PARAPLEGIA IN HYPEREXTENSION CERVICAL INJURIES WITH NORMAL RADIOGRAPHIC APPEARANCES

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This communication deals solely with a group of cases, hitherto rather obscure, in which damage to the cervical part of the spinal cord occurs without radiographic evidence of vertebral injury or displacement. Two alternative explanations of such injury have been postulated. The first is that the spinal cord pressure is due to acute massive prolapse of an intervertebral disc: but our limited experience of this lesion suggests that as a rule this is recognisable on radiographic examination by narrowing of the intervertebral space. The second explanation is that paraplegia is due to hyperflexion dislocation which undergoes spontaneous reduction; but it is doubtful whether this occurs at all, because forward flexion or dislocation of the cervical spine which is sufficient to damage the cord would necessarily be associated with locking of the articular facets, or crush fracture of a vertebra, or both. Neither of these lesions undergoes spontaneous reduction, and moreover both are demonstrable by radiographic examination. We suggest that the usual mechanism of these injuries, in which radiographic examination reveals no significant displacement, is forced hyperextension.

Case Report—A miner aged thirty-three years was admitted to hospital twelve hours after injury sustained by falling downstairs on his face. There was immediate and complete paraplegia to the level of the seventh cervical segment of the cord. Radiographs showed an oblique crack in the body of the second thoracic vertebra, without displacement, which obviously was not related directly to the cord damage, and a crack in the left upper articular process of the seventh cervical vertebra, also without displacement. There was no narrowing of disc spaces. Lumbar puncture showed that the cerebrospinal fluid was clear and that there was free conduction of pressure throughout the theca. Pantopaque was introduced in order to determine whether or not there was compression at the site of the cord lesion. With the patient in both prone and supine positions the oil passed freely to the foramen magnum, and it was thus clear that, whatever may have caused the original cord damage, it was no longer exerting pressure. On the third day the patient showed signs of ascending cord oedema with hyperthermia, tachypnoea, and failure of the peripheral circulation. In this state he died. Autopsy revealed that the anterior longitudinal ligament was ruptured between the sixth and seventh cervical vertebrae; the column had been torn through by detachment of the intervertebral disc from the lower surface of the sixth vertebral body. The upper segment of the column, carrying with it the intact posterior longitudinal ligament, could be displaced backwards on the lower segment with great ease, the disc remaining attached to the upper surface of the seventh vertebra and the posterior longitudinal ligament being lifted from its posterior surface (Fig. 1). At this point there was a sharply localised contusion of the cord, the principal haemorrhage being situated posteriorly, exactly opposite the sharp upper margin of the seventh cervical lamina. The displacement could be replaced easily and it tended to remain in the replaced position (Fig. 2). There is little doubt that this occurred during life, thus accounting for the normal radiographic appearances. It accounts also for the peculiarly localised cord injury which represents a sharp nip, without stretching, and is in contrast with the more diffuse contusions which are encountered in flexion injuries and in which the cord may be both stretched and compressed.

In this case the nature of the injury admits of no doubt. The mechanism is substantiated equally well in the case of a sixty-year old man who fell from a haystack and had signs of a complete transverse lesion of the cord at the fifth cervical level. Radiographs were negative, and there was no subarachnoid block. There was, however, persistent increase in the protein content of the cerebrospinal fluid obtained by lumbar puncture, and after much thought it was decided to explore by laminectomy in order to determine whether or not there was any compressing factor. Exploration was negative, and in particular
there was no intervertebral disc protrusion. Lateral radiographs of this case taken six months after injury showed a calcified shadow at the anterior margin of the fourth-fifth cervical interspace, which was interpreted as heterotopic calcification in the haematoma associated with rupture of the anterior longitudinal ligament.

In many other cases with cord damage and no vertebral displacement it has been suspected that the injuries may have been of this type, but in these instances no evidence has been available from autopsy or operation.

**Discussion**—It is suggested that hyperextension is not only one of the mechanisms of injury in the type of case under discussion, but that it is the usual mechanism. This has not been recognised hitherto, no doubt because two related assumptions have passed into general acceptance and have acquired for orthopaedic surgeons an almost doctrinal significance. The first is that the anterior longitudinal ligament is proof against almost any force, and that against it the extension force used in the reduction of crush fractures of the spine may be applied with safety. Thus Davis (1943) in pointing out that the anterior longitudinal ligament is incompletely elastic and has a rupture strain of 337 lb., writes: "By and large one may say that the anterior longitudinal ligament is secure against horizontal severance either of fracturing force or hyper-extension reduction." Most methods of reduction, and most first-aid measures in the handling of fractured spines, are based on this belief. (Watson-Jones 1938, Taylor 1929, Platt 1938, Brookes 1937.) The second assumption is that if the cervical spine is subjected to sufficient hyperextension strain the brunt of injury will be borne by the atlas and axis vertebrae which may fracture through one or both arches or at the base of the odontoid process. Both these assumptions are partly true, but no more than partly true. The anterior longitudinal ligament is certainly strong, but it does rupture under sufficient strain, and a backward thrust applied through the head does cause dorsal

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**Fig. 1**

*Sagittal section of cervical and upper thoracic spine in hyperextension. The anterior longitudinal ligament is severed and the sixth cervical vertebra sheared from the disc below. The posterior longitudinal ligament is intact but separated from the back of the seventh cervical vertebra, thus allowing backward dislocation with narrowing of the spinal canal.

Inset shows localised contusion of posterior part of spinal cord caused by momentary nipping between the posterior-inferior border of the body of C 6, and the lamina of C 7.*
dislocation or fracture at the lower levels of the cervical spine. Wilson and Cochrane (1929) report, with autopsy photographs but without radiographs, a case of extension dislocation of the spine at the sixth cervical level with rupture of the anterior longitudinal ligament. W. J. Mixter (1941) envisaged such a case when he wrote, "Posterior dislocation is rare on account of the strong supporting structures, and if it does occur, I believe that spontaneous reduction might easily take place."

**Diagnosis**—The differential diagnosis in cases of paraplegia without radiographic evidence of significant displacement calls for reconsideration. We believe that forward dislocation due to flexion injury with spontaneous reduction is highly improbable. Damaging criticism of this conception has been advanced by Cramer and McGowan (1944). In our view the diagnosis lies between extension dislocation with immediate spontaneous reduction, and acute prolapse of an intervertebral disc. These two conditions can be differentiated with relative certainty. The history may indicate clearly whether the injury took place in flexion or extension. Severe facial injury of the type here presented suggests injury in extension. Older patients with fixed kyphotic deformity are still more liable to extension injury. Spinal manometry should be done because the results furnish a useful guide to treatment, particularly in cases of acute disc prolapse, but it does not help greatly in differentiation of the conditions under review. Disc protrusion may or may not cause spinal block, and although hyper-extension dislocation does not result in continued narrowing of the spinal canal at the point of injury, yet spinal block may develop later by reason of oedema of the cord due to contusion. Although we have stressed the normal appearances of radiographs, X-ray examination may help by disclosing minor changes. Narrowing of a disc space, or the suspicion of wedging of a vertebra, create a strong presumption that the compressing agent is a ruptured disc. The tearing of a small flake of bone from the anterior margin of a vertebral body suggests rupture of the anterior longitudinal ligament and therefore extension injury. If doubt still exists myelography should dispel it, because a prolapsed disc of sufficient size to damage the cord should be visualised without difficulty.

Mention should be made of another possibility which was suggested by Cramer and McGowan (1944), namely that "recoil" cervical cord injuries are caused by acute "piston-like" retropulsion of the intervertebral disc in hyperflexion, with immediate complete

FIG. 2

In the position of flexion the dislocation is completely reduced and the anatomical contours are restored. The spinal canal is no longer narrowed. There is no block on spinal manometry, and radiographs taken in this position reveal no abnormality.

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reduction. Burns Plewes (1938) made a similar suggestion on the basis of one case studied at autopsy, but unfortunately the anterior longitudinal ligament was not examined, and it is to be noted that the patient had a wound on the forehead, thus suggesting hyperextension injury. Unless more definite evidence is forthcoming this hypothesis should be treated with some reserve. It seems most unlikely that the inelastic annulus and posterior longitudinal ligament should stretch sufficiently to allow posterior displacement of nuclear material and contusion of the cord without actual rupture.

**Treatment**—The practical value of the recognition of hyperextension injuries lies in treatment. The extended position in which, by well established usage, all cases of traumatic cervical paraplegia are treated is in fact the position most calculated to inflict further damage to the injured cord. If the anterior longitudinal ligament is ruptured the extended position tends to reproduce the conditions of injury, to redislocate the spine, and to narrow the spinal canal at a point where the cord normally bears the greatest ratio of size in relation to the spinal canal. We suggest, therefore, that if the diagnosis is made, or even strongly suspected, the patient should be nursed in a shell, or between sandbags, in the neutral position or even in a position of slight flexion.

The tone of this paper is perhaps dogmatic. It is based on indisputable facts, but they relate only to two cases and there is not yet ground for certainty. Nevertheless the facts have been emphasised deliberately in the hope that attention will be focused on the possibility that so-called recoil injuries may be dislocations in extension. “Recoil injury” or spontaneous reduction is clearly a reality, and it did in fact take place in our case of posterior dislocation by extension, but we remain sceptical of the occurrence of recoil or spontaneously reducing hyperflexion injuries.

**Summary.** 1) A case is reported of paraplegia with normal radiographic appearances in which cervical cord damage was shown at autopsy to have been due to hyperextension injury.

2) The mechanism of such injuries is discussed, together with the differential diagnosis from acute prolapse of an intervertebral disc.

3) The grave dangers of using the fully extended position of the cervical spine in the management of these cases is noted.

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**REFERENCES**


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