

Transcranial Doppler in Acute Stroke Patients Undergoing Endovascular Therapy: Are we Underutilizing the Weapon?

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DEAR EDITOR,

Endovascular therapy has shown long-term benefits in acute large-core established infarcts [1] and in acute medium vessel occlusion [2].

For post-acute stroke intervention in the first 24 hours, current standards recommend keeping the diastolic blood pressure (DBP) below 105 mmHg and the systolic blood pressure (SBP) below 180 mmHg. But with the rise of endovascular therapy and the inclusion of more patients within the eligibility criteria for acute stroke management, are the existing guidelines for blood pressure management in acute stroke patients satisfactory enough? In fact, after the endovascular therapy, the blood pressure target is still lacking a definite guideline. There are both advantages and disadvantages to setting higher or lower blood pressure targets after an endovascular procedure, and it may be challenging to establish a universally acceptable range for all cases. Some research suggests that, on one hand, aggressively lowering SBP might be disadvantageous, and on the other hand, a peak SBP above 160 mmHg in the first 24 hours is linked to worse functional results [3].

Understanding of the cerebral hemodynamics in acute stroke is improving with time. And, we may need to adjust our actions on an individual basis during pre- and post-acute stroke interventions by utilizing this knowledge to improve the potential outcomes. In patients with large vessel occlusion (LVO) stroke and adequate baseline perfusion, a higher SBP dropmax (maximal decrease in systolic blood pressure over two consecutive measurements) before recanalization is significantly associated with an increased infarct

proportion (the volume of final infarct relative to the initial area at risk) and functional recovery in patients. As regional perfusion pressure decreases after LVO, the pial artery dilates to provide maximal blood flow within its capacity, and perfusion from leptomeningeal collaterals saves the ischemic penumbra. The cerebral autoregulation capacity in this region is likely already depleted, meaning that cerebral perfusion pressure will be the sole driving force of regional cerebral blood flow (CBF), with blood pressure serving as a significant determinant of this perfusion pressure [4]. In the presence of moderate LMC, tissue viability primarily relies on systemic perfusion, necessitating clinicians to be attuned to even little fluctuations in blood pressure. [4] Simultaneously, patients with superior or adequate collateral circulation may exhibit greater tolerance to moderate blood pressure variations. Truly, understanding the status of the intracranial and extracranial vasculature and collaterals is insufficient, as it is a dynamic phenomenon and there is a constant change in the equation of demand and supply. For example, the status of LVO may change due to re-occlusion after endovascular therapy (EVT), and cerebral autoregulatory capacity may suddenly fail; therefore, it is essential to assess the status of leptomeningeal perfusion, which maintains blood flow during this critical period. All these factors highlight the importance of managing blood pressure and understanding both the anatomical and functional status of the leptomeningeal circulation (LMC) for achieving successful outcomes in acute stroke treatment after EVT.

Now, transcranial Doppler (TCD) has the capacity of understanding the dynamic changes in these

anatomical substrates, helping us to decide on blood pressure targets for these individuals, especially when situations become more complex, like determining target blood pressure after EVT in large core infarcts.

We know hemorrhagic transformation is a primary cause of early neurological decline following endovascular therapy (EVT). Earlier, it was believed that lower blood pressure after a successful mechanical thrombectomy could prevent hemorrhagic conversion of the infarct. But our present understanding is that haemorrhagic transformation of the infarct is probably due to microvascular ischemic injury that accumulated before recanalization. Hence, it is inferred that we need a better way to predict hemorrhagic conversion of the infarct, as it has a massive influence on the management and prognosis of the patient.

The role of TCD again comes into play here. Following complete recanalization, hyperperfusion may elevate the risk of cerebral edema, particularly in the vicinity of the infarct zone, potentially resulting in hemorrhagic transformation. The diagnosis of hemorrhagic transformation can be complex, as computed tomography findings post-EVT may be due to the extravasation of contrast dye. TCD can assist in identifying patients predisposed to hemorrhagic transformation. For example, detecting a 30% increase in mean blood flow velocity of the affected middle cerebral artery within 72 hours post-successful EVT significantly correlates with hemorrhagic transformation and adverse clinical outcomes at 90 days.^[5] All instances of hyperemia, especially those accompanied by high blood pressure, led to hemorrhagic transformation, as precisely detected using TCD [5]. Thus, in complex patients exhibiting early neurological decline, we can employ TCD to detect hemorrhagic change.

This method can also help us identify those patients who will deteriorate post-EVT and require hemicraniectomy. Early prediction of intractable intracranial pressure and early craniectomy may be life-saving. This is because these patients deteriorate fast, and a sudden decompensation may force the treating team to end up in a helpless situation, especially in odd hours where a staff crisis is a possibility.

In summary, TCD is a safe (no radiation exposure), feasible, bedside, less costly approach that can provide a fund of information during the acute stroke

period without hindering patient care. Post-EVT, this method can assist neurologists in evaluating recanalization success and the extent of reperfusion based on real-time hemodynamic data. It can identify patients who are in crisis and also help us determine a treatment plan. However, there needs to be easy access to the machine and, more importantly, a trained staff able to perform and interpret TCD findings confidently. Large clinical observational studies and clinical trials are welcome to determine the role of TCD in blood pressure targets in acute stroke patients on an individualized basis, whether they undergo acute stroke intervention or not. In reference to that context, there needs to be widespread availability of TCD machines and popularization of TCD training programs to ensure confident stroke management and successful outcomes.

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