Central venous pressure: soon an outcome-associated matter

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Purpose of review
Central venous pressure (CVP) alone has so far not found a place in outcome prediction or prediction of fluid responsiveness. Improved understanding of the interaction between mean systemic pressure (P_{ms}) and CVP has major implications for evaluating volume responsiveness, heart performance and potentially patient outcomes.

Recent findings
The literature review substantiates that CVP plays a decisive role in causation of operative haemorrhage and renal failure. The review details CVP as a variable integral to cardiovascular control in its dual role of distending the diastolic right ventricle and opposing venous return.

Summary
The implication for practice is in the regulation of the circulation. It is demonstrated that control of the blood pressure and cardiac output/venous return calls upon regulation of the volume state (P_{ms}), the heart performance (E_h) and the systemic vascular resistance. Knowledge of the CVP is required to calculate all three.

Keywords
cardiovascular regulation, central venous pressure, heart efficiency, venous return, volume responsiveness

INTRODUCTION

Central venous pressure, the prodigal variable

Goal-directed therapy (GDT), whether employed in the perioperative or intensive care setting, typically relegates central venous pressure (CVP) to a simple numerical target or a variable traditionally reported but with little bearing on the cardiovascular goals targeted. Such meagre appreciation of CVP is based on the poor correlation to the intravascular volume *per se* or rapid manipulations of the same to increase cardiac output (CO), that is fluid responsiveness [1–4]. Gurgel and do Nascimento [5] and Hamilton *et al*. [6] reviewed 32 controlled trials investigating haemodynamic interventions to maintain adequate tissue perfusion in high-risk surgical patients. Usually, CVP was reported as a static pressure in isolation with little or no attention paid to the context in which CVP was measured. The influential Surviving Sepsis Campaign, widely endorsed by intensive care societies, recommends targeting CVP but only provides the GRADE system 1C recommendation (strong recommendation based on well done observational studies) in the context of concurrent measures of mean arterial pressure (MAP), urine output and central venous oxygen saturation ($S_{cv}O_2$) [4,7,8].

In perioperative management, CVP has been the subject of controlled trials in patients undergoing hepatectomy and liver transplantation, uniformly demonstrating a positive, linear relationship between CVP, blood loss, transfusion and length of hospital stay [9–13], which has led to the implementation of low CVP anaesthesia [14,15]. A similar relationship has been observed in retrospective studies in cystectomy and coronary artery bypass surgery patients [1,2]. Retrospective studies in critically ill septic patients have pointed to a linear relationship between risk of acute kidney injury and CVP [16]. Another study in septic patients showed that

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KEY POINTS

- CVP plays a pivotal role in the understanding and regulation of cardiovascular function.
- CVP in Guytonian physiology completes the prevailing Starlingesque understanding of circulation.
- CVP must claim the role as an outcome-associated variable in future studies.

microvascular flow index and perfused small vessels were significantly lower in patients with CVP above 12 mmHg vs CVP of 12 mmHg or less [17]. A post-hoc study of data from the vasopressin in septic shock trial demonstrated that CVP correlated with fluid balance at 12 h and that patients with CVP below 8 mmHg had the lowest mortality rate followed by those with CVP 8–12 mmHg and above 12 mmHg [18]. See Supplementary Digital Content, http://links.lww.com/COAN/A40, for literature review.

The position of CVP in perioperative and intensive care seems confusing and inconclusive. In the current Starling-based concept of volume optimizing GDT, it is denounced as having no role in fluid resuscitation. The recent ARISE [19], ProMISE [20] and ProCESS [21] studies did not show any advantage of GDT compared with standard therapy including measurements of CVP. A subsequent meta-analysis did not specifically address CVP outside the bundle including MAP and ScvO2 in both GDT and control groups [22].

Still, CVP seems to play a vital role in bleeding control and in preserving organ function. It may thus be contemplated to change the paradigm, adopting a cardiovascular model encompassing CVP as a variable associated with outcome.

The Frank–Starling concept dictates that CVP, within limits, is linearly related to CO as has been demonstrated on the 'individual' level by van den Berg *et al.* [23]. The interindividual variation is ascribed to inotropic state and this explains the lack of relationship at the 'group' level. The Starling cardiac function curve does not reveal the fact that CVP, as well as distending the right ventricle, acts as a counterpressure to venous return. It was Arthur Guyton who clarified this in the combined graphing of the CO and venous return curves. Guyton furthermore described the preloading relationship between the pressure gradient from venous capacitance to CVP and venous return, and consequently CO.

It is the opinion of the authors that there is a need for a change in paradigm. The remainder of this review introduces the reader to a model describing the responses of the cardiovascular system in a coherent and parsimonious setting.

The use of this paradigm will be illustrated with data from a study involving colloid and crystalloid fluid resuscitation and a pharmacological study see supplementary digital content, http://links.lww.com/COAN/A40.

Understanding central venous pressure

Control of the circulation physiologically or therapeutically devolves into three elements: control of the volume state, the performance of the heart and resistances. The relationship between the CVP and these elements is often imperfectly understood.

Preload

The volume state has been thought of in terms of 'preload', a measure of end-diastolic myocardial fibre length. Right ventricular end diastolic volume, right ventricular end diastolic pressure and CVP have an increasingly complex and distant relationship with fibre length. Preload is also influenced by intrinsic heart performance and afterload in addition to the influence of the volume state.

In addition, CVP is influenced by the intrathoracic and intrapericardial pressures which are complexly related to preload [24]. We are thus better off considering the effect of the absolute CVP on venous return. The CVP is not a measure of the volume state or a credible measure of preload. Attempts to mathematically relate CVP to stroke volume, CO or volume responsiveness are likely to end in frustration and the view that CVP measurement has no place in volume therapy [3,25]. We need to look at matters in another way by considering the volume state.

Just as we judge the volume state of a tyre by measuring the static pressure within it, equally by the systemic blood volume state, we should imply the average pressure (P_{ms}) within the systemic circulation. This pressure is a consequence of the stressing volume in the elastic circulation above that just required to fill the circulation without wall distension or pressure rise. The normal value of P_{ms} is approximately 7 mmHg in humans but may rise to 30 mmHg or more in patients in heart or renal failure.

$$P_{ms} = \frac{V_s}{C} \quad (1)$$

where V_s is the stressing volume (ml) and C is the systemic compliance (ml/mmHg).

Volume state (P_{ms}) and venous return

P_{ms} is critical to understanding the cardiovascular dynamics. The P_{ms}, in pushing the venous return

back to the heart, forms a quantitative relationship between the volume state and the circulatory dynamics.

$$CO = VR = \frac{P_{ms} - CVP}{RVR} \quad (2)$$

where RVR is the resistance to venous return (mmHg/l/min). The mean systemic filling pressure (P_{ms}) and (similar) mean circulatory filling pressure (P_{mc}) were championed by Guyton, who in a 1980 foreword stated.

> *"It was during this period that we discovered with much intellectual satisfaction the extreme importance of the mean circulatory filling pressure which turned out to be the first measurable quantity that allows one to relate blood volume mathematically to the control of cardiac output and arterial pressure. We hope that the reader will see the beauty of this concept"* [26].

The evidence is that most did not.

Resistance to venous return

The RVR is defined as the resistance encountered by the average element in the circulation in returning to the heart. The RVR thus depends on both arterial and venous resistances and the related disposition of the circulatory elements. This in turn depends on arterial and venous compliances. Changes in arterial and venous resistances together with veno-arterial compliance ratio thus effect RVR.

Equation 2 is an Ohmic relationship (I=V/R) wherein the current venous return is determined by the pressure difference (P_{ms} − CVP) generated by the heart, somewhat like the voltage difference generated by a battery. The resistance of the tissues is determined locally by oxygen requirements. The venous return determines CO (Starling) and oxygen delivery, DO_2. Thus, Equation 2 unifies the role of volume, the tissues, the heart and the kidneys in the determination of cardiovascular dynamics.

Measuring P_{ms} and P_{msa}, the effective volume state

Maas *et al.* [27] approached the problem of measuring P_{ms} by using progressive retardation of venous return with inspiratory hold manoeuvres during positive pressure ventilation while continuously measuring CO. The linear plot of CVP (x) vs CO (y) is extrapolated to CO = 0, wherein the CVP of the x-intercept is taken to indicate P_{ms} [28,29].

An alternative computational approach is to see how far the patient is displaced in volume, arterial resistance and heart performance from a normal resting 'template' of a patient of the same size and age. Consider a minimalist normal resting two-compartment model of the systemic circulation comprising lumped arterial capacitance C_a, venous capacitance C_v and right atrium linked in series with arterial and venous resistances R_a and R_v, see Fig. 1.

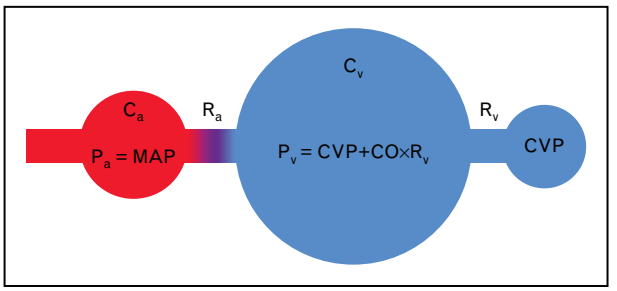

FIGURE 1. Lumped circulatory model consisting of an arterial (C_a) and a venous (C_v) capacitance connected by arterial (R_a) and venous (R_v) resistances. $C_v/C_a = 24$ and $R_a/R_v = 25$ [30].

For a system of n linked capacitances, the 'average pressure' is given by

$$P_{av} = \frac{(P_1 \times C_1) + (P_2 \times C_2) + \ldots + (P_n \times C_n)}{(C_1 + C_2 + \ldots C_n)} \quad (3)$$

For the two compartment model

$$P_{msa} = \frac{(P_a \times C_a) + (P_v \times C_v)}{C_a + C_v} \quad (4)$$

where P_{msa} is the model analogue of P_{ms}, that is

$$P_{msa} = \frac{MAP \times C_a + (CVP + CO \times R_v) \times C_v}{C_a + C_v} \quad (5)$$

dividing by C_a

$$P_{msa} = \frac{MAP + (CVP + CO \times R_v) \times \frac{C_v}{C_a}}{1 + \frac{C_v}{C_a}} \quad (6)$$

Let $x = \frac{C_v}{C_a}$, then

$$P_{msa} = \frac{x}{1+x} \times CVP + \frac{1}{1+x} \times MAP + \frac{x}{1+x} \times R_v \times CO \quad (7)$$

Clinically, we are not in a position to know C_a, C_v or x, the venoarterial compliance ratio or the patient's venous resistance, R_v. Perturbations in these values will alter the effective volume state by changing RVR and, of course, P_{msa} directly. This is a consequence of the venous return Equation 2 wherein changes in P_{ms} produce changes in venous return of opposite sign to changes in RVR. By

inserting a normal resting value of $x = 24$, according to Guyton [30], we obtain

$$P_{msa} = 0.96 \times CVP + 0.04 \times MAP + 0.96 \times RV_{nr} \times CO \quad (8)$$

P_{msa} will equal P_{ms} for changes in volume state, heart performance and arterial resistance. P_{msa} will not equal P_{ms} for unmeasurable changes in venous resistance or venoarterial compliance ratio. In these latter situations, P_{msa} and not P_{ms} will measure the effective volume state. Using the normal resting value of RV, denoted RV_{nr}, for the patient in question [$RV_{nr} = 1/26\times$ normal resting systemic vascular resistance (SVR), mmHg/l/min] together with the patient's actual CVP, MAP and CO, we obtain the effective volume state (P_{msa}).

The normal resting SVR of the patient is age and size-dependent and is known from the normal resting age-dependent MAP divided by the normal resting age-dependent CO [31].

The CO is obtained from the normal resting cardiac index from Brandfonbrener *et al.* [32] multiplied by the body surface area. A normal CVP of zero is assumed.

This approach to measure RV_{nr} scales the CO term in the P_{msa} (Equation 8) for individuals of widely different size and age.

Central venous pressure and the effective volume state, P_{msa}

The CVP term used in the P_{msa} (Equation 8) has a coefficient of 0.96 or nearly unity. That is, the CVP is added to the MAP and CO terms to obtain the volume state. In this sense, the CVP acts like a 'floating ground' in electrical circuits. Although absolute intravascular pressures are measured with respect to atmosphere as the zero (as with a true ground), we are usually interested in the difference between the intravascular pressure and the CVP, as, for example, in Equation 2 or the SVR in Equation 12.

The CVP is usually the point of lowest pressure in the circulation in the supine patient. It may be negative, equal or significantly positive with respect to the atmospheric pressure. The CVP is measured with a transducer placed on the phlebostatic axis and zeroed to atmospheric pressure. The phlebostatic axis is a horizontal plane through the tricuspid valve at the base of the right atrium. The position of the right atrium in the thorax ensures that the normal CVP at rest is close to the atmospheric pressure as with a true ground [33▪▪]. A contentious subject is at which point in the CVP curve to obtain the CVP. Conventionally, it is reported as a mean by the haemodynamic monitor. This, for all purposes, may function as an integral value of respiratory oscillations in atrial pressure and its effects on venous return. For an elaborate discussion on the CVP, see Magder [34].

Measuring the performance of the heart

The value of the CVP and therefore the floating ground primarily represents the interplay between the volume state, the performance of the heart and the RVR. As the role of the heart could be seen as creating a pressure gradient ($P_{msa} - CVP$) for venous return, the dimensionless variable

$$E_h = \frac{P_{msa} - CVP}{P_{msa}}, 1 \geq E_h \geq 0 \quad (9)$$

is useful in defining the global performance of the heart. In addition to factors intrinsic to the heart, E_h is sensitive to mechanical factors that extrinsically effect heart performance (e.g. pericardial tamponade, tension pneumothorax, raised intrathoracic pressure from ventilation, intra-abdominal pressure). Rearranging Equation 9, we note

$$CVP = P_{msa} \times (1 - E_h) \quad (10)$$

from which it follows that when E_h is 1 (normal heart), the CVP floating ground is equal to the true ground and equals zero.

When the heart is stopped, CO and $E_h = 0$, and $CVP = P_{msa}$ at which time the CVP (and MAP) measure the volume state P_{msa}.

A corollary is that the coefficients of CVP and MAP in Equation 8 sum to unity. From the venous return Equation 2, rearranging

$$CVP = P_{msa} - CO \times RVR \quad (11)$$

shows the influence of RVR on CVP.

Controlling resistance

The SVR

$$SVR = \frac{MAP - CVP}{CO} \quad (12)$$

is the sum of the arterial and venous resistances. The venous resistance is normally of the order of 1/25 of the arterial resistance but has a profound effect on the circulation. Increase in arterial resistance will increase MAP while decreasing CO and *vice versa*. The arteriolar resistance may be seen as partitioning the power output of the heart between

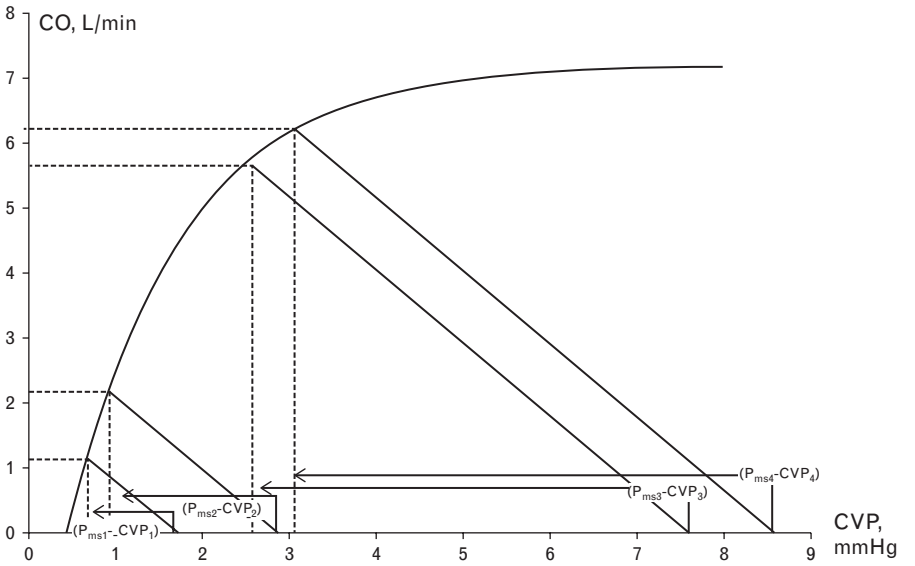

FIGURE 2. Two situations of venous return curves intersecting with cardiac function curve before and after fluid resuscitation. In the first situation, marked with subscripts 1 and 2, a volume bolus increases P_{msa} from P_{ms1} to P_{ms2} and CVP from CVP1 to CVP2. The increase from CVP1 to CVP2 is moderate, the increase in return pressure $\Delta(P_{msa} - CVP)$ is large. Compare this favourable situation with the situation marked with subscripts 3 and 4: an identical increase in P_{msa} generates a larger increase in CVP and thus a smaller $\Delta(P_{msa} - CVP)$. Relating $\Delta(P_{msa} - CVP)$ to ΔP_{msa} in the first instance yields a larger figure than in the second case, indicating a better outcome of fluid resuscitation in first instance compared with second. This is formalized in Equation 13.

potential energy (MAP) and kinetic energy (CO). Thus, a circulation with a designated MAP and CO will require a designated power (MAP × CO), generally achieved with volume and the correct SVR to partition that power between MAP and CO. Increase in the venous resistance will decrease both MAP and CO and *vice versa*.

Volume responsiveness

We are frequently interested in predicting if an increase in volume state will produce a useful rise in CO. A standard procedure is to assess if a volume load of, for example, 3 ml/kg produce a ΔCO of at least 10%. Such a rise would require $\Delta(P_{msa} - CVP)$ of at least 10% if RVR is unaffected (see Equation 2).

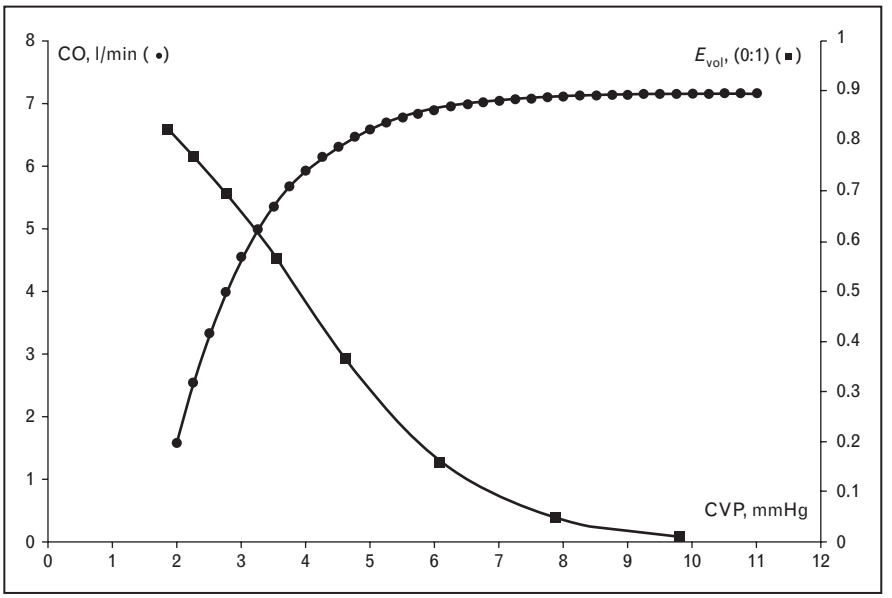

FIGURE 3. Concordant cardiac function curve (filled circle) and E_{vol} (filled square) as P_{msa} is increased stepwise by 2 mmHg.

The P_{msa} rises with a volume load. The response of the CVP will essentially depend upon the performance of the heart, particularly its ability to maintain a difference between P_{msa} and CVP. If heart function is poor, CVP will rise towards P_{msa}; if well maintained, the (P_{msa} − CVP) difference will be sustained or increase and the CVP will remain relatively low. This conforms to the adage, before the days of P_{msa}, that it is the rate of rise of the CVP rather than its absolute value that should guide the rate of volume therapy.

The outcome of fluid resuscitation is illustrated in Fig. 2 in the combination of Starling's cardiac function curve and Guyton's venous return curve. Depending on the position of the intercept of the P_{msa} increase and the cardiac function curve, CVP and CO change in distinct patterns. If the ΔP_{msa} intercepts the ascending limb of the function curve, CVP may rise minimally whilst CO responds significantly. In contrast, if the ΔP_{msa} intercepts the function curve in the flat part, the CO changes minimally and CVP increases substantially.

In response to fluid administration, the measure of 'volume efficiency', E_{vol}, may appropriately answer the question of whether fluid, vasopressor or inotropic therapy is more conducive to the target of increasing CO and DO_2. Parkin has suggested a ratio of change in (P_{msa} − CVP) divided by the change in P_{msa}:

$$E_{vol} = \frac{\Delta(P_{msa} - CVP)}{\Delta P_{msa}} \quad (13)$$

The relationship between the Starling function curve and volume efficiency is illustrated in Fig. 3.

CONCLUSION

Knowledge of the CVP is essential for the measurement of the volume state, the performance of the heart and the SVR. It enters considerations of heart, volume and power efficiency. The CVP provides a floating ground for the differential measurement of intravascular pressures. It does not inherently measure preload or the volume state but its measurement is essential to their calculation. Once the above principles are understood, precise control of the circulation becomes a straightforward mathematically predictable process. Readers can refer to the Supplementary Digital Content, http://links.lww.com/COAN/A40, for worked examples of the algorithm and details of literary review.

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Conflicts of interest

For the first 12 months or so of the 36 months prior to submission, G.P. received a partial salary as employee of Applied Physiology (AP). AP was an Australian registered software company engaged in the commercialization of decision support software for the circulation. During that period, G.P. was a member of the Board following the retirement of another. A.A. and S.S. have no conflicts of interest.

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- ■■ of outstanding interest

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