The Effect of Acute Stroke on Cardiac Functions as Observed in an Intensive Stroke Care Unit

BY SYLVAN LAVY, M.D., ISRAEL YAAR, M.D., ELDAD MELAMED, M.D., AND SHLOMO STERN, M.D.*

Abstract:
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Fifty-two stroke patients, 43 with cerebral ischemia and nine with cerebral hemorrhage, underwent continuous cardiac monitoring in an Intensive Stroke Care Unit shortly after the onset of the acute cerebrovascular accident. In the group of patients with no evidence of pre-existing heart disease, eight of 18 with cerebral ischemia and five of seven with hemorrhage developed ECG abnormalities. Additional ECG changes were noted in 21 of 25 patients with cerebral ischemia and two of two with hemorrhage who were known to have previous heart disease. Both disturbances in rhythm and conduction and "ischemic" ST-T alterations were detected and the frequency of the former exceeded that of the latter. The ECG alterations were transient in 32 patients and permanent in four. New electrocardiographical abnormalities in patients without evidence of heart disease prior to the stroke were associated with poorer prognosis. The pathogenetic mechanisms leading to the appearance of cardiac abnormalities in stroke patients are considered.

Additional Key Words
- cerebral ischemia
- cerebral hemorrhage
- electrocardiographical monitoring
- ischemic heart changes
- arrhythmic heart changes
- intensive stroke unit

Introduction

It is common knowledge that decrease in the cardiac output below a critical level, such as in heart failure, myocardial infarction and rhythm or conduction disturbances, may lead to the development of cerebral hemodynamic and metabolic abnormalities. However, the adverse effect of the brain on cardiac function is less widely recognized and appreciated.

Clinical experience shows that stroke victims may die unexpectedly of cardiac arrest, arrhythmia or severe hypotension in the absence of any pre-existing heart disease. Several authors1-14 reported electrocardiographical (ECG) abnormalities in patients with cerebrovascular accident. However, those were usually chance findings, based mainly on occasional ECG recordings.

Since the establishment of an Intensive Stroke Care Unit (ISCU) in the Department of Neurology, we had the opportunity of performing an intensive follow-up, by both neurologists and cardiologists, of stroke patients and to study among other data electrocardiographical alterations by continuous monitoring shortly after the onset of the cerebrovascular event, for at least 24 hours. The findings are presented in the following report.

Methods

Patients with acute cerebrovascular accidents admitted to the emergency ward of the Hadassah University Hospital in Jerusalem were immediately transferred to the ISCU attached to the Department of Neurology. The clinical diagnosis was confirmed by the history and physical and neurological examinations and supplemented by lumbar puncture, electroencephalogram and cerebral angiogram. On this basis the patients were classified into two categories: those who had cerebral ischemia (in the internal carotid or in the vertebrobasilar arterial system) and those with cerebral hemorrhage. Patients with subarachnoid hemorrhage were not included in this study. Patients in whom admission to the ISCU occurred more than six hours after the appearance of the acute stroke were also excluded.

A careful history of the patient’s cardiac condition prior to the stroke was obtained from the family and referring physician. Previous electrocardiograms of the patients were reviewed when available. A 12-lead ECG recording was performed in the emergency ward before the transfer of the patient to the ISCU.

Clinical evaluation of the cardiac status of the patient was as follows: (a) patients were regarded as having no heart disease prior to stroke if there was no previous history of cardiac disease and if the ECG performed recently or the one recorded in the emergency ward failed to show any abnormality. (b) Patients were considered as having a pre-
existing heart disease if a history of previous cardiac disease was obtained and/or when an ECG performed recently or the one recorded in the emergency ward exhibited abnormalities. Ischemic heart disease was diagnosed if the history disclosed previous anginal pains or a myocardial infarction and/or the ECG showed significant ST-T changes of the ischemic type. Cardiac dysrhythmia was diagnosed according to the history and/or to rhythm and conduction abnormalities disclosed on previous ECG.

In the ISCU the patients were kept under the close supervision of trained nurses or medical students for at least 24 hours. The medical personnel exercised all the functions of intensive coronary and respiratory care with repeated measurements every 15 minutes of heart rate, blood pressure and respiratory rate. Continuous ECG monitoring was performed by the use of an ECG monitoring system which displays the record constantly on its oscilloscope. A complete ECG recording was performed routinely every hour or at any time when changes in heart rate, rhythm, conduction or the ST segment and T waves were observed. Frequent arterial blood gas studies and electrolyte examinations were done. The neurological and cardiological condition of the patients was checked from time to time by one of the senior neurologists and cardiologists.

Patients were regarded as having new ischemic heart changes if the ECG exhibited significant ST segment depression of the ischemic type, lasting for more than 0.1 second with or without inversion of T waves, which did not exist before. Recent dysrhythmias were considered if abnormalities such as sinus bradycardia, supraventricular extrasystoles or tachycardia, atrial flutter or fibrillation, ventricular extrasystoles, nodal rhythm, nodal escape, nodal bradycardia, atrioventricular block, etc., appeared in the ECG.

The new electrocardiographical disturbances, ischemic or dysrhythmic, were regarded as transient if they cleared during the first 72 hours of hospitalization and as permanent if they remained unchanged during that period.

**Results**

Fifty-two consecutive patients with acute cerebrovascular accident admitted to our ISCU are included in the study. Twenty-six (50%) were males and 26 females. Forty-three patients were classified as having cerebral ischemia and nine had intracerebral hemorrhage. The age distribution of the patients ranged between 38 and 78 (table 1).

In 25 patients, 18 with cerebral ischemia and seven with hemorrhage, there was no evidence of heart disease prior to the stroke. In 27 patients with cerebral ischemia and two with hemorrhage, cardiac changes, ischemic and/or arrhythmic, were present before the development of the cerebrovascular accident (table 2).

During the first 24 hours of monitoring, ECG abnormalities of the ischemic and/or arrhythmic type developed in 29 patients (67%) with cerebral ischemia. In 18 patients with no evidence of previous cardiac disease, electrocardiographical changes were demonstrated in eight (44%), one of them had ischemic ECG pattern only, three had both ischemic and arrhythmic changes and four had arrhythmia only. In 21 (84%) of 25 patients with pre-existing cardiac disease, additional abnormalities were observed. Three of them exhibited ST-T changes only, one had both ischemic and dysrhythmic changes and nine had arrhythmia only (table 3). Among the seven hemorrhagic stroke patients without previous heart disease, five exhibited dysrhythmic electrocardiographical changes. The two patients with ischemic heart disease prior to the stroke showed further ECG disturbances — one had arrhythmia, and the other had both arrhythmia and ST-T changes (table 4).

Dysrhythmic abnormalities exceeded the ischemic heart changes in the patients suffering from cerebral ischemia and cerebral hemorrhage (tables 3 and 4). Many types of arrhythmias and conduction disturbances were noted: sinus bradycardia, multiple supraventricular extrasystoles, supraventricular tachycardia, paroxysmal atrial fibrillation, atrial flutter, nodal rhythm, nodal escape, nodal bradycardia, atrioventricular block, etc., appeared in the ECG.

The electrocardiographical abnormalities among the patients with cerebral ischemia were transient in 25 and permanent in four (three with ischemic and one with arrhythmic changes). In the patients with cerebral hemorrhage, seven exhibited transient ECG changes. There was no permanent change in that group (table 5).

By correlating the appearance of ECG abnormalities to the assumed area of the ischemic stroke, it could be demonstrated that they developed in every patient with a cerebrovascular accident in the distribution of the vertebrobasilar arterial system (brain stem...
EFFECT OF ACUTE STROKE ON CARDIAC FUNCTIONS

TABLE 3
Heart Condition Prior to Stroke (Ischemic Stroke, n = 43)

<table>
<thead>
<tr>
<th>ECG changes during the first 24 hours</th>
<th>Arrhythmia</th>
<th>Ischemia</th>
<th>Arrhythmia and ischemia</th>
<th>Total</th>
<th>No change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal heart (n = 18)</td>
<td>4</td>
<td>1</td>
<td>3</td>
<td>8</td>
<td>10</td>
</tr>
<tr>
<td>Previous heart disease (n = 25)</td>
<td>14</td>
<td>9</td>
<td>3</td>
<td>1</td>
<td>13</td>
</tr>
<tr>
<td>Ischemia</td>
<td>7</td>
<td>2</td>
<td>3</td>
<td>1</td>
<td>6</td>
</tr>
<tr>
<td>Arrhythmia</td>
<td>4</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
</tbody>
</table>

ischemia). Among the 36 patients with a cerebrovascular accident in the distribution of the internal carotid arterial system, ECG changes appeared in 29 (table 6).

The prognosis of stroke patients was much worse for those developing cardiac abnormalities. Apparently, no death occurred in the patients who did not exhibit electrocardiographical changes. The outcome of the patients with cerebral ischemia seems to be related more to the changes acquired during the acute stage of the stroke than to previous cardiac condition. Thus, five of eight patients with no pre-existing heart disease who showed further ECG changes, only four deaths occurred (table 7). Similarly all four patients with cerebral hemorrhage who died had electrocardiographical changes during the acute stage of the stroke; none of them had evidence of previous heart disease (table 8).

Monitoring of blood pressure during the first 24 hours of stroke failed to disclose significant variations in either hypertensive or normotensive patients.

Discussion
In 1947 Byer et al. described large and upright T waves and prolonged QT intervals in a patient with subarachnoid hemorrhage. In 1953 Levine reported inverted, deep T waves and ST segment elevations in a patient with a ruptured aneurysm of the circle of Willis, while the next year Burch et al. described a new electrocardiographical pattern consisting of a prolonged QT interval, large and often inverted T waves and large U waves in patients with cerebrovascular accidents. Since then, more reports concerning ECG alterations in stroke patients appeared in the literature. They were described mainly in subarachnoid hemorrhage and less often in cerebral ischemia and intracerebral hemorrhage. The present study which dealt primarily with cerebral ischemia and hemorrhage demonstrates that electro-

TABLE 4
Heart Condition Prior to Stroke (Hemorrhagic Stroke, n = 9)

<table>
<thead>
<tr>
<th>ECG changes during the first 24 hours</th>
<th>Arrhythmia</th>
<th>Ischemia</th>
<th>Arrhythmia and ischemia</th>
<th>Total</th>
<th>No change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal heart (n = 7)</td>
<td>5</td>
<td>—</td>
<td>—</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td>Previous heart disease (n = 2)</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>—</td>
</tr>
</tbody>
</table>

TABLE 5
ECG Abnormalities Monitored During the First 24 Hours of the Stroke

<table>
<thead>
<tr>
<th>Ischemic stroke (n = 43)</th>
<th>Transient (25)</th>
<th>Arrhythmia (15)</th>
<th>Ischemia (4)</th>
<th>Arrhythmia and ischemia (6)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Permanent (4)</td>
<td></td>
<td>Arrhythmia (1)</td>
<td>Ischemia (3)</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Hemorrhage stroke (n = 9)</th>
<th>Transient (7)</th>
<th>Arrhythmia (6)</th>
<th>Ischemia and arrhythmia (1)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No ECG change (2)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
cardiographical abnormalities occur very frequently in these conditions.

The ECG changes described by some authors\textsuperscript{3-16} indicate alteration and delay in the ventricular repolarization which are manifested by a prolonged QT interval, depression or elevation of the ST segment, tall, peaked, flat or deeply inverted T waves and large U waves. In selected reports of cases, changes similar to those observed in cardiac ischemia and infarction were described.\textsuperscript{6, 9, 11} However, we did not observe ischemic-type depression of the ST segment in a previous series of 200 patients with ischemic stroke\textsuperscript{14} and in a series of 45 patients with hemorrhagic stroke.\textsuperscript{15}

In the present study, however, significant ischemic ST-T alterations were observed frequently, in patients with cerebral ischemia and those with hemorrhage. Obviously, the high frequency of these abnormalities detected in this series is best explained by the use of continuous ECG monitoring which enabled us to disclose every short and transient episode of ECG abnormalities, whereas the previous reports were based mainly on occasional routine 12-lead ECG recordings. Apparently, such ischemic changes could be easily missed if the ECG recording was done sporadically. By starting the monitoring shortly after the onset of the cerebrovascular accident, we could demonstrate the frequent electrocardiographical disturbances appearing within 24 hours after the stroke.

Rhythm and conduction disturbances in patients who have cerebrovascular accidents have been less frequently described in ischemic stroke, but they seem to appear mostly in patients with subarachnoid hemorrhage.\textsuperscript{5, 6, 8, 14}

The striking frequency of dysrhythmias is best explained, again, by the system of constant ECG monitoring, a method not used by previous investigators.

The occurrence of cardiac abnormalities in the acute phase of stroke as evidenced by the appearance of new ECG alterations or aggravation of pre-existing ones may raise the possibility that the cardiac pathology pre-existed or even served as a predisposing factor for the appearance of the stroke, as it is known that stroke and heart disease share common vascular etiology in the older age groups. However, the continuous ECG monitoring enabled us to detect dynamic changes which occurred definitely after the onset of the stroke, and were not present in the ECG tracing performed before admission to the unit. It should be stressed that ECG abnormalities were also described in younger age groups,\textsuperscript{13} where the possibility of pre-existing heart disease is remote. With or without a previous cardiac disease, it may be argued that the ECG changes become manifest or aggravated due to extracerebral causes such as electrolyte imbalance, loss of fluid, shock and respiratory disturbances which may accompany the cerebrovascular event. However, in our patients, who were closely watched in an ISCU, the ECG abnormalities occurred in the absence of such disturbances, leaving the impression that in most cases the ECG changes were induced by the brain lesion itself.

The exact cause of the cardiac disturbances occurring in intracranial disease is not utterly certain. Many experiments with animals demonstrated a multitude of transient or permanent electrocardiographical abnormalities following lesions and electrical stimulation of certain areas, cortical or subcortical, of the brain. Thus, stimulating the orbital subfrontal region (area 13, where the vagus is presented),\textsuperscript{5} other parts of the cortex and limbic system,\textsuperscript{17-23} anterior and posterior hypothalamus\textsuperscript{24} and other brain stem structures\textsuperscript{25} resulted in ischemic-like ECG alterations and/or various types of rate rhythm and conduction disturbances. It is believed that the ECG changes were brought about either through parasympathetic overactivity or sympathetic overactivity or by

### Table 6

<table>
<thead>
<tr>
<th>Stroke area</th>
<th>Total</th>
<th>ECG change</th>
<th>No ECG change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Basilar artery</td>
<td>7</td>
<td>7</td>
<td>0</td>
</tr>
<tr>
<td>Carotid artery (R-L)</td>
<td>36</td>
<td>29</td>
<td>7</td>
</tr>
</tbody>
</table>

### Table 7

<table>
<thead>
<tr>
<th>Heart condition prior to stroke</th>
<th>ECG changes</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal heart (n = 18)</td>
<td>Transient/ permanent</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td>No change</td>
<td>10</td>
</tr>
<tr>
<td>Previous heart disease (n = 25)</td>
<td>Transient/ permanent</td>
<td>21</td>
</tr>
<tr>
<td></td>
<td>No change</td>
<td>4</td>
</tr>
</tbody>
</table>

### Table 8

<table>
<thead>
<tr>
<th>Heart condition prior to stroke</th>
<th>ECG changes</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal heart (n = 7)</td>
<td>Transient/ permanent</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>No change</td>
<td>2</td>
</tr>
<tr>
<td>Previous heart disease (n = 2)</td>
<td>Transient/ permanent</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>No change</td>
<td>—</td>
</tr>
</tbody>
</table>

**LAVY, YAAR, MELAMED, STERN**
a combination of both.22-26 Cardiac arrhythmias were reported after manipulation of the circle of Willis and were believed to be due to vasocardiac reflexes.27 Some authors claim that the ECG abnormalities in stroke cases can be attributed in part to the overproduction of catecholamines.28-30 Postmortem examinations of such patients with ECG abnormalities (even of myocardial infarction) showed the heart to be normal in many cases.2,4,6,9 Connor,31,32 however, demonstrated myocardial damage (focal myocytolysis) in patients dying from intracranial disease and Koskelo33 reported subendocardial hemorrhages in cases with intracranial bleeding. Experimental studies also suggest direct heart damage following lesions or stimulation of the brain.34

The EEG alterations induced experimentally are quite similar to those encountered in patients with cerebrovascular accidents. Thus, it may be speculated that the cerebral infarction of hemorrhage, by acting directly or indirectly (through edema or hemodynamic and metabolic changes) on certain susceptible cortical or subcortical areas, may lead to the appearance of the electrocardiographical abnormalities.

The present study demonstrates that the prognosis of patients with co-existing stroke and cardiac abnormalities is grave. The disturbances of cardiac function lead to further hemodynamic and metabolic abnormalities in the brain and further deterioration and even death of the patient. There is no specific treatment for the completed stroke but its complications should be treated vigorously, in order to achieve better prognosis. Therefore, stroke victims have to be watched closely and treated promptly when complications arise. By continuous ECG monitoring, cardiac disturbances, especially the rhythm and conduction abnormalities, can be detected and treated immediately. It is our opinion that the advent of the Intensive Stroke Care Unit may lead to a better outcome for patients with cerebrovascular accidents.

References


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