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Pressure Is Not Perfusion: Reconciling the Strong Association between Intraoperative Hypotension and Organ Injury with Null Randomised Trials of Higher Blood-Pressure Targets

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ABSTRACT

Background. Intraoperative hypotension is common, is graded in both depth and duration against postoperative organ injury, and is directly modifiable. It has accordingly become a central perioperative quality target. Randomised trials of higher intraoperative blood-pressure targets have nonetheless failed to reduce these outcomes. This review addresses the resulting gap between association and intervention.

Methods. We conducted a structured narrative review of the observational, interventional and predictive-monitoring literature on intraoperative hypotension in adult non-cardiac surgery, together with contemporary consensus recommendations. We appraised four candidate explanations for the gap and derived a management framework consistent with current evidence.

Results. Definitional heterogeneity is severe: one review identified 140 definitions across 130 studies, with mean arterial pressure thresholds from below 50 to below 75 mmHg. Around 29 per cent of non-cardiac cases accumulate at least 15 minutes below 65 mmHg, and the association with acute kidney injury is dose-dependent. Against this, a multinational randomised comparison of a hypotension-avoidance strategy targeting 80 mmHg or above with a hypertension-avoidance strategy achieved higher pressures but produced no reduction in acute kidney injury or major vascular complications. Machine-learning prediction reduces process measures of hypotension but has not been shown to improve outcomes, nor clearly to outperform a simple threshold alarm.

Conclusion. The evidence supports avoiding hypotension rather than pursuing a number. Pressure is a surrogate for perfusion, the means used to raise it determines whether perfusion follows, and a vasopressor-driven rise is not the same intervention as preventing hypotension. We propose an individualised floor, cause-directed treatment before pressor escalation, and measurement of cumulative exposure rather than nadir.

1. Introduction

Few perioperative exposures have been studied as intensively over the past decade as intraoperative hypotension, and few have produced a more uncomfortable evidential position. The observational case is about as strong as observational evidence in this field becomes: hypotension is common, its association with organ injury is graded in both depth and duration, the association is reproducible across surgical populations and institutions, and it is biologically coherent, since organ perfusion pressure is the proximate determinant of oxygen delivery in vascular beds with limited autoregulatory reserve.

The exposure is also, uniquely among perioperative risk factors, under the direct minute-to-minute control of the anaesthetist. That combination — strong association, plausible mechanism, immediate modifiability — is precisely what converts an epidemiological finding into a quality target, and intraoperative hypotension has duly become one, appearing in departmental dashboards, in institutional quality metrics and in commercial monitoring products designed to prevent it.

Randomised evidence has not cooperated. Strategies designed to keep intraoperative blood pressure higher succeed in keeping it higher, and do not reduce the outcomes that the association predicted they would. This

review is concerned with that gap. Our purpose is neither to defend the conventional target nor to argue that hypotension is harmless, but to set out what the evidence supports, what it does not, and how an anaesthetist should act in the interval before the question is settled.

Objectives

We set out to characterise the definitional landscape of intraoperative hypotension; to summarise the observational association and its shape; to summarise the randomised evidence on higher blood-pressure targets; to appraise four candidate explanations for the divergence between the two; to assess whether predictive haemodynamic monitoring alters the position; and to derive a management framework that is defensible under the current state of evidence.

2. Methods

This is a structured narrative review rather than a systematic review. No protocol was registered and no formal screening process was applied, and the evidence cited is therefore illustrative of the field rather than exhaustive of it. We searched the published literature for studies of intraoperative hypotension in adult non-cardiac surgery addressing definition, association with postoperative organ injury, randomised comparison of blood-pressure management strategies, and predictive haemodynamic monitoring, and we consulted contemporary consensus recommendations.

No primary data were collected and no individual patient records were accessed. Institutional review board approval and informed consent were therefore not applicable, and no trial registration applies. The management framework in Section 8 is the authors' synthesis and is offered as a proposal consistent with current evidence rather than as a validated protocol or a guideline.

3. One exposure, many definitions

Before the association can be interpreted, the exposure must be defined, and here the literature is in considerable disorder. A systematic review of 130 studies identified 140 distinct definitions of intraoperative hypotension [1]. Reported risk thresholds span mean arterial pressures from below 50 to below 75 mmHg and systolic pressures from below 70 to below 100 mmHg [2] (Figure 1, Table 1).

The heterogeneity is not merely numerical. Definitions divide between absolute thresholds and relative falls from a patient's own baseline; between instantaneous nadir, cumulative time below threshold, area under the threshold, and time-weighted average; and some incorporate the clinician's response, such as the administration of fluid or vasopressor, which makes the exposure partly a function of treatment [1]. Baseline itself is inconsistently derived, sometimes from a preoperative clinic reading, sometimes from the first reading in the anaesthetic room after anxiolysis, sometimes from the first reading after arrival in theatre — values that may differ substantially in the same patient.

Two consequences follow. Pooled estimates across studies using incompatible exposure definitions should be read cautiously. And a departmental quality metric based on nadir pressure measures something different from one based on cumulative time below a threshold, with the latter far closer to what the association literature actually describes.

Table 1. Dimensions along which definitions of intraoperative hypotension differ.

Dimension	Options in use	Consequence for interpretation
Pressure variable	Mean arterial pressure; systolic pressure	Systolic and mean thresholds are not interchangeable across patients
Reference	Absolute threshold; percentage fall from the patient's baseline	Absolute thresholds may be too low for chronically hypertensive patients and unnecessarily high for others
Metric of exposure	Nadir; cumulative minutes below threshold; area under threshold; time-weighted average	Nadir discards duration, which the association literature shows to matter
Baseline derivation	Preoperative clinic reading; pre-induction reading; first theatre reading	Different baselines yield different relative thresholds in the same patient
Treatment incorporation	Some definitions require fluid or vasopressor administration	Makes the exposure partly a function of the clinician's response
CLINICAL BOTTOM LINE Specify the definition before citing a risk estimate or setting a departmental target. A metric based on cumulative time below an individualised threshold is closer to the evidence than one based on the lowest recorded pressure.		

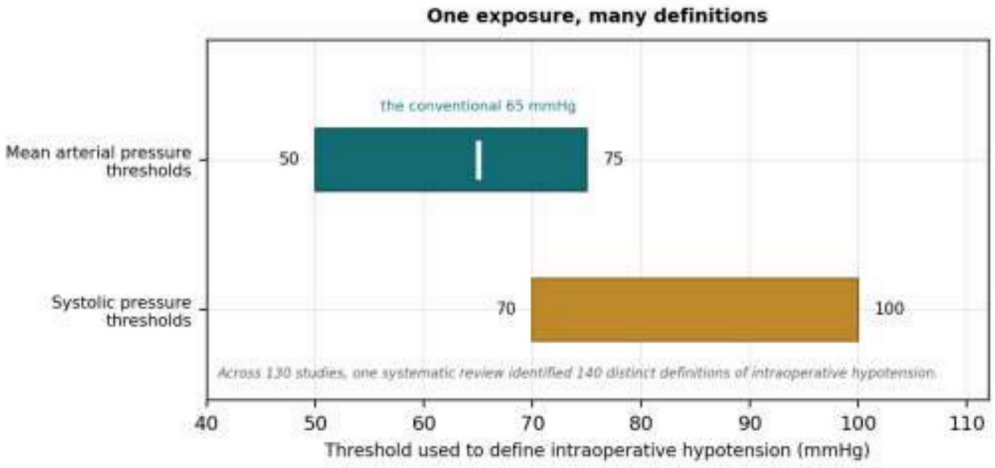

Figure 1. The range of blood-pressure thresholds used to define intraoperative hypotension across the published literature. Data sources: references [1,2].

4. The observational association

Intraoperative hypotension is frequent. In one large community-based analysis, 29 per cent of non-cardiac cases accumulated at least 15 minutes with a mean arterial pressure below 65 mmHg [3]. Associations have been reported with acute kidney injury, myocardial injury after non-cardiac surgery, postoperative delirium, stroke and death, and contemporary cohort work demonstrates that these risks are graded and depend strongly on the cumulative duration of hypotension as well as its depth [3] (Figure 2).

For acute kidney injury specifically, a systematic review concluded that a mean arterial pressure below 65 mmHg sustained for more than five minutes is strongly associated with postoperative injury, with risk rising with both magnitude and duration; the relation was dose-dependent in non-cardiac surgery, and in cardiac surgery appeared dose-independent [2]. Cohort analyses stratifying by pressure band have reported that maintaining a mean arterial pressure at or above 65 mmHg, and at or above 75 mmHg, was associated with lower odds of acute kidney injury relative to pressures below 55 mmHg for at least 10 minutes [4].

Two qualifications belong here and are frequently omitted when these findings are cited in support of targets. First, the association is not uniform across outcomes: in the same analyses in which acute kidney injury and length of stay tracked hypotension, in-hospital and 30-day mortality did not, associating instead with cardiac history [4]. Second, the threshold that matters may differ by patient. In chronically hypertensive patients the renal autoregulatory curve is displaced rightward, and cohort work in this group has suggested a different operative threshold from that derived in unselected populations [5]. A single universal number is unlikely to be correct for everyone.

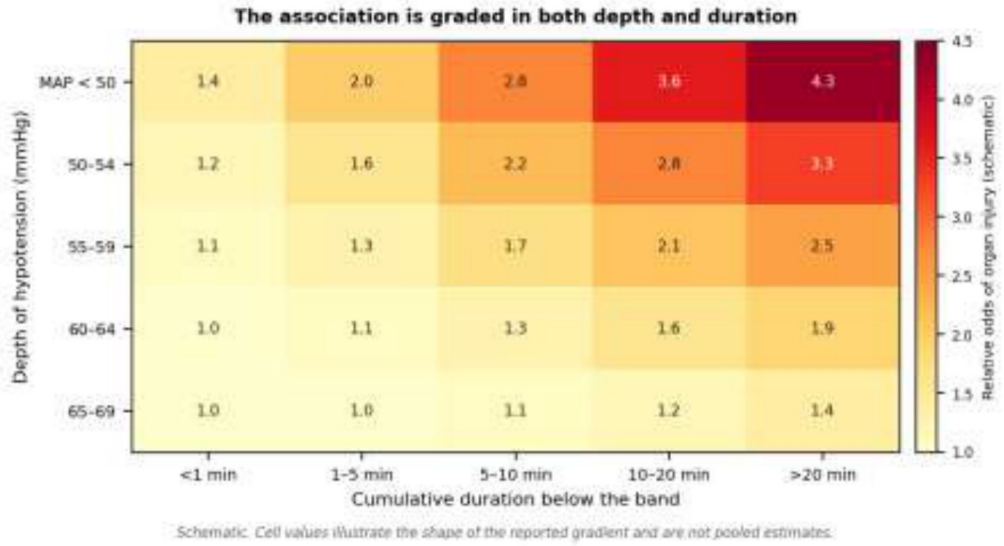

Figure 2. Schematic representation of the graded association between depth and cumulative duration of intraoperative hypotension and postoperative organ injury. Cell values illustrate the reported shape of the gradient and are not pooled estimates. Based on the relationships described in references [2–4].

Table 2. Summary of the observational association in non-cardiac surgery.

Outcome	Nature of the association	Shape	Consistency
Acute kidney injury	Deeper and longer hypotension associated with higher incidence	Dose-dependent in depth and duration	High across cohorts
Myocardial injury after non-cardiac surgery	Associated with hypotensive exposure	Graded	Moderate to high
Postoperative delirium	Associated with hypotensive exposure	Graded	Moderate
Stroke	Associated with hypotensive exposure	Reported but less well characterised	Moderate
Mortality	Inconsistent; in some analyses attenuates on multivariable adjustment	Unclear	Low to moderate

Sources: references [2–5]. Consistency ratings are the authors' appraisal of agreement across the cohorts cited.

5. The randomised evidence

The decisive test is whether a strategy that produces higher intraoperative pressures reduces organ injury. A large multinational partial-factorial randomised trial addressed exactly this. Patients aged 45 years or older, with or at risk of atherosclerotic disease, taking at least one antihypertensive medication and scheduled for non-cardiac surgery, were randomised to a hypotension-avoidance strategy or a hypertension-avoidance strategy [6,7]. The hypotension-avoidance strategy targeted an intraoperative mean arterial pressure of 80 mmHg or above, and on the day of surgery and for two days afterwards held renin–angiotensin–aldosterone system inhibitors and used other antihypertensives in stepwise fashion according to systolic pressure [7].

The prespecified acute kidney injury sub-study included 7,307 patients from 110 hospitals in 22 countries [7]. Despite achieving greater cessation of antihypertensive medication and higher intraoperative mean arterial pressures in the intervention arm, no difference in the rate of postoperative acute kidney injury was observed [7,8]. For the broader endpoint, the trial reported no difference between the two strategies in major vascular complications among patients receiving long-term antihypertensive therapy [6].

This is a well-conducted, adequately sized, multinational randomised comparison that achieved its intended separation in the exposure and found nothing on the outcome. It cannot be dismissed as underpowered or as a failure of protocol adherence, and any account of intraoperative blood pressure management must accommodate it (Figure 3, Table 3).

It is equally important to state what the trial does not establish. It compared two management strategies in a specific population — patients on long-term antihypertensive therapy — and both arms will have received

active blood-pressure management. It did not compare permissive hypotension against strict avoidance, and it does not license indifference to profound or prolonged hypotension, which no trial has tested and which no clinician would defend.

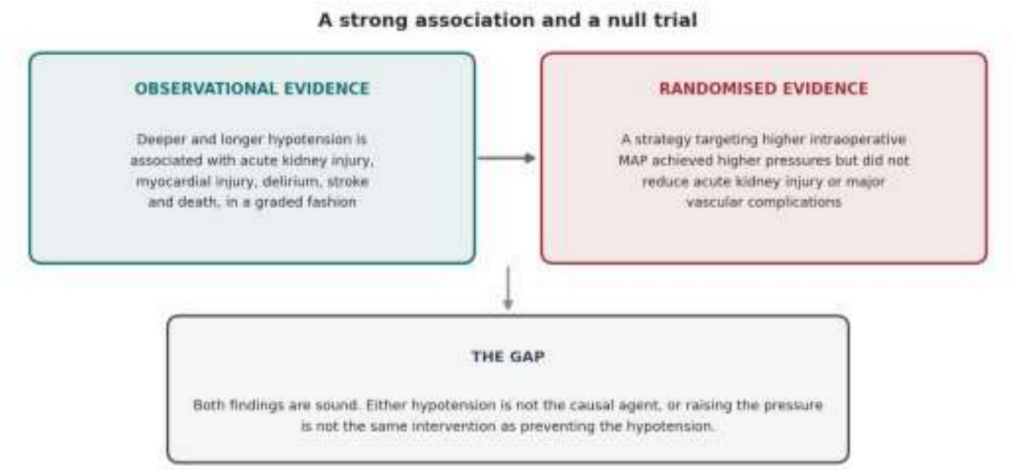

Figure 3. The association–intervention gap. Both bodies of evidence are methodologically sound; reconciling them requires an account of why raising the pressure did not reproduce the benefit the association implied.

Table 3. Principal randomised evidence on intraoperative blood-pressure targets.

Feature	Hypotension-avoidance strategy	Hypertension-avoidance strategy	Result
Intraoperative target	Mean arterial pressure ≥80 mmHg	Usual intraoperative management	Separation achieved
Antihypertensive handling	Hold RAAS inhibitors on day of surgery and for two days after; other agents stepwise by systolic pressure	Continue usual antihypertensive regimen	Greater cessation in the intervention arm
Population	Aged ≥45 years, at or at risk of atherosclerotic disease, on ≥1 antihypertensive, non-cardiac surgery	Same	7,307 patients in the AKI sub-study
Setting	110 hospitals, 22 countries	Same	Multinational
Acute kidney injury	—	—	No difference
Major vascular complications	—	—	No difference

RAAS = renin–angiotensin–aldosterone system. Sources: references [6–8].

6. Four explanations for the gap

Four explanations can account for the divergence, and none has been excluded (Figure 4, Table 4). They are not mutually exclusive.

6.1 Confounding By Indication

Hypotension during anaesthesia is rarely a random event. It accompanies haemorrhage, sepsis, cardiac dysfunction, frailty, and deep anaesthesia — each of which independently predicts postoperative organ injury. A patient who becomes hypotensive is, on average, a sicker patient having a more difficult operation, and multivariable adjustment can address only measured confounders. On this account, hypotension is a marker rather than a mechanism, and correcting the marker leaves the underlying process untouched. The observation that mortality associations attenuate on adjustment while acute kidney injury associations persist [4] is at least consistent with partial confounding.

6.2 The Means of Raising the Pressure

This is, in our view, the most actionable explanation, and it turns on a distinction that the exposure variable obscures. Mean arterial pressure can be raised by lightening anaesthesia, by restoring circulating volume, or by vasoconstriction. The first two raise pressure by restoring flow. The third raises pressure by increasing resistance, and may do so while regional flow falls (Figure 5).

If a substantial part of the pressure difference achieved in a trial is generated by vasopressor, then the trial has tested a composite of a higher number and a larger pressor dose. Vasoconstrictors carry recognised risks of splanchnic and renal hypoperfusion and of arrhythmia, and the benefit of the higher pressure may be offset by the harm of the means used to achieve it. Under this account the association may be causal and the trial still null, because the intervention delivered was not the intervention the association implied.

6.3 The wrong contrast

The randomised comparison set a target of 80 mmHg or above against usual care, in a trial population receiving active management in both arms. If the threshold below which injury occurs lies substantially lower — and consensus recommendations set a considerably lower floor, discussed in Section 7 — then both arms may have spent most of the operation above the region in which harm accrues. A null result in that circumstance is uninformative about the region of the exposure distribution that matters. Testing 80 against usual care is not the same as testing 50 against 65.

6.4 Heterogeneous susceptibility

A universal threshold assumes a common autoregulatory range. Chronic hypertension shifts the cerebral and renal autoregulatory curves rightward, so that a pressure that is comfortably adequate in a normotensive patient may be below the autoregulatory knee in a patient with long-standing untreated hypertension; cohort evidence in hypertensive populations is consistent with a different operative threshold [5]. Averaged across a heterogeneous trial population, a benefit confined to a susceptible subgroup and a harm confined to another may cancel.



Figure 4. Four candidate explanations for the divergence between the observational association and the randomised result.

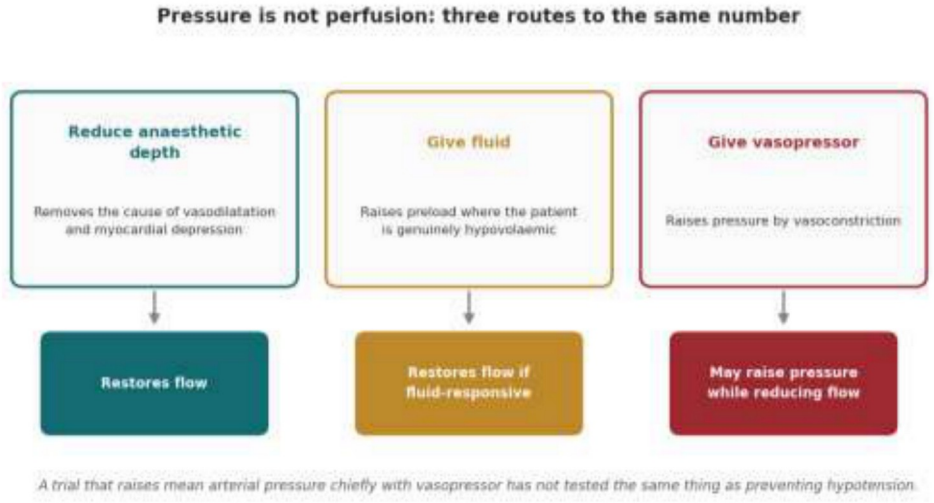

Figure 5. Three routes to the same mean arterial pressure, with differing consequences for organ blood flow.

Table 4. Appraisal of the four explanations.

Explanation	What would support it	What would refute it	Standing
Confounding by indication	Attenuation of associations with fuller adjustment; discordance between outcomes	Persistence of dose-response after rigorous confounder control or within-patient designs	Plausible
The means of raising pressure matters	Trials separating flow-restoring from vasoconstrictor routes to the same pressure	Equivalent outcomes regardless of the agent used to achieve the target	Plausible and testable
The wrong contrast was tested	A trial comparing a low threshold against a moderate one rather than a moderate against a high one	Null result in a trial contrasting low against moderate thresholds	Plausible
Heterogeneous susceptibility	Interaction between baseline blood pressure and target in trial subgroups	Absence of interaction in adequately powered subgroup analysis	Plausible

CLINICAL BOTTOM LINE The gap is not evidence that hypotension is harmless. It is evidence that raising a number is not the same intervention as preventing the physiological state the number stands for. The most useful clinical corollary is to treat the cause of the low pressure preferentially, and to use a vasoconstrictor as a bridge rather than as the treatment.

7. Can we predict and prevent it?

If hypotension is harmful and reactive management is inherently late, then predicting it should permit pre-emptive action. Machine-learning prediction from the arterial pressure waveform has been developed for this purpose, generating a unitless index that indicates the likelihood of an impending mean arterial pressure below 65 mmHg for at least one minute, with an alarm above a defined index value [9,10].

The evidence has a characteristic shape, and it is worth setting out as a ladder of increasingly demanding questions (Figure 6, Table 5). The technology predicts hypotension — that much is established. Meta-analyses of randomised trials find that its use reduces process measures of hypotension such as time-weighted average mean arterial pressure below threshold and the duration of hypotensive episodes [11,12]. Whether it outperforms a simple threshold alarm is less clear: a prospective observational comparison found the index no better than a mean arterial pressure threshold at predicting hypotension during non-cardiac surgery [11]. And whether it improves patient outcomes is not established; trials have been inconsistent, with a small trial suggesting reduced hypotension and a larger trial not, and none powered for organ injury [11,13].

The interpretive difficulty is the same one that runs through this review. Reducing a process measure of hypotension is a benefit only if hypotension is causal and if the means of reducing it is not itself harmful. A monitor that prompts earlier vasopressor administration will reliably reduce the amount of recorded hypotension, and whether that helps the patient is precisely the question the randomised blood-pressure trials left open.

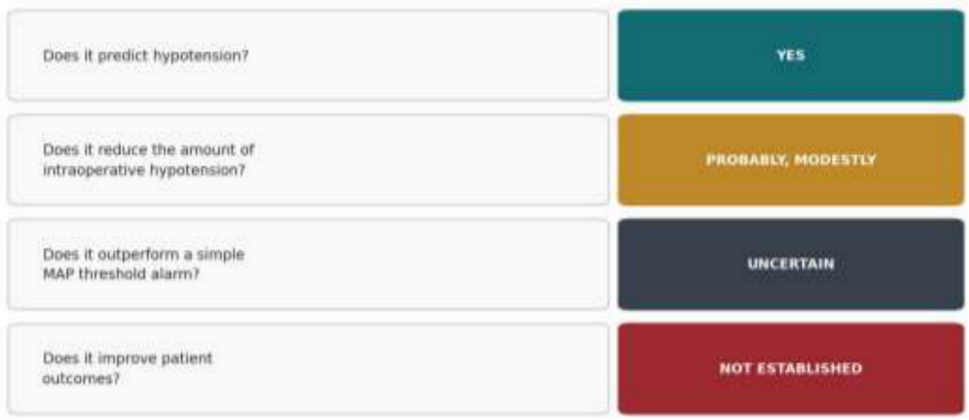

Figure 6. The evidence ladder for predictive haemodynamic monitoring. Each rung is a more demanding question than the one above it, and the evidence weakens as the questions become more clinically consequential.

Table 5. Evidence status for predictive haemodynamic monitoring in intraoperative hypotension.

Question	Evidence	Status	Implication
Does it predict hypotension?	Derived and validated for prediction of mean arterial pressure <65 mmHg for ≥1 min	Established	The prediction task is solved
Does it reduce measured hypotension?	Meta-analyses of randomised trials report reductions in time-weighted average and duration	Probable, modest	Process improvement demonstrated
Does it beat a simple threshold alarm?	Prospective observational comparison found no superiority over a mean arterial pressure threshold	Uncertain	Incremental value over cheaper alternatives unproven
Does it improve patient outcomes?	Trials inconsistent; none powered for organ injury	Not established	Adoption on outcome grounds is premature

Sources: references [9–13].

8. A framework consistent with the evidence

What should be done on Monday morning? Contemporary consensus from a multidisciplinary expert group, using a modified Delphi approach, recommends keeping intraoperative mean arterial pressure at or above 60 mmHg in at-risk patients [14]. That figure is notably lower than the 80 mmHg target tested and found null in the randomised comparison, and lower than the 65 mmHg that dominates the observational literature and departmental dashboards. The discrepancy is instructive: the consensus floor is set where the evidence of harm is strongest, not where the association is most often measured.

Figure 7 and Table 6 set out a framework. Five elements deserve emphasis.

Establish a baseline that means something. If a relative threshold is to be used, the baseline must be a resting, unpremedicated value, not the first anxious reading in the anaesthetic room.

Set an individualised floor. A defensible operating rule is the higher of an absolute floor and a proportional fall from the patient's own baseline, which accommodates the rightward-shifted autoregulation of the chronically hypertensive patient without imposing a high universal target that the randomised evidence does not support.

Diagnose before treating. A fall in pressure is a sign, not a diagnosis. Anaesthetic depth, hypovolaemia, occult bleeding, obstruction and cardiac dysfunction each call for a different response, and the response that corrects the cause restores flow as well as pressure.

Use vasopressors as a bridge. Pressors buy time while the cause is corrected. Sustained pressor infusion to hold a numerical target, in the absence of a diagnosed cause, is the practice for which the randomised evidence offers least support.

Measure exposure, not nadir. Departmental quality metrics should record cumulative depth and duration below the individualised floor, which is what the association literature describes and what a nadir-based metric cannot capture.

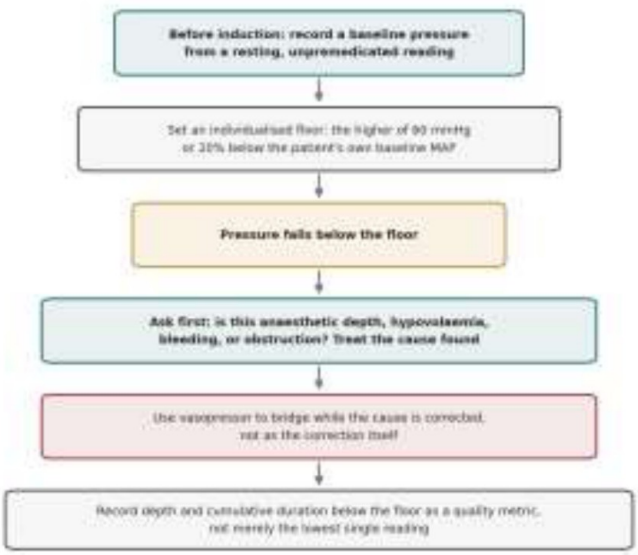

Figure 7. A proposed framework for intraoperative blood-pressure management consistent with the current evidence. This is the authors' synthesis and is not a validated protocol.

Table 6. Proposed management framework with the evidential basis for each element.

Element	Recommendation	Evidential basis	Strength
Baseline	Record a resting, unpremedicated pre-induction pressure	Definitional literature on baseline derivation	Practice standard
Floor	The higher of an absolute floor of 60 mmHg or a 20% fall from the patient's own baseline mean arterial pressure	Consensus recommendation plus cohort evidence of heterogeneous thresholds	Moderate
First response	Diagnose and treat the cause: anaesthetic depth, hypovolaemia, bleeding, obstruction	Physiological; supported by the pressure-versus-flow distinction	Moderate
Vasopressor use	Bridge while the cause is corrected, rather than as the correction	Inferred from the null randomised result and vasoconstrictor pharmacology	Weak to moderate
Metric	Cumulative depth and duration below the individualised floor	Association literature is duration-dependent	Moderate
Predictive monitoring	Not required; if used, on process grounds rather than outcome grounds	Evidence ladder in Table 5	Weak

Table 7. Research priorities arising from this review.

Question	Design that would answer it	Priority
Does the route to a given mean arterial pressure alter outcome?	Randomised comparison of flow-restoring versus vasoconstrictor-led strategies achieving equivalent pressures	High
Where is the threshold below which harm accrues?	Randomised comparison of a low against a moderate floor, rather than moderate against high	High
Does susceptibility vary with chronic blood pressure?	Prespecified interaction analysis of target by baseline pressure in an adequately powered trial	High
Does predictive monitoring improve outcomes?	Trial powered for organ injury, with a simple threshold alarm as comparator	Moderate
Which exposure metric best predicts injury?	Head-to-head comparison of nadir, cumulative time, area under threshold and time-weighted average in a common cohort	Moderate

9. Limitations

This is a structured narrative review, not a systematic review, and no protocol was registered. The evidence cited was selected to illustrate the structure of the argument, and a systematic appraisal might weight individual studies differently or identify contrary findings we have not discussed.

The randomised evidence rests substantially on a single large trial and its sub-studies. That trial was well conducted and multinational, but it examined a specific population — patients aged 45 years and older on long-term antihypertensive therapy having non-cardiac surgery — and its result should not be extended without qualification to obstetric, paediatric, cardiac, neurosurgical or emergency populations, in each of which the perfusion requirements and the relevant thresholds may differ.

The four explanations in Section 6 are advanced as candidate accounts and appraised for plausibility, not tested. We have indicated for each what would support and what would refute it, but the review cannot adjudicate between them, and readers should not take our emphasis on the second as evidence for it.

Figure 2 is schematic. It represents the shape of the reported dose-response relationship and must not be used to derive risk estimates or thresholds for individual patients.

Finally, the framework in Section 8 is the authors' synthesis. It has not been prospectively evaluated, and the numerical floor it proposes is drawn from a consensus recommendation rather than from a trial that tested that floor against an alternative. Its principal claim to attention is that it is consistent with both the observational and the randomised evidence, which few current departmental protocols are.

10. Conclusion

Intraoperative hypotension is common, its association with postoperative organ injury is graded and reproducible, and a large randomised comparison found that targeting a higher intraoperative mean arterial pressure did not reduce that injury. Both findings are sound, and the reconciliation is not that one of them is wrong.

The most defensible reading is that mean arterial pressure is a surrogate for organ perfusion, that the relationship between the two depends on how the pressure is generated, and that a trial raising pressure substantially by vasoconstriction has not tested the same intervention that the association implies. On that reading the clinical instruction changes in an important way: from pursuing a target to preventing and diagnosing the state, and from treating the number to treating its cause.

Until the trials proposed in Table 7 are done, the position that fits the evidence is a modest individualised floor, cause-directed first response, vasopressors as a bridge, and a quality metric that records cumulative exposure rather than the lowest reading of the day.

Declarations

Ethical approval. Not applicable.

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Conflicts of interest. The authors declare that there are no conflicts of interest regarding the publication of this paper.

Data availability. The data that support the findings of this study are available from the corresponding author upon reasonable request.

Author contributions. Makbul Hussain Choudhury: Conceptualization, Methodology, Software, Validation, Investigation, Data curation, Writing – original draft, Writing – review & editing, Visualization, Supervision, Project administration, Funding acquisition.

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