THE MECHANISM OF INJURY TO THE SPINAL CORD IN THE NECK WITHOUT DAMAGE TO THE VERTEBRAL COLUMN

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The discrepancy between damage to the spinal cord and to the vertebral column in cervical injuries has been the subject of comment by many authors. Both cord damage without vertebral column injury, and vertebral fracture and dislocation without cord dysfunction, are recognised. This communication concerns the former group.

The commonly observed anatomical displacement in skeletal injuries of the cervical spine is fracture-dislocation in hyperflexion, and it is caused by hyperflexion. Not unnaturally it was supposed that spinal cord damage without persistent vertebral displacement was caused during acute hyperflexion, involving temporary dislocation, immediately reduced spontaneously by muscular action. This hypothetical series of events was designated "recoil injury of the cervical spine." The improbability of this explanation was demonstrated by Cramer and McGowan (1944). They suggested temporary intraspinal displacement of an intervertebral disc as the explanation, and postulated acute forcible flexion of the neck as the cause of the supposed temporary disc displacement. As evidence, they relied upon the occasional findings of a prolapsed cervical disc as a cause of cord compression after injury, but they did not adduce proof that a temporary prolapse can occur, nor that it could account for spinal cord contusion. Barnes (1948) showed that hyperflexion can cause cord compression only if it is sufficient to dislocate and lock the articular processes. Locking of this type is not susceptible of spontaneous reduction.

As was pointed out by Taylor and Blackwood (1948), injuries causing cervical cord damage in the absence of vertebral fracture or dislocation are usually sustained by forcible extension of the neck rather than by hyperflexion. Such accidents are exemplified by diving with the head back into shallow water, by falling forwards downstairs or by being thrown forwards while the head is arrested by a fixed object, as in motoring accidents. Taylor and Blackwood described a case in which rupture of the anterior longitudinal ligament and temporary dislocation in hyperextension occurred, leaving no trace detectable by radiography. They suggested that this might be a relatively common occurrence and might explain many of the cases in question. Subsequent observations, however, indicate that a simpler mechanism is probably the common cause of damage to the cord. The findings suggest that the injury is caused by hyperextension of the cervical spine without temporary dislocation, and that the agent which impinges on the cord is the forward-bulging ligamentum flavum.

CASE REPORT

A man aged sixty-seven years gave a clear history of injury by forcible hyperextension. He was kicking a golf ball along a roadway, with both hands in his pockets, when he fell forward on to his face. He was unable to free his hands quickly enough to break his fall. He sustained a large bruise on his forehead. He was immediately paralysed in both arms and legs, retaining only shoulder movements, and had anaesthesia below the segmental level of C.6-7. Radiographs of the spine showed no abnormality except senile degenerative changes. After a spirited resistance, he died from urinary infection nine weeks after the injury.

Necropsy—The cervical spine was removed en bloc, after laminectomy and removal of the cord. Radiographs of the specimen showed no abnormality (Figs. 1 and 2). There was no rupture of
Microscopically, to the gliosis. Figure also demonstrates that for which was made hyperextension in the experiments in outline the bulging the radiographs and the spinal cord in the area of the lesion. Fragmentation of the nerve fibres extends from the posterior aspect of the cord almost to the central canal (× 9).

the anterior longitudinal ligament, and careful dissection showed all discs, joints and ligaments to be undamaged.

The spinal cord showed, at vertebral level C.4–5, a small area of softening in its dorsal half. Microscopically, at the site of this softening, there was gross destruction of the posterior columns, with interruption and fragmentation of the nerve fibres (Fig. 3). At its edges there was well marked gliosis. The damage extended into the anterior columns, where compound granular corpuscles were also seen, but with a lesser degree of gliosis and no gross interruption of the nerve fibres.

We felt justified in drawing two conclusions from these findings: first, that it was possible for the spinal cord to be damaged by hyperextension of an intact cervical spine; and second, that the cord had been struck from behind.

ANATOMICAL INVESTIGATIONS

To determine the impinging agent radiographic studies were undertaken on cadavers in which radio-opaque oil had been injected into the cervical spinal canal. Radiographs were made with the spine in various positions. The smooth outline of the opaque fluid column was undisturbed in flexion (Fig. 4) and in the neutral position (Fig. 5). But on forced hyperextension a series of indentations appeared on the posterior surface of the column, opposite the interlaminar spaces (Fig. 6). These appeared to be caused by inward bulging of the compressed interlaminar ligaments. It can be seen from the radiographs that the thecal outline is narrowed by as much as 30 per cent of its total width. These findings were constant in experiments on five cadavers (see also Fig. 7). The experiment was repeated on a young adult patient who was undergoing contrast investigations for a lumbar lesion. Figures 8 and 9 show his cervical spine in moderate and in full hyperextension. In full hyperextension radiographs showed indentations opposite the laminar spaces like those seen in the experiments on cadavers. It was confirmed, on a cervical spine removed intact, from which the cord and dura had been stripped out, that hyperextension did in fact cause an inward bulging of the interlaminar ligaments in the manner suggested by the radiographs.

Figure 4 demonstrates that when the head of a cadaver was maximally flexed so that the chin and the sternum were apposed no indentation appeared on the anterior surface of
the oil column opposite the discs. However, the significance of disc degeneration and adjacent osteophyte formation in senile spines can be seen in Figure 7. The fixed protrusions which they cause further reduce the margin of safety. Lying anteriorly, they act as counter points to the pressure of the forward-bulging ligamenta flava. This accords with the clinical observations of Barnes (1948) on the high incidence of these injuries in the elderly.
DISCUSSION

It seems, after all, that the projection which bruises the cord in these cases is not the maligned intervertebral disc—which has already to bear more than its share of blame for many otherwise unexplained symptoms—but the ligamentum flavum. It is of interest to recall that the ligamentum flavum was blamed in earlier days for much of the sciatic and brachial pain now rightly attributed to lesions of the intervertebral disc. Now the ligament takes its place again as an injurious agent but in a new role.

The observations of farmers and sportsmen suggest that hyperextension is the force that most readily produces spinal cord injury in animals. Those with experience of livestock do not attempt to break the neck of a hen, a rabbit, or a fish by flexion.
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It is stated in Cunningham's Text-book of Anatomy (1937) that "the ligamenta flava are the only markedly elastic ligaments in Man. By their elasticity they can accommodate themselves to separation of the laminae in forward flexion of the vertebral column, without falling, on extension of the column, into folds which might press upon the dura mater or be caught between the laminae they connect." It is true that they do not fall into folds, but by compression-bulging they can press on the theca and its contents. The greatest approximation of the posterior arches during hyperextension is at the point of greatest curvature, namely, C.4-5 (Fig. 6). The contrast studies showed greatest bulging of ligamenta flava between C.3 and C.6. The segmental level at which cord damage occurs in this type of case is usually C.5, 6 or 7. Neural damage at a vertebral level of C. 4, 5, 6 accords with our hypothesis. It will be seen that this lesion conforms to the dictum that traumatic vertebral and cord lesions occur at the sites of greatest spinal mobility.

TREATMENT

From the evidence here adduced we would venture to draw the conclusion that cases of cervical paraplegia without radiographic evidence of bone damage or displacement should be regarded as injuries caused by forcible hyperextension and treated as such by immobilisation in slight flexion. Immobilisation is necessary because there is no means of determining whether the anterior longitudinal ligament is intact or not. Traction has no value and should not be employed.

The conception of cord injury by skeletal displacement in hyperflexion with spontaneous recoil to normal relationships is untenable and should no longer influence practical management. We would urge especially that to nurse such patients in hyperextension is to cause cord ischaemia. Under normal conditions the cord has a smaller margin of safety at the cervical enlargement than anywhere else. The cord swells after contusion, sometimes sufficiently to cause obstruction to the flow of cerebrospinal fluid as judged by Queckenstedt's test. If the primary injury is dislocation by extension, hyperextension causes redislocation. If injury is from projection of ligamenta flava, hyperextension re-forms the projection and compresses the cord.

SUMMARY

A case of cervical traumatic paraplegia is described in which there was no evidence of damage to vertebrae, discs or ligaments. Experimental evidence suggests that such injuries may be caused by inward bulging of the ligamentum flavum during hyperextension. The reasons why this inward bulging may occur, despite the elasticity of the ligamentum flavum, are discussed. Treatment of such cases is considered and the importance of avoiding extension emphasised.

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REFERENCES