Tidsskriftet Tidsskrift for Den norske legeforening

Transcranial ultrasound monitoring in acute stroke

PERSPECTIVES

LARS THOMASSEN

E-mail: lathom2@online.no
Lars Thomassen is a specialist in neurology, professor emeritus at the University of Bergen, a former senior consultant at the Department of Neurology and now a researcher at the Division of Clinical Neuroscience, Haukeland University Hospital.
The author has completed the ICMJE form and declares no conflicts of interest.

ANNETTE FROMM

Annette Fromm, PhD (diagnostic ultrasound), European MSc (stroke medicine) is a specialist in neurology and senior consultant at the Department of Neurology, Haukeland University Hospital.
The author has completed the ICMJE form and declares no conflicts of interest.

SANDER AARLI

Sander Aarli is a PhD candidate at the University of Bergen and works with ultrasound monitoring in the Department of Neurology, Haukeland University Hospital.
The author has completed the ICMJE form and declares no conflicts of interest.

NICOLA LOGALLO

Nicola Logallo, PhD (transcranial diagnostic ultrasound), European MSc (stroke medicine), is a specialist in neurology and senior consultant in the Neurosurgery Intensive Care Unit, Haukeland University Hospital.
The author has completed the ICMJE form and declares no conflicts of interest.

Acute stroke results in unstable cerebral blood circulation and a brain in crisis. Should we be satisfied with a snapshot X-ray image on admission, or do we also need to monitor cerebral blood circulation over time?

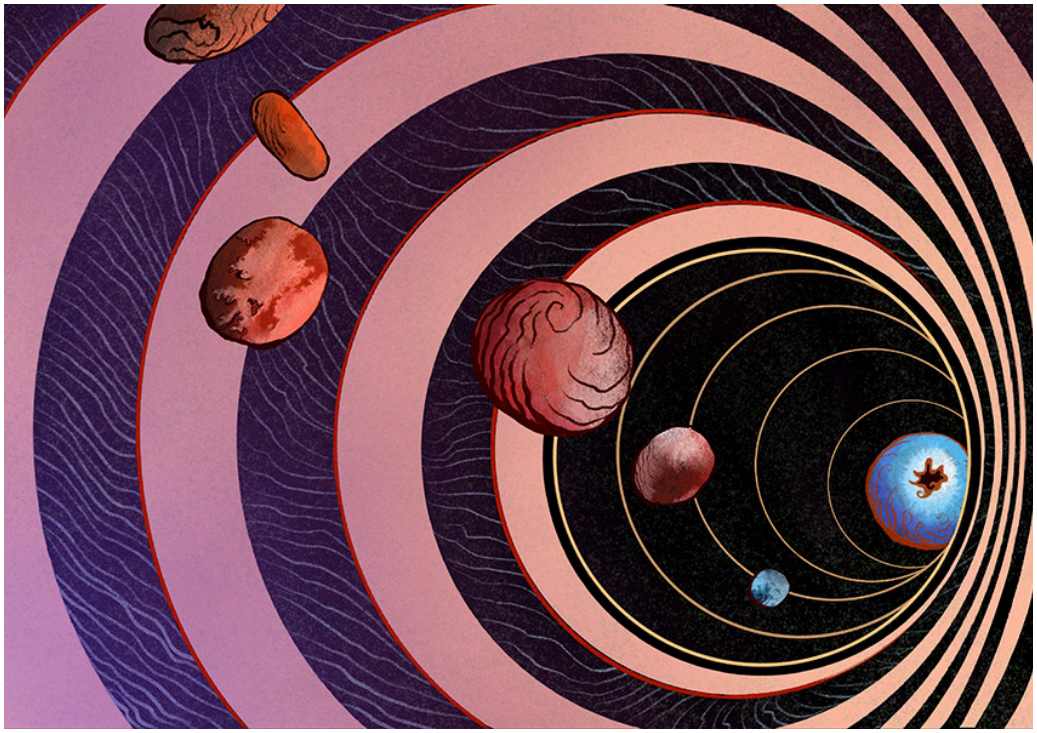

Illustration: Lisbeth Moen

When patients with symptoms of acute cerebral infarction or cerebral haemorrhage arrive at a hospital, CT or MRI diagnostic radiological imaging is performed immediately to ensure the correct acute treatment. When the patient subsequently arrives at the stroke ICU, abundant information is available on the arteries, blood circulation in the brain and brain tissue. However, stroke monitoring has traditionally been limited to monitoring support functions such as heart function, blood pressure, oxygen saturation, temperature, blood sugar and fluid balance, while cerebral circulation is not systematically monitored. The basis for further treatment therefore consists of a pathophysiological status on admission, but little or no information on vascular developments through the following critical hours.

Transcranial ultrasound

Transcranial ultrasound is a non-invasive method of bedside monitoring of cerebral circulation (1–3). Repeated duplex scans or continuous Doppler monitoring enable pathophysiological monitoring over time, which in practice cannot be done with CT or MRI. Ultrasound examinations can be adapted to the patient's disease and the severity of the disease.

The examination starts with a transcranial duplex scan, which shows colour-coded blood flow through the arteries, while a Doppler curve shows flow velocities and haemodynamics. This scan is repeated as needed. Continuous

transcranial Doppler monitoring with bilaterally or unilaterally fixed ultrasound probes (on a headband) yields a continuous Doppler curve which reflects haemodynamic changes, such as recanalisation and reocclusion (4, 5).

Cerebral infarction

Intravenous thrombolysis in the event of arterial occlusions results in extensive recanalisation, haemodynamic normalisation and clinical improvement. However, the condition is unstable, and around 10 % of patients experience early clinical deterioration. Haemodynamic factors play a key part here, but in most cases the cause remains undetermined (6). Around 30 % of patients with confirmed early recanalisation may experience reocclusion with haemodynamic crisis and clinical deterioration (7, 8). With Doppler monitoring, it is possible to follow the degree of recanalisation and any reocclusion in real time over a period of hours. Although approved guidelines state that platelet inhibitors are contraindicated for the first 24 hours after thrombolysis, Doppler findings may strengthen the indication for early antithrombotic therapy. Case reports indicate that repeated intravenous thrombolysis may also be safe in cases of clinical deterioration in the initial hours following the first dose (9).

As a rule, intra-arterial thrombectomy results in good recanalisation, but not always in adequate tissue perfusion (the no reflow phenomenon). Nor is recanalisation always unproblematic, and it may result in cerebral hyperperfusion syndrome, with haemorrhagic complications, cerebral oedema, infarct growth and clinical deterioration. Clinical symptoms are often delayed and unclear in cerebral infarction. Early haemodynamic information in the initial hours is therefore crucial (10). Doppler monitoring may reveal such haemodynamic changes, and prompt rational treatment.

A precerebral occlusion results in focally reduced blood flow, but the degree of perfusion failure and risk of cerebral ischaemia depend on the collateral circulation. A transcranial Doppler scan can show whether the circle of Willis is functionally intact as central collaterals, and whether there is flow diversion to leptomeningeal peripheral collaterals (11). The degree of collateral circulation has a bearing on the intensity of blood pressure therapy.

> ***«Ultrasonography can be used for continuous general bedside monitoring, can be used frequently and does not cause patient stress»***

In cases of precerebral occlusion and reduced cerebral perfusion, the degree of residual capacity for vasodilatation (autoregulation) may be crucial. Doppler velocity measurement in proximal artery segments during dilatation of peripheral artery segments with the aid of intravenous acetazolamide injection (Diamox test) yields answers about the degree of lost dilatation capacity (vasoreactivity) (12). In the absence of vasomotor reactivity, cerebral perfusion is passively dependent on systemic blood pressure, and the blood pressure must therefore not be lowered.

Acute cerebral infarction is generally due to thromboembolism, but the origin of the embolus is unknown. Doppler monitoring is the only means of registering circulating microemboli in vivo (13). Ongoing embolisation occurs most frequently immediately after the cerebral infarct, and the examination should therefore be conducted as early as possible (14, 15). Bilateral emboli indicate a cardiac or systemic source, while unilateral emboli point to a carotid stenosis. Detection of microemboli is thus of significance both for the choice of antithrombotic therapy and for treatment intensity.

Cerebral haemorrhage

In cerebral haemorrhage, an initially growing haematoma volume leads to increasing cerebral injury. There is very close correspondence between transcranial duplex scans of the volume of bleeding and CT scans, and repeated duplex scans provide satisfactory information about early haematoma growth and midline displacement (16, 17). The information has prognostic significance and may provide a basis for attempting haemostatic treatment or intensified blood pressure treatment.

Vasospasm and subsequent ischaemic brain damage are uncommon in primary cerebral haemorrhage, but frequent when there is also intraventricular haemorrhage (18, 19). The ventricles communicate with the subarachnoidal space, and a very unstable condition can develop, as with primary subarachnoidal haemorrhage. Neurosurgical monitoring here encompasses repeated ultrasound scans to pick up signs of increasing vasospasm and to guide nimodipine therapy (20, 21). In a stroke unit, patients with cerebral haemorrhage and intraventricular blood should be offered similar ultrasound monitoring to avoid possible additional ischaemic injury to the brain.

Monitor the brain with ultrasound

Acute neurovascular disease can be life-threatening, and immediate intensive monitoring and treatment is necessary. Milder cases are not necessarily life-threatening, but cerebral circulation is always unstable and brain function is always threatened. In the early hours, repeated radiological scans would be desirable. However, CT has limitations because of the radiation and contrast medium burden, MRI is usually not available at short notice and both CT and MRI involve moving patients. Radiological methods therefore do not lend themselves to monitoring. By way of comparison, ultrasonography can be used for continuous general bedside monitoring, it can be used frequently and does not cause patient stress. In an acute situation with rapid pathophysiological changes, transcranial diagnostic ultrasound may, however, be difficult. Diagnostic workup presupposes correct measuring methods and a thorough knowledge of neurovascular anatomy and physiology. The end product is a spectral pulse curve and physiological flow variables that must be interpreted.

These are probably some of the reasons why relatively few clinicians really master the method, and why many remain sceptical regarding the value of the test (22).

Clinical neurosonology is criticised for being very operator-dependent. The criticism underscores the need for systematic training to be incorporated into daily clinical practice and in theoretical education (23, 24). Ultrasound results are open to interpretation, but in combination with information from initial CT/MRI and a pathophysiological understanding of the complexity of vascular neurology, the results provide support for therapeutic decisions and prognostic assessments. In experienced hands, the methods provide practical useful information about and for the patient over time.

In our experience from the Norwegian Stroke Association, clinical neurosonology and transcranial ultrasound monitoring are not widely used in Norway. It is time to upgrade Norwegian stroke units to assume greater responsibility for the first critical hours of stroke.

LITERATURE

1. Naqvi J, Yap KH, Ahmad G et al. Transcranial Doppler ultrasound: a review of the physical principles and major applications in critical care. Int J Vasc Med 2013; 2013: 629378. [PubMed][CrossRef]

2. Montrief T, Alerhand S, Jewell C et al. Incorporation of Transcranial Doppler into the ED for the neurocritical care patient. Am J Emerg Med 2019; 37: 1144–52. [PubMed][CrossRef]

3. Bonow RH, Young CC, Bass DI et al. Transcranial Doppler ultrasonography in neurological surgery and neurocritical care. Neurosurg Focus 2019; 47: E2. [PubMed][CrossRef]

4. Thomassen L, Waje-Andreassen U, Naess H et al. Doppler ultrasound and clinical findings in patients with acute ischemic stroke treated with intravenous thrombolysis. Eur J Neurol 2005; 12: 462–5. [PubMed] [CrossRef]

5. Molina CA. Monitoring and imaging the clot during systemic thrombolysis in stroke patients. Expert Rev Cardiovasc Ther 2007; 5: 91–8. [PubMed] [CrossRef]

6. Tisserand M, Seners P, Turc G et al. Mechanisms of unexplained neurological deterioration after intravenous thrombolysis. Stroke 2014; 45: 3527–34. [PubMed][CrossRef]

7. Alexandrov AV, Grotta JC. Arterial reocclusion in stroke patients treated with intravenous tissue plasminogen activator. Neurology 2002; 59: 862–7. [PubMed][CrossRef]

8. Baizabal-Carvallo JF, Alonso-Juarez M, Samson Y. Clinical deterioration following middle cerebral artery hemodynamic changes after intravenous

thrombolysis for acute ischemic stroke. J Stroke Cerebrovasc Dis 2014; 23: 254–8. [PubMed][CrossRef]

9. Cappellari M, Tomelleri G, Carletti M et al. Intravenous thrombolysis on early recurrent cardioembolic stroke: 'Dr Jekyll' or 'Mr Hyde'? Blood Coagul Fibrinolysis 2012; 23: 78–81. [PubMed][CrossRef]

10. Lin YH, Liu HM. Update on cerebral hyperperfusion syndrome. J Neurointerv Surg 2020; 12: 788–93. [PubMed][CrossRef]

11. Saqqur M, Khan K, Derksen C et al. Transcranial doppler and transcranial color duplex in defining collateral cerebral blood flow. J Neuroimaging 2018; 28: 455–76. [PubMed][CrossRef]

12. Müller M, Voges M, Piepgras U et al. Assessment of cerebral vasomotor reactivity by transcranial Doppler ultrasound and breath-holding. A comparison with acetazolamide as vasodilatory stimulus. Stroke 1995; 26: 96–100. [PubMed][CrossRef]

13. Bazan R, Luvizutto GJ, Braga GP et al. Relationship of spontaneous microembolic signals to risk stratification, recurrence, severity, and mortality of ischemic stroke: a prospective study. Ultrasound J 2020; 12: 6. [PubMed] [CrossRef]

14. Best LMJ, Webb AC, Gurusamy KS et al. Transcranial doppler ultrasound detection of microemboli as a predictor of cerebral events in patients with symptomatic and asymptomatic carotid disease: A systematic review and meta-analysis. Eur J Vasc Endovasc Surg 2016; 52: 565–80. [PubMed] [CrossRef]

15. Kargiotis O, Psychogios K, Safouris A et al. The role of transcranial doppler monitoring in patients with multi-territory acute embolic strokes: A review. J Neuroimaging 2019; 29: 309–22. [PubMed][CrossRef]

16. Pérez ES, Delgado-Mederos R, Rubiera M et al. Transcranial duplex sonography for monitoring hyperacute intracerebral hemorrhage. Stroke 2009; 40: 987–90. [PubMed][CrossRef]

17. Camps-Renom P, Méndez J, Granell E et al. Transcranial duplex sonography predicts outcome following an intracerebral hemorrhage. AJNR Am J Neuroradiol 2017; 38: 1543–9. [PubMed][CrossRef]

18. Khatri IA, Verma N, Alkawi A et al. Cerebral vasospasm in intracerebral hemorrhage-case report. J Vasc Interv Neurol 2009; 2: 139–41. [PubMed]

19. Regula JU, Schill J, Ringleb PA et al. Cerebral vasospasm and delayed cerebral ischemia in intraventricular hemorrhage. Neurocrit Care 2014; 20: 460–5. [PubMed][CrossRef]

20. Kumar G, Shahripour RB, Harrigan MR. Vasospasm on transcranial Doppler is predictive of delayed cerebral ischemia in aneurysmal

subarachnoid hemorrhage: a systematic review and meta-analysis. J Neurosurg 2016; 124: 1257–64. [PubMed][CrossRef]

21. Samagh N, Bhagat H, Jangra K. Monitoring cerebral vasospasm: How much can we rely on transcranial Doppler. J Anaesthesiol Clin Pharmacol 2019; 35: 12–8. [PubMed]

22. Alexandrov AV, Sloan MA, Tegeler CH et al. Practice standards for transcranial Doppler (TCD) ultrasound. Part II. Clinical indications and expected outcomes. J Neuroimaging 2012; 22: 215–24. [PubMed][CrossRef]

23. Nedelmann M, Stolz E, Gerriets T et al. Consensus recommendations for transcranial color-coded duplex sonography for the assessment of intracranial arteries in clinical trials on acute stroke. Stroke 2009; 40: 3238–44. [PubMed][CrossRef]

24. Robba C, Poole D, Citerio G et al. Brain ultrasonography consensus on skill recommendations and competence levels within the critical care setting. Neurocrit Care 2020; 32: 502–11. [PubMed][CrossRef]

Publisert: 10 August 2021. Tidsskr Nor Legeforen. DOI: 10.4045/tidsskr.21.0180
Received 3.3.2021, first revision submitted 18.3.2021, accepted 7.4.2021.