It has been suggested that lesions in the insula may result in abnormal electrocardiographic (ECG) findings and increase the risk of sudden death. We investigated if computed tomography (CT) detected insular lesions due to acute stroke were related to ECG abnormalities and mortality at three months. Acute insular lesions were diagnosed in 43/179 patients (left insular = 25; right insular = 17; bilateral = 1) with acute stroke (cerebral infarcts = 62 and intracerebral haemorrhage = 17) based on CT scans from 5–8 days after stroke onset; 12 lead ECGs were recorded on admission and ECG telemetry was done in the first 12–24 hours after admission. Information regarding mortality at three months was obtained. Insular lesions were related to sinus tachycardia with heart rate >120 bpm (p = 0.001), ectopic beats >10% (p = 0.032), and ST elevation (p = 0.011). Right insular lesions were related to atrial fibrillation (p = 0.009), atrioventricular block (p = 0.029), ectopic beats >10% (p = 0.016), and inverted T wave (p = 0.040). Right insular lesions, compared with left or no insular lesions, increased the odds of death within three months (OR 6.2, 95% CI 1.5 to 25.2) independent of stroke severity, lesion volume, and age. As the number of patients in the present study is relatively small, our findings need to be confirmed in studies on other populations of stroke patients.

Changes in heart rate and blood pressure following insular stimulation or lesions in animal models as well as in humans are well documented. Electrocardiographic (ECG) abnormalities are frequent in acute stroke. The mechanism by which these abnormalities occur is not well understood but may involve the insular cortex; insular damage may cause activation of the sympathetic–adrenal system because of decreased inhibitory insular activity. Intraoperative insular cortex stimulation is suggestive of right sided dominance in mediation of sympathetic activity. Intraoperative insular cortex stimulation is suggestive of right sided dominance in mediation of sympathetic activity. Insular lesions may result in changes in heart rate and blood pressure following insular stimulation or lesions in animal models as well as in humans are well documented. Electrocardiographic (ECG) abnormalities are frequent in acute stroke. The mechanism by which these abnormalities occur is not well understood but may involve the insular cortex; insular damage may cause activation of the sympathetic–adrenal system because of decreased inhibitory insular activity. Insular lesions may result in changes in heart rate and blood pressure following insular stimulation or lesions in animal models as well as in humans are well documented. Electrocardiographic (ECG) abnormalities are frequent in acute stroke. The mechanism by which these abnormalities occur is not well understood but may involve the insular cortex; insular damage may cause activation of the sympathetic–adrenal system because of decreased inhibitory insular activity. Intraoperative insular cortex stimulation is suggestive of right sided dominance in mediation of sympathetic activity. Intraoperative insular cortex stimulation is suggestive of right sided dominance in mediation of sympathetic activity. Insular lesions may result in changes in heart rate and blood pressure following insular stimulation or lesions in animal models as well as in humans are well documented. Electrocardiographic (ECG) abnormalities are frequent in acute stroke. The mechanism by which these abnormalities occur is not well understood but may involve the insular cortex; insular damage may cause activation of the sympathetic–adrenal system because of decreased inhibitory insular activity. Insular lesions may result in changes in heart rate and blood pressure following insular stimulation or lesions in animal models as well as in humans are well documented. Electrocardiographic (ECG) abnormalities are frequent in acute stroke. The mechanism by which these abnormalities occur is not well understood but may involve the insular cortex; insular damage may cause activation of the sympathetic–adrenal system because of decreased inhibitory insular activity. Intraoperative insular cortex stimulation is suggestive of right sided dominance in mediation of sympathetic activity.
A/S, Denmark) was performed in the first 12–24 hours after admission; the decision to stop telemetry was made by the treating physician, mostly during the morning or evening round. Episodes of pathological activity were automatically registered and kept in the patients’ files.

The ECGs were retrospectively analysed by one observer (AFC) as to the occurrence of atrial fibrillation, atrial flutter, tachycardia (heart rate (HR) >120 beats per minute (bpm)), bradycardia (HR <45 bpm), atrioventricular block, ventricular fibrillation >5 seconds, and ST elevation, ST depression, T wave inversion, and corrected Q-T interval (cQ-T = Q-T/R-R) according to guidelines. Ectopic beats >10% of beats were registered. AFC had access to patient number and ECG but no other clinical information.

**Statistical analysis**

Statistical analysis was performed with SPSS 9.0 for Windows and consisted of the $\chi^2$ test, Mann–Whitney U test, and multiple logistic regression analysis. Variables were chosen based on literature and the tests were performed as a simple enter analyses. Goodness of fit was assessed by the Hosmer–Lemeshow test. The significance level chosen was 0.05 in all tests.

**RESULTS**

The patients’ baseline characteristics are shown in table 1.

In 41 patients (22.9%) acute insular involvement was detected on the day 1 CT scan and in 43 patients (24%) on the day 5–8 scan. In two patients insular lesions were only detected on the second CT scan. Seven patients died before the time of the second scan, however, insular lesions were not visible on the day 1 CT scan of all of these patients. Left insular lesions were detected in 25 patients, right insular lesions were detected in 17 patients, and one patient presented with acute bilateral insular lesions. Sinus tachycardia with HR>120 bpm, ST elevation and ectopic beats >10% were significantly more frequent in patients with stroke lesions involving the insula (table 2). Blood pressure (systolic and diastolic) was not related to presence or laterality of insular involvement, even when stratifying for stroke severity.

When correcting for lesion volume in multiple logistic regression analysis, insular damage (right or left) increased the risk of sinus tachycardia (odds ratio (OR) 3.7, 95% confidence interval (CI) 1.6 to 8.5) and ST elevation (OR 8.3, 95% CI 1.4 to 48.7). In patients with a right insular lesion (table 2), atrial fibrillation, atrioventricular block, ectopic beats, and inverted T wave were significantly more frequent than in patients with left insular lesions. The one patient with bilateral acute insular lesions had an abnormal ECG showing atrial fibrillation, third degree block, ST depression, and inverted T wave.

Right insular involvement independently predicted the three month mortality in a multiple logistic regression model (OR 6.2, 95% CI 1.5 to 25.2), with the three month mortality as dependent variable and right insular involvement versus no radiological involvement of the right insula, stroke severity (SSS) on admission, CT lesion volume on day 5–8, and age as independent variables. The presence of insular involvement when disregarding the side of involvement did not affect the three month mortality in a multivariate logistic regression model, with three month mortality as the dependent variable and insular involvement versus no insular involvement, stroke severity (SSS) on admission, CT lesion volume on day 5–8, and age as independent variables.

The causes of death in patients with right insular involvement as estimated by the attending physician did not differ from those of patients with strokes in other locations.

**DISCUSSION**

In the present study, insular damage, especially right sided insular damage was related to ECG changes in patients with
acute stroke. Right insular lesion predicted death within three months of stroke independent of age, severity of stroke, and infarction volume. Patients with insular involvement significantly more often presented with ECG abnormalities.

A drawback of this study is that we lacked ECGs from before stroke onset and therefore cannot conclude that the observed abnormalities result from the stroke incident. Another weakness is that CT scans do not allow discrimination between lesions in different insular regions as magnetic resonance imaging might have done. Large numbers of ECG findings were compared and multiple comparisons may increase the risk of a chance finding. The strength of this study is that it included the most common ECG abnormalities in more patients than in previous studies and therefore provides a broader approach to the subject. In agreement with other studies we found higher rates of arrhythmias in patients with insular lesions but could not confirm any relation with prolonged QTc interval.

Based on this study, we cannot determine how right insular involvement predicted three month mortality, as the causes of death in these patients did not seem to differ from those in patients without insular lesions. Whether cardiac arrhythmias played a decisive role in any death in the present study is uncertain. The given causes of death are based on best clinical judgement since autopsy was obtained in only one case. The importance of the insular regions for cardiac rhythm was demonstrated by Oppenheimer and our finding that a right insular location of the stroke lesion significantly increases risk of death within three months supports the hypothesis that damage to the right insula directly or indirectly affects cardiac function. If these results were reproduced in subsequent studies on different patient populations, further investigations—for example, Holter monitoring of patients with right insular lesions, might reveal conditions that can be modified by therapeutic intervention and thereby improve outcome.

Authors’ affiliations
H Christensen, G Boysen, Department of Neurology, Bispebjerg Hospital, University of Copenhagen, Denmark
A F Christensen, Department of Radiology, Rigshospitalet University Hospital, Copenhagen, Denmark

H H Johannesen, Department of Radiology, Bispebjerg Hospital, University of Copenhagen, Denmark

Funding: Aase og Ejnar Daniellsens Fond, Augustinus Fonden, Dagmar Marshalls Fond, Else og Mogens Wedell-Wedellsborgs Fond, Foundation for Research in Neurology, Ludvig & Sara Elsæs’ Fond, Lykkefalds Legat

Competing interests: none declared

Correspondence to: Hanne Christensen, Bispebjerg Hospital, Department of Neurology, 2400 Copenhagen NV, Denmark; hc04@bbh.hosp.dk

Received 23 January 2004
In revised form 14 April 2004
Accepted 25 April 2004

REFERENCES

Insular lesions, ECG abnormalities, and outcome in acute stroke

H Christensen, G Boysen, A F Christensen and H H Johannesen

*J Neurol Neurosurg Psychiatry* 2005 76: 269-271
doi: 10.1136/jnnp.2004.037531

Updated information and services can be found at:
http://jnnp.bmj.com/content/76/2/269

These include:

**References**

This article cites 12 articles, 7 of which you can access for free at:
http://jnnp.bmj.com/content/76/2/269#BIBL

**Email alerting service**

Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

**Topic Collections**

Articles on similar topics can be found in the following collections

- Stroke (1449)
- Radiology (1747)
- Radiology (diagnostics) (1309)

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/