Cardiac Arrhythmia Associated With Reversible Damage to Insula in a Patient With Subarachnoid Hemorrhage

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Background The insular cortex has been shown experimentally to contain an arrhythmogenic center that may play an important role in the genesis of cardiac arrhythmias and electrocardiographic changes in patients with intracranial (eg, cerebrovascular) lesions. The description of our case is intended to substantiate this claim with a clinical observation.

Case Description A 37-year-old woman with subarachnoid hemorrhage suffered a severe reversible cardiac arrhythmia after neurosurgical clipping of an arterial aneurysm and removal of an intracerebral hematoma from the region of the left insula.

Conclusions The observed association of a neurosurgical intervention in the region of the left insular cortex with a cardiac arrhythmia supports but does not prove the suggested role of the insula in the causation of heart rhythm disturbances after stroke. (Stroke. 1994;5:1053-1055.)

Key Words • cerebral cortex • electrocardiograph • arrhythmia • subarachnoid hemorrhage
intraoperative complications were noted, and no intraoperative heart arrhythmias were described in the anesthesia protocol.

In the two ECG recordings taken 2 and 6 hours after surgery, frequent premature ventricular complexes were present (Fig 4). The corrected QT period (QTc) was from 0.42 to 0.44 second.

During the first week after SAH, the patient had no significant complications. Plasma norepinephrine concentrations, determined on days 1, 3, and 7 after SAH, were normal. The patient was discharged 16 days after SAH.

An ECG taken 6 months after SAH showed sinus rhythm, 61 beats per minute, an ST segment elevation in lead V3, and a QTc of 0.38 second.

Discussion
Central autonomic regulation of heart rhythm is complex and not yet sufficiently explained. Recent studies8-10 suggest that the insula might be an arrhythmogenic center. In animal studies Oppenheimer et al11 demonstrated an insular chronotropic organization. It is possible that the completely reversible cardiac arrhythmia in our patient, which developed postoperatively in an electrically somewhat unstable myocardium (as shown by the prolonged QTc) and both appearing and disappearing on the day of the operation, was the result of manipulation of the insular region during the neurosurgical procedure or, in view of the absence of a description of heart arrhythmias in...
Electrocardiographic Findings During the First Week After Subarachnoid Hemorrhage

<table>
<thead>
<tr>
<th>Day</th>
<th>Heart Rate, bpm</th>
<th>ST Segment</th>
<th>T Wave</th>
<th>U Wave</th>
<th>QTc, s</th>
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<tbody>
<tr>
<td>1</td>
<td>85-94</td>
<td>Normal</td>
<td>Normal</td>
<td>Lead V2</td>
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</tr>
<tr>
<td>2</td>
<td>89-92</td>
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<td>Normal</td>
<td>Absent</td>
<td>0.44</td>
</tr>
<tr>
<td>3</td>
<td>64-70</td>
<td>Elevated in V2</td>
<td>Normal</td>
<td>Absent</td>
<td>0.40</td>
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<tr>
<td>4</td>
<td>50-56</td>
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<td>Normal</td>
<td>Lead V2</td>
<td>0.38</td>
</tr>
<tr>
<td>4†</td>
<td>64-70</td>
<td>Normal</td>
<td>Normal</td>
<td>Lead V2</td>
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</tr>
<tr>
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<td>70-75</td>
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<tr>
<td>6</td>
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<td>Absent</td>
<td>0.38</td>
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<tr>
<td>6</td>
<td>57</td>
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<td>Normal</td>
<td>Negative in lead III</td>
<td>0.40</td>
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<tr>
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<td>56-60</td>
<td>Normal</td>
<td>Normal</td>
<td>Negative in lead III</td>
<td>0.41</td>
</tr>
</tbody>
</table>

bpm indicates beats per minute; QTc, corrected QT interval.

*Electrocardiogram (ECG) recorded on fourth day after subarachnoid hemorrhage (SAH), before operation.
†ECG recorded on fourth day after SAH, 2 hours after operation.
‡ECG recorded on fourth day after SAH, 6 hours after operation.

We conclude that the observed temporal coincidence of a surgical intervention in the region of the left insula with a transient, reversible cardiac arrhythmia appears to support the suggested role of the insula in the causation of ECG changes after SAH. The data are insufficient to be taken as proof of a causal relation, however.

References
Cardiac arrhythmia associated with reversible damage to insula in a patient with subarachnoid hemorrhage.
V Svigelj, A Grad, I Tekavcic and T Kiauta

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