

Fluid Therapy

Options and Rational Selection

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KEYWORDS

- Endothelial glycocalyx • Hypovolemia • Dehydration • Crystalloid • Colloid
- End points of resuscitation

KEY POINTS

- The discovery of endothelial glycocalyx has dramatically changed the understanding of vascular permeability, and should be considered when developing fluid therapy plans.
- Dehydration and hypovolemia are terms to describe fluid derangements, and each must be addressed uniquely to maximally benefit patients.
- Synthetic colloids have many potential benefits but appropriate patient selection is vital for maximizing patient safety while using them.

INTRODUCTION

Total body water (TBW) comprises 60% of body weight.¹ The 2 main fluid compartments in the body are the intracellular fluid (ICF) and extracellular fluid (ECF). The ICF compartment comprises approximately 60% of TBW, and the ECF compartment makes up the other 40%.¹ ICF is found inside the bilayered cell plasma membrane, and is in osmotic equilibrium with the ECF. The ECF is divided into 3 components: the interstitial compartment, the intravascular compartment, and the third space.¹ The interstitial compartment is the fluid space that surrounds cells and allows movement of ions, proteins, and nutrients across cell membranes. Approximately 75% of ECF is located in the interstitial compartment and is continuously turned over and recollected by the lymphatic vessels. The intravascular compartment comprises approximately 25% of the ECF, and fluids do not normally accumulate in the third space.¹

TRADITIONAL STARLING FORCES

Movement of fluid across capillary walls is essential for maintaining a continuous exchange of oxygen and carbon dioxide between the body's cells and the blood supply. There is a continual exchange between fluid compartments in the body, which

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provides nourishment to cells and removes waste products. Hydrostatic pressure and oncotic pressure work against each other to produce this fluid movement. Hydrostatic pressure is the pressure exerted by any fluid in a confined space. If fluid is in a container, there will be some pressure applied to the wall of that container. Picture a column-shaped container. The pressure pushing against its wall is greater at the bottom than at the top partly because of the force of gravity. Capillaries are the equivalent of a column-shaped container turned on its side. As fluid moves through a capillary, hydrostatic pressure causes fluid to move into the interstitial compartment. This movement also means that the hydrostatic pressure decreases as the blood moves from the arteriolar to the venous end of the capillary. The fluid pushed out through the capillary wall by hydrostatic pressure is called filtrate.

Blood contains plasma proteins that displace some water in blood, and less water in the intravascular compartment creates a concentration gradient between the intravascular and interstitial spaces. The osmotic pressure generated by plasma proteins is called oncotic pressure or colloid osmotic pressure (COP). Plasma proteins pull water into the intravascular compartment, whereas the force of osmosis equalizes the amount of water in the intravascular compartments and the interstitial fluid.

ENDOTHELIAL GLYCOLYX

According to traditional Starling forces described earlier, hydrostatic pressure pushes water out of capillaries and oncotic pressure pulls fluid into the intravascular compartment.² Since Dr Starling originally published his hypothesis, further research regarding fluid dynamics has emerged. It is now known that the luminal surface of endothelial cells is lined with a glycocalyx of membrane-bound macromolecules composed of sulfated proteoglycans, hyaluronan, glycoproteins, and plasma proteins; the endothelial glycocalyx (EG).

The high rate of resorption of interstitial fluid in the venular segments of the microcirculation hypothesized by Dr Starling does not happen. Filtration across the vascular barrier is largely independent of the bulk colloid concentration surrounding the vessel. In regions with high intravascular pressure, the inwardly directed oncotic pressure gradient across the EG prevents flooding of the interstitial space in conjunction with the high resistance to flow within the narrow strand gaps of the endothelium.³ Within low-pressure sections, free and easy access of plasma constituents toward the tissue cells allows a highly effective exchange of nutrients and waste products. Therefore, the fluid shift is modest if the endothelial surface layer is intact because of the low hydrostatic and oncotic pressure gradients in these segments.³

The principal role of the EG is to maintain the vascular permeability barrier.³ Other meaningful functions include shielding vascular walls from direct exposure to blood flow and mediating shear stress-dependent nitric oxide (NO) production.³ The EG also promotes retention of vascular protective enzymes and helps preserve the intravascular concentration of coagulation inhibition factors.³ The EG also helps modulate the inflammatory response by preventing leukocyte adhesion and binding of chemokines, cytokines, and growth factors to the endothelium.³

TISSUE SAFETY FACTORS

Extracellular edema forms when excess fluid accumulates in the interstitial compartment; this accumulation occurs as a result of either abnormal leakage from the intravascular compartment to the interstitial compartment or a failure of the lymphatics to return fluid from the interstitium to the intravascular compartment, or both. Altered capillary filtration occurs because of an increased capillary filtration coefficient,

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