Electrocardiographic Changes and Myocardial Damage in Patients with Acute Cerebrovascular Accidents

JACOB DIMANT, M.D. AND DAVID GROB, M.D.

SUMMARY In 100 consecutive patients with acute cerebrovascular accident, due to cerebral thrombosis in 72, cerebral hemorrhage in 12, embolus in 6, and subarachnoid hemorrhage in 10, there were 90 who had electrocardiographic abnormalities during the first three days after admission, compared to 50% in a control group. The patients with cerebrovascular accident had a 7- to 10-fold higher incidence of ST segment depression, prolonged QTc interval and atrial fibrillation, and a 2- to 4-fold higher incidence of T wave inversion, conduction defects, premature ventricular beats and left ventricular hypertrophy. Patients who died had a 2-, 3- and 5-fold higher incidence of electrocardiographic evidence of recent myocardial infarction, atrial fibrillation and conduction defects than those who survived, but these changes occurred in only 5, 21 and 14% of all patients, and other electrocardiographic changes could not be correlated with mortality.

IT IS WELL KNOWN that patients who have cerebrovascular accidents have an increased incidence of electrocardiographic abnormalities.1 This is due mainly to the association of arteriosclerotic cerebral and coronary artery disease, which have common risk factors. In addition, arrhythmias, myocardial infarction, and bacterial endocarditis may cause cerebrovascular accidents as a result of embolism or hypotension, and cerebrovascular accidents, particularly those due to cerebral or subarachnoid hemorrhage, may be followed by electrocardiographic changes, with post-mortem evidence of myocardial damage. In the present report, the incidence and significance of electrocardiographic changes in patients with cerebrovascular accident have been studied by recording the electrocardiogram and serum level of enzymes which may be derived from cardiac muscle, including creatine phosphokinase (CPK), hydroxybutyrate dehydrogenase (HBDH), lactic dehydrogenase (LDH), and glutamic-oxaloacetic transaminase (GOT).

During the first three days after admission 29 patients had elevation of serum enzymes which may be derived from cardiac muscle, particularly CPK, which was increased 6-fold, compared to 2-fold increases in HBDH, GOT, and LDH. Only 5 of these patients had electrocardiographic evidence of recent myocardial infarction. Patients with elevated serum CPK had a 2-fold higher incidence of ST segment depression, T wave inversion, conduction defects and atrial fibrillation than those with normal CPK, and a mortality of 66%, compared to 30%. Of 41 patients who died, 49% had elevated serum CPK, compared to 15% of 59 patients who survived. These differences were significant ($P < 0.01$). Serum CPK was more frequently helpful than the electrocardiogram in evaluating the extent of cardiac damage and in predicting mortality. Patients with acute cerebrovascular accident should have repeated evaluation of serum CPK and the ECG, and be monitored for arrhythmias.

Results

Historical Factors

The patients with cerebrovascular accident had a higher incidence of history of cerebrovascular disease (29-fold), diabetes and ischemic heart disease (2- and 3-fold), and of hypertension and hypercholesterolemia (4- and 5-fold) than the control group (table 1).

Electrocardiographic Changes

Ninety of 100 patients with cerebrovascular accident had an abnormal electrocardiogram at the time of admission, compared to 50 patients in the control group. The patients with cerebrovascular accident had a 7- to 10-fold higher incidence of ST segment depression, prolonged QTc interval and atrial fibrillation, and a 2- to 4-fold higher incidence of T wave inversion, conduction defects, premature ventricular beats and left ventricular hypertrophy (by voltage criteria) (table 2). Five of the patients with cerebrovascular accident had electrocardiographic evidence of a recent myocardial infarction, including the appearance of Q waves and elevation of ST segments. Five patients had tall T waves and 4 had U waves, despite normal serum levels of potassium, compared to none in the control group. The incidence of non-specific ST-T wave changes and of evidence of old myocardial infarction was the same in the two groups. There were no striking differences between the electrocardiogram of patients with cerebral thrombosis, cerebral or subarachnoid hemorrhage, and cerebral embolus, except that the latter had a higher incidence of atrial fibrillation (100%).

Serum Enzymes

Twenty-nine of 100 patients with cerebrovascular accident had serum levels of CPK above the upper limit of normal of...
130 units, ranging from 145 to 2373 (median 306) units. (table 3). These included 20 of 72 patients with cerebral thrombosis, 2 of 10 with subarachnoid hemorrhage, 6 of 12 with cerebral hemorrhage, and 1 of 6 with cerebral embolus. The remaining 71 patients had serum levels of CPK between 2 and 130 (median 48) units. Other enzymes which may arise from cardiac muscle (HBDH, GOT and LDH) were also elevated in the group of patients with elevated serum CPK, though to a lesser degree (2 times the upper limit of normal or the level in patients with normal CPK). Elevated serum levels of CPK were not encountered in the control group.

Relation of Serum CPK to Electrocardiographic Changes

The 29 patients with elevated serum CPK had the same incidence of abnormal electrocardiogram (93%) as the 71 patients with normal levels (89%) (table 4). However, the incidence of ST segment depression, T wave inversion, conduction defects and atrial fibrillation in patients with elevated serum CPK was twice that in patients with normal levels. The five patients with electrocardiographic evidence of recent myocardial infarction all had elevated serum levels of CPK, but these were no higher than in other patients with elevated levels.

Mortality

Forty-one of 100 patients with cerebrovascular accident died during the period of hospitalization, 4 to 68 (mean 12) days after admission. These included 20 of 72 patients with cerebral thrombosis, all of 12 with cerebral hemorrhage, 6 of 12 with subarachnoid hemorrhage, and 3 of 6 with cerebral embolus. In 13 patients the cause of death appeared to be cerebral, in 11 cardiac, in 6 respiratory infection, and in 3 pulmonary embolism, while in 8 patients the final event was not clear. Mortality from cerebral causes usually occurred during the first week of hospital stay, whereas mortality due to infection or pulmonary embolism usually occurred later. Mortality from cardiac causes occurred with the same incidence during the first week or later.

Relation of Electrocardiographic Changes to Mortality

Of the 41 patients with cerebrovascular accident who died, 39 (95%) had an abnormal electrocardiogram on admission, compared to 86% of the patients who survived, a difference that was not significant (table 5). Those who died had a 2-, 3- and 5-fold higher incidence of electrocardiographic evidence of recent myocardial infarction, atrial fibrillation and
Levels of Creatine Phosphokinase (CPK) in 100 Patients with Prolonged QT c; interval

<table>
<thead>
<tr>
<th>Table 4</th>
<th>Relation of Electrocardiographic Changes to Serum Levels of Creatine Phosphokinase (CPK) in 100 Patients with Cerebrovascular Accident</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal CPK</td>
<td>Elevated CPK</td>
</tr>
<tr>
<td>Number of patients</td>
<td>71</td>
</tr>
<tr>
<td>Abnormal electrocardiogram</td>
<td>63 (89%)</td>
</tr>
<tr>
<td>Left ventricular hypertrophy</td>
<td>17 (24%)</td>
</tr>
<tr>
<td>Prolonged QTc; interval</td>
<td>11 (15%)</td>
</tr>
<tr>
<td>Significant ST segment depression</td>
<td>13 (18%)</td>
</tr>
<tr>
<td>T wave inversion (more than one lead)</td>
<td>18 (25%)</td>
</tr>
<tr>
<td>Tall T waves</td>
<td>2 (3%)</td>
</tr>
<tr>
<td>U waves</td>
<td>2 (3%)</td>
</tr>
<tr>
<td>Evidence of old myocardial infarction</td>
<td>10 (14%)</td>
</tr>
<tr>
<td>Conduction defects</td>
<td>8 (11%)</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>11 (15%)</td>
</tr>
<tr>
<td>Premature ventricular beats</td>
<td>9 (13%)</td>
</tr>
<tr>
<td>Evidence of recent myocardial infarction, including appearance of Q waves</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Died in hospital</td>
<td>22 (30%)</td>
</tr>
</tbody>
</table>

Conduction defects than those who survived, but these changes occurred in only 5, 21 and 14% of all patients, and other electrocardiographic abnormalities could not be correlated with mortality. Two of 10 patients with normal electrocardiograms on admission died.

Relation of Serum CPK to Mortality

Of the 29 patients with cerebrovascular accident who had elevated serum CPK on admission, 66% died, compared to 30% of the 71 patients with normal serum levels (tables 4 and 5). Of the 41 patients with cerebrovascular accident who died, 49% had elevated serum CPK, compared to 15% of the 59 patients who survived. Of the 20 patients with cerebral thrombosis who died, 55% had elevated serum CPK, compared to 17% of the 52 patients who survived. These differences were all significant (p < 0.01). Of the 12 patients with cerebral hemorrhage, all of whom died, 6 (50%) had elevated serum CPK. Of the 6 patients with subarachnoid hemorrhage who died, 2 (33%) had elevated serum CPK, compared to none of the 4 patients who survived. Of the 3 patients with cerebral embolus who died, one (33%) had elevated serum CPK, compared to none of the 3 patients who survived.

Illustrative Patient J.B.

A 26 year old white female, who had previously been in good health, was brought to the emergency room in coma. Three hours previously she had complained of pain and stiffness of the neck, and soon afterward vomited and then became comatose. Physical examination showed an unconscious female with decerebrate rigidity and posture who did not respond to painful stimuli. Blood pressure was 200/100. Respiration was shallow and slow. Pupils were fixed and dilated. The Babinski reflex was positive bilaterally. Spinal tap revealed grossly bloody fluid which did not clear in successive tubes and which had a xanthochromic supernatant. The electrocardiogram on admission (fig. 1) showed sinus tachycardia, with a rate of 130/min., normal axis, prolonged QTc; interval, tall peaked T waves, slight ST segment depression in leads 2, 3, and aVF and small, 3 mm Q waves in these leads, which may be normal for this age. On the second and third day of hospitalization, the electrocardiogram did not change except for inversion of the T wave in lead V6. On the first day of illness, the serum CPK was 408 units (upper limit of normal 130) and HBDH 3640 (upper limit of normal 330). The serum GOT and LDH were 30 and 180 on the first day, and rose to 45 and 250 units on the second day. The patient remained comatose. Endotracheal intubation was performed and respiration had to be supported mechanically after the first hospital day. The patient was not hypotensive or anoxemic at any time, and required no vasopressor drug. On the third hospital day, after brain death was confirmed by flat electroencephalograms, the kidneys were removed for transplantation. Postmortem examination showed a ruptured intracranial aneurysm and subendocardial hemorrhages in the myocardium.

Illustrative Patient M.T.

A 75-year-old white woman was admitted in coma. She had struck her head one day before and had progressively increasing headache during the day. Over a period of several hours she gradually lapsed into coma. There was a history of hypertension for 5 years. For several years she had experienced occasional syncopal episodes and drop attacks. Physical examination on admission showed the patient to be completely comatose, unresponsive, flaccid and areflexic. Pupils were fixed and widely dilated. The Babinski reflexes were negative. The patient became apneic and was promptly intubated and mechanically ventilated. Spinal tap showed an opening pressure of 150 mm H2O and grossly bloody fluid which did not clear. Blood pressure was 140/90 on admission and was within normal limits until shortly before death. The heart was enlarged on physical examination and there was a small infiltrate at both lung bases. Electrocardiogram on admission (fig. 2) revealed normal sinus rhythm, non-specific ST segment depression in leads II, aVF, V4 and V5, and the presence of U waves. On the next day, there was slight ST segment elevation in leads I and aVL with slight ST segment depression in leads II, aVF, V3, V4 and V5, slight widening of QRS, and premature atrial
### TABLE 5
Incidence of Electrocardiographic Changes and Elevated Serum Levels of Creatine Phosphokinase (CPK) in 100 Patients with Cerebrovascular Accident, and Relation to Mortality

<table>
<thead>
<tr>
<th>Number of Patients</th>
<th>Cerebrovascular Accident</th>
<th>Cerebral Thrombosis</th>
<th>Cerebral Hemorrhage</th>
<th>Subarachnoid Hemorrhage</th>
<th>Cerebral Embolus</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>All</td>
<td>Surv.</td>
<td>Died</td>
<td>All</td>
<td>Surv.</td>
</tr>
<tr>
<td>Total Number of Patients</td>
<td>100</td>
<td>59</td>
<td>41</td>
<td>72</td>
<td>52</td>
</tr>
<tr>
<td>Elevated serum CPK</td>
<td>29</td>
<td>9</td>
<td>20</td>
<td>20</td>
<td>9</td>
</tr>
<tr>
<td>Normal Electrocardiogram</td>
<td>10</td>
<td>8</td>
<td>2</td>
<td>8</td>
<td>6</td>
</tr>
<tr>
<td>Abnormal Electrocardiogram</td>
<td>90</td>
<td>51</td>
<td>39</td>
<td>64</td>
<td>46</td>
</tr>
<tr>
<td>Left Ventricular Hypertrophy</td>
<td>23</td>
<td>13</td>
<td>10</td>
<td>17</td>
<td>13</td>
</tr>
<tr>
<td>Prolonged QT Interval</td>
<td>14</td>
<td>7</td>
<td>7</td>
<td>9</td>
<td>7</td>
</tr>
<tr>
<td>Significant ST Segment Depression</td>
<td>22</td>
<td>10</td>
<td>12</td>
<td>14</td>
<td>9</td>
</tr>
<tr>
<td>T Wave Inversion (more than one lead)</td>
<td>31</td>
<td>18</td>
<td>13</td>
<td>22</td>
<td>16</td>
</tr>
<tr>
<td>Non-Specific ST-T Changes</td>
<td>22</td>
<td>11</td>
<td>9</td>
<td>18</td>
<td>13</td>
</tr>
<tr>
<td>Tall T Waves</td>
<td>5</td>
<td>2</td>
<td>3</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>U Waves</td>
<td>4</td>
<td>0</td>
<td>4</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Evidence of Old Myocardial Infarction</td>
<td>14</td>
<td>10</td>
<td>4</td>
<td>10</td>
<td>9</td>
</tr>
<tr>
<td>Evidence of Recent Myocardial Infarction</td>
<td>5</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>Conduction Defects</td>
<td>14</td>
<td>3</td>
<td>11</td>
<td>9</td>
<td>3</td>
</tr>
<tr>
<td>Atrial Fibrillation</td>
<td>21</td>
<td>7</td>
<td>14</td>
<td>10</td>
<td>4</td>
</tr>
<tr>
<td>Premature Ventricular Beats</td>
<td>13</td>
<td>10</td>
<td>3</td>
<td>10</td>
<td>9</td>
</tr>
</tbody>
</table>

**FIGURE 1.** Electrocardiogram of patient J.B. on admission.
The patient developed paroxysmal supra-ventricular tachycardia. The serum enzyme levels determined on the first and second hospital days were CPK 115 and 528 units, HBDH 1,086 and 1,130 units, LDH 250 and 750 units, and SGOT 50 and 135 units. The blood pressure fell at the end of the third hospital day, responding only transiently to vasopressor drugs, and the patient died shortly thereafter. Postmortem examination showed the heart to be enlarged, weighing 420 grams, with left ventricular enlargement. There was scattered fibrosis, and areas of myocytolysis (fig. 4). The lungs showed atelectasis at both bases. The brain showed an extensive hemorrhage which had arisen in the cerebellum from rupture of an arteriosclerotic artery.

**Discussion**

The interrelationship of cerebrovascular disease and cardiovascular disease has been repeatedly emphasized. They have common risk factors, such as hypertension, diabetes, hyperlipidemia, and cigarette smoking, and frequently coexist. Decompensation in one system may adversely affect the other, whether or not the patient has recognized disease of both systems. Cardiac disease may cause acute cerebral manifestations in several ways. Three to 8% (in the current study 6%) of acute cerebrovascular accidents are due to cerebral emboli, usually arising from a fibrillating auricle, or less often from a mural thrombus on a myocardial infarct or from endocarditis. Acute myocardial infarction may be followed by transient or permanent neurologic damage as a result of acute reduction of cerebral blood flow due to hypotension or arrhythmia. Heart block, bradyarrhythmias or tachyarrhythmias may cause transient cerebral symptoms or the Adams-Stokes syndrome. On the other hand, it is well known that patients with acute cerebrovascular accidents have a high incidence of electrocardiographic changes and of arrhythmias. In the current study 90% of such patients had an abnormal electrocardiogram, compared to 50% of a control group. The incidence of ST segment depression, prolonged Q-Tc interval and atrial fibrillation was 7- to 10-fold higher, and of T wave inversion, conduction defects, premature ventricular beats and left ventricular hypertrophy 2- to 4-fold higher. The association of cerebrovascular accident with myocardial infarction has been repeatedly described, though only 5% of the patients with cerebrovascular accident in this study had electrocardiographic changes compatible with recent myocardial infarction. In the other patients with cerebrovascular accident who had an abnormal electrocardiogram, there was no electrocardiographic change that was not present in a smaller proportion of patients in the control group, with the exception of tall peaked T waves in 5 patients and U waves in 4, which were not present in the control group. However, these changes were not sufficiently frequent or distinctive to be of diagnostic help. Patients who died had a 2-, 3- and 5-fold higher incidence of electrocardiographic evidence of recent myocardial infarction, atrial fibrillation and

**Figure 2.** Electrocardiogram of patient M.T. on admission.
conduction defects than those who survived, but these changes occurred in only 5, 21 and 14% of all patients, and other electrocardiographic changes could not be correlated with mortality. Therefore, the electrocardiogram of patients with cerebrovascular accident obtained on admission was usually of limited value in predicting mortality.

In view of the advanced age of the patients and common risk factors for cerebrovascular and cardiovascular disease, it is difficult in patients with cerebral thrombosis, hemorrhage or embolus to distinguish between pre-existing electrocardiographic changes and changes which might indicate cardiac damage resulting from the cerebrovascular accident. In the younger patients with subarachnoid hemorrhage due to berry aneurysm or arteriovenous malformation, the electrocardiographic changes that were observed can be more readily related to cerebral damage. These included ST segment depression in four patients, T wave in-
version in two, prolonged Q-T, interval in two, tall T waves in three and U waves in one. These changes, and, rarely, ST segment elevation, have been previously described.1, 2, 3 In most patients who die of subarachnoid hemorrhage, including those with severe electrocardiographic changes detected prior to the occurrence of hypotension or anoxemia, the heart has been normal at post-mortem examination. This had led to the suggestion that the electrocardiographic changes may be neurogenic in origin. Electrocardiographic changes and arrhythmias have been observed following manipulation of the circle of Willis during surgical procedures,4 and, in experimental animals following stimulation of certain areas of the central nervous system,5-8 the vagus nerve,9 or the stellate ganglion,9 or following catecholamine infusion.10-12 In some experiments the changes could be prevented by cervical cord transection,13, 14 vagal block,15 or propranolol administration,16 and were attributed to interplay between sympathetic and vagal stimulation of the heart. On the other hand, some patients who died of subarachnoid hemorrhage were found to have subendocardial hemorrhages and focal areas of myocardial cell injury and myocytolysis.17-21 The pathologic findings could not be correlated with the presence of electrocardiographic changes.1 One patient had Q waves in the electrocardiogram, but autopsy revealed diffuse myocardial damage rather than myocardial infarction. Subendocardial hemorrhages have been reported in 40%,20 and epicardial hemorrhages or myocytolysis in 8% of patients who died from a variety of intracranial lesions. Similar histologic findings have also been reported following intracranial hemorrhage,22-24 stimulation of the brain,22 vagus,22 or stellate ganglion,25 or catecholamine infusion,26, 27 in experimental animals. Acute myocardial infarction has been reported to occur following prolonged bilateral stimulation of the hypothalamus in cats.22-24 These observations provide evidence that pathologic changes in the myocardium, as well as electrocardiographic changes, may result from cerebral injury and may be mediated by the autonomic nervous system. That the electrocardiographic changes observed in 8 of the 10 patients with subarachnoid hemorrhage might reflect myocardial damage was suggested by elevation of serum CPK and subendocardial hemorrhages or myocytolysis in 2 of these patients. A previous study reported elevation of serum CPK and SGOT in 8 of 20 patients with subarachnoid hemorrhage, and electrocardiographic changes in 6 of the 8.28

The level of serum enzymes that may arise from the heart was more frequently helpful than the electrocardiogram in evaluating the extent of cardiac damage and in predicting mortality in patients with cerebrovascular accident, regardless of cause. Of these enzymes, the serum level of CPK has proved to be the most useful indicator of myocardial damage.29 Serum CPK arises almost entirely from cardiac or skeletal muscle, and minor elevation may follow intramuscular injections or other trauma to muscle. While smaller amounts of CPK are present in the brain,30 the elevation of CPK in the serum that may occur following acute cerebral infarction30 has been identified as muscle rather than brain isoenzyme.31 It is not accompanied by elevation of CPK in the spinal fluid,31 and is not believed to arise from the brain.32 Furthermore, intravenous administration to dogs of CPK isoenzyme which arises from the brain was followed by its rapid disappearance from the blood.33 Serum LDH and SGOT are derived not only from cardiac and skeletal muscle, but also from liver and other tissues, and are less helpful as indicators of myocardial damage. Elevation of α-HBDH, which represents LDH isoenzymes LDH1 and LDH2, may be helpful in the detection of myocardial injury, particularly when accompanied by an elevated ratio of HBDH to LDH. However, this may also occur in other diseases such as megaloblastic anemia, renal infarction, liver disease, and some myopathies and malignancies,34 and is, therefore, less helpful as an indicator of myocardial damage than elevation of serum CPK.

Of 100 patients with acute cerebrovascular accident, 29% had serum levels of CPK that were above the upper limit of normal, with a median level 2.5 times the upper limit of normal and 6 times the level in patients with normal CPK. Serum HBDH, GOT and LDH were increased in these patients to a median level 2 times the upper limit of normal or the level in patients with normal CPK. Only 5 of the 29 patients had electrocardiographic evidence of recent myocardial infarction. In the remainder, the electrocardiographic changes were qualitatively similar to those in patients with normal CPK, but there was a two-fold higher incidence of ST segment depression, T wave inversion, conduction defects and atrial fibrillation. The mortality of the 29 patients with elevated serum CPK (66%) was over twice that of patients with normal CPK (30%), and in the 41 patients who died, the incidence of elevated serum CPK (49%) was three times that in patients who survived (15%), regardless of whether the cerebrovascular accident was due to cerebral thrombosis or embolus or to subarachnoid hemorrhage. While the terminal event appeared to be cardiac in only one-fourth of the patients, the precise cause of death was not always clear, and the occurrence of elevated serum CPK in half the patients who died suggests that myocardial damage may have been a contributory factor in many of these patients. Electrocardiographic evidence of recent myocardial infarction, conduction defects, or atrial fibrillation indicated a poorer prognosis,35 but the serum level of CPK was more frequently helpful than the electrocardiogram in predicting mortality. It is evident that every patient with cerebrovascular accident, whether due to thrombosis, hemorrhage or embolus, should have repeated evaluation of serum CPK and of the electrocardiogram, as a guide to prognosis and management, and should be monitored for arrhythmias, preferably in an acute care unit.

References

7. Sensenbach W: Some common conditions, not due to primary heart dis-
ECG CHANGES IN CBVD ACCIDENTS/Diman & Grob

37. Kortiweg GCJ, Boeles JTF, TenCate J: Influence of stimulation of some
subcortical areas on the electrocardiogram. J Neurophysiol 20: 100–107,
1957
38. Weinberg SJ, Foster JM: Electrocardiographic changes produced by
40. Byer E, Toth L, Ashman R: The electrocardiographic changes induced by
cooling or warming the inner surface of dog's ventricle. Am J Physiol 149:
264–268, 1947
41. Yanowitz F, Preston JB, Abildskov JA: Functional distribution of right
and left stellate innervation to the vessels: Production of neurogenic
electrocardiographic changes by unilateral alteration of sympathetic
42. Chappell CC: Comparison of cardio toxic action of certain sympathetic
43. Rasb W: Key position of catecholamines in functional and degenerative
44. Barger AC, Liebowitz MR, Herda J: Chronic catherization of the cor-
nary artery: infusion of autonomic drugs in the unanesthetized dog. Fed
45. Porter RW, Kinsakah K, Greenhoot JH: Persistent electrocardio-
graphic abnormalities experimentally induced by stimulation of the
46. Mauck HP, Hockman CH, Hof F: ECG changes after cerebral stimulation.
I. Anomalous atrioventricular excitation elicited by electro-
trical stimulation of mesencephalic reticular formation. Am Heart J 68:
99–101, 1964
47. Manning JW, Cotten MD: Mechanisms of cardiac arrhythmias induced
48. Hockman CH, Mauck HP, Hof F: ECG changes resulting from cere-
bral infarction. II. Spectrum of ventricular arrhythmias of symp-
49. Drory Y, Ousakhine G, Kotary IZ, Kellerman JJ: Electrocardiographic
findings in brain death: Description and presumed mechanism. Chest 67:
425–432, 1975
50. Burch GE, Sun SC, Colloough HL, Desaupasque NP, Sohal RS: Acute
myocardial lesions following experimentally induced intracranial hemor-
51. Manning GW, Hall GE, Bank FG: Vagus stimulation and the produc-
52. Kaye MP, McDonald KH, Randel WC: Systolic hypertension and sub-
endocardial hemorrhages produced by electrical stimulation of the stel-
53. Bloom S, Cancilla A: Myocytolysis and microchondrial calcification in rat
myocardium after low dose of isoproterenol. Am J Pathol 54: 373–381,
1969
54. Wester BC, Judd JT, Kittinger GW: Myocardial necrosis induced by is-
55. Melville KI, Blum B, Shister HE, Silver MD: Cardiac ischemic changes
and arrhythmias induced by hypoalimentation. Am J Cardiol 22:
781–791, 1963
56. Hunt D, McRae C, Zapf P: Electrocardiographic and serum enzyme
changes in subarachnoid hemorrhage. Am Heart J 77: 479–488, 1969
57. Sohal BE, Shell WE: Serum enzyme determination in the diagnosis and
58. Madsen A: Creative phosphokinase isoenzymes in human tissue with spec-
59. Acheson J, James DC, Hutchinson EC, Westhead R: Serum creatine
60. Dubo M, Park DC, Pennington RJT, Kulpag RM, Walton JV: Serum
creatine kinase in cases of stroke, head injury or meningitis. Lancet 2:
743–748, 1967
61. Lisak RP, Craig FA: Lack of diagnostic value of creatine phospho-
62. Cohen L: Serum enzyme determinations: Their reliability and value. Med
phosphokinase activity in dogs following intravenous injection of the
64. Rosalki SB, Wilkinson JH: Serum alpha-hydroxybutyrate dehydro-
genase in diagnosis. JAMA 189: 61–63, 1964
Electrocardiographic changes and myocardial damage in patients with acute cerebrovascular accidents.
J Dimant and D Grob

Stroke. 1977;8:448-455
doi: 10.1161/01.STR.8.4.448
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1977 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/8/4/448