# Central venous pressure: we need to bring clinical use into physiological context

S. Sondergaard[1], G. Parkin[2] and A. Aneman[3]

[1]Department of Anaesthesiology and Intensive Care Medicine, Sahlgrenska University Hospital, University of Gothenburg, Gothenburg, Sweden
[2]Intensive Care Unit, Monash Medical Centre, Clayton, Vic, Australia
[3]Intensive Care Unit, Liverpool Hospital, SWSLHD, University of New South Wales, Liverpool BC, NSW, Australia

**Correspondence**
S. Sondergaard, Department of Anaesthesiology and Intensive Care Medicine, Sahlgrenska University Hospital, University of Gothenburg, Blå Stråket 5,5, Gothenburg 413 45, Sweden
E-mail: sondergaard.soren@gmail.com

**Conflict of interest**
Soren Sondergaard and Anders Aneman declare no conflicts of interest. Geoff Parkin declares that he has been a director and employee of Applied Physiology, an Australian software firm, which produced the Navigator Cardiovascular Decision Support device. He was paid by Applied Physiology and held shares in the firm. Applied Physiology is no longer trading. The findings of this publication no longer affect directly or indirectly Dr Parkin's compensation.

**Funding**
The present study received no institutional or other funding.

Submitted 20 December 2014; accepted 31 December 2014; submission 24 April 2014.

**Citation**
Sondergaard S, Parkin G, Aneman A. Central venous pressure: we need to bring clinical use into physiological context. Acta Anaesthesiologica Scandinavica 2015

doi: 10.1111/aas.12490

**Background:** The place of central venous pressure (CVP) measurement in acute care has been questioned during the past decade. We reviewed its physiological importance, utility and clinical use among anaesthetists and intensivists.
**Methods:** A literature search using the PubMed, Cochrane, Scopus and Web of Science databases was performed in regard to details of the physiology, measurement and interpretation of CVP. A questionnaire was conducted among members of the European Society of Intensive Care Medicine concerning knowledge and uses of CVP.
**Results:** Aligning pressure transducers to the phlebostatic axis was handled inadequately. The unsuitability of CVP to assess the intravascular volume state was generally recognised by clinicians. Still, many used CVP to guide volume resuscitation in the absence of a cardiac output monitor, while the literature positioned CVP as a useful haemodynamic variable only in the expanded context of being one determinant of the driving pressure for venous return and hence cardiac output.
**Conclusion:** The correct measurement of CVP is pivotal to its proper clinical application. This relates to defining the pressure gradient for venous return and heart efficiency. The clinical appreciation of CVP should be restored by educational efforts of its physiological context.

**Editorial comment: what this article tells us**
The use of central venous pressure (CVP) was studied, focusing on physiology, measurement and interpretation. Daily practice on CVP was assessed by a questionnaire. Aligning pressure transducers to the phlebostatic axis was incorrectly performed. Although CVP is not adequate to assess intravascular volume, several used it in this context.





Acta Anaesthesiologica Scandinavica
AN INTERNATIONAL JOURNAL OF ANAESTHESIOLOGY AND INTENSIVE CARE, PAIN AND EMERGENCY MEDICINE

Regulating the cardiovascular system in intensive care and during surgery demands deliberation of which variables to monitor, their interpretation and therapeutic interventions.

The performance of the cardiovascular system is dependent on three states: effective intravascular volume, heart performance and vascular resistance. Each and every variable depends on each and every state. Central venous pressure (CVP) is inextricably linked to the correct measurement and physiological interpretation of all three states.[1–4] The clinician's dilemma is to derive the states from the variables, for guidance of (1) adding/removing fluid, changing compliance, and (2) administering cardioactive and (3) vasoactive medication or other interventions (e.g. pacing).

Since the publication of the Frank-Starling law, the volume state is often thought of in terms of preload measures, such as CVP and pulmonary capillary wedge pressure (PCWP). Contemporarily, the volume state is often thought of in terms of volume responsiveness, although this is foiled by a dozen caveats.[5,6] Clinicians have no agreed formality for discreet measurement of the volume state.

The lack of consensus has brought CVP into disrepute. A critical care manual stated: 'The CVP is a relic from the past and should never be measured in modern critical care medicine (except in acute cor pulmonale). The CVP and PCWP are no more useful than the "phases of the moon" in evaluating a patient's volume status'.[7] It has been pointed out, however, that '[t]hese points are true [that CVP cannot indicate volume responsiveness] but from the basic physiology it makes no sense even to ask these questions in the first place'.[8] On the background of such antagonism, we found it of interest to review the basic physiology governing the use of CVP in anaesthesia and intensive care for the purpose of cardiovascular regulation.

A review of the literature was complemented by a survey of clinicians to better understand how the physiological context of CVP is appreciated. We aimed first to highlight CVP and its relation to intravascular volume, cardiac performance and vascular resistance; second to illustrate its current clinical application; and third to identify clinical areas where the use of CVP for haemodynamic monitoring is warranted and could potentially be expanded. We argue that the clinical use of CVP can only be appreciated in the proper physiological context as emphasised in the title of this review.

## Methods

### Literature review and practice

First, the literature was reviewed based on a search of PubMed, Cochrane Database, Scopus, Web of Science, PubMed and authors' library using the terms 'central venous pressure' in combinations with 'measurement', 'physiology', 'cardiovascular system' and 'haemodynamics'. Publications after 1950 relevant to adult, human clinical practice were considered. Papers on insertion techniques, ultrasound examination and non-English papers were excluded.

Second, a survey was performed among members of the European Society of Intensive Care Medicine (ESICM) to assess the agreement between clinical use and physiological context relevant to CVP. The European Critical Care Research Network and the ESICM endorsed the survey and its distribution via the ESICM website for a duration of 3 months. The survey collected information on subspecialisation, age, professional experience, clinical indications for central venous catheter (CVC), procedures related to zeroing, levelling the system and interpreting CVP. A free text field captured optional comments. Descriptive statistics were used to describe the responses and the $\chi^2$ to test differences in proportions between groups based on age or length of experience.

## Results

### Literature review

The review identified 72 unique references from 1950 to present date (Fig. 1).

### How should CVP be measured?

The measurement of right atrial pressure (RAP/CVP) is performed using a CVC inserted via the axillary, subclavian, internal jugular, innominate, femoral or brachial vein (peripherally inserted central catheter, PICC), the position often verified

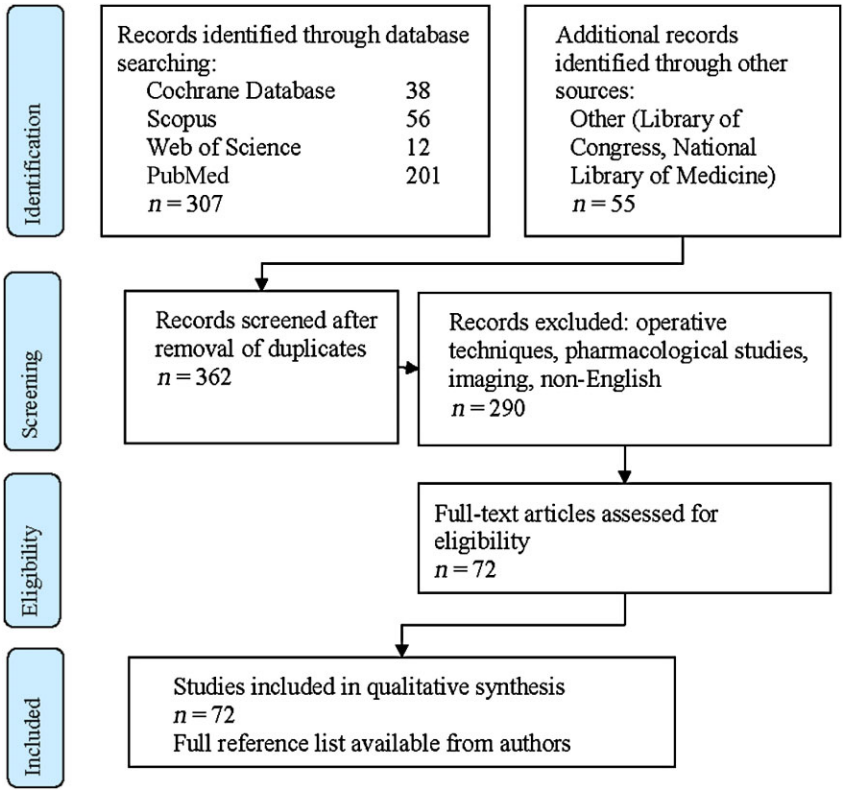


**Fig. 1.** Flow chart showing the number of articles identified and evaluated during the review process.

by X-ray. The measurement is obtained in or in proximity to the right atrium in the superior caval vein. Pressure and curve analysis can also be safely performed with PICC.[9]

The first step in obtaining RAP/CVP is the zeroing of the pressure transducer/amplifier to atmospheric pressure via a three-way stopcock. The second step involves alignment of the pressure transducer to the horizontal plane through the tricuspid valve, known as the phlebostatic axis. Traditionally, intensive care textbooks recommend levelling to the intersection of the mid-axillary line with the fourth intercostal space in the supine subject.[10]

In 1956, Guyton and Greganti investigated the localisation of the phlebostatic axis in dogs defined as the transducer position where change in pressure is minimal during changes in position in the three planes. This was found to be in the midline of the thorax, in a transverse plane 0.8 times the sternal notch-xiphoid tip distance and 0.4 times the anteroposterior (AP) distance within that transverse plane.[11] The position of the human tricuspid valve is remarkably similar (Fig. 2). A radiological study by Pedersen and Husby measured the position of the intersection of a cardiac catheter passing between the ostia of the superior and inferior caval veins in the sagittal fourth intercostal space, and reported this to be at 42.7% of the AP distance with a standard deviation (SD) of 2.9%.[12] Parkin (unpublished data) performed a CT-study to determine the position of the tricuspid valve and reported similar figures. Guyton reasoned that '[t]he apparent reason for such precise localization is that the heart operates as a feedback control system for controlling the end-diastolic pressure in the right ventricle; that is, increasing the end-diastolic pressure increases cardiac output, and this automatically returns the end-diastolic pressure back toward normal'.

If these reference points are identified, the vertical position of the pressure transducer may be *approximating* the change in position of the phlebostatic axis during change in patient position along longitudinal and transverse axes, recognising that the organs of the thoracic cavity may shift. Figg and Nemergut demonstrated how the positioning of the transducer by ICU staff using a supine mannequin had a variance of 4.3 (5.8) mmHg [SD (interquartile range)]. In 30° head up

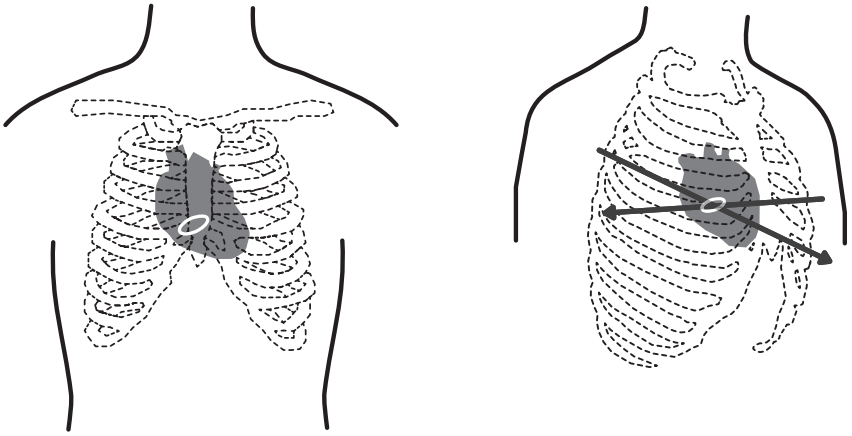

**Fig. 2.** Rib cage with arrows marking the external reference points of the position of the tricuspid valve. The phlebostatic axis is projected to surface anatomy in the midline and to the fourth intercostal space at approximately 40% of the anteroposterior diameter.

tilt, the displacement resulted in a variance of 6.5 (5.7) mmHg with comparable errors in the 15° Trendelenburg position. These were not diminished by the use of a laser level.[13]

## What factors influence CVP?

The midline position of the tricuspid valve implies the physiological advantage of CO being less dependent on rotation around the longitudinal axis. The CVP is an intravascular pressure influenced by changes in the transmural pressure and vessel distensibility. Hence, changes in pericardial, intrathoracic (positive end expiratory pressure, PEEP) and intra-abdominal pressure, vascular resistance and compliance, blood volume, and cardiac pump function all impact on CVP.[14] Measurement of the transmural pressure corrects the effective preload when external pressures are taken into account for the performance of the heart. Such approaches are inherently complex given that preload is itself volume-, resistance- and heart-dependent, and only qualitatively related to the circulatory dynamics.

A more productive and quantitative approach results from using CVP as the lowest pressure point in the circulation at the initiation of ejection of right ventricle (RV) stroke volume when RV pressure exceeds CVP. As backpressure to venous return (VR), we need but consider its absolute value, transmural pressures are not relevant. This allows one to numerically define the effective volume state and the state of heart performance. By the volume state we imply the mean pressure in the systemic circulation, $P_{ms}$, exerted by the volume of blood when the heart is stopped.

## What is the position of CVP in the interpretation of haemodynamics?

An analogue of $P_{ms}$ may be calculated using

$$P_{msa} = a \times CVP + b \times MAP + c \times CO \qquad (1)$$

(MAP: mean arterial pressure, a (= 0.96), b (= 0.04) are dimensionless, c (= 0.3–1.2) is an anthropometrical variable based on age, height and weight).

Since $a \approx 1$, the CVP is essentially additive to the volume state in the manner of a floating electrical ground.[15] Only when the heart is stopped does CVP measure the volume state.*

At steady state, CO equals the VR. The difference between the $P_{msa}$, the volume state and the CVP is the pressure gradient for VR. When the resistance to venous return (RVR) is considered, VR is given by

$$VR = \frac{P_{msa} - CVP}{RVR} \qquad (2)$$

Equation (2) dictates that VR (and CO) can be increased by increasing $P_{msa}$ or decreasing CVP and/or RVR. This is commonly achieved using fluids or a venous vasopressor (increasing $P_{msa}$), an inotrope (lowering CVP) or an inodilator (combining lowering CVP and reducing RVR). Understanding RVR as the resistance encountered by the average element in the circulation in

*Taking an example, for CVP = 0, MAP = 100, c = 0.6 and CO = 5, $P_{msa}$ = 0 + 4 + 3 = 7. If the heart is stopped, CVP = 7, MAP = 7 and CO = 0, $P_{msa}$ = 6.72 + 0.28 + 0 = 7 showing that $P_{ms}$ is unaffected by stopping the heart.

returning to the heart also explains why a vasoconstrictor may reduce VR/CO by increasing RVR, and conversely why adenosine, a potent arterial vasodilator, increases CO by decreasing RVR. While most of the average elements are in the veins and only have to cross the venous resistance, some are in the arteries and have to cross both the arterial and venous resistance.[16]

The gradient ($P_{msa}$ – CVP) is thus pivotal in the realisation why CVP alone will never be indicative of volume or change in volume. The possible outcomes of a fluid bolus from its effect on ($P_{msa}$ – CVP) include an increase in CO if $\Delta P_{msa} > \Delta CVP$, no change if $\Delta P_{msa} \approx \Delta CVP$ and a decrease if $\Delta CVP > \Delta P_{msa}$. This emphasises the double role of the CVP as distending RV (Starling mechanism) and counteracting VR. Several reviews have been published on the physiological concept of VR[17–19], and Cecconi et al. and Gupta et al. have demonstrated this physiological relationship.[20,21]

To further clarify how the heart converts the difference ($P_{msa}$ – CVP) into a global cardiac function, Parkin et al. formulated the quantitative measure of heart efficiency, $E_h$

$$E_h = \frac{P_{msa} - CVP}{P_{msa}}, (0 \le E_h \le 1) \quad (3)$$

## Is there a role for CVP in assessing volume responsiveness?

Fluid optimisation refers to the iterated infusion of boluses to increase CO. A binary approach is applied based on $\Delta CO > 10–15\%$ denoting a responsive state. The abundance of reports using dynamic variables to distinguish fluid responsiveness, i.e. pulse pressure (PPV) and stroke volume variation, relies on the 10–15% increase in CO to define their cut-off values for predictive power. While such binary approaches to defining a volume responsive state appear simple at the bedside and have enjoyed widespread clinical acceptance, the physiological rationale remains debatable.[5] In contemplating volume responsiveness (or rather *volume efficiency*), we are interested in the effect upon $\Delta(P_{msa} - CVP)$ of a volume change, $\Delta P_{msa}$. This leads to a dimensionless variable

$$E_{vol} = \frac{\Delta(P_{msa} - CVP)}{\Delta P_{msa}}, (0 \le E_{vol} \le 1) \quad (4)$$

This concept is attractive for many reasons. It provides a continuous, dimensionless signal in the interval 0–1. $E_{vol}$ relies on sound physiological principles clinically valid irrespective of breathing pattern, airway pressures or volumes, and cardiorespiratory rate and rhythm. It provides guidance whether to use fluids or other means to increase flow, and moves focus on CO from assessment of instantaneous agreement (where 10–15% is the least detectable difference[22]) to trending capability of continuous CO equipment. The gross correspondence between $E_{vol}$ and PPV is illustrated in the study by Cecconi et al. and Gupta et al. showing an $E_{vol}$ of 0.36 and 0.32 in the volume responding group vs. 0.07 and 0.03 in the non-responding group.[20,21]

### Survey results

We recorded 450 unique responses with 53% of respondents working in intensive care and 35% having combined duties in anaesthesia and intensive care. Half of the respondents worked in tertiary hospitals. Clinical indications allocated the CVC as access route for administration of cardioactive, vasoactive and vasoirritant drugs, including intravenous nutrition (87–93% of respondents). As a monitoring device, 78% obtained venous oxygen saturation ($S_{cv}O_2$) for haemodynamic monitoring, 61% measured CVP to guide volume resuscitation, and 21–36% used CVP incorporated in the VR physiology. Monitoring of right ventricular function was stated by 31% of respondents.

### How should CVP be measured?

The identification of the phlebostatic axis by the respondents is illustrated in Fig. 3. Only 3.4% of respondents identified the phlebostatic axis at 40–50% of AP diameter. A majority corrected the position of the pressure transducer to the level of the heart following changes in body position, while 12% ignored correcting the position as the change in CVP was thought to be minimal.

### Correction of factors influencing CVP

Thirty-five per cent of respondents corrected CVP during positive pressure ventilation and PEEP, most commonly (46% of respondents) by adding

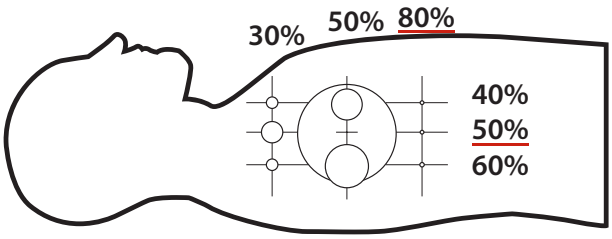


**Fig. 3.** Torso illustrating respondent's positioning of phlebostatic axis. The size of the circles represents the frequency of response in each of the nine coordinates. The majority is found at 50% of anteroposterior diameter/50% of sternal length. Exact numbers are given in the Supporting Information. The correct position at 40–50% of anteroposterior diameter/80% of sternal length was indicated by 3.4% of respondents.

a fraction of PEEP (range 0.3–0.8) and alternatively (35% of respondents) by measuring CVP at zero-end expiratory pressure by disconnecting the ventilatory circuit.

### What is the haemodynamic importance of the CVP?

More than 40% of respondents stated that CVP had no relation to intravascular volume state or changes in volume. There were no significant differences concerning the use of CVP to gauge the volume state between respondents according to length of experience. Between 40% and 50% of respondents still used CVP as a guide for fluid resuscitation, in the absence of a CO monitor, frequently as part of the Surviving Sepsis Campaign (SSC) resuscitation bundle. Elderly (61–70 years) colleagues demonstrated greater proclivity to use CVP for guiding fluid therapy without access to CO measurements compared with young (30–40 years) and medium age (41–60 years) colleagues.

Detailed information on the survey results are reported in the Supporting Information.

## Discussion

There was significant variability in the identification of the phlebostatic axis and the redundancy in accounting for transmural pressure when measuring CVP. The survey suggested that approximately 50% of respondents had a measurement error of 2 mmHg, while 25% had an error of 4 mmHg in the sagittal plane. If the patient were to be tilted 15° with an umbilical centre of rotation, this would introduce errors of 2–4 mmHg in some cases and correct errors in others. The 2–4 mmHg difference may seem negligible, but in the context of VR physiology it amounts to ¼–½ of the pressure gradient from the periphery to the right atrium.

Clinical practice examples may serve to emphasise the importance of correctly measuring CVP. In the anaesthetic management of hepatic surgery, a low CVP regime is often applied to decrease bleeding during the resection phase by enhancing flow through intact liver sinusoids towards the right atrium, as well as lowering pressure gradients towards the transected surfaces.[23,24] In thoracoabdominal aneurysm repair, the association between high CVP, low MAP and the occurrence of paraplegia testifies to the detrimental effect of low perfusion pressure.[25] In orthopaedic and neurosurgery, performed in beach chair position, CVP control is used to counteract the risk of venous air emboli.[26] The importance of correctly measuring CVP is obvious in these situations, and future solutions may eventually involve the use of 3D positioning systems based on external or internal markers.[27]

The survey demonstrated that many clinicians doubt the usefulness of CVP in cardiovascular management but still use it. A minority of senior clinicians indicated an understanding of the physiology behind VR and hence appreciated the need to view CVP in the context of $P_{ms}$.

It is thus necessary to ascertain the $P_{ms}$ to put CVP into physiological context. The concept of mean systemic filling pressure was introduced by Weber.[28] Starling revived the concept in his lecture at the Royal College of Surgeons of England in February 1897: 'It thus follows that the neutral point in the vascular system, where the mean systemic pressure is neither raised nor lowered by the inauguration of the circulation, lies considerably on the venous side of the capillaries . . .',[23] and summarised his Law of the Heart in the Linacre lecture 1915 in Cambridge.[24] Half a century later, Guyton demonstrated the importance of $P_{ms}$ as the measure of the relationship between stressing volume and venous capacitance, based on a series of animal experiments.[29–32] In these experiments, $P_{ms}$ was measured as the equilibrium between arterial and venous pressures during circulatory

standstill following induced ventricular fibrillation. An alternative, clinically feasible method has been described using coincident measurements of CO and CVP during stepwise increases in inspired tidal volume with a short-end inspiratory pause. The increased intrathoracic pressure induces an increase in CVP, and thus decreases in ($P_{ms}$-CVP) and CO. By extrapolating the measurements to the CVP at zero CO, the intersection of the abscissa defines the $P_{ms}$.[33] This method has been further evaluated in patients admitted to the intensive care unit.[34,35] It is suited to sedated patients in controlled ventilation where the excitation of the cardiovascular system can be performed but is not possible to perform in awake, spontaneously breathing patients.

The SSC recommends CVP for volume resuscitation in combination with $S_{cv}O_2$ as a 'physiologic target for resuscitation'. The guideline summarises: 'Although there are limitations to CVP as a marker of intravascular volume status and response to fluids, a low CVP generally can be relied upon as supporting positive response to fluid loading'. The guidelines mirror the shattered interpretation of CVP if it is not realised that it is, basically, a downstream pressure for $P_{ms}$.

Recently, the SSC recommendation of CVP as a resuscitation goal has been linked to the relationship between increased CVP and acute kidney injury.[36] The role of $P_{ms}$ is still under debate as it seems difficult to change the cardiovascular paradigm from the Starling cardiocentric to the Guyton histocentric view. The derivation of $P_{msa}$ is based on a cardiovascular model consisting of CO, MAP, CVP, and arterial and venous compartments characterised by their compliances and resistances. This model has been incorporated into a clinical decision support system to provide a physiologically consistent, comprehensive and predictive cardiovascular model.[15] The model is critically dependent on correct measurement of CVP, CO and MAP, and has been evaluated in intraoperative and intensive care settings.[37,38]

## Conclusion

The literature review demonstrated that CVP is of paramount importance in the understanding and management of cardiovascular physiology combining the cardiac function curve of Starling and the VR curve of Guyton, and is hence of vital importance in circumscribed intraoperative and intensive care cases.

The knowledge and use of CVP among clinicians were not in concordance with the reviewed physiology of CVP. On the basis of literature and questionnaire results, it is suggested that educational efforts relevant to CVP are launched.

## Acknowledgements

The assistance of Guy Francois, Division of Scientific Affairs, Research, ESICM, in managing the questionnaire and of Johanna Åneman in providing the illustrations are greatly appreciated.

## References

1. Cecconi M, Aya HD. Central venous pressure cannot predict fluid-responsiveness. Evid Based Med 2014; 19: 63.
2. Osman D, Ridel C, Ray P, Monnet X, Anguel N, Richard C, Teboul JL. Cardiac filling pressures are not appropriate to predict hemodynamic response to volume challenge. Crit Care Med 2007; 35: 64–8.
3. Cecconi M, Monti G, Hamilton MA, Puntis M, Dawson D, Tuccillo ML, Della Rocca G, Grounds RM, Rhodes A. Efficacy of functional hemodynamic parameters in predicting fluid responsiveness with pulse power analysis in surgical patients. Minerva Anestesiol 2012; 78: 527–33.
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.
5. Mahjoub Y, Lejeune V, Muller L, Perbet S, Zieleskiewicz L, Bart F, Veber B, Paugam-Burtz C, Jaber S, Ayham A, Zogheib E, Lasocki S, Vieillard-Baron A, Quintard H, Joannes-Boyau O, Plantefeve G, Montravers P, Duperret S, Lakhdari M, Ammenouche N, Lorne E, Slama M, Dupont H. Evaluation of pulse pressure variation validity criteria in critically ill patients: a prospective observational multicentre point-prevalence study. Br J Anaesth 2014; 112: 681–5.
6. Sondergaard S. Pavane for a pulse pressure variation defunct. Crit Care 2013; 17: 327–32.
7. Marik PE. Handbook of evidence-based critical care. New York: Springer, 2010.
8. Magder S. Hemodynamic monitoring in the mechanically ventilated patient. Curr Opin Crit Care 2011; 17: 36–42.

9. 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.
10. Tobin MJ. Principles and practice of intensive care monitoring. New York: McGraw-Hill, Health Professions Division, 1998.
11. Guyton AC, Greganti FP. A physiologic reference point for measuring circulatory pressures in the dog; particularly venous pressure. Am J Physiol 1956; 185: 137–41.
12. Pedersen A, Husby J. Venous pressure measurement. I. Choice of zero level. Acta Med Scand 1951; 141: 185–94.
13. Figg KK, Nemergut EC. Error in central venous pressure measurement. Anesth Analg 2009; 108: 1209–11.
14. Gelman S. Venous function and central venous pressure: a physiologic story. Anesthesiology 2008; 108: 735–48.
15. Parkin WG, Leaning MS. Therapeutic control of the circulation. J Clin Monit Comput 2008; 22: 391–400.
16. Zall S, Milocco I, Ricksten SE. Effects of adenosine on myocardial blood flow and metabolism after coronary artery bypass surgery. Anesth Analg 1991; 73: 689–95.
17. Funk DJ, Jacobsohn E, Kumar A. The role of venous return in critical illness and shock-part I: physiology. Crit Care Med 2013; 41: 255–62.
18. Funk DJ, Jacobsohn E, Kumar A. Role of the venous return in critical illness and shock: part II-shock and mechanical ventilation. Crit Care Med 2013; 41: 573–9.
19. Jacobsohn E, Chorn R, O'Connor M. The role of the vasculature in regulating venous return and cardiac output: historical and graphical approach. Can J Anaesth 1997; 44: 849–67.
20. Cecconi M, Aya HD, Geisen M, Ebm C, Fletcher N, Grounds RM, Rhodes A. Changes in the mean systemic filling pressure during a fluid challenge in postsurgical intensive care patients. Intensive Care Med 2013; 39: 1299–305.
21. Gupta K, Sondergaard S, Parkin G, Leaning M, Aneman A. Applying mean systemic filling pressure to assess the response to fluid boluses in cardiac post-surgical patients. Intensive Care Med 2015; doi: 10.1007/s00134-014-3611-2.
22. Nilsson LB, Nilsson JC, Skovgaard LT, Berthelsen PG. Thermodilution cardiac output–are three injections enough? Acta Anaesthesiol Scand 2004; 48: 1322–7.
23. Starling EH. Some points in the pathology of heart disease. Lecture II. Lancet 1897; March 6: 652–5.
24. Starling EH, Chapman CB. Starling on the heart. Facsimile reprints including the Linacre Lecture on the Law of the Heart. Analysis and critical comment Carleton B. Chapman and Jere H. Mitchell. London: Dawson, 1965: 121–47.
25. Etz CD, Luehr M, Kari FA, Bodian CA, Smego D, Plestis KA, Griepp RB. Paraplegia after extensive thoracic and thoracoabdominal aortic aneurysm repair: does critical spinal cord ischemia occur postoperatively? J Thorac Cardiovasc Surg 2008; 135: 324–30.
26. Krier C, Wiedemann K. Luftembolie. Eine Komplikation bei neurochirurgischen Eingriffen in sitzender position. Prakt Anaesth 1978; 13: 386–97.
27. Parkin J, Boyd C. Position monitoring apparatus. In Organization WIP, ed. WO 2005/032364 A8 edn. Australia, 2005.
28. Weber EH. Ueber die Anwendung der Wellenlehre auf die Lehre vom Kreislaufe des Blutes und insbesondere auf die Pulslehre. Berichte über die Verhandlungen der Königlich Sächsischen Gesellschaft der Wissenschaften zu Leipzig: Mathematisch-Physische Klasse 1850; 2: 164–204.
29. Guyton AC. Determination of cardiac output by equating venous return curves with cardiac response curves. Physiol Rev 1955; 35: 123–9.
30. Guyton AC, Lindsey AW, Kaufmann BN. Effect of mean circulatory filling pressure and other peripheral circulatory factors on cardiac output. Am J Physiol 1955; 180: 463–8.
31. Guyton AC, Polizo D, Armstrong GG. Mean circulatory filling pressure measured immediately after cessation of heart pumping. Am J Physiol 1954; 179: 261–7.
32. Guyton AC, Lindsey AW, Abernathy B, Richardson T. Venous return at various right atrial pressures and the normal venous return curve. Am J Physiol 1957; 189: 609–15.
33. Den Hartog EA, Versprille A, Jansen JR. Systemic filling pressure in intact circulation determined on basis of aortic vs. central venous pressure relationships. Am J Physiol 1994; 267: H2255–8.
34. Maas JJ, Geerts BF, van den Berg PC, Pinsky MR, Jansen JR. Assessment of venous return curve and mean systemic filling pressure in postoperative cardiac surgery patients. Crit Care Med 2009; 37: 912–8.
35. Maas JJ, Pinsky MR, Geerts BF, de Wilde RB, Jansen JR. Estimation of mean systemic filling

pressure in postoperative cardiac surgery patients with three methods. Intensive Care Med 2012; 38: 1452–60.
36. Legrand M, Dupuis C, Simon C, Gayat E, Mateo J, Lukaszewicz AC, Payen D. Association between systemic hemodynamics and septic acute kidney injury in critically ill patients: a retrospective observational study. Crit Care 2013; 17: R278.
37. Pellegrino VA, Mudaliar Y, Gopalakrishnan M, Horton MD, Killick J, Parkin WG, Playfordh HR, Raper RF. Computer based haemodynamic guidance system is effective and safe in management of postoperative cardiac surgery patients. Anaesth Intensive Care 2011; 39: 191–201.
38. Sondergaard S, Wall P, Cocks K, Parkin WG, Leaning MS. High concordance between expert anaesthetists' actions and advice of decision support system in achieving oxygen delivery targets in high-risk surgery patients. Br J Anaesth 2012; 108: 966–72.

## Supporting Information

Additional Supporting Information may be found in the online version of this article at the publisher's web-site:

**Fig. S1.** Percentage of each group's use of CVP in haemodynamic monitoring according to length of experience. In each triad, the first column represents experience 0–10 years, the second 11–20 years and the third 21– > 30 years.
**Fig. S2.** Percentage of each group's use of CVP in haemodynamic monitoring according to length of experience. In each triad, the first column represents experience 0–10 years, the second 11–20 years and the third 21– > 30 years.
**Table S1.** Geographical provenience of respondents.
**Table S2.** Gender and age distribution of respondents. Numbers in parentheses indicate percentage of members of ESICM belonging to the age/gender-combination.
**Table S3.** Distribution of workplaces.
**Table S4.** Distribution of length of experience among respondents.
**Table S5.** Percentage distribution of localisation of phlebostatic axis.
**Table S6.** Percentage distribution of phlebostatic axis according to length of experience.
**Appendix S1.** ESICM webpage announcement of CVP questionnaire.