CASE REPORTS

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The Processed Electroencephalogram May Not Detect Neurologic Ischemia during Carotid Endarterectomy


Neurologic testing in the awake patient during carotid endarterectomy is used as a monitor of cerebral perfusion. However, because carotid endarterectomy is often performed under general anesthesia, electroencephalographic (EEG) monitoring (both standard and processed) is often used to record cerebral electrical activity (CEA) for the detection of cerebral ischemia. We describe two cases undergoing carotid endarterectomy under regional anesthesia who were monitored with the Lifescan® EEG monitor (Neurometrics).

The Lifescan® uses aperiodic analysis, which maps each waveform in relation to its frequency, amplitude, and time of occurrence, rather than averaging a large number of waveforms over a given epoch, as in Fourier analysis. The EEG signal is obtained from a two-bipolar-channel, five-lead system with electrodes attached to the frontal and mastoid areas bilaterally and a reference electrode in the midline frontally. We used 3M Red Dot® EKG electrodes, after the skin had been scraped and small amounts of electrode gel applied. The processed EEG is displayed in full color on a high resolution screen as two 3-dimensional parallelograms with frequency versus amplitude on the XY axis and time on the Z axis. Ninety percent edge frequency (frequency beneath which 90% of the summed amplitudes reside) is displayed as a white line on the roof of the parallelogram.

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Case 1. A 73-yr-old woman presented for right carotid endarterectomy. Medical history included non-insulin-dependent diabetes mellitus and hypertension. She had suffered two cerebrovascular accidents, 16 and 17 months previously, both resulting in a right hemiparesis from which she had almost fully recovered. Carotid angiogram had shown complete occlusion of the left internal carotid and a severe stenosis of the right internal carotid. Her medications included 250 mg of alphamethyldopa twice daily and 650 mg of enteric-coated aspirin daily.

Diazepam 10 mg was given orally 1 h before anesthesia. Peripheral venous and radial artery catheters were inserted and electrodes attached to the chest for EKG monitoring, and to the head for EEG monitoring. Oxygen was administered via a face mask with an end-tidal CO₂ sampling tube attached. Deep cervical plexus block was performed by injecting 6 ml of 1.5% lidocaine with epinephrine at C2, C3, and C4 (total 18 ml), and the superficial cervical plexus was blocked with 10 ml of the same solution. Neurologic status was tested intermittently (the frequency depending on stage of surgery) by asking the patient to squeeze the anesthesiologist's hand (using her left hand).

The sequence of changes on the Lifescan® screen is shown in figure 1. After administering heparin 50 mg, a trial cross-clamping of the internal carotid artery was performed. Within 30 s the patient lost power in the left hand and lost consciousness a few seconds later. Almost simultaneously with the loss of consciousness there was a noticeable loss of high frequency in the CEA. The clamp was removed until the patient regained consciousness and movement in the left hand (she was unconscious for approximately 30 s). This was accompanied by a return to the original appearance on the EEG. The carotid artery was subsequently clamped again for 85 s while a shunt was inserted. Occlusion of flow again resulted in loss of power of the left hand followed rapidly by a 60 s loss of consciousness with similar changes on the EEG. These recovered on reestablishment of cerebral flow. The endarterectomy was then performed without complications. Carotid cross-clamping during shunt removal was again associated with transient neurologic and EEG changes similar to the two previous occasions. During the periods of loss of consciousness there were no airway or respiratory difficulties. The patient was returned to the recovery room in a satisfactory condition without further neurologic signs. At this time the CEA was identical to the preoperative level. The patient's postoperative course was uneventful.

Case 2. An 85-yr-old woman presented for right carotid endarterectomy following two reversible episodes of left-sided numbness and weakness occurring 6 and 2 wk previously. No residual sensory or motor deficit was present on admission. A digital subtraction angiogram demonstrated right carotid artery stenosis, and this was associated with a loud right carotid bruit on auscultation. Her only medications were chlora hydrate 500 mg at night and aspirin 100 mg daily, which she had commenced after her second ischemic episode.

Preoperative systolic blood pressures measured on the ward varied between 100 and 190 mmHg. Following premedication with temazepam 10 mg orally the patient arrived in the anaesthetic room relaxed, but awake and alert. Monitoring was established as in case 1, and a similar anesthetic technique and neurologic assessment were used. During the procedure the patient experienced two brief episodes (less than 30 s) of loss of power in the left hand, despite remaining conscious and being able to respond verbally. The first episode occurred within 40 s of trial clamping of the carotid system. The clamp was released with almost immediate return of grip strength. Associated with this episode the systolic blood pressure, which had been 180 mmHg, gradually increased to 220 mmHg over the next 2.5 min and remained
above 300 mmHg for 8 min. There was no loss of grip strength during the clamping of the carotid artery for shunt insertion (110 s). At this time the systolic blood pressure was above 200 mmHg as opposed to 180 mmHg at the time of the first clamping. The carotid endarterectomy was then performed uneventfully. During carotid clamping for removal of the shunt the grip strength was again lost. At this time the systolic blood pressure was 190 mmHg and, as systolic blood pressure once again spontaneously increased to 210 mmHg, the strength in the hand grip returned even before the clamp was removed. Both the left and right CEA remained unchanged throughout the procedure with no obvious changes related to either cross-clamping or loss of grip strength in the left hand. Postoperative recovery was uneventful.

**DISCUSSION**

The EEG has been described as accurate in detecting episodes of cerebral ischemia in patients undergoing carotid endarterectomy under general anesthesia. Processed EEG machines provide an alternative to 16 to 20 lead EEG because they are easy to apply, use, and read. Several methods of processing and displaying CEA are available. Previous studies have investigated the changes in the processed EEG in relationship to the standard EEG and to postoperative neurologic deficits. The relationship between the on-line processed EEG and neurologic assessment in the awake patient has not been reported.

The on-screen changes associated with neurologic deficit in case 1 are of interest. Spackman et al. emphasized that interpretation of the Lifescan® is based on the overall picture rather than a numeric output as in some processed EEG methods. In this case the loss of high frequency components is most clearly represented by a fall in edge frequency on the right side, although a perceptible fall in edge frequency is also visible on the left side. Furthermore, the screen changes were identical and reproducible at each point of cerebral blood flow occlusion. Decreases in the trended edge frequency on screen would seem the best indicator of ischemia.

The second case demonstrates that awake neurologic assessment may reveal evidence of cerebral ischemia, which is undetected by two-channel aperiodic analysis. This brings into question the sensitivity of such monitoring. Several factors must be considered. First, it is known that CEA on the brain surface may fail to reflect ischemia in the deeper regions of the brain. Using a standard EEG in patients undergoing carotid endarterectomy under general anesthesia Chiappa et al. documented that only six of nine patients who awoke with neurologic deficits demonstrated intraoperative EEG changes. Evans et al. noted that the EEG remained unchanged in four of 13 patients under regional anesthesia, who developed clear neurologic deficits on cross-clamping.
distantly spaced electrodes tend to be orientation-sensitive rather than location-sensitive and are therefore less sensitive for detection of localized ischemia. The ischemia in the second case may have been more localized than the first case because no loss of consciousness or verbal communication impairment resulted. Finally, the possibility of technical error must be considered. This seems unlikely because the processed EEG machine used here self-tests and calibrates automatically before each usage and is programmed to signal any fault should it arise.

The second case also demonstrates that adequate cerebral perfusion may be critically dependent on a systolic blood pressure higher than the patient’s normal range. Under general anesthesia in the absence of EEG changes and without the loss of the patient’s hand strength to signal cerebral hypoperfusion, the anesthesiologist may have chosen to decrease the arterial blood pressure pharmacologically and thereby jeopardized cerebral perfusion unknowingly.

The problems of two-channel monitoring in aperiodic analysis are also shared by two-channel Fourier analysis. However, the two methods differ in both method of processing and display. Aperiodic analysis produces a real time interpretation of the raw EEG tracing. The averaging required for the Fourier analysis may compromise the resolutions of the high frequencies, which are important in the detection of ischemic changes.

In summary, we have described and correlated the changes in Lifescan® output with awake neurologic assessment in two cases of eventful carotid endarterectomy. Changes in this processed EEG method are probably best represented by a change in edge frequency. However, the method may fail to detect cerebral hypoperfusion that can be detected by simple neurologic assessment in the awake patient. The performance of aperiodic analysis of the CEA during carotid endarterectomy under both regional and general anesthesia requires further assessment.

REFERENCES


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Posterior Arytenoid Dislocation Following Uneventful Endotracheal Intubation and Anesthesia

EDWARD J. FRINK, M.D.,* BRADLEY D. PATTISON, M.D.†

Arytenoid cartilage dislocation following endotracheal intubation is a rare event. Recognition of its occurrence and subsequent treatment are important to prevent long-term consequences. This report describes a case of left posterior arytenoid dislocation following endotracheal intubation and anesthesia for mastectomy and subsequent myocutaneous flap.

REPORT OF A CASE

A 49-yr-old woman was admitted for right modified radical mastectomy with concomitant reconstruction using rectus abdominis flap. The patient had no previous history of medical or surgical illnesses.

* Fellow in Anesthesiology.
† Resident in Anesthesiology.

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Address reprint requests to Dr. Frink: Department of Anesthesiology, Arizona Health Sciences Center, 1501 North Campbell, Tucson, Arizona 85724.

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