FACTORS CAUSING MASSIVE SPASM FOLLOWING TRANSECTION OF THE CORD IN MAN*

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Since the first World War the idea has become prevalent that when the spinal cord has been completely transected in man, after a brief period during which the reflexes are abolished, there develops, inevitably, a chronic state of heightened reflex activity in the isolated cord, leading gradually to mass reflex spasms, which are considered to be the result of "release" of the isolated portion of the cord from the influence of the brain.

These current views were foreshadowed by those of Romberg, Charcot, Gowers, Riddoch, and others. Romberg wrote:

When motility and cerebral sensibility are entirely lost, spinal or unconscious sensibility manifests itself by reflex action . . . . In spinal injuries some time elapses before these phenomena are manifested, a circumstance probably dependent upon the concussion of the organ . . . . The antagonist muscles assume a state of permanent tension . . . . The contraction of the flexors is occasionally so considerable, that the heel touches the nates.

Charcot in his lectures on spinal injury taught that:

In the first degree, simple paresis is observed with flaccidity of the limbs . . . . At the end of some days or of some weeks, later in certain cases, sooner in others, there supervene, in the paralyzed members, jerks, cramps, a temporary rigidity of the muscular mass,—so many symptoms attaching also to lesion of the lateral columns, but which already indicate that a source of excitation has taken up its abode in the fascicles . . . . Lastly, supervenes permanent contracture of the limbs . . . . It is the rule that this contracture shall first, for some time, maintain the paralyzed limbs in a posture of forced extension, but sooner or later generally speaking, a posture of forced flexion supervenes . . . . In this phase of the disease, under the combined influence of the suppression of the moderator influence of the brain . . . . the reflex properties of the inferior segment of the cord are augmented. Then, we see the paralyzed members rise and become convulsed at the least touch, or when the patient micturates or goes to stool.

In 1886, Gowers said:

An acute lesion in any part of the cord may cause an initial loss of reflex action in the part below, but if the lesion is above the lumbar enlargement, reflex action returns in the course of a few hours. Frequently, there is no initial depression. Subsequently, the reflex action becomes excessive, that from the skin rapidly, that from the muscles more slowly. Ultimately, each attains a high degree of exaltation, . . . Cases of superficial reflex action indicate withdrawal of the cerebral controlling influence of the reflex centers.

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Although these views were briefly challenged by Bastian\textsuperscript{1,2} and others,\textsuperscript{3,4,6,11,12,15} they continued to represent prevailing opinion.

In 1917 Riddoch\textsuperscript{13} published an important paper, which fortified the ideas previously expressed by Romberg, Charcot and Gowers, and has largely influenced all subsequent thought on this subject. Riddoch reported in great detail his observations upon five patients in whom complete transection of the spinal cord was verified at operation, four of whom lived for many months after injury. Each of these four patients, after passing through an initial period of so-called “spinal shock,” eventually developed massive reflex movements of the lower extremities, which Riddoch believed to be the true chronic state of reflex activity in the human following transection of the spinal cord. Although he carefully avoided ascribing these mass reflexes to the “release” of the isolated segments of the cord from the influence of the brain, there is no doubt that that is the general interpretation that was placed upon Riddoch’s observations by others and is the view most widely held today.

In our experience, on the contrary, the chronic reflex state of man following transection of the spinal cord is highly \textit{variable}. Of 12 patients with verified transection at mid and upper thoracic levels, studied by us, 7 developed mass involuntary spasms, while 5 did not. Furthermore, among the 7 cases in which spasms did develop, there was great variation in the interval of time elapsing between the transection of the cord and the onset of the spasms (Table 1).

These facts challenge the prevailing concept that “release” of the cord from the influence of the brain is the \textit{sole} mechanism responsible for the

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<th>Patient</th>
<th>Spasm</th>
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<tr>
<td>1</td>
<td>C-7</td>
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<td>6 mos.</td>
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<td>T-10</td>
<td>“M”</td>
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<td>T-8</td>
<td>“Br”</td>
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development of mass spasm following transection of the cord; and suggest that, in addition to "release," which is common to all this group, there were in those cases in which spasm developed, added factors not heretofore described that contributed to their occurrence. It was felt that these factors were essentially pathologic in nature and inherent in the lesion.

**SURGICAL OBSERVATIONS**

A. *Excision of Extrinsic and Intrinsic Scar Tissue from the Stumps of the Previously Severed Spinal Cord (5 cases).* The considerations above described led, in 5 patients suffering mass spasms, to surgical exploration of the lesions.

In each case the spinal cord was found completely divided, with the proximal and distal stumps connected by a strand of fibrous tissue. The first 1–2 cm. of each stump were grossly distorted and firmly anchored to the dura by massive adhesions. This portion of the cord was unresponsive to both mechanical and electrical stimulation. Immediately beyond this area, however, in the distal stump, there was a "transition zone" in which the cord began to assume a normal appearance; and here, minimal mechanical and electrical stimuli initiated mass contractions which were greater than those obtained with the same stimuli applied to more distal segments. From these observations it would appear that the area of anatomic transition represents a region of hyperexicitability or "trigger zone." Within this zone the maximal responses were obtained from stimulation of the dorsal columns, while lesser responses were elicited from the ventro-lateral portion of the cord and the dorsal roots. Stimulation of the ventral roots produced only a segmental response.

In 4 patients both the distal and proximal stumps of the cord were released from adhesions binding them to the dura, and excised. In one case the proximal stump, alone, was excised. Microscopic examination of these stumps revealed in each instance marked distortion of the normal architecture and extensive degeneration and gliosis.

Following operation there was in each instance an alleviation of spasms ranging from 20 to 90 per cent. This improvement, however, was only temporary, and after varying intervals of time, ranging from 2 to 10 weeks, there began a gradual increase in the spasms.

*Case 1—"B".* On 7 October 1944, this patient received a gunshot wound of the spine at the level of T-4 resulting in immediate complete sensory loss and flaccid motor paralysis below his wound. One month after injury, involuntary spontaneous reflex spasms began, increasing in severity so that they had become almost constant and quite violent, forcing the patient to assume, while in bed, a position in which the thighs were acutely flexed on the abdomen and the legs acutely flexed on the thighs. Even with forceful and sustained traction on the ankles, the legs could be straightened only a few degrees due to the violence of the flexor spasms. There was no evidence of any return of motor, sensory or bladder function.

On 25 September 1945, laminectomy was performed under local anesthesia at the level of his injury. The spinal cord had been divided by his original wound, leaving only a tough, gritty strand of scar. This was glued to the dura by dense adhesions, as were the distal and proximal stumps of the cord itself. The tough fibrous scar, after tests had showed it conducted no nerve impulses, was excised. Adhesions around the diseased ends of the cord were freed.
At the lower end of the scar, in obviously diseased spinal cord, was a trigger zone which was removed with the scar. Microscopic section of this scar showed that no nerve fiber of any kind ran through it. Section through the diseased trigger zone at the lower end of the scar showed considerable loss of myelin, internal scarring and disorganization.

Following this operation, the patient, though not cured of his spasm, was definitely better. On 30 October, 5 weeks after the laminectomy, he was observed continuously for 4 hours, during which time he had only 3 very mild spontaneous spasms of his legs. At that time, it was no longer possible to elicit mass reflexes by cutaneous irritation and it was difficult to initiate them even with strong proprioceptive stimulation. In early November, he traveled to a nearby city to see a football game and in December he made a long journey to his home on a furlough. Since then, however, follow-up reports indicate that there has begun a gradual increase in the frequency and severity of spasms.

*Case 2—"R."* In February, 1944, this patient received a penetrating wound of his spine at the level of T-4 resulting immediately in complete sensory loss and motor paralysis below that level, which have persisted to date without any signs of recovery. Five months after injury, spontaneous mass reflex spasms of his legs began. These spasms increased in frequency and severity. They would occur after any cutaneous or proprioceptive stimulus. They were frequently initiated by a change in position in bed. They often lasted for 20–30 minutes and would be so severe that they would audibly "knock the wind" out of him and leave him exhausted.

On 28 September 1945, under novocain anesthesia, laminectomy was performed. The findings and the procedure were similar to those described in Case 1, but the excision of cica-trix and scarified cord was less radical and complete than in some of the later cases.

Following operation, there was slight but definite improvement. There were no longer reflex spasms following cutaneous stimulation of the legs, and on many occasions it was not possible to elicit spasms by any form of stimulus. On 10 October 1945, it was estimated that his spasms were only about 30 per cent as severe as they had been before his operation. On 12 October 1945, this patient set out with his mother in a car for his home in California. He has not been seen by the writers since that time, but follow-up reports have indicated a gradual return of the spasms of the legs, although not to their original severity.

*Case 3—"N."* In November, 1944, this patient received a gunshot wound of his spine at the level of the 6th thoracic segment which resulted in immediate and total loss of sensation and power below that level, which has shown, to date, no signs of recovery. A few months later, reflex spasms of his legs began.

On 24 September, under novocain anesthesia, his spine was explored at the site of his wound. At that time, his legs were in such a state of spasm that both hip and knee joints were almost continuously flexed to right angles. He had to be forcibly strapped to the table and even then, during the greater part of the operation, his pelvis was well off the table.

The findings were similar to those described in Case 1. Scar tissue and the cicatrized upper and lower stumps of the previously severed cord were excised. On 28 October, 5 weeks after operation, he had only a few spontaneous spasms. He no longer has reflex spasm on cutaneous stimulation of his extremities, although squeezing his quadriceps femoris muscle still causes marked spasm of the muscle. Girdle pains are very much reduced in both frequency and severity. They no longer cause nausea or vomiting. The patient has gained weight. Improvement is estimated at 40–50 per cent.

*Case 4—"Br."* On 21 September 1944, this patient received a gunshot wound of his spine with immediate loss of all sensation and motor power below the level of the 5th thoracic segment, which has persisted to date. Several months later spontaneous massive reflex spasms began which became increasingly worse.

On 9 October 1945, under novocain anesthesia, laminectomy was performed at the site of the injury and the lesion exposed. The cord was reduced for about 2 cm. to a hard, gritty band of tissue having a cross section about 1⁄2 that of the normal cord, tightly bound to
dura with dense adhesions. These adhesions were divided, and the fibrous band and adjacent cord freed of adhesions. Nothing further was done at this session.

Following this first operation the patient had violent spasms of the abdominal muscles and also of the visceral musculature. These latter were associated with nausea, vomiting and simultaneous fecal discharge from the rectum. These continued for several days.

On 12 October 1945, the laminectomy wound was reopened under nitrous oxide-oxygen-ether anesthesia. At this time, the lightest possible touch of the distal stump of the cord with the tip of the forceps, at a point about 1 cm. below the hard fibrous band, was sufficient to set off massive reflex spasms of the legs. The slightest traction on cord or roots at this point produced similar results. Maximum spasms of legs occurred when touch was applied over the dorsal columns.

Electrical stimulation of the hard fibrous band gave no responses at all, even with very strong currents. Stimulation of the dorsal surface of the cord 1 cm. lower gave quick bilateral flexor mass spasms of the legs with equally quick relaxation; while stimulation of the dorsolateral and ventro-lateral surfaces of the cord produced much slower and less violent contractions and relaxations. There were no nerve elements in the scar on microscopic examination.

The hard gritty band of scar tissue was excised, together with about 3 cm. of the distal stump. In this was found a fair-sized syrinx occupying the ventral half of the gray matter and extending from the site of the lesion well beyond the lower limits of the excision and the surgical exposure.

Immediately after the operation, the patient had continuous clonic spasms of his legs which lasted for 12 hours, thus proving the absence of spinal shock following secondary transection in this case. After that, spontaneous reflex spasms gradually subsided. On 28 October they were less than half as severe as they had been before operation. Cutaneous stimulation no longer set off spontaneous spasms, though these could still be induced by proprioceptive stimuli. The legs no longer had to be tied down when the patient was in a wheelchair, which had been the case prior to operation. However, in the latter part of November, there was a gradual increase in the frequency and severity of the spasms, although they never attained their preoperative frequency and severity.

Case 5—"J." This young man sustained a gunshot wound of the spine at the level of the 6th thoracic vertebra in November, 1944. Immediately following this, he had complete motor and sensory loss below the level of his lesion which never showed any sign of recovery. A few months later, he began to have massive spasms of his legs, which gradually increased until his legs had to be tied down even while he was in bed. At the same time, he had severe girdle-like pains at the upper level of his anesthetic area. This pain and the spasms became so bad that the patient could not eat or sleep and death seemed imminent from undernourishment and exhaustion.

On 18 August 1945, under nitrous oxide, oxygen and ether anesthesia, laminectomy was performed primarily for relief of pain through rhizotomy. The cord at the site of injury was found to be shrunken, hard and partly ossified. Both it and the spinal roots were involved in the densest sort of scar tissue. It was found impossible to dissect out the roots in such a way that they could be sectioned between scar and cord. Accordingly, a block resection of about 3 cm. including degenerated cord of the proximal stump was carried out. The distal stump was not resected.

After operation, the patient's pain was entirely gone. He has had a few occasional small spasms of his toes, only. He has gained 15 lbs. in weight and is up in a chair every day.

In contrast to the 5 cases of spasm above described, one additional patient without spasms was explored at the site of earlier traumatic transection for relief of root pain. Essentially the same pathologic picture was found as in the preceding cases and similar effects were obtained upon mechanical and electrical stimulation. After resection of the desired nerve roots, the
cord was freed of adhesions to the dura but none of it was resected. Six weeks later, spasms appeared in this patient for the first time since he had been wounded 9 months prior to the operation. In this patient, once again, an operative procedure upon the distal stump of the cord had apparently affected the reflex activity of the isolated segments.

Case 6—"J.K." In February, 1945, this patient received a shrapnel wound of the spine resulting in complete paralysis below the level of the 7th thoracic segment, associated with severe root pain at the level of the lesion.

On 15 November 1945 laminectomy was performed at the site of the lesion for the relief of the root pain. Essentially the same pathologic picture was found as in the preceding cases, and similar effects were obtained following mechanical and electrical stimulation after section of the desired nerve roots. The cord was then freed of adhesions to the dura, but none of it was resected.

Six weeks after this operation, mass spasms appeared in this patient for the first time since he was wounded, and rapidly assumed major proportions; and it would appear that the surgical manipulation upon the distal stump of the cord had caused a persisting increase in the reflex activity of the isolated segments.

The 6 cases just reported indicate that operative procedures carried out upon the distal stump of a transected cord are capable of affecting the mass reflex activity of the isolated segments.

B. Surgical Section of Dorsal Columns Below the Level of the Spinal Lesion (2 cases). In the preceding cases both mechanical and electrical stimuli had produced maximum contractions when applied to the dorsal columns, thus suggesting that these columns, functioning in an antidromic or descending direction, were important channels for the dispersion of stimuli throughout the isolated portion of the cord. For this reason, in two patients with high thoracic cord transection, suffering spasms, the dorsal columns were divided below the level of injury. Section was carried out in each patient, at two separate levels, at the 10th thoracic segment with the idea of isolating the lumbosacral segments from abnormal impulses arising within the stump, and again at the 12th thoracic segment in an effort to reduce the dissemination of afferent impulses reaching the caudal segments of the cord from the periphery.

Following this procedure, there was striking relief from spasms in one patient which lasted three months. In the second patient, improvement was only transitory.

Case 7—"W." In July, 1944, this patient was wounded in the spine resulting in an immediate and lasting loss of sensory perception and motor power below the level of the 4th thoracic segment. Spasms began in the legs and abdominal muscles 1 month later, and became progressively worse, until they were quite severe and recurred spontaneously almost constantly. They could be initiated if the foot were stroked, the legs passively moved, or the patient ingested food.

On 14 October 1945, under novocain anesthesia, laminectomy was performed from T-8 to T-12 laminae, inclusive. The arachnoid membrane was moderately thickened, suggesting a previous widespread inflammatory reaction. The lightest possible touch applied with the tip of the forceps to the dorsal columns caused massive spasms of the lower extremities. Similarly, any mechanical stimulus applied to the dorsal roots would set off massive spasms.
Electrical stimulation produced maximum effects when applied to the dorsal columns, next when applied to dorsal roots. In each instance, the spasms were quick and the relaxations were quick. Stimulation of the ventral part of the cord gave sluggish, slow contractions of the legs with equally slow relaxations; while stimulation of the ventral roots gave the poorest mass response of all. By using weak stimuli, it was possible to obtain responses on one side of the body only. The dorsal columns were sectioned bilaterally at the level of the 10th and 12th thoracic vertebrae.

Immediately after operation, there was complete abolition of spontaneous massive spasms of the lower extremities; nor could spasms be induced by any form of cutaneous or proprioceptive stimulus.

At the time of writing this report, on 4 November, cutaneous stimuli failed to elicit spasms and the strongest possible proprioceptive stimuli produced only the very slightest movement of withdrawal; while no spontaneous mass movements at all have occurred since operation.

These cases demonstrate that the severity of mass reflex spasms of the lower extremities following transection of the spinal cord in man, may be definitely reduced by surgical section of the dorsal columns below the level of the lesion.

**SEGMENTAL REFLEXES**

The tendon reflexes in patients with transected cords and mass spasms were, prior to surgical resection, generally exaggerated. As regards the Babinski sign, there was up-going of the hallux in some cases, down-going in others, and no response to plantar stimulation in others. After surgical procedures on the distal stump of the divided cords there was marked reduction of tendon reflexes in 4 cases and alteration in the responses of the toes to plantar stimulation.*

**DISCUSSION**

*Clinical studies* show great variation in both the segmental and mass reflex activity of the distal segments of the transected cord in man. No relation appears to exist between the level of the transection and the presence or absence of spasms, nor any constant time-interval between transection and the appearance of spasms.

These considerations make it clear that the occurrence of mass spasms following transection cannot be satisfactorily explained solely on the basis of the "release" of the isolated segments from the influence of the brain. That variations in reflex activity cannot be satisfactorily correlated with damage of particular conducting systems of the spinal cord has already been demonstrated in an experimental manner by Mettler,9,10 while Lassek8 has demonstrated a similar lack of correlation from a pathologic point of view in the human.

*Surgical observations* have revealed marked pathologic changes in and about the distal stump of the traumatically severed cord, which include, in

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*A more detailed description of the segmental reflexes in these cases will appear in a subsequent communication.*
addition to dense adhesions anchoring the stump to the dura, extensive degeneration and gliotic changes within the cord itself. It has also been established that this portion of the cord in the region of the injury has a lowered threshold for both mechanical and electrical stimuli; and that nervous impulses induced within this region, in response to stimuli, are readily transmitted to more distal segments by way of the dorsal columns. It has further been shown that surgical procedures that alter the anatomic condition of the stump, cause alteration of the reflex activity of the cord below that level. Finally, it has been demonstrated that striking amelioration of the spasms may be obtained by interrupting one of the main pathways (dorsal columns) for transmission of impulses from the stump to lower segments.

The conclusion is drawn from these observations that factors operating upon, or within, the isolated portion of the divided cord, especially at the level of the lesion, may play an important role in determining the occurrence, as well as the severity of mass spasms.

An "irritative mechanism" contributing to the development of massive spasms following transection of the cord in man is suggested by these observations. Constant or oft repeated traction upon the adherent stump of the distal segment, or the irritative effect of gliosis within the stump, may increase the irritability of this portion of the cord, and lower its threshold for afferent stimuli. Efferent impulses arising in this hyperexcitable zone are then transmitted to the more distal segments, chiefly, though possibly not entirely, by the dorsal columns in antidromic direction. It is fully recognized that "release" of the cord from the influence of the brain may play a primary role in conditioning the isolated segments for the development of spasms; but it is believed that the actual occurrence of spasms in any given case of a transected cord, is determined by factors operating upon or within the isolated segments, chiefly at the site of the lesion, and primarily irritative in nature.

SUMMARY

1. Evidence has been presented showing that massive involuntary spasms do not invariably follow transection of the spinal cord in man.
2. Studies indicate that when massive spasms do appear, their occurrence is determined not alone by "release" of the cord from the influence of the brain but by additional factors, among which chronic irritation of the cord due to extrinsic or intrinsic scarring of the distal stump at the site of the lesion appears to play an important role.
3. Electrical stimulation of the human spinal cord at the operating table in patients under local as well as general anesthesia is reported.
4. Relief from spasm, in varying degrees, has been obtained by the following surgical procedures:
   (a) Liberation of the ends of severed cord from cicatrices binding them to the dura, and excision of degenerated tissue from the stumps of the cord (5 cases).
(b) Surgical section of the dorsal columns caudad to the lesion (1 case).

5. An "irritative mechanism" contributing to the occurrence of mass involuntary spasms in man following transection of the spinal cord has been proposed.

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