

Roger Desjardins
André Y. Denault
Sylvain Bélisle
Michel Carrier
Denis Babin
Sylvie Lévesque
Raymond Martineau

Can peripheral venous pressure be interchangeable with central venous pressure in patients undergoing cardiac surgery?

Received: 30 July 2002
Accepted: 2 October 2003
Published online: 5 November 2003
© Springer-Verlag 2003

This study was supported by the Plan de Pratique des Anesthésiologistes of the Montreal Heart Institute and the Fonds de la Recherche en Santé du Québec.

R. Desjardins · A. Y. Denault (✉)
S. Bélisle · M. Carrier · D. Babin
S. Lévesque · R. Martineau
Department of Anesthesiology and Surgery,
Montreal Heart Institute,
5000 Belanger Street East, Montreal,
Quebec, H1T 1C8, Canada
e-mail: denault@videotron.ca
Tel.: +1-514-3763330 ext 3732
Fax: 1-514-3768784

Abstract *Objective:* Pressure measurements at the level of the right atrium are commonly used in clinical anesthesia and the intensive care unit (ICU). There is growing interest in the use of peripheral venous sites for estimating central venous pressure (CVP). This study compared bias, precision, and covariance in simultaneous measurements of CVP and of peripheral venous pressure (PVP) in patients with various hemodynamic conditions. *Design and setting:* Operating room and ICU of a tertiary care university-affiliated hospital. *Patients:* Nineteen elective cardiac surgery patients requiring cardiopulmonary bypass were studied. *Interventions:* A PVP catheter was placed in the antecubital vein and connected to the transducer of the pulmonary artery catheter with a T connector. Data were acquired at different times

during cardiac surgery and in the ICU. *Measurements and results:* A total of 188 measurements in 19 patients were obtained under various hemodynamic conditions which included before and after the introduction of mechanical ventilation, following the induction of anesthesia, fluid infusion, application of positive end expiratory pressure and administration of nitroglycerin. PVP and CVP values were correlated and were interchangeable, with a bias of the PVP between -0.72 and 0 mmHg compared to the CVP. *Conclusions:* PVP monitoring can accurately estimate CVP under various conditions encountered in the operating room and in the ICU.

Keywords Central venous pressure · Peripheral venous pressure · Hemodynamics

Introduction

Central venous pressure (CVP) measurements are commonly used in anesthesia practice and in the intensive care unit (ICU) to estimate cardiac preload or circulatory blood volume. The CVP measurement can be accurately measured using an indwelling central venous catheter. However, this technique is not without complications, which include pneumothorax, arrhythmias, carotid artery puncture, and catheter-related infection. Measuring peripheral venous pressure (PVP) is a simpler and safer monitoring technique [1, 2]. The venous return concept originally described by Guyton et al. [3] is based on the existence of a pressure gradient between the periphery

and the right atrium. The gradient is the difference between mean systemic pressure and CVP. This gradient determines venous return. The concept of venous return implies that PVP must be greater than CVP to allow the blood to circulate towards the heart. However, the magnitude of the relationship between CVP and PVP is unknown and could depend on the site chosen for PVP measurement, on the resistance to venous return, and on cardiac systolic and diastolic function. Furthermore, the relationship between CVP and PVP in patients in the operating room and ICU with various cardiac function and under controlled alterations in preload, afterload, and contractility such as following cardiopulmonary bypass has not been reported.

Table 1 Patients' characteristics [NYHA New York Heart Association classification, N/A not available, CAD coronary artery disease, CABG coronary arterial bypass graft, MR mitral regurgitation, MS mitral stenosis, AI aortic insufficiency, AS aortic stenosis,

AoD aortic disease (regurgitation and stenosis), MD mitral disease (regurgitation and stenosis), V valve surgery, C complex surgery (valve and CABG)]

Patient no.	Age (years)	Sex	Weight (kg)	Height (cm)	NYHA	EF (%)	Diagnosis	Surgery
1	60	F	54	170	III	25	CAD, MR	V
2	58	M	93	181	III	69	CAD, AS	C
3	72	F	50	155	II	N/A	CAD	CABG
4	69	F	62	158	II	62	MS, AI	V ×2
5	76	M	77	171	III	N/A	MS	V
6	73	F	73	137	II	65	CAD, AS	C
7	64	M	97	184	II	34	CAD, AI	C
8	51	M	78	171	II	42	CAD	CABG
9	75	F	71	159	III	75	MS+AoD	V ×2
10	60	M	77	170	III	22	CAD	CABG
11	71	M	60	163	II	50	AoD+MD+CAD	C
12	77	M	65	167	III	N/A	CAD	CABG
13	77	M	63	160	II	N/A	MS, AS	V
14	64	M	96	176	III	33	CAD	CABG
15	70	F	58	150	III	22	CAD	CABG
16	76	M	70	165	III	70	MR+CAD	C
17	64	M	84	178	III	N/A	CAD	CABG
18	66	M	73	165	III	42	CAD	CABG
19	76	F	55	152	III	72	AoD+CAD	C
20	76	M	66	168	III	66	CAD, MR	C

The goal of this study was to determine whether PVP measurement can replace CVP monitoring in clinical practice. To achieve this goal, based on the venous return concept, we hypothesized that PVP is higher than CVP, and that covariance is maintained during different loading conditions.

Materials and methods

Between May and July 2000 we recruited 20 patients undergoing an elective cardiac surgical procedure under cardiopulmonary bypass; there were 13 men and 7 women, with a mean age of 69 ± 8 years, mean weight 71 ± 14 kg, and mean height 165 ± 11 cm. Individual data are presented in Table 1. Patients provided written consent and our institutional review board approved the study. Exclusion criteria were emergency surgery and unexpected hemodynamic instability requiring rapid intervention. Only two patients were excluded in the ICU. Data were collected on medication, diabetes, hypertension, prior myocardial infarction, unstable angina, type of surgery, and number of graft vessels. Preoperative ejection fraction value was derived from ventriculography or the echocardiographic examination; this was available in 15 patients and varied between 22% and 75%.

Patients were premedicated with intramuscular morphine (0.1 mg/kg) and scopolamine (0.005 mg/kg). In the operating room (OR), they were sedated using midazolam at 1–2 mg intravenously titrated to achieve a Ramsay score of 3 or 4 during insertion of a 15-cm triple-lumen catheter (CS-12703, Arrow International, Reading, Calif., USA) and a pulmonary artery catheter (7.5F 931HF75, Baxter Healthcare, Irvine, Calif., USA). Monitoring included five-lead electrocardiography, pulse oximeter, infrared CO₂ analyzer, and radial artery catheter. Induction of anesthesia used a combination of fentanyl (5–10 µg/kg) or sufentanil (0.7–1 µg/kg), midazolam (up to 0.1 mg/kg), and pancuronium (0.1 mg/kg). Isoflurane was used to control blood pressure during maintenance of anesthesia.

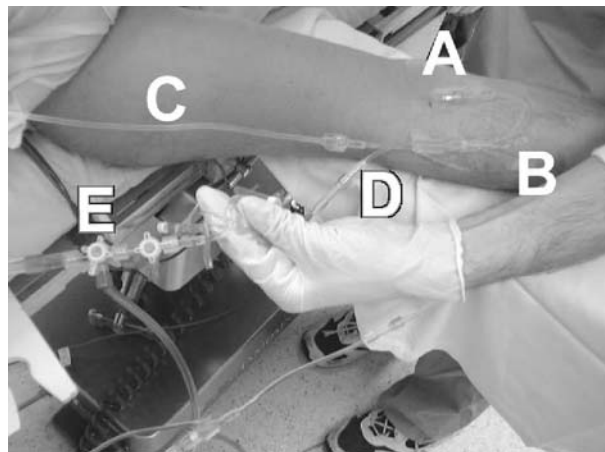


Fig. 1 Peripheral venous pressure monitoring system. In A a 16-gauge short intravenous catheter in the antecubital vein is connected to a Y connector (B). This connector is attached to both the pressure transducer system (C) and the low compliance intravenous tubing (D). Finally in E a three-way stopcock is used to isolate the pressure monitoring system from the fluid infusion site during the measurement periods

PVP was monitored through a short 32-mm 16-gauge catheter (Johnson & Johnson, Mississauga, Ontario, Canada) inserted in a vein of the antecubital fossa (Fig. 1, A). We elected to use this site after a pilot study showed greater reliability and stability of the antecubital region than a dorsal vein of the hand. This catheter was attached to a Y connector (Fig. 1, B). One arm was connected to a low-compliance tubing directly to a pressure transducer (Medex, Ohio, USA; Fig. 1, C), and the other arm of the connector was linked to an intravenous infusion system (Fig. 1, D) with a three-way stopcock (Fig. 1, E). The pressure transducer has a pre-

Table 2 Mean CVP and PVP [CVP central venous pressure, PVP pulmonary venous pressure, PEEP positive end-expiratory pressure, CI confidence interval, ICU intensive care unit, SD standard deviation]

Time point	n	CVP	PVP	Difference	95% CI (one-sided)	Range of agreement
t_1 : baseline	20	7.0±4.05	7.4±3.63	-0.40±1.27	-0.99 to 0.19	-3.06 to 2.26
t_2 : after fluid challenge	18	10.5±4.68	10.5±4.02	0±1.64	-0.82 to 0.81	-3.47 to 3.47
t_3 : baseline postinduction	20	8.5±3.69	8.9±3.19	-0.4±1.35	-1.03 to 0.23	-3.23 to 2.43
t_4 : after fluid challenge	20	11±2.85	11.3±2.64	-0.35±0.87	-0.76 to 0.06	-2.18 to 1.48
t_5 : after PEEP	17	12.2±2.82	12.5±2.58	-0.29±0.92	-0.77 to 0.17	-2.24 to 1.65
t_6 : baseline	11	11.7±2.76	11.7±2.9	-0.45±0.93	-1.08 to 0.17	-2.53 to 1.62
t_7 : after nitroglycerin	11	10.2±2.79	10.9±3.21	-0.72±0.79	-1.25 to -1.19	-2.47 to 1.02
t_8 : baseline ICU	19	9.3±3.87	9.6±3.56	-0.36±1.16	-0.92 to 0.19	-2.81 to 2.07
t_9 : after fluid challenge	16	12.6±2.9	12.4±2.39	0.18±1.22	-0.46 to 0.84	-2.42 to 2.79
t_{10} : after PEEP in the ICU	18	13.8±2.5	13.9±2.48	-0.11±0.58	-0.4 to 0.18	-1.34 to 1.11
t_{11} : baseline ICU	18	11.2±3.79	11.4±4.58	-0.27±1.41	-0.97 to 0.42	-3.24 to 2.68

cision above the AAMI specifications with a time variation below 2 mmHg per 4 h. The display monitor (Siemens Medical Systems, Danvers, Mass, USA) measures the PVP using a built-in algorithm which calculates mean pressure. This system allows the measurement of either pulmonary artery pressure or the PVP simultaneously with the CVP obtained from a different transducer. Measurement of CVP was taken from the distal (16-gauge lumen) end of the triple lumen central venous catheter. During surgery the patient's arm was placed by the side of the patient in a neutral position. The arm was protected with a 60-cm metallic arm protector on the side where PVP monitoring was attached to avoid any compression by the surgeon.

Measurements were taken at several time periods before and after the introduction of positive pressure ventilation (tidal volume of 8 ml/kg, respiratory rate of 10/min) following the induction of anesthesia, fluid challenge, the application of positive-end-expiratory pressure (PEEP), and administration of intravenous nitroglycerin (NTG). Fluid challenge was achieved by giving bolus of NaCl 0.9% in the OR or with Pentaspan (DuPont Pharma, Mississauga, Ontario, Canada) in the ICU to raise the CVP at least 4 mmHg. A maximum of 15 ml/kg of intravenous fluid was used. An expiratory valve was used to obtain 12.5 cmH₂O of PEEP, or it was directly programmed on the ICU ventilator (7200 Series, Puritan-Bennett, Carlsbad, Calif., USA). Finally, small boluses of NTG (10–50 µg) were administered to reduce systolic blood pressure by 15% or to a lower limit of 100 mmHg. Only 11 patients were eligible to receive a small bolus of nitroglycerin (NTG) because of borderline blood pressure.

The sequence of hemodynamic measurements was as follow: t_1 , baseline or before mechanical ventilation and fluid challenge; t_2 , before mechanical ventilation and after fluid challenge; t_3 , baseline after mechanical ventilation following induction of anesthesia; t_4 , after fluid challenge; t_5 following administration of PEEP; t_6 baseline following release of PEEP; t_7 after infusion of NTG; t_8 baseline upon admission in the ICU; t_9 in the ICU after fluid challenge; t_{10} in the ICU following the application of PEEP; and t_{11} after baseline in the ICU. A total of 188 hemodynamic data points were recorded in 20 patients.

Each hemodynamic measurement included heart rate, systolic, diastolic, and mean arterial pressure, and systolic, diastolic, and mean pulmonary artery pressure. PVP and pulmonary artery occlusion pressure were determined at end-expiration and end-diastole. Cardiac output was measured in triplicate using the thermodilution technique (Sirecust 1281, Siemens) in which less than 10% variation was observed. The antecubital vein and transducers were placed at the midthoracic level and a "flush test" was performed prior to each set of measurements to verify the resonance or damping of the system [4]. Hemodynamic variables were obtained from a printed recording of the values obtained from the monitor. Prior to recording any values of the PVP the three-way

stopcock at the Y connection of the intravenous infusion line was closed to avoid artifacts caused by the intravenous tubing.

Difficult separation from bypass (DSB) was defined as systolic blood pressure below 80 mmHg confirmed by central aortic measurement (femoral or aortic) and diastolic pulmonary artery pressure or pulmonary artery capillary wedge pressure less than 15 mmHg during progressive separation from cardiopulmonary bypass, the use of inotropic or vasopressive support (norepinephrine >4 µg/min, epinephrine >2 µg/min, dobutamine >2 µg/kg per minute, or the use of amrinone and milrinone) or mechanical support or intra-aortic balloon pump [5, 6]. By this definition 11 patients (55%) had difficult separation from bypass (DSB); five required more than 8 µg/min noradrenaline, four of whom required milrinone, and two required adrenaline to be weaned from bypass. One patient developed circulatory shock with cyanotic extremities upon arrival in the ICU which precluded further measurements.

Bias was defined as the mean difference between measurements by the two methods, and precision as the standard deviation (SD) of the bias between PVP and CVP measurements [7]. The confidence interval (CI) was calculated, and the lower and upper values of the 95% CI are reported because the mean bias would lie within this value. The range of agreement was defined as the mean bias ±2 SD. Limits of agreement were a priori clinically defined as ±3 mmHg for PVP. Linear regression analysis and covariance analysis were calculated to determine the relationship between PVP and CVP. Results are expressed as mean ±SD.

Results

Absolute values of CVP and PVP ranged from 2 to 27 mmHg. Table 2 summarizes mean CVP and PVP during each measurement period, bias, precision, and range of agreement. The highest mean difference was -0.72±0.78 mmHg (95% CI of -1.25 to -0.19 mmHg) at t_7 . Figure 2 presents a Bland-Altman graph. Because the difference between CVP and PVP were often the same, a three-dimensional Bland-Altman graph was produced with a z-axis representing the number of times or recurrences for which the value was observed. Using our clinically defined limit of agreement or 3 mmHg difference, CVP and PVP were within 3 mmHg in 180 instances, or interchangeable, 96% of the time. Figure 3 presents an example of a patient in whom PVP and CVP were recorded. Here one sees that PVP is greater than

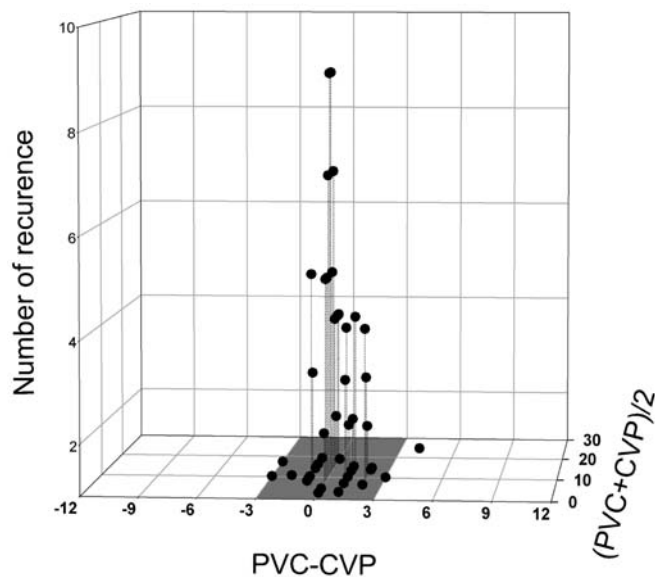


Fig. 2 Three-dimensional Bland-Altman graph. The difference between peripheral venous pressure (PVP) and central venous pressure (CVP) or *x-axis* is plotted against the sum of the two values divided by 2 or *y-axis*. The *z-axis* represents the number of recurrences or number of times for which the same value is obtained

CVP; the values are close at end-diastole, and the atrial waveforms of the central venous tracing are more visible than in the peripheral venous tracing.

Discussion

This study describes a simple, inexpensive, and minimally invasive technique that can be used as a substitute to the monitoring of CVP. We observed that PVP measured in the antecubital fossa lies within 3 mmHg of CVP 96% of the time. Both values demonstrate a covariance under different condition of cardiac function, volume infusion, vasoactive drugs, and administration of PEEP both in the awake and during mechanical ventilation. The various conditions in which PVP was obtained and compared with PVP supports its clinical usefulness in the OR and in the ICU, but it could also be used in the emergency room, coronary care unit, and in every location where a clinician must manage patients in whom estimation of filling pressure is important.

Interestingly, few human studies have reported their experience with the use of PVP. In 1970 Eustace [8] reported his use of PVP in 15 surgical patients under general anesthesia, concluding that the PVP did not provide similar diagnostic information as CVP. A fluid bolus was given to five patients, and the magnitude of change was observed to differ between PVP and CVP. However, PVP was higher than CVP in all patients the, the two values covaried in the same direction, and the location of

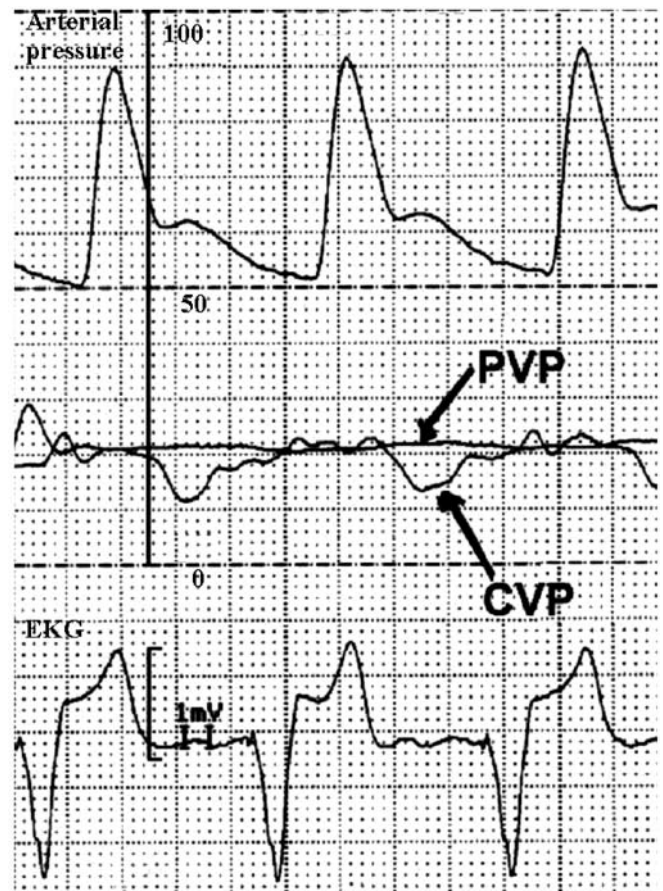


Fig. 3 Illustrates the relationship between central venous pressure (CVP) and peripheral venous pressure (PVP). It can be seen that, peripheral venous pressure was higher on average than central venous pressure, and that the normal atrial waves of the central venous pressure are lost with the peripheral venous pressure

the PVP measurement varied between patients. Sheldon et al. [9, 10] also demonstrated in an animal model that the use of PVP was well correlated with cardiac output and peripheral perfusion measured as peripheral saturation.

More recently Munis et al. [1] reported their experience with PVP in 15 patients undergoing neurosurgical procedures. Paired measurements were obtained under supine ($n=8$) or prone ($n=7$) position. They observed a significant relationship between PVP and CVP ($p<0.001$), with a Pearson correlation coefficient of 0.82. Measurements were obtained following induction of anesthesia, and PVP was obtained with either 18-gauge or 20-gauge catheter in the dorsal vein of the hand or the distal forearm, and hemodynamic data were recorded every 5 min. However, fluid administration and vasoactive medication were not protocolized, and no Bland-Altman was analysis performed. Despite their limitations this study highlighted very well the close correlation between changes in PVP and CVP. In addition, they also

noted that a, c, and v waves and respiratory variations were lost using the peripheral venous tracing, as we observed (Fig. 3). We observed a smaller difference between PVP and CVP than Munis et al. This can be explained by the fact that we used the antecubital fossa and a larger 16-gauge catheter. In our pilot study we observed a larger difference between a more distal PVP monitoring site which is consistent with the concept of venous return predicting higher upstream pressure.

In another recent study Amar et al. [2] reported their experience using PVP in 150 patients undergoing noncardiac surgery. Patients were excluded if ventricular dysfunction or valvular disease was present. They observed that PVP was correlated with CVP in the OR (bias of -1.6 ± 1.7 mmHg) and in the postanesthesia care unit (bias of -2.2 ± 1.9 mmHg). They used several different peripheral sites and did not observe any significant differences. Fluid challenge was performed in ten patients with 2 liters, and the increase in both PVP and CVP was identical. We observed a smaller mean difference in our population because the site was identical in all patients. Their population was also different than ours. We did not exclude patients with abnormal ventricular function; seven of our patients had ejection fraction lower than 50%. Finally, other venous sites of monitoring have been reported. Use of the external jugular vein for measuring PVP produced values that were correlated with those of CVP, but this method was limited by the positional effect of the neck musculature [11]. The femoroiliac vein, iliac vein, and inferior vena cava have also been used as substitute to CVP and have shown good agreement [12, 13, 14].

None of these studies, however, determined the relationship between CVP and PVP under controlled hemodynamic conditions with various techniques and not limited to fluid infusion only. In addition, we document for the first time that this relationship is maintained in patients with abnormal cardiac function (ejection fraction below 50%) which was present in 7 (35%) of our patients and in patients with vasoactive medications (55% of our population)

Measurement of preload using PVP has limitations. The normal CVP waveforms are dampened by the use of PVP. These waveforms can be useful in the diagnosis of pericardial disease and right ventricular dysfunction and also for recognizing arrhythmia [15, 16] and fluid responsiveness [17]. However, clinical appreciation of the value of CVP obtained from a monitor has been shown to be subject to significant interobserver variability [18]. This may potentially be reduced by using PVP because more stable values are observed (Fig. 3).

Precaution must be taken to avoid any compression over the arm. Arm compression results in higher PVP, and for this reason we shielded the arm with a protector. Venous obstruction, ill-deflated pressure cuffs, muscle movement, and shivering can all interfere with the monitoring of PVP. A central catheter also remains our first choice when we plan to infuse vasoactive drugs during an intervention because of the risk and complications of drug extravasation. We observed that the range of agreement between CVP and PVP was broader in awake patients than when under general anesthesia. This could be explained by alteration in the autonomic nervous system during anesthesia which can theoretically reduce the peripheral vasoconstriction that some patients experience when awake and anxious prior to their operation. Finally the same limitations in the clinical value of CVP also apply to PVP [19, 20].

In summary, PVP monitoring can accurately estimate CVP in cardiac surgical patients under awake or positive-pressure ventilation, under general anesthesia condition, and following fluid challenge, PEEP, and the administration of vasodilating drugs. The value of PVP and in stratifying critically ill patients to distinguish between cardiogenic and noncardiogenic causes remains to be determined. Further studies should be conducted to explore the value and limitations of such a noninvasive and inexpensive monitoring device for its use outside of the OR or ICU and in situations in which rapid estimation of right-sided pressure would help to stratify hemodynamically unstable patients [21].

References

1. Munis JR, Bhatia S, Lozada LJ (2001) Peripheral venous pressure as a hemodynamic variable in neurosurgical patients. *Anesth Analg* 92:172–179
2. Amar D, Melendez JA, Zhang H, Dobres C, Leung DH, Padilla RE (2001) Correlation of peripheral venous pressure and central venous pressure in surgical patients. *J Cardiothorac Vasc Anesth* 15:40–43
3. Guyton AC, Lindsey AW, Abernathy B, Richardson T (1957) Venous return at various right atrial pressures and the normal venous return curve. *Am J Physiol* 189:609–615
4. Gardner RM (1981) Direct blood pressure measurement—dynamic response requirements. *Anesthesiology* 54:227–236
5. Hardy JF, Searle N, Roy M, Perrault J (1993) Amrinone, in combination with norepinephrine, is an effective first-line drug for difficult separation from cardiopulmonary bypass. *Can J Anaesth* 40:495–501
6. Bernard F, Denault A, Babin D, Goyer C, Couture P, Couturier A, Buithieu J (2001) Diastolic dysfunction is predictive of difficult weaning from cardiopulmonary bypass. *Anesth Analg* 92:291–298
7. Bland JM, Altman DG (1986) Statistical methods for assessing agreement between two methods of clinical measurement. *Lancet* I:307–310

8. Eustace BR (1970) A comparison between peripheral and central venous pressure monitoring under clinical conditions. *Injury* 2:14–18
9. Sheldon CA, Balik E, Dhanalal K, Belani K, Marino J, Leonard AS (1982) Peripheral postcapillary venous pressure—a new hemodynamic monitoring parameter. *Surgery* 92:663–669
10. Sheldon CA, Cerra FB, Bohnhoff N, Belani K, Frieswyk D, Dhanalal K, Leonard AS (1983) Peripheral postcapillary venous pressure: a new, more sensitive monitor of effective blood volume during hemorrhagic shock and resuscitation. *Surgery* 94:399–406
11. Stoelting RK (1973) Evaluation of external jugular venous pressure as a reflection of right atrial pressure. *Anesthesiology* 38:291–294
12. Yazigi A, Madi-Jebara S, Antakly MC (1999) Iliac venous pressure predicts central venous pressure in spontaneously breathing patients (Abstract) *Crit Care Med* 27:1219
13. Lloyd TR, Donnerstein RL, Berg RA (1992) Accuracy of central venous pressure measurement from the abdominal inferior vena cava. *Pediatrics* 89:506–508
14. Dillon PJ, Columb MO, Hume DD (2001) Comparison of superior vena caval and femoroiliac venous pressure measurements during normal and inverse ratio ventilation. *Crit Care Med* 29:37–39
15. Sharkey SW (1987) Beyond the wedge: clinical physiology and the Swan-Ganz catheter. *Am J Med* 83:111–122
16. Mark JB (1991) Central venous pressure monitoring: clinical insights beyond the numbers. *J Cardiothorac Vasc Anesth* 5:163–173
17. Magder S, Georgiadis G, Cheong T (2003) Respiratory variations in right atrial pressure predict the response to fluid challenge. *J Crit Care* 7:76–85
18. Cook DJ (1990) Clinical assessment of central venous pressure in the critically ill. *Am J Med Sci* 299:175–178
19. Shippy CR, Appel PL, Shoemaker WC (1984) Reliability of clinical monitoring to assess blood volume in critically ill patients. *Crit Care Med* 12:107–112
20. Michard F, Boussat S, Chemla D, Anguel N, Mercat A, Lecarpentier Y, Richard C, Pinsky MR, Teboul JL (2000) Relation between respiratory changes in arterial pulse pressure and fluid responsiveness in septic patients with acute circulatory failure. *Am J Respir Crit Care Med* 162:134–138
21. Wood KE (2002) Major pulmonary embolism: review of a pathophysiologic approach to the golden hour of hemodynamically significant pulmonary embolism. *Chest* 121:877–905