COMPRESSION OF THE CERVICAL SPINAL CORD BY HERNIATED INTERVERTEBRAL DISCS

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THE difficulties in differentiating some instances of degenerative disease of the spinal cord from compression of the spinal cord could be thoroughly illustrated from almost any experienced neurologist's practice. Nevertheless, errors in making such a differentiation continue to be relatively common. Because of the frequency with which obstruction of the spinal subarachnoid space, as demonstrated by Queckenstedt's maneuver, and an elevation in the protein content and a xanthochromia of the spinal fluid are observed with intraspinal neoplasm, it has become commonplace to expect these important diagnostic findings with all lesions that compress the spinal cord. Unfortunately, they are by no means always present. Although numerous examples of spinal cord tumor could be cited in which one, two or even all of these alterations were lacking, it is even more common to find these criteria lacking with non-neoplastic compression of the spinal cord. In fact, it has been our experience that with median herniations of cervical intervertebral discs which impinge upon the spinal cord these findings, upon which such great dependence is often placed in making a differential diagnosis, are often lacking. This fact plus the facts that there are often few or no symptoms pointing to the neck or even the upper extremities, that simple X-ray examinations of the cervical spine are often negative, and that the neurological signs and symptoms are either confined to, or predominate in, the motor system makes differentiation of such herniations from multiple sclerosis, primary lateral sclerosis, and amyotrophic lateral sclerosis often most difficult.

This is not a new observation. Median herniations of the cervical intervertebral discs with compression of the spinal cord have been described by many observers since Stookey,22 in 1928, so clearly delineated this problem (Elsberg;6,7 Adson;1 Peet and Echols;16 Mixter and Barr;16 Mixter and Ayer;14 Hawk;9 Love and Camp;12 Craig and Shelden;5 Stone, Arieff, Kaplan and Brown;21 Péron, Lereboullet, Guillaume and Dumas;17 McKenzie and Botterell;13 Haynes;10 Portugal;18 Epstein and Davidoff;8 Broager;2 Kahn;11 Bradford and Spurling;2 and others). Many of these observers have pointed out the similarity between the symptoms that arise as the result of a median herniation of a cervical intervertebral disc and those commonly associated with degenerative disease of the spinal cord. Nevertheless, it is still not commonly appreciated that spasticity and weakness in the lower extremities, often with considerable disturbance of equilibrium, developing without
obvious cause in a young adult, with no or trivial sensory abnormalities, with or without discomfort in the neck, and with little or no sensory or motor alterations in the hands, and frequently with no, or minimal, changes in the spinal fluid, may be the result of compression of the spinal cord in the cervical region. Furthermore, it is of the greatest importance that such cases be recognized early and promptly treated if serious, irreparable damage to the spinal cord is to be avoided.

It is for the purpose of emphasizing these points, and of illustrating the diagnostic criteria of median herniations of the cervical intervertebral discs that we are reporting 4 recent cases of this type.

Case 1. H. G., truck-driver, 39 years old. Weakness in knees and unsteady gait, 5 weeks; numbness of 3rd, 4th, and 5th fingers, 4 weeks; unable to work, 3 weeks. Constipation. Examination—Mild hypalgesia 3rd, 4th and 5th fingers and ulnar borders of hands; spasticity in legs; unsteady gait; hyperactive tendon reflexes; Hoffmann’s sign; 80 mg. per cent protein in spinal fluid; obstruction of spinal canal to pantopaque. Operation—Removal of herniated intervertebral disc from between C5 and C6. Recovery.

H. G., a 39-year-old truck-driver, referred by Dr. Richard Bubolz of Chicago, was admitted to The Chicago Memorial Hospital on Mar. 25, 1947. For several years he had noted a slight aching pain at the base of his neck posteriorly after driving his truck a hundred miles or so. This is not an uncommon complaint among truck-drivers. Apart from this minor complaint he had been well until 5 weeks before admission, when he noticed a weakness in his knees on awakening in the morning. He was forced to walk with his feet spread widely apart and to take short steps in order to maintain his balance and even then he staggered. He did not fall. These symptoms grew steadily worse. About a week after the onset there developed a numbness of the 3rd, 4th, and 5th fingers of each hand and across the lower part of his back and abdomen. Three weeks before admission he was forced to cease driving his truck. For 2 weeks prior to admission he had been constipated but there was never any disturbance of urination.

His past medical and family histories seemed unrelated to his present complaints.

Examination. He was a very well developed, well nourished man. The general physical examination was entirely negative. There was no stiffness or tenderness of his neck. There was no evidence of any disturbance of the cranial nerves. The only sensory disturbance was a mild hypalgesia of the ulnar aspect of both hands and of the 4th and 5th and the ulnar half of the 3rd fingers. There was no muscular weakness anywhere. There was a slight spasticity in both lower extremities. Coordination on the heel-to-knee test was slightly impaired on both sides. On Romberg’s test he swayed toward the right. He walked with his feet spread widely apart. He could not hop on either foot alone or walk tandem with one foot in front of the other. The knee jerks and ankle jerks were equally hyperactive in both lower extremities. Plantar stimulation elicited a plantar flexion of the great toe on the right side and no response on the left. Hoffmann’s sign was present bilaterally, more marked on the left. The left abdominal reflexes were more vigorous than those on the right side.

Urinalysis, the blood count and the Wassermann and Kahn tests on the blood were all negative.

A lumbar puncture was made. The initial pressure was 90 mm. of fluid. The pressure rose rapidly and freely on jugular compression and fell freely when the compression was released.

At the conclusion of this examination we were confronted with a relatively young man who was suffering from a rather rapidly developing disability of his legs consisting of slight spasticity, hyper-reflexia and ataxia and dysequilibrium so severe that he was forced to cease work. There was no evidence of any obstruction of the spinal canal. It seemed most likely that he was suffering from multiple sclerosis with involvement of the descending motor pathways and the spinocerebellar tracts in the lateral columns of the spinal cord. A few days later the