Eur Arch Psychiatr Neurol Sci (1987) 237:21–28

European Archives of Psychiatry and Neurological Sciences

Vertebral Artery Doppler Sonography

R. Winter, S. Biedert, Th. Staudacher, H. Betz, and R. Reuther

Neurologische Universitätsklinik Heidelberg, Voßstrasse 2, D-6900 Heidelberg, Federal Republic of Germany

Summary. We have examined the vertebral and subclavian arteries in 1,205 patients using directional continuous-wave (c-w) Doppler sonography, and compared the sonographic findings with the results of unilateral or bilateral retrograde brachial arteriographies in the same patients. Doppler sonography revealed 33 false positives among 909 cases with normal angiographic findings. Some types of vertebral artery (VA) lesions allowed an excellent, others a fairly good differentiation by Doppler sonography: the complete subclavian steal syndrome with constant reversal of VA flow was reliably detected (16 cases). In the incomplete steal syndrome (5 cases) sonography was superior to angiography. Two bilateral distal VA occlusions and seven basilar artery occlusions – six in the proximal third and one in the rostral third – were detected sonographically; four basilar occlusions sparing the caudal third and one case exhibiting rete mirabile anastomoses were not identified by Doppler sonography. Our acoustically defined sonographic criteria did not permit an unequivocal assignment to an anatomical variant or a vascular lesion. The sensitivity in the detection of a severe stenosis at the VA origin amounted to 16 out of 31, and to 12 of 25 in cases with a proximal VA occlusion and reconstitution of the distal VA through cervical collaterals. Our results confirm that the conventional hand-held c-w Doppler sonography cannot replace angiography in the evaluation of vertebrobasilar insufficiency. It rather serves as an aid to the decision for or against angiography, and in the follow-up of angiographically proven lesions. However, several therapeutically important lesions are readily diagnosed by sonography.

Offprint requests to: R. Winter

Key words: Ultrasonic Doppler – Vertebral artery – Basilar artery – Vertebro-basilar insufficiency

Introduction

Directional continuous-wave (c-w) Doppler sonography has become one of the most important techniques in non-invasive evaluation of the carotid arteries within the past 10 years. The non-invasive examination of the vertebro-basilar system has been restricted by the system's relatively inaccessible location and its considerable anatomical variability. Furthermore, therapeutic approaches have in the past mainly focused on the carotid artery. Until now, investigations on the validity of directional c-w Doppler sonography of the vertebral arteries (VAs) have been scarce. Therefore, we compared the sonographic findings with retrograde brachial angiograms done on the same patients in a large series.

Materials and methods

The present article refers to 1,205 patients, the majority of whom demonstrated typical symptoms of, or risk factors for, occlusive cerebro-vascular disease. However, many cases with non-specific symptoms suggestive of cerebral ischaemia were included, as well as patients prior to an angiographic investigation for a suspected mass lesion or a vascular malformation. The investigation of the brain-supplying extracranial arteries, including the subclavian arteries, by directional c-w Doppler sonography was part of their routine investigation.

Flow velocity of the VA at the atlas loop and in the supraclavicular fossa near its origin from the subclavian artery was recorded by means of directional c-w Doppler sonography. The device used consisted of a 4 MHz bidirectional Doppler (Dyna, licensed by Delalande) and a Siemens Cardirex 3T 3-channel cardiograph recording unit.

In the sonographic evaluation of the VAs, we employed the criteria developed by Büdingen et al. [8,9]. The VA was identified by its retromastoid location, reversal of flow direction at its atlas loop and repetitive compression at the atlas when examining its origin in the supraclavicular fossa. With some experience, we could regularly record the proximal VA in approximately 95% of all patients, except in those with massive diffuse thyroid enlargement or short necks with reduced mobility. Flow direction was determined in the supraclavicular position. By rhythmical or sustained digital compression of the VA in the region of the atlas, the proximally recorded signal was invariably modulated, thereby distinguishing the proximal VA from other vessels in the lateral cervical region. The VA signal recorded at the atlas could not be followed continuously caudally (downwards), thereby allowing its distinction from that of the internal carotid. The occipital artery was identified by its compression over the occipital squama: this manoeuvre suppressed the flow in the occipital artery almost completely.

In the normal sonogram, there is a marked end diastolic flow component in both VAs. We monitored the VAs for the following phenomena:

1. Subclavian steal, i.e. systolic deceleration and alternating VA flow in the incomplete type [27], and constant flow reversal in the complete one; the diagnosis was confirmed by the arm compression test [26].
2. Bilateral low or absent end diastolic flow components, bilateral sonographic silence or a combination of these findings, as criteria of a severe disturbance of basilar artery supply, i.e. severe stenosis or occlusion of the basilar or retrograde perfusion in cases of bilateral distal VA occlusion.
3. Reconstitution of the distal VA through collateral pathways without evidence of a severe stenosis at the VA origin, in most cases associated with a "soft" VA signal at the atlas loop; a soft sound of the Doppler signal indicated a reduced acceleration steepness of the systolic frequency profile (peak dv/dt significantly decreased).
4. Suspected stenosis at the origin: high Doppler frequency in the area of the probable VA origin with marked decrease of peak frequencies in the distally adjacent segment.
5. Severe stenosis at the origin: extremely high Doppler frequency proximally, cranially followed by a highly disturbed flow signal with low peak frequencies, and an acoustically soft signal (for definition see No. 3) at the atlas loop; in some cases cervical collaterals[1].
6. Pulse curve with a very small or absent end diastolic flow component in all segments of the VA, indicative of a distal (intracranial) increase in vascular resistance.
7. Soft acoustic appearance of the VA signal (for definition see No. 3) at the atlas loop without other pathological findings.
8. Sonographic silence (VA not detectable).
9. Weak signal, which because of low intensity, was not detectable by a zero crossing meter; in most cases even a frequency analysis device did not provide satisfactory information; weak signals in accoustic analysis typically showed a well-preserved end diastolic flow component.

Due to inevitable inaccuracies in the recordings of zero crossing detectors [19,24], especially for weak signals of thin-calibred and/or deeply located arteries, we renounced graphic analysis of the pulse curves and relied on the criteria of acoustic analysis instead. It is noteworthy that the sophisticated computerized frequency analysis does not essentially accomplish more than the human ear with respect to frequency and intensity analysis, except for objectivity and hence reproducibility.

Retrograde brachial angiography was employed as a reference in the present study. The Doppler sonographic diagnosis was always made prior to angiography in this series; the decision for angiography was based on clinical criteria. It must be noted that due to the angiographic technical used, the right VA was also visualized when the right carotid artery was the vessel of interest. In addition, some cases with cerebral or cerebellar mass lesions and vascular malformations were included. The angiographic diagnoses were routinely made by one of us (H.B.). For the purpose of this study, another member of the group (R.W.) re-examined the angiographies with respect to exact classification of stenoses without being aware of the Doppler results. For measurements, a caliper was used.

The degree of stenosis was calculated on the basis of the distal lumen diameter and referred to only one plane of projection in cases of proximal VA stenoses, since the patient's shoulders obscured this region in the lateral projection.

Taking into account different prognoses and therapeutic approaches to vascular pathology, we distinguished between the following abnormal angiographic findings:

1. Unilateral VA hypoplasia type A: predominantly filling the posterior inferior cerebellar artery (PICA), a third-calibred segment merging with the dominant VA.
2. Unilateral VA hypoplasia type B: ending in the PICA.
3. Unilateral VA hypoplasia type C: ending in muscular branches, no brain-supplying artery.
4. Atherosclerotic irregularities and VA stenosis at the origin up to 59% reduction in lumen diameter.
5. VA stenosis at the origin with an at least 60% reduction in lumen diameter.
6. Severe VA stenosis between the proximal segment and its passage through the dura mater.
7. Severe intracranial VA stenosis (distal to its passage through the dura mater).
8. Unilateral complete occlusion of one VA.
9. Proximal VA occlusion with dye filling the distal VA through cervical collaterals.
10. Unilaterals intracranial VA occlusion.
11. Subclavian steal syndrome with flow reversal (complete) or clearly visible delay in foreward VA circulation (incomplete).
12. Basilar artery stenosis.
13. Bilateral distal or complete occlusion of the VAs, combination of VA hypoplasia type B or C with contralateral occlusion, complete or segmental occlusion of the basilar artery.

If several criteria were applicable, the more important one was attributed: atherosclerotic irregularities and stenoses at the origin below 60% reduction in lumen diameter were not considered in cases of hypoplastic VA. Under all other circumstances, hypoplasia was not considered. Ipsilateral extracranial stenoses were not considered in cases of severe intracranial stenoses and occlusions, or in cases of subclavian steal syndrome.

Results

We compared the angiographic findings in 1,556 VAs of 1,205 patients who underwent retrograde brachial arteriography between 1980 and 1985 with the corre-

[1] v. Reutern et al. (1980) [28] described the sonographic identification of collaterals as superficial cervical vessels with a "brain-supplying" flow pattern, which can easily be compressed throughout. The VAs of some healthy individuals can also be compressed by slight pressure, but solely at their atlas loops.

Table 1. Correlation of Doppler sonographic and angiographic findings. The cases with an angiographically proven occlusion of both distal vertebral arteries (VA) or of the basilar artery are not included

Angiography	Normal	Subclavian steal (complete or incomplete)	Reconstitution of distal VA through collaterals	Suspected stenosis at VA origin	Severe stenosis at VA origin	Lack of diastolic VA flow component	"Soft" VA signal	Sonographical silence	"Weak" VA signal	Σ
Normal	872	4	15	1	4	2	2	2	9	909
Hypoplasia type A	54	–	–	–	–	4	4	5	9	76
Hypoplasia type B	35	–	–	–	–	10	1	12	13	71
Hypoplasia type C	2	–	–	–	–	2	1	5	2	12
VA irregularities, minor stenosis	226	1	–	30	3	1	5	–	2	268
Severe stenosis at VA origin	14	–	–	2	6	2	3	–	4	31
Severe VA stenosis between origin and skull	16	–	1	1	1	–	–	1	–	20
Severe intracranial stenosis	18	–	–	–	–	4	–	–	2	24
Complete occlusion (unilateral)	3	–	2	–	–	16	–	24	1	46
Proximal occlusion, collaterals	10	–	6	–	2	1	3	–	3	25
Intracranial occlusion (unilateral)	–	–	–	–	–	8	–	2	2	12
Subclavian steal	–	16	–	–	–	–	–	–	–	16
Σ	1250	21	9	48	13	52	19	51	47	

sponding sonographic findings in the same patients. A further 20 cases were excluded since no segment of the VA was visualized and no distinction could be made among aplasia, complete occlusion and atypical VA origin from the aortic arch. In 16 of these 20 cases, Doppler sonography was normal; it revealed sonographic silence in 2 cases and pulse curves without a diastolic flow component in 2 further cases.

Table 1 illustrates the correlations between the sonographic and angiographic findings, excluding the cases with basilar artery disease. Of 909 cases with normal angiographic findings 37 were judged pathological by Doppler sonography. Four cases exhibiting an alternating or orthograde flow with systolic deceleration in one VA by sonography may well be excluded from this category, since angiography indicated a subclavian stenosis proximal to the VA origin, thus confirming the sonographic diagnoses. In the remaining cases, the rate of false positive Doppler sonographic results amounted to 33 out of 909 (3.63%).

The number of sonographic abnormalities in cases of VA hypoplasia increased with the extent of this variant (by angiography): hypoplasia type A was indicated by sonography in 22 out of 76 cases (29%), hypoplasia type B in 51% and type C in 83%. Differentiation between hypoplastic VA and intracranial occlusions or severe stenoses was not feasible by Doppler sonography.

Of 268 cases with angiographically proven atherosclerotic irregularities and proximal VA stenoses below 60% reduction in lumen diameter 226 were judged normal by Doppler sonography. In 20 out of the 30 cases with a suspected proximal VA stenosis according to Doppler sonographic criteria, angiography revealed moderate stenoses with a reduction in lumen diameter less than 60%. In this angiographic subset, the case with an absent diastolic flow component, the 7 cases with a weak or soft acoustic signal at the atlas and 12 of the 30 cases with the presumptive sonographic diagnosis of a proximal VA stenosis must be considered false positive, since angiography did not reveal major irregularities and/or circumscript stenoses. One case with a sonographically alternating flow and the angiographic diagnosis of a slight proximal VA stenosis exhibited a proximal subclavian stenosis upon angiography.

Only 7 of the 31 cases with an angiographically proven proximal VA stenosis of at least 60% reduction in lumen diamter were diagnosed unequivocally by sonography, and 2 additional stenoses were suspected (sensitivity 29%). In another 7 of these 31 cases, Doppler sonography showed a weak or soft acoustic signal at the atlas. Together with these nonspecific pathological findings, the sensitivity in-

creased to 52%. In 2 cases, we only found an absent diastolic flow component: angiography revealed a hypoplasia type B which probably masked the severe proximal stenosis in the sonographic evaluation.

The severe extracranial stenoses between the proximal and the intracranial segments of the VA, as well as the unilateral intracranial VA stenoses, exhibited a low sensitivity in the sonographic evaluation (Table 1). All 12 cases of unilateral intracranial occlusions induced pathological Doppler findings (sensitivity 100%), but only 8 of the cases were indicated by an absent diastolic flow component as expected from theory. Corresponding changes were found in the sonograms of 43 out of the 46 cases with a complete VA occlusion (sensitivity 93%); in 2 cases, Doppler sonography revealed collateral pathways that were not detected through angiography.

Proximal VA occlusions with reconstitution of the distal VA through cervical collaterals (as shown by angiography) were detected in 6 out of 25 cases by Doppler sonography; 6 further cases exhibited soft or weak acoustic signals at the atlas without further differentiation of the diagnosis, and 2 cases were misinterpreted as severe proximal VA stenoses due to the high flow velocity in the collateral pathway.

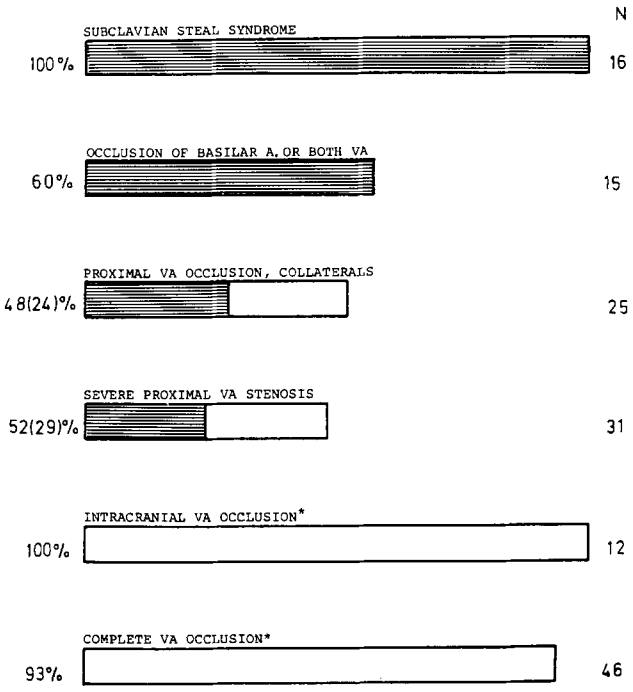

Fig. 1. Sensitivity of Doppler sonography for the most important types of vertebral and basilar lesions. *N* = number of cases. The total length of each bar represents the percentage of pathological sonographic findings for several types of lesions (corresponding percentages of *N* on the left). The *hatched zones* of each bar indicate the percentages of correct specific Doppler sonographic diagnoses (percentages in brackets). * = Unilateral findings

All 16 cases of subclavian steal with flow reversal were correctly diagnosed by directional c-w Doppler sonography.

Among the 8 cases with a basilar artery stenosis above 50% reduction in lumen diameter, only 2 were diagnosed by Doppler sonography, according to the criterion of bilateral absence of diastolic flow components. Six basilar artery occlusions in the proximal third and one in the rostral third were detected sonographically by the above criteria; 2 further occlusions with the same localization were not detected sonographically, 1 probably due to – angiographically demonstrated – rete mirabile anastomoses of the cerebellar arteries, which counteracted the haemodynamic effects of the occlusion on the peripheral resistance and thus on the extracranial VA flow patterns. The remaining 4 basilar artery occlusions missed by sonography did not include the proximal third of the basilar.

The 2 cases with bilateral occlusion of the distal VAs were correctly diagnosed by sonography. A differentiation of the occlusive localization and the degree of basilar stenosis was not achieved by Doppler sonography. False positive results were not obtained in this subset of patients.

Diagnostic Validity of Sonographical Findings[2]

The complete subclavian steal syndrome was correctly diagnosed by sonography in every case. In each of the 5 cases with alternating VA flow, angiography revealed a subclavian stenosis proximal to the origin of the VA[3].

In every case in which a marked disturbance of basilar artery flow was suspected by sonography, the diagnosis was confirmed by angiography.

In 6 out of 9 sonographically suspected cases of cervical collateral pathways in proximal VA occlusions, angiography confirmed the diagnoses. In 2 further cases, angiography did not detect collateral pathways but rather suggested a complete VA occlusion. Generalizing the sonographic criteria to the diagnosis of a severe disturbance of VA circulation, no false positive cases were indicated by Doppler sonography in this subset.

Doppler sonography indicated a VA stenosis at the origin in 48 cases; however, angiography proved a proximal stenosis of at least 60% reduction in lumen diameter in only 2 cases. In 20 further arteries, stenoses with less than 60% reduction in lumen diameter were found by angiography. In 1 case, the angio-

[2] The distribution of pathological and normal cases in our material makes the term "specificity" meaningless. Therefore, we provide only the rate of false positives.

[3] Angiographic findings in Table 1 refer to the VA only.

graphically proven stenosis was located a few centimeters distal to the VA origin. In the remaining 25 VAs, angiography either showed a VA coiling next to its origin, or assumed a compensatory increase in blood flow due to a contralateral subclavian steal syndrome, a severe lesion of the contralateral VA or an external carotid artery occlusion (2 cases).

A severe VA stenosis at the origin was diagnosed (by sonography) in 13 cases, while angiography corresponded in only 6 cases. In 2 additional cases, angiography revealed a stenosis of approximately 40% reduction in lumen diameter (1 case), and a severe stenosis a few centimeters distal to the VA origin (1 case). In another 2 cases, collateral pathways due to proximal VA occlusions were apparently misinterpreted as severe stenoses. There was no angiographic correlation in 3 cases.

Vertebral pulse curves without a diastolic flow component (52 cases) corresponded to VA hypoplasia (types A, B, C) in 16 cases; in 2 cases, the VA hypoplasia masked the co-existing severe proximal VA stenosis. In 28 further patients, angiography detected intracranial VA stenoses and occlusions or complete VA occlusions. The false positive cases amounted to 11.5%.

A soft signal, detected at the atlas loop, indicated a pathological or anatomical variant of the VA in 17 out of 19 cases. It is noteworthy that apparently not all members of our diagnostic ultrasound laboratory agreed upon the acoustic correlation of this criterion, as angiography revealed either a proximal occlusion with collaterals or a severe stenosis at the VA origin in the cases examined by one observer, whereas hypoplasia was found by angiography in 3 out of the 4 cases of another observer.

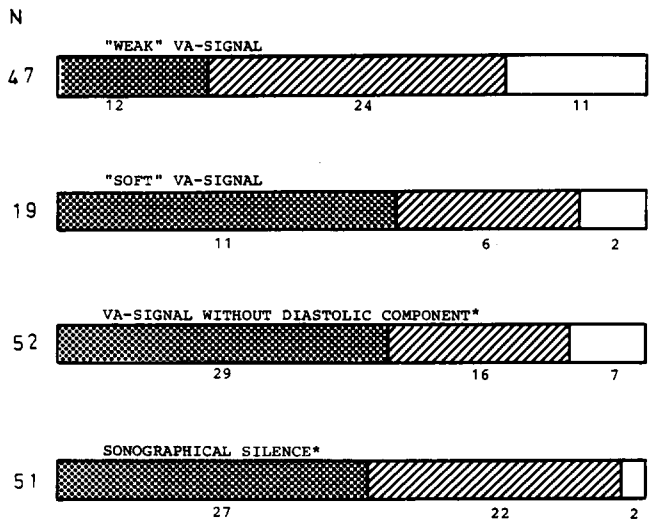

Fig. 2. Correlations of non-specific pathological findings by Doppler sonography (n = number of cases) with angiographic results: pathological findings *(dotted areas)*, anatomical variants *(hatched)* and normal findings *(white)*. * = Unilateral findings

Sonographic silence, i.e. no detectable VA signal, occurred as a unilateral isolated finding in 51 cases and was associated with a complete or an intracranial VA occlusion in 26 cases as demonstrated by angiography. Hypoplasia was found in 22 cases. Definite false positives with normal angiograms amounted to 2 cases (4%).

A weak sonographic signal at the atlas (47 cases) corresponded either to a VA hypoplasia (24 cases) or to a variety of pathological angiographic findings (12 cases). In 11 patients, however, there was no angiographic correlation. Counting only the severe vertebral stenoses at the origin, the intracranial stenoses and the vertebral occlusions as correct sonographic diagnoses, the false positive cases amounted to 74%. Including the angiographically proven cases of vertebral hypoplasia as true positive sonographic diagnoses, the false positive cases by sonography fell to 23%.

Discussion

The great majority of published investigations considering the angiographic-sonographic correlation in VA disease [9, 18, 20, 21, 27–31] have been based on rather small series. Several of these were restricted to a rough analysis of sonographic phenomena and did not include the direct insonation of the proximal VA. An unequivocal assignment of sonographic phenomena to angiographic diagnoses does not exist. All available studies only agree on the high sensitivity and specificity of sonographic detection of the subclavian steal syndrome. The introduction of sonographic examination of the proximal VA [18, 28] promised to substantially increase the diagnostic value of Doppler sonography. However, no study has been published with a number of cases large enough to settle the validity of this technique. In the studies of v. Reutern et al. [28] and Ringelstein et al. [30], reporting 11 and 8 stenoses, respectively, of more than 60% reduction in lumen diameter at the VA origin, the sonographic sensitivity was only 73% and 75%, with a specificity of 100%. V. Reutern et al. [28] described the detection of cervical collaterals in cases of proximal VA occlusions and severe stenoses by Doppler sonography, allowing the diagnosis of extracranial collateral compensation of VA obstruction. However, a substantial rate of false negatives with respect to this diagnosis was observed.

Ringelstein et al. [29] first mentioned the possibility of detecting severe basilar artery disease by indi-

rect sonographic criteria. However, we demonstrated in a previous study that basilar artery occlusions sparing the proximal third and basilar artery lesions that are associated with good collateral circulation through rete mirabile anastomose will not alter the peripheral circulatory resistance, and thus the extracranial flow patterns, sufficiently to result in abnormal sonographic findings [7,35].

In the present study, retrograde brachial arteriography was employed as a reference technique – with its known limitations in the estimation of the degree of proximal VA stenoses, in the detection of collateral pathways, in the distinction of the occlusive localization of VA disease (proximal versus intracranial) and in the clear distinction between a proximal VA occlusion, a VA origin from the aortic arch and a unilateral VA aplasia.

The present study – in accordance with previous investigations – demonstrates the reliability of directional c-w Doppler sonography in the detection and exclusion of some distinct types of VA lesions. However, the sonographic phenomena unilateral lack of a diastolic flow component, softness of the acoustic signal, unilateral sonographic silence and weak, otherwise acoustically normal signal do not permit an unequivocal assignment to an anatomical variant or to a vascular lesion (Fig. 2). Apparently, the haemodynamic effects upon the vertebral circulation may be similar under diverse conditions – orthologically and pathologically – and thus cannot be distinguished by Doppler sonography.

These non-specific pathological sonographic findings can indicate not only a VA occlusion or hypoplasia, but a proximal VA stenosis as well. The phenomenon of a soft signal was often related to proximal stenoses. Since under certain prerequisites proximal VA stenoses are considered clinically important for surgical correction, the detection of such lesions is essential.

Other types of VA lesions are better differentiated by Doppler sonography: the subclavian steal syndrome can reliably be detected or excluded. In the incomplete steal syndrome with peak systolic deceleration or alternating flow, sonography is superior to angiography. The sonographic diagnosis of a proximal VA occlusion with reconstitution through cervical collaterals is also specific, while the sensitivity for the detection of this, according to Fisher [17], clinically benign entity is low.

We conclude from our results that a substantial intracranial unilateral disturbance of VA flow cannot be distinguished from a VA hypoplasia by means of Doppler sonography. However, bilateral intracranial VA occlusion, described as a condition with grave prognosis [11], was detected in each of our two cases. A remarkable percentage of basilar artery occlusions were also found by sonography. A differentiation of the occlusive localization and of the degree of a stenotic lesion in the intracranial vertebro-basilar system was not feasible by Doppler findings. What makes sonography especially valuable in the latter subset of patients is the fact that no false positive findings occurred. As a consequence, in patients having already developed a vertebro-basilar thrombosis but not yet suffering from serious or typical brainstem dysfunction, the Doppler finding of a probable basilar artery occlusion may prompt immediate and vigorous emergency treatment. This applies especially to cases when no neuroradiological unit is readily available. That way, some patients may possible be protected against the expected unfortunate outcome [4] of their diseases.

As already reported in a previous publication [35], many of our patients with segmental basilar artery thrombosis remained in a surprisingly good condition. One may interpret this fact in two ways: it appears plausible that early detection of basilar artery thrombosis and consequent anti-coagulant therapy stop the development of brainstem infarction and markedly improve the course of the disease. It may also be argued that the gravity of basilar artery thrombosis has generally been over-estimated [10, 16] and that Doppler sonography provides a realistic view in demonstrating the true frequency of benign cases that often have been overlooked previously. Since the cases with a thrombosis of the proximal basilar artery are especially well recognized by Doppler sonography and since a grave prognosis is particularly associated with occlusion of the rostral segment, this selection by sonographic criteria may explain the favourable clinical course of our patients. The mode of selection for treatment in our hospital excludes patients in an extremely bad condition. This probably explains the low total number of sonographically detected basilar artery thromboses. In addition, patients with an apparently life-threatening disease of the basilar artery sometimes "bypass" the ultrasound laboratory, since a definite exclusion or diagnosis of basilar thrombosis is impossible without angiography.

The sonographic diagnosis of severe arterial stenosis is generally considered a specific finding. In our series, the specificity with respect to the diagnosis of a severe proximal VA stenosis is surprisingly low with 4 definitely false positives among 13 such sonographic diagnoses. This may be explained by different reliabilities of the results among the members of our group with respect to proximal VA stenoses, indicating severe problems in the insonation of the proximal VA in the supraclavicular fossa. At this location,

signals from compressed veins may obscure arterial signals or even imitate arterial stenoses. High flow velocities in the inferior thyroid artery and in collaterals are also liable to cause error.

On the other hand, limitations in our arteriographic technique, especially in this region, must be considered. Only one plane of projection is available for the proximal VA, and a well opacified subclavian artery may shadow a VA origin located at the dorsal circumference of the subclavian artery. As a consequence, stenoses of the VA origin may be missed by retrograde brachial angiography.

Concerning stenoses of the VA origin, the sensitivity of Doppler sonography was low in our study, reducing the diagnostic value of this technique. The considerably higher sensitivity (up to 75% in the detection of proximal VA stenoses of at least 70% reduction in lumen diameter reported in previous studies [18, 28, 30] may be attributed to the low figures of their angiographic controls, especially in sonographically unsuspected cases. We believe that a large number of proximal VA stenoses were not detected in the present study due to sonographic examination of the proximal VA segment, but not of the true – quite variable [14] – VA origin from the subclavian artery.

How to adequately evaluate patients with vertebro-basilar insufficiency remains an unsettled question. A benign natural history – especially concerning the functional outcome – has been attributed to ischaemic attacks in the vertebro-basilar system by some authors [5, 25, 36]. Others did not find a difference between the prognoses of transient ischaemic attacks (TIAs) in the carotid artery and in the vertebro-basilar system [12, 13, 34]. Faught et al. [15] stated that "real vascular disease" is rarely found in angiograms of patients suffering clinically from vertebro-basilar ischaemic attacks. On the other hand, large infarctions of the cerebellum and the brainstem are undoubtedly life-threatening to a greater degree than hemispheric brain infarctions of equal size.

As a consequence of these divergent findings, the evaluation of patients with clinical symptoms of vertebro-basilar insufficiency has to meet conflicting demands. It is essential that dangerous vascular lesions be diagnosed early enough for effective and preventive therapy. However, invasive diagnostic procedures should be used with reserve due to the low yield of pathological findings. In addition, it must be considered that the great majority of pathological findings in the vertebro-basilar system have no specifical therapeutic consequences. Some new measures have been developed during the last 10 years [2, 3, 22, 29, 32, 33] but their efficacy has by no means been proved yet.

There are four accepted therapeutic approaches to vertebro-basilar insufficiency that exceed general measures such as regulation of blood pressure and treatment of congestive heart failure: (1) operations on stenoses and occlusions of subclavian or innominate arteries in cases of a severe, symptomatic subclavian steal syndrome; (2) operations on severe stenoses at the VA origin – when the contralateral VA is hypoplastic, absent or also severely compromized [6]; (3) operations on high grade carotid artery stenoses when a patent posterior communicating artery can be demonstrated by angiography [23]; and (4) anti-coagulant drugs in cases of severe basilar artery disease.

Each of these measures is rarely applied in comparison to carotid artery TEA and anti-coagulant therapy for hemispherical TIAs or minor strokes.

In the evaluation of carotid artery disease, non-invasive techniques have gained great importance during the last decade. In contrast, the success of the non-invasive investigation of the vertebro-basilar arterial system has been disappointingly small. The main reasons are the great variability in normal anatomy, the relatively inaccessable location of the vertebral and basilar arteries as an impediment to reliable sonographic diagnoses and a certain lack of approved therapeutic measures.

Our results confirm that conventional hand-held c-w Doppler sonography cannot replace angiography in the evaluation of patients with vertebro-basilar insufficiency. Generally speaking, it rather serves as an aid to the decision for or against angiography. Within this limitation, Doppler screening of patients appears worthwhile, since several therapeutically important lesions – severe stenoses and occlusions of the carotid, the subclavian, and the innominate arteries – are readily diagnosed by sonography. The other categories of important lesions – stenoses at the VA origin and a severely compromised basilar artery perfusion – are indicated by Doppler sonography to a substantial degree.

Long-term as well as short-term follow-up studies make Doppler sonography of the VAs even more valuable. Changes in Doppler findings, especially the disappearance of a VA signal, can definitely be interpreted as pathological, since in this case a hypoplasia need not be discussed as a cause of sonographic silence. Once an intracranial occlusion has been proven by angiography as the cause of a VA flow pattern without a diastolic component, Doppler sonography can indicate re-canalization if a normal flow pattern is rapidly re-established. It even appears reasonable to expect that the follow-up of proximal VA stenoses, once confirmed by angiography, can be done by sonography.

It may be expected that the transcranial investigation of the distal VAs and the basilar artery as introduced by Aaslid and coworkers [1] greatly enhances the importance of diagnostic ultrasound in the evaluation of patients with vertebro-basilar disease.

Acknowledgement. The authors gratefully appreciate the reliable and qualified work of Miss M. Stegmaier, the technical assistant of the diagnostic ultrasound laboratory.

References

1. Aaslid R, Markwalder T-M, Nornes H (1982) Noninvasive transcranial Doppler ultrasound recording of flow velocity in basal cerebral arteries. J Neurosurg 57:769–774
2. Allen GS, Cohen RJ, Preziosi TJ (1981) Microsurgical endarterectomy of the intracranial vertebral artery for vertebrobasilar transient ischemic attacks. Neurosurgery 8: 56–59
3. Ausman JI, Nicoloff DM, Chou SN (1978) Posterior fossa revascularization: Anastomosis of vertebral artery to PICA with interposed radial artery graft. Surg Neurol 9:281–286
4. Archer CR, Horenstein S (1977) Basilar artery occlusion, clinical and radiological correlation. Stroke 8:383–390
5. Baker RN, Ramseyer JC, Schwartz WS (1968) Prognosis in patients with transient cerebral ischemic attacks. Neurology 18:1157–1165
6. Berguer R, Bauer R (1981) Vertebral artery reconstruction; a successful technique in selected patients. Ann Surg 193:441–447
7. Biedert S, Winter R, Staudacher T, Betz H, Reuther R (1985) Doppler sonography in basilar artery occlusion. Neuroradiology 27:430–433
8. Büdingen HJ, Reutern GM v, Freund HJ (1876) Die Differenzierung der Halsgefäße mit der direktionellen Doppler-Sonographie. Arch Psychiatr Nervenkr 222:177–190
9. Büdingen HJ, Reutern GM v, Freund HJ (1982) Dopplersonographie der extracraniellen Hirnarterien. Thieme, Stuttgart New York
10. Caplan LR (1979) Occlusion of the vertebral or basilar artery, follow-up analysis of some patients with benign outcome. Stroke 10:277–282
11. Caplan LR (1983) Bilateral distal vertebral artery occlusion. Neurology 33:552–558
12. Cartlidge NE, Whisnant JP, Elveback LR (1977) Carotid and vertebral-basilar transient cerebral ischemic attacks. A community study, Rochester, Minnesota. May Clin Proc 52:117–120
13. Coneally PM, Dyken ML, Futty DE, Poskanzer DE, Calanchini PR, Swanson PD, Price TR, Haerer AF, Gotshall RA (1978) Cooperative study of hospital frequency and character of transient ischemic attacks. JAMA 240:742–746
14. Daseler EH, Anson BJ (1959) Surgical anatomy of the subclavian artery and its branches. Surg Gynecol Obstet 108: 149–174
15. Faught E, Trader SD, Hanna GR (1979) Cerebral complications of angiography for transient ischemia and stroke: Prediction of risk. Neurology 29:4–15
16. Fields WS, Ratinov G, Weibel J, Campos RJ (1966) Survival following basilar artery occlusion. Arch Neurol 15: 463–471
17. Fisher CM (1970) Occlusion of the vertebral arteries causing transient basilar symptoms. Arch Neurol 22:13–19
18. Franceschi C (1977) L'investigation vasculaire par ultrasonographie Doppler. Masson, Paris
19. Johnston KW, Maruzzo BC, Cobbold RSC (1977) Errors and artifacts of Doppler flowmeters and their solution. Arch Surg 112:1335–1342
20. Kaneda H, Irino T, Minami T, Taneda M (1977) Diagnostic reliability of the percutaneous ultrasonic Doppler technique for vertebral arterial occlusive diseases. Stroke 8: 571–579
21. Keller HM, Meier WE, Kumpe DA (1976) Noninvasive angiography for the diagnosis of Vertebral Artery disease using Doppler ultrasound (vertebral artery Doppler). Stroke 7:364–369
22. Khodadad G, Singh RS, Olinger CP (1977) Possible prevention of brain stem stroke by microvascular anastomosis in the vertebrobasilar system. Stroke 8:316–321
23. Lord R, Adar R, Stain R (1969) Contribution of the circle of willis to the subclavian steal syndrome. Circulation 40: 871–878
24. Lunt MJ (1975) Accuracy and limitations of the ultrasonic Doppler blood velocimeter and zero crossing detector. Ultrasound Med Biol 2:1–10
25. Marshall J (1964) The natural history of transient ischaemic vertebro-vascular attacks. Q J Med 33:309–324
26. Mozersky DJ, Barnes RW, Sumner DS, Strandness DE Jr (1973) Hemodynamics of innominate artery occlusion. Ann Surg 178:123–127
27. Reutern GM v, Pourcelot L (1978) Cardiac cycle-dependent alternating flow in vertebral arteries with subclavian artery stenoses. Stroke 9:229–236
28. Reutern GM v, Clarenbach P (1980) Value of Doppler examination of the cervical collateral arteries and the vertebral ostium in the diagnosis of stenoses and occlusions of the vertebral artery. Ultrasonics 1:153–162
29. Ringelstein EB, Zeumer H (1982) The role of continuous-wave Doppler sonography in the diagnosis and management of basilar and vertebral artery occlusions, with special reference to its application during local fibrinolysis. J Neurol 228:161–170
30. Ringelstein EB, Zeumer H, Pohlen R (1982) Wertigkeit und Grenzen der CW-Doppler-Sonographie im vertebrobasilären Kreislauf. Deutsche Gesellschaft für Angiologie, Jahrestagung Ulm/Neu-Ulm, 15.–18. September 1982
31. Ringelstein EB, Zeumer H, Hündgen R, Meya U (1983) Angiologische und prognostische Beurteilung von Hirnstamminsulten. DMW 108:1625–1631
32. Sundt TM Jr, Smith HC, Campbell K, Vliestra RE, Cucchiara RF, Stanson AW (1980) Transluminal angioplasty for basilar artery stenosis. Mayo Clin Proc 55:673–680
33. Sundt TM Jr, Piepgras DG, Houser OW, Campbell JK (1982) Interposition saphenous vein grafts for advanced occlusive disease and large aneurysms in the posterior circulation. J Neurosurg 56:205–215
34. Toole JF, Janeway R, Choi K, Cordell R, Davis C, Johnston F, Miller HS (1975) Transient ischemic attacks due to atherosclerosis: A prospective study of 160 patients. Arch Neurol 32:5–12
35. Winter R, Biedert S, Reuther R (1984) Das Doppler-Sonogramm bei Basilaristhrombosen. Eur Arch Psychiatr Neurol Sci 234:64–68
36. Ziegler DK, Hassanein RS (1973) Prognosis in patients with transient ischemic attacks. Stroke 4:666–673

Received February 23, 1987