Failure of Lumbar Sympathectomy in the Relief of Hyperhidrosis

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Stimulation 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, vasodilatation, 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 obliterator 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 re-growth 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 reflex.

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 conunsectomy was carried out instead. Immediately after the operation the legs appeared flaccid and the sweating disappeared. The skin excoriation in the groins healed subsequently, and the patient has remained anhidrotic in the skin areas of previous hyperhidrosis. The bladder has been converted into a lower motor neuron type.

**Discussion**

The cause of failure of lumbar sympathectomy for the relief of excessive sweating is probably the lack of total sympathetic denervation of the sort that has been noted in sympathectomies undertaken for other reasons. Although the presence of aberrant sympathetic 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{10} in 1921 named these aberrant ganglia "intermediate ganglia." Their presence in the upper lumbar nerve roots was subsequently confirmed by Gruss\textsuperscript{11} 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 communicantes.\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 contained between 160 to 2000 cells, with an occasional large ganglion containing up to 20,000 cells. They also noted interconnecting sympathetic fibers between the nerve roots of T-11, T-12, and L-1 and the corresponding 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 intermediate ganglia in the 8th cervical nerve root and rather infrequently in the 3rd lumbar nerve root. It was Alexander and his co-workers,\textsuperscript{1} however, who furnished the most significant anatomical proof that postganglionic 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, subsequent clinical observations contributed greatly to a better understanding of the functional significance and clinical importance of the aberrant sympathetic fibers. Ray and Console\textsuperscript{15} in 1948 demonstrated residual sympathetic innervation in each of 151 patients subjected to thoraco-lumbar sympathectomy for the relief of hypertension, either immediately or several weeks later. Subsequent 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)