A New Electrocardiographic Pattern
Observed in Cerebrovascular Accidents

By G. E. Burch, M.D., Robert Meyers, M.D., and J. A. Abildskov, M.D.

An electrocardiographic pattern was encountered in patients with cerebrovascular accidents which consisted primarily of T waves of large amplitude and duration. Large U waves were often present which may fuse in part or entirely within the T wave. Because of the presence and fusion of the T and U waves, the prolonged Q-T interval associated with the pattern was most probably a Q-U interval.

During the past several years a distinctive electrocardiographic pattern has been observed in some patients with cerebrovascular accidents at the Charity Hospital at New Orleans. No reference to this association was found in the literature, although others have observed changes in the electrocardiograms of patients with cerebrovascular accidents. Since no detailed description of the electrocardiographic pattern has been published, it is not possible to compare the pattern reported here with the isolated electrocardiographic changes noted by others.

In its most characteristic form, the pattern consists essentially of T waves of considerable amplitude and width and a long Q-T interval. Prolongation of the Q-T interval has been mentioned in association with cerebrovascular accidents, but there has been no detailed description of the configuration of the associated T wave.

Method

No attempt has been made to review a large series of patients’ records to ascertain the incidence of the electrocardiographic syndrome in cerebrovascular accidents, since electrocardiograms were not recorded on all patients with this clinical state. Furthermore, because of sudden or early death, this diagnosis could not always be definitely established.

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A review of the case histories of patients with a diagnosis of cerebral hemorrhage, subarachnoid hemorrhage or unclassified cerebrovascular accidents at Charity Hospital during the year 1950 revealed 10 electrocardiograms with the syndrome. In addition, tracings with this pattern from one private patient, two patients observed in 1949, one in 1951 and three in 1952 were included, making a total of 17 electrocardiograms included in this report.

Table 1 summarizes certain clinical data concerning the patients whose electrocardiograms were studied. In most instances the diagnosis of cerebrovascular accident was established by clinical study alone. Hemorrhage was demonstrated by spinal puncture in 14 of the 17 patients, and in the remaining three parenchymal hemorrhage or arterial thrombosis (classified as C.V.A. in table 1) was diagnosed on the basis of neurologic manifestations.

Results

Results are summarized in table 1 and figures 1, 2 and 3.

Q-T Interval. The Q-T interval was prolonged in all electrocardiograms. The increase beyond normal duration, determined by Ashman’s formula, varied from 7 to 66 per cent, the mean being 29 per cent. The shortest Q-T interval was 0.44 second and the longest 0.80 second (table I).

T Wave. The typical T wave encountered in this electrocardiographic syndrome was a large wave with the same general configuration as found in association with myocardial ischemia (fig 1). The T wave was usually negative in the standard and chest leads, although large
positive T waves were encountered in the chest leads recorded from the right of the transition zone in lead V₃. Some of the widest and largest T waves seen in clinical electrocardiography were recorded in this syndrome (fig. 1). With improvement of the clinical state, the T waves reverted to a pattern dependent upon the underlying cardiac state, normal or abnormal (fig. 1b and c). In some cases, the T waves were so large that they occupied the entire interval between the R and P complexes (fig. 2); this was not always attributable to tachycardia, with associated shortening of the duration of electrical diastole.

**U Wave.** Some tracings contained large U waves (fig. 3) which were usually located within the T waves and were large and distinct in some instances but discernible with difficulty in others. It is possible, if not usual, that the U wave contributed to the deflections usually interpreted as T waves, but an adequate number of serial tracings was not available to clarify this point. Thus, because of the close association of the U wave with the T wave, the

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**Table 1.—Pertinent Clinical and Electrocardiographic Data**

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age (yrs.)</th>
<th>Sex</th>
<th>Diagnosis</th>
<th>Interval Before 1st ECG</th>
<th>Duration Q-T Interval (sec.)</th>
<th>Normal Q-T Interval (sec.)</th>
<th>% Increase</th>
<th>U Wave</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>78</td>
<td>M</td>
<td>Cerebral hemorrhage</td>
<td>—</td>
<td>0.80</td>
<td>0.48</td>
<td>66</td>
<td>Not prominent</td>
</tr>
<tr>
<td>2</td>
<td>79</td>
<td>M</td>
<td>Cerebral hemorrhage</td>
<td>24 hours</td>
<td>0.44</td>
<td>0.37</td>
<td>19</td>
<td>V₁ and V₂</td>
</tr>
<tr>
<td>3</td>
<td>50</td>
<td>M</td>
<td>Cerebral hemorrhage</td>
<td>24 hours</td>
<td>0.60</td>
<td>0.48</td>
<td>25</td>
<td>V leads</td>
</tr>
<tr>
<td>4</td>
<td>47</td>
<td>M</td>
<td>Cerebral hemorrhage</td>
<td>24 hours</td>
<td>0.48</td>
<td>0.41</td>
<td>17</td>
<td>V₂</td>
</tr>
<tr>
<td>5</td>
<td>65</td>
<td>F</td>
<td>Cerebral hemorrhage</td>
<td>24 hours</td>
<td>0.46</td>
<td>0.40</td>
<td>15</td>
<td>Not prominent</td>
</tr>
<tr>
<td>6</td>
<td>63</td>
<td>F</td>
<td>Cerebral hemorrhage</td>
<td>7 days</td>
<td>0.52</td>
<td>0.34</td>
<td>53</td>
<td>Not prominent</td>
</tr>
<tr>
<td>7</td>
<td>74</td>
<td>F</td>
<td>Cerebral hemorrhage</td>
<td>24 hours</td>
<td>0.44</td>
<td>0.40</td>
<td>10</td>
<td>V₂-V₆</td>
</tr>
<tr>
<td>8</td>
<td>34</td>
<td>F</td>
<td>Subarachnoid hemorrhage</td>
<td>24 hours</td>
<td>0.48</td>
<td>0.36</td>
<td>33</td>
<td>Not prominent</td>
</tr>
<tr>
<td>9</td>
<td>58</td>
<td>M</td>
<td>Subarachnoid hemorrhage</td>
<td>24 hours</td>
<td>0.48</td>
<td>0.40</td>
<td>20</td>
<td>V leads</td>
</tr>
<tr>
<td>10</td>
<td>32</td>
<td>F</td>
<td>Subarachnoid hemorrhage</td>
<td>48 hours</td>
<td>0.48</td>
<td>0.44</td>
<td>9</td>
<td>V₂-V₆</td>
</tr>
<tr>
<td>11</td>
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<td>F</td>
<td>Subarachnoid hemorrhage</td>
<td>24 hours</td>
<td>0.64</td>
<td>0.45</td>
<td>42</td>
<td>V₂</td>
</tr>
<tr>
<td>12</td>
<td>70</td>
<td>M</td>
<td>Subarachnoid hemorrhage</td>
<td>4 days</td>
<td>0.60</td>
<td>0.47</td>
<td>27</td>
<td>V₃</td>
</tr>
<tr>
<td>13</td>
<td>63</td>
<td>F</td>
<td>Subarachnoid hemorrhage</td>
<td>48 hours</td>
<td>0.62</td>
<td>0.38</td>
<td>63</td>
<td>Not prominent</td>
</tr>
<tr>
<td>14</td>
<td>71</td>
<td>F</td>
<td>Subarachnoid hemorrhage</td>
<td>72 hours</td>
<td>0.44</td>
<td>0.35</td>
<td>26</td>
<td>All leads</td>
</tr>
<tr>
<td>15</td>
<td>65</td>
<td>M</td>
<td>C.V.A.</td>
<td>24 hours</td>
<td>0.44</td>
<td>0.34</td>
<td>30</td>
<td>III and V₁</td>
</tr>
<tr>
<td>16</td>
<td>47</td>
<td>M</td>
<td>C.V.A.</td>
<td>48 hours</td>
<td>0.44</td>
<td>0.41</td>
<td>7</td>
<td>V₁</td>
</tr>
<tr>
<td>17</td>
<td>58</td>
<td>M</td>
<td>C.V.A.</td>
<td>72 hours</td>
<td>0.44</td>
<td>0.37</td>
<td>19</td>
<td>Not prominent</td>
</tr>
</tbody>
</table>

**Fig. 1.** Example of a typical electrocardiographic pattern associated with cerebrovascular accidents. This tracing was obtained from patient 16 listed in table 1.
Fig. 2. Typical electrocardiographic patterns associated with cerebrovascular accidents. Tracing A was obtained from patient 12, 24 hours after a subarachnoid hemorrhage. Tracing B shows the disappearance of the electrocardiographic pattern six days after the hemorrhage in patient 12.

Fig. 3. Electrocardiographic tracing from patient 2 shows wide T waves occupying the entire interval between the T and P waves although the cardiac rate was 95 per minute.
long Q-T intervals shown in Table I may actually be Q-U intervals. It is interesting that the T and U waves often exhibited configurations which resembled those accompanying electrolyte disturbances.

**Comment**

Four of the 17 electrocardiograms recorded for the patients in this series are presented. As in any biologic process, variations were considerable, the range remaining unknown. Whereas the typical pattern is easily identified, it may not be specific for cerebrovascular accidents and might occur in many forms of acute cerebral trauma, including physical injury to the brain. Detailed aspects of its incidence require further investigation.

The mechanism of the syndrome has not been studied. Opportunities were not available for accurate measurement of electrolytes in this series to determine whether or not the electrocardiographic changes were caused by disturbances in electrolyte metabolism. Such disturbances may conceivably have been at least partially responsible for the pattern, because acute cerebral injuries have been demonstrated to be associated with disturbances in electrolyte metabolism and because some of the electrocardiograms in this series resembled those encountered in patients with such disturbances. Changes in plasma potassium, sodium, pH and bicarbonate levels are known to produce alterations in T waves, and it is possible that other chemical, thermal or hemodynamic changes, as well as alterations in the autonomic nervous system associated with a cerebrovascular accident, may contribute to the electrocardiographic changes.

The most pronounced changes occurred in the subjects with subarachnoid hemorrhage, with the next most striking in those with cerebral hemorrhage. One patient had a moderate elevation in blood urea nitrogen (80 mg. per 100 cc.), but this declined promptly to 10 mg., and in three patients there was a slight elevation up to 29.4 mg. per 100 cc. Although none of the patients were diabetic, four had an elevation of blood glucose in association with the cerebrovascular accident.

The time at which the electrocardiographic changes occurred following the cerebrovascular accident could not be determined because the electrocardiograms were recorded within the first 24 hours of the cerebrovascular accident in nine patients, 24 to 48 hours in three patients, 48 to 72 hours in two patients, four to seven days in two patients, and the interval was not known in the other patient.

This electrocardiographic pattern was maintained for at least nine days in three patients and more than 11 days in one. Because of lack of adequate serial tracings and because of the death of some patients, the duration of the electrocardiographic changes could not be determined from the data available. It is also interesting that five patients had bradycardia. As would be expected, T waves in the records of these patients were of greatest amplitude.
and duration during the period of bradycardia. In no patient did the cardiac rate exceed 100 beats per minute. Because patients who have cerebrovascular accidents have often had chronic antecedent cardiac disease with abnormal electrocardiograms, the electrocardiographic patterns in tracing or serial tracings of any given patient were, of course, modified by such existing electrocardiographic abnormalities. All of these factors contributed to the variations encountered in the patterns.

**Summary**

A distinctive electrocardiographic pattern encountered in patients with cerebrovascular accidents consisting primarily of T waves of considerable amplitude and width, long Q-T interval, and large U waves has been described, but the mechanism responsible for the changes is not known.

**Sumario Español**

Un patrón electrocardiográfico se encontró en pacientes con accidentes cerebrovasculares, que consistió principalmente en ondas T de grande amplitud y duración. Ondas U grandes también fueron encontradas las cuales se fundían en parte o completamente con la onda T. Debido a la presencia y fusión de las ondas T y U, la prolongación del intervalo Q-T asociado con este patrón fue probablemente debido al intervalo Q-U.

**REFERENCES**

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