Applied Physiology of Fluid Resuscitation in Critical Illness

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KEYWORDS

• Effective circulating blood volume • Mean systemic pressure • Venous return
• Stressed volume • Unstressed volume

KEY POINTS

- Venous return defines cardiac output.
- Fluid resuscitation aims to increase mean systemic pressure.
- Both fluids and vasopressors can increase mean systemic pressure and cardiac output.
- Crystalloids distribute across the body, whereas colloids tend to remain intravascular longer.

INTRODUCTION

Cardiovascular instability presenting as hypotension is often associated with decreased effective circulating blood volume and an associated increase in sympathetic tone. In the past, fluid management was the initial treatment of choice for all causes of hypotension. Although fluid management remains controversial, it is generally now agreed that fluids are not a 1-size fits all therapy; rather, it is a tailored management because the goals of care differ based on the cause of cardiovascular collapse. Indeed, many different processes can result in cardiovascular instability, not all of which benefit from fluid resuscitation. Only those patients in whom increasing the mean systemic pressure (Pms) will increase cardiac output are treated with fluid infusions. In the setting of trauma, the mechanism of cardiovascular instability is often hypovolemia secondary to hemorrhage; whereas in sepsis, the initial mechanism is increased unstressed blood volume due to a generalized inflammatory response-induced vasoplegia. However, with massive pulmonary embolism, cardiovascular

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instability is caused by right ventricular (RV) failure due to pulmonary vascular obstruction; in myocardial infarction, it is caused by decreased systolic pump function. Neither of these 2 common causes of circulatory shock is effectively treated by fluid infusion. To illustrate this, this article reviews the determinants of cardiac output, then the various shock causes, and then the fluids used to treat them.

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Why Give Fluids?

In cardiovascular resuscitation, the only reason to give fluids is to increase the Pms in the hope that it will increase the pressure gradient for venous return, thereby increasing cardiac output. Because the back pressure to venous return is right atrial pressure (Pra) and because the heart can only pump the blood it receives, it should be clear that the primary job of the heart with regard to cardiac output is to keep the Pra as low as possible so as to optimize this pressure gradient. Indeed, cardiogenic and obstructive shock can rapidly lead to cardiovascular collapse because the associated sudden increases in the Pra impair venous return and thus impede left ventricular (LV) filling, such that cardiac output and arterial pressure decrease. These points are illustrated on the combined LV function and venous return curves if one plots cardiac output verses the Pra (Fig. 1).

Effective Circulating Blood Volume

The Pms reflects the impact of blood volume distribution on the subsequent stressed volume of the circulation. However, most of the circulating blood volume, although it fills the intravascular spaces, does not directly contribute to the Pms. If one were to take a circularity system, otherwise intact but devoid of volume, and start infusing whole blood into the vascular space, a curious thing would happen: nothing. Venous pressures would remain at 0 and no flow would come back to the heart to allow pumping, even though quite a lot of blood might have been initially infused. Eventually, after enough blood was infused into the circulation, equal to approximately 60% to 70% of the total circulatory blood volume, another curious thing would occur: on infusion of

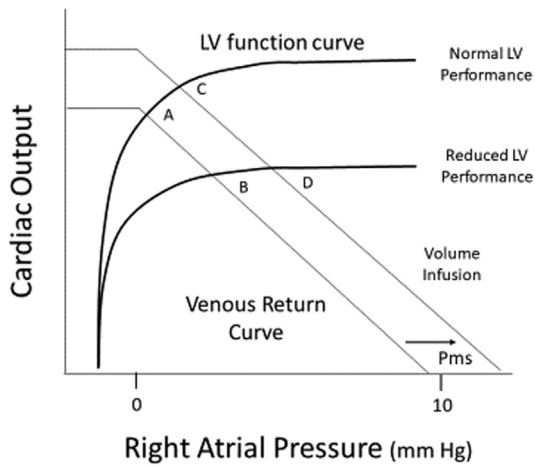

Fig. 1. Relationship between LV contractility (lines A–C and B–D) and intravascular volume changes on venous return (lines A–B and C–D). Fluid resuscitation increases Pms which on this graph is the Pra at the zero cardiac output value.