Hemodynamics of the Upper Extremities in Subclavian Steal Syndrome

By Margaret C. Conrad, Ph.D., James F. Toole, M.D.,
And Richard Janeway, M.D.

Since the initial clinical description of reversal of blood flow in the vertebral artery by Reivich, Holling, Roberts, and Toole in 1961,1 there has emerged an awareness of the importance of obstructive lesions of the proximal subclavian artery in the production of symptoms of cerebral ischemia. Since this is a form of cerebral vascular insufficiency readily improved or cured by surgery, it is imperative that the "steal" syndrome be suspected clinically and that appropriate diagnostic procedures be performed. Symptoms of brainstem ischemia coupled with brachial blood pressure inequality, subclavian bruit, and ipsilateral radial pulse delay as observed by Kesteloot and Houte,2 are the basic clinical criteria for selection of these patients for contrast procedures.

Experiments in which chronic reversal of vertebral flow was created in dogs3,4 suggested that plethysmographically recorded alterations in digital pulse contour, timing, and blood flow in the occluded extremity could be of diagnostic importance. The present studies, carried out on patients with symptomatic subclavian steal, amplify our previous findings in animals and demonstrate pulse delay and the decrease in pulse amplitude in the extremity distal to the subclavian occlusion. The clinically observed diminution or loss of radial pulse following exercise of the arm with subclavian stenosis or occlusion has been confirmed objectively. Furthermore, we have found a compensatory dilatation of cutaneous vascular bed on the side of the obstruction which results in flow on the side of the occlusion which is often greater than that on the normal side. In our experience, signs of brachial arterial obstruction in conjunction with digital flow equal to or greater than that in the opposite extremity are seen only with subclavian steal syndrome. Therefore we believe digital plethysmography to be a valuable screening test for patients with possible reversed vertebral artery flow (subclavian steal).

Methods

All studies were carried out with the patient in the supine position. Preoperative measurements were made in a temperature-controlled room maintained at 20 ± 0.2 °C. The patients were warmed with a blanket and two heating pads, and the extremities were exposed to room air. Plethysmographic cups were sealed to the third finger of each hand with "plethysmoseal" and occlusion cuffs were secured just proximal to the cup with "Velcro" tape. Digital volume changes were recorded on a Visicorder by means of venous-type Statham pressure transducers and Honeywell carrier amplifiers.

Pulses were recorded simultaneously in the two digits for evaluation of "pulse delay" and pulse contour. Flow was measured by venous occlusion plethysmography. Digital systolic artery pressure was evaluated by inflating the occlusion cuff to above systolic pressure and reducing the pressure by increments until blood flowed beneath the cuff as indicated by an increase in plethysmographically measured volume of the digit. Small vessel resistance was calculated as digital artery pressure divided by digital blood flow.

All of the patients were studied before surgical repair of the subclavian lesions. One patient was evaluated during surgical repair and three were studied again following subclavian reconstruction.

Case Reports

Case 1

NCBH 37-35-77. This 55-year-old man had a history of episodic vertigo recurring several times a week for a period of 3 months and claudication

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SUBCLAVIAN STEAL SYNDROME

Table 1
Comparison of Digital Hemodynamics in the Ipsilateral and Contralateral Extremities of Patients with Vertebral Reversal

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Conditions</th>
<th>Extremity</th>
<th>Flow (ml/min)</th>
<th>Flow/pulse (ml/min/ml)</th>
<th>Digital systolic press. (mm Hg)</th>
<th>Resistance (mm Hg/ml/min)</th>
<th>Foot-to-foot pulse delay (sec)</th>
<th>Peak-to-peak pulse delay (sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Preoperative</td>
<td>(normal) L</td>
<td>1.17</td>
<td>487</td>
<td>200</td>
<td>171</td>
<td>0.032</td>
<td>0.108</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(occluded) R</td>
<td>1.68</td>
<td>843</td>
<td>170</td>
<td>100</td>
<td>0.032</td>
<td>0.060</td>
</tr>
<tr>
<td></td>
<td>Preoperative</td>
<td>(normal) L</td>
<td>2.64</td>
<td>220</td>
<td>200</td>
<td>76</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>(occluded) R</td>
<td>3.30</td>
<td>970</td>
<td>170</td>
<td>51</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Preoperative</td>
<td>(normal) R</td>
<td>0.234</td>
<td>146</td>
<td>130</td>
<td>555</td>
<td>0.12</td>
<td>0.20</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(occluded) L</td>
<td>0.131</td>
<td>221</td>
<td>90</td>
<td>687</td>
<td>0.12</td>
<td>0.20</td>
</tr>
<tr>
<td></td>
<td>Postoperative</td>
<td>(normal) R</td>
<td>0.156</td>
<td>160</td>
<td>140</td>
<td>897</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>(occluded) L</td>
<td>0.096</td>
<td>140</td>
<td>100</td>
<td>1,042</td>
<td></td>
<td></td>
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<tr>
<td>3</td>
<td>Preoperative</td>
<td>(normal) R</td>
<td>1.58</td>
<td>210</td>
<td>180</td>
<td>114</td>
<td>0.04</td>
<td>0.07</td>
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<tr>
<td></td>
<td></td>
<td>(occluded) L</td>
<td>1.51</td>
<td>559</td>
<td>110</td>
<td>73</td>
<td>0.04</td>
<td>0.07</td>
</tr>
<tr>
<td></td>
<td>At surgery</td>
<td>(normal) R</td>
<td>0.300</td>
<td>160</td>
<td>90</td>
<td>300</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>pre-repair</td>
<td>(occluded) L</td>
<td>0.720</td>
<td>400</td>
<td>70</td>
<td>97</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>At surgery</td>
<td>(repaired) L</td>
<td>0.68</td>
<td>136</td>
<td>90</td>
<td>132</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Immed. after repair</td>
<td>R</td>
<td>0.246</td>
<td>153</td>
<td>50</td>
<td>203</td>
<td>0.20</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Two days after repair</td>
<td>(repaired) L</td>
<td>0.756</td>
<td>229</td>
<td>120</td>
<td>158</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Preoperative</td>
<td>(normal) R</td>
<td>0.585</td>
<td>278</td>
<td>190</td>
<td>324</td>
<td>0.014</td>
<td>0.090</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(occluded) L</td>
<td>0.810</td>
<td>405</td>
<td>140</td>
<td>173</td>
<td>0.014</td>
<td>0.090</td>
</tr>
<tr>
<td></td>
<td>Postoperative</td>
<td>(normal) R</td>
<td>0.645</td>
<td>184</td>
<td>130</td>
<td>201</td>
<td>0.014</td>
<td>0.090</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(repaired) L</td>
<td>0.690</td>
<td>197</td>
<td>135</td>
<td>195</td>
<td>0.014</td>
<td>0.090</td>
</tr>
</tbody>
</table>

of the right shoulder and arm of 2 months' duration. Physical examination revealed a systolic bruit at the base of the right carotid and in the area of the right subclavian artery. On the left, the brachial artery pressure was 180/110 and on the right 130/100 mm Hg. Cineangiographic studies showed that the aortic arch and the left subclavian, vertebral, and carotid arteries were normal. On the right the subclavian was partially obstructed at its origin, and flow was reversed in the right vertebral. The angiographic appearance of retrograde flow seemed to increase while the patient was exercising his right arm.

Studies

Plethysmographic measurements on two different occasions (table 1) revealed flow in the digit on the side of the subclavian stenosis to be higher than that on the normal side. Flow/pulse ratios were markedly elevated in the affected extremity. An increase in flow/pulse ratio (i.e., a decrease in the pulse amplitude relative to the flow) has been found to reflect the damping of the pulse as the blood flows via the smaller and longer collateral vessels around an occlusion. In this case the collateral channel would be the vertebral arteries. The digital artery pressures reflected brachial pressures and were significantly lower on the stenosed side. In both studies on this patient, the calculated small vessel resistance was lower distal to the occlusion than in the normal extremity, suggesting a dilatation of the skin bed in response to the occlusion that would facilitate a "steal" of blood from the brain by the extremity.

In a comparison of pulse contours (fig. 1), the delay of the peak of the pulse distal to the occlusion with respect to its foot is apparent, and the right pulse has a much more damped appearance than normal, probably due to the longer course and smaller vessels through which the blood is flowing. The foot of the abnormal pulse was only slightly delayed in time over that of the normal digital pulse, but the delay of the peak of the pulse was much greater (table 1).

Case 2

N CBH 38-46-84. This 67-year-old man had a history of sharp pain in his left arm for 9 months,
Figure 1
Normalized digital volume pulse contours in patients with subclavian steal syndrome and the effect of surgical reconstruction of the subclavian.

severe, predominantly left-sided occipital headaches, slurring of speech, a roaring in the left ear, diplopia, and falling. Physical examination revealed a brachial artery pressure of 140/86 on the right and 90/80 on the left, a decreased and delayed radial pulse on the left, and a coarse bruit at the base of the left side of the neck.

Cineangiography showed the innominate, right vertebral, and carotid arteries to be normal. The left subclavian was completely obstructed 1 cm. distal to its origin and blood flowed retrograde in the left vertebral artery (fig. 2).

The thrombus was removed from the subclavian and the artery was repaired with a Teflon patch graft. Following repair the pulse in the subclavian artery was good but no radial pulse could be palpated.

Studies
In this patient the plethysmographically measured flow was lower on the occluded side (table 1). After surgical repair of the subclavian artery, however, the digital flow was still low in spite of return of good pulses in the artery; also, the digital artery pressure was still low on the left in spite of a return of brachial artery pressure to normal. These results indicated a second more distal occlusion in the left extremity. Preoperatively the delays of the foot and peak of the left pulse were much more marked in this individual with two occlusions than in the other cases studied (and the pulse contour was more abnormal, the peak occurring quite late in the pulse cycle [fig. 1]).

Case 3
NCBH 39-47-42. The history included episodes of vertigo and unsteadiness for 3 months. Physical examination revealed a brachial artery pressure of 100/80 on the left and 160/80 on the right. There were bruits over both sides of the neck. Injection of the aortic arch showed all vessels to be patent except the left subclavian, which was occluded 1 cm. distal to its origin. Injection of the right vertebral resulted in immediate opacification of the left vertebral, left subclavian, and axillary arteries.

The occluding plaque was removed. The vessel was cleaned proximally and distally and repaired with a Dacron patch. Flow was re-established in

Figure 2
Reversal in flow in the left vertebral artery as demonstrated by cineangiography. A, dye flowing up right vertebral; B, filling of basilar system; C, dye passing retrograde in left vertebral; D, dye flowing to left arm via the subclavian; E, thrombus removed from the proximal subclavian artery. Depressions in the thrombus produced by pressure in the proximal subclavian on one side and reversed vertebral flow on the other.
the subclavian with an excellent pulse distal to the repair. Postoperatively the blood pressures were equal in the brachial arteries.

Studies
Preoperative digital flows were about equal in the right and left extremities in spite of a pressure difference between the right and left digital arteries of about 70 mm Hg (table 1). Flow/pulse ratios were increased on the occluded side. There was a delay in the left pulse and the left pulse contour was quite abnormal (fig. 1).

At operation, prior to subclavian endarterectomy, digital artery pressures were low and digital flows were lower than preoperatively. The flow on the normal side was decreased much more than on the occluded side due to a greater increase in small vessel tone in this extremity (table 1). Before removal of the plaque, the left vertebral artery was clamped and the left digital pulse decreased to less than a third of its preocclusion value (table 2), suggesting that two thirds of the flow in the left digit could have been supplied via the reversed flow in the left vertebral. Immediately following removal of the plaque in the subclavian artery, the pressure in the left digital artery rose to the level of that on the right, the flow/pulse ratio became normal (table 1) and the contour of the pulse changed toward normal, i.e., the peak of the pulse occurred sooner in the pulse cycle (fig. 1).

Two days after surgery the digital flow/pulse ratio and pressure were normal in the digits of the previously occluded extremity (table 1), and the digital pulse had now regained a notch (fig. 1). At this time the flow in the previously normal extremity was low, the digital artery pressure was 60 mm Hg lower than on the left and the right pulse was slightly delayed over that on the left, all of which indicated a newly developed occlusion in this extremity. This probably occurred as a result of the catheter being inserted into the right brachial artery for the purpose of angiography.

Table 2
Effect of Clamping the Vertebral Artery and Exercise of the Occluded Extremity on Digital Volume Pulse Amplitudes

<table>
<thead>
<tr>
<th>Case</th>
<th>Procedure</th>
<th>Left (occluded)</th>
<th>Right (normal)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Control</td>
<td>Experimental</td>
</tr>
<tr>
<td>3</td>
<td>Effect of clamping left vertebral on digital pulse amplitude</td>
<td>0.0022</td>
<td>0.0005</td>
</tr>
<tr>
<td>4</td>
<td>Effect of exercising left arm on digital pulse amplitude</td>
<td>0.0033</td>
<td>0.0009</td>
</tr>
</tbody>
</table>

Case 4
NCBH 15-13-64. This individual had a history of left-sided headaches since 1952, accompanied by tenderness of the left shoulder. Physical examination revealed a brachial artery pressure of 190/110 on the right and 130/100 on the left, a decrease in left radial pulse, a palpable pulse delay, and a bruit at the junction of the left subclavian and vertebral arteries. Exercise of the left arm resulted in a loss of palpable pulse. Angiography was performed under general anesthesia and consequently the arm could not be exercised at this time. Rapid reversal could not be demonstrated but oscillatory motion of the dye column was noted in the proximal vertebral, and the medium did not ascend the vertebral artery. Left subclavian endarterectomy was performed after which brachial artery pressures were equal bilaterally.

Studies
The preoperative plethysmographic studies followed the pattern described above, i.e., a higher digital flow on the occluded side in spite of a lower digital artery pressure, a high flow/pulse ratio distal to the occlusion, a damped-appearing pulse contour on the left and a small delay in the foot of the left pulse and a more marked delay in the peak (table 1, fig. 1).

The effect of exercise on the digital pulse was recorded. With exercise of the affected extremity, the pulse in the digit decreased to about one third of its preexercise value and there was a small decrease in the digital pulse amplitude of the opposite extremity (table 2) corroborating the clinical impression of loss of radial pulse after exercise of the arm.

Two days after subclavian endarterectomy, flows and arterial pressures were essentially equal in the two extremities, the left flow/pulse ratio had returned to normal and the pulse delay had disappeared (table 1). The contour of the left pulse had changed toward normal (fig. 1).

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Discussion

In all of these patients except for the individual who had a more distal occlusion in addition to that in the subclavian artery, the digital flow on the occluded side was equal or greater than that on the normal side. Since the digital artery pressure was consistently lower on the affected side, this increased flow would require a lower resistance or decreased tone of the small vessel bed distal to the occlusion. This dilatation of the peripheral vessels on the affected side has previously been seen in animals with experimentally produced vertebral reversal\(^3,4\) and apparently represents a chronic compensatory response to the occlusion. While protecting the extremity from nutritional deficiency, this mechanism would result in a greater shunting of blood away from the brain. Of course, only the digital or skin bed is studied here and it is essential to know the state of the vessels supplying the muscles under these same conditions before firm conclusion can be reached.

The increased flow/pulse ratio and the damped pulse contour are abnormalities seen with arterial occlusion at any site. We have noted these findings in conjunction with high flow in the digit distal to the occlusion only with vertebral reversal.

A pulse delay in the radial artery distal to the subclavian occlusion can almost always be detected in these patients by palpation. In all probability the palpable delays are a reflection of the delay in the peak of the pulse recorded plethysmographically and which was present in each of the patients studied.

The marked drop in the digital pulse amplitude with direct occlusion of the vertebral in which flow was reversed suggests that a very large proportion of the blood reaching the digit comes via the vertebral and a more minor proportion from local collateral channels in the area of the subclavian artery.

The decrease in palpable radial pulse with exercise in these patients is confirmed in the recorded changes in digital pulse with exercise of the extremity. The dilatation of the muscle bed apparently shunts blood away from the skin. The decrease in pulse in the digits of the normal resting extremity would be compatible with a decreased distal flow in this arm due to increased vertebral reversal to supply the exercising muscle.

Summary

Digital flow, digital artery systolic pressure, resistance, pulse volume, pulse contour, and delay between right and left pulses were evaluated in the upper extremities of four patients with the subclavian steal syndrome. Studies were made preoperatively in all patients and following surgical reconstruction of the subclavian in three cases.

Digital artery pressures were consistently lower on the side with the subclavian occlusion.

Flow on the occluded side was equal to or higher than that in the opposite extremity except when a second, more distal occlusion was present.

In each case there was a delay in the foot of the digital pulse on the occluded side and a more marked delay in the pulse peak. Distal to the occlusion the pulse amplitudes were decreased and the contours were abnormal.

The amplitude of the digital pulse was markedly decreased with exercise of the affected extremity.

The pulse delay and the disappearance of pulse in the radial artery during exercise previously described clinically has been confirmed by measurement.

Acknowledgment

The authors wish to thank Dr. L. B. Leinbach for permission to publish the cineangiograms and Mr. Harold Cook for their preparation.

References


Realdus Columbus and Michael Servetus

The question of priority between Realdus Columbus and Michael Servetus regarding the true passage of blood through the lungs and the change in color which takes place there has been discussed at great length. On this subject Bainton has wisely written: "Here, as so often in the history of Science, independent investigators came upon the same truth almost coincidentally. There can be no rivalry between Servetus and Columbus, the other contestant, for it has been established that neither knew the discovery of the other. Insofar as the announcement, there is no problem at all." Indeed, Servetus' text dates back to 1553, whereas Columbus' De re anatomica was published six years later. Furthermore, with regard to the time of the observation, Bainton in a short and searching paper has given convincing proof that a manuscript of Servetus in the Bibliothèque Nationale in Paris containing a statement on the pulmonary passage of blood is probably a faithful copy of an earlier draft sent in 1546, to Calvin, who never returned it.—ANDRÉ COURNAND, M.D. Circulation of the Blood. Edited by Alfred P. Fishman, M.D., and Dickinson W. Richards, M.D. New York, Oxford University Press, 1964, p. 24.
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