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Preanesthetic Evaluation of a Patient with Pathologic Q Waves Following Subarachnoid Hemorrhage

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The assessment of a patient's cardiovascular status, including a recent electrocardiogram (ECG), is often an important part of a preanesthetic evaluation. ECG findings may be prognostically significant and must be reviewed carefully before planning surgical anesthesia. Most abnormal tracings usually require further cardiac evaluation before proceeding with elective surgery, and ECG evidence of recent myocardial infarction is often a contraindication to elective surgery. When urgent surgery is indicated for a patient with ECG changes suggestive of myocardial injury or infarction, the risk of delaying or canceling surgery must be weighed against the risk of perioperative cardiac complications, including death.

This situation may be encountered in patients with subarachnoid hemorrhage, who often present with abnormal ECGs. The most frequently noted abnormalities in these patients are elevation or depression of the ST segment, changes in T wave morphology, QT prolongation and various dysrhythmias.¹ Because these findings are widely recognized and are believed to be neurogenic in origin, they usually do not contraindicate surgery. In contrast, pathologic Q waves, which rarely are associated with subarachnoid hemorrhage, cannot be assumed to represent a neurogenic process.^{2,3} Rapid evaluation of this ECG abnormality must be performed to avoid unnecessary cancellation of surgery.

We describe a patient who developed pathologic Q waves during the course of hospitalization for subarachnoid hemorrhage. A complete cardiac evaluation, including serial 12-lead ECGs, continuous limb lead monitoring, cardiac isoenzyme determinations, and myocardial nuclear scanning performed at the time of Q-wave development showed no evidence of myocardial damage.

The patient underwent general anesthesia for surgical treatment of a cerebral artery aneurysm, which was performed successfully without cardiac complications.

REPORT OF A CASE

A 47-year-old man, in good health, experienced a tonic-clonic seizure, followed by a headache and a right-sided hemiparesis. He experienced a second tonic-clonic seizure in the emergency room. His medical history was significant only for alcohol abuse not associated with head trauma or previous seizure activity. He was taking no medications and had no history of cardiovascular disease.

His arterial blood pressure ranged from 186/100 to 220/110 mmHg, with a regular heart rate of 100 bpm. He had an S₄ gallop rhythm, lethargy, disorientation to time and place, mild dysarthria, a right facial palsy, and weakness of his right limbs. His lungs were clear, and there was no evidence of long-standing hypertension or of congestive heart failure. Laboratory values included normal serum electrolytes, renal function tests, and hematology. Analysis of arterial blood gases revealed a mild metabolic acidosis. His chest roentgenogram showed minimal hilar vascular congestion. Admission ECGs were felt to be within normal limits (fig. 1).

After administration of dilantin and dexamethasone, a computerized axial tomogram of the head showed collections of blood in the right and left lateral ventricles without an intracerebral collection. His hypertension was controlled adequately with hydralazine and diuretics. On the day after admission, a cerebral angiogram revealed a small irregular anterior communicating artery (ACA) aneurysm without spasm. The patient remained in the intensive care unit for routine monitoring until his aneurysm could be surgically treated. Nimodipine (30 mg po q4 hours) was started as part of an experimental protocol to reduce the severity of vasospasm.

His course in the ICU was unremarkable until the fourth day after admission, when his MCL monitor lead showed acutely inverted T waves. The patient was hemodynamically stable at this point and remained arousable but not alert. A 12-lead ECG revealed recent development of T wave inversion and pathologic Q waves in leads II, III, and aVF, consistent with inferior wall myocardial infarction (fig. 2). Surgery for clipping of the aneurysm was canceled while a cardiac evaluation was performed. Serial creatinine phosphokinase (CPK) and CPK isoenzyme fractions drawn over a 48-h interval were interpreted as being within normal limits. The abnormal tracings persisted and, in fact, appeared to be evolving on the 12-lead ECG. Despite normal cardiac isoenzymes, it was felt that these ECG changes may have represented myocardial infarction, possibly increasing this patient's operative risk. For that reason, a nuclear scan with pyrophosphate was obtained 48 h after the ECG changes had first been noted, which showed no localized or diffuse increase in tracer uptake by the myocardium. A repeat cerebral angiogram was obtained, which again showed no evidence of spasm.

Because the cardiac isoenzyme determination and the nuclear scan provided strong evidence that the patient had not suffered a myocardial infarction, he was rescheduled for operative treatment of his aneurysm. Eleven days after admission, he had a craniotomy and

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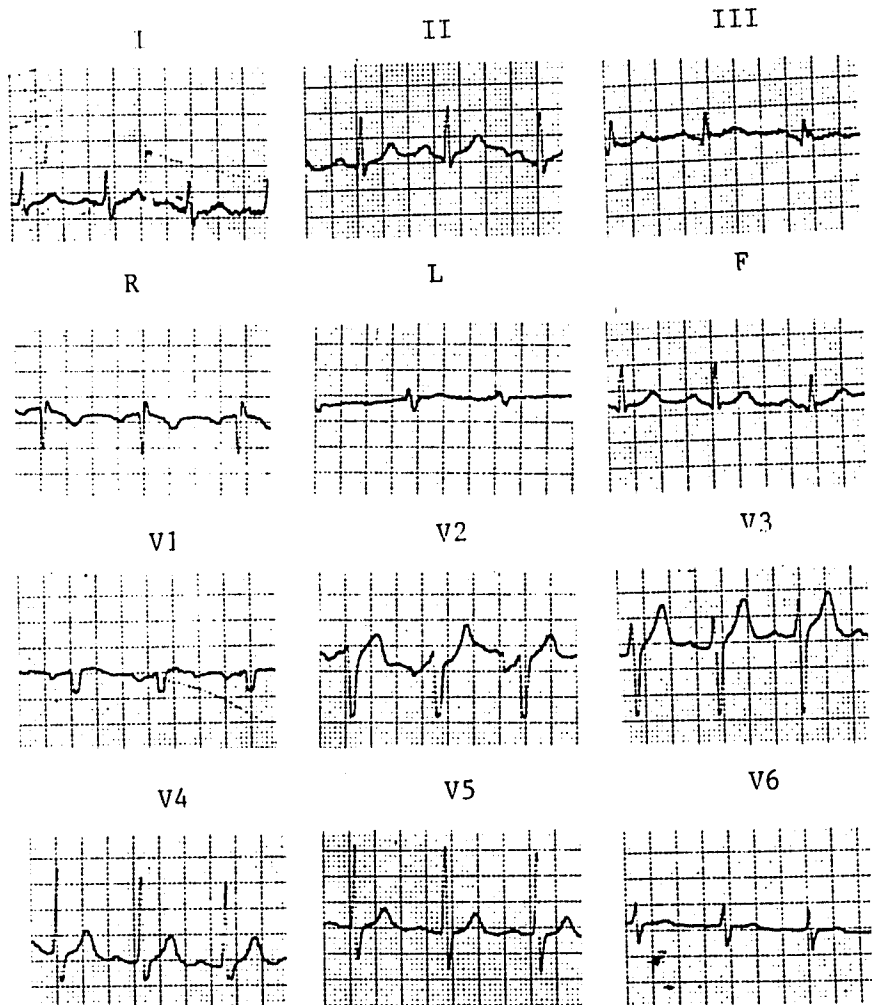


FIG. 1. Admission ECG demonstrating normal appearing inferior lead tracings.

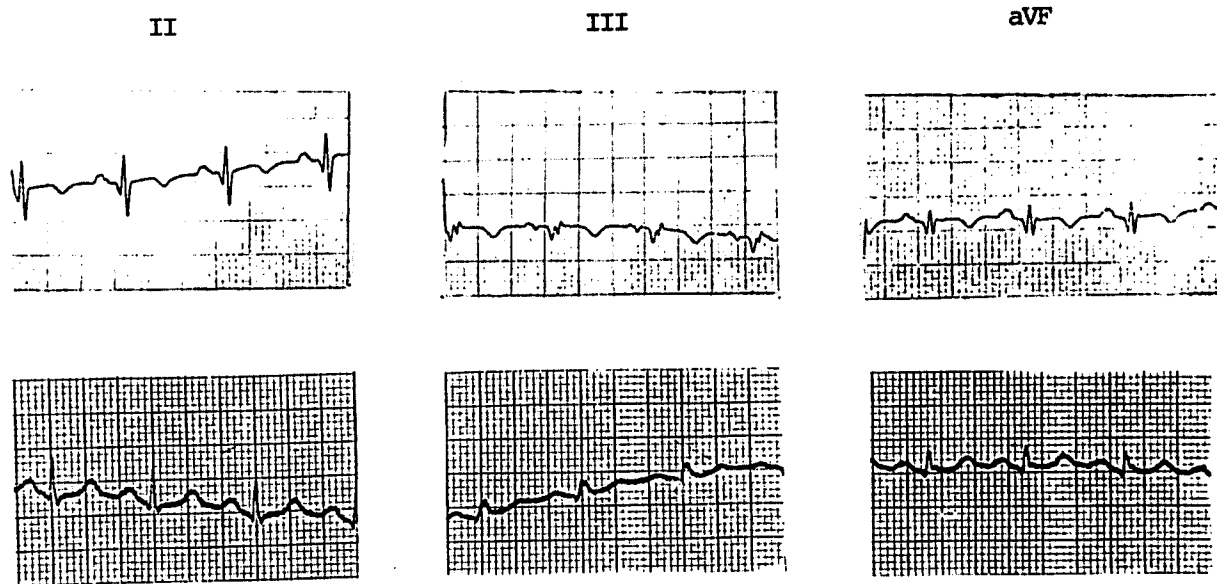


FIG. 2. (Top row) Leads II, III and aVF from a 12-lead ECG obtained after T-wave inversion was noted on the MCL leads. There is T-wave inversion and development of pathologic Q waves in leads II, III, and aVF. (Bottom row) Leads II, III and aVF from a follow-up ECG obtained 5 months after discharge, documenting resolution of the Q- and T-wave abnormalities noted during the patient's hospital course.

clipping of his ACA aneurysm. His intraoperative and postoperative courses were uneventful. He had no further cardiac or hemodynamic complications and was discharged from the hospital 2 weeks later. The patient returned for follow-up 5 months after discharge. An ECG obtained at that time showed resolution of the pathologic Q waves seen on earlier ECGs (fig. 2).

DISCUSSION

The finding of several ECG abnormalities varies from 60 to 100% in patients with subarachnoid hemorrhage.⁴ Most of these abnormalities usually are not associated with myocardial damage. When pathologic Q waves develop, however, they must be differentiated from ECG evidence of true myocardial infarction. Establishing that these ECG changes are not due to primary myocardial injury is important in patients who otherwise would be candidates for immediate surgical treatment (e.g., aneurysm clipping). Prophylactic anticoagulation to reduce the risk of myocardial infarction or cancellation of surgery in these patients is inappropriate and may prove fatal.^{5,6} Surgery immediately following myocardial infarction, however, may have equally catastrophic results.

Much has been written about the neurogenic etiology of ECG abnormalities associated with subarachnoid hemorrhage. Cruickshank *et al.*³ and, more recently, Harries² have summarized the most widely accepted theory of the pathogenesis of these ECG changes. Briefly, subarachnoid hemorrhage leads to cerebrovascular spasm of the small vessels supplying the hypothalamus. Lesions produced by ischemia of the hypothalamus cause stimulation of the heart by catecholamines through sympathetic pathways. Cardiac stimulation via right or left stellate ganglia can produce a variety of ECG abnormalities, including prolonged QT intervals, ST elevation, and changes in T-wave morphology.^{7,8} These findings probably are caused by regional changes in the functional refractory period of the myocardium, which alters the sequence of electrical recovery of the heart.⁸

Although this theory explains the occurrence of the commonly seen ECG changes noted above, it does not explain the occurrence of pathologic Q waves, which are considered by many to be diagnostic of transmural myocardial infarction. In fact, Hunt *et al.*⁹ state that pathologic Q waves are not a feature of the ECG of subarachnoid hemorrhage and may be used to differentiate true myocardial infarction from neurogenic ECG changes.⁹ Although this view has been challenged by isolated reports of Q waves developing with subarachnoid hemorrhage,¹⁰ these ECG findings are always ominous in a patient being evaluated for surgery.

The case presented attracted our attention because it posed a diagnostic dilemma for the anesthesiologist and neurosurgeon. This patient required urgent surgery, which was canceled when abnormal Q waves and

T-wave inversion developed, documented by serial ECGs. A careful preanesthetic cardiac evaluation, however, revealed normal isoenzyme levels and a normal radioactive tracer scan of the myocardium. Pyrophosphate myocardial scanning done 48–72 h after the onset of injury will detect 90% of all myocardial infarctions and 96% of all transmural infarctions, when diffuse activity detected by the scan is interpreted as a positive finding.^{11,12} This patient's negative scan, using the most sensitive criteria for detection of myocardial injury, provided strong evidence that his ECG abnormalities did not represent a myocardial infarction. Although surgical treatment of his aneurysm had been canceled initially, a careful assessment of his cardiac status allowed surgery to be rescheduled within several days. The delay in surgical intervention was brief, and the patient appeared to suffer no ill effects. His uneventful perioperative course and the apparent resolution of the Q waves on subsequent ECGs further support the contention that he had not suffered a preoperative myocardial infarction. A longer delay or cancellation of surgery may have proved fatal for this patient.

Recognition that most ECG abnormalities following subarachnoid hemorrhage may be neurogenic rather than cardiogenic in origin is important for the preanesthetic evaluation of these patients. Assessment of their cardiac and hemodynamic status employing continuous monitoring, isoenzyme determinations, and myocardial nuclear scanning may be an effective way of differentiating neurogenic from cardiogenic causes of ECG changes. This approach is particularly important when evaluating development of pathologic Q waves, an infrequently observed ECG finding with subarachnoid hemorrhage, which may be misinterpreted as being evidence of recent myocardial infarction. Rapid assessment of a patient's cardiac status in this situation may avoid unnecessarily long delays in initiating potentially life-saving surgical intervention.

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Intraoperative Defibrillator Failure

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We wish to describe a cascade of three defibrillator failures that could have been prevented by changes in operating procedure or equipment design.

REPORT OF A CASE

A 62-year-old woman presented for emergency coronary artery bypass grafting. After an uneventful anesthetic induction, the scrub nurse passed the sterile defibrillator cable to the anesthesiologist, who connected the cable to the defibrillator (Model 640, Physio-Control Corp., Redmond, Washington) but found that it would not charge for testing. A second, identical defibrillator was borrowed from the adjacent operating room, connected to the original cable, and charged to approximately five joules. Then the unit was switched off, causing it to discharge through its internal "dump" resistor, completing our usual test procedure.

Surgery proceeded without incident until the aortic cross-clamp was removed, when vigorous ventricular fibrillation developed. Defibrillation was attempted with the second unit, but it too would not charge. A second sterile cable was brought onto the operative field and connected successively to the second and first defibrillators, but neither of these would charge. A third, identical defibrillator was borrowed, but it too would not charge when connected to either cable. While waiting for another defibrillator to arrive, various combinations of defibrillators, internal and external cable sets, wall plugs, and extension cords were tried, all without success.

A fourth defibrillator (Life Pack 6®, Physio-Control Corp.) came with no internal cable, nor would it accept the available internal cables. Four unsuccessful defibrillation attempts were made with 400 joule shocks applied externally just lateral to the sterile field (two of these after the pericardial sac was filled with saline solution).

Finally, when a fifth defibrillator arrived with its own internal cable, the first shock of 10 joules produced normal sinus rhythm. Total fibrillation time was 27 min on full cardiopulmonary bypass at normothermia. The remainder of the operation and recovery were uneventful. The patient's peak creatine phosphokinase MB fraction postoperatively was 4 units/l (normal in our laboratory: ≤ 6 units/l), indicating that no significant myocardial damage occurred.

Subsequent investigation disclosed that the first cable had been steam autoclaved, despite a large red placard stating "Gas Sterilize Only." An earlier model of this cable set had been approved by the manufacturer for steam sterilization, and confusion arose because our inventory of cables contains both the current and the old models. Apparently, the autoclaving caused a short circuit in the cable connector, which, in turn, caused the fuse in the charging circuit of each defibrillator to blow when charging was attempted with the faulty cable connected. The fuse in question is of the "slo-blo" variety, so that the second defibrillator tested successfully because it was only briefly charged and then discharged, but it failed in actual use when the charging was sustained slightly longer. Unfortunately, the faulty cable happened to be the first one connected to the third defibrillator, so that it too failed.

DISCUSSION

Because of the design of this unit, we did not recognize that the source of the problem was in the cable connector. We then attached the faulty cable to two additional defibrillators, rendering them inoperative. The fuses on this unit are located inside the case and are not accessible for inspection or replacement by the user in the event of difficulty. There is no external indication when the charging circuit fuse has blown and the pilot lamp continues to function normally. Although this may appear to be a serious design flaw in this particular unit (which no longer is manufactured), the current voluntary standard for defibrillators‡ makes no statement that

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‡ American National Standard for Cardiac Defibrillator Devices. Arlington, Virginia, Association for the Advancement of Medical Instrumentation Publication No. ANSI/AAMI DF2-1981, 1981.