CERVICAL SPINAL-CORD INJURY*

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A n air of hopelessness surrounds the patient with cervical spinal-cord injury. Properly, the physician holds out little hope for the family as the patient is transferred to a neurosurgical unit, particularly if the lesion apparently has been complete since the accident. It generally is not recognized, however, that patients with incomplete lesions, perhaps with only a trace of movement, show an astonishing potential for recovery particularly when persisting compression can be demonstrated. This brief review of the problem includes our experience with 45 patients with acute, severe cervical spinal-cord injury admitted to the neurosurgical service over the 8-year period from January 1952 to December 1959.

The cervical spine, a site of maximum mobility, commonly is injured by indirect violence through movements of hyperflexion or hyperextension such as those that occur following motor-vehicle accidents, diving into shallow water and falling down stairs. About one-quarter of all vertebral fracture-dislocations are accompanied by severe injury of the spinal cord, but it is well known that serious cord injury may occur without roentgen-ray evidence of bony damage. This occurred in 11 of the 45 cases from our unit. Usually sharp flexion results in compression of one or more vertebral bodies, often with dislocation of the upper forward on the lower vertebra with locking of the facets. The cord then is firmly pinched between the lower vertebral body and the neural arch of the vertebra above. Bursting compression fractures without dislocation can squirt fragments of bone and discal material into the spinal canal causing acute compression of the cord. The supposition that recoil from acute hyperflexion may reduce a momentary dislocation or acute retropulsion of a disc in patients showing no bone injury has not met with favor. Barnes has demonstrated that hyperflexion of normal spines can cause compression only if it is sufficient to dislocate and lock the articular facets. It remained for Taylor to show that cervical spinal-cord injury without fracture or dislocation usually resulted from forcible extension rather than flexion. His experiments on cadavers showed very graphically how the forward bulging of the ligamenta flava with extension of the neck narrow the cervical canal; to a profound degree if a bony ridge of cervical spondylosis lies immediately in front. A bruise on the face or forehead may indicate the mechanism of hyperextension in injury of the older quadriplegic who has fallen down a flight of stairs.

Recently there has been renewed interest in the pathogenesis of spinal-cord injury by these physical mechanisms. The degree of loss of function of the spinal cord was presumed to be related to the degree of contusion by compression. Infarction by acute or chronic injury of the spinal artery was not considered seriously until the search to explain the myelopathy associated with spondylosis. However, Sir Geoffrey Jefferson was inquisitive in 1939 because he found that the cord injury was not in the form of a linear band a few mm. in width, but more often 3 or 5 cm. in length, which explained the common puzzling relationship between the segmental levels found by roentgen-ray and clinical examinations. He attributed the length of the lesion to concomitant vascular injury. Then, in 1954, Schneider et al.10 published a series of cases demonstrating

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what they called the syndrome of "acute central cervical spinal cord injury." This was characterized by disproportionately more motor impairment of the upper than the lower extremities, dysfunction of the bladder, and varying degrees of sensory loss, chiefly senses of pain and temperature and tending to spare senses of light touch and position. He considered the prognosis more favorable with central-cord injury depending on the relative degrees of central edema, contusion and hemorrhage. Motor power returned to the lower extremities first, function of the bladder returning next and finally movement in the upper extremities. Motion of the fingers and wrist was the very last to return. Sensory recovery did not pursue an orderly pattern. At the time Schneider and associates felt that the central-cord lesion seen in these cases was a contusion from momentary compression by the bony ridge in front and the ligamentum flavum posteriorly. Subsequently\(^\text{11,12}\) they considered that the central softening might be the result of a relative insufficiency in circulation of the anterior spinal artery. Their thesis of vascular insufficiency as a cause of traumatic myelopathy is based upon evidence that the vertebral artery may be compressed by cervical subluxation at the atlanto-occipital and atlantoaxial junctions as well as below, particularly where unco-vertebral lipping narrows the canal of the vertebral artery. The secondary relative insufficiency in the anterior spinal and anterior sulcal arteries produces the central softening. As the leg area of the corticospinal tracts, the posterior columns and the lateral spinothalamic tracts lie more peripherally and are nourished by the arteriae coronae (branches of the posterior spinal arteries), these patients show little or no long-tract sensory or motor changes but primarily paresis of the hands and arms. When long-tract damage is present, they believed the injury is a contusion from "squeezing" of the cord.

The same mechanism of vascular insufficiency had been postulated by Symonds\(^\text{13}\) and by Mair and Druckman\(^\text{8}\) to explain the myelopathy of cervical spondylosis. Here, of course, the lower extremities are involved severely in the spastic paresis while the upper extremities tend to escape. These authors claimed that the narrowed anterior spinal artery creates a gradient of hypoxia which is greatest at the functional zone between the areas supplied by the posterior and anterior spinal arteries, i.e., the leg segments of the corticospinal tracts.

It is difficult not to accept wholly this evidence of known vascular anatomy fitting nearly exactly into the pathology. However, traumatic myelopathy is an immediate consequence of injury. The effects of a relative insufficiency in the anterior spinal artery are not likely to be so sudden and delayed syndromes should occur, perhaps in only minutes. Thrombosis of the anterior spinal artery appears to be unusual following trauma\(^\text{6}\) and should be followed, on occasion at least, by Brown-Séquard syndromes, for the sulcal branches of this artery irrigate only one-half of a segment in alternating fashion.\(^\text{4}\)

The effects of a sudden, compressive force on the spinal medulla can not be discounted entirely as an explanation of central-cord injury.\(^\text{7}\) The spinal marrow consists of longitudinal bundles of heavy myelinated nerve fibers arranged around a central soft matrix of grey matter. A sudden squeezing force might well rupture the more fragile core before injuring the ensheathing fiber tracts completely. This would explain the immediate onset, and the varying clinical involvement of the long tracts would then be a factor in the degree of contusion.

Rostral extension of the clinical level for a segment or more after a few hours is not uncommon and may prove fatal with C4–C5 injuries. This has been presumably the result of "ascending edema." The greater caudal extent of many lesions over several segments is explained more readily by vascular insufficiency. The swollen cord within the tight pial envelope could compromise the blood supply of these lower segments which normally are in a zone of poor collateral circulation.\(^\text{17}\) Schneider’s plea for more inquiry into the clinical and pathological nature of these lesions is noteworthy, for our understanding is far from complete.