References

1. Ross S, Scarborough CD: Total spinal anesthesia following brachial plexus block. ANESTHESIOLOGY 1973; 39:458–60

2. DeSarram M: Accidental total spinal analgesia. Anaesthesia 1956; 11:77–79

3. Gupta A, Sjoberg F, Bengtsson M: Spinal anaesthesia for caesarean section following epidural analgesia in labour: A relative contraindication. Int J Obstet Anaesth 1994; 3:153–6

4. Leivers D: Total spinal anesthesia following early prophylactic epidural blood patch. 1990; 73:1287–9

5. Mets B, Broccoli E, Brown AR: Is spinal anesthesia after failed epidural anesthesia contraindicated for cesarean section? Anesth Analg 1993; 77:629–31

6. Gupta A: Spinal anesthesia after failed epidural anesthesia (letter). Anesth Analg 1996; 82:214

7. Waters J, Leivers D, Hullander M: Response to spinal anesthesia after inadequate epidural anesthesia (letter). Anesth Analg 1994; 78: 1033–4

8. Beck GN, Griffiths AG: Failed extradural anaesthesia for caesarean section. Complication of subsequent spinal block. Anaesthesia 1992; 47:690–2

9. Mets B: Spinal anesthesia after failed epidural anesthesia (response letter). Anesth Analg 1996; 82:214

10. Johnson MD, Burger GA, Mushlin PS, Arthur GR, Datta S: Reversal of bupivacaine epidural anesthesia by intermittent epidural injections of crystalloid solutions. Anesth Analg 1990; 70:395–9

11. Lubenow T, Keh-Wong E, Kristof K, Ivankovich O, Ivankovich AD: Inadvertent Subdural Injection: A Complication of an Epidural Block.Anesth Analg 1988; 67:175–9

12. Hodgkinson R: Total spinal block after epidural injection into an interspace adjacent to an inadvertent dural perforation. ANESTHESIOLOGY 1981; 55:593–4

13. Bernards CM, Kopacz DJ, Michel MZ: Effect of needle puncture on morphine and lidocaine flux through the spinal meninges of the monkey *in vitro*. *ANESTHESIOLOGY 1994; 80:853–8*

14. Bromage PR: Mechanism of action, Epidural Analgesia. Philadelphia, W. B. Saunders, 1978, pp 119–59

15. Bromage PR, Joyal AC, Binney JC: Local anesthetic drugs: Penetration from the spinal extradural space into the neuraxis. Science 1963; 140:392–4

16. Martins AN, Wiley JK, Myers PW: Dynamics of the cerebrospinal fluid and the spinal dura mater. J Neurol Neurosurg Psych 1972; 35:468–73

17. Blumgart CH, Ryall D, Dennison B, Thompson-Hill LM: Mechanism of extension of spinal anesthesia by extradural injection of local anesthetic. Br J Anaesth 1992; 69:457–60

18. Takiguchi T, Okano T, Egawa H, Okubo Y, Saito Y, Kitajilma T: The effect of epidural saline injection on analgesic level during combined spinal and epidural anesthesia assessed clinically and myelographically. Anesth Analg 1997; 85:1097–100

19. Stienstra R, Dahan A, Alhadi BZ, van Kleef JW, Burm AF: Mechanism of action of an epidural top-up in combined spinal epidural anesthesia. Anesth Analg 1996; 83:382–6

Anesthesiology
1998; 89:1270–2

Evoked Potential Monitoring and EKG: A Case of a Serious "Cardiac Arrhythmia"

Carol Ann B. Diachun, M.D., John G. Brock-Utne, M.D., Ph.D., F.F.A. (S.A.),† Jaime R. López, M.D.,‡ Jo Ann Ceranski, R.E.E.G.T.§*

INTRAOPERATIVE neurophysiologic monitoring of spontaneous or evoked neural activity has become an important aspect of neuroanesthesia care. Evoked potentials are useful because they monitor the functional integrity of specific neural pathways that may be at risk for injury during surgery[1]; and, thus, are commonly used in many centers to monitor cerebral and spinal cord function during intracranial and spinal instrumentation procedures. Sensory evoked potentials are elicited using various stimulation techniques. Somatosensory evoked potentials (SSEP) can be obtained by electrical stimulation of a peripheral nerve; whereas, dermatomal sensory evoked potentials (DSEP) necessitate cutaneous stimulation of a specific dermatome. We report an occurrence of dermatomal evoked-potential monitoring causing what appeared

* Clinical Instructor of Anesthesia.

† Professor of Anesthesia.

‡ Assistant Professor of Neurology.

§ Neurodiagnostic Technologist.

Received from the Stanford University Medical Center, Stanford, California. Submitted for publication February 16, 1998. Accepted for publication July 6, 1998.

Address reprint requests to Dr. Diachun: Department of Anesthesia, Room H3580, Stanford University Medical Center, Stanford, California 94305-5115. Address electronic mail to: diachun1@leland.stanford.edu.

Key words: Artifacts; dermatomal evoked potential monitoring.

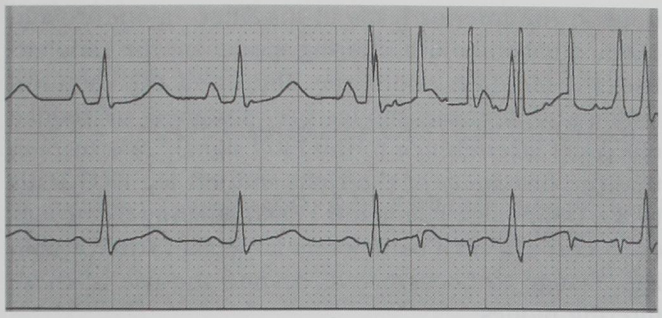

Fig. 1. Electrocardiography (EKG) changes observed in lead II (top) 45 min after incision. Is this a sudden onset of supraventricular tachycardia or interference from the dermatomal-sensory-evoked-potential monitoring? The EKG trace in lead V_5 (bottom) shows the more frequent interference pattern we observed during previous somatosensory-evoked-potential monitoring.

to be a serious cardiac arrhythmia, as seen during electrocardiography (EKG).

Case Report

A 39-yr-old man (American Society of Anesthesiologists status 3) with a diagnosis of a chronic pain syndrome involving the right side of the chest, as a result of an industrial accident, was scheduled for a dorsal root entry zone lesion placement at right T_{10}, T_{11}, and T_{12}. Adequacy of the dorsal root entry zone lesion was assessed using DSEP monitoring (Viking IV, Nicolet, Madison, WI). Relevant medical history included multiple back surgical procedures and obesity. Anesthesia was induced after placement of standard monitors, and the patient was placed in the prone position without incident. Using the Hewlett-Packard (Palo Alto, CA) Component Monitoring System, model M10948, in the EKG monitor-bandwidth filtering mode, EKG skin electrodes from 3M Health Care Red Dot, model 2238 (St. Paul, MN) were placed as follows: right arm lead (white) at the right posterior shoulder, left arm lead (black) at the left posterior shoulder, right leg lead (green) at the right mid-axillary line at the sixth intercostal space, left leg lead (red) at the left midaxillary line at the eighth intercostal space, and the V_5 lead (brown) at the left mid-axillary line at the fifth intercostal space. Bipolar evoked potential stimulator surface skin electrodes were placed at the right T_8, T_{10}, T_{12} and left T_{10} dermatomes at the mid-axillary line. Stimulation parameters were 18.5 mA with a duration of 0.2 milliseconds and a rate of 4.7 Hz.

Forty-five minutes after surgery began, EKG changes consistent with supraventricular tachycardia at a rate of 282 beats/min were observed (lead II in fig. 1); however, the patient's pulse by palpation and as measured by pulse oximetry was 74 beats/min and regular. At the same time, the patient's oxygen saturation was 100%, and blood pressure was unchanged at 110/70 mmHg. A search for possible causes commenced, but within 60 s, the EKG returned to normal sinus rhythm at a rate of 74 beats/min. Patient vital signs remained stable. A few minutes later, the phenomenon reappeared and again lasted less than 1 min. The first episode occurred with excision of bone, which could have caused a catecholamine surge in a lightly anesthetized patient, stimulating cardiac arrhythmia; but the second episode occurred during positioning of the operating microscope with little-to-no surgical stimulation. Venous blood provided normal electrolyte and blood gas values. Throughout these events, the surgeon was not using electrocautery, a common cause of artifact on the EKG.[1] A few minutes later, the phenomena reoccurred and, again, vital signs were stable. Suspecting a temporal relationship between the apparent cardiac arrhythmia and the DSEP electrical stimulation, we requested that the neurologist stimulate each dermatomal site. The supraventricular tachycardia-like changes on the EKG appeared to occur during stimulation of dermatomes T_{10} on the left and T_8 on the right. Throughout the procedure, this EKG interference pattern appeared in the filter-, monitor- and diagnostic-bandwidth filtering modes on our Hewlett-Packard monitor whenever the neurologist stimulated the left T_{10}, the right T_8 dermatomes, or both. Importantly, stimulation of the right T_{10} and T_{12} dermatomes did not result in these EKG changes in any filtering mode. Patient vital signs remained stable throughout surgery and the procedure was completed uneventfully. The patient awakened from anesthesia without problems.

Discussion

The electrocardiogram is a graphic representation of the skin surface voltage changes. These voltage changes reflect the electrical current produced during cardiac depolarization and repolarization. EKG interference patterns occur even with signal filtering.[1] Electrocautery use is the most common source of EKG signal perturbation[1]; but other causes are reported. For example, Kimberly *et al.*[2] and Sliwa and Marinko[3] describe EKG artifact resulting from transcutaneous nerve stimulators with electrodes placed parasternally and at the lower thoracic and lumbar regions. Russell[4] describes tachyarrhythmic-appearing EKG artifact resulting from neurogenic motor evoked potentials when cervical or high thoracic stimulation is used. Legatt and Frost[5] report large EKG artifact on the Datascope 2000 monitor (Datascope, Paramus, NJ) resulting from SSEP electrical stimulation.[5] Transcutaneous nerve stimulators units and evoked-potential monitoring produce an electrical current that can be recorded *via* the EKG skin electrodes, resulting in artifact. This case is that of dermatomal evoked-potential monitoring mimicking serious arrhythmia during EKG.

Similarly, DSEPs are generated after cutaneous electrical stimulation of the corresponding dermatomal regions and can be recorded from appropriate spinal and scalp sites. Changes in the amplitude or latency, or both, of these potentials may indicate damage to the neural tracts.[1] Often we observed interference patterns represented by small sharp deflections on the EKG trace in the diagnostic-bandwidth filtering mode only on the Hewlett-Packard monitors (lead V_5 in fig. 1) during pre-

vious cases using SSEP monitoring with the stimulating electrodes placed at the wrists. This more commonly observed pattern can be misinterpreted easily as pacemaker spikes. In fact, Sliwa and Marinko[3] report that this type of interference (small deflections during EKG) caused by a transcutaneous nerve stimulator unit was interpreted by a cardiologist as a malfunctioning pacemaker.[3] Similarly, the large EKG deflections produced in our inferior leads by DSEP stimulation could have been interpreted easily as sudden onset of supraventricular tachycardia.

We believe the proximal positioning of the EKG electrodes proximal to the DSEP-stimulating electrodes caused the nearly equal amplitude of the evoked-potential deflections and the QRS complex in the inferior leads. Lack of this interference pattern in the lateral leads or with electrical stimulation of the right T_{10} and T_{12} dermatomes helps to confirm our conclusion. Legatt and Frost[5] report a nearly identical interference pattern from SSEP-stimulating electrodes when viewed on the Datascope 2000 monitor screen display and the "delayed" hard copy, but not on the "diagnostic" hard copy mode because of a "pacer enhancement circuit" in the monitor designed to increase the visibility of small pacemaker spikes.[5] During previous SSEP monitoring, we observed small EKG interference deflections (lead V_5 in fig. 1) in the diagnostic-bandwidth filtering mode only, which displays an unfiltered EKG trace.[6] However, during this case using DSEP monitoring proximal to the EKG electrodes, we observed large EKG interference deflections in the inferior leads in the filter-, monitor-, and diagnostic-bandwidth filtering modes on the Hewlett-Packard monitors. Both the filter- and the monitor-bandwidth filtering modes are designed to reduce electrical interference to the EKG signal.[6] We suspect that the proximity of the right and left leg EKG leads to the DSEP right T_8 and left T_{10} stimulating electrodes (< 5 cm) caused a sufficiently large skin surface voltage change to be detected, despite the use of these filtering modes.

This report cautions that evoked-potential monitoring potentially can lead to false EKG signals, which can be misinterpreted easily as pacemaker malfunction or serious cardiac arrhythmia.

References

1. Barash PG, Cullen BF, Stoelting RK: Clinical Anesthesia. 2nd edition. Philadelphia, J. B. Lippincott, 1992, pp 760–1, 771–5, 1067–8

2. Kimberly APS, Soni N, Williams TR: Transcutaneous nerve stimulation and the electrocardiograph. Anesth Intens Care 1987; 15:358–9

3. Sliwa JA, Marinko MS: Transcutaneous electrical nerve stimulator-induced electrocardiogram artifact: A brief report. Am J Phys Med Rehabil 1996; 75:307–9

4. Russell GB: Motor evoked potentials, Primer of Intraoperative Neurophysiologic Monitoring. Edited by Russell GB, Rodichok LD. Boston, Butterworth-Heinemann, 1995, pp 166–7

5. Legatt AD, Frost EAM: EKG artifacts during intraoperative evoked potential monitoring. ANESTHESIOLOGY 1989; 70:559–606. Hewlett-Packard Component Monitoring System Anesthesia/Standard Operating Manual. 9th edition, volume 2. Palo Alto, Hewlett-Packard, 1995, pp 12-24–5