George Garceau, an orthopedic surgeon, is generally credited as the first physician to attribute neurological sequelae to a taut terminal filum, which he did in an article published in the *Journal of Bone and Joint Surgery* in 1953. Garceau’s brief paper described three cases of spastic paralysis in which patients underwent exploratory laminectomies. In each patient, the terminal filum was sectioned; all patients experienced improvement in their symptoms. The three surgeries were performed in 1949 and 1951, and Dr. Robert F. Heimburger, then Associate Professor of Surgery (Neurosurgery), at Indiana University School of Medicine, was acknowledged for his assistance at the end of Garceau’s paper.

Heimburger submitted the paper presented here, which describes cases of scoliosis that had come to clinical attention some decades earlier, for publication in the medical literature. The patients in these cases underwent sectioning of tight terminal filia and all experienced good outcomes. Heimburger’s paper regarding what is now known as the spinal cord traction syndrome, was not well received when submitted for publication more than 50 years ago; Heimburger was rebuked by various editors of that day. Furthermore, it was made clear to Heimburger that if he continued to perform and advocate the procedures described in the paper that his medical license would be in jeopardy.

Half a century after the rejection of Heimburger’s paper, our understanding of the disease entities known as terminal filum syndrome and tethered cord syndrome has increased significantly. Heimburger’s paper, which was never published, is the now-retired surgeon’s original submission. The paper is important from a historical perspective, providing us a unique window into the early cause-and-effect thinking regarding this neurosurgical lesion.

We believe the following paper to be one of the very first descriptions of the tethered cord syndrome and the first description of scoliosis as a consequence of a tight terminal filum.

### Heimburger’s Paper

A definitive cause for scoliosis can be found in only a small number of the patients who suffer from the disease. Rickets, tuberculous spondylitis, poliomyelitis with unequal paravertebral muscle paralysis, emphysema with rib resection, and sciatic pain are primary conditions that can give rise to scoliosis as a secondary complication. The hemivertebrae of congenital scoliosis present a clear
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cause for abnormal spinal curvature; even in such cases, however, no satisfactory primary cause—except that the changes occur during fetal life—can be given for the deviation in the structure of the VBs.

It is a commonly accepted fact that the curvature indicative of idiopathic scoliosis increases most rapidly during the most rapid periods of growth in the spinal column. This observation has rightfully led many investigators to suspect unequal growth of the vertebral epiphyses as the cause. Several ingenious experiments have been performed to strengthen the idea that unequal growth of the vertebral epiphyses is the most likely secondary cause for idiopathic scoliosis. Infectious epiphysitis, discussed in 1925 by Buchman, has not gained enthusiastic proponents among investigators of scoliosis because it has not been demonstrated clinically with adequate frequency. This leaves no widely accepted theory for the primary cause of idiopathic scoliosis, even though unequal growth of vertebral epiphyses is generally believed, both clinically and in the laboratory, to be its secondary cause.

When a primary cause is known the secondary causes of scoliosis tend to be categorized into two groups: first, the conditions that change bone growth patterns, such as rickets, tuberculous spondylitis, or rib resection for emphysema; and, second, changes in the bone growth patterns of the vertebrae resulting from alterations in the soft tissues near the spinal column. Examples of primary causes for this type of scoliosis are poliomyelitis with unequal paravertebral muscle spasm and scoliosis resulting from sciatica. If the soft-tissue changes occur during a period of rapid growth, the vertebral epiphyses are affected and permanent scoliosis results. On the other hand, soft-tissue changes occurring after the bone is fully developed do not result in permanent scoliosis.

The goal of this paper is to present a possible primary cause for idiopathic scoliosis: alterations of the soft tissues within the spinal canal. The discussion will be based on the observation of 11 patients suffering from scoliosis and varying degrees of neurological damage. The usual reasoning with regard to such cases is that the neurological deficit is secondary to the scoliosis. It is hoped that the findings presented in this paper will help those in the medical community consider the possibility that, at least in some instances, a neurological deficit and scoliosis may arise from the same primary cause.

Case Reports

There are 11 cases separated into three groups, and a representative case from each group is described in detail. Six of the 11 cases presented can be classified as congenital scoliosis; in these cases radiographs demonstrated one or more hemivertebrae or other vertebral anomalies. There are four cases of idiopathic scoliosis and one of scoliosis due to tuberculous spondylitis. Because these patients suffered from neurological deficits in addition to long-term scoliosis, each received neurological surgery consultation at the request of orthopedic surgeons.

Case 1

Presentation and Examination. A 14-year-old caucasian boy presented to the James Whitcomb Riley Hospital on August 26, 1944, having been referred by Dr. H. K. Stort of Huntingburg, Indiana. The boy was said to have had an abnormal curve in the upper thoracic portion of his spine since birth. This curve had become more pronounced during the 6 months prior to his admission to the hospital. Examination and radiography at that time revealed a marked kyphoscoliosis at T-3 and T-4. The convexity of the curve was to the left, and there was a so-called razor deformity of the ribs on the left. No treatment was instituted because the scoliosis was asymptomatic. No neurological abnormality was reported.

Second Presentation and First Treatment. Less than a year later weakness developed in the patient’s legs, and he was readmitted to the hospital on July 25, 1945. He was found to be suffering from spasticity of both lower extremities, with bilateral upward plantar responses. There was no sensory loss and no block of spinal fluid as a result of jugular compression. Spinal fluid protein measured 122 mg%. The spasticity of the legs was improved by bed rest and head halter traction. Because this improvement was not permanent, a T3–6 fusion was performed. The fusion was successful, and the spastic paraparesis subsided so that the boy was able to continue his school work for 4 years.

Readmission and Further Treatment. The boy was readmitted to the hospital on October 6, 1949, because of a return of the spastic paraparesis. This time the weakness was more pronounced. In addition to the spasticity, weakness of the lower extremities, and bilateral Babinski responses, there was urinary retention and a level of hypesthesia at T-10 dermatome. Examination of a lumbar puncture specimen revealed no block of spinal fluid flow as a result of jugular compression, but myelography demonstrated no passage of contrast material above T-6. The spinal fluid protein was 115 mg%.

On October 26, 1949, the boy underwent a T6–7 laminectomy. The spinal cord and dural sheath were found to be very small and tightly pressed against the T-3 and T-4 pedicles on the concave side of the curve. The major portion of the spinal canal was filled with loose fatty tissue. The dura mater was opened. The small size of the spinal cord and the narrowness of the surrounding subarachnoid space were thought to be sufficient to explain the neurological deficit. Following this procedure the boy experienced a slight improvement for 6 weeks, and then neurological deficit increased. The laminectomy was reexplored when the patient’s sensory level rose to the T-6 dermatome.

The laminectomy performed on January 5, 1950, again at T-3 through T-6, revealed no change from the previous operation. A catheter could be passed within the subarachnoid space upward to the level of C-6 without meeting obstruction. Spinal fluid was present around the cord to T-6, but none was seen below this level. Some adhesions were released, and an attempt was made to reposition the cord by taking bone from the T-4 and T-5 pedicles on the right. The fat pad in the free portion of the spinal canal was removed. The procedure provided no benefit to the patient, and the paraparesis gradually progressed to paraplegia during the 2 weeks after surgery.

Laminectomy was again performed on January 23, 1950, this time from C-6 through T-2. The cord was found to be tightly stretched across the point of maximum gibbus formation. An attempt was made to remove some of
the T-1 VB to free the cord. The T-2 and T-3 nerve roots were sectioned because they were too tightly stretched from the convex to the concave side of the spinal canal.

The tightly stretched spinal cord over the gibbus at T-1 appeared to be anchored at its lower end. Because the myelogram provided no information regarding the position of the conus medullaris, we decided to explore the lower end of the spinal cord to determine if it was stretched. We believed that this procedure was justified because the patient’s paraplegia had continued to worsen.

An L-2 laminectomy was performed, at the usual position of the conus medullaris, on February 2, 1950. The conus medullaris was not found at L-2 and there appeared to be no decrease in the size of the cord. The laminectomy was continued. The entire sacrum was bifid. At S-2 the spinal cord started to taper slightly, but it was still almost 1 cm in diameter. The nerve roots exited the spinal canal at a point almost at right angles from their origin in the cord. Because no muscular response could be elicited in the minute nerve filaments exiting the cord at S-2, this point was thought to be the junction between the conus medullaris and the terminal filum. The spinal cord was cut at the S-2 level. The two ends immediately sprang apart 1.5 to 2 cm.

Postoperative Course. Improvement started soon after this procedure. The urethral catheter, which had been necessary for 3 months prior to terminal filum surgery, was removed on the 21st postoperative day. Gait training went much more rapidly than it does in cases of complete paraplegia, and the patient was discharged from the hospital June 28, 1950, able to complete a “swinging-through gait.” Sensory return in the lower extremities had begun in spots, not conforming to entire dermatomes.

Follow-Up Examinations and General Notes. The patient was seen periodically for gait training and changes in braces. When last seen in August of 1960, he was walking with drop-foot braces and canes and had regained the use of the muscles controlling his hips and knees. Sensory return was varied below the T-10 dermatome, although some return was apparent in the second, third, and fourth toes on the right, from the convex to the concave side of the spinal canal.

spinal canal during the key growth period. In three of the five patients an abnormality of the terminal filum was revealed during surgery. The distal end of the spinal canal was not observed during surgery in these patients in whom laminectomy was performed at the L5–S1 level; therefore, it cannot be stated definitively that there was in these patients a failure of the spinal cord to migrate cephalad during the growth period. Nevertheless, it can be assumed that there was a failure of this sort at least in three cases, because the terminal filum in three patients was abnormally large and vascular. In each of these patients the terminal filum was situated posteriorly in the spinal canal, which is abnormal. The ends of the terminal filum in each of these patients separated dramatically when cut, indicating that the structure was pulled tightly.

In two of the four cases with congenitally abnormal vertebrae the terminal filum was not thought to be abnormal. In one patient (Case 5) there was a hemivertebra at L-1. The only neurological deficit in this girl was pain/hypalgesia. In the other patient (Case 6) with a normal terminal filum, Klippel–Feil syndrome was the only vertebral abnormality. This patient had undergone removal of an occipital encephalocele at 3 months of age. When she developed a progressive scoliosis and increasing weakness in her arms and legs it was thought that the central nervous system might be under traction as a result of an abnormal terminal filum. This structure proved to be normal at the L5–S1 level. An exploration of the posterior fossa confirmed the presence of an Arnold–Chiari malformation, and it was assumed that the neurological deficit stemmed from that.

It is interesting to note that the patients in each of the four cases of abnormal upper thoracic vertebrae suffered from an abnormality of the terminal filum, whereas the patients with abnormal cervical or lumbar vertebrae demonstrated normal fila.

Case 7

History. A 10-year-old caucasian girl presented at the James Whitcomb Riley Hospital on September 9, 1954, having been referred by Dr. Robert Scott of Charlottesville, Indiana. The patient’s records revealed the appearance of a club foot on her right side at the age of 1 month. Treatment for this condition was started at another hospital when the child was 1.5 years of age. The girl’s treatment began with casting and concluded with a peroneal nerve transplantation at the age of 6 years. When the child was 1.5 years of age her parents had noticed that her back was crooked. That curvature increased more rapidly between the ages of 8 and 10 years, and an ulcer developed on the girl’s right second toe several months before admission.

Examination. Examination revealed a Sprengel deformity on the left and a talipes equinus varus on the right. There was a hemangioma and a hairy patch over the thoracolumbar junction of the back. There was no mass or dimple in this area, and none over the coccyx. A rotatory scoliosis was present with the concavity to the right in the thoracic spine and to the left in the lumbar spine. The apex of the scoliosis appeared on radiographs to be located at the L-2 level. There was still some mobility of the spine upon lateral flexion. The right lower extremity was smaller in diameter and length than the left. Sensory loss was apparent in the second, third, and fourth toes on the right.
Terminal filum syndrome

and the terminal phalanx of the right second toe had ulcerated and sloughed.

Initial Treatment. The girl was treated at home in a body cast for several months and brought into the hospital for neurological evaluation on May 17, 1955. Examination at that time revealed the same conditions found 9 months previously. Laboratory investigation showed normal findings except for a spinal fluid total protein of 105 mg%. Radiographs of the spine demonstrated no changes in the scoliosis during the 9-month observation period.

On June 15, 1955, the L-S1 spinous processes and laminae were exposed. The S-1 vertebra was bifid, although this had been demonstrated on radiographs. An S-1 laminectomy and partial L-5 laminectomy were performed. The dura mater appeared normal in this area. When the dura was opened, a pinkish structure 3 to 4 mm in diameter appeared. As the spinal fluid was removed, the adjacent nerve roots fell to the bottom of the dural canal, but the pinkish structure maintained its position close to the posterior portion of the dura. This structure was identified by electrical stimulation as the terminal filum. It was not extremely taut, but it refused to fall to the bottom of the dural sac as the nerve roots had. When the patient’s lumbar spine flexed, the terminal filum became considerably more tense. It was sectioned between silver clips. The cut ends of the terminal filum immediately separated 3 to 4 cm. This separation measuring 4 cm was demonstrated on postoperative radiographs.

Postoperative Course. The child was allowed to ambulate on the 4th postoperative day. On the 5th postoperative day she admitted that her right foot, instead of being externally rotated as it had been for her entire life, was now straight when she walked. This improvement has been maintained during the 4 months since surgery. There has been no change in the area of sensory loss in the second, third, and fourth toes on the right, but the sensory loss perceived by the patient was less profound. There was no change in the scoliosis demonstrated on radiographs at the examination conducted 2 weeks after surgery.

Comparison With Other Cases. The patients in this group, classified as cases of idiopathic scoliosis, suffered less severe spinal cord damage than the patients with a kyphoscoliosis and angulation of the spinal canal. In each of these cases the gradual curvature usually seen in idiopathic scoliosis was demonstrated. The spinal curvature developed during childhood in each of the patients and was not noticed as early as in the group with abnormal vertebral and kyphoscoliosis. All but the youngest of these patients had suffered from the scoliosis long enough for irreversible changes in the VBs to develop.

It is interesting that the patients in the four cases classified as idiopathic scoliosis were female; the population of patients with scoliosis is 75% female and 25% male. In the group of patients with congenital scoliosis and more severe evidence of spinal cord damage the equal distribution of males and females was equal.

Case 11

First Presentation. A 2-year-old African-American boy presented to the James Whitcomb Riley Hospital on June 28, 1939, having been referred by Dr. Robert Pierson of Spencer, Indiana. At that time the boy was found to be suffering from advanced, healed tuberculosis in the apex of each lung, and marked upper thoracic kyphosis resulting from the collapse of a VB. He was treated for 3.5 years by bed rest and a Bradford frame. He was unable to walk at the termination of this treatment and had upward plantar responses and bilateral ankle clonus until the age of 4 years. The patient was followed until the age of 6 years; he had learned to walk and appeared to be normal except for the kyphoscoliosis.

Second Presentation. No additional follow-up was conducted until the boy presented again, at the age of 13 years, to the James Whitcomb Riley Hospital on February 21, 1951, having been referred this time by Dr. Jack Hatfield, of Indianapolis, Indiana. At the time of admission he exhibited weakness in both lower extremities, more markedly on the left. He exhibited hyperactive reflexes, ankle clonus, and upward plantar responses. Hypalgesia and hypesthesia were evident below T-3, and kyphosis at T-3 or T-4. Slight scoliosis was apparent in the thoracic area, with the convexity to the left. Spinal curvature was also evident in the lumbar area, with the convexity to the right and the apex at L-1.

First Operation. On March 7, 1951, an L-4 laminectomy was performed. The terminal filum appeared thick and vascular; it fell to the floor of the spinal canal along with the nerve roots when the spinal fluid was removed and exhibited no tension. More difficult to identify than the previously described fila, its position was not exaggerated in the posterior portion of the dural canal. The ends of the terminal filum did not separate when it was cut. No improvement resulted from this procedure and the patient’s paraparesis increased.

Second Operation. Because the patient experienced increasing dysfunction in the spinal cord a C7–T3 laminectomy was performed on March 20, 1951. The spinal cord and dura mater were found to be tightly stretched over a gibbus at the T2–3 level. The dura pulsedated above this point but not below it. By removing bone, an attempt was made to relieve the tension of the spinal cord; although no improvement resulted from this procedure, the progress of the paraparesis was stopped. After surgery the patient demonstrated voluntary control in the quadriceps and hamstring muscle groups, along with bladder and bowel control. In addition, some sensation remained below the T-3 level.

The boy led a fairly active existence in a wheelchair until the age of 16 years when he developed pain in the legs. Contrast material oil introduced into the cisterna magna failed to pass below a complete block at C-7. The earlier laminectomy was reexplored on February 10, 1954, and a small amount of bone was removed from the maximum point of the gibbus anterior to the spinal cord. The patient experienced relief of pain but no other improvement as a result of this procedure.

Comparison With Other Cases. Although the terminal filum in this patient appeared abnormally large it exhibited none of the signs that have been thought to indicate tension in the structure. The terminal filum underwent sectioning in this case because it was believed that the spinal cord had been anchored at the apex of the kyphosis during
most of the boy’s growth period. It seemed possible that the cord could be stretched between the kyphosis and the attachment of the terminal filum to the lower end of the spinal canal. Although the terminal filum was abnormally large and vascular, it could lengthen as necessitated by the increase in the length of the spinal column that would result from increasing kyphosis or normal growth.

Discussion

The evidence presented in these 11 cases is insufficient to conclude that either neurological deficit or scoliosis resulted from spinal cord tension. That an abnormal and tense terminal filum was evident in eight of the 10 patients with congenital or idiopathic scoliosis indicates that a failure of the spinal cord to migrate successfully up the spinal canal may have played a role in the syndrome. The absence of a tense terminal filum in the one patient suffering from tuberculous spondylitis and neurological damage is further evidence in support of the theory.

The criteria used to determine whether the terminal filum was normal or abnormal was derived from intraoperative observations of the cauda equina of the normal spine and from Tarlov’s explanation of the normal spine. During a rhizotomy, all elements of the cauda equina fall to the anterior portion of the dural canal when the spinal fluid is removed. The terminal filum is difficult to find among the tangle of nerve roots; in cases in which it is easily located from the surrounding nerve roots the identification is a result of difference in color, size, flexibility, and reaction to electrical stimulation. The terminal filum is similar to fascia, pale, glistening, and inelastic, in contrast to the pinkish, elastic qualities of the nerve roots.

The abnormal terminal filum associated with scoliosis differs in several respects from this description. During the surgeries performed in the cases described here, the abnormally tight terminal filum always appeared prominently, forming a tent in the dura mater even before the dura had been opened. When the dura was opened and the spinal fluid removed, the abnormal terminal filum remained posteriorly in the dural canal while the roots of the cauda equina fell to the floor of the spinal canal. The abnormal terminal filum was too tight to conform to the laws of gravity and fall to the anterior portion of the dural canal as normal filum does. When the lumbar spine was flexed during surgery, the abnormal terminal filum became tense; this increased tension was demonstrated each of the four times it was attempted.

The abnormal terminal filum was quite large in diameter compared with the normal one. Moreover, it appeared pinkish and vascular, resembling a large nerve root. In contrast, the normal terminal filum is, according to Tarlov, the slender glistening prolongation of the end of the conus medullaris.

The possibility of damage to the spinal cord resulting from a failure of the terminal filum to migrate up the spinal canal during growth has been suggested in the medical literature. Lichtenstein and others described the effect of such an abnormality on the brainstem and cerebellum. Katzenstein argued that the spinal cord can be stretched and can deteriorate because of its failure to migrate. The great increase in the length of the terminal filum during growth reported by Tarlov supports the possibility of great damage to the spinal cord if the terminal filum fails to grow as the spinal canal grows (Table 1). Even partial failure of the terminal filum to increase almost fourfold in length from the time of birth to maturity could reduce the blood supply in the spinal cord and cause dysfunction.

Additional information regarding scoliosis can be derived using a map measurer to measure the length of the spinal canal. This method of measuring was applied to radiographs of the spinal canal. In a 2-year-old healthy child the flexed spinal canal is 2 cm longer than it is when extended. This increase in length during flexion must be considerably exaggerated in the adult. From these data, one can infer that the spinal cord is normally placed under tension during periods of spinal column flexion. If the tautness of an abnormally tight terminal filum is added to the normal tension produced by spinal flexion, damage to spinal cord function would appear to be a predictable result. The intraoperative observation that a tight terminal filum becomes tighter when the lumbar spine undergoes flexion further supports this theory. Patients with upper thoracic kyphoscoliosis and spinal cord dysfunction have limited mobility of the spinal column, particularly during flexion. This limited mobility may be an attempt to decrease the damage that flexion and increased traction may cause in the spinal cord.

Further studies with the map measurer show that there is no difference in the length of the right and left side of the spinal canal in normal individuals or those suffering from scoliosis. When no rotation of the spinal canal has occurred, the right and left sides of the spinal canal measure exactly the same length; however, when there has been some rotation of the VBs, causing the spinal canal to lie on the concave side of the thoracolumbar curve, there is a marked difference in the length of the two sides of the canal. The concave side of the canal may be at least 5 cm shorter than the convex side. Rotation of the spinal canal in scoliosis is always to the concave side, which is perhaps the body’s attempt to shorten the distance traveled by the spinal cord.

Spinal cord traction has also been observed in the experimental laboratory. A laminectomy in the lower lumbar area was performed in two young Macaque rhesus monkeys (3 lb) by Dr. L. W. Freeman. The contents of the dura were disturbed for the purpose of another experiment. It was observed that these animals developed a pronounced rotatory scoliosis as they grew. When they were killed 3 years after their original surgery, a marked adherence between the dura mater and its neural contents was observed. The conus medullaris appeared to be lower in the spinal canal than normal; this phenomenon occurred only in animals in which the laminectomy was performed in the lower portion of the spinal canal. Those in which surgery was conducted in the thoracic area often developed a kyphosis at the operative site, but none developed a rotatory scoliosis. No control animals kept in the colony during the same period developed a spinal curvature.

Several trials have been undertaken to repeat this experiment, with the production of scoliosis as the primary goal. Attempts made by Dr. Charles Gregory to shorten the terminal filum of four young monkeys were not successful (personal communication, 1940). In another study, 20 newborn dogs underwent sacral laminectomy. A kaolin
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TABLE 1
Summary of the mean measurements of spinal structure reported by Tarlov

<table>
<thead>
<tr>
<th>Structure</th>
<th>Length (cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>conus medullaris</td>
<td>1.2</td>
</tr>
<tr>
<td>terminal filum</td>
<td>4.2</td>
</tr>
<tr>
<td>intradural</td>
<td>2.2</td>
</tr>
<tr>
<td>extradural</td>
<td>1.7</td>
</tr>
</tbody>
</table>

suspension, shown to cause dense arachnoid adhesions, was injected into and around the dural sac. Although the dogs grew rapidly, none developed scoliosis nor any sign of spinal curvature. When these animals were killed between 1 and 6 months of age, some arachnoid adhesions were apparent at the operative site, but the conus medullaris had progressed up the spinal canal in what was considered a normal fashion.

Because no definite method, except observation or operation, has been developed to demonstrate the level of the conus medullaris, it is not possible to state where this structure was located in the majority of the patients in the aforementioned 11 cases. It was believed that an extensive laminectomy to locate the site of the conus medullaris was justifiable. The conus medullaris was observed in the patient in Case 1 at the S-2 level and was found in its normal position at the L-2 level in the patient in Case 5, in whom a normal terminal filum was also observed.

In each of the 11 cases an abnormally taut, enlarged, and vascular terminal filum was associated with scoliosis. It is possible that the abnormality of terminal filum is primary and that as growth of the spinal cord occurs, traction is exerted on the spinal cord because of the failure of the terminal filum to lengthen. To relieve tension on the spinal cord, rotatory scoliosis may develop to shorten the spinal canal and growth of the vertebral epiphyses may occur to accommodate the scoliosis. In most instances the scoliosis is sufficient to relieve the tension on the spinal cord and no neurological deficit develops. In an occasional case the tension on the spinal cord is so great that the scoliosis is not sufficient compensation and neurological dysfunction results. If traction on the spinal cord becomes severe before the vertebral centers of ossification appear, hemivertebrae and other vertebral anomalies may result.

Most cases of idiopathic scoliosis can be explained on the basis of abnormalities in bone development in the spinal canal. Neurological deficits rarely occur in such cases. Cases involving congenital kyphoscoliosis are associated with a higher incidence of neurological deficit, and a finding that may play a role in the aforementioned hypothesis.

Conclusions

Patients suffering from scoliosis accompanied by neurological deficit demonstrated a tight terminal filum in a majority of the cases reported. The neurological deficits usually improved after the surgery to section the tight terminal filum. In some cases, scoliosis may result when the spinal cord, under the tension caused by a terminal filum that has failed to grow properly, requires a shorter spinal canal. Growth may be altered by the scoliosis and result in abnormal vertebrae and kyphosis formation. Surgery to section the terminal filum should be contemplated if the spinal cord is situated tightly against the concave side of the spinal canal.

References