

The Relationship Between Baseline Blood Pressure and Computed Tomography Findings in Acute Stroke

Data From the Tinzaparin in Acute Ischaemic Stroke Trial (TAIST)

Gillian M. Sare, MRCP; Philip M.W. Bath, FRCP; Laura J. Gray, PhD; Thierry Moulin, MD; France Woimant, MD; Timothy England, MRCP; Chamila Geeganage, MSc; Hanne Christensen, MD, PhD; Peter Paul De Deyn, MD; Didier Leys, MD; Desmond O'Neill, FRCPi; E. Bernd Ringelstein, MD; for the TAIST Investigators

Background and Purpose—High blood pressure (BP) is present in $\approx 80\%$ of patients with acute ischemic stroke and is independently associated with poor outcome. There are few data examining the relationship between admission BP and acute CT findings.

Methods—TAIST was a randomized controlled trial assessing 10 days of treatment with tinzaparin versus aspirin in 1489 patients with acute ischemic stroke (<48 hr) with admission BP of $\leq 220/120$ mm Hg. CT brain scans were performed before randomization and after 10 days. The relationships between baseline BP and adjudicated CT findings were assessed. Odds ratios per 10 mm Hg change in BP were calculated.

Results—Higher systolic BP (SBP) was associated with abnormal CT scans because of independent associations with chronic changes of leukoariosis (OR, 1.12; 95% CI, 1.05–1.17) and old infarction (OR, 1.12; 95% CI, 1.06–1.17) at baseline, and signs of visible infarction at day 10 (OR, 1.06; 95% CI, 1.00–1.13). A lower SBP was associated with signs of acute infarction (OR, 0.94; 95% CI, 0.89–0.99). Hemorrhagic transformation, dense middle cerebral artery sign, mass effect, and cerebral edema at day 10 were not independently associated with baseline BP.

Conclusion—Although high baseline BP is independently associated with a poor outcome after stroke, this was not shown to be through an association with increased hemorrhagic transformation, cerebral edema, or mass effect; trial design may be suboptimal to detect this. Higher SBP is associated with visible infarction on day 10 scans. The influence of changing BP in acute stroke on CT findings is still to be ascertained. (*Stroke*. 2009;40:41-46.)

Key Words: acute stroke ■ CT ■ hypertension ■ ischaemia ■ outcome

High blood pressure (BP) (systolic BP [SBP] >140 mm Hg) is present in $\approx 80\%$ of patients with acute ischemic stroke.¹ Multiple studies have shown that high BP is associated independently with poor outcome,² including both early death and late death/dependency. Mechanisms for this relationship in ischemic stroke include an increased risk of early recurrence^{1,3} and severe cerebral edema.¹ Studies of experimental stroke have shown a relationship between hypertension and hemorrhagic transformation,^{4–6} although this was not found in the International Stroke Trial (IST),¹ a trial of 19 435 patients with ischemic stroke.⁷ To date, there are few published studies exploring the relationship between baseline BP and acute and subacute CT findings in acute stroke;⁸ CT findings such as arterial occlusion (hyperdense artery) and mass effect might be associated with early hypertension.

This study assesses the relationship between blood pressure and CT findings using data from the Tinzaparin in Acute Ischemic Stroke Trial (TAIST). Previous analysis of the data from this trial has shown high BP to be associated with poor outcome.⁹

Methods

Subjects

TAIST compared the safety and efficacy of tinzaparin (low-molecular-weight heparin) given at high dose (175 anti-Xa IU/kg per day) or medium dose (100 anti-Xa IU/kg per day), and aspirin (300 mg od) in patients with acute ischemic stroke.¹⁰ Subjects were included within 48 hours of stroke onset with 47% recruited within 24 hours. Exclusion criteria included significant hypertension (SBP >220 mm Hg or diastolic BP [DBP] >120 mm Hg). All data were collected prospectively as part of the trial protocol.¹⁰

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From Institute of Neuroscience (G.M.S., P.M.W.B., L.J.G., T.E., C.G.), University of Nottingham, Nottingham, UK; Stroke Unit (T.M.), CHU Besancon, University of Franche-Comte, Besancon, France; Department of Neurology (F.W.), Lariboisiere University Hospital, France; Department of Neurology (H.C.), Bispebjerg Hospital, Copenhagen, Denmark; Department of Neurology (P.P.D.D.), A. Z. Middelheim, ZNA, University of Antwerp, Antwerpen, Belgium; Clinique Neurologique (D.L.), CHRU de Lille, Lille, France; Department of Age Related Health Care (D.O.), Adelaide & Meath Hospital, Dublin, Ireland; Klinik für Neurologie (E.B.R.), Universität Münster, Münster, Germany.

Correspondence to Professor Philip Bath, Stroke Trials Unit, University of Nottingham, Clinical Sciences Building, City Hospital campus, Nottingham NG7 2UH UK. E-mail philip.bath@nottingham.ac.uk

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Blood Pressure

SBP, DBP, and heart rate were measured in all patients on 1 to 6 occasions in the 6 hours leading up to randomization. Mean time to randomization was 26.1 hours; therefore, measurements of BP were \approx 24 hours after stroke onset. In the present analysis, the baseline SBP, DBP, and heart rate were calculated from the average of the first 3 measurements. Further hemodynamic measurements were calculated as follows: pulse pressure=SBP-DBP; mean arterial pressure=DBP+1/3 pulse pressure; pulse pressure index=pulse pressure/mean arterial pressure; and rate pressure product=SBP \times heart rate.

CT Scans

All subjects had a baseline CT scan before randomization. Mean time from symptom onset to baseline CT was 15.8 hours. A second CT scan was performed after 10 days of treatment. Each CT scan was independently adjudicated centrally by 2 radiologists blinded to treatment and clinical findings. If there was disagreement, then the scans were reviewed by a third radiologist with the majority decision standing.

CT Scan Definitions

A scan was defined as abnormal if it showed any abnormality outside normal anatomic variation. On baseline scans, early signs of hypodensity were defined as acute infarction; early signs of infarction were other signs of acute infarction, including loss of gray/white matter differentiation (contrast reduction) and sulcal effacement. Dense middle cerebral artery refers to scan evidence of thrombus in the middle cerebral artery, and hemiedema was edema resulting in ventricular effacement on the side of the stroke. Leukoariosis was defined by the presence of periventricular white matter lucency, and old infarction was defined by the presence of established hypodense lesions.

On the day 10 scan, evidence of subacute hypodensity in keeping with ischemic stroke defined infarction evident (>1 focus defined as multiple infarction). Definitions for bleeding parameters in TAIST were used in MAST-E¹¹ and FISS-bis¹² and are as follows: hemorrhagic transformation is definite as petechial bleeding within the infarct; intrainfarct hematoma represents significant confluent bleeding within the infarct; additional hematoma is bleeding unrelated to the infarct; and interventricular bleeds are adjudicated as blood in ventricles. The definitions used in this analysis have been described previously.^{13,14}

Statistical Methods

The relationship between hemodynamic measures and clinical classifications was assessed using Student *t* test. The relationship between hemodynamic measures and CT findings were assessed using unadjusted (*t* test) and adjusted (logistic regression) models; adjustment included age, sex, baseline severity (Scandinavian Stroke Scale), time to treatment, and treatment allocation (day 10 scan only). Odds ratios refer to a change in hemodynamic measure by 10 mm Hg. Significance was taken at $P<0.05$, and SD or 95% CI are given. All analyses were performed using SPSS (version 11.0 for Mac OS; SPSS Inc).

Results

Subjects

Of 1489 randomized patients, emerging exclusion criteria prevented treatment in 15 patients and baseline BP was not recorded in 5 patients; 177 patients had no baseline CT adjudicated and 168 had no day 10 scan adjudicated. Twenty-one patients in both the baseline and day 10 scans had unsatisfactory scan quality; therefore, the information from these scans is reported variably. As such, the denominator for the different imaging variables varies. Baseline characteristics of randomized patients in TAIST have been published

previously.¹⁰ Mean baseline BP in all randomized TAIST patients was 156.4 mm Hg (SD, 22.8). Baseline SBP was significantly related to OCSP clinical subtype;¹⁵ LACS patients had significantly higher SBP than non-LACS patients (mean SBP 160.1 mm Hg vs 155.0 mm Hg; $P<0.0001$); TACS patients had significantly lower baseline SBP than non-TACS patients (mean SBP 154.5 mm Hg vs 157.5 mm Hg; $P=0.01$). Baseline SBP was also significantly related to TOAST subtype¹⁶ ($P<0.0001$; with mean SBPs for lacunar 160.1 mm Hg, large artery=153.8, and cardioembolic=154.5 mm Hg).

Baseline Scan

Baseline scan findings were compared with SBP, DBP, mean arterial pressure, rate pressure product, and pulse pressure. All of the hemodynamic variables were associated with scan findings with the exception of DBP. The results for SBP with odds ratios for the data with and without adjustment for confounding factors are shown in Table 1. Figure 1 shows baseline CT findings and mean SBP with standard deviations. Before adjustment, all of the baseline CT findings were associated with SBP. Patients with a higher baseline SBP were more likely to have had an abnormal scan, with this being related to an increase in leukoariosis and the presence of old infarction. These associations remained statistically significant after adjustment. The rest of the baseline CT findings, including evidence of acute infarction, early signs of infarction such as loss of gray white matter differentiation, mass effect, and hemiedema, were more common in patients with lower BP. With the exception of evidence of acute infarction, all of these factors ceased to be statistically significant after adjustment.

Day 10 Scan

Day 10 scan findings were compared with baseline SBP, DBP, mean arterial pressure, rate pressure product, and pulse pressure. Once again DBP was not associated with scan findings. Of the remaining hemodynamic measurements, the relationship with SBP remained the most consistent. The results for SBP with odds ratios for the data with and without adjustment for confounding factors are shown in Table 2. Figure 2 shows day 10 CT findings and mean baseline SBP with standard deviations. Patients with higher baseline BP were significantly more likely to have an infarct (all infarcts and nonhemorrhagic infarcts) seen on the day 10 scan; this remained significant after adjustment.

In the trial 435 patients (29%) had evidence of hemorrhagic transformation of their infarct, 11 (0.7%) of which were symptomatic.¹⁰ Hemorrhagic transformation (petechial bleeding within the infarct) was not associated with baseline BP after adjustment, nor was the amount of petechial bleeding. Larger bleeds forming intrainfarct hematomas, hematomas not related to the infarct, and interventricular bleeds were less common and were not significantly related to BP.

Patients with symptomatic bleeds had nonsignificantly higher baseline SBP than those without (165.3 mm Hg, SD 14.6 vs 156.4 mm Hg, SD 22.8) after adjustment for confounding factors $P=0.246$. Seventy patients (4.7%) died before day 10. Twenty of these had adjudicated clinical scans

Table 1. Relationship Between Hemodynamic Factors and Baseline CT Findings

CT Findings at Baseline	N of Patients Finding Present/Absent/Missing	SBP OR (95% CI) Unadjusted	SBP OR (95% CI) Adjusted
Abnormal scan	736/560/190	1.13 (1.07–1.18)	1.09 (1.03–1.15)
Acute infarction	395/898/193	0.90 (0.87–0.96)	0.94 (0.89–0.99)
Early signs of infarction	157/1137/192	0.90 (0.83–0.97)	0.94 (0.87–1.02)
Contrast reduction	151/1115/220	0.91 (0.84–0.98)	0.95 (0.88–1.03)
Lentiform nucleus involvement	96/1172/218	0.91 (0.78–0.94)	0.90 (0.81–0.99)
Insular zone involvement	95/1172/219	0.84 (0.77–0.93)	0.90 (0.82–1.00)
Dense MCA	64/1191/1255	0.82 (0.73–0.91)	0.87 (0.78–1.00)
Hemioedema	148/1127/211	0.87 (0.82–1.00)	0.93 (0.87–1.00)
Mass effect	75/1219/192	0.87 (0.78–1.00)	0.92 (0.83–1.00)
Leukoaraiosis	597/696/193	1.15 (1.09–1.21)	1.12 (1.05–1.17)
Old infarctions	483/811/192	1.14 (1.07–1.20)	1.12 (1.06–1.17)

ORs are for each 10-mm Hg change in BP.
MCA indicates middle cerebral artery.
Controlled for age, sex, baseline Scandinavian stroke scale, and time to treatment.
P<0.05 are shown in bold.

performed between day 1 and 10. Of these patients, 14 of 20 (70%) had mass effect evident on CT (*P*<0.0001). The group who died before day 10 did not have a higher baseline SBP than those who did not die (157.8 mm Hg vs 156.4 mm Hg; *P*=0.598).

Discussion

This exploratory analysis of a large, randomized, controlled trial in acute stroke sought to examine whether the association between high BP and poor outcome in stroke is related to mechanisms visible on CT scan. This study shows that BP levels at entry into the trial (≈24 hours after stroke onset) are

associated with CT findings. Early infarction, as visible on the baseline CT, appears to be independently related to lower BP. In contrast, other apparent associations between baseline CT findings and BP (dense middle cerebral artery, hemioedema, mass effect) disappear after adjustment.

The association between lower BP and evidence of acute infarction may be related to clinical subtype; therefore, stroke severity. Data from this study concur with that from IST, which revealed that patients with a total anterior circulation strokes (TACS, Oxford Community Stroke Project classification¹⁵) had lower blood pressures;¹ TACS are more likely to be visible on early CT scans.¹⁷ This is in accordance with

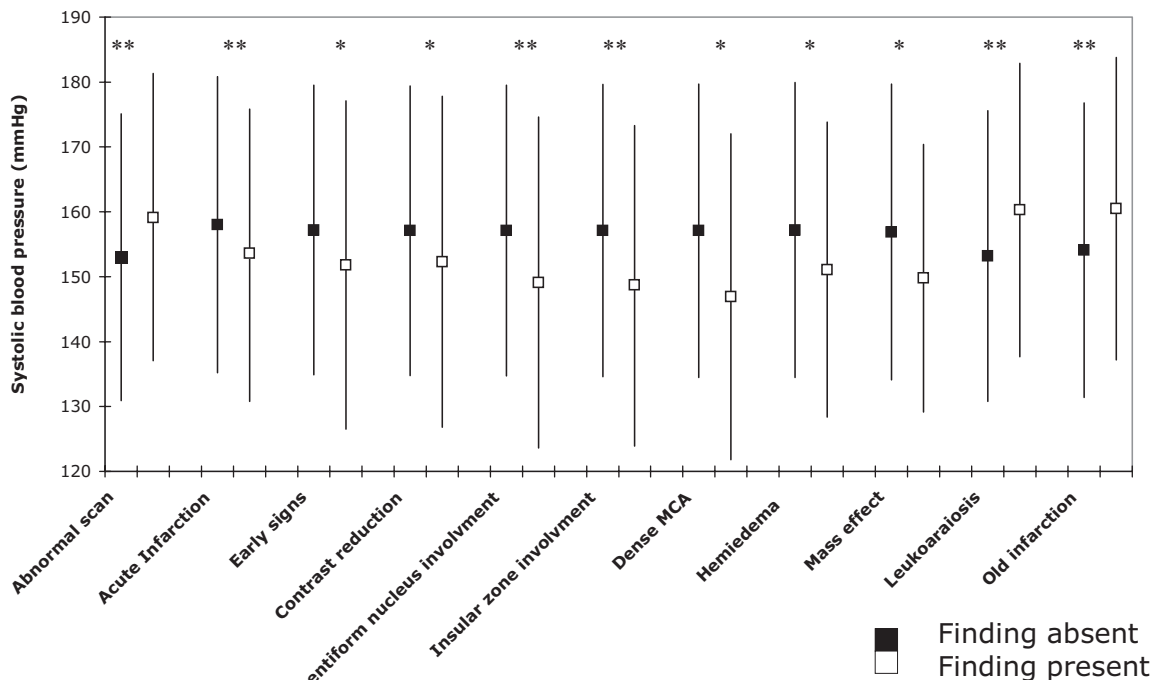


Figure 1. Stock chart showing baseline SBP (mean, SD) in baseline scan by type of abnormality. The BP scale shows the top end of the BP scale and does not cross the x-axis at 0. SD bars; *significant before adjustment; **significant after adjustment; MCA, middle cerebral artery.

Table 2. Relationship Between Hemodynamic Factors and Day 10 CT Findings

CT Findings at Day 10	N of Patients Finding Present/Absent/Missing	SBP OR (95% CI)	
		Unadjusted	Adjusted
Abnormal scan	367/930/189	0.98 (0.93–1.04)	0.99 (0.99–1.00)
Infarction evident	912/383/191	1.02 (0.96–1.07)	1.06 (1.00–1.13)
Nonhemorrhagic infarction	483/815/188	1.08 (1.03–1.14)	1.08 (1.03–1.14)
Hemorrhagic infarction	435/862/189	0.93 (0.87–0.98)	0.96 (0.91–1.02)
Petechial bleeding >50%	143/286/6	1.04 (0.95–1.14)	1.04 (0.94–1.15)
Intrafarcit hematoma	14/1284/188	0.99 (0.79–1.24)	0.98 (0.77–1.23)
Additional hematoma	1/1278/207	0.96 (0.40–2.30)	1.06 (0.46–2.45)
Blood in ventricles	4/1275/207	0.92 (0.60–1.42)	0.90 (0.59–1.38)
Total superficial bleed	48/449/NA	1.03 (0.90–1.17)	1.07 (0.94–1.20)
Partial superficial bleed	343/120/NA	0.95 (0.90–1.00)	0.97 (0.91–1.02)
Deep bleed	237/212/NA	0.93 (0.88–0.99)	0.96 (0.90–1.03)
Multiple infarcts	14/1264/208	0.89 (0.71–1.13)	0.90 (0.71–1.15)
Mass effect	215/1063/208	0.94 (0.88–1.00)	0.98 (0.91–1.06)
Midline shift	23/1254/209	0.90 (0.76–1.10)	1.01 (0.83–1.20)

ORs are for each 10-mm Hg change in BP.

Controlled for age, sex, baseline Scandinavian stroke scale, time to treatment, and treatment group.

$P < 0.05$ are shown in bold.

the observation that patients with early signs of acute infarction had a lower BP in this analysis. Low BP is also associated with poor outcome after stroke after adjustment for stroke severity.¹ This may be because of concurrent medical problems (cardiac failure, sepsis), or it may be hypothesized that low perfusion pressure in the ischemic penumbra causes it to develop into frank infarction.

Additionally, a key confounding variable is time between ictus and scan because BP declines progressively from stroke onset over the first hours and days.¹⁸ CT findings in acute

ischemic stroke take time to develop after clinical presentation so it may be that the lower BP seen with some CT features are explained by longer ictus to scan times; statistical adjustment may not completely control for this.

Analysis of the TAIST data provide some insights into the association between BP and poor outcome. Although the link between high BP and poor outcome after stroke is well-established, the explanation for this relationship remains unclear. Suggested mechanisms include increased recurrence,^{1,9} fatal cerebral edema,¹ and increased hemorrhagic transformation.^{4–6}

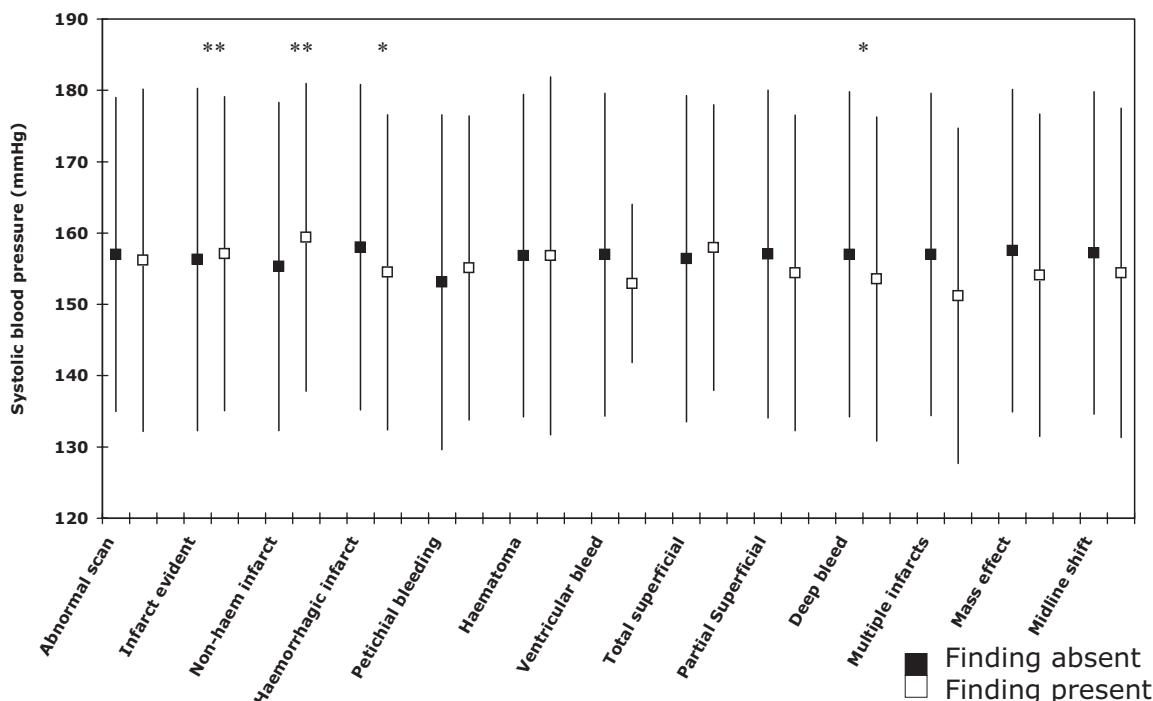


Figure 2. Bar chart showing baseline SBP (mean, SD) in day 10 scan by type of abnormality. The BP scale shows the top end of the BP scale and does not cross the x-axis at 0. SD bars; *significant before adjustment; **significant after adjustment.

Analysis of the TAIST data based on day 0 and 10 CT scans does not reveal a significant association between hypertension and cerebral edema, which was present on 11% of baseline scans. In contrast, data from IST reported a possible relationship between BP and fatal (presumed) cerebral edema; in IST, the outcome of fatal cerebral edema was derived and not dependent on clinical or imaging data.¹ A case-control analysis of patients with ischemic stroke revealed a relationship between fatal cerebral edema and 12-hour systolic hypertension,¹⁹ although this does not prove causality. The cerebral edema may have caused an increase in BP through a Cushing response or, alternatively, been a consequence of raised BP caused by vasoconstriction and breakdown of the blood–brain barrier.

One explanation for the current findings is the timing of the CT scans. Baseline scans were performed on average 15 hours after stroke onset at a time that may be too early to detail many cases of cerebral edema. Similarly, scans at day 10 may also miss the peak incidence of cerebral edema, and 70 patients with severe stroke died before day 10. This is supported by data from 20 of these 70 patients who had an adjudicated clinical scan before death; in this group, early death was strongly associated with the presence of mass effect, although the number was too small to confirm an association with SBP. Another explanation is that exclusion of patients with hemorrhagic transformation on baseline scan from enrollment in the trial. This will have excluded the most severe strokes and, therefore, those most prone to cerebral edema.

Analysis of the TAIST data also fails to confirm experimental studies that show a link between baseline hypertension and hemorrhagic transformation of the infarction.^{4–6} Although patients with hemorrhage on baseline scan were excluded from TAIST, one-third of patients had evidence of hemorrhagic transformation (defined as petechial bleeding within the infarct) at day 10. There was no evidence of an association between baseline BP and subsequent hemorrhagic transformation or amount of petechial bleeding. There were no significant associations between more clinically relevant bleeds (hematoma formation, intraventricular bleeds or symptomatic bleeds) and BP, although less of these events occurred, reducing statistical power. Analysis of IST also failed to show a link between BP and hemorrhagic transformation.¹

However, this analysis cannot provide information on the relationship between BP at onset and hemorrhagic transformation in the hyperacute phase of ischemic stroke, because patients with early evidence of hemorrhagic transformation were excluded from this trial. Further information from hyperacute studies are needed to examine effects of BP in the first few hours after onset. A recent retrospective analysis of 386 placebo-treated patients in the ECASS II thrombolysis trial did not show an association between baseline BP (taken within 6 hours) and hemorrhagic transformation.⁸

The presence of leukoariosis and old infarctions are significantly associated with high BP at baseline. These imaging changes are chronic and will have antedated the index event. TAIST confirms earlier studies showing a link between high BP, leukoariosis, and recurrent infarction.^{20–22}

At day 10, the only CT finding that was associated with high BP after adjustment was the presence of a visible infarction. Statistical adjustment included baseline severity, so the presence of this visible CT lesion is not primarily related to the severity of the presenting stroke. This may be because most strokes were lacunar (42% in the TAIST trial using a modified version of the TOAST classification¹⁶) and, whereas these strokes are less likely to be visible acutely, they are associated with leukoariosis and past history of hypertension, which are associated with high baseline BP.⁹ It may also be that patients with long-standing hypertension, who are more likely to have a higher BP in the days after stroke onset, have more severe atherosclerosis, and, therefore, more severe infarcts.

Although the findings on the relationship between baseline BP and early CT findings are novel, there are several limitations to this study. Importantly, the data come from a randomized controlled trial with specific inclusion–exclusion criteria so that selection bias will be present inherently. In particular, patients with a very high BP (BP >220/120 mm Hg) were excluded from the trial; therefore, there are no data on severely hypertensive patients who may have significant CT findings. However, IST showed patients with SBP over 170 mm Hg to have worse outcomes, and previous analysis TAIST trial data shows hypertension to be related to poor outcome.¹ Additionally, patients with hemorrhagic transformation on baseline scan were excluded, which will have led to an underrepresentation of patients with very severe stroke. Finally, CT scans at day 10 may have missed some infarcts caused by “fogging.”²³ However, 912 of 1484 patients had evidence of infarct at day 10, so this problem appears to be minimal. Nevertheless, the data come from a large, high-fidelity, double-blinded, double-dummy trial, and the nature of the trial with blinded adjudication of CT scans and blinding to treatment arrangement makes analysis of BP and CT findings robust. This article does not address outcome in patients with elevated BP in the TAIST trial, because this has already been assessed and published.⁹ This analysis was designed to assess possible mechanisms from CT findings rather than outcome.

Overall, this analysis provides insight into the association between baseline BP and CT findings in acute and subacute stroke. Although we have not shown cerebral edema or hemorrhagic transformation to be related to BP, patients with elevated SBP are more likely to have visible infarctions by day 10, an intermediate explanation for a poor functional outcome. This is an exploratory analysis, and as such there can be no firm recommendations for the treating physician regarding hypertension in acute ischemic stroke. There are currently large-scale ongoing trials in this area (SCAST and ENOS²⁴) which should provide robust data on which to base clinical practice, as well as the opportunity to prospectively analyze CT data in relation to BP, including the influence of changing BP in acute stroke.

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Disclosures

None.

References

- Leonardi-Bee J, Bath PMW, Phillips SJ, Sandercock PAG, for the IST Collaborative Group. Blood pressure and clinical outcomes in the international stroke trial. *Stroke*. 2002;33:1315–1320.
- Willmot M, Leonardi-Bee J, Bath PM. High blood pressure in acute stroke and subsequent outcome: A systematic review. *Hypertension*. 2004;43:18–24.
- Toyoda K, Okada Y, Kobayashi S. Early recurrence of ischemic stroke in Japanese patients: The Japan standard stroke registry study. *Cerebrovasc Dis*. 2007;24:289–295.
- Bowes MP, Zivin JA, Thomas GR, Thibodeaux H, Fagan SC. Acute hypertension, but not thrombolysis, increases the incidence and severity of hemorrhagic transformation following experimental stroke in rabbits. *Exp Neurol*. 1996;141:40–46.
- Saku Y, Choki J, Waki R, Masuda J, Tamaki K, Fujishima M, Ogata J. Hemorrhagic infarct induced by arterial hypertension in cat brain following middle cerebral artery occlusion. *Stroke*. 1990;21:589–595.
- Fagan SC, Bowes MP, Lyden PD, Zivin JA. Acute hypertension promotes hemorrhagic transformation in a rabbit embolic stroke model: Effect of labetalol. *Exp Neurol*. 1998;150:153–158.
- International Stroke Trial Collaborative Group. The international stroke trial (IST); a randomised trial of aspirin, subcutaneous heparin, both, or neither among 19435 patients with acute ischaemic stroke. *Lancet*. 1997;349:1569–1581.
- Yong M, Kaste M. Association of characteristics of blood pressure profiles and stroke outcomes in the ECASS-II trial. *Stroke*. 2008;39:366–372.
- Sprigg N, Gray LJ, Bath PMW, Boysen G, De Deyn PP, Friis P, Leys D, Marttila R, Olsson J-E, O'Neill D, Ringelstein B, van der Sande J-J, Lindenstrom E, for the TAIST investigators. Relationship between outcome and baseline blood pressure and other haemodynamic measures in acute ischaemic stroke: Data from the taist trial. *J Hypertens*. 2006;24:1413–1417.
- Bath P, Lindenstrom E, Boysen G, De Deyn P, Friis P, Leys D, Marttila R, Olsson J-E, O'Neill D, Orgogozo J-M, Ringelstein B, van der Sande J-J, Turpie AGG. Tinzaparin in acute ischaemic stroke (TAIST): A randomised aspirin-controlled trial. *Lancet*. 2001;358:702–710.
- The Multicenter Acute Stroke Trial - Europe Study Group. Thrombolytic therapy with streptokinase in acute ischemic stroke. *N Engl J Med*. 1996;335:145–150.
- Hommel M, for The FISS bis Investigators Group. Fraxiparine in ischaemic stroke study (FISS bis). *Cerebrovasc Dis*. 1998;8:19(abstract).
- Moulin T, Cattin F, Crepin-Leblond T, Tatu L, Chavot D, Piotin M, Viel JF, Rumbach L, Bonneville JF. Early ct signs in acute middle cerebral artery infarction: Predictive value for subsequent infarct locations and outcome. *Neurology*. 1996;47:366–375.
- Moulin T, Crepin-Leblond T, Chopard JL, Bogousslavsky J. Hemorrhagic infarcts. *Eur Neurol*. 1994;34:64–77.
- Bamford J, Sandercock P, Dennis M, Burn J, Warlow C. Classification and natural history of clinically identifiable subtypes of cerebral infarction. *Lancet*. 1991;337:1521–1526.
- Adams HP, Bendixen BH, Kappelle LJ, Biller J, Love BB, Gordon DL, Marsh EE, The TOAST Investigators. Classification of subtype of acute ischemic stroke. Definitions for use in a multicenter clinical trial. *Stroke*. 1993;24:35–41.
- Mead GE, Lewis SC, Wardlaw JM, Dennis MS, Warlow CP. How well does the Oxfordshire community stroke project classification predict the site and size of the infarct on brain imaging? *J Neurol Neurosurg Psychiatry*. 2000;68:558–562.
- Christensen H, Meden P, Overgaard K, Boysen G. The course of blood pressure in acute stroke is related to the severity of the neurological deficits. *Acta Neurol Scand*. 2002;106:142–147.
- Krieger DW, Demchuk AM, Kasner SE, Jauss M, Hantson L. Early clinical and radiological predictors of fatal brain swelling in ischemic stroke. *Stroke*. 1999;30:287–292.
- Khan U, Porteous L, Hassan A, Markus HS. Risk factor profile of cerebral small vessel disease and its subtypes. *J Neurol Neurosurg Psychiatry*. 2007;78:702–706.
- Fu JH, Lu CZ, Hong Z, Dong Q, Luo Y, Wong KS. Extent of white matter lesions is related to acute subcortical infarcts and predicts further stroke risk in patients with first ever ischaemic stroke. *J Neurol Neurosurg Psychiatry*. 2005;76:793–796.
- Arboix A, Font A, Garro C, Garca-Eroles L, Comes E, Massons J. Recurrent lacunar infarction following a previous lacunar stroke: A clinical study of 122 patients. *J Neurol Neurosurg Psychiatry*. 2007;78:1392–1394.
- De Reuck J, Van de Velde E, Van Maele G, Wissaert W. The prognostic significance of changes in x-ray attenuation on ct in established cerebral infarcts. *Cerebrovasc Dis*. 2003;16:114–121.
- The ENOS Trial Investigators. Glyceryl trinitrate vs. Control, and continuing vs stopping temporarily prior antihypertensive therapy, in acute stroke: Rationale and design of the efficacy of nitric oxide in stroke (ENOS) trial (ISTCRN99414122). *Int J Stroke*. 2006;1:245–249.

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