Thoracic Intervertebral Disc Protrusion with Spinal Cord Compression*

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SINCE the original report of Middleton and Teacher in 1911,10 the sinister sequelae of central thoracic disc protrusion, operated or unoperated, have been increasingly appreciated.1,4,7,9–11,13,16,19,21,22 Their patient lifted a heavy steel plate, rapidly became paraplegic, and died. Necropsy disclosed a massive disc protrusion opposite the twelfth thoracic interspace. Of Muller’s22 four cases, after operation three were left with an almost complete transection of the cord, and the fourth, after a slight, temporary improvement, with a total paraplegia. Of Hawk’s6 three patients who survived operation, all with severe preoperative spinal cord damage, one was made worse and the others showed no benefit from surgery. Two of the four cases reported by Mixter and Barr21 became paraplegic postoperatively.

Of the 17 cases of Love and Kiefer,17 marked improvement in the sensory and motor impairment occurred in only one, and complete recovery in none. Usually the patients with compression of the cord prior to operation had residual disability indicative of irremediable damage to the cord.

Of Logue’s16 11 cases, the three severely disabled prior to operation developed a total transection postoperatively. In the first the prolapse was excised, but in the other two surgical intervention was confined to a decompression. Two patients showing degenerative changes in the cord at operation, and one with a preoperative paraplegia, were rendered worse by removal of the protrusion and then made slow incomplete recoveries over several years, so they were able to get around with the aid of canes. The remaining six cases were improved.

Arseni and Nash2 reported 12 cases, eight of which showed paraparesis, and in none did this disappear. One improved after 6 years, one after 4, one after 3, and one was made worse. Interestingly, complete paraplegia occurred postoperatively in three of the paraparetic patients. In two of these recovery was incomplete, but no indication of improvement was observed in the third.

More recently, Fisher2 described his experience with four cases, in which ventral erosion of the dura occurred in one and dorsal erosion in another. He found that necrosis of the cord meant a neurological deficit regardless of whether conservative or radical measures were used. Moreover, the simple unroofing of the lesion in one instance caused paraplegia such that later removal of the disc was of no value.

In 1965, Love and Schorn19 collected 61 cases, including the 17 reported in 1950 by Love and Kiefer.17 Although they believed the wisdom of operating on protruded thoracic intervertebral discs associated with marked neurologic deficit could be questioned, they emphasized that often the nature of the lesion is unknown until it is surgically exposed.

In visiting several neurosurgeons with considerable experience, Love18 in 1944 was surprised to learn that many had never encountered a protruded thoracic intervertebral disc. That the condition is uncommon, even though increasingly becoming more recognized, is apparent from an incidence of 0.4% reported by Logue16 and by Arseni and Nash.2 An additional indication of their infrequent occurrence is evident in 95 reported cases collected by Arseni and Nash2 in 1960 and by the fact that single case reports make their appearance in the medical literature.

Because of the many diagnostic and surgical problems involved, as well as their infre-

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Cord Compression from Thoracic Disc  

Case Reports

Case 1. A 50-year-old woman slipped off a porch during the winter of 1928. Afterward she was troubled with backache, accentuated by activity. Some 11 years later she noticed weakness of the right leg which increased during the next few months. She was operated on by one of us (H.A.B.) on December 29, 1939. In view of the gradually progressive history and the myelographic findings consistent with those of a neoplasm, a spinal cord tumor seemed the likely diagnosis. Laminectomy of the 8th, 9th, and 10th dorsal vertebrae revealed, following the dural opening, a large bulging mass behind the ventral dura. The major portion of the disc material was removed extradurally. This relieved the hyper-sensitivity of the right leg but not its weakness. A few months after the operation she fell and experienced a temporary setback. By April, 1941, she had shown improvement in both legs but noted some residual spasticity and sensory impairment of the right leg. During May, 1950, she sustained a spiral fracture of the right femur when she slipped on a throw rug. Some 9 months later she was walking with the aid of a cane. In March, 1955, she fell again and noticed a temporary increase in the weakness of the right leg. She was examined from time to time between April, 1955, and August, 1961; her condition remained relatively stationary. The weakness in the right foot was helped by a spring brace. The vibratory sense was diminished in the right leg and the Babinski sign persisted.

Case 2. A 41-year-old married woman was seen in consultation at the Santa Barbara Cottage Hospital on September 2, 1961. During March, 1961, she had noticed numbness of the feet and clumsiness in walking, which she described as “tripping over things.” On several occasions she actually fell but did not hurt herself. Soon after this she noticed hypersensitivity below the umbilicus, a girdle sensation associated with difficulty in voiding and urgency, diminished pain and temperature perception in the legs, and bilateral inguinal pain. Her knees became weak and tended to buckle; she staggered as though intoxicated.

During the previous month she had had an extensive examination at the Sansum Medical Clinic which had included electromyographic studies, spinal fluid examinations, x-ray studies of the skull and the entire spine, as well as an electroencephalogram. All of these tests were normal. During the examinations, however, she had demonstrated intermittent Babinski responses with reduction in vibratory and position sense as well as a sensory level at the umbilicus. The myelographic findings on September 1 included that of a constant filling defect some 2 cm in length at the upper border of T-8. Immediately above, the spinal cord appeared widened.

Examination. When seen on September 2, verification was made of a sensory level just above the umbilicus, no vibratory sensation in the legs, and bilateral Babinski responses.

Operation. On September 7, laminectomy of T-6 through T-9 (D.L.R.) disclosed a firm area 2 cm long corresponding to the myelographic abnormality. Opening of the dura revealed upward displacement of the spinal cord. Careful retraction of the cord exposed a midline herniating intervertebral disc, which had not only compressed the cord but also comprised its blood supply. To relieve pressure on the cord, the dentate ligaments were sectioned on each side. When the capsule was incised, the disc contents were extruded as abnormally soft tissue much like toothpaste. Additional material was removed by curets and suction during careful retraction of the spinal cord. The microscopic diagnosis was that of intervertebral disc tissue and chondromalacia of the nucleus pulposus.

Postoperative course. Postoperatively, the patient was paraplegic. When she was transferred to the Orthopedic Hospital of Los Angeles for rehabilitation on October 10, 1961, sensation and leg movement had improved. A lumbar puncture October 13 showed no spinal fluid block, and the total protein was 21 mg\%o. Radiographs of the midthoracic area demonstrated no evidence of erosion or changes in the vertebral bodies or spaces. By October 31, she was able to move both thighs forward and backward, extend the legs at the knees, plantar flex the feet, and wiggle the toes. There was still some spasticity with sustained patellar and ankle clonus, and some loss of sensation to pain, light touch, vibration, and positioning in both legs.

When seen on March 27, 1962, she was able