

18. Mollison PL: The investigation of haemolytic transfusion reactions. *Br Med J* 1:529-532, 559-561, 1943
19. Braude AI, Sanford JP, Bartlett JE, Mallery OT, Jr: Effect and clinical significance of bacterial contaminants in transfused blood. *J Lab Clin Med* 39:902-916, 1952
20. Elin RJ, Lundberg WB, Schmidt PJ: Evaluation of bacterial contamination in blood processing. *Transfusion* 15:260-265, 1975
21. Pittman M: A study of bacteria implicated in transfusion reactions and of bacteria isolated from blood products. *J Lab Clin Med* 42:273-288, 1953
22. Stenhouse MAE, Milner LV: *Yersinia enterocolitica*: A hazard in blood transfusion. *Transfusion* 22:396-398, 1982.
23. Wetterlow LH, Kay FH, Edsall G: Missed contaminations in biological products: The role of psychrophilic bacteria. *J Lab Clin Med* 43:411-421, 1954
24. Denlinger JK, Nahrwold ML, Gibbs PS, Lecky JH: Hypocalcaemia during rapid blood transfusion in anaesthetized man. *Br J Anaesth* 48:995-999, 1976
25. Stoelting RK: *Pharmacology and Physiology in Anesthetic Practice*. Philadelphia, JB Lippincott, 1987, pp 342-343
26. Rock G, Labow RS, Franklin C, Burnett R, Tocchi M: Hypotension and cardiac arrest in rats after infusion of mono(2-ethylhexyl)phthalate (MEHP), a contaminant of stored blood (letter). *New Engl J Med* 316:1218-1219, 1987
27. Gullbring B: The use of plastics in blood transfusion equipment with special regard to toxicity problems (editorial). *Vox Sang* 9:513-529, 1964
28. Nicholls A: Ethylene oxide and anaphylaxis during hemodialysis (editorial). *Br Med J* 292:1221-1222, 1986
29. Leitman SF, Boltansky H, Alter HJ, Pearson FC, Kaliner MA: Allergic reactions in healthy plateletpheresis donors caused by sensitization to ethylene oxide gas. *N Engl J Med* 315:1192-1196, 1986
30. Vermeylen J, Janssens S, Ceuppens J, Vermeylen C: Transfusion reactions in haemophiliac caused by sensitisation to ethylene oxide (letter). *Lancet* 1:594, 1988
31. Morse AN, Churchill WH, Moore FD, Jr: Complement activation during blood donation. *Transplant Proc* 20:1175-1179, 1988
32. Seltzer JL, Goldberg ME, Larijani GE, Ritter DE, Starsnic MA, Stahl GL, Lefer AM: Prostacyclin mediation of vasodilation following mesenteric traction. *ANESTHESIOLOGY* 68:514-518, 1988
33. Stoelting RK, Dierdorf SF, McCammon RL: *Anesthesia and Co-Existing Disease*. New York, Churchill Livingstone, 1988, pp 284-286
34. Schmidt PJ, Keyv SV: Air embolism: A hazard during phlebotomy. *New Engl J Med* 258:424-427, 1958
35. Shehadi WH, Toniolo G: Adverse reactions to contrast media. *Radiology* 137:299-302, 1980
36. Covino BG: *Clinical pharmacology of local anesthetic agents, Neural Blockade in Clinical Anesthesia and Management of Pain*. Edited by Cousins MJ, Bridenbaugh PO. Philadelphia, JB Lippincott, 1988, pp 111-144
37. Whitwam JG, Russell WJ: The acute cardiovascular changes and adrenergic blockade by droperidol in man. *Br J Anaesth* 43: 581-591, 1971
38. Office of Medical Applications of Research, National Institutes of Health: Perioperative red cell transfusion. *JAMA* 260:2700-2703, 1988

Anesthesiology
74:628-630, 1991

Transient Paraplegia during Posterior Cervical Osteotomy

JOHN C. DRUMMOND, M.D., F.R.C.P.C.,* J.J. ABITBOL, M.D., F.R.C.S.C.,†
T. J. SANFORD, M.D.,‡ S. R. GARFIN, M.D.§

A procedure for correction of severe cervical kyphosis performed under local anesthesia provided an opportunity for unique observations regarding the progression of evoked response changes and physical signs and symptoms during the evolution and regression of paraplegia.

* Staff Anesthesiologist, VA Medical Center, San Diego; Associate Professor of Anesthesiology, University of California, San Diego.

† Assistant Professor of Surgery, University of California, San Diego.

‡ Staff Anesthesiologist, VA Medical Center, San Diego; Clinical Professor of Anesthesiology, University of California, San Diego.

§ Professor of Surgery, University of California, San Diego.

Received from the Department of Anesthesia and the Department of Surgery (Orthopedics) of the VA Medical Center, San Diego, and the University of California, San Diego. Accepted for publication November 19, 1990. Performed at the VA Medical Center, San Diego.

Address reprint requests to Dr. Drummond: University of California—San Diego, Mailstop 0629, La Jolla, California 92093-0629.

Key words: Anesthetic techniques: monitored anesthesia care. Monitoring: evoked potential. Surgery: cervical osteotomy.

CASE REPORT

The patient was a 62-yr-old man with ankylosing spondylitis and a severe cervical kyphosis. He was scheduled for posterior cervical osteotomy and realignment of the neck. Six months prior to the time of surgery, he had sustained a fracture at C7 in association with a fall. That injury resulted in an aggravation of the kyphosis and in numbness and decreased sensation in a right C6 distribution. He was otherwise neurologically intact. The cervical kyphosis was estimated to be 90° (chin-brow angle) and was such that his chin rested continually on his sternum. Mouth opening was limited and his ambulation was impaired because he was unable to look forward. His only regular medication was baclofen. He had last undergone a general anesthetic 6 yr prior to the current procedure. That anesthetic was reported by the patient to have been uneventful, and he could not recall the details of airway management. The anesthetic and surgical plan was to perform the procedure under local anesthesia and to be prepared to induce a brief period of general anesthesia if the realignment maneuver (which entails elective fracturing of the heavily calcified anterior longitudinal ligament) was not tolerated in the awake state. Somatosensory evoked response (SSER) monitoring was to be performed in order to permit

evaluation of the spinal cord during periods in which the patient could not be examined neurologically.

The patient was brought unpremedicated to the operating room. A peripheral intravenous catheter was inserted. Alfentanil and midazolam were administered in small increments to provide sedation consistent with prompt patient responsiveness. A "halo" fixation device was placed after infiltration of the pin sites with 0.25% bupivacaine. Topical anesthesia of the airway was established with a combination of lidocaine ointment and 4% lidocaine administered by atomizer.

A fiberoptic bronchoscope was passed through the nose into the midtrachea. An endotracheal tube was passed over the bronchoscope. A precordial Doppler was placed.

Prior to the passage of the endotracheal tube, a system of hand signals was established. "Thumbs-up" by the patient was to be used to indicate that the patient was in no distress. "Thumbs-down" was to be used to indicate that there was a problem, which would be identified by suggestion and hand signal response. The patient was placed in the sitting position in a barber-type chair. The chair had arm rests and back support up to the level of the superior edge of the scapulae. The patient's head was supported from the ceiling by a rope and pulley system. The rope was attached to the halo, and the pulley was attached to the ceiling over the chair.

The patient was prepared for recording of SSERs. Platinum needle electrodes were placed over the posterior tibial nerves at the ankle. Gold disk recording electrodes (Grass) were placed at CZ' and FPz with a ground electrode over the left mastoid process. Recording electrode impedances were maintained at ≤ 3 kOhm. Stimulation and recording were performed with a Nicolet CA 2000 (Nicolet Biomedical, Madison, WI). Stimulus intensity was adjusted to two times motor threshold. The low and high filters were set at 30 and 500 Hz, respectively. For each SSER, 150 constant current impulses of 100 μ s duration, delivered bilaterally at a rate of 3.1 Hz were averaged. Selected waveforms were recorded on magnetic disc and on paper.

After skin preparation, the skin, fascia and muscle overlying C4-T2 were infiltrated with 0.25% bupivacaine with 1:200,000 epinephrine. The operation proceeded uneventfully. Wide laminectomies were performed from C5 to T1. Additional midazolam, 1 mg, and alfentanil, 250 μ g, were administered. After additional bupivacaine infiltration, posterior osteotomies between C7 and T1 were undertaken. This involved excision of the fused facet joints and undercutting of the adjacent pedicles (the latter to prevent entrapment of the C8 nerve roots in association with realignment). The patient repeatedly gave a thumbs-up sign in response to inquiries as to his well-being. Evoked responses were recorded at intervals of approximately 5 min. Toward the con-

clusion of the phase of the resection of the facet joints, the evoked response to posterior tibial nerve stimulation was noted to be absent. The patient again gave the thumbs-up sign in response to an inquiry as to the presence of any discomfort or distress.

The patient was asked to move his lower extremities. He was unable to move his right leg, and there was a flicker of antigravity movement in the quadriceps mechanism at the left knee. The patient denied pain. Repeat neurologic assessment approximately 1 min later revealed total flaccidity and absence of voluntary movement in both lower extremities. Grip strength in the right arm was noted to be diminished. The right arm weakness appeared to be progressive over the ensuing 2-3 min. The surgeon observed that the widely exposed dura, which previously had been pulsatile, was now without obvious pulsation.

At this point, it was appreciated that the barber chair, which had been relocated early in the procedure to achieve better illumination of the surgical field, was oriented such that the supporting rope between the halo system and the roof mounted pulley was approximately 25° off the vertical. The immediate suspicion was that the patient's head had been falling forward progressively during the resection of the facet joints. The surgeon, who was positioned behind the patient, gripped the halo and extended the neck 15-20°. Spontaneous pulsation of the dura overlying the cervical cord was immediately noted. Within 3-5 min, the patient again had antigravity strength of the lower extremities, and an evoked response waveform was identifiable. By 10 min after the repositioning of the neck, the patient's lower extremity strength and evoked responses were indistinguishable from their postintubation baseline status. Minimal right arm weakness appeared to persist. The patient denied pain or dysesthesia.

The barber chair was repositioned and the roof support rope was tightened. After completion of the resection of the facet joints, and during repeated SSER evaluations and gross motor examinations, the surgeon performed a forced extension of the cervical spine of approximately 30°. This was accomplished without neurologic or electrophysiologic change. The wound was closed, and the halo was secured to the prefabricated jacket and vertical struts.

The patient was taken to the postanesthesia care unit (PACU) with the nasotracheal tube in place. Approximately 1 h later, after verification of constant normal neurologic function in the lower extremities, the trachea was extubated. Repeat neurologic examinations over the ensuing days confirmed persistent normal lower extremity motor function. There was mild, persistent, weakness of the right wrist extensors and of the intrinsic muscles of both hands, the right greater than the left. These were believed to be due to nerve root compression. Two weeks later the patient underwent an anterior interbody fusion with iliac crest autograft. The upper extremity weakness persisted despite a solid fusion. Therefore, 4 months later, he underwent posterior C7, C8, and T1 foramenotomies, after which his motor function returned to normal.

DISCUSSION

Typically, procedures of this nature have been undertaken during anesthesia by local infiltration,^{1,2} although the use of general anesthesia has been reported.³ The practice of performing the operation with local anesthesia has evolved because the procedure entails a substantial risk of neurologic injury. The spinal column is surgically destabilized by laminectomy and excision of the facet joints, after which the anterior longitudinal ligament is electively fractured. Our experience confirms the neurologic hazard inherent to this procedure. Although local anesthesia can be readily justified, it has been the experience of some surgeons that at least a brief period of

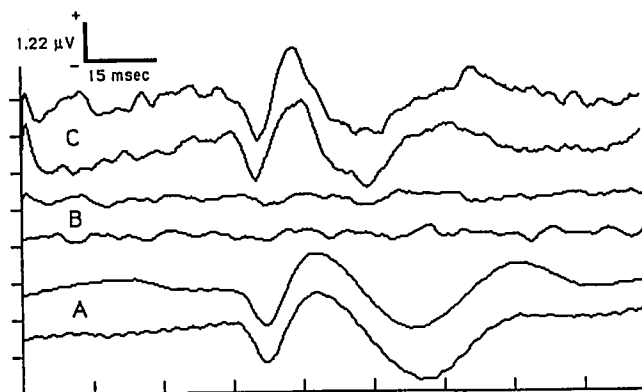


FIG. 1. Cortical somatosensory evoked responses to bilateral posterior tibial nerve stimulation recorded shortly before (A), during (B), and approximately 10 min after (C) resolution of the episode of paraplegia.

unconsciousness may be required because of pain associated with the fracturing of the anterior longitudinal ligament. In some instances this has been accomplished by administration of fixed agents and/or administration of inhaled agents by mask.^{1,2}

In the present case, because of the extreme flexion of the upper airway, there was reluctance to accept the possibility of any period of reduced airway reflexes because of the apparent impossibility of airway management *via* mask. Accordingly, awake bronchoscopic intubation was performed to guarantee the airway under any circumstances. The patient was, in fact, extremely tolerant of surgical manipulation under local anesthesia, and no period of insensibility was required. We nonetheless would advocate this approach on a subsequent exposure to a similar clinical circumstance. We anticipate that management, *via* mask, of airways such as that initially present in our patient would be extremely difficult, and might also be neurologically hazardous in a patient with a surgically destabilized neck. Our group has subsequently undertaken this procedure using halo-jacket fixation with strut loosening and adjustment at the time of the cervical realignment maneuver. The halo jacket apparatus further reduces neck mobility and makes initial elective tracheal intubation even more appropriate.

There were additional lessons learned from the current case. It had been our assumption at the outset that acute cord compression would be a symptomatic event. Although this probably is the case with abrupt cord compression, the current experience suggests that when the onset of the compressive event is sufficiently gradual, it may be unaccompanied by symptomatology. Immediately upon extubation, the current patient was interviewed in the PACU with respect to his recollection of the episode of paraplegia. Despite the midazolam employed for sedation, he had a very clear recollection of his inability to move his lower extremities and was unequivocal in his statement that, prior to being asked to move voluntarily, he was unaware that anything was amiss. Pain, other than some discomfort at the surgical site, was never a feature of the onset or the regression of his neurologic dysfunction.

The current case also provided a rare opportunity to observe the correlation between the onset and regression of paraplegia and the onset and regression of evoked response changes. The interval between the last normal neurologic evaluation and the detection of the evoked response loss was approximately 5 min. We do not know precisely when within that 5-min interval gross neurologic dysfunction would have been detectable had the patient been examined more frequently. However, the temporal relation between loss of neurologic function and the loss of the evoked response was inevitably close (less than 5 min). After repositioning of the head, the neurologic and evoked response examinations were performed at shorter intervals (less than 2 min), and the recovery of the two functions paralleled one another very closely.

In summary, we observed the onset and regression of a transient paraplegia during an episode of cervical cord compression during an extensive cervical laminectomy. The case demonstrated that the onset of paraplegia due to cord compression can occur in an awake subject without conspicuous symptomatology. It points out clearly the need for repeated neurologic examination in such circumstances. In addition, the episode confirms the utility of evoked response monitoring in situations in which cord compression may occur. In this instance, the evoked responses notified the attending physicians of cord compression at an interval such that measures could be taken to correct the situation prior to the insult's becoming irreversible. The current observations confirm that it is not inevitably "too late" by the time changes in SSERs provide evidence of cord compromise.

REFERENCES

1. Simmons EH: Surgery of the spine in rheumatoid arthritis and ankylosing spondylitis, Surgery of the musculoskeletal system, Volume 4. Edited by Evarts CM. New York, Churchill Livingstone, 1983, pp 4:85-4:151
2. Chang J: Anaesthesia for cervical osteotomy. *Can Anaesth Soc J* 21:83-91, 1974
3. Harris AG, Heron JS, Renwick WA: Anaesthesia for posterior cervical osteotomy. *Can Anaesth Soc J* 22:84-90, 1975