Hypovolaemia

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Abstract

The determination of intravascular volume in haemodynamically unstable patients is key to subsequent management. Administration of fluid remains the cornerstone of treatment but fluid loading in those patients not responsive to fluid is detrimental to outcome, as interstitial oedema impedes tissue oxygenation. Traditionally, static markers of cardiac preload such as central venous pressure have guided fluid therapy. Such markers have been shown to poorly predict fluid responsiveness. This has led to increased interest in dynamic variables such as the fluctuation of blood pressure and stroke volume in response to mechanical ventilation. These variables use the cyclical changes induced by positive pressure ventilation on right and left ventricular loading to measure changes in left ventricular stroke volume and arterial pressure. These dynamic indices, termed systolic pressure variation, pulse pressure variation and stroke volume variation, are superior in predicting fluid responsiveness in mechanically ventilated patients with a regular cardiac rhythm. In patients with spontaneous respiratory effort, the response of stroke volume to a passive leg raise is also an accurate indicator of fluid status.

Keywords Fluid responsiveness; heart–lung interactions; hypovolaemia; passive leg raise; pulse pressure variation; stroke volume variation; systolic pressure variation

Royal College of Anaesthetists CPD Matrix: 1A01, 2A02, 2A05

Learning objectives

After reading this article, you should be able to:

- define hypovolaemia
- define fluid responsiveness
- summarize the Frank–Starling curve and heart–lung interactions during mechanical ventilation
- recognize the limitations of central venous pressure in assessing fluid responsiveness
- understand the principles of dynamic indices of fluid responsiveness and their limitations

Definition

Hypovolaemia is defined as inadequate filling of the circulation. In health, one-third of total body fluid is distributed in the extracellular compartment (ECF), of which one-quarter resides within the vasculature. The causes of fluid loss from the intravascular compartment are broad but can be divided for ease into absolute and relative hypovolaemia. Absolute hypovolaemia is usually caused by excessive fluid loss, such as blood loss from trauma or surgery, plasma loss from burns, or ECF loss from diarrhoea and vomiting. Relative hypovolaemia occurs in sepsis and anaphylaxis, where total body fluid content is high but the intravascular compartment is deplete.

Pathophysiology

Lack of adequate circulatory volume results in reduced organ blood flow, oxygenation and organ damage. To compensate, the body initially responds with arterial and venous vasoconstriction to decrease the capacitance of the circulatory system and redistribute fluid from the skin and gastrointestinal tract. These compensatory mechanisms can maintain blood pressure until 30% of circulating volume has been lost. With insufficient intravascular volume cardiac output will fall secondary to inadequate left ventricular filling and hypotension will ensue.

Fluid responsiveness

Optimizing cardiac output and tissue oxygenation in haemodynamically unstable patients is of vital importance. Fluids remain the principal management of haemodynamically unstable patients, however studies have shown that only 50% of critically ill patients will be fluid responsive (i.e. respond to a fluid challenge by increasing stroke volume and cardiac output).[1] The dangers of insufficient fluid resuscitation have been recognized for years, but there is increasing awareness that excessive fluid administration increases morbidity and mortality in the critically ill. Therefore accurate predictors of fluid responsiveness are key in the management of haemodynamic instability – guiding the fluid requirements for those who are fluid responsive or indicating the need for vasopressors and inotropes and avoiding the detrimental effects of excess fluid in those who are not.

The Frank–Starling curve and central venous pressure

The Frank–Starling principle states that with increasing preload, left ventricular stroke volume will increase until an optimal preload is reached and stroke volume will not increase despite further fluid loading (Figure 1). A patient who is fluid responsive is on the ascending limb of the Frank–Starling curve and those who are non-responsive are on the plateau.

Central venous pressure (CVP) is traditionally used to guide fluid administration. As a measure of right atrial pressure, it has been assumed that CVP is a good indicator of right ventricular preload and, indirectly, left ventricular preload. However, studies have shown that CVP is a poor predictor of fluid responsiveness, with neither the initial value nor the response to fluid accurately indicating where a patient lies on the Frank–Starling curve.[1]

Heart–lung interactions in the mechanically ventilated

Positive pressure ventilation produces cyclical variation in the loading of the right and left ventricles (Figure 2). During inspiration, venous return to the right ventricle is reduced due to increased pleural pressure and a decrease in the venous return pressure gradient. Right ventricular afterload is increased due to an increase in trans-pulmonary pressure. This results in

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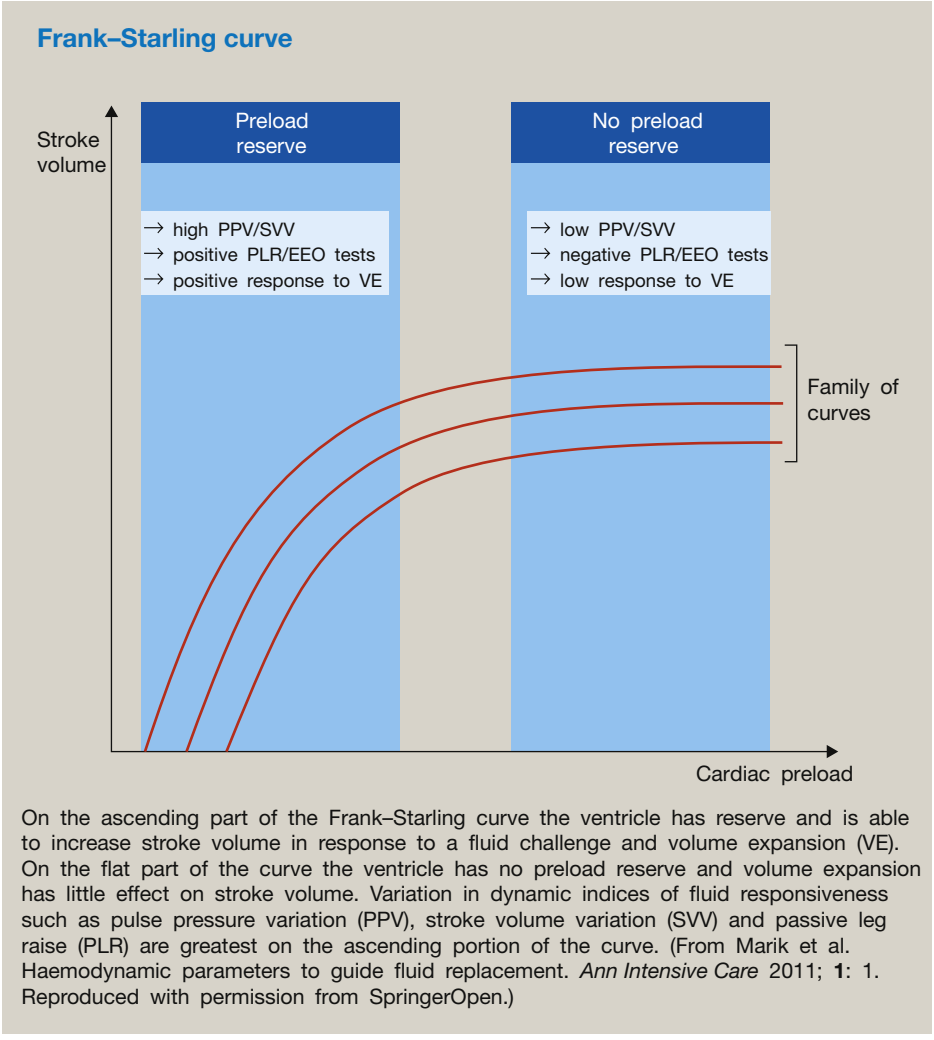

On the ascending part of the Frank–Starling curve the ventricle has reserve and is able to increase stroke volume in response to a fluid challenge and volume expansion (VE). On the flat part of the curve the ventricle has no preload reserve and volume expansion has little effect on stroke volume. Variation in dynamic indices of fluid responsiveness such as pulse pressure variation (PPV), stroke volume variation (SVV) and passive leg raise (PLR) are greatest on the ascending portion of the curve. (From Marik et al. Haemodynamic parameters to guide fluid replacement. *Ann Intensive Care* 2011; **1**: 1. Reproduced with permission from SpringerOpen.)

Figure 1

decreased right ventricular output. Conversely, during inspiration, the left ventricular output is transiently increased due to an increase in venous return from increased trans-pulmonary pressures, and decreased afterload. The decrease in right ventricular output seen during inspiration is echoed in a decrease in left ventricular output during expiration, the lag being accountable for by the transit time of pulmonary blood flow.[2]

The magnitude of variation will be greater when operating on the ascending portion of the Frank–Starling curve and will be exacerbated by hypovolaemia and diminished by fluid administration (Figure 1). The studies investigating these dynamic indices are only replicable in mechanically ventilated patients with tidal volumes of at least 8 ml/kg with a regular cardiac rhythm.

Dynamic indices of fluid responsiveness

Systolic pressure variation (SPV)

SPV can be calculated from the arterial waveform based on the fact that systolic pressure is proportional to left ventricular stroke volume. A variation in systolic pressure between 8 and 10 mmHg reflects a high likelihood that the patient will be fluid responsive.[3–5] However, systolic pressures may be more susceptible to variations in transmural and pleural pressures during mechanical ventilation and other dynamic indices such as pulse pressure variation have been shown to be superior.

Pulse pressure variation (PPV)

Pulse pressure variation is derived from the arterial waveform. It is defined as the maximum pulse pressure in a respiratory cycle minus the minimum pulse pressure divided by the mean of these two values. It has been suggested as superior to SPV as changes in pleural pressures will affect both systolic and diastolic pressures and thus PPV will reflect changes in stroke volume alone. Studies suggest variations greater than 13–15% are highly accurate in assessing fluid responsiveness.[3–5]

Stroke volume variation (SVV)

With the advent of cardiac output monitoring technology the estimation of actual variation in stroke volume through pulse contour analysis, rather than using surrogates such as PPV or SPV, is now possible. Variation between 9% and 13% has been reliably shown to predict fluid responsiveness.[5]

Spontaneously breathing patients

A limitation of the dynamic indices considered above is that they cannot be used in spontaneously ventilating patients. A recent