Electrocardiographic Patterns in Patients with Cerebrovascular Accidents

By Vagn Fentz, M.D., and Joins Gormsen, M.D.

Electrocardiographic changes without demonstrable cardiac cause have been found in some patients with cerebrovascular accidents.1-5 These changes have usually affected the T wave, which is flattened and often inverted, not infrequently of a typically coronary shape. Large, positive T waves have also been described. The T-wave alterations are most clearly demonstrable in leads I, aV_{1}, and V_{4} to V_{6}. In addition, there have been ischemic changes in the RS-T segments, prolongation of the Q-T interval, and large U waves. Thus, the electrocardiographic changes may resemble those seen in acute myocardial infarction, and misinterpretation has led to a delay in operative treatment of subarachnoid hemorrhage.2,5

These changes have been reported predominantly in patients with subarachnoid hemorrhage, but also in a few patients with other forms of intracranial bleeding. They have also been described in one patient with cerebral thrombosis diagnosed on the basis of focal electroencephalographic abnormalities4 and in three patients (of a group of 17) in whom the neuropathologic diagnoses, based solely on clinical findings, were given as parenchymal hemorrhage or arterial thrombosis.3

The most important cause of electrocardiographic changes in patients with cerebral accidents is of course heart disease, which is very common in these cases—and often a provocative factor.6 Frequently, it is difficult to be sure that electrocardiographic changes are not due to heart disease, possibly latent, but rendered manifest by anoxia. Other extracerebral causes of circulatory, metabolic, or respiratory nature, due to shock, loss of fluid or electrolytes, changes in blood pressure, hepatic or renal disease, and accumulation of secretion in the air passages must be considered. It is not until these conditions have been ruled out as a cause of the electrocardiographic changes that a cerebral cause can be looked for. The electrocardiographic changes demonstrated in patients with cerebral accidents without heart disease might be imagined to be due to local anoxia or to simple mechanical irritation of cortical areas 13 and 24 (the orbital surface of the frontal lobe and the anterior gyrus cinguli), since stimulation of these centers is known to influence cardiac function.2,5,7,8

The mentioned series have been small, and definitive neuropathologic assessment is lacking in the few cases with nonhemorrhagic cerebral accidents. We are therefore presenting studies on a fairly large number of patients with acute cerebral infarct and a smaller number with intracranial hemorrhage.

Results

Acute Cerebral Infarction

The electrocardiographic findings are presented in 69 cases of acute cerebral infarction in which the diagnosis was based on medical-neurologic-angiographic-encephalographic studies.8 A priori, we excluded all patients with severe heart disease, since in these cases

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Patients with Acute Cerebral Infarction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Electrocardiogram</td>
<td>Abnormal but explicable</td>
</tr>
<tr>
<td>Carotid arteriography</td>
<td>Number of cases</td>
</tr>
<tr>
<td>Normal</td>
<td>16</td>
</tr>
<tr>
<td>Oclusion</td>
<td>11</td>
</tr>
<tr>
<td>Atherosclerosis only</td>
<td>24</td>
</tr>
<tr>
<td>Retarded blood flow</td>
<td>3</td>
</tr>
<tr>
<td>Not performed</td>
<td>15</td>
</tr>
<tr>
<td>Total</td>
<td>69</td>
</tr>
</tbody>
</table>

From the Copenhagen County Hospital, Hellerup, Denmark.
Case no. 6, a 75-year-old man. Electrocardiogram (top) on day of admission for acute thrombosis of right internal carotid artery and (bottom) 7 days later. No history or autopsy evidence of organic heart disease.

Figure 1
the criteria for assessing new changes are uncertain. Thus all cases of atrial flutter or fibrillation, of present or past myocardial infarction, and of severe hypertensive electrocardiographic changes were omitted. We did not aim at any other systematic selection. Of the 69 patients, 12 died and 42 were discharged for outpatient observation. Fifteen were not in a condition to accept outpatient care.

All the patients had a history of acute neurologic loss, varying from mild, temporary, localizing signs to unconsciousness with widespread paralysis. The history was taken minutely on a special form, partly by questioning the patients' next-of-kin. All the patients were submitted to careful medical and neurologic examination and had routine laboratory tests (including determination of SGO-transaminase), lumbar puncture, electrocardiogram, and electroencephalogram. Moreover, the majority had carotid arteriography within a few days of the acute attack. Serum electrolytes were studied when the clinical appearances raised the suspicion of shifts, but these tests did not show material changes.

Table 1 shows the distribution of these 69 patients with acute cerebral infarction according to arteriographic and electrocardiographic findings. In most cases the electrocardiographic grouping was carried out on the basis of two sets traced in five (45 patients) or 12 (25 patients) leads at a minimum interval of one day; thus, the tables do not represent a single event. As a rule, the first electrocardiogram was taken on the day of admission, i.e., in most cases a few hours after the acute attack.

The electrocardiograms were assessed on the basis of all current criteria. The classifi-
Electrocardiograms in Patients with Acute Cerebral Infarction

<table>
<thead>
<tr>
<th>Electrocardiogram Abnormalities</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td></td>
</tr>
<tr>
<td>Developed posterior wall infarction pattern; No stenocardia</td>
<td></td>
</tr>
<tr>
<td>Gradually horizontally lowered; autopsy: no coronary occlusion</td>
<td></td>
</tr>
<tr>
<td>Lowered in further tracings; no stenocardia</td>
<td></td>
</tr>
<tr>
<td>Developed coronary T waves; no clinical sign of coronary occlusion</td>
<td></td>
</tr>
<tr>
<td>Developed posterior wall infarction pattern; coronary occlusion unlikely on clinical grounds; autopsy: no coronary occlusion or liver disease</td>
<td></td>
</tr>
<tr>
<td>Increasingly inverted, 3 weeks later again slightly positive; patient has chronic cor pulmonale but no cardiopulmonary complaints</td>
<td></td>
</tr>
</tbody>
</table>

Table 3

Patients with Intracranial Hemorrhage

<table>
<thead>
<tr>
<th>Electrocardiogram Abnormalities</th>
<th>Electrocardiogram Abnormal but explicable Abnormal Mean QTc</th>
</tr>
</thead>
<tbody>
<tr>
<td>Site of hemorrhage</td>
<td>Number of cases Normal Explicable Abnormal Mean QTc</td>
</tr>
<tr>
<td>Subarachnoid</td>
<td>5 2 1 2 0.45</td>
</tr>
<tr>
<td>Intracerebral</td>
<td>16 2 1 13 0.43</td>
</tr>
<tr>
<td>Total</td>
<td>21 4 2 15 0.43</td>
</tr>
</tbody>
</table>

As "normal electrocardiogram," we grouped tracings in which deviations did not exceed the normal range, whereas "abnormal but explicable electrocardiogram" signifies cases in which the alterations might be the result of heart disease, possibly of simultaneous coronary occlusion, suspected from an increase in the SGO-transaminase and clinical findings, or of fairly severe hypertension. In the absence of other likely cause, elevated SGO-transaminases must be interpreted in some cases as coming from infarcted brain tissue.

The last group, "abnormal electrocardiogram," thus comprises 11 cases in which extracerebral causes could not reasonably be held responsible for the changes present or arising during the course of the disease. A detailed tabulation of these 11 patients is given in table 2. Six became ambulatory, four died, and one was discharged without being able to accept outpatient care. The RS-T and T changes were completely or partially rever-

Circulation, Volume XXV, January 1962
possible. In half the cases the U wave disappeared, whereas the QTc changes often persisted.

**Intracranial Hemorrhages**

This group comprises 21 patients with non-traumatic intracranial bleeding confirmed by spinal fluid, arteriographic or postmortem findings. Table 3 gives the distribution of the series, the electrocardiographic findings being classified as in table 1. Of the five patients with subarachnoid bleeding three died, and of the 16 patients with intracerebral bleeding 10 died. Autopsy was performed in 10 of the 13 fatal cases.

Table 4 lists the 15 patients in whom extracerebral causes could not reasonably explain the electrocardiographic changes. In only one case (no. 19) was a pulmonary embolus demonstrated (or likely), and this patient survived for several days after the tracing. This table does not give electroencephalographic findings, since only a few of the hemorrhagic cases had this test.

**Discussion**

According to our findings a considerable number of patients with cerebrovascular accidents show electrocardiographic changes that are apparently inexplicable by cardiac or other extracerebral causes. In our series, these changes were considerably more common in patients with cerebral hemorrhage than in patients with primary infarctions, 71 as compared with 15 per cent.

The electrocardiographic changes often resemble the findings in acute coronary occlusion. There may be an alteration of the RS-T segment, usually a depression, and large inverted T waves. Prolongation of QTc exceeding 0.45 second was observed in five of 69 cases of infarction and in seven of 21 cases of hemorrhage. We did not observe very large or negative U waves. The changes of the RS-T
segments, T waves, and U waves were completely or partially reversible, whereas the change in QTc usually persisted (figs. 1-3).

Our series is too small to relate the findings to age, sex, electroencephalographic findings, site (most infarcts affect the left hemisphere), or prognosis. At a rough estimate, however, the electrocardiographic changes—considered together—appear to bear no relation to the arteriographic findings in cases of infarction.

Our studies seem to support the previous reports on electrocardiographic changes without demonstrable extracerebral explanation in patients with intracranial hemorrhage. Moreover, we have found that these changes may occur also in patients with acute cerebral infarction. Even when extracerebral causes of electrocardiographic abnormalities are considered, as in our series, a fairly large group of patients remains showing changes inexplicable according to the ordinary concepts. We are unable to explain why these changes are not found in all patients with cerebrovascular accidents; perhaps it is due to the marked variability in the site of the lesion. Some of the electrocardiographic changes simulate myocardial ischemia or even infarction; so there is reason to urge a particularly critical assessment of such electrocardiographic abnormalities in patients with cerebrovascular accidents.

Summary

In two series of 69 patients with acute cerebral infarction and 21 patients with intracranial hemorrhage, 11 and 15 respectively showed electrocardiographic changes that could not be ascribed to the usual cardiac or other extracerebral causes. These changes
often simulate myocardial ischemia or even infarction.

References

The Pulse Rate by Sanctorius

With the fame of Sanctorius as the discoverer of insensible perspiration, and with the inconceivable success of his aphorisms we have nothing to do, nor yet with his theories, or his morals, which seem not to have interfered with his appropriation of another man's inventions. . .

In his Commentary on Avicenna, he quotes Galen as to "the need to know the amount of departure from the natural state, which is only to be reached by conjecture." . . .

Then he describes what must have been the form of Galileo's pulsilogon. . .

Fig. 11 is a scale and a bullet marked with a central white line. We swing the pendulum and note the pulse with fingers. If the pendulum be faster than the pulse, we lengthen the line; if slower, we shorten it until they coincide. "Then," he says, "we keep this degree in mind until the next day and compare it with a new record. And so we can study the pulse of health and disease." Also he defines the values and defends the accuracy of the pendulum; but of Galileo, not a word.

Finally we are assured that "what other physicians acquire by conjecture concerning the pulse, we are able to attain unerringly by the infallible skill of the pulsilogon." What a comfort he must have found it!

When Sanctorius died, Sydenham was a boy of nine. There is not a pulse count among all those vigorous sketches which this great Englishman drew with a master's hand, and only once does Harvey speak of their number, which he says is from 1,000 to 4,000 in the half hour.—S. WEIR MITCHELL, M.D. Transactions of the Congress of American Physicians and Surgeons, Second Triennial Session held at Washington, D.C., 1891. New Haven, The Congress, 1892, p. 174.

Circulation, Volume XXV, January 1962
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Circulation. 1962;25:22-28
doi: 10.1161/01.CIR.25.1.22
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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