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Fluid therapy recommendations for major abdominal surgery. Via RICA recommendations revisited. Part III: Goal directed hemodynamic therapy. Rationale for maintaining vascular tone and contractility

Recomendaciones de fluidoterapia perioperatoria para la cirugía abdominal mayor. Revisión de las recomendaciones de la Vía RICA. Parte III: Terapia hemodinámica guiada por objetivos. Fundamento para el mantenimiento del tono vascular y la contractilidad

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Introduction
Non-cardiac surgery in high-risk patients is associated with an increased incidence of postoperative complications and mortality, with multiple organ failure being a main cause of death in these patients. Only about 10% of all anaesthetic procedures are performed in high-risk surgical patients; however, these patients account for more than 80% of perioperative deaths. Poor cardiopulmonary reserves limit the patient’s ability to respond to the stressful insult and prevent the body compensating for the increased oxygen demand and, in essence, defines the “high-risk surgical patient”.3

Even though perioperative goal directed hemodynamic therapy (GDHT) has shown improved patient outcomes, and despite recommendations from experts in the United States and Europe, assessment of oxygen delivery (DO2) during high-risk surgery is not widely used. Although most anaesthetists use some kind of therapeutic goals and hemodynamic interventions, some of those common goals may not be appropriate. Stroke volume (SV), cardiac output (CO), and arterial blood pressure (BP) are the main components of hemodynamic optimization in the operating room. Lower SV, lower CO, or lower BP and its cumulative duration are associated with postoperative morbidity and mortality. BP and SV are weakly correlated, but complement microcirculatory perfusion. Insufficient tissue perfusion and cellular oxygenation due to hypovolaemia, heart dysfunction or both are main determinants of impaired outcomes. GDHT should be used on an individual basis, always from a physiological point of view, resulting in a more appropriate use of fluids, vasopressors, and inotropes.

Rationale for maintaining vascular tone

Hypotension
Hypotension is frequent between the induction of anaesthesia and the beginning of surgery. Disputed definitions of intraoperative hypotension include systolic BP below 80 mmHg, mean arterial pressure (MAP) below 55–60 mmHg, and a 20–25% decrease in systolic or mean BP from baseline. However, there is evidence that intraoperative hypotension is associated with acute kidney injury (AKI), myocardial injury, stroke, and mortality; untreated hypotension could contribute to increased postoperative morbidity by damaging major organs, such as the brain, heart, and kidney due to poor organ perfusion and ischemia. Moreover, prolonged intraoperative hypotension is associated with a decrease in both short and long-term survival. In critically ill patients, values of MAP of 72 mmHg or higher may be essential in order to prevent AKI and myocardial injury. A case control study conducted in patients undergoing non-cardiac and non-neurological surgeries concluded that a sustained decrease in intraoperative MAP of more than 30% from baseline values “was significantly associated with postoperative stroke”. Bilker et al. found that MAP of less than 50 mmHg had the greatest independent association with death in their study in 1705 patients undergoing noncardiac surgery.

Triple low
Sessler et al. discussed the concept of triple low (MAP <75 mmHg, low bispectral index (BIS) <45, and low minimal alveolar concentration (MAC) <0.80) in the context of excessive length of stay and increased risk of 30-day mortality. The authors concluded that a double combination of low MAP and low MAC was a strong predictor of mortality, and even more so when associated with low BIS values. Drummond suggested considering ”variability in normal population” when discussing published scientific evidence regarding cerebral blood flow (CBF) autoregulation with ”declining MAP” and the incidence of stroke.

Monk et al. observed that 1-year mortality increased by 3.6% for every minute that systolic blood pressure was less than 80 mmHg. In a retrospective review of perioperative deaths, Lienhart et al. found that intraoperative hypotension and anaemia were closely associated with postoperative myocardial ischaemic events. Intraoperative hypotension has also been linked to non-cardiac complications and adverse outcome after surgery. Episodes (not necessarily contiguous) of hypotension lasting longer than 15 min have been associated with an increase in mortality.

Rationale for vasopressor therapy
The goal of any hemodynamic perioperative intervention is to maintain or improve tissue perfusion. When MAP decreases below an autoregulatory threshold of about 60–65 mmHg, organ perfusion becomes pressure dependent. Within the microcirculation, the distribution and magnitude of blood flow represent a coordinated interplay between arteriolar, capillary, and venular segments based on local and regional metabolic demand. The rationale for vasopressor therapy in hypotensive states is based on the knowledge that in all regional circulations, including the renal, splanchic, cerebral and coronary beds, blood flow is autoregulated. Nonetheless, and contrary to what is often believed, tissue and microcirculatory perfusion is physiologically regulated by changes in blood flow, vessel density and local vasomotor responses, and not BP. Under physiological conditions, regulation of blood flow occurs autonomously in the tissues and is driven by metabolic demand. Atasever et al. demonstrated the biphasic response of the human microcirculatory system to NTG-induced hypotension in a clinical setting. This response to a relatively large dose of NTG is characterized by an initial increase in arteriolar diameter and a reduction in systemic BP, promoting microcirculatory flow. Then, when BP gets too low, this is followed by a phase in which microcirculatory flow can no longer be sustained. Although severe loss of BP results in alterations in microcirculatory perfusion, within the physiological range of CO and MAPm the relationship between systemic hemodynamics and microcirculation is relatively loose. It is to be expected that venoconstriction produced by a vasopressor allows the transfer of blood from the splanchic beds to the heart increasing right ventricular filling and CO. Alternatively, this is expected to increase ventricular afterload and thereby decrease SV. These two mechanisms are not mutually exclusive, and likely compete against one
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Together with the complex interaction between many factors regulating splanchnic circulation, they determine the effect of vasopressors on CO. As Maas et al. showed, since stroke volume variation (SVV) is a measure of how SV varies with changes in preload, patients with high SVV are operating on the steep portion of the cardiac function curve. Because the effect of norepinephrine on mean systemic pressure generally exceeds the effect on venous vascular resistance, increased preload with norepinephrine resulted in a significant increase in CO in this group of patients. In contrast, patients with low SVV are operating on the flat portion of their cardiac function curves, and the negative effect of increased afterload likely exceeds the small benefit of augmented venous return (VR), thereby decreasing CO.

Recently, Rebet et al. showed similar effects following administration of phenylephrine after episodes of hypotension in ventilated patients under general anaesthesia during surgery. They found that in preload-dependent patients, cardiac index (CI) and SV remained unchanged; whereas in preload-independent patients, administration of phenylephrine decreased CI and SV. Therefore, the response in terms of increasing SV and CO following the administration of vasopressors can be predicted by baseline SVV or PPV, because these give a picture of patient status within the Frank-Starling curve.

Although the deviating effects of noradrenaline and phenylephrine on MAP and CO are known, SV and CO monitoring will provide crucial information; however, most clinicians still rely on BP, heart rate and oxygen saturation during intraoperative hemodynamic management of patients undergoing high-risk surgery. There is some evidence of lack of benefit in increasing MAP

**Figure 1** Autoregulation of blood flow to the tissues driven by the perfusion pressure curve. The red line represents the autoregulatory responses in which flow changes relatively little despite a major change in perfusion pressure. In situations of severe hypotension (mean arterial pressure <50 mmHg), blood flow to the tissues is decreased, leading to hypoxia. There is a pressure below which an organ is incapable of autoregulating its flow because it is maximally dilated. This perfusion pressure may be between 50 and 70 mmHg, depending upon the organ. Below this perfusion pressure, blood flow decreases passively in response to further reductions in perfusion pressure.

**Figure 2** Stroke volume and afterload. Afterload is the resistance against which the ventricles pump, so greater afterload makes it harder for the ventricles to eject the stroke volume (SV). All else constant, an increase in vascular resistance would decrease SV. This usually does not occur, as contractility increases to maintain SV and thus cardiac output. A decrease in afterload increases SV.
Vasopressors in goal directed hemodynamic therapy

Wuethrich et al. compared a liberal maintenance fluid therapy versus a restrictive fluid therapy accompanied by continuous infusion of norepinephrine in patients undergoing planned open cystectomy with thoracic epidural. In the norepinephrine group, postoperative zero fluid balance, lower in-hospital and 90-day postoperative complication rates, and reduced hospitalization time were observed. Remarkably, only a slight increase in serum lactate was found in the restrictive group; there were no differences in other tissue perfusion parameters or hemodynamic indices at the end of the intervention. Unfortunately, the authors did not compare a GDHT group versus control. However, they showed that administration of norepinephrine counteracts the decrease in sympathetic tone and vasodilatation induced by epidural analgesia, anaesthetics, and analgesics, and may be more physiologic at compensating for a plegic vascular system than the liberal use of intravenous fluids. The induced reduction in enlarged unstressed blood volumes caused by norepinephrine, and the restoration of stressed blood volumes can maintain an adequate hemodynamic balance.

In spite of the positive results obtained by Wuethrich et al., it seems more physiological to base vasopressor administration on hemodynamic algorithms, rather than on continuous infusion. Salzwedel et al. randomized 160 patients undergoing elective major abdominal surgery and showed that GDHT using PPV, CI trending and MAP that involved the use of fluids, vasopressors and inotropes, reduced the total number of complications and the number of patients with complications. The benefits of such algorithms have been widely demonstrated. Although GDHT protocols aim to optimize tissue perfusion, it is currently unclear whether systemic hemodynamic parameters accurately reflect the final impact on perfusion at local tissue level. Interestingly, Stens et al. investigated whether GDHT based on PPV, MAP and CI (similar to Salzwedel et al.) improves microcirculatory perfusion when compared to a MAP-based strategy in patients undergoing elective abdominal surgery. The authors found that although the GDHT group showed an improvement in hemodynamic parameters, this did not correlate with an improvement in microcirculation or lactate measurements on the first postoperative day. These findings suggest that the relation between systemic hemodynamics and microcirculation is not fixed, especially when CO and BP values are within normal ranges. However, high-risk surgery is associated with microvascular dysfunction, and these alterations may play a role in the development of postoperative organ dysfunction. Defining the adequacy of GDHT requires attention to both global and regional perfusion.

Rationale for maintaining contractility

"Normal" cardiac output

Cardiac output is one of the most important physiological parameters, as it directly and proportionally reflects the
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![Graph: The Frank-Starling curves. Increasing EDV is equivalent to pre-extension of the muscle heart muscle, which will affect contraction tension through the length-force relation. However, as increased diameter also increases total load, the effect on stroke volume may be somewhat less than the effect seen in isolated muscles. Contractility increases with inotropic stimuli and decreases with heart failure, being a state of reduced contractility.](image)

metabolism of the entire organism. It is the primary determinant of global oxygen transport from the heart to the tissues, since it is the main contributor to $\dot{V}O_2$. Cardiac output is the sum of the systemic flow per minute, and is the product of SV and HR. The ability of the body to adapt to increased workload and hence metabolism is the result of the ability of the heart to increase HR and SV. The normal CO value depends on metabolic demand and individual characteristics (Fig. 4). Cardiac index (CI) is the ratio of left ventricular CO in 1 min to body surface area (BSA), thus relating heart performance to the size of the individual. The mean CI values reported are 3.1 ml/min/m$^2$ for women and 3.2 ml/min/m$^2$ for men.$^{68}$ Interestingly, Carlsson et al. found no difference between the CI at rest in normal individuals and elite athletes, showing that CI is primarily dependent on the basal metabolism.$^{45}$ The fundamental characteristic of elite athletes is an increase in basal SV, due to a higher total heart volume (THV),$^{69}$ due to either an increase in ventricular size or improved pumping mechanics, and a decrease in HR. This supports the higher reserve capacity of athletes for increasing CI through HR increase during stress. The larger THV of athletes can generate greater CO due to higher SV at similar HR.$^{68}$ In patients with congestive heart failure, CI is lower compared to the healthy population, primarily due to a lower SV. At rest, heart failure patients often maintain a normal CO until later stages of the disease, when CO becomes too low to meet the metabolic demands of the body.$^{70}$ Ageing is associated with a sedentary lifestyle which decreases metabolism, so CI decreases with age.$^{71}$ Age-associated changes in cardiac and vascular function are identified as a major risk factor for cardiovascular morbidity and mortality, with older patients having a higher risk of morbidity and mortality.$^{72,73}$ At rest $\dot{V}O_2$ exceeds the oxygen consumption of all tissues ($\dot{V}O_2$) combined. The optimal level of $\dot{V}O_2$ varies according to metabolic demands, but an inadequate $\dot{V}O_2$ increases the oxygen extraction ratio (OER), thereby maintaining aerobic metabolism. Physiologically, every time there is a reduction in $\dot{V}O_2$, there will be an increase in tissue oxygen extraction in order to stabilize $\dot{V}O_2$. The OER will keep increasing up to a critical $\dot{V}O_2$ below which $\dot{V}O_2$ becomes supply-dependent and anaerobic metabolism will occur.$^{74}$ When this process begins, $\dot{V}O_2$ is called critical $\dot{V}O_2$ and $\dot{V}O_2$/DO$_2$ dependence is established$^{15}$ (Fig. 5).

**Perioperative oxygen requirements**

Oxygen absorption during exercise is not directly comparable with oxygen absorption in the perioperative patient. However, as with exercise, oxygen consumption during and after major surgery is high.$^{76}$ Major surgical trauma increases oxygen requirements from an average of 110 ml/min/m$^2$ at rest to an average of 170 ml/min/m$^2$ in the postoperative period.$^{77}$ Anaesthesia is associated with cardiovascular depression and delay or failure to respond to fluid and blood loss, anaemia, and pre-existing comorbidities, such as cardiac, pulmonary or renal insufficiencies. The activation of an inflammatory response to surgery resulting in hemodynamic active substances such as cytokines is a
Figure 5  Relationship between oxygen delivery, oxygen consumption, oxygen uptake rate and lactate. Initially, as metabolic demand (VO2) increases or DO2 diminishes, O2ER increases to maintain aerobic metabolism and consumption remains independent of delivery. However, maximum O2ER is reached at a point called critical DO2 (cDO2). This is believed to be ~70%. Beyond cDO2, any further increase in VO2 or decline in DO2 must lead to tissue hypoxia and anaerobic metabolism (lactate production is a surrogate for this).

contributing factor that alters tissue oxygenation and normal values. Compromised physiologic reserves and multiple comorbidities in combination with extensive surgery seem to be a hallmark of high complication and mortality rates, because these patients are less likely to meet the increased oxygen demand that occurs during major surgery. While healthy or older individuals or patients with heart failure are able to maintain adequate DO2 at rest, this parameter is compromised in stressful situations such as exercise, or surgery, which is associated with significant systemic inflammatory response which is in turn associated with increased oxygen demand.

The supranormal oxygen delivery approach
Alterations in oxygen transport leading to tissue hypoxia and impaired microvascular flow are associated with the development of organ failure and death. The lack of an early marker for tissue hypoxia coupled with the fact that pioneering studies in which normal DO2 goals were used found no benefit suggests that normal values may not be adequate during surgical trauma; therefore, treating patients to achieve a high DO2 was seen as an attractive alternative. This prompted Shoemaker to hypothesize that deliberately increasing DO2 may prevent the development of organ failure. The ultimate aim of GDHT is still to prevent tissue oxygen debt by maintaining tissue perfusion. Supranormal DO2I has been shown to reduce both morbidity and mortality in the peri operative period. However, the clinically important question is whether there is an identifiable subset of patients who may benefit from supranormal DO2I targets.

Certain authors have studied survival rates in patients with a high CI and high DO2. The suggested DO2 value of 600 ml/min/m2 has yet to be confirmed. Nevertheless, when absolute values of CI or DO2 are used as therapeutic targets, they are often predefined. The use of individual goals instead of a pre-established arbitrary value of >600 ml/min/m2 is more rational and would avoid potential adverse events related to the GDHT. There is widespread controversy at to which values are the most suitable, and for which patients. Supra-normal values of DO2 should be defined in relation to pre-operative (i.e. normal) values of DO2 and not in relation to the “magic number” of 600 ml/min/m2. From an “energy debt” perspective, it is certainly much more important to consider the DO2 VO2 relationship than to indicate a specific value of DO2 or CI as a goal.

The oxygen supply
The most commonly used methods to assess global VO2/DO2 are mixed venous oxygen saturation (SvO2) and its surrogate, central venous oxygen saturation (ScvO2). During anaesthesia, it is reasonable to assume that ScvO2 reflects CO and oxygen supply. ScvO2 may therefore be a useful physiologic indicator to guide fluid responsiveness and administration. The main factors that influence ScvO2 are haemoglobin, arterial oxygen saturation of haemoglobin, CO, and oxygen consumption, or ScvO2 = SaO2 – (VO2/[CO x Hb x 1.34]).
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Theoretically, if three of these factors remain constant (SaO₂, SvO₂ and VO₂) the ScvO₂ value will reflect the changes in VO₂. There are multiple physiologic, pathologic, and therapeutic factors that influence ScvO₂, such as anaemia, hypovolaemia, contractility and bleeding. More importantly, both low and high levels of ScvO₂ can be pathologic.

The normal range for SvO₂ is 65–75%. Although SvO₂ above 70% does not necessarily reflect adequate tissue oxygenation, a persistently low SvO₂ (<30%) is associated with tissue ischemia and bad outcome, whereas normal or supranormal ScvO₂ values do not guarantee adequate tissue oxygenation. Therefore, additional hemodynamic...
Inotropics in GDHT

Fluid boluses alone may be sufficient to achieve CI, ScvO₂ and DO₂ goals, and GDHT using only fluids has been shown to improve outcomes in certain groups of surgical patients.7,8,97,98 Nonetheless, fluids are often not enough to achieve these goals, and inotropics and vasodilators may be necessary. Jhanji et al. highlighted the important pathophysiological mechanisms underlying the benefit of GDHT. They showed that SV-targeted colloid administration coupled with a fixed infusion rate of dopexamine improved oxygen DO₂, central ScvO₂, micro-vascular blood flow, and tissue oxygenation, and that fluid therapy alone led to additional modest improvements.99 However, the OPTIMISE trial, which examined the effect of GDHT (algorithm based on intravenous fluid boluses and an inotrope) in high-risk gastrointestinal surgical patients on outcomes following surgery64 did not confirm previous data suggesting the benefit of GDHT. Although a decrease was found in the primary outcome – a composite of predefined moderate or major postoperative complications and mortality at 30 days following surgery (OR 0.73; 95% CI, 0.53–1.00; p = 0.05) and cumulative mortality at 180 days (OR 0.61; 95% CI, 0.36–1.04; p = 0.07), these reductions were not significant. However, the study was underpowered, as the sample size was calculated based on an expected 30-day complication incidence of 50% in the control group and a 37.5% incidence in the GDHT group, whereas the incidence of complications was only 43.4% in the control group and 36.6% in the GDHT group (p = 0.07). Thus, the initial sample size calculation was based on a much higher incidence of postoperative complications than expected. Interestingly, in the pre-specified adherence-adjusted analysis conducted using established methods, the observed treatment effect was strengthened when the 65 patients whose care was non-adherent were assumed to experience the same outcome as if they had been allocated to the alternative group (RR, 0.80; 95% CI, 0.61–0.99; p = 0.04). Moreover, a significant interaction (p = 0.02) was found for timing of recruitment; the intervention was associated with a reduction in the primary outcome for patients recruited later (RR, 0.59; 95% CI, 0.41–0.84) compared with earlier at each site (RR, 1.51; 95% CI, 0.75–3.01). This shows that when the GDHT protocol was consistently applied, the treatment effect was strengthened. Arulkumaran et al. found a reduction in morbidity in patients who were treated to achieve supranormal DO₂ targets with the use of fluids and inotropes, without finding an increase in cardiac complications due to the use of inotropes.100

The ideal CO monitoring system should fulfil the following requirements: presentation of non-invasive, continuous, real-time data, easy to apply, easy to operate, non-operator dependent, accurate and reliable, and easy to interpret.101

There is currently no monitoring system that meets this ideal. Selecting the most appropriate hemodynamic monitoring device may be an important first step in reducing the risk of complications.102 However, no single hemodynamic goal or monitoring method has been accepted in the literature,103 and all variables measured must be correctly interpreted and applied to the individual patient104 (Fig. 6).

Conclusions

The concept of GDHT is based on anticipation. Predefined interventions with pre-specified goals are organized in a specific fashion in order to provide the best possible care to patients undergoing a high-risk intervention. Fluid loading, vasopressor or inotropic therapy could be adapted to each patient and each situation using comprehensive hemodynamic monitoring. Local algorithms should be available to optimize all hemodynamic components during this high-risk period.

Conflict of interest

JRM: received travel funding from Deltex Medical and hono- raria for lectures from Fresenius Kabi, Edwards Lifesciences, Deltax Medical and Merck Sharp & Dohme.

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