Central Venous Pressure

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3.1 Summary

Central venous pressure (CVP) reflects the pressure in the major veins, namely, vena cava *superior* and *inferior*. From the physiological point of view, the central venous pressure is a product of the complex interplay between potential heart performance and venous return; therefore, the response of CVP to the similar hemodynamic interventions can be opposite in different ICU patients. Historically, CVP was often used for the assessment of hemodynamics, volume status, and fluid responsiveness. However, over the last decades, multiple studies have demonstrated the absence of correlation of both absolute values and changes in CVP with end-diastolic left ventricle volume and cardiac output. Not surprisingly, CVP is unable to predict changes in cardiac output in response to fluid challenge. Nowadays, a certain "renaissance" of CVP seems to be possible since new studies show that increased baseline values and/or fast increment of this parameter are associated with progression to acute kidney injury, multiple organ failure, splanchnic congestion, and death. Thus, the therapy aiming to decrease CVP may improve organ function and clinical outcome. Obviously, there are many

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questions to be addressed concerning CVP in critically ill patients before the decision on when and how to use this hemodynamic parameter.

3.2 Introduction

The blood enters the heart chambers under certain force known as "filling pressure." In the case of the right atrium, the filling pressure is called the central venous pressure (CVP); to be more precise, CVP approximates the end-diastolic pressure in the right atrium. Monitoring of this parameter is widely available as central venous access is one of the routine invasive procedures performed in the majority of ICU patients.

A typical point of the CVP measurement is a proximal part of the vena cava *superior* near the junction with the right atrium where the tip of the central venous catheter is placed. As a rule, the geometric center of the right atrium should be taken as the baseline level for the CVP measurement. Pressure transducer is zeroed to atmospheric pressure at the "point of the right atrium" which can be easily determined by lowering the perpendicular of about 5-cm long (for an adult) from the front surface of the chest starting from the level of the sternum angle to the point of junction of the sternum and second rib [1]. In practice, the "phlebostatic point" is located at the intersection of the middle axillary line with the fifth rib or fourth intercostal space. It is easy to identify, but measurements will only be possible in a horizontal position. The values measured in the projection of the "phlebostatic point" exceed those at the level of the "point of the right atrium" approximately by 3 mmHg [2].

3.3 Morphology of the Central Venous Pressure Curve

The shape of the CVP curve has some distinct similarities with one of the systemic (arterial) blood pressure. According to the classic representation, five segments can be distinguished in the curve, three of those are peaks (waves *a*, *c*, and *v*) and two are descents (waves *x* and *y*) (Fig. 3.1). It is generally accepted that *c*, *x*, and *v* are of systolic origin, while wave *a* and descent *y* are diastolic. The most noticeable element of the CVP curve is wave *a*, which reflects the contraction of the right atrium that occurs after the completion of cardiac diastole. In approximate, wave *a* corresponds to the P wave on the electrocardiogram. With the beginning of the right atrium relaxation, wave *a* fades out and is interrupted by a small dicrotic *c* wave that is associated with isovolumetric contraction of the right ventricle and "prolapse" of the closed tricuspid valve toward the atrium.

If measured in a more distal section of the venous bed, for example, in the superior bulb of the internal jugular vein, wave *c* may be associated with the pulsation transmitted from the internal carotid artery ("carotid wave") [3]. Wave *c* corresponds to the onset of ventricular systole and, in part, to the period of early ejection. Atrial pressure continues to decline throughout the ventricle systole, turning into a descent, or cut *x*. At the end of the ventricle systole, a second rise in CVP is observed with the wave *v* associated with venous filling of the atrium during diastole, The wave *v* approximately corresponds to the T wave on ECG and is followed by a further decrease in the pressure curve with the formation of descent *y*, associated with the decline of the right atrium pressure during the ejection of blood into the ventricle (diastolic collapse) and the opening of the tricuspid valve. In some cases, plateau *h* can be recorded, persisting from the middle to the end of the diastole [3].

The most important argument in favor of analyzing the shape of the CVP curve is the possibility of early recognition of arrhythmias [4].

3.4 Determinants of the Central Venous Pressure

The resulting value of CVP is a product of the interplay of two key factors: the function of "venous return" characterizing the blood backflow to the right heart and the function of the heart (cardiac output and contractility) (Fig. 3.2) [5].

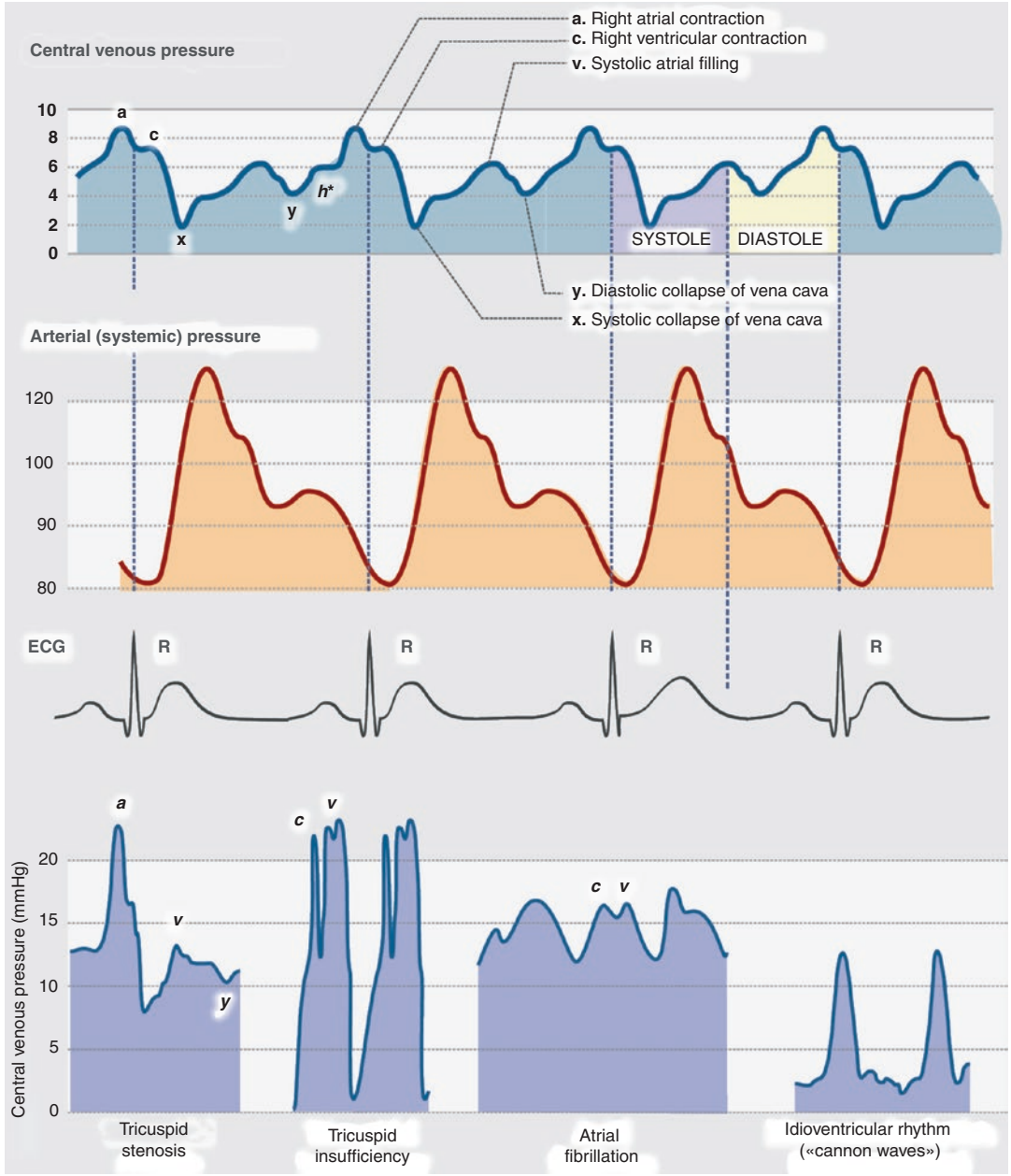

Fig. 3.1 Normal shape and elements of the central venous pressure curve (upper panel) and its changes under certain clinical conditions (lower panel)

The central venous pressure is largely dependent on the tone (resulting compliance) of the venous reservoir. It is considered that CVP is determined by the correspondence between the volume of blood and the capacity of the venous vascular bed, the condition of the main veins and heart valves (to a greater extent, the tricuspid valve), as well as the compliance of the right ventricle and pulmonary artery pressure [6]. These numerous factors significantly impede the straightforward clinical interpretation of the baseline value and changes in CVP.

According to the Guyton's model of circulation, there are three main determinants of cardiac

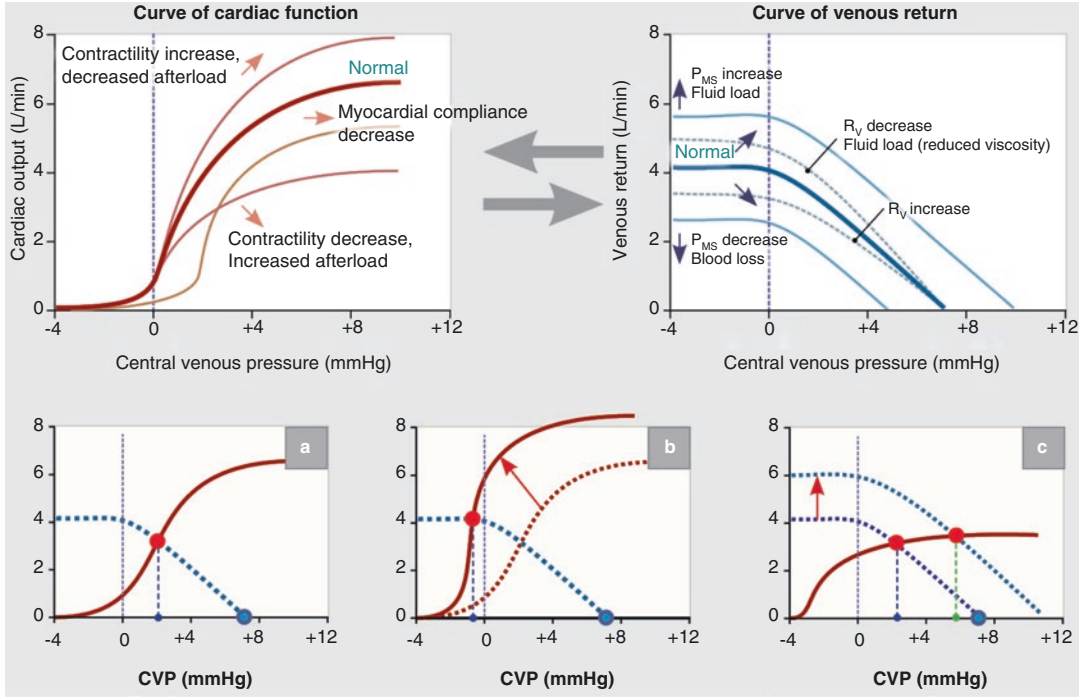

Fig. 3.2 A model of interplay between the venous return and the heart function (contractility). (**a**) An intersection of cardiac function and venous return curves gives resulting values of cardiac output (CO), venous return, and CVP. (**b**) Maximal venous return in low (negative) CVP ("waterfall" phenomenon) at the vertical position when the increase in contractility does not result in further CO increase. (**c**) Venous return curve crosses the plateau of the cardiac function curve; therefore, the augmentation of venous return does not result in further increase in CO. Please note that in accordance with the simultaneous position shift of the curves, the resulting CVP values can both increase and decrease depending on the personalized response to similar intervention (*i.e.,* fluid load, vasopressors). *CVP* central venous pressure, *CO* cardiac output, P_{MS} mean circulatory filling pressure, R_V resistance to the venous return

output—the pumping function of the heart, peripheral resistance to blood flow, and the volume of the circulatory system. Since venous return is equivalent to cardiac output, an increase in the latter can be achieved by increasing the mean systemic pressure and lowering the resistance to venous return or CVP. This is confirmed by the observations of an increase in cardiac output and a simultaneous decrease in CVP during moderate physical activity (Fig. 3.2) [7].

3.5 Interpretation of the Central Venous Pressure

The central venous pressure reflects the ability of the heart to "pump" an inflowing volume of blood and characterizes the filling pressure of the right ventricle. In this term, clinicians often use CVP as an indirect indicator of ventricular preload and intravascular volume.

Under the conditions of normal heart function and adequate intravascular volume, when patient is standing up or in the sitting position, CVP is usually below zero (atmospheric pressure). This can be explained by the "suction" function of the ventricles during diastole [8]. This effect must be considered during surgical interventions performed in the upright position (neurosurgery) due to the risk of air embolism.

Moreover, in most cases, CVP transducer is "calibrated" against relatively constant atmospheric pressure, not taking into account the changes in airway pressure [9]. However, in assessing CVP, one should consider the phase, type, and other characteristics of respiration. During spontaneous inspiration, a decrease in CVP (following a decrease in intrapleural

pressure) and an increase in blood flow into the heart (preload) can be observed because of the "suction" action of the chest. In contrast, inspiration during mechanical ventilation is accompanied by an increase in CVP and, in addition, results in a certain decrease in the volume of the heart due to the restriction of blood flow. In the routine clinical practice, CVP is usually evaluated at the end of expiration that provides the most accurate assessment of transmural pressure. The intrapleural pressure gradually returns to zero (atmospheric pressure) by the end of the passive spontaneous expiration or when the patient is disconnected from the mechanical ventilation and the transmural pressure matches CVP most closely.

Under pathological conditions, the role of CVP is not limited by indicating changes of the intravascular volume only (Fig. 3.3). For example, in the case of increased cardiac output (distributive shock, hyperdynamic state), we can observe reduced CVP despite normo- or hypervolemia. On the contrary, the increased values of CVP can be registered both in true volume overload and in normovolemia when the patient has severe heart dysfunction or pulmonary hypertension (*e.g.,* pulmonary embolism). The dynamic changes in myocardial compliance (*e.g.,* due to the use of various beta-adrenergic drugs) can further complicate these interactions.

3.6 The Current Place of the Central Venous Pressure in Clinical Practice

The mean value of CVP and its dynamic changes have been used for decades as indirect markers of the blood volume inflowing to the heart and, therefore, of ventricle preload. The physiological basis for the use of CVP as a guide for fluid therapy was first introduced in the 1950s by Hughes and Magovern in patients who underwent thoracotomy [10]. Later, the clinical value of CVP as a marker of preload has become a subject of constructive criticism [11, 12]. However, the question about the optimal ventricle preload indicator—volume or pressure—remains unresolved. Since the right and left parts of the heart are functionally combined, when the right ventricle reaches its functional plateau, the ejection of the left ventricle also becomes limited. The recognition of this phenomenon has led to the statement "Success of the left ventricle is impossible without the success of the right ventricle." On this basis, Magder advocates the view that it is unacceptable to use PAOP and the size of the left ventricle to optimize preload [9]. It should be recognized that the left ventricle can eject only the volume of blood that the right one delivers, and, *vice versa*, the right ventricle can dispose only the volume that the left one is able to accept.

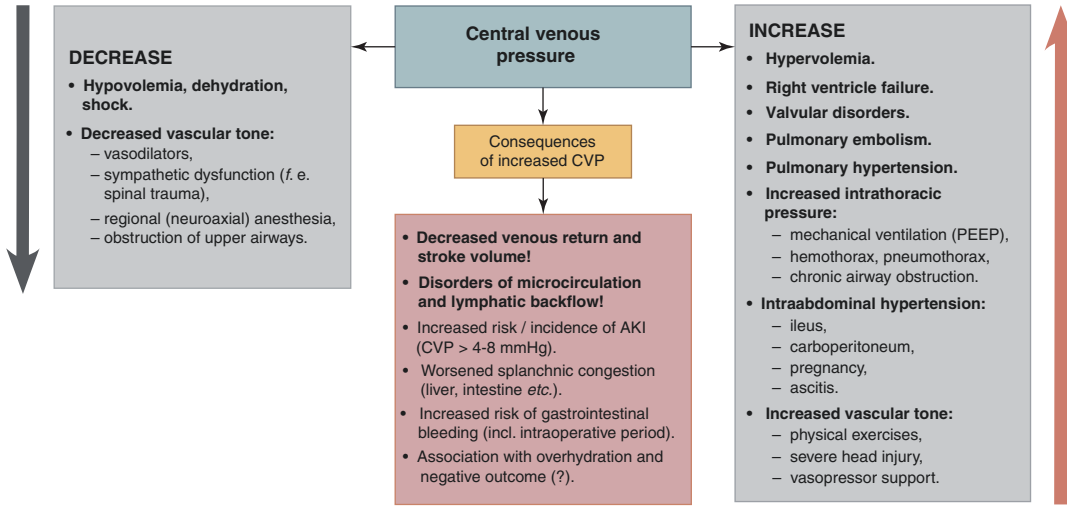

Fig. 3.3 The factors resulting in the changes of the central venous pressure (CVP) and the risks of increased CVP

A number of studies and recent systematic reviews have demonstrated that using the absolute values of CVP to predict fluid responsiveness is unreliable in most critically ill patients [12, 13]. Notably, the prognostic value of CVP regarding the preload of the left ventricle becomes completely unacceptable in patients with intraabdominal hypertension or increased airway pressure (*e.g.,* in COPD). At the same time, the question "How to treat a patient with shock and normal CVP, or, conversely, a stable patient with a low value of this parameter?" remains unresolved [14].

Practical Advice

The central venous pressure is an unreliable predictor of fluid responsiveness. Therefore, the use of CVP for this purpose should be discontinued.

3.7 Risks of Increased Central Venous Pressure and Further Perspectives

Recent studies have shown that liberal (or "aggressive") infusion therapy in critically ill leading to a rise in CVP above 8–12 mmHg is accompanied by increased risk and incidence of acute kidney injury, multiple organ dysfunction, and death [15–18]. As reported, a rise in CVP by 1 mmHg results in an increase of the risk of AKI by almost 2% [19]. It has also been demonstrated that an increase in CVP ≥12 mmHg in patients with sepsis is associated with profound microcirculation disorders [20]. Of note, the *Surviving Sepsis Campaign* no longer targets a central venous pressure of 8–12 mmHg as a goal of fluid resuscitation [21]. Recently, Xing et al. demonstrated the advantages of early renal replacement therapy aiming to reduce CVP in regard to the recovery of renal function in patients with sepsis-induced acute kidney injury [22]. Moreover, fluid de-escalation strategy in patients with ARDS leading to a decrease in CVP was associated with fewer days of mechanical ventilation [23]. Recent meta-analysis demonstrated that the application of different strategies (Trendelenburg position, nitroglycerine, furosemide, fentanyl, control of infusion rate, and clamping the intrahepatic vena cava) aiming at controlled low CVP (or even targeted "zero" CVP) in liver resection significantly reduces blood loss and requirement in blood transfusion [24].

Practical Advice

The central venous pressure has a potential to be considered as one from the parameters of safety of fluid therapy. During fluid load, the physician should be aware of the multiple risks of CVP exceeding 12 mmHg.

Nevertheless, the "optimal" CVP value has not been established yet. It should be personalized and kept as low as possible [25]. In addition, the visual analysis of the CVP curve is still useful in cardiac surgery and may give information about the tricuspid and mitral valve function, the hemodynamic effects of rhythm disturbances, and the presence of constrictive pericarditis and pericardial tamponade [26]. Thus, some authors advocate further studies aiming to evaluate the potential benefits of CVP monitoring [27].

3.8 Conclusion

The measurement of CVP requires knowledge of the methodology and cardiovascular physiology, while the analysis of the wave contour of CVP can help to detect cardiac disturbances. Obviously, CVP cannot be recommended for further use as a reliable predictor of fluid responsiveness. However, monitoring of changes in CVP has a potential to provide important information on the safety of fluid therapy and to detect the risk of acute kidney injury and microcirculatory distress. The future studies should address the association of CVP with overhydration and peripheral tissue edema and answer the question about possible role of this parameter in the personalized algorithms for de-escalation of fluid therapy.

Keynotes

- The central venous pressure (CVP) is a static parameter reflecting preload and function of the right ventricle only; thus, in many clinical situations, CVP does not correlate with the work of the left heart.
- The central venous pressure is unreliable as a predictor of fluid responsiveness.
- In critically ill patients, increased CVP is associated with development of organ dysfunction, especially acute kidney injury and splanchnic congestion.
- In certain clinical scenarios, therapy guided to decrease the elevated values of CVP might attenuate organ dysfunction and has a potential to improve outcome.

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