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Early decompression of the spinal cord following injury: arguments for and against

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Introduction

The management of the traumatized spinal cord has remained controversial over the past half century. Arguments for and against each treatment modality exist. Each is equally likely to be adopted or disparaged until possibly all factors hindering spinal cord recovery are identified. Direct comparison between surgery and conservative management in a series of unselected patients would contribute to solving the controversy (Lewis and McKibbin 1974).

The difficulties with design of studies and statistical evaluation of the results should not be underestimated. Problems of designing such comparative clinical studies are to be expected, since, even in the controlled animal studies, reproducibility is not always possible.

The idea that the words of Guttmann should be reiterated almost a decade after his death brings home the stark reality that we have come but a little way in determining the truth in the ever-present conflict over the operative as against the dynamic conservative management of these patients.

The current management varies from a conservative to a surgical approach. Surgery consists of either decompression or stabilization or a combination of both.

Basis of management

Most clinicians base their arguments on clinical experience and belief, as well as on the experimental and clinical work available in the literature. Frankel *et al.* (1969), Bedbrook (1969), Guttmann (1976), Burke *et al.* (1976), and other proponents of conservative management have concluded, following decades of experience, that most of the damage to the spinal cord occurs at the time of the accident, and that surgical decompression is in the majority of cases unlikely to alter the course of events favourably. Spinal cord decompression may result in neurological deterioration which is either transient (Bohlman and Eismont 1981; Gertzbein *et al.* 1988) or permanent

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(Morgan *et al.* 1971; Wiberg and Hauge 1988). El Masri *et al.* (in press) believe that this neurological deterioration is probably related to a 'physiological instability' in an injured spinal cord. This 'instability' is possibly due to a combination of spinal cord microvascular alterations, loss of autoregulation, and biochemical and enzymatic changes in patients with impaired autonomic functions.

Furthermore, this instability is not always associated with or directly related to the biomechanical instability of the bony column nor to the decompressive surgery.

It is possible that with extra attention to the haemodynamics of the traumatized patient, neurological deterioration may be reduced or avoided.

The concept of the secondary insult following injury dates back to the early 1950s, when Freeman and Wright (1953) proposed that loss of vascular perfusion secondary to bony encasement of an oedematous haemorrhagic contused spinal cord may cause a secondary injury. Many pharmaceutical agents and surgical procedures were tried in the hope of manipulating favourably this secondary insult.

The drug studies, however, failed to give uniform results: almost all agents have in some study failed to show an effect as tested in one or more laboratories (Collins 1983). Corticosteroids, the commonest clinically used drugs, were demonstrated to improve neurological recovery in animals. Ducker and Hamit (1969), Young and Dexter (1978), and many others administered steroids both in low and high doses. A multicentre trial comparing both dose regimes failed to demonstrate any statistically significant improvement (Collins 1983; Bracken *et al.* 1984, 1985). A randomized controlled trial of methyl prednisolone, naloxone, and placebo (Bracken *et al.* 1990) suggested, however, statistically significant neurological improvement with high doses of methyl prednisolone administered within 8 hours from injury. Information about the size of the population who improved and the degree of improvement, i.e. whether a small number of patients improved significantly or a large number of patients showed little improvement; information about whether it was improvement of density or level, or whether the improvement was local or distal, and the number of patients with Brown-Séquard Syndrome in each group all require consideration in the evaluation of the clinical significance of the statistics.

The advocates of surgical decompression believe that mechanical compression by bony fragment and/or soft tissue is likely to be responsible for some of the persistent neurological deficit, especially in patients with incomplete spinal cord injury. Complete anatomical transection of the spinal cord is rare (Bohlman 1979). Of 67 patients who had a clinically complete injury, 50 showed some continuity across the injured segments (Kakulas 1988).

Whether it is possible to halt or reverse the secondary injury by early decompression is still unclear in the clinical situation.

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Pathophysiology of spinal cord injury

The macro- and microscopic pathology of spinal cord injuries has been well described by Hughes (1978). The area of interest in the traumatized spinal cord is centred around the microvascular circulation (Turnbull 1971; White 1975). Of note is the absence of any interconnection of the terminal arteries in the spinal cord, though there is an extensive network of interlocking capillaries around the cord (Turnbull 1971). The capillaries are significantly increased in the central grey matter to accommodate the increased metabolic demand in this area, to the extent that each neuron is connected to at least one capillary.

Understanding the spinal cord blood-flow will lead to the realization that extraspinal circulatory changes are very likely to affect the injured cord. Korbine *et al.* (1975) reported loss of autoregulation in the lateral columns of the traumatized cord. As such the cardio-vascular status and haemoglobin with its oxygen-carrying capacity must not be seen as unrelated or isolated. Turnbull (1971), Ducker and Perot (1971), Assenmacher and Ducker (1971), Ducker *et al.* (1971), Dohrmann *et al.* (1971, 1973) and White *et al.* (1969) have indicated that the microvascular changes following a spinal cord injury are reflected initially in the grey matter, and that there is a correlation between the neurological deficit and the degree of subsequent involvement of the white matter. The biochemical changes at the site of impact also correlate with the vascular status in that the cord oxygen drops almost immediately at the site of the traumatized segment, as noticed in the dog (Kelly *et al.* 1970, 1972). Ducker and Perot (1971) confirmed this but found that it occurs between 2 and 3 hours after the injury. It is preceded by a reduction in blood-flow.

Within about 1½ minutes a significant rise in the lactate levels lasting for about 24 hours was observed by Locke *et al.* (1971). By 1 hour after injury multiple areas of haemorrhage are present in the central grey matter, with generalized loss of cellular architecture in the grey matter. This is followed by the coalescence of the micro-haemorrhages within the grey matter at 2–4 hours following the injury (White *et al.* 1969).

An increase in the water content of the spinal cord appears as early as 5 minutes after trauma, and persists for as long as 15 days (Yashon *et al.* 1973). It is clear that there are a number of parameters that can be measured in the experimental situation, though with some difficulty. Different experimental treatment regimes have been undertaken in the hope of manipulating these parameters. As yet a correlation between the efficiency of this manipulation and its neurological outcome in the laboratory with events in the clinical situation remains to be proven.

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Problems of the experimental model

Osterholm (1978) highlighted the difficulties encountered with the reproducibility of results in the controlled laboratory environment. The experiments of Allen in 1911 suggested that the rapidity of the initial insult has a significant bearing on the outcome of cord injury. Slow compression irrespective of size may give rise to minimal neurological fall-out. A small acute compression, however, has a profound effect, frequently causing complete cell destruction. Olsson (1971) and Dohrmann and Panjabi (1976) demonstrated that by dropping different weights from different heights to achieve a 400 gm/cm impact on the cat spinal cord, using an impounder, the volume of the lesion produced in the spinal cord was variable despite the controlled experimental design.

They demonstrated that the amount of energy transferred to the spinal cord was dependent upon factors such as height, impounder mass, and velocity. The latter authors in 1978 also stated that 'force may well be a most reasonable means of quantitating experimental spinal cord trauma'. These factors are virtually impossible to measure in the clinical situation. To date there are no investigative procedures to measure accurately the initial volume of the spinal cord lesion. The previous factors can explain the difficulties in comparing outcome of different management procedures and producing convincing statistical evidence for the superiority of one method of management over others.

Improvement following spinal surgical decompression is currently suggested in various experimental studies. In Bohlman and colleagues' (1979) study of anterior screw compression on the spinal cord of dogs, it is noteworthy that all animals became paralysed to varying degrees. However, despite the ongoing compression some began to improve their cord function. Of the eight dogs with piston compression, three did not require any decompressive surgery to regain their pre-operative neurological status. Although the remaining five animals required decompression to recover, their pre-operative neurological status is not revealed.

Dolan *et al.* (1980) used the clip compression model, with forces ranging from 16 gm–178 gm and duration of compression ranging from 3 seconds to 900 seconds, in four different groups of rats. They suggest that following decompression, the subsequent functional recovery over a period of 8 weeks was related to the duration of compression regardless of the actual force. Among the difficulties in extrapolating this to the human situation is the fact that the latest decompression in the experimental model was done 15 minutes after compression.

The same laboratory in 1987 (Guha *et al.* 1987) used the same injury model but with a smaller range of forces applied to the cord. These forces ranged between 2.3 and 53.0 gm, and decompression was performed after 15, 60, 120, and 240 minutes of compression. Functional recovery was

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assessed weekly for 8 weeks. They found that animals injured with the low forces (2.3 gm clips) performed significantly better than those injured with higher forces for all times until decompression. There was also a significant difference in recovery between the groups injured by the 16.9 and 53.0 gm clips, although only for the group decompressed at 15 minutes. Early decompression did not improve the neurological outcome with the 53 gm clip. The animals compressed at 2.3 gm and decompressed after 15 or 16 minutes had significantly better function than those decompressed after long periods (120–240 minutes).

The authors concluded that, with lower forces, decompression as early as possible and within 240 minutes (simulating the clinical situation) can be beneficial.

We feel that, apart from the control group of rats whose spinal cords were not compressed the inclusion of another group of rats with unrelieved compression might have helped to simulate the clinical situation with its attendant controversy. The neurological outcome of rats with unrelieved compression, especially of the lower magnitude, would have been most interesting to observe.

Decompression by establishing anatomical reduction

Most clinicians would agree that reduction and restoration of vertebral alignment is accompanied by some decompression of the spinal cord (Jacobs and Casey 1984; Dickson *et al.* 1978; Yosipovitch *et al.* 1977; Fletsch *et al.* 1977; White *et al.* 1980).

Wiberg and Hauge (1988) found that decompression was not always achieved following the application of distraction rods and realignment of the spine in thoracic and lumbar injuries. On re-exploration of the ventral aspect of the spinal canal, there was significant compression from bone fragments in 43 per cent of patients in the latter study. McAFee *et al.* (1985) and Jelsma *et al.* (1982) reported similar cases where following restoration of alignment by distraction rods there was persistent compression.

Frankel *et al.* (1969) found no correlation between the bony lesions, the various degrees of reduction, and the neurological lesion. Durward *et al.* (1981) observed poor correlation between the degree of canal compromise as measured by computerized tomography and the resulting neurological deficit.

The following case history demonstrates that reduction is not imperative for the achievement of neurological improvement. The value of conservative management is postulated:

A 74-year-old lady was involved in a road-traffic accident at the age of 60. She sustained a bilateral facet dislocation of C5 upon C6 as well as a fracture of the pedicle of C2. The initial clinical presentation indicated a symmetrical weakness of the wrist

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extensors and the triceps, with minimal power in the intrinsic muscles of the hand. There was no power in the lower extremities; no control of the bladder or bowel, but complete sensory preservation. Her reflexes in the biceps and triceps were present, but absent in the lower limbs.

The patient was managed in skull traction without effectively reducing the cervical dislocation. Despite this she began to recover function to the lower limbs and the hands at 2 days. Skull traction was discontinued at 6 weeks. At 3 months after the injury the patient was walking and demonstrated control of her bowel and bladder.

After 13 years the patient had full power in the legs, with hyperreflexia of the lower limbs. The only deficit was confined to the left hand, which demonstrated minimal loss of full wrist extensor power [4/5] and the loss of power of the intrinsic muscles of the hand, which did not recover.

The first X-ray demonstrates the fracture of the pedicle of C2 and bilateral dislocation of the facets at C5/C6 (Fig. 1.1). The second X-ray (10 years after the injury) confirms the union of the above injury, still in the dislocated position (Fig. 1.2).

In our experience late reduction of the cervical spine (after 48 hours) can result in a significant neurological deterioration. We believe that it is particularly hazardous for the inexperienced clinician to attempt closed reduction at this late stage.



Fig. 1.1. Lateral radiograph of the cervical spine with a fracture of the pedicle in C2, and a bilateral dislocation of the facet joints C6 upon C7.

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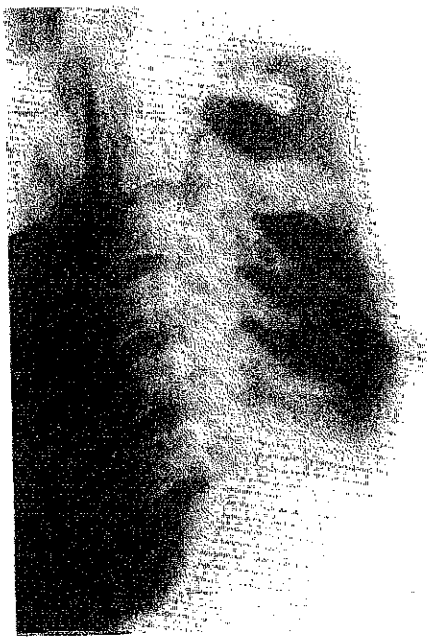


Fig. 1.1. Lateral radiograph of the cervical spine with a fracture of the pedicle in C2, and a bilateral dislocation of the facet joints C6 upon C7.

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Fig. 1.2. Lateral radiograph of the cervical spine ten years later. Follow-up demonstrating the healed pedicle fracture of C2 and the union of C6 to C7 in the dislocated position.

The current difficulty in predicting which type of injury will benefit from decompression by establishing anatomical reduction is probably due to the fact that the commonly available diagnostic tools do not allow complete and exact identification of the details of the injury. It may be that bony fragments attached to the undamaged posterior longitudinal ligament encroaching on the canal and/or spinal cord would be stretched away from the canal and its contents by distraction/reduction. In this instance, establishing anatomical alignment would be beneficial. If, however, the posterior longitudinal ligament is torn or the bony fragment is not attached to this ligament, reduction or retraction are unlikely to be accompanied by decompression. Another cause of failure of the decompression is 'locking' of the bony fragments. Further work with Magnetic Resonance Imaging (MRI), with its capacity to delineate ligaments, may contribute to the identification of those injuries in which decompression is likely to succeed with distraction and or realignment (see Figs. 1.3, 1.4, and 1.5).

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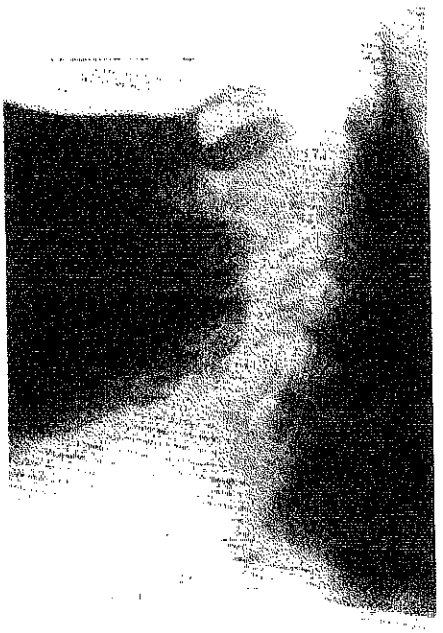


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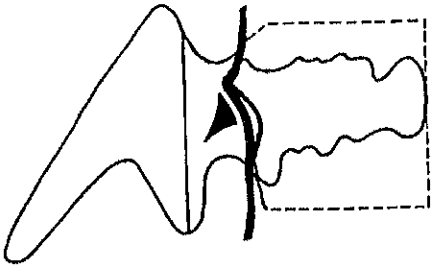


Fig. 1.3. Sagittal section of the vertebral body indicating the bony fragment separate from the posterior longitudinal ligament, precluding it from effective reduction by distraction.

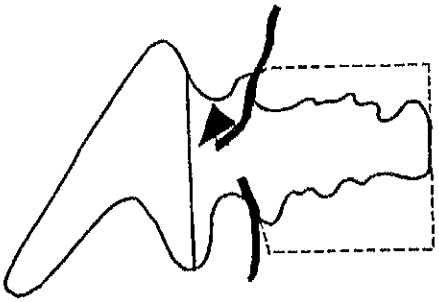


Fig. 1.4. Sagittal section through the vertebral body demonstrating a torn posterior longitudinal ligament and its inability to produce reduction despite traction.

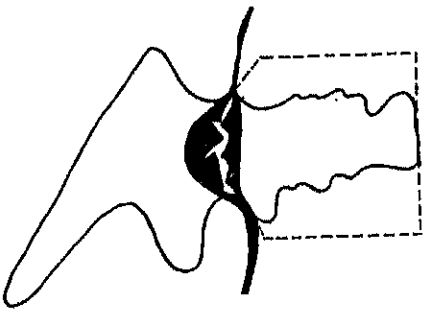


Fig. 1.5. Sagittal section through the vertebral body showing 'locking' of the bony fragments, which in turn prevent complete reduction despite traction.

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Decompression by laminectomy

Routine decompressive laminectomy has little to offer to patients with spinal injury (Seljeskog 1982; Wiberg and Hauge 1988; McAFee *et al.* 1985). Furthermore it can lead to and/or increase instability and spinal deformity (Verbiest 1963; Jacobs and Casey 1984; Bradford *et al.* 1977; Bohlman and Eismont 1981; McAFee *et al.* 1985).

Neurological deterioration following laminectomy was highlighted by Guttmann (1949), Benassy *et al.* (1967), Morgan *et al.* (1971), Bohlman (1979), and Dickson *et al.* (1978).

In the presence of the narrowed spinal canal and an injured spinal cord the jaws of the rongeur removing the laminae can inflict further damage to the cord (Verbiest 1963). Adequate decompression cannot be achieved by laminectomy when the compressive element is in the anterior aspect of the canal (Bradford *et al.* 1977; Fleisch *et al.* 1977). Laminectomies are still done in conjunction with other surgical procedures such as posterior instrumentation (Seljeskog 1982) and pedicular screwing of the Roy-Camille's plate, (Lagarrigue *et al.* 1980).

Our indication for laminectomy is empirically restricted to patients who may deteriorate with an ascending neurological deficit of more than 2–3 segments, when computerized tomography reveals posterior compression of the spinal cord or cauda equina by bony fragments or soft tissue. This is however a rather infrequent occurrence.

Anterior decompression

During this last decade we have seen enthusiasm in some surgical quarters for decompression of the anterior aspect of the spinal canal through an anterior approach or a postero-lateral approach, depending on the level and the type of injury. This is possibly related to the disappointing results of routine laminectomy, the lack of convincing evidence that surgical realignment and internal fixation improved the neurological outcome, the possible neurological deterioration with sublaminar wiring (Rossier and Cochran 1984), and the advent of computerized tomography, revealing anterior compression of the neural tissue as well as narrowing of the sagittal diameter of the canal. The proponents of anterior decompression believe that in patients with an incomplete neurological lesion, improvement can be achieved by the surgical removal of the elements encroaching on the canal. This is suggested as a treatment modality for the cervical spine by Bohlman and Eismont (1981), and in the dorso-lumbar spine by Kadena *et al.* (1984), McAFee *et al.* (1985), McEvoy and Bradford (1985), and Dunn (1986). Indeed further collapse of the vertebral column has been observed by Gertzbein *et al.* (1988) following anterior decompression. A further surgical stabilizing procedure is usually

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necessary in view of the additional instability that could occur. Dislodging of an anterior bony graft has been observed by the authors in the cervical spine following decompression without stabilization.

While it is possible to combine anterior decompression with posterior stabilization and fusion or anterior plating, in a one-stage procedure, this is likely to be associated with increased mortality and morbidity if contemplated within the first 50 hours from injury in a patient with a multi-system deficit.

Decompression is usually performed at the site of the vertebral fracture or fracture dislocation. Assenmacher and Ducker (1971) demonstrated that in an experimental traumatic paraplegia the vascular and pathological changes seen in irreversible cord lesions can extend up to 5 times the length of the traumatized segment. Indeed in the two post-mortem specimens we examined following the death of two patients with complete spinal cord injuries, the cord lesion did extend beyond the traumatized unit of motion. It was argued that in these two patients anterior decompression restricted to the site of the fracture would have been incomplete. The incidence of this problem is not yet known. Furthermore we are in agreement with Bedbrook (1975, 1979) that early myelography is of no clinical value in distinguishing the extent of damage in the spinal cord, nor of management value, in determining the need for decompression (Guttmann 1973). It might be possible in the near future to determine the volume and the extent of the cord lesion by MRI imaging and spectroscopy.

Myelotomy

Allen in 1911 reported that in dogs with severe cord injury, death would occur unless myelotomy was performed. Freeman and Wright (1953) performed myelotomy following experimental spinal injuries on dogs, and reported better than expected neurological improvement compared to the untreated animals.

The beneficial effects of myelotomy may be explained by a number of possible mechanisms. The removal of pressure, blood, and necrotic tissue from within the spinal cord has been postulated by Allen (1914) and Campbell *et al.* (1973); by decreasing cavitation, thus protecting the surviving axons from further damage (Freeman and Wright 1953), or by improving spinal cord blood-flow from the reduction in tissue pressure and vasoactive substances (Rivlin and Tator 1979). However the procedure could cause irreversible cord damage (Scarff 1960) or some further destruction of cord tissue (Tarlov 1957). Benes in 1968 performed myelotomy in 20 patients, four of whom were eventually able to walk with crutches. Unfortunately the completeness of the lesion prior to surgery could not be ascertained. The death-rate following the procedure was 40 per cent.

Although we have no direct experience with the procedure we know of

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cases where 'toothpaste-like' cord material had exuded through dural tear during surgical decompression. We are not aware of any surgeon undertaking this procedure routinely in the present time.

Timing of surgical decompression

There is no general agreement between surgeons as to the optimum timing of surgical decompression by whichever method. Immediate or early decompression is advocated by Bedoiseau *et al.* (1971), Jacobs (1980), and Dunn (1986).

Wiberg and Hauge (1988) believe that early surgery may have been beneficial, with neurological improvement, in two of his patients who presented with complete cord injuries and who were decompressed within 10 hours after the accident.

On the other hand Larson *et al.* (1976), McAFee *et al.* (1985), Bedbrook (1975, 1979), Bradford *et al.* (1977), and Willen *et al.* (1985) believe that early or immediate surgical decompression does not appear to provide additional neurological recovery in the severely contused spine.

By comparing the neurological outcome of patients with cauda equina lesions who had undergone immediate, early, and late decompression, Fleisch *et al.* (1977) could not show any difference in the neurological recovery.

It is difficult to determine the value of early decompression in view of the dynamic nature of the neurological picture. It is a well-known fact that a certain degree of spontaneous neurological recovery occurs in many patients with incomplete neurological lesions and in some patients with complete paralysis on presentation. Furthermore, neurological deterioration associated with active intervention cannot be measured or ruled out.

Complications of surgical decompression and conservative management

The injured swollen cord is probably especially vulnerable to any further trauma, however minimal. Rossier and Cochran (1984) reported deterioration in a patient with dorsal injury and another patient with dorso-lumbar injury, following open reduction and sublaminar wiring with Harrington compression rods. Wiberg and Hauge (1988) reported cord swelling during surgical decompression converting incomplete (subtotal) lesions into complete lesions. They reported protrusion of 'oedematous cord substance ... like toothpaste through dural rents'. Transient neurological deterioration was also observed immediately following surgery in 'several cases' undergoing an anterior decompression and/or a posterior procedure by Gertzbein *et al.* (1988). McAFee *et al.* (1985) reported on 70 patients with spinal cord injury and dorso-lumbar fractures treated by anterior spinal cord

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decompression: three of these cases had transient neurological deterioration from radiculopathy; while Bohlman and Eismont (1981) reported three patients who deteriorated following post-operative improvement in a series of 100 patients who had undergone late decompression of the cervical spine.

Dunn (1986) states that significant complications occur following anterior stabilization and decompression for thoraco-lumbar injuries. He feels, however, that on balance the neurological recovery of his patients with incomplete lesions was more favourable than those of the Frankel *et al.* (1969) and the Dickson *et al.* (1978) series.

Non-neurological complications such as pneumonia, respiratory distress, pulmonary compromise, oesophageal perforation, thoracic duct tear, graft extrusion, and graft collapse have been reported in the literature following surgery, as well as meningitis and brain damage.

With conservative management and frequent, regular, accurate neurological examination we have observed in a small number of patients a transient ascent of the neurological level involving one or two segments above the level of the lesion. This usually occurs within 48–120 hours following the injury. This is almost invariably temporary, and we confirm the observation by Frankel (1969) that within a few weeks the initial neurological level descends to correspond to that found immediately following the injury, or even to a lower level.

A less common form of deterioration, with permanent ascent in the level of lesion of more than 2 segments and up to 7 segments, occurs in about 1.5 per cent of patients with mainly dorso-lumbar injuries, usually within the first two weeks following the injury and less commonly in the third week following the accident (Frankel 1969). The pathology of this condition is not clear, however: the final neurological outcome in six out of the seven patients documented by Frankel was flaccid paralysis, suggesting a vascular aetiology. The one patient who had two laminectomies performed did not improve. The incidence of transient neurological ascent of 1 or 2 levels of lesion is not well documented in the operative management series. The topography extent, and type of paralysis when deterioration occurs following surgery is not usually well described. Indeed it may be difficult to determine post-operatively if a neurological deterioration is due to the natural injury process or is related in someway to the surgical procedure (Stauffer 1989).

When approaching the management of spinal injury one needs to consider the 'spinal cord instability' as well as the vertebral column instability. In the unstable vertebral injury there is a potential risk for further displacement and mechanical compression on the spinal cord resulting in further neurological deterioration. This instabilty is usually contained by either conservative or surgical means. Neurological deterioration can occur, however, owing to

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factors unrelated to mechanical compression. The pathophysiological changes in the spinal cord suggest the presence of an inherent physiological instability following trauma (El Masri *et al.*, in press). Indeed hypotension, hypothermia, hypoxia, or septicaemia can result in neurological deterioration or slow down the neurological recovery rate in patients with incomplete lesions.

This association has been observed by us as well as by clinicians from both the conservative and the surgical schools. The effect of anaesthetic agents on the traumatized spinal cord requires thorough assessment.

Patients with spinal cord injury initially have a multi-system deficit which can be the source of many complications, and may result in hypotension, hypoxia, and infections. Anaesthetic and/or surgical procedures can, at least potentially, increase the risk of these complications. Whether extreme care would completely abolish the extra risk of neurological deterioration is arguable.

Neurological outcome of management

To date there is no convincing evidence that the final neurological outcome is favourably altered by surgical decompression. Young and Dexter (1978) found no statistically significant difference between the neurological progression of their patients and those reported in the earlier Stoke Mandeville study by Frankel *et al.* (1969). We have found no convincing evidence of any advantageous neurological outcome following surgery by carefully analysing McAFee and colleagues' series (1985) of anterior decompression for thoraco-lumbar fractures with incomplete neurological deficit; Wiberg's and Hauge's series (1988) of surgery for the thoracic and lumbar spine treated with Harrington distraction device and additional anterior decompression in 43 per cent of patients; or the Gertzbein *et al.* series of 1988. The last also concluded that there was no clear difference in the outcome between anterior versus posterior surgery, regardless of the level of the lesion. Osti *et al.* (1989) reviewed their patients with cervical injuries and concluded that early surgical stabilization of the cervical spine using a dowel interbody fusion seemed to impair neurological recovery in patients with neurological deficit. Tator *et al.* (1987) compared the outcome of surgical and conservative management in 208 patients with acute spinal cord injury. They have shown that statistically there was no difference between operated and non-surgically treated patients in length of stay or in neurological recovery. Wilmot and Hall (1986) found no difference in the neurological outcome between surgically and conservatively treated patients with paraplegia.

Folman and El Masri (1989) reviewed 70 patients with closed traumatic incomplete injuries of the spinal cord admitted within 72 hours of their accident to the Midland Centre for Spinal Injuries, and treated conservatively.

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There were 50 patients with incomplete cervical and 20 patients with incomplete thoracic cord injuries. No patients with cauda equina or root lesions were included. The Frankel classification was used for an average follow-up of 4.7 years. Of the 37 patients who presented with sensory sparing only (group B) 24 patients [65 per cent] recovered useful motor function, of whom 5 patients made full recovery. Of the 21 patients presenting with useless motor sparing (group C) 18 patients [86 per cent] recovered useful motor function, of whom 12 [60 per cent] recovered completely. Of the 12 patients who presented with useful motor sparing (group D) 9 patients [75 per cent] recovered completely. Gotfried and El Masri are presently reviewing a series of patients with dorso-lumbar lesions treated conservatively. Our impression is that patients with cauda equina lesions have an even better neurological outcome than patients with cord lesions, and that the results of conservative management compare favourably with surgical decompression.

The following case history highlights the value of conservative management of the dorso-lumbar fracture with a cauda equina lesion:

JS, a 19-year-old girl, was involved in a motor-cycle accident on 1/8/87. She presented a burst fracture of the first lumbar vertebra. Her clinical picture demonstrated weakness of the lower limbs, with a symmetrical loss of power in the hip muscles of grade 2/5 and a power of 2/5 in the rest of the lower limb. The knee and ankle reflexes were absent on admission. She had analgesia to pinprick in the S1 to S5 areas bilaterally, and no bladder or bowel control. Her initial X-rays demonstrate the burst fracture of L1, and the CAT scan confirms the retropulsion of fragments into the canal, almost obliterating it.

The patient was treated conservatively with bed-rest for a period of 6 weeks, and at that time her power in the legs had improved to grade 4/5 throughout and the sensory deficit had improved in the S3 dermatome. During this period the S3–S5 dermatome was still analgesic to pinprick, and the bladder and bowels required manual expression.

At about three months after the accident the patient had regained her bladder function as well as a partial return of sensation in the sacral dermatomes.

At the two-year follow-up the patient is able to walk as far as she wants and the power in the legs is normal. She still has a diminished sensory perception to pinprick in the sacral segments, but her bladder and bowel control is normal. Both the knee and the ankle reflexes have returned. She demonstrates improvement despite the ongoing neural compression. The range of movement in her dorso-lumbar spine is normal (Figs. 1.6, 1.7, and 1.8).

There is concern amongst some clinicians that bony fragments left unremoved in the spinal canal may produce further neurological deterioration either in the short or the long term. This is not borne out by our own experience. Evidence of remodelling of the spinal canal with receding of the bony fragments has been reported recently by Weinstein *et al.* (1988) and Fidler (1988). The recession or disappearance of the bony fragment from the canal in some cases may explain why late neurological deterioration, other

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than that due to post-traumatic syringomyelia, is most unlikely to happen, especially in the cauda equina lesions.

Most studies attempting to define the place of surgery in the management of spinal cord injured patients have compared a group of patients treated by a single method with either a historical control group from the same institution or from the literature treated by another method (Tator *et al.* 1987). Although prospective randomized controlled studies would be of value in determining the benefits of any surgical procedure including late decompression, there are a number of problems which may explain why such a study has not been done. The multitude of patterns of injuries and of the opinions for the treatment of any particular injury, the dynamic nature of the pathology and the clinical neurological progression, the difficulty of accurate assessment of the rate of the neurological progress, the variable times of presentation of the patient with spinal cord injury to the specialist units, the different emphasis on the importance of certain medications, the lack of an easy objective method for prospective evaluation, the unavailability of accurate investigative diagnostic procedures, the poor resources in some specialist units, and the need for several specialized centres to co-operate in such a study are some of the reasons why it has not yet been done. This is not to say that it is beyond the ingenuity of our caring profession.



Fig. 1.6. Anterior radiograph of the lumbar vertebrae showing a burst fracture of L1.

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Fig. 1.6. Anterior radiograph of the lumbar vertebrae showing a burst fracture of L1.

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Fig. 1.7. Lateral radiograph of the lumbar vertebrae demonstrating the burst fracture of L1.



Fig. 1.8. CAT scan of the L1 vertebra showing the retropulsion of the 'body' of L1 almost obliterating the canal.

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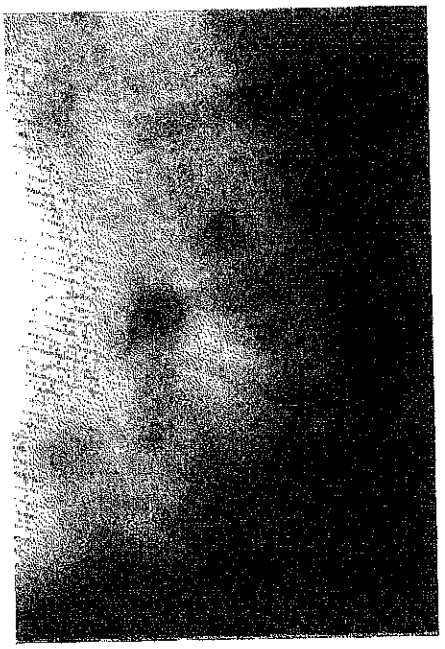


Fig. 1.7. Lateral radiograph of the lumbar vertebrae demonstrating the burst fracture of L1.



Fig. 1.8. CAT scan of the L1 vertebra showing the retropulsion of the 'body' of L1 almost obliterating the canal.

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Conclusions

Early surgical decompression has yet to prove particularly beneficial to the neurological outcome of patients with spinal cord injury. In our experience the great majority of patients presenting with sensory or motor sparing as early as 6 hours following the injury or later will make some neurological recovery with conservative management. In general the more neurological sparing is evident at presentation, the greater the degree of expected recovery. Recovery is likely to continue for about 18 months following the injury; however, the rate of the recovery is usually much higher in the first 6 months. During the first 2 months following injury, the spinal cord is physiologically unstable and vulnerable to hypoxia, hypotension, hypothermia, and septicaemia. Anaesthesia and surgery present an extra potential hazard to the injured cord in inexperienced hands. To date we have reserved decompression for the occasional patient who may show significant neurological deterioration. However, we have no evidence of its beneficial value. We feel that MRI imaging and spectroscopy, when refined, are likely to contribute to our understanding of the pathological processes in the spinal cord, and may help us improve our understanding of the role of early decompression in the management of spinal cord injury. We would be prepared to consider early surgical decompression if and when objective evidence of its beneficial value becomes available.

)urst fracture



e 'body' of L1

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