Anesthesiology
83:208–210, 1995

Lippincott–Raven Publishers

Detection of Acute Embolic Stroke during Mitral Valve Replacement Using Somatosensory Evoked Potential Monitoring

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STROKE is a well recognized complication of cardiac operations. Although the precise etiology for stroke during cardiac operations remains controversial, experimental and clinical studies have suggested that cerebral embolism may be an important cause of neurologic injury in this group of patients.[1,2] A problem encountered in establishing the relationship between operative events that result in embolization and intraoperative stroke has been, in part, the limited ability to detect acute neurologic injury in anesthetized patients. In experiments, cortical somatosensory evoked potentials (SEPs) are sensitive to changes in regional cerebral blood flow caused by vascular occlusion or gas embolism,[3,4] but no data support the clinical use of SEP monitoring to detect acute central nervous system injury during cardiac operations. The following report demonstrates that SEP monitoring detected an acute embolic stroke during mitral valve replacement, localized the region of injury, and established the operative event that was temporally related to the injury.

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Received from the Departments of Anesthesiology, Neurology, and Surgery, University of Pennsylvania, Philadelphia, Pennsylvania. Submitted for publication January 9, 1995. Accepted for publication March 9, 1995.

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Key words: Brain: somatosensory evoked potentials; stroke. Complications: stroke. Monitoring: somatosensory evoked potentials. Surgery: cardiac.

Case Report

A 70-yr-old, 69-kg man was referred for mitral valve replacement because of severe mitral regurgitation, congestive heart failure, and atrial fibrillation. The patient had an aortic valve replacement with a 31-mm Carpentier-Edwards bioprosthetic valve (Baxter Healthcare Corp, Santa Ana, CA) 14 yr earlier for rheumatic aortic regurgitation.

General anesthesia was induced and maintained by the administration of fentanyl, midazolam, pancuronium, and 100% O_2. Volatile anesthetics were not administered. Bilateral upper extremity SEPs were monitored (Viking II, Nicolet Biomedical, Madison, WI) and recorded every 3 min throughout the operation in a clinical protocol approved by the University of Pennsylvania Committee on Studies Involving Human Beings to detect brachial plexus injury during cardiac operations. The somatosensory stimulus consisted of 25-mA, 0.5-ms electrical pulses applied to the right and left ulnar nerves at the wrist. Recordings were made from electrodes placed at the right and left Erb's points, the neck, and the C3′ and C4′ scalp electrode positions. The latency of the Erb's point potential arising from the brachial plexus, the N13 arising from the cervicomedullary junction, N18 arising from the brainstem, N20 arising from the thalamus and cortex, and P22 arising from the somatosensory cortex were measured as well as the amplitude of the N20–P22 complex. Intraoperative transesophageal echocardiography showed no thrombus in the left atrial appendage and no atherosclerotic plaques in the ascending or descending thoracic aorta.

Cardiopulmonary bypass was instituted using a membrane oxygenator (Maxima Plus, Medtronic, Anaheim, CA) and in-line arterial filter (Bard H-690, Bard, Tewksbury, MA). A pulmonary artery vent was inserted while the patient was cooled to 28°C. After ventricular fibrillation, the ascending aorta was cross-clamped with an extra-large Fogarty clamp. Cardiopulmonary bypass was maintained at a mean flow rate of 3.7 l/min (cardiac index of 2.2 $l \cdot min^{-1} \cdot m^2$) and mean arterial pressure of 65 mmHg. The native mitral valve was excised and replaced with a 31-mm Carpentier-Edwards bioprosthesis.

Rewarming was initiated during closure of the atriotomy. Within 90 s after removal of the ascending aortic cross-clamp, while the heart was asystolic, there was a complete disappearance of the N20 and P22 potential arising from the right thalamus and sensory cortex (fig. 1). The latencies and amplitudes of the Erb's point, N13, and N18 potentials did not change. Cardiopulmonary bypass flow was immediately increased to 5.0 l/min, 100% O_2 was administered into the membrane oxygenator, phenylephrine was administered to maintain a mean arterial pressure of greater than 60 mmHg, and an infusion of 50 $\mu g \cdot kg^{-1} \cdot min^{-1}$ intravenous propofol was initiated.

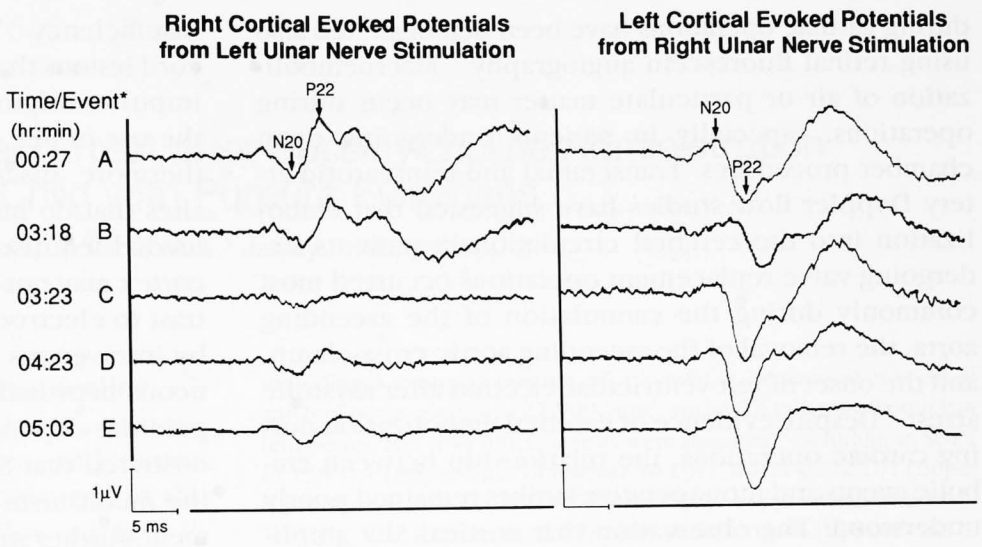

Fig. 1. Representative cortical N20–P22 somatosensory evoked potentials (SEPs) from right ulnar nerve stimulation (*right*) and left ulnar nerve stimulation (*left*) obtained at five elapsed times after the start of operation (time 0). The acute loss in amplitude of the right cortical N20–P22 SEP from left ulnar nerve stimulation was observed at an elapsed time of 03:23 (h:min). The aortic cross-clamp was removed at an elapsed time of 03:21 (h:min). No significant differences between right and left Erb's point, N13, and N18 SEPs were detected during the operation (not shown). *SEPs were recorded in relation to the events shown in figure 2.

Transesophageal echocardiography examination did not show an excessive quantity of air in the cardiac chambers. Separation from cardiopulmonary bypass was accomplished with ventricular pacing, 4 μg/min intravenous epinephrine, and 100 $\mu g \cdot kg^{-1} \cdot min^{-1}$ intravenous propofol. The amplitude of the right thalamic and cortical N20–P22 potentials increased gradually to 35% of their baseline value over the ensuing hour (fig. 2).

On postoperative day 1, the patient had a dense left hemiplegia. There were no detectable upper extremity peripheral neuropathies. A computed tomography scan showed a small right frontal periventricular infarction in a watershed distribution and no intracranial hemorrhage. Perioperative creatine kinase-BB isoenzyme fractions increased from 0 to a peak value of 6.2% at 3 h after the release of the aortic cross-clamp and decreased to 1% by postoperative day 2. During the next 9 days, the patient had gradual recovery of left-sided motor and sensory function, although moderate left leg weakness persisted that required the aid of a walker for ambulating. On postoperative day 12, a repeat computed tomography scan of the head showed no new changes. On postoperative day 13, the patient was discharged to a rehabilitation unit.

Discussion

Monitoring of the cortical SEPs established the time at which the stroke occurred, the region of the brain that was injured, and a temporal link between an operative event and the acute onset of neurologic injury. The removal of the ascending aortic cross-clamp and the almost immediate loss of right cortical SEPs suggested that an acute arterial embolism into the cerebral circulation caused the stroke. The mechanism and site of injury based on the observed changes in the intraoperative SEP recordings were later verified by the neurologic examination, computed tomography scan of the head, and biochemical markers of central nervous system injury.

Cerebral embolism has been implicated as a major cause of neurologic injury complicating cardiac operations.[1,2] Small capillary and arteriolar dilations observed in postmortem studies of the brains of animals and patients after cardiopulmonary bypass have suggested that cerebral microembolization may be common during heart operations.[5,6] Microembolization

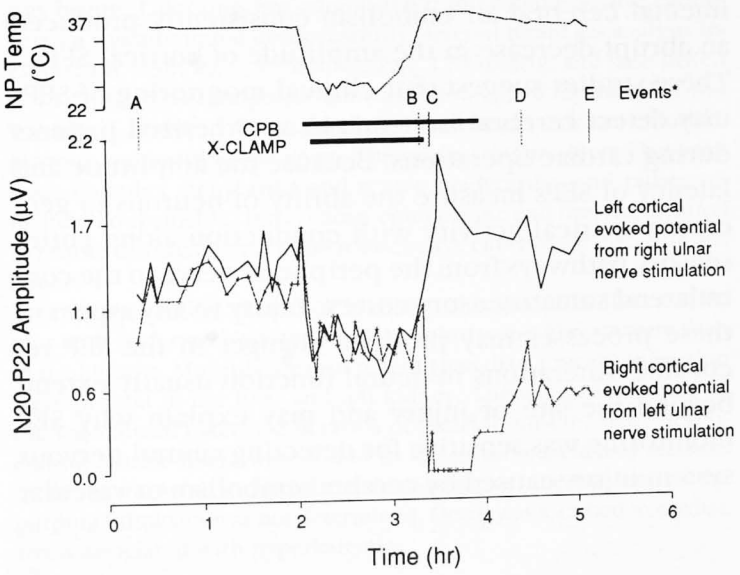

Fig. 2. Peak-to-peak amplitudes of N20–P22 somatosensory evoked potentials (SEPs) arising from left (— + —) and right (– – –) ulnar nerve stimulation during the operation. The symmetric changes in amplitude before removal of the aortic cross-clamp were caused by the decrease in body temperature. The asymmetric changes in the amplitude of the N20–P22 SEPs after removal of the aortic cross-clamp suggested acute injury to the right thalamus or right somatosensory cortex. CPB = cardiopulmonary bypass; NP Temp = nasopharyngeal temperature; X-clamp = ascending aorta cross-clamped.

during cardiac operations have been demonstrated also using retinal fluorescein angiography.[7] Macroembolization of air or particulate matter may occur during operations, especially in patients undergoing open chamber procedures. Transcranial and transcarotid artery Doppler flow studies have suggested that embolization into the cerebral circulation in patients undergoing valve replacement operations occurred most commonly during the cannulation of the ascending aorta, the removal of the ascending aortic cross-clamp, and the onset of left ventricular ejection after asystolic arrest.[2] Despite evidence of cerebral embolization during cardiac operations, the relationship between embolic events and intraoperative strokes remained poorly understood. The observation that cortical SEP amplitude decreased almost immediately after removal of the aortic cross-clamp provided compelling evidence that cerebral embolization was an important contributor if not the cause of the stroke during the cardiac operation.

In experimental studies, the amplitude of cortical SEPs decreased within minutes after regional cerebral blood flow was reduced to critical levels associated with cerebral ischemia (<16 ml $\cdot$ 100g^{-1} $\cdot$ min^{-1}). Cortical SEPs were abolished at regional cerebral blood flow rates of less than 12 ml $\cdot$ 100 g^{-1} $\cdot$ min^{-1}.[3] Experimental cerebral air embolism consistently produced an abrupt decrease in the amplitude of cortical SEPs.[4] These studies suggest that clinical monitoring of SEPs may detect cerebral ischemia in anesthetized patients during cardiac operations. Because the amplitude and latency of SEPs measure the ability of neurons to generate electrical activity with conduction along entire sensory pathways from the peripheral nerve to the contralateral somatosensory cortex, injury to any aspect of these processes may produce changes in the SEP recording. Alterations in neural function usually extend beyond the site of injury and may explain why SEP monitoring was sensitive for detecting central nervous system injury caused by cerebral embolism or vascular insufficiency.[3,4] However, peripheral nerve or spinal cord lesions that block the conduction of afferent nerve impulses originating from peripheral sites may prevent the use of SEP monitoring to detect brain injury. Furthermore, discrete central nervous system injuries to sites that do not affect the function of the brainstem, medial lemniscus, internal capsule, or somatosensory cortex may not be detected by SEP monitoring. In contrast to electroencephalography, SEPs are less affected by intravenous anesthetics, do not depend on spontaneous neuronal activity, and display predictable temperature-dependent changes.[8] Although this case demonstrated that SEP monitoring provided insight about the mechanism of stroke during mitral valve replacement, further study is required to determine the clinical utility of SEP monitoring for cardiac operations.

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Anesthesiology
83:211–213, 1995

Hyperkalemic

ALTHOUGH hyperkalemia a
ministration has been associ
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muscular disease,[1–4] it has r
setting of purpura fulminan
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Received from Oregon Health Sc
University of Washington and Child
Seattle, Washington. Submitted for
Accepted for publication March 1

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98105.

Key words: Ions, potassium: hy
ants: succinylcholine. Purpura fu