Assessment of intracranial hemodynamics in carotid artery disease by transcranial Doppler ultrasound

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Noninvasive transcranial Doppler recordings were correlated to the angiographic findings in 77 patients with carotid artery disease. Stenoses reducing the luminal area of the internal carotid artery by 75% or more also reduced the pulsatility transmission index (PTI) of the ipsilateral middle cerebral artery (MCA). The PTI is the pulsatility index of the artery under study expressed as a percent of the pulsatility index of another intracranial artery with presumed unimpeded inflow in the same individual. For stenoses in the 75% to 89% category, PTI reduction was significantly greater in patients with bilateral carotid stenosis, indicating an impaired potential for collateral flow in these patients. The PTI reduction probably reflects both the pressure drop across the stenosis and the cerebral autoregulatory response. Two criteria proved useful in demonstrating collateral MCA supply through the circle of Willis. On the recipient side, retrograde flow in the proximal anterior cerebral artery was demonstrated in 29 of the 31 patients when this flow pattern was disclosed angiographically. In 26 of these patients, the anterior cerebral artery on the supplying side also had clearly increased flow velocity. Increased flow velocities in the proximal posterior cerebral artery were present in 26 of the 30 vessels that were acting as a collateral channel to the ipsilateral MCA.

Key Words: intracranial blood flow, carotid artery disease, ultrasound, pulsatility transmission index

Stenoses of the internal carotid artery (ICA) most commonly occur extracranially. Direct examination by Doppler ultrasound is accurate in diagnosing such lesions. However, the effect on the flow to the cerebral arteries is governed not only by the severity of the extracranial ICA lesion, but also by the system of collateral vessels circumventing the obstruction. Due to the anatomical variants of the circle of Willis, the capacity of these collateral vessels has important differences in individual patients. For this reason, extracranial Doppler techniques alone are not satisfactory in evaluating the net hemodynamic effect of carotid stenosis or occlusion.

Unlike the extracranial carotid arteries, which are uniquely situated for investigation with Doppler ultrasound, the basal cerebral arteries usually affected in stroke-prone adult individuals are surrounded by the bony cranium. Nevertheless, the noninvasive extracranial Doppler technique can be used to record flow velocities in the middle, anterior, and posterior cerebral arteries (MCA, ACA, and PCA) in most individuals.

The present study was designed to establish a procedure for assessing the effect of ICA stenoses on flow patterns in basal cerebral arteries and to compare this noninvasive technique with angiographic parameters.

Theoretical Considerations

Stenosis of an artery causes a pressure loss that is reflected by a damping of blood flow velocity waveforms recorded distally. The MCA can be regarded as being an end artery. Hence, it was expected that the net hemodynamic effect of ICA stenoses could be assessed from recordings of MCA flow velocity. Gosling and King introduced a dimensionless parameter, the pulsatility index (PI), for quantifying Doppler waveforms. The PI is the amplitude of the flow velocity waveform, or spectrum outline, divided by its time-mean value (Fig. 1). Since systemic cardiovascular characteristics differ between individuals, the PI also varies considerably. In the present study, the pulsatility transmission index (PTI) was introduced to correct for individual variation. The PTI is the PI measured...
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**Fig. 1.** Left: Determinations of systolic, diastolic, and time-mean values of the Doppler velocity spectrum outline ($V_s$, $V_d$, and $V$, respectively). The pulsatility index, $PI = (V_s - V_d)/V$. Center: Transcranial flow velocity recordings. Probe position 1 is preferred for recordings from the middle and posterior cerebral arteries (MCA and PCA). The sampling volume is shown in position for MCA recordings. The sampling depth setting for these recordings is usually about 45 mm. The PCA is identified by aiming the probe slightly more posteriorly, using depth settings of about 65 mm. Probe position 2 is preferred for recordings from the anterior cerebral artery in individuals with more than one ultrasonic window. Right: Location of areas where the adult cranium usually is sufficiently thin to allow penetration of ultrasound for transcranial recordings of flow velocity (“ultrasonic windows”). Numbers correspond to probe positions shown center.

for the MCA under study ($PI_{MCA}$), expressed as a percentage of the $PI$ for another basal cerebral artery with presumed unimpeded inflow in the same individual ($PI_{REF}$), so that $PTI = (PI_{MCA}/PI_{REF}) \times 100$.

**Clinical Material and Methods**

**Control Groups**

Control Group A consisted of 20 healthy volunteers aged 20 to 35 years (median age 28 years). Control Group B consisted of 20 individuals aged 49 to 63 years (median age 55 years) without cerebral symptoms or signs, who were scheduled for major cardiovascular operations. Asymptomatic carotid stenosis was ruled out in all control individuals by Doppler examinations of the extracranial carotid arteries.

**Patient Population**

A total of 77 patients with angiographically proven carotid artery disease, investigated in Bern (16 patients) or in Oslo (61 patients), were admitted to the study. Their ages ranged from 42 to 76 years (median age 61 years). The routine clinical examination included Doppler ultrasonography of the extracranial carotid and vertebral arteries. These findings are not considered here. Biplane selective carotid angiograms of acceptable technical quality, showing the extracranial carotid arteries and the basal cerebral arteries, were available in all patients. Patients with angiographically evident stenosis or occlusion of intracranial arteries were not included.

The luminal diameter of the stenosed ICA was measured with calipers to the nearest half millimeter in two projections at the point of maximum narrowing (measurements a and b) and again at about 4 cm distally (measurements A and B). The result is expressed as a percentage area stenosis so that $1 - (a/A \times b/B) \times 100 = \text{stenosis (\%)}$. All 77 patients had at least one ICA stenosis that reduced the luminal diameter by 40% or more. Twenty-eight patients had bilateral ICA stenosis of more than 40%. Hence, the other 49 patients had one ICA that was, for the purpose of this study, considered normal. These 49 patients were denoted as having unilateral ICA stenosis. Angiographically, all ICA stenoses of 40% or more were allocated to one of the following categories: 40% to 74%, 75% to 89%, 90% to 99%, and 100% stenosis (total ICA occlusion).

The stenosis percentages for vessels in patients with unilateral ICA stenosis and in patients with bilateral ICA stenosis were randomly distributed within each of these categories ($p > 0.25$). Unilateral selective vertebral angiograms were obtained in 38 patients. The intracranial films were reviewed for evidence of collateral filling.

**Transcranial Doppler Recordings**

Laboratory prototype 2-MHz pulsed-wave Doppler instruments with focused transducers were used. The depth of the sampling location can be selected by the operator through range-gating. The Bern instrument has been described previously. The Oslo instrument differs mainly in that it incorporates a dedicated microcomputer for on-line 64-point fast Fourier transform (FFT) frequency analysis of the Doppler-shifted spectra. Eighty consecutive spectral analyses (2.2 seconds each) were displayed on a monitor and transferred to a dot matrix printer for hard copy production.

The standard examination procedure has been reported by Aaslid, et al. Some details, particularly pertaining to the examination of patients with carotid artery disease, are presented here. There are basically two sources of information for identification of the various intracranial arteries: 1) the depth setting and
FIG. 2. Transcranial Doppler identification of basal cerebral arteries in a healthy individual during a test occlusion of the common carotid artery (CCA) in the neck. The proximal segments of the middle and posterior cerebral arteries (MCA and PCA) have a normal flow direction toward the transducer. An ipsilateral CCA test occlusion reduces the absolute MCA flow velocity level, which reflects MCA inflow through collateral vessels (center). By contrast, the response in the PCA signal to this test is an increased velocity level, indicating the potential of this vessel as a collateral source (right).

FIG. 3. Transcranial Doppler identification of the anterior cerebral artery (ACA) in the same healthy individual as monitored in Fig. 2. In the proximal anterior cerebral artery (ACA), the normal flow direction is away from the probe. Normally directed ACA velocity spectra are therefore shown below the zero line (left). Test occlusions of the common carotid artery (CCA) give rise to characteristic responses in the ACA flow velocity patterns.
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![Graph](image-url)

**Fig. 4.** In the 49 patients with unilateral internal carotid artery (ICA) stenosis, the middle cerebral artery (MCA) flow velocity (V_{MCA}) on the side with ICA stenosis is presented, expressed as a percentage of V_{MCA} on the normal side, and correlated with the percentage diameter of ICA stenosis. The median value decreases for ICA stenosis over 90%. Nevertheless, the ranges are wide with values from patients with total ICA occlusion (100% stenosis) overlapping even those from control individuals, owing to the MCA blood flow and V_{MCA} being maintained when cerebral autoregulation is operative. The V_{MCA} value is therefore quite insensitive to the hemodynamic effect of ICA stenosis. The numbers over each column denote the number of patients in each group.

The aiming direction combined with the directional spectral display of the signal; and 2) the response of the Doppler signal to test occlusions of the common carotid artery (CCA) in the neck. The CCA is the only artery emerging laterally from the circle of Willis toward the probe position (Fig. 1 center and right). By successive reduction of the sampling depth, the signal from the CCA can be followed outward to about 30 mm from the transducer, where the branches of the CCA bifurcation can often be differentiated. In tracking the CCA signal into the depth of the Sylvian fissure, the recording of simultaneous signals directed toward and away from the probe indicates where the terminal ICA bifurcates into the MCA and the ACA. This is a useful landmark for spatial orientation. The PCA is identified by aiming the probe slightly more posteriorly. The proximal PCA can be scanned inward to the basilar artery tip where the contralateral PCA flows in the opposite direction. It is also possible to record from more distal PCA segments with blood flowing away from the probe. The ACA can be tracked across the midline where a signal directed toward the transducer appears. This signal is from the contralateral ACA and indicates a recording from the anterior communicating artery region. Test occlusions of the CCA elicit characteristic patterns of flow velocity responses (Figs. 2 and 3).

**Data Analysis**

The angiographic and transcranial Doppler findings were interpreted independently. The time-mean flow velocity for the MCA, PCA, and ACA (V_{MCA}, V_{PCA}, and V_{ACA}, respectively), and the PI for the MCA and PCA (PI_{MCA} and PI_{PCA}, respectively), were determined from the frozen spectral display (Fig. 1 left). All figures represent the average from 10 consecutive cardiac cycles. The PTI was calculated for the MCA on both sides in all patients. The highest value determined for the PI_{PCA} represented the PI_{REF}. Statistical evaluations were performed using the Wilcoxon test for two samples. The correlation between transcranial Doppler and angiography findings was determined in terms of sensitivity and specificity and expressed as percentages.

**Results**

**Middle Cerebral Artery Studies**

**MCA Flow Velocities.** The V_{MCA} mean values and standard deviations (SD) were 67 ± 7 cm/sec (range 52 to 88 cm/sec) in Control Group A and 58 ± 10 cm/sec (range 38 to 82 cm/sec) in Control Group B (difference statistically significant, p < 0.01). In the 49 patients with unilateral ICA stenosis, the V_{MCA} on the normal side was 59 ± 12 cm/sec, with a range of 38 to 94 cm/sec (not significantly different from Control Group B). In control individuals, the V_{MCA} for the left side was expressed as a percentage of the V_{MCA} for the right side. The mean and SD was 100% ± 11% and 102% ± 14% for Control Groups A and B, respectively (not significantly different).

In the 49 patients with unilateral ICA stenosis, the absolute MCA flow velocity (V_{MCA}) on the stenosed side was expressed as a percentage of V_{MCA} on the normal side and correlated with the percentage ICA stenosis, as shown in Fig. 4. The median flow value dropped in ICA stenoses of more than 90%, but the ranges for all stenosis categories were wide and even overlapped those of control individuals. This indicates that the hemodynamic effect of ICA stenoses cannot be satisfactorily assessed from the absolute MCA flow velocity alone, even when an MCA with normal inflow conditions is available for comparison.

**Pulsatility Transmission Index.** The values for PI_{MCA} in Control Groups A and B were 0.71 ± 0.10 and 0.94 ± 0.14, respectively. This significant difference (p < 0.015) was due to lower heart rates in Control Group B (p < 0.02). In assessment of the PTI in control individuals, the PI_{MCA} for the left side was expressed as a percentage of the right side PI_{MCA}, or of the PI_{PCA}, whichever was the highest. These PTI values were 100 ± 4 and 99 ± 4 for Control Groups A and B, respectively (not significantly different). In the 49 patients with
unilateral ICA stenosis, the PI_{MCA} on the normal side was 0.96 ± 0.16, with a range of 0.74 to 1.62 (not significantly different from Control Group B).

Figure 5 shows the correlation between the PTI and ipsilateral ICA stenosis in all patients. The PTI value showed a definite drop when the ipsilateral ICA stenosis exceeded 75%. Of a total of 81 patient hemispheres with an ICA stenosis of 75% or more, only three of the hemispheres had PTI values of over 92 (sensitivity 96%). All PTI values from patient hemispheres where the ICA was stenosed by less than 75% were above 92 (sensitivity 100%). A comparison of PTI findings from patients with unilateral and with bilateral ICA stenosis, respectively, showed significantly lower PTI values for the 75% to 89% stenosis category in patients with bilateral ICA stenosis (p < 0.025). The potential for collateral flow is generally impaired in these patients. This is illustrated by the finding of retrograde flow in 29 of the 31 patients with this cross-flow pattern disclosed angiographically (sensitivity 93%, specificity 100%).

The MCA flow velocity level (V_{MCA}) generally correlated poorly with the degree of ipsilateral ICA stenosis (Fig. 4). A concomitant reduction of PTI and V_{MCA} was nevertheless observed in some patients. This is illustrated in Fig. 6. For PTI values below 60, the V_{MCA} was reduced to levels significantly below those observed when the PTI was 60 or higher (p < 0.02).

**Circle of Willis Flow Studies**

Bilateral recordings of ACA flow velocities were made in 18 individuals from Control Group A and in 16 individuals from Control Group B. In six control individuals, ACA recordings were obtained on one side. The ACA flow velocity (V_{ACA}), calculated as a percentage of V_{MCA} on the normal side, showed significant correlation with the corresponding pulsatile transmission index (PTI). The values show no deviation from findings in control individuals or in the other patients (p < 0.02). Also, the successively lower V_{MCA} with further PTI reduction. The numbers over each column denote the number of patients in each group. The findings in patients with total ICA occlusion (circled numbers) showed a great diversity owing to individual variations in collateral capacity. Results in Control Groups A and B are given for comparison.

**Fig. 5. Pulsatility transmission index (PTI) for middle cerebral artery (MCA) recordings correlated with percentage diameter of stenosis of the ipsilateral internal carotid artery (ICA) in patients with unilateral (open columns) and bilateral ICA stenosis (cross-hatched columns).** A definite PTI drop occurred for ICA stenoses over 75%. For stenoses in the 75% to 89% range (darker column), PTI was significantly lower in patients with bilateral ICA stenosis compared to patients with unilateral ICA stenosis (p < 0.025). The potential for collateral flow is generally impaired in these patients.

**Fig. 6. In the 49 patients with unilateral internal carotid artery (ICA) stenosis, the middle cerebral artery (MCA) flow velocity (V_{MCA}) on the side with ICA stenosis is presented, expressed as a percentage of V_{MCA} on the normal side.** Correlation with the corresponding pulsatile transmission index (PTI) values shows no deviation from findings in control individuals or patients with PTI over 70, but PTI values below 60 were associated with a V_{MCA} asymmetry that was significantly greater than in control individuals or in the other patients (p < 0.02). Also, the successively lower V_{MCA} with further PTI reduction. The numbers over each column denote the number of patients in each group. The findings in patients with total ICA occlusion (circled numbers) showed a great diversity owing to individual variations in collateral capacity. Results in Control Groups A and B are given for comparison.
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**FIG. 7.** Transcranial Doppler findings in a 58-year-old man in whom left carotid angiography confirmed total internal carotid artery (ICA) occlusion, right carotid angiography revealed no ICA stenosis but did indicate collateral filling across the midline into the left middle cerebral artery (MCA), and the vertebral angiograms were normal with no filling of collateral vessels. The left MCA velocity outline has a reduced pulsatility index (PI = 0.76) when compared to the recording from the right MCA (PI = 1.00). The absolute velocity levels ($V_{MCA}$) are about equal. The right MCA and the posterior cerebral artery (PCA) velocity outlines show identical pulsatility. The pulsatility transmission index (PTI) for the left MCA is 76. On the right side, an anterior cerebral artery (ACA) collateral channel to the left MCA is indicated since the $V_{ACA}$ (upper right) is increased to 175% of the right $V_{MCA}$. Retrograde flow velocities in the left ACA confirm this finding (lower right).

grade ACA velocities. On the side providing collateral flow, the $V_{ACA}$ level was increased to over 150% of the ipsilateral $V_{MCA}$ level in 26 of the 31 patients with an ACA collateral branch to the opposite MCA (Fig. 7). Such high $V_{ACA}$ levels were also seen in two patients with double filling of both distal ACA's (pericallosal arteries) from one side (sensitivity 84%, specificity 92%). The ACA findings are further detailed in Table 1.

An increase in $V_{PCA}$ to over 125% of the ipsilateral $V_{MCA}$ indicated that the PCA flow was collateral to the MCA flow (Fig. 8) in 26 of the 30 patients in whom this filling pattern was disclosed (sensitivity 86%, specificity 92%). In identifying the PCA, it should be noted that test occlusion of the carotid artery obscured the PCA when PCA inflow was from the distal intracranial ICA (Table 2). In that situation, the PCA inflow conditions are identical to those of the ipsilateral MCA, and the PI of another vessel must be substituted when determining PTI.

**Discussion**

Individual differences in heart rate, blood pressure, vascular compliance, and arterial pCO₂ account for great individual variation in the pulsatility index (PI) of the Doppler spectrum outlines.¹¹,¹³ For clinical investigation, it is therefore essential to correlate PI values to reference values from each individual studied. The purpose of the pulsatility transmission index (PTI) is to correct for individual variations. This was confirmed by the PTI being 100 ± 4 (mean ± SD) in healthy individuals. This standard deviation reflects a methodological inaccuracy. Theoretically, 95% of normal individuals will fall within ± 2 SD from the mean value. Thus, PTI = 92 seems to be a clinically useful limit for detecting impaired MCA inflow. For a total of 81 ICA's stenosed by 75% or more, the PTI for the ipsilateral MCA was over 92 on only three occasions (sensitivity 96% and specificity 100%).

Experimental studies indicate that a mean pressure drop of 5 mm Hg or more and a reduced PI occur in all cases of artery stenoses with about an 80% reduction in area.⁷,¹⁴,²⁵ The present findings of reduced PTI values for ICA stenoses of over 75% fit with those observations. For ICA stenoses in the 75% to 89% category, the PTI was even lower in patients with bilateral ICA stenoses (p < 0.025, Fig. 5). Such cases do exhibit impaired potential for collateral vessel flow.²³ Stenoses reducing the luminal area by over 90% significantly

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reduce ICA volume flow. When a very tight ICA stenosis is present, the MCA inflow conditions may therefore depend as much upon the capacity of the circumventing collateral vessels as upon the ICA stenosis per se. The wide range of PTI values found in patients with total ICA occlusion reflects the fact that the collateral vessel capacity varies in different individuals.

In spite of severe ICA stenosis or even total ICA occlusion, the ipsilateral cerebral hemisphere often has a blood flow within normal limits. In response to reduced inflow pressures, cerebral autoregulation maintains blood flow by lowering cerebrovascular resistance. In effect, MCA volume flow and flow velocity remain relatively constant when cerebral autoregulation is operative. Therefore, the PTI is more sensitive than is \( V_{MCA} \) to the net hemodynamic effects of ICA stenoses. The PTI reduction reflects a combination of reduced MCA inflow pressure and lowered cerebrovascular resistance. The relative importance of these two components cannot be assessed from the present data.

Flow velocities in basal cerebral arteries have a wide normal range. The present series confirms this. The individual variation observed in \( V_{MCA} \) probably parallels individual vessel caliber variation. The \( V_{MCA} \) was significantly lower in persons with systemic atherosclerosis (Control Group B) than in healthy individuals (Control Group A). This probably reflects the fact that widening of arteries is part of this process. The values for \( V_{MCA} \) on opposing sides were nevertheless within 30% of each other in all control individuals (Fig. 4). Slightly different vessel diameters on the two sides in one individual seem to be common and explain findings of different \( V_{MCA} \) values on opposing sides. Some patients with very low PTI values also had a concomitant \( V_{MCA} \) reduction to levels significantly below those seen under less extreme circumstances (Fig. 6). Such findings might suggest that MCA inflow is at or even below the lower limit of autoregulation. However, the present data permit no firm conclusions in this respect.

High velocities in collateral channels reflect the increased perfusion territory and demand for blood flow through these vessel segments. The ACA and PCA flow velocities were clearly increased when these vessels conveyed collateral flow into the MCA territory. If collateral channels dilate with time, allowing for increased flow rates without correspondingly increased

**FIG. 8.** Transcranial Doppler findings in a 66-year-old man in whom right carotid angiography revealed 90% stenosis of the extracranial internal carotid artery (ICA) and proximal anterior cerebral artery (ACA) filling by the right and left pericallosal arteries (distal ACA's) only, left carotid angiography showed total ICA occlusion, and vertebral angiography demonstrated left middle cerebral artery (MCA) filling through a collateral channel from the left posterior cerebral artery (PCA) but no filling of the left ACA. The pulsatile index and velocity for the MCA (\( P_{MCA} \) and \( V_{MCA} \), respectively) are about equal. The signal from the right PCA is more pulsatile (upper center), indicating impaired inflow to both MCA's (pulsatile transmission index: left MCA 72; right MCA 73). The left \( V_{PCA} \) is 200% of the left \( V_{MCA} \), indicating collateral flow. The right \( V_{ACA} \) (upper right) is 170% of the right \( V_{MCA} \). No flow was recorded in the left ACA.
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flow velocities, this may explain findings of only moderately elevated flow velocities in some collateral channels (Tables 1 and 2). Filling of both pericallosal arteries (distal ACA’s) from one side may be due to the hemodynamic effect of a contralateral ICA stenosis. However, anatomical variations of the circle of Willis also account for such filling patterns.\(^9\)

Cerebral perfusion in carotid artery disease is related to the conditions of inflow to the basal cerebral arteries. The MCA, which can be regarded as an end artery, may originate from stenosed carotid arteries as well as from circumventing collateral channels. Conventional Doppler examinations are useful in identifying extracranial carotid lesions.\(^2\) The transcranial Doppler technique extends noninvasive tests to include assessment of net circumventing collateral channels. Conventional Doppler recordings complement the anatomic and hemodynamic information obtained angiographically. Together, these methods improve the understanding of the individual cerebral circulatory state. The PTI seems to permit grading of the cerebral inflow conditions. Studies of the relationship between PTI and the pressure drop across carotid artery stenoses are in progress.

Results from extracranial-intracranial bypass operations correlate well with the MCA perfusion pressure.\(^2\) Methods to assess the conditions of inflow to the basal cerebral arteries thus seem highly relevant to the question of hemodynamic benefit from cerebral revascularization.

**References**


15. Nornes H: The role of the circle of Willis in graded

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**TABLE 1**

<table>
<thead>
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<th>Transcranial Doppler:</th>
<th>Angiographic Findings</th>
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<th>Total Vessels</th>
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<tr>
<td>V(_{AC}) (%) ipsilat</td>
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<td>A(_{CA}) Filling</td>
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<tr>
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<td>&gt; 150</td>
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<td>A(_{CA}) flow</td>
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<td>total vessels</td>
<td>60</td>
<td>16</td>
<td>31</td>
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* V\(_{AC}\) and V\(_{MCA}\) = time-mean flow velocity measured from Doppler spectrum outlines recorded from the ACA and middle cerebral artery (MCA).

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**TABLE 2**

<table>
<thead>
<tr>
<th>Transcranial Doppler:</th>
<th>Normal PCA Filling To MCA</th>
<th>Collateral PCA Filling To MCA</th>
<th>PCA Filling To MCA From ICA</th>
<th>Total Vessels</th>
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<td>V(<em>{PCA}) (%) ipsilat V(</em>{MCA})</td>
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<tr>
<td>&lt; 75%</td>
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<tr>
<td>total vessels</td>
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<td>30</td>
<td>7</td>
<td>76</td>
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* Selective vertebral angiography was performed in 38 patients. V\(_{PCA}\) and V\(_{MCA}\) = time-mean flow velocity measured from Doppler spectrum outlines recorded from the PCA and middle cerebral artery (MCA).

† Identification of the proximal PCA segment was not possible when the PCA originated from the intracranial internal carotid artery (ICA).

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