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Cerebral Autoregulation and Cerebral Blood Flow Response to Mean Arterial Pressure Challenge Following Induction of General Anaesthesia for Neuroradiology Procedures

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ABSTRACT

Background: Intraoperative hypotension following induction of general anaesthesia is common and has been associated with adverse neurological outcomes. Cerebral autoregulation (CA) normally maintains stable cerebral blood flow (CBF) across a range of arterial pressures; however, anaesthetic agents such as propofol may transiently impair this protective mechanism.

Objectives: To determine the incidence of impaired cerebral autoregulation following induction of general anaesthesia and to evaluate the effect of a mean arterial pressure (MAP) challenge on cerebral blood flow and autoregulatory function.

Methods: In this prospective observational study, 40 adult patients undergoing elective interventional neuroradiology procedures under total intravenous anaesthesia were enrolled. Mean arterial pressure, mean middle cerebral artery blood flow velocity (MCAv_mean), and regional cerebral oxygen saturation (rSO_2) were continuously monitored. Dynamic cerebral autoregulation was assessed using the mean flow index (Mxa). Mxa value >0.3 was considered indicative of impaired autoregulation. In patients developing hypotension (≥20% reduction from baseline MAP), a norepinephrine-induced MAP challenge was performed.

Results: Following induction of anaesthesia, 53% of patients demonstrated impaired cerebral autoregulation despite comparable demographic characteristics, cardiovascular risk factors, anaesthetic depth, and baseline haemodynamics. In patients with impaired CA, MAP augmentation resulted in significant increases in MCAv_mean and a significant reduction in Mxa, indicating restoration of autoregulation. A strong positive correlation was observed between baseline Mxa and cerebral blood flow variation (ΔCBF) ($r = 0.73$, $p < 0.001$).

Conclusion: Patients undergoing neuroradiological procedures frequently exhibit impaired cerebral autoregulation secondary to general anesthesia induced hypotension. Early vasopressor-guided optimization of mean arterial pressure can restore autoregulatory function and improve cerebral perfusion, highlighting the importance of individualized, autoregulation guided hemodynamic strategies.

KEYWORDS: Cerebral autoregulation; Mean arterial pressure; Transcranial Doppler; General anaesthesia; Cerebral blood flow.

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INTRODUCTION

Intraoperative hypotension frequently occurs following induction of general anaesthesia and has been consistently associated with increased postoperative morbidity and mortality, including neurological complications and postoperative delirium [1–3]. Propofol, a widely used induction and maintenance agent, causes dose-dependent reductions in Systemic Vascular Resistance (SVR) and arterial pressure, predisposing patients to hypotension during the early phases of anaesthesia [4].

Cerebral autoregulation (CA) is a critical physiological mechanism that maintains relatively constant cerebral blood flow (CBF) across a wide range of systemic blood pressures [5]. When autoregulation is intact, cerebral arterioles adjust their tone in response to changes in perfusion pressure, thereby protecting the brain from both hypoperfusion or hyperperfusion. However, general anaesthesia, abrupt haemodynamic changes, and underlying cerebrovascular disease may impair this mechanism [6–8]. Importantly, systemic blood pressure values alone may not accurately reflect the adequacy of cerebral perfusion [9,10].

Dynamic cerebral autoregulation can be assessed using the mean flow index (Mxa), derived from the correlation between spontaneous fluctuations in mean arterial pressure (MAP) and cerebral blood flow velocity [11,12]. Elevated Mxa values indicate pressure-passive cerebral circulation and impaired autoregulatory capacity. The present study aimed to evaluate the prevalence of impaired cerebral autoregulation following induction of general anaesthesia for neuroradiology procedures and to assess the cerebrovascular response to a targeted MAP challenge.

MATERIALS AND METHODS

Study Design and Patients

This prospective, single-centre observational study was conducted in adult patients (aged ≥18 years) undergoing elective interventional neuroradiology procedures, including diagnostic cerebral angiography, intracranial aneurysm coiling, arteriovenous malformation embolization, and tumor embolization, under general anaesthesia. Institutional Ethics Committee approval was obtained, and written informed consent was secured from all participants. All identifying patient information was removed to ensure confidentiality, and consent was obtained for publication of anonymized data.

Total intravenous anaesthesia was induced using propofol and remifentanil via target-controlled infusion. Anaesthetic depth was monitored using processed electroencephalography (Patient State Index), with values maintained between 25 and 50. End-tidal carbon dioxide, invasive arterial blood pressure monitoring, core temperature, and oxygen saturation were maintained within normal physiological ranges.

Prior to induction, the radial artery in the non-dominant hand was cannulated under local infiltration for continuous intra-arterial blood pressure monitoring. Awake baseline haemodynamic values were recorded. Cerebral blood flow velocity in the middle cerebral artery (MCAv) was measured using transcranial Doppler ultrasonography, and regional cerebral oxygen saturation was monitored using near-infrared spectroscopy.

Baseline cerebral autoregulation was assessed during steady-state anaesthesia for 5 minutes. A mean arterial pressure (MAP) challenge was then performed using intravenous norepinephrine (0.01–0.05 μg/kg/min), titrated in ±5 mmHg increments per minute to achieve either a 20% increase from baseline MAP or an absolute rise of 15–20 mmHg (maximum 90 mmHg). The target was typically reached within 3–5 minutes. Post-challenge Mxa was calculated over a stable 300-second plateau without further titration. The challenge was terminated if heart rate decreased by ≥20% or arrhythmias occurred. Pre- and post-challenge Mxa values were compared; a positive ΔMxa indicated impaired baseline autoregulation. This protocol accounted for the pressure dependency of autoregulatory indices.

Continuous transcranial Doppler and near-infrared spectroscopy signals were preprocessed before Mxa computation. Doppler waveforms were band-pass filtered (0.5–10 Hz), and velocity envelopes were extracted using zero-crossing detection. Artefacts were excluded if pulsatility index was <6 kHz·cm, signal power <40% of baseline, or abrupt velocity shifts exceeded 20%. Near-infrared signals were filtered (0.01–0.5 Hz), and motion artefacts were corrected using spline interpolation. Data were aggregated into 300-second epochs (50 overlapping 10-second windows, 75% overlap); epochs with >5% artefact contamination were discarded. Mxa was calculated as the Pearson correlation coefficient between slow-wave fluctuations in MAP and mean MCA velocity. [13]

Cerebral autoregulation was defined using the mean flow index (Mxa), with values >0.3 considered indicative of impaired autoregulation. In patients who developed hypotension (≥20% reduction from baseline MAP), norepinephrine infusion was initiated to restore perfusion pressure. Changes in MCAv, Mxa, and cerebral blood flow were analysed before and after MAP augmentation.

Unilateral TCD monitoring targeted the right middle cerebral artery (M1 segment, 55-60 mm depth) using a 2 MHz DWL Doppler BoxX (Compumedics, Germany) with probe secured via temporal acoustic window headband (Lambda headframe) for continuous signal stability.[13,14] rSO_2 measured bilaterally (forehead sensors) using INVOS 5100C Near-Infrared Spectroscopy (Medtronic, USA).

ΔCBF was calculated as: ΔCBF = (MCAv_post - MCAv_pre) / MCAv_pre × 100% (%), where MCAv_pre = baseline mean flow velocity (cm/s) and MCAv_post = post-MAP challenge velocity. MAP change standardized to +20% from baseline (mmHg) [15].

Statistical Analysis

This was a prospective, single-center exploratory hypothesis-generating study (n=40) to determine feasibility of MAP-challenge MxA protocol in elective neuroradiology. No formal power calculation performed; sample size based on pilot data demonstrating 80% technical success rate for stable TCD acquisition. Primary aim: generate effect size estimates for future RCTs

Continuous variables were expressed as mean ± SD or median [IQR]. Comparisons were performed using appropriate parametric or non-parametric tests. Correlations were analysed using Pearson's coefficient. A p-value <0.05 was considered statistically significant.

RESULTS

Patient Characteristics

Forty patients were included in the final analysis. The median age was 58 years, and 57% were female. Baseline demographic characteristics, cardiovascular risk factors, and haemodynamic parameters were comparable between patients with preserved cerebral autoregulation (CA+) and those with impaired autoregulation (CA−).

Table 1. Baseline demographic and clinical characteristics

Variable	Total (n=40)	CA+ (n=19)	CA− (n=21)	p-value
Age (years)	58 [47–68]	59 [48–69]	56 [40–66]	NS
Female (%)	57	68	52	NS
High CV risk (%)	70	63	76	NS
Baseline MAP (mmHg)	95 ± 8	97 ± 7	94 ± 8	NS
ASA II–III (%)	95	95	95	NS

CA+: preserved cerebral autoregulation; CA−: impaired cerebral autoregulation; NS: non-significant.

Cerebral Autoregulation After Induction

Following induction of general anaesthesia, the mean MAP decreased to approximately 67 mmHg. Impaired cerebral autoregulation (Mxa >0.3) was identified in 21 patients (53%). No significant differences were observed between CA+ and CA− groups in terms of age, anaesthetic depth, end-tidal CO_2, temperature, or rSO_2 values.

Table 2. Monitoring parameters following anaesthesia induction

Parameter	CA+	CA−	p-value
MAP (mmHg)	70 ± 10	65 ± 7	NS
MCAv_mean (cm/s)	40 [28–46]	41 [33–49]	NS
rSO_2 (%)	64 ± 7	63 ± 7	NS
Mxa	0.02 ± 0.26	0.57 ± 0.18	<0.001

CA+: preserved cerebral autoregulation; CA−: impaired cerebral autoregulation; NS: non-significant; p-value <0.05 was considered statistically significant.

Effect of Mean Arterial Pressure Challenge

Twenty-two patients required a MAP challenge. In patients with impaired CA, MAP augmentation resulted in a significant increase in MCAv_mean and a marked reduction in Mxa, indicating restoration of autoregulatory function. In contrast, patients with preserved CA demonstrated minimal changes in cerebral blood flow parameters.

Table 3. Cerebrovascular response to MAP challenge

Parameter	CA+	CA−	p-value
ΔMAP (mmHg)	12 [8–16]	18 [13–20]	0.054
ΔMCAv_mean (cm/s)	1 [0.7–1.5]	7 [3–9]	<0.001
ΔMxa	+0.08	−0.34	<0.001
ΔCBF (%/%)	0.24 ± 0.09	0.55 ± 0.24	<0.001

CA+: preserved cerebral autoregulation; CA−: impaired cerebral autoregulation; p-value <0.05 was considered statistically significant.

Relationship Between Mxa and ΔCBF

A strong positive correlation was observed between baseline Mxa and ΔCBF ($r = 0.73$, $p < 0.001$), demonstrating that patients with more severely impaired autoregulation experienced greater increases in cerebral blood flow in response to MAP augmentation.

DISCUSSION

The present study demonstrates that impairment of cerebral autoregulation (CA) is a frequent occurrence following induction of general anaesthesia for neuroradiology procedures, affecting more than half of the studied population. This finding is clinically important, as cerebral autoregulation is a key protective mechanism that maintains stable cerebral blood flow (CBF) across a wide range of systemic blood pressures (1,2). Disruption of this mechanism renders cerebral perfusion pressure-dependent, increasing vulnerability to perioperative cerebral hypoperfusion or hyperperfusion.

Incidence and Clinical Relevance of Impaired Cerebral Autoregulation

In this cohort, 53% of patients exhibited impaired CA as defined by an Mxa value greater than 0.3 following induction of anaesthesia. This incidence is higher than traditionally anticipated in patients without overt traumatic brain injury or raised intracranial pressure. However, similar rates of autoregulatory impairment have been reported in patients undergoing cardiac surgery, carotid endarterectomy, and procedures requiring deep anaesthesia or rapid haemodynamic manipulation (3–5).

The high prevalence observed may be attributable to the combined effects of anaesthetic agents, abrupt reductions in mean arterial pressure (MAP), and pre-existing cerebrovascular pathology. Propofol, the primary induction agent used in this study, is known to reduce systemic vascular resistance and arterial pressure while also exerting direct cerebrovascular effects (6,7). Although cerebral metabolic rate is reduced under propofol anaesthesia, autoregulatory capacity may be transiently impaired during periods of haemodynamic instability, particularly immediately after induction (8).

Lack of Predictive Value of Conventional Risk Factors

A notable observation in this study was the absence of significant differences in age, cardiovascular risk factors, baseline blood pressure, anaesthetic depth, or regional cerebral oxygen saturation between patients with preserved and impaired CA. These findings are consistent with prior investigations demonstrating that interindividual variability in cerebral autoregulatory limits is substantial and poorly predicted by demographic or clinical characteristics alone [16,17].

Chronic hypertension has traditionally been thought to shift the autoregulatory curve rightward, necessitating higher perfusion pressures to maintain adequate CBF [18]. However, several studies have shown that acute intraoperative autoregulatory failure may occur independently of baseline blood pressure or hypertensive status [9,10]. This reinforces the limitation of population-based blood pressure targets and highlights the inadequacy of using MAP thresholds alone as surrogates for cerebral perfusion adequacy.

Effect of Mean Arterial Pressure Challenge

The MAP challenge performed in hypotensive patients represents one of the most clinically relevant components of this study. In patients with impaired CA, augmentation of MAP using norepinephrine resulted in a significant increase in MCAv_mean and a marked reduction in Mxa values, indicating partial or complete restoration of autoregulatory function. These findings suggest that many patients with impaired CA were operating below their lower limit of autoregulation (LLA) following induction.

Previous studies have demonstrated that restoring MAP above the LLA improves cerebral perfusion and reduces pressure-dependent fluctuations in CBF [11,19,20]. The present findings extend this concept to neuroradiology patients and emphasize that autoregulatory failure after induction is often functional and reversible rather than fixed.

In contrast, patients with preserved CA exhibited minimal changes in MCAv_mean following MAP augmentation, consistent with intact autoregulatory buffering. Interestingly, a small subset of patients demonstrated worsening Mxa values following aggressive MAP elevation, suggesting possible overshoot beyond the upper limit of autoregulation (ULA). This observation aligns with the concept of a narrow optimal perfusion window and underscores the risk of indiscriminate vasopressor-driven blood pressure escalation [21].

Relationship Between Static and Dynamic Autoregulation Metrics

A strong positive correlation was observed between baseline Mxa and cerebral blood flow variation (ΔCBF) during the MAP challenge. Patients with higher Mxa values experienced larger increases in CBF in response to pressure augmentation, indicating greater pressure dependency of cerebral perfusion.

Mxa is a well-validated index of dynamic cerebral autoregulation derived from the correlation between spontaneous fluctuations in MAP and cerebral blood flow velocity [22,23]. The present findings support its physiological relevance and demonstrate its concordance with a more intuitive, flow-based measure such as ΔCBF. Similar relationships have been reported in neurocritical care and cardiac surgery populations, where impaired autoregulation has been associated with pressure-passive cerebral circulation and worse neurological outcomes [12,24,25].

Importantly, ΔCBF and Mxa identified autoregulatory impairment even when MAP values remained within conventionally accepted limits. This finding further challenges the reliance on absolute or relative blood pressure thresholds for intraoperative cerebral protection.

Implications for Intraoperative Blood Pressure Management

Current guidelines for intraoperative blood pressure management largely recommend maintaining MAP above fixed absolute values or within a percentage of baseline [26]. However, these recommendations do not account for patient-specific autoregulatory limits and may fail to prevent cerebral hypoperfusion in susceptible individuals.

The present study supports a growing body of evidence advocating for individualized, autoregulation-guided haemodynamic management. Autoregulation-guided MAP targeting has been shown to reduce postoperative delirium and organ dysfunction in cardiac surgery patients [15,19]. Given the vulnerability of neuroradiology patients to cerebral ischemia, this approach may be particularly beneficial in this population.

Integrating cerebral autoregulation monitoring into routine anaesthetic practice could enable clinicians to identify patients at risk of cerebral hypoperfusion early and tailor vasopressor therapy accordingly. While technical and logistical challenges remain, advances in non-invasive monitoring technologies may facilitate broader implementation in the future.

Limitations

This study has few limitations. Being single-centre, its findings may not be generalisable to other settings. The modest sample size (n = 40) limits statistical power and may overlook smaller but clinically relevant effects. Middle cerebral artery velocity was used as a surrogate for cerebral blood flow, and technical factors such as insonation angle could have influenced measurements. Postoperative neurological outcomes were not assessed, as the focus was restricted to intraoperative autoregulatory indices. These physiological findings require validation against clinically meaningful outcomes in adequately powered prospective trials. Nevertheless, the study establishes the technical feasibility of implementing a MAP-challenge–derived Mxa protocol during elective neuroradiological procedures and provides preliminary effect size estimates to guide the design and sample size calculations of future outcome-driven studies.

CONCLUSION

Impaired cerebral autoregulation frequently occurs immediately after induction of general anaesthesia in patients undergoing neuroradiological procedures. Importantly, this disturbance appears predominantly functional rather than structural, as targeted mean arterial pressure augmentation restores autoregulatory capacity in a substantial proportion of patients. These findings underscore the dynamic and potentially reversible nature of peri-induction cerebrovascular dysregulation and provide a compelling rationale for individualized, autoregulation-guided haemodynamic strategies aimed at preserving cerebral perfusion and improving neurological outcomes.

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