SCOLIOSIS WITH PARAPLEGIA

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Our interest in the problem of scoliosis with paraplegia, excluding spinal deformities due to tuberculous disease, was aroused in 1925 when we operated on an eighteen-year-old youth who had severe scoliosis of the thoracic spine with motor and sensory involvement of the lower limbs and trunk (Fig. 1). After surgical decompression of the spinal cord there was striking improvement. This case was reported in 1927 and, since that time, four others have been added to our series: these five, together with reports of comparable cases in the literature, make up a total of forty-one cases now available for study. Most of these patients were males—which is odd, because orthopaedic surgeons are almost invariably impressed by the relative frequency of scoliosis in females. The deformity was always in the dorsal spine, and as a rule there was a marked kyphotic element.

![Figure 1](image1)

Case 1. S. S. The dural sac as it was exposed at operation (Fig. 1). Herniation of the cord through the dural incision illustrates the importance of dividing the dura if pressure on the cord is to be relieved (Fig. 2).

In all but eight cases the onset of symptoms due to interference with the spinal cord occurred between the ages of twelve and nineteen years. Two patients were aged six years, and six were twenty to twenty-three years of age. With three exceptions, early evidence of cord damage heralded constant deterioration which usually terminated in profound motor and sensory loss but seldom in interference with sphincteric control. The level of cord damage nearly always corresponded to the apex of the deformity—a fact which was demonstrated by intrathecal injection of lipiodol or pantopaque.
There is no record of the treatment of three patients, one of whom died; twenty-four were at some period treated conservatively (though ten were operated upon later); and, in all, twenty-four patients were operated upon. The most obviously successful results were gained by laminectomy, but in this group there were three post-operative deaths.

Case 1. S. S. Clinical appearances of the patient in 1926.

Case 1. S. S. Clinical appearances (Fig. 3) and radiographic findings (Figs. 4 and 5) as recorded in 1926 when the patient was eighteen years of age. Severe kyphoscoliosis, probably of idiopathic type, measuring 109 degrees. The lipiodol block is just above the apex of the curve at the seventh dorsal level (Fig. 4). The curved lines shown in Fig. 5 mark the extent of the laminectomy, and the two silver clips indicate the point of maximal compression of the cord. After an interval of twenty-one years there has been no appreciable increase in the curve.

The paucity of case reports of scoliosis with paraplegia, and the fact that surgeons who see many patients with severe scoliosis have never seen one with paraplegia, indicates the rarity of the syndrome. It seems clear that there must be some factor other than the deformity itself that accounts for paralysis.
Case 2. M. L. The patient is seen as a boy, aged fifteen years, in 1935 (Fig. 6); and as a man, aged twenty-seven years, in 1947 (Fig. 7). The antero-posterior radiograph (Fig. 8) taken in 1947, shows congenital deformities in both upper and lower dorsal regions of the spine. Laminographic studies of the greater deformity (antero-posterior projection in Fig. 9, and lateral projection in Fig. 10) show a hemivertebra which is outlined. The kyphosis measured 120 degrees and the block occurred at the apex.

Case 5. C. F. Patient in 1947, aged twelve years, is seen in Fig. 11. The curve measured 80 degrees. Laminographic studies showed its congenital nature with a hemivertebra and extra rib (Fig. 12, inset). A myelogram showed the level of block at D.4 (Fig. 12) and strikingly exposed the very eccentric relation of the cord to the vertebral bodies caused by the rotation, and by the tight dural sac hugging the inner wall of the spinal canal.
The problem has been reviewed again for a number of reasons. 1) The operative findings in one patient were unique in our experience and, so far as we know, have not been described before (Case 3, Table I). In this unusual case a congenital band was stretched across the dural canal, and resection of the band cured the paralysis. This case will be presented in detail. 2) Since treating the first patient in 1925 we have operated on four others, and we are now in a position to speak with more certainty regarding the long-term prognosis. 3) Technical points of the operation deserve mention. 4) In previous reports there has been little discussion as to the type of scoliosis which may be associated with paralysis, whereas in our study this has been considered carefully. 5) It was hoped, by careful review not only of our own cases but of others reported in the literature, that we might be able to determine the best method of handling these problems and elucidating their origin.

**CLINICAL MATERIAL**

**REPORT OF FIVE PATIENTS WITH SCOLIOSIS AND PARAPLEGIA**

**TABLE I**

<table>
<thead>
<tr>
<th>Case</th>
<th>Neurological findings</th>
<th>Date and type of operation</th>
<th>Result</th>
</tr>
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<tbody>
<tr>
<td>Case 1</td>
<td>S. S.</td>
<td>Male</td>
<td>18 years</td>
</tr>
<tr>
<td>Case 2</td>
<td>M. L.</td>
<td>Male</td>
<td>15 years</td>
</tr>
<tr>
<td>Case 3</td>
<td>R. W.</td>
<td>Male</td>
<td>16 years</td>
</tr>
<tr>
<td>Case 4</td>
<td>E. L.</td>
<td>Male</td>
<td>29 years</td>
</tr>
<tr>
<td>Case 5</td>
<td>C. F.</td>
<td>Female</td>
<td>12 years</td>
</tr>
</tbody>
</table>
Case 1—S. S.—The first patient was operated upon in 1925 and was reported in detail (McKenzie 1927). The clinical appearances, and radiographs of the spine, are seen in Figs. 3–5. The taut and narrowed dural canal, and the marked release of cord pressure when the dura was opened, are shown in Figs. 1 and 2. Certain features of the operation which are believed to be important are discussed later (page 170). After operation there was rapid recovery from the motor paralysis and sensory disturbance which had progressed for two years and reached the point where the patient had been bedridden for two weeks. When he was re-examined in December 1947, twenty-two years after operation, there was normal muscle power in the lower limbs, no abnormality of reflexes, and no sensory disturbance: in fact, the neurological picture was so normal that this was quite evidently the best result in our series of five patients.

Case 2—M. L., and Case 5—C. F. had operative findings comparable to those in Case 1. The essential points pertaining to these patients are shown in Table I.

Case 4—E. L.—At the time of operation this patient with scoliosis and paraplegia showed no neurological signs above the level of spinal curvature. Four years later, lower motor neuron signs and sensory disturbance developed in the upper limbs. It was then found that he was suffering not from pressure paraplegia but from syringomyelia with congenital lesions of the cerebellar fossa—the Arnold-Chiari syndrome. The scoliosis was probably the result, and certainly not the cause, of the neurological lesion.

Case 3—R. W., is reported in detail because the cause of paraplegia was quite different from that of other patients in this series and other cases recorded in the literature. This sixteen-year-old boy was admitted to the Toronto General Hospital in August 1941. At the age of seven, while riding a toboggan, he hurt his back, but within a few days he was well. His birth and early development had been normal except that the left arm was always shorter than the right. Two years before admission it was noticed that his back was not straight; the right shoulder was more prominent than the left. There was no history to suggest infantile paralysis or any acute infection.

Clinical Examination—There was a dorsal scoliosis, convex to the right (Figs. 15–17) with no weakness of the abdominal muscles which might suggest a paralytic origin. For no obvious reason the left humerus was two inches shorter than the right. Neurological findings—There was no weakness of any isolated group.
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Case 3. R. W. Photograph in 1947, at the age of 23 years.

Fig. 15

Case 3. R. W. Photograph of the patient, at the age of twenty-three years, taken in 1947, shows that the scoliosis is not severe (Fig. 15). This is confirmed in the antero-posterior radiograph taken in the standing position at the age of sixteen years in 1941 (Fig. 16). The block to lipiodol occurred at the level of D.3, in the lesser curve, which is not in accord with the findings in typical cases. An antero-posterior radiograph taken in 1947 in the supine position (Fig. 17) shows that there has been no increase in the curves. Arrows point to silver clips which mark the cut extremities of the congenital band.
of muscles such as might be expected in poliomyelitis. There was a marked spastic paraplegia. He was just able to get about with a stiff, shuffling gait which had come on gradually during the previous two or three months, and was first noticed in the right lower limb. There was general diminution of sensation over the lower limbs and trunk, suggesting a lesion at about the second or third thoracic level, and precipitancy of urination. *Radiographic examination*—Comparison of radiographs taken at the age of sixteen years, and again at twenty-three years, showed no change in measurement of the spinal curves. Neither was marked: the left curve, extending from D.1 to D.4, measured 20 degrees; and the right curve, extending from D.4 to D.9, 30 degrees. There was no significant lumbar curve. A mild round-back was associated with the scoliosis. *Lipiodol myelography* disclosed an incomplete block at D.3. The findings were compatible with idiopathic scoliosis. The case did not fall into the usual group because the deformity was not severe. Lipiodol was put into the cistern, and it trickled slowly past the area of scoliosis, showing that the block was not complete. Nevertheless, we were able to satisfy ourselves that there was a marked and prolonged hold-up at the level of the second or third thoracic vertebrae, thus fitting in with the neurological findings.

![Image](https://via.placeholder.com/150)

**Fig. 18**

Case 3. R. W. Illustration of the congenital membrane crossing the dural canal from side to side and compressing it. Marks indicate the line of excision of the band which was necessary to relieve pressure on the cord. After excision, recovery of the paralysis was complete. It is possible that this band, of developmental origin, was the cause of the so-called idiopathic scoliosis.

*Operation*—(K. G. McK.), August 15, 1941—The surgeon's note, made immediately after operation, reads: "Laminectomy was done in the suspected region. The extradural fat, instead of being soft and easily handled as it normally is, proved to be very tough and interspaced with firm fibrous bands; it was at least one-eighth of an inch in thickness. When this was dissected away, I was still confronted with an abnormal situation. There was a firm, extremely tight band of tissue stretched over the dural canal. Passing downwards, this band cut across the dural canal, to be anchored in the left lower part of the field. The band was about the thickness and consistency of dura; it was just as tight as a bow-string and undoubtedly was causing compression of the dural canal. Superficially, this membrane had some light attachment to the overlying extradural fat. Where it was adjacent to the dura it was easily dissected or separated—by that, I mean that it did not have any intimate connection with the dura, so that I think it is some
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sort of congenital abnormality rather than inflammatory tissue. The membrane was firmly anchored above and below beyond the operative bone defect. This membrane was partly dissected out and I feel that we have completely removed the pressure on the dural canal. Unfortunately, the specimen was lost, only the overlying extradural fat being saved for section. If, however, the patient does not do well, further operation should be carried out higher up. The dura was not opened. Doctor Harris was called in to see this, as I had never seen anything like it before. We wondered if this abnormally tight band could have produced the scoliosis. Two silver clips have been left in; they will show the upper and lower extent of removal of this band, and show its relationship to the scoliosis" (Fig. 17). Pathological Report—The extradural fat showed no evidence of inflammation. Unfortunately the band which was partly removed at operation was lost.

Follow-up Reports—Three weeks after operation the patient was discharged from hospital walking, with the sensory loss much improved. One year after operation—Still walking with a stiff gait. Able to stand on toes. Numbness gone from legs. No longer has precipitancy of urination. Gait still improving. Six years after operation—In good health, able to walk several miles without difficulty. Definite stiffness and spasticity of the lower limbs, especially the right. All sensory disturbance has cleared up. There is bilateral clonus, more marked on the right; and bilateral extensor response to plantar stimulation. Subjectively the right leg is not as strong as the left, but each individual muscle group shows excellent strength: neither dorsi-flexion nor plantar-flexion of the ankle can be overcome by the resistance of the examiner. The residual neurological signs are presumably due to intrinsic damage to the spinal cord from prolonged interference with circulation, rather than to continued pressure.

This case may represent no more than an unusual finding, not previously reported in the literature. On the other hand, it may be of importance if similar lesions are found in other cases of idiopathic scoliosis. Certainly the band was sufficiently strong and tight to have produced the scoliosis itself. We believe that it was a developmental abnormality—possibly a remnant of the outer layer of dura which, in the spinal canal, is normally separated from the inner layer comprising the spinal dural canal. At any rate we propose to look for such a lesion in a few cases of severe idiopathic scoliosis which are to be treated by spinal fusion with the object of preventing further progression of deformity. It is possible that in some patients, such a lesion may be the cause of "idiopathic" scoliosis.

COMMENTS ON THE RADIOGRAPHIC FINDINGS

Two facts became evident from our study of the radiographic findings in our own cases and those reported in the literature. Almost without exception the paraplegia was associated with severe deformity; and in patients who were examined by myelography there was clear evidence of compression of the cord near the apex of the curve. In earlier reported cases the etiology and type of scoliosis was often obscure, clinical description and radiographic reproduction being inadequate. Many were described as congenital even although there was no substantive evidence other than the early onset and the marked wedging of apical vertebrae. It seems probable, however, that most cases were in fact congenital in origin. The second largest group was probably idiopathic in the modern sense of the word. A few were attributable to infantile paralysis; some were associated with rickets; and two were examples of Von Recklinghausen's neurofibromatosis involving bone.

There is little or no connection between the liability of a case of scoliosis to develop paraplegia and the origin of the scoliosis. What is more important is the severity of the curve, and probably the rapidity of its development.

COMMENTS ON OPERATIVE TREATMENT

In nearly all patients the cause of paraplegia is compression of the spinal cord by the dura. This compression is relieved by opening the dura on the convex side of the curve. The dural opening should be sufficiently long to uncover the deep sub-arachnoid spaces above and below (usually three to six inches). This is important in order to avoid nipping
of the cord if it should swell after operation. The dura should be opened carefully so that there will be no tearing of the arachnoid. If the arachnoid is opened, by mischance, pledgets of fibrin or gel-foam should be left over the area so that an accumulation of cerebro-spinal fluid between the exposed cord and the overlying muscles may be prevented. In Case 1 of this series, in which the best result was gained, not only was the dura opened but two tight nerve roots were divided because it was thought that tension arising from them might interfere with the circulation of the cord. For the same reason, it may sometimes be wise to cut several dentate ligaments. On several occasions we have felt that adequate decompression necessitated lateral cuts, as well as a longitudinal cut in the dura. Occasionally it may not be necessary to open the dura at all (as in Case 3 of this series—in which the compression was due to an extradural band).

The results of operative treatment—Of the forty-one cases that have been studied, twenty-four were treated by laminectomy—five of our own patients, and nineteen reported by others. Three patients died: two died within a few hours of operation, the exact cause of death being uncertain; one died several weeks after operation from decubitus ulceration and cellulitis. Fifteen patients who were traced for more than seven months after operation made a recovery that could be classified as good or excellent; three gained only slight improvement; and three were not improved at all. The six poor results warrant further study.

(i) Case reported in this series (Case 4—E. I., Table 1)—At operation there was some doubt as to whether the dura was tight enough to be causing cord pressure. Later, an ascending lesion of the cord proved to be due to syringomyelia associated with herniation of the cerebellar tonsils (Arnold-Chiari syndrome).

(ii) Case reported by Ellmsie (1925)—A fourteen-year-old boy had neurological symptoms of two years' duration. When the dura was opened it was apparent that the cord was squeezed in the usual manner. Recovery was slow and the patient was just beginning to walk at the end of one year. In view of the long duration of symptoms, delayed and incomplete recovery might have been expected.

(iii) Case reported by Grobelski (1932)—In this patient, kyphoscoliosis was first associated with symptoms referable to the cord at the age of four to six years. Progress was arrested by conservative treatment which was continued over a long period of time. Nevertheless complete freedom of walking was never restored. At the age of ten years, cord symptoms became prominent once more and the patient was bedridden in a few weeks. Every type of conservative treatment was again tried and continued for three years. Finally, at the age of thirteen years, laminectomy was performed. The dura was very tight, and the cord was flattened and pale. Two years later the condition was unchanged. This patient had been profoundly paraplegic for three years before operation. It must be concluded that, when definite neurological signs develop, decompression of the cord should not be delayed.

(iv) Case reported by Hewery (1944)—In a thirteen-year-old patient with cord symptoms of only three months' duration, deformity due to Von Recklinghausen's disease was short and sharp. The dura was tight, and the cord was pale and thin. No improvement was noted in the year after operation. In this case there was no record of examination by myelography and it is possible that an extradural neurofibroma, elsewhere than at the site of operation, was missed. It is also possible, however, in view of the pallid nature of the cord, that extreme pressure had produced irreversible damage.

(v) Case reported by Ruhlkin and Albert (1941—Case 3)—This was a patient, aged twenty-one years, with cord involvement of nine months' duration and marked dorso-lumbar kyphoscoliosis. After conservative treatment for twelve weeks, with some improvement, laminectomy and decompression of the cord was complicated by the development of a post-operative haematoma which had to be evacuated. Three years later there was no improvement. This case illustrates the importance, when the dura is opened and the spinal cord left exposed, of arresting all haemorrhage and leaving the operative field dry.
(vi) Case reported by Ruhlin and Albert (1941—Case 4)—This case was also complicated by the formation of a post-operative haematoma requiring evacuation, again illustrating the importance of a dry field when the cord is exposed. If a haematoma is suspected, because the neurological signs increase after operation, immediate exploration should be undertaken in order to prevent sustained pressure on the cord.

Of the fifteen patients that did well the dura was left open in thirteen. Recovery was complete in five; and in eight cases, despite residual spasticity, there was no complaint and the patients walked well. It is reasonable to believe that irreversible changes in the cord occur with less frequency if operation is performed soon after the first appearance of neurological signs. In two cases there was no reason to open the dura because the cause of obstruction was apparent—in one, a congenital band (Case 3, Table 1); and in the other, a bone outgrowth, removal of which was followed by recovery (Heyman 1937).

The benefit gained from operation was clearly due to release of tension on the spinal cord by opening the dural sac. It was usually noted that the cord herniated through the dural slit, and often that there was return of pulsation in the cord which previously had been absent. Eleven of the patients with good results have been traced for more than one year after operation. In the three cases of decompression reported in this series, which were traced for twenty-two years, twelve years, and seven years respectively, the improvement gained from operation was maintained and there was no evidence of recurrent symptoms. It would seem that if decompression of the cord is successful, the long-term prognosis is assured.

Decompression combined with spinal fusion—In only two cases reported in the literature was spinal fusion performed some months after decompression, presumably because there was fear of increased deformity and recurrence of cord symptoms (Heyman 1937, Ruhlin and Albert 1941). Such fear, however, is not substantiated by long-term results in our own cases and others reported in the literature. Spinal fusion should not be combined with decompression at the initial operation because it increases the likelihood of haematoma formation. Moreover, since the good results of simple decompression have been lasting, with no recognisable increase in deformity even when cases have been observed for many years, we believe that spinal fusion is seldom indicated even at a later date. We would consider such a procedure only if there was definite evidence of increasing paralysis despite laminectomy; and this does not appear to be the usual consequence of the operation.

COMMENTS ON CONSERVATIVE TREATMENT

Twenty-four patients were treated conservatively for varying periods: twenty-three by traction and the application of plaster jackets and corsets; and one by spinal fusion without decompression of the cord. In the case of spinal fusion the cord symptoms increased despite sound consolidation of bone (Ruhlin and Albert 1941). Of all the cases treated conservatively eleven were improved; in one there was temporary arrest in the progress of paralysis; and in twelve there was no change at all. Of the eleven cases which improved, six were traced for eleven months or longer, and in five the clinical records are sufficiently complete to show that the late-result was good or excellent. Of the twelve failures, ten were explored later with good results in eight, a poor result in one, and death in one.

It is unfortunate that better records are not available in cases treated conservatively, for we find ourselves unable to make adequate comparison with operative methods. There seems to be no doubt that in five cases there was remarkable improvement, and so far as we can tell the improvement was maintained. It is to be noted, however, that relatively long periods of treatment were necessary: in one instance there was no evidence of improvement for two months (Kleinberg 1923) and since we know that prolonged cord pressure may cause
permanent impairment of function, the treatment is perhaps dangerous unless there is evidence of recovery soon after treatment is started. It was the lack of early response that prompted surgeons to perform laminectomy in ten cases which failed to show improvement after conservative treatment. Furthermore, if paraplegia is not due to dural pressure, relief cannot be expected from conservative treatment, and this applies to the third case in this series where there was a congenital band (R. W., Table I), and to the case reported by Heyman (1937) where the pressure was due to a spur of bone.

In view of the facts: that in skilled hands decompression of the cord is not a dangerous procedure; that there is often constricting pressure due to a tight dura, a congenital band, or a spur of bone; and that sustained compression of the cord may lead to irreversible changes causing permanent loss of function: we must conclude that early laminectomy is advisable and that it should not be delayed too long.

COMMENTS AS TO THE CAUSE OF PARAPLEGIA

Study of twenty-four cases treated by operation shows that, with the exception of three (bone block, congenital band, and syringomyelia), the cause of paraplegia was the combination of a tightly stretched dura and a sharply angulated spinal canal, the point of maximal pressure by the dura being localised at the angle of the spinal canal. That the cord itself is not under longitudinal tension is borne out by the ready escape which may be observed at operation through the linear release incision made in the dura. If then the cord is in fact compressed, which is established not only by the findings at many operations but also at two autopsies (Valentin and Putschar 1932, Thomas, Sorrel, and Sorrel-Derjerine 1933) the compression must be from the tightness of the dural sac. The sac is attached more firmly to the foramen magnum above, and the sacrum below, than it is to the sides of the spinal canal by its prolongations on the nerve roots. If it is taut, it will resist the tendency of the spinal cord to be displaced from a straight line by deformity, thus explaining localisation of the lesion to the apex of the curve. The lumen of the dural sac must be narrowed still further by rotational displacement.

It may be that in all cases of severe scoliosis there is some longitudinal dural tension, a degree that is usually insufficient to cause symptoms but is ready nevertheless to respond to anything which might tip the scales. This was suggested by Jaroschy (1928) who demonstrated by intrathecal lipiodol an incomplete block at the apex of the curve in two patients whose scoliosis was not associated with paraplegia. It is corroborated in the case reported by Grobelski (1932)—a young patient, in whom neurological symptoms first appeared at the age of six years: progress in the neurological symptoms was arrested by conservative treatment, and over a period of four years there was neither improvement nor deterioration; at the age of ten years distortion of the spinal column increased rapidly and was accompanied by increased difficulty in walking; within a few weeks paraplegia was complete.

It seems probable that in these rare cases of scoliosis with paraplegia the exciting cause which precipitates paralysis is the rapid growth of the spinal column together with the inability of a tight dura to accommodate itself to such growth. In all but six of the forty-one cases that have been reported, the age at which there was recognition of abnormal neurological signs corresponded to the years of most rapid growth. One exception is explained by the final diagnosis of syringomyelia (Case 4, this series); and, in a second case, doubt may be cast on the validity of diagnosis because the cord symptoms showed many remissions and no steady progress (Roger and Schacter 1940). In the few cases in which paraplegia first developed in later years, after growth had ceased, the precipitating cause is obscure; but some light may perhaps be thrown on this matter by future observations at operation and autopsy.

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SUMMARY

1. Five cases of scoliosis with paraplegia are reported, and thirty-six comparable cases from the literature are reviewed. These forty-one cases have been studied with the object of determining the etiology of scoliosis, the reason why cord compression sometimes develops, and the results of conservative and operative treatment of such compression of the cord.

2. The cause of paraplegia is nearly always compression of the spinal cord by the dura, which, in severe scoliosis, is under longitudinal tension because of its firm attachment to the foramen magnum above and the sacrum below. Such tension, resisting displacement of the spinal cord from the straight line, may be shown to cause incomplete spinal block even when there is no paralysis.

3. When paralysis occurs it usually develops during the years of most rapid growth, the tight dura being unable to accommodate itself to the rate of growth of the spinal column; cord compression is probably increased by narrowing of the dural sac by rotational displacement.

4. The most striking results have been secured by laminectomy with section of the dura and sometimes division of dentate ligaments and tight nerve roots. After such division there is evidence of release of compression: the cord herniates through the dural slit; and spinal pulsation returns.

5. It is important to control bleeding in order to avoid post-operative compression by blood clot; and to prevent leakage of cerebro-spinal fluid through the arachnoid.

6. It is unwise to perform spinal fusion at the same time as decompression because it increases the danger of haematoma formation. Moreover the improvement gained by decompression is maintained even if no fusion of the spine is performed.

7. Conservative treatment of scoliosis with paraplegia should not be continued for long periods unless there is evidence of early and progressive improvement because prolonged compression causes irreversible changes in the cord.

8. In three cases, paraplegia was not due to dural compression: one turned out later to be a case of syringomyelia; one, reported by Heyman, was due to the pressure of a bone spur; and one, reported in this series, was due to a congenital tight band of developmental origin which might have caused the scoliosis as well as the paralysis, and in which, after resection of the band, recovery from the paralysis was complete.

DISCUSSION

Dr Arthur Steindler (Iowa City, Iowa)—This series of cases of paraplegia in scoliosis is a valuable and most welcome addition to our knowledge. As the speakers have indicated, earlier reports of this complication were indeed scarce. Since the first description by Mauclaire in 1913, and the report by Ridlon in 1916, there are hardly two score cases on record. From our clinic in Iowa City, Ruhlin and Albert reported seven cases, five being treated by laminectomy. Our oldest observation goes back to 1915, and it concerned a boy, aged ten years, with a severe dorso-lumbar curve and a marked degree of rotatory deformity. Spastic paraplegia was progressive and it did not yield to conservative treatment though temporary improvement was gained by traction. Laminectomy was refused. Another case, treated by fusion alone without laminectomy, likewise showed no improvement in the degree of paralysis though the fusion remained solid. Of the five cases that were dealt with by laminectomy after unsuccessful conservative treatment, one died shortly after operation from pneumonia, and the other four were greatly improved or recovered completely.

The pathological findings were of interest. Case 1 (H. C.) had a pulsating cord with no direct pressure, but there was distinct tension on the cord due to kinking. One month after operation there was clinical improvement in sensory findings and no abnormality of reflexes. Case 2 (J. S.)—There was a question of neurofibromatosis. Laminectomy from the seventh cervical to the sixth dorsal level showed a pulsating cord but with a definite kink. After operation all symptoms were exaggerated but improvement began three months later, including improvement in bowel and bladder function. Case 3 (M. L.)—Severe right
thoracic curve. Laminecotomy from third to seventh thoracic vertebrae; distinct kink at fifth; rapid improvement; complete recovery after three months. Case 5 (C. N.)—Severe left cervico-thoracic curve. Laminecotomy fourth cervical to fifth dorsal vertebrae; pulsation absent but returned after decompression; severe kink at apex. Under observation for more than two years; marked improvement but not complete recovery.

The most interesting feature in the series reported by Dr McKenzie and Dr Dewar is the finding of a congenital band in one case. This conforms with the observation that most of these cases of scoliosis are of the congenital type. One wonders if the mechanical factor alone is responsible, or whether congenital anomalies have anything to do with the production of paralysis. So far as the pathomechanics are concerned, we believe that torsion of the cord is a more important cause of ischaemia than direct compression. How much distortion will the cord tolerate? And why does paralysis appear so seldom in severe scoliosis? In our series it developed in less than 0.3 per cent. of cases. Many questions remain unanswered. But the careful study of the authors' cases has furnished an important contribution to our knowledge and I deem it a privilege to be permitted to discuss their paper.

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