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PERFUSION BEYOND ARTERIAL PRESSURE IN THE PERIOPERATIVE PERIOD: FROM MACRODYNAMICS TO MICRODYNAMICS AND THE EFFECTIVE PERFUSION GRADIENT

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Abstract : Mean arterial pressure (MAP) has traditionally been used as the main target for perioperative hemodynamic management; however, its normalization does not guarantee effective tissue perfusion. Perfusion occurs at the microvascular level and depends on the interaction between pressure, flow, capillary distribution, and the effective perfusion gradient, defined by the difference between arterial inlet pressure and venous or compartmental outlet pressures. In the perioperative period, multiple factors such as anesthesia, surgical inflammation, vasoplegia, hemodilution, use of vasopressors, and positive pressure ventilation can cause a loss of hemodynamic coherence, in which acceptable macrodynamic parameters coexist with persistent tissue hypoperfusion. This discordance explains the onset of organ dysfunction, particularly renal dysfunction, even in the presence of a MAP considered adequate. The assessment of perfusion requires a multimodal approach, integrating clinical and metabolic markers of microperfusion (capillary refill time, perfusion index, lactate, and diuresis), along with imaging tools such as point-of-care ultrasound (POCUS) to assess cardiac function, venous congestion, and compartment pressures. In this context, increased central venous pressure or intra-abdominal pressure can significantly reduce the perfusion gradient and compromise effective oxygen delivery. An integrative perioperative approach is proposed, based on a sequential assessment of macrodynamics, microdynamics, and effective perfusion gradient, followed by interventions guided by hemodynamic phenotype and dynamic reassessment. This paradigm shift promotes personalized, physiologically oriented hemodynamic management focused on actual perfusion, with the potential to optimize

outcomes and reduce perioperative organ dysfunction.

Keywords: hemodynamic monitoring, tissue perfusion, microcirculation.

INTRODUCTION

Mean arterial pressure (MAP) has traditionally been the main target of perioperative hemodynamic management, as it is considered a practical surrogate for tissue perfusion. This approach is supported by evidence linking intraoperative hypotension to adverse outcomes such as acute kidney injury, myocardial damage, and increased mortality(1) .

However, persistent clinical observation of organ dysfunction in patients who maintain MAP values within "acceptable" ranges has called into question the validity of blood pressure as the sole marker of effective perfusion.

Tissue perfusion depends on the interaction between blood flow, functional microcirculation, and effective pressure gradient, rather than on an isolated pressure reading. In the perioperative period, factors such as the inflammatory response to surgery, vasoplegia, hemodilution, the use of vasopressors, and mechanical ventilation can cause a dissociation between macrodynamics and microdynamics, a phenomenon known as loss of hemodynamic coherence(2)

In this scenario, normalization of MAP does not guarantee adequate tissue oxygenation or efficient oxygen delivery at the capillary level.

Additionally, the perfusion of vulnerable organs, such as the kidney and brain, is determined by the perfusion gradient between arterial inflow pressure and venous or compartmental outflow pressures(3)

Venous congestion, increased intra-abdominal pressure, or right heart dysfunction can significantly reduce this gradient, precipitating organ hypoperfusion even with apparently adequate MAP values. This understanding has driven a conceptual shift toward more individualized hemodynamic targets oriented toward actual perfusion(4) .

The objective of this narrative review is to synthesize the current state of knowledge on perioperative perfusion beyond blood pressure, integrating the concepts of macrodynamics, microdynamics, and effective perfusion gradient, and to discuss their clinical implications for more precise and personalized hemodynamic management in the perioperative patient.

TISSUE PERFUSION

Tissue perfusion is not synonymous with blood pressure. In physiological terms, perfusion means delivering blood with the capacity to transport oxygen and substrates to the functional capillary bed, in sufficient quantity and with adequate distribution to sustain cellular metabolism. Therefore, perfusion involves three inseparable elements: effective flow, competent microcirculation, and a useful pressure gradient between the inlet and outlet of the vascular territory(1) .

It is important to differentiate between three concepts that are often confused in practice:

- Pressure (MAP): average driving force of the arterial system. It is necessary to maintain perfusion in pressure-dependent territories, but

it does not "guarantee" capillary exchange.

- Flow (CO/DO_2): volume of blood per unit of time. It provides overall availability, but may be poorly distributed (shunts, heterogeneity).
- Perfusion (microcirculation + gradient): final result at the tissue level. If the capillary is not recruited or the flow is heterogeneous, the cell may be hypoperfused even if the monitor shows an "acceptable" MAP.

This framework is crucial in the perioperative period because surgery and anesthesia simultaneously modify macrohemodynamics, vasomotor tone, endothelial inflammation, and "outflow" pressures (venous/compartmental). Therefore, perfusion should be understood as a multilevel phenomenon, and the clinical goal is not only to "correct numbers" but to restore effective oxygen delivery where it is actually used(2) single centre, randomised controlled trial (Bottomline-CS trial .

MACRODYNAMICS: CLINICAL UTILITY AND PHYSIOLOGICAL LIMITS

Macrodynamics parameters

Macrodynamics describes the overall state of the cardiovascular system and allows minimum hemodynamic safety to be ensured. Its most commonly used variables in the perioperative period include:

Mean arterial pressure (MAP): operational indicator of average arterial perfusion pressure.

Cardiac output (CO)/cardiac index: determinant of overall flow.

/Variation in systolic volume: useful for evaluating response to systolic volume and the impact of interventions.

Arterial oxygen saturation (SaO_2) and hemoglobin (Hb): determine arterial oxygen content and, therefore, global oxygen delivery (DO_2).

These variables remain essential: critically low MAP, insufficient CO, or inadequate SaO_2/Hb directly compromise perfusion capacity. The problem is not their use, but overinterpretation: assuming that "MAP in range" equals "guaranteed perfusion"(5) .

Why MAP is not perfusion

MAP is a necessary component of perfusion, but it does not define it. There are three main physiological reasons for this:

Microcirculation can be dysfunctional even if MAP is normal.

Metabolic exchange depends on perfused capillaries and homogeneous microvascular flow. In states of inflammation, vasoplegia, endothelial damage, or glycocalyx alteration, flow can become heterogeneous: areas with shunting ("fast" flow without exchange) and areas with stasis (hypoxia). In this context, raising MAP with vasopressors can increase pressure without improving capillary recruitment(6).

- Actual perfusion depends on flow and its distribution, not just pressure. Adequate MAP with low CO (or unfavorable regional redistribution) can maintain pressure but not deliver effective flow to

vulnerable organs, especially if autoregulation is impaired (elderly, chronic hypertension, perioperative sepsis, major surgery)(7).

- MAP ignores the "outflow side" of the system: venous congestion and compartment pressures.

Even with "good" MAP, if venous pressure or intra-abdominal pressure increases, the effective perfusion gradient falls and organ perfusion may decrease. In practice, this is the most common mechanism of "correct MAP with organ failure," especially in the kidney.

In the perioperative period, this decoupling is amplified by typical situations: general anesthesia (vasodilation), blood loss, hemodilution by fluids, use of vasopressors, positive pressure ventilation, pneumoperitoneum, and systemic inflammatory response. Therefore, MAP should be treated as a "safety threshold" and not as a "perfusion equivalent"(8) .

MICRODYNAMICS: THE TRUE DETERMINANT OF PERFUSION

Microdynamics parameters

Microdynamics refers to what occurs in arterioles, capillaries, and venules, where actual tissue delivery is defined. The most important conceptual parameters are:

Functional capillary density: how many capillaries are actually perfused.

Capillary erythrocyte flow: whether the flow is continuous and effective for transporting O_2.

Flow heterogeneity: uneven distribution produces "patchy hypoxia" even with sufficient global DO_2.

Extraction capacity: whether the tissue can use the delivered O_2; it is affected by edema, shunting, and mitochondrial dysfunction.

In the perioperative period, microdynamics are altered by inflammation, endogenous/exogenous catecholamines, thermal changes, anemia/hemodilution, hypocapnia/hypercapnia, and the surgical environment itself. The clinical value of this concept is that it explains why two patients with the same MAP/CO can evolve very differently(9) .

CLINICAL AND METABOLIC MARKERS OF MICROPERFUSION

Perfusion index (PI)

The perfusion index (PI) is derived from the plethysmographic signal and reflects changes in peripheral perfusion and vasomotor tone. It is attractive because it is continuous, noninvasive, and available on most monitors(10) .

How to use it effectively in the perioperative period:

Interpret it as a trend (rises/falls with intervention) rather than as a universal absolute threshold.

Contextualize it with temperature, analgesia, vasopressors, hypovolemia, and sympathetic stress.

Integrate it with MAP/CO: a low PI with acceptable MAP may suggest periph-

eral vasoconstriction or redistribution, not necessarily hypovolemia.

Critical limitation: high interindividual variability and sensitivity to peripheral conditions (cold, vasospasm, edema), so it should not be an isolated therapeutic goal.

Lactate

Lactate is a useful metabolic marker, but it is not synonymous with hypoxia. It can be elevated by hypoperfusion (anaerobiosis), but also by increased aerobic glycolysis (catecholamines), liver dysfunction, seizures, hyperthermia, or sepsis(7)effectively accomplished by vascular remodeling .

Clinical key in the perioperative period:

- Kinetic (trend and clearance) is more important than an isolated value.
- A decrease in lactate with clinical improvement usually supports overall recovery of perfusion/metabolism.
- Persistent lactate should not automatically trigger fluids or vasopressors without checking: CO, bleeding, oxygenation, anemia, congestion, and liver status.

• Capillary refill time (CRT)

CRT is a simple and powerful tool for approximating peripheral perfusion. Its usefulness lies in the fact that it responds quickly to hemodynamic changes and can reflect microvascular tone and redistribution(11) .

Practical points:

- It should be measured in a standardized manner (site and pressure time).
- It is most valuable as a serial monitor: if it improves after intervention, it suggests peripheral improvement.
- It is not specific: hypothermia, vasopressors, and pain can prolong it without central hypoperfusion.
- In a multimodal strategy, CRT helps to decide whether "the macro" is translating into "the micro."

Diuresis

Diuresis is a functional marker, not exclusively hemodynamic. In the perioperative period, oliguria may reflect renal hypoperfusion, but also neurohormonal response (ADH), surgical stress, pain, anesthesia, and, very importantly, venous congestion or elevated intra-abdominal pressure, which reduce the renal perfusion gradient(12) .

Mechanism	Macrodynamic effect	Microdynamic effect
Vasoplegia	Low/normal MAP with vasopressor	Microvascular shunt
Hemodilution	Normal CO	↓ O_2 transport
Vasopressors	MAP ↑	Capillary vasoconstriction
Inflammation	Variable CO	Flow heterogeneity

Table 1. Causes of macro-micro discordance in the perioperative period

Sublingual microcirculation

Sublingual microcirculation (videomicroscopy) allows direct evaluation of the microvascular bed: perfused capillary density, flow quality, and heterogeneity. In research, it has been fundamental in demonstrating that microcirculation can remain altered despite normalization of MAP/CO(11) .

POCUS AND VENOUS CONGESTION: BRIDGE BETWEEN MACRO AND ORGAN

Hemodynamic assessment with POCUS

POCUS allows hemodynamics to be integrated physiologically: left/right ventricular function, approximate intravascular volume, indirect signs of preload/afterload, and response to intervention. Its strength lies in transforming "assumptions" into findings(13) .

In the perioperative period, POCUS is especially useful when:

- there is hypotension refractory to target MAP,
- there is uncertainty between hypovolemia vs. vasoplegia vs. ventricular dysfunction,
- congestion is suspected (positive balance, positive pressure ventilation, elevated venous pressure).

VExUS and venous congestion

The central concept here is that the problem may not be "lack of pressure" but rather excess venous pressure. The VExUS approach integrates venous information to estimate systemic congestion and risk of organ damage (especially renal). In cardiac surgery and critically ill patients, venous congestion is associated with AKI and worse outcomes(14).

- VExUS operationalizes the concept of perfusion as a gradient: if the output pressure rises, the gradient falls.
- This allows us to argue that "avoiding over-resuscitation" is not only prudent, but also applied physiology: excess volume can reduce effective perfusion.

EFFECTIVE PERFUSION GRADIENT

The concept of effective perfusion gradient represents a necessary evolution from the classic approach focused on blood pressure. From a physiological perspective, tis-

Assessment	Finding	Hemodynamic implication
LV	Low output	Optimize flow
RV	Dilated	Congestion / ↑ PVC
VExUS	Altered venous flow	AKI risk
Lung	B lines	Overload

Table 2. Role of POCUS in assessing the perfusion gradient

sue perfusion does not depend on the absolute value of mean arterial pressure (MAP), but rather on the actual pressure gradient that drives flow through the organ, defined by the difference between arterial inlet pressure and venous or compartmental outlet pressure. When this gradient is reduced, perfusion decreases, even in the presence of apparently adequate MAP values(11) .

In simplified terms, organ perfusion can be expressed as:

Effective perfusion ≈ Inlet pressure – Outlet pressure

This principle, well established in physiology, takes on critical relevance in the perioperative period, where multiple interventions and clinical conditions can elevate outflow pressure without this being evident when monitoring MAP alone(15).

PERIOPERATIVE THERAPEUTIC IMPLICATIONS

Once the model is understood, therapy becomes more rational:

- Fluids: useful when they increase effective flow and recruit microperfusion; harmful when they induce congestion/edema and reduce gradient.
- Vasopressors: useful for restoring pressure-dependent perfusion, but must be titrated to avoid excessive vasoconstriction and considering peripheral microperfusion.
- Inotropics: relevant when the problem is flow (CO) rather than pressure, or when there is ventricular dysfunction with compromised perfusion.
- Avoid over-resuscitation: this is not a dogma; it is the physiological consequence of protecting the effective gradient and microdynamics.

CONCLUSIONS

Mean arterial pressure is necessary for perioperative hemodynamic safety, but it is not equivalent to tissue perfusion. Effective perfusion is defined at the microvascular level and depends on functional capillary flow, its adequate distribution, and the actual pressure gradient between arterial inflow and venous or compartmental outflow.

The macro-micro discordance explains why normalization of MAP and cardiac output can coexist with persistent tissue hypoperfusion. In the perioperative period, inflammation, vasoplegia, hemodilution, and the use of vasopressors alter microcirculation and limit the usefulness of uniform pressure targets.

The effective perfusion gradient integrates arterial pressure, venous congestion, and compartment pressures and is a key determinant of organ perfusion. Venous congestion and increased intra-abdominal pressure can reduce this gradient and precipitate organ dysfunction, even with apparently adequate MAP.

A multimodal and dynamic approach, combining macrodynamics, microsignals, bedside ultrasound, and perfusion gradient assessment, allows for the identification of hemodynamic phenotypes and guides more rational interventions. The future of perioperative hemodynamic management is personalized, oriented toward actual perfusion, and focused on physiological response rather than isolated numbers.

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Traditional approach	*Integrative approach*
MAP as a target	Perfusion as a target
Single pressure	Macro + micro + gradient
Reflective fluids	Phenotype-directed
Delayed reaction	Dynamic reassessment

Table 3. Comparison: traditional approach vs. integrative approach

Scenario	*MAP*	*Actual problem*	*Mechanism*
Postoperative oliguria	70 mmHg	AKI	Venous congestion
Persistent lactate	Normal	Microshunt	Inflammation
Peripheral coldness	Normal	Vasoconstriction	Vasopressors
Delirium	Normal	Cerebral hypoperfusion	Impaired autoregulation

Table 4. Clinical examples of hypoperfusion with "adequate" MAP

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