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Review
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THE ROLE OF NEUROSONOLOGY IN VERTIGO

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Summary

Vertigo is a common symptom, and may represent a serious neurological disorder. If it develops suddenly, it may be a symptom of an acute stroke.

Neurosonology can be used in cerebrovascular disorders in order to assess the vessel patency, or to present different craniocervical artery diseases. Atherosclerotic changes may be seen, as well as inflammatory diseases, dissections, vasculopathies or vascular malformations. By means of a transcranial Doppler the intracranial hemodynamics can be assessed. A development of collateral pathways in extra- or intracranial occlusive diseases can be presumed, cerebral vasomotor reactivity can be tested, and, with the application of new softwares, microembolic signals can be detected.

Neurosonology can be used in a variety of neurological disorders presenting with vertigo.

Key words: Vertigo, Color Doppler, Transcranial Doppler, carotid artery, vertebral artery

INTRODUCTION

Vertigo is a common symptom. Since it may represent a neurological disorder, it is a frequent reason for neurological consultations. A sudden onset may be the symptom of an ischemic or hemorrhagic stroke, especially in the posterior circulation. It may represent different cerebrovascular disorders developing from vessel variability like hypo-

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plasia, aplasia, vascular malformation, steno-occlusive changes in the vertebrobasilar circulation, or subclavian steal syndrome. A vessel inflammation or vasculopathies may lead to vertebrobasilar circulation disorders or can cause dissections. Besides vascular disorders, brain tumors in posterior fosse or demyelization, they may cause vertiginous symptoms.

Neurosonological studies are routinely performed in neurological clinics. Their greatest advantage is the real-time, bedside evaluation of the morphology and hemodynamics of brain vessels. The main goal is to identify large obstructive lesions in the extracranial and intracranial basal arteries and to monitor and facilitate spontaneous or drug-induced thrombolysis in the majority of patients. It also enables the identification of vascular lesions amenable for interventional treatment. The detection of rare causes of an ischemic stroke, such as dissections, vasculopathies and other less frequent etiologies is facilitated by a systematic use of ultrasound studies. Therefore, its usage is recommended as a part of comprehensive stroke treatment [1-7].

Vertigo in the emergency room

In acute vertigo a brain imaging study like CT scan is mostly used to exclude structural brain lesions that may imitate a stroke [1,2,3]. Rarely, in the first few hours, it will display ischemic changes. Therefore, neurosonological investigations may point at underlying stroke mechanisms. Also, in some centers, CT scan is not available on a 24-hour basis. Therefore, this useful test in clinicians' hands may help in patient management. Neurosonology has several advantages: it can be performed at the bedside and repeated as needed or applied for continuous monitoring; its usage is less expensive, and more available. It consists of extracranial color Doppler imaging of carotid and vertebral arteries and transcranial color Doppler sonography for intracranial evaluation. In acute vertigo, with an experienced clinician, it may help to distinguish an ischemic from hemorrhagic stroke by means of the underlining mechanism detection: macroangiopathic, cardioembolic, vasculopathy or dissection; or to raise the suspicion on the signs of vascular malformations. It may also point out the advantages of brain hemodynamic monitoring.

Extracranial evaluation of vertebral arteries

One fourth of ischemic strokes are related to the vertebrobasilar territory. Noninvasive investigations of vertebral arteries have become popular in the last decade with the invention of the color Doppler, so that vertebral occlusions have been seen more often, clinically presented with TIA or a mild stroke. Also, vertebral dissections have been recognized more often.

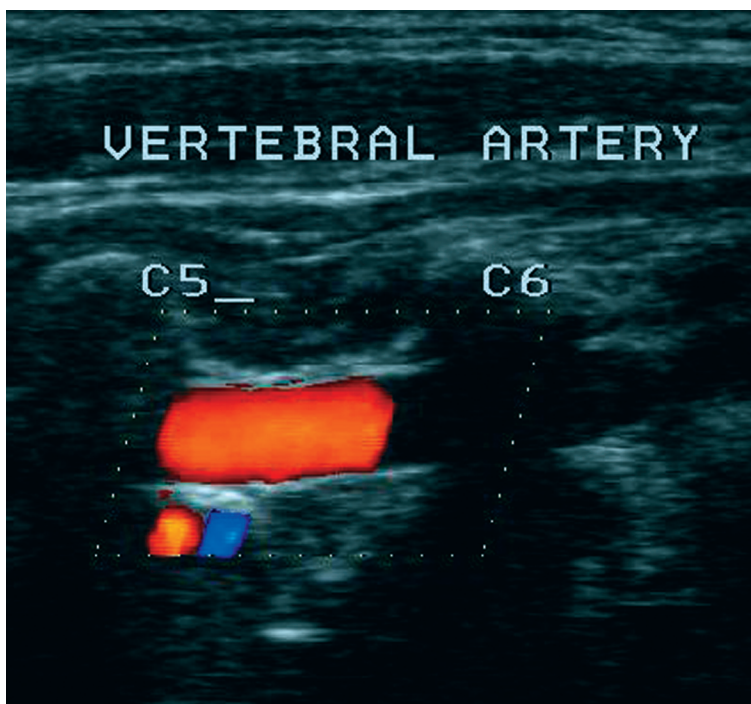


Figure 1. Normal vertebral artery

By means of vertebral artery color Doppler sonography (VCDS) normal vertebral arteries may be displayed (Fig. 1). Asymmetry of vertebral arteries may be found in up to 30% of healthy individuals, with the left vertebral artery wider, and more frequent as dominant [8,9,10,11]. Hypoplasia of a vertebral artery may be found in up to 10% of cases, slightly more often of the right than of the left vertebral artery [8,9,10,11]. Blood flow velocities and the mean diameter don't change with age. Females have higher blood flow velocities, and thinner vertebral arteries than males.

In hypoplasia [8,9,10] the findings may include poor color flow opacification, low flow velocities and increased resistance (Fig. 2). A hypoplasia in the vertebrobasilar system was found to predispose the adults to a posterior circulation ischemia [12].

VERTEBRAL artery occlusion and posterior circulation ischemia

The visualization of vertebral artery occlusion depends on the location, diameter and blood flow volume in the artery and collaterals [13]. The hemodynamic spectra may help in localizing the site of occlusion [13,14]. In patients with a distal occlusion, color

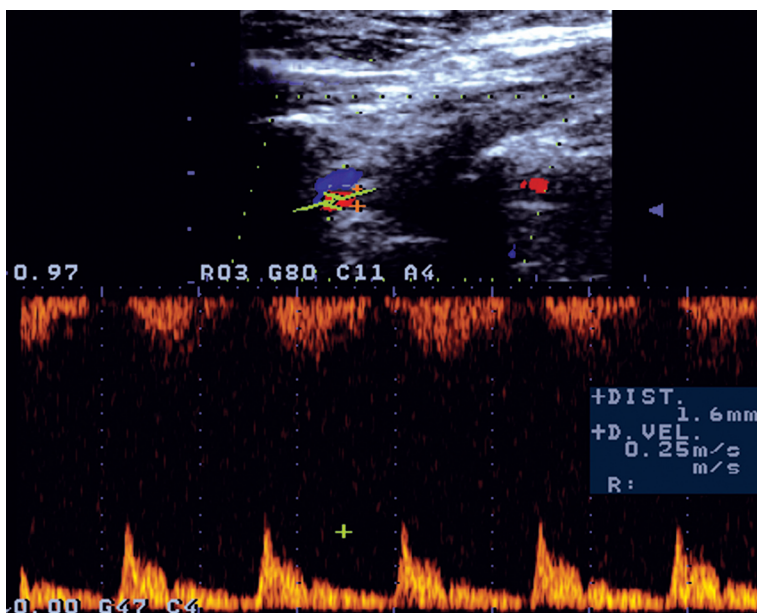


Figure 2. Hypoplastic vertebral artery

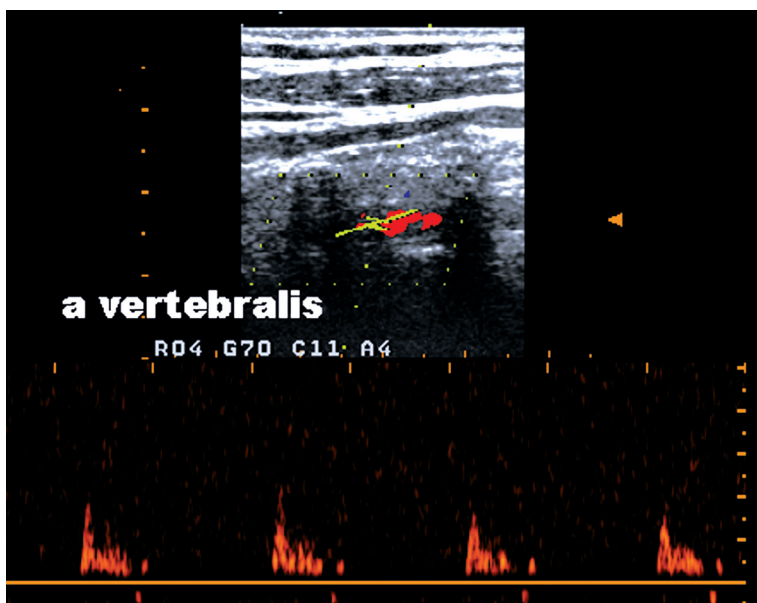


Figure 3. Vertebral artery occluded in the distal part, before the branching of the posterior inferior cerebellar artery

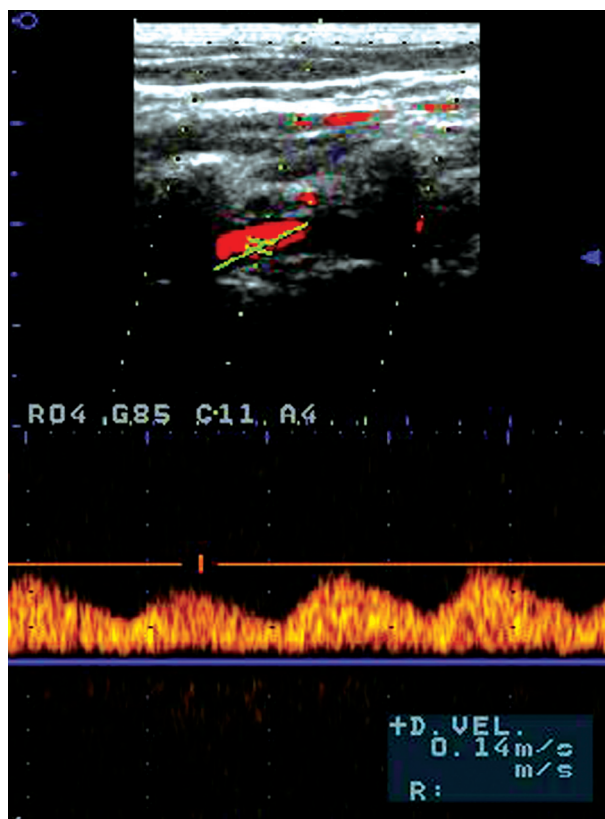


Figure 4. Vertebral artery occluded in the distal part, after the branching of the posterior inferior cerebellar artery

Doppler filling may be reduced due to similar hemodynamic changes as in the hypoplastic vertebral artery (Fig. 3). Difficulties in distinguishing the site of occlusion exist, since the collateral flow may resemble a vertebral artery. A power-enhancement Doppler enables visualization of a vessel with very low flow velocities, as in those vertebral arteries with a dampened flow due to distal or proximal occlusive lesions and a tortuous course [13,14,15]. VCDS can therefore distinguish the site of vertebral artery occlusion according to the following criteria. Vertebral artery occlusion at the origin (V1 occlusion) (Fig. 4): lumen filled with plaques, absence of directional or power Doppler flow within the lumen, presence of collateral flow [13,14,15,16]. Vertebral artery occlusion before the branching of the posterior inferior cerebellar artery (PICA): vertebral artery filled with color, absence of diastolic flow [13,14] (Fig. 4). Vertebral artery occlusion after the branching of the PICA: vertebral artery filled with color, damped flow (blood flow

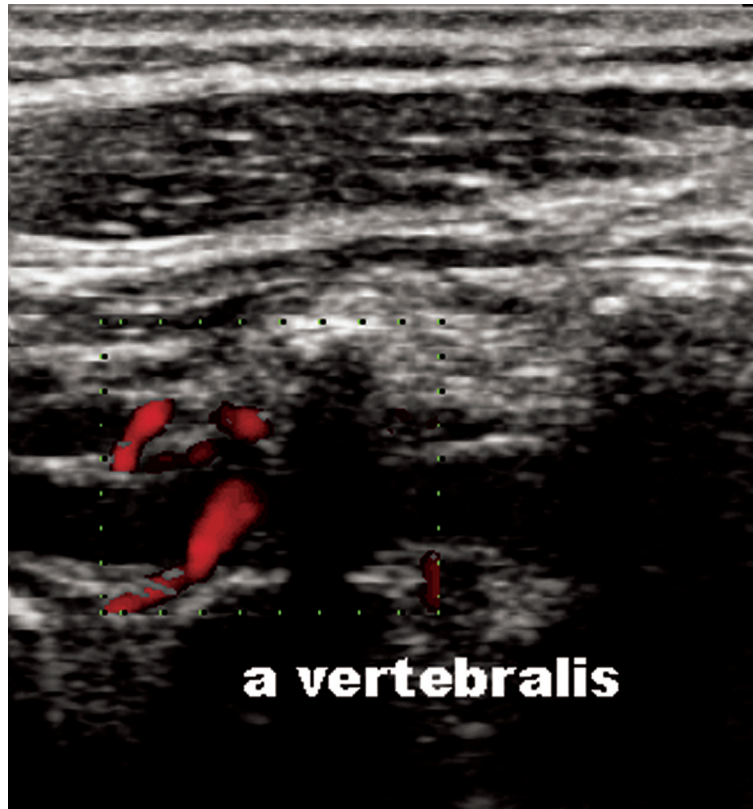


Figure 5. Vertebral artery occluded at the origin

velocities reduced more than 30% to contralateral side, presence of diastolic flow) (Fig. 3). The differences in end-diastolic flow velocities between the last two groups are thought to be based on the differences in the peripheral vascular resistance. In the group with occlusion after the branching of the PICA, blood can flow through the vertebral artery as far as the PICA. Therefore, the peripheral vascular resistance in this group is lower than in the previous group, before the branching of the PICA, thus resulting in higher end-diastolic flow velocities. If the occlusion is proximal of the branching of PICA, a higher resistance pattern with absent diastolic flow is expected. In patients with a basilar artery occlusion, in both vertebral arteries high resistance pattern would be found.

Most data on ischemia in the posterior circulation derive from the New England Medical Center Posterior Circulation Registry (NEMC PCR) [17,18], consisting of a consecutive series of 407 patients with signs and symptoms of posterior circulation ischemia seen during a 10-year period.

Clinical presentations in these 407 NEMC PCR patients [18] were as follows: 59% had strokes without transient ischemic attacks (TIAs), 24% had TIAs then strokes, and 16% had only TIAs. Embolism was the commonest stroke mechanism (40% of patients including 24% of cardiac origin, 14% intra-arterial, 2% cardiac and arterial). In 32% of patients large artery occlusive lesions caused a hemodynamic brain ischemia. Infarcts most often included the distal posterior circulation territory (rostral brainstem, superior cerebellum and occipital and temporal lobes); the proximal (medulla and posterior inferior cerebellum) and middle (pons and anterior inferior cerebellum) territories were equally involved. Severe occlusive lesions (>50% stenosis) involved more than one large artery in 148 patients; 134 had one artery site involved unilaterally or bilaterally. The most common occlusive sites were: extracranial vertebral artery (52 patients, 15 bilateral) intracranial vertebral artery (40 patients, 12 bilateral), basilar artery (46 patients). Intra-arterial embolism was the most common mechanism of brain infarction in patients with a vertebral artery occlusive disease. Thirty-day mortality was 3.6%. Embolic mechanism, distal territory location, and basilar artery occlusive disease carried the poorest prognosis. The best outcome was in patients who had multiple arterial occlusive sites; they had position-sensitive TIAs for months to years.

According to Wytick [17], out of 407 patients chosen for study from the NEMC PCR, 80 (20%) had V1 segment lesions, either higher-grade stenosis or occlusion. Patients were classified into 5 groups: 22 patients had V1 disease and a coexistent severe intracranial occlusive disease of the posterior circulation, 19 had V1 disease with an evidence of artery-to-artery embolism, 20 had a suspected V1 disease with an artery-to-artery embolism, but with other potential causes of the stroke or a less certain vascular diagnosis, 13 had V1 disease associated with hemodynamic transient ischemic attacks and 6 had proximal vertebral arterial dissection. Hypertension, cigarette smoking, and a coronary artery disease were common risk factors. Clinical features, the location of infarct and the outcome differed between the groups and reflected the presumed mechanisms of stroke.

The use of VCDS increases diagnostic confidence of the sonographic examination in patients with suspected vertebral artery disease, like stenosis, occlusion or dissection.

Vasculitis, vasculopathies, dissections

Vasculitis of the nervous system includes a group of disorders characterized by the histological feature of an inflammation of blood vessels. The diagnosis is suspected by a clinical presentation, and confirmed by signs of inflammation obtained with laboratory analysis, or biopsy. The use of CCDS may help in a noninvasive visualization of the disease [19]; by direct visualization if the location of the disease is present in a segment that is accessible to the ultrasound investigation, like the affection of the branches of the aortic arch [20], by indirect signs in the hemodynamics of the carotid or vertebral arter-



ies, like the subclavian steal syndrome in one of the vertebral arteries, or by visualizing dark halo around the pin like the color-coded flow in the temporal [21] or occipital artery [22]. In this way a hypoechoic vessel wall thickening of the temporal or occipital artery is described, most likely reflecting an inflammatory vessel wall edema. Fibrotic healing would increase the level or echogenicity of the vessel wall, and later on improve the flow. Color Doppler imaging enables differentiation between a spontaneous dissection and giant cell arteritis of the vertebral artery [23], which may have a similar clinical and sonographic appearance at first sight. Clinically, both diseases may be accompanied by an occipital headache and neck pain with an acute or subacute onset, with a later presentation of ischemic symptoms. Sonographically, both entities may display a hypoechoic vertebral artery with a residual channel of color flow. In dissection the intramural hematoma leads to a stenosis or occlusion of a vertebral artery, and a similar finding would be the result of an inflammatory hypoechoic vessel wall change in giant cell arteritis. The difference is that the hypoechogenicity in a dissection is extending along the extracranial vertebral artery, and is usually eccentric and crescent-shaped, often with a spiraling course. A concentric halo of a vertebral artery may have a good positive predictive value for the presence of vasculitis, especially if found in another vessel like the temporal artery. Vasculitis affecting smaller arteries may alter the intracranial hemodynamics, which can be measured as impaired vasoreactivity as a marker of smaller vessel involvement.

Between vasculopathies, Moyamoya disease [24] and fibromuscular dysplasia [25] can be displayed, and may predispose to a dissection. Dissections are lately more often recognized as relatively common causes of a stroke, particularly among young patients. Dissections lead to ischemic strokes through artery-to-artery embolism or by causing a significant stenosis and occlusion of the proximal vessel, and in some cases, dissections may lead to the formation of a pseudoaneurysm, which can also serve as a source of thrombus formation. Intracranial dissections in the vertebrobasilar territory have a higher risk of rupture, leading to a subarachnoidal hemorrhage (SAH). Dissections may appear as different findings in the color-coded Doppler mode [26-30]. When extending from the aortic arch, double lumens can be seen. Bifurcation stenosis may dissect leading to the formation of a color-coded flow in the plaque base. In younger persons dissections are usually affecting the distal parts of internal carotid or vertebral arteries. Hypoechoic stenosis of the vessels in distal parts can be seen, or, when located intracranially leading to a complete occlusion, the indirect signs of distal occlusions are present. Such signs include dampened flow, with a high resistance pattern, and possible inversed hemodynamics during the diastole. The goals of the therapy, when treating patients with dissections and an ischemic stroke, are to prevent further ischemic strokes and to promote healing of the dissected vessel. Carotid CDS may help in monitoring the vessel healing,

parallel with the emboli detection that may show a reduction in embolic signals [31-34]. One of the first neurosonological investigations of vertebral artery dissections [28] verified by angiography, magnetic resonance imaging or both, revealed that the most common dissected segments in 14 patients were as follows: atlantoaxial (V-3) in 6, V-3 and intertransverse (V-2) in 3, V-3 and intracranial (V-4) in 3, and V-2 in 2 patients. Extracranial and transcranial Doppler examination of the atlas loop, involving 12 patients, showed an absent flow signal in 5, low bidirectional flow signal in 1, and poststenotic low blood flow velocities in 3 patients. Seven of these patients had a high-grade stenosis or occlusion. The stenotic segment with an increased flow signal was identified directly in 2 patients. Duplex examination of the intertransverse segment confirmed an absent flow in 4 patients, making technically insufficient examination unlikely. In the 2 patients with directly detected stenosis, duplex examination showed low flow velocities before the stenosis. The combined use of extracranial and transcranial Doppler and duplex sonography increased the diagnostic yield to detect vertebral artery pathology. If abnormal sonographic findings were considered, the yield was 86%; relying only on the definitively abnormal findings (absent flow signal, severely reduced vertebral artery blood flow velocities, no diastolic flow, bidirectional flow, and a stenosis signal) the yield was 64%. Such results pointed out that there was no pathognomonic ultrasound finding for vertebral artery dissection. If a patient presents with suggestive symptoms, an ultrasound may corroborate the clinical suspicion and aid in the decision regarding an early anticoagulant treatment, and the definite diagnosis can be made with other imaging techniques demonstrating hematoma in the vessel wall.

In another series of vertebral artery dissections in 24 patients with 28 vertebral artery dissections in the neck (4 occurring bilaterally) [29], with 83% of dissections temporally related to trauma, and without an underlying vascular disease, the major initial manifestation was pain in the occipital or neck region. The next most common symptoms were vertigo and nausea in 17 patients. Clinical manifestations were: 5 patients with vertebrobasilar TIA (in 2 patients vestibulocerebellar TIA, in 1 patient visual TIA, in 1 patient motor TIA, and in 1 patient brain stem TIA with perioral paresthesia), 10 patients with a cerebellar infarction (in 4 patients bilateral), 5 patients with a brainstem infarction, in one a posterior cerebral artery territory infarction, and multiple vertebrobasilar ischemic lesions in 3 patients. Typical ultrasonographic findings were: irregular stenosis, dissecting membrane with a true and false lumen, localized increase in the diameter of the artery, pseudoaneurysm, intramural hematoma, and a tapering stenosis with a distal occlusion; while typical angiographic findings were irregular narrowing of the vessel lumen or a tapering stenosis with a distal occlusion. Magnetic resonance imaging showed a thickened vessel wall with a hematoma signal at the site of the dissection. Duplex color-flow imaging was valuable for the early diagnosis of an extracranial verte-



bral artery dissection and for follow-up examinations. In 43% of cases the most frequent localizations of dissections were the distal V1- and the proximal V2-segment (at the level of the C6 vertebra). The outcome was favorable, except for 2 patients with a basilar artery occlusion. In an earlier series by the same authors [35] in the follow-up examination a good regression of pathological findings was found in 70.8% dissections, with two occlusions completely reanalyzed.

Lately, the biggest series of 195 vertebral artery dissections in 169 patients were published [36]. Brain ischemia occurred in 131 patients (77%; ischemic stroke in 67% and TIA in 10%). Three patients with an ischemic stroke also showed signs of a subarachnoid hemorrhage (SAH) and 3 (2%) had SAH without ischemia. Head and/or neck pain was present in 118 out of 134 patients (88%) with brain ischemia or SAH, and pulsatile tinnitus in seven (5%) patients. The remaining 35 patients (21%) had an isolated head and/or neck pain in 21 (12%) cases, asymptomatic spontaneous vertebral artery dissection in 13 (8%), and cervical radiculopathy in one case (1%). Location of the spontaneous vertebral artery dissection was more often in the pars transversaria (V2; 35%) or atlas loop (V3; 34%) than in the prevertebral (V1; 20%) or intracranial (V4; 11%) segments ($P=0.0001$). A favorable outcome (mRA 0-1) was found in 82% of 107 ischemic stroke patients with follow up, and two (2%) patients died. Independent predictors of a favorable outcome were a low baseline National Institutes of Health Stroke Scale score and a younger age.

Transcranial evaluation of stroke, vessel occlusion or hemorrhage

Transcranial Doppler (TCD) measures the local blood flow velocity (BFV) and direction in the proximal portions of large intracranial arteries [6,7,37,38]. It is a "blind method", therefore operator dependent and requires training and expertise to perform and interpret the results. Several studies evaluated the ultrasound in comparison with neuroradiological imaging methods in the acute stroke setting [5,7]. Non-contrast-enhanced TCD was reported to have a sensitivity of 80% and a specificity of 90%, compared with digital subtraction angiography (DSA) in patients presenting within 5 hours of a middle cerebral artery (MCA) stroke [39], but the sensitivity for detecting internal carotid, basilar or vertebral artery occlusion is lower (SE 55-81%, SP 96%) [7]. By means of transcranial color coded Doppler (TCCD) flow imaging the sensitivity to detect advanced (greater than 50%) MCA, vertebral or basilar artery stenosis is higher (SE 100%, SP 100%) [40]. Therefore TCD or TCCD may be used as a screening test to determine the need for further angiographic studies. The bedside availability, convenience to the patient, and continuous monitoring possibility make TCD particularly suitable and practical for emergency evaluations. TCD also allows real-time assessment of the BFV, pulsatility, and microembolization, information which is not available with angiography.



Intracranial arterial occlusions detected by TCD are associated with poor neurological recovery, disability, or death after 90 days [41], whereas normal results predict early improvement [42]. In patients with an acute ICA territory stroke TCD findings, stroke severity at 24 hours, and CT lesion size were independent predictors of the outcome after 30 days [41]. When combined with the carotid duplex sonography, the presence and total number of arteries with suspected steno-occlusive lesions by TCD in TIA or stroke patients, were associated with a poor outcome [43,44], an increased risk of further vascular events and death within 6 months [45]. Such a combined stroke patient evaluation can identify lesions amenable for interventional treatment (LAIT) in patients with an acute cerebral ischemia [46], achieving 100% accuracy.

Several TCCS studies have shown that the detection of a homogeneously hyperechoic area, sharply demarcated from the surrounding brain tissue, is diagnostic for an acute intracerebral hemorrhage (ICH) [47,48]. It is also possible to monitor the midline shift (MLS) in patients with space-occupying MCA infarcts, since MLS displacement may predict a fatal outcome in patients with malignant MCA infarcts [49]. TCCS can identify stroke complications like a hemorrhagic transformation, ventricular bleeding or MLS, and differentiate between an intracerebral hemorrhage and an ischemic stroke with a 95% sensitivity and 94% specificity [50]. Thus, if a CT scan is not readily available, TCCS may help in identifying patients with a primary brain hemorrhage or secondary hemorrhagic complications.

TCD in subarachnoidal hemorrhage (SAH)

Some patients with a subarachnoidal hemorrhage present with a headache and vertigo. In these patients TCD and TCCD is helpful for the assessment of cerebral vasospasm (VSP) and in differentiating a VSP from angiomas feeding vessels, or to directly visualize the vascular malformation.

Cerebral VSP is a delayed narrowing of the large capacity arteries at the base of the brain after SAH, often associated with the radiographic or cerebral blood flow evidence of diminished perfusion in the distal territory of the affected artery, and can be easily detected by means of TCD [51]. It has a typical temporal course with onset 3 to 5 days after the hemorrhage, reaching its maximal at 5 to 14 days, and gradually resolving over 2 to 4 weeks. In about one half of the cases a VSP is manifested by the occurrence of a delayed neurological ischemic deficit, which may resolve or progress to cerebral infarction [51].

Large and medium-sized cerebral aneurysms located in the proximal segments of the circle of Willis can be sometimes detected as colored oval structures of a pulsatile nature adjacent to large parent arteries [52,53]. Aneurysms located beyond the field of

scanning and those that are thrombosed cannot be detected. TCCS can detect 76 to 91% of nonthrombosed intracranial aneurysms of >6 mm in size [52,53], and the use of echo contrast agents or power Doppler may increase the rate of detection, including aneurysms >5 mm in size [52-54].

Arteriovenous malformations (AVMs) can be displayed as areas with a color mosaic, which is related to the focal accumulation of vascular convolutions and spectral hemodynamic abnormalities similar to those in the feeding vessels [55] TCD and TCCS can suggest the presence of AVMs by detecting abnormal increased systolic and especially end diastolic flow velocities and a decreased pulsatility in the feeding arteries [55,56].

CONCLUSION

Neurosonological investigations are useful in the evaluation of patients with vertigo. They can display the vertebrobasilar steno-occlusive disease, dissections, vasculitis or vasculopathies, and thus raise suspicion on acute ischemia. Noninvasive monitoring of the disease is possible. By means of TCCS, hyperdense signals may suggest a hemorrhage and vascular malformations can be seen.

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Sažetak

Uloga neurosonologije u vertigu

Vertigo je čest simptom koji može predstavljati i ozbiljan neurološki poremećaj. Ako vertigo naglo nastane, može biti simptom akutnog moždanog udara.

Neurosonologija se može upotrijebiti u cerebrovaskularnih poremećaja kako bi se procijenilo stanje krvnih žila, ili u dijagnostici različitih bolesti kraniocervikalnih arterija. Mogu se prikazati aterosklerotske promjene, upalne bolesti, disekcije, vaskulopatije i vaskularne malformacije. Primjenom transkranijuskog doplera može se procijeniti intrakranijska hemodinamika, može se prikazati nastanak kolateralnih puteva u ekstra- i intrakranijakim i okluzivnim bolestima, može se ispitati cerebralna vazomotorna reaktivnost, a primjena novih softwera omogućuje detekciju mikroembolijskih signala.

Neurosonologija se može primijeniti u različitim neurološkim poremećajima koji imaju vertigo kao simptom.

Ključne riječi: Vertigo, obojeni dopler, transkranijuski dopler, karotidna arterija, vertebralna arterija