NEUROLOGICAL COMPLICATIONS OF THE REDUCTION OF CERVICAL SPINE DISLOCATIONS

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We have studied the case records of 16 patients with dislocations of the cervical spine who deteriorated neurologically during or after reduction. The dislocations were reduced by skull traction in four patients, by manipulation in four and by operation in seven.

This complication was not related to age, sex, mechanism of injury, or the level and the type of dislocation. Fourteen patients made substantial recoveries, one made a partial recovery and one patient remained totally paralysed and died three months later. The causes and prevention of spinal-cord damage at this stage of management are discussed, and the early use of MRI or CT myelography is recommended.

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Traumatic facet dislocation of the cervical spine, unilateral or bilateral, may or may not be associated with a fracture. Severe neurological damage is usual, but some patients sustain little or no injury to the spinal cord or nerve roots.

The primary aim of treatment is to achieve reduction and stability while preserving neurological function, using slowly increasing skull traction, closed manipulation or open reduction with surgical fusion. Spinal-cord injury may result during the reduction. Neurological deterioration has been reported to occur after skull traction (Burke and Berryman 1971; Fried 1974), during operative reduction (Bohman 1978) and after disc prolapse (Eismont, Arena and Green 1991; Robertson and Ryan 1992).

Secondary neurological damage is a major complication with considerable morbidity. We have studied the causes in a retrospective study of 16 consecutive patients seen at a secondary referral centre.

PATIENTS AND METHODS

We reviewed the medical records of 1321 patients admitted to the National Spinal Injuries Centre (NSIC) with cervical spinal injuries from 1944 to December 1991. Of these 341 had traumatic dislocations of the cervical spine, 291 with major cord injury. Fifty patients had minimal or no cord involvement. One patient had a nerve-root injury alone.

There were 15 patients who had presented initially to the referring hospital with dislocation of the cervical spine and with minimal or no features of spinal-cord involvement, who had later developed cord damage as a result of reduction. After the onset of secondary paralysis, they had been transferred to us for further treatment and rehabilitation. In addition, one patient who was never admitted to the NSIC is included. These form the basis of our study.

There were 14 men and two women; their average age was 43 years (24 to 73). The causes of the injuries were road-traffic accidents in nine, a fall in four, sport in two and diving in one. Nine patients had local neck pain, and six had neck pain with radiation to the arms. There was no record of neck pain in one patient. Records were inadequate to define other clinical parameters such as tenderness.

Radiology. The initial prereduction radiographs of the cervical spine were examined to determine the level and type of dislocation, associated fractures, spondylitic changes and the amount of anterior subluxation. The sagittal diameter of the cervical spinal canal at the level of the dislocation on the postreduction lateral film was measured.

All available radiological investigations of each patient after paralysis had developed were studied to establish the adequacy of the reduction, the size of the spinal canal, and the nature of cord compression.
RESULTS

The results of our retrospective review of 16 patients are outlined in Figure 1. Four cases are illustrated and described in Figures 2 to 5.

Neurology (Table I)

Before reduction. Seven patients were normal, eight had minimal cord damage and one patient had a nerve-root palsy. Two patients gave a history of transient paralysis immediately after the accident, which recovered within a few minutes.

During and after reduction. Seven patients were found to have complete paralysis, six after operation and one after manipulation. One of these patients also developed cerebellar infarction. There was complete motor paralysis with some sparing of sensation in five patients, in two
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Fig. 2

MRI of a 41-year-old man who sustained unilateral facet dislocation at C5-C6 with no neurological involvement. Operative reduction carried out using Halifax clamps resulted in severe cord involvement. After transfer to the NSIC, MRI showed a disc prolapse. Treatment was conservative and there has been slow but incomplete neurological recovery.

Fig. 3a

A 28-year-old man was treated overseas for bilateral facet dislocation at C5-C6 with no neurological involvement (a). Treatment by skull traction using 41 kg traction resulted in gross over-traction and caused nerve-root palsy (b).

after skull traction and in three after manipulation. One patient had increasing paralysis after skull traction, involving the upper limbs only and one developed mild paralysis of all limbs during the application of a plaster cervical collar in a sitting position. A nerve-root palsy developed in one patient when a skull caliper slipped at the weight of 41 kg (90 lb). One patient had mild paralysis of all limbs after an operation.

Recovery. Some neurological recovery occurred in the majority of these cases as reported under final outcome.

Radiology

Before reduction. The levels of dislocation were C1-C2 (1), C2-C3 (2), C3-C4 (1), C4-C5 (3), C5-C6 (4) and C6-C7 (5). Eight patients had bilateral facet dislocation and five had unilateral dislocation. One patient had nonunion of a previous fracture of the odontoid process which gave a C1-C2 dislocation, and the two with dislocations at C2-C3 had associated fractures.

The average forward shift in unilateral facet dislocation was 5 mm (4 to 6), which is less than one-third of the width of the vertebral body. In bilateral facet dislocation the average shift was 10 mm (8 to 12), equal to or more than half the width of the vertebral body. The sagittal diameter of the cervical canal at the sites of bilateral facet dislocation on the postreduction lateral film was between 16 mm and 20 mm (average 18.6). Ten patients had associated minor fractures of the spinous
process or facet at the site of dislocation. Spondylitic changes were found in only five patients, but always resulted in narrowing of the spinal canal. One patient had bony ankylosis of the cervical spine below the level of dislocation.

After attempted reduction. Radiographs of the cervical spine showed complete reduction in eight patients, partial reduction in two, and failure to reduce in two. Redislocation occurred in two patients. In one patient, unilateral dislocation became bilateral during manipulation, and in another, bilateral dislocation became unilateral when the skull caliper disengaged under traction of 41 kg. This patient and one other showed over-distraction on plain radiographs. The two patients

A 19-year-old man, not included in our series, sustained a bilateral facet dislocation at C4-C5 with no neurological involvement (a). An operation to reduce the dislocation was carried out three days after injury, and 18 hours later he developed a dense paralysis. Myelography showed gross swelling of the spinal cord with a block at C4-C5 but no extrinsic compression (b). Paralysis remained complete.

A 73-year-old man sustained a C3-C4 bilateral facet dislocation in a spondylitic cervical spine with very minimal neurological involvement (a). An immediate attempt at reduction, with a lengthy and difficult intubation, resulted in dense tetraplegia and cerebellar infarction, shown on MRI (b). He died three months later.
with redilocation and the two with a change of
dislocation were all fully reduced later.

Myelograms were performed in six patients at this
stage. There was a complete block without extrinsic cord
compression in one, a small filling defect in one, acute
angulation of the cord in one, and no block in two. One
patient had a disc protrusion. CT was performed after
myelography on three patients; these all showed intrinsic
cord swelling with no extrinsic compression. CT scans
alone in one patient were inconclusive.

MRI was performed on two patients. One showed a
posterior disc prolapse and a haematoma, the other
cerebellar infarction and spinal-cord swelling.

**Treatment.** In most cases, the dislocation was difficult
to reduce, and necessitated the use of multiple procedures
before or during the onset of paralysis. In nine patients
these were: a cervical collar and manipulation (1), two
episodes of skull traction (1), skull traction and operation
(3), collar, traction and operation (1) and late operation
of the cervical spine.

| Table I: Progression of paralysis in 16 patients who
deteriorated during or after reduction of dislocation of
the cervical spine |
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<td>Frankel grade</td>
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<td>Outcome</td>
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<td>A</td>
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<td>7</td>
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<td>B</td>
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<td>Nerve-root palsy</td>
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| Table II: Timing of reduction leading to neurological deterioration |
|----------------------|----------------------|----------------------|----------------------|----------------------|
| Treatment            | Interval between injury and treatment (hours) |
|                      | 0 to 6 | 6 to 12 | 12 to 24 | 24 to 48 | 48 to 72 | > 72 |
| Skull traction       | -      | 2       | 1        |          |          | 1    |
| Manipulation         | 1      | -       | 2        | 1        |          |      |
| Operation            | -      | -       | 1        |          | 2        | 4    |
| Application of cervical collar | -      | -       | 1        |          |          |      |

for instability (3). Thus 20 procedures were carried out
on these nine patients. Seven patients had only one form
of treatment: one had a cervical collar, three had skull
traction and three had manipulation.

The timing of each type of treatment is recorded in
Table II. In 11 patients, definitive reduction was
attempted between 4 and 72 hours after the injury. In the
other five, late reduction was attempted, the longest
interval being eight weeks. All 16 patients became
paralysed within 48 hours of the last attempt at reduction,
only five showing immediate neurological deterioration
(Table III, Fig. 1). After development of spinal-cord
damage, skull traction was continued in eight patients,
using minimal weight to maintain or improve reduction.
Three patients had an operation to restore the integrity
of the spinal canal and to achieve stability. Five patients
were given a cervical collar to provide external support.
Four patients had steroids and a diuretic as adjunctive
therapy; one had diuretics but no steroids.

**Final outcome.** The dislocation was completely reduced
in 12 patients, partially reduced in two and unreduced in
two. Bone injury, however, was found to be stable, and
eventually healed by bone union in all 16 cases.

All 15 patients who were admitted to the NSIC had
a comprehensive rehabilitation programme. Neurological
progress was assessed by Frankel’s classification
(Frankel et al 1969). Of the seven patients in the whole
series who were completely paralysed, five made a very
substantial recovery, one made a partial recovery, and
one remained completely paralysed. The last, not admitted
to the NSIC, was completely paralysed after
operation, remained so and eventually died after three
months from respiratory complications. All six patients
who had only motor paralysis made a very substantial
recovery. The remaining three patients with partial
paralysis all recovered completely. Three of the four
patients whose dislocations could not be reduced com-
pletely made a substantial recovery, and the fourth made
a partial recovery (Fig. 1).

**DISCUSSION**

The NSIC is a secondary referral centre for patients with
spinal-cord injuries and consequently we rarely see cases
of traumatic dislocation of the cervical spine without
neurological involvement. We cannot, therefore, assess
the incidence of injury to the cord during reduction but
recognise it as one of the most devastating complications
experienced by orthopaedic surgeons or neurosurgeons.
The 16 patients that we report represent about 1% of the
cervical spinal injuries referred to us from other hospitals
between 1944 and 1991, but the true incidence of this
complication is probably much lower.

The usual mechanism of cervical dislocation has
been described by Bauze and Ardran (1978). Force
applied along the axis of the semi-flexed neck ruptures
the posterior ligaments, and causes the posterior facet or
facets to slide upwards and dislocate. The forward shift of the cranial vertebra completes the rupture of the capsule of the apophyseal joint and the annulus and disc are inevitably disrupted. Primary neurological damage is caused when the cranial vertebra sweeps forward and injures the cord. If dislocation is relatively slow and gentle, particularly when it is associated with rotation, a unilateral facet dislocation may not damage the cord. More rarely a bilateral facet dislocation occurs without neurological damage, when the spinal canal is capacious relative to the size of the spinal cord. Secondary damage may occur as swelling develops as a result of oedema or evolving vascular injury. All the patients that we report had minimal or no neurological damage before an attempt was made to reduce the dislocation. We found no relation to age, sex, mechanism of injury, type of accident, and type and level of dislocation.

The timing of the onset of paralysis in relation to the treatment varied (Table III, Fig. 1). In one group of five patients, paralysis was immediate, during an attempt to reduce the dislocation. In a second group, of 11 patients, there was no immediate neurological damage but paralysis appeared later.

In the first group damage was probably due to mechanical factors causing direct injury to the spinal cord, rather than intrinsic cord swelling. The anatomical and pathological factors that must be considered are the stability of the fracture, allowing cord injury by reproducing the acute injury, any reduction of canal diameter, disc protrusion, spondylitic changes, vascular problems, and the influence of traction and mechanical manipulation on the spinal cord.

The diameter of the spinal canal is critical, since it is recognised that a congenitally narrow canal puts the cord at risk even when there is no complete dislocation (Payne and Spillane 1957; McMillan and Silver 1987). We measured the canal diameter in all cases. In five patients, the original unilateral facet dislocation had occurred with no spinal-cord or nerve-root involvement. There had been only minimal forward displacement and the diameter of the cervical canal was not grossly reduced. All eight injuries with bilateral facet dislocation were below the third cervical vertebra, and the mean sagittal diameter of the canal was 18.6 mm, larger than the normal mean value of 17.6 mm (Pegington 1985). It seemed that these capacious canals had initially allowed more room for the cervical cord, which is about 10 to 13 mm in diameter at this level. In such cases, anaesthesia will relax the spasm of muscles which may be protecting the cord by preventing movement of an unstable injury. When reduction is attempted, the canal diameter is reduced (Breig 1989).

Other mechanisms may be important. Disc disruption and prolapse can occur in association with dislocation of the cervical spine (Eismont et al 1991; Robertson and Ryan 1992), and the extruded disc material may be displaced further into the spinal canal during reduction. This occurred in one of our patients and was demonstrated by MRI. We are aware of another patient not in our series who developed cord damage after a posterior cervical spinal fusion. Disc herniation pressing on the cord was demonstrated by myelography and CT-myelography, and there was neurological improvement after anterior disectomy and fusion with cord decompression.

In another patient not in our series, disc protrusion was seen on MRI at the site of an unreduced bilateral facet dislocation, but there was no spinal-cord involvement. Anterior disectomy was performed, and followed by open reduction of the dislocated facets, with interspinous wiring and posterior fusion.

In our series, collected since 1944, six patients had myelograms, only one of which showed disc prolapse, and two had MR scans, of which one showed disc prolapse. The other eight patients had neither myelogram nor MRI. The association of disc lesions with dislocations was recognised by Bohlman (1979), and MRI, which now enables us to visualise the canal and the spinal cord without the use of a myelogram, has shown that this occurs in up to 50% of cases (Eismont et al 1991). It is difficult to visualise how a bifacetal dislocation can occur without there being significant disc disruption.

We found spondylitic changes in the cervical spine in five patients aged 61 to 73 years. In all of these, the canal was narrow and showed posterior osteophytes, which can damage the cord by causing kinking in the narrow canal.

During reduction, injury to the vertebral arteries resulting in ischaemic damage to the brain is possible. This is surprisingly rare, since the vertebral arteries pass through the transverse processes and must be compromised by a bilateral facet dislocation (Pratt-Thomas and Berger 1947; Louw et al 1990). This complication was seen in only one of our patients, who developed cerebellar infarction in addition to spinal-cord damage. The rarity of this is probably due to the rich anastomosis between the basilar artery and the anterior cerebral circulation: in young patients the cerebral circulation can function adequately after total occlusion of one vertebral artery.

Traction, particularly if it is prolonged, excessive or applied in the wrong direction, may cause damage by stretching the cord. The commonly used method of closed reduction which involves flexion before lifting the facets clear will initially reduce the diameter of the spinal canal, potentially increasing cord compression (Breig 1989).

Our second group of patients, 11 in number, had later onset of paralysis after an interval of from 6 to 48 hours. Where canal integrity had been restored and the reduction was stable, the late deterioration was probably due to intrinsic cord factors. One recognised factor is cord oedema, which may develop early. This ill-understood swelling of the cord can now be demonstrated non-invasively by MRI. Before this became available, cord oedema could only be shown by myelography, which was believed to be contraindicated because of the
possibility of causing cord damage, especially in the presence of blood. Four of the nine patients on whom specialised radiological investigations were performed showed cord swelling (MRI 1; CT-myelogram 3). This swelling of the cord may have resulted from the initial accident or from the process of reduction.

Experimental work in animals shows that cord oedema starts after a few minutes to four hours, and can last for two weeks after the injury (Braakman and Penning 1971; Ducker, Kindt and Kempe 1971; Ducker et al 1984). In our series, an attempt had been made to reduce the dislocation within this critical period (four hours to two weeks) in 13 patients; five of these developed cord damage immediately and eight some hours later (see Fig. 1). The swollen and oedematous cord may be particularly vulnerable during this period. Any direct trauma to the cord at operation, or indirect trauma by traction, may aggravate the oedema, and the swollen cord may then be compressed within a reduced bony canal, by fragments of vertebra, disc protrusion or damage to the ligamentum flavum, leading to a progression to paralysis (Mahale and Silver 1992).

An operation to reduce dislocated facets must cause further damage to ligaments and soft tissues, increasing any instability. It may also compromise the local blood supply to the cord, which enters along the nerve roots. Thus repeated unsuccessful attempts at reduction, particularly if they are late, will aggravate cord swelling. In some patients fracture dislocation is unstable, and this problem is not easily recognised. In one patient a skull caliper disengaged, which led to displacement and further damage to the cord.

MRI, CT, myelography and CT-myelography are currently available to show cord compression. MRI and CT-myelography are the most appropriate investigations, and are important, especially when neurological function is intact below the level of dislocation. The evidence from our series and other reports (Eismont et al 1991; Robertson and Ryan 1992) suggests that these investigations should be carried out before any reduction is attempted. This would help to prevent neurological complications with their attendant morbidity. We conclude that such patients should be admitted immediately after cervical injury to specialised centres where these investigations are available, and where urgent and appropriate treatment can be provided.

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REFERENCES


Payne EE, Spillane JD. The cervical spine: an anatomicopathological study of 70 specimens (using a special technique) with particular reference to the problem of cervical spondylosis. Brain 1957; 80:571-96.


Pratt-Thomas HR, Berger KE. Cerebellar and spinal injuries after chiropractic manipulation. JAMA 1947; 133:600-3.