Effect of Contralateral Carotid Artery Stenosis on Carotid Ultrasound Velocity Measurements

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Background and Purpose—Carotid ultrasonography is being increasingly performed as the sole investigation to assess internal carotid artery (ICA) stenosis. A potential source of error in using ultrasound peak systolic velocity (PSV) measurements is that the redistribution of blood flow due to severe stenosis in a contralateral carotid artery may lead to artificially elevated values.

Methods—Ultrasonography was performed before and after carotid endarterectomy in symptomatic patients who participated in the North American Symptomatic Carotid Endarterectomy Trial (NASCET). The mean change in PSV in the unoperated artery was assessed across all degrees of angiographically defined stenosis. A simple theoretical resistance model of the cerebral circulation was also derived.

Results—Complete bilateral ultrasound examinations were performed within 90 days of the initial scan in 386 patients. In the presence of a contralateral severe (70% to 99%) ICA stenosis, the PSV in the unoperated artery was artificially elevated by a mean of 84 cm/s ($P=0.03$; 95% CI, 10 to 159 cm/s). The mean elevation was less pronounced for lesser degrees of stenosis (11 to 21 cm/s). Small elevations (3 to 12 cm/s) were observed when the contralateral artery had <70% stenosis. The patterns of observed results were congruent with those from the theoretical model.

Conclusions—The present study showed that a severely stenosed contralateral ICA can artificially elevate ultrasound PSV. Since the effect was greatest when bilaterally severe stenoses were present, caution must be exercised when assessing the degree of ICA stenosis on the basis of ultrasonography PSV measurements alone. (Stroke. 2000;31:2636-2640.)

Key Words: carotid artery disease • carotid endarterectomy • stenosis • ultrasonography

Carotid ultrasonography is being increasingly performed as the sole investigation before carotid endarterectomy in estimating the degree of internal carotid artery (ICA) stenosis on the basis of velocity or frequency measurements.1,2 Several studies have reported that a potential source of error in using these blood flow measurements is that the presence of severe contralateral ICA stenosis or occlusion may artificially elevate the peak systolic velocity (PSV) or frequency (PSF) values used to quantify the degree of stenosis in the artery of interest.2,9,11 In fact, the phenomenon of increased blood flow was astutely noted by Thomas Willis in 1681, centuries before the advent of ultrasonography. He writes in archaic English, describing an autopsy, “When his Skull was opened, we beheld these things belonging to the Head, and found the right Carotides, rifing within the Skull, plainly bony or rather ftony, its cavity being almoft wholly flut up . . . that the blood excluded from the right Carotidick Artery, when at firft it rufhed more impetuouly in the left, had diftended the Membrane.”11 It has been reported that the magnitude of increased blood flow appears to be related to increasing severity of stenosis.2,7,8,10 This is of particular concern when carotid endarterectomy is contemplated solely on the basis of ultrasonography, since some arteries may be erroneously judged to be severe, when in fact they have not reached the stenosis threshold to warrant surgery.

To our knowledge, 3 studies have assessed the magnitude of elevation in a direct manner by recording the change in PSV10 or PSF3,12 in an ICA, before and after a unilateral endarterectomy performed on the contralateral artery. The rationale for this approach was the hypothesis that with endarterectomy restoring blood flow in a diseased ICA, flow would have to be altered in the unoperated artery to maintain the same level of blood volume to the brain as before the surgery. This hypothesis has been referred to as the compensatory flow phenomenon.3

The aim of the present study was to examine the effect of contralateral ICA stenosis on carotid ultrasound PSV measurements in more detail than the aforementioned studies, with specific interest in estimating the magnitude of artificial elevation. The PSV is considered the cornerstone of the...
ultrasound examination. It has been reported that when bilaterally severe stenoses are present, the PSV may be artificially elevated on both sides without clear distinction as to which artery is exerting the greater effect on flow parameters. This confounding effect potentially biases the estimate of elevation. A feature of the present study was to eliminate this confounding effect by using the PSV values obtained before and after an endarterectomy, in addition to using angiography to objectively assign the degree of stenosis to an ICA. Data from the North American Symptomatic Carotid Endarterectomy Trial (NASCET) were used. To assess the validity of the results from the observed trial data, a simple theoretical resistance model of the major pathways and collaterals that form and supply the circle of Willis was developed. The theoretical model provided, independent of anatomic and patient variability, a simple circuit-based examination of the effect of a contralateral ICA stenosis when bilateral ICA stenoses are present. It is noted that the present study made no attempts to correlate the ultrasound measurements with angiographic stenosis because of the reported limitations of doing so.

Subjects and Methods

Study Subjects

NASCET was a prospective randomized multicenter trial designed to determine the role of carotid endarterectomy in patients with symptomatic ICA stenosis. Details of the NASCET inclusion criteria, patient baseline characteristics, and outcome have been described elsewhere. Ultrasonography was performed at the enrolling institution concurrently with the entry angiogram and again approximately 1 month after surgery. Data were sent to the central office, where all available information was reviewed by an experienced ultrasonographer without knowledge of the angiography results or of the clinical status of the patient. Patients who underwent carotid endarterectomy were included in the present study if the entry and follow-up ultrasound data were complete for both the left and right ICAs, and the follow-up ultrasound scan was obtained within 90 days of the initial ultrasound scan. Patiens with any occluded ICA determined by angiography or ultrasonography during the present study period were excluded.

The ultrasound examinations had been performed in tertiary and teaching hospitals using standard techniques and included both longitudinal and transverse views of the carotid bifurcation and relevant stenosis to obtain Doppler readings. The primary measurement used in the present study was the PSV of the ICA and common carotid artery (CCA). Approximately one quarter of NASCET patients with ultrasound data had PSF measurements recorded, and for these patients a PSV was estimated with the use of the Doppler formula with an angle of 60 degrees assigned.

Entry carotid angiograms were reviewed in a blinded fashion by the principal neuroradiologist, and the degree of ICA stenosis was calculated according to NASCET criteria. For the purpose of the present study, each artery was assigned to 1 of 4 categories of stenosis: minimal (0% to 29%), mild (30% to 49%), moderate (50% to 69%), and severe (70% to 99%).

Theoretical Resistance Model

The circuit diagram shown in Figure 1 represents a simplified model of the cerebral circulation. The numbered node points correspond approximately to the following physical locations: 1, aortic arch; 2 and 3, carotid bifurcations; 4 and 6, ophthalmic arteries; 5, circle of Willis; and 7, capillary bed. The resistances (R) corresponding to the vessels between nodes are defined in the Appendix.

![Figure 1. A simple theoretical resistance model of the cerebral circulation. The numbered node points correspond approximately to the following physical locations: 1, aortic arch; 2 and 3, carotid bifurcations; 4 and 6, ophthalmic arteries; 5, circle of Willis; and 7, capillary bed. The resistances (R) corresponding to the vessels between nodes are defined in the Appendix.](http://stroke.ahajournals.org/)

The primary outcome in the present study was the PSV in the unoperated ICA and CCA, before and after endarterectomy in the contralateral ICA. The mean change in PSV was considered an estimate of artificial elevation and was assessed for statistical significance with a paired t test. We also calculated 95% CIs for the mean change.

The theoretical resistance model was used in the following manner. For each patient, their predicted PSV in the unoperated ICA before endarterectomy was calculated from the model by using their degree of angiographically defined, entry ICA stenosis for both the left and right ICAs. The predicted PSV after endarterectomy was calculated from the model by using the value zero for the degree of stenosis in the operated artery while using the same value as the entry angiographic stenosis for the unoperated artery. Each artery was then assigned a stenosis category, and the mean change in predicted PSV was computed and tested for significance.

Results

Of the 1415 patients assigned to the surgical arm in NASCET, 386 had complete ultrasound data for both carotid arteries at entry and within 90 days of the initial scan after endarterectomy. The mean time interval between scans was 51 days, ranging from 10 to 90 days. The mean age of the patients in the present study was 66 years.

Figure 2, top panel, shows the mean PSV across the 4 categories of angiographically defined ICA stenosis in the unoperated artery, before and after an endarterectomy was performed in the contralateral artery of moderate and severe stenoses. The greatest change in PSV was observed in patients with bilaterally severe stenosis. Before endarterectomy, the mean PSV in the unoperated ICA was 376 cm/s. Compared with the mean PSV after endarterectomy of 292 cm/s, it is estimated that the PSV was artificially elevated by...
an average of 84 cm/s (95% CI, 10 to 159 cm/s) by the prior presence of a severe contralateral stenosis ($P = 0.03$). The mean level of elevation in PSV measurements was reduced for lesser degrees of stenosis in the unoperated arteries (21 cm/s for moderate, 20 cm/s for mild, and 11 cm/s for minimal stenosis). In contrast, minimal to moderate stenoses in the operated artery had very little effect. Values of elevation ranged from a mean of 3 cm/s for minimal stenosis in the unoperated artery to 12 cm/s when the arteries had severe stenosis. None of these changes were statistically significant.

The results in Figure 3, top panel, are similar to those in the top panel of Figure 2 in regard to the ICA/CCA PSV ratio. This is explained by the CCA PSV in the unoperated artery remaining unaffected by the performance of carotid endarterectomy. In the case of bilaterally severe stenoses, the ICA/CCA PSV ratio was estimated to be artificially elevated by an average of 2.6 units ($P = 0.01; 95\% CI, 0.7$ to 4.5 units). The bottom panels of Figures 2 and 3 show predicted ICA PSV and ICA/CCA PSV ratios from the theoretical resistance model. In comparison to the observed data, the patterns of results are congruent. The artificial elevation in PSV becomes more pronounced as the arteries approach bilateral severe stenoses.

**Discussion**

In the present study both the clinical and theoretical results demonstrated that the ICA PSV was artificially elevated by the presence of a severely stenosed contralateral ICA. Data
from Figure 2 can be used to estimate the magnitude of this artificial elevation. It has been suggested that the ICA/CCA PSV ratio may reduce the effect on velocities. This does not appear to be the case. Since there was little increase in PSV for the CCA, a similar pattern of results was observed when the ICA/CCA PSV ratio was used. The artificial elevation will affect the correlation between PSV values alone and angiographically defined ICA stenosis, but whether this results in a change in treatment decision will depend on the ultrasound cut points that are used for assigning the degree of stenosis.

The present results are similar to a recently reported study of 38 patients that used a broader grouping of 60% to 99% stenosis determined by ultrasonography. This study found a significant mean decrease in PSV of 48 cm/s in the 60% to 99% stenosis group after a contralateral carotid endarterectomy. In another study of 85 patients, the mean PSF significantly decreased by 1175 Hz (approximately 35 cm/s). Although the decrease was averaged across all degrees of stenosis of the unoperated artery, it was observed from the reported regression line that the largest decrease in PSF occurred for the more severely stenotic arteries. A small, nonsignificant mean decrease of 213 Hz (approximately 6 cm/s) was reported in 26 patients. The decrease in PSF was averaged across all degrees of stenosis, and notably few arteries were severely stenosed. Cross-sectional studies, in which different patients with bilateral carotid disease were grouped together into different categories of stenosis severity, are not directly comparable to the before-after endarterectomy studies. Nevertheless, the reported elevations of ultrasound velocity contralateral to a diseased artery are in general agreement.

An independently derived theoretical resistance model of the cerebral circulation corroborated the observed artificial velocity elevations. The theoretical results lend credence to the clinical findings being a genuine phenomenon rather than coincidental. In addition, the theoretical resistance model, detailed in the Appendix, can be used to estimate the PSV for each degree of stenosis in both arteries and the magnitude of artificial elevation. The model used resistances derived from flow values obtained from the literature, which were within reasonable limits of other studies. The theoretical results are surprisingly similar to the observed data given the many modeling assumptions that were made, such as steady blood flow, ignoring complex flow effects through stenosis, and including only the major arteries. A more complicated model could have been derived to account for some of these other factors, but it is unlikely that the findings of the present study would have changed.

A total of 113 patients had PSV estimated from their PSF measurements using the Doppler formula, which converts the unit of ultrasound measurement from frequency to velocity. In this study the primary outcome was the difference between the before and after endarterectomy measurements, and these results remained largely the same when the patients with only PSV measurements were analyzed as a separate group.

The ultrasound examinations in the participating NASCET centers were performed by trained ultrasonographers at the same laboratory using standard techniques at the time of the trial. These standard techniques included ultrasound measurements at an angle of insonation of approximately 60 degrees. While every attempt was made to conduct the ultrasonography examination at this angle, in some patients this may not have been possible because of factors such as body habitus. However, these factors should reasonably be the same between the before and after endarterectomy examinations.

In conclusion, there is a significant artificial elevation of ultrasound PSV in the presence of a contralateral severe ICA stenosis. The effect is greatest when both arteries have severe stenoses. Bilateral severe stenoses cannot be inferred from ultrasound PSV measurements alone.

Appendix

Details of the Theoretical Resistance Model

The equations governing the flow circuit are given by the following:

\[
(1) \quad Q_{24} + Q_{35} = Q_{12} \\
(2) \quad Q_{35} + Q_{46} = Q_{13} \\
(3) \quad Q_{45} + Q_{56} = Q_{24} \\
(4) \quad Q_{56} + Q_{65} = Q_{35} \\
(5) \quad Q_{15} + Q_{25} + Q_{35} + Q_{45} + Q_{56} = Q_{37} \\
(6) \quad R_{CCA} Q_{13} + (R_{IPS} + R_{ICA}) Q_{35} = R_{BA} Q_{15} \\
(7) \quad R_{CCA} Q_{13} + (R_{CON} + R_{ICA}) Q_{35} = R_{BA} Q_{15} \\
(8) \quad R_{ECA} Q_{24} + R_{COL} Q_{45} = (R_{IPS} + R_{ICA}) Q_{25} \\
(9) \quad R_{ECA} Q_{36} + R_{COL} Q_{56} = (R_{CON} + R_{ICA}) Q_{35} \\
(10) \quad R_{COL} Q_{45} + R_{BA} Q_{35} = R_{EXT} Q_{17} \\
(11) \quad R_{COL} Q_{45} + R_{BA} Q_{35} = R_{EXT} Q_{17} \\
(12) \quad R_{BA} Q_{15} + R_{BA} Q_{35} = P_{17}
\]

where \( R \) represents the resistance values; \( Q \), mean flow rates between nodes \( i \) and \( j \) in Figure 1; \( P \), total pressure drop across the circuit; IPS, ipsilateral; and CON, contralateral. Other abbreviations are defined in the Table.

The values for the constant (ie, nonstenosis) resistances were determined using median ICA, external carotid artery, and basilar artery flow rates for normal controls and patients with bilateral ICA occlusion. It was further assumed that there was no collateral flow in the normal controls and that flow to the external circulation was the same for both normal controls and patients. These flow rates were substituted into the above equations, which, when solved, yielded the resistance values given in the Table.

The resistances of the stenosis segments were determined from a combination of Poiseuille’s and Bernoulli’s laws, as follows:

<table>
<thead>
<tr>
<th>Resistance</th>
<th>Value (PRU*)</th>
<th>Corresponding Artery</th>
</tr>
</thead>
<tbody>
<tr>
<td>( R_{CCA} )</td>
<td>0.0187</td>
<td>CCA from aortic arch to bifurcation</td>
</tr>
<tr>
<td>( R_{ICA} )</td>
<td>0.0358</td>
<td>ICA past stenosis to circle of Willis</td>
</tr>
<tr>
<td>( R_{ECA} )</td>
<td>0.0498</td>
<td>External carotid artery distal to bifurcation</td>
</tr>
<tr>
<td>( R_{BA} )</td>
<td>0.128</td>
<td>Vertebral and basilar arteries</td>
</tr>
<tr>
<td>( R_{SC} )</td>
<td>0.166</td>
<td>Brain circulation</td>
</tr>
<tr>
<td>( R_{BA} )</td>
<td>0.385</td>
<td>Ophthalmic arteries</td>
</tr>
<tr>
<td>( R_{EXT} )</td>
<td>0.581</td>
<td>External circulation</td>
</tr>
</tbody>
</table>

*1 PRU=1 mm Hg/mL/min.
where $D_{IPS}$ and $D_{CON}$ are the diameters of the ipsilateral and contralateral stenoses, $D_{ICA}$ is the diameter of the normal distal ICA, $L$ is the stenosis length, $\mu$ is the blood viscosity, and $\rho$ is the blood density.

The normal distal ICA diameter, $D_{ICA}$, was set to 5.2 mm, and the stenosis length, $L$, was equal to $D_{ICA}$. Although stenosis length varies with severity of stenosis, for the purpose of model simplicity it was assumed to be constant. Additionally, the value is approximately the same as in a previous study. The blood viscosity and density were set to 4 centipoise and 1.06 g/cm$^3$, respectively.

With the use of these resistance values in combination with those from the Table and an assumed pressure drop across the circuit of 100 mm Hg, the mean flow rates in the arteries in the circuit model could be calculated for any combination of ipsilateral and contralateral stenosis severity by solving Equations 1 to 12. Owing to the dependence of stenosis resistances on the ICA flow rates, these equations were solved iteratively until the computed flow rates differed by $<0.01$ mL/min.

The PSV in a given vessel was calculated by the following formula:

$$PSV = k f Q / A$$

where $k$ is the ratio of peak to mean velocity at peak systole, $f$ is the ratio of peak systolic to mean flow rate, $Q$ is the mean flow rate in the vessel, and $A$ is the arterial lumen area.

It was assumed that the peak systolic flow rate was twice the mean flow rate (ie, $f=2$). Owing to the anticipated presence of a nearly blunt velocity profile in both the CCA and stenosis throat at peak systole, a peak mean velocity ratio of 1 (ie, $k=1$) was also assumed, in which case PSV was computed as twice the mean flow rate divided by the arterial cross-sectional area. For the ICA, the cross-sectional area was computed from the stenosis diameter. For the CCA, the diameter was assumed to be constant at 8 mm.

Acknowledgments

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References

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