Author/s:
Lloyd-Donald, P;Fujino, M;Waldman, B;Miles, LF

Title:
Measurement and interpretation of central venous pressure: a narrative review

Date:
2025-09-01

Citation:
Lloyd-Donald, P., Fujino, M., Waldman, B. & Miles, L. F. (2025). Measurement and interpretation of central venous pressure: a narrative review. Anaesthesia, 80 (9), pp.1093-1102. https://doi.org/10.1111/anae.16633.

Persistent Link:
https://hdl.handle.net/11343/362728

Anaesthesia 2025, 80, 1093–1102 doi:10.1111/anae.16633

Core Topics

Measurement and interpretation of central venous pressure: a narrative review

Patryck Lloyd-Donald, Midori Fujino, Boris Waldman and Lachlan F. Miles

Summary

Introduction Central venous pressure has been a key component of haemodynamic monitoring for several decades, but its clinical utility is still debated. While central venous pressure has been used traditionally to assess intravascular volume status and fluid responsiveness, mounting evidence suggests that its absolute value alone is an unreliable predictor of such states. This narrative review explores the historical development and physiological foundations of central venous pressure, its measurement techniques and the available evidence regarding central venous pressure monitoring in various clinical scenarios.

Methods We conducted a literature search to identify relevant articles. The abstracts of identified articles were assessed for relevance, and their references were screened for further relevant publications.

Results We identified 229 articles, with 72 undergoing full text extraction and data synthesis. Preliminary work in central venous pressure monitoring began in canines, before early human studies and the development of Guytonian principles of haemodynamics. Physiologically, central venous pressure reflects intraluminal pressure of the superior vena cava, with measurement affected significantly by transducer position, catheter position and intrathoracic pressures. In right ventricular dysfunction, the central venous pressure waveform loses its x-descent, as the y-descent becomes exaggerated. Central venous pressure in isolation is considered a poor predictor of volume responsiveness; however, elevated central venous pressure is linked with impaired tissue perfusion and poor clinical outcomes, including acute kidney injury and higher risk of mortality in certain patient cohorts.

Discussion Although reliance on central venous pressure as a singular guide for fluid resuscitation is discouraged, it remains a valuable tool when interpreted in conjunction with waveform analysis and trend monitoring, offering insights into right heart function, venous congestion and organ perfusion. As patient cohorts grow increasingly complex, a nuanced understanding of central venous pressure waveforms and trends is essential for optimising management strategies in peri-operative and critical care settings.

For full author affiliations, see end of article
Correspondence to: Lachlan F. Miles
Email: lachlan.miles@unimelb.edu.au
Accepted: 3 April 2025
Keywords: central venous catheter; central venous pressure; haemodynamic monitoring; right atrial pressure
X: @Austin_Health; @CritCareUniMelb; @LachieMiles

Introduction

Central venous pressure (CVP) is an often controversial physiological parameter in critical care medicine. Central venous pressure measurement has been integrated into clinical practice for > 70 years, from preliminary studies in animal models [1], to promotion as a marker of volume status and a resuscitation target [2], and subsequently contributing to advanced haemodynamic

monitoring [3] to guide treatment options for patients who are critically ill.

The ongoing utility of CVP measurement is still subject to debate [4, 5]. Current evidence suggests CVP does not predict volume responsiveness across a range of patient cohorts [6, 7], leading some commentators to move away from monitoring the parameter altogether [4]. However, the clinical utility of targeting specific CVP values, both in isolation and as part of a trend, remains uncertain in different surgical and non-surgical populations, as does the mechanism by which these targets may influence clinical outcomes.

A comprehensive review of this topic was performed by Eskesen et al., who re-examined datasets from over 1000 patients, concluding that predictive values of *"all specific CVP values assessed"* were low for fluid responsiveness, with the exception of very high or very low values [6]. Building further upon this, our review focuses both on fluid responsiveness and subsequent new developments, including non-invasive measurement techniques utilising ultrasonography, and the role of CVP as a critical determinant of organ perfusion.

We address the historical background of CVP monitoring; principles of measurement; physiological underpinnings; how the parameter is influenced in certain pathophysiological states; and examine the evidence behind its use in clinical medicine, including new and emerging areas.

Methods

We conducted a literature search (MEDLINE and Embase) on 1 October 2024 to identify relevant articles. Key search terms included: 'central venous pressure'; 'CVP'; 'venous pressure monitoring'; 'right atrial pressure'; 'jugular venous pressure'; and 'venous filling pressures'. A date limit of 1960 onwards was prespecified. The abstracts of identified articles were assessed for relevance, along with screening of their references for further relevant publications. A review of 229 articles was undertaken by three authors (PLD, MF and BW), with another author (LM) used to resolve conflicts; 72 articles underwent full text screening.

Results

Early concepts in central venous pressure measurement

The first reported CVP measurement occurred in 1733, using glass tubing to access the jugular vein of an injured horse [8]. Experiments in canines – showing a closed circulation and the relationship between arterial, capillary and venous pressure – were first described by Bayliss and Starling in 1894 [9]. Further canine experiments lead to the development of the Frank-Starling 'law of the heart', wherein ex vivo heart preparations showed a *"direct connection between venous pressure, diastolic filling and output"* until the heart began to fail [10]. Starling's own experiments did not generally treat preload as an independent variable; however, reproductions of the classic Frank-Starling curve usually placed preload on the x-axis, with subsequent physiology texts inferring that CVP was equivalent to ventricular preload [10–12] and implying that CVP could be used as a marker of intravascular filling and volume responsiveness. Further canine studies in the 1920s described the transient elevation and subsequent fall of CVP in response to fluid bolus administration [1]. The first human central venous catheterisation was performed in 1905 via the antecubital fossa by Bliechröder but this was not formally reported until 1929 after self-catheterisation was performed by Forssmann [8, 13].

Technological advancement meant that by the mid-20th century, measurement and interrogation of CVP and right heart catheterisation were feasible and widespread. This enabled the clinician to calculate cardiac output and characterise cardiac and pulmonary pathology [14]. Measurement of CVP in clinical medicine and its application to disease states became an area of interest in the 1950s, using cannulation of the internal jugular vein and a water manometer [11]. Preliminary volunteer studies documented a temporary rise in CVP with fluid infusion and a fall with exsanguination [15]. Thereafter, interest in CVP monitoring, particularly as a marker of fluid responsiveness, expanded further, even as contradictory evidence had emerged by the 1970s.

By 1973, pioneering work by Arthur Guyton advanced the clinical understanding of haemodynamics, filling pressures and ultimately CVP. Contrary to received wisdom, Guyton cautioned against relying on CVP as a marker of fluid responsiveness and promoted a model that incorporated physiological forces that contribute to venous return to describe the circulation [16]. This gave physicians a more holistic model on which to interpret changes in the circulation and reduced reliance on the importance of a single variable [12]. Guyton's model depicted CVP as being influenced by both upstream and downstream pressures, with cardiac output represented by the intersection of a cardiac function curve and a venous return curve. The cardiac function curve has similarities with the Starling curve, whereby increasing right atrial pressure leads to increasing cardiac output. In contrast, the venous return curve is defined by the equation:

$$\text{Venous return} = \frac{\text{Mean systemic filling pressure} - \text{right atrial pressure}}{\text{Resistance to venous return}}$$

Therefore, increasing right atrial pressure reduces venous return and cardiac output. Furthermore, infusing fluid will only increase venous return if it leads to an increase in mean systemic filling pressure greater than right atrial pressure, or decreases resistance to venous return through recruitment of collapsed abdominal veins [16] (Fig. 1).

Measurement and monitoring

The gold standard for measuring CVP is via a central venous catheter or pulmonary artery catheter as it transitions through the right atrium [17]. While techniques for non-invasive measurement of CVP exist (i.e. using ultrasound to interrogate various central veins), the specifics of these methods and their validity are beyond the scope of this article. Common insertion sites for a central venous catheter are via the internal jugular or subclavian vein with the tip sitting in or close to the junction of the superior vena cava and the right atrium. A central catheter inserted peripherally can also measure static and dynamic CVP values accurately [18].

When CVP is measured from a point below the diaphragm (i.e. via the femoral vein), factors influencing intra-abdominal pressure must be considered [19]. In the absence of obstruction or compression between a catheter tip in the inferior vena cava and the right atrium, the inferior vena cava measurement is concordant within 0.23 mmHg of readings taken from the superior vena cava [20]. However, in the presence of altered abdominal pressure or obstruction, thrombosis or other disruption of the inferior vena cava, sub-diaphragmatic measurement of CVP becomes less accurate and reflects true values poorly [21].

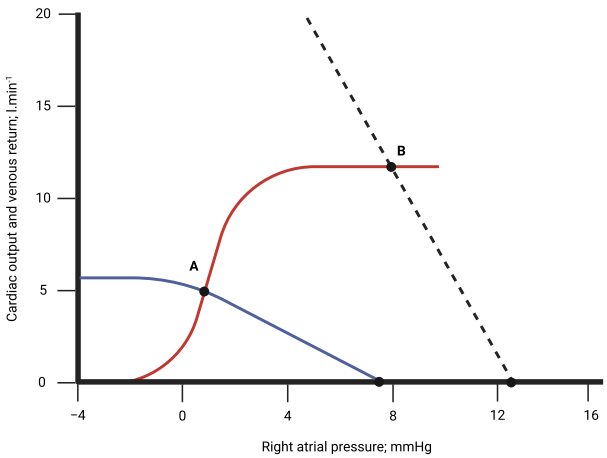

Figure 1 Guyton's combination of two curves to model the circulation. Red line, cardiac performance curve; blue line, venous return curve. The intersection of the two (A) determines the cardiac output. Where venous return is higher (black dashed line), the cardiac output similarly increases (B).

To ensure transduced values reflect desired physiology accurately, the clinician must ensure that the pressure transducer is correctly positioned and zeroed to atmospheric pressure [17]. As CVP has the lowest normal absolute value of the commonly transduced intravascular pressures, it is the most sensitive to discrepancies between the point of measurement and the height of the pressure transducer. Classically, CVP is measured in line with the tricuspid valve; externally, this equates to 5 cm ventral to the sternal angle or one-third of the anteroposterior diameter of the chest [22]. This reference point is accurate whether the patient is supine or sitting up as the right atrium is an anterior structure. This is more accurate than measuring at the midpoint of the chest, which is only accurate in supine patients [22]. In contrast, aligning the transducer incorrectly with the sternal angle corresponds to a 10 cm relative movement of the transducer when moving from supine to sitting, which is equivalent to a 7.6 mmHg measurement error [23].

Pressure transducers are zeroed to atmospheric pressure via a three-way tap, and repeated periodically to account for cable faults or baseline drift [17]. Given the various pieces of equipment used to monitor CVP, and to account for the natural frequency and damping coefficient, a fast-flush test to elicit a square waveform measures the dynamic response of the system to assess over under- or overdamping [24].

In contemporary practice, CVP waveforms and values are displayed digitally; however, this is also an inherent source of error. First, cardiac mechanical events (and thus changes in the pressure waveform) follow the corresponding electrical events with a delay of 0.2 s [25]. Second, the concept of a CVP 'value' is abstract, derived using various filtering techniques that incorporate peak and trough values; the difference between the instantaneous CVP value and the derived value can be up to 4 mmHg in patients whose lungs are ventilated spontaneously or mechanically [26]. Measurement timed to right ventricular end diastolic pressure, or when the tricuspid valve closes, provides the most accurate indication of cardiac preload [27].

The most accurate methods for obtaining a CVP numerical value involve: reading a waveform directly; or freezing the bedside monitor at the base of the c-wave at end expiration over three breaths (where pleural pressure is

closest to atmospheric in patients whose lungs are both spontaneous and mechanically ventilated, thereby reducing the effects of intrathoracic pressure) [25–28]. However, if there is an elevated end expiratory pressure from obstruction or bronchospasm, then using this method results in a falsely elevated CVP [25].

The mean (SD) difference between mean CVP and CVP at end diastole during expiration in patients who are paralysed and receiving mechanical ventilation is 0.58 (0.81) mmHg [29]. Despite end diastolic CVP being slightly higher than mean CVP, this difference is not clinically significant. Overall, the difference between the 'reference' CVP and the displayed value using contemporary digital monitoring is within 1 mmHg of agreement [28].

Physiological underpinnings of CVP

Central venous pressure refers to the pressure measured within any central intrathoracic vein and is generally taken to mean the superior vena cava. The term is used interchangeably with right atrial pressure; in the supine position there is little pressure difference between the intrathoracic central veins and the right atrium [17]. Central venous pressure is a fundamental physiological variable with several key determinants and is generated by the interaction of: upstream pressure, which in turn depends on blood volume and vascular capacitance; downstream pressure, specifically the resistance to right atrial outflow caused by pressure transmitted from the right heart, which in turn depends on right ventricular diastolic compliance and the pressures within the pulmonary vasculature; and the lateral pressures, specifically intrathoracic pressures and intrapericardial pressures, which are most influenced by mechanical ventilation [30].

The relationship between total blood volume and mean systemic filling pressure is not linear [31]. Furthermore, 70% of blood is contained in the venous system and, of that, 25% is 'stressed' at baseline (i.e. held in veins with a transmural pressure above zero). The remaining 75% of venous volume is unstressed (i.e. held in veins with a transmural pressure of near zero). The stressed portion contributes to mean systemic filling pressure, which in turn drives venous return. Volume can be redistributed between 'unstressed' and 'stressed' states with venoconstriction [31].

Central venous pressure and right atrial diastolic pressure are not entirely interchangeable; CVP is a measure of intravascular pressure alone, whereas the true right atrial diastolic pressure is a composite of the intravascular pressure minus intrathoracic pressure [27]. Furthermore, the effects of such lateral pressures were not part of Starling's original model, due to the lungs of the experimental subjects being ventilated mechanically with a thoracotomy, which reduced the transmural pressure to zero [10]. Factors which alter intrathoracic pressure will also alter the measurement of CVP, in addition to any factors affecting right ventricular compliance, cardiac rhythm and blood volume [27] (Fig. 2).

The CVP waveform is generated by transmitted intraluminal pressure, plotted over time and varying with changes in the cardiac cycle and respiration. A typical CVP wave is split into a-, c- and v-waves with x- and y-descents interspersed between them. In health, the a-wave is generated by the contraction of the atrium at the end of diastole and the c-wave is caused by the bulging of the tricuspid valve into the atrium during isovolumetric right ventricular contraction. The x-descent represents the emptying of the right ventricle during which the right atrium is distracted downward, increasing the chamber volume and decreasing pressure. The subsequent v-wave is caused by atrial filling against a closed tricuspid valve and the y-descent is caused by the opening of the tricuspid valve at the onset of diastole [16, 17] (Fig. 3).

The mean CVP is typically 2–3 mmHg in healthy patients who are awake [17]. In individuals who are positioned upright, CVP ranges from below atmospheric to as high as 4 mmHg during exertion [32]. As CVP provides an estimation of right atrial pressure, it can also be a surrogate for right ventricular diastolic pressure [27].

Central venous pressure in pathological states

The characteristic descents and waves of the CVP waveform (representing atrial and ventricular pressures during specific phases of the cardiac cycle) undergo distinct changes in the presence of certain pathologies. As such, CVP waveform analysis offers a dynamic view of right heart haemodynamics, with pathological changes in waveform morphology offering more detailed and nuanced information than an isolated mean CVP value [17, 32].

In severe tricuspid regurgitation, the v-wave becomes exaggerated and merges with the c-wave resulting in a large 'c-v wave', also known as the 'Lancisi sign'. This reflects elevated right atrial pressure during right ventricular systole due to regurgitant volume ejected from the incompetent tricuspid valve. While c-v waves are not specific for severe tricuspid regurgitation, they are highly suggestive, and may supplement echocardiographic findings [33]. In clinical situations where the c-wave is absent (i.e. children with distensible atria), either the average pressure during the a-wave or the pressure corresponding with the end of

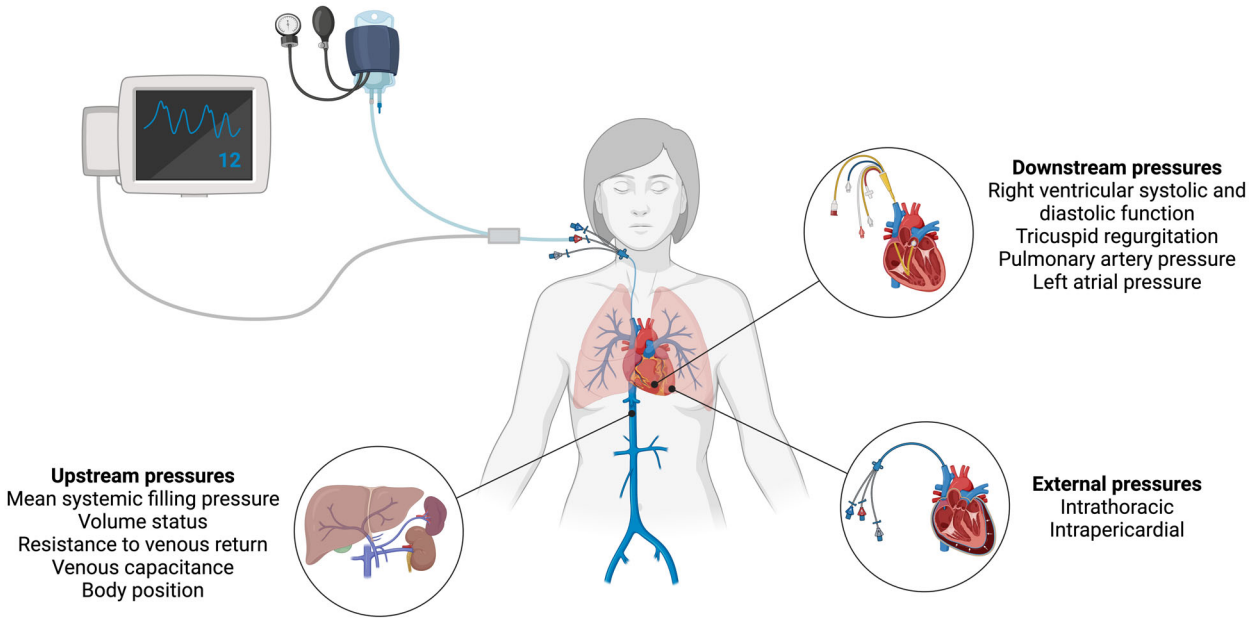

Figure 2 Interplay of factors affecting central venous pressure.

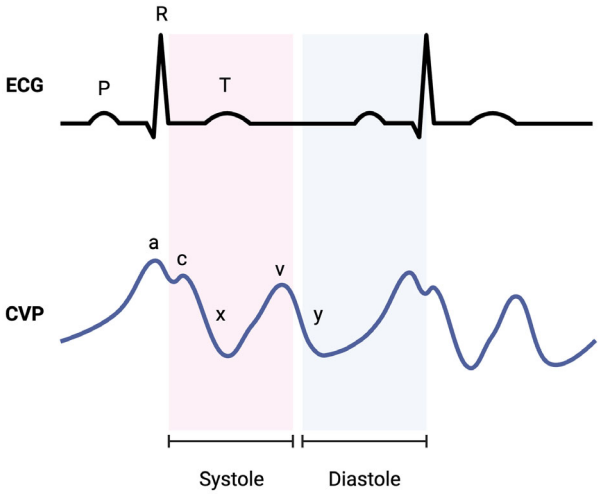

Figure 3 Mechanical components of the central venous pressure waveform presented against the electrical events of the ECG. Systole (red field) includes the c-wave, x-descent and v-wave; diastole (blue field) includes the a-wave and the y-descent.

the QRS complex should be used to ensure greatest accuracy [27].

Worsening right ventricular diastolic dysfunction is associated with changes in the CVP waveform, affecting particularly the x- and y-descents. Early right ventricular diastolic dysfunction is associated with an attenuated or absent x-descent due to impaired atrial relaxation, and a simultaneously exaggerated y-descent, secondary to elevated right atrial pressure in early diastole in order to provide a pressure gradient for flow into a poorly distensible right ventricle, which falls rapidly when the tricuspid valve opens [34]. An exaggerated y-descent is not specific for impaired right ventricular lusitropy per se, but is a generic indicator of elevated right atrial pressure secondary to any cause of poor right ventricular compliance, which may also be observed in other conditions such as constrictive pericarditis [35] (Fig. 4).

Central venous pressure has been used traditionally as a surrogate of right atrial pressure, and therefore a marker of cardiac preload but its use to guide fluid therapy is controversial. The 1960s saw increased interest in CVP monitoring and the emergence of case reports and case series, which supported the use of CVP as a marker of peri-operative fluid resuscitation [36]. Conversely, a 1966 study investigated a population of over 100 patients undergoing major cancer resection, concluding that CVP was not a useful measure of volume status, nor as a blood loss indicator, unless haemorrhage was rapid [30]. The authors instead observed that CVP served as an indicator of myocardial competence and right ventricular diastolic filling pressures, concluding that CVP *"had little correlation with blood loss and was instead useful for judging the heart's ability to tolerate rapid fluid replacement"* [30].

This conflicting evidence culminated in a 1971 editorial concluding: *"there is, in fact, ample evidence that changes in blood volume may be reflected in changes of CVP and, that fluid replacement based on CVP measurement may be*

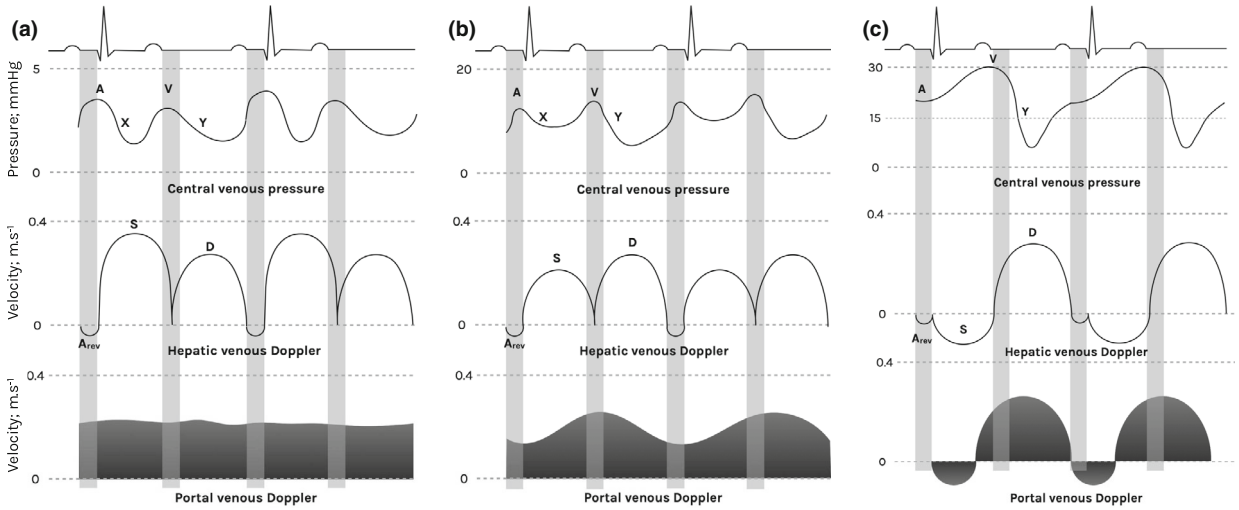

Figure 4 Correlation between right ventricular diastolic dysfunction, pathological central venous pressure waveform variants and Doppler ultrasonography of the hepatic and portal veins. (a) normal physiology; (b) early right ventricular diastolic dysfunction; (c) late right ventricular diastolic dysfunction.

the best way to treat acute hypovolaemia" [37]. Subsequent clinical approaches generally considered CVP an independent variable, leading to a focus on utilising CVP as an indicator of volume status and fluid responsiveness. Contemporary evidence suggests CVP reflects poorly the adequacy of cardiac preload – consistent with the observations of Ryan and Howland – and is a poor predictor of fluid responsiveness in patients who are critically ill [7, 12]. A 2013 meta-analysis including 43 studies and 1802 participants (including healthy volunteers, patients in the intensive care unit and patients undergoing major surgery) obtained a summary correlation coefficient between CVP and the change in stroke volume or cardiac index in response to a fluid challenge of 0.18 (95%CI 0.10–0.25), concluding CVP is unable to predict fluid responsiveness in the assessed cohorts [4]. This builds on a 2008 meta-analysis by the same group, who analysed CVP and fluid therapy (then including 24 studies involving 803 patients) which also concluded that CVP correlated poorly with measured blood volume, and has limited accuracy when guiding fluid therapy decisions, particularly in dynamic settings (pooled correlation coefficient 0.16, 95% CI 0.03–0.28) [38]. Similarly, a 2016 systematic review of 51 studies, and re-analysis of over 1000 patient datasets, concluded that CVP value in isolation is a poor predictor (both negative and positive) of fluid responsiveness at values 0–20 mmHg [6]. It was postulated that the poor predictive value of CVP and fluid responsiveness is explained by the complex interactions between venous return, right ventricular systolic and diastolic function, pulmonary vascular tone and intrathoracic pressure [5]. Therefore, given the wide variability of cardiac volume and myocardial contractile performance between individuals, isolated CVP measurement cannot predict individual response to fluid administration reliably [12]. Reliance on CVP for fluid responsiveness assessment is now discouraged [4, 5]. Moreover, current evidence suggests CVP values in isolation do not provide an accurate reflection of total blood volume status and should not be used as a single resuscitation target.

Central venous pressure and right ventricular diastolic dysfunction

Elevated CVP may reflect impaired right ventricular filling secondary to impaired ventricular compliance [12, 39]. However, CVP cannot be used to diagnose the cause of right ventricular dysfunction, which is a consequence of a several conditions, including elevated left atrial pressure; pulmonary hypertension; pulmonic stenosis; right ventricular outflow tract obstruction; and primary right ventricular dysfunction [39, 40].

In the normal heart, right ventricular end diastolic volume is determined by the transmural pressure and myocardial compliance. Increased intrapericardial and intrathoracic pressures reduce right ventricular transmural pressure, and impaired right ventricular relaxation requires a higher CVP to facilitate filling. As an example, following cardiac surgery, a patient living with obesity, who has World Health Organization group 3 pulmonary hypertension due to obstructive sleep apnoea,

and whose lungs are mechanically ventilated at a mean airway pressure of 12 mmHg may have a CVP of 15 mmHg despite being euvolaemic. Conversely, a patient breathing spontaneously in acute left heart failure may still have a low CVP [16].

Despite being non-specific for right ventricular dysfunction or volume status, monitoring of CVP trends may be useful in conditions such as pulmonary hypertension or left-sided heart failure, in which the development of acute right ventricular diastolic dysfunction is associated with high morbidity and poorer overall prognosis [3, 41]. The relationship between CVP and right ventricular dysfunction is complex, as elevated pressures can trigger a compensatory increase in afterload, worsening right ventricular filling [3, 39]. Nevertheless, CVP provides critical insights into the haemodynamic burdens placed on the right heart and can be useful to monitor the progression of right ventricular diastolic dysfunction over time (Fig. 4).

Anaesthetists and intensivists are called upon increasingly to care for patients at risk of right heart failure secondary to pulmonary hypertension. It is estimated to affect 1% of the global population, with increased prevalence in those aged > 65 y. Most cases are secondary to left heart disease or lung disease (World Health Organization group 2 disease). These patients have a peri-operative mortality of 15–50% when undergoing emergency surgery [42]. The 2022 International Society for Heart and Lung Transplantation guidelines recommend CVP be monitored during general anaesthesia where significant fluid shifts are expected [42]. Importantly, CVC insertion and monitoring is within the core skill set of all anaesthetists, whereas peri-operative echocardiography or pulmonary artery catheterisation may not be. In such cases, the absolute value, trends and waveform are all of importance. A CVP rising progressively combined with a falling mean arterial pressure may indicate a failing right ventricle, requiring consideration of inotropic and/or vasoactive support, careful volume management and inhaled pulmonary vasodilators. Changes in the CVP waveform also alert the anaesthetist to worsening right ventricular function [17, 42].

Doppler ultrasound findings, particularly in hepatic, portal and femoral veins, have been investigated to assess their relationship with CVP and right ventricular dysfunction. Emerging evidence suggests Doppler wave profiles in large veins may correlate with changes in CVP. An observational study investigating femoral venous analysis and right heart pressures on 238 patients found a statistically significant inverse correlation between both CVP and Doppler a-wave (atrial systole), and CVP and Doppler v-wave (atrial diastole, $r = -0.4297$, $p < 0.001$ and $r = -0.4893$, $p < 0.001$, respectively) [43]. Similarly, a study involving 200 patients concluded that flow patterns within the inferior vena cava can diagnose 'normal' (defined as < 7 mmHg) CVP with 94% sensitivity and 92% specificity [44]. Emerging evidence suggests Doppler venous analysis of major intra-abdominal veins (notably the hepatic and portal veins) has an association with right ventricular dysfunction and patient outcome, with a single-centre study reporting that elevated portal flow pulsatility fraction is associated with right ventricular dysfunction (defined as fractional area change of < 35% or a tricuspid annular plane systolic excursion of < 16 mm) and major complications (OR 5.83, 95%CI 2.04–16.68, $p = 0.001$) [45]. While further outcome-based data are required, Doppler changes in the hepatic and portal veins align closely with pathological CVP waveform variants. These changes are detectable using surface ultrasound or transoesophageal echocardiography and include the loss of the x-descent and exaggerated y-descent on interrogation of the CVP waveform, serving as complementary markers of right ventricular diastolic dysfunction [44]. Consequently, combining Doppler waveform analysis with CVP waveform analysis, CVP trends and absolute CVP values may provide further information regarding global haemodynamic status and right ventricular function (Fig. 4).

There is increasing evidence that CVP affects tissue perfusion, which could contribute to visceral organ injury. In health, a low CVP detracts minimally from the pressure gradient driving systemic perfusion (mean arteriovenous pressure gradient) which is normally > 80 mmHg and is represented by the gradient between CVP and mean arterial pressure [46]. However, during general anaesthesia, with a mean arterial pressure of 60 mmHg, an increase in CVP from 5 mmHg to 10 mmHg lowers the mean arteriovenous pressure (and thus the gradient driving organ perfusion) by 10%. Additionally, a raised CVP increases capillary pressure and changes the hydrostatic gradient between the intravascular and interstitial spaces. This can enable fluid to enter the interstitial space and cannot be overcome with an increase in mean arterial pressure [32].

There is evidence that a raised CVP is a predictor of acute kidney injury and mortality in patients undergoing cardiac surgery, independent of cardiac output [47]. Additionally, there is emerging evidence that mean arteriovenous pressure predicts kidney injury independent of CVP in patients in the intensive care unit, providing a rationale for higher mean arterial pressure targets in patients with a raised CVP [48]; however, prospective evidence supporting this practice is lacking [32, 46].

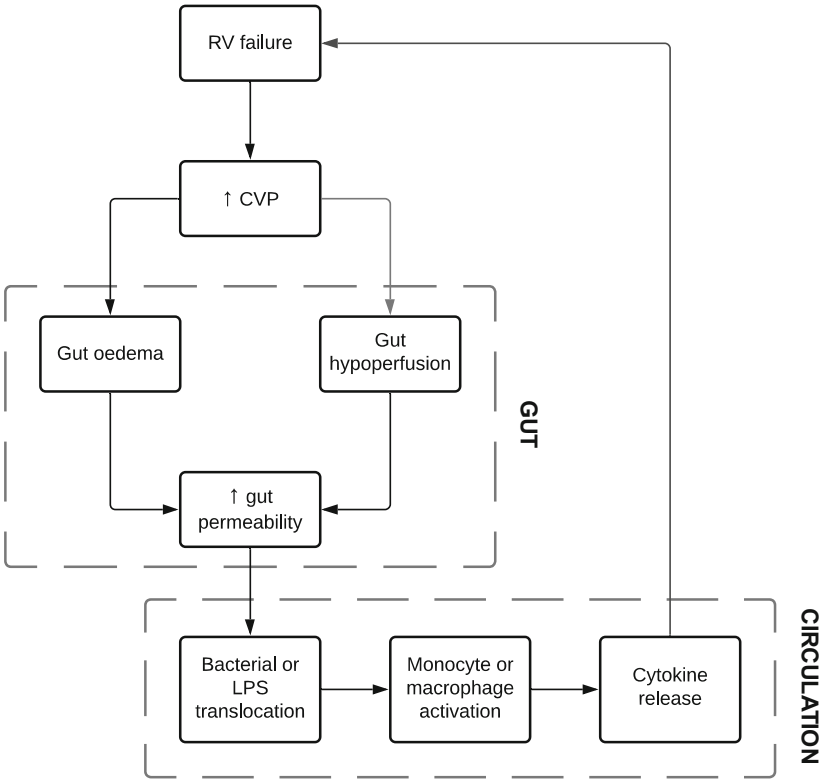

Figure 5 Pathogenesis of the cardio-intestinal syndrome [50]. RV, right ventricle; CVP, central venous pressure; LPS, lipopolysaccharide.

Elevated CVP in the absence of a corresponding increase in mean arterial pressure reduces mean arteriovenous pressure and therefore reduces organ perfusion [49]. In the gastrointestinal system, reduced mean arteriovenous pressure is implicated in the pathogenesis of cardio-intestinal syndrome, a condition in which inadequate splanchnic perfusion leads to ischaemia, altered gut motility and bacterial translocation [50]. Elevated CVP in patients with this condition may contribute to reduced venous return and, in turn, decreased forward flow through splanchnic circulation and worsening of the disease state [49, 50]. Prospective observational studies have reported that elevated CVP (> 12 mmHg) in sepsis reduces microcirculatory perfusion [51]. Moreover, it has been suggested that splanchnic congestion itself may contribute to worsening right heart failure; worsening venous congestion results in decreased mean arteriovenous pressure and enterocyte ischaemia, followed by intracellular acidosis. This results in upregulation of sodium-hydrogen exchanger-3 and pro-inflammatory cytokines, causing increased enteral sodium absorption, subsequent increased water absorption and worsening fluid overload [52]. Central venous pressure monitoring may provide the clinician with an early marker of impaired venous return which, when interpreted alongside mean arterial pressure and mean arteriovenous pressure, may warn of decreased microcirculatory perfusion and impending deranged organ perfusion and/or systemic congestion (Fig. 5).

Conclusion

Central venous pressure remains a fundamental yet controversial parameter in haemodynamic monitoring. While evidence suggests that the absolute CVP value alone is a poor predictor of fluid responsiveness, integrating CVP with waveform analysis and trend monitoring provides valuable insights into right heart function, venous congestion and organ perfusion. Emerging evidence suggests there is valuable interplay between CVP and critical physiological variables, such as right ventricular diastolic dysfunction, microcirculatory perfusion and Doppler-derived venous waveforms, reinforcing the relevance of CVP monitoring in peri-operative and critical care settings.

As surgical and critically ill patient populations become increasingly complex, the role of CVP should be

reconsidered – not as an isolated predictor, but as a component of comprehensive haemodynamic assessment. Understanding the nuances of CVP trends, waveform morphology and its interactions with other haemodynamic markers remains integral to informing accurate clinical decision-making in critical care medicine. Future research directions may include refining the integration of CVP with advanced monitoring modalities, while minimising reliance on outdated paradigms of volume assessment to optimise patient outcomes.

Author affiliations

Patryck Lloyd-Donald,[1] Midori Fujino,[1] Boris Waldman,[1] and Lachlan F. Miles,[1,2]

1 Department of Anaesthesia, Austin Health, Melbourne, VIC, Australia

2 Department of Critical Care, Melbourne Medical School, The University of Melbourne, Melbourne, VIC, Australia

Acknowledgements

The authors gratefully acknowledge the contribution of Kathryn Rough of the Australian and New Zealand College of Anaesthetists library for her assistance in developing the search strategy, and Professor Laurence Weinberg of the Austin Health Department of Anaesthesia who reviewed the article before submission. The authors' institution has received funding from BD Advanced Patient Monitoring (formerly Edwards Lifesciences) unrelated to the presented work. LM is an Editor of *Anaesthesia*. No data or statistical code were generated. No other competing interests declared. Open access publishing facilitated by The University of Melbourne, as part of the Wiley - The University of Melbourne agreement via the Council of Australian University Librarians.

References

1. Yeomans A, Porter RR, Swank RL. Observations on certain manifestations of circulatory congestion produced in dogs by rapid infusion. *J Clin Invest* 1943; **22**: 33–45. https://doi.org/10.1172/jci101366.
2. Singh NK, Sangwan G, White P. Central venous pressure as popular resuscitation surrogate: not totally unjustified. *Chest* 2008; **134**: 1352. https://doi.org/10.1378/chest.08-2024.
3. Whitehead EH, Thayer K, Burkhoff D, et al. Elevated central venous pressure as a trigger for right heart failure evaluation in patients receiving left-sided mechanical support for cardiogenic shock. *J Card Fail* 2020; **26**: S55. https://doi.org/10.1016/j.cardfail.2020.09.161.
4. Marik PE, Cavallazzi R. Does the central venous pressure predict fluid responsiveness? An updated meta-analysis and a plea for some common sense. *Crit Care Med* 2013; **41**: 1774–81. https://doi.org/10.1097/CCM.0b013e31828a25fd.
5. Meyer TE. Assessment of the jugular venous pressure. UpToDate June 2024. https://www.uptodate.com/contents/assessment-of-the-jugular-venous-pressure (accessed 22/10/2024).
6. Eskesen TG, Wetterslev M, Perner A. Systematic review including re-analyses of 1148 individual data sets of central venous pressure as a predictor of fluid responsiveness. *J Intensive Care Med* 2016; **42**: 324–32. https://doi.org/10.1007/s00134-015-4168-4.
7. Rex S, Brose S, Metzelder S, et al. Prediction of fluid responsiveness in patients during cardiac surgery. *Br J Anaesth* 2004; **93**: 782–8. https://doi.org/10.1093/bja/aeh280.
8. Kalso E. A short history of central venous catheterization. *Acta Anaesthesiol Scand* 1985; **29**: 7–10. https://doi.org/10.1111/j.1399-6576.1985.tb02313.x.
9. Bayliss WM, Starling EH. Observations on venous pressures and their relationship to capillary pressures. *J Physiol* 1894; **16**: 159–318. https://doi.org/10.1113/jphysiol.1894.sp000498.
10. Patterson SW, Starling EH. On the mechanical factors which determine the output of the ventricles. *J Physiol* 1914; **48**: 357–79. https://doi.org/10.1113/jphysiol.1914.sp001669.
11. Ball C, Westhorpe RN. Central venous pressure monitoring. *Anaesth Intensive Care* 2009; **37**: 689. https://doi.org/10.1177/0310057X0903700535.
12. Berlin DA, Bakker J. Starling curves and central venous pressure. *Crit Care* 2015; **19**: 55. https://doi.org/10.1186/s13054-015-0776-1.
13. Forssmann W. Probing the right heart. *Klin Wochenschr* 1929; **8**: 2085–7. https://doi.org/10.1007/BF01875120.
14. West JB. The beginnings of cardiac catheterization and the resulting impact on pulmonary medicine. *Am J Physiol Lung Cell Mol Physiol* 2017; **313**: L651–8. https://doi.org/10.1152/ajplung.00133.2017.
15. Gauer OH, Henry JP, Sieker HO. Changes in central venous pressure after moderate hemorrhage and transfusion in man. *Circ Res* 1956; **4**: 79–84. https://doi.org/10.1161/01.RES.4.1.79.
16. Guyton AC, Jones CE. Central venous pressure: physiological significance and clinical implications. *Am Heart J* 1973; **86**: 431–7. https://doi.org/10.1016/0002-8703(73)90132-4.
17. Mark JS, Atilio B, McCarthy G. Cardiovascular monitoring. In: Gropper M, ed. *Miller's Anesthesia*, 10th edn. Philadelphia, PA: Elsevier, 2024: 994–1042.
18. Latham HE, Rawson ST, Dwyer TT, Patel CC, Wick JA, Simpson SQ. Peripherally inserted central catheters are equivalent to centrally inserted catheters in intensive care unit patients for central venous pressure monitoring. *J Clin Monit Comput* 2012; **26**: 85–90. https://doi.org/10.1007/s10877-012-9337-1.
19. Jenkins LC, Screech G. Central venous pressure monitoring in anaesthesia. *Can Anaesth Soc J* 1966; **13**: 513–29. https://doi.org/10.1007/bf03003618.
20. Walsh JT, Hildick-Smith DJR, Newell SA, Lowe MD, Satchithananda DK, Shapiro LM. Comparison of central venous and inferior vena caval pressures. *Am J Cardiol* 2000; **85**: 518–20. https://doi.org/10.1016/S0002-9149(99)00787-0.
21. Lee BK, Lee HY, Jeung KW, Jung YH, Lee GS. Estimation of central venous pressure using inferior vena caval pressure from a femoral endovascular cooling catheter. *Am J Emerg Med* 2013; **31**: 240–3. https://doi.org/10.1016/j.ajem.2012.06.034.
22. Magder S. Central venous pressure: a useful but not so simple measurement. *Crit Care Med* 2006; **34**: 2224–7. https://doi.org/10.1097/01.CCM.0000227646.98423.98.
23. Haywood GA, Joy MD, Camm AJ. Influence of posture and reference point on central venous pressure measurement. *BMJ (Clinical Research Ed)* 1991; **303**: 626–7. https://doi.org/10.1136/bmj.303.6803.626.
24. Kleinman BS, Powell S, Gardner RM. Equivalence of fast flush and square wave testing of blood pressure monitoring systems. *J Clin Monit* 1996; **12**: 149–54. https://doi.org/10.1007/bf02078135.
25. Mark JB. Central venous pressure monitoring: clinical insights beyond the numbers. *J Cardiothorac Vasc Anesth*

1991; **5**: 163–73. https://doi.org/10.1016/1053-0770(91)90333-O.
26. Ahrens TS, Schallom L. Comparison of pulmonary artery and central venous pressure waveform measurements via digital and graphic measurement methods. *Heart Lung* 2001; **30**: 26–38. https://doi.org/10.1067/mhl.2001.112504.
27. Smith T, Grounds RM, Rhodes A. Central venous pressure: uses and limitations. In: Pinsky MR, Payen D, eds. *Functional Hemodynamic Monitoring. Update in Intensive Care and Emergency Medicine*. Volume **42**. Berlin: Springer, 2005: 99–110. https://doi.org/10.1007/3-540-26900-2_8.
28. Roger C, Muller L, Riou B, et al. Comparison of different techniques of central venous pressure measurement in mechanically ventilated critically ill patients. *Br J Anaesth* 2017; **118**: 223–31. https://doi.org/10.1093/bja/aew386.
29. Fujita Y, Hayashi D, Wada S, Yoshioka N, Yasukawa T, Pestel G. Central venous pulse pressure analysis using an r-synchronized pressure measurement system. *J Clin Monit Comput* 2006; **20**: 385–9. https://doi.org/10.1007/s10877-006-9035-y.
30. Ryan GM, Howland WS. An evaluation of central venous pressure monitoring. *Anesth Analg* 1966; **45**: 754–9.
31. Gelman S. Venous function and central venous pressure: a physiologic story. *Anesthesiology* 2008; **108**: 735–48. https://doi.org/10.1097/ALN.0b013e3181672607.
32. Pesenti A, Slobod D, Magder S. The forgotten relevance of central venous pressure monitoring. *J Intensive Care Med* 2023; **49**: 868–70. https://doi.org/10.1007/s00134-023-07101-z.
33. Senguttuvan NB, Karthikeyan G. Jugular venous c-v wave in severe tricuspid regurgitation. *N Engl J Med* 2012; **366**: e5. https://doi.org/10.1056/NEJMicm1012843.
34. Samura T, Yoshioka D, Asanoi H, et al. Right atrial pressure waveform predicts right ventricular failure after left ventricular assist device implantation. *Ann Thorac Surg* 2019; **108**: 1361–8. https://doi.org/10.1016/j.athoracsur.2019.04.050.
35. Doshi S, Ramakrishnan S, Gupta SK. Invasive hemodynamics of constrictive pericarditis. *Indian Heart J* 2015; **67**: 175–82. https://doi.org/10.1016/j.ihj.2015.04.011.
36. Wilson JN, Grow JB, Demong CV, Prevedel AE, Owens JC. Central venous pressure in optimal blood volume maintenance. *Arch Surg* 1962; **85**: 563–78. https://doi.org/10.1001/archsurg.1962.01310040035005.
37. Kelman GR. Interpretation of CVP measurements. *Anaesthesia* 1971; **26**: 209–15. https://doi.org/10.1111/j.1365-2044.1971.tb04764.x.
38. Marik PE, Baram M, Vahid B. Does central venous pressure predict fluid responsiveness? A systematic review of the literature and the tale of seven mares. *Chest* 2008; **134**: 172–8. https://doi.org/10.1378/chest.07-2331.
39. Konstam MA, Kiernan MS, Bernstein D, et al. Evaluation and management of right-sided heart failure: a scientific statement from the American Heart Association. *Circulation* 2018; **137**: 578–622. https://doi.org/10.1161/cir.0000000000000560.
40. Arrigo M, Huber LC, Winnik S, et al. Right ventricular failure: pathophysiology, diagnosis and treatment. *Card Fail Rev* 2019; **5**: 140–6. https://doi.org/10.15420/cfr.2019.15.2.
41. Sanders JL, Koestenberger M, Rosenkranz S, Maron BA. Right ventricular dysfunction and long-term risk of death. *Cardiovasc Diagn Ther* 2020; **10**: 1646–58. https://doi.org/10.21037/cdt-20-450.
42. McGlothlin DP, Granton J, Klepetko W, et al. ISHLT consensus statement: perioperative management of patients with pulmonary hypertension and right heart failure undergoing surgery. *J Heart Lung Transplant* 2022; **41**: 1135–94. https://doi.org/10.1016/j.healun.2022.06.013.
43. Cozcolluela MR, Sarría L, Sanz L, Martinez-Berganza MT, de Vera JM, Bernal A, García S. Correlation of central venous pressure with Doppler waveform of the common femoral veins. *J Ultrasound Med* 2000; **19**: 587–92. https://doi.org/10.7863/jum.2000.19.8.587.
44. Ranjan R, Mostafavi Toroghi H, Pressman GS, Schiller NB. Doppler recognition of low or normal central venous pressure from continuous flow from inferior vena cava into right atrium. *Am J Cardiol* 2019; **124**: 448–52. https://doi.org/10.1016/j.amjcard.2019.04.044.
45. Eljaiek R, Cavayas YA, Rodrigue E, et al. High postoperative portal venous flow pulsatility indicates right ventricular dysfunction and predicts complications in cardiac surgery patients. *Br J Anaesth* 2019; **122**: 206–14. https://doi.org/10.1016/j.bja.2018.09.028.
46. Raman J, Hu R, Bellomo R. Mean perfusion pressure and venous congestion: important but often forgotten aspects of heart failure. *Heart Lung Circ* 2024; **33**: 263–4. https://doi.org/10.1016/j.hlc.2024.03.001.
47. Li J, Wang R, Wan J, Zhu P, Xiao Z, Wang X, Zheng S. Postoperative central venous pressure is associated with acute kidney injury in patients undergoing coronary artery bypass grafting. *Front Cardiovasc Med* 2022; **9**: 1016436. https://doi.org/10.3389/fcvm.2022.1016436.
48. Ostermann M, Hall A, Crichton S. Low mean perfusion pressure is a risk factor for progression of acute kidney injury in critically ill patients – a retrospective analysis. *BMC Nephrol* 2017; **18**: 151. https://doi.org/10.1186/s12882-017-0568-8.
49. Mohamedali B, Yost G, Doukky R, Bhat G. Mean arterial pressure to central venous pressure ratio – a novel marker for right ventricular failure after left ventricular assist device placement. *J Heart Lung Transplant* 2016; **35**: S381. https://doi.org/10.1016/j.healun.2016.01.1098.
50. Sundaram V, Fang JC. Gastrointestinal and liver issues in heart failure. *Circulation* 2016; **133**: 1696–703. https://doi.org/10.1161/circulationaha.115.020894.
51. Vellinga N, Ince C, Boerma EC. Elevated central venous pressure in septic patients is associated with impairment of microcirculatory blood flow. *Crit Care* 2012; **16**: P200. https://doi.org/10.1186/cc10807.
52. Polsinelli VB, Marteau L, Shah SJ. The role of splanchnic congestion and the intestinal microenvironment in the pathogenesis of advanced heart failure. *Curr Opin Support Palliat Care* 2019; **13**: 24–30. https://doi.org/10.1097/spc.0000000000000414.