Functional Anatomy of the Cervical Sympathetic Trunk

To the Editor:—I would comment on the clinical report of Drs. Jaffe and McLeskey concerning position-induced Horner’s syndrome. The rarer the complication the more elusive the explanation so there are both alternative possibilities and suggestions for further study in this matter.

Contrary to the statement in the discussion, sympathetic innervation of the head does not arise from the superior cervical ganglion alone. Rather, preganglionic nerves going to the head arise approximately in the first and second thoracic spinal segments, traverse the combined first thoracic-inferior cervical ganglion (stellate) to synapse with postganglionic fibers in the middle and superior cervical ganglia, largely the latter.

Jaffe and McLeskey quote the “work” of Nicholson and McAlpine to explain why stretch of the cervical sympathetic trunk might have caused temporary paralysis. Actually, the latter merely cite Seddon and Denny-Brown for experiments done in connection with nerve injury. Notably, both investigators examined somatic motor nerves and both reiterate, according to the classic investigations of Gasser and Erlanger, that large-diameter myelinated fibers are far more susceptible than the smaller, unmyelinated variety to damage either by compression or stretch. Accordingly, Seddon coined the term neuropraxia to account for first-degree injury and transient dysfunction, in contrast to second-degree (axonotmesis) and third-degree (neurotmesis) lesions where paralysis is prolonged owing to crushing or section, both followed by Wallerian degeneration. Neither Seddon nor Denny-Brown described the histologic changes in transient paralysis as quoted by Jaffe and McLeskey. Furthermore, it is unlikely that unmyelinated, gray postganglionic fibers would show such change. As the sympathetic trunk lies anterior to the cervical vertebral transverse processes, the gross diagram of a torso (fig. 1 of the article) does little to abet the concept of stretch injury. The writers did attempt to support the head properly.

Ischemia secondary to vascular embarrassment might have played a role as stellate ganglion and cervical trunk are nourished by vessels in the vicinity—thyrocervical trunk of the subclavian, first intercostal and vertebral artery. Did the patient’s pulse weaken in the lateral decubitus? Were blood pressures equal in both arms? A bruise found in the carotid? Does the chest x-ray of this 71-year-old woman reveal vessel calcification, evidence of a superior thoracic outlet syndrome (Pancoast) or cervical arthritis? At the time there were signs of sympathetic blockage or dysesthesia over the first and second brachial dermatomes? In brachial plexus injury involving the first and second intercostal nerves, Horner’s syndrome may evolve.

While the authors may be correct in their surmise, the supporting evidence is weak. Unable to translate from the German, I could not ascertain whether the only other report of this complication provides better support for the “stretch” etiology.

LEROY D. VANDAM, M.D.
Professor of Anaesthesia Emeritus
Harvard Medical School
Department of Anesthesiology
Brigham and Women's Hospital
75 Francis Street
Boston, Massachusetts 02115

REFERENCES


(Accepted for publication February 2, 1982.)

In reply:—We would like to thank Dr. Vandam for his comments of clarification and historical interest. We were indeed in error when we stated that sympathetic innervation to the head arises from the superior cervical ganglion. It was our intent to imply that most sympathetic fibers innervating the head must first traverse the superior cervical ganglion where they may be susceptible to injury.

Dr. Vandam states that we misquoted “work” of Seddon and Denny-Brown, summarized by Nicholson and...