The Cause of Dissociated Motor Loss in the Upper Extremity with Cervical Spondylosis*

A Case Report

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The cause of atrophic motor loss in the upper extremity in cervical spondylosis has been difficult to interpret because of its common association with spinal cord symptoms and the relative absence of sensory loss. There has been a tendency to attribute this dissociated motor loss to cystic degeneration in the anterior horns of the cord. However, some cases with extensive upper extremity paralysis do not present any long tract spinal cord symptoms. Because of these, the causative pathology has remained in doubt. We recently have had the opportunity to follow such a case to postmortem examination, where it became clear that the dissociated motor loss was due to independent compression of the motor roots intradurally, not to cord pathology. This study clarifies some misconceptions concerning this syndrome and seemed worthy of report.

Case Report

This patient, K.G., first noticed upper extremity paralysis in 1949 at the age of 56. It began, on arising one morning, with moderate pain in his right shoulder and inability to flex or abduct his arm. He was able to flex and extend his wrist and fingers. A tingling numb sensation was noted in the thumb and index finger, but the pain subsided in a few days. The motor loss persisted, with small contractions and great atrophy of the shoulder and arm muscles (Fig. 1). Four years later, in 1953, the same sequence of events occurred in the left shoulder and arm, again on awakening one morning. The patient had not been aware of any neck symptoms and no x-rays of his cervical spine were taken; a diagnosis of progressive muscular atrophy was given. Two years later (1955), in different medical hands, x-rays of his cervical spine (Fig. 2), showed such extensive hyperostosis around C3-4, 4-5, 5-6 and 6-7 intervertebral discs that he was referred for neurological examination. Oblique x-rays (Fig. 3) showed great narrowing of C3-4, 4-5 and 5-6 intervertebral canals. There was little sensory loss and no spinal cord symptom. The neurological interpretation was that the shoulder and arm motor loss corresponded to the distribution of the 5th and 6th cervical nerve roots, and was probably caused by the cervical spondylosis.

Because of the preceding medical diagnosis of progressive muscular atrophy and the poor prospect of benefit from treatment at this late stage, he was referred to Dr. Derek Denny-Brown of Boston, Mass., who reported that the motor loss involved the supranuclear deltoid, biceps and supinator muscles bilaterally, with normal scapular fixation, triceps function and flexion and extension of the wrists and fingers. Biceps and supinator jerks were absent, triceps reflexes present, knee jerks sluggish, ankle jerks absent, and plantar reflexes clearly flexor. Sensory testing showed slight reduction in several modalities (Fig. 4), interpreted as extending from the upper cervical to the 8th cervical segment. The x-rays showed encroachment on the 3rd to 6th intervertebral foramina and reduction of the diameter of the spinal canal to 12–13 mm. opposite C3–4, 4–5, and 5–6 discs. Dr. Denny-Brown’s conclusion was that the condition represented the result of damage to a series of nerve roots or segments from C4 to C8 and that the mild segmental sensory loss and severe motor disturbance could be caused by a cyst of the gray matter of the 4th to 8th cervical segments on both sides. He felt the syndrome was not characteristic of progressive muscular atrophy or syringomyelia.

Surgery was not advised because experience has indicated that cutting of the dentate ligaments does not benefit this chronic type of multiple spondylosis. The patient continued under medical care until his death in 1964 at 61 years of age with no change in the upper extremity paralysis and no spinal cord symptoms. Death was due to complications related to vascular insufficiency of the legs.

Postmortem Examination. A cervical myelogram was done after embalming, but before removal of the spinal cord. The postero-anterior films (Fig. 5), showed sur-

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Fig. 1. Patient, K.G., bilateral atrophy and paralysis of shoulder and arm muscles.
prisingly normal outline of spinal cord and nerve root pouches at the C5 and C6 neurological level of major involvement, the oil pooling laterally and extending between nerve root pouches, thinning a little over the intervening spaces. This contrasted with the lateral x-ray (Fig. 6), which showed prominent posterior and anterior oil defects at C3-4, 4-5 and 5-6 intervertebral levels, interpreted radiologically as thickening or infolding of the ligamenta flava, transverse ridging and constriction of the spinal canal. Discussion of these contradictory findings will be given later.

The cervical spine was removed intact with its enclosed spinal cord, from C3 to T1 vertebrae inclusive, and fixed for 1 week in 5 per cent formalin solution before dissection. The spines and laminae were removed with rongeur. There was no thickening or infolding of the ligamenta flava or crowding of the dural canal. Firm elevations were palpated beneath the 4th, 5th and 6th cervical nerve roots, less beneath the 7th root. These extended into the intervertebral canals but a small probe could be passed through the cephalad portion of these canals, indicating some remaining space for nerve roots and blood vessels. Medially the hyperostoses were less prominent beneath the spinal cord. The articular processes were removed on the right side to expose the nerve roots, showing extension of the hyperostoses through the canals, displacing the vertebral artery at the C4-5 level. Measurement of these hyperostoses showed an average length of 90 mm, width 10 mm and thickness 5 mm.

The dura was opened longitudinally (Fig. 7), to expose the spinal cord and nerve roots intradurally. The spinal cord appeared normal but the observation was made that the 6th cervical nerve roots showed complete separation of sensory and motor portions near their entrance into their common dural sheath, with the motor root tightly stretched and flattened over a prominent "high spot" of the underlying hyperostosis. The 5th nerve roots were similarly involved but less separated; the right 4th root was greatly flattened over a large hyperostosis not present on the left. The other roots showed the normal anatomy of overlapping sensory and motor components. A drawing was made of this region (Fig. 8), for better delineation. Cutting and retracting the 2 dentate ligaments adjoining the left 6th nerve root (Fig. 9) did not release this root appreciably over the hyperostosis. The adjoining sensory root was freely movable and appeared uninvolved at this site. This discrete intradural involvement of the motor roots intradurally seemed to give an explanation of the dissociated motor loss in the upper extremities of this case with extensive chronic cervical spondylosis.

Further dissection consisted of cutting the nerve roots intradurally on the right side allowing retraction of the spinal cord to the left. There was no evidence of pressure grooving of the formalin-fixed cord and only slight elevation of the dura over midline disc ridges. The dural canal with its spinal cord and nerve roots was dissected free and the remaining spine proteolytically cleaned to show in better manner the paired hyperostoses around intervertebral discs (Fig. 10). The medial "high spots" of the hyperostoses are clearly seen in this oblique view which also shows the remaining space in the left intervertebral canals.