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Special Article

SPECIAL ARTICLES

Perioperative Quality Initiative consensus statement on the physiology of arterial blood pressure control in perioperative medicine

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Abstract

Background: Perioperative arterial blood pressure management is a physiologically complex challenge influenced by multiple factors.
Methods: A multidisciplinary, international working subgroup of the Third Perioperative Quality Initiative (POQI) consensus meeting reviewed the (patho)physiology and measurement of arterial pressure as applied to perioperative medicine. We addressed predefined questions by undertaking a modified Delphi analysis, in which primary clinical

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research and review articles were identified using MEDLINE. Strength of recommendations, where applicable, were graded by National Institute for Health and Care Excellence (NICE) guidelines.
Results: Multiple physiological factors contribute to the perioperative physiological importance of arterial pressure: (i) arterial pressure is the input pressure to organ blood flow, but is not the sole determinant of perfusion pressure; (ii) blood flow is often independent of changes in perfusion pressure because of autoregulatory changes in vascular resistance; (iii) microvascular dysfunction uncouples microvascular blood flow from arterial pressure (haemodynamic incoherence). From a practical clinical perspective, we identified that: (i) ambulatory measurement is the optimal method to establish baseline arterial pressure; (ii) automated and invasive arterial pressure measurements have inherent physiological and technical limitations; (iii) individualised arterial pressure targets may change over time, especially in the perioperative period. There remains a need for research in non-invasive, continuous arterial pressure measurements, macro- and micro-circulatory control, regional perfusion pressure measurement, and the development of sensitive, specific, and continuous measures of cellular function to evaluate blood pressure management in a physiologically coherent manner.
Conclusion: The multivariable, complex physiology contributing to dynamic changes in perioperative arterial pressure may be underappreciated clinically. The frequently unrecognised dissociation between arterial pressure, organ blood flow, and microvascular and cellular function requires further research to develop a more refined, contextualised clinical approach to this routine perioperative measurement.

Keywords: arterial pressure; blood pressure monitoring; haemodynamics; micro-circulation; perioperative care; perioperative medicine

Editor's key points

- Measurement of arterial pressure is fundamental to perioperative medicine, but has recognised limitations.
- An expert consensus meeting reviewed the physiology and measurement of arterial pressure in the perioperative period using a modified Delphi approach to create recommendations.
- Multiple interacting factors contribute to dynamic changes in perioperative arterial pressure that may be underappreciated clinically.
- Further research is needed to refine our understanding and management of arterial pressure in the perioperative period.

The measurement of arterial blood pressure is a fundamental tenet of modern perioperative practice, yet the limitations of using blood pressure to guide clinical management have long been recognised.[1] With the increasing complexity of clinical interventions and cardiorespiratory comorbidity, the interpretation of this measurement has become increasingly challenging and scrutinised. The development of novel monitoring technologies,[2] coupled with recent trials demonstrating the need for a reappraisal of chronic arterial hypertension management,[3] further demand a re-evaluation of applied bedside physiology to everyday perioperative practice. Here, we summarise the key aspects of arterial blood pressure physiology relevant to the perioperative period by focusing on applied physiological principles to guide the rational interpretation of this common, but frequently over-simplified, clinical measure. We also provide tractable clinical examples that highlight the need for the constant re-evaluation of perioperative blood pressure regulation.

Methods

The Perioperative Quality Initiative (POQI) is an international, multidisciplinary non-profit organisation that organises consensus conferences on clinical topics related to perioperative medicine. Each conference assembles a collaborative group of diverse international experts from multiple healthcare disciplines who are tasked with using a modified Delphi technique to develop consensus-based recommendations in perioperative medicine.

The POQI-3 consensus conference on perioperative blood pressure management took place in London, UK, from July 1 to 3, 2017. The objective of POQI-3 was to produce consensus statements and practice recommendations pertaining to the definition and management of perioperative arterial blood pressure, and to identify research priorities. The participants in the POQI consensus meeting were recruited based on their expertise in perioperative medicine and blood pressure management (Supplementary material, Appendix 1). Conference participants were divided into four work groups: Group 1 reviewed the physiology and measurement of blood pressure with relevance to the perioperative setting (this paper), whereas Groups 2, 3, and 4 were focused on preoperative,[4] intraoperative,[5] and postoperative[6] blood pressure, respectively.

The POQI process is based on an established modified Delphi process used in the Acute Dialysis Quality Initiative (ADQI) conferences[7] that includes the following iterative steps before (steps 1 and 2) and during (step 3) the conference: (1) building consensus around the most important questions related to the topic, (2) a literature review of the topic raised by each question, and (3) sequential steps of content development and refinement until agreement is achieved and a consensus document is produced. This final step of content development and refinement involves a modified Delphi process of alternating breakout and plenary sessions. In the breakout sessions, work groups addressed the issues in their assigned topic area and formulated consensus statements and practice recommendations. In the plenary sessions, the findings and deliberations of each work group are presented, debated, and refined. Consensus on some statements and recommendations may be achieved in the first plenary session. Other statements and recommendation required further refinement by the work groups before re-presentation to the plenary group in the next cycle. At the end of the conference, plenary group members vote to signal either formal agreement with the final statements/recommendations, or signal their

disagreement. In the latter case, a statement of disagreement would be included in the manuscript. All recommendations were unanimously approved, unless stated otherwise.

This workgroup of the POQI-3 consensus meeting reviewed the (patho)physiology and measurement of arterial blood pressure as applied to perioperative medicine. Before the meeting, a literature search was conducted in Medline based on predefined questions (Supplementary material, Appendix 2).

Results

Consensus statements

Consensus statement 1: *Different measures and values of arterial blood pressure reflect multivariable, complex physiology that are not interchangeable.*

Blood pressure is a composite measurement comprising several values with differing physiological roles and origins

Arterial blood pressure is determined by the interaction between left ventricular cardiac contraction, the hydraulic load of the arterial system, and extravascular, intra-thoracic, and intra-abdominal mechanical forces.[8]

Common measures of arterial blood pressure include systolic, diastolic, mean arterial, and pulse pressure

These different measures are not constant and reflect fundamentally different components shaping the physiology of blood pressure regulation (Fig 1).[9] Systolic pressure is the maximal aortic pressure achieved after the left ventricle has ejected blood into the aorta. During left ventricular relaxation and refilling, aortic pressure declines to a nadir, termed the diastolic blood pressure. Pulse pressure represents the difference between systolic and diastolic pressures, representing the interaction between stroke volume and arterial tone.[10]

Systolic pressure is determined by the pattern and duration of left ventricular ejection (stroke volume), the compliance (distensibility) of the arterial vessels, the velocity of the pressure wave in large arteries and vasomotor tone in peripheral arteries, which regulates the magnitude of reflection of pressure waves.[8] Increased transmission velocity of both the forward and reflected pressure waves leads to arrival of the reflected wave in the central aorta during systole, augmenting systolic pressure.[11] Hypertension, the prevalence of which increases with age, has a multifactorial aetiology including loss of elasticity in central arteries.[12] Hence, arterial stiffening augments systolic and pulse pressures. In essence, this means that central aortic pressure serves as a surrogate of ventricular wall tension, and is the most accurate measure of afterload.

Systolic pressure increases progressively towards the peripheral arterial tree through augmentation of the arterial pulse wave. As systolic pressure increases, diastolic pressure decreases slightly, because of branching vessels reflecting pressure waves in combination with the decreased arterial compliance of the distributing arteries.[9] As a result of increased resistance and reduced compliance in smaller arteries and arterioles, the amplitude of pulsation decreases until becoming minimal in the capillaries.[13] The capacitive ('reservoir') function is determined by the compliance of the aorta and large elastic arteries and largely determines the morphology of the pulse waveform.[14] The arterial reservoir declines with age as compliance decreases, leading to changes in the aortic pressure waveform.

Diastolic pressure depends on arterial compliance (stiffness), heart rate, and the resistance and distribution of the vascular network, which is arranged in series and parallel. As humans age, diastolic pressure increases until ~50 yr of age and then typically declines thereafter. Laminar and turbulent flow characteristics, as well as blood viscosity, also influence systolic and diastolic pressures. Taken together, changes in vascular tone and viscosity associated with pathology affect both the amplitude and timing of the reflected waves, meaning that measured peripheral pressures rarely equate to central arterial pressure. Therefore, when tone, viscosity, or contractility varies rapidly, organ input pressure is unlikely to reflect central pressure. This, in part, explains the inconsistent and variable threshold values of blood pressure associated with pathophysiology.

The clinical importance of calculating MAP

MAP is the average pressure value during the arterial pulse pressure cycle. As there is relatively low resistance in the arterial tree down to the smaller arteries, MAP declines by only a small degree as the aortic pressure pulse travels away from the aorta and to the distributing arteries. By analogy to Ohm's law, MAP is a function of cardiac output and arterial resistance with

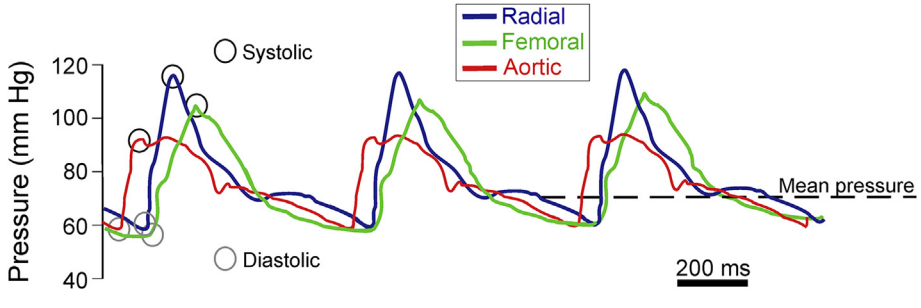

Figure 1. Variation in arterial pressure by site of measurement. Simultaneously measured arterial pressure waveforms from the radial artery, femoral artery and ascending aorta. Although diastolic and mean arterial pressures are similar regardless of the site at which arterial pressure is measured, systolic pressure increases as the monitoring site is further away from the ascending aorta. Figure reused with the permission of the Perioperative Quality Initiative (POQI).

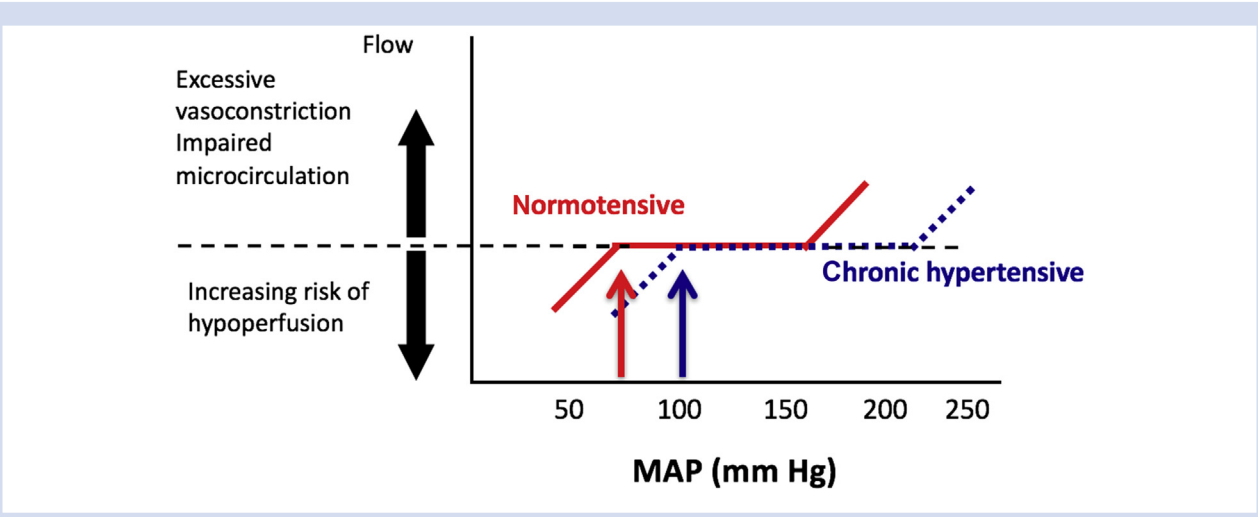

Figure 2. Autoregulation of blood flow. Schematic diagram showing altered relationship between flow and arterial pressure in normotensive and hypertensive individuals. Autoregulation allows optimal blood flow to be maintained for a wide range of pressure values. Autoregulation is present also in hypertensive individuals, but the lower pressure limit is higher and the curve is shifted to the right compared with healthy subjects. Blood flow outside the autoregulation areas leads to either excessive vasoconstriction and impaired microcirculation, or to low blood flow and hypoperfusion. Figure reused with the permission of the Perioperative Quality Initiative (POQI).

the arterial resistance determined primarily in the downstream small arteries and arterioles. This also means that MAP can be used as a reference value along the entire central arterial system to estimate organ input pressure. However, MAP does not accurately reflect left ventricular afterload because the hydraulic impedance encountered by the left ventricle comprises static (total peripheral resistance) and pulsatile elements more accurately quantified by the combination of hydraulic pressure, arterial elastance, and compliance.

Arterial blood pressure may be dissociated from intact autoregulation mechanisms

Across different organs, maintenance of tissue perfusion requires autoregulatory mechanisms that counteract extreme variations in arterial pressure (Fig 2).[15,16] Autoregulation in the cerebral circulation, for example, involves myogenic, neurogenic, and metabolic feedback mechanisms to optimise brain perfusion.[17] However, interactions between these three regulatory mechanisms are poorly understood, particularly under anaesthesia, in the presence of systemic inflammation, or both. Experimental data suggest that neurogenic (sympathetic autonomic) control is a key player in rapid cerebral autoregulatory adjustments during acute changes in arterial pressure.[18] In hypertension, autoregulation is impaired, at least in part because of impaired neurovascular responses to carbon dioxide compared with normotensive subjects.[19] Impairment of cerebral autoregulation in hypertension may extend beyond a rightward shift, with a marked narrowing, or complete loss, of the plateau range of pressure over which constant flow is ensured.[20]

Circadian and neural/hormonal changes influence blood pressure over time

Circadian rhythms regulate cardiovascular physiology through alterations in metabolism, feeding, sleep, and wakefulness, coupled with coordinated neurohormonal secretion.[21] The master circadian clock situated in the suprachiasmatic nuclei of the hypothalamus is synchronised to the external environment primarily by signals from the visual system, providing information about light–dark cycles. Interoceptive stressors similarly shape circadian signalling. Beyond the brain, peripheral circadian clocks also regulate circadian oscillations.[21]

Arterial pressure is substantially lower during sleep in healthy individuals.[22] Loss of central diurnal rhythms, peripheral diurnal rhythms, or both, that alter activity, metabolism, and hence neurohormonal release, profoundly influence blood pressure and other cardiovascular functions that contribute to the development of cardiometabolic disease.[23] Many (hypertensive) individuals fail to show such marked declines in blood pressure at night,[24] a feature associated with end-organ damage and a higher incidence of cardiovascular complications.[25] Acute inflammation and anaesthetic drugs are additional potent triggers for disrupting normal circadian regulation of arterial pressure.[26]

Essential hypertension is a complex, multi-organ disease

The complexity of blood pressure regulation is amplified in an estimated 25% of adults with essential hypertension.[27] The uncoupling of mechanisms regulating blood volume, ventricular function, central and peripheral autonomic control, neurohormonal activation via the renin–angiotensin–aldosterone system, and endothelial release of nitric oxide disrupts blood pressure, blood pressure variability, or both. The role of salt sensitivity in hypertensive individuals highlights the potential impact of increased sodium administration, which is a crucial regulator of blood volume.[28] Chronic systemic inflammation driven by perturbations in innate and adaptive immune cells acting at both vascular and non-vascular substrates further contribute to the multifaceted pathophysiology of hypertension.[29] There are limited clinical data on how perioperative interventions are affected by, or impact on, various pathophysiological drivers of hypertension.

Perioperative implications of consensus statement 1

The contribution of arterial pressure measurement to clinical management is dependent on the context within which that measurement occurs. A single measurement divorced from both acute and chronic clinical contexts is highly unlikely to provide clinically useful information.

Consensus statement 2: *Arterial blood pressure is necessary to ensure adequate blood flow to meet cellular metabolic demands.*

Adequate blood flow that meets the metabolic demands of tissues is usually reflected by pulse pressure (reflecting stroke volume) and MAP (reflecting cardiac output) remaining within a population-defined normal range. Recent trials suggest that end-organ damage in chronic hypertension occurs at lower than previously accepted arterial pressure thresholds,[3,30] and that preoperative elevated pulse pressure is associated with perioperative myocardial injury.[31] Moreover, within organs, significant heterogeneity in intra-organ blood flow occurs as a result of intrinsic variability in local microvascular resistance that is likely to be chiefly determined by regional and local metabolic requirements. Micro-circulatory perfusion is frequently perturbed by acute hypotension for prolonged periods, even after brief episodes, resulting in metabolically compromised, dysoxic, or hypoxic tissues.[32] Despite evidence for cellular dysfunction after tissue hypoperfusion in vulnerable tissues, such as the gastric mucosa during controlled haemorrhage in healthy conscious volunteers, arterial pressure may remain within its normal range during significant hypovolaemia.[32] Thus, the physiological response to hypovolaemia maintains arterial pressure, which is dissociated from cardiac output for a variable length of time.[33] Attempts to reverse hypotension may therefore not be effective in restoring micro-circulatory perfusion.[34] Loss of haemodynamic coherence between the macro-circulation and the micro-circulation occurs when either spontaneously, or through clinical intervention, systemic arterial pressure is restored yet deficiencies in micro-circulatory perfusion and oxygen delivery persist.[35] Macro- and micro-circulatory incoherence is likely to promote therapeutic measures targeted towards macrovascular variables that potentially cause harm, such as the inappropriate administration of fluids, vasopressor drugs, or both. This may explain why correcting macrovascular haemodynamic variables to normalise, or supranormalise, systemic oxygen delivery may be ineffective once systemic inflammation is established.[36,37] Thus, even though macrovascular (systemic arterial pressure) parameters may appear to be adequate in both acute and chronic pathological states, this does not necessarily reflect intra-organ microvascular blood flow. In other words, adequate arterial pressure is necessary to ensure adequate blood flow to meet cellular metabolic demands, but is not sufficient to guarantee such flow.

Perioperative implications of consensus statement 2

Arterial blood pressure measurement alone cannot ensure that adequate blood flow meets cellular metabolic demands. Therefore, confirmatory measures are required to establish whether a particular clinical arterial pressure target is adequate. Confirmatory measures may require simple measures (e.g. central–peripheral temperature gradient), additional sophisticated measures, or both (e.g. lactate, mixed venous oxygen saturation).

Consensus statement 3: *Blood flow is often independent of changes in perfusion pressure as a consequence of autoregulatory changes in vascular resistance.*

Within a broad range of organ-specific perfusion pressures, autoregulatory mechanisms ensure that flow is preserved (Fig 2). Pharmacological (e.g. anaesthetic agents) and pathological (e.g. sepsis) perturbation of autoregulatory control renders organ blood flow pressure dependent.[38] These observations partly explain why perioperative complications are frequently observed in organs (kidney, heart, brain) that require highly autoregulated, yet individualised, control of arterial pressure. Extremes of arterial pressure (hypotension, hypertension) are associated with perioperative injury in these organs.[39]

Perioperative implications of consensus statement 3

An arterial blood pressure reading deemed 'normal' for any individual may lead to the erroneous conclusion that this accurately reflects normovolaemia, adequate cardiac output, or both. For example, progressive haemorrhage in surgical patients often fails to manifest as a decline in arterial pressure when compensatory mechanisms are intact. Moreover, in the presence of concomitant pain or exogenous catecholamine infusion, arterial pressure measurements may mask an injurious decline in organ perfusion.

Consensus statement 4: *Arterial blood pressure is the input pressure to organ blood flow, but is not the sole determinant of perfusion pressure.*

Total systemic vascular resistance has long been defined by electrical circuit theory, which assumes that a constant pressure decrease from input to output sites exists. However, laboratory and human studies demonstrate that two separate pressure gradients are likely to exist, enabled by the presence of Starling resistors residing within arteriolar or precapillary loci. The arterial gradient is generated from the central arterial circuit to the critical closing pressure, whereas a venous pressure gradient exists between mean systemic pressure and central venous pressure (Fig 3). The input pressure to an organ is determined by the difference between the central arterial pressure and organ-specific arterial critical closing pressure, the pressure threshold that coincides with cessation of blood flow at an inflow pressure higher than outflow venous pressure.[40] Perfusion pressure to an organ is determined by the input pressure minus the outflow pressure, which in turn is determined by the surrounding organ pressure (e.g. interstitial pressure) and right atrial pressure. Input pressure thresholds and outflow pressure vary significantly between organs, highlighting the importance of arterial and venous resistances which determine a 'vascular waterfall' that ensures organ perfusion even in low-flow conditions.[41] The presence of two separate, but in-series, vascular resistances ensures that a pressure gradient within an organ (i.e. critical closing pressure > mean systemic pressure) is maintained for a finite length of time even during profound hypotension (when MAP decreases to the critical closing pressure). These data suggest that common perioperative scenarios such as hypotension—characterised by a short-lasting dissociation between MAP and cardiac output—are unlikely to be rationally addressed by conventional clinical intervention(s) (Fig 3).

Perioperative implications of consensus statement 4

Raised local intra-abdominal organ pressure (e.g. insufflation during laparoscopy, intra-abdominal organ oedema) may

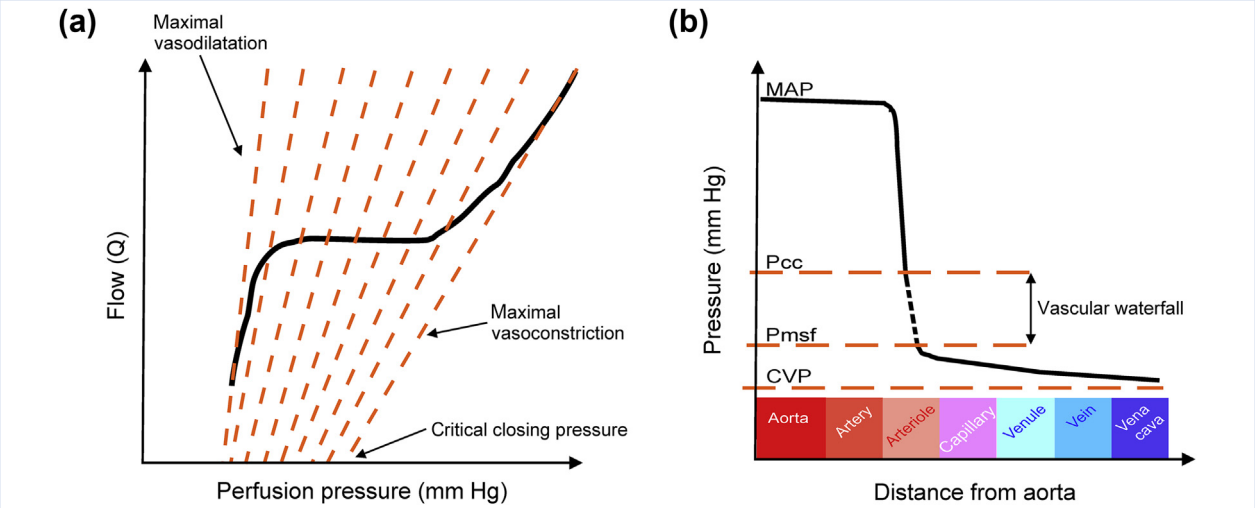

Figure 3. Arterial input pressure and flow. (a) Theoretical relationship between pressure and flow (black line) showing the autoregulation of vascular tone to sustain a constant blood flow despite varying arterial input pressures. The orange dashed lines illustrate how changes in vascular tone alter the relationship between instantaneous arterial input pressure and blood flow subject to autoregulation. The point at which arterioles spontaneously collapse (zero blood flow) limiting arterial pressure decrease is referred to as the critical closing pressure (Pcc), which also varies with changes in vasomotor tone. (b) Theoretical vascular pressure profile throughout the circulatory tree. MAP is constant for most of the arterial tree because larger arteries serve mainly as vascular capacitors holding stored blood under pressure. By contrast, vascular pressure decreases rapidly once blood reaches smaller arteries that branch into arterioles and precapillary sphincters. The vascular waterfall is approximated by the critical closing pressure (Pcc) mirroring how water flowing over a waterfall is unaffected by how far it falls once over the edge. Thus, the decline in pressure from arterioles to venules, or changes in downstream venous pressure, does not influence either arterial pressure or blood flow. Mean systemic filling pressure (Pmsf) represents the upstream pressure driving venous return against downstream central venous pressure (CVP). Figure reused with the permission of the Perioperative Quality Initiative (POQI).

result in inappropriate systemic arterial pressure targets aimed at maintaining regional organ perfusion.

Consensus statement 5: *Measurement of arterial blood pressure has inherent limitations because of inaccurate values, interpretation, or both.*

On physiological and sampling frequency grounds alone,[42] gold-standard measurement of arterial pressure necessitates an intra-arterial catheter,[43] taking into account several well-established factors including the site of catheter placement, waveform damping, and catheter dimensions. Manual mercury sphygmomanometry remains the gold standard to assess the accuracy of automated oscillometric devices, the most widely used technique in the perioperative setting to measure arterial blood pressure. Manually measured blood pressures often differ from those obtained using automated devices, and this adversely influences correct blood pressure classification. Although easy to use, two key inaccuracies are likely to contribute to the variability in measurement of cuff pressure.[44] First, fixed deflation rates (typically 2 mm Hg s^{-1}) set a limit of resolution that is dependent on incident heart rate. Second, measurement of the maximal rate of pressure increase during arterial pressure oscillation throughout the cardiac cycle is imprecise. The pressure level at which the rate of increase is maximal defines MAP; a proprietary algorithm uses this value to estimate systolic and diastolic blood pressure.[45] Absolute arterial pressure, differences in arterial pressure between left and right arms, variability between different devices/manufacturers, cuff size, posture, environment, and ambient temperature may all adversely affect accuracy.[45] Failing to take these limitations into account may lead to iatrogenic harm when single or intermittent measurements shape clinical decisions.

Perioperative implications of consensus statement 5

The site and mode of arterial blood pressure management yield different values. Accordingly, clinical management (including blood transfusion and vasopressor use) may differ depending on the method used, as suggested by observational database studies.[46]

Consensus statement 6: *Ambulatory arterial blood pressure measurement is the optimal method to establish baseline values.*

Guidelines from multiple international bodies recommend that adults with elevated arterial pressure in a clinical setting should undergo ambulatory measurements to exclude white coat hypertension before diagnosis.[47–50] Ambulatory blood pressure measurements are a stronger predictor of all-cause and cardiovascular mortality than one-off blood pressure measurements made in clinics. However, masked hypertension[51] is associated with higher mortality than overt hypertension.[52] Ambulatory blood pressure measurement also reduces the risk of misdiagnosing hypertension, which occurs in up to 18% of the general population when clinic or home-based measurements are made.[53] Inappropriate treatment of apparent white coat hypertension after a clinic-based measurement has been associated with adverse outcomes, chiefly through hypotension.[54,55] A further ~15–30% patients exhibit

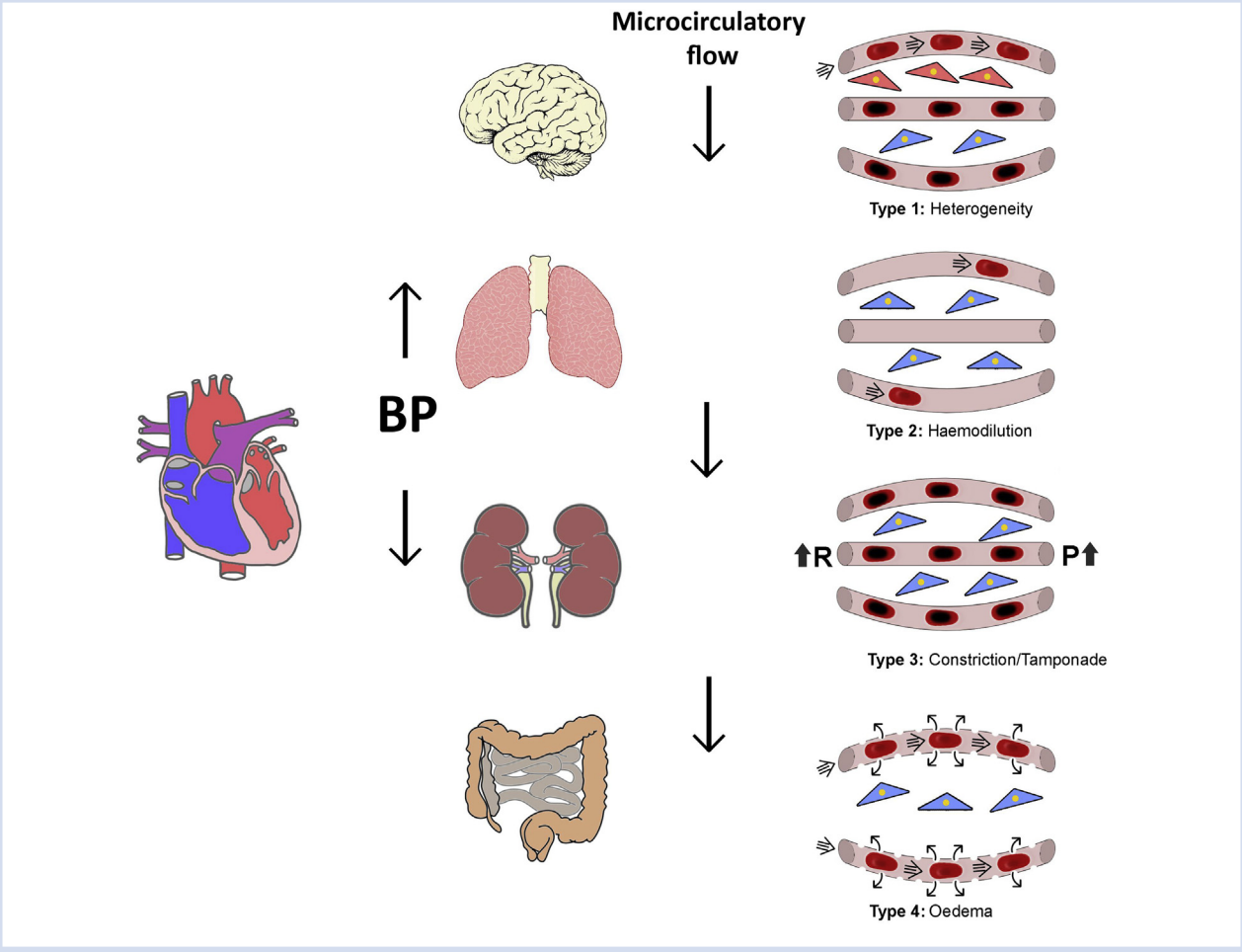

Figure 4. Microcirculation and arterial pressure. Microcirculatory conditions where control of arterial blood pressure under conditions of hypotension or hypertension impairing tissue perfusion are not effective in improving microcirculatory perfusion. Such conditions occur when there is a loss of haemodynamic coherence characterised by normalised systemic haemodynamic variables but persistent microcirculatory dysfunction leading to a lack of oxygen availability in tissue (as indicated by blue cells). Four distinct aetiologies of haemodynamic incoherence leading to microcirculatory shock include. Type 1: heterogeneous perfusion of the microcirculation as seen in septic patients with obstructed capillaries next to perfused capillaries resulting in heterogeneous oxygenation of tissue cells. Type 2: haemodilution of microcirculatory blood resulting in the loss of erythrocyte-filled capillaries and increasing diffusion distance between erythrocytes in capillaries and tissue cells. Type 3: stasis of microcirculatory erythrocyte flow induced by altered systemic variables (e.g. increased arterial vascular resistance (R) and or increased venous pressures caused by tamponade). Type 4: alterations involve oedema caused by capillary leak syndrome which results in increased diffusion distances from erythrocytes to tissues and reduced ability of the oxygen to reach tissue cells. Figure reused with the permission of the Perioperative Quality Initiative (POQI).

masked hypertension, where clinic measurements are normal but breach hypertension thresholds outside the clinical setting.[56] The Ambulatory Blood Pressure Collaboration in Patients With Hypertension meta-analysis found that both a blunted nocturnal decline (dipping) in arterial pressure and more extreme dipping in untreated hypertensives were associated with excess cardiovascular morbidity and mortality, independent of ambulatory blood pressure measurements averaged over 24 h.

Perioperative implications of consensus statement 6

Intraoperative arterial pressure management is frequently based on a very limited number of preoperative readings that are unlikely to be a true representation of an individual's long-term blood pressure control. The hypertension literature implies that a non-representative preoperative, one-off, clinic-based value is likely to be misleading in >30% of patients.

Consensus statement 7: *Arterial pressure targets may change over time for any individual patient.*

With advancing age, a U-shaped association develops between systolic arterial pressure and all-cause mortality.[57] These observational data challenge the findings of the Systolic Blood Pressure Intervention Trial (SPRINT), reinforcing the view that lower targets may require a more personalised approach.[58] Lower systolic pressure appears to be associated with mortality linked to non-cardiovascular causes,[57] which may reflect subclinical cardiac failure/deconditioning.[59,60] In the acute setting, the perioperative period is characterised by

heterogenous metabolic demands across disease states and different organs.[61] Fixed arterial pressure targets may lead to unintended adverse effects of interventions, as they are likely to be incompatible with maintaining a state of haemodynamic coherence, where macro- and micro-circulatory flow are matched.[35] Additionally, the arterial baroreflex plays an important role in long-term control of arterial pressure.[62] Impaired responses through loss of baroreflex sensitivity, a key autonomic regulatory mechanism, are associated with poorer perioperative outcomes[63] and linked mechanistically with organ injury.[64,65] The loss of haemodynamic coherence may occur in a highly heterogeneous, organ-specific pattern (Fig 4).

Perioperative implications of consensus statement 7

Dynamic perioperative alterations in arterial pressure regulation require repeated evaluation of clinical targets. Therefore, arterial pressure management requires repeated, contextualised assessment of systemic targets in conjunction with other clinical haemodynamic parameters (e.g. cardiac output monitoring) and markers of organ perfusion, including metabolites (e.g. lactate), enzyme function (hepatic transferases), and biomarkers for injury (e.g. troponin, B-type natriuretic peptide).

Recommendations for research

From the consensus points developed above, we recommend that further research relevant to the perioperative period should include:

1. Methods for non-invasive, continuous arterial pressure measurements.
2. Impact of perioperative arterial pressure therapies on autoregulatory, micro-circulatory, and autonomic control.
3. Methods to evaluate regional perfusion pressures to enable the assessment of individualised organ responses to alterations in arterial pressure control.
4. Identify sensitive, specific and continuous measures of cellular function that enable a more refined evaluation of arterial pressure management.

Strengths and limitations

POQI uses an established modified Delphi process which has been used in more than 25 ADQI and POQI conferences in the past 20 yr. The combination of a literature review with expert opinion aims to produce a practical consensus statement focusing on areas of clinical uncertainty. This methodology does not incorporate a formal systematic review or meta-analysis. We acknowledge that by primarily focusing on perioperative issues, many complex areas of blood pressure (patho)physiology have been considered briefly. However, as this process is based partly on expert opinion, there remains some risk of bias. Areas of uncertainty have been clearly signposted in the discussions accompanying each statement.

Conclusions

Changes in the optimal management of perioperative arterial pressure are very likely given the shifting clinical landscape in diagnosis and management of chronic changes in arterial pressure. However, there is a current lack of evidence linking the latest international guidelines on chronic management of arterial pressure with targets for perioperative practice. Inevitably, this has major implications for perioperative medicine, and reinforces the need to refine our understanding and management of this complex physiological measure in the perioperative period.

Authors' contributions

Drafting of the first version of the manuscript: GLA, CSB, MC, CI, MGI, JL, MRP.
Review and editing of revised manuscript: GLA, CSB, MC, CI, MGI, JL, MRP, MPWG, MGM, MRE, TEM.
Participation in the conference: GLA, CSB, MC, CI, MGI, JL, MRP, MPWG, MGM, MRE, TEM.
Submission of the manuscript: MPWG, MGM, MRE, TEM.
Chair of the physiology group: GLA.
Member of the physiology group: CSB, MC, CI, MGI, JL, MRP.
POQI conference organiser: MPWG, MGM, MRE, TEM.

Declarations of interest

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Appendix A. Supplementary data

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