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.2,4,7 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 sciotic 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.

The child was then transferred to the University Hospital where a review of the earlier x-rays showed a dorsal kyphosis of 60° angulation with its apex at a wedged fifth-dorsal hemivertebra (Fig. 1). Failure of segmentation of the second, third, and fourth dorsal vertebrae was noted (block vertebrae) X-rays of the skull, cranio-spinal junction and cervical spine were normal. Examination revealed spastic paraplegia and atrophy of the legs, extensor plantar responses, and decreased sensation below the ninth dorsal segment. Urinary and fecal incontinence was evident.

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 neurosurgical 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-
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.

ing 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
this progressive neurological pattern is difficult to say. However, the separation of the cord segments at operation obviously released tension in the cephalic segment. This was followed by a remarkable and prompt return of neurological function. Therefore, it is hard to deny the role of "traction" as a major factor. The arterial supply to the spinal cord was actually further compromised by the transection of the anterior spinal artery.

Precise measurements of spinal cord movement and stretch have been made in cadavers. O'Connell\(^9\) has shown that from maximum extension to maximum flexion of the neck, the spinal canal can increase in length up to 5 cm between the foramen magnum and the first dorsal vertebra. Between these same extremes of neck and body extension and flexion, the length of the spinal cord can change 7 1/2 cm, as measured between the brain stem and the conus medullaris.\(^1\)

Each segment of the spinal cord maintains its position in relation to its appropriate area of the spinal canal because two mechanisms are in effect; first, the relative movement of the cord with respect to the spinal canal, and second, the stretch of the dural sac and plastic elongation of the spinal cord. The principal migration (plastic elongation of the cord and stretch of the dura) is cephalad in the cervical area. Lesser changes transpire in the dorsal and lumbar areas.\(^1\) The dentate ligaments effectively link the spinal cord and the dura so that there is little relative movement between the cord and the dura along the axis of the spinal canal.\(^1\)\(^11\)

Between the extremes of extension and flexion, the cervical cord is estimated to lengthen from 18 to 24%\(^1\)\(^11\)\(^13\).

Normally, relative elongation of the spinal canal in flexion of the neck and trunk is compensated for by plastic elongation of the cord distributed over a considerable length of cervical and thoracic cord. If a dorsal kyphosis increases the disproportion between the lengths of the spinal canal and the spinal cord and also fixes the position of the thoracic cord, then the full effect of flexion, perhaps beyond tolerance, must be accommodated by elongation of that length of spinal cord isolated above the kyphosis. We feel that this can result in neurological deficits first in the area of greatest tension, which experimentally is nearest the apex of spinal angulation,\(^1\) and later at points progressively more remote from the point of fixation, as transmitted tension is dissipated by plastic elongation of the cord.

The tight filum terminale syndrome is also felt to be a cord traction syndrome, with maximal neurological deficit close to the point of fixation, generally in the legs.\(^2\)\(^7\) However, one case of this sort was reported in which neurological changes existed in the arms.\(^4\)

**Summary**

We have reported a case in which an ascending neurological deficit developed above a marked dorsal kyphosis. This deficit was completely reversed by transection of an obviously atrophic segment of spinal cord stretched over a bony ridge at the level of the apex of the kyphosis. We suggest that increasing traction stretched the cervical cord and produced the neurological changes.

**References**