Short Communication

Transcranial Doppler Ultrasonography of Carotid–Basilar Collateral Circulation in Subclavian Steal

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The combination of a carotid–basilar and a vertebro–vertebral collateral circulation was verified directly in a patient with a complete subclavian steal by means of transcranial Doppler ultrasonography. The patient showed permanently reversed blood flow in the basilar artery. The subclavian steal influenced the hemodynamics of the circle of Willis at rest and during functional tests of the collaterally supplied arm. Our investigation provides the first direct experimental evidence of increased blood flow velocity in the carotid artery after decompressing the collaterally supplied arm. (Stroke 1988; 19:1036-1042)

Different forms of collateral pathways1-4 can be differentiated in patients with complete subclavian steal5-10 resulting from occlusion of the proximal subclavian or innominate artery (permanently reversed blood flow in the vertebral artery throughout the whole cardiac cycle). In the most common form of a vertebro–vertebral collateral circulation, the obstruction is bypassed by retrograde blood flow from the ipsilateral vertebral into the subclavian artery distal to the occlusion, and the reversed flow is fed by the contralateral vertebral artery. If the capacity of the contralateral vertebral artery is additionally reduced by stenosis or if this artery is occluded, a carotid–basilar collateral circulation with reversed basilar artery blood flow can develop. Furthermore, collateral circulation can arise from external carotid branches (occipital artery) that anastomose with muscular branches of the vertebral artery and with the thyrocervical and costocervical trunks. Only in innominate artery occlusion is there the possibility of a carotid–subclavian collateral circulation with reversed carotid and vertebral artery blood flow.

The majority of these collateral pathways can be detected by directional continuous-wave (CW) Doppler ultrasonography. With vertebro–vertebral collateral circulation, the Doppler pulse curve of the ipsilateral vertebral artery is characterized by an end-diastolic blood flow velocity near 0 due to the higher peripheral flow resistance in the arm vessels than in the cerebral vessels, whereas the contralateral vertebral artery shows a normal or slightly reduced end-diastolic flow velocity. Furthermore, reversed flow in the ipsilateral vertebral artery and blood supplied by the contralateral vertebral artery can be determined by functional tests (compression of the upper arm or closure of the fist). For the following reasons the diagnosis of a carotid–basilar collateral circulation by means of CW Doppler ultrasonography has until now been made by exclusion and not directly: first, the intracranial vessels are not accessible for direct examination with this technique; second, as far as we know, the changes of carotid hemodynamics that one would expect when the collaterally supplied arm is subjected to functional tests have not been detected by Doppler ultrasonography.11

We describe findings in a patient who initially appeared to have a vertebro–vertebral collateral pathway but who was later found by means of transcranial Doppler ultrasonography (TCD) to have a combination of carotid–basilar and vertebro–vertebral collateral circulation.

Case Report

During a routine physical examination, a 58-year-old male office worker was found to have no measurable blood pressure in his right arm while in his left arm the blood pressure measured 100/60 mm Hg. This difference had not been noticed before, and the patient had no subjective symptoms. He did report that on two earlier occasions, 5 and 19 years...
FIGURE 1. Transcranial Doppler ultrasonographic recordings of (a, b) left vertebral artery (VA L), (c, d) right vertebral artery (VA R), (e, f) both vertebral arteries before confluence (bidirectional signal), and (g–k) basilar artery (BA). Insonation from suboccipital region at rest, during compression (bar above Doppler recordings), and decompression (arrow) of upper arm supplied by collateral circulation. Symbols (at top right of each panel) indicate flow direction of Doppler signals in positive range of velocity scale in relation to probe (=> [, flow toward probe; <= [, flow away from probe). Cursor indicates peak systolic velocity for each trace. Orthograde perfusion of VA L (<= [), retrograde perfusion of VA R (=> [). Cardiac cycle-dependent alternating blood flow in initial part of BA (depth 80–85 mm); completely reversed blood flow in remaining course of BA at rest (depth 90–100 mm).

previously, he had experienced sudden short spells of unconsciousness, lasting a few seconds, which had occurred during periods of otherwise perfect well-being. A tentative diagnosis of subclavian obstruction was made, and the patient was referred for further diagnostic evaluation.

On physical examination, the radialis pulse was not palpable in his right wrist, nor was the blood pressure measurable. Blood pressure in his left arm was 120/70 mm Hg. Skin temperature was the same in both hands. Prolonged fist-clenching did not cause pain or fatigue. Further internal and neurologic examination findings were normal.

Electrocardiogram, chest x-ray, computed tomography, and electroencephalogram were normal. His only risk factor for vascular disease was a 16 pack-
A microprocessor-controlled pulsed Doppler device operating at 2 MHz (TC 2-64, EME, Überlingen, FRG) was used for the TCD examinations. Measurements were taken from selected areas at depths ranging from 25 to 155 mm at steps of 5 mm by a range-gated transducer. Pulse repetition frequencies were 6.8-18 kHz, depending on depth. Bidirectional signals were recorded with a 10-kHz low-pass filter and a 150-Hz high-pass filter. The emitted ultrasonic power varied between 10 and 100 mW/cm². The spectrum was analyzed with a fast-Fourier transformation and 64-point resolution. Spectral information was displayed either as kHz frequency shift or as velocity. The relation between Doppler frequency shift ($f$ in kHz) and flow velocity ($v$ in cm/sec) for measurements performed with an ultrasonic instrument operating at 2 MHz is $v = 39f$. It is not possible to determine the angle between the ultrasonic beam and the direction of the artery; however, if this angle is presumed to be <30°, the error, which is a function of the cosine of the angle, will be <15%. The TCD examination techniques have been described in detail.

The patient's extracranial vascular status was determined with a directional CW Doppler device.
FIGURE 3. Doppler ultrasonographic recordings of various segments of (a, c, e, f) right internal carotid artery (ICA R) and (b, d) left internal carotid artery (ICA L) during compression (bar above Doppler recordings) and decompression (arrow) of upper arm supplied by collateral circulation. (a, b) CI segment of ICA R or L with temporal placement of probe. (c, d) Extracranial part of ICA R or L with submandibular probe location. Initial part of ICA R examined with (e) continuous-wave Doppler ultrasonography and (f) duplex scanner. (=>, flow toward probe; <=, flow away from probe.) For further explanations see text.

(4 MHz emitting frequency; Delalande/Dyna, Paris, France). Additionally, the common carotid arteries and the carotid bifurcation were examined by duplex scan (CV 400, Diasonics, Milpitas, California).

The Doppler ultrasonographic findings of the vertebral arteries confirmed our suspicion of a subclavian steal. Figure 1, a–d, shows TCD recordings of the left and right vertebral arteries at depths of 60 mm by insonating from the suboccipital region. The left vertebral artery at rest is shown in Figure 1a. Compression of the right upper arm did not affect the flow signal of the left vertebral artery, whereas decompression was followed by an increase of both systolic and especially end-diastolic blood flow velocity (Figure 1b). Blood flow in the right vertebral artery was completely reversed with cessation of the end-diastolic blood flow (Figure 1c). Compressing the right upper arm led to a slight orthograde flow component during diastole. After releasing compression, there was a marked increase of systolic and end-diastolic blood flow velocity in the retrograde direction (Figure 1d).

Both the retrogradely perfused right vertebral artery and the orthogradely perfused left vertebral artery could be recorded in the same sample volume as a bidirectional signal at depths of 65–75 mm (Figure 1, e and f). Analogous to the anatomic variants described for the vertebrobasilar system,16,17 this finding indicated that the two vertebral arteries had closely adjacent, parallel courses at these sam-
ple volume depths. Although recent studies suggest that the confluence of the vertebral arteries is often at a greater depth, in the present case the transition to the basilar artery was recorded at a depth of 80 mm (Figure 1g) due to a clear differentiation from both of the vertebral arteries; a cardiac phase-dependent change in the direction of blood flow, with considerable retrograde flow during a part of systole and less orthograde flow in diastole, was found. At depths of >90 mm, a permanently retrograde perfusion with cessation of the end-diastolic blood flow (Figure 1i) could be recorded. Compression of the right upper arm again led to the small amount of orthograde flow in diastole (Figure 1, j and k). It was possible to trace the basilar artery to a depth of 100 mm. Decompressing the arm led to a marked increase of systolic and end-diastolic blood flow velocities in the reversed direction (Figure 1k).

In view of the hemodynamic findings in the basilar artery, the existence of a carotid–basilar collateral circulation in addition to the vertebr–vertebral collateral circulation was suspected. To prove this, it was necessary to investigate the hemodynamics in other arteries of the circle of Willis and its main branches. Figure 2 shows the TCD recordings (insonation from the temporal region) of the right anterior cerebral artery (ACA) (Figure 2a), the right middle cerebral artery (MCA) (Figure 2b), and the right posterior cerebral artery (PCA) (Figure 2, c–e). Neither compression nor decompression of the right upper arm resulted in any changes of the hemodynamics of the MCAs (M1 segment, depth 50 mm), the ACAs (A1 segment, depth 75 mm), or the P2 segment of the PCAs (depth 65 mm). However, blood flow in the P1 segment of the PCAs (depth 70 mm) was reversed (Figure 2c). Furthermore, the index of cerebral circulatory resistance (R) according to Pourcelot was markedly higher in the P1 segment (R = 0.74) than in the MCA (R = 0.56), the ACA (R = 0.52), and the P2 segment of the PCA (R = 0.50). R was calculated from the recorded flow patterns as $R = \frac{[\text{maximal systolic flow velocity} - \text{end-diastolic flow velocity}]}{\text{maximal systolic flow velocity}}$. Following release of compression of the right upper
arm, blood flow velocity in the P1 segment increased considerably in the retrograde direction (Figure 2d).

These findings led us to conclude that the increased blood flow in the P1 segment during functional testing was fed by the carotid arteries, and the Doppler ultrasonographic findings of the right and left internal carotid arteries (ICAs) verified this assumption. Various segments of the ICAs are shown in Figure 3. By placing the probe over the temples (depth 70 mm), it was possible to examine the CI segment of both ICAs (Figure 3, a and b). After decompressing the right upper arm, a slight increase of systolic blood flow velocity (e.g., from an average of 75 cm/sec to a maximum of 80 cm/sec in the right ICA) and a more marked increase during end-diastole (e.g., from an average of 35 cm/sec to a maximum of 48 cm/sec in the right ICA) was registered. We then detected a slight increase of diastolic blood flow velocity in the extracranial segments of both ICAs after arm decompression (Figure 3, c and d: submandibular position of the TCD probe; Figure 3, e and f: CW Doppler recordings and duplex scan of the initial segment of the right ICA).

An arterial digital subtraction angiogram (Figure 4) demonstrated an occlusion of the right proximal subclavian artery and a hypoplastic left vertebral artery.

Discussion

Our investigations yielded two essential facts:
1) TCD enabled the verification of a combined carotid–basilar and vertebro–vertebral collateral circulation, and
2) the hemodynamic changes occurred in the basilar artery, in various segments of the ICAs, and in other arteries of the circle of Willis at rest and during functional tests.

Initially, the combination of an orthograde perfusion of the left vertebral artery with its distinct end-diastolic blood flow velocity and marked decompression effect together with flow reversal in the right vertebral artery with missing end-diastolic blood flow seemed to prove an exclusive vertebro–vertebral collateral circulation. However, retrograde blood flow found in the basilar artery suggested the existence of further collateral pathways such as a carotid–basilar collateral circulation.

Changes of the Doppler pulse curve in the topographic course of the basilar artery both at rest and during functional tests allowed conclusions about existing pressure gradients in the vertebrobasilar system: a cardiac phase-dependent change in blood flow direction from reverse to forward still existed in the initial part of the basilar artery to a depth of 85 mm. Assuming that forward flow was the consequence of the amount of diastolic blood flow in the orthogradely perfused left vertebral artery, there was obviously a sort of watershed in the basilar artery at a depth of 85–90 mm. Compressing the right upper arm resulted in a shift of this watershed centrally due to the marked increase of peripheral blood flow resistance of the right collaterally supplied arm; correspondingly, forward flow during diastole could be observed in the basilar artery to a depth of 100 mm.

The TCD results showed that the hemodynamics of the MCA, the ACA, and the P2 segment of the PCA were not affected by the carotid–basilar collateral circulation either at rest or during functional tests.
tests. However, a marked increase of blood flow velocity in the PI segment of the PCA after decompression of the right upper arm was detected. In the present case, the PI segment exhibited reversed blood flow and supplied both the brain and the right arm. As a result of the greater peripheral blood flow resistance, a considerably higher R was observed in this segment than in the MCA, the ACA, and the P2 segment of the PCA.

The slight systolic and distinct end-diastolic blood flow velocity increase in the C1 segment and the cervical part of the ICA bilaterally could be taken as further evidence of a subclavian steal phenomenon via carotid–basilar collateral circulation. The existing collateral pathways leading from both ICAs to the right subclavian artery via the posterior communicating arteries, the PCAs, the basilar artery, and the right vertebral artery were deduced from blood flow directions, the various flow patterns, and the influence of functional tests on hemodynamics. They are shown schematically in Figure 5.

The ultrasonicographic findings pointed to an occlusion of the subclavian artery proximal to the origin of the right vertebral artery. Since no pathologic Doppler ultrasonicographic findings of the left vertebral artery could be detected with certainty, the exact reasons for the development of the combined vertebral–vertebral and carotid–basilar collateral circulation in the present case could not ultimately be determined by these findings alone. The development of both of the demonstrated collateral pathways became intelligible after a hypoplastic left vertebral artery was found by means of digital subtraction angiography (Figure 4).

In previous investigations concerning the frequency of various collateral pathways,4,21 the carotid–basilar collateral circulation was rare. In our ongoing study of the hemodynamics of the verteobasilar system in subclavian steal, the present patient was the only one of 52 investigated so far who exhibited permanently reversed blood flow in the PCA.

It is noteworthy that even long-lasting arm ischemia did not cause subjective symptoms or objective signs of verteobasilar insufficiency.22 There was no computed tomographic evidence of preceding diminished perfusion in the areas supplied by the ACA, the MCA, or the PCA or by the basilar artery.

It is often difficult to evaluate the hemodynamics of the basilar artery and other arteries of the circle of Willis by means of angiography under pathological conditions such as the different forms of collateral pathways in subclavian steal.23–25 TCD findings together with CW Doppler ultrasonography enable one to assess noninvasively the hemodynamic effects of a subclavian steal on arteries of the circle of Willis.

References


Key Words • subclavian steal syndrome • ultrasonics • collateral circulation
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