Subclavian Steal in Takayasu's Arteritis
A Hemodynamic Study by Means of Ultrasonic Doppler Flowmetry

SHOTARO YONEDA, M.D., TADAATSU NUKADA, M.D., KUNIHIKO TADA, M.D., MASATOSHI IMAIZUMI, M.D., TAKASHI TAKANO, M.D., AND HIROSHI ABE, M.D.

SUMMARY Blood flow in the vertebral artery and the upper extremity was studied in five cases of Takayasu's arteritis with subclavian steal by use of ultrasonic Doppler flowmetry and finger plethysmography.

The diagnosis of subclavian steal was made by observation of flow reversal in the vertebral artery on the subclavian steal side during grip exercise and, in addition, the vertebral flow change with brachial artery occlusion.

The blood flow increase of both internal carotid and non-affected (non-subclavian steal side) vertebral arteries during a common carotid compression was almost normal in patients with Takayasu's arteritis in this study.

During carotid compression on the side of the subclavian steal, ipsilateral vertebral blood flow greatly decreased, and the amplitude of the ipsilateral finger plethysmogram decreased slightly or moderately.

It is suggested that there are significantly important factors in suppressing symptoms of vertebrobasilar ischemia in these patients with Takayasu's arteritis with subclavian steal. These factors are believed to be (1) good function of the circle of Willis, (2) good blood supply to the brain stem, and (3) collateral circulation to the distal subclavian artery not via the vertebral artery.

THE SUBCLAVIAN steal syndrome consists of transient episodes of vertebrobasilar ischemia due to obstruction or severe stenosis of the proximal subclavian artery with reversal of direction of blood flow in the ipsilateral vertebral artery and stealing blood from the contralateral vertebral artery. Up to the present, angiography has been the only available method for diagnosis. Angiography is primarily a morphological rather than a functional assessment of the condition and is difficult to perform repeatedly.

Ultrasonic Doppler flowmetry is a non-invasive, reproducible, and semi-quantitative method which has been widely applied in medicine. In this study the ultrasonic Doppler technique was used to examine the hemodynamic change in vertebral arteries in subclavian steal from Takayasu's arteritis. The usefulness of the technique in the diagnosis of subclavian steal and the factors concerned with vertebrobasilar ischemia were studied.

Patients and Methods

Patients

The study included five female patients with Takayasu's arteritis with subclavian steal. None of the patients had complaints suggesting vertebrobasilar ischemia. The clinical data are summarized in table 1.

The criteria for subclavian steal were: (1) arteriographic evidence of subclavian or innominate artery occlusion or marked stenosis; (2) retrograde vertebral artery flow, as demonstrated by serial angiography; (3) patency of both vertebral arteries.

The subclavian arterial lesions responsible for the subclavian steal were occlusion in four patients and a severe stenosis in one patient. Lesions of Takayasu's arteritis of the affected (subclavian steal side) and non-affected (non-subclavian steal side) vertebral arteries (table 1) were seen in the arteriograms of Cases 1, 2, 3, and 5 (fig. 1, A, B, & C). The pathway for collateral circulation in the five patients was either through anastomoses between the external carotid artery and the costocervical trunk or through anastomoses between the two thyrocervical trunks via the two inferior thyroid arteries (fig. 1, A, B, & C).

Methods

A directional Doppler flowmeter (Hitachi, EUD-3B, 5MHz) and a real-time spectral ultrasonic rheograph (Hitachi, EUD-4, 5MHz) were used. The method of the directional Doppler flowmetry was previously reported in detail.

The real-time spectral ultrasonic rheograph recorded the velocity distribution of blood flow in real time. This ultrasonic rheograph consisted of a directional ultrasonic Doppler rheometer, a real-time frequency analyser, an analog computer and a recorder. The Doppler beat was sampled every 50 µs, and the analog data were converted to digital. The memory could store 256 bits of data. Since each bit contained the datum for 50 µs seconds, the memory could store the data for 12.8 m seconds. The data for 12.8 m seconds were divided into 128 equal parts in a range from 0 to 10 KHz and then analysed by the frequency analyser. During analyses one bit of processed datum was erased every 50 µs seconds, and another bit of new datum stored in its place. Thus, in every 50 µs seconds the analyser analysed the 256 bits of data which included one bit of newly stored datum.

The blood flow velocity curve from the directional Doppler flowmeter was integrated every 3 seconds. This value was employed to simplify the measurement of the blood flow change. The blood flow velocity, the integrated blood flow velocity, electrocardiogram and sometimes ear plethysmogram and finger plethysmogram were recorded simultaneously on a polygraph (Hitachi ECP-1).

The technique of vertebral flow detection used in this study has been described elsewhere. Briefly, in the supine position the patient's head was turned away from the side of the examination. A probe was placed beneath the posterior-inferior margin of the mastoid process and the ultrasonic beam directed medially and slightly superiorly.
### TABLE 1  Summary of Patient Data

<table>
<thead>
<tr>
<th>Case</th>
<th>Age</th>
<th>Sex</th>
<th>Subclavian steal side</th>
<th>Lesion of vertebral artery</th>
<th>Lesion of carotid artery*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Affected side</td>
<td>Non-affected side</td>
</tr>
<tr>
<td>1</td>
<td>55</td>
<td>F</td>
<td>Left</td>
<td>N</td>
<td>t ~ §</td>
</tr>
<tr>
<td>2</td>
<td>31</td>
<td>F</td>
<td>Right</td>
<td>t</td>
<td>N</td>
</tr>
<tr>
<td>3</td>
<td>34</td>
<td>F</td>
<td>Left</td>
<td>§</td>
<td>N</td>
</tr>
<tr>
<td>4</td>
<td>37</td>
<td>F</td>
<td>Left</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>5</td>
<td>41</td>
<td>F</td>
<td>Left</td>
<td>occlusion of distal portion</td>
<td>N</td>
</tr>
</tbody>
</table>

*Includes common and internal carotid considered as one unit; N : normal; t : mild stenosis; t : moderate stenosis; § : severe stenosis

---

**FIGURE 1.** (A, B and C). Case 3. Occlusion of proximal left subclavian artery. Reversal of flow in left vertebral artery. Severe stenosis of proximal left subclavian artery. Collateral connection between the two thyrocervical trunks and between the left external carotid artery and costocervical trunk contribute to supply to distal left subclavian artery.
266 STROKE VOL 8, No 2, MARCH-APRIL 1977

L-Vertebral Artery
Forward Flow
Reverse Flow
ECG
L-Brachial Arterial Flow Cessation

A.N. 55 TAKAYASU'S ARTERITIS

Figure 2. Blood flow velocity pattern of the left (affected) vertebral artery in Case 1. Note little forward flow during resting state and blood flow cessation of the brachial artery on the affected side.

Diagnostic Procedure

The measurement of blood flow velocity in the vertebral artery was performed at rest, with hand grip exercise and during blood flow cessation of the brachial artery by tourniquet. Hand exercise on the subclavian steal side was done for one minute at a frequency of ninety times per minute. Brachial blood flow was halted by inflation of a cuff to a suprasystolic pressure on the upper arm.

During digital compression of the ipsilateral common carotid artery, the contralateral internal carotid and both vertebral arterial blood flow changes were measured. Complete obstruction of the carotid artery was confirmed by a plateau wave of the ipsilateral ear plethysmogram recorded simultaneously. No significant change in blood pressure and heart rate was observed during carotid compression.

Results

Figure 2 shows the blood flow velocity pattern in the affected vertebral artery in Case 1 by the real-time ultrasonic Doppler flowmeter. During resting state and occlusion of the brachial artery on the affected side, little forward flow was detected. Similar results were found in the other cases.

The integrated blood flow velocity in the vertebral artery on the affected side, measured by the directional Doppler flowmeter, indicated reversed flow. On the other hand, the integrated blood flow velocity in the vertebral artery on the non-affected side presented the integrated forward flow velocity because of little reversed flow.

Figure 3 shows the blood flow velocity change in the affected vertebral artery in Case 2 using the directional Doppler flowmeter. The ipsilateral grip exercise caused an increase in vertebral flow velocity. During cessation of the brachial flow in the ipsilateral upper arm, the affected vertebral artery flow decreased abruptly; following deflation of the tourniquet the flow velocity increased. The vertebral blood flow on the non-affected side increased to some degree during grip exercise of the affected side. The vertebral arterial flow on the non-affected side decreased abruptly when flow was stopped in the brachial artery on the affected side (fig. 4). Similar results were obtained in the other four cases.

Table 2 shows that blood flow velocity increased in both internal carotid arteries and the vertebral artery on the non-affected side during carotid compression. Blood flow increased from 26 to 55% in the internal carotid arteries of four patients and from 36 to 62% in the vertebral arteries of three. Flow increases of the internal carotid and vertebral arteries in young controls were 44.5 ± 8.4 and 42.9 ± 6.5%. In the patients with Takayasu’s arteritis, the blood flow increase of the non-affected vertebral artery was the same as that in young healthy adults and in the internal carotid artery it was normal or subnormal.

Blood flow in the vertebral artery on the affected side during ipsilateral carotid compression was reduced from 45 to 93% (table 3, fig. 5). During carotid compression on the affected side, the amplitude of the ipsilateral finger plethysmogram decreased moderately in Case 1 (fig. 5) and to a slight degree in Cases 2 and 4.

Discussion

Grossman investigated the subclavian steal syndrome using a non-invasive ultrasonic Doppler technique and reported measurement of Doppler pulse wave of the brachial artery in cases with the syndrome and abnormal pulse delay of 0.03 to 0.06 seconds on the affected side.

In the present study, a direct transcutaneous Doppler recording was attempted from the vertebral artery. Flow in
both vertebrals is increased by grip exercise with the hand on the affected side. Vertebral flows are decreased by sudden occlusion of the brachial artery on the affected side (figs. 3 and 4). This change in vertebral flow is not seen in healthy subjects. The diagnosis of subclavian steal could be made by observation of such flow change in addition to vertebral artery reverse flow using Doppler flowmetry.

Conrad\textsuperscript{19} reported that in one case with subclavian steal syndrome, the ipsilateral digital pulse amplitude was reduced by one-third during vertebral artery clamping, indicating that a very large proportion of blood reaching the digit came via the vertebral artery.

Reports\textsuperscript{4, 14, 18} from arteriographic studies indicate that in subclavian steal the blood supply to the arm may depend on collateral pathways other than the vertebral artery. Newton\textsuperscript{14} reported that in some cases anastomoses between the two thyrocervical trunks via the inferior thyroid arteries and between the external carotid artery and thyrocervical trunk appeared to be the main collateral pathway to the distal subclavian artery. Baker\textsuperscript{2} showed the rich collateral pathway between the two thyrocervical trunks. North\textsuperscript{19} found that excellent collateral circulation came by way of the internal thoracic and inferior thyroid arteries. These functional collateral pathways may account for the failure of simple exercise to produce basilar artery insufficiency. Our results suggest that blood to the upper extremity on the affected side was supplied to a considerable extent through collateral pathways other than the vertebral artery. And during arm exercise stealing of blood from the cerebral circulation did not occur which would account, at least in part, for lack of vertebrobasilar ischemia symptoms in cases with Takayasu's arteritis. Because of early onset of Takayasu's arteritis, excellent collateral circulation may have developed.

During a carotid compression, the blood flow increases in the internal carotid and vertebral arteries in young healthy adults were 44.4 ± 8.4 and 42.9 ± 5.5%. Nornes\textsuperscript{19} believes that a contralateral internal carotid flow increase at occlusion in excess of 40–50% of the preocclusive level indicates sufficient intercarotid shunting capacity of the circle of Willis. Low contralateral internal carotid flow increase (14–17% of control) is a sign of poor intercarotid shunting capacity through the circle of Willis. In the five patients studied, the blood flow increase in the common carotid and vertebral arteries of 35–40% of the preocclusive level indicates sufficient shunting capacity through the circle of Willis. Bosniak\textsuperscript{4} and Handa\textsuperscript{10} suggested that anomalies of the circle of Willis might explain why some patients with subclavian steal were asymptomatic and others were not. Lord\textsuperscript{20} has reported that anomalous development of the circle of Willis contributed to the symptoms of patients with subclavian steal. The findings in patients with Takayasu's arteritis in this study supported the suggestion that one of the factors in vertebrobasilar ischemia was use of the circle of Willis as a collateral pathway.

In conclusion, the results of this study indicate that there are several factors suppressing symptoms of vertebrobasilar ischemia in patients with Takayasu's arteritis with subclavian steal. These are (1) good function of the circle of Willis, (2) good blood supply to the brain stem, (3) collateral circulation to the distal subclavian artery not via the vertebral artery.

The ultrasonic Doppler technique promises a simple non-invasive method for diagnosis and long-term and semi-quantitative observation of hemodynamic change in the subclavian steal.

### Table 2 Blood Flow Changes of Internal and Vertebral Arteries During Common Carotid Artery Compression

<table>
<thead>
<tr>
<th>Case</th>
<th>R-Internal carotid artery compression</th>
<th>L-Internal carotid artery compression</th>
<th>Right common carotid artery compression</th>
<th>Left common carotid artery compression</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>+30%</td>
<td>+30%</td>
<td>+48%</td>
<td>+62%</td>
</tr>
<tr>
<td>2</td>
<td>+48</td>
<td>+43</td>
<td>+48%</td>
<td>+50</td>
</tr>
<tr>
<td>3</td>
<td>+46</td>
<td>+50</td>
<td>+48%</td>
<td>+30</td>
</tr>
<tr>
<td>4</td>
<td>+26%</td>
<td>+55</td>
<td>+48%</td>
<td>+36</td>
</tr>
</tbody>
</table>

### Table 3 Blood Flow Change of Steal Sided (Affected) Vertebral Artery and Decrease of Amplitude of Steal Sided (Affected) Finger Plethysmogram During Ipsilateral Carotid Artery Compression

<table>
<thead>
<tr>
<th>Case</th>
<th>Blood flow change of vertebral artery</th>
<th>Decrease of amplitude of finger plethysmogram</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>-93%</td>
<td>moderate</td>
</tr>
<tr>
<td>2</td>
<td>-45</td>
<td>slight</td>
</tr>
<tr>
<td>4</td>
<td>-68</td>
<td>slight</td>
</tr>
</tbody>
</table>
L-Common Carotid Artery
Compression

L-Common Carotid Artery
Compression

Integrated L-Vertebral
Arterial
Blood Flow Velocity

L-Vertebral Arterial
Blood Flow Velocity

L-Finger Plethysmogram

Figure 5. Integrated left (affected) vertebral flow velocity and left vertebral flow changes and amplitude change of left finger plethysmogram in Case 1 during left common carotid compression. Note severe reduction of vertebral flow and moderate decrease of amplitude of finger plethysmogram during carotid compression.

References

Subclavian steal in Takayasu's arteritis. A hemodynamic study by means of ultrasonic Doppler flowmetry.
S Yoneda, T Nukada, K Tada, M Imaizumi and T Takano

Stroke. 1977;8:264-268
doi: 10.1161/01.STR.8.2.264

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/8/2/264