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Perioperative fluid therapy: defining a clinical algorithm between insufficient and excessive ☆

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Abstract In the perioperative scenario, adequate fluid and volume therapy is a challenging task. Despite improved knowledge on the physiology of the vascular barrier function and its respective pathophysiologic disturbances during the perioperative process, clear-cut therapeutic principles are difficult to implement. Neglecting the physiologic basis of the vascular barrier and the cardiovascular system, numerous studies proclaiming different approaches to fluid and volume therapy do not provide a rationale, as various surgical and patient risk groups, and different fluid regimens combined with varying hemodynamic measures and variable algorithms led to conflicting results. This review refers to the physiologic basis and answers questions inseparably conjoined to a rational approach to perioperative fluid and volume therapy: Why does fluid get lost from the vasculature perioperatively? Whereto does it get lost? Based on current findings and rationale considerations, which fluid replacement algorithm could be implemented into clinical routine?

1. Introduction

Because fluid management influences patient's outcome, basic and clinical research addressed the numerous different aspects contributing to fluid and volume administration in the perioperative period. Basic research improved knowledge on the function of the endothelial vascular barrier and its functional changes leading to vascular leakage. Clinical studies, proclaiming different approaches to fluid management, have shown conflicting results and do in most instances not refer to the vascular barrier's physiologic basis. The same accounts for studies that primarily focused on clinical goals to guide perioperative volume therapy. However, a rationale should generally be derived from physiologic facts and significant, comparable studies. Thus, this review summarizes the relevant knowledge on the physiology of the endothelial vascular barrier, on the effect of different intravenous fluids, and on the opportunities of hemodynamic monitoring to answer questions inseparably conjoined to the search for a rationale strategy for perioperative fluid therapy: Why does fluid get lost from the vasculature perioperatively? Whereto does it get lost? Which fluid replacement strategy should be implemented?

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2. Why does fluid get lost from the vasculature perioperatively?

2.1. The double-barrier concept

Human body fluid is located in the intracellular compartment, containing two-thirds of fluid, and in the extracellular compartment, which in turn divides into blood plasma and interstitial space. As the basis for cell metabolism, both compartments communicate across the vascular barrier to enable exchange of electrolytes and nutrients. In 1896 already, Ernest Starling [1] suggested that under physiologic conditions, large molecules, such as proteins and colloids, could not cross the barrier in relevant amounts. The generated interstitial colloid osmotic pressure is far below the intravascular pressure and opposes the hydrostatic pressure, continuously forcing blood toward the interstitial space [1]. According to the Starling [1] principle, only the endothelial cell line is responsible for the vascular barrier function. Opposing this concept, it has been shown that the interstitial colloid osmotic pressure is in fact nearly 70% to intravascular osmotic pressure without causing interstitial edema [2]. To understand this contradiction, the role of the endothelial glycocalyx (GLX) must be explained. Every healthy vascular endothelium is coated by syndecans and glypicans containing heparan sulfate chains, which together constitute the endothelial GLX [3]. Dissolved plasma proteins are loading the GLX to the about 1-μm–thick endothelial surface layer (ESL), which binds approximately 800 mL blood plasma and is subject of a periodic constitution and degradation [4,5]. The GLX acts as a molecular filter increasing the oncotic pressure within the endothelial surface layer, whereas a small space between the anatomical vessel wall and the ESL remains nearly protein-free [2]. Accordingly, fluid loss across the vascular barrier is limited by an oncotic pressure gradient within the ESL [6]! Starling's classic principle therefore complemented the "double-barrier concept" in which not only the endothelial cell line but primarily the ESL constitutes the vascular barrier [2,6].

2.2. Causes and consequences of vascular barrier dysfunction

Various agents and pathologic states impair the GLX scaffolding. Cited examples are postischemic reperfusion, which led to a 30-fold increased shedding of heparan sulfate [7], or hypervolemia, which causes GLX impairment by liberation of atrial natriuretic peptide from atrial cells [8]. Inadequately high fluid administration may therefore cause iatrogenic GLX damage. Furthermore, it is well recognized that exposure to inflammatory mediators, such as tumor necrosis factor–α, cytokines, proteases, and heparanase, as well as major surgery, reduces the thickness of the endothelial GLX [7-10]. A clinical investigation showing increased plasma levels of syndecan-1 and heparan sulfate in patients with global or regional ischemia undergoing major vascular surgery approves these experimental data [11]. The dramatic consequences of an only rudimentary GLX, which loses the bigger part of its ability to act as a second barrier, are a strongly increased transendothelial permeability and formation of interstitial edema [7,8]. Accentuating the clinical relevance, increased plasma levels of glycosaminoglycans and syndecan-1 were found in septic patients, whereas median glycosaminoglycan levels were higher in patients who did not survive [12]!

3. Whereto does intravascular fluid get lost?

3.1. Preoperative fastening and insensible perspiration

In the healthy human, urine production and insensible perspiration are physiologically replaced by water absorbed from the gastrointestinal system and primarily affect the extravascular space. Only if they are pathologically increased or if the physiologic replacement is limited does the physician have to compensate a perioperative loss artificially by infusing crystalloids. Preoperative hypovolemia after an overnight fasting period is regularly described in anesthesia textbooks [13,14], and current literature seems to confirm this declaration. Bundgaard-Nielsen et al [15] found a functional volume deficit in 70% of patients before low invasive surgery. A closer look at the experimental protocol reveals that blood volume was not measured directly, but cardiac stroke volume response to fluid administration was estimated by esophageal Doppler after induction of general anesthesia [15]. Because only minimal amounts of fluid were needed to reach a maximal stroke volume, one could assume that their data rather reflect the vasodilating effect of anesthesia induction than existing preoperative hypovolemia. Furthermore, direct measurements approve that blood volume is normal after an overnight fasting period [16]. Hypovolemia does not occur regularly in all patients, and fluid reloading is therefore unjustified at least in cardiovascular healthy patients prior to low invasive surgery [16]. Fluid loss from insensible perspiration is also overestimated in many patients, and infusion rates up to 8 mL/(kg h) plus occurring additional losses are advocated [13,14]. In fact, even when the abdominal cave is opened, a loss of only 1 mL/(kg h) occurs [17]! Theoretically, it should be adequate to compensate only insensible perspiration and urine production and to substitute occurring blood loss to maintain a normal blood volume. In practice, mainly based on the assumption that a generous fluid administration could prevent hypotension and postoperative renal failure, frequently, much greater amounts of crystalloids are infused perioperatively [18], although there is no evidence that the incidence of renal failure can be decreased by a liberal infusion regimen during surgery [19]. Nevertheless, in daily clinical routine, patients indeed seem to require much more intravenous fluids than suggested by the considerations above.