Transcranial Doppler detection of anterior cerebral artery vasospasm

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Abstract

The performance of transcranial Doppler in the detection of anterior cerebral artery vasospasm and vasospasm in patients after subarachnoid haemorrhage was analysed. Transcranial Doppler and cerebral angiography were performed within the same 24 hours on each of 41 patients with acute subarachnoid haemorrhage. Sensitivity and specificity of transcranial Doppler to classify middle cerebral arteries, anterior cerebral arteries, and patients with angiographic vasospasm were determined at mean velocities of 120 and 140 cm/s. Accuracy of transcranial Doppler was better at 140 than at 120 cm/s. For the middle cerebral artery, sensitivity was 86%, specificity 98%. For the anterior cerebral artery, sensitivity was 13%, specificity 100%. Among all patients, sensitivity was 45%, specificity 96%. Among patients with anterior communicating artery aneurysms, sensitivity was 14%, specificity 90%. Therefore, transcranial Doppler accurately differentiates between middle cerebral arteries with and without vasospasm on angiography, but has a very low sensitivity for detecting anterior cerebral artery vasospasm and vasospasm in patients with anterior communicating artery aneurysms. Since vasospasm may involve anterior cerebral arteries while sparing middle cerebral arteries, especially after rupture of an anterior communicating artery aneurysm, caution should be exercised in using negative transcranial Doppler results to make treatment decisions based on the assumed absence of vasospasm.

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Transcranial Doppler (TCD) has been successfully applied to the detection of vasospasm following subarachnoid haemorrhage. Mean velocity in the middle cerebral artery (MCA) measured by TCD correlates well with vessel lumen diameter on angiography[1-4] and is significantly higher in patients who develop delayed cerebral ischaemia compared with those who do not.[5] TCD has good sensitivity and specificity for diagnosing MCA vasospasm.[3 4 6 7]

Substitution of TCD for angiography to diagnose vasospasm of specific arteries could prove invaluable for research to determine the causes of vasospasm and delayed cerebral ischaemia, assess treatment of these conditions, and delineate predictors of outcome. Application of TCD to the clinical setting may assist in making decisions regarding timing of aneurysm surgery and selection and adjustment of therapy to alleviate vasospasm.

The accuracy of TCD in the diagnosis of MCA vasospasm has been encouraging with regard to both research and clinical issues. However, since the development of the TCD, it has been recognised that for the anterior cerebral artery (ACA), the anatomical course, most of it at right angles to the Doppler beam, and vascular interconnections may interfere with both measurement and interpretation ACA velocities by this non-invasive test.[1 8-10] This report analyses the performance of the TCD test compared with angiography in the detection of ACA vasospasm.

Methods

During a 12 month period, all of the authors' patients with subarachnoid haemorrhage whose onset was within 14 days of admission to the Neurological Institute of New York were studied by TCD. The study includes all 41 patients who had a TCD within 24 hours of cerebral angiography and no surgical procedure between the Doppler and angiographic studies. No patient was included in this report more than once.

TCD examinations were performed with a pulsed, range-gated 2 mHz TCD (EME TC2–64B, Eden Medizinische Elektronik GmbH, Uberlingen, Germany) using the standard transtemporal technique to insonate and identify arteries.[11] The maximum mean velocity and pulsatility index for each basal artery studied were recorded. On angiography, vasospasm was diagnosed if focal or diffuse narrowing of the MCA M_1, ACA A_1, or ACA A_2 segments was not explained by atherosclerosis or a congenital anomaly. TCD and angiography were performed and the results interpreted independently by reviewers having no knowledge of the patients' clinical status.

The sensitivity and specificity of TCD compared with cerebral angiography to diagnose vasospasm of a specific artery were determined at mean velocities of >140 cm/s and >120 cm/s for the MCA and ACA. In addition, the sensitivity and specificity of TCD to classify patients as having vasospasm were determined at these two mean velocities.

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Patients who for technical reasons had no arterial flow detected by TCD are not included in the analysis of TCD sensitivity and specificity but are reported in the description of TCD test results. Comparison of means of arterial mean velocities and comparison of means of pulsatility indices were performed using Student's t test with $a = 0.05$.

Results

The study included 41 subjects, 28 women and 13 men with a mean age of 52 years (range 22–85). Thirty four had bilateral and seven unilateral carotid angiograms. All had bilateral TCD studies. Four patients (10%) had no ultrasonic window at either temple; one patient had a left but not a right temporal window. Aneurysm locations are shown in table 1.

Seventy five MCAs in 41 patients were studied angiographically and by TCD. An ultrasonic window was present in 66 and MCA flow was detected by TCD in all 66. On angiography, seven of the 66 arteries were in spasm. Based on a mean velocity of >140 cm/s to diagnose vasospasm, TCD correctly classified six of the seven in spasm and 58 of the 59 not in spasm (sensitivity 86%; specificity 98%). Using a mean velocity of >120 cm/s to diagnose vasospasm, TCD sensitivity was unchanged and specificity dropped to 86% as a result of seven additional false positives. MCAs in spasm on angiogram had significantly higher mean velocities (mean 153 cm/s) than MCAs not in spasm (mean 72 cm/s, $p < 0.01$). Pulsatility indices were significantly lower in the MCAs in spasm (spasm mean 0.57 versus no spasm mean 1.04, $p < 0.01$).

Seventy five ACAs in 41 patients had both angiographic and TCD studies. One of these ACAs was aplastic. Nine arteries were not detected by TCD because there was no ultra-

sonic window. TCD detected no flow in nine additional ACAs despite detection of flow in the ipsilateral MCA. On angiography, 15 of 66 ACAs studied by TCD were in spasm. Based on the >140 cm/s mean velocity criterion for vasospasm, TCD correctly classified only two of the 15 in spasm (sensitivity 13%), 10 had mean velocities <140 cm/s, and three had no flow detected. Using the >140 cm/s vasospasm criterion, TCD correctly classified all 51 ACAs not in spasm on angiogram (specificity 100%). Changing the TCD vasospasm criterion to >120 cm/s did not improve the sensitivity, while two false positives lowered the specificity to 96%. There was no significant difference between the mean velocities of those ACAs in spasm (mean 88 cm/s) versus those not in spasm (mean 66 cm/s, $0.05 < p < 0.10$) or between pulsatility indices of ACAs in spasm (mean 0.83) or not (mean 1.01, $p > 0.10$).

Ten patients had vasospasm of one (four patients) or both (six patients) ACAs on angiogram (table 2). Seven had anterior communicating artery (ACom) aneurysms, three posterior communicating artery aneurysms. TCD correctly diagnosed vasospasm of the ACAs of only one patient. This patient had a posterior communicating artery aneurysm and diffuse, severe vasospasm affecting both middle and anterior cerebral arteries. Three other patients had vasospasm of both anterior and middle cerebral arteries, but TCD detected only the MCA vasospasm. Two of these patients had posterior communicating artery aneurysms, one an ACom aneurysm.

Six patients had vasospasm on angiography of ACAs, but not MCAs. All had ACom aneurysms. Ten of their 12 ACAs were in spasm, but five had spasm of only the post-communicating ACA segment (A_2), not the precommunicating ACA segment (A_1). TCD failed to detect the vasospasm in all six patients and all nine ACAs in spasm that had ultrasonic windows.

Of the 37 patients with ultrasonic windows, 11 had vasospasm on angiography of at least one middle or anterior cerebral artery. At mean velocity >140 cm/s, TCD sensitivity was 45% and specificity 96%. All of the false negatives occurred in patients with ACom aneurysms. Among the 19 ACom aneurysm patients in the study, 17 had ultrasonic win-

Table 1   Aneurysm locations

Anterior communicating artery	19
Pericallosal artery	3
Posterior communicating artery	10
Middle cerebral artery	1
Basilar artery apex	2
Posterior cerebral artery	1
Posterior inferior cerebellar artery	2
No aneurysm	3

Table 2   Comparison of angiographic and transcranial Doppler findings in patients with anterior cerebral artery vasospasm

Aneurysm	Angiography vasospasm						Transcranial Doppler velocity (cm/s)			
	LMCA	RMCA	LA_1	RA_1	LA_2	RA_2	LMCA	RMCA	LACA	RACA
ACOM	Yes	Yes	Yes	Yes			188	86*	116*	42*
ACOM	No	No	No	No	Yes	Yes	122	NW	42*	NW
ACOM	No	No	No	No	Yes	Yes	56	64	64*	ND*
ACOM	No	No	No	Yes			106	72	114	96*
ACOM	No	No	Yes	Yes			66	114	ND*	96*
ACOM	No	No	Yes	No		Yes	34	50	20*	42*
ACOM	No	No	Yes	No			120	122	ND*	86
PCOM		Yes		Yes				162		78*
PCOM	Yes	Yes	Yes	Yes			166	198	140	206
PCOM	Yes	No	Yes	No			160	166	116*	110

ACOM = anterior communicating artery; PCOM = posterior communicating artery; LMCA = left middle cerebral artery; RMCA = right middle cerebral artery; LA_1 = left A_1 segment, anterior cerebral artery; RA_1 = right A_1 segment, anterior cerebral artery; LA_2 = left A_2 segment, anterior cerebral artery; RA_2 = right A_2 segment, anterior cerebral artery; ND = no flow detected; NW = no ultrasonic window.
* False negative transcranial Doppler.

dows and seven of these 17 had vasospasm on angiography. At mean velocity >140 cm/s, TCD had a sensitivity of 14% (one of seven) and specificity of 90% (nine of 10). The one ACom aneurysm patient with a positive TCD had MCA as well as ACA vasospasm; the six ACom patients with negative TCDs but positive angiograms had only ACA vasospasm.

Discussion

In this study, mean velocity measured by TCD had the high sensitivity and specificity for diagnosis of MCA vasospasm previously reported.[3][4][6][7] By contrast, TCD had a low sensitivity and high specificity for the detection of angiographically diagnosed ACA vasospasm. Also in this study, TCD had a low sensitivity for diagnosing vasospasm in patients with ACom aneurysms.

Most reports of TCD after subarachnoid haemorrhage have emphasised results of MCA insonation. Those studies which have also noted results of ACA insonation have found that ACA velocities add no information to MCA measurements,[12] that there is a weak correlation between ACA diameter and ACA mean velocity,[1-3] and that MCA vasospasm versus no vasospasm groups have significantly different maximum mean velocities but ACA vasospasm versus no vasospasm groups do not.[7] Contributing to the low TCD sensitivity for detection of ACA vasospasm may be anatomy, test conditions and technique, and diagnostic criteria.

The course of the ACA A_1 segment makes transtemporal TCD detection of the A_1 more difficult than detection of the MCA.[1][8-10] TCD may fail to provide a measure of velocity in more than 20% of ACAs[1][10][11][13] due to the frequency of no transtemporal ultrasonic window (4/41 patients in this study and 2·7–10% of patients in other reports[1][4][9][10][14]) and the rate of failure to detect ACA flow even when there is a good MCA signal and no ACA vasospasm.[1][9][10] A_1 hypoplasia or aplasia, affecting 5–12% of ACAs,[15-17] may add to difficulty in detecting ACA flow.

The usual ACA A_1 course provides an angle of transtemporal TCD insonation which may result in an underestimate of mean velocity and therefore an underdiagnosis of A_1 vasospasm based on mean velocity criteria. Severe A_1 vasospasm which markedly reduces flow may increase the difficulty in locating and measuring A_1 flow.[18] The ACA A_2 segment is beyond the reach of transtemporal TCD so when vasospasm involves the A_2 but not the A_1 segment, TCD gives a false negative result. ACA vasospasm may be diagnosed in error when high ACA velocities reflect low vascular resistance distal to the point of insonation. For example, left internal carotid artery (ICA) occlusion may lead to increased flow, and therefore high velocities, in the right ACA if this artery becomes an important source of collateral blood flow to the left ICA territory.

TCD operator experience and skill are important for accuracy of vessel identification and velocity measurement. In this study, the accurate classification of MCA vasospasm by TCD makes it unlikely that poor TCD operator skill alone would be a sufficient explanation of the low TCD sensitivity for detecting ACA vasospasm.

The TCD diagnostic criteria for vasospasm tested in this study, >120 cm/s and >140 cm/s were selected based on reports of TCD performance at these velocity limits and the reported correlation between MCA velocity and lumen area. The MCA velocity criteria for vasospasm assessed by other investigators include 110 cm/s (sensitivity 85%, specificity 98%[3]) and 120 cm/s (sensitivity 59%, specificity 100%,[4] and sensitivity 78%[9]) At 120 cm/s, TCD sensitivity to diagnose ACA vasospasm has been reported as 71%.[9] In the MCA, mean velocity >140 cm/s correlates with reduction in lumen diameter to <1·5 mm[13], corresponding to >75% reduction in lumen area.[20] Because this degree of lumen stenosis significantly reduces blood flow, mean velocity 140 cm/s has been recommended as the minimum velocity to diagnose vasospasm.[3] Brain infarction[5] and delayed ischaemic deficits[2] due to vasospasm may be rare at velocities below 140 cm/s, whereas the incidence of ischaemia due to vasospasm rises at velocities above 140 cm/s.[2][7]

In this study, mean velocity >140 cm/s proved a satisfactory TCD measure for diagnosing MCA vasospasm. Mean velocity >140 cm/s was a highly specific but very insensitive classifier of ACA vasospasm. Classification was no more accurate at mean velocity >120 cm/s. Other measures, not examined in this study, such as comparison of ipsilateral internal carotid artery and ACA mean velocities and pulsatility indices, or determination of change in ACA velocities over time, may prove more accurate classifiers of ACA vasospasm.

Low TCD sensitivity to vasospasm in a patient despite high TCD sensitivity to MCA vasospasm has been reported previously and attributed to vasospasm affecting arteries other than the MCA stem.[4] In this study, the patient characteristic which explained TCD insensitivity to vasospasm in a patient was aneurysm location, with all false negatives occurring in patients with ACom aneurysms. Anatomy and technical problems may contribute to this low sensitivity in patients with ACom aneurysms.

Vasospasm following rupture of ACom aneurysms is most likely to involve the ACAs. Although some patients with ruptured AComs will experience vasospasm of other arteries as well, others will have only ACA vasospasm[4] (and six of our seven patients with ACom aneurysms and vasospasm) and some will have vasospasm of the A_2 but not the A_1 segments[15] (and three of our seven patients with ACom aneurysms and vasospasm). As discussed above, TCD has a low sensitivity for both A_1 and A_2 vasospasm.

The condition of the patient affects the ease or difficulty of performing TCD. The

restless, agitated patient may be unable to keep his head still long enough for accurate vessel identification and velocity measurement. The patient with a ruptured ACom aneurysm and consequent ACA vasospasm leading to frontal lobe ischaemia may be just the restless, agitated patient who cannot have a technically satisfactory TCD study.

In summary, the high specificities of TCD for classifying MCA and ACA vasospasm establish that the diagnosis of vasospasm can be made with confidence when mean velocities exceed 140 cm/s. Treatment decisions based on the presence of vasospasm, such as timing of surgery, institution of volume expansion, modification of calcium channel blocker dose, and selection of patients for percutaneous transluminal angioplasty of spastic cerebral arteries may be supported by such positive results and have been endorsed by other investigators.[2 5 7 9 21 22] However, because TCD has such a low sensitivity for detecting ACA vasospasm and vasospasm in patients with ACom aneurysms, caution should be exercised in using negative TCD results (mean velocity <140 cm/s) to make treatment decisions based on the assumed absence of vasospasm.

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