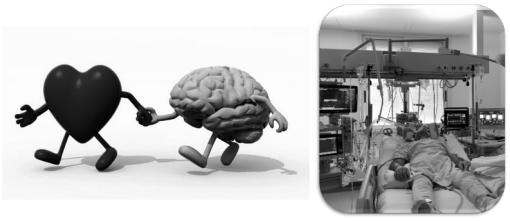

Neuromonitoring in the Stroke Unit

Claudio Baracchini MD, PhD, FESO
Director of the Stroke Unit and Neurosonology Lab
University of Padua School of Medicine, Italy

1

Dynamic Management

Coronary Care Units

The main feature of coronary care is **continuous cardiac monitoring**.
This **allows early intervention**, **improving the prognosis**.

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2

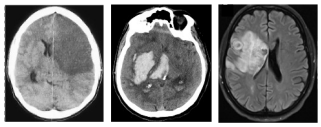
Neurological Surveillance in Stroke Unit/NICU

"Traditional" Neurological surveillance is achieved by:

1. **Neurological monitoring**
2. **Parenchymal imaging** (cerebral CT/MRI)

However, it is **limited**

- It is not easily repeatable
- It needs transport of sometimes unstable patients.



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3

Cerebral Blood Flow Control

To sustain neuronal function, the brain has evolved **very complex regulatory processes** to ensure a continuous and constant blood supply:

1. **Cerebral Autoregulation**
2. **Neurovascular Coupling**
3. **Cerebral Vasomotor Reactivity**
4. **Neurogenic Regulation**

An **inflexible structure surrounding the brain**: **the skull**.
Therefore, it is of utmost importance to have tightly regulated mechanisms to maintain a relatively constant CBF.

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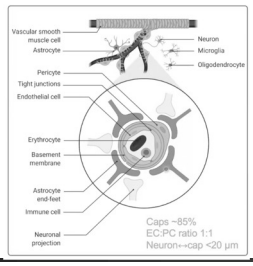
4

THE NEUROGLIOVASCULAR UNIT

the Neurovascular Unit
is Essential to all CBF regulatory processes

INFRASTRUCTURE:
- Small Arteries
- Arterioles
- Capillaries
- Venules

IMPORTANT PLAYERS:
- Endothelial Cells
- Pericytes
- Astrocytes
- Perivascular Nerves



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5

CEREBRAL AUTOREGULATION

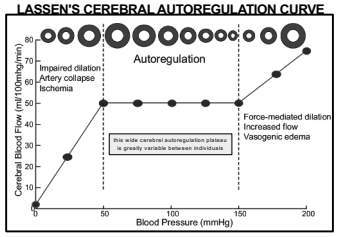
Autoregulation assures relatively constant CBF, despite fluctuation of perfusion pressure (CPP)

CPP = MAP – ICP (mmHg)

MECHANISM:
Intrinsic Ability of the **Arterioles to dilate/constrict when intravascular pressure decreases/increases,** respectively, to maintain CBF relatively constant **over a range of systemic blood pressures**

TYPES OF CA:
1. **STATIC CA:** response of CBF to steady-state changes of arterial blood pressure.
2. **DYNAMIC CA:** CBF responses to transient fluctuations of arterial blood pressure.

LASSEN'S CEREBRAL AUTOREGULATION CURVE



Lassen, N. A. (1959). Cerebral blood flow and oxygen consumption in man. *Physiological Reviews*, 39(2), 183–238.

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6

CEREBRAL AUTOREGULATION

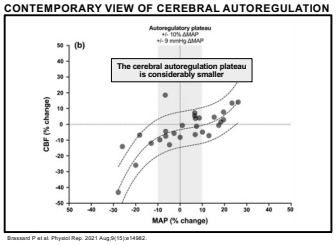
Autoregulation assures relatively constant CBF, despite fluctuation of perfusion pressure (CPP)

CPP = MAP – ICP (mmHg)

CBF regulation is far more pressure-passive in nature than traditionally believed

Asymmetry in the cerebral autoregulation response: the brain seems better adapted to compensate for transient increases compared to decreases in MAP

CONTEMPORARY VIEW OF CEREBRAL AUTOREGULATION



Brassard P et al. Physiol Rep. 2021 Aug;9(15):e14962.

7

NEUROVASCULAR COUPLING

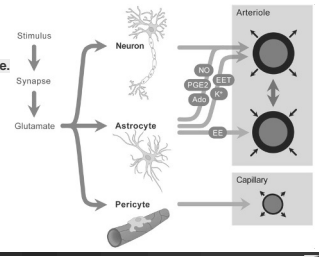
It refers to the brain's ability to increase CBF to regions where neurons are metabolically active.

MECHANISMS:

Metabolic messengers (adenosine, lactate) contribute to functional hyperemia through glutamate-induced prostaglandin signaling to blood vessels.

The effect is a **dilatation of the arterioles** which leads to a **CBF increase.**

Recent data have also suggested the important **role of pericytes** in regulating CBF through the **control of capillary diameter.**



8

CEREBRAL VASOMOTOR REACTIVITY

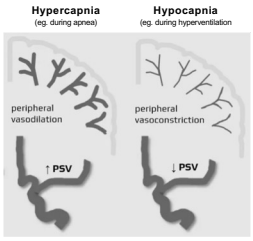
It refers to the brain's ability to increase/decrease CBF in response to chemical messengers.

MECHANISMS:

Chemical messengers (eg. pCO_2, pH, ~pO_2) contribute to functional hyperemia:

↑ **pCO_2** The effect is a **dilatation of the arterioles** which leads to a **CBF increase.**

↓ **pCO_2** The effect is a **constriction of the arterioles** which leads to a **CBF decrease.**



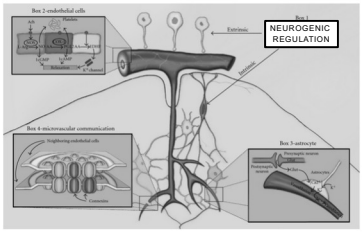
9

NEUROGENIC REGULATION

The third mechanism is the **neurogenic regulation** whereby **extensive arborization of perivascular nerves play a role in controlling CBF.**

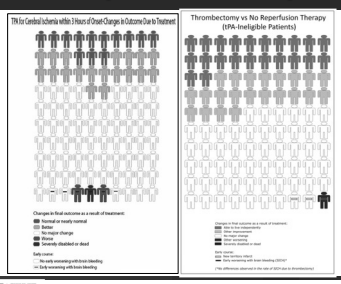
Extrinsic perivascular innervation:
1. The trigeminal ganglion (sensory)
2. The superior cervical ganglion (sympathetic)
3. The sphenopalatine ganglion (parasympathetic)

Intrinsic perivascular innervation: local interneurons (mainly GABAergic) also play a role in the regulation of microvascular tone.



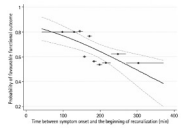
10

Acute Reperfusion Therapies in Ischemic Stroke



IVT reduces disability by 12-19%
IVT does not reduce mortality

EVT reduces disability by 34-44%
EVT reduces mortality by 3-5%



These effects are time-dependent

Gadhia J, et al. Stroke. 2010 Feb;41(2):300-6
Tokumbah I, et a.. Stroke. 2018 Jan;49(1):90-97
Tsivgoulis G, et al. J Stroke. 2020 Jan;22(1):130-140

11

Acute Reperfusion Therapies: Periprocedural Complications

- Contrast allergy
- Airway control, Oxygenation and Sedation
- Hypertension and blood pressure variability
- Hyperglycemia an temperature regulation
- Arterial access site complications
- Vessel injury, Vasospasm, Device Retention
  - Re-occlusion
  - Hemorrhage and Edema
- Temperature dysregulation, Arrhythmias
- **Swift Changes!!!!!**
  - Infection (pneumonia/ UTI), Stress ulcers, Pressure ulcers, Peripheral venous thrombosis, Fall risk
  - Timing of extubation, tracheostomy, PEG. Prognosis and rehabilitation

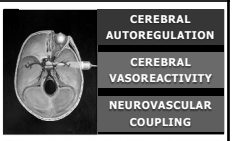
Jadhav AP et al. Care of the Post-Thrombectomy Patient. Stroke. 2018;49:2801-2807

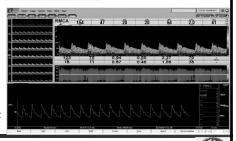
12

Neurological Surveillance in Stroke Unit/NICU

"Dynamic" Neurological surveillance is achieved by:

1. **Close neurological monitoring**
2. **Parenchymal imaging** (cerebral CT/MRI)
   However, it is **limited**
   - It is not easily repeatable
   - It needs transport of sometimes unstable patients.
3. **Bedside Hemodynamic Monitoring**
   - It can be **performed directly by the clinician**: i.e. **inexpensive**
   - **without side effects (non-invasive)** even including US contrast agent application
   - can be **repeated in short intervals (easily repeatable).**
   - High Sensitivity and Specificity for significant stenosis
   - Only modality able to detect **embolic signals**
   - It offers not only Imaging (CTA, MRA) but mainly a **real-time beat-to-beat hemodynamic evaluation and longer monitoring**





13

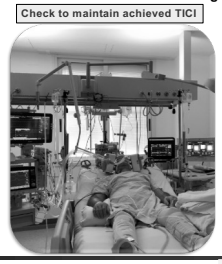
Lack of Neurological Exam in Intubated patients

COGIF grade	Hemodynamic pattern	DSA/TCD Correlation: sensitivity 91%, specificity 93%
1	No flow	
2	Low flow velocities without diastolic flow	
3	Low flow velocities with diastolic flow	
4	Established perfusion:	
	a) flow velocities equal to contra-lateral side	
	b) high focal flow velocities (i.e. stenosis)	
	c) high segmental flow velocities (hyperperfusion)	

Consensus On Grading Intracranial Flow Obstruction Nedelmann M et al, Stroke 2009

TCCD Monitors the Residual Flow Signals

Check to maintain achieved TICI



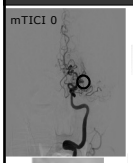
14

Neurological Worsening

- **Reocclusion ?**

15

REAL TIME MONITORING AFTER MECHANICAL THROMBECTOMY

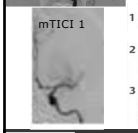


TIBI Classification

0 **Absent flow signal**
Complete proximal occlusion

No antegrade residual flow

No Doppler Signal:
- Re-Occlusion



1 **Minimal flow signal**
Absend end-diastolic flow

No antegrade residual flow

2 **Blunted flow signal**
Delayed systolic flow acceleration with reduced mean flow velocity and PI <1.2

3 **Dampened flow signal**
Pulsatile signal with normal acceleration and decrease of mean flow velocity >30% compared with normal side

Sluggish antegrade flow

HYPOPERFUSION
Infarct Expansion

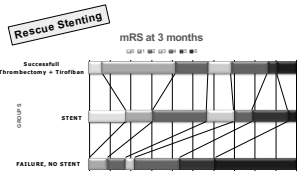
Decreased Velocity

Beware!
Resistance changes can occur quickly:
Look at diastolic flow.

16

Stentriever Thrombectomy Failure: A Challenge in Stroke Management
Claudio Benedetto[1], Filippo Ferlo[1], Matteo Sozo[1], Federica Viaro[1], Silvia Favaretto[1], Anna Palmieri[1], Caterina Kulyk[1], Enzo Ballotta[1], Lorena Nico[1], Giacomo Center[1], Francesco Causin[1]

Rescue Stenting

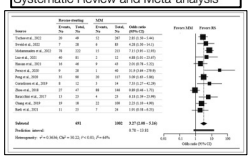


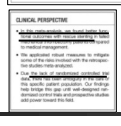
Better Outcome in stented pts: mRS 0-2: 56.5% vs 17.4%

CONCLUSIONS: Permanent stenting might be a feasible solution in patients with acute LVO **after stentriever thrombectomy failure.**

World Neurosurg. 2017 Jul;103:57-64.

Rescue Stenting for Failed Mechanical Thrombectomy in Acute Ischemic Stroke: Systematic Review and Meta-analysis



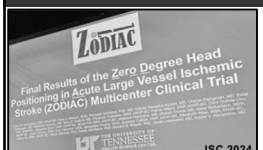


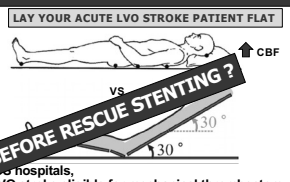
Rescue Stenting

Stroke Vasc Interv Neurol. 2023;3:e000881

17

REAL TIME MONITORING BEFORE MECHANICAL THROMBECTOMY





REAL TIME MONITORING BEFORE RESCUE STENTING ?

Prospective Multicenter Study con[illegible] hospitals,
92 patients (mean age roughly [illegible] an LVO stroke eligible for mechanical thrombectomy,

Patients positioned at [illegible] degree angle:
Reduced early [illegible] deterioration (2.2% vs 55.3%; P < 0.001).
No cases of [illegible] cerebral hemorrhage or hospital-acquired pneumonia
Lower rate of [illegible] death at 90 days (4.4% vs 21.7%; P = 0.03).
More likely to have an improvement in NIHSS score at 24 hours (86.7% vs 60.9%; P = 0.008) and at 7 days or discharge (86.7% vs 67.4%; P = 0.045).
By 90 days, functional outcomes tended to favor zero-degree positioning, although the differences were not statistically significant.

18

Neurological Worsening

- **Reocclusion ?**
- **Increased Intracranial Pressure ?**

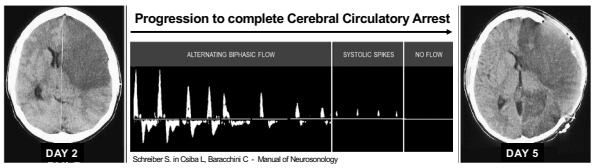
19

INDIRECT DEPICTION OF INTRACRANIAL HYPERTENSION

20

Malignant MCA infarction and Decompressive Craniectomy

Large hemispheric infarcts occur in **up to 10% of all ischemic strokes** and can cause devastating disability.



Schreiber S. in Csiba L, Baracchini C - Manual of Neurosonology

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Neurological Worsening

- **Reocclusion ?**
- **Increased Intracranial Pressure ?**
- **Continuing Embolization ?**

22

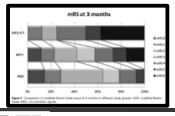
REAL TIME MONITORING AFTER MECHANICAL THROMBECTOMY

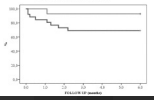
Possible EMBOLIZATION
1. From the same site causing the index event
2. Procedural release of clot fragments

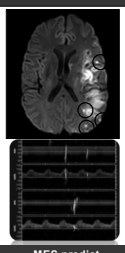
Prognostic Role of Microembolic Signals After Endovascular Treatment in Anterior Circulation Ischemic Stroke Patients

Filippo Farina[1], Anna Palmieri[2], Silvia Favaretto[1], Federica Viaro[1], Giacomo Cester[1], Francesco Causin[2], Claudio Baracchini[1]

Study Objectives:
To assess the prognostic value of Micro-Embolic Signals (MES) after Thrombectomy.







MES predict
Worse Outcome and Stroke Recurrence

23

MES MONITORING IN ESUS

The Role of Transcranial Doppler Monitoring in Patients with Multi-Territory Acute Embolic Strokes: A Review

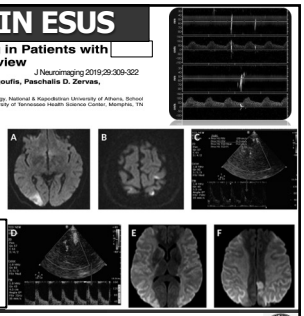
J Neuroimaging 2019;29:309-322

Odysseas Kargiotis, Klearchos Psychogios, Apostolos Safouris, Georgios Magoufis, Paschalis D. Zervas, Eleftherios Stamboulis, Georgios Tsivgoulis

From the Stroke Unit, Metropolitan Hospital, Piraeus, Greece (OK, KP, AS, GM, ES); Second Department of Neurology, National & Kapodistrian University of Athens, School of Medicine, "Attikon" University Hospital, Athens, Greece (AS, PDZ, GT); and Department of Neurology, The University of Tennessee Health Science Center, Memphis, TN (GT).

Table 2. Sonographic Characteristics of Microembolic Signals

Characteristic acoustic sound
High intensive amplitude of at least 3 db greater than the spectral background
Transient signals with short duration of less than 300 ms
Unidirectional signals
Detected within the flow spectrum



The presence and burden of MES is clearly associated with an **increased risk of symptomatic, recurrent embolization**, and thus **can justify a more aggressive treatment** approach (clopidogrel load followed by dual antiplatelet therapy or alternatively therapeutic dose of low-molecular-weight heparin).

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MES MONITORING IN ATRIAL FIBRILLATION

Detection of microemboli in patients with acute ischaemic stroke and atrial fibrillation suggests poor functional outcome

European Stroke Journal
1–9
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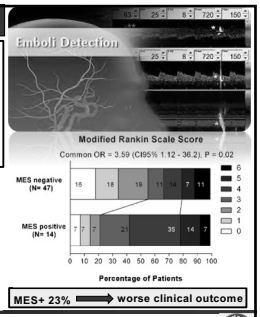
Sage

Pedro Castro[1*], J Ferreira[2*], Branko Malojcic[1], Danira Bazadona[1], Claudio Baracchini[1], Alessio Pieroni[1], David Skoloudik[3], Elsa Azevedo[4] and Manfred Kaps[1]

Eur Stroke J. 2023 Dec

Abstract
Introduction: We investigated the burden of microembolic signals (MES) in patients with acute ischaemic stroke (AIS) and atrial fibrillation (AF), assessing their impact on functional outcomes.
Patients and methods: This multicentre international prospective cohort study involved patients with AIS and either a known or newly diagnosed anticoagulant-naive AF. All centres utilised the same transcranial Doppler machine for 1-h monitoring with bilateral 2 MHz probes within 24h of symptom onset. Recordings underwent MES analysis by a blinded central reader. The primary objectives were to ascertain the MES proportion and its association with functional outcomes assessed by the modified Rankin scale (mRS) score at 90 days.
Results: Between September 2019 and May 2021, we enrolled 61 patients, with a median age of 78 years (interquartile range 73–83) and a median stroke severity score of 11 (interquartile range 4–18). MES were observed in 14 patients (23%), predominantly unilateral (12/14, 86%), with a median rate of 6 counts/hour (interquartile range 4–18). MES occurrence was higher post-thrombectomy and among those with elevated brain natriuretic peptide levels ($p < 0.05$). A worse mRS score of 3–6 was more frequent in patients with MES, occurring in 11/14 (79%), compared to those without MES, 20/47 (43%), with an adjusted odds ratio of 5.04 (95% CI, 1.15–39.4), $p = 0.04$.
Conclusions: Nearly a quarter of patients with AIS and AF exhibited silent microembolization after the index event. Detecting MES within 24h post-stroke (using transcranial Doppler) could signify a marker of poor functional outcomes. Subsequent trials will assess if very early antithrombotic treatment might enhance outcomes in this highly selective group of cardioembolic stroke patients. (ClinicalTrials.gov ID: NCT05078606).



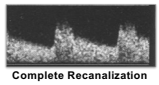
MES+ 23% ⟹ worse clinical outcome

25

Neurological Improvement

TIBI Classification

5 Normal flow signal
Without relevant difference to the contralateral side (<30% difference)



Complete Recanalization

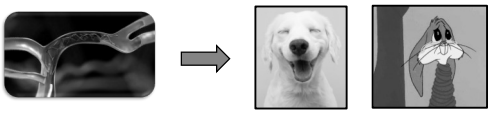
Even if Dramatic Neurological Improvement with rtPA and/or MT

⬇

Continue to monitor Cerebral Hemodynamics

26

Acute Reperfusion Therapies ⟹ Not always a success!



Despite High recanalization rate (>90%) after EVT

>1/3 pts develop **Early Neurological Deterioration within 72 hrs** after EVT and **<50%** of patients are **functionally independent (mRS 0-2) at 3 months.**

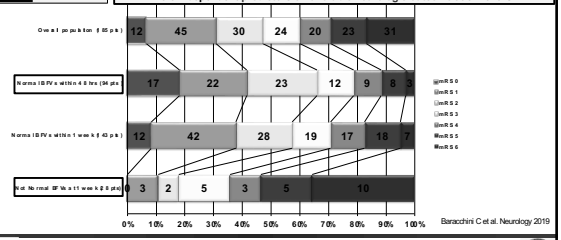
Why is there such a discrepancy between clinical outcome and recanalization rates?

27

REAL TIME MONITORING AFTER MECHANICAL THROMBECTOMY

Prospective study in **185 patients** who had a first-ever anterior circulation LVO stroke and underwent EVT within 8h from stroke onset.

Early (<48 hours) normalization of the PSV ratio and successful recanalization were **independent** predictors of favorable neurologic outcome **at 3 months.**



Baracchini C et al. Neurology 2019

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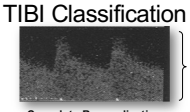
Neurological Improvement but…

TIBI Classification



Increased Velocity !!!

Complete Recanalization

Even if Dramatic Neurological Improvement with rtPA and/or MT

⬇

Continue to monitor blood flow velocities:
Check for Increase of Velocity along the entire recanalized segment

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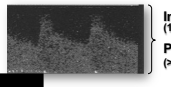
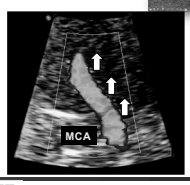
REAL TIME MONITORING AFTER MECHANICAL THROMBECTOMY

TIBI Classification



Increased Velocity
(1.5 times compared to the contralateral artery)

PI decrease
(>30% vs unaffected side)



Increase of Velocity along the entire recanalized segment:
- Hyperemia?
- Hyperperfusion?

HYPERPERFUSION* ⟹ **Hemorrhagic Transformation**

* Analogous to cerebral hyperperfusion syndrome after carotid endarterectomy

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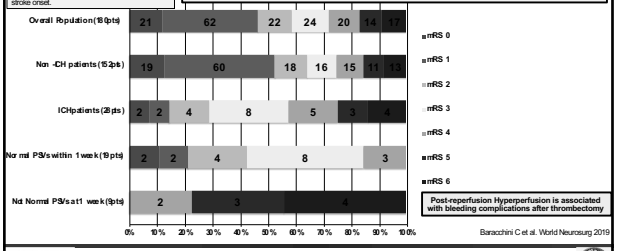
REAL TIME MONITORING AFTER MECHANICAL THROMBECTOMY

Prospective study in **180 patients** who had a first-ever anterior circulation LVO stroke and were **successfully recanalized** by EVT within 6h from stroke onset.

Early detection of high PSV MCA Ratio in successfully recanalized stroke patients indicates an increased risk of ICH and a poor outcome at 3 months.



Post-reperfusion Hyperperfusion is associated with bleeding complications after thrombectomy

Baracchini C et al. World Neurosurg 2019

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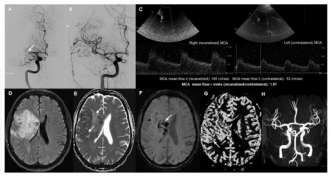
REAL TIME MONITORING AFTER MECHANICAL THROMBECTOMY

Post-reperfusion hyperperfusion after endovascular stroke treatment: a prospective comparative study of TCD versus MRI

Kneihsl M, *et al. J Neurointervent Surg* 2023 Oct;15(10):983-988

Prospective study in **226 patients successfully recanalized** by EVT. In the subgroup for which both TCD and perfusion MRI were available, 29 patients (31%) had increased ipsilateral MCA flow velocities on TCD. Of these, 25 patients also showed parenchymal hyperperfusion on MRI (sensitivity 85%; specificity 62%). Hyperperfusion severity on MRI correlated with MCA flow velocities on TCD (rs=0.379, p<0.001).

TCD vs MRI



Hyperemic Reperfusion occurred in 1/3 (75/226) of successful thrombectomies. **Hyperperfusion severity on MRI correlated with MCA flow velocities on TCD.**

WHAT IS ALREADY KNOWN ON THIS TOPIC

- Increased middle cerebral artery (MCA) blood flow velocities on transcranial duplex sonography (TCD) have been identified in individual patients after successful mechanical thrombectomy (MT) and were associated with intracranial hemorrhage (ICH) and poor outcome. However, the underlying mechanisms and the relationship with parenchymal brain perfusion still remained to be determined.

WHAT THIS STUDY ADDS

- This prospective study identified post-reperfusion hyperperfusion as a frequent mechanism explaining elevated MCA blood flow velocities on TCD after MT. TCD completed well with perfusion MRI and helped in increased risk of post-interventional reperfusion injury (ICH, vasogenic brain edema).

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

- TCD can serve as a reliable bedside tool to monitor cerebral hemodynamic changes after MT and has the potential to serve as an early prognostic tool in this setting.

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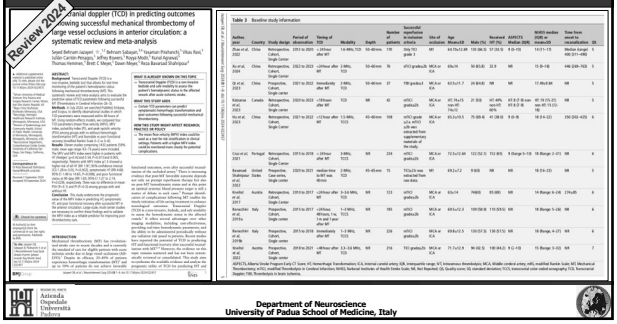
REAL TIME MONITORING AFTER MECHANICAL THROMBECTOMY



Review 2024

Transcranial doppler (TCD) in predicting outcomes following successful mechanical thrombectomy of large vessel occlusions in anterior circulation: a systematic review and meta-analysis

Seyed Behnam Jazayeri[1,2*], Behnam Sabayan[3,4], Yasaman Pirahanchi[5], Vilias Rava[1], Julián Carrión-Penagos[1], Jeffrey Bowers[1], Roopa Modi[1], Kunal Agrawal[1], Thomas Hemmen[1], Brett C Meyer[1], Dawn Meyer[1], Reza Bavarsad Shahripour[1]

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Blood Pressure Management after IVT and/or MT

- **Proper Target ?**
- **Intensive vs Standard BP Treatment ?**
- **Role of Transcranial Ultrasound ?**

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BLOOD PRESSURE MANAGEMENT

Blood Pressure before IVT bolus <185/110mmHg

Blood Pressure after IVT < 180/105mmHg for 24hrs post treatment

N.B. Any violation of these rules increases the sICH risk: OR 2.59; 95% CI 1.07-6.25, P=0.034

AHA/ASA Guideline

Guidelines for the Early Management of Patients With Acute Ischemic Stroke: 2019 Update to the 2018 Guidelines for the Early Management of Acute Ischemic Stroke

A Guideline for Healthcare Professionals From the American Heart Association/American Stroke Association

EMINENCE-BASED GUIDELINES!!!

These BP values are part of the guidelines, but they were chosen arbitrarily by some experts!

Are these recommendations reasonable for all patients before/after EVT?

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Prospective Randomized Studies evaluating post-EVT BP

REFERENCES	YEAR	TRIAL NAME AND TYPE	STATUS	SAMPLE SIZE	BLOOD PRESSURE COMPARISONS	OUTCOMES/GOALS
Mazighi et al.	2021	**BP TARGET**; randomized, controlled, open-label trial. Patients were enrolled at 4 clinical sites	Completed	324 patients post EVT with **TICI 2b-3**	Patients were randomized within 1 hour after EVT to BP target of **100-129mmHg vs 130-185mmHg**	Primary outcome: Radiographic ICH Secondary outcome: NIHSS at 24 h, and 3-month mRS Results: There was no difference in the rate of radiographic ICH or any of the secondary clinical efficacy outcomes.
Song et al.	2022	**ENCHANTED 2**; prospective, randomized trial	Stopped early after review of outcome data	821 patients post-EVT with **TICI 2b-3**	SBP target of **<120 vs 140-180mmHg** during first 72 h after EVT	Primary Outcome: 90-day mRS Results: the more intensive treatment group had more early neurological deterioration and major disability at 90 days. There were no significant differences in symptomatic ICH, serious adverse events or mortality
Nam et al.	2023	**OPTIMAL BP**; prospective, multicenter randomized trial	Stopped early because of safety concerns	644 patients post-EVT with **TICI 2b-3**	SBP target **<140mmHg vs <180mmHg** during first 24 h after EVT	Primary Outcomes: 90-day mRS, symptomatic ICH at 36 h, death at 90 days. Results: intensive BP management for 24 hours led to a lower likelihood of functional independence at 3 months compared with conventional BP management.
Mistry et al	2023	**BEST-II**; prospective, randomized trial	Completed	120 patients post-EVT with **TICI 2b-3**	Assigned to **SBP target of <180, <160, or <140mmHg** during first 24 h after EVT	Primary Outcomes: final infarct volume and utility-weighted mRS at 90 days. The study did not find a significant difference in the treatment groups.

Intensive BP target should be avoided after EVT for acute ischemic stroke
(worse neurological recovery and decreased rates of functional independence compared to the standard BP target).
The optimum systolic blood pressure after MT is yet to be defined.

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BLOOD PRESSURE MANAGEMENT AFTER MT

Optimum BP Value: **There should be individual patient decision-making.**

On what basis?
- Degree of Recanalization after MT
- Neurological Status

CURRENT PRACTICE:

EXPERTS ADVICE

TICI 3 + Dramatic neurologic recovery: < 140/80mmHg

TICI 3 + No neurologic improvement: < 160/90mmHg

TICI 2 ± Neurologic improvement: < 160/90mmHg

No recanalization < 160/90mmHg or permissive hypertension < 180/90mmHg?

Strict BP control to avoid variability in all groups

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BLOOD PRESSURE MANAGEMENT AFTER MT

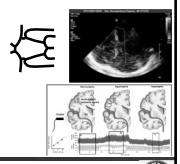
Optimum BP Value: **There should be individual patient decision-making.**

On what basis?
- Degree of Recanalization after MT
- Neurological Status
- Pre-Cerebral Vessel Occlusion

In stroke patients with a Pre-Cerebral Occlusion

TCD/TCCD scan can show:

- If the **circle of Willis is functionally intact**
- If there is **activation of primary collaterals (AcomA, AcomP)**
- If there is **flow diversion to leptomeningeal peripheral collaterals**.
- If **Vasomotor Reactivity** is preserved or compromised.
- If **Cerebral Autoregulation** is preserved or compromised.



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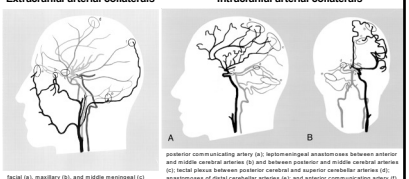
COLLATERAL PATHWAYS

In case of vessel obstruction, Activation of Collateral Pathways is very important for the clinical outcome of the patient.

- **Primary Collaterals**
  - AcomA
  - PcomA
- **Secondary Collaterals**
  - Ophthalmic Art.
  - Leptomeningeal Art.

Extracranial arterial collaterals

Intracranial arterial collaterals



facial (a), maxillary (b), and middle meningeal (c) arteries to the ophthalmic artery and dural arteriolar anastomoses from the middle meningeal artery (d) and occipital artery through the mastoid foramen (e) and parietal foramen (f).

posterior communicating artery (a); leptomeningeal anastomoses between anterior and middle cerebral arteries (b) and between posterior and middle cerebral arteries (c); tectal plexus between posterior cerebral and superior cerebellar arteries (d); anastomoses of distal cerebellar arteries (e); and anterior communicating artery (f).

Liebeskind DS, *Stroke*. 2003;34:2279-2284

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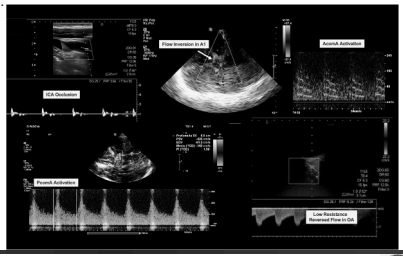
COLLATERAL PATHWAYS

In case of vessel obstruction, Activation of Collateral Pathways is very important for the clinical outcome of the patient.

Occlusion of the ICA

- **Primary Collaterals**
  - AcomA
  - PcomA
- **Secondary Collaterals**
  - Ophthalmic Art.
  - Leptomeningeal Art.



Baracchini C – Brain-POCUS in press

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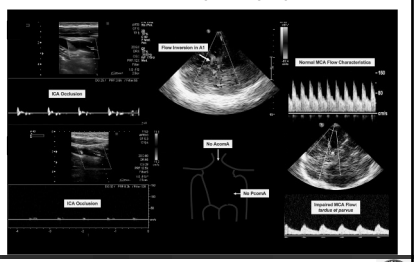
COLLATERAL PATHWAYS

In case of vessel obstruction, Activation of Collateral Pathways is very important for the clinical outcome of the patient.

Occlusion of the ICA

- **Primary Collaterals**
  - AcomA
  - PcomA
- **Secondary Collaterals**
  - Ophthalmic Art.
  - Leptomeningeal Art.



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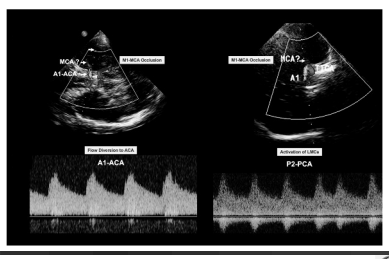
COLLATERAL PATHWAYS

In case of vessel obstruction, Activation of Collateral Pathways is very important for the clinical outcome of the patient.

Occlusion of M1-MCA

Pre-MT or Post-MT Failure

- **Secondary Collaterals**
  - Flow Diversion
  - Leptomeningeal Art.



Baracchini C – Brain-POCUS in press

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COLLATERAL PATHWAYS and BLOOD PRESSURE MANAGEMENT AFTER MT

The degree of collateral circulation has a bearing on the intensity of blood pressure therapy

Stroke: Vascular and Interventional Neurology

ORIGINAL RESEARCH

Transcranial Doppler After Endovascular Treatment to Evaluate Collateral Status and Outcomes in Patients With Large-Vessel Occlusion

Small prospective study on 15 LVO stroke patients undergoing MT

RB Shahripour Stroke Vasc Interv Neurol. 2022;2:e000214

- Most patients with poststroke disability (modified Rankin scale>2) had either flow diversion or active leptomeningeal collateral flow in the presence of elevated blood pressure (≥170/93 mm Hg).
- In cases without early neurological deterioration and in those with a modified Rankin scale<2, the authors did not observe any flow diversion or active leptomeningeal collateral.

Role of TCD in identifying high-risk patients who need close clinical monitoring:
The persistence of active leptomeningeal collaterals and flow diversion despite a successful EVT indicates the possibility of inadequate cerebral flow and is associated with a poor clinical outcome.

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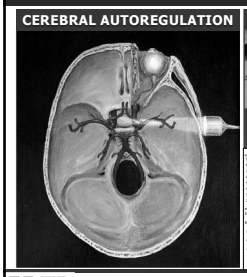
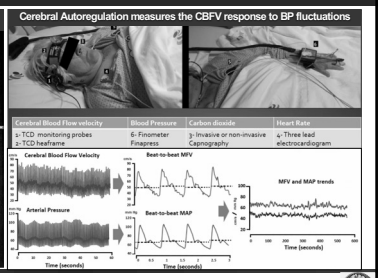
TCD and Cerebral Blood Flow Control

CEREBRAL AUTOREGULATION



Cerebral Autoregulation measures the CBFV response to BP fluctuations



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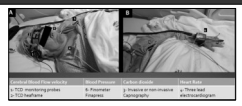
CEREBRAL AUTOREGULATION TESTING

brain sciences — MDPI

Sheriff et al. Brain Sci. 2020, 10, 641

Brief Report

Dynamic Cerebral Autoregulation Post Endovascular Thrombectomy in Acute Ischemic Stroke



Results:

- Complete (TICI) 3) recanalization was associated with a more favorable autoregulation profile compared with TICI 2b or poorer recanalization ($p < 0.05$).
- **Less effective autoregulation** in the first 24 h was associated with **increased rates of hemorrhagic transformations** (PH1–PH2).
- These data suggest that **patients with incomplete recanalization and poor autoregulation** (especially within the first 24 h post-LSW time) may warrant closer blood pressure monitoring and control in the first few days post ictus.

When cerebral autoregulation is damaged,
BP management becomes more important:
Low BP → cerebral hypoperfusion → infarct expansion
Elevated BP → excessive flow → cerebral edema and hemorrhagic conversion.

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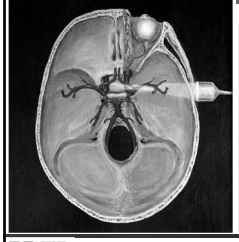
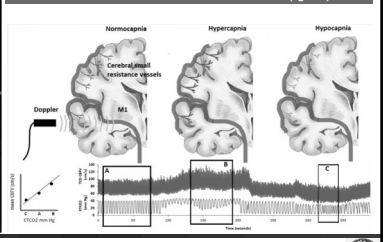
TCD and Cerebral Blood Flow Control

CEREBRAL VASOREACTIVITY

Cerebral Vasomotor Reactivity measures of the responsiveness of the cerebral blood vessels to either vasodilator or vasoconstrictor stimuli (eg. CO_2).





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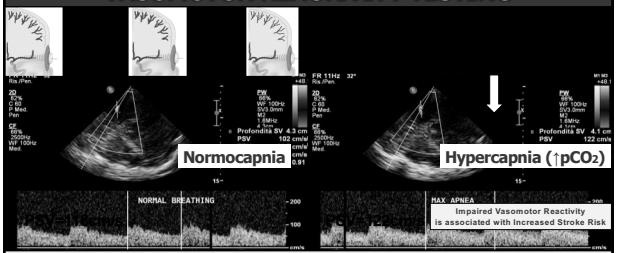
VASOMOTOR REACTIVITY TESTING



Impaired Vasomotor Reactivity is associated with Increased Stroke Risk

In the absence of vasomotor reactivity, cerebral perfusion is passively dependent on systemic blood pressure, and **the blood pressure must therefore not be lowered.**

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VASOMOTOR REACTIVITY TESTING

Reversed Robin Hood Syndrome in Acute Ischemic Stroke Patients

Andrei V. Alexandrov, MD; Vijay K. Sharma, MD; Annabelle Y. Lao, MD; Georgios Tsivgoulis, MD; Marc D. Malkoff, MD; Anne W. Alexandrov, PhD

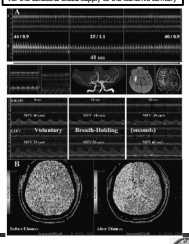
Background and Purpose—Recurrent hemodynamic and neurological changes with persisting arterial occlusions may be attributable to cerebral blood flow steal from ischemic to nonaffected brain.

Methods—Transcranial Doppler monitoring with voluntary breath-holding and serial NIH Stroke Scale (NIHSS) scores were obtained in patients with acute middle cerebral artery or internal carotid artery occlusions. The steal phenomenon was detected as transient, spontaneous, or vasomotory stimuli-induced velocity reductions in affected arteries at the time of velocity increase in normal vessels. The steal magnitude (%) was calculated as [(MFVm−MFVb)/MFVb]×100, where m=minimum and b=baseline mean flow velocities (MFV) during the 15- to 30-second period of a total 30 second of breath-holding.

Results—Six patients had steal phenomenon on transcranial Doppler (53 to 73 years, NIHSS 4 to 15 points). Steal magnitude ranged from −15.0% to −43.2%. All patients also had recurrent neurological worsening (>2 points increase in NIHSS scores) at stable blood pressure. In 3 of 5 patients receiving noninvasive ventilatory correction for snoring/sleep apnea, no further velocity or NIHSS score changes were noted.

Conclusions—Our descriptive study suggests possibility to detect and quantify the cerebral steal phenomenon in real-time. If the steal is confirmed as the cause of neurological worsening, reversed Robin Hood syndrome may identify a target group for testing blood pressure augmentation and noninvasive ventilatory correction in stroke patients. (***Stroke.*** 2007;38:3045-3048.)

Mechanism in RRH Syndrome:
vasodilation in non-ischemic brain regions
(↑pCO_2, anesthesia) → ↓ BP in pial arterial network → ↓of the collateral blood supply to the ischemic territory



Reversed Robin Hood Syndrome ? ⟹ **Blood Pressure Augmentation**

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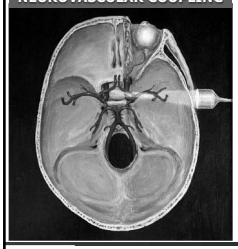
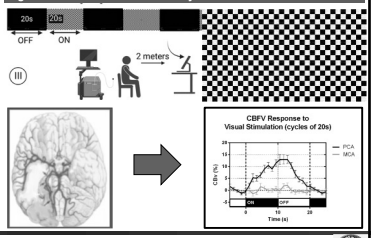
TCD and Cerebral Blood Flow Control

NEUROVASCULAR COUPLING

Neurovascular Coupling measures the brain's ability to increase CBF to regions where neurons are metabolically active.





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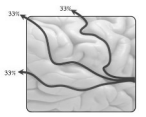
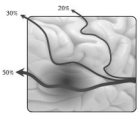
NEUROVASCULAR COUPLING TESTING

Luxury Perfusion

represents a **Mismatch between Cerebral Blood Flow and local Metabolic Requirement** that are usually tightly coupled. As neuronal and glial cells succumb to ischemia, their metabolic requirements also reduce. This should result in a reduction in cerebral blood flow. Instead, in luxury perfusion, this is not the case, presumably due to a failure of autoregulation. Instead, there is an overabundance of blood supplied to infarcted tissue. This, in turn, results in a decrease in the regional oxygen extraction fraction.

Mechanism: Regional Vasoparalysis brought about by the release of acidic metabolites from the ischemic tissue





RISK

- Cerebral Edema
- Hemorrhagic Transformation

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Neurovascular Ultrasound

Dynamic Management

is an Extension of the Neurological Examination and an Indicator of high quality and competence in stroke management.

- **Lack of neurological exam in intubated patients:**
  - Check to maintain achieved TICI
- **Neurological worsening:**
  - Re-occlusion?
  - Increased Intracranial Pressure?
  - Continuing embolization?
- **Neurological improvement:**
  - Minimize the risk of symptomatic ICH (sICH) after reperfusion (IVT and/or EVT): avoid hyperemia/hyperperfusion.
- **Optimal blood pressure?**
  - Set the range
  - Avoid fluctuations (variability)

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FUTURE DIRECTIONS

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ORIGINAL WORK

Robotic Assisted Transcranial Doppler Monitoring in Acute Neurovascular Care: A Feasibility and Safety Study

Alvise Fattorello Salimbeni[1*†], Caterina Kulyk[2†], Francesco Favruzzo[1], Ludovica De Rosa[1], Federica Viaro[1], Alessio Pieroni[1], Stefano Mozzetta[1], Milan R. Vosko[2] and Claudio Baracchini[1]

Ra-TCD

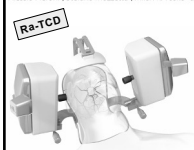

Fig. 1 Robotic assisted transcranial color Doppler (NovaGuide Intelligent Ultrasound, NeuraSignal Incorporated) in Angio Suite (original image)

AI in Stroke Unit

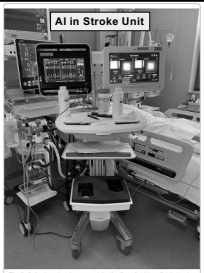

Fig. 2 Robotic assisted transcranial color Doppler (NovaGuide Intelligent Ultrasound, NeuraSignal Incorporated) in intensive care unit. (original image)

Neurocrit Care. 2024 Sep 19. doi: 10.1007/s12028-024-02121-x.

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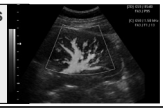
ULTRAFAST ULTRASOUND IMAGING

Breaking the Fundamental Limits of Ultrasound Imaging

1. **Breaking Time Resolution**
2. **Breaking Spatial Resolution**

Overcoming the Limitations of Conventional Pulsed-Doppler US

- Slow frame rate (b/c mult. Tx pulses/line, many lines)
- Poor sensitivity (to slow, small-volume flow)
- Poor resolution of small vessels (b/c long pulse, line spacing, etc.)



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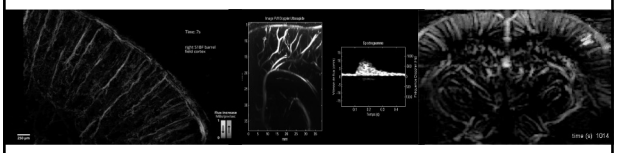
ULTRAFAST ULTRASOUND

Breaking Time and Space Resolution in Ultrasound
Enabling Super-Resolution Functional Ultrasound



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JOIN US

Claudio Baracchini MD, PhD, FESO
Director of the Stroke Unit and Neurosonology Lab
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