

CHEST[®]

Official publication of the American College of Chest Physicians



Does Central Venous Pressure Predict Fluid Responsiveness?: A Systematic Review of the Literature and the Tale of Seven Mares

Paul E. Marik, Michael Baram and Bobbak Vahid

Chest 2008;134;172-178
DOI 10.1378/chest.07-2331

The online version of this article, along with updated information and services can be found online on the World Wide Web at:
<http://chestjournal.org/cgi/content/abstract/134/1/172>

CHEST is the official journal of the American College of Chest Physicians. It has been published monthly since 1935. Copyright 2007 by the American College of Chest Physicians, 3300 Dundee Road, Northbrook IL 60062. All rights reserved. No part of this article or PDF may be reproduced or distributed without the prior written permission of the copyright holder (<http://www.chestjournal.org/misc/reprints.shtml>). ISSN: 0012-3692.

A M E R I C A N C O L L E G E O F



P H Y S I C I A N S[®]



Does Central Venous Pressure Predict Fluid Responsiveness?*

A Systematic Review of the Literature and the Tale of Seven Mares

Paul E. Marik, MD, FCCP; Michael Baram, MD, FCCP; and Bobbak Vahid, MD

Background: Central venous pressure (CVP) is used almost universally to guide fluid therapy in hospitalized patients. Both historical and recent data suggest that this approach may be flawed.

Objective: A systematic review of the literature to determine the following: (1) the relationship between CVP and blood volume, (2) the ability of CVP to predict fluid responsiveness, and (3) the ability of the change in CVP (Δ CVP) to predict fluid responsiveness.

Data sources: MEDLINE, Embase, Cochrane Register of Controlled Trials, and citation review of relevant primary and review articles.

Study selection: Reported clinical trials that evaluated either the relationship between CVP and blood volume or reported the associated between CVP/ Δ CVP and the change in stroke volume/cardiac index following a fluid challenge. From 213 articles screened, 24 studies met our inclusion criteria and were included for data extraction. The studies included human adult subjects, healthy control subjects, and ICU and operating room patients.

Data extraction: Data were abstracted on study design, study size, study setting, patient population, correlation coefficient between CVP and blood volume, correlation coefficient (or receive operator characteristic [ROC]) between CVP/ Δ CVP and change in stroke index/cardiac index, percentage of patients who responded to a fluid challenge, and baseline CVP of the fluid responders and nonresponders. Metaanalytic techniques were used to pool data.

Data synthesis: The 24 studies included 803 patients; 5 studies compared CVP with measured circulating blood volume, while 19 studies determined the relationship between CVP/ Δ CVP and change in cardiac performance following a fluid challenge. The pooled correlation coefficient between CVP and measured blood volume was 0.16 (95% confidence interval [CI], 0.03 to 0.28). Overall, $56 \pm 16\%$ of the patients included in this review responded to a fluid challenge. The pooled correlation coefficient between baseline CVP and change in stroke index/cardiac index was 0.18 (95% CI, 0.08 to 0.28). The pooled area under the ROC curve was 0.56 (95% CI, 0.51 to 0.61). The pooled correlation between Δ CVP and change in stroke index/cardiac index was 0.11 (95% CI, 0.015 to 0.21). Baseline CVP was 8.7 ± 2.32 mm Hg [mean \pm SD] in the responders as compared to 9.7 ± 2.2 mm Hg in nonresponders (not significant).

Conclusions: This systematic review demonstrated a very poor relationship between CVP and blood volume as well as the inability of CVP/ Δ CVP to predict the hemodynamic response to a fluid challenge. CVP should not be used to make clinical decisions regarding fluid management.

(CHEST 2008; 134:172–178)

Key words: anesthesia; blood volume; central venous pressure; fluid responsiveness; fluid therapy; hemodynamic monitoring; ICU; preload; stroke volume

Abbreviations: AUC = area under the curve; CI = confidence interval; CVP = central venous pressure; Δ CVP = change in central venous pressure; ROC = receiver operator characteristic

Central venous pressure (CVP) is the pressure recorded from the right atrium or superior vena cava. CVP is measured (usually hourly) in almost all patients in ICUs throughout the world, in emergency

department patients, well as in patients undergoing major surgery. CVP is frequently used to make decisions regarding the administration of fluids or diuretics. Indeed, internationally endorsed clinical

guidelines¹ recommend using CVP as the end point of fluid resuscitation. The basis for using CVP to guide fluid management comes from the dogma that CVP reflects intravascular volume; specifically, it is widely believed that patients with a low CVP are volume depleted while patients with a high CVP are volume overloaded. This concept is taught to medical students as well as to residents and fellows across a wide range of medical and surgical disciplines. Indeed an authoritative textbook² of cardiovascular physiology states as a key concept that “[the] central venous pressure gives clinically relevant information about circulatory [and volume] status.” The chapter on cardiovascular monitoring in a standard anesthesiology text³ states that “the most important application of CVP monitoring is to provide an estimate of the adequacy of circulating blood volume”, and “[that] trends in CVP during anesthesia and surgery are also useful in estimating fluid or blood loss and guiding replacement therapy.” Over 25 years ago, the “5–2” rule for guiding fluid therapy was popularized.⁴ According to this rule, the change in CVP following a fluid challenge is used to guide subsequent fluid management decisions. This rule is still widely used today. Recently, the idea that the CVP reflects blood volume has been challenged. Since CVP plays such a central role in the fluid management strategy of hospitalized patients, the goal of this study was to systemically review the evidence that supports this practice.

MATERIALS AND METHODS

Identification of Trials

Our aim was to identify all relevant clinical trials that analyzed the relationship between CVP and measured blood volume as well as those studies that determined the ability of CVP to predict fluid responsiveness (*ie*, an increase in stroke index/cardiac index following a fluid challenge). Studies that compared CVP with volumetric measurements (right and left ventricular end-diastolic volumes, global left heart volume, central blood volume) but did not report the ability of CVP to predict volume responsiveness were not included. We restricted this analysis to human adults; however, there was no restriction as to the type of patient or the setting where the

study was performed. We used a multimethod approach to identify relevant studies for this review. All authors independently searched the National Library of Medicine MEDLINE database for relevant studies in any language published from 1966 to June 2007 using the following medical subject headings and key words; central venous pressure (explode) AND blood volume, or fluid therapy or fluid responsiveness. In addition, we searched Embase and the Cochrane Database of Systematic Reviews. Bibliographies of all selected articles and review articles that included information on hemodynamic monitoring were reviewed for other relevant articles. In addition, the authors reviewed their personal files and contacted experts in the field. This search strategy was done iteratively until no new potential citations were found on review of the reference lists of retrieved articles. We performed this metaanalysis according to the guidelines proposed by the Quality of Reporting of Meta-analyses group.⁵

Study Selection and Data Extraction

Only studies that reported either of the following were included in this analysis: (1) the correlation coefficient between CVP and measured blood volume, or (2) the correlation coefficient or receiver operator characteristic (ROC) between CVP or change in CVP (Δ CVP) and change in stroke index/cardiac output following a fluid challenge. All authors independently abstracted data from all studies using a standardized form. Data were abstracted on study design, study size, study setting, patient population, correlation coefficients and area (including 95% confidence intervals [CIs]) under the ROC curve, the percentage of patients responding to a fluid challenge as well as the baseline CVP in the fluid responders and nonresponders. In general, an increase in the stroke index or cardiac index > 10 to 15% was used as an index of fluid responsiveness.^{6,7}

The random-effects models (Comprehensive Meta-analysis 2.0; Biostat; Englewood NJ) was used to determine the pooled area under the curve (AUC) and correlation coefficients.^{8,9} Summary effects estimates are presented with 95% CIs. We calculated the Cochran Q statistic to test for statistical heterogeneity. Values of Q significantly > 0 ($p < 0.1$) were considered evidence of heterogeneity. When not reported in the primary paper, the correlation coefficients were calculated from the raw data (when available) [NCSS 2007; NCSS; Kaysville, UT].

RESULTS

The initial search strategy generated 206 citations; of these, 189 were excluded due to trial design or failure to report an outcome variables of interest. An additional seven studies were identified from the bibliographies of the selected articles and review articles. Of the 24 studies included in this analysis, 5 studies compared CVP with the measured circulating blood volume while 19 studies determined the relationship between CVP and change in cardiac performance following a fluid challenge (generally defined as a > 10 to 15% increase in stroke index/cardiac index). In all, 830 patients across a spectrum of medical and surgical disciplines were studied^{10–33} (Tables 1, 2). In three studies,^{10,15,33} the correlation coefficients were not reported in the article but were calculated from the raw data.

*From the Division of Pulmonary and Critical Care Medicine, Thomas Jefferson University, Philadelphia, PA.

The authors have no financial conflicts of interest in any of the products mentioned in this article.

Manuscript received September 20, 2007; revision accepted November 14, 2007.

Reproduction of this article is prohibited without written permission from the American College of Chest Physicians (www.chestjournal.org/misc/reprints.shtml).

Correspondence to: Paul Marik, MD, FCCP, Chief, Pulmonary and Critical Care Medicine, Thomas Jefferson University, 834 Walnut St, Suite 650, Philadelphia, PA 19107; e-mail: paul.marik@jefferson.edu
DOI: 10.1378/chest.07-2331

Table 1—Summary of Studies of Blood Volume*

Source	Setting	Type	Patients, No.	Methodology	r, Blood Volume
Baek et al, ¹⁰ 1975	ICU	General surgery	69	¹²⁵ I-albumin	0.19
Shippy et al, ¹¹ 1984	ICU	ICU	118	¹²⁵ I-albumin	0.27
Hoefl et al, ¹² 1994	OR/ICU	CABG	11	Indocyanine green	0.12
Oohashi et al, ¹³ 2005	ICU	Esophagectomy	16	Indocyanine green	0.17
Kuntscher et al, ¹⁴ 2006	ICU	Burns	16	COLD system†	0.02
Pooled value					0.16

*OR = operating room; CABG = coronary artery bypass graft surgery.

†COLD Z-021 system (Pulsion Medical Systems; Munich, Germany).

The pooled correlation coefficient between the CVP and measured blood volume was 0.16 (95% CI, 0.03 to 0.28; $r^2 = 0.02$). Heterogeneity was present between studies. Figure 1 illustrates the relationship between CVP and measured blood volume from the study of Shippy et al.¹¹ Overall $56 \pm 16\%$ (mean \pm SD) of the patients included in this review responded to a fluid challenge. The pooled correlation coefficient between baseline CVP and change in stroke index/cardiac index (reported in 10 studies) was 0.18 (95% CI, 0.08 to 0.28). The pooled area under the ROC curve (reported in 10 studies) was 0.56 (95% CI, 0.51 to 0.61). The pooled correlation between Δ CVP and change in stroke index/cardiac index (reported in

seven studies) was 0.11 (95% CI, 0.01 to 0.21). The baseline CVP (reported in 11 studies) was 8.7 ± 2.3 mm Hg in the responders, as compared to 9.7 ± 2.2 mm Hg in nonresponders (not significant; $p = 0.3$). The Q statistic was not significant for the pooled correlation and area under the curve statistic.

DISCUSSION

The results of this systematic review are clear: (1) there is no association between CVP and circulating blood volume, and (2) CVP does not predict fluid responsiveness across a wide spectrum of clinical

Table 2—Summary of Studies of Volume Challenge*

Source	Setting	Type	Patients, No.	Methodology	AUC†	r, CVP/SI	r, Δ CVP/SI	CVP-R	CVP-NR
Calvin et al, ¹⁵ 1981	ICU	Mixed ICU	28	PAC/Scint		0.16	0.26	4.7	4.8
Reuse et al, ¹⁶ 1990	ICU	ICU	41	PAC		0.21		8.5	8.4
Godje et al, ¹⁷ 1998	ICU	CABG	30	PAC, COLD system‡			0.09		
Wagner and Leatherman, ¹⁸ 1998	ICU	ICU	25	PAC		0.44		7.4	10.1
Wiesenack et al, ¹⁹ 2001	OR	CABG	18	PAC, TPT			0.09		
Berkenstad et al, ²⁰ 2001	OR	Neurosurgery	15	TPT	0.49	0.05	0.08	9.3	9.3
Michard et al, ²¹ 2000	ICU	ICU	40	PAC	0.51				
Reuter et al, ²² 2002	ICU	CABG	20	TPT	0.42				
Reuter et al, ²³ 2003	ICU	CABG	26	PAC, TEE	0.71				
Barbier et al, ²⁴ 2004	ICU	Sepsis	20	TEE	0.57			10	9
Kramer et al, ²⁵ 2004	ICU	CABG	21	PAC	0.49	0.13		13.5	13.3
Marx et al, ²⁴ 2004	ICU	Sepsis	10	PAC, TPT		0.41	0.28		
Preisman et al, ²⁷ 2005	OR	CABG	18	TPT, TEE	0.61			8.7	10
Perel et al, ²⁸ 2005	ICU	Vascular surgery	14	TEE		0.27		9.6	12.2
Hofer et al, ²⁹ 2005	OR	CABG	40	PAC, TEE	0.54	0.02	0.2		
De Backer et al, ³⁰ 2005	ICU	ICU	60	PAC	0.54			10	12
Kumar et al, ³¹ 2004	ICU	Healthy volunteers	12	PAC/Scint		0.32	0.22		
Osman et al, ³² 2007	ICU	Septic	96	PAC	0.58			8	9
Magder and Bafaqeeh, ³³ 2007	ICU	CABG	66	PAC		0.36		5.9	8.7
Pooled					0.56	0.18	0.11	8.7	9.7

*PAC = pulmonary artery catheter; TEE = transesophageal echocardiography; Scint = radionuclide scintigraphy; TPT = transpulmonary thermodilution; CVP-R = baseline CVP of responders; CVP-NR = baseline CVP of nonresponders; SI = fluid responsiveness; see Table 1 for expansion of abbreviations.

†Area under ROC curve of CVP and fluid responsiveness.

‡COLD Z-021 system (Pulsion Medical Systems; Munich, Germany).

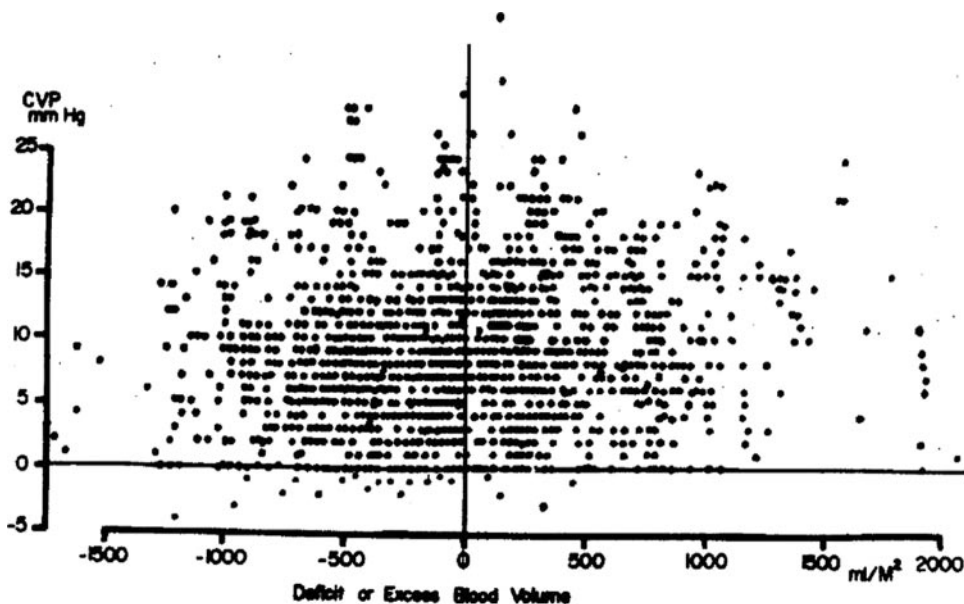


FIGURE 1. Fifteen hundred simultaneous measurements of blood volume and CVP in a heterogeneous cohort of 188 ICU patients demonstrating no association between these two variables ($r = 0.27$). The correlation between Δ CVP and change in blood volume was 0.1 ($r^2 = 0.01$). This study demonstrates that patients with a low CVP may have volume overload and likewise patients with a high CVP may be volume depleted. Reproduced with permission from Shippy et al.¹¹

conditions. In none of the studies included in this analysis was CVP able to predict either of these variables. Indeed, the pooled area under the ROC curve was 0.56. The ROC curve is a statistical tool that helps assess the likelihood of a result being a true positive vs a false positive. As can be seen from Figure 2, an ROC of 0.5 depicts the true-positive rate equal to the false-positive rate; graphically, this is represented by the straight line in Figure 1. The higher the AUC, the greater the diagnostic accuracy of a test. Ideally, the AUC should be between 0.9 to

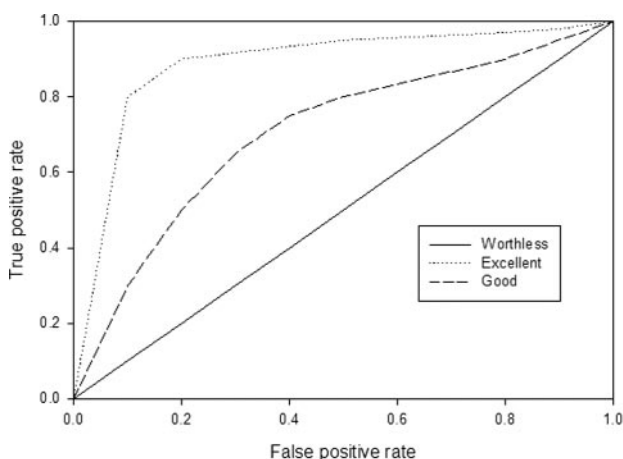


FIGURE 2. Comparison of ROC curves showing tests with different diagnostic accuracies.

1 (0.8 to 0.9 indicates adequate accuracy with 0.7 to 0.8 being fair, 0.6 to 0.7 being poor, and 0.5 to 0.6 indicating failure). In other words, our results suggest that at any CVP the likelihood that CVP can accurately predict fluid responsiveness is only 56% (no better than flipping a coin). Furthermore, an AUC of 0.56 suggests that there is no clear cutoff point that helps the physician to determine if the patient is “wet” or “dry.” It is important to emphasize that a patient is equally likely to be fluid responsive with a low or a high CVP (Fig 1). The results from this study therefore confirm that neither a high CVP, a normal CVP, a low CVP, nor the response of the CVP to fluid loading should be used in the fluid management strategy of any patient.

The strength of our review includes the rigorous selection criteria used to identify relevant studies as well as the use of quantitative end points.^{8,9,34} Furthermore, the studies are notable for the consistency (both in magnitude and direction) of their findings. This suggests that the findings are likely to be true.^{8,9,34} The results of our study are most disturbing considering that 93% of intensivists report using CVP to guide fluid management.³⁵ It is likely that a similar percentage (or more) of anesthesiologists, nephrologists, cardiologists, and surgeons likewise use CVP to guide fluid therapy. It is important to note that none of the studies included in our analysis took the positive end-expiratory pressure levels or changes in intrathoracic pressure into account when

recording CVP. This is important because right ventricular filling is dependent on the transmural right atrial pressure gradient rather than the CVP alone.³⁶ However, in the real world, transmural filling pressures are rarely if ever calculated.

As demonstrated by this study, only about a half of patients administered a fluid bolus will demonstrate a positive hemodynamic response to the intervention. With an ROC of 0.56, the play of chance (or a dice) will be as helpful as CVP in predicting which patients will respond to a fluid challenge. If fluid resuscitation is guided by CVP, it is likely that patients will have volume overload and pulmonary edema. Indeed the practice parameters for hemodynamic support of sepsis in adult patients concludes that “fluid infusion should be titrated to a filling pressure” and that “pulmonary edema may occur as a complication of fluid resuscitation and necessitates monitoring of arterial oxygenation.”³⁷ Should volume overload and pulmonary edema be the end point of fluid resuscitation?³⁸ This is clinically important because a positive fluid balance in both ICU patients and those undergoing surgery has been associated with increased complications and a higher mortality.^{39–41} It is however equally likely that resuscitation guided by CVP will result in inadequate volume replacement. Furthermore, the use of diuretics based on CVP may result in intravascular volume depletion leading to poor organ perfusion and ultimately renal failure and multiorgan failure because a “high” CVP does not necessarily reflect volume overload.

Fundamentally the only reason to give a patient a fluid challenge is to increase the stroke volume.⁶ This assumes that the patient is on the ascending portion of the Frank-Starling curve and has “recruitable” cardiac output. Once the left ventricle is functioning near the “flat” part of the Frank-Starling curve, fluid loading has little effect on cardiac output and only serves to increase tissue edema and to promote tissue dysoxia. It is therefore crucial during the resuscitation phase of all critically ill patients to determine whether the patient is fluid responsive or not; this determines the optimal strategy of increasing cardiac output and oxygen delivery.⁴² The results from this article clearly demonstrate that CVP should not be used for this purpose.

The notion that CVP does not reflect intravascular volume and is a misleading tool for guiding fluid therapy is not new. In an article published in 1971, Forrester and colleagues,⁴³ the pioneers of hemodynamic monitoring, concluded that “CVP monitoring in acute myocardial infarction is at best of limited value and at worst seriously misleading.” In their landmark article that was published in 1975, Baek and colleagues¹⁰ convincingly established that “there was no correlation of blood volume with central

venous pressure” and suggest that “inaccurate physiologic evaluation of critically ill patients is likely to jeopardize survival by inviting inappropriate and ineffectual therapy.” In 1977, Dr. Burch,⁴⁴ a well-respected cardiologist, noted that “to accept non-critically the level of central venous pressure as a quantitative index of blood volume can only lead to physiologic and/or therapeutic errors.” The observations of Forrester et al,⁴³ Baek and colleagues,¹⁰ and Burch⁴⁴ have now been confirmed by 23 more recent studies. Indeed, limited data support using CVP to guide fluid therapy. The only study⁴⁵ we could find demonstrating the utility of CVP in predicting volume status was performed in seven standing, awake mares undergoing controlled hemorrhage! In addition, Magder and colleagues⁴⁶ reported that the respiratory variation in CVP in spontaneously breathing patients was predictive of fluid responsiveness. Additional studies are required to support using the respiratory variation in CVP to guide fluid management. In addition, it should be noted that in the ARDSnet fluid management trial,⁴⁷ those patients randomized to the “CVP conservative-strategy” group had significantly more ventilator-free days and a shorter length of ICU stay. It is unclear from this study whether CVP or the conservative fluid strategy was the important intervention because there was no “no-CVP” study arm. It should also be recognized that CVP was a component of early goal-directed therapy in the landmark article by Rivers and colleagues.⁴⁸ However, both the control and intervention groups had CVP targeted to 8 to 12 mm Hg. Based largely on the results of the early goal-directed therapy study, the Surviving Sepsis Campaign guidelines¹ for management of severe sepsis and septic shock recommend a CVP of 8 to 12 mm Hg as the “goal of the initial resuscitation of sepsis-induced hypoperfusion” and “a higher targeted central venous pressure of 12–15 mm Hg” in patients receiving mechanical ventilation. The results of our study suggest that these recommendations should be revisited.

The origins of CVP monitoring can be traced back to Hughes and Magovern,⁴⁹ who in 1959 described a complicated technique for right atrial monitoring as a guide to blood volume replacement in post-thoracotomy patients. These authors described a fall in CVP with blood loss and a relationship between the CVP and blood transfusion. The technique of CVP monitoring was further popularized by Wilson and Grow⁵⁰ and soon became routine in patients undergoing thoracic surgery. Based on scarce data, CVP became the standard tool for guiding fluid therapy, initially in the operating room and then in the ICU. However, what was not generally appreciated is that the CVP is a

measure of right atrial pressure alone; and not a measure of blood volume or ventricular preload. Based on the results of our systematic review, we believe that CVP should no longer be routinely measured in the ICU, operating room, or emergency department. However, measurement of the CVP may be useful in select circumstances, such as in patients who have undergone heart transplant, or in those who have suffered a right ventricular infarction or acute pulmonary embolism. In these cases, CVP may be used as a marker of right ventricular function rather than an indicator of volume status.

ACKNOWLEDGMENT: The authors are grateful to Dr. "Moose" Ahmed for his insight into animal physiology, and Dr "JVP" De Caro for his valuable clinical perceptiveness.

REFERENCES

- Dellinger RP, Carlet JM, Masur H et al. Surviving Sepsis Campaign guidelines for management of severe sepsis and septic shock. *Crit Care Med* 2004; 32:858–873
- Central venous pressure: an indicator of circulatory hemodynamics. In: Mohrman DE, Heller LJ, eds. *Lange cardiovascular physiology*. 6th ed. New York, NY: McGraw-Hill, 2006
- Mark JB, Slaughter TF. Cardiovascular monitoring. In: Miller RD, ed. *Miller's anesthesia*. 6th ed. Orlando, FL: Churchill Livingstone, 2005
- Weil MH, Henning RJ. New concepts in the diagnosis and fluid treatment of circulatory shock: thirteenth annual Becton, Dickinson and Company Oscar Schwidetsky Memorial Lecture. *Anesth Analg* 1979; 58:124–132
- Moher D, Cook DJ, Eastwood S, et al. Improving the quality of reports of meta-analyses of randomised controlled trials: the QUOROM statement; quality of reporting of meta-analyses. *Lancet* 1999; 354:1896–1900
- Michard F, Teboul JL. Predicting fluid responsiveness in ICU patients: a critical analysis of the evidence. *Chest* 2002; 121:2000–2008
- Michard F, Teboul JL. Using heart-lung interactions to assess fluid responsiveness during mechanical ventilation. *Crit Care* 2000; 4:282–289
- Ng TT, McGory ML, Ko CY, et al. Meta-analysis in surgery: methods and limitations. *Arch Surg* 2006; 141:1125–1130
- Moher D, Cook DJ, Eastwood S, et al. Improving the quality of reports of meta-analyses of randomised controlled trials: the QUOROM statement; QUOROM Group. *Br J Surg* 2000; 87:1448–1454
- Baek SM, Makabaki GG, Bryan-Brown CW, et al. Plasma expansion in surgical patients with high central venous pressure (CVP): the relationship of blood volume to hematocrit, CVP, pulmonary wedge pressure, and cardiorespiratory changes. *Surgery* 1975; 78:304–315
- Shippy CR, Appel PL, Shoemaker WC. Reliability of clinical monitoring to assess blood volume in critically ill patients. *Crit Care Med* 1984; 12:107–112
- Hoelt A, Schorn B, Weyland A, et al. Bedside assessment of intravascular volume status in patients undergoing coronary bypass surgery. *Anesthesiology* 1994; 81:76–86
- Oohashi S, Endoh H, Oohashi S, et al. Does central venous pressure or pulmonary capillary wedge pressure reflect the status of circulating blood volume in patients after extended transthoracic esophagectomy? *J Anesth* 2005; 19:21–25
- Kuntscher MV, Germann G, Hartmann B. Correlations between cardiac output, stroke volume, central venous pressure, intra-abdominal pressure and total circulating blood volume in resuscitation of major burns. *Resuscitation* 2006; 70:37–43
- Calvin JE, Driedger AA, Sibbald WJ. The hemodynamic effect of rapid fluid infusion in critically ill patients. *Surgery* 1981; 90:61–76
- Reuse C, Vincent JL, Pinsky MR. Measurements of right ventricular volumes during fluid challenge. *Chest* 1990; 98:1450–1454
- Godje O, Peyerl M, Seebauer T, et al. Central venous pressure, pulmonary capillary wedge pressure and intrathoracic blood volumes as preload indicators in cardiac surgery patients. *Eur J Cardiothorac Surg* 1998; 13:533–539
- Wagner JG, Leatherman JW. Right ventricular end-diastolic volume as a predictor of the hemodynamic response to a fluid challenge. *Chest* 1998; 113:1048–1054
- Wiesenack C, Prasser C, Keyl C, et al. Assessment of intrathoracic blood volume as an indicator of cardiac preload: single transpulmonary thermodilution technique versus assessment of pressure preload parameters derived from a pulmonary artery catheter. *J Cardiothorac Vasc Anesth* 2001; 15:584–588
- Berkenstadt H, Margalit N, Hadani M, et al. Stroke volume variation as a predictor of fluid responsiveness in patients undergoing brain surgery. *Anesth Analg* 2001; 92:984–989
- Michard F, Boussat S, Chemla D, et al. Relation between respiratory changes in arterial pulse pressure and fluid responsiveness in septic patients with acute circulatory failure. *Am J Respir Crit Care Med* 2000; 162:134–138
- Reuter DA, Felbinger TW, Kilger E, et al. Optimizing fluid therapy in mechanically ventilated patients after cardiac surgery by on-line monitoring of left ventricular stroke volume variations: comparison with aortic systolic pressure variations. *Br J Anaesth* 2002; 88:124–126
- Reuter DA, Kirchner A, Felbinger TW, et al. Usefulness of left ventricular stroke volume variation to assess fluid responsiveness in patients with reduced cardiac function. *Crit Care Med* 2003; 31:1399–1404
- Barbier C, Loubieres Y, Schmit C, et al. Respiratory changes in inferior vena cava diameter are helpful in predicting fluid responsiveness in ventilated septic patients. *Intensive Care Med* 2004; 30:1740–1746
- Kramer A, Zygun D, Hawes H, et al. Pulse pressure variation predicts fluid responsiveness following coronary artery bypass surgery. *Chest* 2004; 126:1563–1568
- Marx G, Cope T, McCrossan L, et al. Assessing fluid responsiveness by stroke volume variation in mechanically ventilated patients with severe sepsis. *Eur J Anaesthesiol* 2004; 21:132–138
- Preisman S, Kogan S, Berkenstadt H, et al. Predicting fluid responsiveness in patients undergoing cardiac surgery: functional haemodynamic parameters including the Respiratory Systolic Variation Test and static preload indicators. *Br J Anaesth* 2005; 95:746–755
- Perel A, Minkovich L, Preisman S, et al. Assessing fluid-responsiveness by a standardized ventilatory maneuver: the respiratory systolic variation test. *Anesth Analg* 2005; 100:942–945
- Hofer CK, Muller SM, Furrer L, et al. Stroke volume and pulse pressure variation for prediction of fluid responsiveness in patients undergoing off-pump coronary artery bypass grafting. *Chest* 2005; 128:848–854
- De Backer D, Heenen S, Piagnerelli M, et al. Pulse pressure variations to predict fluid responsiveness: influence of tidal volume. *Intensive Care Med* 2005; 31:517–523
- Kumar A, Anel R, Bunnell E, et al. Pulmonary artery occlusion pressure and central venous pressure fail to predict ventricular filling volume, cardiac performance, or the re-

- response to volume infusion in normal subjects. *Crit Care Med* 2004; 32:691–699
- 32 Osman D, Ridell C, Ray P et al. Cardiac filling pressures are not appropriate to predict hemodynamic response to volume challenge. *Crit Care Med* 2007; 35:64–68
 - 33 Magder S, Bafaqeeh F. The clinical role of central venous pressure measurements. *J Intensive Care Med* 2007; 22:44–51
 - 34 Bigby M, Williams H. Appraising systematic reviews and meta-analyses. *Arch Dermatol* 2003; 139:795–798
 - 35 Boldt J, Lenz M, Kumle B, et al. Volume replacement strategies on intensive care units: results from a postal survey. *Intensive Care Med* 1998; 24:147–151
 - 36 Magder S. Central venous pressure monitoring. *Curr Opin Crit Care* 2006; 12:219–227
 - 37 Hollenberg SM, Ahrens TS, Annane D, et al. Practice parameters for hemodynamic support of sepsis in adult patients: 2004 update. *Crit Care Med* 2004; 32:1928–1948
 - 38 Singer M. The Surviving Sepsis guidelines: evidence-based . . . or evidence-biased? *Crit Care Resusc* 2006; 8:244–245
 - 39 Vincent JL, Sakr Y, Sprung CL, et al. Sepsis in European intensive care units: results of the SOAP study. *Crit Care Med* 2006; 34:344–353
 - 40 Wheeler AP, Bernard GR, Thompson BT, et al. Pulmonary-artery versus central venous catheter to guide treatment of acute lung injury. *N Engl J Med* 2006; 354:2213–2224
 - 41 Brandstrup B, Tonnesen H, Beier-Holgersen R, et al. Effects of intravenous fluid restriction on postoperative complications: comparison of two perioperative fluid regimens: a randomized assessor-blinded multicenter trial. *Ann Surg* 2003; 238:641–648
 - 42 Marik PE, Baram M. Non-invasive hemodynamic monitoring in the intensive care unit. *Crit Care Clin* 2007; 23:383–400
 - 43 Forrester JS, Diamond G, McHugh TJ, et al. Filling pressures in the right and left sides of the heart in acute myocardial infarction: a reappraisal of central-venous-pressure monitoring. *N Engl J Med* 1971; 285:190–192
 - 44 Burch GE. Of central venous pressure [abstract]. *Am Heart J* 1977; 94:391
 - 45 Magdesian KG, Fielding CL, Rhodes DM, et al. Changes in central venous pressure and blood lactate concentration in response to acute blood loss in horses. *J Am Vet Med Assoc* 2006; 229:1458–1462
 - 46 Magder SA, Georgoadis G, Cheong T. Respiratory variations in right atrial pressure predict response to fluid challenge. *J Crit Care* 1992; 20:29–42
 - 47 Comparison of two fluid-management strategies in acute lung injury. *N Engl J Med* 2006; 354:2564–2575
 - 48 Rivers E, Nguyen B, Havstad S, et al. Early goal-directed therapy in the treatment of severe sepsis and septic shock. *N Engl J Med* 2001; 345:1368–1377
 - 49 Hughes RE, Magovern GJ. The relationship between right atrial pressure and blood volume [abstract]. *Arch Surg* 1959; 79:238
 - 50 Wilson JN, Grow JB. Central venous pressure in optimal blood volume maintenance. *Arch Surg* 1962; 85:55

Does Central Venous Pressure Predict Fluid Responsiveness?: A Systematic Review of the Literature and the Tale of Seven Mares

Paul E. Marik, Michael Baram and Bobbak Vahid

Chest 2008;134;172-178

DOI 10.1378/chest.07-2331

This information is current as of July 16, 2008

Updated Information & Services	Updated information and services, including high-resolution figures, can be found at: http://chestjournal.org/cgi/content/full/134/1/172
References	This article cites 48 articles, 17 of which you can access for free at: http://chestjournal.org/cgi/content/full/134/1/172#BIBL
Permissions & Licensing	Information about reproducing this article in parts (figures, tables) or in its entirety can be found online at: http://chestjournal.org/misc/reprints.shtml
Reprints	Information about ordering reprints can be found online: http://chestjournal.org/misc/reprints.shtml
Email alerting service	Receive free email alerts when new articles cite this article sign up in the box at the top right corner of the online article.
Images in PowerPoint format	Figures that appear in CHEST articles can be downloaded for teaching purposes in PowerPoint slide format. See any online article figure for directions.

A M E R I C A N C O L L E G E O F



P H Y S I C I A N S ®