Cardiac Injury and Subarachnoid Hemorrhage*
A Clinical, Pathological, and Physiological Correlation

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Electrocardiographic (ECG) abnormalities associated with central nervous system disorders are well known, having been reported with such diverse conditions as status epilepticus, subarachnoid hemorrhage, meningitis, cerebral infarction, and intracranial mass lesions. A variety of ECG alterations have been reported, seen most consistently with subarachnoid and intracerebral hemorrhages. The most common abnormalities are depression or elevation of ST segments, prolongation of Q-T intervals, and inversion of T waves.

No pathological changes in the heart were described until 1964 when Koskelo, et al., reported three cases of subarachnoid hemorrhage with ECG changes, and mentioned several small subendocardial petechial hemorrhages seen at postmortem. No other cardiac abnormalities were noted, and the pathogenesis of the electrographic abnormalities has remained unclear.

We have seen a variety of structural changes in hearts of patients dying with subarachnoid hemorrhage, several of whom had abnormal ECG's, and we have produced similar changes experimentally in cats. In this communication we will present our clinical material, the experimental results, and some comments on the pathology of this heart lesion.

Case Reports

Case 1. A 56-year-old white woman had a subarachnoid hemorrhage on January 2, 1966, with residual left hemiparesis. The ECG on January 2, 1966, was normal. The patient was recovering from her neurological deficit when she bled again on February 2, 1966, and died approximately 18 hours later. No ECG was done at the time of the second bleeding episode.

Postmortem examination revealed a right middle cerebral artery aneurysm with infarction in the distribution of the middle cerebral artery. Examination of the heart showed no gross changes, but microscopic sections revealed multifocal areas of acute myocardial cell injury. Dense eosinophilic transverse bands replaced the normal striated appearance of the cytoplasm (myofibrillar degeneration), and was unassociated with an inflammatory reaction (Fig. 1).

This patient died 18 hours after the second subarachnoid hemorrhage. The changes in myocardium are typical of those seen in other patients dying acutely with subarachnoid hemorrhage, and show disruption of myocardial cell architecture without infiltration by inflammatory cells or macrophages.

Case 2. The sudden onset of subarachnoid hemorrhage in this 14-year-old girl was accompanied by coma, decerebration, a blood pressure of 182/130, fixed dilated pupils, and massive pulmonary edema. Despite her grave condition she responded dramatically to 40 mg intravenous Ritalin and medical therapy for pulmonary edema. Twenty-four hours following the initial hemorrhage the patient was awake, moving all extremities well, speaking coherently, and able to recognize her family. Five hours later she had a second hemorrhage. She died 2½ days following the first hemorrhage. No ECG was performed.

Autopsy showed subendocardial hemorrhage extending 1 mm into the left ventricle. The brain showed a ruptured anterior communicating artery aneurysm with blood both in the interhemispherical fissure and filling the
basal cisterns around the hypothalamus and brain stem. A large left frontal lobe intracerebral hematoma communicated with the subarachnoid hemorrhage and had ruptured into the right lateral ventricle. Microscopic examination of the hypothalamus revealed scattered perivascular hemorrhage in the anterior septal region, and occasional perivascular lymphocytic cuffs.

Sections of the heart revealed a large subendocardial area of interstitial hemorrhage and acute inflammatory cells surrounding unremarkable myocardial cells. In the wall of the left ventricle were multiple small scattered foci of myocardial cell injury, manifested by myofibrillar degeneration and infiltration of the necrotic debris by mononuclear cells (Fig. 2). The cardiac lesion was thought to represent an evolving acute change and the infiltration with macrophages and mononuclear cells consistent with a lesion 2½ days old. The subendocardial hemorrhage was probably related to the second and fatal hemorrhage.

Case 3. A 49-year-old white woman collapsed at home and was seen shortly thereafter in the emergency room where she had bilateral pulmonary edema, bilateral Babinski signs, a dilated left pupil, and a mild right hemiparesis. Subarachnoid hemorrhage was confirmed by lumbar puncture. Arteriography showed two aneurysms, one of the left middle cerebral artery and the other of the right pericallosal artery; it also suggested a basilar artery aneurysm.

The ECG’s done on admission and the day of surgery showed peaked T-waves and “subendocardial ischemia.” Frontal craniotomy was performed and both aneurysms inspected. Neither appeared to have bled.

Five days postoperatively the patient failed rapidly, developed a right hemiparesis, and became semicomatose. The ECG at this time showed premature ventricular contractions and Q waves diagnostic of apical diaphragmatic myocardial infarction (Fig. 3). Subsequent weekly serial tracings confirmed this impression. Her neurological condition continued to deteriorate, and she died 14 days following the first subarachnoid hemorrhage and 1 week following the second. A series of SGOT, LDH, and serum electrolyte studies were normal.

Postmortem examination revealed pulmonary edema and hepatic congestion. In addition to the aneurysms previously shown, there was a ruptured aneurysm at the bifurcation of the basilar artery, which had bled upward into the posterior third ventricle, distending the third ventricle, and filling the entire ventricular system with clot.

Microscopic examination of the posterior hypothalamus showed neurons undergoing acute change in the posterior hypothalamus,