Subarachnoid hemorrhage is a serious neurological disorder that is often complicated by the occurrence of electrocardiographic abnormalities unexplained by preexisting cardiac conditions. These morphological waveform changes and arrhythmias often are unrecognized or misinterpreted, potentially placing patients at risk for inappropriate management. Many previous investigations were retrospective and relied on data collected in an unsystematic manner. More recent studies that included use of serial electrocardiograms and Holter recordings have provided new insight into the high prevalence of electrocardiographic changes in subarachnoid hemorrhage. Research on the prevalence, duration, and clinical significance of these electrocardiographic abnormalities and on associated factors and etiological theories is reviewed. (American Journal of Critical Care. 2002;11:48-56)

**Purpose**

This article describes the range of ECG abnormalities associated with SAH, as reported in the published literature, and reviews implications for patients’ management and prognosis. An overview is presented of research findings on the prevalence, duration, and clinical significance of these ECG changes, factors thought to be associated with the changes, and theories about etiology. Areas for future research are suggested.

**Significance of ECG Abnormalities in SAH**

The phenomenon of ECG abnormalities in patients with SAH was first reported in 1947, but the
prevalence, characteristics, and prognostic significance of the abnormalities have still not been fully explored. The etiology remains unclear, and the association with cardiac pathological changes is uncertain. Because of all these unknown factors, healthcare professionals who manage patients with SAH are confronted with special challenges. Some commonly used therapeutic strategies, such as infusion of vasopressors and large volumes of intravenous fluids, may not be optimal in patients with cardiac abnormalities.10

Because ECG changes commonly experienced by patients with SAH can mimic changes associated with coronary ischemia or infarction, or can predispose patients to serious and possibly life-threatening arrhythmias, healthcare professionals must be able to anticipate and recognize these changes. Several case reports published since the 1950s indicate the confounding effect that ECG abnormalities can have in the management of patients with SAH. Beard et al11 described the demise of a 37-year-old woman admitted with ST-segment elevation and T-wave inversion. After myocardial ischemia was diagnosed, anticoagulation therapy was started. Although the patient never showed neurological signs, other than brief syncope and stupor immediately before admission, she died several days later of SAH. An autopsy revealed a normal heart and a ruptured intracranial aneurysm. Another report12 described postponement of surgery on a patient with SAH because of ECG changes consistent with anterior myocardial infarction. The patient died, and again autopsy findings included ruptured aneurysm and no cardiac abnormalities.

Evidence from a number of studies indicates that patients with SAH are at high risk for malignant ventricular arrhythmias, including ventricular tachycardia, torsades de pointes, and ventricular fibrillation, particularly if the corrected QT (QTc) interval is prolonged.13-15 A decrease in cardiac output due to alterations in cardiac rate associated with SAH, such as sinus bradycardia, sinus tachycardia, or rapid atrial fibrillation, also can adversely affect patients’ clinical status.

The relationship between ECG abnormalities and patients’ outcomes is not clearly established. In earlier studies,13,16 of the prognostic importance of ECG changes, sample sizes were small and the results were equivocal. In a more recent retrospective study, Zaroff et al10 examined mortality due to cardiac abnormalities and to all causes in 58 patients with SAH who had ECG changes consistent with myocardial ischemia or infarction. The results indicated that ECG abnormalities were not a significant predictor of mortality. However, 20% of patients in the source SAH database were excluded from the study because their medical records did not include ECG findings, perhaps leading to selection bias. This study was further limited by its small sample size and inclusion of only 3 “snapshot” ECG recordings per subject. To date, patients’ outcomes have not been studied in a prospective investigation that included a large sample size.

Although SAH mortality rates have decreased slowly during the past 2 decades, they remain high.1 If ECG abnormalities develop during hospitalization, the heart of a patient with SAH who has brain death is not accepted as a donor organ because of the possibility of cardiac abnormalities. Learning more about the nature, reversibility, and optimal clinical management of ECG changes may help increase the availability of donor hearts from this population of patients with SAH.

Prevalence of ECG Abnormalities in SAH

The reported prevalence of ECG changes in patients with SAH ranges from 27% to 100%.10,13,14,17,18 This variation may be due to differences in study design, investigators’ definitions of ECG abnormalities, or the methods used to evaluate ECG changes.

Retrospective studies in which subjects are selected on the basis of having one or more ECG tracings in their medical record may produce falsely exaggerated prevalences of ECG abnormalities. Because ECGs are often not recorded routinely for all patients with diagnoses of neurological abnormalities, selection based on the availability of an ECG may result in a higher proportion of subjects who had some cardiac signs or symptoms during the hospital course.

Additionally, ECGs recorded before the SAH occurred were not included in most investigations, limiting the differentiation of preexisting ECG abnormalities from those associated with SAH. Although some researchers may consider the comparison unnecessary because patients with SAH are members of a relatively young and generally healthy population, ECG tracings obtained before SAH should, whenever available, be compared with ECG recordings obtained after SAH.

Timing of ECG data collection may influence conclusions about the prevalence of abnormalities. In a 12-lead ECG investigation, Brouwers et al13 found that the most pronounced ECG changes occurred during the first 72 hours after SAH. Di Pasquale et al14 found that 90% of patients had ECG abnormalities in the first 48 hours, suggesting that studies in which surveillance is started later in the course of illness may miss significant data.
Duration of ECG Abnormalities

No investigation to date has included continuous 12-lead ECG monitoring of patients with SAH, and only a few have included examination of serial tracings. Therefore, whether the observed ECG changes are transient or permanent is unclear.

In the study by Burch et al, duration of ECG abnormalities varied among subjects, exceeding 11 days for one patient. Brouwers et al found resolution of virtually all morphological changes and arrhythmias within 12 days. In a more recent study of 23 patients with SAH with ST elevation, the elevation normalized within 1 week, but changes in the T wave persisted for months. This finding confirms the observations of an earlier investigation in which T-wave inversion was persistent. ECG changes in patients with SAH also may partially resolve but then recur during a second SAH.

Morphological ECG Changes

ECG changes associated with SAH primarily reflect repolarization abnormalities involving the ST segment, T wave, U wave, and QTc interval. Because of the combination of ST-segment elevation or depression and abnormal T-wave morphology, myocardial ischemia or infarction is often suspected in patients with SAH. Reported frequencies of specific morphological ECG abnormalities are summarized in Table 1.

In an early study of 29 patients after surgery for SAH, Cropp and Manning found abnormal or questionable Q waves in 4 patients and T-wave changes indicative of myocardial ischemia in 15. One case report describes cancellation of surgery in a patient with SAH because of sudden onset of T-wave inversion and pathologic Q waves in leads II, III, and aVF consistent with inferior myocardial infarction. Despite persistent ECG changes, no cardiac enzymes indicative of myocardial infarction were detectable in serial serum samples collected during the next 48 hours, and findings on nuclear scans of the heart were normal. Surgery was performed 1 week later without incident. ECG changes had resolved 5 months after hospitalization.

An abnormally prolonged QTc interval with large T waves is a common finding in patients with SAH. Burch et al described “some of the widest and largest T waves seen in clinical electrocardiography.” The pattern of broad, slurred, inverted T waves associated with long QTc intervals is commonly termed “cerebral,” “neurogenic,” or “giant” T wave (see Figure). Other researchers have found flattening and notching of T waves.

Although widely reported, the prolonged length of the QTc interval was disputed by Shuster. He cautioned against inadvertently including the U wave in the QTc interval. The U wave, which is often 1 mm or greater in amplitude in patients with SAH, can be mistakenly interpreted as part of a notched T wave if the U wave occurs early during repolarization. De Swiet reported an unusual U-wave abnormality in a patient with SAH. The ECG obtained 1 day after admission had deeply inverted U waves and elevated ST segments in the precordium. These changes were partially resolved on an ECG obtained 5 days later.

<table>
<thead>
<tr>
<th>Study</th>
<th>N</th>
<th>P wave</th>
<th>PR interval</th>
<th>Corrected QT interval</th>
<th>ST segment</th>
<th>T wave</th>
<th>U wave</th>
</tr>
</thead>
<tbody>
<tr>
<td>Shuster,20 1960</td>
<td>12</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td>5 (42)</td>
<td>11 (92)</td>
<td>NR</td>
</tr>
<tr>
<td>Cropp and Manning,19 1960</td>
<td>29</td>
<td>3 (10)</td>
<td>NR</td>
<td>19 (66)</td>
<td>13 (45)</td>
<td>15 (52)</td>
<td>8 (28)</td>
</tr>
<tr>
<td>Hersch,7 1964</td>
<td>20</td>
<td>8 (40)</td>
<td>NR</td>
<td>9 (45)</td>
<td>10 (50)</td>
<td>4 (20)</td>
<td>8 (40)</td>
</tr>
<tr>
<td>Melin and Fogelholm,20 1983</td>
<td>76</td>
<td>5 (7)</td>
<td>6 (8)</td>
<td>8 (11)</td>
<td>20 (26)</td>
<td>16 (21)</td>
<td>3 (4)</td>
</tr>
<tr>
<td>Di Pasquale et al,14 1987</td>
<td>120</td>
<td>17 (14)</td>
<td>1 (0.8)</td>
<td>50 (42)</td>
<td>44 (37)</td>
<td>15 (12)</td>
<td>19 (16)</td>
</tr>
<tr>
<td>Rudehill et al,23 1987</td>
<td>406</td>
<td>NR</td>
<td>17 (4)</td>
<td>94 (23)</td>
<td>62 (15)</td>
<td>129 (32)</td>
<td>190 (47)</td>
</tr>
<tr>
<td>Brouwers et al,11 1989</td>
<td>61</td>
<td>10 (16)</td>
<td>19 (31)</td>
<td>24 (39)</td>
<td>31 (51)</td>
<td>36 (59)</td>
<td>27 (44)</td>
</tr>
<tr>
<td>Arruda and de Lacerda Junior,24 1992</td>
<td>15</td>
<td>NR</td>
<td>NR</td>
<td>8 (53)</td>
<td>5 (33)</td>
<td>4 (27)</td>
<td>1 (7)</td>
</tr>
</tbody>
</table>

Values are number of patients (%). N indicates sample size; NR, not reported.
Although most early reports of ECG changes in SAH are case reports or studies of fewer than 60 patients, a few larger studies confirmed earlier findings. Melin and Fogelholm found ECG changes in 86% of 14 subjects who died within 7 days of SAH and in 73% of 62 subjects who survived more than 7 days. A total of 26 other subjects died within 6 hours of SAH before an ECG could be obtained. The most common abnormalities were pathological Q waves, ST-segment elevation or depression, inverted or peaked T waves, and QTc interval greater than 430 ms. These findings were based on a single ECG tracing that was obtained within 72 hours of admission.

In the study by Brouwers et al, a total of 61 patients with SAH had serial 12-lead ECG and continuous cardiac monitoring for arrhythmia. The study period for each patient was 12 days or until the patient died or had surgery. The study protocol differed slightly in the 2 centers involved: 26 patients in one center had daily ECG, whereas 43 patients in the other institution had ECG 3 times per week. Predominant morphological changes included ST-segment abnormalities, prominent U waves, “ischemic” T waves, prolongation of the QTc interval, indications of left ventricular hypertrophy, flat or isoelectric T waves, and short PR intervals.

In the largest study to date, a single preoperative 12-lead ECG from each of 406 patients with SAH was examined. ECG findings included high-amplitude R waves in 19% of subjects, ST depression in 15%, T-wave abnormalities in 32%, U waves greater than 1 mm in amplitude in 47%, and prolonged QTc interval (>440 ms) in 23%.

During a 4-year period, di Pasquale et al prospectively studied 120 patients with SAH. A 12-lead ECG was obtained at the time of admission, and 24-hour Holter monitoring was started on the same day for detection of arrhythmias. Major morphological changes detected in the 12-lead ECG tracing included ST-segment changes in 37% of patients, prominent U waves in 16%, and T-wave abnormalities in 12%. In addition, 42% of patients had a prolonged QTc interval. Transitory ST-segment depression greater than 1.5 mm lasting 10 to 30 minutes was detected via the Holter monitoring in 7 patients. ST-segment elevation lasting 20 minutes occurred in 1 patient during cerebral angiography and concurrent with bigeminal premature ventricular complexes.

Abnormalities involving atrial depolarization, particularly peaked P waves (>2.5 mm in amplitude) and short PR intervals (<100 ms), have also been reported.

**Arrhythmias**

Numerous studies of cardiac arrhythmias in patients with SAH have been conducted. Reported frequencies of specific arrhythmias associated with SAH are summarized in Table 2. In earlier studies,
arrhythmias were detected by using occasional 12-lead ECG and whatever cardiac monitoring apparatus was used in the clinical setting. Consequently, early reports of the prevalence of arrhythmias most likely were underestimations. Despite this flaw, similar types of arrhythmias, which generally included sinus bradycardia and sinus tachycardia, wandering atrial pacemaker, and atrial fibrillation, have been detected in several studies. Premature atrial, junctional, and ventricular complexes; ventricular tachycardia; and atrioventricular block have also been detected occasionally.

Several case reports describe episodes of life-threatening arrhythmias, such as ventricular tachycardia and torsades de pointes. Carruth and Silverman reported a dramatic case in which several runs of nonsustained ventricular tachycardia developed in a patient with SAH shortly after admission. Subsequently, the patient had a prolonged period of torsades de pointes, which also terminated spontaneously. On an ECG obtained afterward, the QTc interval was 750 ms. After several days of treatment with intravenous propranolol, ventricular arrhythmia did not recur, and the QTc interval returned to 450 ms.

More recently, 24-hour 2-channel Holter monitoring has been used to detect arrhythmias in patients with cerebrovascular accident and SAH. In the largest study to date in which Holter technology was used, Di Pasquale et al first obtained a 12-lead ECG at the time of admission from a sample of 120 patients with SAH. Holter monitoring was started on the same day, and a total of 107 adequate Holter recordings were obtained. Cardiac arrhythmias were detected in 96 (90%) of the 107 patients. Premature ventricular complexes, including multiform premature ventricular complexes, couplets or triplets, and R-on-T phenomenon, were detected in 49 patients (46%). Five of the patients with frequent premature ventricular complexes also had nonsustained ventricular tachycardia (defined as 3 or more consecutive premature ventricular complexes). Torsades de pointes occurred in 4 patients and progressed to ventricular fibrillation and asystole in 1 of the 4. Holter monitoring was repeated in 48 hours for all patients with malignant ventricular arrhythmias, but no further similar arrhythmias were recorded.

Of the 107 patients, 39 (36%) had supraventricular arrhythmias, including premature supraventricular

<table>
<thead>
<tr>
<th>Study</th>
<th>N</th>
<th>ST</th>
<th>SB</th>
<th>PSVC</th>
<th>SVT</th>
<th>AF</th>
<th>PVC</th>
<th>VT</th>
</tr>
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<tr>
<td>Eisalo et al,^24^ 1972</td>
<td>20</td>
<td>5</td>
<td>7</td>
<td>NR</td>
<td>NR</td>
<td>2 (10)</td>
<td>2 (10)</td>
<td>NR</td>
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<tr>
<td>Cruickshank et al,^26^ 1974</td>
<td>40</td>
<td>13</td>
<td>9</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td>5 (12)</td>
<td>NR</td>
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<tr>
<td>Estañol et al,^28^ 1979</td>
<td>15</td>
<td>10</td>
<td>2</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td>3 (20)</td>
<td></td>
</tr>
<tr>
<td>Melin and Fogelholm,^29^ 1983</td>
<td>76</td>
<td>7</td>
<td>9</td>
<td>21</td>
<td>28</td>
<td>7 (9)</td>
<td>8 (10)</td>
<td>NR</td>
</tr>
<tr>
<td>Andreoli et al,^29^ 1987</td>
<td>70</td>
<td>10</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td>5 (7)</td>
<td>NR</td>
<td>3 (4)</td>
</tr>
<tr>
<td>Di Pasquale et al,^30^ 1987</td>
<td>107</td>
<td>32</td>
<td>42</td>
<td>29</td>
<td>27</td>
<td>7 (7)</td>
<td>3 (3)</td>
<td>49 (46)</td>
</tr>
<tr>
<td>Rudehill et al,^31^ 1987</td>
<td>406</td>
<td>9</td>
<td>20</td>
<td>12</td>
<td>3</td>
<td>NR</td>
<td>NR</td>
<td>12 (3)</td>
</tr>
<tr>
<td>Stober et al,^32^ 1988</td>
<td>52</td>
<td>44</td>
<td>12</td>
<td>17</td>
<td>33</td>
<td>NR</td>
<td>2 (4)</td>
<td>Unifocal, 17 (33)</td>
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<tr>
<td>Brouwers et al,^33^ 1989</td>
<td></td>
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<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Arruda and de Lacerda Junior,^34^ 1992</td>
<td>61</td>
<td>12</td>
<td>31</td>
<td>9</td>
<td>15</td>
<td>4 (7)†</td>
<td>4 (7)†</td>
<td>9 (15)§</td>
</tr>
</tbody>
</table>

Values are number of patients (%).
AF indicates atrial fibrillation; N, sample size; NR, not reported; PSVC, premature supraventricular complexes; PVC, premature ventricular complexes; SB, sinus bradycardia; ST, sinus tachycardia; SVT, supraventricular tachycardia; VT, ventricular tachycardia.
*Combined nonsustained and sustained VT, ventricular fibrillation, and torsades de pointes.
†Combined PSVCs, premature nodal complexes, and PVCs.
‡Combined supraventricular arrhythmias: atrial flutter, AF, and SVT.
§Combined ventricular arrhythmias: PVCs, VT, ventricular flutter, and ventricular fibrillation.
complexes, nonsustained supraventricular tachycardia, and atrial fibrillation. Thirty-two patients (30%) had sinus tachycardia (heart rate >120/min), 32 (30%) had sinus arrhythmia, 42 (39%) had sinus bradycardia (heart rate <50/min), and 23 (21%) had sinoatrial block. Several instances of wandering pacemaker, sinus arrest greater than 3 seconds, 2:1 atrioventricular block, atrioventricular dissociation, and idioventricular rhythm were recorded.

In correlating arrhythmias with time elapsed since SAH, Di Pasquale et al found that both frequency and severity of arrhythmias were higher in the 62 subjects studied within 48 hours of SAH. In these patients, they found no significant difference in the duration of the QTc interval between those with and those without ventricular tachyarrhythmias. However, all patients with malignant ventricular arrhythmias had a much longer QTc interval (590 ± 52 ms) and serum levels of potassium less than 3.5 mmol/L.

Interestingly, Di Pasquale et al found no correlation between clinical condition, site of aneurysm, extent of intracranial hemorrhage, age, or preexisting heart disease and the frequency and severity of arrhythmias.

This study is the most significant to date in detecting arrhythmias in patients with SAH. Because continuous 24-hour monitoring was used, the findings may be closer to the true prevalence of arrhythmia, at least for the phase of illness during which the patients were studied.

**Etiological Theories**

Theories about the underlying causes of ECG abnormalities in SAH are controversial, and intense investigation is ongoing. Originally, the cause was thought to be preexisting coronary artery disease exacerbated by the physiological demands of critical illness. However, only hypertension and smoking are risk factors in both SAH and coronary artery disease, and many patients with SAH are premenopausal women with no history of coronary artery disease. Chest pain, a frequent symptom in coronary artery disease, has not been reported in association with ECG abnormalities that occur in patients with SAH. Additionally, both coronary angiography and autopsy have revealed normal coronary vasculature in patients with SAH who had marked ECG changes. Cardiac injury due to elevated myocardial wall stress associated with tachycardia and hypertension has also been suggested as a causative factor. Yuki et al proposed that coronary vasospasm and reversible posts ischemic “stunned myocardium” may influence the development of ECG changes in patients with SAH.

Increasingly, however, evidence indicates a neurogenic etiology for ECG abnormalities in SAH. Animal studies suggest interesting links between brain structures and the heart. Rogers et al produced increases and decreases in the amplitude of the T wave in cats by stimulating the right and left sides of the hypothalamus and stellate ganglia, respectively. These authors suggested that the mechanism is unilateral alteration of sympathetic tone to the heart. Studies in animals and humans have indicated that injury to the insula, an area of the cortex thought to be involved in arrhythmogenesis, might be implicated in both abnormal cardiac rhythm and the focal myocardial lesions that sometimes occur after SAH.

Currently, much research focuses on the release of catecholamines, either systemically or within the myocardium, as a cause of ECG abnormalities. Autopsies have revealed areas of characteristic subendocardial myocardial lesions, called contraction band necroses, in the hearts of patients with SAH. The myocardial damage resembles lesions produced in animal experiments by infusion of norepinephrine. The characteristic pattern of myocardial lesions suggested to some researchers that the damaging catecholamines are released from intramyocardial nerve endings rather than from the general circulation. This possibility was reinforced by investigators who did not find a significant relationship between plasma levels of norepinephrine and the occurrence of ECG abnormalities in patients with SAH.

Additional evidence of pathologic changes in cardiac structure includes the elevated serum levels of cardiac enzymes and left ventricular dysfunction that occur in some patients with SAH. Investigations of the relationship between levels of creatine kinase and creatine kinase-MB and the occurrence of ECG abnormalities associated with SAH yielded conflicting results. More recently, studies focused on cardiac troponin I, a highly sensitive and specific biomarker of myocardial damage. Parekh et al found that patients with elevations in cardiac troponin I were more likely than patients without elevations in the biomarker to have ECG abnormalities.

Echocardiograms have shown transient abnormalities in left ventricular wall motion in patients with SAH. The link between left ventricular dysfunction and ECG abnormalities in patients with SAH, however, remains unclear. Kono et al compared 2 groups of patients with SAH, one group with and one group without ST-segment elevation. Left ventricular wall motion was significantly decreased in the group with ST-segment elevation compared with the group without ST-segment elevation. In another study, 5 subjects
from a total sample of 57 patients with SAH had abnormalities in left ventricular wall motion, and a significant association was observed between abnormal findings on echocardiograms and symmetrically inverted T waves. Conversely, in a study of 45 patients with SAH, Davies et al. found that ECG was not an accurate predictor of myocardial function in 4 subjects with SAH who had abnormal findings on echocardiograms. All such investigations to date, however, have been limited by small sample sizes, and studies with larger sample sizes are needed to clarify the relationship between left ventricular dysfunction and ECG abnormalities. Samuels has theorized that neurogenic influence on cardiac function might exist as a continuum, with mild, reversible ECG changes at one end of the continuum and severe, irreversible myocardial degeneration at the other end.

The relationship between ECG abnormalities and neurogenic pulmonary edema in patients with SAH has not been determined. Although the pathogenesis of neurogenic pulmonary edema is not completely understood, most researchers believe that the abnormality is noncardiogenic, resulting primarily from injury to the pulmonary circulation. However, in a study of patients with SAH and subsequent neurogenic pulmonary edema, Mayer et al. found evidence that neurogenic left ventricular dysfunction, in combination with noncardiogenic mechanisms, may contribute to the formation of pulmonary edema. Further investigation is needed to clarify the pathogenesis of pulmonary edema in patients with SAH and to determine if the edema is associated with ECG abnormalities.

Factors that may influence the development of arrhythmias in patients with SAH include cerebral vasospasm, hypoxia, electrolyte imbalance, and sudden increase in intracranial pressure triggering a sympathetic or vagal discharge due to compression of brain structures. Injection of blood into the subarachnoid space of rats can produce sinus bradycardia and tachycardia and a large number of other arrhythmias. This finding suggests that the basic hemorrhagic nature of SAH may play a role in producing arrhythmias in the period immediately after hemorrhage.

Limitations of Existing Studies

The first report of ECG abnormalities in patients with neurological problems was published more than 50 years ago. Research began largely with case reports describing the phenomenon, followed by studies with small sample sizes comparing the prevalence of ECG changes in various neurological conditions such as cerebral vascular accident, trauma, meningitis, and intracranial tumors. It soon became apparent that ECG abnormalities were more frequent and pronounced in patients with SAH than in patients with other neurological disorders.

Studies specifically concerned with ECG changes in SAH have examined prevalence, characteristics, and associated factors, but many questions remain. Many of the earlier studies were retrospective reviews of medical records. This method presents a potential weakness, because most likely ECGs were not obtained routinely and may have been reserved for patients who were clinically unstable. Another weakness in virtually all previous research is a lack of systematic collection of ECG data on either a continuous or a consistent basis.

Rudehill et al. collected ECG data on a very large sample (N = 406) of patients with SAH. However, ECGs were limited to a single preoperative tracing for each patient and were not obtained at any particular time after diagnosis of SAH. Unlike the situation in most previous studies, a well-documented and widely accepted classification system was used to evaluate ECG abnormalities. Although this investigation yielded a great deal of data, the episodic nature of the ECG recordings somewhat limits its usefulness.

Brouwers et al. were among the first to collect data prospectively in a relatively systematic manner. However, the ECG data were limited to a single tracing for each 24 hours in the study for some patients and to only one tracing in 72 hours for the remainder of the patients. Most likely, ECG changes were missed because of the snapshot nature of the recordings.

Perhaps the most complete and informative investigation to date was that of Di Pasquale et al. Data from their one-time 24-hour Holter recordings were striking. These authors reported some of the highest prevalences of arrhythmias, most likely because of the continuous nature of the monitoring. ST-segment changes and transient arrhythmias of every type were detected in the 107 patients who had Holter monitoring. Of particular interest were the 4 cases of torsades de pointes. Although the results are limited because the patients had Holter monitoring only once, this study provides some of the best information thus far on cardiac arrhythmias in patients with SAH. The findings also confirm that methods used in the past were inadequate to detect the true scope of arrhythmias in patients with SAH. This study was also limited because the continuous Holter monitoring reflected only 2 ECG leads and thus possibly missed QT or ST changes that occurred in unrecorded leads.

Directions for Future Research

Much remains to be learned about the phenomenon of ECG abnormalities in patients with SAH. Unanswered questions include those about prevalence,
contributing factors, etiology, and prognostic significance. Some specific areas for future research are listed in Table 3.

Carefully designed, prospective studies with large sample sizes are needed to effectively address these questions. Continuous, systematic multilead—preferably 12-lead—acquisition of ECG data is necessary to establish the prevalence, duration, and timing of ECG abnormalities in the clinical course of SAH. Precise, well-accepted definitions and accurate measurement of ECG amplitudes and intervals are essential elements of any study of ECG abnormalities.

**Summary**

SAH is a serious neurological disorder that is often complicated by the occurrence of ECG abnormalities unexplained by preexisting cardiac conditions. In particular, ECG changes that occur during cardiac repolarization, such as abnormalities in the ST segment and the T wave, must be interpreted in the context of the patient’s neurological abnormalities. Neurologically mediated ECG changes are often misdiagnosed as myocardial ischemia or infarction, resulting in delayed treatment of the primary problem. Routine measurement of the length of the QTc interval in patients with SAH may help detect predisposition to potentially lethal tachyarrhythmias, particularly if the patient also has low serum levels of potassium.

Investigation into the frequency, characteristics, and prognostic significance of ECG abnormalities in patients with SAH will provide essential information about the underlying neurological, biochemical, or cardiac processes. Systematic ECG monitoring for both morphological changes and arrhythmias could add significantly to what is known about the nature of these ECG changes and their clinical implications. Previous research on ECG abnormalities in patients with SAH has just touched the surface, and a great deal must be learned for optimal management of patients with SAH.

**REFERENCES**


**Table 3**  Areas for future research in patients with subarachnoid hemorrhage

<table>
<thead>
<tr>
<th>Question</th>
<th>Details</th>
</tr>
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<tbody>
<tr>
<td>What morphological ECG abnormalities are common in patients with SAH?</td>
<td></td>
</tr>
<tr>
<td>What cardiac arrhythmias occur in patients with SAH?</td>
<td></td>
</tr>
<tr>
<td>What is the frequency and duration of ECG abnormalities, and at what phase of the clinical course do they occur?</td>
<td></td>
</tr>
<tr>
<td>Do ECG abnormalities associated with SAH resolve over time, or are they permanent?</td>
<td></td>
</tr>
<tr>
<td>What clinical variables are associated with these ECG abnormalities? Variables may include increased intracranial pressure, cerebral vasospasm, and measures of neurological status such as Glasgow Coma Scale score and Hunt-Hess SAH grade.</td>
<td></td>
</tr>
<tr>
<td>What demographic variables are associated with the phenomenon? Variables include age, sex, ethnic group, and coronary risk factors.</td>
<td></td>
</tr>
<tr>
<td>What are the effects of common nursing and medical interventions on ECG abnormalities? Interventions may include neurosurgery, neurovascular interventional procedures, cerebral angiography, “HHH” therapy (hypervolemia, hypertension, hemodilution), and calcium channel blocker therapy for prevention of vasospasm (nimodipine).</td>
<td></td>
</tr>
<tr>
<td>What is the association between these ECG abnormalities and indices of abnormal myocardial function, such as detection of abnormal serum levels of cardiac enzymes or ventricular wall motion abnormalities?</td>
<td></td>
</tr>
<tr>
<td>Are any of these ECG abnormalities predictive of in-hospital mortality?</td>
<td></td>
</tr>
<tr>
<td>Do any of these ECG abnormalities disqualify the use of an SAH patient’s heart for cardiac transplantation?</td>
<td></td>
</tr>
</tbody>
</table>

ECG indicates electrocardiographic; SAH, subarachnoid hemorrhage.
Electrocardiographic Abnormalities in Patients With Subarachnoid Hemorrhage
Claire E. Sommargren

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