Peripheral Venous Pressure as a Hemodynamic Variable in Neurosurgical Patients

James R. Munis, MD, PhD, Sanjay Bhatia, MD, and Leonardo J. Lozada, MD

Section of Anesthesia for Neurosurgery, Department of General Anesthesiology, The Cleveland Clinic, Cleveland, Ohio

Neurosurgical patients undergoing either craniotomy or complex spine surgery are subject to wide variations in blood volume and vascular tone. The ratio of these variables yields a pressure that is traditionally measured at the superior vena cava and referred to as "central venous pressure" (CVP). We have investigated an alternative to CVP by measuring peripheral venous pressure (PVP), which, in parallel animal studies, correlates highly with changes in absolute blood volume ($r = 0.997$). We tested the hypothesis that PVP trends parallel CVP trends and that their relationship is independent of patient position. We also tested and confirmed the hypothesis, during planned circulatory arrest, that PVP approximates mean systemic pressure (circulatory arrest pressure), which reflects volume status independent of cardiac function. PVP was compared with CVP across 1026 paired measurements in 15 patients undergoing either craniotomy (supine, $n = 8$) or complex spine surgery (prone, $n = 7$). Repeated-measures analysis of variance indicated a highly significant relationship between PVP and CVP ($P < 0.001$), with a Pearson correlation coefficient of 0.82. The correlation was best in cases with significant blood loss (estimated blood loss >1000 mL; $r = 0.885$) or hemodynamic instability (standard deviation of CVP > 2; $r = 0.923$).

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Neurosurgical patients undergoing either craniotomy or complex spine surgery are at risk for significant blood loss and hemodynamic changes while under anesthesia. A portion of that risk is associated with the surgery itself, and the remainder is because of the effects of anesthetics, mechanical ventilation, and positional changes on cardiac function, relative blood volume, and vascular tone. For patients having procedures with a likelihood of significant blood loss, or for patients in whom hemodynamic changes are especially threatening because of coincident cardiovascular disease, central venous pressure (CVP) monitoring has been widely used since the introduction of percutaneous jugular vein cannulation in 1969 (1). CVP is a direct measure of pressure, not volume. However, pressure is assumed to reflect the ratio of blood volume to vascular compliance, and it is a way to assess "relative blood volume" in patients who are either critically ill or undergoing major surgery (2). In this capacity, and in the normal range of hemodynamic values, CVP appears to have more utility as a trend monitor than for titration to an absolute end point (3,4).

The measurement of CVP requires the insertion and maintenance of a catheter in the thorax. Typically, the catheter is positioned in the superior vena cava from an insertion site in an external or internal jugular vein, subclavian vein, or, less often, from a femoral or antecubital vein (5,6). A small but potentially serious risk accompanies every CVP placement (7–11), in addition to adding time and monetary costs to surgery.

This study specifically addresses an alternative to the CVP as a physiological volume monitor. Peripheral venous pressure (PVP) reflects an "upstream" venous variable that is coupled to CVP by a continuous column of blood, analogous to the fluid continuity that exists between a pulmonary artery occlusion catheter and the left atrium. We tested the hypothesis that PVP values are easily obtained, free from local occlusion artifact, and closely correlated with CVP trends in a variety of major neurosurgical procedures, patient positions, and degrees of hemodynamic instability. We also tested the hypothesis that PVP reflects mean systemic pressure (circulatory arrest pressure), which is the unique pressure to which all vascular beds converge in the absence of cardiac function. Because mean systemic pressure is defined as the simple ratio of blood volume to vascular compliance, it is a uniquely useful variable for volume assessment, but

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Address correspondence to James Munis, MD, PhD, Head, Section of Anesthesia for Neurosurgery, Department of General Anesthesiology/E-31, The Cleveland Clinic, 9500 Euclid Avenue, Cleveland, OH 44195. Address e-mail to munisj@ccf.org.

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has not been accessible to clinical measurement techniques during intact circulatory function.

Methods

IRB approval was obtained for an observational study without the requirement for written consent to evaluate hemodynamic data from patients undergoing either craniotomy or complex spine surgery using existing central and peripheral venous lines. Criteria for inclusion included presentation for either craniotomy or complex spine instrumentation surgery, acceptable access for central venous catheter placement, and acceptable access for customary peripheral IV placement. One patient was excluded from the study on the basis of inadequate peripheral IV access.

Hemodynamic data were recorded beginning with placement and calibration of CVP and PVP catheters and positioning of the patient for surgery, and ending at the conclusion of surgery. Real-time waveforms were displayed throughout the case for both CVP and PVP, and numeric pressure values were retained by the same monitoring system (GE Marquette 9500 Patient Monitor with Tram 350 Module; GE Marquette, Milwaukee, WI). Central venous catheters (either 7F 2-lumen or 12F 3-lumen, 16 cm., Arrow International, Reading, PA) were placed through either the left or right internal jugular approach. Peripheral IV catheters (either 18-gauge or 20-gauge, 1.16 in., B-D Angiocath™, Becton Dickinson Infusion Therapy Systems, Inc., Sandy, UT) were placed, or used *in situ*, from dorsal hand or distal forearm veins. The PVP catheter site was maintained at midthorax height throughout each case. After flushing and room-air zero calibration (Becton-Dickinson Transducer Sets; Becton-Dickinson Critical Care Systems Pte. Ltd., Singapore), the transducer sets were mechanically flushed with saline and also maintained at midthorax level throughout surgery. Continuity of the PVP catheter with the downstream venous system was demonstrated at the beginning of each case by observing coincident pressure changes in the PVP waveform during circumferential, proximal arm occlusion. All hemodynamic data were recorded to the nearest 1 mm Hg at 5-min intervals throughout the recording period.

No alterations were made in the usual anesthetic technique for the cases. Anesthesia was induced with IV sodium thiopental or propofol, and maintained by nitrous oxide/oxygen with isoflurane; or by IV propofol and remifentanil infusion with nitrous oxide/oxygen and small-dose isoflurane. Muscle relaxation was maintained by intermittent IV doses of either pancuronium or rocuronium, except in two spine surgery cases (4 and 10) where electromyography was used to monitor spinal cord integrity and no muscle relaxant was administered after an intubating dose of succinylcholine. All patients were intubated and mechanically ventilated throughout surgery. Arterial pressure was monitored with a standard 20-gauge radial arterial line throughout surgery. Decisions regarding IV fluid administration, blood or blood product administration, and treatment with vasoactive medications were made according to the usual judgement of each staff anesthesiologist without regard to PVP readings.

One adult patient who underwent repair of a giant basilar artery aneurysm under deep hypothermic circulatory arrest was studied. Simultaneous measurements of radial arterial pressure (ART), pulmonary arterial pressure (PAP), CVP, and PVP were made throughout the case. After systemic heparinization and cannulation of the right femoral artery and vein, the patient was placed on cardiopulmonary bypass (CPB) and cooled to a target temperature of 18°C with a chilled bypass circuit. After temperature stabilization, CPB was abruptly stopped and the femoral venous cannula connecting the patient to the venous reservoir was simultaneously occluded. Real-time continuous pressure measurements of ART, PAP, PVP, and CVP were recorded before, during, and after circulatory arrest.

The hemodynamic data are reported as single values (in mm Hg, referenced to midthorax level), rounded to the nearest 1 mm Hg unless otherwise specified. Time intervals are reported as minutes after the initiation of recording. All venous pressures were measured at 5-min intervals throughout the surgical procedure. To test for a significant relationship, PVP and CVP trends were compared by repeated-measures analysis of variance. PVP and CVP trends were also compared by simple regression analysis to obtain Pearson correlation coefficients, and reanalyzed according to significant estimated blood loss (defined as $>$1000 mL), and hemodynamic instability (defined as SD of CVP $>$2). Scatter plots of CVP versus PVP and mean arterial pressure (MAP) versus PVP were made using all 1026 respective paired measurements. Because PVP was expected to have an overall physiological offset more than simultaneous CVP measurements (and, therefore, a physiological "bias"), and because our hypothesis concerned trend correlation rather than equivalence of absolute values, we applied statistical tests of trend correlation rather than of bias.

Results

Table 1 summarizes the demographic and hemodynamic features of each case. Patient ages ranged from 16 to 76 yr, and ASA physical status ranged from II-IV. Estimated surgical blood loss ranged from 200 mL to 8500 mL. All 15 surgeries were considered "major," either craniotomy for tumor or vascular malformation,

Table 1. Patient Characteristics and Cumulative Hemodynamic Profiles For Each Surgery

Case No.	Age/ sex	Wt. (kg)	ASA	Position	Surgery	EBL (mL)	Crystalloid (mL)	Hetastarch (mL)	PRBC (mL)	Intraoperative Diuretics	UOP (mL)
1	42 F	54	3	Supine	Craniotomy—AVM	300	3000	0	0	M	1220
2	44 F	90	3	Supine	Craniotomy—tumor	700	3000	500	0	M	1300
3	64 M	110	3	Supine	Craniotomy—tumor	1200	2900	1000	0	None	1400
4	58 F	80	2	Prone	Redo complex spine	8500	12000	1000	4650	None	1075
5	53 M	86	3	Prone	Redo Complex spine	600	3500	0	0	None	1700
6	76 F	68	3	Supine	Craniotomy—tumor	200	1600	0	0	M	400
7	68 M	55	4	Prone	Complex spine	2400	6250	0	1575	None	1600
8	45 M	126	3	Supine	Craniotomy—tumor	350	3600	0	0	M	800
9	33 M	85	3	Supine	Craniotomy—tumor	300	2600	0	0	None	480
10	16 M	55	3	Lateral/prone	Scoliosis repair	2400	6200	1000	PRBC 970, FFP 541	None	600
11	58 M	82	3	Prone	Complex spine	5800	9200	1000	PRBC3350, FFP 2200	None	2300
12	44 M	80	3	Supine	Craniotomy—tumor	1200	5800	1000	PRBC 770	None	870
13	74 M	88	3	Prone	Complex spine	2400	6700	1000	1400	None	1025
14	50 M	90	4	Supine	Craniotomy—aneurysm	250	4500	0	0	Lasix 10 mg	1000
15	27 M	69	2	Supine/prone	Complex spine	900	5000	1000	300	None	1000

ASA = American Society of Anesthesiologists preoperative physical status; EBL = estimated surgical blood loss; Crystalloid = Normal saline or lactated Ringer's solution; PRBC = packed red blood cells; UOP = urine output; AVM = cerebral arteriovenous malformation; M = mannitol; FFP = fresh frozen plasma.

or complex spinal instrumentation surgery. Patient positioning was supine for craniotomy (n = 8), or prone for spine surgery (n = 6). Two cases (repair of scoliosis, 10; complex spine surgery, 15) were conducted in two immediately sequential phases during the same operation: lateral (Case 10), or supine (Case 15) for anterior release, then prone for posterior spinal instrumentation. Hemodynamic monitoring continued throughout both phases and positions.

PVP was easily obtained in all 15 patients, in supine, lateral, and prone positions. Figure 1 displays a 24-s recording of simultaneous PVP and CVP in the same patient. Panel A shows uninterrupted waveforms, and panel B demonstrates a proximal arm occlusion and release that was used in each case to test for the continuity of measured PVP with the downstream venous system. The coincidence of an abrupt pressure change in the PVP waveform when the arm was occluded or released at a site far removed from the PVP catheter site demonstrates this continuity. The CVP tracing reveals higher amplitude respiratory variations, as well as characteristic a, c, and v-wave excursions. In no case was "wall-artifact" encountered, or developed, after initial calibration and positioning of the recorded limb. Panel C demonstrates the range of PVP–CVP gradient among three separate cases and reveals the relatively increased degree of respiratory variation seen in CVP tracings compared with PVP tracings.

Simultaneous PVP and CVP tracings for each of the 15 cases are displayed in Figure 2. In most cases, an offset (PVP > CVP) is evident, but the degree of offset tended to remain relatively constant throughout each case. Periods of hemodynamic instability unmasked a tight dynamic correlation between the two variables.

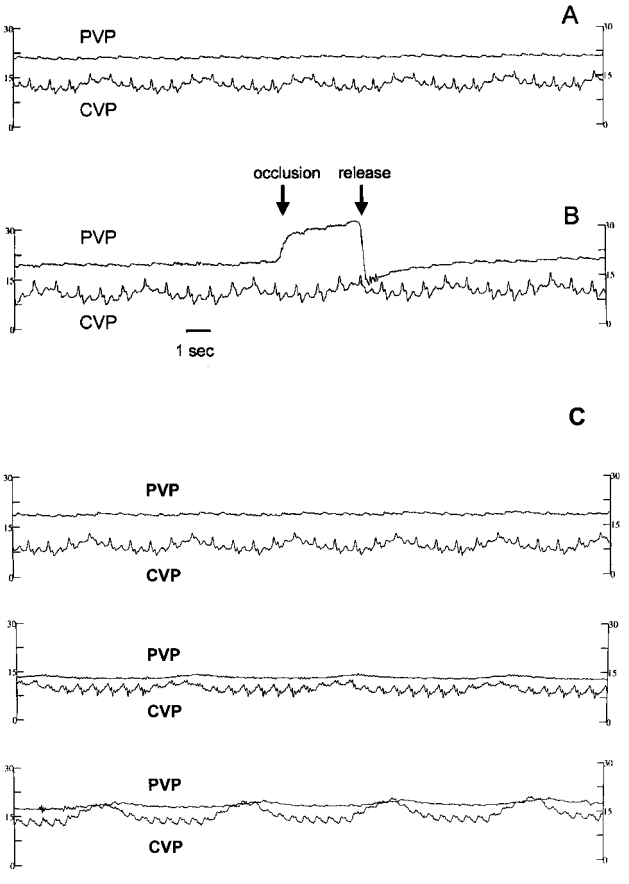

Figure 1. Pressure waveforms from simultaneous peripheral venous pressure (PVP) and central venous pressure (CVP) measurements at the beginning of case 4. Panel A displays an uninterrupted 24-s recording interval. Panel B displays the effect of manual, circumferential, proximal arm occlusion followed by release. Pressure values are in mm Hg. Panel C demonstrates the variable gradient between PVP and CVP waveforms, and the relative difference in respiratory variation seen between PVP and CVP during three separate cases. The time scale is the same for all panels.

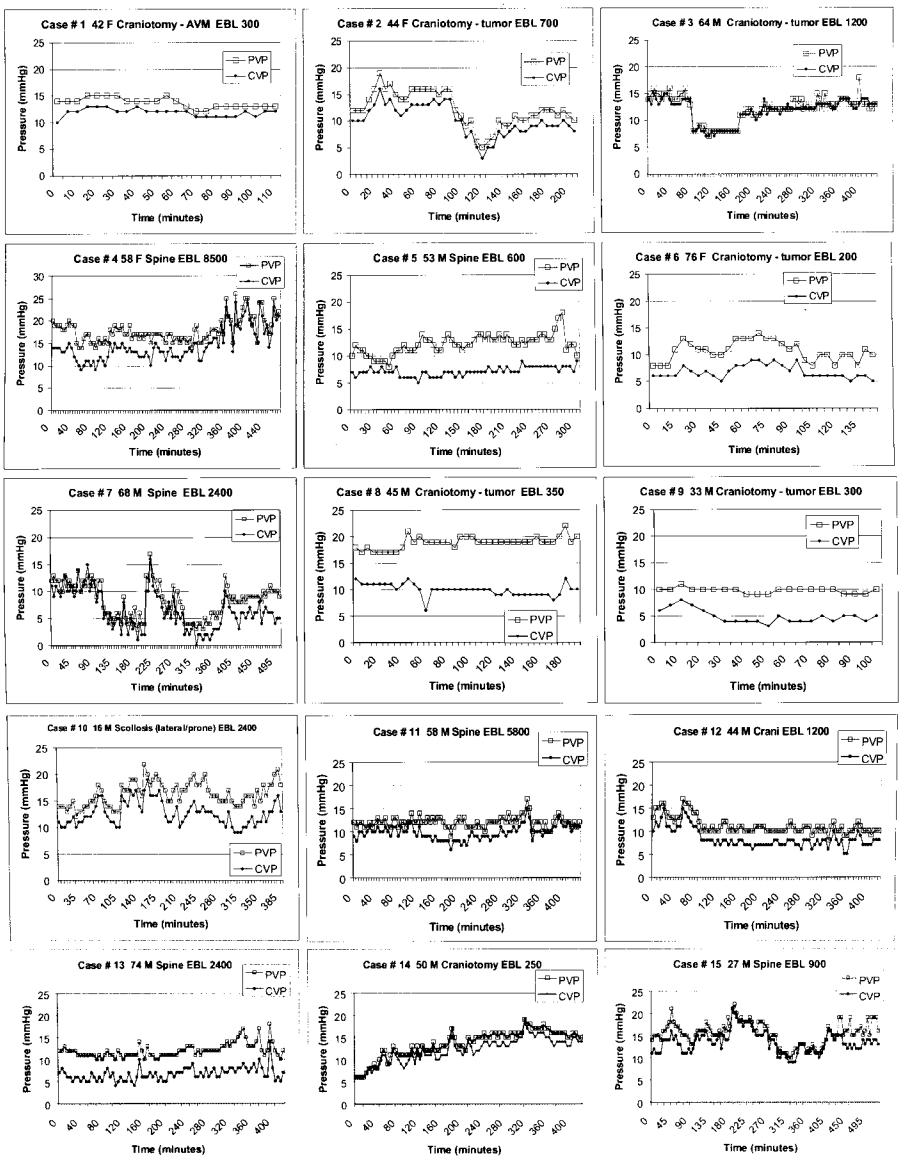

Figure 2. Hemodynamic profiles for peripheral venous pressure (PVP) and central venous pressure (CVP) in all 15 cases. Pressures and time intervals are as labeled in each panel. All 1026 data measurement intervals are displayed. Patient and surgery characteristics are abbreviated in each, and appear in detail in Table 1.

In contrast, cases with little hemodynamic (CVP) instability (for example, 5 and 8) also revealed little change in PVP, and the correlation coefficient was predictably small in those cases. Repeated-measures analysis of variance revealed a significant relationship between PVP and CVP ($P < 0.001$). Table 2 displays statistical tests of correlation for individual cases, as well as the overall correlation from 1026 paired measurements ($r = 0.82$). Tests of correlation for cases with large estimated blood loss (>1000 mL; $r = 0.885$), and large CVP variability (SD CVP > 2; $r = 0.923$) revealed essentially the same result as is visually evident from Figure 2: where CVP trends changed rapidly, so did PVP trends, and in synchrony with each other. The mean CVP value in these patients was 10 mm Hg and the mean PVP value was 13 mm Hg, with an overall difference of 3 mm Hg. Our data revealed no consistent relationship between absolute PVP values, or the PVP–CVP gradient, and catheter site (data not shown) or position (Table 2). Similarly, no consistent relationship was evident between type of surgery, anesthetic technique, or patient position and the PVP–CVP gradient.

We counted the individual sampling intervals that demonstrated either a movement of PVP and CVP in opposite directions (7 out of 921), or movement of one variable by >2 mm Hg without a corresponding movement in the other variable (6 out of 921). The cumulative frequency of either of these events was 13/921 (1.4%).

Figure 3A reveals the relationship between CVP and PVP in a scatter plot that includes all 1026 data points.

Table 2. Summary Statistics for 15 Cases

Case	AVG. PVP (mm Hg)	SD PVP (mm Hg)	AVG. CVP (mm Hg)	SD CVP (mm Hg)	EBL (mL)	r
1	13.7	0.93	11.8	0.78	300	0.617
2	11.2	3.29	10.1	2.92	700	0.989
3	12.2	2.44	11.7	2.22	1200	0.915
4	17.8	2.77	14.5	3.62	8500	0.906
5	12.2	1.82	7.2	0.81	600	0.215
6	10.7	1.84	6.8	1.25	200	0.792
7	8.3	3.14	6.4	3.50	2400	0.908
8	18.9	1.12	10.0	1.15	350	−0.127
9	9.8	0.54	4.9	1.26	300	0.553
10	16.4	2.23	12.6	2.30	2400	0.818
11	12.1	1.19	9.8	1.49	5800	0.603
12	11.2	1.88	8.4	2.05	1200	0.943
13	12.0	1.61	6.7	1.50	2400	0.758
14	13.6	3.09	12.2	2.81	250	0.966
15	15.5	2.63	13.5	2.41	900	0.805
Overall	13.19	3.68	10.19	3.72	—	0.819
Cases with EBL > 1000	—	—	—	—	—	0.885
Cases with SD CVP > 2	—	—	—	—	—	0.923

AVG. = average value; PVP = peripheral venous pressure; CVP = central venous pressure; EBL = estimated blood loss; r = correlation coefficient.

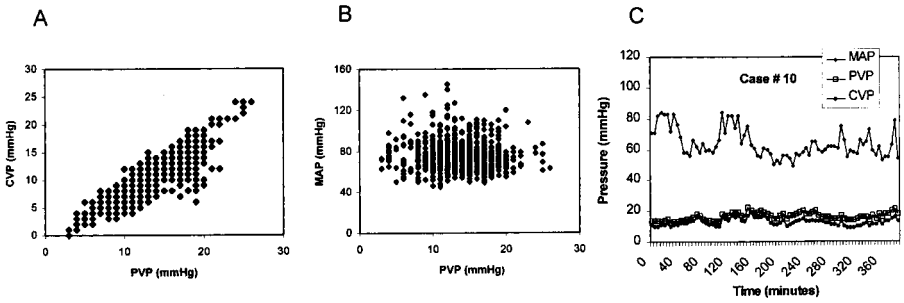

Figure 3. Correlation between peripheral venous pressure and central venous pressure, and absence of correlation between mean arterial pressure and peripheral venous pressure. Panel A displays central venous pressure (CVP) versus peripheral venous pressure (PVP) for all cases. Each marked data point (diamond) contains at least one measured CVP-PVP value, but may include more than one measured value. Panel B displays mean arterial pressure (MAP) versus PVP in the same format as Panel A, for all cases. Panel C displays simultaneous MAP, PVP, and CVP hemodynamic profiles for case 10.

Because of the large number of measurements, each point on the graph represents at least one, but may represent more than one, paired measurement. In contrast, the scatter plot of MAP versus PVP in Figure 3B reveals no consistent relationship between the two variables. This is also graphically evident in each case where MAP trends are included along with PVP and CVP trends (representative example in Fig. 3C).

Figure 4 displays data before CPB (A), immediately before and during (B), and after (C, D) circulatory arrest. Compared with each of the other hemodynamic variables (ART, PAP, and CVP), PVP revealed a more stable waveform, and was the only pressure that remained constant through the arrest transition. After arrest, ART and PAP decreased, and CVP increased to converge on an essentially unchanging PVP. That pattern defined PVP as equivalent to mean systemic pressure for the case. It is not clear why the arterial pressure plateaued briefly before converging to PVP, but this deviation from a simple monoexponential decay is characteristic of other circulatory arrests in our institution (data not shown). Panel 4E displays hemodynamic data from a separate patient with intact circulatory function. During each diastolic runoff, ART decreased and CVP trended upward (arrows), but PVP remained stable, revealing the same tendency toward pressure convergence to PVP as during circulatory arrest, but interrupted by the next systole.

Discussion

It is not surprising that PVP and CVP trends are linked, given that the two sites of measurement are part of the same venous continuum. The demonstration of that relationship in surgical patients raises several questions, however. First, why has the coupling of PVP to CVP not been noticed and taken

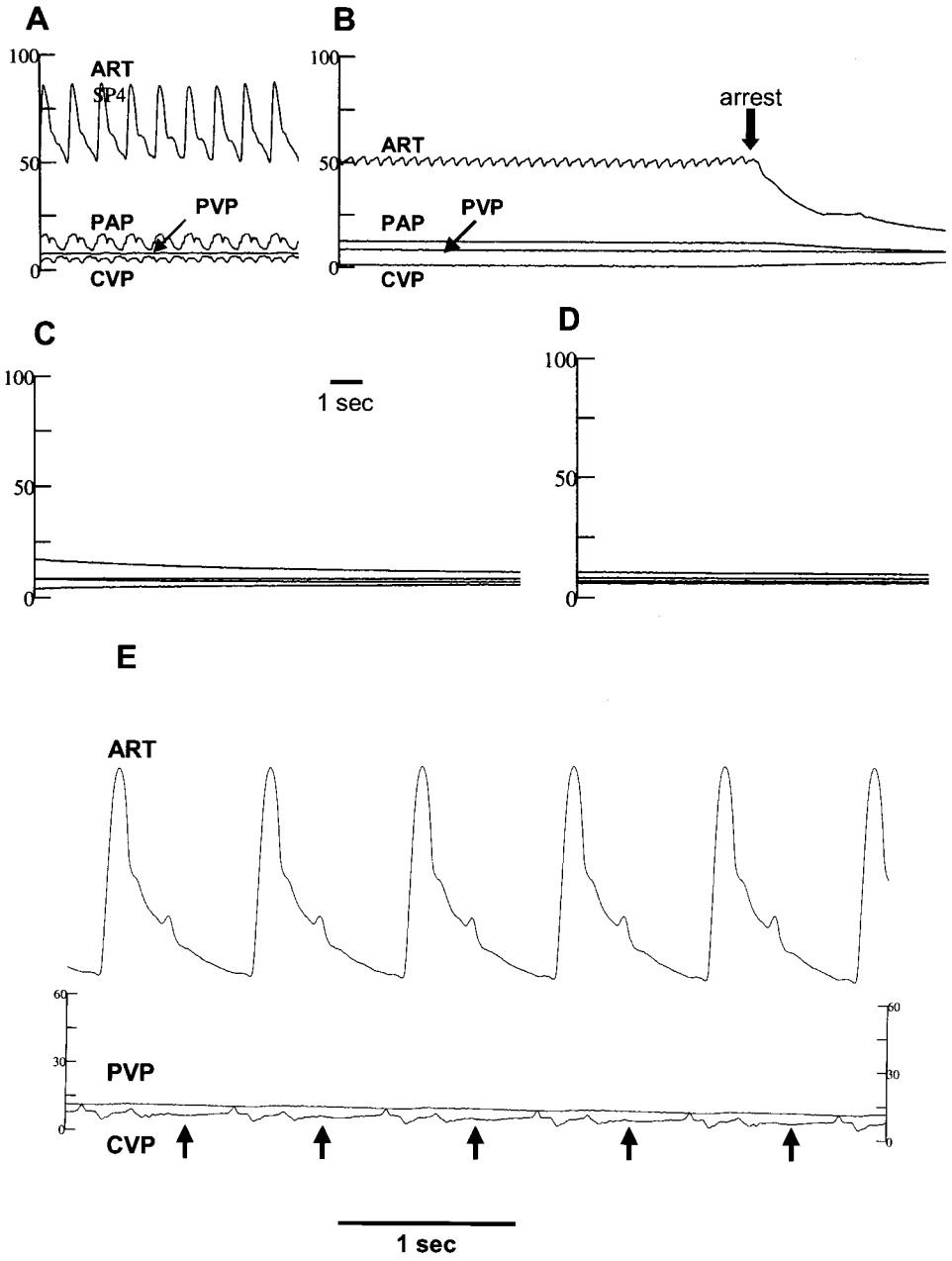

Figure 4. Hemodynamic variables during controlled circulatory arrest (panels A-D), and during diastolic runoff in the intact circulation (panel E). Panel A, before cardiopulmonary bypass. Panel B, immediately before and during circulatory arrest. Panels C and D, continuation of Panel B. ART = arterial blood pressure; PAP = pulmonary artery blood pressure; PVP = peripheral venous pressure; CVP = central venous pressure.

advantage of before? The expectation that PVP and CVP are related is not new. Since Holt's (12) demonstration in 1943 that the pressure in the antecubital vein tracks intrathoracic pressures in spontaneously breathing patients, several studies have attempted to define that coupling as a diagnostic tool (13). With the availability of cardiac catheterization techniques in 1952, Pedersen (14) measured the pressure gradients between the antecubital vein and the right atrium in 18 spontaneously breathing adults. He found that the gradient varied between 0.7 and 5.8 cm H_2O, with a mean value of 2.6 cm H_2O. He also noted that the respiratory and cardiac excursions evident in the right atrium were largely absent in the periphery. Those observations are in general accord with our own, and precede ours by a half-century.

Each of the early studies that examined PVP-CVP gradients revealed a significant variability between subjects. Given that variability, and the primitive techniques for hemodynamic measurement in that era, it is not surprising that PVP measurement never matured into a clinically useful tool. The claim by Sykes (15), in 1963, that measurement of CVP was a useful adjunct to volume resuscitation was criticized for being technically unwieldy, and never widely adopted. Even in 1970, when Eustace (16) explored the relationship between PVP and CVP in surgical patients, the use of fluid manometers, widely separated measurement

points, and an inability to separate mean pressure values from maximum and minimum manometric excursions handicapped any effort to define a dynamic relationship between PVP and CVP.

More recently, we have demonstrated that PVP correlates to blood volume changes in an animal model (r = 0.997 for PVP compared with hemorrhage volume; unpublished data). This result is consistent with the observations of another group (17–20), which used a retrograde venous balloon-occlusion catheter in anesthetized dogs during either hemorrhage or volume loading to examine the relationship between "postcapillary occlusion pressure" and volume status. The result is also consistent with reports of single-point comparisons between PVP and CVP during surgery (21), and in the postanesthesia care unit (22). For this clinical study, we chose to use standard peripheral IV catheters. Despite its simplicity, the technique of measuring PVP from existing or standard arm vein catheters is apparently free from vessel wall or occlusion artifact, and lends easy accessibility to the technique.

A second question raised by our results is whether the measurement of PVP reveals local (peripheral vein), rather than systemic, physiological information. On an empirical level, the correlation between PVP and CVP argues that PVP reflects a systemic phenomenon. At that same empiric level, we chose to study a wide variety of patient and arm positions, surgeries, blood loss and hemodynamic profiles, and catheter sites. If the measurement of PVP was prejudiced by local venous tone and position variables, it is unlikely that such a robust coupling to CVP trends would have been demonstrated. We also know from clinical experience that, in the absence of extravasation or obstruction by clotting, IV catheters continue to flow unimpeded into the central circulation throughout surgery. This implies that fluid continuity is maintained between PVP and CVP sites despite changes in venous geometry that may occur with repositioning, and despite any venous valves that may intervene between the PVP site and the central circulation. Such valves are, by definition, open during steady state venous flow, and should therefore not disrupt fluid continuity between the two sites.

At a physiological level, the PVP reflects "downstream" pressures in the right atrium analogous to the pulmonary artery occlusion catheter reflecting pressure phenomenon in the left atrium. Because the PVP site is postcapillary, vessel occlusion is not required to eliminate arterial pressure influences. The isolation of the PVP waveform, and PVP steady-state pressures, from arterial pressure changes is demonstrated in Figures 1 and 3 B and C, respectively.

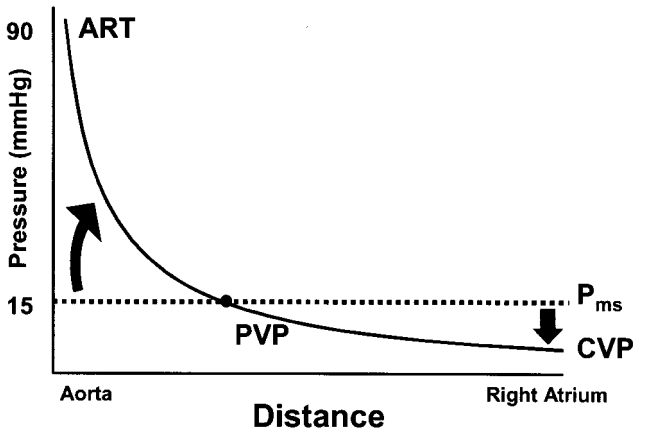

Figure 5. Schematic diagram of the systemic pressure decay curve. For clarity, systolic pressure excursions are eliminated from the arterial region of the diagram. The dotted horizontal line represents mean systemic pressure (P_{ms}–defined in Discussion), which is equivalent to circulatory arrest pressure. The arrow on the left represents the increase of arterial pressure higher than P_{ms} by cardiac activity. The arrow on the right represents the depression of central venous pressure (CVP) less than P_{ms} by cardiac activity. Peripheral venous pressure (PVP) approximates P_{ms}, and therefore, is relatively independent of cardiac influences and left to reflect volume status alone. ART = arterial pressure.

Our interest in PVP goes beyond its potential role as a substitute for CVP monitoring in selected patients. The initial physiological rationale for our study was to measure vascular pressure at a point in the systemic circulation close to mean systemic pressure (P_{ms}), which is a static, circulatory arrest pressure first defined by Starling and others (23), and refined by Guyton et al. (24) as part of an analysis of venous return (see Fig. 5). During controlled circulatory arrest in humans, all vascular pressures equalize to approximately 15 mm Hg (unpublished observations from planned adult circulatory arrest anesthetics at our institution), which is coincident with the observed arrest pressure noted by Levy (25) in a dog model. This value is simply the ratio of blood volume to vascular compliance. It is precisely this ratio that is useful in defining "relative blood volume." With steady-state cardiac activity, arterial pressures are increased, and CVP depressed, according to the ratio of arterial-to-venous compliances. CVP reflects, then, the operation of cardiac activity on P_{ms}, whereas PVP, as an "upstream" venous pressure, is by definition closer to P_{ms} and less influenced by cardiac activity.

The variable offset between patients may reflect either technical variables, such as catheter tip height differences, or physiological variables, such as differences in venous flow and resistance. The gradient between PVP and CVP was remarkably constant within subjects, however, and the small intersubject variability should not detract from the use of PVP as a trend monitor, in exact analogy to the use of CVP as a trend

monitor. Throughout the 1026 measurement intervals, the frequency of single point PVP and CVP divergence was only 1.4%, suggesting that PVP trend information could have functioned as an equivalent physiological monitor for these cases. This observation is consistent with the data from parallel animal studies indicating that PVP was at least as good as CVP in estimating volume changes (unpublished data).

In summary, these results are consistent with the results of a parallel animal study that demonstrate a high degree of correlation between PVP and changes in absolute blood volume. The results are also consistent with the hypothesis that PVP reflects mean systemic pressure; and therefore PVP measurement may provide a method of estimating mean systemic pressure during normal circulatory function. Together with the observation of a strong correlation between PVP trends and CVP trends in both craniotomy and complex spine surgery, PVP appears to warrant further investigation as a clinical volume monitor.

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