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Hemiparesis Following Carotid Endarterectomy: Comparison of Monitoring Methods

BETTY L. GRUNDY, M.D.,* ARTHUR C. SANDERSON, PH.D.,† MARSHALL W. WEBSTER, M.D.,‡
ELDRED T. RICHEY, M.D.,§ PHYLLIS PROCOPIO, B.A.,¶ PERCY N. KARANJIA, M.D.**

Intraoperative monitoring of brain function may reduce the incidence of neurologic morbidity associated with carotid endarterectomy.[1] Some surgeons measure carotid artery stump pressure[2] or regional cerebral blood flow[3] to assess cerebral circulation during carotid occlusion. Others routinely employ carotid artery shunts.[4] Brain function can be monitored using clinical neurologic assessment in patients operated upon under regional anesthesia,[5] or electroencephalography (EEG) during general anesthesia.[6-8] With either of these monitoring methods, routine insertion of a carotid artery shunt and its complications[9,10] can be avoided in those patients with collateral circulation adequate to maintain cerebral function during carotid artery occlusion. We had the opportunity to compare intraoperative neurologic assessment, on-line EEG monitoring, and off-line EEG signal analysis in a patient who developed mild hemiparesis after carotid endarterectomy.

REPORT OF A CASE

This case was part of a study approved by the Institutional Review Board, University of Pittsburgh School of Medicine. A 62-year-old right-handed man with hypertensive and arteriosclerotic cardiovascular disease experienced recurrent transient ischemic attacks consisting primarily of weakness in the right shoulder and arm. The neurologic examination was normal. Arterial blood pressure ranged from 120/70 to 140/80 torr. Angiography showed stenosis with an ulcerated plaque at the origin of the left internal carotid artery and a small plaque in the right internal carotid.

Left carotid endarterectomy was performed under cervical plexus block, with continuous monitoring of electrocardiogram, blood pressure recorded from a cannula in the radial artery and EEG. We assessed neurologic function every five minutes throughout the procedure by asking the patient to perform a simple calculation, recall a simple fact, and perform a simple motor task with the right hand. In addition, the patient counted aloud for two minutes when the carotid artery was clamped and again when clamps were removed from the vessel.

Three EEG monitors were employed on-line: conventional 16-channel strip chart recording,†† paired Cerebral Function Monitors,‡‡ and four channels compressed spectral array.§§ Two channels of EEG were recorded on magnetic tape for later analysis. Gold cup electrodes were placed according to the International Ten Twenty System[11] and attached to the scalp with collodion. Electrode impedances were less than 3000 ohms. A specially constructed junction box allowed us to record on multiple devices from the same electrodes, so that differences among methods of signal analysis and display could not be attributed to differences in electrode placement. The four-channel compressed spectral array analyzed signals from F3-C3, C3-P3, F4-C4 and C4-P4. The paired Cerebral Function Monitors and tape recorder used C3-P3 and C4-P4. These electrode locations are over the area of cortex supplied by the middle cerebral artery, where ischemic or embolic injury during carotid endarterectomy would be most likely to occur.

Transient bradycardia and hypotension associated with initial manipulation of the carotid bifurcation (heart rate 35 beats/min and arterial pressure 105/40 torr for approximately one minute) responded to local infiltration of 0.5 per cent lidocaine and intravenous administration of 0.8 mg atropine. Arterial blood pressure was otherwise stable between 130/70 and 170/100 torr. The three on-line EEG monitors were stable throughout to visual inspection. Carotid occlusion time was 20 min. No shunt was employed. No neurologic change was appreciated intraoperatively or in the recovery room. The patient was alert and well-oriented. He moved his face and extremities on command, and his grip strength was clinically symmetrical.

On initial ambulation the day after surgery, the patient complained of weakness in the right shoulder and stated that his right hand "felt light." The first detailed postoperative neurologic examination (30 hours after operation) showed mild right hemiparesis characterized by pronation drift of the right hand and weakness in a pattern typical of upper motor neuron lesions (deltoid, external rotators of shoulder, triceps, finger extensors, hip flexors and leg dorsiflexors). The rest of the neurologic examination was normal.

Three methods of EEG analysis not yet available for on-line use were employed after surgery to determine whether these potentially more sensitive signal processing techniques might reflect intraoperative EEG changes not apparent with the on-line EEG monitors. Our off-line methods of analysis are briefly described in the Appendix. Electroencephalographic (EEG) signals tape recorded during operation were analyzed by the method of Sanderson.[12] In addition, we plotted power and frequency ratios derived from power spectra of EEG activity in symmetrical channels from the two sides of the head. Finally, similar power and frequency ratios were derived from the output of the paired Cerebral Function Monitors.

Sanderson's method of signal analysis showed EEG change in the distribution of the middle cerebral artery during carotid clamping and

* Assistant Professor of Anesthesiology and Neurosurgery, University of Pittsburgh.

† Associate Professor of Electrical and Bio-Medical Engineering, Carnegie-Mellon University.

‡ Associate Professor of Surgery, University of Pittsburgh.

§ Associate Professor of Psychiatry and Neurology, University of Pittsburgh.

¶ Research Associate in Anesthesiology, University of Pittsburgh.

** Assistant Professor of Neurology, University of Pittsburgh.

Address reprint requests to Dr. Betty L. Grundy: Assistant Professor of Anesthesiology and Neurosurgery, 1060 E Scaife Hall, Pittsburgh, Pennsylvania 15261.

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†† Grass Instrument Corporation, Quincy, Massachusetts.

‡‡ CFM Model 870, Critikon, Inc., Tampa, Florida.

§§ Nicolet MED 80, Nicolet Biomedical, Madison, Wisconsin.

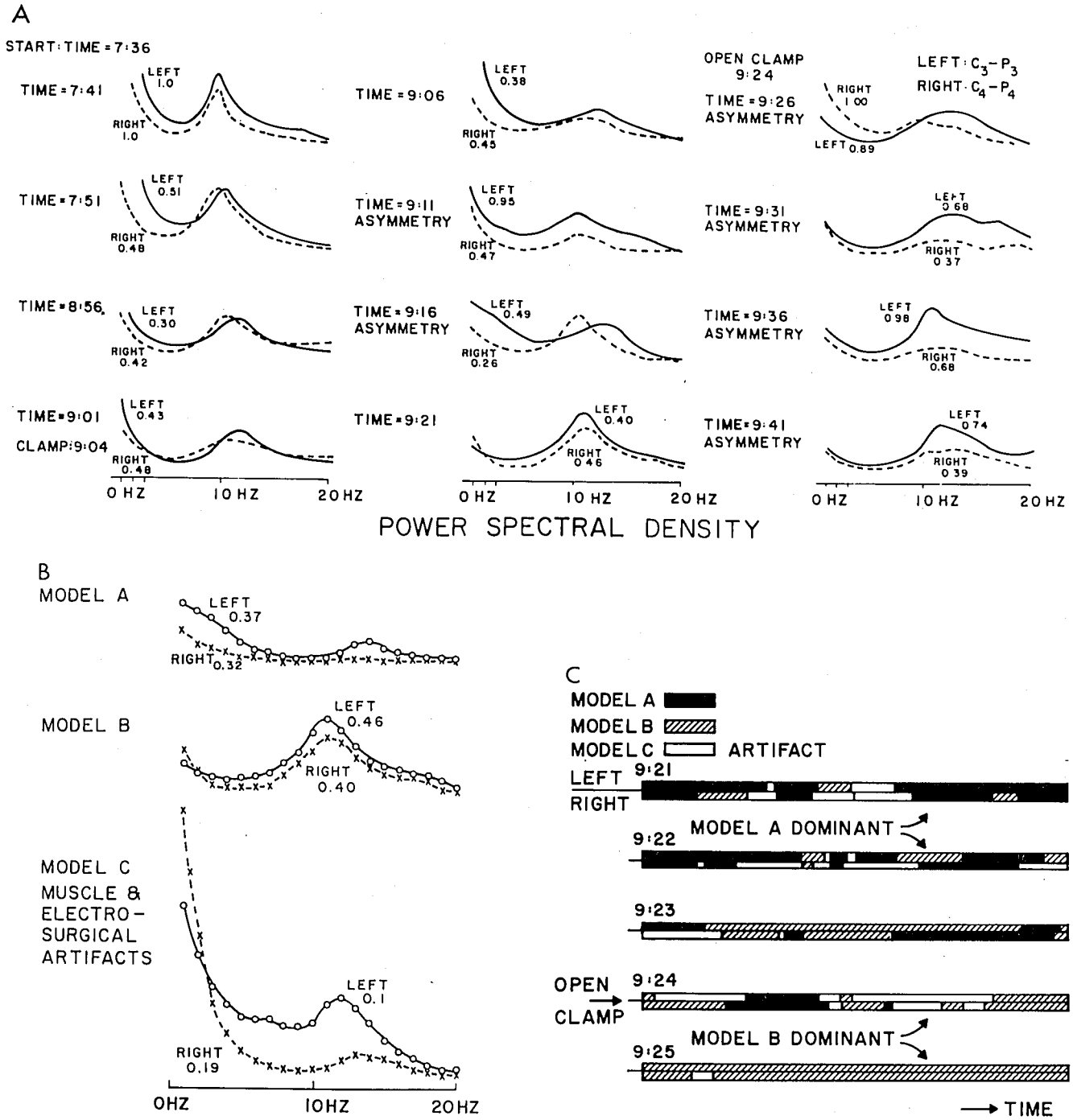

FIG. 1. Hierarchical modeling and pattern analysis by method of Sanderson.[12] *A*. Each spectrum describes a time segmented model computed for a five-minute period. The vertical axis of each plot indicates relative spectral power. Number labels indicate the proportion of the particular five-minute time period occupied by a given model. For many periods, time segments including excessive artifact due to muscle or the electrosurgical machine were omitted. These periods were automatically excluded by the segmentation technique. Models occupying only small proportions of the time period were also omitted. *B*. Spectral models for time period 9:21 to 9:26. *C*. Sequence of model segments for time period 9:21 to 9:26. The sequence of spectra illustrate the development of asymmetry during clamping of the carotid and after unclamping. The pattern change after unclamping is readily apparent in the graphic presentation *C*. showing new dominance of Model B.

EEG asymmetry within the first minute after unclamping (fig. 1). The left-to-right ratio of power in the alpha frequency band changed within five minutes after unclamping (fig. 2). Power and frequency ratio plots derived from the Cerebral Function Monitors were unrevealing and reexamination of the clinical 16-channel EEG record again failed to detect any intraoperative changes apparent to visual inspection.

The patient went home three days after the operation, the mild hemiparesis resolving and not limiting his usual activities.

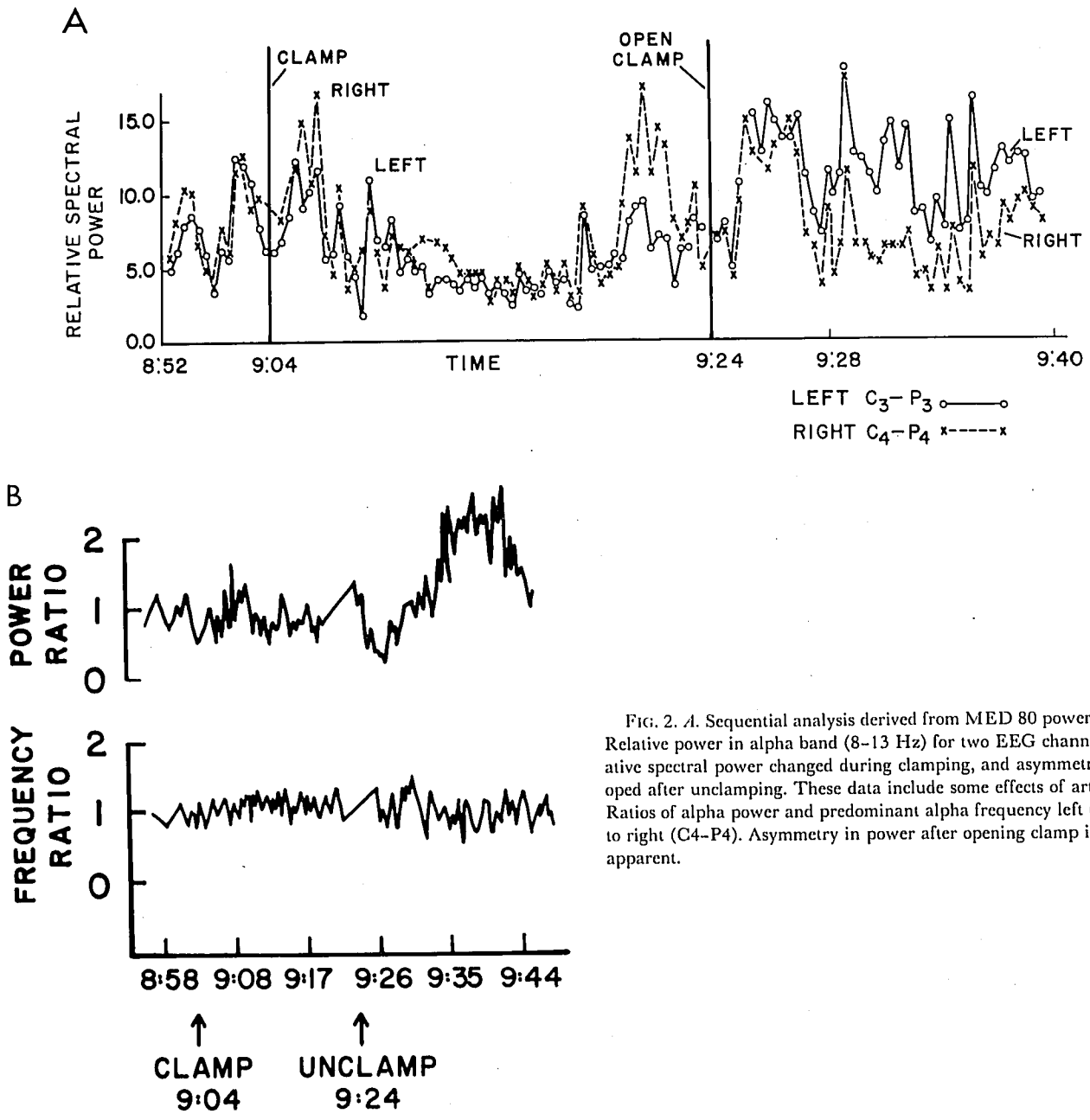

FIG. 2. *A*. Sequential analysis derived from MED 80 power spectra. Relative power in alpha band (8–13 Hz) for two EEG channels. Relative spectral power changed during clamping, and asymmetry developed after unclamping. These data include some effects of artifact. *B*. Ratios of alpha power and predominant alpha frequency left (C3–P3) to right (C4–P4). Asymmetry in power after opening clamp is readily apparent.

DISCUSSION

Clinical neurologic assessment during surgery is constrained by limitations on access to the patient and by time factors. To monitor function, we should repeat observations at frequent intervals; yet we must not interfere with the surgical procedure or exhaust the patient. Our tests depended on verbal communication with the patient and unobtrusive movements of one hand. These tests failed to detect intraoperative change in this patient. Repeated assessment of strength in the shoulders and upper arms might have detected an asymmetry in motor function as EEG changes developed, but this kind of examination could not have been performed during carotid endarterectomy without disrupting the operative field.

EEG monitoring in the operating room, suggested more than 40 years ago,[13] has been hampered by difficulties in signal acquisition and data reduction.[14] With currently available technology, both of these problems are clearly amenable to solution.[15]

Computer-assisted methods of EEG analysis may enhance the value of EEG monitoring during anesthesia and surgery. Once we define quantitative measures of intraoperative EEG change that are associated with postoperative neurologic deficit, we can develop EEG mon-

itors with alarms to give early warning of cerebral dysfunction. The EEG measurements of interest can be learned partly from animal studies, but only careful measurements of intraoperative EEG change in patients at high risk for neurologic injury will allow us to derive electrophysiologic measures predictive of outcome in patients.

This case is of interest because computer-assisted EEG signal analysis revealed subtle changes in brain function during carotid endarterectomy that were not detected either by intraoperative neurologic assessment or by conventional 16-channel EEG recording. Machines cannot develop new information, but they can process large amounts of information more efficiently than can humans. Our off-line computer programs extracted relevant information from the overwhelming amount of data in the complex EEG signal and presented this information in a format readily amenable to interpretation by clinicians (figs. 1*C*, 2*B*). Intraoperative EEG changes thus detected were associated with a mild but definite new neurologic deficit indicating cortical damage in the region of intraoperative EEG alteration. In this case, the off-line EEG analyses proved more sensitive to functional change than did any of the monitoring methods used on-line. Such sensitive measures may prove valuable for monitoring brain function during anesthesia and surgery.

Sanderson's method of analyzing EEG signals seems to offer several advantages for monitoring brain function in the operating room. Segments of the signal heavily contaminated by artifact can be more accurately recognized and discarded with this method than with methods of analysis that reject artifact using only amplitude criteria. Information of interest (*e.g.*, acute changes in pattern, appearance of patterns associated with subsequent development of neurologic deficit, or other untoward outcomes) can be presented in convenient and compressed format for clinical interpretation (fig. 1*C*). Sanderson's technique has been applied to normal and abnormal clinical EEG recordings[12] but has not been used in animal studies. This is the only report to date of its application to EEG recorded intraoperatively.

Because the most noticeable EEG changes in this case occurred immediately after unclamping of the carotid artery while the patient was otherwise stable, we must consider embolism as a possible mechanism of injury.

The decrease in EEG power seen on the side of operation during carotid artery occlusion is the kind of EEG change previously reported with cerebral hypoperfusion,[1,3,6-8] but the increase in alpha power after unclamping of the carotid artery is not. EEG correlation with intraoperative artery-to-artery embolism has not been systematically described, and, therefore, we cannot say whether the changes we saw were typical of embolic injury. Many observations such as ours will be required to define characteristic EEG patterns. Though monitors of brain function probably cannot prevent neurologic damage from arterial embolism during carotid endarterectomy, other insults to brain function (*e.g.*, ischemia, hypotension, hypoxia) might produce similar EEG alterations long before irreversible damage occurred, allowing timely intervention to improve outcome.

Reperfusion of a previously ischemic area with free flow at systemic arterial blood pressures should also be considered as a possible mechanism of injury in our patient.[16] Conceivably, this kind of injury could be minimized by controlling arterial blood pressure if an EEG monitoring method such as Sanderson's were available to guide therapy. Once again, however, systematic description of characteristic EEG patterns would be required.

To be effective, monitors must give early warning of dangerous situations. Because the brain is very sensitive to physiologic, pathophysiologic, and pharmacologic perturbations of interest to the anesthesiologist, computer-assisted EEG analysis for monitoring brain function during anesthesia and operation may prove valuable even for relatively low-risk procedures. If the potential value of EEG monitoring during anesthesia and surgery is to be realized, patterns associated with adverse neurologic outcomes must be defined so that they can be recognized intraoperatively before permanent damage occurs. When dangerous situations are detected early, the anesthesiologist can often intervene to prevent injury. Observations such as ours can assist in the development of effective monitors by defining patterns of intraoperative EEG change associated with postoperative neurologic deficit.

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APPENDIX

Off-line Methods of EEG Signal Analysis

These methods, not yet available for real time use in the operating room can be developed for on-line monitoring if they prove clinically useful.

Method 1. Hierarchical modeling and pattern analysis by the method of Sanderson.[12]

This technique is based on an autoregressive method of analysis. It recognizes specific power spectral patterns and formulates models for each of these patterns (fig. 1*A*). Once models have been constructed for a particular EEG record, the computer tracks the EEG signal and detects each change in pattern. A sequence of time segmented power spectral models is shown for each five-minute epoch of EEG (fig. 1*B*). The sequence of model segments for two symmetrical EEG channels is then depicted graphically (fig. 1*C*) to show pattern changes that might not otherwise be apparent. Sanderson's method may be more sensitive to subtle changes in EEG than other available methods of analysis because it adapts to time-varying EEG patterns.

Method 2. Power and frequency ratios in specific frequency bands.

The power spectral analysis performed on-line by the Nicolet MED 80 prints numerical power and frequency data as well as plotting the compressed spectral array. Ratios of the values from symmetrical channels on either side of the head are plotted to make subtle EEG asymmetries apparent. Numerical values for total power and predominant frequency in each of four specific frequency bands (theta, 4-8 Hertz; alpha, 8-13 Hertz; beta, 13-25 Hertz; and theta plus alpha, 4-13 Hertz) are compared by dividing values obtained from the EEG channel ipsilatural to the side of operation (C3–P3) by values obtained from the symmetrical contralateral EEG channel (C4–P4). Figure 2*A* shows numerical values for relative alpha power in each of these channels plotted against time. Figure 2*B* shows the derived ratios for alpha power and frequency.

Method 3. Power and frequency ratios derived from Cerebral Function Monitors.

Ratios are derived from digital values displayed on the two CFM 870® monitors by dividing values for the EEG channel ipsilateral to the side of operation (C3–P3) by values from the symmetrical contralateral channel (C4–P4).

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Coronary Artery Spasm during General Anesthesia

CHARLES W. BUFFINGTON M.D.,* AND TOM D. IVEY, M. D.*

Coronary artery spasm abruptly reduces coronary blood flow and oxygen delivery to the myocardium. The systemic hemodynamic pattern associated with this condition differs from that associated with effort-induced ischemia. Myocardial ischemia caused by coronary spasm occurs suddenly and without provocative increases in blood pressure or heart rate. Often recurrent, the episodes of ischemia are brief and paroxysmal in nature. Spontaneous, rapid resolution of ischemia may be accompanied by an overshoot in blood pressure and heart rate. Recognition of this pattern allows correct diagnosis and successful treatment. This is a report of coronary vasospasm during general anesthesia.

* Assistant Professor.

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Address reprint requests to Dr. Buffington: University of Washington School of Medicine, Department of Anesthesiology, RN-10, Seattle, Washington 98195.

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REPORT OF A CASE

A 55-year-old white man presented for coronary bypass surgery with a six-month history of angina pectoris. The pain occurred primarily during exertion but occasionally occurred at rest. He was selected for surgery because his angina was refractory to medical management with 60 mg propranolol, four times daily, 20 mg isosorbide dinitrate, every four hours, and sublingual nitroglycerin as needed. Coronary arteri-