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UNDERSTANDING THE DISEASE

The forgotten relevance of central venous pressure monitoring

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Central venous pressure (CVP) monitoring is widely available in critically ill patients but its usefulness is frequently criticized. It is said that CVP neither indicates blood volume nor fluid responsiveness [1]. However, these arguments miss the real importance of CVP. CVP provides an ongoing indication of the equilibrium between the return of venous blood and the heart's ability to handle it. We argue that the emphasis on fluid responsiveness has obscured the clinical importance of CVP monitoring.

Measurement and normal values

Pressures measured with fluid filled systems are relative to a reference level. In cardiovascular physiology, the level is the mid-point of the right atrium, approximately 5 cm below the sternal angle. CVP in the upright posture is normally sub-atmospheric. Even at peak exercise, CVP only rises to about 4 mmHg and is in a similar range when supine. The heart is surrounded by pleural pressure (Ppl), whereas the body is at atmospheric pressure. When CVP is used to evaluate preload and cardiac function, what counts is the difference between the CVP relative to atmosphere and Ppl. This is called transmural pressure (CVPtm). Here, we focus on the downstream pressure determining venous return, CVP relative to atmosphere, and emphasize its important role in organ function and outcome.

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Impact of high CVP values

Outcome

Decreased survival of critically ill patients with elevated CVP is well described. A re-analysis of the VASST trial found that patients with a CVP < 8 mmHg during shock had a mortality 35% lower than those with a CVP of > 8 mmHg [2]. Similarly, critically ill patients who spent a longer time with a CVP > 10 mmHg had worse outcomes [3]. These studies, however, only indicate association and not that an elevated CVP causes increased mortality. To get closer to this point, examining the relationship between a high CVP and organ dysfunction can be helpful.

Kidney

Elevated CVP values are associated with increased risk of acute kidney injury (AKI) in critically ill patients [4]. In isolated animal kidneys, raising venous pressure decreased urine output, and stopped it at CVP > 25 mmHg [5]. The proposed mechanism is that increased interstitial pressure within the encapsulated kidney produces renal tamponade [6]. In patients with cardiovascular disease, CVP > 6 mmHg was associated with worsening renal function and decreased survival [7]. An acute rise in CVP proved more important than a chronic increase [8]. A retrospective study in critically ill patients, found that a 1 cmH_2O increase in CVP above 7 cmH_2O on admission was associated with a 2% higher risk of AKI [9]. Similarly, CVP > 14 mmHg early after cardiac surgery was an important determinant of AKI [10]. Ostermann (cited by [1]) showed that a decrease in mean perfusion pressure (MPP) was associated with progression from AKI stage I to stage III; CVP was the component of MPP that had an independent impact on AKI progression whereas mean arterial pressure was not. While these studies are still associative, the progressive

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decreases in function with higher CVP supports a causal relationship.

Liver

An elevated CVP is associated with liver injury and failure. Sherlock demonstrated in patients with heart failure that elevation of bilirubin was related to elevations of CVP but not to cardiac output [11]. In animal studies, CVP elevation directly increased portal venous pressure and reduced portal venous and hepatic arterial flow [12] indicating a mechanistic process.

How could CVP elevation per se cause organ damage?

Causality also is supported by identifying a credible mechanism for harm from an elevated CVP. This comes down to simple biophysics. Fluid filtration in the microcirculation is determined by the force driving fluid out of the vessel (hydrostatic pressure) and the force retaining fluid (intravascular oncotic pressure). Any increase in CVP directly increases capillary hydrostatic pressure, assuming no change in venous resistances (Fig. 1). To make matters worse, albumin, the main determinant of oncotic pressure, often is reduced in the critically ill. It, thus, is likely that a CVP > 10 mmHg will overload the filtration balance in the capillaries and produce tissue edema. Inflammatory conditions, e.g., sepsis or trauma, increase capillary permeability and exacerbate this phenomenon. Filtered fluid from capillaries into the interstitial space drains back to central veins through the lymphatic system, which is also hindered by CVP elevation [1]. The impact of an elevated CVP is likely much greater in encapsulated organs in which the increased parenchymal volume tamponades blood flow, and collapses urinary tubules, hepatic sinusoids, and venules.

Changes in Ppl induced by mechanical ventilation

The CVP value important for venous return is the value relative to atmosphere, and not CVPtm. A fundamental role of the right heart is to keep CVP as close to zero as possible. While in spontaneous breathing Ppl decreases during inspiration, during passive mechanical ventilation Ppl increases during inspiration. Moreover, application of positive end-expiratory pressure (PEEP), always makes Ppl positive. Accordingly, to maintain preload (i.e., the same CVPtm), a higher CVP is needed (Fig. 1). The body physiologically counteracts the effects of a decreased CVPtm by retaining sodium and water. Corresponding increases in CVP are transmitted to the portal veins [12] causing jaundice [13] and liver swelling [14]. Thus, positive pressure ventilation always creates a conflict between the higher CVP required to maintain preload (CVPtm), and prevention of organ congestion and damage. Whereas a decrease in CVPtm could be addressed by increasing inotropy, decreasing right ventricular afterload, or increasing heart rate, there are few ways to reduce tissue congestion except by lowering venous pressure.

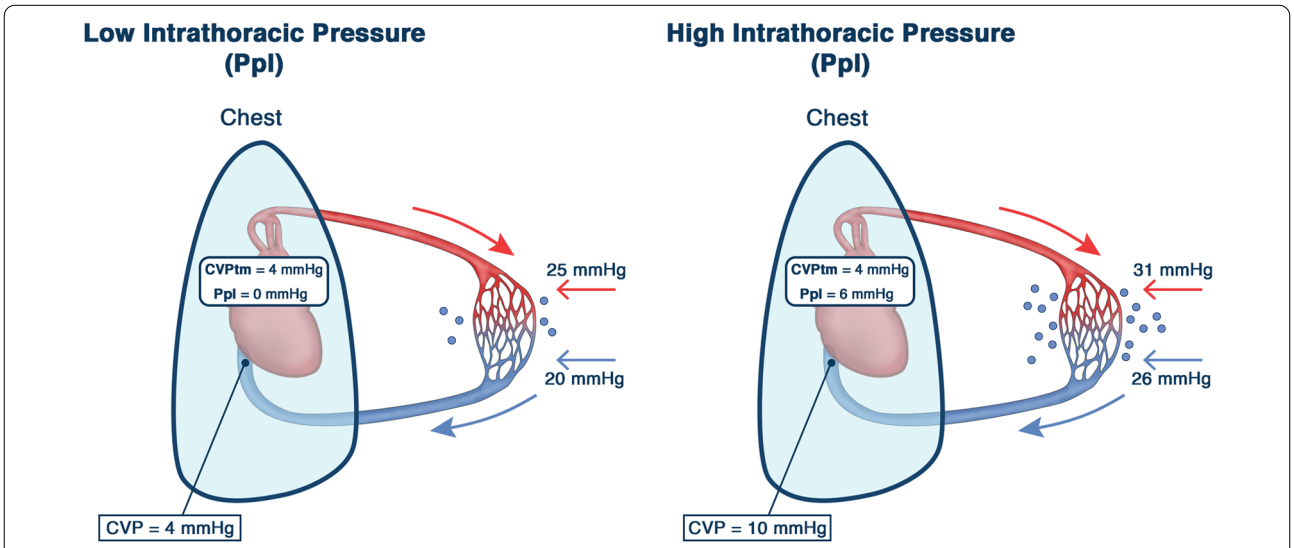

Fig. 1 Effects of an increase of intrathoracic pressure (Ppl). *CVP* central venous pressure, *Ppl* intrathoracic pressure, *CVPtm* transmural central pressure or right ventricular preload (CVP–Ppl). The heart pumps through the arterial system to resistance vessels and capillaries (shown as narrow lines). The pressure in the capillaries on the arterial side is ~ 25 mmHg and on the venous side 20 mmHg. When Ppl increases from 0 mmHg (left) to 6 mmHg (right), the same CVPtm is only possible if CVP is increased of the same amount, from 4 to 10 mmHg. The capillary pressure on the arterial side would be 31 mmHg. No other physiological adaptations are taken into account

In an animal study, fluid accumulation was directly related to mechanical ventilation, and further increased by adding PEEP [15]. A positive fluid balance in mechanically ventilated patients also was associated with mortality [16]. In patients with acute respiratory distress syndrome (ARDS), survivors had much smaller daily fluid balances than non-survivors [17]. Application of PEEP requires higher systemic venous pressures to maintain cardiac output. Thus, if CVP starts high, the tissue consequences are worse, and may hinder weaning.

Take-home message

CVP is normally low; higher than normal CVP values proportionally increase the risk of systemic tissue edema. Increases in CVP carry a price and costs must be balanced with benefits. When CVP is elevated, strategies such as keeping tidal volumes and airway pressures lower and/or using inotropes should be considered, particularly in the presence of increased capillary permeability. Emphasis on fluid responsiveness has obscured appreciation of the potential harm caused by a high CVP. CVP and its change over time are important safety parameters to monitor.

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Declarations

Conflicts of interest
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