Failure of Lumbar Sympathectomy in the Relief of Hyperhidrosis

DAVID M. LEVY, M.D., DAVIT TOVI, M.D., AND ERICH G. KRUEGER, M.D.

Surgical Service, Section of Neurosurgery, Bronx Veterans Administration Hospital, and the Department of Neurological Surgery, College of Physicians and Surgeons, Columbia University, New York, New York

Sympathetic or overactivity of sympathetic nerve fibers supplying the extremities produces vasoconstriction, pallor, coolness of the skin, and sweating. Interruption of sympathetic innervation causes the reverse, namely, vasodilation, a red, warm skin, and anhidrosis. Sympathectomies have been performed in the past mainly for the improvement of the peripheral vascular circulation in Raynaud's disease or obliterative arterial conditions, for the alleviation of arterial hypertension, and, less frequently, for the relief of pain in causalgia and for the elimination of excessive sweating.

An apparently adequate sympathectomy may fail to relieve the symptoms for which it was performed, either immediately or after a prolonged interval. Reinnervation or regrowth of severed sympathetic nerve fibers has been postulated as the cause of failure in the second instance. However, another cause of failure is due to the presence of aberrant sympathetic ganglia which do not synapse in the sympathetic ganglionic chain and are therefore preserved following technically adequate sympathectomies.

Case Report

A 34-year-old white man was admitted to the neurosurgical section on February 20, 1967. The patient had sustained a spinal cord injury at T-10 in an automobile accident in August, 1959. Shortly after the accident, the patient developed marked sweating in the lower abdomen, groin, and legs together with gradually increasing mass reflex spasticity. An immediate decompressive laminectomy of T-9 and T-10 had produced no improvement in the pre-existing total cord lesion. In July, 1960, posterior rhizotomies, left T-8 to T-10 and right T-9, were followed by relief of radicular pain. The spinal cord appeared pale and narrow at the site of the injury.

On February 10 and 17, 1967, subarachnoid lumbar phenol (5%) instillations afforded moderate relief of the spasticity in both legs but had no effect on bladder and bowel function, nor did they improve the distressing hyperhidrosis which was most marked in areas corresponding to the T-12 and L-3 dermatomes. The patient had marked excoriations of the skin in both groins which resisted all medical treatment.

Examination. On February 20, 1967, the patient showed anesthesia and motor paralysis below the tenth thoracic cord segment, with mild muscle spasticity in both legs. Urological evaluation revealed a hypertonic bladder with a low capacity and ureteral reflux.

First Operation. On February 27, a bilateral lumbar sympathectomy was performed. The sympathetic chain was resected for the length of four ganglia on the right and three ganglia on the left. This was confirmed by the histological examination of the surgical specimen. In spite of this, 3 hours postoperatively the patient showed excessive cold sweating in a restricted bilateral area below the level of the cord lesion, most marked from the 12th thoracic to the 3rd lumbar dermatomes. However, both legs were pink and warm. The sweating continued unabated, and an intraspinal procedure for the total interruption of the sympathetic outflow was decided upon.

Second Operation. On March 15, 1967, an additional laminectomy of T-11 to L-1 was performed and the lower thoracic cord from T-10 to the conus as well as the cauda equina exposed. A tentatively planned longitudinal cordotomy was abandoned because the spinal cord appeared to have a central cavity. Since the presence of a cystic central
cavity had previously been reported as a cause of failure of deafferentation\textsuperscript{17,18} in the alleviation of spasticity or rigidity, a conu-
sectomy was carried out instead. Immediately after the operation the legs appeared flaccid and the sweating disappeared. The skin excoriation in the groins healed sub-
sequently, and the patient has remained anhid-
rotic in the skin areas of previous hyperhid-
drosis. The bladder has been converted into a lower motor neuron type.

**Discussion**

The cause of failure of lumbar sympathecto-
tomy for the relief of excessive sweating is prob-
ably the lack of total sympathetic denervation of the sort that has been noted in sympathectomies undertaken for other rea-
sons. Although the presence of aberrant sympa-
thetic ganglia in the upper lumbar nerve roots, usually at the origin of the white ramus communicans, was demonstrated by Marinesco and Minea\textsuperscript{19} as early as 1908, their functional significance as the cause of failure of incomplete sympathetic denervation was not appreciated until much later. Hirt\textsuperscript{6} in 1921 named these aberrant ganglia “intermediate ganglia.” Their presence in the upper lumbar nerve roots was subsequently confirmed by Gruss\textsuperscript{2} and Wrete,\textsuperscript{21-23} who also found them in the lower roots of the brachial plexus. It was felt at that time that the intermediate ganglia had not migrated far enough from the neural crest and that their postganglionic fibers passed through the white rami to the ganglionic chain to join the main postganglionic fibers in their usual course through the gray rami communica-
tes.\textsuperscript{16}

Alexander, *et al.*,\textsuperscript{1} found the intermediate sympathetic ganglia in human material next to or imbedded in the white and gray rami, or in the spinal roots proper at the first and second thoracic and first and second lumbar levels. These small ganglia usually contained between 160 to 2000 cells, with an occasional large ganglion containing up to 20,000 cells. They also noted intercon-
necting sympathetic fibers between the nerve roots of T-11, T-12, and L-1 and the corre-
spanding nerve roots of T-12, L-1, and L-2 which did not pass through the ganglionic chain. Kirgis and Kuntz\textsuperscript{20} and Kuntz and Dillon\textsuperscript{12} demonstrated the presence of in-
termediate ganglia in the 8th cervical nerve root and rather infrequently in the 3rd lum-
bar nerve root. It was Alexander and his co-workers,\textsuperscript{1} however, who furnished the most significant anatomical proof that post-
ganglionic fibers of the intermediate ganglia did not traverse the ganglionic chain but proceeded directly through the segmental nerves to the periphery (Figs. 1 and 2).

In the light of these anatomical data, sub-
sequent clinical observations contributed greatly to a better understanding of the func-
tional significance and clinical importance of the aberrant sympathetic fibers. Ray and Console\textsuperscript{15} in 1948 demonstrated residual sympathetic innervation in each of 151 pa-
ients subjected to thoraco-lumbar sympa-
thectomy for the relief of hypertension, ei-
ther immediately or several weeks later. Sub-
sequent spinal anesthesia, paravertebral blocks, or anterior rhizotomy of the first to third nerve root would interrupt the remain-

![Fig. 1. Schematic longitudinal representation of sympathetic nervous system. The classically described pathways are on the left side; the pathways circumventing the paravertebral chain are on the right.](image-url)
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Fig. 2. Schematic transverse section of spinal cord, nerve roots, and ganglionic chain showing the classical and aberrant sympathetic outflow.

ing sympathetic pathways. Bridges and Yahr demonstrated sympathetic responses in the upper extremities during stimulation of the anterior first thoracic and 8th cervical nerve roots after previous cervico-thoracic sympathectomies. In two patients with Raynaud's disease who, after a prolonged interval following sympathectomy, showed recurrent symptoms, Bridges succeeded in eliminating the residual sympathetic innervation by anterior rhizotomy of C-8 and T-1.

We have had two failures of lumbar sympathectomy for the relief of hyperhidrosis that can only be explained by the existence of aberrant sympathetic ganglia in the segmental nerves. The first patient, observed previously by one of us (E.G.K.), had undergone two lumbar sympathectomies on the same side by an experienced general surgeon in another hospital without the slightest effect on the sweating in an amputation stump. The sweating was so severe that the patient was unable to wear a prosthesis. The administration of a ganglionic blocking agent (hexamethonium chloride) as a test totally eliminated the sweating temporarily. A subsequent unilateral anterior rhizotomy of T-12 to L-2 relieved the patient of the incapacitating hyperhidrosis permanently.

Our second such case is the one reported here; it suggests the same cause for failure of lumbar sympathectomy to eliminate excessive sweating. In this case a conusectomy was necessary to complete the therapeutic denervation.

It would be interesting to know whether a longitudinal cordotomy of the Bischof type would have been successful in eliminating the spastic phenomena including the hyperhidrosis in this patient. Our limited experience with the longitudinal myelotomy has convinced us of its efficacy in eliminating severe, incapacitating, painful spasticity with preservation of residual voluntary motor function. Although in our patient a myelotomy had been planned, the discovery of a fusiform central cystic cavity containing a clear fluid led us to abandon our plan in favor of a conusectomy, which had also been considered (Fig. 3). We were particularly influenced by Tarlov's observation of two comparable paraplegic patients with cystic cavities in the conus who failed to respond to deafferentation by bilateral posterior rhizotomy.

The immediate failure of sympathectomy has generally been explained on the basis of anatomical data and clinical observations as being due to incomplete interruption of the classical sympathetic pathways, either because of anatomical variations or because of technical, surgical considerations. However, the underlying mechanism for the recurrence of sympathetic functions after originally satisfactory sympathectomies is even more difficult to explain. There may be individual differences in the relative abundance of aberrant sympathetic ganglia. In cases of great paucity of these elements it might take time for these few ganglia to compensate functionally for the overwhelming loss of the predominant ganglia in the ganglionic chain.
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Fig. 3. Cross sections of conusectomy specimen from T-10 to S-1 demonstrating posttraumatic hydromyelia.

Also, the presence of a greater number of intermediate sympathetic ganglia in the cervico-thoracic area may account for the fact that lumbar sympathectomies are, in general, more satisfactory clinically than thoraco-cervical procedures.20

Therefore, to assure total sympathetic denervation of the leg, it appears necessary to section the anterior roots of T-12 to L-2, and in some instances also of either T-11 and L-3 if the lumbar sympathectomy fails. This can be done with a relatively acceptable functional impairment. However, total sympathetic denervation of the arm by anterior rhizotomy of C-8 and T-1 can result in a prohibitively severe loss of essential hand motor function, and in this region it should therefore be restricted to exceptional cases.

Summary

We have described a patient with pathologic hyperhidrosis who did not respond to a lumbar sympathectomy but obtained complete denervation following a subsequent rhizotomy. We have emphasized the related role of aberrant or intermediate ganglia which do not send postganglionic fibers through the ganglionic chain but directly through the segmental nerves, and believe the variable results after sympathectomy can be explained by individual differences in the presence or number of intermediate ganglia.

References


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