HYPEREXTENSION INJURY OF THE NECK WITH PARAPLEGIA

C. R. Berkin and Cedric Hirson, London, England

From the West Herts Hospital, Hemel Hempstead

An example of paraplegia due to hyperextension of the neck is described in which neurological and post-mortem examination demonstrated the nature and mechanism of the injury.

CASE REPORT

A man of sixty-three years, suffering from influenza, fainted and struck his forehead on a wall. When he regained consciousness about a minute later, he could not move his limbs and had paraesthesiae and numbness of the extremities. He was known to have had pain and stiffness of his neck for four years. Examination the day after injury showed abrasions of the forehead (Fig. 1). The heart and lungs were normal and the blood pressure was 135/85 millimetres Hg. The cranial nerves and optic discs were normal and there was no sign of meningeal irritation. Motor system—Voluntary power below the neck was limited to weak movements in the following groups: elevators of the shoulders, adductors of the arms, flexors of the left thumb and all groups in the lower limbs. The arm jerks were all present and the upper limbs were flaccid. The lower limbs were spastic and the jerks were lost. The abdominal reflexes were absent, the plantar responses were doubtfully extensor, and there was retention of urine with faecal incontinence. Diaphragmatic movement was normal. Sensory system—There was reduction of sensation to pinprick and cotton wool, and impaired temperature sensation from the upper chest downwards. Position sense was absent from...
Antero-posterior radiographs of spine with lipiodol in theca. Figure 3—Spine flexed. Figure 4—Spine hyperextended.

Lateral radiographs of spine with lipiodol in theca. Figure 5—Spine flexed. Figure 6—Spine hyperextended.
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all limbs and vibration was poorly felt at the wrists and left leg, and not felt in the right leg. Radiographs of the neck showed marked hypertrophic lipping and no fracture. Radiographs of the skull and chest were normal.

Treatment and progress—A skull caliper was applied and moderate flexion was maintained by light traction. In the following three days the only sign of neurological improvement was the recovery of some movement of all muscle groups in the upper limbs. On the fourth day broncho-pneumonia supervened and the patient died the next day.

Necropsy—Post-mortem examination showed extensive broncho-pneumonia. There was no other significant lesion outside the cervical spine, where there was a complete transverse tear of the anterior longitudinal ligament between the third and fourth cervical vertebrae (Fig. 2).

Detailed study of the cervical spine—The cervical spine was removed, lipiodol was injected into the theca, and radiographs were taken with the neck fully flexed and extended (Figs. 3 to 6). In the extended position the column of opaque fluid is seen to be severed at the level of the rupture of the anterior longitudinal ligament. The lateral view shows the filling defect to be posterior. Lesser posterior indentations are seen at all other segmental levels.

The spine was divided in the median sagittal plane (Figs. 7 and 8) and the cord was replaced by a plasticine model. Figure 9 shows the indentations produced by hyperextension.

Mechanism of the cord injury—By relating the radiographs and the sagittal section of the spine in hyperextension (Figs. 6 and 8), it can be seen that the agent deforming the cord was the lamina of the fourth cervical vertebra with its overlying ligamentum flavum. This has moved upwards and forwards relative to the body of the third cervical vertebra, reducing the antero-posterior diameter of the canal by half. Smaller movements of the laminae above and below the lesion are seen to have caused the lesser indentations.
Nature of the cord injury—The neurological signs in the nervous system were those of a nearly symmetrical, predominantly posterior cord lesion at about the level of C.4–5. The loss of cutaneous sensation and motor function below the lesion was incomplete, but the loss of posterior column sensation was nearly complete, which suggested that the main force of the damage had been exerted from behind. A section of the cord (Fig. 10) at the level of the lesion supports these findings. No vascular lesion was seen, and sections made above and below the lesion showed no significant abnormality.

DISCUSSION

Paraplegia from hyperextension injury of the neck has been reported a number of times in recent years. Stress has been laid on the absence of radiological evidence of injury, and Barnes (1948, 1951) considered that pre-existing rigidity in an osteoarthritic neck was an important clinical feature. The normal spine has enough flexibility to disperse amongst its several segments the angulation occurring during hyperextension, whereas such dispersion does not take place in a rigid spine, which snaps like a dry stick, concentrating angulation at one level. This is evidenced by the occurrence of paraplegia after spinal injury in patients with ankylosing spondylitis.

Of the mechanisms that have been put forward to explain the cord injury, Jefferson (1948) has dismissed as “an absurd notion” the proposal that there is spinal contusion without actual mechanical contact. Barnes (1948) considered that nerve traction might explain a lesion higher than the level of the ligamentous damage to the neck but he was unable to support this hypothesis experimentally. Taylor and Blackwood (1948) observed that the anterior longitudinal ligament might be torn without radiographic evidence of bony injury. Taylor (1951) suggested that the posterior injury to the cord was caused by forward bulging of the ligamenta flava at the moment of hyperextension. He showed experimentally that such bulging occurs in normal and pathological subjects and that the level of injury in his case corresponded to the level of greatest intrusion of the ligaments into the canal.

Our observations confirm that the ligamenta flava bulge forwards during hyperextension sufficiently to deform a lipiodol column within the theca or to dent a plasticine model. However, we found no evidence of cord damage at these sites. We believe that the injury
to the cord in this case was produced by compression of the cord between the sharp upper edge of the lamina of C.4 and the flat posterior surface of the body of C.3. A similar mechanism appears to have operated in the first case of Taylor and Blackwood (op. cit.).

**SUMMARY**

1. The clinical and post-mortem findings are described of a patient who sustained a hyperextension injury of the neck with paraplegia.
2. There was no radiological evidence of fracture: osteoarthritis of the spine was present.
3. Evidence is presented that the cord was damaged from behind by the lamina of the vertebra below a tear in the anterior longitudinal ligament.

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**REFERENCES**