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Transcranial Doppler in Neurointervention: Applications in Endovascular Thrombectomy and Carotid Artery Stenting

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Recent advances in neurointerventional strategies, including the endovascular thrombectomy (EVT) and carotid artery stenting (CAS) techniques, have revolutionized ischemic stroke management by expanding treatment options and improving patient outcomes. Conventional transcranial Doppler (TCD), a tool long used in stroke care, lacks the ability to directly visualize vessels, limiting its application. However, TCD is still valuable as a non-invasive adjunctive monitoring strategy in neurointervention, owing to its ability to provide continuous, real-time bedside hemodynamic monitoring, offering precise numerical data, high repeatability, low invasivity, and no risk of radiation or contrast agent exposure. This unique capacity for continuous monitoring is particularly useful when integrated with artificial intelligence (AI) for data interpretation. This review explored the potential of TCD as an adjunct tool in neurointervention, emphasizing its roles in EVT and CAS. In EVT, TCD aids to evaluate post-recanalization blood flow, predicting clinical outcomes by assessing cerebral autoregulation and collateral status, and identifying patients at risk of hyperperfusion syndrome. TCD can further aid in stroke risk assessment in patients with asymptomatic carotid stenosis, as well as the selection of candidates for CAS by detecting microemboli, assessing the cerebrovascular reserve, and evaluating ophthalmic artery flow reversal. It can also be used to detect cerebral hyperperfusion following CAS. The utility of TCD extends to other endovascular procedures beyond neurointerventions, where automated and AI-assisted devices enhance its real-time intraoperative neuromonitoring abilities. This review discusses the potential of TCD to refine patient selection, predict outcomes, and enhance the efficacy of neurointerventional procedures.

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INTRODUCTION

In recent years, the role of neurointervention techniques, such as endovascular thrombectomy (EVT) and carotid artery stenting (CAS), in ischemic stroke management has significantly expanded owing to the development of advanced devices, improved outcomes, and the accumulation of supporting evidence.[1] EVT is currently considered a cornerstone treatment for acute ischemic stroke in patients with large vessel occlusion (LVO), if provided within 24 hours of symptom onset.[2] Furthermore, the utility of EVT has been extensively investigated in patients with more challenging clinical presentations, such as posterior circulation occlusion,[3] medium vessel occlusion,[4] mild neurological symptoms,[5] and lesions with a large ischemic core.[6] While carotid endarterectomy (CEA) remains the standard therapy for carotid revascularization, CAS has recently emerged as a comparable alternative.[7-10]

Transcranial doppler (TCD) has a long history of use in the management of patients with ischemic stroke, as a tool for the evaluation of cerebral vessels by allowing real-time bedside hemodynamic monitering.[11] However, conventional TCD has limitations; most notably that it cannot directly visualize vessel anatomy, instead only providing indirect flow data. In addition, the TCD is operator-dependent, while the lack of acoustic windows remains a technical challenge. These limitations, along with the growing availability of advanced imaging modalities such as computed tomography angiography (CTA) and magnetic resonance angiography (MRA), have reduced its clinical utility. Despite these drawbacks, TCD has significant potential as a valuable non-invasive adjunctive technique in the field of neurointervention. Specifically, TCD allows for continuous, real-time bedside monitoring of hemodynamic information, thus providing exact numerical values, and offering the significant advantages of being non-invasive, repeatable and free of radiation or contrast agents.[11] Moreover, continuous hemodynamic measurements can offer more dynamic and actionable insights compared to static angiographic imaging, particularly when interpreted using recent advance in artificial intelligence (AI).[12] In fact, the continuous monitoring of numerical values is becoming increasingly important in various clinical fields, including blood glucose monitoring,[13] blood pressure management,[14] and electroencephalogram analysis. Similarly, the ability of TCD to provide ongoing, real-time hemodynamic data makes it a valuable tool for neurointervention.

This narrative review examines the potential utility and applications of TCD as an adjunct tool within the standard care pathway for stroke patients in the context of modern neurointervention, specifically focusing on the following:

1. EVT
   1) Evaluation of blood flow following recanalization
   2) Monitoring post-hyperperfusion
2. CAS
   1) Enhancing the selection of patients with asymptomatic CAS.
   2) Detecting cerebral hyperperfusion syndrome
   3) Evaluating microemboli in CAS and other fields

Overall, this review highlights the role of TCD in improv-

Grade 0: **Absent**	- absent flow signals are defined by the lack of regular pulsatile flow signals despite varying degrees of background noise.
Grade 1: **Minimal**	- systolic spikes of variable velocity and duration - absent diastolic flow during all cardiac cycles based on a visual interpretation of periods of no flow during end diastoli. Reverberating flow is a type of minimal flow
Grade 2: **Blunted**	- flattened systolic flow acceleration of variable duration compared to control. - positive end diastolic velocity and pulsatility index < 1.2.
Grade 3: **Dampened**	- normal systolic flow acceleration - positive end diastolic velocity - decreased mean flow velocities (MFV) by >30% compared to control
Grade 4: **Stenotic**	- MFV of >80 cm/s AND velocity difference of >30% compared to the control side or - if both affected and comparison sides have MFV <80 cm/s due to low end-diastolic velocities, MFV >30% compared to the control side AND signs of turbulence.
Grade 5: **Normal**	- <30% mean velocity difference compared to control - similar waveform shapes compared to control

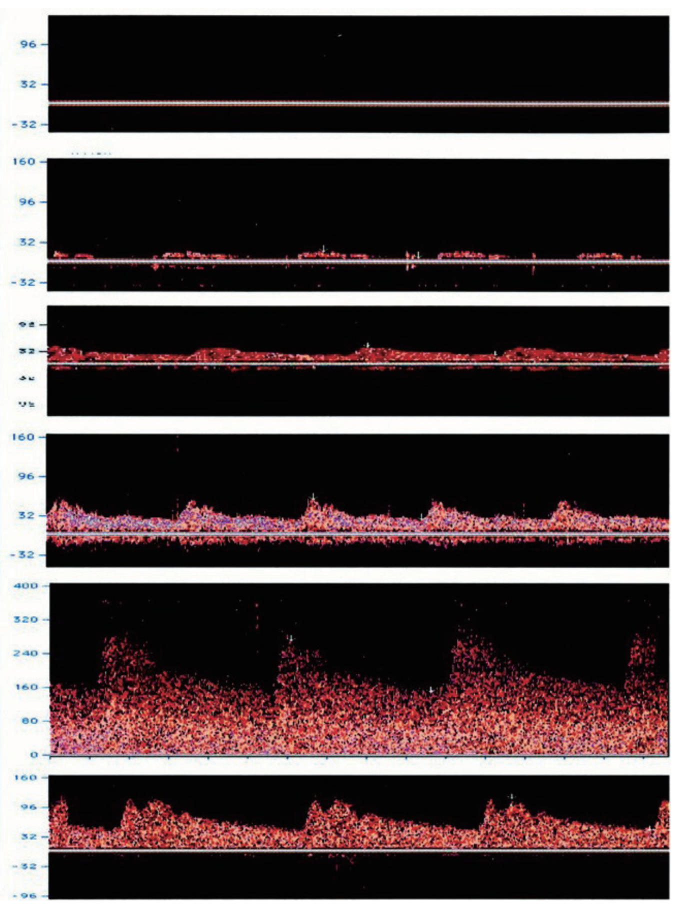

Fig. 1. Overview of the Thrombolysis in Brain Ischemia (TIBI) classification. Fig. 1 was obtained from Demchuk et al.[18], and has been included here with permission from Wolters Kluwer Health, Inc.

ing patient outcomes following neurointerventional procedures.

THE ROLE OF TCD IN EVT

Among patients with acute LVO, the evaluation of the vascular anatomy, including identification of the exact location and shape of the occluded vessel and assessment of the aortic arch structure to evaluate accessibility; monitoring of ischemic damage using the Alberta Stroke Program Early CT Score (ASPECTS) on non-contrast CT; and assessment of the core-penumbra mismatch using perfusion imaging are all critical aspects in the current era of EVT.[1,2] However, all of this information, which essential for guiding treatment decisions, cannot be provided solely by TCD, which limits its role as a standalone modality in EVT. Nevertheless, TCD still plays a valuable role in neurointervention. As TCD can be used to detect middle cerebral artery (MCA) occlusion prior to EVT, it has been recommended as a useful aspect of acute stroke care during intravenous thrombolysis (IVT), as it does not cause treatment delays, and has been linked to better functional outcomes.[15] However, it is important to note that most investigations studying the role of TCD in acute stroke management were conducted before the widespread adoption of EVT. However, although these earlier studies focused on IVT, they still offer indirect insights into the potential utility of TCD in conjunction with EVT. More recent studies conducted in the EVT era have provided further data to explore the evolving role of TCD.

1. Blood flow after recanalization

1) Thrombolysis in Brain Ischemia classification

Successful angiographic recanalization, graded using the Treatment in Cerebral Ischemia (TICI) scale on angiography, is a critical determinant of prognosis among patients with LVO.[16] However, some patients still experience poor outcomes despite achieving a good recanalization status, thereby highlighting the need for additional imaging markers.[17] The recanalized vessel status following EVT is one potential marker, which may be associated with complications and could thus help to predict prognosis. The MCA flow is graded in this context. Comparison with the contralateral MCA flow is necessary to ensure accurate

Table 1. Summary of the selected TCD parameters applicable in EVT

Index	Patients	Independent variable	Outcome	Ref
TIBI grading				
	215 patients anterior EVT, TICI 2b–3	TIBI 0-4 (vs. TIBI 5)	Poor mRS score (3–6) at 3 months: OR 2.4 (95% CI 1.3–4.6)	19
	155 patients anterior EVT, TICI 2b–3	TIBI 0-3 (vs. TIBI 4-5)	Favorable mRS (0–2) at discharge: OR 0.09 (95% CI 0.01–0.81)	20
Leptomeningeal collateral flow	15 patients MCA EVT, TICI 0–3	Leptomeningeal flow at a depth of 20–30 mm via a temporal window	Early neurological deterioration, hemorrhagic transformation (descriptive data only)	27
MCA MBF velocity index				
	123 patients anterior EVT TICI 2b-3	>30% increased MCA MBF velocity index (vs. contralateral)	ICH: OR 3.6 (95% CI 1.1–13.2) Poor 3 months mRS (3–6): OR 3.2 (95% CI 1.1–9.7)	28
	226 patients Anterior EVT TICI 2b–3	>30% increased MCA MBF velocity index (vs. contralateral)	Large infarct size (>2/3 MCA territory): OR 4.3 (95% CI 1.9–9.4) ICH: OR 2.7 (95% CI 1.4–5.3) **equivocal poor 3 months mRS Severe hyperperfusion on MRI: sensitivity 86%, specificity 62%	29

CI, confidence interval; EVT, endovascular therapy; ICH, intracerebral hemorrhage; MBF, mean blood flow; MCA, middle cerebral artery; MRI, magnetic resonance imaging; mRS, modified Rankin Scale; OR, odds ratio; TCD, transcranial Doppler; TICI, Thrombolysis in Cerebral Infarction; TIBI, Thrombolysis in Brain Ischemia.

assessment, while patients with high-grade stenosis or occlusion of the contralateral internal carotid artery (ICA) or MCA should be excluded. In 2001, the Thrombolysis in Brain Ischemia (TIBI) classification, which requires the use of TCD to non-invasively monitor residual flow signals in the intracranial vessels, was developed in patients treated with IVT (Fig. 1).[18] This score could also be employed in patients with EVT. In one study of 215 patients with anterior LVO, the TIBI classification was evaluated within 72 hours of EVT (median 8 hours).[19] Among patients who achieved successful recanalization (TICI 2b–3), 36% exhibited abnormal sonographic MCA flow (TIBI 0–4), which was associated with a 2.4-fold increased risk of poor 90-day outcomes compared with patients with TIBI grade 5 (OR 2.4, CI 1.3–4.6) (Table 1).[19] In another study of 155 patients with successfully recanalized anterior LVO, 35 (23%) had TIBI grade 4 and 19 (12%) had TIBI grade ≤3;[20] the latter grading was associated with a lower likelihood of both favorable discharge mRS (OR 0.09, 95% CI 0.01–0.81) and favorable destination (OR 0.22, 95% CI 0.07–0.71) (Table 1).[20] Other TCD predictors of recanalization include increased end-diastolic velocity (EDV), microembolic signals distal to the occlusion, flow distal to the occlusion site, absence of tandem occlusion, and good collateral status.[11]

2) Cerebral autoregulation impairment

Cerebral autoregulation is defined as the ability of the cerebral vasculature to maintain a stable cerebral blood flow in the face of fluctuations in systemic blood pressure.[21-23] This mechanism protects the brain from ischemic and hyperemic damage. Cerebral autoregulation is impaired post-stroke, consequently causing both reperfusion injury and infarct expansion. Given that cerebral autoregulation can be used to assess how cerebral perfusion pressure responds to changes in systemic blood pressure, with static autoregulation referring to steady-state responses, TCD assessments traditionally utilize linear mathematical models to evaluate the coherence, gain, and phase lead of the cerebral blood flow relative to the blood pressure fluctuations.[24]

In one study of 107 patients undergoing EVT, TCD revealed that impairments in cerebral autoregulation, defined by the extent of the linear relationship between arterial pressure and cerebral blood flow fluctuations, were observed 24–96 hours after the last normal measurement.[25] These impairments gradually recovered thereafter, influencing both the ipsilateral and contralateral hemispheres, and thus indicating global autoregulation dysfunction. If autoregulation is intact, changes in the arterial blood pressure can cause minimal changes in cerebral blood flow velocity, which could explain why recent randomized trials have failed to show any benefit at reducing the risk of hemorrhage from hyperperfusion by actively controlling systemic blood pressure in patients following successful EVT.[26] Such impairments in autoregulation were more pronounced in patients with incomplete recanalization (TICI 2a or 2b) compared to those achieving complete recanalization (TICI 3).[25] Considering these aspects, TCD could serve as a gatekeeping test to guide patient selection (e.g., those with incomplete recanalization who may have poor cerebral autoregulation) for future trials targeting post-EVT reperfusion injury. However, this issue warrants further investigation to ensure clarity.

3) Leptomeningeal collaterals

Early neurological deterioration remains a significant concern following successful recanalization, while TCD can help identify patients at risk of deterioration. A comparison of TCD performed in the emergency room before and immediately after (within 60 min) EVT can efficiently evaluate early re-occlusion and hyperemia. Additionally, leptomeningeal collaterals (assessed using TCD at a depth of 20–30 mm in the temporal window) and elevated blood pressure were both associated with hemorrhagic transformation.[27] Active collateral flows on TCD following successful EVT (TICI 2b–3) were frequently observed in patients with post-stroke disabilities (Table 1).[27]

2. Post-reperfusion hyperperfusion

The MCA mean blood flow (MBF) velocity index, measured using TCD, can be used to assess hyperperfusion following EVT. To account for inter-individual physiological differences in blood flow velocities, and to exclude factors that could influence individual MBF, patients with contralateral high-grade ICA/MCA stenosis or occlusion should be excluded. Subsequently, the index can be calculated as the MBF velocity of the ipsilateral MCA divided by that of the contralateral MCA, as in the formula below:

$$\text{MCA MBF velocity index} = \frac{\text{recanalized MCA MBF velocity}}{\text{contralateral MCA MBF velocity}}$$

In one study, among 123 patients with anterior LVO, those with ICH had a 30% increased MCA MBF velocity index (1.32 vs. 1.02, p<0.001). A higher MCA MBF velocity index was associated with the occurrence of ICH (OR 3.6, 95% CI 1.1–13.2) and poor modified Rankin scale (mRS) outcomes at 3 months (mRS 3–6; OR 3.2, CI 1.1–9.7) (Table 1).[28] Based on these results, in a further prospective study of 226 patients, a high MCA MBF velocity index of >1.3 was found to be associated with a larger infarct size (≥2/3 of MCA territory, OR 4.3, 95% CI 1.9–9.4, p<0.001), and risk of intracranial hemorrhage (HT2–PH2, OR 2.7, 95% CI 1.4–5.3, p=0.004), indicating that an increase in MBF of more than 30% could be an optimal threshold to predict parenchymal hyperperfusion (Table 1).[29] Furthermore, this factor was found to be associated with hyperperfusion on MR perfusion imaging (sensitivity 86%, 95% CI 0.64–0.95; specificity 62%, 95% CI 0.49–0.74; diagnostic accuracy 76%) (Table 1).[29] Nevertheless, these results require cautious interpretation, as hemodynamic changes in the recanalized MCA may be induced by local vasospasm due to the interventional procedures.[30]

THE ROLE OF TCD IN CAS

1. TCD as an adjuvant method to guide decisions regarding stenting in patients with asymptomatic carotid stenosis

Carotid revascularization is recommended for patients with symptomatic carotid stenosis >50% or asymptomatic carotid stenosis >60–70%.[8-10,31] While CEA remains the standard therapy for carotid revascularization, CAS has also emerged as a comparable alternative.[7-9] However, unlike in patients with symptomatic stenosis, the role of CAS in asymptomatic patients remains uncertain. Further, the optimal treatment for asymptomatic carotid stenosis remains debatable, while the criteria for selecting patients who could benefit from CAS are still largely unclear.[31] Currently, most imaging-based risk assessments for stroke rely on the degree of arterial narrowing, while the degree of stenosis remains the primary criterion for deciding whether to perform CAS in current clinical practice. Conventionally, 60–70% stenosis is the cut-off for CAS in patients with asymptomatic carotid stenosis.[8,9,31] However, additional information from TCD may be helpful in deciding whether to perform CAS in patients with severe

Table 2. Adjuvant TCD parameters helpful in identifying CAS in patients with carotid stenosis

Parameter	Study design	Patients	Supportive findings	Ref
Microemboli	Prospective cohort F/U 2 years	467 patients with asymptomatic carotid stenosis ≥70%	Ipsilateral stroke and TIA: HR 2.54 (95% CI 1.20–5.36, p=0.015).	32
CVR impairment	Meta-analysis of cohorts F/U 33 months	991 patients with high-grade symptomatic and asymptomatic carotid stenosis	Stroke: OR 4.78 (95% CI, 2.71–8.42)*	34
Reversed OA flow				
	Retrospective, cross-sectional study	81 patients with pathology measured ICA stenosis	Residual lumen of <1.5 mm at the origin of the ICA: specificity 100%, sensitivity 31%	35,36
	Retrospective, cross-sectional study	84 patients with digital subtraction angiography	Severe (≥70%) carotid stenosis: specificity 100%, sensitivity 48%	36

This association was observed only in meta-analyses; as such, these results require careful interpretation.
CAS, carotid artery stenting; TCD, transcranial Doppler; CVR, cerebrovascular reserve; F/U, follow-up; HR, hazard ratio; CI, confidence interval; TIA, transient ischemic attack; ICA, internal carotid artery; OR, odds ratio; OA, ophthalmic artery.
*The meta-analysis was conducted in patients assessed using both TCD and nuclear medicine scintigraphy, whereas the provided value was derived from a subset of patients evaluated using TCD alone. A preplanned sub-study of 106 patients from the ACES failed to demonstrate an association between impaired CVR and stroke risk.[34]

asymptomatic carotid stenosis by providing supplementary data to classify patients at high-risk of stroke, in addition to the degree of stenosis.

1) Micro-emboli detection

The detection of asymptomatic embolization on TCD can help to identify patients with asymptomatic carotid stenosis at a higher risk of stroke, and may further be beneficial in selecting patients likely to benefit from CAS.[32] In the Asymptomatic embolization of prediction of stroke in the Asymptomatic Carotid Emboli (ACES) study, a prospective observational study of 467 patients with asymptomatic carotid stenosis >70%, embolic signals on TCD were documented in 77 patients.[32] Over 2 years of follow-up, this patient cohort had a 2.5-fold higher risk of ipsilateral stroke and transient ischemic attack (TIA) (HR 2.54, 95% CI 1.20–5.36, p=0.015) (Table 2). Based on the results of repeated TCD evaluations performed every 6-months, the presence of embolic signals on TCD was associated with a 2.6-fold higher risk of ipsilateral stroke and TIA (HR 2.63, 95% CI 1.01–6.88, p=0.049) in the 6-months following evaluation, compared to those without embolic signals. These associations were stronger when the focus was placed solely on stroke.

2) Cerebrovascular reserve

As previously described, cerebral autoregulation of the brain refers to the physiological response that adjusts the resistance of cerebral blood vessels to maintain a constant degree of cerebral blood flow, despite fluctuations in blood pressure.[21-23] This resistance is primarily controlled by arterioles, which dilate to compensate and sustain cerebral blood flow when perfusion pressure decreases. In other words, if patients with severe asymptomatic carotid stenosis have hemodynamically- and functionally-problematic stenosis, they may lack the reserve required to maintain autoregulation, subsequently developing an impaired cerebrovascular reserve (CVR), placing them at a higher risk of stroke.

In one study of 60 patients with CAS, patients were stratified based on when TCD was performed: the day prior CAS, within 6 h after CAS, and 30 days later.[33] Without using acetazolamide or CO2 inhalation; an increase in arterial PCO2 was induced by having patients hold their breath for a minimum of 15 s following normal inspiration. An increase of <20% in the basal MCA MBF velocity was considered indicative of impaired CVR. CAS resulted in a significant improvement in CVR from 26.0% to 37.0% on the side ipsilateral. In a separate meta-analysis of 991 patients with high-grade ICA stenosis or occlusion (including the ACES study), baseline CVR impairment was associated with a 3.9-fold increased risk of stroke (OR 3.86, 95% CI 1.99–7.48) over a mean follow-up of 32.7 months in the whole population, regardless of symptomatic or asymptomatic status, and this association remained consistent in patients with TCD (OR 4.78, 95% CI 2.71–8.42) (Table 2).[34] However, this result requires careful interpretation, as no such association was found in the preplanned sub-study of the ACES study involving 106 patients.[34]

3) Reversal of ophthalmic artery flow and other TCD parameters

Invasive digital subtraction angiography (DSA) is the gold standard for stenosis measurement. However, to avoid unnecessary invasive angiography and enable one-stage CAS, the degree of stenosis could be easily assessed using carotid Doppler imaging, CTA, or MRA. TCD may also provide indirect but valuable information for the evaluation of the degree of stenosis. Among the various measurement parameters, the most important and easily applicable is ophthalmic artery (OA) flow reversal, as it is simple to measure, intuitive, requires no additional calculations, and shows a high specificity (100%) at predicting severe carotid stenosis.[35-37]

Among 81 patients with severe carotid stenosis confirmed by lumen diameter calculation from *en bloc* endarterectomy specimens, the strongest indicator of hemodynamically significant stenosis (defined as a residual lumen diameter of <1.5 mm at the origin of the ICA) was reversed ipsilateral OA flow (100% specificity, 31% sensitivity) (Table 2).[35] Additionally, a >35% difference in ipsilateral MCA peak systolic velocity relative to the contralateral MCA, or a >50% difference in the contralateral anterior cerebral artery (ACA) peak systolic velocity relative to the ipsilateral ACA, showed a 100% specific at identifying a residual lumen diameter of <1.5 mm. The sensitivities of these modalities were 32% and 43%, respectively.[35] In one study of 84 patients with DSA-confirmed severe carotid stenosis, OA flow reversal showed 100% specificity and 48% sensitivity.[36] The combination of TCD findings including OA and ACA reversal, low MCA flow acceleration (<290 cm/s2), and low pulsatility index (<0.88), were found to be associated with ≥70% carotid stenosis on DSA.[36] Flow acceleration was further calculated as the difference in velocity between the minimum at the systolic upstroke of the Doppler waveform and the maximum at the systolic peak of Doppler waveform, divided by the time differential. The pulsatility index was further calculated as the difference between the peak systolic and end-diastolic velocities, divided by the mean velocity.

2. Cerebral hyperperfusion syndrome after CAS

The TCD can be used to evaluate the post-CAS hyperperfusion. Cerebral hyperperfusion syndrome (CHS), defined as an increase in cerebral blood flow of >100% compared to baseline on perfusion imaging, TCD, or single-photon-emission CT, is characterized by clinical features and the evidence of hyperperfusion.[21] CHS presents with ipsilateral headache, seizure, and focal neurologic deficit, and can result in severe brain edema, cerebral hemorrhage, and even death. It occurs in 0–3% of cases following carotid revascularization. One meta-analysis of 4,446 patients following carotid revascularization reported incidences of 1.16% and 0.74% for CHS and ICH, respectively.[23] The exact underlying mechanism is still unclear; however, it is believed to result from impaired cerebral autoregulation.[21-23] In settings of chronic low-flow due to severe carotid stenosis, cerebral autoregulation trigger maximum downstream vasodilation. Eventually, these vessels lose their ability to autoregulate; subsequently, after blood flow is restored following CAS, they lose the ability to constrict themselves in response to an

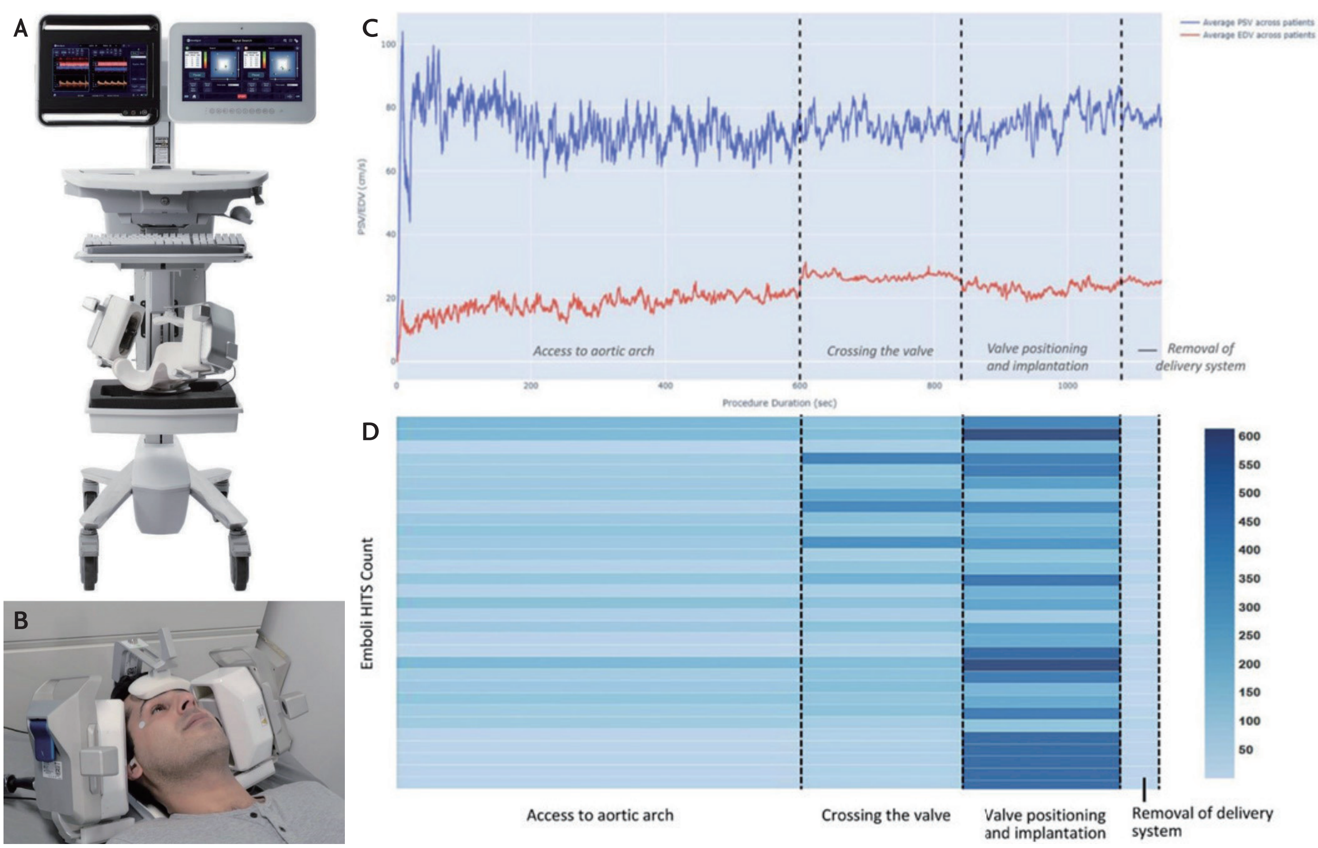

Fig. 2. Set up of the robotic transcranial doppler and example of an analysis workflow. (A) Photograph of the robotic probe pods and Doppler robotic unit on the TCD cart NovaGuide (NovaSignal Corp, Los Angeles, CA, USA). (B) Photograph of the head cradle, showing the probe pods positioned on both sides of the subject in the supine position for examination. (C) Cerebral perfusion parameters. Line graph showing the mean PSV and EDV throughout the entire procedure. (D) Heat map showing varying frequencies of emboli HITS during the four critical phases of the procedure. (A) and (B) were adapted from O'Brien et al.[47], and have been included here with permission from the Korean Society of Neurosonology. (C) and (D) were adapted from Baig et al.[12], and have been included here with permission from the *British Medical Journal*. TCD, transcranial Doppler; PSV, peak systolic velocity; EDV, end-diastolic velocity.

increase in systemic blood pressure, resulting in cerebral hyperperfusion, loss of vessel integrity, disruption of the blood–brain barrier, and eventually, cerebral edema and/or cerebral hemorrhage. Autoregulatory dysfunction typically recovers within a few days to weeks, which overlaps with the timeframe for CHS occurrence; while most cases develop within the first few days following revascularization, delayed presentation is also possible. Because the diameter of the MCA is not altered by autoregulation, changes in the flow velocity show a strong correlation with changes in MCA perfusion, which makes TCD a good tool for predicting CHS.[21] However, it is worth noting that an increase in the peak systolic velocity of >100% on TCD demonstrated high specificity (93.6%), but low sensitivity (47.6%).[38] This indicates that the classical criterion of a >100% increase compared to baseline as evidence of hyperperfusion for diagnosing CHS may result in the underdiagnosis of this severe complication. Further studies and the development of improved measurement methods and predictive tools are required to enhance the accuracy of CHS predictions.

3. Microemboli evaluation in CAS and other fields

The detection of microemboli on TCD has various applications in addition to patient selection for asymptomatic carotid stenosis. Continuous TCD monitoring during CAS can help to identify the stages of the procedure that produce more embolic material, thus guiding surgeons to proceed with greater caution. In one study involving 30 patients with CAS using a distal protection device (Filter Wire EZ Embolic Protection System; Boston Scientific, Natick, MA, USA), both malignant and micro emboli were most frequently observed during stent deployment, followed by deployment of the protection device.[39] Malignant emboli detected using continuous TCD monitoring during CAS were also significantly associated with post-procedure diffusion-weighted imaging lesions.[39] In another study which considered emboli with a relative energy index of micro signal >1.0 as malignant, indicating that embolic signals could also be evaluated quantitatively.[40]

Microemboli detection can be applied to various procedures, and may help to direct future procedural innovations. The efficacy of embolic protection devices in trapping emboli could be evaluated using dual-probe TCD, with probes placed both before and after the protection device.[41] One early study of transcarotid artery revascularization (TCAR) in 2006 demonstrated the feasibility of this technique by showing a reduction in microembolic signals during TCAR compared to conventional transfemoral CAS, using continuous TCD monitoring.[42] Although no study has yet conducted a direct head-to-head comparison between TCAR and transfemoral CAS, the theoretical expected benefit of TCAR would include a decreased risk of embolic infarctions by avoiding unprotected passage through the stenosis site and employing a physiologically superior protection method.[43] Furthermore, one recent paper proposed a protocol for the real-time monitoring of MCA blood flow using intraoperative TCD monitoring during TCAR.[44]

Novel techniques beyond the field of neurointervention may also be assessed using TCD. Continuous TCD monitoring has previously been employed to evaluate the efficacy of cerebral protection devices in various endovascular procedures, including thoracic endovascular aortic repair and transcatheter aortic valve implantation.[45,46] Recently, a fully automated robotic TCD system, termed the NovaGuide (NovaSignal Corp., Los Angeles, CA, USA), was developed to enhance operability and analysis, thereby offering the potential to expand TCD applications in clinical settings (Fig. 2A, 2B).[47] Following the integration of continuous data and AI interpretation, real-time and continuous intraoperative neuromonitoring could be performed more easily and feasibly. For example, one recent study evaluated the efficacy of a protection device based on reductions in microemboli, using continuous TCD in conjunction with AI interpretation (Fig. 2C, 2D).[12]

In conclusion, our results show that TCD serves as a valuable adjunct tool in neurointervention, by enhancing the clinical outcomes of EVT and aiding in better candidate selection for CAS among patients with asymptomatic carotid stenosis. Its ability to provide real-time hemodynamic data and non-invasive monitoring underscores its significant utility. Future studies should explore the role of TCD in optimizing neurointervention outcomes, particularly with advancements in automated and AI-integrated monitoring systems.

Ethics Statement

The Institutional Review Board process and patient consents were not proceeded because this is a review article.

Availability of Data and Material

The data that support the findings of this study are available in the text.

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Conflicts of Interest

M.B. reports receiving a research grant from Daewoong Pharmaceutical.

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