The Electrocardiogram in Stroke: Relationship to Pathophysiologcal Type and Comparison with Prior Tracings

DAVID S. GOLDSTEIN, M.D., PH.D.

SUMMARY The author reviewed electrocardiographic records of 150 patients with acute stroke and 150 age- and sex-matched controls, to assess the relative frequencies of ECG abnormalities among the pathophysiologic categories of stroke, and to distinguish new abnormalities at the time of the stroke from those noted on prior tracings. Of the 150 patients with stroke, 138 (92%) showed ECG abnormalities. The most common abnormalities were also changes from prior tracings: QT prolongation (68 patients, 45%), ischemic changes (59, 39%), U waves (42, 28%), tachycardia (42, 28%), and arrhythmias (41, 27%). Patients with cerebral embolus had a significantly increased frequency of atrial fibrillation (9 patients, 47%); and with subarachnoid hemorrhage an increased frequency of QT prolongation (20, 71%) and sinus arrhythmia (5, 18%). The frequencies of QT prolongation and ischemic changes related strongly to admission systolic pressure but not to mortality. Stroke patients had an increased frequency of pathologic Q waves (30 patients, 20%) and left ventricular hypertrophy (39, 26%), but these were not new findings at the time of the stroke.

The results are consistent with an interaction of underlying hypertensive or atherosclerotic cardiovascular disease, sympathetic hyperactivity, and possibly myocardial necrosis, in producing ECG changes.

Cerebrovascular accidents between January, 1975, and November, 1977, using computer-generated chart lists arranged according to ICDA (International Classification of Diseases, Applied to the United States) coding for cerebral thrombosis, cerebral embolus, subarachnoid hemorrhage, and acute cerebrovascular accident, etiology undetermined. All patients with strokes were included, unless: 1) a legible copy of the patient’s ECG, done within 24 hours of the onset of neurological symptoms, was unavailable; 2) head trauma occurred within 1 week prior to the stroke; 3) there was a documented history of subdural hematoma; 4) the stroke occurred in the setting of dissecting aortic aneurysm; 5) the patient had a functioning artificial pacemaker; or 6) alternative diagnoses were not excluded.

Each patient was assigned to 1 of 5 pathophysiologic diagnostic categories according to the following criteria. Cerebral thrombosis was diagnosed if the patient suffered the gradually progressive onset of a focal neurologic deficit and had one of the following: 1) a history of transient ischemic attacks in the same vascular distribution as the subsequent stroke; 2) previously documented carotid occlusive disease or a carotid bruit on the same side as the cerebral infarction; 3) documented hypotension just prior to the onset of symptoms; or 4) autopsy-proven cerebral infarction without evidence for cerebral embolization. Cerebral embolus was diagnosed if the patient had the abrupt onset of a focal neurologic deficit, and had one of the following: 1) systemic embolization; 2) simultaneous multiple neurologic defects in different vascular distributions; 3) intracardiac clot, endocarditis, rheumatic vegetations, or tumor; 4) a prosthetic heart valve with recent discontinuation of oral anticoagulation; 5) recent electric cardioversion; or 6) diagnostic autopsy findings. Subarachnoid hemorrhage was diagnosed if 1) the patient suffered the sudden onset of headache and stiff neck without lateralizing neurologic signs, and had grossly bloody
cerebrospinal fluid; or 2) angiography or autopsy findings were diagnostic. Intracerebral hemorrhage was diagnosed if 1) the patient suffered the sudden onset of headache with progressive lateralizing signs, progressive loss of consciousness, and bloody cerebrospinal fluid in the setting of chronic hypertension; or 2) angiography, computerized tomography, or autopsy findings were diagnostic. Stroke of indeterminate etiology was diagnosed if the patient did not fall into one of the above categories. This includes patients suffering the abrupt onset of a focal neurologic deficit without autopsy proof of cerebral thrombosis and without satisfying the other diagnostic criteria for cerebral embolus.

In analyzing the ECGs, the author considered U waves as significant if they were visible in more than 2 leads, and defined sinus arrhythmia as a variation of R-R interval-derived heart rate of more than 30 beats per minute, based on the admission 12-lead ECG. QT intervals were corrected for age, sex, and heart rate according to the criteria listed above. The study, therefore, included 150 stroke patients: 49 with cerebral thrombosis, 38 with stroke of indeterminate etiology, 28 with subarachnoid hemorrhage, 19 with cerebral embolus, and 16 with intracerebral hemorrhage. The average patient age was 66.4 years, ranging from 21 to 92, with 72 males and 78 females.

The author also analyzed the admission ECGs of 150 age- and sex-matched inpatients without acute stroke, myocardial infarction, or a functioning artificial pacemaker. These ECG's were randomly selected from ECG record room files. The most recent previous ECG for each patient also was analyzed if available.

Statistical testing of the data was performed using Yates-corrected chi-square analyses.

### Results

Of 585 charts reviewed, 435 (74%) were excluded according to the criteria listed above. The study, therefore, included 150 stroke patients: 49 with cerebral thrombosis, 38 with stroke of indeterminate etiology, 28 with subarachnoid hemorrhage, 19 with cerebral embolus, and 16 with intracerebral hemorrhage. The average patient age was 66.4 years, ranging from 21 to 92, with 72 males and 78 females.

<table>
<thead>
<tr>
<th>Finding</th>
<th>Stroke, indeterminate etiology</th>
<th>Subarachnoid hemorrhage</th>
<th>Cerebral embolus</th>
<th>Intracerebral hemorrhage</th>
<th>Stroke, overall</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>7 (14%)</td>
<td>4 (11%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>12 (8%)</td>
<td>53 (35%)</td>
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<td>Abnormal</td>
<td>42 (86%)</td>
<td>34 (80%)</td>
<td>28 (100%)</td>
<td>10 (100%)</td>
<td>138 (92%)</td>
<td>97 (65%)</td>
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<tr>
<td>Prolonged QT</td>
<td>15 (37%)</td>
<td>13 (35%)</td>
<td>20 (71%)**</td>
<td>7 (37%)</td>
<td>68 (45%)</td>
<td>18 (12%)</td>
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<tr>
<td>T wave inversion</td>
<td>12 (24%)</td>
<td>14 (37%)</td>
<td>7 (25%)</td>
<td>4 (21%)</td>
<td>43 (29%)</td>
<td>14 (9%)</td>
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<tr>
<td>U waves</td>
<td>17 (37%)</td>
<td>19 (46%)</td>
<td>9 (32%)</td>
<td>4 (21%)</td>
<td>8 (25%)</td>
<td>12 (8%)</td>
</tr>
<tr>
<td>Increased HR</td>
<td>7 (14%)</td>
<td>10 (26%)</td>
<td>10 (36%)</td>
<td>2 (10%)</td>
<td>3 (24%)</td>
<td>14 (9%)</td>
</tr>
<tr>
<td>ST depression</td>
<td>12 (24%)</td>
<td>12 (32%)</td>
<td>8 (11%)</td>
<td>5 (26%)</td>
<td>41 (27%)</td>
<td>15 (10%)</td>
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<tr>
<td>LVH</td>
<td>12 (24%)</td>
<td>10 (26%)</td>
<td>7 (25%)</td>
<td>3 (16%)</td>
<td>39 (26%)</td>
<td>18 (12%)</td>
</tr>
<tr>
<td>Q waves</td>
<td>11 (22%)</td>
<td>6 (16%)</td>
<td>6 (21%)</td>
<td>4 (21%)</td>
<td>3 (19%)</td>
<td>30 (20%)</td>
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<tr>
<td>Atrial fibr.</td>
<td>0 (0%)</td>
<td>8 (21%)</td>
<td>3 (11%)</td>
<td>9 (47%)***</td>
<td>3 (10%)</td>
<td>21 (14%)</td>
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<tr>
<td>Axis deviation</td>
<td>7 (14%)</td>
<td>9 (24%)</td>
<td>3 (11%)</td>
<td>1 (5%)</td>
<td>1 (8%)</td>
<td>21 (14%)</td>
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<tr>
<td>PVCs</td>
<td>8 (16%)</td>
<td>6 (16%)</td>
<td>2 (7%)</td>
<td>1 (5%)</td>
<td>5 (3%)</td>
<td>5 (3%)</td>
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<tr>
<td>1* heart block</td>
<td>3 (6%)</td>
<td>4 (11%)</td>
<td>3 (11%)</td>
<td>1 (5%)</td>
<td>6 (4%)</td>
<td>4 (3%)</td>
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<tr>
<td>Decreased HR</td>
<td>4 (8%)</td>
<td>3 (8%)</td>
<td>0 (0%)</td>
<td>2 (11%)</td>
<td>2 (13%)</td>
<td>8 (5%)</td>
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<tr>
<td>Sinus arrhythmia</td>
<td>3 (6%)</td>
<td>1 (3%)</td>
<td>5 (18%)**</td>
<td>0 (0%)</td>
<td>10 (7%)</td>
<td>7 (5%)</td>
</tr>
<tr>
<td>Other arrhythmia</td>
<td>1 (2%)</td>
<td>2 (5%)</td>
<td>4 (14%)</td>
<td>1 (5%)</td>
<td>2 (7%)</td>
<td>10 (7%)</td>
</tr>
<tr>
<td>PACs</td>
<td>4 (8%)</td>
<td>1 (3%)</td>
<td>3 (11%)</td>
<td>0 (0%)</td>
<td>10 (7%)</td>
<td>7 (5%)</td>
</tr>
<tr>
<td>RBBB</td>
<td>5 (10%)</td>
<td>5 (10%)</td>
<td>2 (7%)</td>
<td>0 (0%)</td>
<td>8 (5%)</td>
<td>3 (2%)</td>
</tr>
<tr>
<td>ST elevation</td>
<td>2 (4%)</td>
<td>4 (11%)</td>
<td>3 (11%)</td>
<td>0 (0%)</td>
<td>9 (6%)</td>
<td>2 (1%)</td>
</tr>
<tr>
<td>LVE</td>
<td>1 (2%)</td>
<td>3 (8%)</td>
<td>2 (7%)</td>
<td>1 (5%)</td>
<td>8 (5%)</td>
<td>3 (2%)</td>
</tr>
<tr>
<td>LBBB</td>
<td>0 (0%)</td>
<td>2 (5%)</td>
<td>1 (4%)</td>
<td>0 (0%)</td>
<td>3 (2%)</td>
<td>4 (3%)</td>
</tr>
<tr>
<td>Other</td>
<td>1 (2%)</td>
<td>1 (3%)</td>
<td>1 (4%)</td>
<td>1 (5%)</td>
<td>4 (3%)</td>
<td>0 (0%)</td>
</tr>
</tbody>
</table>

**p < 0.001 difference from all other stroke groups combined.
***p < 0.01 difference from all other stroke groups combined.
****p < 0.05 difference from stroke groups combined.
beats per minute (28%), ST depression (27%), and left ventricular hypertrophy (26%). Arrhythmias of any type occurred in 27% of patients.

Few ECG findings characterized particular types of stroke. Atrial fibrillation occurred in 9 out of 19 (47%) patients with cerebral embolus, compared with 12/131 (9%) patients with all other types of stroke ($x^2 = 15.80$, $p < 0.001$), and compared with 0/49 patients with cerebral thrombosis ($x^2 = 21.43$, $p < 0.001$). QT prolongation occurred more frequently in patients with subarachnoid hemorrhage (20/28, 71%) than in other types of stroke (28/122, 39%; $x^2 = 9.46$, $p < 0.01$), as did sinus arrhythmia (5/28, 18% vs 5/122, 4%; $x^2 = 4.30$, $p < 0.05$). Combined QT prolongation and U waves occurred more frequently in patients with intracranial bleeding (subarachnoid hemorrhage or intracerebral hemorrhage: 11/44, 25%) than without (8/106, 8%; $x^2 = 7.28$, $p < 0.01$).

Fifty-three of the patients with stroke (35%) and 63 of the controls (42%) had prior available ECGs. The median intervals between tracings were 4 months for the patients with stroke and 3 months for the controls, with ranges of 1 day to 7 years and 1 day to 5 years, respectively. Prior ECGs were abnormal in 48/53 (91%) of the patients with stroke and 45/63 (71%) of the controls ($x^2 = 6.64$, $p < 0.01$). New abnormalities appeared on the current ECGs of 39 (74%) of the patients with stroke and 9 (14%) of the controls ($x^2 = 41.71$, $p < 0.001$). Table 2 illustrates that patients with stroke showed significantly greater frequencies of new QT prolongation (32% vs 2%), arrhythmias of any type (25% vs 3%), ischemic changes, i.e., ST depression or T wave inversion (21% vs 3%), U waves (13% vs 0%), and atrial fibrillation (9% vs 0%) than control patients.

### Table 2

<table>
<thead>
<tr>
<th>Finding</th>
<th>Stroke</th>
<th>Control</th>
<th>Chi-square</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prolonged QT</td>
<td>17 (32%)</td>
<td>1 (2%)</td>
<td>18.40***</td>
</tr>
<tr>
<td>T wave inversion</td>
<td>8 (15%)</td>
<td>0 (0%)</td>
<td>7.12**</td>
</tr>
<tr>
<td>U waves</td>
<td>7 (13%)</td>
<td>0 (0%)</td>
<td>6.78**</td>
</tr>
<tr>
<td>ST depression</td>
<td>7 (13%)</td>
<td>1 (2%)</td>
<td>4.47*</td>
</tr>
<tr>
<td>Arrhythmia</td>
<td>13 (25%)</td>
<td>2 (3%)</td>
<td>10.01**</td>
</tr>
<tr>
<td>Sinus</td>
<td>2 (4%)</td>
<td>0 (0%)</td>
<td>N.S.</td>
</tr>
<tr>
<td>Atrial fibr.</td>
<td>5 (9%)</td>
<td>0 (0%)</td>
<td>4.25*</td>
</tr>
<tr>
<td>Ventricular</td>
<td>4 (8%)</td>
<td>1 (2%)</td>
<td>N.S.</td>
</tr>
<tr>
<td>Other</td>
<td>2 (4%)</td>
<td>1 (2%)</td>
<td>N.S.</td>
</tr>
<tr>
<td>PACs</td>
<td>3 (6%)</td>
<td>0 (0%)</td>
<td>N.S.</td>
</tr>
<tr>
<td>PVCs</td>
<td>3 (6%)</td>
<td>0 (0%)</td>
<td>N.S.</td>
</tr>
<tr>
<td>Either ST depression or T inversion</td>
<td>11 (21%)</td>
<td>2 (3%)</td>
<td>7.40**</td>
</tr>
<tr>
<td>Bradycardia</td>
<td>4 (8%)</td>
<td>0 (0%)</td>
<td>N.S.</td>
</tr>
<tr>
<td>Tachycardia</td>
<td>1 (2%)</td>
<td>2 (3%)</td>
<td>N.S.</td>
</tr>
<tr>
<td>Overall number changed</td>
<td>39 (74%)</td>
<td>9 (14%)</td>
<td>41.71***</td>
</tr>
</tbody>
</table>

*p < 0.05 difference between stroke and control groups
**p < 0.01 difference between stroke and control groups
***p < 0.001 difference between stroke and control groups
N.S. = no significant difference.
**U Waves**

U waves occurred in 42/150 (28%) patients with acute stroke, and new U waves in 7/53 (13%) patients who had prior available tracings. No relationship was obtained between the frequency of U waves and type of stroke. Serum potassium levels were measured on admission in 32 (76%) patients with U waves, with a mean level of 3.77, and in 76 (70%) patients without U waves, with a mean level of 4.00 — a non-significant difference. Twenty-five of 32 (78%) patients with U waves had potassium levels greater than 3.5, suggesting in these cases that the U waves were not due to hypokalemia.

**Arrhythmias**

Arrhythmias of any type occurred in 41/150 (27%) patients with acute stroke, and new arrhythmias occurred in 13/53 (25%) patients who had prior available tracings.

Atrial fibrillation was the most common arrhythmia, occurring in 21/150 (14%) patients. Nine of 19 (47%) patients with cerebral embolus had atrial fibrillation, compared with 0/49 with cerebral thrombosis. Atrial fibrillation also occurred significantly more frequently in patients with strokes of indeterminate origin (8/38, 21%) than with cerebral thrombosis (χ² = 9.07, p < 0.01). Of 13 patients with cerebral embolus who had prior available tracings, 8 (62%) showed atrial fibrillation on the current ECG, and in 4 of these (50%), atrial fibrillation was a new finding. No significant association appeared between atrial fibrillation and history of mitral or rheumatic heart disease.

Sinus arrhythmia occurred in 10/150 (7%) patients with acute stroke and was a new finding in 2/53 (4%) patients with prior available tracings. Patients with subarachnoid hemorrhage had a significantly greater frequency of sinus arrhythmia than patients with other types of stroke.

Ventricular arrhythmias occurred in 7/150 (5%) patients of acute stroke and in those patients with prior available tracings, ventricular arrhythmias were always new but uncommon (4/53, 8%) findings. Mortality among patients with ventricular tachycardia, ventricular fibrillation, or asystole (4/5, 80%) was significantly greater than that of patients without these abnormalities (25%; χ² = 4.82, p < 0.05).

Other arrhythmias — paroxysmal atrial tachycardia, atrial bigeminy, junctional, atrial flutter, and wandering atrial pacemaker — occurred uncommonly.

**"Classic" or Bizarre Changes**

The combination of QT prolongation, U waves, and T wave changes (inverted or wide, large, and upright) occurred in 12/150 (8%) patients with acute stroke and in 1/150 (1%) controls (χ² = 17.36, p < 0.001). This combination related significantly to intracranial bleeding and to severe systolic hypertension (10/35 = 29% of patients with pressure greater than or equal to 200, vs 8/109 = 7% of patients with pressure less than 200. χ² = 9.08, p < 0.02), but not to mortality.

**Mortality**

Overall mortality during the current hospitalization was 37/150 (25%) in patients with acute stroke. As shown in table 3, mortality varied significantly with level of consciousness, type of stroke, level of CPK, hypotension on admission, and the occurrence of malignant ventricular arrhythmias. Mortality did not relate significantly with any other ECG abnormality, history of hypertension or ischemic heart disease, extreme hypertension — even if new — or radiographic evidence of cardiomegaly or pulmonary congestion.

**Myocardial Infarction**

**ECG ST Elevations and Q Waves**

Nine of 150 (6%) patients with acute stroke showed ECG ST elevations, compared with 2/150 (2%) controls, a non-significant difference; and 30/150 (20%) showed Q waves, compared with 14/150 (9%) controls (χ² = 6.82, p < 0.01). These ECG changes did not

| Table 3: Factors Related to Mortality in Patients with Stroke |
|---------------------------------|-----------------|----------------|
| Factor                         | Mortality       | Chi-square    |
| Level of consciousness         |                 |               |
| Alert                          | 3/81 (4%)       |               |
| Lethargic                      | 5/24 (21%)      |               |
| Stuporous                      | 8/17 (47%)      |               |
| Comatose                       | 21/28 (75%)     | 57.05***      |
| Type of stroke                 |                 |               |
| Cerebral thrombosis            | 4/49 (8%)       |               |
| Indeterminate                  | 3/38 (8%)       |               |
| Cerebral embolus               | 6/19 (32%)      |               |
| Subarachnoid hemorrhage        | 15/28 (54%)     |               |
| Intracerebral hemorrhage       | 9/16 (56%)      | 30.40***      |
| Level of CPK                   |                 |               |
| Less than 100                  | 0/13 (0%)       |               |
| 101-499                        | 4/14 (29%)      |               |
| 500 or more                    | 6/9 (67%)       | 8.63**        |
| Admission systolic pressure    |                 |               |
| Less than 100 mm Hg            | 6/9 (67%)       |               |
| More than 100 mm Hg            | 30/135 (22%)    | 6.05**        |
| Ventricular arrhythmias        |                 |               |
| Tachycardia, fibrillation, or asystole | 4/5 (80%) |               |
| None                           | 33/145 (23%)    | 4.82*         |

*p < 0.05  **p < 0.00  ***p < 0.0001

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Autopsy Data
dial hemorrhage. Four had microscopically normal
healed infarcts, another left ventricular hypertrophy,
dial infarction but with elevated CPK died and were
showed diffuse myofibrillar loss, and another, who had
acute myocardial infarction. One patient showed old,
myocardia. One patient, with normal cardiac
received cardiopulmonary resuscitation, intramyocar-
the patients showed gross or microscopic evidence of
hemorrhage. Three had ischemic ECG changes
tracranial bleeding (x² = 8.89, p < 0.01). As discussed
above, the frequency and severity of CPK elevations
correlated with mortality.

Of 10 patients without recent myocardial infarctions
who had CPK isozymes assayed, 4 (40%) had
positive CPK cardiac isozyme fractions. Of 21
patients who had LDH isozymes assayed, 4 (19%) had
reversals of LDH isozymes 1 and 2. Three of the 4
patients with positive cardiac CPK isozymes had intra-
cranial bleeding.

Cardiac Enzymes

Thirty-eight patients with stroke (25%) had CPK
levels measured during their hospitalization, including
5 patients whose strokes occurred while being
hospitalized for myocardial infarction. Of the 33
patients with stroke without recently diagnosed myocar-
dial infarction, 20 (61%) had CPK levels above 100
and 9 (27%) had levels above 500. Lactic dehydrogenase (LDH) levels were elevated in 29/97
(29%). All 11 of the patients with intracranial bleeding
whose CPK levels were measured showed elevations, compared with 9/22 (41%) patients without in-
tracranial bleeding (x² = 8.89, p < 0.01). As discussed
above, the frequency and severity of CPK elevations
related to the setting of stroke or mortality. None of the
patients with prior available tracings showed new ST
elevations or new Q waves on their current ECGs.
With serial ECGs, none of the patients with ST
elevations showed changes consistent with evolving
myocardial infarction, and this group did not have a
higher frequency of CPK elevation than patients
without ST elevations.

Stroke in the Setting of Myocardial Infarction

Five patients (3%) suffered strokes in the setting of
recent myocardial infarction. All the strokes occurred
within 3 days of the infarcts; 3 were embolic, 1 thrombo-
otic, and 1 of indeterminate etiology. At the time of
their strokes, none of the patients showed new Q
waves, 3 showed ischemic changes, and 2 showed no
ECG signs of ischemia or injury. Three patients showed QT prolongation, and 2 of the 3 showed new
QT prolongation compared with their admission
tracings. One patient died during the hospitalization.

Autopsy Data

Eight patients without clinical evidence of myocardial
infarction but with elevated CPK died and were
autopsied. All 8 had died from intracranial hemorrhage. Three had ischemic ECG changes
without ST elevations or Q waves. At autopsy, none of
the patients showed gross or microscopic evidence of
acute myocardial infarction. One patient showed old,
healed infarcts, another left ventricular hypertrophy,
and 2 others pulmonary congestion. One patient showed diffuse myofibrillar loss, and another, who had
received cardiopulmonary resuscitation, intramyocar-
dial hemorrhage. Four had microscopically normal
myocardia. One patient, with normal cardiac
isozymes, no clinical evidence of acute myocardial
infarction, and with anterior ST elevations in the setting
of subarachnoid hemorrhage, died, and at autopsy
showed focal areas of myocardial necrosis, cytoplasmic banding, loss of nuclear staining, and
increased cytoplasmic eosinophilia.

Discussion

The patient with signs of a stroke and with an ab-
normal ECG represents a common diagnostic
challenge to the clinician. Cardiac diseases such as
myxoma, mural thrombus, endocarditis, and atrial
septal defect with deep venous thrombosis, may event-
tuate in cerebral emboli; arrhythmias, heart block,
and myocardial infarction associated with decreased
cardiac output may precipitate cerebral ischemia. In
addition, patients often have simultaneous coronary
cerebral atherosclerosis, or have hypertension,
leading to ECG abnormalities and to stroke inde-
dependently. To this complexity can be added the
results of the numerous reports demonstrating that
primary neurologic abnormalities may produce ECG
changes, with or without myocardial lesions.

Despite the extensive literature on the subject,
however, few studies have actually assessed the overall
frequencies of these abnormalities in the population of
patients with acute stroke. None has included ade-
quate numbers of patients to assess statistically the
relative frequencies of these abnormalities among the
pathophyslogic categories of stroke. No previous
studies have included detailed comparisons with prior
ECGs to document which changes associated with
stroke actually were new. In the present study, 91% of
patients with stroke with prior available ECGs showed
some ECG abnormality before the stroke, making this
deficiency in the literature particularly glaring.

In the current study, the most common abnor-
malities were also the most common changes from
prior tracings: QT prolongation (45%), ischemic
changes (39%), arrhythmias (27%), tachycardia (28%),
and U waves (28%). Except for potentially lethal
arrhythmias such as ventricular tachycardia, these ab-
normalities were unrelated to mortality.

The frequency of QT prolongation and sinus
arrhythmia was especially high in patients with sub-
arachnoid hemorrhage, and patients with intracranial
bleeding (subarachnoid or intracerebral hemorrhage)
showed an increased frequency or combined QT
prolongation and U waves. A strong association oc-
curred between atrial fibrillation and cerebral emb-
bolus: 47% of patients with cerebral emboli were in
atrial fibrillation, in contrast with none of 49 patients
with cerebral thrombosis. On the basis of this finding,
one might predict that a high proportion of strokes of
indeterminate etiology in patients with atrial fibrilla-
tion are in fact due to cerebral embolization.

In contrast with the lack of association between
ECG abnormalities and mortality in patients with
acute stroke, mortality was strongly related to level of
consciousness, type of stroke, CPK levels, hypoten-
sion on admission, and potentially lethal arrhythmias.

A combination of ECG abnormalities thought to be
characteristic of acute stroke — QT prolonga-
tion, U waves, and T wave changes — was uncommon, occurring in 8% of patients. However, only 1 of the 150 control patients (a patient with profound hypokalemia) showed this combination, and none of the 53 patients with stroke with prior available tracings showed this combination on the prior tracings. The combination of QT prolongation, U waves, and T wave changes may be relatively specific for acute stroke, especially if hypokalemia is absent.

None of the patients with acute strokes, including those with autopsy findings of myocytolysis, developed new pathologic Q waves. A review of the medical literature revealed only 2 cases in which acute strokes produced Q waves without autopsy evidence of infarction. New pathologic Q waves, in contrast to ischemic changes, occur rarely in stroke.

CPK and LDH levels were often elevated in patients with acute stroke, thereby limiting their differential diagnostic value in excluding associated myocardial infarction or necrosis. CPK was elevated in 61% of the 33 patients in whom it was measured, and in all of the 11 patients with intracranial bleeding in whom it was measured. Four of the 10 patients who had CPK isozymes assayed showed positive cardiac isozyme fractions, and 26% of the 23 patients who had LDH isozymes assayed showed reversal of fractions 1 and 2. Since these isozyme assays are the most specific currently available tests for myocardial necrosis, \(^{19}\) it seems reasonable to conclude that a proportion of patients with acute stroke — especially with intracranial bleeding — suffer some degree of myocardial necrosis. Direct autopsy evidence for such was obtained in 2 patients with subarachnoid hemorrhage.

Recent experimental evidence in humans with stroke suggests that increased sympathetic nervous system activity accounts in part for the ECG abnormalities associated with stroke. Cruickshank and Dwyer\(^{19}\) reported ECG changes and elevated catecholamine levels in patients with subarachnoid hemorrhage; prropanolol modified most of the changes. Feibel, Campbell, and Joynt\(^{20}\) noted a relationship between catecholamine secretion and severity of ECG changes in patients with subarachnoid hemorrhage of cerebral infarction. Lack of statistical testing limits inferences from these studies.

Increased sympathetic activity may explain the striking relationships obtained in the current study between extreme hypo- or hypertension on admission and the frequency of QT prolongation and new ischemic changes. On the basis of the cited studies and the results of the present one, ECG abnormalities associated with stroke may be viewed as due to 3 interacting processes: a) underlying atherosclerotic or hypertensive cardiovascular disease, producing a high frequency of left ventricular ischemic changes, Q waves, arrhythmias, bundle branch blocks, and left ventricular hypertrophy prior to the stroke; b) ischemic, arrhythmic, and repolarization changes due to increased sympathetic outflow during the stroke; and c) myocardial necrosis, precipitated by (a) or (b) or both.

From a retrospective study by one author, inferences should be made with awareness of the limitations of the research design. In this study there was prior knowledge of the clinical findings when ECG interpretations were made and the author relied on the written records available. The stringent criteria listed in the Methods section for categorizing the types of stroke and interpreting ECGs were devised to minimize observer bias.

The use of the Ashman and Hull tables for determining QT prolongation may also be questioned, since 12% of the control patients showed QT prolongation using these tables. Nevertheless, QT prolongation was almost 4 times as common in the patients with stroke, and was a documented new finding in almost 1/3 of the stroke patients who had prior available tracings. It seems reasonable to conclude that QT prolongation is a common new change associated with acute stroke.

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Transient Ischemic Attacks. Retrospective Study of 150 Cases of Ischemic Infarct in the Territory of the Middle Cerebral Artery

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SUMMARY

Transient ischemic attacks (TIA) are episodes of abrupt beginning, consisting of subjective or objective neurological dysfunction of short duration, with complete recovery of neurological function in the course of 24 hours. With this definition, the authors carried out a retrospective study of 150 patients suffering from ischemic infarct in the brain in the territory of the middle cerebral artery. Thirty-eight percent of the patients had had TIAs before their cerebral infarct. The symptoms, in order of frequency, were motor, sensory deficits, alterations of speech and vision. Most of the patients had a definite cerebral infarct, occurring one month after the last TIA; the symptoms of both processes were remarkably similar.

The authors studied the angiographic characteristics, pharmacological and toxic antecedents, and associated diseases in these patients. The study indicates that TIA may be the first manifestation of cerebral vascular disease.

Methods

One hundred and fifty patients, suffering from ischemic cerebral infarct in the portion of the brain supplied by the middle cerebral artery, who had been admitted to the neurology service of the Hospital de la Santa Cruz y San Pablo, were studied. None of these patients had previously suffered a cerebral infarct.

Diagnosis of the ischemic infarct in the territory of the middle cerebral artery was established from the clinical history, neurological examination, and data supplied by the skull x-ray, EEG and cerebral arteriography. The diagnostic criteria for cerebral infarction are those of the Joint Committee for Stroke Resources.

Results

One hundred men and 50 women were studied. The age range was 25–89 with an average age of 63.7 years (table 2). Of the 150 patients with infarct in the territory of the middle cerebral artery, 58 (38.6%) had a TIA prior to the infarct. The age distribution of patients with TIA is shown in table 3. Patients between 50 and 59-years-old had, proportionally, the greater number of TIAs. The frequency distribution of TIA is shown in table 4. Of the 58 patients 42 (72.4%) had only one episode of TIA.

The duration of the TIAs and the area of brain...
The electrocardiogram in stroke: relationship to pathophysiological type and comparison with prior tracings.

D S Goldstein

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