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Perioperative Quality Initiative consensus statement on goal-directed haemodynamic therapy

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Summary

Perioperative goal-directed haemodynamic therapy (GDHT) includes a variety of protocolised approaches to the assessment and management of the circulatory system and blood flow for patients undergoing surgery. Here we present updated consensus statements on perioperative GDHT developed during the 11th Perioperative Quality Initiative (POQI) consensus conference meeting held in London, UK in June, 2023. Statements relating to the definitions, components, and underlying physiology surrounding GDHT are proposed. We recommend considering use of GDHT in specific settings including during cardiopulmonary bypass (CPB), after cardiac surgery, and during hip fracture surgery. However, the level of evidence is weak in these settings. Clinicians can consider use of GDHT protocols on an individual patient basis for moderate- to high-risk patients undergoing major noncardiac surgery; however, we recommend against use of fixed low-dose inotrope infusions as part of GDHT protocols. We do not recommend routine use of GDHT protocols for patients undergoing major elective abdominal surgery. There is currently insufficient evidence to recommend routine use of GDHT during emergency abdominal surgery. Future research should focus on individualisation of GDHT to individual patients' haemodynamic requirements, newer paradigms such as technology-assisted delivery of GDHT protocols, and the role of predictive models using artificial intelligence.

Keywords: cardiac output; fluid therapy; goal-directed haemodynamic therapy; inotropes; perioperative blood pressure

Editor's key points

- The Perioperative Quality Initiative is an international multidisciplinary organisation that organises consensus conferences on clinical topics related to perioperative medicine.
- The 11th Perioperative Quality Initiative (POQI) consensus conference met in 2023 to update best practices in perioperative medicine including goal-directed haemodynamic management, perioperative blood pressure, and fluid therapy in perioperative medicine.

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- This consensus statement reports 11 updated consensus statements on perioperative goal-directed haemodynamic therapy (GDHT).
- GDHT can be beneficial in specific settings including during cardiopulmonary bypass, after cardiac surgery, and during hip fracture surgery.
- Future research should focus on individualisation of GDHT to individual patients' haemodynamic requirements and technology-assisted delivery of GDHT protocols including predictive models and artificial intelligence.

The assessment and management of patients' haemodynamic status throughout the physiological and pathophysiological stress response to surgery is a core aspect of high-quality perioperative care. Interventions include administration of i.v. fluids and inotropic or vasoactive medications to improve perfusion and oxygen delivery to tissues, thereby reducing complications and improving clinical outcomes. Haemodynamic status can be assessed using a range of simple physiological measurements including arterial blood pressure, heart rate, and fluid balance to more advanced assessments such as estimates of cardiac stroke volume, cardiac output, and systemic vascular resistance. Certain relative or absolute values of these measured variables can be used as intervention targets within perioperative algorithms. Achieving optimised haemodynamic status has been proposed to improve postoperative patient outcomes, but despite clinical and physiological plausibility, little definitive clinical effectiveness evidence exists regarding these goal-directed interventions. Suggested mechanisms of benefit relate to the adequacy of blood flow supplying oxygen and nutrients to organs and tissues to maintain their function while aiming to avoid iatrogenic harm.

Given the importance of such goal-directed haemodynamic therapy (GDHT) approaches and the evaluation of their clinical effectiveness, the 11th meeting of the Perioperative Quality Initiative (POQI-11) was convened in 2023, which included a subgroup tasked with updating and building on previous POQI guidance on GDHT for perioperative clinicians.[1–3] Here we present our recommendations with relevance to current clinical practice, and propose future research questions in this area.

Methods

The Perioperative Quality Initiative (POQI) is an international multidisciplinary nonprofit organisation that organises consensus conferences on clinical topics related to perioperative medicine. Each POQI conference assembles a collaborative group of diverse national or international experts from multiple healthcare disciplines to develop consensus-based recommendations in perioperative medicine. The group members were reimbursed for travel, accommodation, and meals but did not receive honoraria. The POQI methodology combines elements of both evidence appraisal and expert opinion, while acknowledging the limitations of available literature to provide practical recommendations.

The POQI-11 was convened on July 4 and 5, 2023, in London, UK, to update previously published consensus statements on best practices in perioperative medicine by three work groups covering goal-directed haemodynamic management, perioperative blood pressure, and fluid therapy in perioperative medicine.[1–4] The outputs of the POQI-11 work groups covering perioperative blood pressure and fluid therapy are published separately.[5,6]

A modified Delphi method was used, designed to garner the collective knowledge of the diverse group of experts to answer clinically important questions around perioperative blood pressure, fluids, and haemodynamic therapy. The members of the whole POQI-11 group (Appendix 1) were recruited based on their expertise in perioperative management of patients undergoing surgery, and members of the GDHT work group (the authors of this manuscript) had particular expertise in perioperative fluid and haemodynamic monitoring and therapy (Supplementary Fig. 1). Before the conference, topics for discussion at the consensus meeting were longlisted, and the three work groups of several members each were assembled to systematically review and create a bibliography of relevant literature on their topic. This list was used to identify important questions to be addressed in the conference.

For publications to be included in this paper, Ovid MEDLINE was searched from 1990 to June 2023 using the following search terms: '((controlled clinical trial).ti,ab,pt OR (randomized).ti,ab OR (randomised).ti,ab OR (randomly).ti,ab OR (trial).ti) AND ((haemodynamic).ti,ab OR (haemodynamics).ti,ab OR (hemodynamics).ti,ab OR (hemodynamic).ti,ab OR (fluid).ti,ab) AND ((cardiac output).ti,ab OR (cardiac index).ti,ab OR (oxygen delivery).ti,ab OR (oxygen consumption).ti,ab OR (stroke volume).ti,ab OR (optimization).ti,ab OR (optimisation).ti,ab OR (Goal-directed).ti,ab OR (Goal-orientated).ti,ab OR (Algorithm).ti,ab OR (guided).ti,ab OR (goal directed).ti,ab OR (goal orientated).ti,ab OR (oxygenation).ti,ab OR (individualised).ti,ab OR (individualized).ti,ab) AND ((surgery).ti,ab OR (perioperative).ti,ab OR (intraoperative).ti,ab)'. The references of relevant articles were reviewed and further articles retrieved if deemed relevant.

At the first plenary session of the conference, work groups from the blood pressure, fluids, and GDHT research groups presented draft consensus statements and the evidence base on which these had been constructed to the whole POQI-11 group. The full POQI-11 group then split into the work groups for discussion. In subsequent plenary sessions, each work group summarised the breakout discussions and any modifications to the consensus statements to the assembled whole POQI-11 group. Feedback and assistance was received, facilitated by four meeting chairs, to refine the consensus statements. During a total of three rounds of work group discussion and plenary presentation and feedback, the statements were further refined before nonanonymous voting took place to determine whether unanimous consensus could be achieved on each statement presented. During the final plenary session, POQI-11 group members voted to signal either formal agreement with the final statements, or signal their disagreement. In the latter case, a statement of disagreement would be included in the report. All statements were unanimously approved, unless stated otherwise. As a large trial of GDHT (OPTIMISE II)[7] had recently been completed but was not published at the time of the POQI meeting, a small number of statements were constructed as drafts without knowledge of the trial results by the GDHT work group, with alternatives based on the possible trial outcomes. These statements were then ratified by the whole POQI-11 group by online discussion after the meeting when the trial results were released.

The Grading of Recommendations Assessment, Development, and Evaluation (GRADE) system was used to rate the

certainty of evidence underpinning recommendations (high, moderate, low, or very low).[8] Where evidence was lacking and consensus could not be reached, recommendations for future research were generated. After the meeting, statements were presented at the Evidence Based Perioperative Medicine (EBPOM) 2023 World Congress in London on July 6, 2023, where attendees were invited to participate in an anonymous vote to indicate their agreement/disagreement for each recommendation using Slido (Slido, Bratislava, Slovakia; https://www.slido.com). After a brief explanatory presentation, each statement was presented in turn by the three work group chairs. There was no presentation of the evidence or rationale that had led to each statement. Attendees voted either 'agree' or 'disagree' using the Slido app or website, or could abstain from voting on a statement-by-statement basis. Voting for each statement was closed when there were no further votes accumulating. Attendees were only able to see the results of voting after voting for each statement was closed. To preserve anonymity, we did not document the institutional affiliation or professional category of these respondents. The composition of the whole POQI-11 group and of the work groups, and the POQI-11 work flow is shown in Supplementary Figure 1.

In this report, we summarise the consensus statements on GDHT and present the results of the anonymous votes of 91 attendees of the EBPOM 2023 World Congress on July 6, 2023 (Table 1).

Summary of consensus statements and supporting evidence

See Table 1.

QUESTION 1. What is meant by 'goal-directed haemodynamic therapy' (GDHT)?

Table 1 Perioperative Quality Initiative 11 (POQI-11) goal-directed haemodynamic therapy (GHDT) consensus statements and recommendations. N/A, not applicable; not presented for voting at the Evidence Based Perioperative Medicine (EBPOM) conference because the statement does not include a recommendation, or * because the statement was not presented pending the results of the OPTIMISE II clinical trial.

		Strength	Level of evidence	Agreement by EBPOM delegates % (no. of votes)
Statement 1	GDHT is an umbrella term for a complex intervention using monitoring techniques and physiological targets to help guide administration of fluids, vasopressors, and inotropes	N/A	N/A	N/A
Statement 2	The goals of haemodynamic management are to optimise tissue oxygenation and support normal cellular metabolic function	N/A	N/A	N/A
Statement 3	We recommend that GDHT protocols should have clearly defined component parts and physiological targets	Strong	Very low	99 (83)
Statement 4	The GDHT evidence base is complex as it comprises a variety of protocols, interventions, monitoring technologies, surgical procedures, patient factors, and outcomes	N/A	N/A	N/A
Statement 5	Fluid responsiveness is a key component of GDHT and is best defined as an increase in stroke volume in response to intravascular fluid administration	N/A	N/A	N/A
Statement 6	Vasopressors and inotropes are additional GDHT components that can be titrated to achieve haemodynamic goals	N/A	N/A	N/A
Statement 7	Dynamic variables (such as pulse pressure variation and stroke volume variation) can be used to assess fluid responsiveness but have limitations	Strong	Moderate	N/A
Statement 8	We recommend that an increase (>10–15%) in stroke volume in response to a fluid bolus should be used to identify fluid responsiveness	Strong	High	92 (86)
Statement 9	We recommend that GDHT protocols aim to optimise stroke volume or cardiac output and mean arterial blood pressure with fluids, vasopressors, and inotropes	Strong	Moderate	94 (87)
Statement 10	Clinical trials of GDHT have been conducted in a range of surgical situations using a variety of different protocols with mixed results	N/A	N/A	N/A
Statement 11	We do not recommend routine use of GDHT protocols for patients undergoing major elective abdominal surgery	Strong	High	N/A*
Statement 12	We recommend considering use of GDHT protocols on an individual patient basis for moderate- to high-risk patients undergoing major noncardiac surgery	Weak	Moderate	N/A*
Statement 13	We recommend against routine inclusion of fixed low-dose inotrope infusions in GDHT protocols	Strong	High	N/A*
Statement 14	We recommend considering use of goal-directed perfusion during cardiopulmonary bypass to reduce the incidence of acute kidney injury	Weak	Moderate	97 (35)
Statement 15	We recommend considering use of GDHT after cardiac surgery to reduce postoperative complications	Weak	Moderate	96 (46)
Statement 16	There is currently insufficient evidence to recommend routine use of GDHT protocols in patients undergoing emergency abdominal surgery	Weak	Low	N/A
Statement 17	We recommend considering use of GDHT to reduce perioperative complications in patients with hip fracture	Weak	Low	83 (84)

Statement 1: GDHT is an umbrella term for a complex intervention using monitoring techniques and physiological targets to help guide administration of fluids, vasopressors, and inotropes.

Statement 2: The goals of haemodynamic management are to optimise tissue oxygenation and support normal cellular metabolic function.

Technologies developed in the 1970s allowed the detailed measurement and manipulation of the cardiovascular system. Early studies suggested that survivors of major high-risk surgery displayed higher values of cardiac output and global oxygen delivery (DO_2) than nonsurvivors.[9] This led to the hypothesis that targeting goals for cardiac output and DO_2 (initially at 'supranormal/survivor' levels) for all patients undergoing surgery would reduce postoperative morbidity and mortality.[10]

DO_2 is the total amount of oxygen in millilitres delivered by the cardiovascular system to tissues per minute:

$$DO_2\ (ml\ min^{-1}) = cardiac\ output\ (CO)\ (L\ min^{-1}) \times arterial\ oxygen\ content\ (CaO_2)$$

The arterial oxygen content is optimised by increasing arterial oxygen saturation and haemoglobin, with dissolved oxygen contributing very little. This leaves cardiac output as the major variable that can be manipulated perioperatively to increase DO_2 with the aim of matching cellular metabolic demands.

Adequate organ perfusion pressure is also important in maintaining organ blood flow, and is achieved through maintaining mean arterial pressure (MAP) within the organ's autoregulatory range.[11] This is a fundamental haemodynamic goal and can be supported by maintaining MAP >60–65 mm Hg.[2,5]

In order to achieve optimal perfusion pressures, blood flow, tissue perfusion, and tissue oxygenation, GDHT uses monitoring and algorithm bundles to guide achievement of haemodynamic endpoints, optimising preload, afterload, and contractility by administration of fluids, vasopressors, and inotropes, respectively. The optimisation of preload is an individualised approach with dynamic tests, assessing whether patients respond to fluids or alternatively to an autotransfusion by passive leg raise.

The approach of GDHT is supported by the observation that preload and cardiac output optimisation after major surgery are associated with optimised microvascular flow and tissue oxygenation.[12] This association has been named 'haemodynamic coherence' indicating that improved macrocirculation and improved global oxygen supply result in improved microcirculation and tissue oxygen supply.[13] However, this coherence can be lost at the tissue level in some organs during shock, reperfusion injury, inflammation, and infection resulting in reduced tissue oxygen supply despite optimised macrocirculation.

In summary, GDHT is an individualised approach to maintain or restore tissue perfusion by optimising global cardiovascular dynamics including organ perfusion pressure, blood flow, and thus oxygen delivery to the tissues.

QUESTION 2. What are the components of GDHT protocols?
Statement 3: We recommend that GDHT protocols should have clearly defined component parts and physiological targets (strong recommendation; very low-quality evidence).

As detailed below and in Figure 1, there are a number of strategies available to achieve the overarching aims of GDHT. As a result, not all GDHT interventions are directly comparable. The term GDHT is best considered an umbrella term which in all approaches should be defined more clearly in relation to its components, namely monitoring technology used, primary and secondary physiological target(s), interventions used to achieve these targets, and the period during which the intervention is applied.

Statement 4: The GDHT evidence-base is complex as it comprises a variety of protocols, interventions, monitoring technologies, surgical procedures, patient factors, and outcomes.

Although there are common themes and approaches within GDHT, when discussing and evaluating this intervention the breadth of approaches, technologies, and clinical settings should be acknowledged (Fig. 1). Early approaches to GDHT from the 1980s and 1990s used the pulmonary artery catheter to target deliberate increases in cardiac output and DO_2 using fluids, inotropes, and red blood cell transfusion in a variety of major surgeries.[10] Contemporary trials typically use minimally invasive (arterial pulse wave analysis or oesophageal Doppler-based) devices, target a range of variables including cardiac stroke volume or so-called dynamic cardiac preload variables, and might use fluids with or without inotropes and vasopressors in the intraoperative phase, postoperative phase, or both and in a wider variety of surgical procedures. In the intervening period, numerous background changes in surgical and perioperative care have been introduced (e.g. minimal access surgery, enhanced recovery protocols, improved preoperative screening and preparation) that might modify the impact of perioperative haemodynamic management. Although there are common conceptual goals as stated above, the differences in approach and time taken for the evidence base to accumulate hamper the interpretation of most meta-analyses in this area. A further issue when comparing outcomes from GDHT research is not only the heterogeneity of interventions but also of the outcome measures used in the trials, an issue highlighted as a key limitation in evidence syntheses.[14] These factors underscore the need for large, robust clinical effectiveness trials aiming to provide definitive evidence of the effects of GDHT in a contemporary setting.

Statement 5: Fluid responsiveness is a key component of GDHT and is best defined as an increase in stroke volume in response to intravascular fluid administration.

Administration of i.v. fluid, guided by markers of cardiac output, has always been a key part of GDHT interventions, either in isolation or combined with inotropes and vasopressors. Conceptually, the aim is to achieve the best possible cardiac performance based on the Frank–Starling mechanism, describing the ability of a normal ventricle, at a set level of inotropy and afterload, to increase contractility when increased venous return leads to a raised end-diastolic volume (preload). This results in increased cardiac stroke volume. However, this mechanism has a maximum plateau of stroke volume. Further increases in venous return beyond this point will increase left ventricular end-diastolic pressure and volume without associated increases in stroke volume. The finding that stroke volume increases in response to a rapid increase in venous return caused by bolus fluid administration is described as 'fluid responsiveness'. The GDHT framework views such increases in stroke volume as beneficial, even

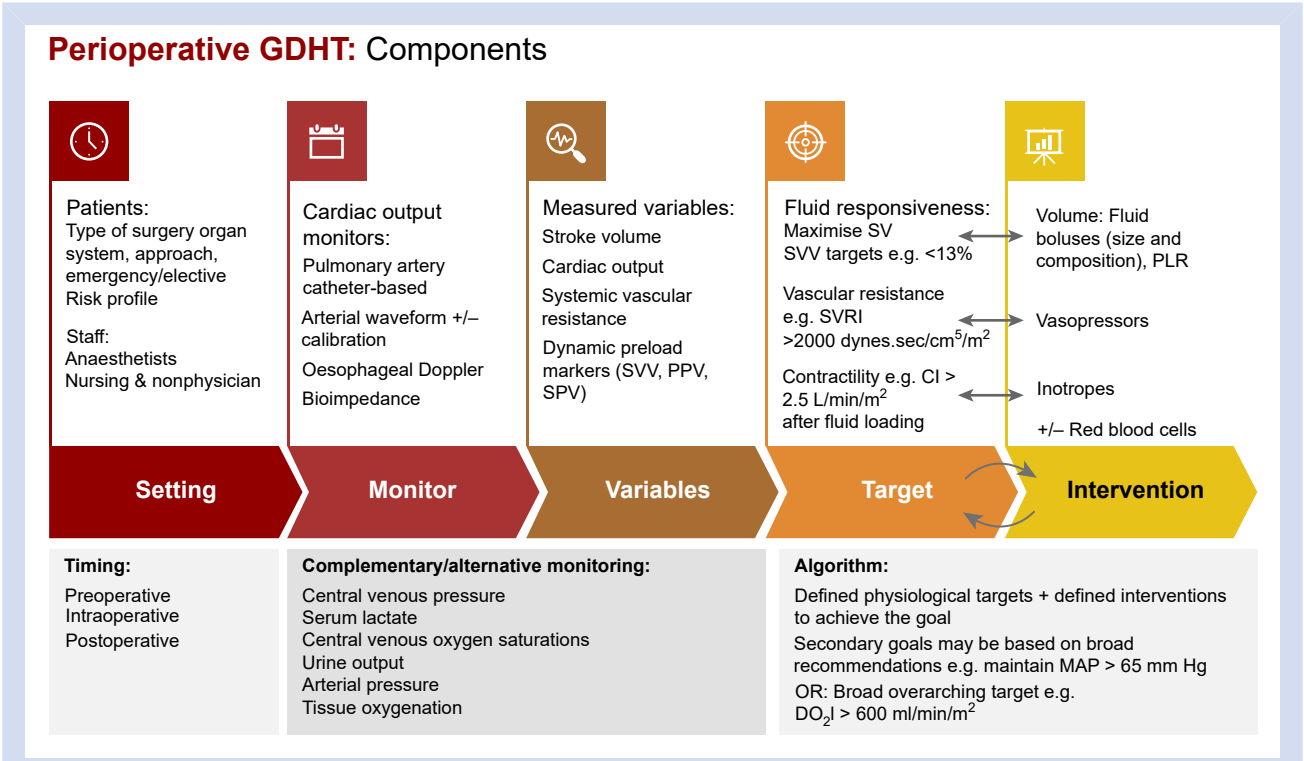

Fig 1. Component parts of any goal-directed haemodynamic therapy (GDHT) approach, including the clinical setting, monitoring technology, physiological variables measured, and the interventions used to target the chosen physiological goals. CI, cardiac index; DO_2I, indexed global oxygen delivery; MAP, mean arterial pressure; PLR, passive leg raise; PPV, pulse pressure variation; SPV, systolic pressure variation; SV, stroke volume; SVRI, systemic vascular resistance index; SVV, stroke volume variation. The target values included do not constitute practice recommendations by the Perioperative Quality Initiative (POQI) GDHT work group, but illustrate a range of values that have been targeted in GDHT protocols tested in clinical trials.

though it considers fluid administration only in the isolated context of cardiac performance without reference to intravascular stressed volume (the volume exerting distending pressure against the vessel walls), unstressed volume (the volume up to the point of filling the vessels but without exerting pressure) or total body water. A healthy unfasted subject with normal baseline circulating volume usually meets the definition of fluid responsiveness. Fears that this paradigm encourages unhelpful 'fluid overload' in more healthy patients[15] have been partly allayed by the finding that intervention and control groups in more recent GDHT trials ultimately receive similar volumes of fluid.[16]

Alternative techniques to identify improvements in cardiac output from increased venous return, and thence cardiac preload, have been described, notably the passive leg raise manoeuvre to deliver a fluid autotransfusion. Proponents argue that this avoids the situation of administering exogenous fluid as a diagnostic test when the ventricle is already working at near-maximal end-diastolic volume. However, the practicality of the passive leg raise manoeuvre is limited in the perioperative phase given the need to frequently re-establish the presence or absence of fluid responsiveness within GDHT approaches. The relatively small volume of fluid boluses used within GDHT algorithms (~250 ml) might provide a safety margin in this regard, with a negative response indicating that further bolus fluid administration should cease. At this point, stroke volume or cardiac output is continuously monitored, with further fluid challenge indicated only if the values decrease below those initially achieved.

Statement 6: Vasopressors and inotropes are additional GDHT components that can be titrated to achieve haemodynamic goals.

Vasopressors increase vascular tone, systemic vascular resistance, and cardiac afterload, whereas inotropic agents increase cardiac contractility, often with a positive chronotropic effect. Depending on the degree of receptor affinity, individual drugs can have relatively pure actions, or a mixture of vasopressor and inotropic effects depending on dose (e.g. epinephrine, norepinephrine). These agents can form part of a GDHT algorithm in several ways. Firstly, some GDHT protocols only use fluid loading and cardiac output responses for the mainstay of their intervention,[17] but give guidance on broader haemodynamic targets such as MAP and heart rate, with the expectation that clinicians will use vasopressors or inotropes to achieve these secondary targets. Secondly, GDHT algorithms can include a range of secondary targets derived from cardiac output monitors, and protocolise the titration of inotropes and vasopressors to achieve these targets. For example in the FEDORA trial, after fluid loading and attainment of an 'optimal' stroke volume (i.e. when there was no further increase in response to a 250 ml fluid load), MAP and cardiac

index were then examined. Vasopressors or inotropes were commenced and titrated as needed to achieve a MAP >65 mm Hg and cardiac index >2.5 ml min^{-1} min^{-2}, respectively.[18] Many trials have used a similar sequential approach of vasopressor and inotrope titration to achieve a range of haemodynamic targets.[19–21] Thirdly, a smaller number of trials have primarily focussed on stroke volume optimisation with fluids, along with broader haemodynamic goals, but also incorporated a fixed low-dose infusion of an inodilator (dopexamine or dobutamine that have mixed inotropic and mild vasodilatory effects).[16,22] The rationale for the inclusion of these agents is that at the low doses used they are likely to have marginal effects on cardiac inotropy but could have beneficial effects on microvascular blood flow (likely leading to improved tissue oxygenation) and systemic inflammation.[12,23]

Statement 7: Dynamic variables (such as pulse pressure variation and stroke volume variation) can be used to assess fluid responsiveness, but have limitations (strong recommendation, moderate-quality evidence).

The advent of minimally invasive cardiac output monitors using arterial pulse wave analysis provides a range of variables that could be derived and explored as therapeutic targets. Analysis of the beat-to-beat changes in pulse pressure, systolic pressure, or area under the arterial waveform curve throughout the respiratory cycle leads to derived variables such as pulse pressure variation (PPV), systolic pressure variation (SPV), and stroke volume variation (SVV). Given that variation in these measures has a relationship with preload (assuming constant vascular compliance and cardiac contractility), with less beat-to-beat variation with increasing stroke volume, they have been termed dynamic markers of preload responsiveness. It has been suggested that these variables could simplify approaches to GDHT, and reduce the volume of fluid administered to determine fluid responsiveness. An SVV of >12–13% has previously been proposed as a threshold above which fluid responsiveness is very likely,[24,25] and is used as a trigger for fluid bolus in certain GDHT algorithms.[21]

However, the reliable and accurate measurement of haemodynamic variations throughout the respiratory cycle depends on a number of factors, including a regular cardiac rhythm and the presence of mechanical ventilation with sufficient tidal volume, typically >8 ml kg^{-1}. These conditions limit the applicability of these markers, particularly with the current awareness of the potential risks of higher intraoperative tidal volume.[26] A large pragmatic study on the accuracy of SVV and PPV in predicting a future positive response of stroke volume (>10% increase) with a fluid bolus suggested they were not suitable for routine use, particularly in spontaneously ventilating patients after surgery.[27] Despite this, they might have some utility, for example at very low values (e.g. <5% SVV) the probability of fluid responsiveness is very low, so a fluid bolus can be avoided even if suggested by the current cardiac stroke volume measurement.[17,28]

Statement 8: We recommend that an increase (>10–15%) in stroke volume in response to a fluid bolus should be used to identify fluid responsiveness (strong recommendation, high-quality evidence).

Bearing in mind the above limitations of the dynamic markers of preload responsiveness and of alternative means of increasing venous return to test fluid responsiveness, we maintain that a stroke volume response to a rapidly administered (within 5 min) fluid bolus of ~250 ml should be used as the primary means of determining fluid responsiveness. An increase of at least 10% in stroke volume shortly after the end of the bolus should be considered a positive response.

The threshold of a 10–15% increase in stroke volume has been well-established, originally from studies determining the smallest change in stroke volume that could be reliably detected with the newer-generation minimally invasive cardiac output monitors.[29–31] Although monitoring technologies have evolved since this early work (towards autocalibrated or uncalibrated devices that are more suited to detecting trends rather than giving accurate absolute values of cardiac output),[32–34] these SV thresholds became embedded in algorithms investigated in perioperative clinical efficacy trials, with promising results.[16,35]

In order to elicit potentially beneficial increases in cardiac output, pharmacodynamic studies have shown that a fluid challenge should be administered rapidly, taking a maximum of 5–10 min to administer.[30] Slower infusions over 15–30 min can be subject to redistribution rather than a 'challenge' *per se*. Also, changes in the wider cardiovascular system such as the degree of sympathetic stimulation or changes in administered vasoactive agents can present confounding variables in interpreting the response. The maximal effect on stroke volume is likely to be seen 1 min after completion of a rapid bolus, so this should be taken as the point of assessing whether a response has been positive.[36]

Despite early protocols in critical care suggesting fluid volumes of 500 ml or greater could be used for fluid challenges, it has been shown that smaller volumes of 4 ml kg^{-1}, or ~250 ml, are sufficient to identify a similar proportion of 'fluid responders'.[37] Although fluid boluses in these algorithms have a combined diagnostic and therapeutic action, in the case of fluid 'nonresponders', fluid infusion is by definition unnecessary. As the core aim of haemodynamic therapy is to avoid iatrogenic harm from fluid excess, the volume of fluid bolus should be no more than essential. Between 40% and 70% of boluses administered in perioperative studies did not lead to an improvement in stroke volume and were therefore unnecessary.[27,30,38,39] Although 250 ml is a reasonable compromise with minimal risk of iatrogenic harm (particularly in protocols where a low SVV is used as a 'brake'[17,28]), there has been more recent interest in even lower volumes of fluid bolus.

The 100 ml 'mini fluid challenge' given over 1 min has been proposed as an indicator of a patient's subsequent response to the remainder of a 250 ml bolus, allowing cessation of the bolus if the response is negative. Studies suggest that a 5–6% increase in SV can be reliably detected by certain pulse wave analysis technologies if the cardiovascular status is otherwise stable, and serves as a reasonable predictor of the response to a full 4ml kg^{-1} bolus.[38,40] However, this has not been investigated rigorously in large effectiveness trials incorporating a wider range of surgical settings and monitoring technologies. Other manoeuvres using heart–lung interactions to detect fluid responsiveness without giving fluids (e.g. the end-expiratory occlusion test) appear less reliable than the mini fluid challenge technique or less suited to the perioperative setting.[39,41]

Statement 9: We recommend that GDHT protocols aim to optimise stroke volume or cardiac output and MAP with fluids, vasopressors, and inotropes (strong recommendation; moderate-quality evidence).

Cellular metabolic function depends on adequate blood oxygen content and tissue perfusion (blood flow). Given the dependency of tissue blood flow on blood volume, vascular tone, and cardiac performance (contractility, rate, and relaxation), GDHT protocols include interventions that can address these key areas. It is almost impossible to modify any of these factors in isolation because of their complex interactions and the mixed effects of most available therapies. Most GDHT algorithms have a primary target (e.g. fluid responsiveness, to address circulating volume and volume-related cardiac performance), but typically will give either broad or protocolised guidance on other aspects such as vascular resistance (or in a simpler expression, systemic arterial pressure) and possibly cardiac output. Data from the INPRESS[42] and FEDORA[18] trials, although targeting different primary physiological goals, underscore the potential benefits of incorporating MAP targets after optimisation of cardiac stroke volume with fluid bolus therapy. In most GDHT trials that have suggested benefit, stroke volume 'optimisation' is taken to mean a value of stroke volume that no longer increases (by $\geq$10%) in response to a further fluid challenge. At this stage the patient's cardiovascular system is considered to have been brought to the point of maximum cardiac output for a particular cardiac function curve, although the cardiac output measured will almost certainly be less than a true absolute 'maximum' that could be achieved in different circumstances such as hard exercise. Additional targets can then be considered, such as maintaining MAP values that have been associated with a lower risk of postoperative organ injury (e.g. >60–70 mm Hg).[5] Arterial oxygen content is a factor in DO_2, and is particularly dependent on arterial oxygen saturation and haemoglobin concentration. Using ventilation and oxygen administration to ensure adequate arterial oxygen saturation and maintaining haemoglobin concentration above a lower threshold is also advised to support GDHT while avoiding supranormal values.[17,28] Measures of the balance between oxygen delivery and utilisation, particularly central or mixed venous oxygen saturations, have also been studied, but have not been used extensively as a primary target of perioperative GDHT.

The rationale for including both fluids and cardiovascular drugs in GDHT algorithms is that although each of these therapies can have overlapping effects, no single intervention can address all the potential haemodynamic therapeutic needs of a patient undergoing major surgery. Fluid boluses can address deficits in circulating volume and optimise volume-dependent cardiac performance, but alone might not reliably improve systemic arterial pressure, particularly in patients with compliant arterial systems under anaesthesia. Conversely, although certain adrenergic drugs can increase venous return from the splanchnic and unstressed circulatory compartments, and increase cardiac contractility and arterial pressure, in isolation these effects could mask an underlying total intravascular volume deficit. Balancing the various aspects of haemodynamic status using these available therapies is a rational approach that has been investigated in numerous studies.

Although currently this is the predominant approach that has been developed through serial iterations in clinical trials, there are conceptual limitations. Firstly, none of the haemodynamic devices currently used in clinical care directly measure tissue perfusion. Furthermore, most of the available monitored variables are surrogates. For example, fluid responsiveness does give some information about blood volume status, but there are no available direct measures of stressed and unstressed blood volumes. Estimates of stroke volume and cardiac output are often based on arterial pulse wave analysis, with attendant limitations. Although echocardiography can measure both stroke volume and contractility more directly, practical limitations have hampered its uptake into widespread use in protocolised GDHT. MAP is by definition impacted by cardiac output and vascular resistance. Secondly, available therapies (fluids, inotropes, vasopressors) can be considered to be relatively nonspecific in that they have whole-system actions tending to increase DO_2, whereas a deficit in perfusion of a particular organ might be owing to impaired regional vascular tone or flow mismatch, or a relative deficit in either the stressed or unstressed volume compartment.

QUESTION 3. Does GDHT improve postoperative outcomes?
Statement 10: Clinical trials of GDHT have been conducted in a range of surgical situations using a variety of different protocols with mixed results.

Depending on the definitions, >100 randomised trials of perioperative GDHT have been conducted since the late 1980s.[14,43,44] A majority have been conducted in major abdominal and gastrointestinal surgery, with other trials a heterogeneous mix of multiple specialties and settings (elective or emergency) or focussed on a single specialty. The heterogeneity introduced by these diverse populations is further increased by almost all available variants of monitoring devices, physiological targets and interventions included in the protocols, and by the wide-ranging definitions of many of the outcomes studied.

Numerous systematic reviews, with meta-analysis where appropriate, have attempted to synthesise this complex evidence base.[14,16,43] A number of similar broad conclusions have been drawn. Firstly, the evidence base remains dominated by smaller trials (n<500) with significant risk of bias. Although there tends to be no small studies effect found, it is notable that no trial with >200 patients has found a mortality benefit. Secondly, the heterogeneity of interventions and outcomes means that the aggregated findings tend to be of low to moderate certainty, often with wide confidence intervals. Thirdly, there are inadequate data to comment on certain patient groups, particularly those undergoing emergency surgery.

With these caveats, prior evidence syntheses suggest that use of GDHT can reduce complications after surgery, with the strongest suggested benefit around reducing postoperative infections (e.g. odds ratio [OR] for surgical site infection 0.54 [95% CI 0.45–0.66], for pneumonia 0.69 [0.55–0.88]), and for anastomotic leakage (OR 0.61 [0.43–0.87]), with a trend towards a reduction in postoperative kidney injury.[14] Reductions in postoperative hospital length of stay and possibly mortality are also suggested,[14] although these findings became less certain in later syntheses.[43] There is a suggestion of a greater benefit in gastrointestinal and abdominal surgery and in higher-risk patients, again with limited certainty. However, a consistent conclusion is that larger, definitive trials of a size that would reliably detect modest differences in standardised, patient-centred outcomes are needed.

Statement 11: We do not recommend routine use of GDHT protocols for patients undergoing major elective abdominal surgery (strong recommendation, high-quality evidence).

Statement 12: We recommend considering use of GDHT protocols on an individual patient basis for moderate- to high-

risk patients undergoing major noncardiac surgery (weak recommendation, moderate-quality evidence).

Statement 13: We recommend against routine inclusion of fixed low-dose inotrope infusions in GDHT protocols (strong recommendation, high-quality evidence).

The OPTIMISE II trial was proposed to address ongoing uncertainty in the perioperative GDHT evidence base. Despite accrual of further evidence from smaller trials during the trial recruitment period, results remained mixed,[18,19,45,46] and the need for a definitive trial remained. This international trial recruited 2498 patients of ASA physical status ≥2 and aged ≥65 yr undergoing elective surgery on the gastrointestinal tract of at least 90 min duration. Patients were randomised to a GDHT intervention or usual care, with the trial intervention period running throughout and for 4 h after surgery. The intervention used an uncalibrated pulse wave analysis cardiac output monitor and 250 ml fluid boluses to optimise stroke volume (based on 10% increases), with the addition of a fixed (non-titrated) low dose of inodilator once volume loading had started. A secondary 'check point' ensured that SVV was >5% before each sequential fluid bolus was given. Either dopexamine or dobutamine could be chosen as the inodilator, but owing to availability only dobutamine was used. In the control group, a pragmatic approach was taken, but with a structured fluid maintenance prescription (1 ml kg^{-1} h^{-1}) and general haemodynamic targets (MAP 60–100 mm Hg, heart rate <100 beats min^{-1}) recommended for all participants.

There was no difference in the primary outcome of postoperative infection (23.2% intervention *vs* 22.7% control group); however, more intervention patients suffered an acute cardiac event within 24 h of surgery that required treatment (3% *vs* 1.7%, P=0.03). This was because of a higher incidence of arrhythmias and was ascribed to the effect of routine dobutamine administration in this group. The incidence of cardiac events had equalised by 30 days after surgery and there were no differences in secondary outcomes including acute kidney injury (AKI), hospital length of stay, or mortality within 6 months of surgery.

OPTIMISE II was designed to test 'real-world' clinical effectiveness; it had broad inclusion criteria and was conducted in a variety of hospitals globally, so the results have wide generalisability. It also had robust methodology, with high intervention compliance, high rates of data follow-up, and numerous measures to remove bias. When combined with the large randomised sample size, it seems unlikely that further trials of this iteration of GDHT in this patient group will have different findings. This is the basis for recommending that this type of GDHT approach should not be used as routine care in broad patient groups similar to those in OPTIMISE II. Furthermore, inclusion of routine fixed-dose dobutamine appears to have led to adverse cardiac effects with at least moderate short-term consequences (i.e. medical intervention was required), and so should be avoided.

The reasons behind the lack of benefit can only be speculated. The overall incidence of postoperative infection was lower than that in previous studies,[16] and the proportion of patients having minimal access surgery had increased. It is possible that such temporal improvements in wider perioperative care reduced the potential impact of haemodynamic interventions.

Within a large trial in a broad patient population such as in OPTIMISE II, there can be heterogeneity of treatment effect, so subgroups that benefit more than the average effect size might not be identified. Exploring this thoroughly would be a significant research challenge. OPTIMISE II also did not overturn the proposed underlying physiology of GDHT; there is no reason to believe that the concepts of fluid loading and volume-related cardiac performance are flawed just because clinical benefit in a broad population was not seen. Aside from the presumed harmful effects of routine dobutamine infusion, there was no other signal of harm from the intervention. Together this leads us to suggest that in selected cases, for example high-risk surgical settings with particularly complex haemodynamics, experienced clinicians can reasonably choose to use cardiac output monitoring and aspects of GDHT interventions to assess and manage haemodynamic status. This is in keeping with prior evidence syntheses suggesting that there might be more benefit from GDHT interventions in very high-risk surgery or patients.[43]

Clinicians should also consider the degree to which control group care in any GDHT trial with neutral results is reflective of their own fluid therapy practice before dismissing the findings outright. For example, in order to reduce unwarranted variation, many trials give guidance on good practice in relation to fluid maintenance volumes (e.g. 1 ml kg^{-1} h^{-1} of hypotonic fluid in the OPTIMISE II trial, with separate discrete boluses of 250 ml isotonic fluid for volume loading based on clinical judgement in the control group). It is notable that observational studies of clinical practice suggest that personnel are a stronger driver of fluid volume variation than patient or surgical factors, and that extreme high or low volumes are associated with worse outcomes.[47–49] It remains possible that such apparently unexplained variation in fluid practice is harmful, and the rational, structured approach seen in trial control groups might have benefits itself.

Cardiac surgery

Statement 14: We recommend considering use of goal-directed perfusion (GDP) during CPB to reduce the incidence of AKI (weak recommendation, moderate-quality evidence).

During CPB, cardiac output is determined by the flow of the CPB machine, which has been historically based on patient body surface area and core temperature. Pump flows of 2.2–2.8 L min^{-1} m^{-2} were considered adequate. However, observational studies have demonstrated an independent association between pump flow-determined oxygen delivery and postoperative AKI, identifying an optimal indexed DO_2 (DO_2I) of >270 ml min^{-1} m^{-2}.[50,51] These observations were subsequently confirmed by two RCTs. Ranucci and colleagues[52] demonstrated in a European multicentre RCT that GDP maintaining DO_2I at ≥280 ml min^{-1} m^{-2} compared with conventional perfusion (based on body surface area and temperature) was effective in reducing stage 1 AKI. Similarly, Mukaida and colleagues[53] demonstrated in a single-centre RCT in Japan that GDP (with DO_2I >300 ml min^{-1} m^{-2} during CPB) resulted in a significantly reduced incidence of AKI (from 30.4% to 14.6%). A recent meta-analysis including three RCTs (with n=777) confirmed that GDP reduces AKI with a relative risk of 0.52 (95% CI 0.38–0.70).[54] Apart from reduced AKI, no further treatment effects of GDP have been studied or demonstrated, and therefore any benefit regarding morbidity other than AKI and mortality are unknown. Nevertheless, the rationale of optimising tissue oxygenation by individualised oxygen delivery is well-justified, bearing in mind that the CPB prime volume induces haemodilution, and the decrease in

haemoglobin during CPB can then be adjusted by an individualised increase in pump flow to maintain adequate DO_2. The beneficial effect of GDP is also reflected in recent cardiac Enhanced Recovery After Surgery (ERAS) recommendations that include GDP as an intervention which plays a role in preventing organ injury associated with CPB.[55]

Statement 15: We recommend considering the use of GDHT after cardiac surgery to reduce postoperative complications (weak recommendation, moderate-quality evidence).

Although mortality after cardiac surgery has been decreasing over the past decade, high-risk patients undergoing complex surgery have a significant incidence of postoperative organ failure, including AKI, myocardial injury, or cognitive disorder, resulting in increased lengths of hospital stay and increased cost.[56–58] These high-risk patients frequently have limited physiological reserve and the plausibility for a beneficial application of GDHT appears to be strong based on the aim to individually optimise tissue oxygen delivery perioperatively and thus to avoid organ injury and haemodynamic decompensation. In the postoperative setting after cardiac surgery, this is achieved by individualised fluid optimisation, targeted usage of inotropes, and optimised haemoglobin concentration by RBC transfusions. Several studies have investigated GDHT after cardiac surgery, and a recent RCT with 126 high-risk patients undergoing coronary artery bypass grafting or valve repair demonstrated a treatment effect of reducing postoperative mortality plus major complications as a composite outcome from 45% to 27% (P=0.04). In addition, ICU and hospital lengths of stays were reduced.[59] In an associated meta-analysis (including six trials and 825 patients) and one additional meta-analysis (five trials and 699 patients), a lower rate of postoperative complications was demonstrated, with no reduction in mortality.[59,60]

The application of a GDHT algorithm as part of an AKI-prevention bundle in high-risk surgical patients is an interesting concept, which has been assessed in a multicentre RCT in patients undergoing cardiac surgery (n=278) and is currently being studied in patients undergoing major surgery.[61,62] In addition to GDHT, interventions of the AKI-prevention bundle include discontinuation of ACE inhibitors or angiotensin receptor blockers, tight glycaemic control, and avoidance of nephrotoxic drugs or radiocontrast agents. This treatment strategy was of benefit to cardiac surgical patients at high risk of developing AKI in the above-mentioned study by Zarbock and colleagues.[61] In the intervention group, 65% of patients received the complete bundle *vs* only 4.2% in the control group (P<0.001); although AKI rates were similar in both groups, the occurrence of moderate and severe AKI was lower in the intervention group (14% *vs* 24%; 95% CI 0.9–19.1; P=0.034).[61]

Similar to GDP, GDHT after cardiac surgery is one of the recommendations in the most recent ERAS guidelines after cardiac surgery emphasising that GDHT can guide perioperative resuscitation and prevent postoperative organ injury.[55]

Emergency surgery

Statement 16: There is currently insufficient evidence to recommend routine use of GDHT protocols in patients undergoing emergency abdominal surgery (weak recommendation; low-quality evidence).

A lack of data on patients undergoing emergency abdominal surgery has been highlighted as one of the key limitations of the GDHT evidence base to date. Of those trials incorporating mixed surgical populations, emergency surgery typically made up <10% of all participants. Patients presenting for emergency surgery can have important pathophysiological differences from those undergoing planned surgery. They can have coexisting critical illness, bleeding, or other factors that can disrupt normal haemodynamics even before surgery starts. It is therefore rational to consider that they might respond differently to GDHT, and to assess them separately in trials. A small pilot study[63] has been followed up by two efficacy studies, the smaller of which (n=43) compared two variants of a GDHT approach.[21,64] The larger GAS-ART trial (n=312) did not find any reduction in the composite outcome of complications or mortality within 90 days.[64] Given the modest size of the few trials currently available, there are insufficient data available to make firm recommendations. The ongoing FLO-ELA trial[17] will study >3000 emergency abdominal surgical cases in the UK, and will report in the coming years.

Hip fracture

Statement 17: We recommend considering use of GDHT to reduce perioperative complications in patients with hip fracture (weak recommendation; low-quality evidence).

Hip fractures are common in older individuals with high 30-day mortality rates (6–9%).[65–67] In addition, patients have a high risk of perioperative complications owing to their limited cardiopulmonary reserve based on surgery-associated stress and their fracture.[68] Early surgery has been introduced as a concept to improve outcomes and reduce mortality.[65,66] Furthermore, ERAS pathways have been described with beneficial effects, including reduced length of hospital stay and reduced postoperative complications.[69] GDHT as part of an ERAS pathway is relatively novel and was assessed only recently in a cohort study comparing patients receiving perioperative GDHT (n=279) *vs* patients without GDHT (n=272).[70] The GDHT group had improved outcomes regarding the primary endpoint with fewer patients presenting with intraoperative haemodynamic instability (37.5% *vs* 28.0%; P=0.017). Secondary endpoints showed fewer postoperative cardiovascular, respiratory, and infectious complications in the intervention group (21% *vs* 3.9%; P<0.001), reduced hospital length of stay (11 *vs* 8 days, P<0.001), and higher 1-yr survival (73% *vs* 84%, P<0.003). Future studies will be necessary to validate these results from a single-centre nonrandomised study, describing beneficial outcomes with GDHT in patients with hip fracture.

QUESTION 4. What are the future directions and research questions for perioperative GDHT?

Could technological assistance support physiological compliance with GDHT algorithms?

Smaller efficacy trials of GDHT interventions suggest good achievement of the planned physiological goals.[35,71] Larger clinical effectiveness trials, although suggesting good overall compliance with the conduct of the intervention protocols, might not report the degree of this physiological compliance (i.e. the extent to which algorithm targets were met). Other studies that have reviewed individual patient records have

shown that implementation of GDHT protocols by anaesthesia providers is highly variable, and the time-in-target for haemodynamic parameters was less than would be expected to improve clinical outcomes.[72] Could technological assistance such as closed-loop systems be implemented into GDHT to help maximise physiological compliance? Given the absence of benefit seen in OPTIMISE II where a relatively simple GDHT approach was used, such developments will need rigorous assessment of their effects on clinical outcomes.

Would further 'individualisation' of GDHT interventions improve clinical outcomes?

Although a large pragmatic approach to GDHT (OPTIMISE II) in a relatively unselected population did not improve clinical outcomes, are there as-yet undefined or unexplored patient endotypes or phenotypes that could benefit more or less from GDHT protocols? Alternatively are there individual components of GDHT protocols that might be more or less beneficial to patients, and can efficient trials be designed to help answer these questions? Is there a role for platform trials or other novel trial designs in exploring different components of GDHT protocols?

Is the prediction of future haemodynamic changes feasible and clinically beneficial?

Recent studies have examined the prediction of future haemodynamic changes using machine learning.[73,74] The core rationale is that an early warning of future haemodynamic instability would allow pre-emptive treatment before inadequate organ perfusion occurs. Studies have not shown benefit consistently,[73,75] but as technology evolves can these variables aid clinicians in predicting major haemodynamic changes? As with any developments in this field, robust assessment of clinical effectiveness will be required, in particular to explore whether or not these technologies are superior to currently available measurements.[76,77] A related, broader question is the degree to which artificial intelligence-based technologies can assist in addressing perioperative haemodynamic monitoring and therapy.

Strengths and limitations

We used a well-established modified Delphi process combining literature review with expert interpretation. The practical consensus statements and recommendations focus on important clinical areas where variation in clinical practice exists. The diverse group of experts was carefully selected to be from a variety of professional groups, institutional types, and locations. We included experts from all subspecialties of anaesthesia for which recommendations were made.

Our work has some limitations.[78] The methodology did not include a formal systematic review or meta-analysis of the literature. The POQI-11 group did not include lay members, patients, or representatives of the target population (i.e. patients who receive haemodynamic therapy during surgery). The process is partly based on expert interpretation; although a diverse group of experts was selected, it remains a discussion between a limited sample of clinicians, so there is some risk of bias. We did not formally document iterations of statements and recommendations during the review and revision process in the work group and plenary (whole POQI-11 group) sessions. We used the GRADE framework but did not formally document the process of agreeing on the classification of the strength of recommendations and the quality of evidence. We highlight areas of uncertainty or persisting discord in the explanatory text and rationale.

During the POQI meeting, the results of the large OPTIMISE II trial were not available. Given the predicted impact of those results, a number of hypothetical statements were discussed and refined through the iterative rounds, pending release of the trial results. The need to finalise and confirm consensus on the statements influenced by the OPTIMISE II results is a deviation from the usual POQI methodology. However, we do not feel this reduces the validity of these statements. Although voting by attendees of the EBPOM 2023 World Congress cannot be considered formal expert peer review, it is an interesting and novel methodological development to explore the response of a large informed and interested audience to 'fresh' recommendations by an expert group. In this regard, the voting was conducted anonymously using the Slido software to minimise bias associated with public declaration of views in front of peers. Several members of the whole POQI-11 group have been supported by industry partners for education or research work in the topic areas of this consensus conference. All potential interests have been declared below. None of the entities listed had any role in the design, conduct, or reporting of the POQI recommendations, nor in the preparation or submission of the manuscript.

Conclusions

Recent large clinical effectiveness trials have contradicted the previous evidence base suggesting the benefits of GDHT in broad, relatively unselected populations, so routine use of the current approach to GDHT is not recommended. Despite this, much of the underlying physiology remains relevant, and perioperative haemodynamic therapy might still have an impact on patient outcomes. A number of avenues remain open to explore newer approaches to this key area of perioperative care.

Authors' contributions

Literature review: all authors
Development of statements: all authors
Writing of the manuscript: all authors
Approval of the final manuscript: all authors

Declarations of interest

Members of the 11th *POQI meeting:* MO has received research funding from Baxter Healthcare (Deerfield, IL, USA), Biomerieux (Marcy-l'Étoile, France), and LaJolla Pharma (San Diego, CA, USA). JR has received consulting fees from Octapharma (Lachen, Switzerland) and Avania Medical (Bilthoven, Netherlands). AZ has received consulting fees from Biomerieux, Baxter, Bayer (Leverkusen, Germany), Novartis (Basel, Switzerland), Guard Therapeutics (Stockholm, Sweden), AM Pharma (Utrecht, Netherlands), Paion (Aachen, Germany), Fresenius Kabi (Bad Homburg, Germany), research funding from Biomerieux, Fresenius, Baxter, and speakers fees from Biomerieux, Fresenius, and Baxter. ADS has served as a consultant for Novartis, Alexion (Boston, MA, USA), AM Pharma, Renibus (Southlake, TX, USA), and Retia Medical (White Plains, NY, USA). TJG has received honoraria from Edwards Lifesciences (Irvine, CA, USA), Medtronic (Minneapolis, MN, USA) and Merck (Rahway, NJ, USA). MRE has received an honorarium for lecturing for Edwards Lifesciences and grant funding from the UK National

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.bja.2025.05.033.

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References

1. Thiele RH, Raghunathan K, Brudney CS, et al. American Society for Enhanced Recovery (ASER) and Perioperative Quality Initiative (POQI) joint consensus statement on perioperative fluid management within an enhanced recovery pathway for colorectal surgery. *Perioper Med* 2016; **5**: 24
2. Martin GS, Kaufman DA, Marik PE, et al. Perioperative Quality Initiative (POQI) consensus statement on fundamental concepts in perioperative fluid management: fluid responsiveness and venous capacitance. *Perioper Med* 2020; **9**: 12

3. Prowle JR, Forni LG, Bell M, et al. Postoperative acute kidney injury in adult non-cardiac surgery: joint consensus report of the Acute Disease Quality Initiative and PeriOperative Quality Initiative. *Nat Rev Nephrol* 2021; **17**: 605–18
4. Sessler DI, Bloomstone JA, Aronson S, et al. Perioperative Quality Initiative consensus statement on intraoperative blood pressure, risk and outcomes for elective surgery. *Br J Anaesth* 2019; **122**: 563–74
5. Saugel B, Fletcher N, Gan TJ, et al. PeriOperative Quality Initiative (POQI) international consensus statement on perioperative arterial pressure management. *Br J Anaesth* 2024; **133**: 264–76
6. Ostermann M, Auzinger G, Grocott M, et al. Perioperative fluid management: evidence-based consensus recommendations from the international multidisciplinary PeriOperative Quality Initiative. *Br J Anaesth* 2024; **133**: 1263–75
7. OPTIMISE II Trial Group. Cardiac output-guided haemodynamic therapy for patients undergoing major gastrointestinal surgery: OPTIMISE II randomised clinical trial. *BMJ* 2024; **387**, e080439
8. Granholm A, Alhazzani W, Møller MH. Use of the GRADE approach in systematic reviews and guidelines. *Br J Anaesth* 2019; **123**: 554–9
9. Shoemaker WC. Cardiorespiratory patterns of surviving and nonsurviving postoperative patients. *Surg Gynecol Obstet* 1972; **134**: 810–4
10. Shoemaker WC, Appel PL, Kram HB, Waxman K, Lee TS. Prospective trial of supranormal values of survivors as therapeutic goals in high-risk surgical patients. *Chest* 1988; **94**: 1176–86
11. Ackland GL, Brudney CS, Cecconi M, et al. Perioperative Quality Initiative consensus statement on the physiology of arterial blood pressure control in perioperative medicine. *Br J Anaesth* 2019; **122**: 542–51
12. Jhanji S, Vivian-Smith A, Lucena-Amaro S, Watson D, Hinds CJ, Pearse RM. Haemodynamic optimisation improves tissue microvascular flow and oxygenation after major surgery: a randomised controlled trial. *Crit Care* 2010; **14**, R151
13. Ince C. Hemodynamic coherence and the rationale for monitoring the microcirculation. *Crit Care* 2015; **19**: S8
14. Grocott MPW, Dushianthan A, Hamilton MA, Mythen MG, Harrison D, Rowan K. Perioperative increase in global blood flow to explicit defined goals and outcomes following surgery. *Cochrane Database Syst Rev* 2012; **11**: CD004082
15. Lai CW, Starkie T, Creanor S, et al. Randomized controlled trial of stroke volume optimization during elective major abdominal surgery in patients stratified by aerobic fitness. *Br J Anaesth* 2015; **115**: 578–89
16. Pearse RM, Harrison DA, MacDonald N, et al. Effect of a perioperative, cardiac output-guided hemodynamic therapy algorithm on outcomes following major gastrointestinal surgery: a randomized clinical trial and systematic review. *JAMA* 2014; **311**: 2181–90
17. Edwards MR, Forbes G, Walker N, et al. Fluid Optimisation in Emergency Laparotomy (FLO-ELA) Trial: study protocol for a multi-centre randomised trial of cardiac output-guided fluid therapy compared to usual care in patients undergoing major emergency gastrointestinal surgery. *Trials* 2023; **24**: 313
18. Calvo-Vecino JM, Ripollés-Melchor J, Mythen MG, et al. Effect of goal-directed haemodynamic therapy on postoperative complications in low–moderate risk surgical patients: a multicentre randomised controlled trial (FEDORA trial). *Br J Anaesth* 2018; **120**: 734–44
19. Pestaña D, Espinosa E, Eden A, et al. Perioperative goal-directed hemodynamic optimization using noninvasive cardiac output monitoring in major abdominal surgery: a prospective, randomized, multicenter, pragmatic trial. *Anesth Analg* 2014; **119**: 579–87
20. Nicklas JY, Diener O, Leistenschneider M, et al. Personalised haemodynamic management targeting baseline cardiac index in high-risk patients undergoing major abdominal surgery: a randomised single-centre clinical trial. *Br J Anaesth* 2020; **125**: 122–32
21. Pavlovic G, Diaper J, Ellenberger C, et al. Impact of early haemodynamic goal-directed therapy in patients undergoing emergency surgery: an open prospective, randomised trial. *J Clin Monit Comput* 2016; **30**: 87–99
22. Pearse R, Dawson D, Fawcett J, Rhodes A, Grounds RM, Bennett ED. Early goal-directed therapy after major surgery reduces complications and duration of hospital stay. A randomised, controlled trial [ISRCTN38797445]. *Crit Care* 2005; **9**: R687–93
23. Bangash MN, Patel NS, Benetti E, et al. Dopexamine can attenuate the inflammatory response and protect against organ injury in the absence of significant effects on hemodynamics or regional microvascular flow. *Crit Care* 2013; **17**: R57
24. Cannesson M, Le Manach Y, Hofer CK, et al. Assessing the diagnostic accuracy of pulse pressure variations for the prediction of fluid responsiveness: a "gray zone" approach. *Anesthesiology* 2011; **115**: 231–41
25. Cecconi M, Monti G, Hamilton MA, et al. Efficacy of functional hemodynamic parameters in predicting fluid responsiveness with pulse power analysis in surgical patients. *Minerva Anestesiol* 2012; **78**: 527–33
26. Futier E, Constantin JM, Paugam-Burtz C, et al. A trial of intraoperative low-tidal-volume ventilation in abdominal surgery. *N Engl J Med* 2013; **369**: 428–37
27. MacDonald N, Ahmad T, Mohr O, et al. Dynamic preload markers to predict fluid responsiveness during and after major gastrointestinal surgery: an observational substudy of the OPTIMISE trial. *Br J Anaesth* 2015; **114**: 598–604
28. Edwards MR, Forbes G, MacDonald N, et al. Optimisation of Perioperative Cardiovascular Management to Improve Surgical Outcome II (OPTIMISE II) trial: study protocol for a multicentre international trial of cardiac output-guided fluid therapy with low-dose inotrope infusion compared with usual care in patients undergoing major elective gastrointestinal surgery. *BMJ Open* 2019; **9**, e023455
29. Cecconi M, Dawson D, Grounds RM, Rhodes A. Lithium dilution cardiac output measurement in the critically ill patient: determination of precision of the technique. *Intensive Care Med* 2009; **35**: 498–504
30. Toscani L, Aya HD, Antonakaki D, et al. What is the impact of the fluid challenge technique on diagnosis of fluid responsiveness? A systematic review and meta-analysis. *Crit Care* 2017; **21**: 207
31. Critchley LA, Lee A, Ho AM-H. A critical review of the ability of continuous cardiac output monitors to measure trends in cardiac output. *Anesth Analg* 2010; **111**: 1180–92
32. Monnet X, Vaquer S, Anguel N, et al. Comparison of pulse contour analysis by Pulsioflex and Vigileo to measure and track changes of cardiac output in critically ill patients. *Br J Anaesth* 2015; **114**: 235–43

33. Saugel B, Kouz K, Scheeren TWL, et al. Cardiac output estimation using pulse wave analysis-physiology, algorithms, and technologies: a narrative review. *Br J Anaesth* 2021; **126**: 67–76
34. Slagt C, Malagon I, Groeneveld ABJ. Systematic review of uncalibrated arterial pressure waveform analysis to determine cardiac output and stroke volume variation. *Br J Anaesth* 2014; **112**: 626–37
35. Noblett SE, Snowden CP, Shenton BK, Horgan AF. Randomized clinical trial assessing the effect of Doppler-optimized fluid management on outcome after elective colorectal resection. *Br J Surg* 2006; **93**: 1069–76
36. Aya HD, Ster IC, Fletcher N, Grounds RM, Rhodes A, Cecconi M. Pharmacodynamic analysis of a fluid challenge. *Crit Care Med* 2016; **44**: 880–91
37. Aya H, Rhodes A, Grounds R, Cecconi M. Minimal volume for a fluid challenge in postoperative patients. *Crit Care* 2015; **19**, P189
38. Biais M, de Courson H, Lanchon R, et al. Mini-fluid challenge of 100 ml of crystalloid predicts fluid responsiveness in the operating room. *Anesthesiology* 2017; **127**: 450–6
39. Messina A, Lionetti G, Foti L, et al. Mini fluid chAllenge aNd End-expiratory occlusion test to assess flUid responsiVEness in the opeRating room (MANEUVER study): a multicentre cohort study. *Eur J Anaesthesiol* 2021; **38**: 422–31
40. de Courson H, Ferrer L, Cane G, et al. Evaluation of least significant changes of pulse contour analysis-derived parameters. *Ann Intensive Care* 2019; **9**: 116
41. Monnet X, Shi R, Teboul JL. Prediction of fluid responsiveness. What's new? *Ann Intensive Care* 2022; **12**: 46
42. Futier E, Lefrant JY, Guinot PG, et al. Effect of individualized vs standard blood pressure management strategies on postoperative organ dysfunction among high-risk patients undergoing major surgery: a randomized clinical trial. *JAMA* 2017; **318**: 1346–57
43. Jessen MK, Vallentin MF, Holmberg MJ, et al. Goal-directed haemodynamic therapy during general anaesthesia for noncardiac surgery: a systematic review and meta-analysis. *Br J Anaesth* 2022; **128**: 416–33
44. Hamilton MA, Cecconi M, Rhodes A. A systematic review and meta-analysis on the use of preemptive hemodynamic intervention to improve postoperative outcomes in moderate and high-risk surgical patients. *Anesth Analg* 2011; **112**: 1392–402
45. Diaper J, Schiffer E, Barcelos GK, et al. Goal-directed hemodynamic therapy versus restrictive normovolemic therapy in major open abdominal surgery: a randomized controlled trial. *Surgery* 2021; **169**: 1164–74
46. de Waal EEC, Frank M, Scheeren TWL, et al. Perioperative goal-directed therapy in high-risk abdominal surgery. A multicenter randomized controlled superiority trial. *J Clin Anesth* 2021; **75**, 110506
47. Thacker JKM, Mountford WK, Ernst FR, Krukas MR, Mythen MMG. Perioperative fluid utilization variability and association with outcomes: considerations for enhanced recovery efforts in sample US surgical populations. *Ann Surg* 2016; **263**: 502–10
48. Lilot M, Ehrenfeld JM, Lee C, Harrington B, Cannesson M, Rinehart J. Variability in practice and factors predictive of total crystalloid administration during abdominal surgery: retrospective two-centre analysis. *Br J Anaesth* 2015; **114**: 767–76
49. Miller TE, Mythen M, Shaw AD, et al. Association between perioperative fluid management and patient outcomes: a multicentre retrospective study. *Br J Anaesth* 2021; **126**: 720–9
50. Magruder JT, Crawford TC, Harness HL, et al. A pilot goal-directed perfusion initiative is associated with less acute kidney injury after cardiac surgery. *J Thorac Cardiovasc Surg* 2017; **153**: 118. 25.e1
51. Newland RF, Baker RA, Woodman RJ, Barnes MB, Willcox TW. Australian and New Zealand Collaborative Perfusion Registry. Predictive capacity of oxygen delivery during cardiopulmonary bypass on acute kidney injury. *Ann Thorac Surg* 2019; **108**: 1807–14
52. Ranucci M, Johnson I, Willcox T, et al. Goal-directed perfusion to reduce acute kidney injury: a randomized trial. *J Thorac Cardiovasc Surg* 2018; **156**: 1918. 27.e2
53. Mukaida H, Matsushita S, Yamamoto T, et al. Oxygen delivery-guided perfusion for the prevention of acute kidney injury: a randomized controlled trial. *J Thorac Cardiovasc Surg* 2023; **165**: 750. 60.e5
54. Gao P, Liu J, Zhang P, Bai L, Jin Y, Li Y. Goal-directed perfusion for reducing acute kidney injury in cardiac surgery: a systematic review and meta-analysis. *Perfusion* 2023; **38**: 591–9
55. Grant MC, Crisafi C, Alvarez A, et al. Perioperative care in cardiac surgery: a joint consensus statement by the Enhanced Recovery After Surgery (ERAS) Cardiac Society, ERAS International Society, and The Society of Thoracic Surgeons (STS). *Ann Thorac Surg* 2024; **117**: 669–89
56. Ostermann M, Kunst G, Baker E, Weerapolchai K, Lumlertgul N. Cardiac surgery associated AKI prevention strategies and medical treatment for CSA-AKI. *J Clin Med* 2021; **10**: 5285
57. Milne B, Gilbey T, Gautel L, Kunst G. Neuromonitoring and neurocognitive outcomes in cardiac surgery: a narrative review. *J Cardiothorac Vasc Anesth* 2022; **36**: 2098–113
58. Hausenloy DJ, Candilio L, Evans R, et al. Remote ischemic preconditioning and outcomes of cardiac surgery. *N Engl J Med* 2015; **373**: 1408–17
59. Osawa EA, Rhodes A, Landoni G, et al. Effect of perioperative goal-directed hemodynamic resuscitation therapy on outcomes following cardiac surgery: a randomized clinical trial and systematic review. *Crit Care Med* 2016; **44**: 724–33
60. Aya HD, Cecconi M, Hamilton M, Rhodes A. Goal-directed therapy in cardiac surgery: a systematic review and meta-analysis. *Br J Anaesth* 2013; **110**: 510–7
61. Zarbock A, Küllmar M, Ostermann M, et al. Prevention of cardiac surgery-associated acute kidney injury by implementing the KDIGO guidelines in high-risk patients identified by biomarkers: the PrevAKI-multicenter randomized controlled trial. *Anesth Analg* 2021; **133**: 292–302
62. von Groote T, Meersch M, Romagnoli S, et al. Biomarker-guided intervention to prevent acute kidney injury after major surgery (BigpAK-2 trial): study protocol for an international, prospective, randomised controlled multicentre trial. *BMJ Open* 2023; **13**, e070240
63. Harten J, Crozier JEM, McCreath B, et al. Effect of intraoperative fluid optimisation on renal function in patients undergoing emergency abdominal surgery: a randomised controlled pilot study (ISRCTN 11799696). *Int J Surg* 2008; **6**: 197–204

64. Aaen AA, Voldby AW, Storm N, et al. Goal-directed fluid therapy in emergency abdominal surgery: a randomised multicentre trial. *Br J Anaesth* 2021; **127**: 521–31
65. Bretherton CP, Parker MJ. Early surgery for patients with a fracture of the hip decreases 30-day mortality. *Bone Joint J* 2015; **97-B**: 104–8
66. Pincus D, Ravi B, Wasserstein D, et al. Association between wait time and 30-day mortality in adults undergoing hip fracture surgery. *JAMA* 2017; **318**: 1994–2003
67. Sheikh HQ, Hossain FS, Aqil A, Akinbamijo B, Mushtaq V, Kapoor H. A comprehensive analysis of the causes and predictors of 30-day mortality following hip fracture surgery. *Clin Orthop Surg* 2017; **9**: 10–8
68. Cowan R, Lim JH, Ong T, Kumar A, Sahota O. The challenges of anaesthesia and pain relief in hip fracture care. *Drugs Aging* 2017; **34**: 1–11
69. Liu SY, Li C, Zhang PX. Enhanced recovery after surgery for hip fractures: a systematic review and meta-analysis. *Perioper Med* 2021; **10**: 31
70. Lorente JV, Reguant F, Arnau A, et al. Effect of goal-directed haemodynamic therapy guided by non-invasive monitoring on perioperative complications in elderly hip fracture patients within an enhanced recovery pathway. *Perioper Med* 2022; **11**: 46
71. Funcke S, Schmidt G, Bergholz A, et al. Cardiac index-guided therapy to maintain optimised postinduction cardiac index in high-risk patients having major open abdominal surgery: the multicentre randomised iPEGASUS trial. *Br J Anaesth* 2024; **133**: 277–87
72. Boekel MF, Venema CS, Kaufmann T, van der Horst ICC, Vos JJ, Scheeren TWL. The effect of compliance with a perioperative goal-directed therapy protocol on outcomes after high-risk surgery: a before-after study. *J Clin Monit Comput* 2021; **35**: 1193–202
73. Maheshwari K, Shimada T, Yang D, et al. Hypotension prediction index for prevention of hypotension during moderate- to high-risk noncardiac surgery. *Anesthesiology* 2020; **133**: 1214–22
74. Davies SJ, Sessler DI, Jian Z, et al. Comparison of differences in cohort (forward) and case control (backward) methodologic approaches for validation of the hypotension prediction index. *Anesthesiology* 2024; **141**: 443–52
75. Wijnberge M, Geerts BF, Hol L, et al. Effect of a machine learning-derived early warning system for intraoperative hypotension vs standard care on depth and duration of intraoperative hypotension during elective noncardiac surgery: the HYPE randomized clinical trial. *JAMA* 2020; **323**: 1052–60
76. Mulder MP, Harmannij-Markusse M, Fresiello L, Donker DW, Potters JW. Hypotension prediction index is equally effective in predicting intraoperative hypotension during noncardiac surgery compared to a mean arterial pressure threshold: a prospective observational study. *Anesthesiology* 2024; **141**: 453–62
77. Yang SM, Cho HY, Lee HC, Kim HS. Performance of the Hypotension Prediction Index in living donor liver transplant recipients. *Minerva Anestesiol* 2023; **89**: 387–95
78. Howell SJ. The need for rigour in consensus statements and guidelines. *Br J Anaesth* 2024; **133**: 1123–5

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