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SPECIAL ISSUE INSIGHT

Central venous pressure (CVP)

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Until recently, the central venous pressure (CVP) was the preferred variable to guide fluid therapy [1]. The interest for CVP has declined in the last few years, mainly after the publication of the 2016 version of the surviving sepsis campaign (SSC) guidelines, which no longer recommend it to guide fluid management in septic patients [2]. Nevertheless, CVP is a pivotal hemodynamic variable [3], since it is a major determinant of both the global cardiac function–through the Frank–Starling mechanism–and the venous status as it is the downstream pressure for venous return and for organ perfusion.

In this article, we underline how important it is to measure CVP to assess at best the hemodynamic status of patients with shock and thus select appropriate therapeutic options.

CVP should be measured properly

A fundamental prerequisite for correctly interpreting CVP is the quality of its measurement as many sources of errors may exist.

CVP measurements need a fluid-filled central venous catheter connected to an electronic pressure transducer linked to a monitor displaying a continuous pressure wave. The tip of the catheter should be located in the superior vena cava upstream to the right atrium.

The transducer should be positioned at the level of the midpoint of the right atrium [4]. The point at a vertical distance 5 cm below the sternal angle seems to be the most suitable. Proper levelling is crucial as even small errors in levelling might result in important consequences for interpreting CVP (Fig. 1) [5].

The CVP measurements must be taken at end expiration, a time when the intrathoracic pressure–the pressure surrounding the superior vena cava–is at its lowest value. In case of spontaneous breathing, the end-expiratory value is close to the highest value seen on the curve. If expiration is active, a CVP measurement early in the expiration is preferred as contraction of abdominal and respiratory muscles increases intrathoracic pressure during expiration [4]. In case of mechanical ventilation, the end-expiratory value is close to the lowest value seen on the curve. Nevertheless, when tidal volume is not high (6–8 mL/kg), taking the average value displayed by the monitor could be acceptable as it overestimates the end-expiratory value by only 1 mmHg [6]. In case of positive end-expiratory pressure (PEEP), the intrathoracic pressure is positive at end expiration. Therefore, to estimate the transmural CVP (end-expiratory CVP–end-expiratory intrathoracic pressure), one needs to correct for the value of PEEP transmitted to the thorax, which could be as high as 4 mmHg for a PEEP of 10 cmH_2O [7]. By analogy with what was shown for the pulmonary artery occlusion pressure (PAOP), the ratio of the difference between end-inspiratory and end-expiratory CVP values over the difference between airway plateau pressure and PEEP could represent the percentage of transmission of the airway pressure into the thorax [7]. This percentage of transmission must be then multiplied by the PEEP value to estimate the transmitted PEEP. Note that this method was used with CVP in a previous study [6] but not as validated as it was for PAOP [7].

Interpretation of CVP

CVP as a reflection of the right-ventricular (RV) filling pressure

The CVP is assumed to reflect the RV filling pressure, provided that its transmural pressure is obtained. An elevated transmural CVP suggests the presence of RV

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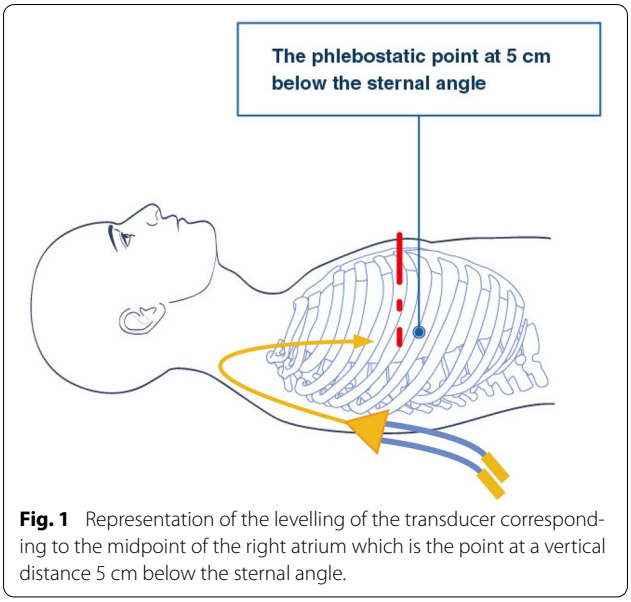

Fig. 1 Representation of the levelling of the transducer corresponding to the midpoint of the right atrium which is the point at a vertical distance 5 cm below the sternal angle.

dysfunction. Such a finding should encourage clinicians to perform an echocardiographic examination to confirm and find what the responsible mechanisms are. Vieillard-Baron et al. proposed to combine RV dilatation and CVP ≥ 8 mmHg to define RV failure with potential implications for fluid management [8]. Cardiac tamponade, severe pulmonary embolism, extended RV ischemia, and tension pneumothorax are among the acute pathologies responsible for such conditions.

Even if CVP reflects the RV filling pressure, it is now well established that CVP (or its changes) cannot be used to predict fluid responsiveness [9, 10]. These findings are explained by the fact that CVP is a static marker of preload, such that a given CVP value can be associated with preload responsiveness or preload unresponsiveness (through the Frank–Starling mechanism) in function of cardiac contractility.

To summarize, if correctly measured, the transmural CVP is a good means to suspect the presence of RV dysfunction but not to predict fluid responsiveness.

CVP as the downstream pressure for organ perfusion

The CVP also reflects the downstream pressure for perfusion of most vital organs (e.g., brain and kidney). The mean perfusion pressure (MPP) of such organs is the difference between mean arterial pressure (MAP) and CVP. For this purpose, the measured CVP but not the transmural CVP must be considered. Ostermann et al. demonstrated that MPP and not MAP was an independent factor associated with progression of acute kidney injury (AKI), with a cut-off value of 60 mmHg [11]. In cases of insufficient MPP due to elevated CVP, the best option is to reduce CVP whenever possible as this also reduces the risk of venous organ congestion, which may contribute to organ dysfunction [12]. In this regard, a recent meta-analysis showed that an elevated CVP is associated with an increased risk of AKI and of death in critically ill patients [13]. If CVP cannot be rapidly reduced, the alternative option is to restore MPP by increasing MAP, but this cannot prevent venous congestion. As CVP also reflects the downstream pressure of the lung lymphatic vessels, an elevated CVP can decrease lung lymphatic flow and lung edema resorption [14].

Take-home message

The CVP provides helpful information on RV function and organ perfusion, provided that proper measurements are performed. As it recommended to insert central venous catheters in patients with shock [15], it would be regrettable not to use them for measuring CVP to assess at best their cardiovascular status.

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Declarations

Conflicts of interest
OH is a member of the medical advisory board of AOP ORPHAN. J-LT is a member of the medical advisory board of Pulsion/Getinge

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