Anesthesiology
57:139, 1982

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.[1] 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.

Jaffee and McLeskey quote the "work" of Nicholson and McAlpine[2] to explain why stretch of the cervical sympathetic trunk might have caused temporary paralysis. Actually, the latter merely cite Seddon[3] and Denny-Brown[4] 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 (neurotomesis) lesions where paralysis is prolonged owing to crush 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 bruit 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 were there 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[5] provides better support for the "stretch" etiology.

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

REFERENCES

1. Jaffe TB, McLeskey CH: Position-induced Horner's syndrome. ANESTHESIOLOGY 56:49–50, 1982
2. Nicholson MJ, McAlpine FS: Neural injuries associated with surgical positions and operations, Positioning in Anesthesia and Surgery. Edited by Martin JT. Philadelphia, WB Saunders, 1978, pp 213–216
3. Seddon HJ: Three types of nerve injury. Brain 66:237–288, 1943
4. Denny-Brown D, Doherty MM: Effects of transient stretch or compression of peripheral nerve. Arch Neurol Psychiatr 54:116–129, 1945
5. Gerber H, Maar K: Horner's syndrome. Anaesthetist 26:357–358, 1977

(Accepted for publication February 2, 1982.)

Anesthesiology
57:139–140, 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

McAlpine[1] concerning histologic changes in transient paralysis. Instead of paraphrasing, let us this time quote the original source. "The effects of stretching a peripheral nerve beyond the limit of physiologic elasticity . . . {produce} damage to epineurial vessels, with resultant patches of ischemic changes in nerve fibers."[2] Granted these observations were made on somatic fibers, but we theorize a milder form of this same stretch injury may explain the transient postoperative sympathetic dysfunction observed in our patient.

According to Dr. Vandam, our "gross diagram of a torso" did not illustrate the anterior relation of the cervical sympathetic trunk to cervical transverse processes. However, we felt it adequate to illustrate the concept of misalignment of the thoracic and cervical vertebrae (which was its primary purpose). Figure 1 illustrates the cervical sympathetic chain in greater detail, further demonstrating how it may be stretched from inadequate head support in a patient placed in the lateral position.

In answer to raised questions, intraoperative blood pressures were monitored via a right radial artery catheter and a left arm blood pressure cuff. Both pressures were approximately equal and unchanged in the lateral position. Blood pressure remained within 20 per cent of control throughout the entire anesthetic. When Horner's syndrome was observed postoperatively, there were no signs or symptoms to suggest a brachial plexus insult. No carotid bruits were auscultated, although occult disease may have existed. Chest x-ray did not demonstrate evidence of Pancoast tumor or cervical rib but did reveal lumbar and thoracic spine arthritis. It was on this basis that we assumed cervical vertebral arthritis and/or spurring may have existed.

Although stretch of the cervical sympathetic chain is unusual, in this case, we are still unable to conjure any other diagnosis to better explain the findings. The need for strict attention to spinal alignment in patients placed in the lateral position remains essential.

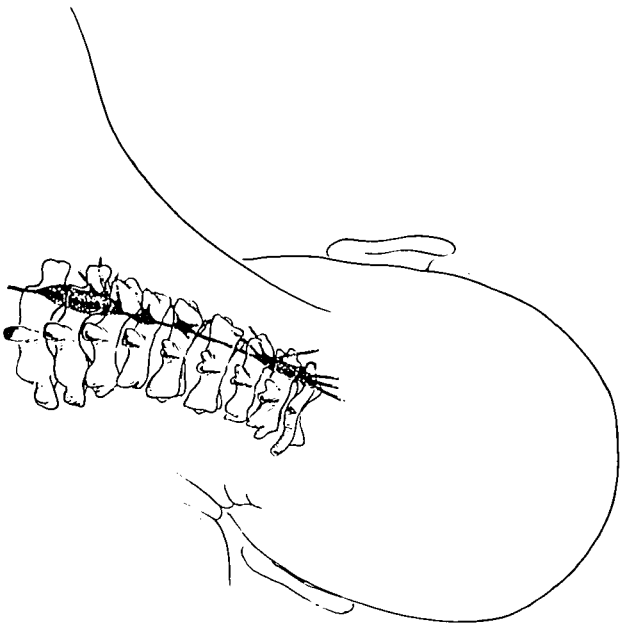

FIG. 1. Cervical sympathetic chain.

CHARLES H. MCLESKEY, M.D.
Assistant Professor

TODD B. JAFFE, M.D.
Fellow

Department of Anesthesia
Bowman Gray School of Medicine
Wake Forest University
Winston-Salem, North Carolina

REFERENCES

1. Nicholson MJ, McAlpine FS: Neural injuries associated with surgical positions and operations, Positioning in Anesthesia and Surgery. Edited by Martin JT. Philadelphia, WB Saunders, 1978, pp 213–216
2. Denny-Brown D, Doherty MM: Effects of transient stretching of peripheral nerve. Arch Neurol Psychiatr 54:116–129, 1945

(Accepted for publication February 23, 1982.)

Anesthesiology
57:140–141, 1982

Further Considerations Regarding the Components of an Effective Test Dose Prior to Epidural Block

To the Editor:—In the recent clinical report by Moore and Batra,[1] it was stated that the purpose of the study was to determine the components of a "*single*" test dose of a local anesthetic solution which, within two minutes from its injection, would produce clinical evidence that the needle has penetrated either a blood vessel or the dura. In the report, it was shown that the addition of epinephrine 1:200,000 to 3 ml of local anesthetic produces a clinically detectable transient tachycardia if injected into a vein. However, the effect of a subarachnoid injection of 3 ml of local anesthetic was not studied. Three ml of 3 per cent chloroprocaine (90 mg) should produce an immediate block,[2] as also should 3 ml of 0.75 per cent bupivacaine (22.5 mg)[3] but the level of anesthesia might reach the upper thoracic dermatomes.[4] However, neither 3 ml of 1.5 per cent mepivacaine nor