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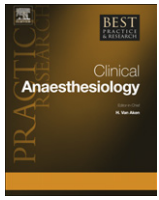

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Effects of perioperative fasting on haemodynamics and intravascular volumes

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Maintaining cardiac preload throughout the perioperative period is a generally accepted target. As perioperative fasting is believed to cause intravascular hypovolaemia it traditionally triggers aggressive preemptive intravenous fluid infusion. Physiology suggests that extracellular losses via urinary output and evaporation decrease the extracellular compartment. Representing a relevant part of the latter, the intravascular space is also affected, even without blood loss. Measurements in humans, however, have revealed that even a prolonged fasting period does not decrease absolute blood volume. Beyond that, modern fasting guidelines recommend to refrain from clear liquids only two hours prior to surgery. Nevertheless, an intravenous colloid challenge can increase stroke volume after induction of anaesthesia in the majority of surgical patients. While perioperative stroke volume maximisation in high-risk surgery probably improves outcome, the implication of this observation for the routine patient remains unclear. It appears as though there are two important targets to preserve cardiac preload: normovolaemia and vasotension.

Introduction

There is a broad consensus that intravascular hypovolaemia has to be avoided in the perioperative period.[1–7] Already a 10% deficit in blood volume significantly compromises splanchnic perfusion,[8] and the treating anaesthesiologist tries everything to avoid this clearly unwanted condition. Traditionally, preoperative fasting is identified as the main opponent of stable haemodynamics, still being blamed to

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have a significant and potentially harmful influence on cardiac preload.[9] Unfortunately, the widely spread established therapeutical answers appear too global and hardly target-oriented: More or less complicated regimes guide physicians to infuse large amounts of crystalloids and/or free water before or during induction of anaesthesia.[9–13] Even colloids have been used in a recent arguable study to replace an assumed intravascular deficit related to preoperative fasting.[14] In fact, it has been demonstrated that after induction of general and/or neuraxial anaesthesia cardiac performance is not individually maximal and can be increased by intravenous volume challenge in the vast majority of our surgical patients.[15] But is maximisation actually necessary in this situation? And what is the pathomechanism behind this decrease in the cardiocirculatory state?

The outcome-based evidence comparing different regimes to deal with cardiocirculatory instability after preoperative fasting and induction of anaesthesia is still limited. This article helps the reader to grasp the actual extent of the problem concerning fluid homeostasis related to preoperative fasting, based on physiology and scientific facts. It reveals how to rationally choose an adequate therapeutical approach for preoperative patients with a decreased cardiac output after induction of general and/or neuraxial anaesthesia.

Composition and basic physiology of compartments and barriers: the theory

55–60% of an adult's body mass is water. As water is not sufficiently retained at any barrier within the organism, its distribution is exclusively related to that of osmotically and oncotically active substances.[1] Two main barriers retain those substances within the three main compartments of the human body. While the intracellular space is surrounded by a cellular membrane, being largely impermeable for almost all solutes, the extracellular space is further divided by the vascular barrier into the intravascular and the interstitial space (Fig. 1).[1] Following increasingly upcoming knowledge the latter, beyond the well recognised endothelial cell line, functionally depends on the integrity of the endothelial glycocalyx.[16,17] This microscopically small molecular structure attached to the endothelial surface consists of proteoglycans and glycosaminoglycans and forms, together with bound plasma constituents, the physiologically active endothelial surface layer in vivo (Fig. 2).[16,18] This layer allows free passage of small solutes like electrolytes in both directions, but prevents a quantitatively relevant exchange of large molecules such as proteins or artificial colloids between the interstitial space and the intravascular compartment.[1] Therefore, the ionic compositions of plasma and interstitial space are comparable, while that of the intracellular compartment is composed completely different (Table 1). Moreover, large

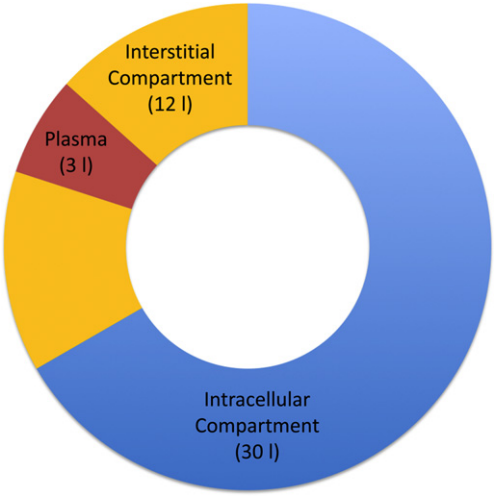

Fig. 1. Distribution of the body water in a cardiocirculatory compensated, normal-weighed male adult (average values). Extracellular compartment (15 l) = plasma + interstitial compartment.