Paraplegia secondary to cord compression is well recognized in marked kyphoscoliosis. Reports have been published, as well, on the treatment of spinal cord traction syndromes which result in paresis and spasticity of the legs as the result of a tight filum terminale. However, the following case of neurological dysfunction of the cervical segments of the spinal cord secondary to traction exerted from the upper dorsal area is unique in our experience.

Case Report

An 11-year-old girl was first seen to be limping by her seventh-grade teacher in May, 1961. Four months later the child was noticeably dragging her right foot.

Examination at another hospital revealed that the child had marked kyphosis, maximal at D-5. It had been present since infancy and was regarded as congenital. The kyphosis was associated with a mild scoliotic deformity. She had always walked with her head bent forward, but had had no difficulty with her gait. A decompressive laminectomy of the upper dorsal vertebrae revealed stretching of the spinal cord across a bony ridge centering at about D-5. The neurological deficit increased postoperatively so that there was complete motor paralysis and an incomplete sensory loss below the D-9 dermatome.

On December 11, 1961, an attempt was made to decompress the spinal cord by costotransversectomy from D-4 to D-7. Postoperatively the patient’s neurological status continued to deteriorate. By the second postoperative month, numbness of both hands and apparently symmetrical ulnar nerve palsies were noted. Initially, this was thought to be due to pressure from the frame on the region of the median epicondyles. The patient was put in a half-shell cast; 2 weeks later only symmetrical shoulder motion remained. The tone and deep tendon reflexes in the arms had markedly increased and there was now a general decrease in sensation below the fifth cervical dermatome. Ten days later it was necessary to place the child in a respirator because of symmetrical diaphragmatic paralysis.

The patient was transferred to the neurological service where a re-exploration of the previous dorsal laminectomy was done on March 13, 1962. Operative findings revealed a very atrophic segment of spinal cord where it was stretched tightly over the apex of the dorsal kyphosis at D-5. The spinal cord was then simply transected above this atrophic area at about the third or fourth dorsal cord segment. Upon transection, the cephalic segment of spinal cord was noted to rise upwards 1½ cm. A small segment of the distal spinal cord was taken for pathological examination and the dura was tightly closed.

The child resumed diaphragmatic breath-
John A. Maxwell and Edgar A. Kahn

FIG. 1. Laminograms showing a complex congenital anomaly of the upper dorsal spine. Left. Lateral view showing a sharply-localized dorsal kyphosis with failure of segmentation of the second, third, and fourth dorsal vertebrae. The body of D-5 is wedged and remains posteriorly. A bony process extends anterior to the spinal column at the apex of the kyphosis. Right. Anterior-posterior view showing minimal scoliosis and a complete pantopaque block at the level of the sixth dorsal vertebra.

Recovery was within 24 hours of surgery and was removed from the respirator. During the following 2 weeks, normal shoulder and elbow motion returned and the long flexors of the wrist and fingers improved markedly; function of the intrinsic musculature and long extensors of the digits was poor. Eight months postoperatively, however, both motor and sensory examinations of the upper extremities were normal.

Discussion

The relative importance of movement, stretch, repeated trauma, vascularity, and compression of the human spinal cord in the development of a neurological deficit is not yet well understood. The discrepancy which may exist between the severity of the neurological disorder and the lack of definite evidence of cord compression is often surprising, especially in cervical spondylosis. The most common explanation is that of pressure on the spinal cord from a bony protrusion into the neural canal. This contact may lead to vascular compromise as well as direct cord compression.

Our case documents a progressively-ascending neurological deficit which spread upward from a mid-dorsal point of abnormal spinal-cord fixation, and which eventually involved the motor supply to the phrenic nerves. This sequence substantiates the idea that spinal cord traction can be a factor in the production of a neurological deficit. After this abnormal “pull” on the cervical cord had been released by transection of the spinal cord in the high dorsal area, a sequential reversal of the neurological defect was noted. Diaphragmatic control returned quickly, followed by function of each cervical segment one after another. Two weeks following cord transection, all segments except the eighth cervical and first dorsal were completely normal. At later neurological examination in the outpatient clinic the upper extremities were completely normal.

Whether cord traction per se or a vascular compromise from tension was the etiology of