Which Parameters of Beat-to-Beat Blood Pressure and Variability Best Predict Early Outcome After Acute Ischemic Stroke?

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Background and Purpose—In hypertensive populations, increasing blood pressure (BP) levels and BP variability (BPV) are associated with a greater incidence of target organ damage. After stroke, elevated 24-hour BP levels predict a poor outcome, although it is uncertain whether shorter-length BP recordings assessing mean BP levels and BPV have a similar predictive role. The objectives of this study were to compare the different measures of beat-to-beat BP and BPV on outcome after acute ischemic stroke and assess whether these parameters were affected by stroke subtype.

Methods—Ninety-two consecutive admissions with a CT-confirmed diagnosis of acute ischemic stroke were recruited, of whom 54 had cortical infarction, 29 subcortical, and 9 posterior circulation infarction. Casual and two 5-minute recordings of beat-to-beat BP (Finapres, Ohmeda) were made under standardized conditions within 72 hours of ictus, with mean BP levels taken as the average of this 10-minute recording and BPV as the standard deviation. Outcome was assessed at 30 days as dead/dependent or independent (Rankin ≤2). The effects of BP, BPV, and stroke subtype on outcome were studied with the use of logistic regression. Stroke subjects were subsequently divided by BP quartiles and within each quartile into low- and high-variability groups; the influence of high BPV on outcome was also assessed.

Results—The odds ratio for death/dependency was significantly higher in cortical strokes compared with subcortical and posterior circulation strokes even after controlling for differences in BP and BPV (OR 4.19, *P* = 0.002). Beat-to-beat systolic BP (SBP), diastolic BP (DBP), and mean arterial pressure (MAP ± SD) levels were higher in the dead/dependent group compared with the independent group (MAP 106 ± 20.4 mm Hg vs 97 ± 19.1 mm Hg, *P* < 0.02), as was MAP variability: 6.1 (interquartile range 4.5 to 7.4 mm Hg) versus 4.9 (3.8 to 6.4 mm Hg, *P* = 0.02). The odds ratio for a poor outcome was 1.38 (*P* = 0.014) for every 10–mm Hg increase in MAP and 1.32 (*P* = 0.02) for every 1–mm Hg increase in MAP variability. Casual BP measurements had no prognostic significance. For the group as a whole when separated into BP quartiles, those with a high MAP and DBP but not SBP variability within each quartile had a worse prognosis compared with those with a low BPV.

Conclusions—A poor outcome at 30 days after ischemic stroke was dependent on stroke subtype, beat-to-beat DBP, and MAP levels and variability. Important prognostic information can be readily obtained from a short period of noninvasive BP monitoring in the acute stroke patient. These findings have important implications, particularly regarding the use of hypotensive agents in the acute stroke period. (Stroke. 2000;31:463-468.)

Key Words: blood pressure, stroke, acute stroke, ischemic, prognosis

Eighty percent of patients with a diagnosis of acute stroke are hypertensive on admission to hospital, and although the elevated blood pressure (BP) levels spontaneously decline over the subsequent 7 to 10 days, 30% of patients still may be classified as hypertensive (BP >160/95 mm Hg) at long-term follow-up. These BP changes appear to be the result of the stroke per se and not of hospital admission, as demonstrated by case-control studies. Increasing BP levels in acute stroke are particularly associated with black ethnic origin, primary intracerebral hemorrhage, and a history of hypertension. Although there is an increasing body of evidence indicating that raised BP levels are associated with a poor prognosis, some reports suggest that they are of little prognostic value or may even indicate a good prognosis. However, these differences simply may be related to methodological differences between the studies, for example, the use of single casual readings compared with 24-hour BP recordings. With the recent suggestion that early antihypertensive therapy may improve outcome in subjects in whom there is coexistent cerebral edema, and with the implications that the introduc-
tion of thrombolysis as an acute therapy may have on the management of BP.15,16 Clarification of the situation is required.

The underlying pathophysiological mechanisms for the acute increase in BP levels after both cerebral infarction and hemorrhage are unknown but could be related to an increase in sympathetic nervous system activity, as evidenced by raised plasma catecholamine and corticosteroid levels17,18 and damage to the autonomic nervous system, in particular the baroreceptor reflex arc. The baroreceptor reflex arc is intrinsically concerned with the beat-to-beat control of BP, and earlier work from this department has shown a reduction in cardiac baroreceptor sensitivity (BRS) in acute stroke19 and impairment of the vasomotor arm of the reflex arc,20,21 both of which imply damage to the central autonomic modulation system. The reduction in the diurnal BP change after acute stroke22–24 is also in keeping with autonomic nervous system dysfunction. It is becoming increasingly appreciated that cerebral infarction involving the insular cortex and the amygdala regions may be particularly important in the genesis of these abnormalities, being associated with increased plasma norepinephrine levels and increased sympathetic and reduced parasympathetic nervous activity.25–28

As well as being able to assess the diurnal BP rhythm, the advent of noninvasive but accurate BP monitors has made it possible to measure beat-to-beat BP variability (BPV) in a large number of subjects,29–35 which would not normally be possible with the use of previous methods that have relied on intra-arterial cannulation. Increased BPV is associated with increased evidence of target organ damage in the elderly and possible with the use of previous methods that have relied on intra-arterial cannulation. Increased BPV is associated with increased evidence of target organ damage in the elderly and hypertensive populations30,31 and in cardiovascular events.32 A previous study has shown that BPV is increased after acute stroke compared with age- and sex-matched control subjects,33 although the prognostic relevance of this finding is currently unknown.

To our knowledge, the influence of stroke subtype on beat-to-beat BPV has not been previously studied, nor has the relation between BPV and outcome after acute stroke been reported. Hence the aim of this study was first, to attempt to assess if mean BP levels and BPV taken from a single, 10-minute beat-to-beat recording performed within 72 hours of ictus could predict 30-day outcome in terms of death or dependency; and second, to examine the effect of stroke subtype on these measures.

Subjects and Methods

Subjects

Patients with a clinical diagnosis of acute stroke were recruited from admissions to the 3 Leicester hospitals. Each subject was assessed (by either T.G.R. or S.L.D.) and examined according to the Oxfordshire Community Stroke Project (OCSP) classification36 and the Barthel Index. All patients were hemodynamically stable, did not require intravenous or subcutaneous fluid administration, and were not biochemically dehydrated. Exclusion criteria were atrial fibrillation, either past or present, diabetes mellitus, diseases known to affect the autonomic nervous system, coexistent chronic diseases limiting independent function, diminished conscious level, and drugs affecting either the cardiovascular or autonomic nervous systems. Twenty-five patients had the diagnosis of isolated systolic hypertension (systolic BP [SBP] ≥160 mm Hg, diastolic BP [DBP] <95 mm Hg) and 29 had the diagnosis of combined hypertension (SBP ≥160 mm Hg, DBP ≥95 mm Hg). Patients taking antihypertensive medication could be included because hospital protocol was to stop these on admission, as long as the recordings were made at least 24 hours after the last dose. All patients had a CT-confirmed diagnosis within 10 days of symptom onset. With the use of the CT and the OCSP classification, patients were subdivided into cortical (total anterior circulation and partial anterior circulation), subcortical (lacunar stroke), and posterior circulation infarcts.

The study was supported by the local ethics committee, and all participants (or their carers where appropriate) gave written informed consent.

Protocol

Subjects were studied in a quiet, dedicated research room kept at a constant ambient temperature (21°C) and dimly lit to minimize external stimuli. They were asked to have abstained from caffeine-, nicotine-, and alcohol-containing products for ≥12 hours and to be ≥2 hours postprandial; they were encouraged to micturate before recording. All subjects were studied supine on a couch, with their head supported by 2 pillows and their arm supported at atrial height within 24 to 72 hours of symptom onset. Casual BP was measured with a standard mercury sphygmomanometer and an appropriately sized cuff (phase V diastole); the average of 3 readings was taken.

Three standard surface ECG leads were attached to the subject, and a Finapres 2300 (Ohmeda Monitoring Systems) noninvasive BP monitor (NIBPM) was fitted to the middle finger of the hemiparetic arm with the use of an appropriately sized cuff. The Finapres has now been widely validated against intra-arterial measurements and has been shown to accurately demonstrate BP trends.37 Once the Finapres readings showed <5% variability over 5 minutes, two 5-minute recordings of ECG and NIBPM were made, with at least a 2-minute interval between them, onto a dedicated PC. During the 2 recording periods, the Servo-adjusted mechanism on the Finapres was disabled, and the subjects were asked to lie quietly (but not sleep) and maintain a respiratory rate ≥12 breaths per minute; in some this was facilitated by the use of a metronome, and no subjects had any abnormal respiratory pattern.

Data Analysis

The analogue outputs from the NIBPM and the ECG were downloaded onto the dedicated PC at a sampling rate of 200 Hz/channel. Specially written software allowed the recording, calibration, and editing of the digitized signal and the subsequent derivation of the beat-to-beat SBP, DBP, mean arterial BP (MAP), and pulse pressure (PP), along with pulse interval. BPV was calculated with the use of the standard deviations of the beat-to-beat recordings obtained during the study period. The mean of the results from the 2 periods was used in the final analysis.

Statistical Analysis

Any association between stroke subtype and BP/BPV was assessed with the use of ANOVA. It was believed that the underlying distributions of BPV were likely to be positively skewed, therefore the logarithm transformation was used to obtain approximate normal distributions with similar variances in each group.

Stroke patients were divided into dead/dependent or independent (Rankin ≤2) groups, depending on their functional ability at 30 days after ictus (as assessed by S.L.D. or T.G.R.) at patient interview. The relation of BP and BPV to outcome was investigated with the use of logistic regression. The fit of each model was checked with the use of the Hosmer and Lemeshow goodness-of-fit test. Each BP measure, that is, SBP, DBP, MAP, and PP, were used in a single variable logistic model and then adjusted for age, sex, pulse rate, and stroke type. Where there was a statistically significant association between a BP measure and outcome in the single variable model, the predictive performance of the measures was assessed by calculating the proportion of the explained variation (R^2)38 and the area under the receiver operating characteristic curve.38 The calibration of the model was also investigated by dividing the subjects into quartiles.
according to their predicted outcome possibilities and reporting the actual dead/dependent rates.

To examine the effect of BPV at different BP levels, as had been similarly described in previous studies,31–33 subjects were divided into 4 groups according to each BP measure to give groups of approximately equal size. Each group was then divided according to the appropriate group median BPV into low-variability (≤median) and high-variability (>median) subjects. The relation between BPV and outcome was assessed with the use of the \( \chi^2 \) test.

Results are presented as mean ± SD for normally distributed data and median and interquartile ranges for non-normally distributed data. Data summary and analysis were carried out with the use of SAS 6.12. Statistical significance was set at the 5% level.

### Results

The 92 patients studied (47 men, 45 women, mean age 69.3 ± 11.6 years, range 39 to 89 years) had a mean casual BP of 165 ± 32/90 ± 17 mm Hg and mean casual pulse rate of 73 ± 14 bpm, with a median Barthel Index of 47.5 (interquartile ranges 30, 78).

Division of subjects by stroke subtype revealed that 54 had cortical infarcts, 29 subcortical, and 9 posterior circulation strokes. The odds ratio for death/dependency at 30 days was significantly increased in cortical strokes compared with subcortical and posterior circulation events, with an odds ratio of 4.19 (1.73 to 10.12, \( P < 0.002 \)), with 69% being dead/dependent compared with 31% in the groups, respectively. However, stroke subtype did not significantly influence SBP, DBP, MAP, or PP levels or BPV (see Table 1), and for further analysis of outcome data, all stroke subtypes have been combined.

Adjusted mean beat-to-beat SBP, DBP, and MAP but not PP levels were higher in the dead/dependent group than in the independent group (see Table 2), such that for a 10-mm Hg

### Table 1. Finapres Mean BP Levels and Variability (taken as SD of All Values From a 10 Minute Recording Period) by Stroke Subtype

<table>
<thead>
<tr>
<th>Stroke Type</th>
<th>TAC/PAC</th>
<th>LAC</th>
<th>POC</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>54</td>
<td>29</td>
<td>9</td>
<td>92</td>
</tr>
<tr>
<td>Blood pressure, mm Hg</td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
</tr>
<tr>
<td>SBP</td>
<td>151 (32.1)</td>
<td>148 (30.7)</td>
<td>160 (38.7)</td>
<td>151 (32.1)</td>
</tr>
<tr>
<td>DBP</td>
<td>76 (16.3)</td>
<td>80 (21.5)</td>
<td>83 (14.1)</td>
<td>78 (17.9)</td>
</tr>
<tr>
<td>PP</td>
<td>76 (27.9)</td>
<td>69 (23.7)</td>
<td>78 (30.2)</td>
<td>74 (26.8)</td>
</tr>
<tr>
<td>MAP</td>
<td>100 (19.1)</td>
<td>102 (22.3)</td>
<td>109 (20.8)</td>
<td>102 (20.2)</td>
</tr>
<tr>
<td>Blood pressure variability, mm Hg</td>
<td>Media (IQR)</td>
<td>Media (IQR)</td>
<td>Media (IQR)</td>
<td>Media (IQR)</td>
</tr>
<tr>
<td>SBPV</td>
<td>9.6 (6.8, 11.2)</td>
<td>9.5 (6.8, 10.7)</td>
<td>5.7 (4.9, 9.9)</td>
<td>8.8 (6.4, 11.2)</td>
</tr>
<tr>
<td>DBPV</td>
<td>4.3 (3.2, 5.6)</td>
<td>4.7 (2.8, 5.7)</td>
<td>3.3 (2.6, 3.7)</td>
<td>4.3 (3.0, 5.6)</td>
</tr>
<tr>
<td>PPV</td>
<td>6.2 (4.5, 8.0)</td>
<td>5.6 (4.7, 7.2)</td>
<td>4.1 (3.7, 7.0)</td>
<td>5.9 (4.4, 7.4)</td>
</tr>
<tr>
<td>MAPV</td>
<td>5.4 (4.4, 7.1)</td>
<td>6.4 (4.0, 7.4)</td>
<td>4.0 (3.3, 5.3)</td>
<td>5.4 (4.1, 7.1)</td>
</tr>
</tbody>
</table>

SBP indicates systolic blood pressure; DBP, diastolic blood pressure; PP, pulse pressure; MAP, mean arterial pressure; TAC/PAC, total/partial anterior circulation; LAC, lacunar; POC, posterior circulation; n, number in each group; IQR, interquartile range.

No significant differences were seen between stroke subtypes for any parameters.

### Table 2. Influence of Mean BP Levels and Variability on Outcome Taken as Independent or Dead/Dependent at 30 Days After Acute Cerebral Infarction

<table>
<thead>
<tr>
<th>Status at 30 d</th>
<th>n</th>
<th>Dead/Dependent</th>
<th>Independent</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>50</td>
<td>42</td>
<td>92</td>
<td></td>
</tr>
<tr>
<td>Blood pressure, mm Hg</td>
<td>Mean(SD)</td>
<td>Mean(SD)</td>
<td>Mean(SD)</td>
<td></td>
</tr>
<tr>
<td>SBP</td>
<td>158 (33.5)</td>
<td>143 (28.7)*</td>
<td>151 (32.1)</td>
<td></td>
</tr>
<tr>
<td>DBP</td>
<td>81 (18.8)</td>
<td>75 (16.3)†</td>
<td>78 (17.9)</td>
<td></td>
</tr>
<tr>
<td>PP</td>
<td>78 (30.7)</td>
<td>69 (20.3)</td>
<td>74 (26.8)</td>
<td></td>
</tr>
<tr>
<td>MAP</td>
<td>106 (20.4)</td>
<td>97 (19.1)†</td>
<td>102 (20.2)</td>
<td></td>
</tr>
<tr>
<td>Blood pressure variability, mm Hg</td>
<td>Media (IQR)</td>
<td>Media (IQR)</td>
<td>Media (IQR)</td>
<td></td>
</tr>
<tr>
<td>SBPV</td>
<td>9.2 (6.79, 11.96)</td>
<td>8.3 (6.38, 10.39)</td>
<td>8.8 (6.40, 11.16)</td>
<td></td>
</tr>
<tr>
<td>DBPV</td>
<td>5.1 (3.64, 6.28)</td>
<td>4.1 (2.68, 5.14)*</td>
<td>4.3 (2.99, 5.58)</td>
<td></td>
</tr>
<tr>
<td>PPV</td>
<td>5.8 (4.25, 7.28)</td>
<td>6.0 (4.67, 7.79)</td>
<td>5.9 (4.38, 7.41)</td>
<td></td>
</tr>
<tr>
<td>MAPV</td>
<td>6.1 (4.45, 7.40)</td>
<td>4.9 (3.79, 6.37)*</td>
<td>5.4 (4.11, 7.06)</td>
<td></td>
</tr>
</tbody>
</table>

*\( P < 0.05 \) for difference between Dead/Dependent and Independent groups adjusted for by age and stroke subtype.

†\( P < 0.02 \).
TABLE 3. Odds Ratios for Death/Dependency at 30 Days After Acute Cerebral Infarction Adjusted for by Age and Stroke Type for Every 10–mm Hg Increase in Mean BP Levels and 1–mm Hg Increase in BP Variability for Finapres and casual BP Measurements

<table>
<thead>
<tr>
<th></th>
<th>Odds Ratio</th>
<th>95% CI</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Finapres</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP, mm Hg</td>
<td>1.19</td>
<td>1.0–1.4</td>
<td>&lt;0.03</td>
</tr>
<tr>
<td>SBPV, mm Hg</td>
<td>1.07</td>
<td>0.9–1.2</td>
<td>0.29</td>
</tr>
<tr>
<td>DBP, mm Hg</td>
<td>1.42</td>
<td>1.1–1.9</td>
<td>&lt;0.02</td>
</tr>
<tr>
<td>DBPV, mm Hg</td>
<td>1.33</td>
<td>1.1–1.7</td>
<td>&lt;0.03</td>
</tr>
<tr>
<td>MAP, mm Hg</td>
<td>1.38</td>
<td>1.1–1.8</td>
<td>&lt;0.02</td>
</tr>
<tr>
<td>MAPV, mm Hg</td>
<td>1.32</td>
<td>1.1–1.7</td>
<td>&lt;0.03</td>
</tr>
<tr>
<td>PP, mm Hg</td>
<td>1.09</td>
<td>0.9–1.38</td>
<td>0.39</td>
</tr>
<tr>
<td>PPV, mm Hg</td>
<td>0.91</td>
<td>0.8–1.1</td>
<td>0.39</td>
</tr>
<tr>
<td><strong>Casual</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP, mm Hg</td>
<td>1.14</td>
<td>0.96–1.35</td>
<td>0.13</td>
</tr>
<tr>
<td>DBP, mm Hg</td>
<td>1.19</td>
<td>0.90–1.57</td>
<td>0.22</td>
</tr>
<tr>
<td>MAP, mm Hg</td>
<td>1.21</td>
<td>0.94–1.55</td>
<td>0.14</td>
</tr>
</tbody>
</table>

See Table 1 for abbreviations.

increase in MAP, the risk of death/dependency rose by 38% when adjusted for by age and stroke type. DBP variability and MAP variability but not SBP variability or PP variability were also significantly greater in the dead/dependent group (P = 0.001 and 0.011, respectively), such that a 1 mm Hg increase in MAP variability increased the risk of death and dependency at 30 days by 32% (see Table 3). None of the casual BP measures or age and pulse rate had a significant effect on outcome (see Table 3).

By dividing the patients by BP into quartiles and further splitting them into high and low BPV (see statistical section), those in the high DBP and MAP variability were significantly at greater risk of poor outcome than those with low variability, but SBP and PP variability did not influence outcome (see Table 3 and the Figure). Of the main predictors of 30-day outcome, for example, DBP and MAP variability and mean SBP, DBP, and MAP levels, no significant difference was found between these measures in their predictive value. From the logistic regression model age, stroke type, and MAP and DBP levels and variability were significant independent predictors of outcome, with no significant interaction between BPV and mean BP levels. By taking the 5 main predictors of 30-day outcome, it can be seen from Table 4 that the best predictors of death and dependency were DBP variability and MAP variability with the highest R² values and area under the receiver operating characteristic curve. These data demonstrate that for the 25% of the subjects with the best predicted outcome by DBP variability, 39.1% were actually observed to have a bad outcome, whereas for the 25% of subjects with the worst predicted outcome, 78.3% actually had a bad outcome.

**Discussion**

This study has shown a 4-fold increase in the risk of death/dependency for cortical strokes when compared with other subtypes, in keeping with previous reports of a 90% mortality rate within 12 months for large middle cerebral infarcts, in contrast to a rate of 50% for restricted cortical, lacunar, or brain stem infarcts. The main objectives of this work were to identify any potential prognostic differences between the various measures of BP, for example, SBP versus MAP and their variability on outcome. It has confirmed previous reports of a greater risk in terms of death and/or dependency after acute cerebral infarction with increasing beat-to-beat but not casual SBP, DBP, and MAP levels but not PP. However, although we found no relation between SBP variability and outcome at 30 days, increasing DBP and MAP variability did significantly relate to outcome, the greater the variability the poorer the outcome, with this relation holding true even when mean BP levels were taken into account. This prognostic information was obtained just from a single 10-minute noninvasive recording of beat-to-beat BP.

For BP and outcome, we found an odds ratio of 1.38 for death/dependency at 30 days with each 10–mm Hg increase in beat-to-beat MAP, with slightly smaller odds ratios for SBP and DBP values, but interestingly no relation with PP. Previous work from this department has reported an odds ratio of 1.88 for every 10–mm Hg increase in 24-hour SBP levels for similar outcome measures. In this present study, the lower odds ratios may represent differences in the study populations—the previous study had a higher percentage of cortical strokes (65% vs 58%) and fewer posterior circulation strokes (2% vs 10%) and included patients with intracerebral hemorrhage. In addition, the use of different methods of BP assessment, for example, Finapres recordings versus 24-hour BP monitor readings, and the fact that BP measurements were taken within 24 hours of ictus in the previous study compared with up to 72 hours in this study, may explain some of the differences. The Finapres device tends to underestimate MAP and DBP compared with intra-arterial measurements, but SBP values are similar; MAP and DBP variability are also...
similar for the 2 devices, but SBP variability is larger with the Finapres. Finapres mean BP values are consistently lower for SBP and DBP compared with casual BP measurements and therefore cannot be directly compared. The beat-to-beat BPV results presented here are comparable to those previously reported after stroke when longer recording periods were taken; Robinson et al reported SBP variability of 13.0 (4.6) mm Hg in acute cerebral infarct patients, with no significant change in BPV at follow-up 10 to 14 days later. No control group was used in this study because the main objectives of the investigation were to examine the effect of mean BP levels and BPV on outcome in stroke patients. We did not detect any difference in BPV between stroke subtypes, though the study was too small to establish whether the increased BPV was related to any particular stroke site such as the insular cortex. The exact mechanisms underlying the beat-to-beat BPV after stroke are unknown, but a reduction in cardiac BRS after acute cerebral infarction is well established and decreased BRS is well known to result in a rise in BPV. In keeping with these findings, Tokgozoelu et al have shown that heart rate variability is reduced after acute stroke, particularly where the lesion lies in the region of the insular cortex. The relation between short-term measurements of beat-to-beat BPV and target organ damage has not been studied in great detail, and we believe this is the first report that has looked at beat-to-beat BPV after acute stroke and outcome. Frattola et al, who used intra-arterial BP measurements and a type of analysis similar to the one conducted in this study, have shown that BPV is significantly related to the development of target organ damage. Again, it might appear that DBP and MAP variability (both variables being strongly statistically related) may be better indicators of outcome than mean BP levels themselves. This might not be surprising because the increase in variability may directly affect cerebral blood flow and hence perfusion to the ischemic penumbra as dynamic cerebral autoregulation processes to beat-to-beat changes in BP are impaired after acute stroke.

It also may appear surprising that mean MAP and DBP levels appeared to be better prognostic indicators than mean SBP, with PP levels having no statistical significant effect on outcome. There is increasing evidence that the pulsatile component of BP, that is, PP, which reflects arterial compliance, is strongly related to the development of atherosclerosis and is a potent risk factor for cardiovascular events in a particular coronary artery disease. However, recent publications have suggested that mean arterial pressure, which can be taken as reflecting the steady-state component of BP, may be a greater risk factor for stroke than PP, though others have been unable to confirm this. These findings may be important when considering BP reduction after stroke and which antihypertensive agents are more efficacious in terms of reducing MAP more than PP levels. However, these studies have only assessed the effects of MAP and PP as predictors of primary stroke, and there are virtually no data looking at these parameters on outcome after stroke, either in the acute or subacute phases. Our study was too small to assess the effects of previous hypertension type or treatment on outcome, but this may well be important.

Further large-scale studies are needed to establish whether these initial findings can be confirmed and what influence other important factors such as hypertension type, for example, isolated systolic hypertension as opposed to combined hypertension, have on outcome. Similarly, it will be interesting to assess whether measures of arterial compliance such as pulse wave velocity or augmentation index may have a better prognostic value than measures of BP or BPV. With increasing evidence of a poorer outcome with higher beat-to-beat and 24-hour BP levels on admission as provided by this and other studies and the new finding that beat-to-beat BPV may be equally as important if not more so than absolute BP levels, a potential therapeutic opportunity has been identified. Whether these findings hold true for cerebral hemorrhage is unknown at present. Although to date there has been much debate as to whether it is safe to introduce antihypertensive agents in the acute stroke period, these results would imply that the introduction of an agent that leads to a gradual reduction in BP, improves BPV, and does not negatively affect cerebral blood flow, for example, a centrally acting agent or an angiotensin-converting enzyme inhibitor, may have a positive role to play in improving prognosis after stroke, even if confined to certain stroke subgroups. This may have particular importance with regard to BP control if thrombolysis therapy in acute stroke is too...

**TABLE 4. Predictive Ability for Death/Dependency of Single Variable Logistic Models with the 5 Best Predictors of Outcome for All Measures of BP and BP Variability Assessed in the Study**

<table>
<thead>
<tr>
<th>Measure</th>
<th>R²</th>
<th>C</th>
<th>1st</th>
<th>2nd</th>
<th>3rd</th>
<th>4th</th>
</tr>
</thead>
<tbody>
<tr>
<td>DBP variability</td>
<td>0.066</td>
<td>0.665</td>
<td>39.1</td>
<td>39.1</td>
<td>60.9</td>
<td>78.3</td>
</tr>
<tr>
<td>MAP variability</td>
<td>0.066</td>
<td>0.654</td>
<td>43.5</td>
<td>34.8</td>
<td>69.6</td>
<td>69.6</td>
</tr>
<tr>
<td>Mean SBP</td>
<td>0.055</td>
<td>0.629</td>
<td>43.5</td>
<td>52.2</td>
<td>52.2</td>
<td>69.6</td>
</tr>
<tr>
<td>Mean MAP</td>
<td>0.047</td>
<td>0.621</td>
<td>43.5</td>
<td>69.6</td>
<td>69.6</td>
<td>60.9</td>
</tr>
<tr>
<td>Mean DBP</td>
<td>0.033</td>
<td>0.600</td>
<td>43.5</td>
<td>52.2</td>
<td>52.2</td>
<td>65.2</td>
</tr>
</tbody>
</table>

R²: Proportion of explained variation; C, area under receiver operating characteristic curve.
widely used. However, until this work has been performed, the debate surrounding BP therapy in the acute stroke situation is set to continue.

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References
Which Parameters of Beat-to-Beat Blood Pressure and Variability Best Predict Early Outcome After Acute Ischemic Stroke?
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