Should central venous pressure be used to guide critical care management?

Central venous pressure is no longer routinely used to guide therapy in UK intensive care units, owing to evidence that it poorly predicts fluid responsiveness. This article reviews whether central venous pressure monitoring should be used to guide critical care management in certain patients.

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Introduction

Haemodynamic monitoring involves the use of real-time cardiovascular measurements to inform fluid, vasopressor and inotropic therapy. Central venous pressure has traditionally been a core component of haemodynamic assessment of patients in intensive care. It is easily measured by connecting the lumen of a central venous catheter via a continuous column of saline to a transducer placed at the level of the right atrium. The mean central venous pressure value in mmHg and the waveform are displayed on the monitoring device.

Methods of haemodynamic monitoring in critical care have evolved significantly over the past 40 years. More recently, less invasive methods of monitoring that derive cardiac output measurements from the arterial waveform (such as LiDCO and PiCCO) have become commonplace. They are used during 'dynamic' tests to assess a patient's response to interventions such as fluid challenges and have reduced reliance on static variables. The importance of central venous pressure has steadily declined and its role has become increasingly unclear.

The decline of central venous pressure monitoring

In the 1990s central venous pressure emerged as a widely used indicator of fluid responsiveness, with boluses administered until increases in central venous pressure were seen (Weil and Henning, 1979). As most intensive care patients had central venous catheters, central venous pressure was an accessible measurement and represented an alternative to the pulmonary artery catheter, which was more invasive and growing evidence indicated did not improve patient outcomes. The concept of central venous pressure as an indirect marker of left ventricular preload led to its widespread use to guide fluid therapy (Kastrup et al, 2007).

Subsequently, a series of systematic reviews has shown central venous pressure to poorly reflect fluid status or responsiveness (Marik et al, 2008). Consequently, it is no longer recommended to guide therapy in the Surviving Sepsis guidelines (Evans et al, 2021). This led to a more rigorous examination of what the central venous pressure value represents physiologically. It is now understood to represent a complex interaction between cardiac output, venous return, venous compliance and the variation in intrathoracic pressure during ventilation. It is even influenced by intra-peritoneal pressure. Furthermore, while central venous pressure measures right ventricular end-diastolic pressure, it does not necessarily correspond with right ventricular end-diastolic volume. Beyond its physiology, central venous pressure's low pressure range means that it is disproportionately affected by transducer placement and its measurement is prone to error. Unsurprisingly, central venous pressure is increasingly viewed as a redundant marker.

A potential return?

Despite this, elevated central venous pressure is fairly consistently associated with poor outcomes, including significantly higher levels of acute kidney injury and all-cause mortality (Boyd et al, 2011). Although this does not indicate causation, it is plausible that grossly

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elevated venous pressure could impair outcomes by reducing organ perfusion pressure, a situation that can occur in right ventricular dysfunction.

Right ventricular dysfunction, broadly defined as impaired right ventricular filling or contraction (Haddad et al, 2008), is a challenging condition to treat and its impact on patient outcomes was highlighted by the COVID-19 pandemic. A large multicentre study found intensive care unit patients with COVID-19 who had right ventricular dysfunction on echocardiogram had double the adjusted mortality of those without (Huang et al, 2022).

The right ventricle operates at lower pressures than its left-sided counterpart, and deals poorly with increases in afterload because of its smaller muscle mass. A spectrum of variably defined right-sided disease exists, with raised pulmonary pressures (or pulmonary hypertension) the most common cause of right ventricular dilation and/or dysfunction. Progressive distension can cause a vicious cycle with worsening tricuspid regurgitation causing further dilation, increased right ventricular wall stress and ischaemia. Acute right ventricular failure may develop where the right ventricle becomes unable to eject sufficient blood, causing systemic congestion with renal and hepatic dysfunction. Ventricular interdependence can develop as the enlarged right ventricle occupies an increasing volume within the pericardial space, limiting left ventricular filling.

In patients with right ventricular dysfunction, additional fluid may improve or worsen overall cardiac function. To complicate matters, dynamic markers of fluid responsiveness, such as stroke volume variation, are unreliable in patients with severe right ventricular dysfunction. Thus, optimal fluid status is crucial but also difficult to achieve, with small changes having an unpredictable but potentially significant impact. In such cases, central venous pressure monitoring can help clinicians remove fluid appropriately. In patients with right ventricular dysfunction, fluid removal may reduce distension and provide more space in the pericardium for left ventricular filling. Removing fluid, through diuresis or renal replacement therapy, until a normal central venous pressure is observed, can significantly improve left ventricular stroke volume (Hua et al, 2021).

Ventilation also has an important impact on right ventricular function. Acute respiratory distress syndrome is commonly associated with right ventricular dysfunction (Mekontso Dessap et al, 2016). Management is extremely difficult, as clinicians try to optimise alveolar recruitment (and oxygenation) while limiting right ventricular afterload. The level of positive-end expiratory pressure is critical. Its ability to recruit collapsed alveoli is well established, but excessive positive-end expiratory pressure can compress pulmonary vessels. In non-recruitable lungs this may increase oxygenation, but does so as the increased right ventricular afterload reduces cardiac output and the amount of shunted blood (Dell'Anna et al, 2022). This can increase right ventricular work while reducing oxygen delivery to tissues. Disproportionate jumps in central venous pressure while titrating positive-end expiratory pressure may indicate significant compression of pulmonary vessels.

An additional consideration is that it is the 'transmural' central venous pressure that is clinically useful – the pressure difference between the vessel fluid (the measured central venous pressure) and its surrounding intrathoracic pressure. This pressure difference across the vessel determines right ventricular wall tension, and can be calculated if intrathoracic pressure is measured. This may produce a more clinically meaningful value for central venous pressure.

Conclusions

Evidence suggests central venous pressure is a poor indicator of left ventricular filling and it has rightly been superseded as a marker of fluid responsiveness by other parameters. However, central venous pressure monitoring may have a role to play in guiding management of the failing right side of the heart. The vastly increased mortality of patients with right ventricular dysfunction who had COVID-19 indicates its clinical significance and that there is room for improvement in its treatment. While clear evidence is lacking, it seems reasonable to use central venous pressure monitoring as an adjunct to titrating positive-end expiratory pressure and facilitating fluid removal in patients with right ventricular dysfunction, potentially improving the function of both ventricles.

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