Yield and Accuracy of Urgent Combined Carotid/Transcranial Ultrasound Testing in Acute Cerebral Ischemia

Oleg Y. Chernyshev, MD, PhD; Zsolt Garami, MD; Sergio Calleja, MD; Joon Song, MD;
Morgan S. Campbell, MD; Elizabeth A. Noser, MD; Hashem Shaltoni, MD; Chin-I Chen, MD;
Yasuyuki Iguchi, MD, PhD; James C. Grotta, MD; Andrei V. Alexandrov, MD

Background and Purpose—We routinely perform an urgent bedside neurovascular ultrasound examination (NVUE) with carotid/vertebral duplex and transcranial Doppler (TCD) in patients with acute cerebral ischemia. We aimed to determine the yield and accuracy of NVUE to identify lesions amenable for interventional treatment (LAITs).

Methods—NVUE was performed with portable carotid duplex and TCD using standardized fast-track (<15 minutes) insonation protocols. Digital subtraction angiography (DSA) was the gold standard for identifying LAIT. These lesions were defined as proximal intra- or extracranial occlusions, near-occlusions, $\geq 50\%$ stenoses or thrombus in the symptomatic artery.

Results—One hundred and fifty patients (70 women, mean age 66 ± 15 years) underwent NVUE at median 128 minutes after symptom onset. Fifty-four patients (36%) received intravenous or intra-arterial thrombolysis (median National Institutes of Health Stroke Scale (NIHSS) score 14, range 4 to 29; 81% had NIHSS ≥ 10 points). NVUE demonstrated LAITs in 98% of patients eligible for thrombolysis, 76% of acute stroke patients ineligible for thrombolysis (n=63), and 42% in patients with transient ischemic attack (n=33), $P<0.001$. Urgent DSA was performed in 30 patients on average 230 minutes after NVUE. Compared with DSA, NVUE predicted LAIT presence with 100% sensitivity and 100% specificity, although individual accuracy parameters for TCD and carotid duplex specific to occlusion location ranged 75% to 96% because of the presence of tandem lesions and 10% rate of no temporal windows.

Conclusions—Bedside neurovascular ultrasound examination, combining carotid/vertebral duplex with TCD yields a substantial proportion of LAITs in excellent agreement with urgent DSA. (*Stroke*. 2005;36:32-37.)

Key Words: carotid stenosis ■ embolism ■ stroke, acute ■ thrombolysis ■ ultrasonography, Doppler

Bedside neurovascular ultrasound can play an important role in the diagnostic work up of acute stroke patients.[1–6] Urgent detection, localization, and grading severity of the arterial obstruction help triage patients with acute cerebral ischemia and patient selection for invasive angiography. Several reports evaluated the accuracy of either transcranial Doppler (TCD) or carotid duplex ultrasound (CD) in acute stroke setting.[2,7,8] Few studies reported the combined use of TCD and CD in stroke patients.[3,9–11] None reported both the yield and accuracy of combined ultrasound assessment in acute cerebral ischemia. Combined TCD and CD can be used as a screening test for lesions amenable for interventional treatment (LAITs).[12]

Considering previous publications[2,3,5,7,13] and our own experience, we developed a fast-track neurovascular ultrasound examination (NVUE) protocol for an experienced sonographer to urgently evaluate acute stroke patients at bedside. We also developed detailed diagnostic criteria for normal, stenosed, and acutely occluded intracranial and extracranial vessels.[13–17]

We routinely perform urgent bedside NVUEs with both standard fast-track TCD and carotid/vertebral duplex in patients with acute cerebral ischemia. We aimed to determine the yield and accuracy of these combined modalities to identify LAITs.

Subjects and Methods

To perform NVUE at bedside, we used portable 2 MHz power-motion or single-channel TCD (PMD 100, Spencer Technologies; Ez-Dop, DWL; Companion III, Nicolet) and portable carotid duplex ultrasound (Sonosite 180 Plus) equipped with B-mode, power-mode, angle-corrected spectral Doppler and 5 to 10 MHz linear probe.

An experienced sonographer and stroke neurologist arrived concurrently. The former performed and the latter evaluated urgent bedside NVUE results. To avoid delays in routine patient evaluation

TABLE 1. Fast-Track Neurovascular Ultrasound Examination

Use portable devices with bright display overcoming room light. Stand behind patient headrest. Start with TCD because acute occlusion responsible for the neurological deficit is likely located intracranially. Extracranial carotid/vertebral duplex may reveal an additional lesion often responsible for intracranial flow disturbance. Fast-track insonation steps follow clinical localization of patient symptoms.

A. Clinical Diagnosis of Cerebral Ischemia in the Anterior Circulation

    STEP 1: Transcranial Doppler

1. If time permits, begin insonation on the nonaffected side to establish the temporal window, normal MCA waveform (M1 depth 45–65 mm, M2 30–45 mm), and velocity for comparison to the affected side.

2. If short on time, start on the affected side: first assess MCA at 50 mm. If no signals detected, increase the depth to 62 mm. If an antegrade flow signal is found, reduce the depth to trace the MCA stem or identify the worst residual flow signal. Search for possible flow diversion to the ACA, PCA, or M2 MCA. Evaluate and compare waveform shapes and systolic flow acceleration.

3. Continue on the affected side (transorbital window). Check flow direction and pulsatility in the OA at depths 40–50 mm followed by ICA siphon at depths 55–65 mm.

4. If time permits or in patients with pure motor or sensory deficits, evaluate BA (depth 80–100+ mm) and terminal VA (40–80 mm).

    STEP 2: Carotid/Vertebral Duplex

1. Start on the affected side in transverse B-mode planes followed by color or power-mode sweep from proximal to distal carotid segments. Identify CCA and its bifurcation on B-mode and flow-carrying lumens.

2. Document if ICA (or CCA) has a lesion on B-mode and corresponding disturbances on flow images. In patients with concomitant chest pain, evaluate CCA as close to the origin as possible.

3. Perform angle-corrected spectral velocity measurements in the mid-to-distal CCA, ICA and external carotid artery.

4. If time permits or in patients with pure motor or sensory deficits, examine cervical portion of the vertebral arteries (longitudinal B-mode, color or power mode, spectral Doppler) on the affected side.

5. If time permits, perform transverse and longitudinal scanning of the arteries on the nonaffected side.

B. Clinical Diagnosis of Cerebral Ischemia in the Posterior Circulation

    STEP 1: Transcranial Doppler

1. Start suboccipital insonation at 75 mm (VA junction) and identify BA flow at 80–100+ mm.

2. If abnormal signals present at 75–100 mm, find the terminal VA (40–80 mm) on the nonaffected side for comparison and evaluate the terminal VA on the affected side at similar depths.

3. Continue with transtemporal examination to identify PCA (55–75 mm) and possible collateral flow through the posterior communicating artery (check both sides).

4. If time permits, evaluate both MCAs and ACAs (60–75 mm) for possible compensatory velocity increase as an indirect sign of basilar artery obstruction.

    STEP 2: Vertebral/Carotid Duplex Ultrasound

1. Start on the affected side by locating CCA using longitudinal B-mode plane, and turn transducer downward to visualize shadows from transverse processes of midcervical vertebrae.

2. Apply color or power modes and spectral Doppler to identify flow in intratransverse VA segments.

3. Follow VA course to its origin and obtain Doppler spectra. Perform similar examination on another side.

4. If time permits, perform bilateral duplex examination of the CCA, ICA, and external carotid artery as described above.

ACA indicates anterior cerebral artery; CCA, common carotid artery; ECA, external carotid artery; ophthalmic artery, OA; PCA, posterior cerebral artery; BA, basilar artery; and VA, vertebral artery.

and thrombolytic therapy, bedside ultrasound examination was carried out simultaneously with clinical assessment, blood draws, computed tomography (CT), and chest x-ray, etc. Sonographers used standardized fast-track (<15 minutes) insonation protocols (Table 1) to identify suspected arterial obstruction. The neurological evaluation guided the sequence of vessels targeted by NVUE. Ultrasound results were interpreted at bedside without knowledge of angiographic results using previously published diagnostic criteria[13–19] (Table 2). In patients with no temporal windows, we performed a non-contract–enhanced TCD examination of the orbital and posterior circulation vessels and carotid/vertebral duplex.

Patients received recombinant tissue plasminogen activator intravenously within 3 hours in a dose of 0.9 mg/kg as the standard of care.[20] Patients were also screened for eligibility for experimental intra-arterial rescue using institutional review board (IRB)–approved protocols. At our center, both ultrasound studies are initiated emergently without specific written informed consent in order not to delay institution of appropriate therapies or management strategies or select patients for experimental treatment. IRB approval was obtained in all cases when patients participated in our experimental protocols. All IRB approvals of

our hyperacute experimental treatment protocols have mention of ultrasound tests as part of initial evaluation for symptoms of stroke. Patients presenting with sustained neurological deficits outside protocol time windows from symptom onset or hypoattenuation on CT scan did not receive interventional treatment. Patients with resolved neurological deficits at the time of examination were considered having transient ischemic attacks (TIAs).

Stroke neurologists decided to perform digital subtraction angiography (DSA) if patients either met clinical CT criteria for an IRB-approved experimental intra-arterial rescue protocol or ultrasound showed persisting arterial occlusion or reocclusion after full-dose intravenous recombinant tissue plasminogen activator therapy. DSA was the gold standard for the diagnosis of LAIT. LAIT was defined as an occlusion or near-occlusion, or ≥50% stenoses or thrombi in an artery (arteries) supplying brain area(s) affected by ischemia. Hence, current definition of LAIT may include chronic lesions, and caution should be exercised if these lesions are directly responsible for current patient symptoms. Patients with TIA, lacunar syndromes, and normal ultrasound findings were least likely to undergo urgent DSA.

TABLE 2.   Ultrasound Screening Criteria for Lesions Amenable for Intervention

Lesion Location	TCD Criteria (At Least One Present)	CD Criteria
M1/ M2 MCA	Primary:	
	Thrombolysis in brain infarction (TIBI) grades 0–4 (absent, minimal, blunted, dampened, or stenotic) at depths <45 mm (M2) and 45–65 mm (M1)	Extracranial findings may be normal or showing decreased ICA velocity unilateral to lesion
	Secondary:	
	Flow diversion to ACA, PCA, or M2 Increased resistance in unilateral TICA Embolic signals in MCA Turbulence, disturbed flow at stenosis Nonharmonic and harmonic covibrations (bruit or pure musical tones)	
TICA	Primary:	
	TIBI grades 0–4 at 60–70 mm Increased velocities suggest anterior cross-filling or collateral flow in posterior communicating artery	Decreased ICA velocity unilateral to lesion or normal extracranial findings
	Secondary:	
	Embolic signals in unilateral MCA Blunted unilateral MCA, MFV>20 cm/s	
Proximal ICA	Primary:	
	Increased flow velocities suggest anterior cross-filling through ACommA or collateral flow through PcommA Reversed OA Delayed systolic flow acceleration in or blunted ipsilateral MCA, MFV>20 cm/s	B-mode evidence of a lesion in ICA±CCA; Flow imaging evidence of no flow or residual lumen;
	Secondary:	ICA >50% stenosis: PSV >125 cm/s; EDV >40 cm/s; ICA/CCA PSV ratio >2
	Embolic signals in unilateral MCA Normal OA direction due to retrograde filling of siphon	ICA near-occlusion or occlusion: Blunted, minimal, reverberating or absent spectral Doppler waveforms in ICA
Tandem ICA/MCA stenosis/occlusion	Primary:	
	TIBI grades 0–4 And: Increased velocities in contralateral ACA, MCA, or unilateral PCommA Or: Reversed unilateral OA	B-mode evidence of a lesion in ICA±CCA; Or: Flow imaging evidence of residual lumen or no flow;
		ICA >50% stenosis: PSV >125 cm/s; EDV >40 cm/s; ICA/CCA PSV ratio >2
	Secondary:	
	Delayed systolic flow acceleration in proximal MCA or TICA Embolic signals in proximal MCA or TICA	ICA near-occlusion or occlusion: Blunted, minimal, reverberating, or absent spectral Doppler waveforms in ICA
Basilar artery	Primary:	
	TIBI flow grades 0–4 at 75–100 mm	Extracranial findings may be normal or showing decreased VA velocities or VA occlusion
	Secondary:	
	Flow velocity increase in terminal VA and branches, MCAs, or PCommAs High resistance flow signals in VA(s) Reversed flow direction in distal basilar artery (85 mm)	
Vertebral artery	Primary (intracranial VA occlusion):	
	TIBI flow grades 0–4 at 40–75 mm	Extracranial findings may be normal (intracranial VA lesion) or showing decreased VA velocities or VA occlusion
	Primary (extracranial VA occlusion):	
	Absent, minimal, or reversed high resistance flow signals in unilateral terminal VA	
	Secondary:	
	Embolic signals Increased velocities or low pulsatility in contralateral VA	

LAITs were defined as obstruction/near obstruction or ≥50% stenosis of (1) M1 or M2 segments of MCA, (2) ICA, (3) tandem ICA/MCA, or (4) vertebrobasilar arteries (VB).

TICA indicates terminal internal carotid artery; TIBI, Thrombolysis In Brain Infarction; ACommA, anterior communicating artery; PCommA, posterior communicating artery.
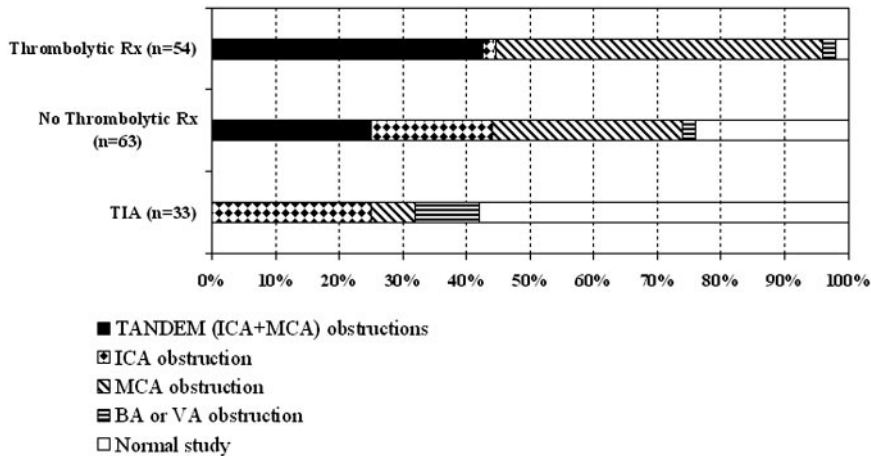
Figure 1. Yield of NVUE in acute cerebral ischemia. Ultrasound screening for LAIT was positive in 98% of patients eligible for thrombolysis, 76% of acute stroke patients ineligible for thrombolysis, and 42% in patients with TIAs ($P<0.001$).

After neuroradiologists dictated DSA reports, we compared NVUE results to DSA to determine true-positive, true-negative, false-positive, and false-negative ultrasound results. We calculated accuracy parameters, including sensitivity, specificity, and positive and negative predictive values. We used χ^2 analysis for dichotomous variables, Student t test for normally distributed continuous variables, and Wilcoxon 2-sample test for variables with nonparametric distribution. We determined statistical significance at a 2-tailed $P<0.05$.

Results

We studied 150 consecutive patients (80 men, 70 women, mean age 66 ± 15 years) with NVUE on average 238 ± 323 minutes (median time 128 minutes) after symptom onset.

Fifty-four patients (36%) received either IV (n=24) or intra-arterial (IA, n=5) thrombolytic therapy or both (n=25). Their median National Institutes of Health Stroke Scale (NIHSS) score was 14 points (range 4 to 29); 81% had NIHSS ≥10 points. No delay in treatment resulted from ultrasound testing. Sixty-three (42%) patients were found ineligible for thrombolysis. Thirty-three patients (22%) had TIAs.

NVUE showed abnormal results consistent with our ultrasound criteria for LAIT in 98% of patients eligible for thrombolysis, 76% of acute stroke patients ineligible for thrombolysis, and 42% in patients with TIA, $P<0.001$ (Figure 1). In the thrombolysis group, 44% had internal carotid artery (ICA) lesions with or without middle cerebral artery (MCA) lesions, 52% had isolated MCA lesions, 2% had lesions in the vertebrobasilar (VB) system, and 2% had normal ultrasound findings. In patients ineligible for thrombolysis, 44% had ICA lesions; 30%, isolated MCA lesions; 2%, VB lesions; and 24% had normal findings. In the TIA group, 25% had isolated ICA lesions; none had tandem ICA+MCA lesions; 7%, isolated MCA lesions; 10%, VB lesions; and 58% had normal findings (Figure 1).

A total of 30 patients (20%) underwent urgent DSA at mean time of 230 ± 207 minutes (maximum delay 308 minutes) after initial NVUE. DSA demonstrated LAIT presence in all patients with abnormal NVUE without false-positive ultrasound studies (sensitivity and specificity for LAIT presence 100%). DSA localized LAIT to the MCA in 10 patients, ICA in 4 patients, tandem ICA/MCA in 15 patients (Figure 2), and VB circulation in 1 patient (Figure 3).
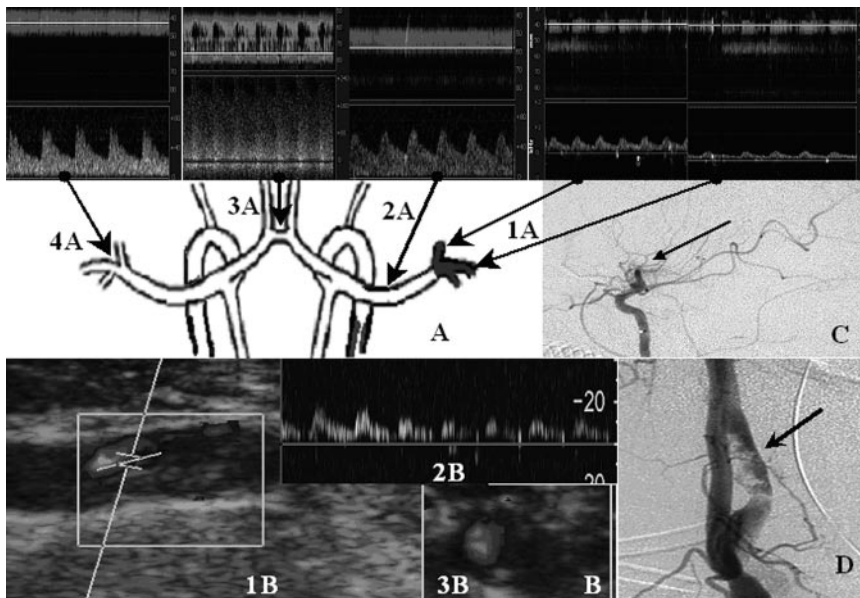
Figure 2. Ninety minutes after the right MCA symptom onset, a 55-year-old woman presented with NIHSS 12 points. Ultrasound showed proximal right ICA and right distal M1/M2 MCA tandem acute occlusions with frequent embolic signals. DSA confirmed lesion localization 1 hour later. Patient underwent experimental intra-arterial thrombolysis with ICA stenting procedures. A, Power-motion TCD showed M1/M2 segments MCA occlusion with microemboli (1A); increased flow at proximal MCA with microemboli (2A); anterior cross-filling through anterior communicating artery (3A); and normal flow in distal MCA on opposite (nonaffected) side (4A). B, Carotid duplex showed proximal ICA obstruction on B-mode image with residual flow lumen on power mode (longitudinal plane 1B and transverse plane 3B). Spectral Doppler showed a minimal flow signal at obstruction site (2B). DSA images showed M1 MCA (C) and proximal ICA obstruction (D).

	TCD +	TCD -
DSA +	25	1
DSA -	1	3

	CD +	CD -
DSA +	18	1
DSA -	1	10

+

	NVUE +	NVUE-
DSA +	30	0
DSA -	0	0

Figure 3. Ultrasound Versus Angiography (n=30). + Indicates positive findings; −, normal findings. DSA was used as the gold standard. Location of LAITs on DSA: MCA=10; ICA/MCA=15; ICA=4. BA/VA=1. BA/VA indicates basilar artery and vertebral artery.

Compared with DSA, the accuracy parameters for each component of NVUE were as follows: TCD 96% sensitivity, 75% specificity, 96% positive, and 75% negative predictive values (given the observed 10% rate of absent temporal windows). Carotid/vertebral duplex had 94% sensitivity, 90% specificity, 94% positive, and 90% negative predictive values (Table 3).

TCD and carotid/vertebral duplex ultrasound each showed 1 false-negative and 1 false-positive study (Figure 3). Tandem ICA/MCA lesions were responsible for these errors because of difficulties identifying the unilateral MCA occlusion with delayed collateral flow in the presence of bilateral carotid diseases and unilateral distal ICA stenosis beyond the reach of carotid duplex scanning area.

Discussion

Our study showed that an urgent combined intra- and extracranial ultrasound examination yields a substantial number of vascular lesions in patients with acute cerebral ischemia. Nearly all patients receiving thrombolysis had lesions amenable for intervention likely due to preponderance of severe strokes and earlier arrival times. Three quarters of patients who had strokes, but did not qualify to receive thrombolysis, still had detectable lesions. These rates are higher than previously reported in studies using solely TCD or CD for stroke evaluation[1,3,7,21,22] likely due to combined testing at the earliest possible time after symptom onset (ie, on admission). NVUE also showed large arterial lesions in 42% of patients with TIAs. Previous studies carried with TCD or CD alone showed detection of arterial lesions in 25% to 35% of these patients.[7,23] Diagnostic tests have even lower yield if done electively.[24,25] Identification of an arterial lesion that persists outside the time window or despite symptom resolution may have serious prognostic implications and deserves further studies regarding acute management of these patients. These findings may ascertain stroke mechanism at bedside and may select most appropriate management or next most informative test. Perhaps, these vascular abnormalities can help to identify the "high-risk TIA" group for early stroke recurrence.

The accuracy of NVUE demonstrated in this study is based on a relatively small sample of urgent invasive angiograms, and the selection bias introduced by abnormal ultrasound findings cannot be ruled out. Therefore, our findings should be interpreted with caution. However, TCD and CD taken separately have lesser accuracy parameters within the same patient sample. Knowledge of shortcomings of each test and the complimentary value of information derived from extra- and intracranial vessels helped to compensate for these shortcomings. Patients with posterior circulation symptoms, greater variability of posterior circulation vessels, and technical difficulties may further affect the performance of these tests.[18,26] In our study, accuracy parameters were largely driven by findings in the anterior circulation. Individual accuracy parameters for TCD and CD were comparable to previous studies.[2,3,5,7,27,28]

Our study has limitations, including the need for considerable sonographer skill and expertise to complete testing promptly and efficiently. Other factors that can affect the yield and accuracy of NVUE include potential selection bias for diagnostic angiography and greater preponderance of patients with severe stroke symptoms or admitted very early after symptom onset. Also, our study had an average delay of over 3 hours between initial ultrasound and DSA that may provide ample time for thrombus propagation, dissolution, or reocclusion to occur. Our study also identified clinical situations when neither TCD nor CD could be reliably interpreted, including tandem lesions with poor collateral flow, limited arterial visualization, and absent temporal windows. Some of these shortcomings can be overcome by using ultrasound contrast agents.[5,19,29–31]

Clinical implications of our findings affirm that rapid bedside ultrasound screening can be used in patient selection for interventional treatment.[32–34] With further developments in portable noninvasive technology, such examination can be feasibly taught to vascular neurologists, emergency physicians, and vascular technologists. The small devices required can become the new "stethoscope" for bedside evaluations.

In conclusion, urgent bedside NVUE yields a substantial proportion of LAITs in patients with acute cerebral ischemia. Transcranial and carotid ultrasound tests, if performed together by experienced sonographers, compensate for each

TABLE 3.   Diagnostic Accuracy of TCD, Carotid Duplex, and Neurovascular Ultrasound Examination

	Sensitivity	Specificity	Positive Predictive Value	Negative Predictive Value
TCD	96	75	96	75
CD	94	90	94	90
NVUE	100	100	100	100

other's shortcomings and can achieve 100% accuracy in detection of lesions amenable for intervention.

References

1. Grolimund P, Seiler RW, Aaslid R, Huber P, Zurbruegg H. Evaluation of cerebrovascular disease by combined extracranial and transcranial Doppler sonography: experience in 1039 patients. *Stroke*. 1987;18:1018–1024.

2. Chambers BR, Merory JR, Smidt V. Doppler diagnosis in cases of acute stroke. *Med J Aust*. 1989;150:382–384.

3. Carmelingo M, Casto L, Censori B, Ferraro B, Gazzaniga GC, Mamoli A. Transcranial Doppler in acute ischemic stroke of the middle cerebral artery territories. *Acta Neurol Scand*. 1993;88:108–111.

4. Iannuzzi A, Wilcosky T, Mercuri M, Rubba P, Bryan FA, Bond MG. Ultrasonographic correlates of carotid atherosclerosis in transient ischemic attack and stroke. *Stroke*. 1995;26:614–619.

5. Lee DH, Gao FQ, Rankin RN, Pelz DM, Fox AJ. Duplex and color Doppler flow sonography of occlusion and near occlusion of the carotid artery. *AJNR Am J Neuroradiol*. 1996;17:1267–1274.

6. Razumovsky AY, Gilliard JH, Bryan RN, Hanley DF, Oppenheimer SM. TCD, MRA, MRI in acute cerebral ischemia. *Acta Neurol Scand*. 1999; 99:65–76.

7. Alexandrov AV, Demchuk AM, Wein TH, Grotta JC. Yield of transcranial Doppler in acute cerebral ischemia. *Stroke*. 1999;30:1604–1609.

8. Garami Z, Calleja S, Labiche L, Chernyshev OY, Campbell M, Malkoff M, Alexandrov AV. Yield of urgent carotid scanning in acute cerebral ischemia. *Stroke*. 2003;34:266. Abstract.

9. Wada K, Kimura K, Minematsu K, Yasaka. M, Uchino M, Yamaguchi T. Combined carotid and transcranial color-coded sonography in acute ischemic stroke. *Eur J Ultrasound*. 2002;15:101–108.

10. Sturzenegger M, Mattle HP, Rivoir A, Baumgartner RW. Ultrasound findings in carotid artery dissection: analysis of 43 patients. *Neurology*. 1995;45:691–698.

11. Comerota AJ, Katz ML, Hosking JD, Hashemi HA, Kerr RP, Carter AP. Is transcranial Doppler a worthwhile addition to screening tests for cerebrovascular disease? *J Vasc Surg*. 1995;21:90–95.

12. IMS Study Investigators. Combined intravenous and intra-arterial recanalization for acute ischemic stroke: the Interventional Management of Stroke Study. *Stroke*. 2004;35:904–911.

13. Grant EG, Benson CB, Moneta GL, Alexandrov AV, Baker JD, Bluth EI, Carroll BA, Eliasziw M, Gocke J, Hertzberg BS, Katanick S, Needleman L, Pellerito J, Polak JF, Rholl KS, Wooster DL, Zierler E. Carotid artery stenosis: grayscale and Doppler US diagnosis: Society of Radiologists in Ultrasound Consensus Conference. *Radiology*. 2003;229:340–346.

14. Demchuk AM, Burgin WS, Christou I, Felberg RA, Barber PA, Hill MD, Alexandrov AV. Thrombolysis in Brain Infarction (TIBI) transcranial Doppler flow grades predict clinical severity, early recovery, and mortality in patients treated with intravenous tissue plasminogen activator. *Stroke*. 2001;32:89–93.

15. Burgin WS, Wojner AW, Demchuk AM, Felberg RA, Barber PA, Hill MD, Malkoff M, El-Mitwalli A, Piriyawat P, Newcommon N, Button A. Validity and reliability of the Thrombolysis In Brain Infarction (TIBI) flow grades. *Stroke*. 2001;32:324. (Abstract).

16. El-Mitwalli A, Saad M, Christou I, Malkoff M, Alexandrov AV. Clinical and sonographic patterns of tandem internal carotid artery/middle cerebral artery occlusion in tissue plasminogen activator-treated patients. *Stroke*. 2002;33:99–102.

17. Christou I, Felberg RA, Demchuk AM, Grotta JC, Burgin WS, Malkoff M, Alexandrov AV. Accuracy parameters of a broad diagnostic battery for bedside transcranial Doppler to detect flow changes with internal carotid artery stenosis or occlusion. *J Neuroimaging*. 2001;11:236–242.

18. Demchuk AM, Christou I, Wein TH, Felberg RA, Malkoff M, Grotta JC, Alexandrov AV. Accuracy and criteria for localizing arterial occlusion with transcranial Doppler. *J Neuroimaging*. 2000;10:1–12.

19. Furst G, Saleh A, Wenserski F, Malms J, Cohnen M, Aulich A, Neumann-Haefelin T, Schroeter M, Steinmetz H, Sitzer M. Reliability and validity of noninvasive imaging of internal carotid artery pseudo-occlusion. *Stroke*. 1999;30:1444–1449.

20. National Institute of Neurological Disorders and Stroke rt-PA Stroke Study Group. Tissue plasminogen activator for acute ischemic stroke. *N Engl J Med*. 1995;333:1581–1587.

21. Zanette EM, Fieschi C, Bozzao L, Roberti C, Toni D, Argentino C, Lenzi GL. Comparison of cerebral angiography and transcranial Doppler sonography in acute stroke. *Stroke*. 1989;20:899–903.

22. Brown PB, Zwiebel WJ, Call GK. Degree of cervical carotid artery stenosis and hemispheric stroke: duplex US findings. *Radiology*. 1989; 170:541–543.

23. Alexandrov AV, Felberg RA, Demchuk AM, Christou I, Burgin WS, Malkoff M, Wojner AW, Grotta JC. Deterioration following spontaneous improvement: sonographic findings in patients with acutely resolving symptoms of cerebral ischemia. *Stroke*. 2000;31:915–919.

24. Rolak LA, Gilmer W, Strittmatter WJ. Low yield in the diagnostic evaluation of transient ischemic attacks. *Neurology*. 1990;40:747–748.

25. Johnston SC, Gress DR, Browner WS, Sidney S. Short-term prognosis after emergency department diagnosis of TIA. *JAMA*. 2000;284: 2901–2906.

26. Barber PA, Demchuk AM. Transcranial Doppler sonography in acute stroke. *J Neurol Neurosurg Psychiatry*. 2000;68:532–541.

27. Felberg RA, Christou I, Demchuk AM, Malkoff M, Alexandrov AV. Screening for intracranial stenosis with transcranial Doppler: the accuracy of mean flow velocity thresholds. *J Neuroimaging*. 2002;12:9–14.

28. Humphrey P, Sandercock P, Slattery J. A simple method to improve the accuracy of non-invasive ultrasound in selecting TIA patients for cerebral angiography. *J Neurol Neurosurg Psychiatry*. 1990;53:966–971.

29. Nabavi DG, Droste DW, Kemeny V, Schulte-Altedorneburg G, Weber S, Ringelstein EB. Potential and limitations of echocontrast-enhanced ultrasonography in acute stroke patients: a pilot study. *Stroke*. 1998;29: 949–954.

30. Gahn G, Gerber J, Hallmeyer S, Hahn G, Ackerman RH, Reichmann H, von Kummer R. Contrast-enhanced transcranial color-coded duplex sonography in stroke patients with limited bone windows. *AJNR Am J Neuroradiol*. 2000;21:509–514.

31. Santalucia P, Feldmann E. The basic transcranial Doppler examination: technique and anatomy. In: Babikian V, Wechler L, Eds. *Transcranial Doppler Ultrasonography*. 2nd ed. Boston, Ma: Butterworth-Heinemann; 1999:13–31.

32. Nakano S, Yokogami K, Ohta H, Yano T, Ohnishi T. Direct percutaneous transluminal angioplasty for acute middle cerebral artery occlusion. *AJNR Am J Neuroradiol*. 1998;19:767–772.

33. Song JK, Cacayorin ED, Campbell MS, Fisher S, Malkoff MD, Alexandrov AV, Grotta JC. Intracranial balloon angioplasty of acute terminal internal carotid artery occlusion. *AJNR Am J Neuroradiol*. 2002; 23:1308–1312.

34. Eckstein HH, Schumacher H, Dorfler A, Forsting M, Jansen O, Ringleb P, Allenberg JR. Carotid endarterectomy and intracranial thrombolysis: simultaneous and staged procedures in ischemic stroke. *J Vasc Surg*. 1999;29:459–471.

Stroke

American Heart Association

American Stroke Association®

Yield and Accuracy of Urgent Combined Carotid/Transcranial Ultrasound Testing in Acute Cerebral Ischemia

Oleg Y. Chernyshev, Zsolt Garami, Sergio Calleja, Joon Song, Morgan S. Campbell, Elizabeth A. Noser, Hashem Shaltoni, Chin-I Chen, Yasuyuki Iguchi, James C. Grotta and Andrei V. Alexandrov

The online version of this article, along with updated information and services, is located on the World Wide Web at:

http://stroke.ahajournals.org/content/36/1/32