THE SYNDROME OF ACUTE ANTERIOR SPINAL CORD INJURY*

RICHARD C. SCHNEIDER, M.D.

Department of Surgery, Section of Neurosurgery, University of Michigan Medical School,
Ann Arbor, Michigan

(Received for publication October 11, 1954)

Immediately following trauma to the spine a syndrome of acute anterior spinal cord injury may sometimes be observed. This is characterized by an immediate complete paralysis with hypesthesia and hypealgesia to the level of the lesion together with the preservation of touch, motion, position and some of vibration sense. This syndrome may be the result of acute anterior spinal cord compression by a dislocated bone fragment or herniated disc or actual destruction of the anterior portion of the cord. Since a surgical lesion causing spinal cord compression is indistinguishable from a nonsurgical destructive one, the author feels that all of these patients should have an exploratory laminectomy and many of them should have a spinal fusion secondarily 2 or 3 weeks later. Emphasis should be placed upon the fact that these patients have attained their maximal neurological deficit and they do not present the usual criteria for surgery, namely, progression of neurological signs or evidence of blockage of the cerebrospinal fluid on the jugular vein compression test.

Thirteen cases are presented in detail to illustrate the pathological conditions producing this neurological pattern and the problems involved in their treatment.

In 1947 Kahn published a report which first described the role of the dentate ligaments in chronic anterior spinal cord compression by a posteriorly protruded cervical nucleus pulposus. Spasticity, hyperreflexia, disturbance of gait, weakness, and subjective sensory disturbances suggested the diagnosis of lateral sclerosis. Previously it had been thought that these symptoms were on the basis of compression of the anterior spinal artery, but this paper suggested that the spinal cord usually was firmly fixed over a posteriorly displaced disc by strong dentate ligaments, and that there was exertion of stress just above the dentate ligaments, resulting in pyramidal tract involvement. Kahn stated:

"In anterior spinal cord compression I believe that, with pressure over a period of time, the pyramidal tracts, because of the greater stress on them and the large size of their fibers, have more disturbance of conductivity than the pain fibers of the spinothalamic tracts, even though the latter are closer to the compressing mass, be it midline herniated nucleus pulposus or tumor. Touch is preserved because, even

* A part of the material in this paper was presented in the Symposium on Trauma at the Clinical Congress of the American College of Surgeons, New York City, September 25, 1952.
though the touch fibers of the ventral spinothalamic tracts may fail to conduct, there is still sufficient sensation carried in the more protected posterior columns to prevent the clinical detection of touch disturbance. Postural sense is preserved because the attachment of the dentate ligaments prevents the posterior columns, which are farthest from the compressing mass, from being pressed against the unyielding laminae (Fig. 1).

**Fig. 1. Case 1.** "Showing lines of stress in anterior spinal cord compression. Greatest stress is anterior on tracts disturbance of which would not be demonstrable by clinical tests. Secondary stress is directly on pyramidal tracts. The leg area is most lateral in pyramidal tracts, while the hand area is most medial, explaining usual sparing of the hands." (Courtesy of Dr. Edgar A. Kahn)

Bucy et al.\(^1\) regarded this mechanism as a possible explanation of the gradual development of neurological symptoms in these chronic cases.

In 1951 the author reported the first 2 cases\(^3\) of what was regarded as an acute phase of Kahn's chronic cervical cord compression mechanism. This topic will be further developed in this paper. The photographs and a brief summary of the original 2 cases are reprinted here to aid in portraying the entire problem.

**CASES**

**Case 1.** J.S., a 36-year-old male, was pinned under his truck in an accident on Aug. 21, 1948. At St. Luke's Hospital, Cleveland, he was found to have had immediate complete paralysis with a level of hypesthesia and hypalgesia to the C7 dermatome with preservation of touch. Cervical roentgenograms showed no fracture-dislocation; a lumbar puncture with jugular vein compression test showed no block. After no change in neurological status for 11 days a preoperative diagnosis of pos-