A young woman who sustained severe head trauma presented to the operating room for emergent surgical intervention. Her electrocardiogram (ECG) exhibited signs of myocardial ischemia, which resolved several days postoperatively.

ECG changes suggestive of cardiac pathology can be associated with intracranial pathology, most notably subarachnoid hemorrhage. Delay of operative therapy may have catastrophic results. Experimental data indicates massive sympathetic outflow results from stimulation of the lateral and posterior hypothalamic regions. Large amounts of norepinephrine are released into the systemic circulation, resulting in hypertension, tachycardia, dysrhythmias and ECG changes. Myocardial ischemia and injury can occur from the effects of this excessive sympathetic stimulation. In certain case reports, neurologic patients who experienced ECG changes had normal hearts on postmortem examination. This implies that myocardial recovery can occur despite the appearance of an abnormal ECG in the neurologic patient.

Anesthetic management of these patients involves prevention of further increases in intracranial pressure and avoidance of hyperventilation in the presence of hypotension or vasospasm. Continuous ECG monitoring is essential. Ventricular dysrhythmias may prove resistant to conventional pharmaceutical management. Beta blockade may be indicated to prevent excessive cardiac stimulation by endogenous catecholamines, and, in addition, may prevent the formation of the microscopic cardiac lesions typical of this hyperstimulation.

Key words: Electrocardiogram, myocardial ischemia, subarachnoid hemorrhage, torsade de pointe.

Patient presentation
A 30-year-old woman was beaten with a lead pipe and sustained multiple facial and skull fractures and a left subdural hematoma. An electrocardiogram (ECG) was obtained soon after admission and revealed sinus tachycardia with horizontal ST depression in leads III and AVF, consistent with myocardial ischemia. She was taken to the operating room shortly after admission for evacuation of the subdural hematoma. A medical history was unavailable on admission but was later obtained and noted to include no preexisting medical problems, with a negative review of systems. Three days later, a second ECG was obtained, which displayed sinus rhythm with accelerated A-V conduction (short PR interval). The previously noted ischemic changes were not present. The patient did well during her
hospitalization and was allowed to go home approximately five weeks postinjury.

Discussion

The association between central nervous system (CNS) disease and ECG changes was first described in 1938. These ECG changes were further detailed and categorized, and their frequent association noted in the presence of spontaneous subarachnoid hemorrhage (SAH). It is estimated that approximately 50% of patients with aneurysmal SAH will have ECG abnormalities. One case report describes a patient with a known aneurysmal SAH who had a preoperative ECG consistent with an anterior wall myocardial infarction. For this reason the operation was canceled, and the patient died soon afterward of a second hemorrhage. The autopsy revealed a normal heart with no evidence of recent infarction.

The anesthesia team is responsible for the preoperative assessment of all patients presenting for surgery. The ECG is perhaps the primary instrument which is employed to evaluate the well-being of the heart. Any patient with an abnormal ECG, or new changes noted on ECG, warrants further cardiac work-up to determine the cause and significance of such findings; however, the neurologic patient with subarachnoid hemorrhage who has such an ECG should not have surgery delayed. The anesthetist must be aware of ECG findings which can occur in the presence of intracranial disease and should examine the origins and prognostic implications such findings carry with them, since a good number of these patients require operative intervention (Table I).

| Table I |
| Conditions associated with subarachnoid hemorrhage |
| Hypertension |
| Cerebral aneurysm |
| A-V malformation |
| Trauma |
| Brain tumor |
| Blood dyscrasias |
| Angiopathies |
| Septic emboli |
| Leukemia |
| Anticoagulation therapy |
| Eclampsia |
| Cocaine toxicity |

Electrocardiographic changes

The most common abnormalities noted in the ECG of the patient with CNS disease are prolongation of the Q-T interval, large and often inverted T waves and prominent U waves. The normal Q-T interval is defined as one-half of the preceding R-R interval. The risk of development of ventricular arrhythmias is increased in the presence of a prolonged Q-T interval, because the delay in ventricular repolarization predisposes to the development of reentrant arrhythmias. A particular form of ventricular tachycardia, known as torsade de pointe, has been described in several case reports of patients with SAH. This rhythm may either terminate spontaneously or progress to ventricular fibrillation. Other ECG changes that are encountered in these patients include: peaked P waves, short P-R intervals, S-T segment depression and S-T segment elevation.

Pathogenesis of ECG and myocardial findings

An understanding of the role the CNS plays in the genesis of ECG abnormalities has been obtained through numerous animal studies. Apparently, the stimulation of certain central autonomic centers results in a massive sympathetic outflow, with a consequent release of large amounts of norepinephrine from the adrenal medulla. These centers in humans have been identified as lying in the lateral and posterior hypothalamic regions. These regions are stimulated by increased intracranial pressure, the irritant effect of blood, ischemia or a combination of these factors. Once stimulated, central efferent pathways produce norepinephrine release into the general circulation, with resultant acute hypertension and cardiac manifestations (Table II).

Experimental evidence indicates that elevated myocardial levels of norepinephrine lead to a non-uniform rate of repolarization, which predisposes to ventricular arrhythmias. Experimental data also suggest that myocardial damage can result from ischemia, which involves primarily the subendocardium of the left ventricle. These changes include
microscopic areas of focal necrosis, subendocardial petechial hemorrhage, mononuclear infiltrates and myofibrillar degeneration. Some researchers believe that, in certain cases, this damage is reversible, and therefore may not be present on postmortem examination.2

Alpha-receptor stimulation from norepinephrine may result in both peripheral and cerebral arterial spasm. This can increase the workload of the heart through a shift of blood volume to the central circulation.1 A recent case series reported that 30-70% of patients with SAH developed pulmonary edema during their hospitalization. It has not been determined whether the appearance of pulmonary edema is due to pump failure secondary to left ventricular lesions, acute circulatory overload, neurogenic pulmonary edema due to increased sympathetic or vagal tone or a combination of these factors.

Pharmacologic treatment
Pharmacological therapy involves the use of sympathetic blocking agents. Alpha blockers may be employed to prevent peripheral and cerebral arterial spasm. Beta-adrenergic blockade can be used to prevent or treat ventricular tachyarrhythmias which are produced by excessive sympathetic stimulation. Torsade de pointe ventricular tachycardia is resistant to or may even be aggravated further by treatment with the Class I antiarrhythmic drugs, such as lidocaine, procainamide or quinidine.3 One case report describes successful treatment of this dysrhythmia with propranolol, without recurrence, in a patient with SAH, intracerebral hemorrhage and prolongation of the Q-T interval.4

Evidence indicates that treatment with beta blockers is effective in preventing the development of the characteristic subendocardial lesions.2 In animals, the ECG changes and ventricular arrhythmias produced by stimulation of specific brain areas are prevented by beta-adrenergic blockade. In addition, one study in a human population demonstrated that in two groups of patients treated for SAH, the group who received propranolol had significantly fewer neurological deficits one-year postbleed.5 Propranolol may have a central effect which could account for these beneficial results. This hypothesis is supported by the fact that propranolol readily crosses the blood-brain barrier and is known to decrease the cerebral oxygen requirements in patients with stroke or cerebral ischemia.

Anesthetic considerations
Neurosurgical patients frequently present to the operating room in a coma with no available medical history, which limits the anesthetist's preanaesthesia assessment to information obtained from physical examination and laboratory testing. The patient's past medical history should be obtained, if possible, prior to operation. It is difficult to determine the implications of abnormal laboratory tests without information about the patient's previous medical history. For example, the neurosurgical patient with symptomatic preexisting cardiac disease and the previously healthy patient would show variation in their tolerance to sympathetic stimulation, i.e., tachycardia and hypertension. A preoperative ECG suggesting myocardial ischemia, even though the etiology of the changes is thought to be of neurologic origin, would be much more worrisome in the patient with preexisting cardiac disease.

The outcome of the patient with SAH is determined primarily by the degree of intracranial hemorrhage present, whether cerebral arterial vasospasm is present or if rebleeding occurs after the acute episode is successfully treated.

The anesthetic management of these patients is therefore directed toward prevention of further increases in intracranial pressure and systemic blood pressure, which could worsen the bleed. These patients should not be hyperventilated, especially when hypotension or vasospasm is present preoperatively, since this will reduce cerebral blood flow and oxygen delivery to the already hypoxic brain tissue.7 Continuous intraoperative ECG monitoring is an established standard of care, and knowledge of the pathogenesis of ECG changes which may arise in the presence of SAH is important for proper medical management.8

Some authors suggest using morphine as an analgesic for neurosurgical patients since there is a propensity for the development of pulmonary edema. An increase in pulmonary vessel capacitance would tend to prevent extravascular fluid shifts which could occur due to an increase in vessel tone from excessive sympathetic stimulation.

In summary, ECG tracings suggestive of the presence of myocardial ischemia or infarction can be encountered in patients with intracranial pathology, most notably subarachnoid hemorrhage (Table I). Surgery is usually not delayed in order to obtain a full cardiac work-up on these patients, since these ECG findings are felt to be attributable to the effects of a central neurogenic mechanism, rather than of cardiogenic origin.8 Whenever possible, the past medical history should be obtained to determine the status of the cardiovascular system prior to the onset of neurological pathology. Postponement of operative intervention could result in further
morbidity and mortality from progression of the CNS lesion. 8, 9

Patients with SAH also have a tendency to develop ventricular dysrhythmias when a prolonged Q-T interval is present, and these dysrhythmias may prove resistant to conventional antiarrhythmic therapy. 4-6 Beta blockade appears to effectively treat and prevent these dysrhythmias by blocking the cardiac stimulatory effects of endogenous catecholamines. Beta blockade also appears effective in preventing the development of the microscopic myocardial lesions which can form in response to high catecholamine levels.

REFERENCES

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