Plasma B-Type Natriuretic Peptide Levels Are Associated With Early Cardiac Dysfunction After Subarachnoid Hemorrhage

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Background and Purpose—Serum B-type natriuretic peptide (BNP) is elevated after subarachnoid hemorrhage (SAH), as well as in the setting of congestive heart failure and myocardial infarction. The aim of this study was to prospectively quantify the relationship between BNP levels and cardiac outcomes after SAH.

Methods—Plasma was collected for BNP measurements as soon as possible after enrollment; a mean of 5±4 days after SAH symptom onset. On days 1, 3, and 6 after enrollment, troponin I (cTi) was measured and 2-dimensional echocardiography was performed. The following cardiac variables were collected and treated dichotomously: left ventricular ejection fraction (LVEF), regional wall motion abnormalities (RWMA), diastolic dysfunction, pulmonary edema, and cTi.

Results—There were 57 subjects. The median BNP level was 141 pg/mL (range, 0.8 to 3330 pg/mL). Higher mean BNP levels were present in those with RWMA (550 versus 261 pg/mL; $P=0.012$), diastolic dysfunction (360 versus 44; $P=0.011$), pulmonary edema (719 versus 204; $P=0.016$), elevated cTi (662 versus 240; $P=0.004$), and LVEF <50% (644 versus 281; $P=0.015$).

Conclusion—Early after SAH, elevated BNP levels are associated with myocardial necrosis, pulmonary edema, and both systolic and diastolic dysfunction of the left ventricle. These findings support the hypothesis that the heart releases BNP into the systemic circulation early after SAH. **(*Stroke*. 2005;36:1567-1571.)**

Key Words: aneurysm ■ echocardiography ■ natriuretic peptides, atrial ■ stroke ■ sympathetic nervous system

B-type natriuretic peptide (BNP) is released from the heart in patients with myocardial infarction[1] and congestive heart failure caused by systolic or diastolic dysfunction.[2] Elevated BNP levels also occur after subarachnoid hemorrhage (SAH),[3] although the source of BNP in this setting is controversial. Previous studies have shown that BNP levels increase soon after SAH and return to baseline in 1 to 2 weeks.[4] Proposed mechanisms include catecholamine release that increases the load on the cardiac ventricles,[4] hypoxia of the hypothalamus,[5] and endothelin-1 release.[6] In a small study of 18 patients,[7] plasma but not cerebrospinal fluid levels of BNP increased by 2- to 3-fold, suggesting that the brain is not the source of BNP.

We hypothesized that SAH may result in cardiac dysfunction and release of BNP from the heart. Therefore, the aim of this study was to quantify the relationships between plasma BNP levels and cardiac dysfunction after SAH.

Materials and Methods

This is a substudy from a prospective SAH cohort. The inclusion criteria for the cohort were age older than 21 years and a diagnosis of SAH by computed tomography or lumbar puncture. Patients were included in the substudy if they consented for storage of plasma samples and excluded if they had history (obtained from the patient, family, or medical records) of myocardial infarction or congestive heart failure. The study protocol was approved by the UCSF Committee on Human Research and informed consent was obtained from each patient or an appropriate designee.

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The patients were enrolled as soon as possible after admission. Clinical data were collected from patient and family interviews and the medical record. Blood samples were collected in EDTA tubes, centrifuged, and stored at −70°C. After study completion, BNP was measured using the Triage BNP assay (Biosite, San Diego, Calif).

On days 1, 3, and 6 after enrollment, serum specimens were collected, echocardiography was performed, and a chest x-ray was reviewed. The level of cardiac troponin I (cTi) was measured using a fluorescent enzyme immunoassay (Abbot Diagnostics, Abbott Park, Ill).

Transthoracic echocardiography was performed using an Acuson Sequoia 6.0 system (Mountain View, Calif). For each echo, standard images[8] and Doppler recordings of mitral inflow and pulmonary venous flow were obtained. All echocardiographic analyses were performed off-line (ProSolv; Indianapolis, Ind) by a blinded ob-

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TABLE 1. Clinical Characteristics

Patients, no.	57
Age, mean±SD†	55±16
Female, no. (%)	41 (72)
Admission Hunt Hess grade, no. (%)	
I. Mild to moderate headache	20 (35)
II. Ssevere headache	8 (14)
III. Lethargy, confusion, or focal deficit	17 (30)
IV. Stupor	8 (14)
V. Coma	4 (7)
Risk factors for coronary artery disease, no. (%)	
History of hypertension	31 (46)
History of diabetes	4 (7)
History of hyperlipidemia	6 (11)
History of smoking	22 (39)
Body surface area, mean±SD	1.9 (0.28)
Anterior location of aneurysm, no. (%)	37 (77)
History of coronary artery disease, no. (%)	4 (7)

server. Left ventricular ejection fraction (LVEF) was measured using the biplane Simpson's method of discs.[8] Regional wall motion abnormalities (RWMA) were defined as hypokinesis, akinesis, or dyskinesis of any of the 16 LV segments.[8] Using established criteria,[9] diastolic function was categorized as normal or abnormal (impaired relaxation, pseudonormal, or restrictive).

The cardiac abnormalities were treated as dichotomous variables based on an abnormal result on any of the 3 study days. Abnormal results were defined as cTi >1.0 μg/L, pulmonary edema on chest x-ray, LVEF <50%, RWMA, and abnormal diastolic function. We performed Wilcoxon rank-sum tests to compare mean BNP levels among patients with and without each cardiac abnormality.

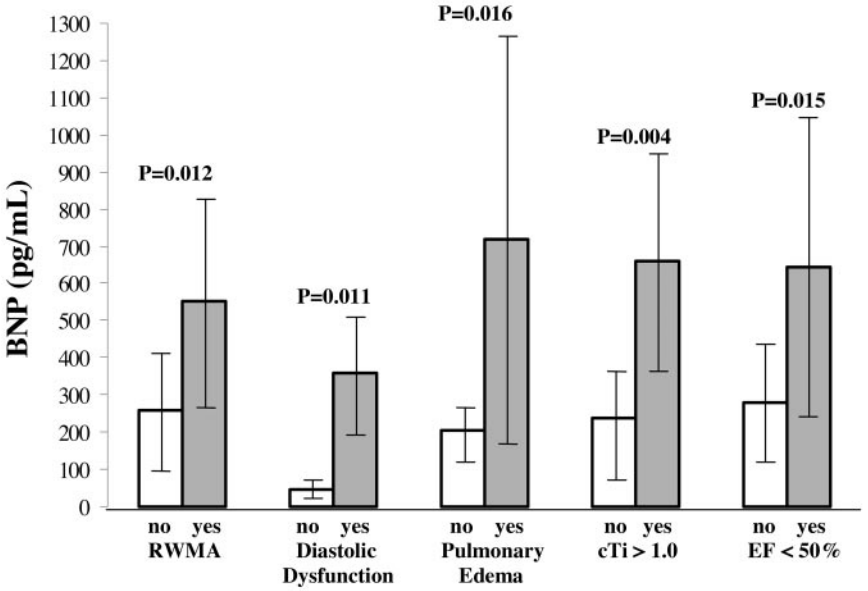
The relationship between BNP levels and hospital discharge disposition (home, acute hospital, rehabilitation hospital, death) was quantified using linear regression (and logBNP as the dependent variable to normalize the BNP distribution). All statistical analyses were performed using commercially available software (STATA, College Station, Tex) and $P \leq 0.05$ was considered significant.

Results

The parent cohort study included 174 subjects. Institutional review board approval was obtained for stored blood samples starting with patient 101. The substudy included all 57 patients who consented for blood storage (of 74 eligible patients). The patients' characteristics are shown in Table 1. There were no significant differences between the substudy patients and the other 117 patients in the cohort, except that more substudy patients had an anterior aneurysm (67% versus 49%; $P=0.043$). However, there were no significant associations between aneurysm location and the cardiac outcomes.

The mean time from SAH symptom onset to enrollment into the study was 3.3±3.2 days. BNP was measured at a mean of 5.1±3.5 days after SAH. The median BNP level was 141 pg/mL (interquartile range, 51 to 396 pg/mL). There was a trend for higher BNP levels in patients with an admission Hunt-Hess grade of 3 to 5 (449±668 pg/mL versus 189±241 for grade 1 to 2 patients, Wilcoxon rank-sum $P=0.086$). Patients with a history of hypertension had higher mean BNP levels than patients without hypertension (481±690 versus 188±251, Wilcoxon rank-sum $P=0.044$) and a higher frequency of diastolic dysfunction.

A total of 12 subjects (21%) had RWMA on any study day, 89% had evidence of diastolic dysfunction, 23% had pulmonary edema, 19% had a cTi level >1 μg/L, and 13% had a LVEF <50%. The mean of the subjects' peak cTi levels was 2.7±8.2 μg/L (median, 0.3 μg/L; interquartile range, 0.3 to 0.5). The mean of the subjects' lowest LVEF was 64% ± 15% (median 65%). Among the 50 subjects with diastolic dysfunction, 31 (55% of study cohort) had mild dysfunction (impaired relaxation) and 19 (34%) had high-grade dysfunction. The average time from SAH to each of 3 study days was 3.5±3.1, 5.3±2.6, and 8.3±2.7 days. The proportion of patients with a cTi level >1 μg/L was higher on study day 1 (13%) than study day 3 (6%). However, the other cardiac outcomes had similar rates across the 3 study days.



BNP levels and cardiac outcomes. The column height indicates the mean BNP and the error bars indicate 95% confidence intervals. Probability values indicate results of Wilcoxon rank-sum tests. RWMA, regional wall motion abnormality on any study day. Diastolic dysfunction, abnormal on any study day. Pulmonary edema, present on chest x-ray on any study day. cTi, cardiac troponin I >1.0 μg/L on any study day. EF, left ventricular ejection fraction <50% on any study day.

TABLE 2. BNP Levels and Discharge Disposition

	No. (%)	BNP (median, IQR)
Home	19 (33)	74, 49–241
Rehabilitation	12 (21)	80, 25–225
Acute hospital	16 (28)	193, 123–454
Died	10 (18)	522, 45–1140

Expressed as pg/mL.
IQR indicates interquartile range.
Rehabilitation: transferred to an inpatient rehabilitation facility.
Acute hospital: transferred to another acute care hospital.

The Figure shows the relationships between plasma BNP levels and the measured cardiac end points. If the diastolic dysfunction analysis excluded patients older than age 65, for whom impaired relaxation is typical, the relationship between diastolic dysfunction and BNP levels was not markedly affected ($P=0.054$).

There was an increase in BNP levels in relation to worsening short-term outcomes (Table 2). By linear regression, patients who died during the hospitalization had higher log BNP levels ($P=0.035$).

Discussion

This is the first study to our knowledge to demonstrate that cardiac injury and dysfunction occurring early after SAH are associated with elevated plasma BNP levels. Our findings are consistent with the hypothesis that BNP is released from the heart after SAH.

Cardiac injury and dysfunction are known to occur after SAH and the most likely mechanism is excessive myocardial catecholamine release.[10] The results of the present study provide indirect evidence that the heart is the primary source of elevated BNP levels after SAH. The strong associations observed between cardiac dysfunction and BNP are consistent with what is known to occur in patients with congestive heart failure. The findings are novel in comparison to 2 previous SAH studies, which showed no correlation between levels of natriuretic peptides and either electrocardiographic abnormalities or troponin release.[6,7]

Previous SAH studies have described an association between BNP levels and the development of cerebral vasospasm.[11] In the present study, we observed that high BNP levels were significantly associated with inpatient mortality, which has not previously been reported.

In conclusion, this study provides novel evidence that cardiac injury and dysfunction occurring after SAH are associated with elevated plasma levels of BNP. These findings support the hypothesis that the heart releases BNP into the systemic circulation early after SAH. Furthermore, elevated BNP levels may have prognostic value, supporting the hypothesis that cardiac dysfunction contributes to poor neurological outcome after SAH.

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Editorial Comment

Brain Natriuretic Peptide and Early Cardiac Dysfunction After Subarachnoid Hemorrhage

Subarachnoid hemorrhage (SAH) can be a catastrophic event for the brain and patients' neurocognitive function; however, SAH also exerts cardiac adverse effects causing rhythm disturbances or myocardial necrosis in up to 40% of the patients.[1–3] Evidence suggests that an increased sympathetic nervous activity and excessive catecholamine release during and after the event may cause deterioration of cardiac function.[4] In this context, Tung et al demonstrated previously that the degree of neurological injury correlates with the extent of cardiac damage.[5] Cardiac dysfunction after SAH adversely affects patients' overall prognosis[3] and, more specifically, the clinical sequelae of heart failure, namely low-output and hypotension, negatively influence neurological outcome after hemorrhagic brain injury. Early identification and monitoring of cardiac dysfunction thus is becoming increasingly recognized as a critical issue in patients with SAH.[6]

A variety of laboratory parameters may yield prognostic information on cardiac function in SAH patients. Among the panel of promising biomarkers, mainly parameters indicative for myocardial necrosis like cardiac troponins or creatine kinase MB fraction have been studied,[7] and, particularly, troponin I seems to be useful in predicting ischemia-related myocardial dysfunction.[7] During the past decade, natriuretic peptides emerged as novel and potentially powerful cardiovascular risk predictors and unequivocally have been shown to predict outcome of patients with heart failure and coronary artery and valvular heart disease.[8–11] Brain natriuretic peptide (BNP) is synthesized as a pro-hormone in ventricular cardiocytes in response to cardiac wall stress and pressure overload, and is cleaved into the active BNP and inactive N-terminal pro-brain natriuretic peptide (NT-proBNP). Levels of BNP correlate with left ventricular dilatation and dysfunction[12] and were initially recognized mainly as markers of chronic heart failure.[13] More recently, BNP was established also as a sensitive prognostic parameter in patients with myocardial ischemia.[8,9] It has been shown that even transient myocardial ischemia results in an immediate increase of BNP, with the magnitude of the increase proportional to the severity of ischemia.[14] It is well-recognized, that BNP is elevated after SAH, but the source of its release and its prognostic impact remained to be determined.

In this issue of *Stroke*, Tung et al[15] report the association between serum BNP levels measured in 57 patients after SAH and occurrence of cardiac dysfunction by echocardiography and cardiac troponin I elevation. BNP was significantly associated with all measures of cardiac dysfunction: higher BNP values were observed in patients with regional wall motion abnormalities, reduced left ventricular function, diastolic dysfunction, pulmonary edema, and cardiac troponin I elevation. Furthermore, BNP levels were associated with in-hospital mortality. The authors concluded that cardiac injury occurring early after SAH is associated with BNP elevation, supporting the hypothesis that the increase of BNP levels frequently observed after SAH is caused by BNP release from the heart. Importantly, elevated BNP was also identified as a prognostic marker with respect to early mortality in these patients.

Discussing the implications of this report several interesting issues arise. The data from Tung et al[15] seem to support the notion that the heart is the source of BNP release after SAH rather than being derived from the brain. However, BNP elevation after SAH seems to directly reflect the extent of cardiac deterioration in these patients, which may be useful for routine clinical applications. BNP is a global indicator of cardiac dysfunction,[13] sensitive both for systolic and diastolic cardiac deterioration.[12,16] After SAH, electrocardiogram has its limitations in detecting myocardial necrosis as changes in waveforms are largely neurally mediated and myocardial lesions tend to be small and patchy.[1] Currently, echocardiography therefore is considered the golden standard to detect cardiac dysfunction after SAH, but daily investigations and close monitoring of SAH patients by echocardiography does not seem feasible on a routine basis, and many SAH patients will undergo echocardiography only in cases of clinical evidence for heart failure. Routine measurements of BNP thus may provide a tool to monitor cardiac function and enable early identification of patients with incipient heart failure after SAH. Importantly, patients with higher BNP levels had an increased risk for in-hospital mortality, but the question whether therapeutic interventions may improve the prognosis of patients with BNP elevation after SAH remains unresolved and needs further evaluation. In the context of coronary artery disease, controversial data exist. A substudy of FRISC II indicated a survival benefit from early revascularization of patients with elevated levels of NT-proBNP,[17] TACTICS-TIMI-18,[18] in contrast, suggested that revascularization did not benefit patients with elevated BNP levels.

Some limitations of the study by Tung et al[15] seem worth further consideration. First, although "prospective," the analytic character of this study remains cross-sectional. Blood samples for BNP measurements were obtained on average 5 days after onset of SAH symptoms and BNP was correlated with abnormal cardiac results "on any of the 3 study days." Without knowing the baseline levels of BNP before the event (which rarely can be obtained) or at least very early BNP measurements, it is virtually impossible to differentiate between BNP elevation caused by preexistent chronic cardiac pathologies and BNP increase in parallel with acute cardiac dysfunction caused by SAH. Second, only a subset of 57 of 174 patients was available to study BNP levels and clinical follow-up was limited to hospital discharge. Confirmation of these data in larger patient series and prolonged neurological

and cardiovascular follow-up seems important before considering BNP measurements after SAH in clinical routine.

In conclusion, the findings by Tung et al suggest that patients with cardiac injury and dysfunction early after SAH can be identified by measurement of BNP levels and that elevation of BNP may be of immediate prognostic importance.

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