Deafferentation to Relieve Spasticity or Rigidity: Reasons for Failure in Some Cases of Paraplegia*

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In 1911, Foerster\(^1\) divided the posterior spinal nerve roots in man to decrease spasticity and paroxysmal spasms by interrupting the exaggerated stretch reflexes. He recommended sectioning the 2nd, 3rd, and 5th lumbar and the 2nd sacral posterior roots for the leg, and the 4th, 5th, 7th and 8th cervical and 1st thoracic posterior roots for the arm. The intervening roots were spared in an effort to avoid anesthesia and ataxia. He stated that the resulting sensory disturbances were negligible or minor and that besides lessening spasms and spasticity the procedure was followed by markedly improved motor activity. Clark and Taylor\(^2\) published results of similar studies. In 1936, Lehmann\(^3\) reported that 210 patients had been operated upon by the Foerster technique. He made a long follow-up study of 25 patients, 15 of whom had shown a very favorable outcome. Foerster was also able to abolish rigidity occurring in palilidal disease by his operation. Pollock and Davis\(^4\) obtained complete flaccidity in a case of postencephalitic parkinsonism after sectioning the posterior roots from the 4th cervical through the 4th thoracic segments unilaterally. The tremor continued although changed in rate.

It is indeed possible to abolish spasticity and spasms by posterior spinal root section or by interrupting the myotatic reflex are within the spinal cord as reported by Pourpré,\(^5\) and by Gonsette and Andre-Balisaux.\(^6\) On the other hand, the following cases show that deafferentation of a joint or of an extremity may fail to abolish severe hypertonus and spasms in paraplegia.

Case Reports

Case 1. B. M., a 38-year-old housewife, entered the hospital in March, 1954, because of pains in her feet for 1 year. Her past history contained no facts relevant to her present illness except possibly the delivery of her 2 children by Caesarian section, one in 1946 and the other in 1950, followed by intestinal obstruction requiring an emergency operation. The operations were done under spinal anesthesia.

Examination. The abnormal findings on examination were a tender 5th lumbar spinous process, markedly weakened eversion of both feet, and decreased ankle jerks. A myelogram showed a complete block at the 1st lumbar level. We considered a herniated intervertebral disc most likely.

Operation. On March 18, 1954, a laminectomy of T-12, L-1 and L-2 was done. The nerve roots on the right side were markedly adherent to the meninges and to each other. The spinal cord underlying T\(_{12}\) was considerably enlarged. A 26-gauge needle introduced through the midline of the spinal cord at this level yielded 2 cc. of faintly yellow fluid, evacuation of which left the cord collapsed. The cyst fluid contained 17 white blood cells (10 polymorphonuclear leucocytes and 7 lymphocytes) per cu. mm. and a faint trace of albumin on Pandy testing. The cerebrospinal fluid contained 8 white cells per cu. mm. and 190 mg. % of protein. The impression was central cavitation of unexplained origin. Nothing further was done.

Second Admission. Over the next 8 years the patient's condition gradually deteriorated. By 1963, she complained of marked painful flexor spasms of the lower limbs. The hips and knees were acutely flexed an angle of about 70° and attempts to extend these joints demonstrated markedly hypertonus up to about 120° beyond which contracture prevented further extension. There was barely perceptible voluntary movement at the knees and ankles but she could flex her hips through a range of about 30°. There was a bilateral loss of position sense in the great toes and ankles. Perception of pinprick was absent or markedly decreased below the level of the costal margin. The knee jerks were hyperactive and the ankle jerks absent. There was no response on plantar stimulation. The patient stated that by straining she could void an essentially normal quantity of urine. She had no sensation of voiding however, nor of defecating. Pantopaque myelography done through a cisternal tap showed a partial arrest in descent of the medium at the level of T\(_{1}\) and a complete arrest at T\(_{11}\).
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Second Operation. In view of her steadily deteriorating condition, and her violently painful spasms, laminectomy of T_7-T_{10} and re-exploration of the former laminectomy site (T_{11}-T_{12}) was done under general anesthesia. The anesthesia abolished the lower limb hypertonus but it was impossible to extend the hips or the knees beyond an angle of about 120° because of contracture. The arachnoid was thickened throughout. The spinal cord appeared normal underlying T_7 vertebra but just below this level it was soft and bluish. A longitudinal incision 1 cm. in length was made in the midline of the dorsum of the spinal cord and a few drops of fluid escaped. There was a cavity within the cord 5–6 mm. in diameter continuous with the previously exposed one. In an attempt to relieve this patient of her painful spasms and of the marked hypertonus so that one might institute rehabilitative measures a bilateral posterior rhizotomy, L_1 through L_4, was done. As the patient seemed to be able to control her urination, the sacral nerve roots were spared. Her ability to void after operation continued as before.

There was a slight temporary lessening of the lower limb hypertonus for a few weeks but then the hypertonus resumed its former severity. Painful flexor spasms of the lower limbs continued, the pain being abdominal. Her condition remained unchanged when last examined, 8 years following the rhizotomy.

Comment. In all probability this patient’s intense lower limb hypertonus and flexor spasms were due to the central cavitation of the spinal cord demonstrated at operation possibly secondary to arachnoiditis. Extensive destruction of spinal interneurones with resulting synaptic denervation of motoneurones must have occurred. The subsequent hyperexcitability resembled that we demonstrated in dogs. Such a mechanism for intense rigidity is more clearly shown in the next report of a paraplegic patient in whom total deafferentation of the lower limbs as well as spinal cord transection above the lumbar-sacral enlargement failed to overcome her severe hypertonus.

Case 2. M. W., a 38-year-old woman, had been stabbed at the level of C_{5-6} with resulting quadriplegia 6 years before her present hospitalization. A cervical laminectomy done 7 months after her injury was followed by severe painful flexor spasms of the completely paralyzed and anesthetic lower limbs, so that they were acutely flexed at the hips and knees. There was uniformly intense resistance to extension at these joints. The knee and ankle jerks were absent, as were abdominal reflexes, and there were no responses upon plantar stimulation. Nursing care was difficult and it was impossible to have her seated in a wheel chair.

Operation. Laminectomy of T_{11}, T_{12} and L_1 was done in August, 1964, in order to sever nerve roots and thus eliminate the hypertonus and severe flexion spasms. Under general anesthesia the hypertonus was abolished and the extremities were easily extended to angles of about 120° at the hips and knees, beyond which further extension was limited by contracture. The spinal cord was enlarged, translucent, and fluctuant. Incision through its mid-dorsum yielded over 30 cc. of clear colorless cerebrospinal fluid. The cavity occupied most of the spinal cord. Nothing further was done at this operation.

The patient’s spasms and hypertonus continued unchanged after operation. We suspected that the spasms and rigidity might be due to extensive interneuronal destruction and decided to try posterior rhizotomy, then cordotomy, and finally, anterior rhizotomy, if necessary, to relieve her of the severe discomfort, and to facilitate rehabilitation.

Second Operation. On September 2, 1964, the previous laminectomy site was exposed and extended through L_4. No anesthesia was required. The posterior roots from L_1 through the sacral roots were cut bilaterally. The severe rigidity persisted. The spinal cord was then severed between T_{12} and L_1 segments and again there was no effect on the rigidity. Finally, section of the anterior lumbar-sacral roots immediately relieved the patient of the rigidity and flexor spasms. Her rehabilitation was facilitated by the operation.

Histologic study of the isolated lower end of the spinal cord, which had been removed, revealed a large central cavity surrounded by a thin rim of grey and white matter (Fig. 1). Counts of motoneurones (determined by counting nerve fibers in the ventral root, all other neurones being interneurones) and interneurones in the L_4 spinal cord segment revealed an interneurone to motoneurone ratio of 3:2 for the ventral horn. The comparable ratio in normal man has not yet been determined. However, in the dog whose neuronal population is almost certainly less than in man, studies showed an interneurone to motoneurone ratio of 13-14:2 in the L_7 ventral horn. These data suggest the magnitude of interneuronal destruction in this patient.

Comment. The increased resistance to manipulation of the extremity would classify the hypertonus as rigidity. Hyperactive reflexes, clonus, and the lengthening reaction ("clasp-knife" phenomenon) so characteristic of spasticity, were absent. Rigidity and spasticity can easily be differentiated from...