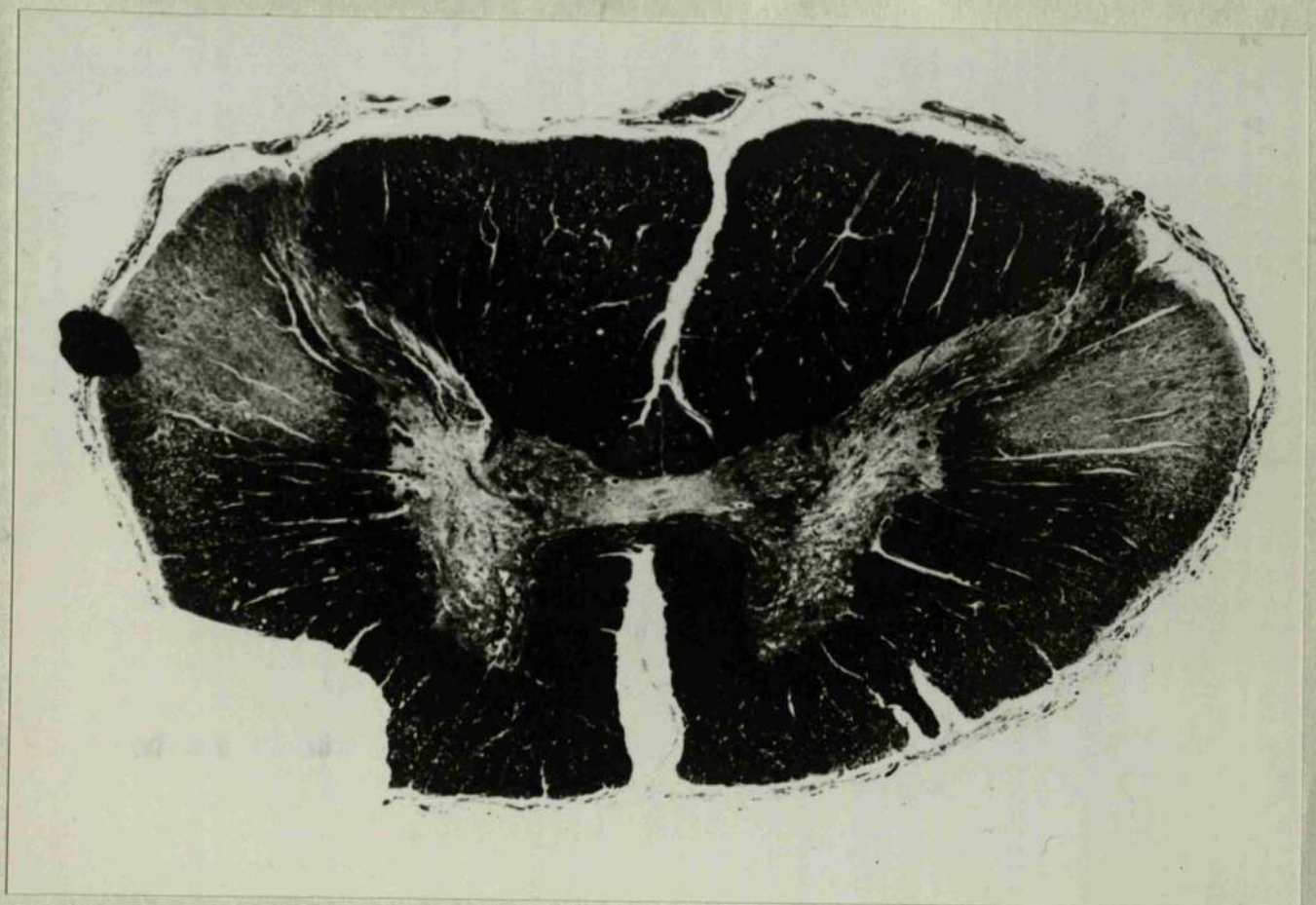


PLATE 8.

Photomicrograph of the anterior horn of cord at the cervico-dorsal junction in Case 7. Cord examined 2 years after injury.

There is pyknosis and shrinkage indicating destruction of anterior horn cells. The rest of the grey matter structure is poorly defined.

(unna Pap X 135)

PLATE 8.

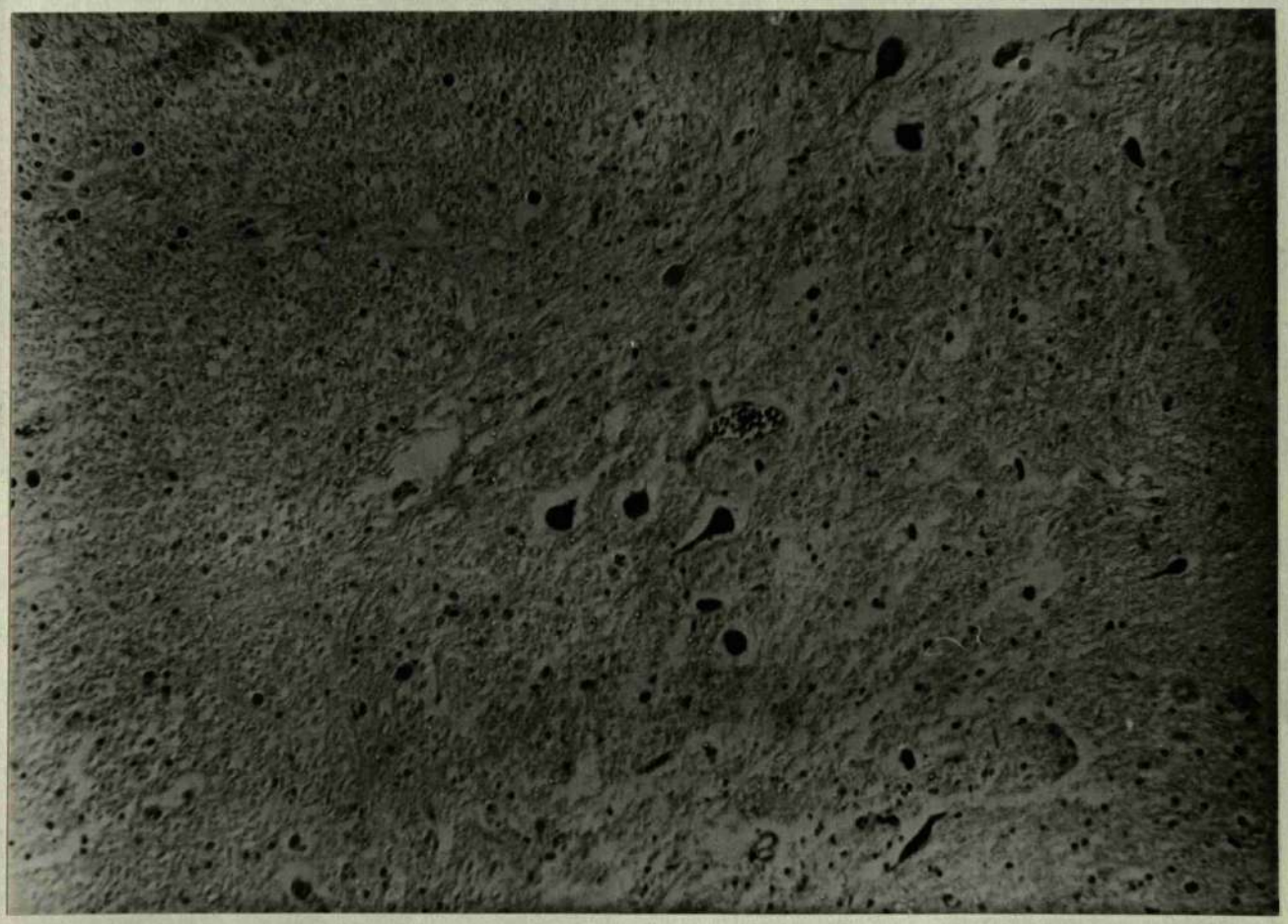


PLATE 9.

Photomicrograph of the demyelinated lateral columns of cord at the cervico-dorsal junction in Case 7. Cord examined 2 years after injury.

Axis cylinders are well preserved. Note the presence of numerous corpora amylacea.

(Bielschowsky X 225)

PLATE 9.

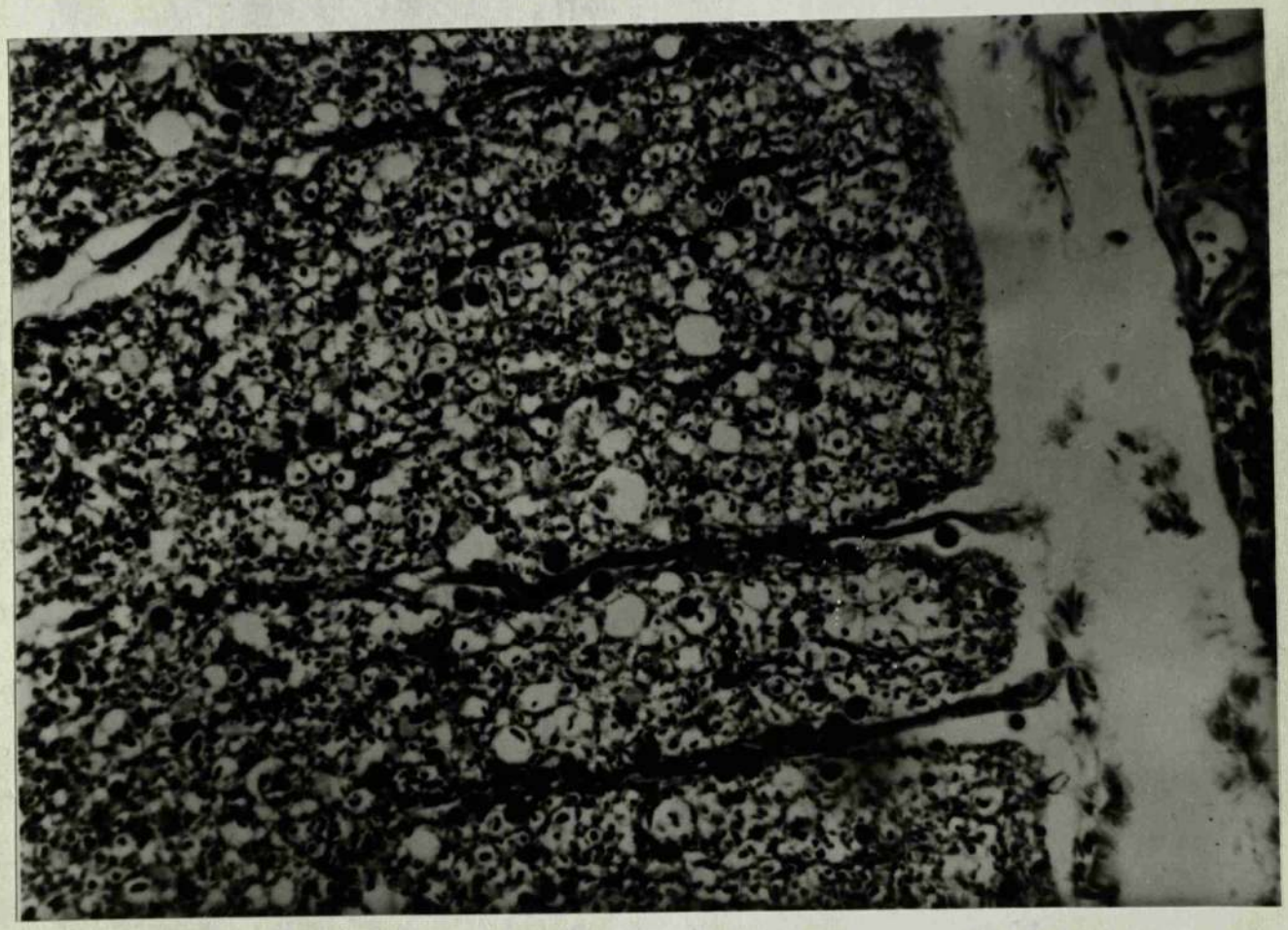


PLATE 10.

Photomicrograph of dorsal column at the cervico-dorsal junction in Case 7. Cord examined 2 years after injury.

The upper plate shows focal accumulations of axons radiating from small cystic spaces. (Bielschowsky X

The lower plate shows a detail of this. Smudges are present in these areas, indicating collagen deposition.

(Bielschowsky X 225)

PLATE 10.

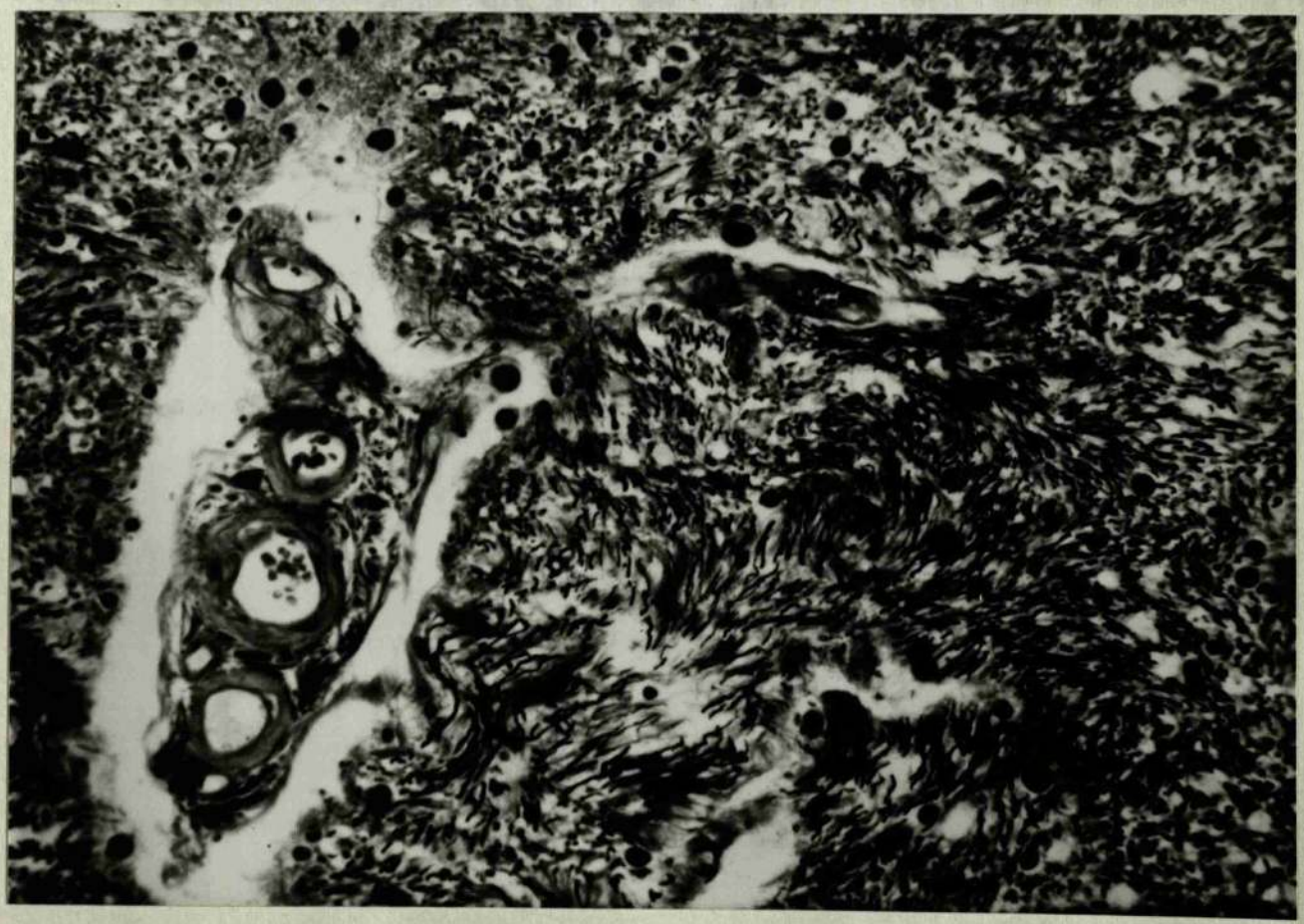


PLATE 11.

Photomicrograph of upper cervical cord in Case 7. Cord examined 2 years after injury.

There is extensive demyelination of the dorsal columns and some demyelination of lateral and antero-lateral columns.

(Weigert X 12)

PLATE 11.

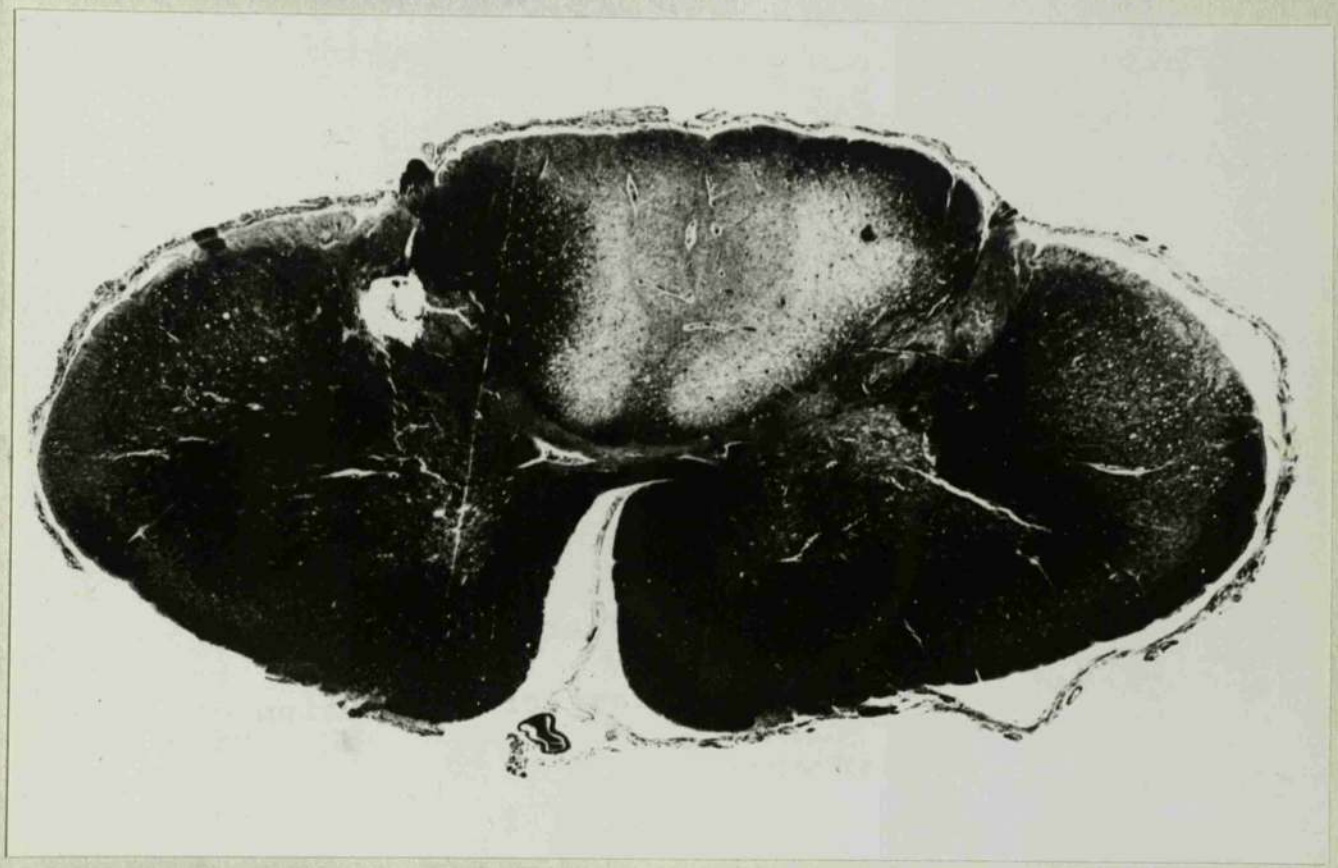


PLATE 12.

Photomicrograph of anterior horn of upper cervical cord in Case 7. Cord examined 2 years after injury.

Anterior horn cells are relatively well preserved. There is some loss of definition of supporting grey matter structure.

(Unna Pap X 135)

PLATE 12.

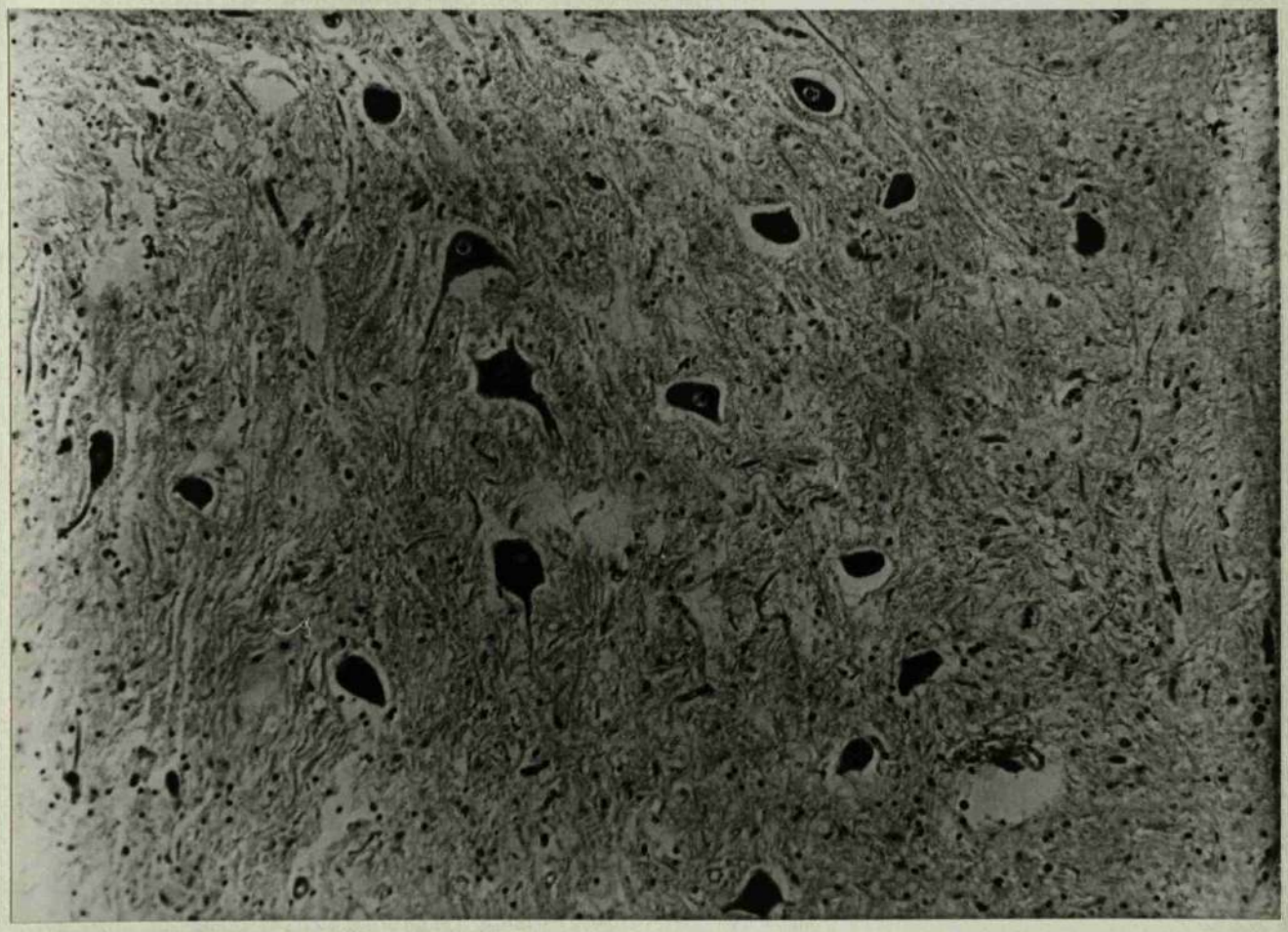


PLATE 13.

Photomicrograph of demyelinated dorsal columns of upper cervical cord in Case 7. Cord examined 2 years after injury.

Many axon sheaths are swollen and some axons appear to have fallen out of their sheaths. Neuroglial cells are increased in number and avidity for silver stain, and possess coarse protoplasmic feet.

(Bielschowsky X 90)

PLATE 13.

PLATE 14.

Photomicrograph of lumbar cord in Case 7. Cord removed 2 years after injury.

There is a small wedge of demyelination in the dorsi-lateral columns.

(Weigert X 15)

PLATE 14.

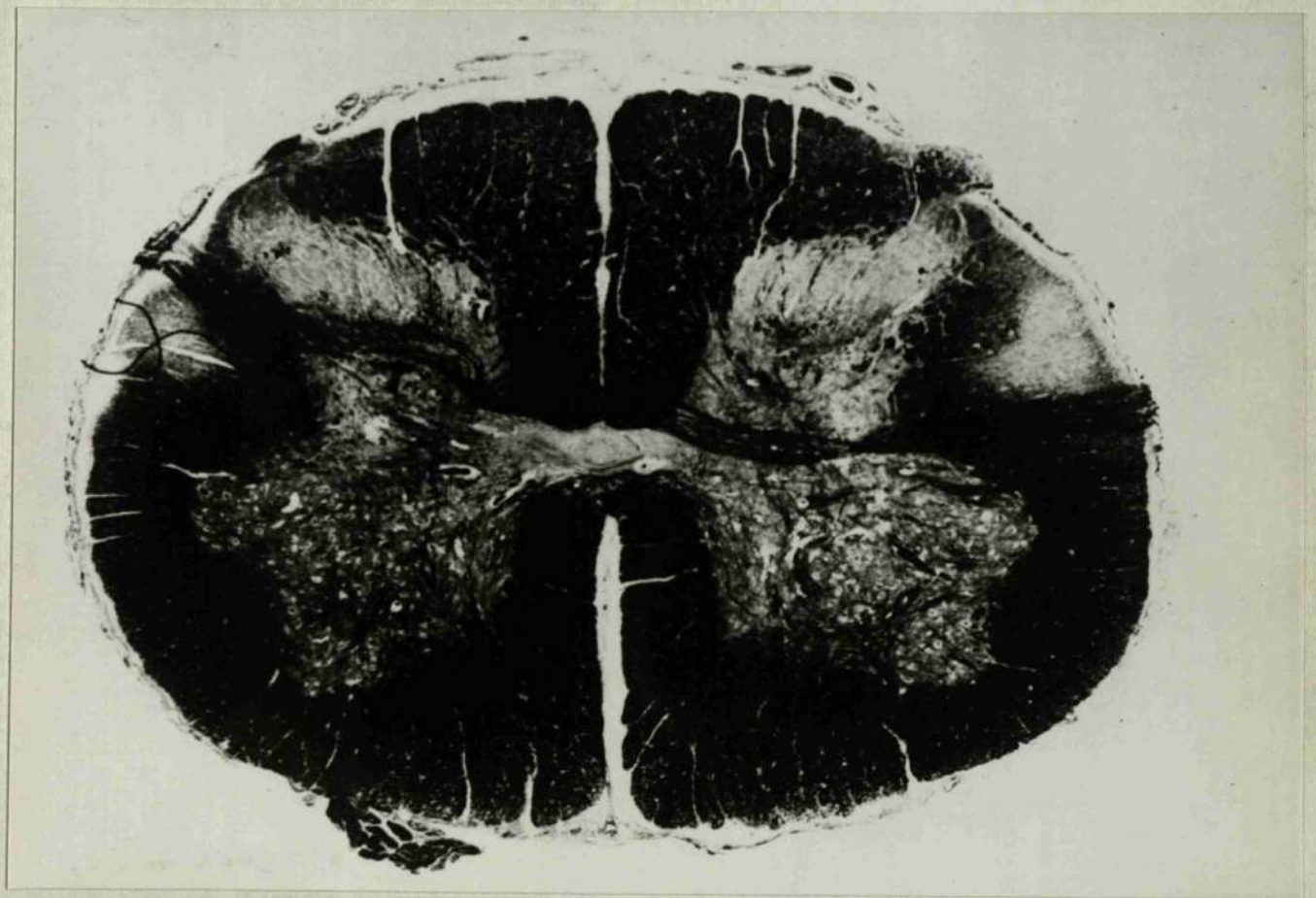


PLATE 15.

Photomicrograph of anterior horn of lumbar cord in Case 7. Cord examined 2 years after injury.

Anterior horn cells are relatively well preserved. There is some loss of definition of the supporting grey matter structure.

(Unna Pap X 135)

PLATE 15.

PLATE 16.

Photomicrograph of demyelinated dorsi-lateral column of lumbar cord in Case 7. Cord examined 2 years after injury.

Many axis cylinders have been preserved. Neuroglial cells are increased in number and avidity for silver, and exhibit coarse protoplasmic feet.

(Bielschowsky X 90)

PLATE 16.

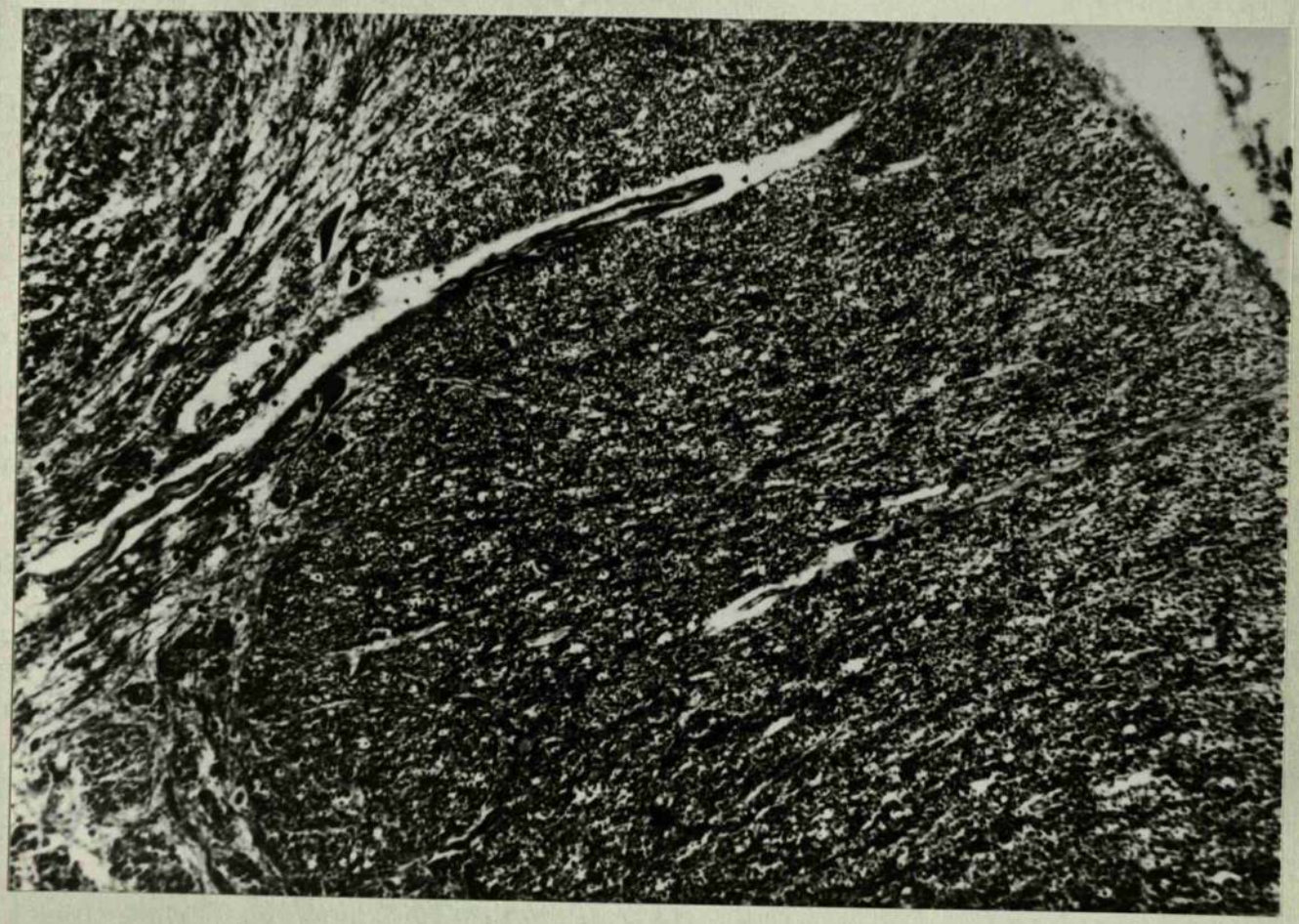


PLATE 17.

Photomicrograph of segment of cord at lumbo-dorsal junction in Case 8. Cord examined 4 years after injury.

Compression upon the cord has left an impression on it, indicated by an arrow. There is a band of demyelination extending cranially from the median parts of the dorsal columns and some demyelination of the lateral columns. The apparent gap in the cord is caused by its being twisted upon itself.

(Weigert X 4)

PLATE 17.

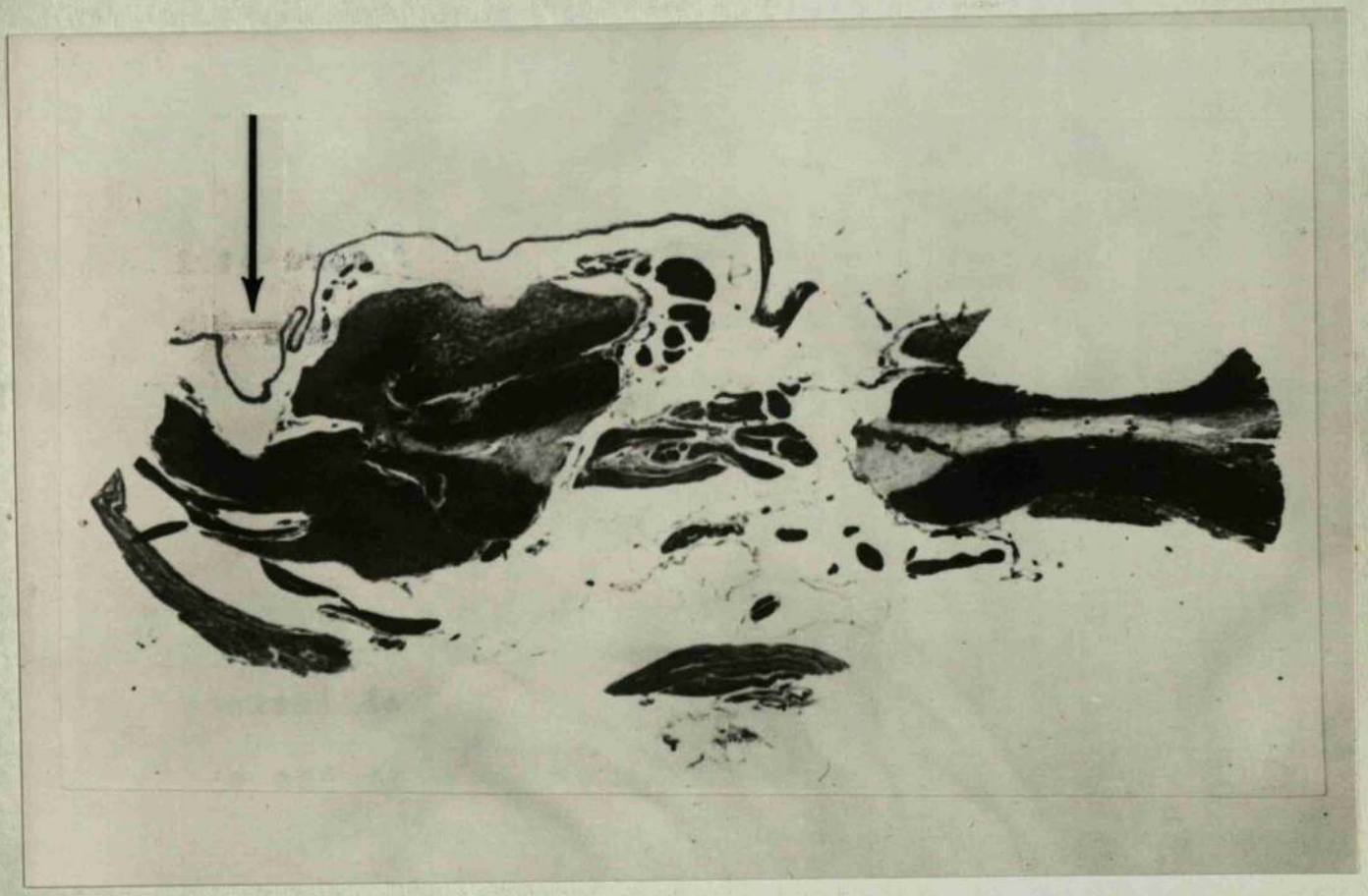


PLATE 18.

Photomicrograph of anterior horn of segment of cord adjoining the compressed area shown in Plate 17. Cord examined 4 years after injury.

Anterior horn cells exhibit some pyknosis. Otherwise grey matter structure is preserved.

(Bielschowsky X 200)

PLATE 18.

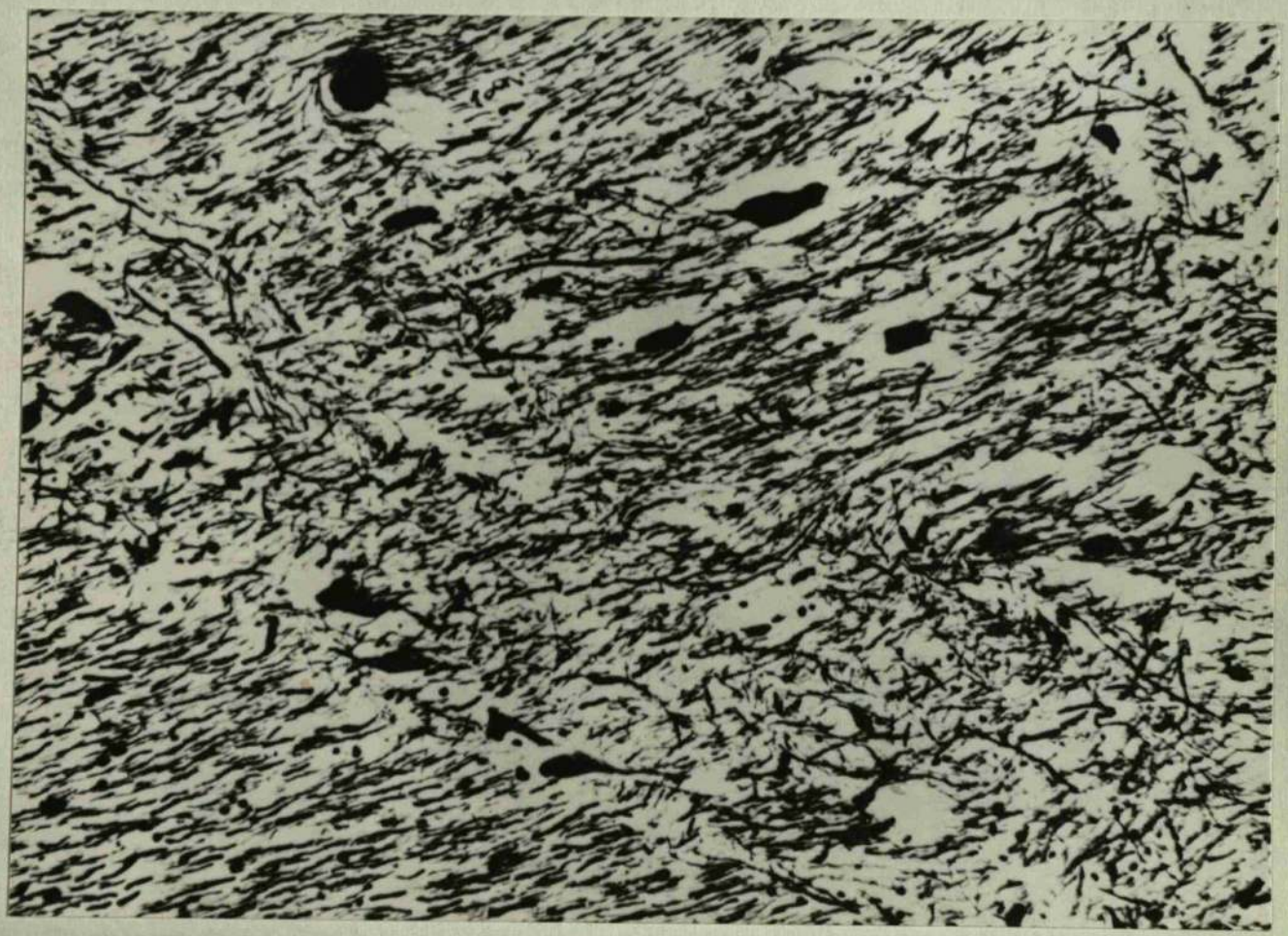


PLATE 19.

Photomicrograph of partially delyelinated lateral columns adjoining the compressed area shown in Plate 17 Cord removed 4 years after injury.

Axis cylinders are preserved. There are one or two large argentophil neuroglial cells with protoplasmic feet, indicated by an arrow.

(Bielschowsky × 90)

PLATE 19.

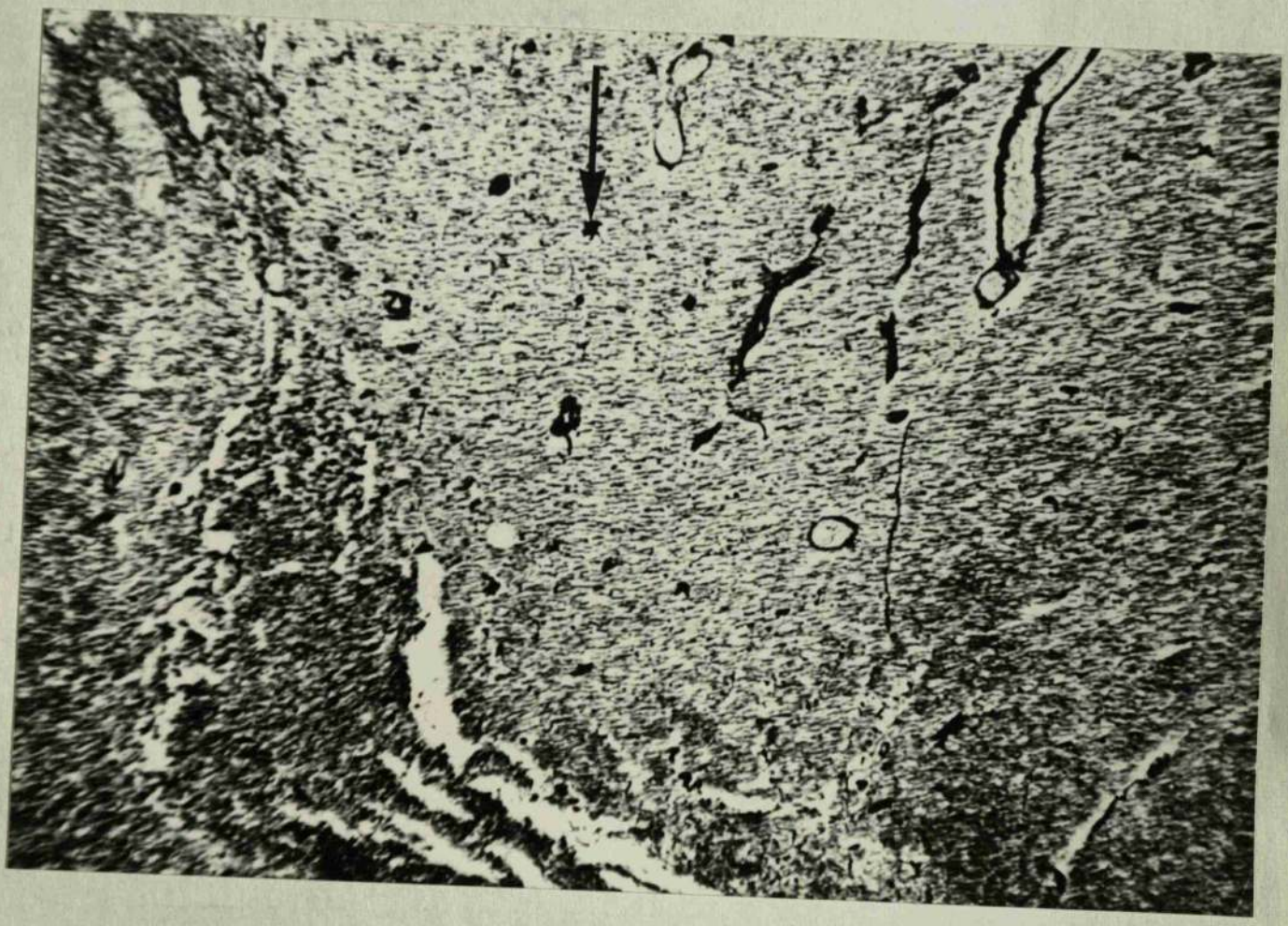


PLATE 20.

Photomicrograph of periphery of lateral columns adjoining the compressed area shown in Plate 17. Cord examined 4 years after injury.

Several wavy bands of collagen have been laid down. These bands have caused some neighbouring axis cylinders to assume a corresponding wavy form.

(Bielschowsky X 90)

PLATE 20.

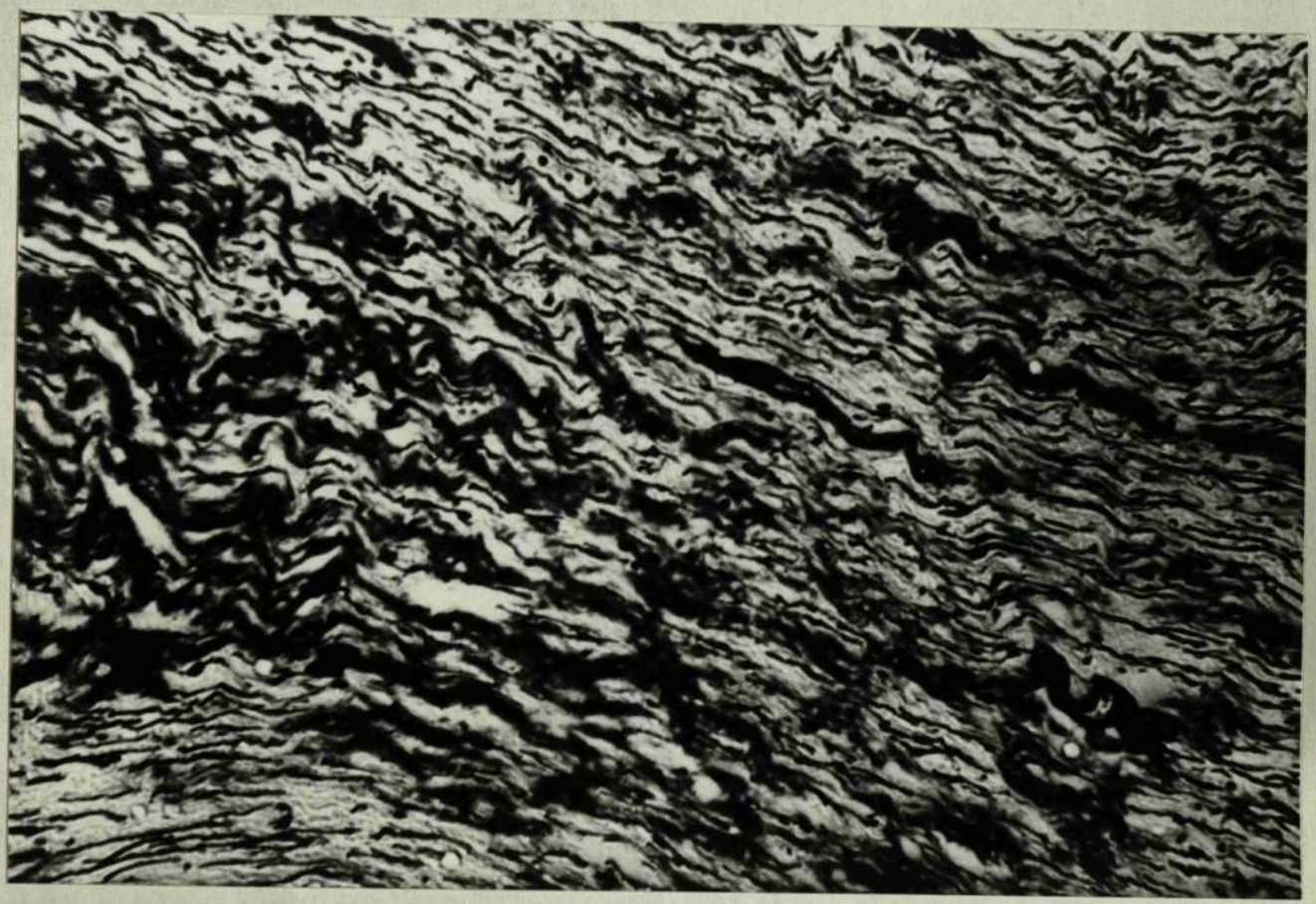


PLATE 21.

Photomicrograph of cauda equina in Case 8. Cord examined 4 years after injury.

There is extensive loss of definition, only a few myelinated nerve bundles surviving.

(Weigert X 19)

PLATE 21.

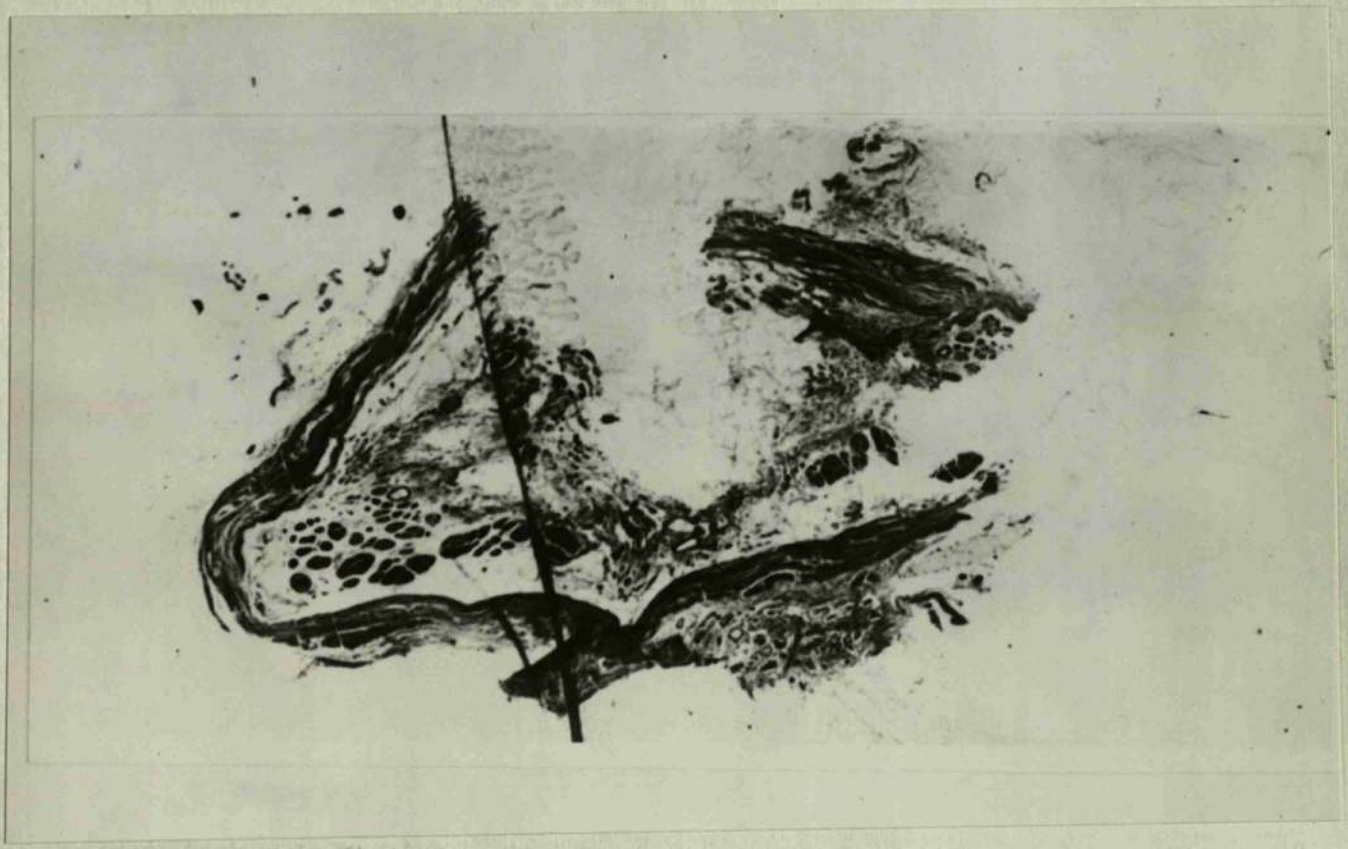


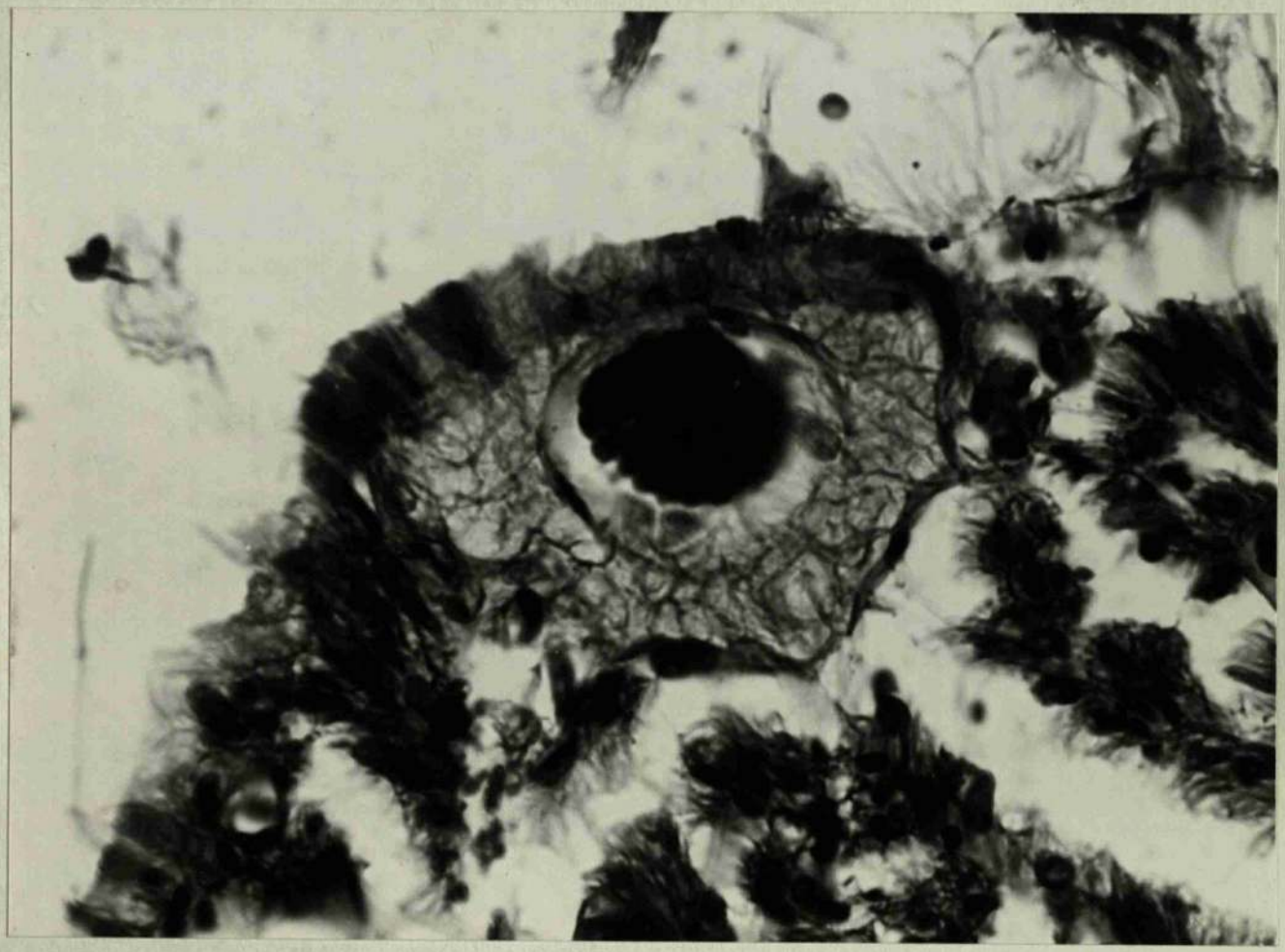
PLATE 22.

Photomicrograph of a cauda equina rootlet in Case 8. Cord examined 4 years after injury.

There is hypertrophy of the endothelium in a capillery, the swollen cells having a glassy appearance.

(H & E X 790)

PLATE 22.



PART THREE.

BLOOD PRESSURE RESPONSES EVOKED BY EXPERIMENTAL COMPRESSION OF THE SPINAL CORD IN CATS.

LEGENDS AND FIGURES

FIGURE 1.

Blood pressure response evoked by momentary compression of the third thoracic segment of the spinal cord. Compression applied at the arrow D.

FIGURE 1.

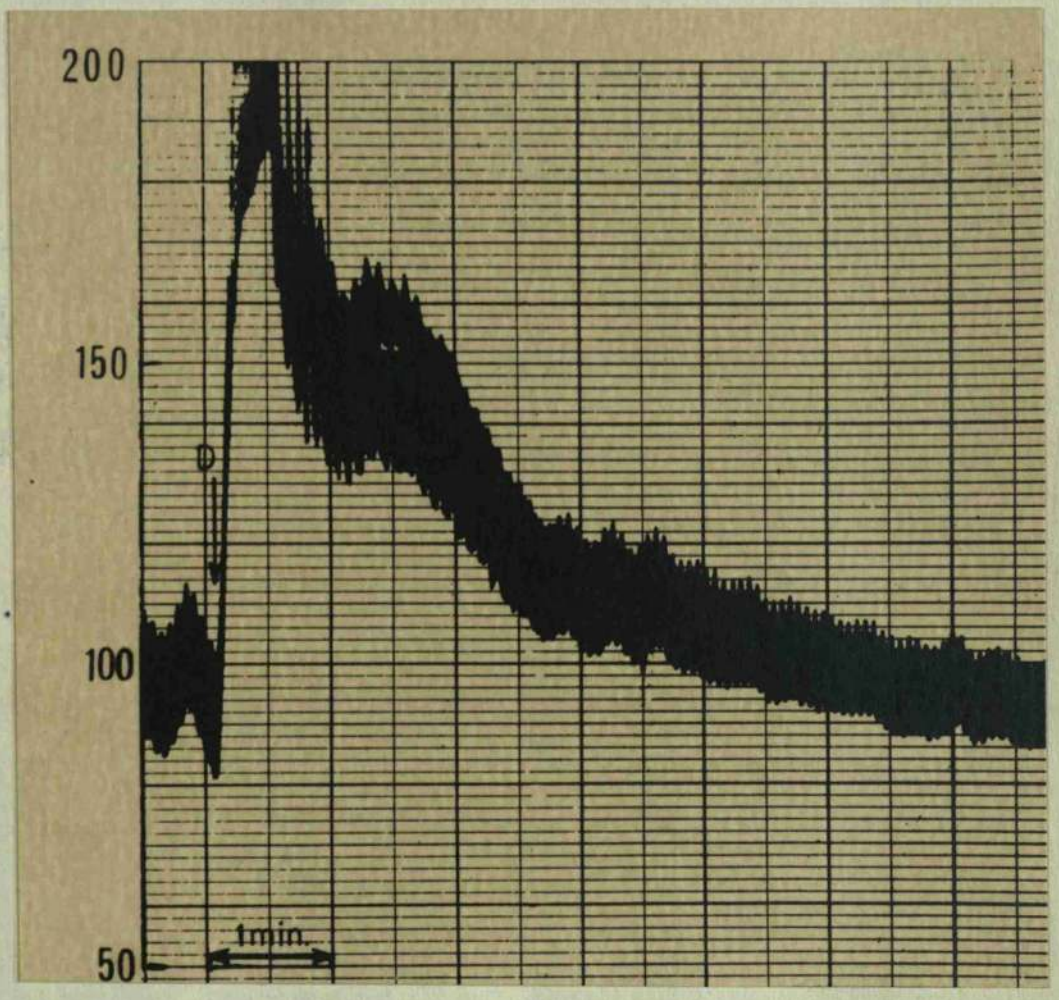


FIGURE 2.

Blood pressure responses evoked by compression of the spinal cord and intra-arterial administration of adrenaline. A'B' represents a two minute period of compression at the second thoracic segment at 200 mm. mercury balloon pressure. At C 0.2 ml. of 1:1000 adrenaline chloride was administered.

FIGURE 2.

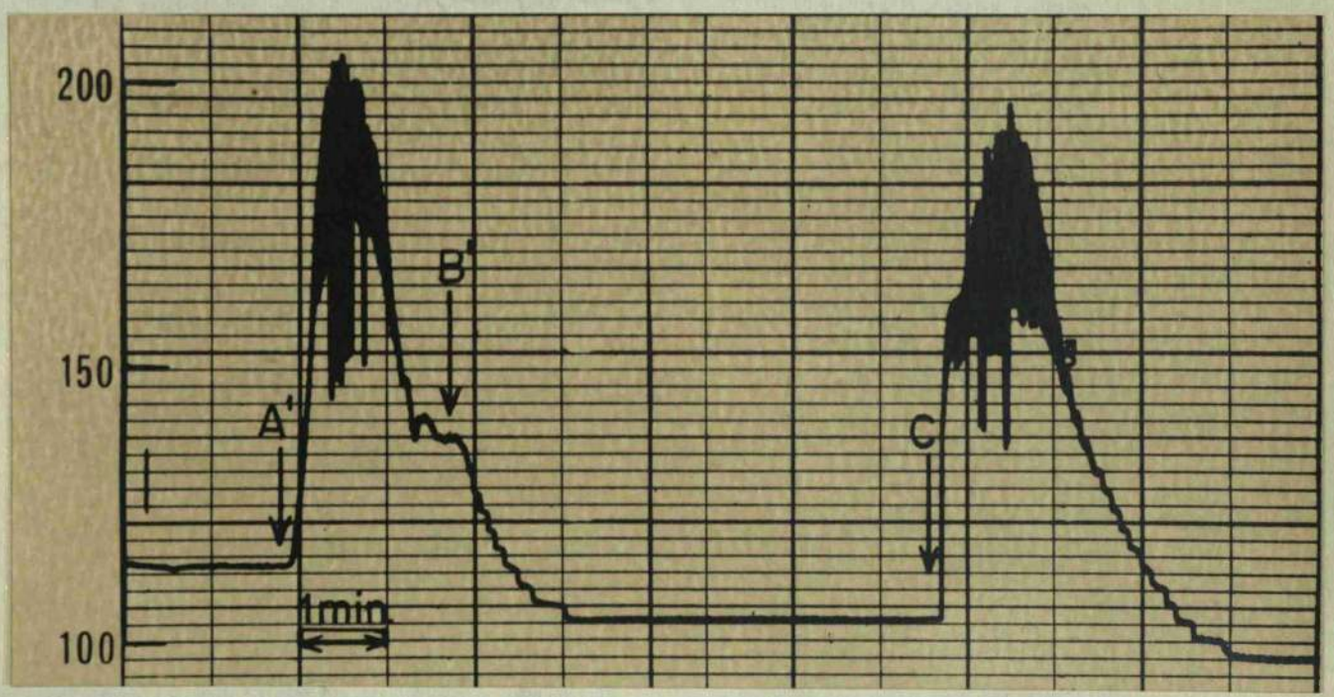


FIGURE 3.

Blood pressure responses evoked by different degrees of spinal cord compression at the fifth thoracic segment. AB represents a period of compression at 100 mm. mercury and A'B' at 200 mm. mercury, balloon pressure.

FIGURE 3.

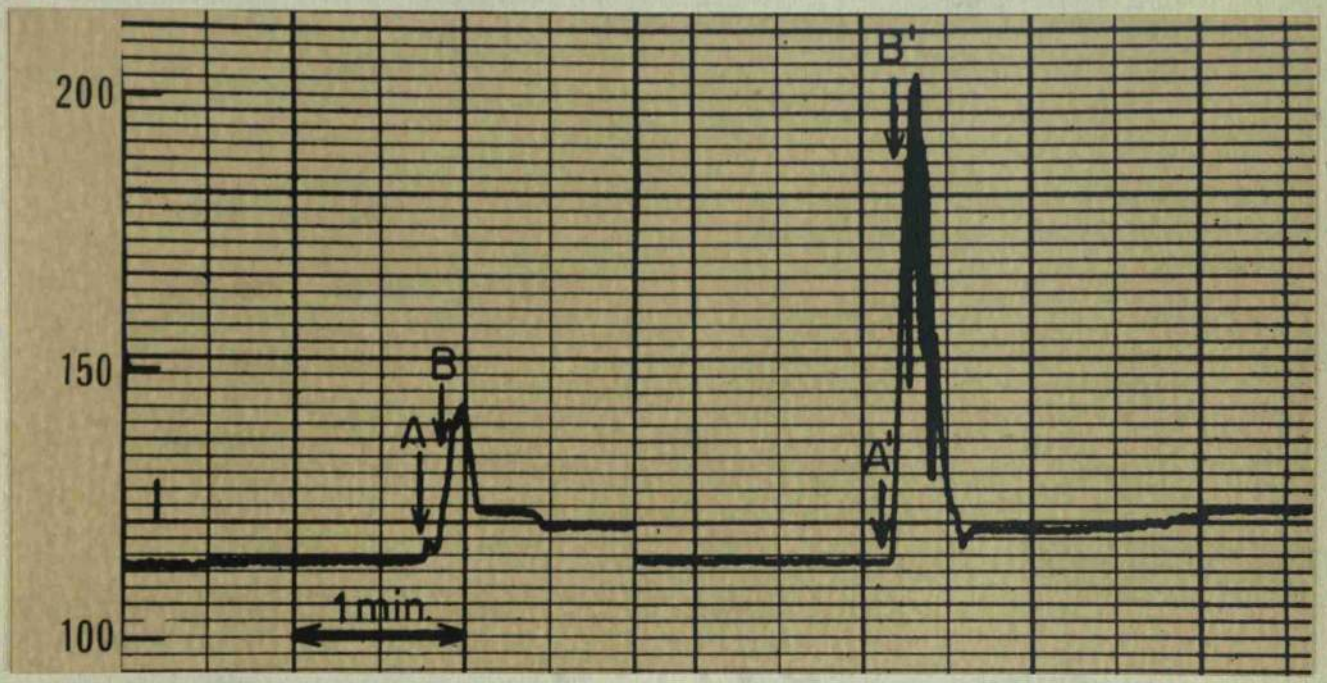


FIGURE 4.

Blood pressure responses evoked by different degrees of spinal cord compression at the seventh thoracic segment. $\overset{\circ}{A}\ \overset{\circ}{B}$ represents a period of compression at 50 mm. mercury, AB at 100 mm. mercury and A'B' at 200 mm. mercury, balloon pressure.

FIGURE 4.

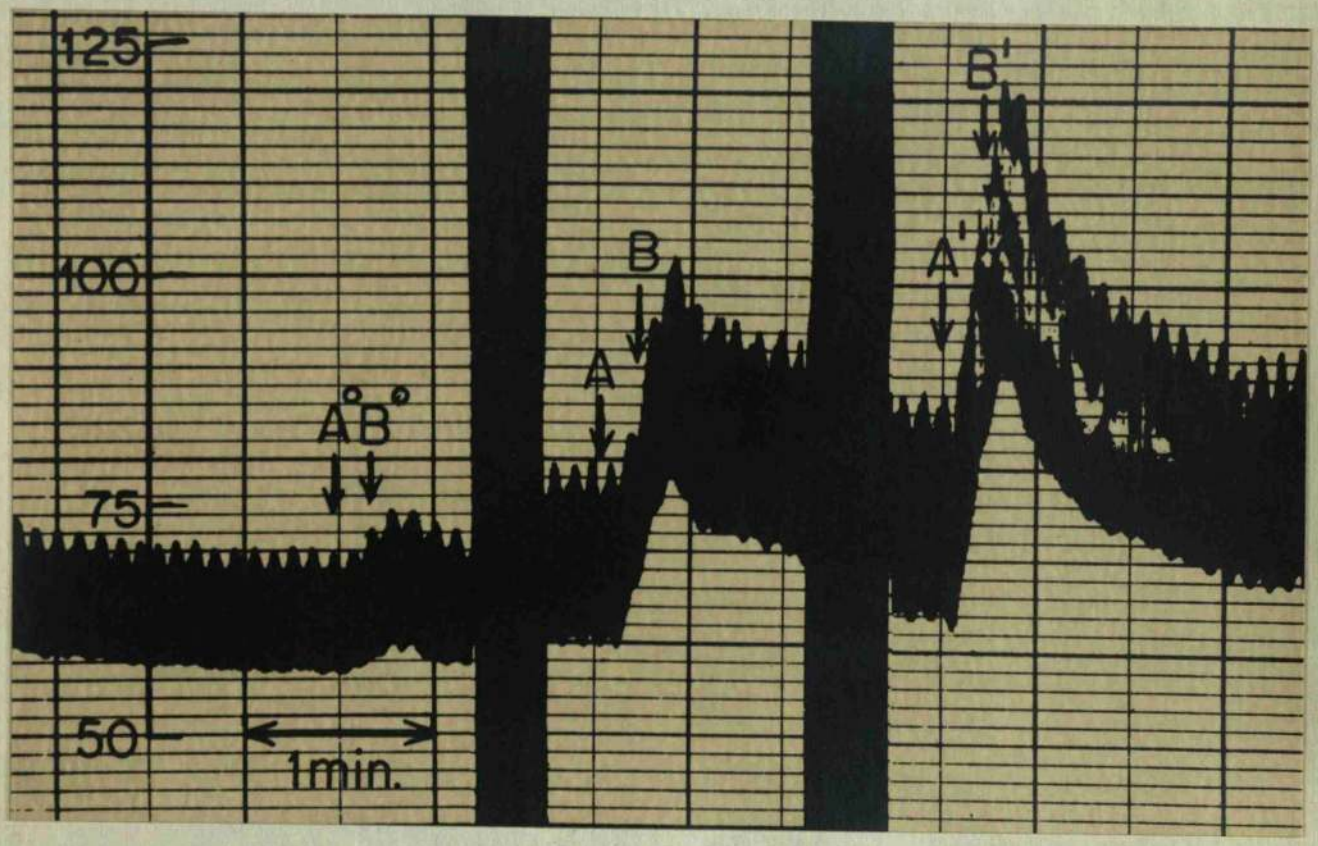


FIGURE 5.

Rises in mean blood pressure in nine cats following compression of different segments of spinal cord at 200 mm. mercury balloon pressure.

FIGURE 5.

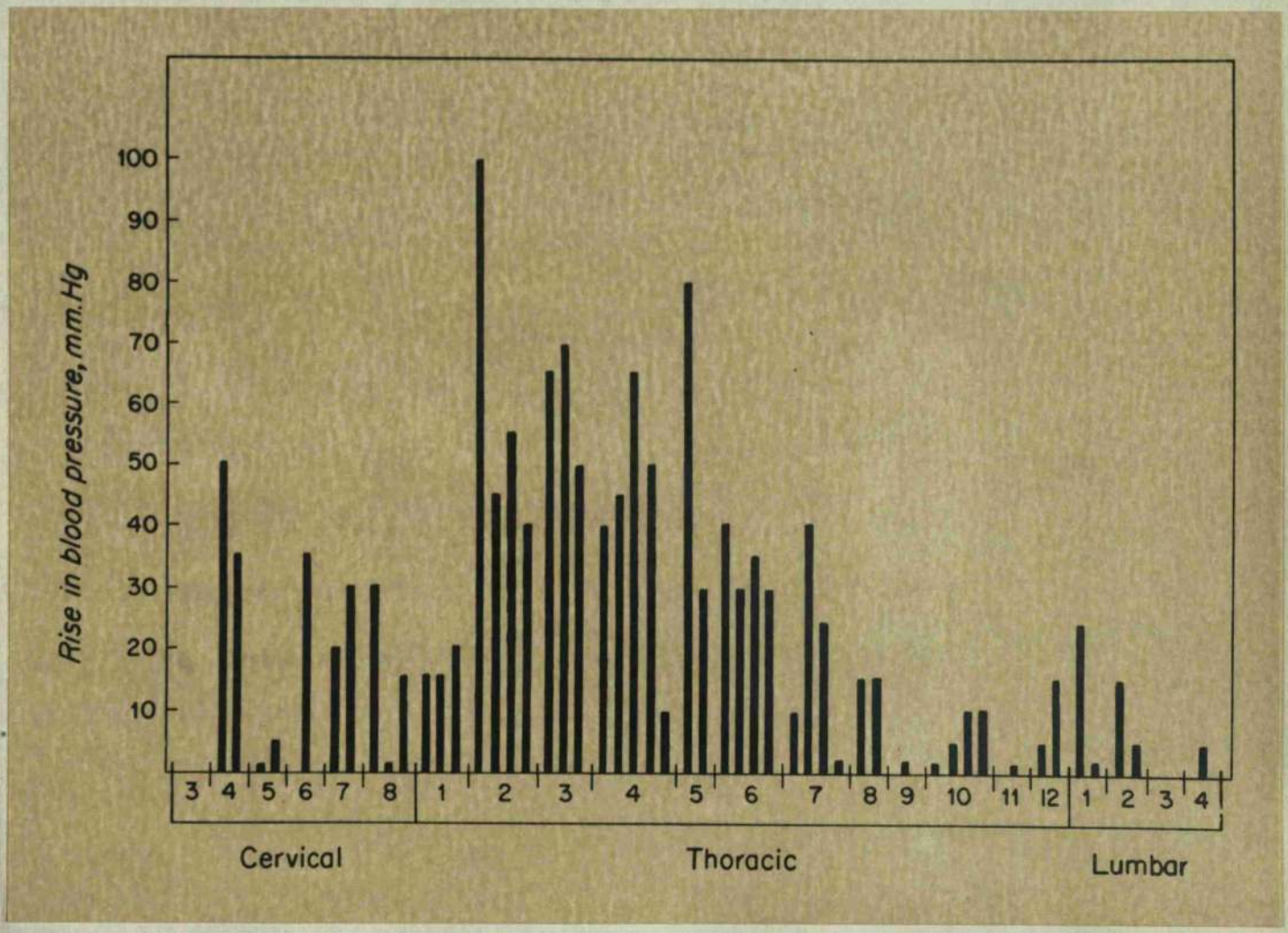
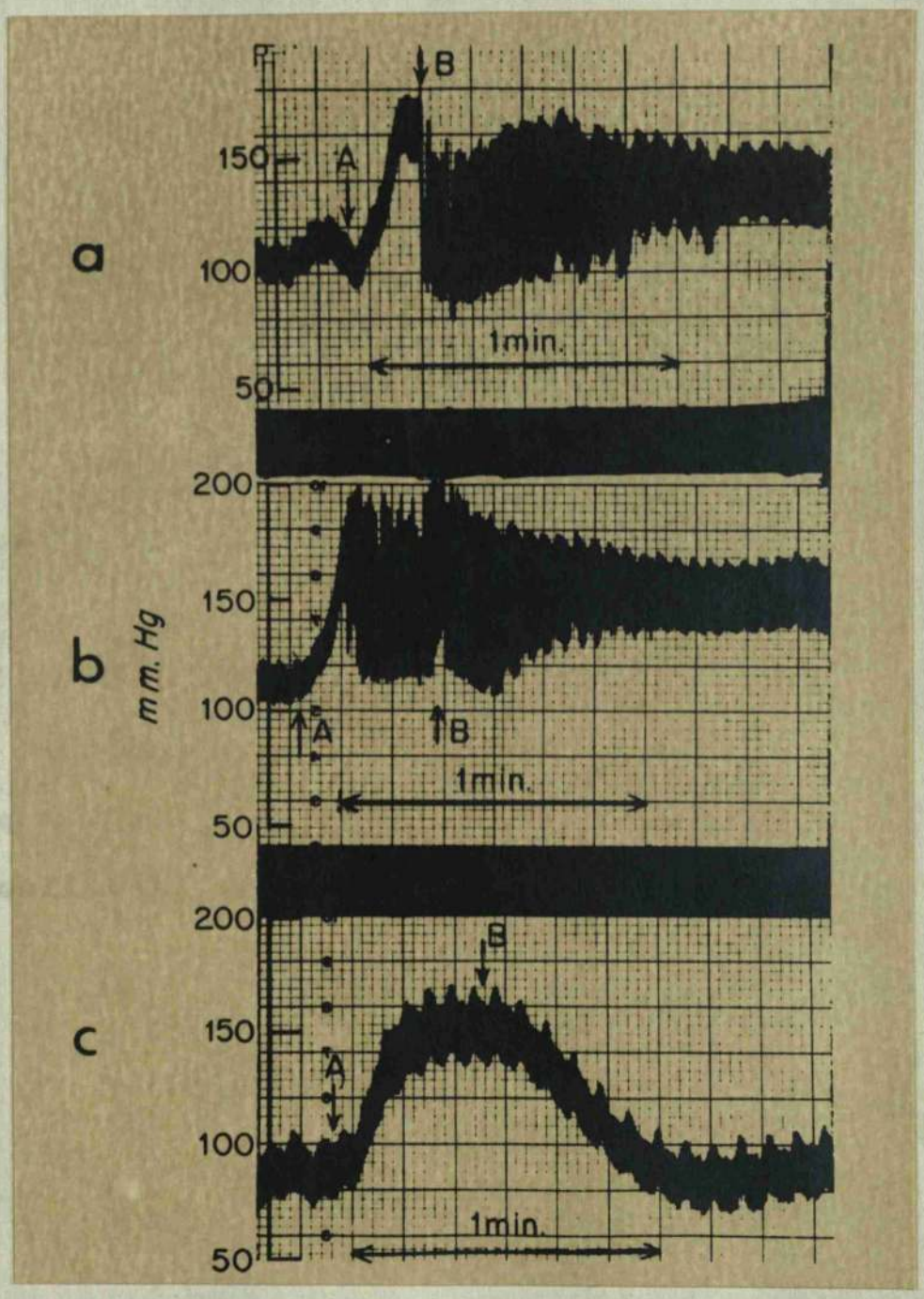

Rise in blood pressure, mm.Hg
100
90
80
70
60
50
40
30
20
10
3 4 5 6 7 8
1 2 3 4 5 6 7 8 9 10 11 12
1 2 3 4
Cervical
Thoracic
Lumbar

FIGURE 6.

Blood pressure responses from second thoracic segment after de-afferentiation (a & b). Upper three dorsal roots have been severed four days prior to compression. (c) sinu-aortic denervation has been performed. AB represents a period of compression at 100 mm. mercury, balloon pressure.

FIGURE 6.



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(1961): Bull. Los Angeles neurol. Soc. 26:172-185

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	Age	Sex	Date of Injury	Cause of Accident	Neurological Level	Defect	Early Mortality Due to Spinal Injury	Early Mortality Due to Other Injury	X-ray
1	73	M	20-7-53	TRAFFIC.	C 5	COMPLETE	ASPHYXIA		N.B.I. O.A
2	28	M	18-2-53	MACHINERY	L-D	COMPLETE			N.B.I
3	53	F	2-12-57	UNKNOWN	C 5	INCOMPLETE			N.B.I
4	63	M	27-6-57	WT. ON BACK INDUSTRIAL	T 10	COMPLETE	PULM. OEDEMA	DIAPH. HERNIA	CRUSH # D. 9.10.11
5	61	M	3-2-55	TRAFFIC	C 5	COMPLETE	HYPOTHERMIA		# DIS. C.V. 4.5
6	68	F	28-8-55	TRAFFIC	C 6	INCOMPLETE		RUPTURED VISCUS	CRUSH # C.V 6
7	31	M	4-4-49	WT. ON BACK MINER	L-D	COMPLETE			# DIS L.V 1-2
8	32	M	18-6-51	WT ON BACK MINER	L-D	COMPLETE			CRUSH # L.V 1
9	42	M	18.5.49	WT. ON BACK MINER	T 11	COMPLETE			# DIS L.V. 2-3
10	46	M	13.12.54	WT. ON BACK MINER	L-D	INCOMPLETE			# DIS D.V. 12 L.V 1.
11	37	M	20.4.54	WT. ON BACK MINER	L-D	COMPLETE			# DIS L.V 1-2
12	25	M	18.8.48	WT ON BACK MINER	L-D	COMPLETE			# DIS L.V. 4.5.
13	45	M	16.8.56	FELL 30'	C 5	COMPLETE	PULM. OEDEMA		# DIS C.V. 5-6
14	23	M	24.1.56	FELL OFF ROOF	DORSAL	INCOMPLETE			CRUSH # D.V 9.10.11.12
15	17	M	8.9.54	FELL 3 STORIES	L-D	COMPLETE		RUPTURED VISCUS	UNKNOWN
16									
17	48	M	13.12.57	TRAFFIC	C 7	COMPLETE	BRONCHO PNEUMONIA		# DIS C.V 7.8
18	40	M	18.8.48	FELL 30'	L-D	COMPLETE			# DIS D.V. 11-12
19	32	M	29.5.54	FELL 50'	L-D	INCOMPLETE			# DIS L.V. 1-2
20	54	M	19 5.45	FELL 30'	DORSAL	INCOMPLETE			CRUSH # D.V. 8
21	21	F	1940	UNKNOWN	L-D	INCOMPLETE			CRUSH # L.V. 1.
22	26	M	17.11.49	UNKNOWN	L-D	COMPLETE			# DIS L.V. 1.
23	56	M	9.6.47	FELL 40'	L-D	INCOMPLETE			# T.P
24	?24	F	14.8.54	TRAFFIC	L-D	COMPLETE		ABDOM. BLEEDING	UNKNOWN

C = CERVICAL
L-D = LUMBO-DORSAL

N.B.I. = NO BONE INJURY
O.A. = OSTEO ARTHRITIS

	Age	Sex	Date of Injury	Cause of Accident	Neurological Level	Defect	Early Mortality Due to Spinal Injury	Due to Other Injury	X-ray
25	20	M	18·5·54	FELL OFF ROOF	L-D	INCOMPLETE			CRUSH # L.V.1.
26	29	M	14·8·54	TRAFFIC	L-D	COMPLETE			#DIS L.V.1.
27	19	M	27·12·45	TRAFFIC	L-D	INCOMPLETE			#DIS L.V.2-3
28	18	M	24·8·53	FELL 20'	C 7	INCOMPLETE			CRUSH # C.V.5
29	46	M	27·8 57	MACHINERY	C 6	INCOMPLETE			DIS C.V.5-6
30	36	M	19 8·56	FELL DRUNK	C5-6	INCOMPLETE			N.B.I.
31	48	M	14 10·56	WT. ON BACK MINER	C 7	INCOMPLETE			N.B.I. O.A.
32	61	M	26·5·57	WT. ON BACK	C5-6	COMPLETE	PULM. OEDEMA		DIS C.V.5-6 #SKULL
33	41	M	21 8·55	TRAFFIC	C 5	INCOMPLETE			N.B.I.
34	24	M	3·10·55	FELL 4 STORES	L-D	INCOMPLETE			CRUSH # D.V.12
35	21	M	26·11·49	FELL DRUNK	L-D	INCOMPLETE			CRUSH # L.V.4.
36	51	M	18·9 48	WT. ON BACK MINER	L-D	COMPLETE			#DIS D.V. 11.
37	25	M	20·10·51	FELL 4 STORIES	L-D	INCOMPLETE			#DIS L.V.2-3
38	50	M	26·11·51	FELL DRUNK	L-D	INCOMPLETE			NIL O.A
39	61	M	7·1·58	FELL DRUNK	C7-8	INCOMPLETE			NIL O.A
40	53	M	30·9·58	FELL DRUNK	DORSAL	INCOMPLETE			CRUSH # D.V.7-10
41	62	M	19·10·58	TRAFFIC	C 6	COMPLETE	PULM COLLAPSE		NIL O.A
42	37	M	19 7 56	WT. ON BACK	L-D	COMPLETE			#DIS L.V.1-2.
43	61	M	22·7 55	FELL SCAFFOLD	C 6-7	COMPLETE	PULM. OEDEMA		DIS C.V.6-7
44	44	M	7 12·50	WT. ON BACK MINER	L-D.	COMPLETE			#DIS L.V.
44a	42	M	22·10 52	WT. ON BACK MINER	L-D	COMPLETE			UNKNOWN
45	48	M	18·11·56	WT. ON BACK MINER	L-D	COMPLETE			#DIS D.V. 12.
45a	31	M	23·2·51	WT. ON BACK MINER	L-D	INCOMPLETE			#DIS D.V. 11-12
46	35	M	27 3·46	WT. ON BACK MINER	L-D	INCOMPLETE			#DIS L.V. 1-2
46a	44	M	25·9·51	UNKNOWN	L-D	INCOMPLETE			UNKNOWN
47	31	M	21·4 53	FELL SHAFT	DORSAL	COMPLETE			CRUSH # DV 10.11.12
47a	38	M	17 2·55	FELL 20'	L-D	COMPLETE			#DIS L.V. 1.
48	34	M	23·3·58	FELL 35'	D 6	COMPLETE			CRUSH # D.V.4-6.
49	29	M	26·2·56	FELL LADDER	L-D	COMPLETE			#DIS L.V. 1

	ORTHOPAEDIC TREATMENT			TROPHIC SKIN ULCERATION			
	Conservative	Closed Reduction	Operative	Residual Spinal Movement	Incidence	Grafted	Necessitated Readmission
1	ORD BED						
2	ORD BED			GOOD	SEVERE TRANSIENT	YES	NO
3	ORD BED + COLLAR			UNKNOWN	UNKNOWN		
4	ORD. BED						
5	ORD. BED						
6	ORD. BED						
7		MANIPULATION COMPLETE PLASTER		RESTRICTED	SEVERE PERMANENT	YES	YES
8	ORD. BED 5/52 PLASTER SHELL			GOOD	SEVERE PERMANENT	YES	YES
9	HIR BED			BAD	SEVERE PERMANENT	NO	NO
10			ORD. BED PLATED	GOOD	MINOR TRANSIENT	NO	NO
11			PLATED TURNING FRAME	UNKNOWN	SEVERE UNKNOWN	UNKNOWN	UNKNOWN
12			REDUCTION COMPLETE PLASTER	RESTRICTED	SEVERE PERMANENT	YES	YES
13		SKULL TRACTION ORD. BED					
14			LAMINECTOMY TURNING FRAME	GOOD	NONE	NO	NO
15	ORD. BED						
16							
17		SKULL TRACTION ORD. BED					
18	COMPLETE PLASTER TURNING FRAME 5/52			RESTRICTED	SEVERE PERMANENT	YES	YES
19	MECCANO BED			GOOD	SEVERE TRANSIENT	NO	NO
20	ORD. BED 7/12		LAMINECTOMY 7/12 BONE FUSION TURNING FRAME	RESTRICTED	MINOR TRANSIENT	NO	NO
21	COMPLETE PLASTER			UNKNOWN	SEVERE PERMANENT	UNKNOWN	UNKNOWN
22			PLATED ORD. BED	RESTRICTED	SEVERE PERMANENT	NO	YES
23	ORD. BED			GOOD	UNKNOWN		
24	ORD. BED						

ORD. = ORDINARY

	ORTHOPAEDIC TREATMENT				TROPHIC SKIN ULCERATION		
	Conservative	Closed Reduction	Operative	Residual Spinal Movement	Incidence	Grafted	Necessitated Readmission
25			LAMINECTOMY BONE FUSION TURNING FRAME		SEVERE PERMANENT	NO	NO
26			PLATED TURNING FRAME	GOOD	SEVERE PERMANENT	YES	YES
27			BONE FUSION TURNING FRAME	GOOD	SEVERE UNKNOWN	UNKNOWN	UNKNOWN
28			LAMINECTOMY COMPLETE P.O.P	GOOD	NONE	NO	NO
29		SKULL TRACTION COMPLETE P.O.P		GOOD	MINOR TRANSIENT	NO	NO
30	ORD. BED			GOOD	NONE	NO	NO
31	ORD. BED			GOOD	NONE	NO	NO
32		SKULL TRACTION ORD. BED					
33	ORD. BED			GOOD	SEVERE TRANSIENT		
34	ORD. BED			GOOD	NONE	NO	NO
35	PLASTER SHELL			UNKNOWN	UNKNOWN		
36			LAMINECTOMY TURNING FRAME	UNKNOWN	SEVERE PERMANENT	NO	YES
37			PLATED TURNING FRAME	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN
38			LAMINECTOMY ORD. BED	GOOD	NIL	NO	NO
39	COMPLETE PLASTER + TURNING FRAME			RESTRICTED	SEVERE PERMANENT	NO	
40	ORD BED		G.	GOOD	NIL		
41	ORD. BED						
42			PLATED TURNING FRAME	UNKNOWN	SEVERE TRANSIENT	NO	YES
43	ORD. BED						
44	WATER BED			RESTRICTED	SEVERE PERMANENT	NO	NO
44a	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN	SEVERE PERMANENT	NO	NO
45	ORD. BED			GOOD	MINOR TRANSIENT	NO	YES
45a	ORD. BED			GOOD	SEVERE TRANSIENT	NO	NO
46			UNSUCCESSFUL WIRING BONE GRAFT	GOOD	MINOR TRANSIENT	NO	NO

P.O.P = PLASTER OF PARIS

46a	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN
47	TURNING FRAME			GOOD	SEVERE TRANSIENT	YES	NO
47a	TURNING FRAME			GOOD	SEVERE PERMANENT	NO	NO
48	TURNING FRAME			GOOD	SEVERE TRANSIENT	NO	NO
49			PLATED TURNING FRAME	GOOD	SEVERE PERMANENT	NO	NO

NEUROSURGICAL PROBLEMS

	Clinically progressive Initially	Later	L.P. Block	Operations	Outcome
1	NIL	NIL			
2	NIL	NIL			
3	NIL	NIL			
4	NIL	NIL			
5	NIL	NIL			
6	NIL	NIL			
7	NIL	NIL			
8	NIL	NIL			
9	YES	NIL			
10	NIL	NIL			
11	UNKNOWN	UNKNOWN			
12	UNKNOWN	NIL			
13	NIL	NIL			
14	NIL	NIL	UNKNOWN	EARLY LAMINECTOMY EXTRA DURA CLOT	UNAFFECTED
15	NIL	NIL			
16					
17	NIL	NIL			
18	NIL	NIL			
19	NIL	NIL			
20	NIL	YES 5/12	COMPLETE BLOCK	LATE LAMINECTOMY BONE RIDGE	IMPROVED
21	UNKNOWN	UNKNOWN			
22	NIL	NIL			
23	NIL	YES 10/YEARS	NO BLOCK		

NEUROSURGICAL PROBLEMS

	Clinically progressive Initially	Later	L.P. Block	Operations	Outcome
24	NIL	NIL			
25	YES	NIL	UNKNOWN	EARLY LAMINECTOMY	UNAFFECTED
26	YES	NIL	COMPLETE BLOCK	EARLY LAMINECTOMY ANT. COMPRESSION	UNAFFECTED
27	NIL	NIL	UNKNOWN		IMPROVED
28	YES	NIL	COMPLETE BLOCK	EARLY LAMINECTOMY COMPRESSION	IMPROVED
29	NIL	NIL			
30	NIL	NIL	NO BLOCK		
31	NIL	NIL			
32	NIL	NIL			
33	YES	NIL	NO BLOCK		
34	NIL	NIL	NO BLOCK		
35	NIL	NIL			
36	NIL	NIL	UNKNOWN	EARLY LAMINECTOMY DISC FRAGMENT	UNAFFECTED
37	YES	UNKNOWN	COMPLETE BLOCK	NIL	UNKNOWN
38	NIL	NIL	COMPLETE BLOCK	EARLY LAMINECTOMY	UNAFFECTED
39	NIL	NIL	NIL	NIL	
40	NIL	NIL	NO BLOCK	NIL	
41					
42	NIL	NIL			
43					
44	NIL	NIL	UNKNOWN		
44a	UNKNOWN	UNKNOWN		UNKNOWN	
45	NIL	NIL		NIL	
45a	NIL	YES	NO BLOCK	LATE LAMINECTOMY NO BLOCK	IMPROVED
46	NIL	NIL	COMPLETE BLOCK	LATE LAMINECTOMY CONSTRICTING LAMINA	IMPROVED
46a	UNKNOWN				
47	NIL	NIL	NO BLOCK	NIL	
47a	NIL	NIL		NIL	
48	NIL	NIL		NIL	
49	NIL	NIL		NIL	

URINARY PROBLEMS

	Initial	Sepsis	Stones	Residual State	Readmission for Urinary Sepsis
1					
2	NIL	NIL		CONTINENT	
3	NIL	NIL		CONTINENT	
4					
5					
6					
7	TIDAL DRAINAGE	SEVERE		INCONTINENT DIED OF PYELONEPH.	YES
8	TIDAL DRAINAGE	SEVERE		AUTONOMOUS BLADDER	NO
9	UNKNOWN	SEVERE		INCONTINENT	NO
10	INDWELLING CATH.	MINOR		INCONTINENT	NO
11	UNKNOWN	SEVERE		UNKNOWN	UNKNOWN
12	UNKNOWN	SEVERE		AUTOMATIC BLADDER	YES
13					
14	INDWELLING CATH.	MINOR		NORMAL	NO
15					
16					
17					
18	TIDAL DRAINAGE	SEVERE		DIED SUPRA PUBIC CATH.	YES
19	INDWELLING CATH.	SEVERE		INCONTINENT	YES
20	NIL	NIL		CONTINENT	NO
21	SUPRA PUBIC CATH.	UNKNOWN		DIED AUTOMATIC BLADDER	UNKNOWN
22	MANUAL COMPRESSION	SEVERE		DIED SUPRA PUBIC CATH.	YES
23	UNKNOWN	UNKNOWN		INCONTINENT	NO

CATH. = CATHETER

URINARY PROBLEMS

	Initial	Sepsis	Stones	Residual State	Readmission for Urinary Sepsis
24					
25	INDWELLING CATH.	SEVERE		INCONTINENT DIED OF SUPPURATION	NO
26	INDWELLING CATH.	SEVERE		INCONTINENT	NO
27	UNKNOWN	MINOR		CONTINENT	NO
28	NIL	NIL		CONTINENT	NO
29	INDWELLING CATH.	SEVERE		INCONTINENT	YES
30	NIL	NIL		NORMAL	NO
31	NIL	NIL		NORMAL	NO
32					
33	INDWELLING CATH.	SEVERE		CONTINENT	NO
34	INDWELLING CATH.	MINOR		AUTOMATIC BLADDER	NO
35	UNKNOWN	UNKNOWN		UNKNOWN	UNKNOWN
36	INDWELLING CATH.	SEVERE		INCONTINENT DIED OF AMYLOID	
37	INDWELLING CATH.	MINOR		UNKNOWN	
38	INDWELLING CATH.	MINOR		NORMAL	
39	GIBBON CATH.	SEVERE		DIED INCONTINENT	
40	NIL	NIL		NORMAL	
41					
42	FOLEY CATH	MINOR		AUTOMATIC	
43					
44	INDWELLING CATH.	SEVERE		INCONTINENT	
44a	INDWELLING CATH.	SEVERE		INCONTINENT DIED PYELONEPH.	
45	INDWELLING CATH.	MINOR		AUTOMATIC WITH PRECIPITENCE	
45a	INDWELLING CATH.	SEVERE		CONTINENT WITH PRECIPITENCE	YES
46	TIDAL DRAINAGE	SEVERE		CONTINENT	YES
46a	UNKNOWN	UNKNOWN		UNKNOWN	YES
47	INDWELLING C.ATH.	SEVERE		AUTOMATIC	NIL
47a	TIDAL DRAINAGE	SEVERE		INCONTINENT	NIL
48	GIBBON CATH	SEVERE		INCONTINENT	NIL
49	TIDAL DRAINAGE	SEVERE		INCONTINENT	NIL

ABDOMINAL SYMPTOMS

	Dyspepsia	Diarrhoea	Abdominal pain	Investigations (X-rays)	Abdominal Operations
1					
2	NIL	NIL	NIL	NIL	NIL
3	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN
4					
5					
6					
7	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN
8	NIL	SEVERE BOUTS	SEVERE	NEGATIVE STOOL CULTURE	NIL
9	SEVERE	NIL	MILD	?DIAPHRAGMATIC HERNIA	NIL
10	NIL	NIL	NIL	NIL	NIL
11	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN
12	NIL	NIL	NIL	NIL	NIL
13					
14	SEVERE	NIL	NIL	NIL	NIL
15					
16					
17					
18	SEVERE	SEVERE BLOOD & MUCUS	UNKNOWN	NEGATIVE STOOL CULTURE ?AMYLOID DISEASE ?ULCERATIVE COLITIS	NONE
19	NIL	NIL	NIL	NIL	NIL
20	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN
21	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN
22	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN
23	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN
24					

ABDOMINAL SYMPTOMS

	Dyspepsia	Diarrhoea	Abdominal pain	Investigations (X-rays)	Abdominal Operations
25					
26	SEVERE COLD WEATHER	NIL	NIL	DUODENAL SPASM	NEGATIVE LAPAROTOMY FOR SUSPECTED PERFORATION
27	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN
28	SEVERE	NIL	NIL	NEGATIVE BARIUM MEAL	NIL
29	SEVERE WITH HAEMATEMESIS	SEVERE BOUTS	SEVERE	NEGATIVE BARIUM MEAL ? ACUTE EROSIONS NEGATIVE STOOL CULTURE	NIL
30	SEVERE	NIL	MILD	NIL	NIL
31	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN
32					
33	NIL	NIL	NIL	NIL	NIL
34	NIL	NIL	NIL	NIL	NIL
35	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN
36	NIL	SEVERE BOUTS	MILD	AMYLOID DISEASE NEGATIVE STOOL CULTURE	NIL
37	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN
38	MILD				
39		SEVERE BOUTS	SEVERE	NIL NEGATIVE STOOL CULTURE	NIL
40	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN
41					
42	SEVERE			NEGATIVE BARIUM MEAL	NIL
43					
44	NIL	NIL	NIL	NIL	NIL
44a	UNKNOWN				
45	MILD	NIL		NIL	NIL
45a	SEVERE	NIL	NIL		NEGATIVE LAPARCTOMY FOR PYLORIC STENOSIS
46	SEVERE	SEVERE BOUTS		NEGATIVE BARIUM MEAL	NEGATIVE LAPAROTOMY CHOLECYSTECTOMY
46a	UNKNOWN				
47	SEVERE HAEMATEMESIS	NIL	SEVERE	NEGATIVE BARIUM MEAL	NIL
47a	NIL	NIL	MILD	NIL	NIL
48	NIL	NIL	NIL	NIL	NIL
49	NIL	NIL	SEVERE	NIL	NIL

NEUROLOGICAL FINDINGS

	Initial Severity	Late Reflex Pattern	Recovery	Involuntary Movements Extent Early	Extent Late	Type
1	COMPLETE					
2	COMPLETE	U.M.N.	FULL MOBILE	MODERATE	MILD	FLEXOR
3	INCOMPLETE	U M.N.	FULL MOBILE	MODERATE	UNKNOWN	FLEXOR
4	COMPLETE					
5	COMPLETE	HYPOTHERMIA				
6	INCOMPLETE					
7	COMPLETE	L.M.N.	NIL	NIL	NIL	NIL
8	COMPLETE	L.M N.	NIL		MILD	EXTENSOR
9	COMPLETE	L.M.N.	NIL	NIL	NIL	NIL
10	INCOMPLETE	U.M.N.	SLIGHT	NIL	NIL	NIL
11	COMPLETE	UNKNOWN	UNKNOWN	UNKNOWN		UNKNOWN
12	COMPLETE	L.M.N.	NIL	MODERATE	MILD	FLEXOR
13	COMPLETE					
14	INCOMPLETE	U.M.N.	ALMOST FULL STIFF	MILD	MILDER	EXTENSOR
15	COMPLETE					
16						
17	COMPLETE					
18	COMPLETE	L.M N.	NIL	NIL		NIL
19	INCOMPLETE	L.M.N.	NIL	NIL		NIL
20	INCOMPLETE	U.M.N.	ALMOST FULL STIFF	UNKNOWN		UNKNOWN
21	INCOMPLETE	L.M.N.	NIL	UNKNOWN		UNKNOWN
22	COMPLETE	L.M.N.	NIL		UNKNOWN	UNKNOWN
23	INCOMPLETE	U.M.N.	NIL	UNKNOWN	MODERATE	FLEXOR

U.M.N. = UPPER MOTOR NEURON
L.M.N. = LOWER MOTOR NEURON

NEUROLOGICAL FINDINGS

	Initial Severity	Late Reflex Pattern	Recovery	Involuntary Movements Extent Early	Late	Type
24	COMPLETE					
25	INCOMPLETE	L.M.N.	NIL			
26	COMPLETE	L.M.N.	NIL	NIL		NIL
27	INCOMPLETE	L.M.N.	SLIGHT	UNKNOWN		UNKNOWN
28	INCOMPLETE	U.M.N.	ALMOST FULL STIFF	NIL		NIL
29	INCOMPLETE	U.M.N.	SLIGHT	SEVERE	MODERATE	FLEXOR + EXTENSOR
30	INCOMPLETE	U.M.N.	ALMOST FULL STIFF		MODERATE	EXTENSOR
31	INCOMPLETE	U.M.N.	FULL MOBILE	NIL		NIL
32	COMPLETE					
33	INCOMPLETE	U.M.N.	SLIGHT	SEVERE		FLEXOR
34	INCOMPLETE	L.M.N.	FULL	NIL		NIL
35	INCOMPLETE	L.M.N.	SLIGHT	UNKNOWN		UNKNOWN
36	COMPLETE	UNKNOWN	NIL	UNKNOWN		UNKNOWN
37	INCOMPLETE	L.M.N.	UNKNOWN	UNKNOWN		UNKNOWN
38	INCOMPLETE	U.M.N.	ALMOST FULL STIFF	MILD		INDETERMINATE
39	INCOMPLETE	U.M.N.	SLIGHT	MILD		INDETERMINATE
40	INCOMPLETE	NORMAL	FULL NORMAL	NIL		NIL
41	COMPLETE					
42	COMPLETE	L.M.N.	NIL	UNKNOWN		UNKNOWN
43	COMPLETE					
44	COMPLETE	L.M.N.	NIL	NIL		NIL
44a	COMPLETE	UNKNOWN	UNKNOWN	UNKNOWN		
45	COMPLETE	MIXED	NIL	VERY MILD		? FLEXOR
45a	INCOMPLETE	U.M.N.	SLIGHT STIFF	SEVERE	MODERATE	EXTENSOR + FLEXOR
46	INCOMPLETE	L.M.N.	SLIGHT	VERY MILD		? EXTENSOR
46a	INCOMPLETE	L.M.N.	SLIGHT	UNKNOWN		UNKNOWN
47	COMPLETE	L.M.N.	NIL	VERY FAINT		NIL
47a	COMPLETE	MIXED	NIL	MODERATE	MILD	EXTENSOR + FLEXOR
48	COMPLETE	MIXED	NIL	SEVERE	MODERATE	EXTENSOR + FLEXOR
49	COMPLETE	MIXED	SLIGHT	MILD		EXTENSOR

U.M.N. = UPPER MOTOR NEURON

L.M.N = LOWER MOTOR NEURON

	Pain Type	Late Sex State
1		
2	NIL	NORMAL - VIABLE CONCEPTION 7YRS
3	UNKNOWN	UNKNOWN
4		
5		
6		
7	VISCERAL	UNKNOWN
8	ROOT AND VISCERAL	DESIRE NORMAL NO SENSATION HAS ERECTIONS + EMISSIONS
9	VISCERAL	NO ERECTIONS, EMISSIONS, SENSATION OR DESIRE
10	NIL	UNKNOWN
11	UNKNOWN	UNKNOWN
12	BURNING DIFFUSE	UNKNOWN
13		
14	VISCERAL	HAS ERECTIONS, EMISSIONS LOST DESIRE
15		
16		
17		
18	UNKNOWN	UNKNOWN
19	NIL	2 VIABLE CONCEPTIONS
20	UNKNOWN	UNKNOWN
21	UNKNOWN	UNKNOWN
22	BURNING DIFFUSE	UNKNOWN
23	UNKNOWN	UNKNOWN
24		

	Pain Type	Late Sex State
25		
26	BURNING DIFFUSE, VISCERAL	HAS EMISSIONS, DESIRE NO ERECTIONS
27	UNKNOWN	UNKNOWN
28	VISCERAL	NORMAL 2 VIABLE CHILDREN
29	BURNING DIFFUSE	HAS EMISSIONS NO DESIRE OR ERECTIONS
30	VISCERAL	NORMAL
31	UNKNOWN	UNKNOWN
32		
33	NIL	NORMAL
34	NIL	HAS ERECTION, DESIRE, PREMATURE EMISSION, NO INTERCOURSE
35	UNKNOWN	UNKNOWN
36	UNKNOWN	SPONTANEOUS ERECTIONS
37	UNKNOWN	UNKNOWN
38	BURNING DIFFUSE PAIN BACK AND HIPS	HAS ERECTIONS, EMISSIONS LIB.DO REDUCED
39	VISCERAL	UNKNOWN
40	UNKNOWN	UNKNOWN
41		
42	UNKNOWN	UNKNOWN
43		
44	SEVERE LOW SPINAL PAIN	NO ERECTIONS, EMISSIONS OR REFLEXES
44a	UNKNOWN	UNKNOWN
45	ROOT	NO DESIRE, ERECTIONS! OR EMISSIONS
45a	VISCERAL	NORMAL - VIABLE CHILD
46	ROOT	HAS DESIRE, SPONTANEOUS EMISSIONS. NO ERECTIONS, INTERC
46a	BURNING DIFFUSE BILATERAL CORDOTOMY	UNKNOWN
47	ROOT BURNING DIFFUSE	HAS ERECTIONS DESIRE REDUCED
47a	VISCERAL	NO EMISSIONS, INTERCOURSE HAS ERECTIONS - REDUCED DESIRE
48	ROOT	HAS ERECTIONS NO EMISSIONS - SUBDUED DESIRE
49	VISCERAL	HAS ERECTIONS LATE EMISSION - SUBDUED DESIRE

MORBIDITY

	Initial Hospitalisation	Ultimate State	Gainful Employment	Late Oedema	Late Ossification
1					
2	18 MONTHS	FULLY ACTIVE	FULL	NIL	NIL
3	5 MONTHS	FULLY ACTIVE	UNKNOWN	UNKNOWN	UNKNOWN
4					
5					
6					
7	8 MONTHS	DIED 10 YRS CHRONIC SEPSIS MYELONEPHITIS	NIL	NIL	NIL
8	24 MONTHS	CHAIR RIDDEN	NIL	NIL	NIL
9	16 MONTHS	SEMI BEDRIDDEN	NIL	SEVERE	SEVERE - HIPS
10	18 MONTHS	CHAIR RIDDEN	NIL	NIL	NIL
11	16 MONTHS	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN
12	34 MONTHS	SEMI BEDRIDDEN	NIL	NIL	NIL
13					
14	5 MONTHS	FULLY ACTIVE	NIL	NIL	NIL
15					
16					
17					
18	33 MONTHS	DIED 10 YEARS CHRONIC SEPSIS	NIL	NIL	NIL
19	7 MONTHS	FULLY WT. BEARING	NIL	TRANSIENT	NIL
20	13 MONTHS	FULLY WT. BEARING	UNKNOWN	NIL	NIL
21	14 MONTHS	DIED 13 YEARS CHRONIC SEPSIS	1 YEAR IN 12	UNKNOWN	UNKNOWN
22	48 MONTHS	DIED 4 YEARS CHRONIC SEPSIS	NIL	NIL	NIL
23	1 MONTH	FULLY WT. BEARING	UNKNOWN	NIL	NIL
24					
25	3 MONTHS	DIED 3 MONTHS ACUTE SEPSIS	NIL	NIL	NIL

MORBIDITY

	Initial Hospitalisation	Ultimate State	Gainful Employment	Late Oedema	Late Ossification
26	17 MONTHS	CHAIR RIDDEN	NIL	NIL	NIL
27	8 MONTHS	FULLY WT. BEARING	UNKNOWN	NIL	AROUND KNEE
28	5 MONTHS	FULLY ACTIVE	FULL	NIL	NIL
29	18 MONTHS	SEMI BEDRIDDEN	NIL	NIL	NIL
30	3 MONTHS	FULLY WT. BEARING	SPORADIC	NIL	NIL
31	10 DAYS	FULLY ACTIVE	UNKNOWN	NIL	NIL
32					
33	22 MONTHS	CHAIR RIDDEN	NIL	NIL	NIL
34	5 MONTHS	FULLY ACTIVE	SPORADIC	NIL	NIL
35	5 MONTHS	FULLY WT. BEARING	UNKNOWN	NIL	NIL
36	29 MONTHS	DIED 4 YEARS CHRONIC SEPSIS	UNKNOWN	SEVERE	NIL
37	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN
38	4 MONTHS	FULLY ACTIVE	FULL	NIL	NIL
39	24 MONTHS	DIED 2 YEARS CHRONIC SEPSIS BED RIDDEN	NIL		
40	3 WEEKS	FULLY ACTIVE	UNKNOWN	NIL	NIL
41					
42	5 MONTHS	SEMI BEDRIDDEN	NIL	NIL	NIL
43					
44	29 MONTHS	CHAIR RIDDEN	NIL	NIL	NIL
44a	14 MONTHS	DIED 7 YEARS CHRONIC SEPSIS BED RIDDEN	NIL	UNKNOWN	UNKNOWN
45	UNKNOWN	JUST WT. BEARING	NIL	NIL	NIL
45a	10 MONTHS	FULLY ACTIVE	NIL	NIL	NIL
46	20 MONTHS	WT. BEARING CALLIPER	NIL	SEVERE	NIL
46a	UNKNOWN	ACTIVE	UNKNOWN	UNKNOWN	UNKNOWN
47	5 MONTHS	CHAIR RIDDEN	NIL	NIL	NIL
47a	10 MONTHS	CHAIR RIDDEN	NIL	NIL	NIL
48	5 MONTHS	CHAIR RIDDEN	NIL	NIL	NIL
49	10 MONTHS	CHAIR RIDDEN	NIL	MODERATE	NIL