Experimental Cord Stretchability and the Tethered Cord Syndrome

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Much remains to be learned about the embryology and pathophysiology of the "tethered cord syndrome." Of particular importance in tethered cord syndrome is spinal cord stretchability and its relation to resultant cord dysfunction. In this study, cord elongation was observed in fresh fetuses and in animals by applying weights or forceps traction at the conus medullaris–cauda equina region. The results show that maximum cord elongation occurs in the lumbar region, some occurs in the thoracic area, and minimal to no elongation occurs in the cervical region. These results are concordant with clinically observed lumbosacral cord dysfunction and oxidative and electrophysiologic impairment of the lumbosacral cord in tethered cord syndrome as shown by other investigators.

The embryology, pathophysiology, and neurologic dysfunction due to longitudinal traction of the conus medullaris, the so-called "tethered cord syndrome," are poorly understood. Little is known about stretchability of the normal and abnormal living cord and the ensuing relation between the deranged cord morphology and the altered physiology of the neural tissue. To ascertain the longitudinal elongation of various segments of the cord, we applied weights or forceps traction at the cauda equina in fresh fetuses and at the conus medullaris–cauda equina region in anesthetized animals to assess cord elongation in the living state. The results of these experiments are shown and their clinical pertinence to tethered cord syndrome briefly discussed.

Materials and Methods

We studied cord elongation in six fresh human fetuses and three living animals. The approximate gestation periods of the fetuses were 6 months (n = 4), 7 months (n = 1), and 9 months (n = 1). The animals studied were one white laboratory rat (500 g), one New Zealand white rabbit (3.2 kg), and one mongrel dog (18.2 kg).

In each fetus, the entire spinal cord covered with dura mater was exposed by laminectomy. The preparation, consisting of the head and the spinal column, was hung vertically, the head resting in a metal ring (fig. 1). A black suture was tied around the cord at approximately the midcervical, midthoracic, and T11–L1 levels. A pin was placed in the tissue outside the cord at exactly the same level as the suture and served as a reference point for measuring elongation. At this stage, the cord response to filum terminale transection was examined.

A suture loop was then formed at the cauda equina and a paper clip hung from it to accept weights (metal nuts), which were added in increments of 5 g. When cord extension reached its maximum with weights, forceps were used to create traction at the cauda equina eliciting maximal cord elongation. All the nerve roots were then severed and the spontaneous effect on the cord observed. Forceps traction was again applied at the cauda equina and cord lengthening noted. This routine was followed in the first three fetuses. After determining that the maximal cord lengthening by weights was identical to that elicited by forceps traction and that the latter provided the most useful information on cord elongation, we dispensed with the weights in favor of the forceps method in the remaining experiments. In fetus 1, the dentate ligaments were dissected, thus permitting us to observe the severing effect on the cord.

To avoid time-consuming total laminectomy and the subsequent blood loss resulting in possible death of the animals, we performed only three-level laminectomies, in the midcervical, midthoracic, and lumbar levels. Cord elongation in these animals was created by forceps traction at the conus medullaris–cauda equina region.

We used a ruler and/or a caliper and a magnifying lens to measure elongation. Measurements at the cervical and thoracic levels were difficult to obtain due to limited cord lengthening at these points. All the measurements were corrected to the next 0.25 mm.

Results

The results of these experiments establish that cord elongation is approximately proportional to the magnitude of the applied force until the point of maximum stretchability is reached. (This is the point at which no further elongation could be elicited, not at which the cord broke.) Figure 2 represents progressive cord elongation in fetus 2 at various cord levels concomitant with application of graduated weights. Similar cord elongation was noted in fetuses 1 and 3. The cord stretches maximally in the lumbar region, slightly in the thoracic region, and minimally to not at all in the cervical region (tables 1 and 2). Severing the nerve roots resulted in slight spontaneous ascent (1 mm) of the cord in all fetuses. Also, with forceps traction at the cauda equina, severed nerve roots resulted in greater cord stretchability than intact nerve roots in every fetus (fig. 3). The cord elongation clearly is greatest nearest the point of force application (figs. 4 and 5). Elasticity of the cord tissue was demonstrated by its ability to regain its original position at each level upon

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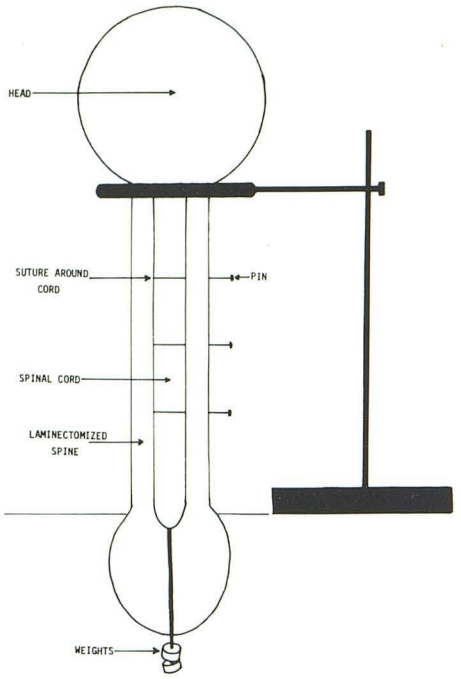

Fig. 1.—Fetal preparation in experiments.

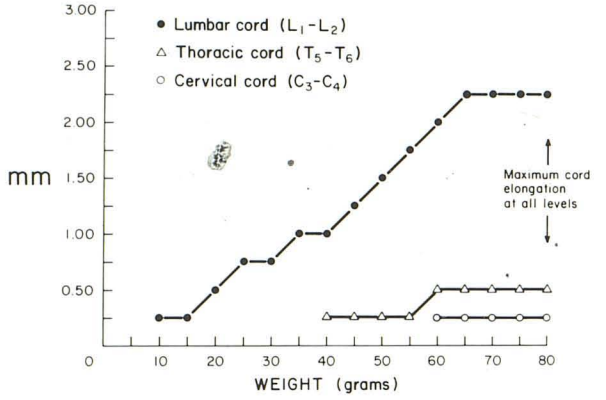

Fig. 2.—Fetus no. 2. Cord elongation with graduated application of weight.

cessation of forceps traction.

In all specimens, the filum terminale curled up at the point of transection and no spontaneous ascent of the cord was seen. As a result of cutting the dentate ligaments in fetus 1, the spinal cord descended 1 mm at all levels, thus demonstrating the anchoring effect of these ligaments on the cord.

In these experiments we have assumed that cord elongation with and without the covering dura mater is about the same. In two fetuses, the dura mater was incised at the midline over a small area at each cord level and cord lengthening measured. We found that the cord stretched to about the same degree as it did when covered with dura mater. The cord tissue in fresh fetus denuded of the entire covering dura mater is so friable that no satisfactory stretching experiments can be conducted.

Discussion

Despite their great clinical importance in tethered cord syndrome, the dynamic and biomechanical changes of the spinal cord in

TABLE 1: **Cord Elongation in Fetuses with Nerve Roots Intact and Cut on Forceps Traction at the Cauda Equina**

Fetus No.: Cord Location	Cord Elongation (mm)	
	Nerve Root Intact	Nerve Root Cut
1:		
Midcervical	0.25	0.75
Midthoracic	0.5	1.25
Lumbar	2.5	4.0
2:		
Midcervical	0.25	1.0
Midthoracic	0.5	1.0
Lumbar	2.25	3.5
3:		
Midcervical	0.25	1.0
Midthoracic	0.5	1.25
Lumbar	1.75	3.5
4:		
Midcervical	0.25	1.0
Midthoracic	0.75	1.75
Lumbar	1.75	3.5
5:		
Midcervical	0.25	0.5
Midthoracic	0.5	1.5
Lumbar	2.5	4.0
6:		
Midcervical	0.25	1.0
Midthoracic	0.5	1.5
Lumbar	3.0	4.5

Note.—Lumbar readings taken at the T11–L1 level.

TABLE 2: **Cord Elongation in Experimental Animals on Forceps Traction at Conus Medullaris–Cauda Equina Region**

Animal: Cord Location	Cord Elongation (mm)
Dog:	
Midcervical	0.25
Midthoracic	1.75
Lumbar (L1–L2)	6.5
Rabbit:	
Midcervical	None
Midthoracic	None
Upper lumbar (T12–L1)	1.0
Lower lumbar (L3–L4)	1.25
Rat:	
Midcervical	None
Midthoracic	0.5
Upper lumbar	1.25

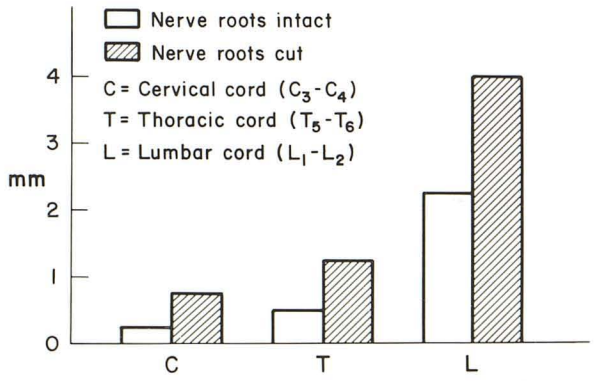

Fig. 3.—Fetus no. 1. Cord elongation with nerve roots intact and cut on applying forceps traction at cauda equina.

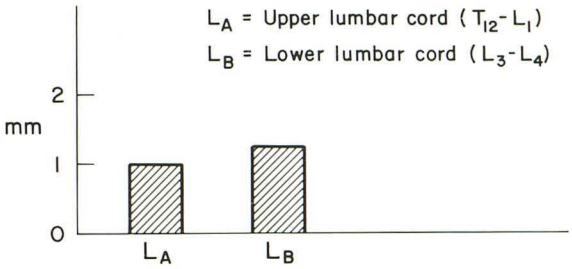

Fig. 4.—Rabbit. Cord elongation by forceps traction at L7 level. No elongation was observed at thoracic and cervical levels.

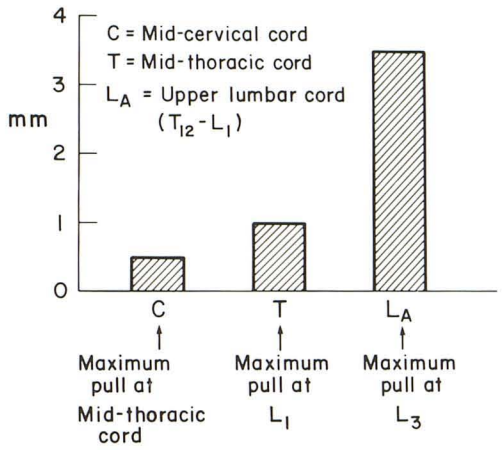

Fig. 5.—Rabbit. Maximum cord elongation occurs nearest to point of force application. No cord elongation at cervical and thoracic levels was seen in this animal when forceps traction was applied at conus–cauda equina region.

normal and pathologic circumstances have not been studied adequately. Breig [1] studied extensively the biomechanics of the central nervous system in cadavers. His studies primarily pertained to physiologic deformation of the brainstem and the spinal cord in forward, backward, and lateral spine bending with emphasis on the cervical region. Nevertheless, his work clearly established the dynamism of the cord (e.g., lengthening and shortening of the cervical cord in ventral and dorsal flexion, respectively). Barry et al. [2] showed that the thoracic cord segments were abnormally longer in one fetus in which the cephalic margin of myeloschisis was in the lower thoracic region. In another fetus, the lumbar cord segments were abnormally large and cephalic margin of the anomaly was in the lower lumbar area. These researchers stated that such segmental cord enlargement was due to caudad traction on the cord exerted by the anomaly. In these fetuses, the cord elongation was greatest near the cephalic margin of the anomaly and became progressively less marked away from it. Smith [3] used posture in monkeys to show changes in length and position of spinal cord segments. Our study attempts to fulfill the need for quantitative experimental study on cord stretchability whereby the cord is directly visualized during traction of the cauda equina.

The fact that cord elongation occurs maximally in the lumbar region and slightly at the thoracic level is consistent with the clinically observed symptoms and signs of lumbosacral cord dysfunction. These observations are concordant with pathophysiologic changes of the lumbosacral cord in tethered cord syndrome shown by Yamada et al. [4]. These investigators studied the oxidative metabolic functioning and electrophysiologic changes in tethered cord syndrome and concluded that "symptoms and signs of tethered cord syndrome are concomitant with lumbosacral neuronal dysfunction, which could be due to impairment of mitochondrial oxidative metabolism under constant or intermittent cord stretching." Such metabolic changes are very likely due to stretching with distortion of vascular structures. These authors showed that untethering improves oxidative metabolism and possibly facilitates neuronal reparative mechanisms. The corollary of these observations is that symptoms and signs of thoracocervical cord dysfunction seen in a patient with known tethered cord syndrome should not be attributed automatically to the syndrome; another cause should be sought (e.g., concomitant syringohydromyelia and Chiari malformation).

By extrapolating the results of our experiments, we can safely assume that the degree of cord elongation will be greater in the longer cords of children and adults. This is borne out by observing at surgery the separation of closely applied clips when the thickened filum terminale is transected between them. The ascent of the proximal clip can reach 1–2.5 cm [5].

The variability of clinical neurologic deficit and the surgical outcome [5, 6] probably are related to the tolerance to stretch of neural tissue in each individual; the locomotion of each patient; and the length of time the cord has been subjected to this abnormal stretching influence. Presumably, if the stretch-induced neural tissue damage has become extensive or irreversible, amelioration of neurologic deficit after untethering will be minimal. Logically, then, the sooner the untethering procedure is performed, the more likely it is that the reparation of the neurologic deficit will be satisfactory.

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