DYNAMIC PERFUSION COMPUTED TOMOGRAPHY IN THE DIAGNOSIS OF CEREBRAL VASOSPASM

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OBJECTIVE: The aim of the study was to correlate absolute cerebral blood flow (CBF) and mean transient time (MTT) measured by dynamic perfusion computed tomographic (PCT) scanning with the clinical course, vasospasm severity, and perfusion abnormality in patients with cerebral vasospasm after aneurysmal subarachnoid hemorrhage.

METHODS: Forty-six patients with vasospasm after aneurysmal subarachnoid hemorrhage had 63 PCT images obtained during the course of vasospasm. All patients had transcranial Doppler measurements, 28 had an angiography study, and 38 had ^{99m}Tc single-photon emission computed tomographic imaging performed in conjunction with the PCT scan.

RESULTS: The average minimal regional CBF (rCBF) and maximal regional MTT in patients with delayed ischemic deficit were significantly different in comparison with patients without delayed ischemic deficit (22.6 $\pm$ 11.2 cm^3/100 g/min versus 45.2 $\pm$ 21.3 cm^3/100 g/min, $P < 0.001$; 7.3 $\pm$ 2.5 s versus 3.3 $\pm$ 1.7 s, $P < 0.05$). The average minimal rCBF and maximal regional MTT in middle cerebral vascular territories in which severe middle cerebral artery vasospasm was measured by transcranial Doppler were significantly different in comparison with middle cerebral vascular territories in which no vasospasm was measured by transcranial Doppler (29.3 $\pm$ 1.7 cm^3/100 g/min versus 54.1 $\pm$ 25.4 cm^3/100 g/min, $P < 0.01$; 4.5 $\pm$ 2.4 s versus 2.8 $\pm$ 1.1 $P < 0.001$). The average minimal rCBF and maximal rMTT in vascular territories with estimated severe hypoperfusion on single-photon emission computed tomographic imaging were significantly different in comparison with values in vascular territories with unimpaired perfusion as estimated by single-photon emission computed tomographic imaging (18.9 $\pm$ 6.9 cm^3/100 g/min versus 54.2 $\pm$ 23.4 cm^3/100 g/min, $P < 0.001$, 0.001; 8.1 $\pm$ 1.9 s versus 2.5 $\pm$ 0.39 s, $P < 0.001$).

CONCLUSION: The present study suggests that, in general, quantitative measurements of rCBF and regional MTT by PCT show high concordance rates with the clinical course, vasospasm severity, and hemodynamic impairments in patients with cerebral vasospasm aneurysmal subarachnoid hemorrhage.

KEY WORDS: Angiography, Computed tomography, Perfusion, Perfusion computed tomography, Single-photon emission computed tomography, Subarachnoid hemorrhage, Transcranial doppler, Vasospasm

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Several perfusion methods are available for the assessment of cerebral blood flow (CBF) after aneurysmal subarachnoid hemorrhage (aSAH), all of which seem to be sensitive for the detection of vasospasm-related perfusion abnormality (5, 6, 9, 11, 13, 15, 17, 19). However, none of these methods are ideal because of their expense (positron emission tomography [PET] and magnetic resonance imaging [MRI]), lack of availability in all or most institutions on an emergency basis (PET, MRI, single-photon emission computed tomography [SPECT]), their time intensiveness (PET, MRI, SPECT), their low resolution (xenon computed tomography), their provision of relative CBF measurements (MRI, SPECT), and, lastly, their general unsuitability for unstable patients (SPECT, PET, MRI).

Dynamic perfusion computed tomographic (PCT) scanning is now widely accepted for the

diagnosis of acute stroke (7, 8, 17, 20, 21). PCT can be performed using current computed tomographic (CT) technology and readily available computer software (8, 20, 21). PCT methods have many limitations, however, related to the deconvolution algorithm (2–4) used for calculation and assuming normal cerebral hemodynamic physiology and intact blood-brain barrier, which are not necessarily intact after aSAH. Furthermore, perfusion values are highly dependent on the arterial input function chosen for the measurements. Nevertheless, PCT has emerged recently as a convenient imaging tool of investigation for cerebral hemodynamics, and, in recent comparative studies, Bisdas et al. (5) found satisfactory correlation between the PCT and MRI assessments of ischemic brain injury, and Sase et al. (16) reported close correlation between CBF values obtained with PCT and stable xenon CT.

In SAH, only a few small cohort studies have shown some correlation between quantitative absolute PCT-CBF values and the clinical course (8,14), and the role of PCT in the diagnosis of vasospasm has not yet been comprehensively evaluated. The aim of the present study was to evaluate correlation between quantitative CBF and mean transit time (MTT) measurements by PCT with the clinical course, vasospasm severity, and perfusion abnormality in patients with vasospasm after aSAH.

TABLE 1. Clinical and demographic data

Characteristic	No. of patients
No. of patients	46
Age	50.2 ± 10.4
Sex (M/F)	19/27
Hunt and Hess grade	
I–III	31
IV–V	15
Fisher's score	
I–II	21
III	12
IV	12
Aneurysmal location	
Anterior circulation	39
Posterior circulation	7
Hydrocephalus	16
Treatment modality	
Surgery	32
Endovascular	14
Glasgow outcome score in 3 months	
Favorable (4–5)	29
Unfavorable (1–3)	17

MATERIALS AND METHODS

Patients

The study was approved by the local ethical committee. The study population included 46 patients (19 men and 27 women; average age, 50.2 ± 10.4 yr; range, 29–75 yr) with cerebral vasospasm after aSAH, all treated at Harborview Medical Center between March 2003 and June 2004. Clinical and demographic data are presented in *Table 1*. Patients included in the study represent a consecutive series of patients in whom aneurysms were secured by craniotomy or coiling within 72 hours, and PCT was performed as clinically indicated in cases of ischemic deterioration or transcranial Doppler (TCD) measurements, suggesting severe middle cerebral artery (MCA) vasospasm for 2 sequential days. Patients with intracerebral hematoma, early brain infarction (appearing on CT scans within 72 h from the initial bleeding or within 24 h after securing the aneurysm), adverse reaction to contrast material, impaired renal function, and patients with a contraindication for hypertension therapy were excluded from the study. All patients received hypervolemia, hemodilution, and hypertension (triple H) therapy guided by the use of central venous catheters and arterial catheters and had mean arterial pressures of greater than 90 mmHg during the test. Clinical data collection was performed prospectively, and detailed imaging analysis was performed by two independent observers who were blinded to the patient's clinical and other imaging data.

PCT studies were obtained between Days 2 and 17 after the initial hemorrhage, and of 71 studies performed, eight were discarded because of quality issues. A total of 51 PCT studies were available for comparison with ^{99m}Tc SPECT imaging (38 patients, some patients had more than one PCT scan and SPECT imaging available for comparison). For 28 patients (31 PCT scans), angiographic imaging was performed on the same day as the PCT scan. All patients included had daily TCD measurements of blood flow velocity of the MCA. Delayed ischemic deterioration (DID) was defined as a worsening of the neurological condition for more than 30 minutes (a drop of two points in Glasgow Coma Score in patients with altered consciousness and a new hemiplegia or hemiparesis or a drop of two points in the muscle scale [on a scale of 0–5] in a previously hemiparetic patient) that could not be attributed to rebleeding, postoperative complications, hydrocephalus, or systemic complications.

Dynamic PCT Scan

PCT imaging was performed using a multislice helical CT scanner (LightSpeed, GE Medical Systems, Milwaukee WI). Scanning consisted of an initial noncontrast head CT scan followed by two PCT acquisitions. The perfusion scans were obtained at the centrum semiovale. Four contiguous 5-mm thick sections were scanned over 55 seconds using cine techniques. Visipaque 300 mg I/ml (Amersham Health, Princeton NJ) was injected at 4 cm^3/s for a total dose of 50 cm^3 during scan acquisition. After the completion of scanning, data were transferred to a workstation and analyzed using commercial PCT software (CT PERFUSION II; GE Medical Systems, Waukesha, WI). For most patients, the larger of the two anterior cerebral arteries was chosen for placement of regions of interest that provided the arterial input function, and the

superior sagittal sinus was chosen as the venous outflow function.

Parametric maps of CBF and MTT were calculated by a deconvolution algorithm and were evaluated using regions of interest in the cortical area of the specific brain area (anterior frontal, parasagittal, posterior frontal and parietal, representing the anterior cerebral artery and MCA territories, respectively) (22).

For calculation and comparison with other imaging and the clinical course, the minimal regional CBF (rCBF) and the maximal regional MTT (rMTT) measured in each vascular territory were used (23).

TCD Measurements

Initial TCD evaluation was performed within the first 48 hours after onset of SAH in all patients. The intracranial MCAs and mean flow velocities were measured through the temporal windows, and vasospasm was diagnosed according to criteria suggested by Aaslid et al. (1) and Lindegaard et al. (12) (severe MCA vasospasm as mean flow velocity $>$ 200 cm/s and hemispheric index $>$ 6).

Angiography Imaging

All angiographies were performed on a biplane system (Integris 5000; Philips Medical Systems, Andover, MA) using selective catheterization of either the right or left internal carotid artery. Measurements of the MCA, anterior cerebral artery, and posterior cerebral artery diameters were performed with image magnification and digital calipers on an electronic image viewing system (Centricity; GE Medical Systems). Diagnostic angiography performed within 48 hours after the initial bleeding was used as a baseline. Narrowing was graded for mild (0–24% narrowing), moderate (25–49% narrowing), and severe (50% narrowing and greater).

SPECT Imaging

The ^{99m}Tc ethyl cysteinate dimer (ECD) SPECT imaging technique used, data acquisition methods, and interpretation have been reported recently (18). For analysis and descriptive purposes, hypoperfusion was defined as mild (70–85% of baseline SPECT uptake), moderate (50–70% of baseline uptake), and severe (20–50% of baseline uptake). This grading is consistent with our current clinical practice (15, 18). All patients had baseline 99mTc ECD-SPECT imaging performed within 72 hours of the initial hemorrhage.

Statistical Analysis

For all data presented as mean $\pm$ standard deviation, the various subgroups were compared by analysis of variance, Student's *t* test, and categorical variables were compared by Fisher's exact test. Differences were considered significant when they reached a *P* value of less than 0.05.

RESULTS

rCBF and rMTT Correlation with Delayed Ischemic Deficits

The average minimal rCBF and maximal rMTT were 22.6 $\pm$ 11.2 cm^3/100 g/min and 7.3 $\pm$ 2.5 seconds in 24 patients who experienced DID during the time of PCT scan compared with 44.2 $\pm$ 21.3 cm^3/100 g/min and 3.3 $\pm$ 1.7 seconds for 22 patients who did not experience DID ($P < 0.0001$ and $P < 0.05$ for CBF and MTT, respectively) (*Table 2*). Nineteen out of 26 (73%) patients in whom the minimal rCBF was less than 25 cm^3/100 g/min experienced DID, whereas only 5 out of 20 (25%) patients in whom the minimal rCBF was greater than 25 cm^3/100 g/min experienced DID ($P < 0.001$) (*Table 3*). DID was found in 17 out of 21 (27%) patients in whom the maximal rMTT was greater than 6.5 seconds, whereas 7 out of 25 (28%) patients in whom the maximal rMTT was less than 6.5 seconds experienced DID ($P < 0.001$) (*Table 4*).

rCBF and rMTT Correlation with Vasospasm Severity

Correlation with TCD measurements was evaluated for the MCAs. A total of 198 sides were evaluated before or after

TABLE 2. Minimal perfusion computed tomographic regional cerebral blood flow and maximal regional mean transient time in relationship with delayed ischemic deficit, transcranial Doppler-middle cerebral artery vasospasm severity, degree of middle cerebral artery and anterior cerebral artery narrowing on angiography and estimated perfusion by ^{99m}Tc ethyl cysteinate dimer-single-photon emission computed tomographic imaging[a]

	Minimal rCBF (cm^3/100 g/min)	*P* value	Maximal rMTT (sec)	*P* value
Delayed ischemic deficit	45.2 $\pm$ 21.3		3.3 $\pm$ 1.7	
No	22.6 $\pm$ 11.2	$<$ 0.001	7.3 $\pm$ 2.5	$<$ 0.05
Yes				
MCA-TCD measurements				
No vasospasm	54.1 $\pm$ 25.4		2.8 $\pm$ 1.1	
Severe vasospasm	29.3 $\pm$ 17	$<$ 0.01	4.5 $\pm$ 2.4	$<$ 0.001
MCAs + ACAs on angiography	48.5 $\pm$ 27.6		3.2 $\pm$ 1.7	
$<$50% narrowing	26.4 $\pm$ 10.3	$<$ 0.001	5.8 $\pm$ 2.7	$<$ 0.001
$>$50% narrowing				
^{99m}TC ECD-SPECT				
Unimpaired perfusion	54.2 $\pm$ 23.4		2.5 $\pm$ 0.39	
Severe hypoperfusion	18.9 $\pm$ 6.9	$<$ 0.001	8.1 $\pm$ 1.9	$<$ 0.001

[a] rCBF, regional cerebral blood flow; rMTT, regional mean transient time; MCA, middle cerebral artery; TCD, transcranial Doppler; ACA, anterior cerebral artery; ^{99m}TC ECD, ^{99m}Tc ethyl cysteinate dimer; SPECT, single-photon emission computed tomographic imaging.

TABLE 3. Relationship between delayed ischemic deficit, vasospasm severity, hypoperfusion estimated by ^{99m}Tc ethyl cysteinate dimer-single-photon emission computed tomographic imaging and perfusion computed tomographic regional cerebral blood flow values[a]

		PCT rCBF		*P* value	Sensitivity (%)	Specificity (%)	+PV (%)	−PV (%)
		<25 cm^3/100/min	>25 cm^3/100/min					
Delayed ischemic deficit	No	7	25					
(per PCT scan)	Yes	24	7	< 0.001	73	76	73	78
Hypoperfusion on ^{99m}TC ECD-SPECT	No or mild	17	119					
(per vascular territory)	Severe	25	1	< 0.001	96	87.5	59.5	99
Angiography findings:	<50% narrowing	11	66					
MCAs + ACAs	>50% narrowing	29	16	< 0.0001	64.5	86	72.5	80.5

[a] PCT, perfusion computed tomography; rCBF regional cerebral blood flow; +PV, positive predictive values; −PV, negative predictive value; ^{99m}TC ECD, ^{99m}Tc ethyl cysteinate dimer.

TABLE 4. Relationship between delayed ischemic deficit, vasospasm severity, hypoperfusion estimated by ^{99m}Tc ethyl cysteinate dimer-single-photon emission computed tomographic imaging and perfusion computed tomographic regional mean transient time values[a]

	PCT rMTT		*P* value	Sensitivity (%)	Specificity (%)	+PV (%)	−PV (%)
	>6.5 s	<6.5 s					
Delayed ischemic deficit (per PCT scan)	6	26					
No	22	9	< 0.001	71	81	78.5	74
Yes							
Hypoperfusion on ^{99m}TC ECD-SPECT (per vascular territory)	21	115					
No or mild	21	6	< 0.001	78	84.5	50	96
Severe							
MCAs + ACAs on angiography	7	72					
<50% narrowing	26	19	< 0.0001	58	91	75	79
>50% narrowing							

[a] PCT, perfusion computed tomography; rMTT, regional mean transient time; +PV, positive predictive values; −PV, negative predictive value; ^{99m}TC ECD, ^{99m}Tc ethyl cysteinate dimer.

scanning in all 45 patients. Of these, severe vasospasm was found in 38 sides, whereas no vasospasm was found in 76 sides. The average minimal rCBF and maximal rMTT in the MCA territories with severe vasospasm was 29.3 ± 17 cm^3/100 r/min and 4.5 ± 2.4 seconds compared with 54.1 ± 25.4 cm^3/100 r/min and 2.8 ± 1.1 seconds for territories in which no vasospasm was found ($P = 0.0042$ and $P < 0.0001$ for rCBF and rMTT, respectively) (*Table 2*). There were cerebral angiographic images available for comparison with 31 PCT scans. Severe narrowing was found in 45 of 124 arteries evaluated (anterior cerebral artery, MCA). The average minimal rCBF and maximal rMTT in territories that angiography revealed severe narrowing of the parent artery were 26.4 ± 10.3 cm^3/100 g/min and 5.8 ± 2.7 seconds compared with 48.5 ± 27.6 cm^3/100 g/min and 3.2 ± 1.7 seconds for 79 territories in which angiography revealed mild, moderate, or no narrowing ($P < 0.0001$ and $P < 0.0001$ for CBF and MTT, respectively) (*Table 2*).

rCBF and rMTT Correlation with Estimated CBF by ^{99m}Tc ECD SPECT Imaging

Fifty-one PCT images and 51 ^{99m}Tc ECD-SPECT images were available for comparison. The PCT-rCBF values measured in brain areas, in which ^{99m}Tc ECD-SPECT disclosed severe hypoperfusion, were significantly lower than values in areas that ^{99m}Tc ECD-SPECT had disclosed moderate or mild hypoperfusion or normal perfusion (normal 54.2 ± 23.4; mild 45.6 ± 16.3; moderate 32.4 ± 13.0, severe 18.9 ± 6.9 cm^3/100 g/min, respectively; $P < 0.001$). The PCT-MTT values measured in territories in which ^{99m}Tc ECD-SPECT disclosed severe hypoperfusion were significantly higher than values in areas that ^{99m}Tc ECD-SPECT had disclosed moderate or mild hypoperfusion or normal perfusion (2.5 ± 0.39, 3.4 ± 0.71, 5.4 ± 1.2 and 8.1 ± 1.9 s, respectively; $P < 0.001$). Cutoff values for rCBF and rMTT and their sensitivity and specificity for the outcome parameters are presented in *Tables 2* and *4*.

DISCUSSION

In the present study, we evaluated the value of quantitative measurements of CBF and MTT by PCT scanning in patients with vasospasm after aSAH. To the best of our knowledge, this is the only comprehensive study performed on the role of this relatively new imaging technique in the evaluation and diagnosis of vasospasm after aSAH. In stroke patients, CBF can be judged as relative to CBF found on the healthy side (17, 20, 21). However, in vasospasm, because multiple territories may be involved, this comparison may not be reliable. Therefore, we tried to evaluate whether absolute CBF and MTT values provide reliable information regarding the hemodynamic status of patients with vasospasm after aSAH.

Using quantitative CBF and MTT values, we found a high concordance rate with ischemic deterioration, and both values were found to be in high correlation with MCA vasospasm severity and degree of arterial narrowing as well as with perfusion impairments as estimated by ECD-SPECT imaging. The findings suggest that patients with an absolute rCBF lower than 25 cm^3/100 g/min and an rMTT greater than 6.5 seconds are at a higher risk for ischemic deterioration and have greater chances for severe arterial narrowing.

Almost all patients with severe hypoperfusion on SPECT imaging showed reduced rCBF and prolonged rMTT on PCT in the same vascular territory. Furthermore, most regions with unimpaired perfusion or mildly impaired rCBF on ECD-SPECT imaging were unimpaired or mildly impaired on PCT scans. To evaluate the perfusion status more precisely, the radionuclide material should be injected at the time of the PCT study. In the present study, SPECT and PCT images were obtained in close temporal proximity; given the dynamic nature of vasospasm, there could be bias related to the time discrepancy. Furthermore, we evaluated the perfusion maps at the level of the centrum semiovale, which do not necessarily cover the calcarine sulcus area that is absolutely supplied by the posterior cerebral arteries (23). It should be considered that, in cases of posterior circulation aneurysm, other scanning can be performed at the level of the third ventricle that better covers the posterior cerebral artery territories.

In the current study, we did not use CBV for correlation with all measured outcome parameters because severe CBV impairments often are associated with an already existing infarction, which was less relevant for our study purposes (10). Nevertheless, it should be considered that a very low measured CBV with impaired rCBF and rMTT values in one territory might suggest a significant ongoing vasospasm in the parent artery that may adversely influence perfusion in other regions of the vascular territory. Furthermore, perfusion values were evaluated in several areas within the vascular territories. Although the absolute extreme values were taken for comparison with the different outcome parameters, one must consider that variations in perfusion within the vascular territory are possible, and the magnitude of involvement of the vascular territory is an important factor in judging the severity of hemodynamic impairment. For example, a moderate CBF and MTT defect in all the vascular territory of the MCA may be suggestive of more severe vasospasm in the parent artery (M1 and M2) than a severe but very focal disturbance in the distal MCA territory.

Although good correlation was reported between CBF measurement by PCT and xenon CT in normal subjects as well as with perfusion MRI scans (5, 16), one must remember that PCT methods have many limitations related to the deconvolution algorithm (2–4) used for calculation and assume normal cerebral hemodynamic physiology and intact blood-brain barrier, which is not necessarily so in the vasospasm patient. Therefore, we suggest that the absolute rCBF and rMTT values should be judged and used as guiding values, adding them to other data, such as the clinical course, and other tests, such as TCD measurements, to provide more reliable information on the patient's hemodynamic status. Nevertheless, the findings in the present study show that normal rMTT and rCBF, if measured by PCT, carry high negative predictive values for significant hemodynamic impairments, which can provide assurance for unimpaired perfusion in uncertain cases.

Regardless of the drawbacks of PCT techniques, it has significant advantages over other perfusion methods in the acute setting of vasospasm when rapid treatment decisions are necessary (20). PCT scanning can be performed immediately after conventional CT scanning and is highly suitable for the evaluation of unstable or uncooperative patients. More importantly, because a helical CT scanner with the appropriate software is all that is needed for perfusion assessment, PCT scanning can be a solution for many institutions with limited financial resources. Currently, we believe that PCT scanning alone or in conjunction with TCD measurements provides significant assurance of the hemodynamic status of patients with cerebral vasospasm. We see two major applications of PCT scanning in the diagnosis of hemodynamic impairment resulting from vasospasm. First, in poor grade patients in whom the clinical examination may not be reliable, we have found PCT scans to be useful when there is uncertainty regarding deterioration (in the case of a heavily sedated and ventilated patient) or when TCD measurements show significant vasospasm or dynamic changes toward significant vasospasm. In these patients, PCT scanning can be performed to rule out or to identify significant perfusion impairments, thereby helping in the decision-making regarding the need for intervention. Secondly, in conscious patients who develop delayed deterioration whose cause is uncertain (e.g., concomitant fever, increase intracranial pressure secondary to hydrocephalus), PCT scans provide additional data regarding hemodynamic status and ischemic deterioration.

CONCLUSION

PCT scans can provide data regarding hemodynamic impairments in patients after aSAH. Although PCT scanning has many drawbacks that reduce the reliability of absolute perfusion values, the present study suggests that, in general, absolute measurements of CBF and MTT by PCT scanning show

high concordance rates with the clinical course, vasospasm severity, and SPECT imaging. Further studies should be performed to evaluate the role of PCT scans in the diagnosis of cerebral vasospasm.

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COMMENTS

Perfusion computed tomography (PCT) is emerging as a method to assess blood flow in patients with subarachnoid hemorrhage (SAH) and vasospasm. Other methods, such as magnetic resonance imaging (MRI)-based perfusion, xenon computed tomography, and single-photon emission computed tomography (SPECT) have been used, but have not obtained widespread use because of the difficulties involved with performing them. PCT is easy to perform when the patient undergoes a computed tomographic (CT) angiogram and plain CT scan a week after SAH. The difficulties with the technology are the assumption of an intact blood-brain barrier and variability in the arterial input function, which make the obtained values variable and difficult to standardize. Neurosurgeons wishing to use this technique should work with a neuroradiologist and analyze the calculations of a few cases to determine the variations in the flow values when the sampled arteries and brain areas are slightly varied. This is not a simple method by which to obtain accurate quantitative values, but it can be done. An indication of the limitations is suggested by the exclusion of eight out of 71 scans (11%) because of poor quality.

The authors present a series of carefully analyzed patients. They measured vasospasm quantitatively by diameter measurements of baseline and follow-up angiograms. In the absence of the angiograms, we have comparisons of one less than 100% accurate test (CT perfusion) with several other less than 100% accurate tests (transcranial Doppler [TCD], SPECT). Therefore, one cannot obtain an accurate representation of the procedure. The values are also derived from detailed analysis of mean maximums and minimums, which suggest that in the real-time setting of patient management, sensitivities, and specificities might be less. The simplicity of CT-based imaging, however, makes me anticipate that this method, or some variant of it, will become easier to standardize and use in management of patients with SAH.

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Sviri et al. present the PCT data from a series of 46 patients with vasospasm secondary to aneurysmal SAH. All patients had TCD measurements, cerebral angiography, or SPECT performed in proximity with the PCT scan. The authors found that the mean regional

cerebral blood flow (rCBF) and maximal regional mean transient time (rMTT) in patients with delayed ischemic deficit (DID) were significantly different compared with patients without DID. They also found that the minimal rCBF and maximal rMTT in middle cerebral vascular territories determined to be in spasm by TCD were significantly different from those with normal TCD values. Finally, they found that minimal rCBF and maximal rMTT in vascular territories with severe hypoperfusion on SPECT imaging were significantly different from those with normal spectroscopy. Based on these findings, the authors conclude that quantitative measurements of rCBF and rMTT by PCT highly correlate with the clinical course, severity of disease, and hemodynamic impairment in patients with cerebral vasospasm.

When interpreting the conclusions of this article, it is important to consider the major limitations of PCT findings in this clinical scenario. The derivation of absolute values for rCBF, cerebral blood volume, and rMTT from PCT data using the deconvolution method assumes ideal physiologic parameters and is highly contingent upon selection of the arterial input function (AIF). This model assumes a rapid and even distribution of contrast from the chosen artery to the output vessel (usually the superior sagittal sinus). This model also assumes an intact blood-brain barrier. In animal models and in patients with normal intracranial physiology (intact autoregulation, normal cerebral perfusion, and systemic blood pressure, etc.), absolute values for rCBF, rMTT, and cerebral blood volume can, theoretically, be derived from PCT studies. However, in the setting of acute aneurysmal SAH (aSAH) and cerebral ischemia secondary to vasospasm, none of these assumptions can reliably be made.

Nevertheless, with increasing availability, PCT represents a valuable tool for evaluating aSAH patients that may allow assessment of vasospasm severity and assist in identifying patients at risk for permanent cerebral infarction.

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The authors describe 46 patients with cerebral vasospasm after SAH from intracranial aneurysms. All patients in the study had TCD measurements, 28 had angiographic studies, and 38 had SPECT studies performed in conjunction with a perfusion CT scan. The authors found a high association of rCBF and rMTT by PCT.

We continue to search for the best way of evaluating patients as they experience vasospasm and are treated for vasospasm after SAH. With the use of mobile CT scanners, PCT scanning is now available at a number of institutions. As in other reports, this article demonstrates a fairly high correlation between the findings on perfusion studies and those of other measures of vasospasm. Vasospasm is a dynamic process with great variability in different areas of the brain, depending on the degree of collateral supply and the amount of blood in that particular area of the brain after hemorrhage. In most centers, there is a multimodality approach of imaging in the evaluation of spasm. As in the current study, it is typical to combine the clinical findings with the TCD measurements and some radiographic study of blood flow. Despite this multimodality approach, the sensitivity and specificity for spasm remains variable. The current study offers further definition of the use of dynamic PCT scan in the diagnosis of cerebral vasospasm. The authors are to be commended for their work.

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The development of vasospasm after SAH is still a problem in the treatment of patients with aneurysms and is associated with high rates of morbidity and mortality. Patients with massive bleeding and high Hunt and Hess grades on admission are at a risk of developing vasospasms. However, in these patients, a solid clinical diagnosis is hampered by their unconscious state. The specificity of TCD ultrasound, although widely used, is a matter of debate because the results depend on anatomical relationships and the experience of the investigator. Digital subtraction angiography and MRI techniques are invasive, time-consuming, expensive, and elaborate for critically ill patients. Therefore, it is desirable to find a technique that may predict or, at least, diagnose vasospasm with an accurate sensitivity and specificity and that is suitable for wide use in the critically ill.

In the present study, the authors evaluate the value of quantitative measurements of rCBF and rMTT by PCT in patients with vasospasm after SAH. The comparatively new technique of PCT was compared with TCD data, SPECT measurements, and angiographic studies. Sixty-three patients received PCT and TCD. Angiography was performed in 31 patients, and 51 SPECT images were available for comparison. The authors found a correlation with clinical aspects of DID, with the severity of vasospasm measured using TCD and angiography, and with SPECT imaging. These data have a high practical impact on the diagnosis and treatment of SAH-induced vasospasm, which needs to be confirmed in further studies.

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Sviri et al. present one of the more comprehensive studies to date evaluating PCT in the assessment of vasospasm associated with SAH. From an imaging perspective, PCT presents technically interesting challenges, and it has recently attracted attention in the neuroradiology community. However, the "real-life" clinical impact and/or utility of PCT information has not been fully delineated at this time, specifically in regards to how much it actually affects treatment decisions independent of angiographic, TCD, and clinical data. This may be one of the reasons that enthusiasm for studying this technique has been relatively tepid since first reports were published several years ago. It's difficult to imagine, even given the high negative predictive value of a "negative" PCT study, that this test would trump angiographic and clinical data. PCT can delineate perfusion deficits secondary to small distal branch vasospasm that shows an essentially unremarkable angiographic (or CT angiographic) appearance of the proximal large and mid-sized vessels. In this instance, PCT can yield reassuring information and provide a rationale for application of triple H therapy. Thus, PCT in patients with SAH may have at least some clinical utility in the majority of cases. There are notable exceptions. As the authors report, clinical findings in patients with SAH can be equivocal or difficult to assess, and it is in this situation that direct knowledge of perfusion status becomes the most useful.

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