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Background and Purpose—The clinical and hemodynamic impacts of basilar artery (BA) vasospasm (VS) after aneurysmal subarachnoid hemorrhage (SAH) are ill-defined. The purpose of the present study was to evaluate the relationship between BA-VS and regional cerebral blood flow (rCBF) with posterior circulation after aneurysmal SAH.

Methods—Daily transcranial Doppler (TCD) measurements of posterior and anterior circulation arteries were conducted in 162 patients with aneurysmal SAH. rCBF to the brain stem (BS) and other brain territories was assessed by multiple single-photon emission computed tomography with ^{99m}Tc ethyl cysteinate dimer single-photon emission computed tomography (ECD-SPECT) imaging during the course of VS.

Results—SPECT imaging showed delayed BS hypoperfusion in 29 patients (17.9%). Of them, 23 patients (79.3%) were found to have BA-VS. Patients with very high BA flow velocities (FVs; >115 cm/s) had a 50% chance of developing delayed BS ischemia. BA-VS was found at a higher rate in patients who experienced reduced rCBF in the cerebellum (56.3%), thalamic nuclei (68.4%), and occipital lobe (81.8%). Although patients with delayed BS hypoperfusion did not present with a higher clinical grade, their clinical outcome was significantly worse (Glasgow Outcome Score after 30 days 2.48±1.16 versus 3.3±1.27; $P=0.001$).

Conclusions—These findings suggest for the first time that BA-VS after aneurysmal SAH is associated with hypoperfusion to BS and other posterior circulation territories. The risk for delayed BS ischemia increased significantly when TCD BA-FVs were >115 cm/s. **(*Stroke*. 2004;35:1867-1872.)**

Key Words: basilar artery ■ vasospasm ■ cerebral blood flow ■ tomography, emission computed ■ ultrasonography, Doppler, transcranial ■ subarachnoid hemorrhage ■ brain stem ■ ischemia

Transcranial Doppler (TCD) has become a reliable and sensitive method for diagnosis of vasospasm (VS) after subarachnoid hemorrhage (SAH), and many studies have shown a correlation between TCD measurements and angiography findings.[1–3] Although TCD measurements do not provide information on cerebral blood flow (CBF) and cerebral tissue perfusion,[1,4,5] significant arterial narrowing resulting from VS is associated with perfusion impairment, and many studies have demonstrated that cerebral VS in the anterior circulation is associated with reduced cerebral perfusion in the affected territories.[5,6]

Unlike anterior circulation VS, little is known about VS in the vertebrobasilar system,[7–10] and with an absence of data regarding CBF disturbances in the posterior circulation brain territories after aneurysmal SAH, many clinicians may not be well guided in the monitoring and treatment of vertebrobasilar VS.[10,11]

In the present study, we used ^{99m}Tc ethyl cysteinate dimer single-photon emission computed tomography (ECD-SPECT) imaging to determine the incidence of delayed brain stem (BS) ischemia and posterior circulation territory ischemia that may be related to VS after aneurysmal SAH.

Materials and Methods

Patients

Records of 354 consecutive patients with aneurysmal SAH admitted between January 2001 and September 2002 were reviewed. Study inclusion criteria for the 162 patients comprised daily anterior and posterior circulation TCD measurements, baseline SPECT imaging done within 72 hours of the initial bleed, and at least 1 other study later in the acute clinical phase. Patients who died within 7 days of bleeding were excluded from the study. The severity of neurological impairment at admission was assessed by the Hunt and Hess grading system (H&H grade).[12] Delayed ischemic neurological deficit (DIND) was defined as a worsening of the neurological condition that could not be attributed to rebleeding or systemic or postoperative complications. For analysis purposes, DINDs were divided into focal neurological deficits or reduced level of consciousness (RLC). Final neurological outcome was assessed by the Glasgow Outcome Score

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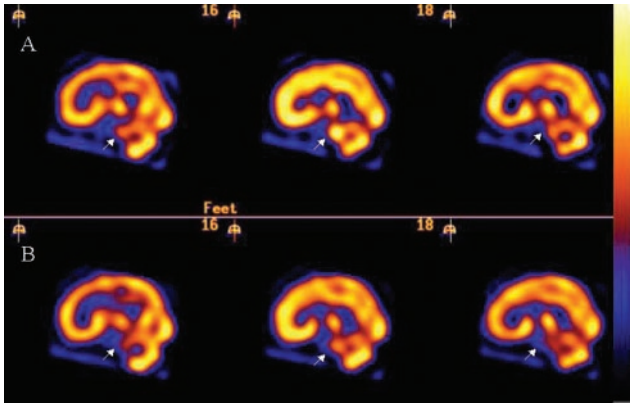

Figure 1. A 6-mm-thick sagittal plane reconstructed ^{99m}Tc SPECT imaging of a 44-year-old female with SAH after rupture of an anterior communicating artery aneurysm. White arrows point to the BS. A, Baseline BS perfusion is normal, as can be seen by the intense and bright uptake signal in the pons area (arrows), which is equal in intensity compared with the rest of the brain territories. B, On day 7 after the initial hemorrhage, the patient presented with RLC and elevated BA TCD-MFVs (110 cm/s). SPECT imaging disclosed moderate reduction in BS rCBF, (shown as decreased uptake intensity signal, arrows) compared with baseline imaging (A) and other brain territories.

(GOS) score[13] for all patients after 1 month. The bleeding intensity was scored according to the Fisher classification.[14]

The University of Washington Human Subjects Committee approved the study.

TCD Recording

Initial TCD evaluation was performed on all patients within the first 48 hours after SAH onset. Mean flow velocities (FVs) of >120 cm/s and FVs of 3-fold greater than that of the FVs in the extracranial internal carotid artery were selected as criteria for VS in the middle cerebral artery and the anterior cerebral artery (ACA), according to severity criteria suggested by Aaslid et al[1] and Lindegaard et al.[2] The basilar artery (BA) FVs were measured through the foramen magnum according to the technique described by Fujioka and Douville.[15] BA-VS defined whenever the FV was >85 cm/s according to criteria suggested by Sloan et al.[7]

SPECT Studies

Each patient was injected with ≈1110 MBq (30 mCi) ^{99m}Tc ECD (Bristol-Myers Squibb Medical Imaging), and images were obtained ≈35 minutes later. All images were acquired using a Prism 3000 triple-headed tomographic scanner (Philips Medical Systems) and low-energy, high-resolution collimators. A 20% window was centered on the 140 keV photopeak of ^{99m}Tc. SPECT images were acquired in a step-and-shoot manner with 64 steps, each lasting 25 seconds, acquired over 360° using clockwise rotation. Images were processed with a Wiener prefilter and RAMP filter for resolution recovery. Software attenuation correction with a coefficient of 0.11 cm^{-1} was used in all patients with intact cranial bones. Hypoperfusion was defined as mild, moderate, or severely decreased uptake compared with cerebellar and global cerebral hemispheric uptake. (Brain perfusion SPECT has been used since the early 1990s to show BS activity with high spatial resolution.[16,17] Normal regional cerebral perfusion data are available in adults and show that on average, uptake in BS is 88% of whole-brain cortical average blood flow, in thalamus 112%, and in cerebellum 100% of whole-brain average).[18] Operationally, values ranging from 80% to 70% in BS, described as mild hypoperfusion, have been reported in ^{99m}Tc hexamethylpropylenamine oxime SPECT in chronic fatigue syndrome and are visually apparent as a mild reduction in uptake.[19] Values that span from 60% to 70% reflect moderate hypoperfusion (Figure 1) and those <60% reflect severe hypoperfusion (Figure 2).

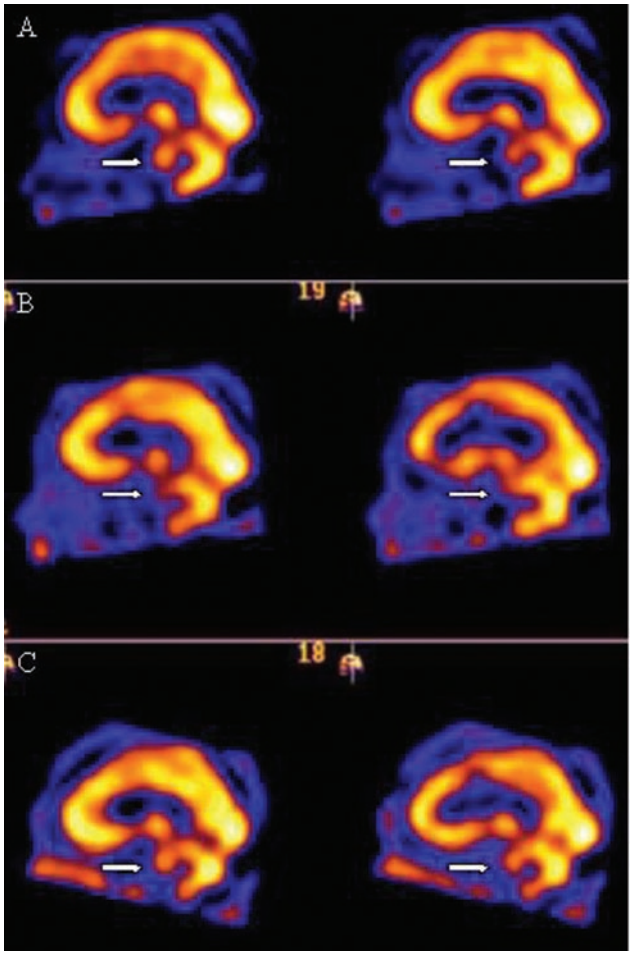

Figure 2. A 6-mm-thick sagittal plane reconstructed ^{99m}Tc SPECT imaging of a 54-year-old female with SAH after rupture of anterior communicating artery aneurysm. White arrows point to the BS (pons area). A, Baseline BS perfusion is normal, as can be seen by intense and bright uptake signal in the pons area (arrow), which is equal in intensity compared with the rest of the brain territories. B, On day 6 after the initial hemorrhage, the patient presented with RLC and elevated BA TCD-MFVs (145 cm/s). SPECT imaging disclosed severe reduction in the BS rCBF (arrows; note the disappearance of the pons uptake signal compared with baseline imaging [A]). C, The patient had angiography, which revealed severe (60%) diffuse narrowing of the BA and subsequently had balloon angioplasty. SPECT imaging obtained after angioplasty revealed significant improvement in BS rCBF perfusion (arrows; note reappearance of the pons uptake signal compared with baseline imaging [A]).

Statistical Analysis

For all data presented as mean±SD, the various subgroups were compared using parametric ANOVA and Student *t* test. For categorical variables, χ^2 and the Fisher exact test were used. The Spearman correlation was used for correlation analysis between TCD-BA mean FVs (MFVs) and BS perfusion impairments. Differences were considered significant when they reached $P<0.05$.

Results

Patients

A total of 162 patients (103 females, 59 males) with aneurysmal SAH were included in the study. Patients in the current study matched to 192 patients with aneurysmal SAH who were excluded from study in their age (50.9±10.3 versus

52.4±9.7; $P>0.05$), H&H grade (2.63±0.91 versus 2.57±0.95; $P>0.05$), Fisher score (2.895±0.91 versus 2.69±0.89; $P>0.05$), and outcome (GOS after 30 days 3.11±1.27 versus 3.23±1.31; $P>0.05$). However, patients included in the present study had a higher rate of ACA aneurysm (32.7% versus 24%; $P=0.015$), posterior circulation aneurysm (12.3% versus 7.8%; $P=0.0037$), and presented more with DIND (41.9% versus 27.6%; $P=0.0071$).

TCD Findings

BA-VS was found in 61 (38%) of 162 patients, (BA-MFVs >85 cm/s). Of them, 7 (11.5%) patients had VS only in the BA, whereas in 54 (88.5%) patients, the VS was in the ACA and the BA. VS limited to anterior circulation was found in 63 (38.9%) patients, whereas in 38 (23.5%) patients, no VS was found according to TCD measurements (Table 1).

Patients with BA-VS had higher Fisher scores compared with patients with only anterior circulation VS or patients without VS (3.2±0.81; 2.81±0.93; 2.55±0.89; respectively; $P<0.05$), presented more with DIND (75.4% versus 30.2%; 7.9%; $P<0.0001$), and had worse outcome (GOS after 30 days 2.64±1.2; 3.17±1.24; 3.47±1.31; $P<0.05$; Table 1).

SPECT Imaging Findings

Delayed BS ischemia was found in 29 (17.9%) of 162 patients. In 9 of them, ischemia was severe, in 10 moderate, and 8 experienced mild hypoperfusion. Patients with delayed BS ischemia had higher Fisher scores than patients without BS ischemia (3.24±0.54 versus 2.8±0.96; $P=0.0006$), presented more with DIND (72.4% versus 35.3%; $P<0.0002$), and had worse outcome (GOS after 30 days 2.48±1.16 versus 3.3±1.27; $P=0.001$; Table 2). DIND was found in 7 of 7 of patients with severe BS ischemia (all of them had an RLC), 8 of 12 patients with moderate ischemia (of whom 7 had an RLC), and 6 of 12 patients with mild ischemia (of whom 5 had an RLC; Table 2).

Delayed cerebellum ischemia was found in 32 (20%) of 162 patients. A total of 38 patients (23.5%) experienced thalamic nuclei ischemia, and 11 (6.8%) experienced posterior cerebral arteries (PCAs) ischemia.

TCD and SPECT Correlation

Most of the patients who experienced delayed BS hypoperfusion were found to have TCD measurements consistent with BA-VS (79.3%). TCD-MFVs in the BA artery were >115 cm/s in 33 patients. In 19 of these patients, SPECT imaging showed delayed reduced regional CBF (rCBF) in the BS (57.6%). However, only 4 of 28 patients (14.3%) with BA-MFVs between 85 cm/s and 115 cm/s, and 6 of 101 patients (7%) with BA-MFVs <85 cm/s had SPECT imaging that showed delayed BS ischemia (Figure 3). Severe BS hypoperfusion (Figure 2) was found in 7 patients, all of whom had BA-MFVs >115 cm/s. Moderate BS hypoperfusion (Figure 1) was found in 12 patients, 9 had BA-MFVs >115 cm/s. The other 3 patients with moderate hypoperfusion had 87, 81, and 41 cm/s BA-MFVs. Mild hypoperfusion was found in 10 patients, 4 had BA-MFVs <85 cm/s, 3 had BA-FVS between 85 and 115 cm/s, and 3 had BA-FVs >115 cm/s (Figures 3 and 4).

Discussion

The study findings suggest for the first time that patients with aneurysmal SAH may develop delayed BS ischemia, which is associated with BA-VS. To evaluate perfusion in the posterior circulation territories, we used ^{99m}Tc perfusion SPECT imaging of the brain, which has been well established in the assessment of regional cerebral perfusion in cerebral VS.[6,20,21] High spatial resolution brain perfusion SPECT has been used since the early 1990s to show BS activity,[16,17] and brain perfusion SPECT with ^{99m}Tc ECD has been reported in association with interventional treatment of BS ischemia resulting from BA stenosis.[22] In the present study, delayed BS ischemia as revealed by ^{99m}Tc ECD-SPECT was found to be associated with higher bleeding intensity, DIND, higher BA-FVs, and worse outcomes. The incidence of delayed BS ischemia in the study population is high (17.9%) and overestimates the true incidence in the general aneurysmal SAH population. The bias is caused by inclusion of patients who had at least 1 more SPECT imaging study beside the baseline. These are usually patients for whom the clinical course indicates imaging, whether because of delayed clinical deterioration, and patients for whom clinical evaluation was unreliable and TCD measurements showed VS. Nevertheless, because patients with aneurysmal SAH who died within the first 7 days after the initial hemorrhage were not included, the overall outcome of included and excluded patients was the same.

In the present study, 34.4% of the patients that meet criteria for BA-VS experienced from delayed BS hypoperfusion. Furthermore, 75.8% of the patients who experienced delayed BS hypoperfusion were also found to have elevated MFVs in the BA (>85 cm/s). All except 1 patient with BS ischemia had BA-MFVs >60 cm/s. According to Sloan at el,[7] BA-MFVs >95 cm/s are associated with 100% specificity and 100% positive predictive value for BA-VS; however, the sensitivity was found to be 39%. Using 60 cm/s as criteria for BA-VS, the sensitivity increased up to 70%. We used FVs of >85 cm/s as criteria for BA-VS, which are associated with >90% specificity and 50% sensitivity for narrowing. Clearly, not all arterial narrowing is associated with perfusion abnormality, and some patients with elevated BA-FVs could actually experience hyperemia.[2,8,9]

Unlike the cortex, blood flow to the BS is mainly through perforating arteries emerging at a 90° angle from the BA. BA-VS might result in reduced perfusion to the perforating arteries feeding the BS. Using a phantom model designed to simulate the anatomy of the perforating arteries, Soustiel et al[23] showed that with significant narrowing of the parent vessel, perforating vessel flow is significantly impaired. As the narrowing in the parent vessel worsens or extends in length, perforating vessel flow is not only reduced, but flow separation appeared in the parent vessel, which, in turn, produced a Venturi-like effect responsible for pressure collapse at the aperture of the perforating vessels. Soustiel et al[23] suggested that this phenomenon could exist in the vertebrobasilar system and can result in reduced flow to the perforating arteries. This is consistent with the finding that patients with very elevated BA-MFVs (>115 cm/s) are at higher risk (57.6%)

TABLE 1. BA-VS as Revalued by TCD Measurements (MFVs >85 cm/s) Compared With Clinical Presentation, Clot Thickness, Aneurysmal Location, SPECT Imaging Findings, DIND, and Outcome in 162 Patients With Aneurysmal SAH

	No Vasospasm	Only Anterior Circulation VS	Anterior Circulation and BA Vasospasm	Only BA Vasospasm	*P*
No. of patients=162	38	63	54	7	
Males/females: 59/103	12/26	25/37	20/34	2/5	
H&H classification	2.5±0.80	2.56±0.91	2.82±0.96		*NS
					**NS
Fisher score	2.55±0.89	2.81±0.93	3.2±0.81		*0.0142
					**0.0003
Aneurysmal location					
Anterior circulation	36 (94.7%)	57 (90.5%)	44 (81.5%)	5 (71.4%)	*NS
					**NS
ICA	20 (52.6%)	20 (31.7%)	9 (16.7%)	1 (17.3%)	NS
					**0.0005
MCA	9 (23.7%)	19 (30.2%)	11 (%)	0	*NS
					**NS
ACA	7 (18.4%)	18 (28.6%)	24 (44.4%)*	4 (57.1%)	NS
					**0.0133
Posterior circulation	2 (5.3%)	6 (9.5%)	10 (18.5%)	2 (28.6%)	*NS
					**NS
BA	3 (7.9%)	3 (4.8%)	8 (14.8%)	2 (28.6%)	*NS
					**NS
Other			2 (18.5%)	2 (28.6%)	
Delayed reduced rCBF					
BS	2 (5.3%)	4 (6.3%)	21 (38.9%)	2 (28.6%)	*<0.0001
					**0.0002
Cerebellum	4 (10.5%)	10 (15.9%)	17 (31.5%)	1 (18.4%)	*NS
					**0.0232
Thalami nuclei	3 (7.9%)	9 (14.3%)	23 (42.6%)	3 (42.9%)	*0.0008
					**0.0003
PCA territories	0	2 (3.2%)	9 (16.7%)	0	*0.0225
MCA and ACA territories	4 (10.5%)	29 (46%)	31 (57.4%)	2 (28.6%)	*NS
					**<0.0001
DIND	3 (7.9%)	19 (30.2%)	43 (79.6%)	3 (42.9%)	*<0.0001
					**<0.0001
Focal	2 (5.3%)	16 (25.4%)	27 (50%)	0	*0.0073
					**<0.0001
Reduced consciousness level	2 (5.3%)	8 (12.7%)	35 (64.8%)	3 (42.9%)	*<0.0001
					**<0.0001
1 month GOS	3.47±1.31	3.17±1.24	2.64±1.2		*0.0171
					**0.0017

*Significance comparing patients with BA and anterior circulation vasospasm patients with vasospasm limited to the anterior circulation.

**Significance comparing patients with BA and anterior circulation vasospasm patients with no vasospasm.

For H&H grade, Fisher score, and outcome, the group of patients with vasospasm limited to the BA were included with the group of patients with BA and anterior circulation VS.

MCA indicates middle cerebral artery; ICA, internal carotid artery.

for developing BS hypoperfusion. Furthermore, significant BS ischemia (moderate or severe hypoperfusion) was associated mainly with higher BA-MFVs, which may suggest that those patients experienced more significant narrowing of the BA in a way that flow through the perforating arteries was impaired.

Patients with high BA-MFVs had a higher rate of delayed reduced perfusion to the cerebellum as well as to the PCA

TABLE 2. Delayed BS Hypoperfusion as Disclosed by ^{99m}Tc SPECT Imaging Compared With Clinical Presentation, Fisher Score, Aneurysmal Location, DIND, and Outcome in 162 Patients With Aneurysmal SAH

	Delayed BS Ischemia	Normal BS Perfusion	*P*
No. of patients	29	133	
H&H classification	2.70±0.91	2.49±0.92	NS
Fisher score	3.24±0.54	2.8±0.96	0.0006
Aneurysmal location			
Anterior circulation	21 (72%)	120 (90%)	NS
Internal carotid artery	5 (17%)	44 (33%)	
Middle cerebral artery	4 (14%)	35 (26.%)	NS
Anterior cerebral artery	12 (41%)	41 (31%)	NS
Posterior circulation	7 (24%)	13 (10%)	NS
Basilar artery	6 (20%)	10 (7.5%)	NS
Other	2 (3.5%)	3 (2%)	NS
DIND	21 (72.4%)	47 (33.3%)	0.0002
1 month GOS	2.48±1.16	3.3±1.27	0.001

territories and the thalamus. Although blood supply to the thalamus and PCA territories comes also from the anterior circulation, and most of the patients with BA-VS also had anterior circulation VS, BA-VS may lead to further reduction of CBF by decreasing collateral flow to the thalamic area or by decreasing direct flow to the PCAs. Furthermore, patients with elevated BA-MFVs had a higher rate of anterior circulation ischemia. Elevated BA-FVs may also reflect hyperemia or collateral effects related to severe anterior circulation VS and may indicate more significant VS process in the anterior circulation. One explanation for BA involvement in the VS process is related to the fact that most of the aneurysms were located in the anterior circulation and that patients who eventually developed BA-VS had higher Fisher bleeding scores. Although Fisher score is based on clot thickness measurements, many of the patients with higher scores have more intense and diffuse SAH. Higher bleeding intensity could lead to disruption of the posterior arachnoid membranes and deposition of the clot around the posterior circulation arteries.[9] The increased intensity of bleeding may be responsible for more significant anterior circulation VS as well as the coexistence of BA-VS, which is reflected in the present study by a higher proportion in patients who had BA-VS and presented with DIND.

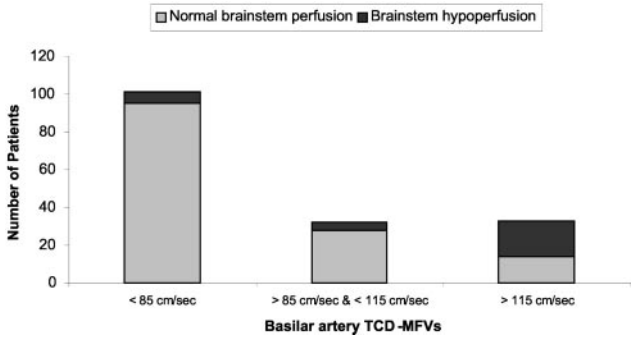

Figure 3. TCD BA-MFVs values and BS perfusion impairments as assessed by ^{99m}Tc SPECT imaging in 162 patients with aneurysmal SAH. (P=0.006 for differences between patients with BA-MFVs >115 cm/s and BA-MFVs >85 cm/s and <115 cm/s; P<0.0001 for differences between patients with BA-MFVs >115 cm/s and BA-MFVs <85 cm/s).

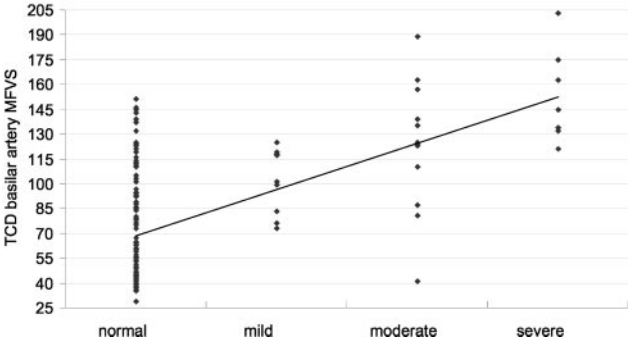

Figure 4. Correlation between TCD BA-MFVs and degree of BS hypoperfusion as disclosed by ^{99m}Tc SPECT imaging in 162 patients with aneurysmal SAH. Normal indicates normal BS perfusion; mild, mild reduction in perfusion; moderate, moderate reduction in perfusion; severe, severe reduction in perfusion (P<0.0001; r=0.448).

BA-VS influence on outcome was reported by Lee et al,[8] and Soustiel et al[9] suggested that elevated vertebrobasilar system FV is associated with poorer neurological outcomes, especially after severe head injury. Because most of the patients with BA-VS also had anterior circulation VS as well as higher rates of anterior circulation perfusion impairments, we cannot suggest that BA-VS is an independent factor that influences the patient outcome after SAH. However, the present findings suggest that elevated TCD BA-FVs are associated with more clinically significant VS and more hemodynamically significant impairments.

Conclusion

BA-VS after aneurysmal SAH is associated with more clinically and hemodynamically significant VS, and patients with BA-VS have a higher tendency to develop BS ischemia. Routine TCD BA-FVs measurements may identify patients who are at higher risk for symptomatic VS, and BA-MFVs of >115 cm/s may be used as a diagnostic threshold to identify patients who are at high risk for BS ischemia.

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