Vertebral Artery Doppler Waveform Changes Indicating Subclavian Steal Physiology

OBJECTIVE. The goal of this study was to characterize and classify changes in antegrade vertebral artery waveforms that may represent the early stages of subclavian steal physiology.

SUBJECTS AND METHODS. A prospective examination of waveforms from 1914 vertebral arteries produced a total of 40 that had a transient sharp decline in velocities at mid or late systole. In these patients, an ECG tracing was synchronized with the pulsed Doppler waveform, and reactive hyperemia was induced in the ipsilateral arm with a blood pressure cuff. The same protocol was performed in a control group of 52 patients with normal vertebral artery waveforms. Correlation between the waveforms and subclavian disease shown on angiography was made in 10 cases collected from the prospective study and in an additional 10 cases identified from a record search.

RESULTS. Four prototypic waveforms were identified on the basis of the degree of flow deceleration in mid systole. Flow velocity at the nadir of the mid systolic notch was greater than that of the end diastole for type 1 waveforms, equal to the end diastole for type 2, at the baseline for type 3, and below the baseline for type 4. The blood pressure cuff maneuver induced a change to more abnormal waveforms in 36 of 40 patients but did not change the waveforms of the control group. The correlation between waveform type and subclavian disease was statistically significant (p = 0.03).

CONCLUSION. Identifiable changes in the pulse contour of antegrade vertebral artery waveforms seem to represent the early stages of subclavian steal physiology. These changes can be organized into waveform types that indicate increasingly abnormal hemodynamics.
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in the left vertebral artery in 29 patients and in the right vertebral artery in 11 patients. Vertebral arteries with waveforms showing retrograde flow throughout systole were excluded.

Of the 40 patients, 26 were women and 14 were men (age range, 48–82 years old; mean age, 66 years).

Indications for the carotid Doppler sonography included coronary artery disease (n = 10 patients), transient ischemic attacks (n = 8), cerebrovascular accident (n = 3), syncope (n = 2), visual impairment (n = 2), carotid bruits (n = 2), and miscellaneous other reasons such as evaluation before surgery, peripheral vascular disease, fatigue, headaches, hypertension, and altered mental status (n = 13). Only five patients had symptoms referable to the vertebrobasilar circulation. Of the five, two patients complained of arm weakness, one of arm claudication, and two of syncope.

Patients were examined in a supine position. Bilateral carotid sonography was performed using standard sonographic equipment (HDI 3000; Advanced Technology Laboratories, Bothell, WA) and multifrequency (7–4 MHz) linear transducers. After gray-scale and color Doppler imaging of the carotid and vertebral arteries, representative Doppler spectral waveforms were obtained in the internal carotid artery, external carotid artery, common carotid artery, and vertebral artery. Sampling the vertebral arteries was performed in the mid portion of the extracranial segment of the artery. The measured angle of insonation was less than 60° in all cases.

ECG tracings were synchronized with the pulsed Doppler waveform in all 40 patients with an abnormal vertebral artery waveform. The ECG tracing was displayed on the same image as the pulsed Doppler recording and recorded on film. Reactive hyperemia was subsequently induced in the ipsilateral arm by cuff inflation and immediately after deflation. ECG tracings were synchronized with the pulsed Doppler tracing. The result was that antegrade flow in the vertebral artery was restored in diastole for waveform types 1–3 and preceded for type 4 waveforms (Fig. 1), equal to the level of end diastole for type 2 waveforms (Fig. 2), at the level of the baseline (0 cm/sec) for type 3 waveforms (Fig. 3), and below the baseline for type 4 waveforms (Fig. 4). The flow direction was entirely antegrade in the vertebral artery for waveform types 1–3 and predominantly antegrade for type 4. Comparison with the ECG tracing showed that antegrade flow in the vertebral artery was restored in diastole for all waveform types.

The prospective collection of 40 patients comprised eight (20%) type 1 waveforms, 16 (40%) type 2 waveforms, five (12.5%) type 3 waveforms, and 11 (27.5%) type 4 waveforms.

Angiographic Correlation

Of the 40 patients prospectively identified with abnormal antegrade waveforms, 10 had undergone aortic arch angiography within 6 weeks of the Doppler study. To augment the number of patients with angiographic correlation, we reviewed the records of 1256 consecutive carotid Doppler examinations performed during a 2-year period just before the prospective study for reports of abnormal vertebral artery waveforms. An abnormal waveform in the vertebral artery was mentioned in 84 reports. When the images of these 84 studies were examined for evidence of mid systolic flow deceleration, 50 cases were found. Of these 50 cases, 10 patients (seven women, three men) had angiography performed within 6 weeks of the sonographic study. Seven cases involved the left vertebral artery; three involved the right.

Carotid angiography was performed with intraradial digital subtraction or standard cut-film techniques after catheterization of the aortic arch and selective carotid catheterization. Images of the subclavian and innominate arteries were retrospectively reviewed for evidence of stenosis by an investigator unaware of sonographic results. The percentage of stenosis was calculated by comparing the lumen diameter measured at the point of maximum stenosis to the diameter of a disease-free segment of the subclavian or innominate artery distal to the stenosis.

The composite group of 20 patients from the prospective and retrospective case collections contained two type 1 waveforms, 10 type 2 waveforms, three type 3 waveforms, and five type 4 waveforms. The relationship between the vertebral waveform type and the corresponding degree of stenosis of the subclavian or innominate arteries at angiography was assessed using Pearson’s correlation coefficient. All statistical studies were considered statistically significant at a p value of 0.05 or less.

Results

Four distinctive waveforms were identified. The unifying feature of the waveforms was an abrupt decline in flow velocity after the early systolic upstroke. This deceleration could be definitively located at mid systole by comparing the Doppler tracing with the synchronously recorded ECG tracing. The result was that within systole two peaks developed. The first was sharp; the second was blunt. Waveform types were defined by the depth of the mid systolic notch. The flow velocity at the nadir of the notch was greater than the flow velocity at end diastole for type 1 waveforms (Fig. 1), equal to the level of end diastole for type 2 waveforms (Fig. 2), at the level of the baseline (0 cm/sec) for type 3 waveforms (Fig. 3), and below the baseline for type 4 waveforms (Fig. 4). The flow direction was entirely antegrade in the vertebral artery for waveform types 1–3 and predominantly antegrade for type 4. Comparison with the ECG tracing showed that antegrade flow in the vertebral artery was restored in diastole for all waveform types.
Doppler Sonography of Subclavian Steal Physiology

Fig. 2.—Type 2 waveform in 53-year-old woman with coronary artery disease.
A. In this waveform, more pronounced and deeper cleft is evident between two systolic peaks. Nadir of this cleft reached velocity at or just below that of end diastole. Second systolic peak tends to be smaller and broader than corresponding peak of type 1 waveform.
B. Drawing delineates waveform changes in cardiac cycle. Outline resembles body profile of rabbit and is sometimes referred to as “bunny waveform.”

Fig. 3.—Type 3 waveform in 54-year-old man with angina.
A. Nadir of mid systolic cleft is at or below baseline, but rapid recovery of forward flow before diastole is shown.
B. Drawing shows waveform outline.

Fig. 4.—Type 4 waveform in 81-year-old man with coronary artery disease.
A. Nadir of mid systolic cleft falls well below baseline signifying greater reversal of flow during systole. Forward flow is restored in diastole.
B. Drawing indicates outline of pulse profile.
The blood pressure cuff maneuver caused changes in the waveform contour in 36 (90%) of these 40 vertebral arteries with abnormal antegrade waveforms (Table 1). When a change in waveform shape was elicited, the waveform after the provocative maneuver was always more abnormal (i.e., a higher waveform type), with a greater decline in mid systolic velocities (Figs. 5 and 6). The blood pressure cuff maneuver never caused a change to a lower waveform type. This finding suggests that the four waveform types represent steps in the progression of disease.

No waveform changes were observed in response to the provocative blood pressure maneuver in the control group. A statistically significant difference was found in the proportion of vertebral arteries that exhibited waveform changes in the control group as compared with the case group ($p < 0.0001$).

Angiography performed in the 20 patients showed evidence of atherosclerotic disease of the subclavian or innominate arteries on the same side as the abnormal vertebral arterial waveform in all patients. The mean diameter of the stenosis of the ipsilateral subclavian artery was 45% for the type 1 waveforms (SD, 7.1%); 53% for type 2 waveforms (SD, 21.5%); 72% for type 3 waveforms (SD, 10.4%); and 78% for type 4 waveforms (SD, 2.9%). The correlation coefficient between the increasing waveform type and the increasing degree of subclavian stenosis was 0.5 ($p = 0.03$), indicating a moderately good correlation. This correlation coefficient, calculated with individual values for waveform type and stenosis estimates, was not based on averages.

**Discussion**

In this study, we identified changes in the Doppler tracing of vertebral arteries with antegrade flow that appear to represent early manifestations of subclavian steal physiology. These Doppler findings are evident in patients who, for the most part, do not yet have vertebrobasilar symptoms. When a blood pressure cuff is used to exacerbate the abnormal hemodynamics of a subclavian stenosis, the waveform shape will be changed such that a hierarchy of waveform types can be constructed ranging from subtle changes in the pulse contour to a full subclavian steal. The waveform changes induced by the blood pressure cuff were found only in patients with the

**TABLE I**

Waveform Changes Evoked by the Reactive Hyperemia Maneuver

<table>
<thead>
<tr>
<th>Waveform Type</th>
<th>No. of Patients</th>
<th>Conversion</th>
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<tbody>
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<td>No.</td>
<td>To</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>8</td>
<td>6 Type 2</td>
<td>2</td>
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<td>16</td>
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<tr>
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<td>5</td>
<td>3 Type 4</td>
<td>1</td>
</tr>
<tr>
<td>4</td>
<td>11</td>
<td>8 Full steal</td>
<td>1</td>
</tr>
</tbody>
</table>

**Fig. 5.**—67-year-old woman with stroke in whom type 2 waveform converted to type 4.

**A.** Doppler tracing of left vertebral artery shows typical type 2 waveform. Note antegrade flow throughout cardiac cycle.

**B.** Doppler tracing obtained after deflation of blood pressure cuff shows transient flow reversal in mid systole.

**Fig. 6.**—82-year-old woman in whom type 4 waveform converted to full subclavian steal.

**A.** Doppler waveform shows transient flow reversal of type 4 waveform.

**B.** After deflation of blood pressure cuff, retrograde flow throughout cardiac cycle indicates full subclavian steal.
abnormal vertebral waveforms, not in the control group of patients with normal waveforms. The synchronous ECG tracing provided a reference by which the stages of the cardiac cycle could be defined and correlated with specific features of the waveform contour, both before and after the provocative maneuver.

The earliest manifestation of the subclavian steal physiology is a transient sharp deceleration of blood flow after the first systolic peak. The synchronous ECG tracing reveals that this deceleration occurs in mid systole, not early diastole as is seen with high-resistance triphasic waveforms of other peripheral arteries. The flow deceleration produces a notch in the pulse contour and gives rise to two systolic peaks: the first sharp, the second blunt and rounded. As the subclavian steal hemodynamics become increasingly abnormal, the systolic notch becomes more pronounced and the second systolic peak diminishes and broadens. The nadir of the notch becomes progressively lower until it reaches and eventually crosses the baseline. The reversal of flow during systole is at first minimal and transient but becomes increasingly more substantial until complete reversal of flow throughout the cardiac cycle is seen.

The physiologic explanation for these Doppler findings may be twofold: a decrease in pressure in which blood flow velocity abruptly increases, and a loss of energy in which 78% disturbed or turbulent flow is present [6]. By the Bernoulli equation, the potential energy and the kinetic energy of a hemodynamic system are inversely related. When the velocity of flow (kinetic energy) increases, the pressure (potential energy) decreases. When the flow velocity increases across a focal stenosis in the subclavian artery, the pressure in that vascular segment decreases and causes an abrupt decline in flow velocity in the vertebral branch. This version of the Venturi effect is familiar to any chemistry student who has drawn water from a precipitate by attaching a hose from the side port of a faucet with running water. The suction effect created by the running water is analogous to the pressure drop in a stenotic subclavian artery. This pressure drop will be greatest during peak systole, when the blood flow across the stenosis is fastest. A transient decline in flow velocity during peak systole in the vertebral branch occurs, and this decline is reflected in the prominent mid systolic notch found in the Doppler waveforms recorded there.

A systolic notch in the pulse contour of a vertebral artery is an uncommon finding. In our study, this notch was found in only 2% of vertebral arteries prospectively examined for this finding. A subtle systolic notch might be normal in some vertebral arteries. We found that two (25%) of eight type 1 waveforms did not change in response to reactive hyperemia. Perhaps then the conversion of a type 1 waveform to a type 2 waveform in response to physiologic maneuvers might distinguish an early manifestation of the subclavian steal phenomenon from a normal—though uncommon—mid systolic retraction.

Angiographic correlation indicates that the hemodynamic changes evident in the vertebral arteries on Doppler sonography are associated with the severity of disease in the subclavian and innominate arteries. A statistically significant correlation was discerned between the increasing waveform grade and increasing subclavian stenosis.

In summary, this study shows a continuum of changes in the pulse contour of antegrade vertebral artery waveforms. This change seems to represent the earliest signs of subclavian steal physiology. These waveform changes occur before the development of frank retrograde flow and are manifest largely in the systolic portion of the cardiac cycle. The cardinal features of these waveform changes are the transient sharp decrease in blood flow velocity at peak systole. Recognition of these early changes could identify patients at risk for the eventual development of the subclavian steal syndrome. Long-term longitudinal follow-up of such patients may confirm the clinical evolution of the findings in this study.

Acknowledgment
We thank Susan Murray for assistance with manuscript preparation.

References