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Ultrasound-guided neuromonitoring methods in the intensive care unit

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ABSTRACT

Ultrasonography (USG) is a non-invasive, portable, bedside, reproducible, radiation-free, inexpensive, and easily accessible imaging method. It provides morphological and functional information. It allows for diagnosis, monitoring, and guiding treatment. The usage areas of USG in the ICU are broad. In interventional procedures (thoracentesis, vascular interventions, percutaneous tracheostomy), evaluation of lung pathologies (pneumothorax, pleural effusion, pulmonary edema, consolidation, A-line, B line), diaphragm evaluation, abdominal imaging (trauma, kidney, liver), diagnosis and follow-up of deep vein thrombosis and the assessment and follow-up of fluid resuscitation (IVCI) and neuromonitoring. USG-guided neuromonitoring can detect stenosis or occlusion of intracranial arteries, monitor the development of patients with vasospasm after subarachnoid hemorrhage, detect cerebral embolism, evaluate the cerebral collateral system, and determine brain death. It can also indirectly calculate intracranial pressure (ICP) and cerebral perfusion under USG guidance.

Keywords: Ultrasound, intensive care unit, neuromonitoring, intracranial pressure, optic nerve sheath diameter, transcranial doppler

INTRODUCTION

Ultrasonography (USG) is an imaging method based on the detection and conversion of ultrasound waves reflecting from tissue surfaces of different densities and converting them into images (Pulse-Echo). The introduction of USG technology into clinical use was in the 1950s. The use of USG in intensive care units has gained popularity in the last 20 years.

USG is a valuable tool because of its ability to quickly and efficiently identify or rule out specific pathologies. In the literature, especially in studies conducted in intensive care units, USG has been defined as the stethoscope of the 21st century for the evaluation of many organs.[1-3]

The goals of USG protocols for the critically ill are to be a part of the physical examination of the critically ill, to reduce complications in interventional procedures in the intensive care unit, and to predict changes in the treatment decision and treatment process.

USG is a non-invasive, portable, bedside, reproducible, radiation-free, inexpensive, and easily accessible imaging method. It provides morphological and functional information. It allows for diagnosis, monitoring, and guiding treatment. Although it has disadvantages, it has many significant advantages.

Advantages:

- Easily accessible
- Easy to apply
- Cheap
- Does not cause severe discomfort to the patient
- No risk of ionizing radiation
- Can be applied at the bedside
- Can be repeated at frequent intervals
- Does not require opaque material
- Provides functional, morphological, and physiological information
- Often crucial in the assessment of vascular flow
- Cross-sectional modalities typically only show vascular patency, whereas USG can detect flow direction, velocity, and waveform

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USG-guided neuromonitoring can detect stenosis or occlusion of intracranial arteries, monitor the development of patients

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EVALUATION OF INTRACRANIAL PRESSURE

The average intracranial pressure value is 7-15 mmHg, and ICP >20 mmHg is a high ICP. It has been reported that increased intracranial pressure is associated with increased mortality and morbidity.[7] Increased intracranial pressure (ICP) can result from various cerebral conditions, such as stroke, bleeding, malignancy, and trauma. Timely diagnosis and treatment of high ICP can prevent dramatic consequences such as cerebral ischemia and herniation and reduce mortality.

Invasive methods (intraventricular and intraparenchymal monitors) are still the gold standard in diagnosing and following increases in ICP. However, such complications as infection, bleeding, parenchymal damage, and misplacement may develop. It also requires neurosurgery expertise. Non-invasive alternative methods are recommended to be used to evaluate ICP; cranial CT and cranial MRI are the measurements of changes in tympanic membrane displacement, intraocular pressure, venous ophthalmic dynamometry, transcranial doppler and optic nerve sheath diameter (ONSD).[8]

Although non-invasive techniques such as computed tomography (CT) and magnetic resonance (MRI) can be used, these techniques are expensive, require time to achieve results, and cannot be applied at the bedside. Therefore, in unstable patients in the intensive care unit (deep hypoxia, hypotension, etc.), problems may be encountered during patient transport, limiting accessibility.[9]

This article will explain ONSD and transcranial Doppler, which can be measured by USG in intensive care neuromonitoring.

OPTIC NERVE SHEATH DIAMETER

The optic nerve is still surrounded by the dural sheath at the point where it emerges from the intracranial space into the orbital cavity. Therefore, the subarachnoid area surrounding the optic nerve is adjacent to the intracranial subarachnoid area. The anatomical relationship between the optic nerve and the subarachnoid space causes enlargement of the optic nerve sheath through the CSF in cases of increased intracranial pressure. Thus, optic nerve sheath diameter can provide information about ICP.[10,11]

According to the modified Dandy criteria, papilledema is a criterion for diagnosing idiopathic intracranial hypertension.[12] As papilledema is optic disc edema caused by high ICP transmitted through the optic nerve sheath, the increase in ONSD must occur before the development of papilledema. Therefore, ONSD helps to recognize increased ICP earlier than the presence of papilledema. Recently, the measurement of optic nerve sheath diameter (ONSD) has grown as a non-invasive, accurate, safe, reproducible, and low-cost method to evaluate ICP.

In comparative studies, ONSD among patients with high ICP was significantly higher than among patients with normal ICP. Several studies have demonstrated a correlation between invasively measured ICP and ultrasonographic ONSD diameter (ONSD) measurements, with 95% sensitivity and 92% specificity for detecting high ICP. In addition, ONSD can be measured using CT and MRI, but the accuracy of measurements made with these two methods is lower than those made with USG.[13,14]

In the study conducted by Lee et al.[15], patients with better outcomes were found in patients with an ONSD <5mm in post-cardiac arrest. They reported that it may help estimates. In their study, Chelly et al.[16] found high ONSD associated with poor prognosis and increased mortality in cardiac arrest patients. Robba et al.[17] reported that ICP changes were correlated with ONSD changes in their meta-analysis. In addition, in this meta-analysis, ONSD was also correlated with ICU mortality.[17] In another study by Robba et al.[18], patients with traumatic brain injury were followed up with ONSD, and it was reported that it was correlated with invasive ICP. In this study they developed a formula with ONSD for non-invasive measurement of ICP in their study (nICPONSD=5xONSD-14 (nICPONSD in mmHg, ONSD in mm)). In addition, the same study reported that ONSD was correlated with intensive care mortality and was an independent predictor of mortality.

Although the evaluation of changes in ICP by measuring ONSD with USG is in parallel with the results obtained with invasive methods in many clinical studies, it has been reported that the measurement is 'applicant-dependent' as the technique's weakness.[19,20] Since the accuracy of ONSD measurement with USG is related to the experience of the person measuring it, Tayal et al.[21] reported that learning to measure ONSD with USG is more accessible than learning to measure ONSD with Doppler USG or assessing papilledema. Moretti et al.[22] stated that it would be sufficient to observe ten average and three pathological optic nerve diameters to perform optic nerve USG.

It can be used with repetitive measurements both in ICP evaluation and when monitoring of ICP is required. Thus, it can assist in clinical decisions and early therapeutic interventions in neurocritical care. ONSD value in terms of clinical decision;

- If <5 mm, ICP did not increase
- If >6 mm, clinically reflects a significant increase in ICP
- Clinical correlation/presence of papilledema should be examined in ONSD values between 5 and 6 mm.[23]

ONSD should be measured 3 mm behind the optic disc, as this distance is where the optic nerve sheath is exposed to maximum diameter fluctuations due to ICP.[24] Studies have reported that the mean ONSD does not change with age, weight, or height but varies with gender.[25]

Ideal ONSD measurement technique (Figure):

- The patient is placed in the supine position.
- If the patient is conscious, the eyes are asked to close their eyelids in a neutral position (facing forward).
- Gel or sterile water is applied to the eyelids.
- Linear probes in the 7.5-10 MHz range are used for imaging according to current protocols.
- The depth on the USG device is set as 4-5 cm. This adjustment provides optimum contrast between the optic nerve sheath and the periorbital adipose tissue when focusing on the retrobulbar area.
- The transducer is placed on the eyelids, taking care not to apply pressure.
- In the retro bulbar position, the sheath diameter is measured 3 mm posterior to the globe in the longitudinal axis.

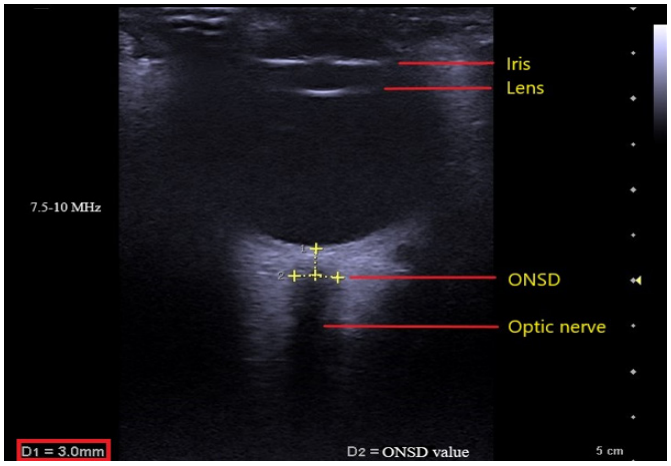

Figure. ONSD measurement technique

Two measurements should be made for each optic nerve. The first is the transverse plane in which the probe is placed horizontally; the second is the sagittal plane in which the probe is placed vertically. The result ONSD is the average of these measurements.

ONSD ultrasonography is a non-invasive, reproducible, and bedside method. This technique can be life-saving, especially when invasive ICP monitoring is contraindicated (e.g., due to coagulopathy) or when a specialist is unavailable for invasive monitor placement in an emergency.

TRANSCRANIAL DOPPLER ULTRASONOGRAPHY

Transcranial Doppler (TCD) ultrasound is a painless, non-invasive imaging method that can evaluate the brain's blood circulation using sound waves and detect medical problems that affect this circulation. With the widespread use of ultrasound in intensive care units (ICU), TCD has become an excellent alternative for patients requiring close follow-up. In particular, the fact that these patients require close monitoring, receive multiple treatments, often have unstable hemodynamics, and take critical drugs for this makes it difficult and sometimes even not permissible to transfer patients from ICU to computed tomography (CT) and magnetic resonance (MR). These problems cause time delays in the imaging of the patients, and as a result, serious issues can be experienced in diagnosis, treatment, and follow-up. TCD is an easy-to-use, reliable method that can be performed at the bedside without the need for patient transfer and does not interfere with the treatment and monitoring of patients.

TCD is mainly used in clinical practice for detecting and monitoring vasospasm in cerebral vessels in subarachnoid hemorrhage due to aneurysm rupture. In addition, ultrasound can detect stenoses and occlusions in the arterial system (Willis polygon and vertebrobasilar), evaluate the vessels in the intracranial circulation, detect cerebral microemboli, evaluate the reactivity of cerebral vasomotor activity, diagnose acute cerebrovascular diseases, evaluate arteriovenous malformations associated with encephalic sepsis, and treat encephalovenous malformations and associated brain dysfunction and encephalic sepsis-related brain dysfunction. Available. Ultrasound can be used as an auxiliary diagnostic method in confirming the clinical diagnosis of brain death.

Doppler takes its technical name from Christian Andreas Doppler, who developed a device using double-bright colored lights in Prague in 1843. In the 1950s, Satomura and Kaneko used the first Doppler, the "rheograph".[26]

Ultrasonography is applied with different methods. Information about the vessel wall, lumen width, and presence of atherosclerotic plaques can be obtained in B-mode ultrasonography and information about blood flow velocity with the Doppler technique. The principle of Doppler ultrasonography is that the radiating ultrasound waves emitted by the crystals detected by the Doppler probe applied on the vessel reflect the blood column in motion. If the reflected blood column is continuous, it is called a continuous wave; if it is in short bursts, it is called an intermittent wave. It is defined as "duplex ultrasonography," which is a combination of "real-time" imaging of the vessel (B-mode) and Doppler ultrasonography. Ultrasonography provides information about the vessel wall, while Doppler includes information about blood flow turbulence and the degree of stenosis if any.[27]

Acoustic Windows

Bone windows are areas where ultrasonographic signals can be received at a certain depth during TCD application, and the bone thickness is thinner. There are three main bone windows:

a. Orbital window: The transorbital approach can examine the bilateral ophthalmic artery and internal carotid artery siphon.

b. Temporal bone window: Bilateral anterior, middle, and posterior cerebral arteries (ACA, MCA, and PCA, respectively) can be examined with the transtemporal approach.

c. Foramen magnum window: Intracranial vertebral arteries and basilar arteries are examined with a transforaminal or suboccipital approach.[28]

Clinical Applications of Transcranial Doppler

1. Blood flow velocities:

a. Examination of extracranial vessels: Atherosclerotic lesions developing in the carotid artery bifurcation or carotid bulb may cause cerebrovascular events. The ischemic event may be due to an embolism ruptured from atherosclerotic plaque, or it may occur with hemodynamic failure. Small lesions do not affect the hemodynamic profile; however, lesions that cause narrowing of around 60% cause changes in blood flow velocities in the intracranial vessels. Minor atherosclerotic changes in the carotids result in distortions in waveforms in TCD. To summarize the findings to be obtained with TCD in carotid artery stenosis/occlusion, systolic and mean blood flow velocity and pulsatile index (PI) (PI=Systolic velocity-Distolic velocity/Average velocity) decrease in ipsilateral MCAs; if the anterior communicating artery (ACoA) is open, mean and systolic blood flow velocities in the contralateral ACAs increase and flow in the ipsilateral ACA A1 segment reverses or disappears; if the posterior communicating artery (PCoA) is open, the mean and systolic blood flow velocities of the PCA, vertebral artery, basilar artery increase and the blood flow velocity in the ophthalmic artery is reversed.[29,30]

b- Examination of intracranial vessels: Blood flow velocities in intracranial arteries can give essential findings. Generally, PI elevation is seen at low blood flow velocities. Diffuse blood flow velocity may indicate hemodynamic insufficiency; focal decreased blood flow velocity can be seen in focal arterial ectasia or proximal to the artery feeding the arteriovenous malformation. If there is stenosis in the artery, the blood flow velocity proximal to the stenosis is low, the PI is high, the blood

flow velocity is increased in the stenosis segment, and the blood flow velocity and PIs are low above the stenosis segment. Blood flow velocity changes can be detected with TCD only when the lumen narrowing exceeds 50-60%. In addition, it can be observed that while there is an occlusion in one artery due to the opening of the collaterals, the blood flow velocities in the other artery increase as a compensatory factor. For example, if ACoA is open in MCA occlusion, blood flow velocity increases in ACA and reverses blood flow velocity in the A1 segment. However, evaluating blood flow velocities at different depths and with contralateral arterial blood flow velocity is essential. For example, if the difference in blood flow velocity between the two MCAs is more significant than 30 cm/sec, there is unilateral stenosis or spasm. Focal stenosis should be considered if a blood flow velocity difference of more than 20 cm/sec is obtained in measurements made at different depths. To evaluate the difference between stenosis and vasospasm, if blood flow velocity is obtained from the same artery twice at a few days' intervals, it will be seen that the increase in blood flow velocities in stenosis remains the same. At the same time, there is a change in vasospasm.[31] The blood flow velocities of the intracranial arteries and the depths at which they are detected are shown in Table.

Table. Normal cerebral blood flow velocity (mm/s) values measured by transcranial Doppler[32]

	Systolic peak V	Diastolic end V	V mean
Middle cerebral artery	91±17	46±10	58±12
Anterior cerebral artery	86±20	41±8	53±10
Posterior cerebral artery	60±20	28±7.5	36±10
Vertebral artery/basilar artery	59±17	29±8	36±11

V: velocity

2. Detection of the presence of embolism

Ideally, the bloodstream has no significant particulate suspensions, aggregates, or bubbles. In some cases, however, such particles may be present in the circulating bloodstream. Conditions such as carotid artery disease, prosthetic heart valve, atrial fibrillation, surgical interventions such as carotid endarterectomy, and cardiopulmonary bypass pose a potential risk for arterial-to-arterial embolism. The left ventricle, aorta, and carotid are the primary sources of embolism.[33] Doppler sonography, a non-invasive examination, can be a helpful method in detecting cerebral embolism and regulating and monitoring the treatment of cerebrovascular diseases. Embolism in cerebral arteries is sought, especially in MCAs and preferably bilaterally. For the detected high-intensity transients to be accepted as a "micro embolic sign," they must last less than 300 ms, be at least 3 dB greater than the ground activity, be unilateral, and have the characteristic "chirp" sound. It is difficult to distinguish between clinical and laboratory carotid and cardiac embolisms. Although not characteristic, it can be said that carotid embolisms are more superficial and cortical and are detected unilaterally by TCD.[34,35]

3. Examination of vasospasm

Many studies have reported that vasospasm is a complication of subarachnoid hemorrhages. In mild and moderate vasospasm, blood flow velocity increases along the segment where the spasm develops due to arterial narrowing; however, cerebral perfusion does not appear to accompany it. In severe vasospasm, irreversible changes occur with impaired cerebral perfusion, and infarcts may accompany the picture. TCD is the most essential tool in the diagnosis and follow-up of vasospasm. The presence and severity of vasospasm can be routinely monitored in many clinics using TCD.[36]

4. Vasomotor reactivity and dynamic autoregulation tests

Static autoregulation tests are performed by evaluating arterial blood pressure responses to some pharmacological agents. In recent studies, dynamic autoregulation tests are taking the place of static tests. Fluctuations in arterial blood pressure were observed in dynamic tests, which were more reliable. In the most commonly used method, a cuff is attached to the calf of the patient in the supine position, and the vein is inflated with a pressure above 20 mmHg and waited for two minutes. Meanwhile, MCAs are detected at three depths, blood flow velocities are continuously displayed invasively or non-invasively, and cerebrovascular resistance (Cerebrovascular resistance=Arterial blood pressure/mean MCA blood flow velocity) is calculated. The cuff is suddenly loosened, and blood pressure, MCA velocity, and cerebrovascular resistance (CVR) are evaluated. This test gives valuable results in patients with symptoms of syncope and vertebrobasilar ischemia attack.[37]

Vasomotor reactivity testing is performed by inhaling CO_2 or assessing blood flow velocity changes after acetolosamide injection. The patient is inhaled 5% CO_2 for 2-3 minutes or is injected with acetazolamide, a carbonic anhydrase inhibitor. Ordinary individuals will have a 50% increase in MCA blood flow velocities; increases below 30% are considered pathological. With this test, the cerebral effects of hypo- or hypertension can be investigated, and objective information can be obtained about the need for surgery before carotid endarterectomy.[38,39]

5- Intracranial pressure imaging

Cerebral circulation takes place in a closed environment surrounded by the skull. This environment includes brain tissue, blood volume, and cerebrospinal fluid. An increase in one of these three compartments due to edema due to infarcts, intracranial hematoma, tumors or head trauma may result in increased intracranial pressure. Cerebral perfusion pressure is the difference between arterial blood pressure and intracranial pressure. Therefore, increased intracranial pressure may result in impaired cerebral perfusion. Transcranial Doppler is a reliable tool for monitoring intracranial pressure. Changes in waveforms make it possible to follow changes in intracranial pressure. In cases where intracranial pressure increases, diastolic blood flow velocities in the cerebral arteries decrease and PIs increase.[40]

6. Bubbles test

TCD can be used to diagnose "patent foramen ovale," a critical risk factor for stroke. In this test, salt or galactose particles are injected intravenously after MCAs are detected by TCD. Five seconds after the injection, the Valsalva maneuver is performed; if the patient has a right-left shunt, the passage of particles in the MCA bloodstream is observed during the Valsalva maneuver. Detection of the right-left shunt with the Bubbles test is more reliable than transesophageal echocardiography; its sensitivity and specificity are 90%.[41]

7. Brain death

TCD is a tool that can be used as an aid in the diagnosis of brain death. Although blood flows cannot be obtained with TCD in the case of brain death, it is possible to provide data such as loss or reversal of the diastolic blood flow wave or early and small systolic spikes.[42]

CONCLUSION

USG-guided ONSD and TCD applications are used as easy, bedside and non-invasive techniques for neuromonitoring in intensive care units.

ETHICAL DECLARATIONS

Referee Evaluation Process: Externally peer-reviewed.

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