Interpretation of carotid and vertebral ultrasound

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ICA Doppler Criterion, Neurosonology Lab, WFU

<table>
<thead>
<tr>
<th>% Stenosis</th>
<th>Systolic V</th>
<th>Diastolic V</th>
<th>ICA/CCA</th>
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<tbody>
<tr>
<td>&lt; 50</td>
<td>&lt; 140 cm/s</td>
<td>&lt; 40 cm/s</td>
<td>&lt; 2.0</td>
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<tr>
<td>50 – 74</td>
<td>&gt; 140 cm/s</td>
<td>&lt; 110 cm/s</td>
<td>2.1 – 2.9</td>
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<tr>
<td>75 – 95</td>
<td>&gt; 140 cm/s</td>
<td>&gt; 110 cm/s</td>
<td>&gt; 2.9</td>
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<tr>
<td>&gt; 95</td>
<td>Variable</td>
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ECA Doppler Criterion, Neurosonology Lab WFU

<table>
<thead>
<tr>
<th>% Stenosis</th>
<th>&lt; 75% ICA stenosis</th>
<th>&gt; 75% ICA stenosis</th>
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<tbody>
<tr>
<td>&lt; 50</td>
<td>&lt; 140 cm/s</td>
<td>&lt; 190 cm/s</td>
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<tr>
<td>&gt; 50</td>
<td>&gt; 140 cm/s</td>
<td>&gt; 190 cm/s</td>
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CAROTID DUPLEX DATA

II. B-Mode Imaging

III. Additional Findings

- Spectral Broadening
- Post-stenotic Turbulence
- Collateral Circulation
- Volume Flow Rate
- TCD Findings
Consensus Panel Gray-Scale and Doppler US Criteria for Diagnosis of ICA stenosis

<table>
<thead>
<tr>
<th>Degree of</th>
<th>Primary Parameters</th>
<th>Additional Parameters</th>
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<tbody>
<tr>
<td>Stenosis</td>
<td>ICA PSV (cm/sec)</td>
<td>Plaque Estimate (%)</td>
</tr>
<tr>
<td>Normal</td>
<td>&lt; 125</td>
<td>None</td>
</tr>
<tr>
<td>&lt; 50</td>
<td>&lt; 125</td>
<td>&lt; 50</td>
</tr>
<tr>
<td>50–69</td>
<td>125–230</td>
<td>≥ 50</td>
</tr>
<tr>
<td>&gt; 70 but less than near occlusion</td>
<td>&gt; 230</td>
<td>≥ 50</td>
</tr>
<tr>
<td>Near Occlusion</td>
<td>High, low, or undetectable</td>
<td>Visible</td>
</tr>
<tr>
<td>Total Occlusion</td>
<td>Undetectable</td>
<td>Visible, no detectable lumen</td>
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* Plaque estimate: diameter reduction with gray-scale and color Doppler US

Plaque Features Descriptors / Parameters

- Location: Specific vessel, Segments involved
- Surface Features: Smooth, Irregular, Crater/Ulcer/Niche
- Texture/Composition: Homogeneous, Heterogeneous, Mixed, Possible Intraplaque hemorrhage
- Echodensity: Hypoechoic, Echogenic, Hyperechoic
  - Dense with or without acoustic shadowing
- Plaque Motion: Radial, Longitudinal

Plaque Size Descriptor Criterion

<table>
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<tr>
<th>Plaque Descriptor</th>
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<tr>
<td>Normal / Wall Thickness</td>
<td>&lt; 1.1 mm</td>
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<tr>
<td>Minimal / Mild</td>
<td>1.1 – 2.0 mm</td>
</tr>
<tr>
<td>Moderate</td>
<td>2.1 – 4.0 mm</td>
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<tr>
<td>Large / Severe</td>
<td>&gt; 4.0 mm</td>
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Graph that demonstrates that volume flow will decrease during a Grade II & III stenosis (70% occlusion), as flow velocity first spikes before dropping during a Grade IV stenosis (90% occlusion).
Waveforms
Direct Effect

1. Acute elevation of blood flow velocities
2. Flow disturbances
3. Decreased blood flow

Indirect effect

- Downstream
  - Flow disturbances
  - Damping waves
  - Low flow acceleration
- Upstream
  - Decreased velocity
  - Increased pulsatility
DDx between ICA and ECA

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<th></th>
<th>ICA</th>
<th>ECA</th>
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<tr>
<td>Wave form</td>
<td>Low resistance</td>
<td>High resistance</td>
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<tr>
<td>Caliber</td>
<td>Larger</td>
<td>Smaller</td>
</tr>
<tr>
<td>Branch</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Temporal pulsation</td>
<td>No response</td>
<td>Response</td>
</tr>
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</table>

Principles of Doppler ultrasound

\[ V = \frac{(F_d \times C)}{(2f_t \times \cos \theta)} \]
Diagnosis
Quantitation of Carotid Stenosis with Continuous-Wave (C-W) Doppler Ultrasound

MERRILL P. SPENCER, M.D. AND JOHN M. REID, PH.D.

SUMMARY  Two methods for determining the degree of stenoses developing on the origin of the internal carotid were tested using non-invasive Doppler ultrasonic imaging (DOPSCAN) of the carotid bifurcations. Spectral analysis of Doppler audio recordings was utilized in determining the maximum frequencies found within the stenosis, as well as the ratio of the frequency downstream to the stenosis, to the frequency within the stenosis. The theoretical relationships between blood flow, velocity, and pressure drop are defined for all grades of stenosis and they predict that carotid flow will not be reduced unless the lumen diameter is less than 1.5 mm. At critical diameter reductions, below 1 mm, the frequencies in human carotids do not exceed 16 KHz because turbulence limits peak velocities. If the maximum systolic frequency exceeds 5 KHz, when 5 MHz probes are directed at a 30° angle from the body axis, there is always present stenosis up to diameters of less than 3.5 mm by x-ray angiographic measurements. Frequency ratio studies confirm that plaque growth is not symmetrical but they did not improve x-ray angiography correlations because of the limitations of x-ray in measuring cross sectional areas from projection films and limitations of the spot size of x-ray tubes.

THE ADVERSE CLINICAL EFFECTS of atherosclerotic plaques on the carotid artery are manifest in the patient’s eye and brain through reduction of blood perfusion following stenosis of the channel or by embolization from the site of the plaque. It is generally agreed that more than one-third of strokes result from cervical arterial disease and primarily from plaques occurring on the origin of the internal carotid artery. For stroke prevention the identification of carotid plaques and quantitation of stenosis is of primary importance.

Non-invasive diagnostic methods are needed to evaluate patients with symptoms of cerebrovascular insufficiency because of the inherent dangers and costs of the alternative, x-ray contrast angiography. In addition, they are needed for medical or surgical follow up in the study of the natural history of the atherosclerotic plaque. With the general availability of non-invasive Doppler ultrasound, which provides blood velocity signals from the carotid arteries, it is important to fully utilize this information to evaluate the degree of stenosis and the attendant collateral circulation. This paper presents a system for determining the degree of stenosis using the increased Doppler audio frequencies within the stenotic segment.

Methods

Carotid blood velocity was measured with 5 MHz continuous-wave (C-W) directional Doppler ultrasonic equipment designed and built in the Bioengineering Center of this Institute. The ultrasonic probe consists of a dual-crystal lens-focusing transducer mounted on a position sensing arm and directed toward the carotid arteries at a 60° angle from the body axis. With this equipment and procedure, a 1 KHz Doppler frequency shift represents a blood velocity of 30 cm/sec. The probe is placed against the neck with intervening coupling jelly and the Doppler shifted frequencies are recorded. A Doppler image of the carotid bifurcation (DOPSCAN), including the common carotid and its external and internal...
branches, is developed. Magnetic tape recordings are made of selected audio signals for later spectral analysis. Diameters calculated from Doppler findings were compared with the minimal diameter measurements found on x-ray contrast angiographic films.

Three methods were tested to determine the lumen cross section at the origin of the internal carotid artery. All methods used spectral analysis of Doppler signals to determine the maximum systolic frequency ($f_{max}$), (fig. 1). Though the mean frequency ($f$) is preferable because it represents the mean velocity ($v$), $f_{max}$ is substituted because it can be more accurately determined than $f$ which must be derived from the zero crossing meter. Though zero crossing meters are available, their accuracy in determining $f$ is questionable.

The first method tried, and the simplest to perform, utilized only the greatest frequency found at the site of the stenosis, $f_{max}$. The other 2 methods utilized the frequency ratio between the internal carotid signals found at the angle of the jaw $f_{max}$, downstream to the origin, and $f_{max}$ (fig. 2).

The theoretical basis for our first method was the concept that a decreasing cross sectional area within a stenotic segment would produce an increase in velocities and corresponding Doppler shifted frequencies. Theoretical model predictions were carried out to determine the maximum range of Doppler frequencies that might be expected with internal carotid stenosis. We assumed a linear relationship between resistance ($R$) and blood flow ($F$). $R$ was calculated in dyne-centimeter-seconds from the following equation:

$$R_{dy,cm, sec} = \frac{8\eta L}{\pi r^4}$$

Where $\eta$ represents the viscosity of the blood (nominal value of 0.04 Poise), $L$ represents the length of the stenotic segment (nominal value of 0.2 cm) and $r$ represents artery radius. $R$ is converted to clinical terms of mm Hg/ml/min by dividing by 79,380, and flow for any given stenosis was calculated from:

$$F = \Delta P/R$$

The mean velocity ($v$) was calculated from the following equation:

$$v = \frac{F}{60 \pi r^2}$$

The calculations considered a network model (fig. 3) in which the origin of the internal carotid artery was represented by a linear resistance $R_1$. We also compared both the relationship of the ratio $f_s/f_i$ and the $\sqrt{f_s/f_i}$ to the minimum x-ray diameter using the principle of continuity of flow in the unbranching internal carotid artery (fig. 4). The rationale for using $f_s/f_i$, without a square root function, is based on the concept of plaque development on one side of the artery lumen and growing across the artery lumen. The differences expected from symmetric and asymmetric stenosis are illustrated in figure 5. In order to test which of these assumptions was most correct the x-ray angiographic diameter at the origin of the internal carotid was compared with each frequency ratio method.

X-ray contrast angiographic films were analyzed and compared with Doppler frequencies from 95 internal carotid arteries from 64 patients, representing all usable studies by both methods in 2 Seattle vascular laboratories during one calendar year. The minimum diameter found at the origin of the internal carotid was measured with a micrometer utilizing all available films. If no stenosis was present the diameter was measured at a distance of 0.5 cm from the bifurcation to represent $D_1$. $D_2$ was measured 5-6 cm downstream.

From the best available films the minimum $D_1$ could not be measured with certainty within 0.5 mm, and greater uncertainty was often present. The least measureable x-ray diameter was found, using phantom wires, to be 0.8 mm and probably represented limitations caused by cathode spot size. The magnification ratio of the x-ray projections was found to vary, from 1.2 to 1.4 but adjustments were not made for this error in correlation considerations.

Results

Model Predictions

Figure 6 represents the theoretical relationships between $F$, $v$, and lumen diameter ($D$) as well as the expected mean Doppler frequency in KHz. Control flow was set at 300 ml/min, $P_t$ at 100 mm/Hg and brain resistance ($R_b$) and resistance of the collateral channels ($R_d$) were assumed to be equal at 0.333 PRU's. It is apparent, from the model data, that increasing degrees of axisymmetric stenosis will not diminish the blood flow through the artery below 10% of its control value until the diameter within the stenosis is less than 1.5 mm. During this early phase, termed Grade I stenosis, blood velocity and corresponding Doppler frequencies progressively increase in an exponential manner proportional to the inverse square of the diameter. Below a diameter of 1 mm a critical phase is reached when a small decrease in

![Figure 1. Doppler spectrum of frequencies (velocities) in the normal internal carotid.](image)
DOPSCAN spectra angiogram

**Figure 2.** DOPSCAN image of the carotid bifurcation in a patient with a "tight" stenosis of the internal carotid. Frequencies within the stenosis (f₁) are elevated while downstream frequencies (f₂) are decreased below normal.

**Figure 3.** Resistive model for internal carotid circulation to the brain. Normal flow through R₁ is also primarily through R₁ with a small amount through R₄.

Since: \( \bar{f}_1 A_1 = \bar{f}_2 A_2 \) and \( \bar{f}_1 D_1^2 = \bar{f}_2 D_2^2 \)

**Figure 4.** Rationale for calculating arterial stenosis from Doppler signals. \( D_1 \) represents the diameter at the origin of the internal; \( D_2 \) represents the downstream diameter; \( f_1 \) represents the mean Doppler frequency found within the stenotic segment on the origin of the internal carotid; and \( f_2 \) represents the mean Doppler frequency downstream to the origin.

Lumen diameter produces a great decrease in blood flow. This critical phase is termed Grade III stenosis. In Grade III stenosis, velocity reaches its greatest values but variations in collateral resistance around the stenosis greatly affects F and v. In Grades IV and V, velocities decrease again through the frequency range of Grades I and II and flow is greatly diminished to zero at occlusion.

**Figure 5.** The difference in relationship between diameter and cross sectional area for asymmetric and axisymmetric stenosis.
Carotid Diameters and Doppler Frequencies

The spectral distribution of frequencies representing blood velocities in the internal carotid arteries of a healthy subject, age 21, are seen in figure 1, where a concentration of energy near the maximum frequency edge ($f_{max}$) of the spectrum provides the normal "smooth" or "breezy" quality to the audio signal.

Figure 7 illustrates the relationship between $f_{max}$ and the x-ray minimal diameter in each of 95 human internal arteries. The horizontal lines represent greater than usual uncertainty of the x-ray measurements. For 77 diameters greater than 1.5 mm, $D_1 = 8.77 f_{max}^{0.67}$ with a coefficient of correlation of 0.74. The close correspondence of $f_{max}$ and $D_1$ to the inverse square relationship is apparent. Progressive deviation from the theoretical relationship develops progressively but becomes severe when the diameter decreases below 2 mm. No stenoses less than 0.5 mm were found on the films as predicted from the phantom measurements. The highest Doppler frequencies measured were 15–16 KHz and occurred in the diameter range of 0.75 to 2 mm.

Frequency Ratios

Figure 8 illustrates the first results obtained when we utilized the square root of the frequency ratio ($\sqrt{f_{max}}$). In this method, the downstream diameter $D_2$ is assumed to be 5 mm because a series of x-ray film measurements determined that this figure represented the median diameter of the internal carotid at the...
The most important problem with x-ray resolvability of stenotic lesions is its inability to represent the cross sectional area of a stenotic segment. Because plaques do develop on one side of the artery and expand asymmetrically toward the axis, because the number of x-ray projections are limited and resolvability appears greater than 0.8 mm, the true cross sectional area of the lumen cannot be measured. The Doppler frequency, which is related to velocity, is, however, closely related to the cross sectional area as well as to volumetric flow. The differences between Doppler and x-ray may be expected on the basis of x-ray inaccuracies alone, and the final test of Doppler awaits a better standard for comparison.

Precision in measurement of carotid stenosis is probably only needed for the higher degrees of stenosis where blood velocities are low, resulting in dangers of large thromboembolisms. In this situation, Doppler may find its greatest role in stroke prevention measures. For stenoses greater than 70% (diameter 1.5 mm or less), predicted by a Doppler systolic frequency of 10 KHz or greater, Doppler provides a 63% sensitivity, 85% specificity, and an overall accuracy of 95%.

Discussion

The findings that the Doppler frequency ratio, rather than its square root, provides a better prediction of the least x-ray diameter, confirms the observations of both pathologists and radiographers that plaque development is, in fact, asymmetric. Though figure 5 illustrates the relationship between the cross sectional area and the least diameter in only one type of asymmetric stenosis, many variations in the form of asymmetry produce a similar effect and all differ from the axisymmetric case by lying closer to a linear relation than does the axisymmetric case.

Acknowledgment

This research was supported by the National Institutes of Health, Grant #HL 19341. We thank the Departments of Radiology at the Providence Medical Center and Northwest Hospital in Seattle for their cooperation. The skill of clinical physiology technicians, Sheryl Clark, Lou Granado, Dave Moseley, John O'Brien, and Karmann Titland is acknowledged. The special encouragement of Drs. Edwin C. Brockenbrough and George I. Thomas has greatly enhanced the quality of this study.

References

The Spencer’s Curve: Clinical Implications of a Classic Hemodynamic Model

Andrei V. Alexandrov, MD

ABSTRACT

Merrill P Spencer and John M Reid applied the Hagen-Poiseuille law, continuity principle, and cerebrovascular resistance to describe a theoretical model of the relationship between the flow velocity, flow volume, and decreasing size of the residual vessel lumen. The model was plotted in a graph that became widely known as the Spencer’s curve. Although derived for a smooth and axis-symmetric arterial stenosis of a short length in a segment with no bifurcations being perfused at stable arterial pressures and viscosity, this model represents a milestone in understanding cerebral hemodynamics with long-lasting practical and research implications. This review summarizes several hemodynamic principles that determine velocity and flow volume changes, explains how the model aids interpretation of cerebrovascular ultrasound studies, and describes its impact on clinical practice and research.

Key words: Hemodynamics, ultrasound, carotid, transcranial Doppler.

“A theory is a good theory if it satisfies two requirements: It must accurately describe a large class of observations on the basis of a model that contains a few arbitrary elements, and it must make definite predictions about the results of future observations.”

Stephen W. Hawking
A Brief History of Time

Introduction

Merrill P Spencer and John M Reid applied the Hagen-Poiseuille law, continuity principle, and cerebrovascular resistance to build a hypothetical flow model with a view to illustrate the relationship between arterial blood flow velocities, flow volume, and decreasing size of the residual lumen as it applies to the internal carotid artery (ICA). In 1979, they published a simple and clear graph (shown here in Fig 1) that has since been reproduced in many textbooks, including major textbooks on cerebrovascular ultrasound. This model has since been widely used for interpretation of cerebrovascular ultrasound studies as means of explaining the velocity behavior with various degrees of the ICA and, most recently, intracranial arterial stenoses.

This model, now known as the Spencer’s curve, represents a milestone in understanding cerebral hemodynamics that has long-lasting practical and research implications. This review summarizes several hemodynamic principles that determine velocity and flow volume changes, explains how the model aids interpretation of cerebrovascular ultrasound studies, and describes its impact on clinical practice and research.

Key Principles of Hemodynamics Reflected in the Curve

The Spencer’s curve is a polynomial curve of the third order since the predicted arterial blood flow velocity shows both linear and nonlinear components in its rise with a subsequent decrease to the zero level. This means that...
the peak systolic velocity (PSV) is inversely proportionate to several functions of the residual lumen diameter \(d\):

\[
\text{PSV} \sim \frac{1}{d + d^2 + d^3 \ldots}
\]

An explanation how the first and second powers of vessel diameter influence the velocity behavior is provided below. The fourth power of the vessel diameter \(d^4 = (2r)^4\), where \(r\) is the vessel radius, is also likely to play a role as it directly influences flow volume and resistance to flow as it will be shown below. However, it is the cubic function \(d^3\) that really explains the turn of the curve from the upslope down to the downslope with the most severe vessel narrowing. Higher powers of radius may also play a role but their contribution in construction of the model is practically negligible.

Spencer and Reid used a vessel with straight walls and no bifurcations in their model of an axis-symmetric and smooth-surface arterial stenosis. In this situation, the flow velocity and cross-sectional areas \(A\) are linked in the so-called continuity principle\(^2\):

\[
A_1 \times \text{PSV}_1 = A_2 \times \text{PSV}_2
\]

Since fluid is noncompressible and since the applied pressure remained the same in the Spencer and Reid model, the maximum stenotic \(\text{PSV}_2\) velocity increases by the amount inversely proportionate to the squared function of the residual vessel diameter:

\[
\text{PSV}_2 = \frac{A_1 \times \text{PSV}_1}{A_2}, \quad \text{or} \quad \text{PSV} \sim \frac{1}{\pi r^2}, \quad \text{or} \quad \frac{1}{d^2}.
\]

Hence, the prestenotic \(\text{PSV}_1\) velocity is not shown in the graph (Fig 1), but the graph contains the initial velocity value with 0 degree stenosis, or normal vessel patency. This could be used as a reference point (or range) in subsequent estimations of disease severity by the velocity changes. As discussed below, subsequent research showed that velocity ratios could complement absolute measurements of the maximum velocity despite the presence of bifurcations.

Flow acceleration begins at the stenosis entrance where the pressure energy of flow (ie, blood pressure) is...
converted into the kinetic energy of flow with increased velocities. This conversion of energy is described by the Bernoulli effect:

\[ P_1 - P_2 - \Delta P = \frac{1}{2 \rho} (V_1^2 - V_2^2) \]

where \( \rho \) is the density of the fluid that has not been commented on but presumably remained stable in the Spencer and Reid model.\(^1\) The velocity changes are therefore mainly driven by the arterial pressure (\( P \)) gradient and the size of the residual lumen. Assuming that arterial blood pressure remained constant across various degrees of the carotid stenosis, the model showed that the initial PSV increase compensated for the flow volume through the residual lumen (Fig 1), yet velocity should not be equated with flow volume even though both are driven by the pressure gradient. Further PSV increase with severe stenoses becomes insufficient, and the flow volume starts to decrease particularly with \( \geq 80\% \) stenosis (Fig 1). Hence, the commonly used term “hemodynamically significant” stenosis refers to a significant pressure or flow volume drop across the stenosis that prompts recruitment of collateral flow to compensate for this arterial lesion.

This flow volume decrease will occur particularly if the cerebrovascular resistance also does not decrease distal to a severe stenosis, as it was assumed for simplicity of the model. Notably, the flow volume per unit of time directly depends on the pressure difference described in the Hagen-Poiseuille law:

\[ \text{Flow volume} = \frac{\pi(p_1 - p_2) \eta^4}{8\pi L} \]

where \( p_1 \) is the pressure at the beginning and \( p_2 \) is the pressure at the end of the flow system, \( \eta \) is the radius of the lumen, \( \pi \) is a constant, \( \eta \) is the fluid viscosity, and \( L \) is the length or distance that flow has to travel between the pressure points.

In the Spencer and Reid model, no compensatory poststenotic vasodilation was introduced and the fluid viscosity as well as the length of the arterial stenosis also remained stable. Thus, an axis-symmetric, smooth-surface, presumably short-length and circular arterial narrowing produced, not surprisingly, a perfect correlation between the arterial flow velocity, flow volume, and increasing degree of the carotid stenosis under these controlled and ideal circumstances. The model, based on a few elements, was proposed to predict the arterial flow velocity behavior across the entire spectrum of carotid stenosis, and to derive diagnostic criteria for spectral Doppler ultrasound for grading the stenosis.\(^1\)

**Interpretation of Cerebrovascular Ultrasound Studies**

Application of hemodynamic principles to interpretation of vascular ultrasound studies is a complex task that requires careful clinical and pathophysiological considerations. In reality, most arterial stenoses are axisymmetric with irregular surface, and have variable lesion length and compliance of the vessel wall. The blood viscosity, pressure, and distal resistance also vary between patients and within an individual over time. Therefore, the Spencer’s curve could best serve as a guide rather than a source of actual velocity values for grading an arterial stenosis. In fact, Spencer and Reid applied this model in their own exploration of the predictive value of spectral Doppler measurements in 64 patients against cerebral angiography to identify the size of the residual lumen and percent arterial stenosis. They achieved good results for ultrasound prediction of the severe ICA disease in their laboratory.\(^1\) However, the actual frequency parameters found in their study and the proposed grades of an arterial stenosis were not directly adopted into practice at other laboratories since direct and angle-corrected ultrasound imaging methods were introduced\(^6\) and the need for further intralaboratory validation was emphasized (www.icavl.org). Many subsequent independent validation studies have been performed in this important clinical field laying foundation for the 2003 multidisciplinary consensus criteria.\(^7\)\(^8\)

However, the model reflected the general direction of hemodynamic changes with carotid disease and, as it will be shown below, it did survive the test of time as a basis to explain individual hemodynamic changes and to understand the results of clinical research.

To interpret any given blood flow velocity value, one must consider whether this velocity was found on the upslope or on the downslope, or “the other side” of the Spencer’s curve. For example, an abnormally elevated flow velocity is most likely to be found on the upslope of the Spencer’s curve, ie, within the 50% to 90% ICA diameter reduction range. How can one decide if a given velocity is abnormal? If there is a reference velocity value such as an unobstructed ICA before the stenosis or on the contralateral side, an arterial stenosis of about 50% diameter reduction will double the velocity value assumed normal for a particular patient. Since the ICA has a bulb that normally has low velocities and could be affected by an axisymmetric plaque, the common carotid artery (CCA) velocity and the ICA/CCA PSV ratios were introduced to compensate for this clinical uncertainty.\(^3\) Despite wide interindividual variations,\(^10\) the PSV itself remains the single best predictor of the stenosis\(^11\) since when it exceeds...
125 cm/sec, one can say with a great degree of certainty that this patient with an atheroma, free of abnormal systemic hemodynamic changes, has ≥50% ICA stenosis.\(^8,11\) Remarkably, these interindividual PSV variations\(^8\) generally follow the shape of the Spencer’s curve (see Graph in the 2003 Consensus criteria, ref. 8). The 2003 Consensus criteria identify ICA PSV, ICA/CCA PSV ratio, and ICA EDV as useful velocity parameters that should be used together with gray scale and color flow imaging findings to interpret carotid ultrasound studies.\(^8\) Additional factors that affect velocity measurements include angle correction and interlaboratory equipment/protocol differences.\(^8\)

However, elevated, “normal,” and decreased velocities can also be found on the “other side” of the Spencer’s curve, ie, with the so-called angiographic “string” signs or near-occlusions indicating most severe arterial stenoses. The differential diagnosis includes the use of the velocity ratios between the prestenotic and stenotic segments, ie, the ICA/CCA PSV ratios, velocity asymmetry between homologous segments on bilateral examinations, and spectral waveform analysis. For example, the ICA/CCA ratio of <4 identifies the range of <70% ICA stenoses that most often present with the PSV of <230 cm/sec.\(^11\) The same relatively low velocities can be found with evidence of a significant impedance to flow such as ICA/CCA ratio greater than 4, the poststenotic blunting of arterial waveforms and signs of flow diversion or collateralization.\(^4\) The latter will likely show higher contralateral velocities and lower ratios implying compensatory flow. Flow velocities must be interpreted in the context of waveform appearance, flow velocity ratios, and putative determinants of flow velocity, if available to the interpreter.

In clinical practice, this helps to identify hemodynamically significant stenoses that may have variable velocities but are likely to be on the “other side” of the Spencer’s curve. The multidisciplinary consensus criteria identify these very severe lesions as having variable velocities,\(^8\) and application of the Spencer’s curve principle in conjunction with other hemodynamic and imaging considerations helps to sort out these changes.

### The Spencer’s Curve and Research Studies

Application of a nonstandardized ultrason sound screening in a large scale multicenter clinical trial led to one of the most frustrating observations\(^15\) fortunately overshadowed by other rigorously designed and successful studies.\(^10–12,14–16\) Nevertheless, if one expects a suboptimal performance of ultrasound screening for carotid stenosis, plotting velocities vs. percent stenosis should result in somewhat random scatter. During the North American Symptomatic Carotid Endarterectomy Trial (NASCET), nonstandardized PSV values from over 1100 patients seen at almost 50 centers were plotted against percent diameter reduction of the ICA on catheter angiography.\(^13\) Figure 2A shows this scatter-plot that remarkably reveals a nonrandom behavior of the PSV. Almost all measurements fit under the area under a hypothetical Spencer’s curve! This happened because different sonographers used different machines at the study centers. Each lab has its own diagnostic criteria with variable local velocity cutoffs leading to individual Spencer’s curves of different amplitude (Fig 2B). But when these individual measurements were summed up together, the resulting scatter clearly followed the basic hemodynamic model. Since this is a post hoc interpretation, NASCET data set offers only indirect support to the Spencer’s curve. Remarkably, this information present in the scatter-plot escaped attention of the NASCET coordinating center investigators. An editorial that accompanied their report identified other significant flaws and biases.\(^17\)

This observation,\(^13\) along with critical analysis of the velocity behavior by Grant et al.\(^10\) pointed out that a single velocity parameter may not perform equally well between patients or laboratories, and that a local validation study is required at any given laboratory to use any selected diagnostic criteria with any confidence in their performance.

To enable clinical researchers to reliably screen for a significant carotid artery disease, the Asymptomatic Carotid Atherosclerosis Study trialists successfully employed a prospective multicenter standardization strategy.\(^11\) Their experience revealed that despite large interlaboratory variations, standardization and optimized noninvasive screening using various ultrasound methods and cutoffs is possible.\(^14\) Evaluation of the on-site performance was done using locally validated diagnostic criteria that take into account both the hemodynamic prediction and specific instruments that produce flow velocity measurements.

### The Clinical Impact

Self-validation of locally adopted diagnostic criteria specific to laboratory personnel and particular equipment, continuing education, and quality control (as laboratory performance can change over time) are paramount components of successful practice of cerebrovascular ultrasound (www.icavl.org). The Spencer’s curve, whether it is being called that or not during any specific lecture, became an excellent teaching tool for both the beginners and advanced ultrasound users. This hemodynamic...
model is evoked in one aspect or another to interpret individual results in everyday practice, and to understand the laboratory performance through self-validation studies. Implications of this model are now being discussed in transcranial Doppler studies of intracranial vessels, with particular emphasis on grading intracranial stenosis including diffuse lesions. It is difficult to understate its importance as it should be an integral part of any textbook, educational course, or quality assessment whenever we deal with applied principles of hemodynamics.

References