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Quantitative Mr Perfusion Imaging Post Endovascular Therapy: Defining Reperfusion Biomarkers in Acute Ischemic Stroke

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Abstract

Quantitative MR perfusion imaging has emerged as a critical tool in evaluating tissue-level reperfusion following endovascular therapy (EVT) for acute ischemic stroke (AIS). While angiographic recanalization signifies macrovascular success, it often fails to capture microvascular dynamics that influence clinical outcomes. This review comprehensively explores the utility of MR perfusion techniques—including dynamic susceptibility contrast (DSC) and arterial spin labeling (ASL)—to assess key hemodynamic parameters such as cerebral blood flow (CBF), cerebral blood volume (CBV), mean transit time (MTT), time to peak (TTP), and Tmax. Post-EVT imaging frequently reveals phenomena like impaired microvascular reperfusion (IMR) or "no-reflow," even in patients with complete angiographic reperfusion (TICI 3), and these conditions have been strongly linked to infarct progression, hemorrhagic transformation, and worse functional outcomes. Conversely, hyperperfusion patterns may indicate either successful tissue salvage or an ominous sign of blood-brain barrier disruption, depending on extent and context. Recent studies underscore the prognostic value of perfusion biomarkers in stratifying post-EVT patients and guiding blood pressure management, ICU monitoring, and rehabilitation planning. However, variability in imaging protocols, thresholds (e.g., relative CBF, Tmax >6s), and timing limits generalizability. This review highlights the need for standardized definitions and imaging workflows, while reinforcing the translational potential of MR perfusion imaging in enhancing personalized post-stroke care and advancing future clinical trials focused on microvascular targets.

Keywords: Acute Ischemic Stroke, Endovascular Therapy (EVT), Magnetic Resonance Perfusion Imaging, Dynamic Susceptibility Contrast (DSC), Arterial Spin Labeling (ASL), Cerebral Blood Flow (CBF), Cerebral Blood Volume (CBV), Mean Transit Time (MTT), Time to Peak (TTP), Tmax, No-Reflow Phenomenon, Impaired Microvascular Reperfusion (IMR), Post-Stroke Hyperperfusion, Perfusion Biomarkers, Stroke Prognostication, Tissue Reperfusion, Functional Outcome Prediction, Microvascular Perfusion, Neuroimaging in Stroke, Reperfusion Injury

INTRODUCTION

Magnetic resonance (MR) perfusion imaging has emerged as a critical tool for characterizing tissue viability in acute ischemic stroke (AIS) and guiding reperfusion therapy. In AIS, timely restoration of

blood flow via endovascular therapy (EVT) can salvage the ischemic penumbra – brain tissue at risk but not yet infarcted – and improve outcomes. MR perfusion imaging quantifies cerebral blood flow (CBF) and related parameters, allowing differentiation of infarct core from penumbra. For example, as shown in Figure 1, dynamic susceptibility contrast (DSC)-MRI perfusion maps including mean transit time (MTT), CBF, and cerebral blood volume (CBV) can delineate a large core infarct (markedly prolonged MTT, severely reduced CBF/CBV) and surrounding penumbra (prolonged MTT with preserved CBV). Such imaging is increasingly used to inform treatment decisions in AIS, especially in extended time windows. After major trials (DAWN, DEFUSE-3), perfusion imaging became central to patient selection for thrombectomy in late time windows.

In an MRI scan of the brain during a stroke, we look at special images called perfusion maps to understand how blood is flowing through different parts of the brain. Three key maps they use are:

- **MTT (Mean Transit Time):** how long it takes blood to pass through the brain tissue
- **CBF (Cerebral Blood Flow):** how much blood is flowing
- **CBV (Cerebral Blood Volume):** how much blood is present in the tissue

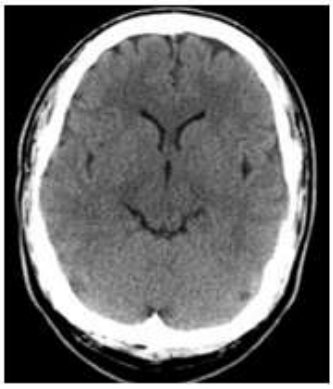
These maps helps to find:

1. **The ischemic core** – the part of the brain that's already severely damaged because it hasn't been getting enough blood for too long. On the scan, this shows up as:

- **MTT is prolonged (blood is slow)**
- **CBF is low (not much blood is flowing)**
- **CBV is low (not much blood left in the area)**

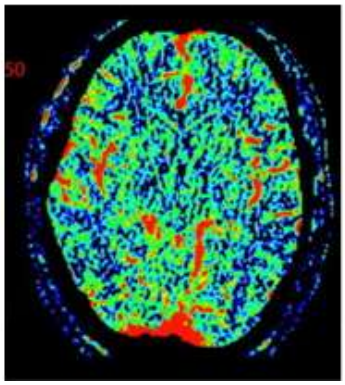
2. **The penumbra** – the part of the brain that is at risk but still alive and can be saved with fast treatment. This shows up as:

- **MTT is prolonged (blood is delayed)**
- **CBF is reduced**
- **But CBV is still normal or preserved** (which means the brain is trying to compensate and keep the area alive)



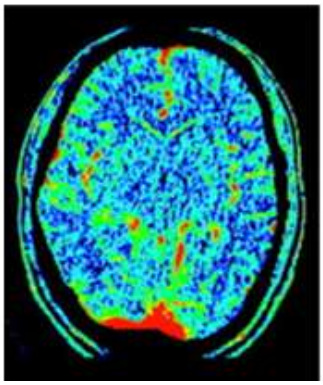
Picture: 1

NCCT (Non-Contrast Computed tomography)



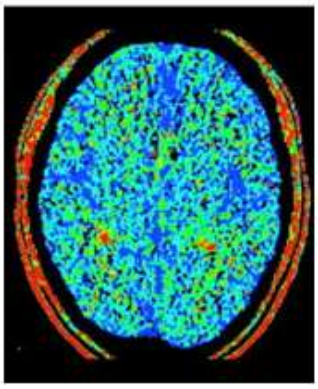
Picture: 2

CTP parametric maps, Cerebral Blood Flow (CBF)



Picture: 3

Cerebral Blood flow (CBF)



Picture: 4

Mean Transit Time (MTT)

This information helps to decide if a patient can benefit from endovascular therapy (EVT) – a treatment to remove the clot and restore blood flow – by showing which brain tissue can still be saved.

Normal brain perfusion is demonstrated by symmetrical blood flow patterns across both hemispheres.

By convention, perfusion color maps use red to represent higher values and blue to indicate lower values.

Post-EVT, however, angiographic recanalization (e.g. TICI 2b–3) does not always equate to tissue reperfusion. Microvascular dysfunction may limit perfusion despite open arteries, a phenomenon sometimes termed "no-reflow". In some patients, MRI after EVT reveals persistent hypoperfusion in the infarct region despite complete recanalization, which has been linked to worse outcomes. Conversely, other studies have reported hyperperfusion (so-called "luxury perfusion") in reperfused tissue, with conflicting prognostic implications. This suggests that quantitative perfusion metrics – including CBF, CBV, MTT, time-to-peak (TTP), and Tmax (time to maximum of the residue function) – may serve as biomarkers of effective reperfusion or microvascular failure.

In this article, we review recent literature on quantitative MR perfusion imaging after EVT, focusing on post-reperfusion biomarkers in AIS. We summarize imaging metrics and thresholds (Table 1), technical acquisition methods (DSC, arterial spin labeling [ASL], etc.), and findings from key trials and case series in the past 5–7 years. We highlight how perfusion parameters correlate with infarct evolution, secondary injury, and clinical outcome, and discuss advances in imaging protocols, analytical methods, and limitations. Our goal is to clarify the translational value of MR perfusion as a predictor of outcome and as a tool for guiding post-EVT management in stroke patients

Methods (Perfusion Imaging Techniques and Metrics)

MR Perfusion Acquisition

Quantitative MR perfusion in stroke primarily uses gadolinium-based **dynamic susceptibility contrast** (DSC) MRI or non-contrast **arterial spin labeling** (ASL). In DSC, a bolus of contrast agent is tracked through T2*-weighted imaging, generating a concentration–time curve for each voxel. From this curve, one derives multiple hemodynamic maps: CBV (area under the curve), CBF (via central volume principle), MTT (mean transit time), Tmax (delay of residue function), and TTP (time to peak of the curve). DSC is the most common perfusion technique in acute stroke (even in many clinical centers), owing to its speed and availability. DSC allows rapid whole-brain coverage and is well-validated for penumbra assessment. A typical post-EVT DSC protocol acquires multiple slices through the infarct immediately after contrast injection, often with 2–3 mm in-plane resolution. Dedicated software (e.g. RAPID, Olea, syngo.via) then performs deconvolution of arterial input functions to calculate quantitative CBF, CBV, MTT, TTP, and Tmax maps.

ASL is a contrast-free perfusion method that magnetically labels inflowing blood water as an endogenous tracer. The labeled blood exchanges with tissue, and subtracting labeled vs control images yields a CBF map. ASL is particularly attractive post-EVT because it avoids additional contrast (critical in renally impaired patients) and can be repeated. However, ASL requires longer acquisition (multiple label-control cycles) and may have lower signal in severely ischemic tissue. Early reports suggest ASL's feasibility in hyperacute stroke and correlation with outcomes. In practice, ASL is often added to an MR stroke protocol (alongside diffusion, TOF MRA, FLAIR). After EVT, ASL can reveal hyperperfusion (elevated CBF) in treated territories or persistent hypoperfusion (low CBF) if microvascular flow is impaired.

Table 1 summarizes key perfusion metrics. Notably, an **ischemic core** typically has severely reduced CBF and CBV, with markedly prolonged MTT and TTP. In contrast, the **penumbra** (tissue at risk but potentially salvageable) shows moderately reduced CBF, normal or elevated CBV (from collateral

dilation), and similarly prolonged transit times. In practice, a threshold of *Tmax > 6 seconds* (on deconvolved DSC maps) has been widely adopted to define critically hypoperfused tissue (core+penumbra). Subtracting the diffusion-defined core from this region estimates the volume of penumbra. Likewise, a relative CBF (rCBF) threshold of ~30% of normal is often used to approximate core.

Metric	Definition	Core vs Penumbra	Threshold (typical)
CBF (Cerebral Blood Flow)	Flow rate of blood through tissue (ml/100g/min)	Markedly ↓↓ in core; moderately ↓ in penumbra	Core often defined as <30% of normal
CBV (Cerebral Blood Volume)	Blood volume in tissue (ml/100g)	↓↓ in core; normal or ↑ in penumbra (collateral recruitment)	–
MTT (Mean Transit Time)	Average time for blood to traverse capillary bed	↑↑ in both core and penumbra (due to slow flow)	–
TTP (Time to Peak)	Time to peak contrast enhancement	↑ in ischemic tissue (core and penumbra)	–
Tmax (Time to Peak of Residue Function)	Delay of peak from arterial input (deconvolved)	↑ in hypoperfused tissue; delineates penumbra	>6 s often identifies critically hypoperfused region

Imaging Protocols and Analysis

Perfusion MRI is usually performed at specific post-EVT intervals. Some centers obtain **immediate post-procedure perfusion** (either via flat-panel CT during EVT or urgent MRI) to assess reperfusion on the spot. More commonly, follow-up perfusion is done at ~24 hours after EVT (often combined with follow-up DWI) to evaluate tissue fate. For instance, Mujanovic et al. (Stroke 2024) acquired MRI perfusion maps ~24h post-EVT to stratify TICI3 patients by microvascular flow. In the prospective study by Valls-Carbó et al. (Front Neurol 2025), consecutive AIS patients had perfusion MRI pre-EVT, within 2h post-EVT, and at day 5. We focus on studies using MR perfusion either in the acute (hours) or subacute (days) phase after EVT, as these inform immediate outcomes and guide early management.

Post-processing can use **absolute** or **relative** metrics. Many perfusion analyses express values relative to the contralateral hemisphere to account for global variability. For example, Ng et al. (Neurology 2022) defined "no-reflow" as ≥15% hemispheric asymmetry in rCBV or rCBF within the infarct. Similarly, Valls-Carbó et al. defined hypoperfused voxels as >15% reduction in rCBV compared to the contralateral mirror region. Such thresholds remain empirical; as evidence accumulates, consensus definitions (and standardized algorithms) for impaired reperfusion in MRI are evolving.

Contemporary imaging often leverages automated deconvolution software (e.g. RAPID®, Olea®, syngo.via) that provides volumetric outputs of perfusion lesions. These tools expedite quantification of ischemic core and penumbra and reduce operator variability. For instance, ischemic core can be estimated by DWI or rCBF thresholds, whereas penumbra is calculated as tissue with Tmax >6s beyond the core. Importantly, MR perfusion can detect **luxury perfusion** (hyperperfusion) after reperfusion – an effect not captured on DWI. Clinicians must be aware that luxury perfusion may mask infarct on perfusion maps, necessitating correlation with DWI.

Results (Key Findings from Recent Studies)

Perfusion Patterns and Clinical Outcomes

No-Reflow/Impaired Microvascular Reperfusion: A growing body of literature has documented that persistent tissue-level hypoperfusion (no-reflow) after successful thrombectomy is a common phenomenon associated with poor outcomes. Ng et al. analyzed pooled data from EXTEND-IA trials and found that 25.3% of patients with successful angiographic reperfusion (mTICI 2c–3) exhibited "no-reflow" on 24h perfusion imaging. These patients had significantly lower rCBV and rCBF in the infarct, and no significant change in MTT, indicating microvascular flow failure. Critically, no-reflow was strongly associated with adverse events: adjusted odds of hemorrhagic transformation were elevated (aOR 1.79) and patients had substantially greater infarct growth (+11.0 mL). Most notably, no-reflow predicted poorer 90-day outcome: the odds of being dead or dependent were nearly four times higher (aOR 3.72) in no-reflow patients. Neurology's Class II evidence statement concluded that "no-reflow" detected on follow-up perfusion imaging portends post-treatment complications and poor functional recovery.

The importance of no-reflow has been corroborated in MRI-only cohorts. Valls-Carbó et al. prospectively imaged AIS patients pre-EVT and shortly post-EVT with MRI perfusion. They defined **Impaired Microvascular Reperfusion (IMR)** as regions within the infarct with ≥15% rCBV reduction vs contralateral. Significant IMR (volume >5 mL) occurred in ~24% of patients (18% even in TICI3 cases). Initially, larger IMR volumes correlated with worse 24h NIHSS and final outcomes, although this did not hold after multivariable adjustment. Nonetheless, Valls-Carbó et al. emphasize that IMR is not rare and likely contributes to early neurological deterioration. They stress the need for standardized definitions: small differences in timing, threshold (they used 15% vs prior 40%), and exclusion criteria can drastically change reported prevalenceIn sum, persistent hypoperfusion on MRI – whether termed IMR or no-reflow – emerges as a potential imaging biomarker of microvascular failure that mitigates the benefit of EVT.

Hyperperfusion: The converse phenomenon – post-EVT hyperperfusion – has also been explored. Mujanovic et al. clustered TICI3 patients based on 24h perfusion status. They found that about a third showed "hyperperfusion" (increased CBV and CBF) within the infarct, a small fraction showed hypoperfusion, and others were normo- or mixed-perfusion. Interestingly, compared to normoperfusion, the hyperperfusion cluster tended toward *better* outcomes (adjusted OR for independence ~3.3), whereas microvascular hypoperfusion was associated with worse outcomes (aOR 0.3). This suggests that microvascular reperfusion heterogeneity exists even after perfect angiographic success. In contrast, Yang et al. used ASL to categorize hyperperfusion globally (in ≥50% of the territory) vs focal vs none. They reported that "global hyperperfusion" (GHP) was linked to **worse** outcomes: GHP patients had much larger infarcts (median 99 mL vs 13.5 mL) and higher NIHSS, and GHP was an independent predictor of infarct size. Their mediation analysis showed infarct volume fully mediated the relationship between GHP and 90-day mRS. Thus, these studies yield mixed results: while small-scale microvascular hyperemia may reflect effective reperfusion, extensive global hyperperfusion (luxury flow) might simply mark large, severe infarcts with poor collaterals. This discrepancy underscores that the **extent** and context of hyperperfusion matter – a focal luxury perfusion in a small infarct could be benign, whereas diffuse hyperemia after a large stroke might portend damage.

Perfusion Metrics as Biomarkers of Reperfusion

Several analyses have sought to distill quantitative perfusion biomarkers predictive of infarct growth and outcome. For instance, de Sousa et al. (AJNR 2021) performed a voxel-wise analysis of pre- and post-EVT perfusion maps in 84 patients. They found that lower post-recanalization CBF within the initially ischemic territory was the strongest predictor of infarct expansion, outperforming baseline core or penumbra volumes. In their logistic model, persisting hypoperfusion (CBF reduction) had greater weight than other factors in predicting infarct growth. Although the full text is proprietary, the abstract suggests that quantitative CBF measurements after EVT provide unique insight into tissue fate beyond what angiographic scores convey.

Murayama et al. (Stroke 2024) analyzed the IMR metrics in their cohort and found that patients with significant IMR had higher 5-day and 24h NIHSS scores, indicating worse clinical status. However, they did not find a significant independent link with 90-day mRS, possibly due to sample size. On the other hand, the EXTEND-IA pooled analysis showed that any degree of no-reflow portended worse NIHSS improvement and functional outcome. These data collectively argue that MRI perfusion biomarkers – such as the **volume of tissue with delayed Tmax**, **fraction of infarct with low rCBF/rCBV**, or **presence of global hyperperfusion** – correlate with complications (hemorrhage, edema) and recovery. At a minimum, perfusion imaging offers a physiologic "tissue clock" that refines the binary angiographic view.

Technical Advances and Case Studies

Several recent studies have illustrated or advanced MR perfusion protocols. For example, the **CHOICE** trial imaging substudy is investigating flat-panel CT perfusion acquired immediately post-thrombectomy to predict outcomes. While not MR, this underlines the trend toward ultra-early tissue perfusion assessment. In MR, emerging work on arterial spin labeling (ASL) is notable. The Yang et al. (2024) Sci Rep study used ASL maps within hours post-EVT to identify hyperemia – demonstrating the feasibility of noncontrast CBF mapping in this setting. Case series from centers with rapid MRI (e.g. Swiss study by Mutke et al.) have reported on perfusion imaging at 24h to explain "futile reperfusion." These anecdotal experiences complement larger cohort analyses.

Another technical consideration is **parameter selection and post-processing**. Most MR perfusion studies of stroke employ standardized thresholds (e.g. Tmax >6s for penumbra), but some explore alternative metrics. For example, hypoperfusion intensity ratio (HIR), defined on Tmax maps (Tmax>10s / Tmax>6s), has been used in CT perfusion to gauge collateral quality. Its applicability to MR perfusion is not yet established, but similar concepts (e.g. ratio of very delayed to moderately delayed tissue) could emerge. Machine-learning approaches have also appeared: d'Esterre et al. (Br J Radiol 2020) used machine learning to define optimal perfusion thresholds for penumbra, suggesting dynamic definitions rather than fixed cut-offs. Such methodological advances await MR equivalents.

Discussion

Translational Implications of MR Perfusion Biomarkers

Quantitative MR perfusion after EVT provides complementary information to angiographic scores. While mTICI or TICI grading assesses macrovascular patency, perfusion imaging probes microcirculatory flow and tissue health. **Translationally, MR perfusion biomarkers have potential roles in:**

- **Predicting outcome/futile reperfusion:** Persistent hypoperfusion (IMR/no-reflow) identifies patients at high risk of deterioration. For example, patients with significant no-reflow had ~3.7-fold higher odds of poor 90-day outcome. In the post-EVT setting, this could flag those who might need closer monitoring, ICU care, or neuroprotective strategies. Conversely, absence of perfusion deficits may reassure clinicians of effective reperfusion.
- **Guiding post-procedure therapy:** Knowledge of microvascular status could inform interventions. For instance, strict blood-pressure control is debated after EVT; some suggest that modest hypotension may worsen already underperfused regions. In the ASL study, Yang et al. speculated that intensive BP lowering in patients with hyperperfusion might be harmful. If MRI shows hyperemia, one might avoid aggressive BP reduction. Alternatively, pronounced IMR might prompt consideration of pharmacologic reperfusion enhancers (e.g. lytic adjuvants) though none are proven.
- **Augmenting patient selection and prognostication:** In extended time windows, perfusion imaging is already used to select EVT candidates. Post-EVT, perfusion maps might refine prognoses beyond final infarct volume. For example, if two patients both achieve TICI3, the one with residual perfusion deficits could be predicted to recover more slowly. This could influence rehabilitation planning and family counseling.
- **Stimulating therapeutic research:** Recognizing IMR as a pathologic process motivates research into therapies targeting microvascular no-reflow (e.g. anti-thrombotics, anti-inflammatories, stem cells). Imaging biomarkers allow stratification of patients in trials and objective endpoints. The recent Stroke 2025 report "Persistent Tissue-Level Hypoperfusion Negates Benefit" (abstract) signals a new era where imaging-defined endophenotypes may direct clinical trials.

Limitations and Challenges

Despite promise, MR perfusion faces hurdles. First, **access and logistics:** Many stroke centers rely on CT for initial triage due to speed and availability. MRI after EVT can be limited by equipment availability, patient instability, and contraindications. Even in centers with MRI, acquiring perfusion scans adds time and may require ICU monitoring. These constraints mean that MRI perfusion is less ubiquitous than CTP in practice. However, in research and select clinical settings, it is feasible (as shown by Swiss and Spanish cohorts).

Second, **standardization:** As highlighted, studies use varying definitions (15% rCBV drop vs visual vs Tmax thresholds) and timing (2h vs 24h). Until consensus is reached, cross-study comparisons are hard. The Valls-Carbó group noted that definitions dramatically changed reported IMR rates. They advocate for harmonization based on pathophysiology and pathology correlation. For now, most investigators use relative perfusion changes within the infarct as proxy markers.

Third, **interpretation complexity:** Perfusion maps can be confounded by luxury perfusion (which may mask core) and by edema/hemorrhage. As [57] cautions, DWI remains the gold standard for core volume, and perfusion must be interpreted in context (the "luxury perfusion" phenomenon). Similarly, PET studies have shown that ASL CBF and DSC-derived CBF may not always match absolute flow, especially in severely infarcted tissue. Radiologists must be cautious in labeling tissue salvageable vs infarcted purely by perfusion maps.

Finally, **dynamic changes:** Perfusion status evolves over time after EVT. Some "no-reflow" regions on ultra-early imaging may normalize by 24h, or vice versa. The Valls-Carbó protocol acquired MRI twice post-EVT, recognizing this variability. This raises the question: when is the optimal time to measure

reperfusion? Early imaging (minutes to hours) assesses immediate microvascular response, while later imaging (day 1–2) reflects secondary injury. Both are informative, but standardized timepoints would aid biomarker validation.

Conclusion

Quantitative MR perfusion imaging post-EVT offers a window into tissue-level reperfusion beyond angiography. By measuring CBF, CBV, MTT, TTP, Tmax and related parameters, clinicians can detect microvascular hypoperfusion or hyperperfusion that influence stroke recovery. Recent literature has established that persistent perfusion deficits (no-reflow/IMR) are relatively common and portend worse outcomes, while the implications of post-reperfusion hyperperfusion are context-dependent. These perfusion biomarkers thus have significant translational value: they can refine prognostic models, identify patients for targeted interventions, and ultimately improve post-stroke care.

Going forward, standardizing imaging protocols and thresholds is imperative. Larger multicenter studies should evaluate MR perfusion in conjunction with clinical trials to validate these biomarkers. Meanwhile, incorporating advanced perfusion imaging into stroke workflows (when feasible) can enrich clinical decision-making. As this review has shown, a deep understanding of MR perfusion metrics and their relationship to tissue fate is critical for translating neuroimaging into better outcomes for stroke patients.

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