THE FILUM TERMINALE SYNDROME: (The Cord-Traction Syndrome)

George J. Garceau
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THE FILUM TERMINALE SYNDROME

(THE CORD-TRACTION SYNDROME)*

BY GEORGE J. GARCEAU, M.D., INDIANAPOLIS, INDIANA

From the James Whitcomb Riley Hospital, Indiana University School of Medicine, Indianapolis

This communication is a report of our observations made during the study and treatment of three patients presenting the symptoms of a progressive spastic paralysis. It concerns especially the pathological changes found when these patients were subjected to exploratory laminectomy. A diligent search of the available literature failed to reveal a similar clinical report, although such may exist.

In 1945 we became interested in the Arnold-Chiari syndrome. This symptom complex, usually associated with deformities of the spine and spinal cord, was first described by Arnold in 1894 and by Chiari in 1895. Many articles have appeared in the neurological literature since the publication of these original papers.

In a recent publication, McKenzie and Dewar concluded that in twenty-one of twenty-four patients operated upon for scoliosis with paraplegia, the cause of the paralysis was a tight dura stretching over the angulated spine. The review of the literature on the subject of paralysis associated with scoliosis supports that opinion. The onset of the paralysis in thirty-three of forty-one cases reported was between the ages of thirteen and nineteen years. Two of our patients were thirteen years old at the onset of paralysis. The third patient was fourteen years old at the onset of the symptoms; however, although he recovered and remained well for four years, the symptoms again recurred at the age of seventeen years.

Ingraham and Lowrey reported that about 25 per cent. of children have defects of the vertebral laminae and that incomplete closure is frequently observed.

Steele reported the Arnold-Chiari malformation to be a frequent factor in cases of hydrocephalus, causing a block of the fourth ventricle and obstruction of the cerebrospinal-fluid circulation.

Lichtenstein described the distant (remote) neuro-anatomical complications of spina bifida. He reported that the cauda equina may be short, causing retention of the infantile position of the conus medullaris.

Harmeier in 1933 and Tarlov in 1938 described the normal structure of the filum terminale and concluded that it contained all the histological tissues of the spinal cord.

Reimann and Anson and later Anson alone studied an instance of a sacral cord in an anatomical laboratory specimen, the only sacral cord in 129 specimens. The spinal cord terminated at the first and second lumbar vertebral level in 95 per cent. of 129 adult specimens.

Kleinberg recently reported a case of paraplegia occurring in a patient with a congenital scoliosis. He concluded that, if the symptoms do not improve with non-operative treatment, laminectomy supplemented by the necessary additional surgery is indicated.

In the patients who form the basis of this report, progressive spastic palsy developed while they were under observation for other conditions. The first had a congenital scoliosis; the second had recovered from tuberculosis of the spine; the third had been under treatment for an idiopathic scoliosis.

CASE 1. In August 1944 a thirteen-year-old white boy was admitted to the Orthopaedic Service for observation and treatment for a rapidly progressing left thoracic kyphoscoliosis, due to extensive congenital malformations of the spine from the second to the seventh thoracic vertebrae (Fig. 1-A). He had

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FIG. 1-A
August 1944

FIG. 1-B
November 1946

FIG. 1-C
November 1946

Minimal increase in left thoracic scoliosis due to congenital anomalies of thoracic vertebrae and of the second, third, and fourth lumbar vertebrae. Lumbar and sacral laminectomy revealed a tight filum terminale and conus medullaris at the level of the second sacral vertebra. There was slow but good recovery.

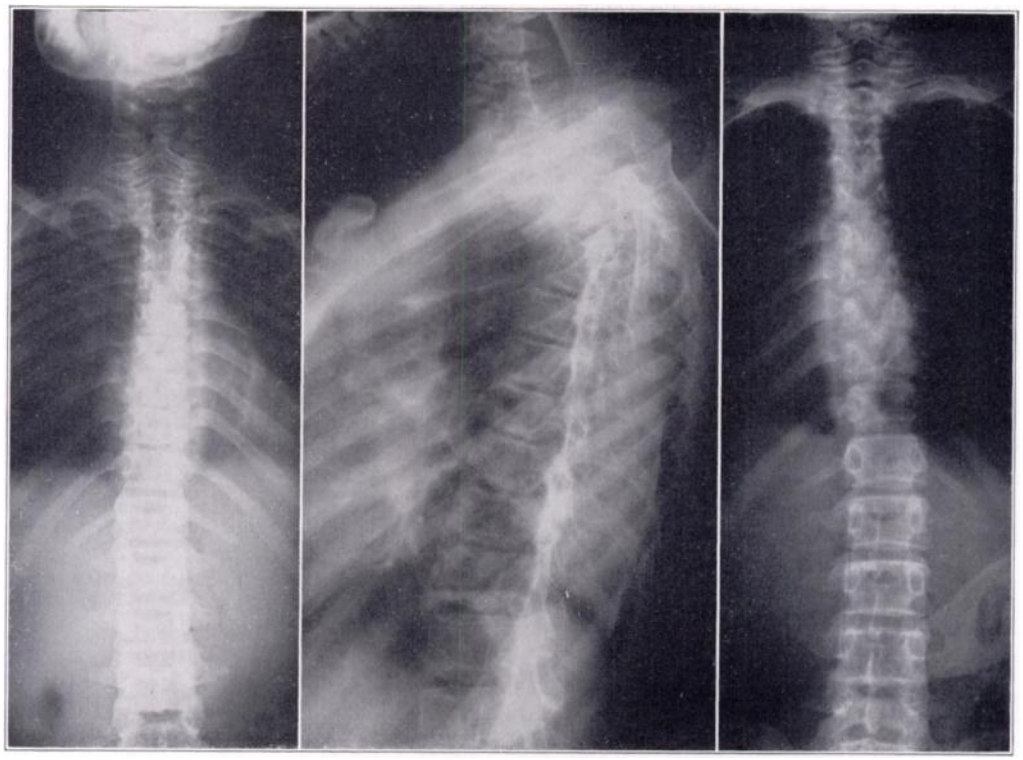

FIG. 2-A
November 1945

FIG. 2-B
September 1949

FIG. 2-C
September 1949

Case 2, H. S. Tuberculous spondylitis from the second to the fifth thoracic vertebrae inclusive. Sound fusion had occurred spontaneously. The filum was sectioned and there was partial recovery.

recently complained of pain in the back on exertion. There was a severe razor deformity of the back. The neurological examination was negative. Eleven months later he was again admitted for further study because of stiffness and clumsiness in the legs. There was spasticity of the legs, equinus of the ankles, bilateral Babinski reflexes, depressed knee jerks, and dull cremasteric reflexes; the abdominal reflexes were absent. The spinal-fluid total protein was 122 milligrams per 100 cubic centimeters and the globulin was ++. Improvement occurred after treatment on a frame with head-and-neck traction. A spine fusion was performed on December 13, 1945. Three months later he had completely recovered.

The patient was not observed again until October 1949, at the age of seventeen years, when he reported for a recurrence of the paralytic symptoms. There was paralysis of the bladder and of the bowel. The examination suggested a lesion at the tenth thoracic segment of the spinal cord. The spinal fluid was about the same as on the initial admission. Three laminectomies were performed by the neurosurgeon, the cord being uncovered from the third cervical to the sixth thoracic vertebra. No constrictions were encountered. The spine fusion was found to be sound. A fourth laminectomy from the second lumbar to the second sacral vertebrae was performed on February 2, 1950. The spinal cord was found to extend to the level of the second sacral segment and was anchored by a large filum terminale. The filum terminale was sectioned. Slow but progressive improvement followed. Bladder and bowel function returned. He now walks well without support.

Case 2. A thirteen-year-old colored boy was admitted to our Service on February 21, 1951, with the complaint of tingling, weakness, and stiffness in the right leg. He had a quiescent tuberculosis of the spine from the second to the fifth thoracic vertebra, inclusive, which had responded to three years of conservative treatment on a frame (Figs. 2-A, 2-B, and 2-C). He had become ambulatory at the age of five years. The physical examination revealed a tall, thin colored boy with a severe kyphosis in the upper thoracic spine. He stood with a wide base. There was weakness in both legs, more in the right than in the left. All the reflexes in the lower extremities were hyperactive. There were Babinski and Rossolimo reflexes bilaterally. Vibratory sense was diminished and positional sense was altered. Pain and touch sensation appeared intact. All symptoms became aggravated while he was under conservative treatment and bed rest. Because a filum terminale was suspected, a laminectomy was performed on March 7, 1951. A thick, tough, and tight filum

terminale was found. Stimulation of the filum gave no response; the filum was therefore resected. Microscopic examination of the specimen revealed a fibrous-tissue band with a few scattered nerve fibers. Because of fear of overlooking pathological changes at the thoracic kyphos, a laminectomy was performed at that level on March 20, 1951. Sound fusion had occurred spontaneously. There was severe angulation of the cord (90 degrees). No actual compression was observed, although pulsation was not definite below the gibbus. The postoperative course was marked by a transient total paralysis of the right lower extremity and increased weakness in the left. There was also hypo-aesthesia of the lower extremities. Slow improvement has occurred.

Case 3. A fifteen-year-old white girl had been admitted to the James Whitcomb Riley Hospital in April 1947, for observation and treatment of a right idiopathic thoracolumbar scoliosis (Fig. 3-A). At that time good correction was obtained in a turnbuckle jacket. Spine fusion from the third thoracic to the second lumbar vertebrae, inclusive, was performed on July 18, 1949. She was followed in the Out-Patient Department. Lengths of the lower extremities remained equal. One-fourth inch of atrophy developed in the right calf. The neurological examination remained negative. In July 1950, she complained of nausea, vomiting, dizziness, and dragging of the left leg. The only positive neurological observations were a loss of tendon reflexes in the left upper extremity, and a possible Babinski reflex on the left side. Four months later a Babinski and an unsustained ankle clonus were found on the left side. In February 1951, a slight weakness of the left grip was noted. Spinal-fluid examination was normal. Severe headaches developed, as well as a tendency to fall, and weakness and pain in the right hand. Examination revealed the fundi to be normal and no signs of cranial involvement were found. There was weakness in the right hand and in the left lower extremity. The left leg was hyperalgesic. Position sense was intact. Co-ordination was normal. Deep reflexes were absent in the arms. Abdominal reflexes were absent. There was a reversed radial reflex (that is, when the distal end of the radius was tapped, the elbow did not flex and the fingers had a tendency to extend) as well as a Hoffmann sign on the right. The left knee jerk was hyperactive. The ankle jerks were equal. There was unsustained patellar and ankle clonus on the left side. Babinski, Chaddock, and Rossolimo reflexes were present on the left side. Our impression at that time was that a high cervical cord compression, due to cord traction, existed. A spinal-puncture attempt was discontinued for fear of injury to a sacral cord if such existed.

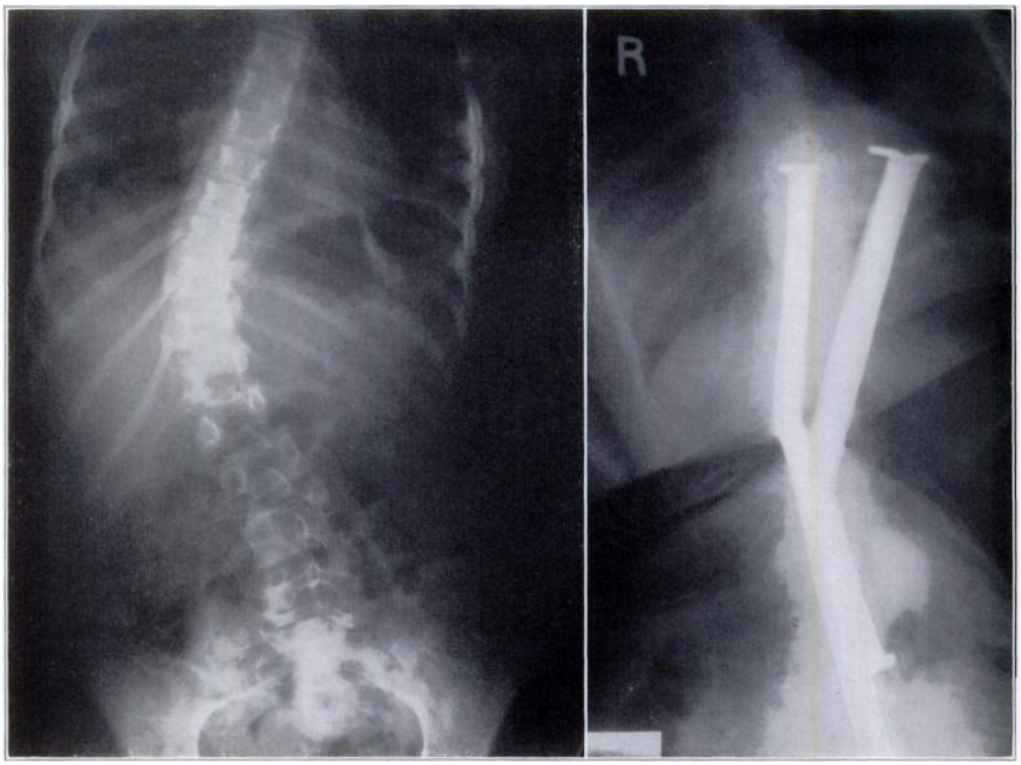

Fig. 3-A

April 1949

Case 3. A. H. Idiopathic right thoracolumbar scoliosis of 50 degrees.

On May 16, 1951, a laminectomy of the fifth lumbar and first sacral vertebrae was performed. A mild spina bifida was found. The ligamentum flavum appeared to constrict the dural sac. Two small dural bands stretching across the dura at the fifth lumbar level were severed. The arachnoid was normal. The filum was five millimeters in diameter and very tense. When the filum was severed, the ends separated one centimeter. The postoperative course was uneventful and almost complete recovery has occurred (Fig. 3-C).

The paresis in this patient was not caused by a tightness of the dura over the angulated spine. There were neurological symptoms in the upper extremities which could not have been caused by a thoracolumbar scoliosis.

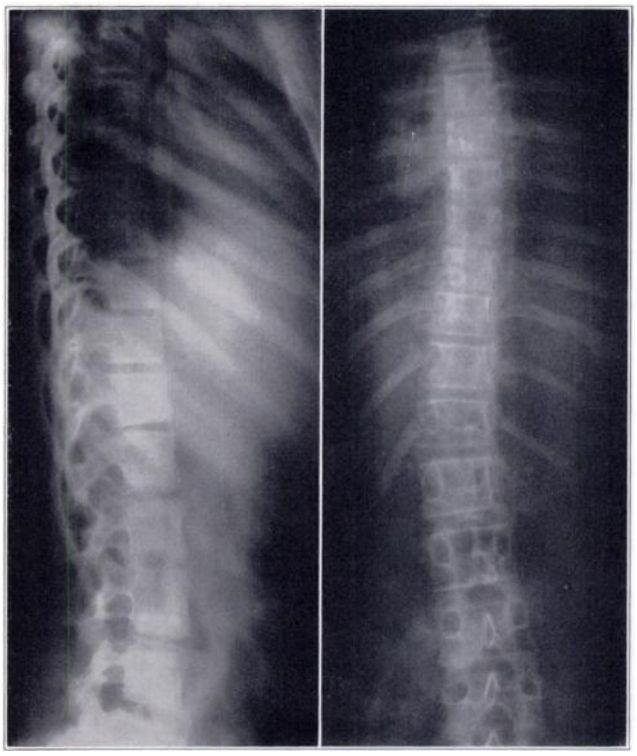

FIG. 3-B

July 1950

Correction maintained. Onset of symptoms of nausea, vomiting, and ataxia. Filum terminale was sectioned in May 1951, followed by good recovery.

DISCUSSION

The cord-traction symptoms associated with dysraphism have been recognized by neurosurgeons for many years. The neurosurgical literature abounds with excellent articles. The syndrome has been associated with simple spina bifida occulta, meningocele, myelomeningocele, diastematomyelia, congenital scoliosis, Klippel-Feil syndrome, cranium bifidum, tight fibrous bands, and bony spicules. All of these conditions may be associated with congenital deformities, especially in the lower extremities. These are usually evident at birth. The neurological abnormalities may be progressive.

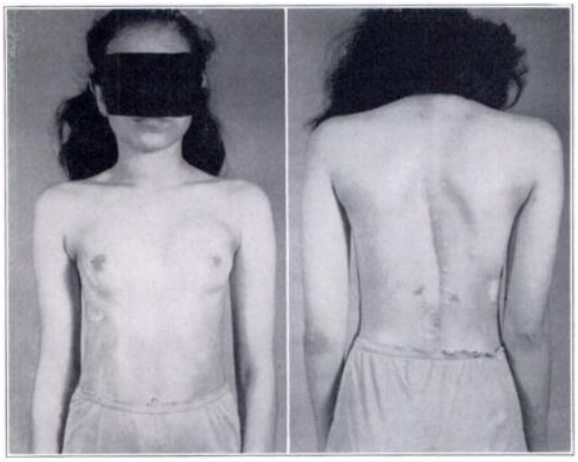

FIG. 3-C

January 15, 1952

Shows satisfactory maintenance of correction of scoliosis.

The associated pathological lesions were different in the three cases here reported. In the first patient the tight filum terminale had prevented the distal migration of the spinal column, so that the conus medullaris had retained the foetal position. Paralysis occurred during the period of rapid growth.

In the patient with tuberculosis of the spine the signs and symptoms of paraplegia developed during the period of rapid growth. Recovery followed sectioning of the filum terminale. Exploration at the area of kyphosis failed to reveal the cause of the paralysis.

In the patient with idiopathic thoracolumbar scoliosis, the filum separated one centi-

meter on sectioning, indicating the tightness of the structure. The symptoms in the upper extremities could not have been caused by stretching of the filum over the angulated spine. Almost complete recovery followed sectioning of the filum.

Unfortunately we do not know the level of the conus medullaris in two of the cases. In the patient with the idiopathic scoliosis we could assume that it was below the usual level of the first or second lumbar vertebra, because it separated one centimeter when cut.

Our treatment of the patients could be questioned. Decompression of the foramen magnum could have given the same result. We are following these patients and may later find that foramenotomy will be necessary.

The tight filum terminale may cause spinal-cord compression over an angulated spine. It may also cause cord traction, pulling the hind brain into the foramen magnum.

The question of the role of the tight filum terminale as a cause of idiopathic scoliosis is suggested but remains unanswered. We are now attempting to produce scoliosis in monkeys by preventing the migration of the spinal column away from the conus medullaris.

CONCLUSIONS

A tight filum terminale may cause the cord-traction symptoms known as the Arnold-Chiari syndrome. The orthopaedic surgeon frequently is the first to observe the patient presenting the symptoms, and, therefore, may direct the patient properly.

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