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REVIEW ARTICLE

Venous System: Evaluation with Central Venous Pressure – Circulatory and Systemic Filling Pressures

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ABSTRACT

Effective hemodynamic management is fundamental in anesthesia practice to ensure adequate organ perfusion and stability of organ function during and after major surgical procedures or in critically ill patients. The advent of advanced monitoring techniques has expanded the understanding of arterial hemodynamic parameters, enhancing decision-making in fluid management and the use of vasoactive agents. However, similar advancements in understanding the venous system—comprising 70% of the total blood volume—remain limited. The venous system's compliance, characterized by its unstressed and stressed volumes, plays a critical role in determining venous return and cardiac output. Central venous pressure (CVP), a routine clinical parameter, provides insights into venous return but has limitations when used in isolation. Advanced concepts such as mean systemic filling pressure (Pmsf) offer a more comprehensive understanding of venous hemodynamics. Techniques like Pmsf(hold), involving inspiratory hold maneuvers, and Pmsf(analog), based on mathematical modeling, have been developed to measure Pmsf in clinical settings. These approaches highlight the dynamic interplay between venous return, stressed volume, and right atrial pressure under various clinical conditions, including hypovolemia, cardiogenic shock, and septic shock. Recent studies suggest that combining CVP analysis with Pmsf evaluations improves fluid management and individualized hemodynamic control. However, clinical application of these measurements remains challenging, necessitating further validation through experimental and clinical studies. This review underscores the importance of an integrated approach to venous and arterial systems in optimizing hemodynamic management, paving the way for more precise and evidence-based patient care.

Keywords: Central venous pressure, fluid responsiveness, hemodynamic management, mean systemic filling pressure, venous return

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Introduction

Effective hemodynamic management is crucial for anesthesiologists in order to achieve adequate organ perfusion in patients both during and after surgery, hence preserving the stability of organ function. In major surgical procedures such as cardiovascular surgery or in the management of critically ill patients, hemodynamics can change rapidly, requiring a number of monitoring tools for anesthesiologists to manage this challenging process.

Aside from the basic hemodynamic parameters now employed in our anesthesiology practice, the advancement of monitoring techniques has facilitated the utilization of advanced hemodynamic parameters to enhance our decision-making algorithms.

The primary components of the cardiovascular system are clearly the left ventricle and the aortic system. The evaluation of cardiac functions is based on factors such as preload, afterload, and contractility. However, compliance, elastance, resistance, or impedance—which are broad expressions of these parameters—play a major role in determining the features of the arterial system within this paradigm. Minimally invasive hemodynamic monitoring, a technique that has gained popularity in recent years, utilizes

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pulse contour analysis to provide advanced hemodynamic data related to the arterial system. Consequently, the hemodynamic management of critically ill patients, particularly their fluid management and hemodynamic control with vasoactive medications, can be carried out in a more individualized and evidence-based manner rather than following traditional approaches.

However, similar advancements are not observed for the venous system, which comprises approximately 70% of the total blood volume. The venous system's compliance, being 24 times greater than that of the arterial system, can be acknowledged as a fundamental challenge in establishing a behavioral model for the venous system compared to the arterial system. Nevertheless, it should be well recognized that the assessment of circulatory and systemic filling pressures, including central venous pressure, pertaining to the venous system, will provide invaluable insights for anesthesiologists in fluid management and hemodynamics.

"a strong heart that is filled with blood empties itself more or less completely, in other words, changes the extent of the contractile power."

The relationship between diastolic volume and cardiac contractility was first described in the literature with this expression by Ludwig in 1861.[1] In 1914, Starling, in an experimental model utilizing anesthetized dogs, directed blood from an ascending aortic cannulation into a venous reservoir, thereby collecting it and directing it to the right atrium.[2] By controlling this flow with a limiter, he demonstrated the effect of preload changes on cardiac contractility and stroke volume. While an increase in preload initially augmented stroke volume up to a certain point, as per Starling's proposition, the heart seemed to tire beyond a certain threshold. At this juncture, despite a rapid increase in right atrial pressure, stroke volume no longer increased. Thus, the Starling curves were established, enhancing our understanding of the connections between preload and stroke volume. The contemporary version that we currently use has been modified by Guyton.[3]

Central venous pressure (CVP), reflecting the right atrial hydrostatic pressure, stands as a fundamental hemodynamic parameter of the venous system that enables us to comment on venous return in clinical practice. While its normal range is between 8–12 mmHg, monitoring its temporal changes beyond numerical values holds significant importance in our fluid management. Central venous pressure is affected by blood volume and sympathetic tone. Additionally, it is imperative to bear in mind that elevated CVP values are always pathological, and their presence warrants elucidation and further investigation.

Wave analysis, as in many other monitoring techniques, is crucial for validating data in the assessment of central venous pressure (CVP). On the other hand, alterations in CVP waves may signify certain pathological conditions within the right side of the heart. The 'a' wave, formed with atrial contraction, is lost in atrial fibrillation. In contrast, junctional rhythms, ventricular pacing, and atrioventricular complete block result in the simultaneous contraction of the atrium and ventricle, leading to giant 'a' waves. Additionally, tricuspid stenosis gives rise to a prominent 'a' wave, while tricuspid regurgitation is associated with a prominent 'v' wave and a large 'v-c' wave. In constrictive pericarditis, the accentuation of tricuspid annular contraction deepens the 'x' descent, and rapid ventricular early filling enhances the 'y' descent. Consequently, a prominent 'x-y' descent is observed in the wave analysis of central venous pressure. In cardiac tamponade, the loss of the 'y' wave occurs due to right ventricular restriction.

Undoubtedly, the significance of our knowledge regarding central venous pressure and wave analysis is undeniable. However, relying just on CVP and wave analysis to assess the venous system may be insufficient, particularly when it comes to the hemodynamics and fluid management of critically ill patients.

What Is the Determinant of Cardiac Output?

To comprehend the limitations inherent in the isolated utilization of central venous pressure in the assessment of the venous system, the paramount inquiry necessitates an understanding of the determinants of cardiac output. Cardiac output is defined by the venous return to the heart through the venous system—that is, the venous return—and the factors that prevent this return.

Although the venous system has high compliance, it is not infinite. As in the example of a balloon, which contains some air even when not actively inflated, preventing complete deflation, the venous system maintains a certain volume of blood that keeps the vascular bed open to prevent collapse of the vascular bed. This volume, which keeps the vessel open, is referred to as unstressed volume. On the other hand, if the balloon is further inflated, the elastic tension in its walls causes air to escape outward. Similarly, in the venous system, exceeding a certain volume generates elastic tension in the vessel wall, facilitating the flow of blood toward the right atrium; this volume is termed stressed volume (Fig. 1).[4]

Magder explained the venous system with a bathtub model.[5] The faucet of the bathtub symbolizes cardiac output, while the drain at the very bottom represents venous return. However, Magder diverges by defining the drain of the bathtub not at the bottom but rather in the middle. In this

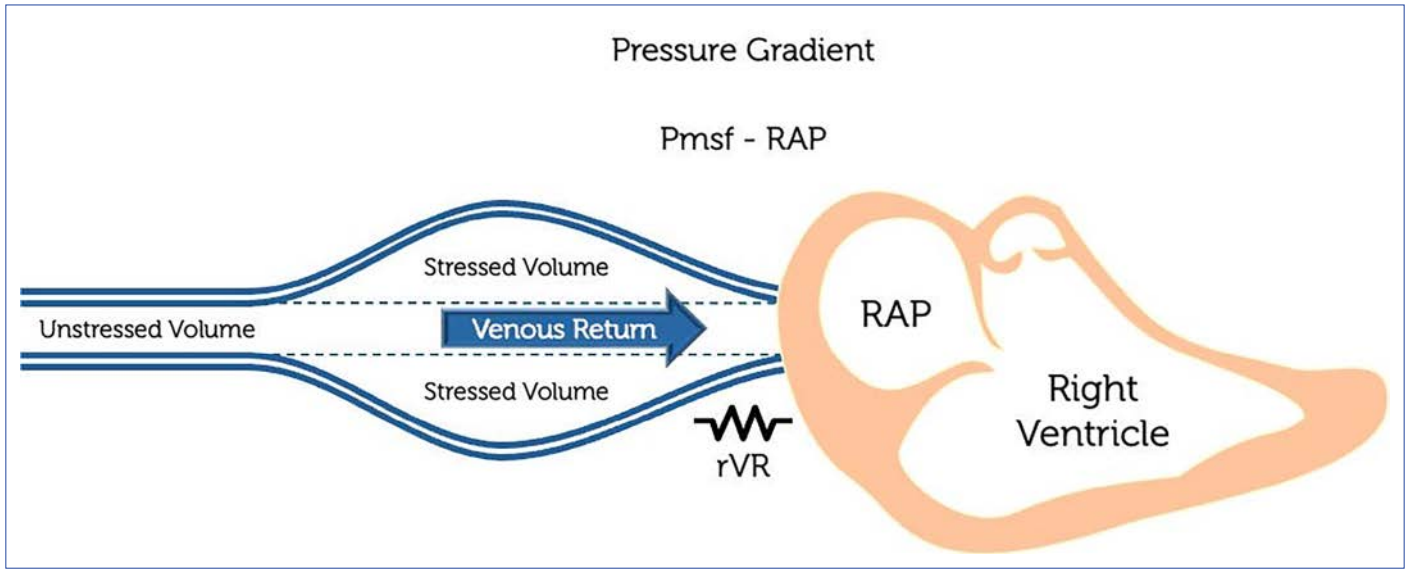

Figure 1. Regulators of venous return. Venous return is determined by the pressure gradient between the Pmsf, generated by the stressed volume, and the RAP. The unstressed volume does not contribute to venous return, whereas the stressed volume creates the driving force for blood flow towards the right ventricle. The resistance to venous return (rVR) modulates this process.

Pmsf: Mean systemic filling pressure; RAP: Right atrium pressure.

manner, the portion below the drain retains the filled volume of the bathtub (unstressed volume), while the volumes above it, due to gravitational flow, facilitate venous return (stressed volume). Consequently, the stressed volume was measured by performing hypothermic circulatory arrest at 19°C in five patients undergoing thoracic arch and vena cava resection due to renal malignancy. Venous cannula was fully opened into a pre-marked reservoir to complete venous return, and the stressed volume was determined to be 1290±296 ml (20.2±1.0 ml/kg). The fundamental limitations of this method include its application under hypothermia and its clinical impracticality, making it challenging to measure and apply.

The pressure created by the stressed volume, which ensures the venous return of blood to the right atrium, is defined as filling pressure. Traces of the concept of filling pressure in the literature date back to 1894, with Starling mentioning "mean general blood pressure".[6] Starling proposed that, in the circulatory system, when the heart stops and the motion generated by the heart ceases, the pressures measured from any point in the circulatory system would eventually equalize. Subsequently, Guyton, through his experimental models, proposed the definition of "mean circulatory filling pressure".[7]

Is It Possible to Measure Filling Pressures in Clinical Settings?

The initial clinical measurements of circulatory filling pressures, other than experimental models, are grounded in cardiologists' electrophysiological studies. In individuals where ventricular fibrillation was induced through a pacemaker, it was demonstrated that central venous pressure and mean arterial pressure converged at a common value in approximately 7 seconds.[8] Moreover, in patients expected to experience cardiac arrest in intensive care units, it has been demonstrated that pressures stabilized at a single point for a brief period following arrest, similar to the time frames observed in other studies.[9] However, like Magder's stressed volume measurements, circulatory filling pressure measurements were not applicable.

The mean systemic filling pressure (Pmsf) is a pressure distinct from circulatory filling pressures, as it represents the equilibrium of arterial and venous system pressures at any location outside the heart and pulmonary circulation. Theoretically interchangeable, in clinical practice, methods for measuring systemic filling pressure are considerably more practicable. There are two basic methodologies defined in literature.

The first among these methodologies is the Pmsf(hold) technique, introduced by Mass et al.[10] in 2009. This approach involves the calculation of Pmsf in the intensive care setting post-cardiac surgery, particularly among intubated and mechanically ventilated patients. Notably, this technique allows for the assessment of cardiac and pulmonary interactions without the necessity of inducing cardiac arrest. In this technique, sequential inspiratory holds of 12 seconds each are applied at airway pressures of 5, 15, 25, and 35 cmH_2O, allowing simultaneous measurements of central venous pressure and cardiac output. Data points are obtained by conducting identical measurements for each

pressure level, and the data are plotted on a graph with central venous pressure on the x-axis and cardiac output on the y-axis. Extrapolating linear regression through these points towards the x-axis at zero flow (x(CO)=0) defines the pressure at zero flow as the mean systemic filling pressure.

Research has demonstrated that the utilization of fluids including crystalloids and colloids leads to an increase in the Pmsf(hold) value.[11,12] Furthermore, research conducted on noradrenaline and propofol dose titrations has demonstrated the sensitive responsiveness of the Pmsf(hold) value.[13,14] Pmsf(hold) measurements in the literature exhibit a range of 19–33 mmHg.[11,15]

Nevertheless, this approach is subject to some limitations. Initially, it necessitates the use of mechanical ventilation and central venous catheterization for measuring purposes. In addition, elevated airway pressures might lead to hemodynamic instability.

The calculation of the mean systemic filling pressure can be achieved using a mathematical model known as the Guytonian model—Pmsf(analog)—which was developed by Parkin and Leaning.[16] In this mathematical model, the right atrial pressure, mean arterial pressure, and cardiac output must be known. Separate resistance constants are assigned to these three parameters: 'a' for right atrial pressure, 'b' for mean arterial pressure, and 'c' for cardiac output. 'a' and 'b' are dimensionless variables, and their sum is equal to '1'. The constant 'c' requires patient-specific information such as height, weight, and age.

This approach has some limitations, such as the requirement for central venous catheterization and the limited validity of calculations based on patients' anthropometric measurements, particularly in critically ill individuals.

In the literature, Pmsf(analog) values vary within the range of 14–21 mmHg.[17,18] Studies have shown that Pmsf(hold) values exhibit a precise increase in fluid applications involving crystalloids and colloids.[19,20]

In the year 2022, Meisj demonstrated a high degree of compatibility between the mean systemic filling pressures calculated using these two methods, employing a correlation coefficient of 0.89.[21]

Venous return is determined by the disparity between the systemic filling pressure, generated by stressed volume to aid blood movement towards the right atrium, and the right atrial pressure that needs to be overcome. Optimal venous return occurs when filling pressure is high and right atrial pressure is low (up to but not exceeding a value of 0). Conversely, venous return is compromised when filling pressure is low (due to low stressed volume, hypovolemia, or vasodilation) and right atrial pressure is high (indicative of heart failure).

The Combined Assessment of Mean Systemic Filling Pressure and Central Venous Pressure in Different Clinical Scenarios

In hypovolemia, the decrease in total blood volume will further diminish stressed volume, leading to a reduction in mean systemic filling pressure. The decrease in the gradient between right atrial pressure and mean systemic filling pressure results in a diminished venous return.[10]

If fluid replacement is administered to this patient, and the patient is in the fluid-responsive range of the Starling curve, there will be an increase in the gradient between systemic filling pressure and right atrial pressure, resulting in an increase in venous return. Nevertheless, when administering fluid replacement to a patient who does not respond to preload, the right atrial pressure will increase along with the filling pressure. Consequently, as the gradient does not increase, venous return remains unchanged, but elevated central venous pressures are measured.[22]

In cardiogenic shock, while the mean systemic filling pressure remains unchanged, the right atrial pressure increases, leading to a reduced gradient and decreased venous return. Consequently, the Starling curve is restructured for lower cardiac output and higher central venous pressures.[23]

In septic shock, widespread vasodilation reduces the stressed volume, leading to a decrease in mean filling pressures. However, since right atrial pressures also decrease, venous return is maintained. If myocardial damage occurs, right atrial pressures increase, resulting in reduced venous return, similar to the scenario in cardiogenic shock.[24,25]

On the other hand, a study conducted by Adda in 2021 on patients with septic shock revealed that fluid administration following the augmentation of the stressed volume compartment through norepinephrine application resulted in higher filling pressures.[26]

In hypovolemia, low Pmsf accurately signals reduced venous return, yet CVP's inability to distinguish between preload responsiveness and overfilling complicates fluid titration. Similarly, in cardiogenic shock, elevated CVP masks underlying Pmsf stability, risking misdiagnosis of fluid status. In septic shock, vasodilation lowers Pmsf, but CVP's variability hinders precise management. These limitations underscore the need for integrated approaches: combining Pmsf with echocardiography or dynamic indices (e.g., pulse pressure variation) could enhance accuracy. Clinically, we recommend initial Pmsf assessment to guide fluid boluses, followed by CVP trend monitoring to avoid over-resuscitation.

Recent clinical investigations have further highlighted the clinical relevance of mean systemic filling pressure (Pmsf) as a dynamic marker of effective circulatory volume and venous return. In a multicenter study involving patients with acute circulatory failure, Mallat et al.[27] demonstrated that while Pmsf increased after both fluid expansion and passive leg raising, only fluid-responsive patients exhibited a significant rise in venous return pressure gradient (Pmsf–CVP) and a reduction in resistance to venous return, in line with Guyton's physiology. This finding reinforces the concept that interpreting absolute values of Pmsf is insufficient; the interplay between Pmsf and CVP must be considered to assess preload responsiveness. Similarly, Hahn et al.[28] showed that Pmsf is lower in fluid responders and that general anesthesia itself increases unstressed volume significantly (up to 1.2 L), requiring careful fluid titration to avoid hemodynamic instability in the perioperative period.

Additionally, Chen and Du evaluated Pmsf in patients with constrictive pericarditis post-pericardial stripping and found that elevated Pmsf values at 24 hours were independently associated with 28-day mortality, even outperforming traditional markers like CVP in prognostic models.[29] Meanwhile, a prospective observational study in liver transplant recipients showed that MSFP significantly declined during the anhepatic phase, with paradoxical negative correlation to cardiac output despite strong positive correlation with CVP—further emphasizing the complexity of venous return physiology in different surgical contexts.[30] Together, these studies support the notion that Pmsf provides valuable insight beyond static preload markers and may enhance precision hemodynamic management when integrated with clinical context and dynamic assessments.

From a clinical perspective, the integration of mean systemic filling pressure (Pmsf) into hemodynamic assessment provides a more physiologically grounded and individualized approach to volume management. Rather than relying solely on central venous pressure (CVP), which often fails to distinguish between preload responsiveness and volume overload, evaluating the Pmsf–CVP gradient allows clinicians to infer the driving force of venous return and thus optimize cardiac output. For instance, a high Pmsf with a narrow Pmsf–CVP gradient may indicate venous congestion with impaired forward flow, suggesting the need for vasodilators or inotropes rather than fluids. Conversely, a low Pmsf with preserved gradient may reflect hypovolemia and guide fluid resuscitation. Clinical studies have demonstrated that dynamic changes in Pmsf following passive leg raising or volume expansion strongly correlate with fluid responsiveness, making it a valuable adjunct in critically ill or perioperative patients. Therefore, combining Pmsf with CVP trend monitoring and functional indices (e.g., cardiac output or pulse pressure variation) can support a stepwise, physiology-based decision algorithm in hemodynamic management, potentially reducing the risks of fluid overload and improving end-organ perfusion.

In conclusion, when evaluating the vascular system, it is essential to carefully assess the venous system with its unique characteristics, in addition to the arterial system. Through the use of central venous pressure measurements and wave analysis, which are part of our routine clinical practice, alongside the evaluation of mean systemic filling pressure, we can achieve a better understanding of venous return. In recent years, studies focusing on the calculation of mean systemic filling pressure have increasingly attracted attention in the literature. However, it is evident that further clinical and experimental studies are needed to validate or identify the limitations of measurements performed under different clinical conditions and using various methodologies.

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