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Measurement of Pulmonary Artery Diastolic Pressure From the Right Ventricle

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Objectives. **This study evaluated the feasibility of estimating pulmonary artery end-diastolic pressure from within the right ventricle. If feasible, this could have important implications for long-term hemodynamic monitoring.**

Background. **Right ventricular pressure at the time of pulmonary valve opening closely approximates pulmonary artery end-diastolic pressure. Because maximal first derivative of right ventricular pressure (dP/dt) can be easily measured, if it occurs at or very near pulmonary valve opening, right ventricular pressure at maximal right ventricular dP/dt would be an estimation of pulmonary artery end-diastolic pressure.**

Methods. **In 10 patients undergoing routine right and left heart catheterization, simultaneous measurements were made using micromanometers in the right ventricle and pulmonary artery at baseline, during isometric work and Valsalva maneuver. Right ventricular pressure at maximal right ventricular dP/dt was considered the estimated pulmonary artery end-diastolic pressure and was compared with the actual pulmonary artery end-diastolic pressure.**

Results. **At baseline, estimated and actual pulmonary artery end-diastolic pressures were (mean ± SD) 17.7 ± 6.6 and 16.7 ± 6.7 mm Hg, respectively (p = NS). During isometric stress, estimated and actual pulmonary artery end-diastolic pressures were 30.4 ± 12.7 and 28.4 ± 10.1 mm Hg, respectively (p = NS). During Valsalva maneuvers, estimated and actual pulmonary artery end-diastolic pressures were 36.5 ± 17.8 and 38.0 ± 16.1 mm Hg, respectively (p = NS).**

Conclusions. **Although more extensive testing is necessary to evaluate validity in different physiologic and pathologic situations, it appears that right ventricular pressure at maximal right ventricular dP/dt can provide accurate estimation of pulmonary artery end-diastolic pressure.**

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It has long been recognized that pulmonary artery end-diastolic (1–3) and pulmonary artery wedge (1–4) pressures are comparable in value to left ventricular end-diastolic pressure and, hence, are acceptable variables of left ventricular preload in a majority of physiologic (and pathophysiologic) states. Today, this concept is utilized regularly in cardiovascular medicine. The balloon-tipped, flow-directed catheter is commonly used in emergency cardiac care units, medical and surgical intensive care units and perioperatively for indirect left ventricular preload evaluation by measurement of pulmonary artery end-diastolic or pulmonary artery wedge pressure, or both. Continuous electrocardiographic (ECG) and other monitoring techniques in these emergency settings minimize the risk that use of these catheters will induce clinically significant arrhythmias or other complications, or both.

Long-term monitoring of left ventricular preload, including monitoring in the ambulatory setting, might be very useful but is currently limited by technologic and conceptual constraints. There are preliminary reports of a fully implantable system capable of monitoring pulmonary artery end-diastolic pressure by means of a transvenously delivered pressure-sensing catheter with a sensor positioned in the pulmonary artery (5,6). In that system the proximal connector is attached to a battery-powered device in a permanent pacemaker-like configuration that telemeters the pressure signal. Potential difficulties, related to lack of catheter stability at the distal end in the pulmonary artery, make such a system problematic. Ventricular arrhythmias induced by the catheter as it passes through the right ventricle and displacement of the catheter tip forward into the pulmonary artery wedge position or backward into the right ventricle could each represent a serious complication. On a technologic level, long-term left ventricular preload monitoring might be more easily accomplished if a variable reflective of left ventricular preload could be measured using a catheter fixed in the right atrium or right ventricle, much like a pacemaker lead.

To our knowledge, no right atrial or right ventricular pressure or other variable has yet been shown to be reflective of left ventricular preload. We therefore based this preliminary study on the hypothesis that right ventricular pressure at the time of pulmonary valve opening is equal to pulmonary artery

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Table 1. Clinical Characteristics and Hemodynamic Variables for 10 Study Patients

Pt No./ Gender	Age (yr)	Underlying Disease	HR (beats/min)	Fick CO (liters/min)	Fick CI (liters/min per m^2)	LVEDP (mm Hg)	LVEF (%)	SVR (dynes·s·cm^{-5})	PVR (dynes·s·cm^{-5})
1/M	58	CAD, valvular disease (AS)	53	4.58	2.08	40	35	1,208	213
2/F	29	Valvular disease (with MS)	66	3.20	1.88	16	58	1,556	21
3/M	31	Idiopathic cardiomyopathy	110	5.56	2.32	NA	NA	1,151	273
4/M	62	CAD, valvular disease (AS)	48	4.51	2.37	15	67	1,440	196
5/M	50	s/p cardiac transplantation	75	6.0	2.40	18	73	1,166	173
6/M	38	CAD, CHF	76	9.67	4.83	20	36	713	17
7/F	42	Valvular disease (with MS)	94	5.80	3.87	12	59	1,393	138
8/F	57	Dyspnea, valvular disease (AS)	74	7.60	4.0	20	62	1,263	126
9/M	50	CAD	65	5.60	3.29	10	59	1,861	131
10/F	45	Valvular disease (AR, MR)	68	5.14	3.21	20	66	1,717	67
Mean	46.2		72.9	5.77	3.03	19.0	57.2	1,346.8	135.5
±SD	11.3		18.2	1.79	0.97	8.7	13.2	325.8	83.0

AR (MR) = aortic (mitral) regurgitation; AS (MS) = aortic (mitral) stenosis; CAD = coronary artery disease; CHF = congestive heart failure; CI = cardiac index; CO = cardiac output; F = female; HR = heart rate; LVEDP = left ventricular end-diastolic pressure; LVEF = left ventricular ejection fraction; M = male; NA = not available; Pt = patient; PVR = pulmonary vascular resistance; SVR = systemic vascular resistance.

end-diastolic pressure, and we evaluated the relation between variables of right ventricular and pulmonary artery end-diastolic pressure, an established and widely accepted index of left ventricular end-diastolic pressure.

Methods

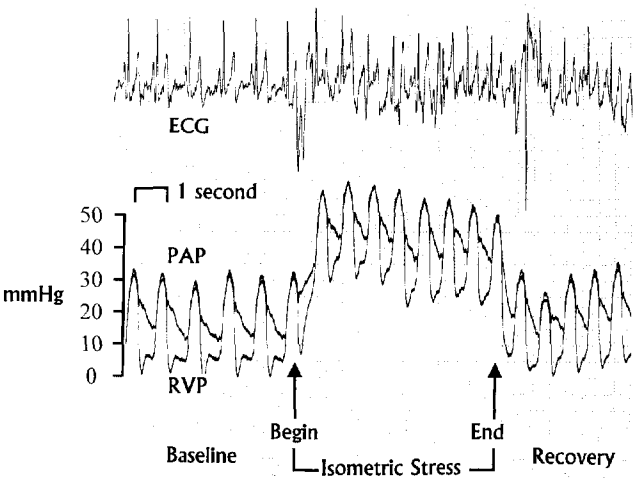
Patient selection. Ten randomly selected patients undergoing right and left heart catheterization were studied (four women, six men; mean age 46.2 years, range 29 to 62). Clinical characteristics and hemodynamic variables are shown in Table 1. Hemodynamic variables were measured or derived during the same cardiac catheterization, but data for fluid administration, timing with respect to angiography and other potential hemodynamic influences were acquired at substantially different times and frequently at different conditions compared with the primary data of this report. Institutionally approved informed written consent was obtained from all patients before cardiac catheterization.

Millar catheter placement. A custom-made 7F Millar catheter was introduced into the pulmonary artery through the femoral vein by means of an 8F 100-cm introducer sheath (Bard USCI). The catheter had one micromanometer pressure transducer positioned 6 cm proximal to the distal tip and a second similar transducer 5 cm proximal to the first. The catheter was positioned such that the distal transducer was in the common pulmonary artery, with the proximal transducer in the right ventricle (7).

Data recording. Three surface ECG leads, pulmonary artery pressure and right ventricular signals were displayed on the catheterization laboratory recorder (Electronics for Medicine) and continuously recorded on a magnetic tape recorder (Hewlett-Packard model #3968A Instrumentation Recorder) for subsequent analysis. Figure 1 is an example of a strip chart record of these variables both at rest and during one of the two physiologic stresses (isometric exercise).

Pressure transducer calibration. The dual transducers of the Millar catheter were zeroed and balanced before and after each recording in each patient. Pressure calibration signals (100 mm Hg/V) were also recorded. Transducer zeroing and balancing were done according to manufacturer instructions by making these adjustments while the transducers were slightly submerged in the horizontal plane in physiologic saline solution. However, unless the two transducers are both precisely in a horizontal plane when inserted into the subject, hydrostatic pressure gradients between the transducers can result. Two pressure transducers separated by 5 cm will, if positioned vertically in a fluid column, read a pressure difference of 3.5 mm Hg (5 cm of water × 0.7 mm Hg/cm of water). In this study, we did not document the physical locations of the two transducers positioned in the patients. Because none of these

Figure 1. Actual strip chart recording of several cardiac cycles 5.5 s before, 6 s during and 4.5 s after isometric stress. ECG = electrocardiogram; PAP = pulmonary artery pressure; RVP = right ventricular pressure.



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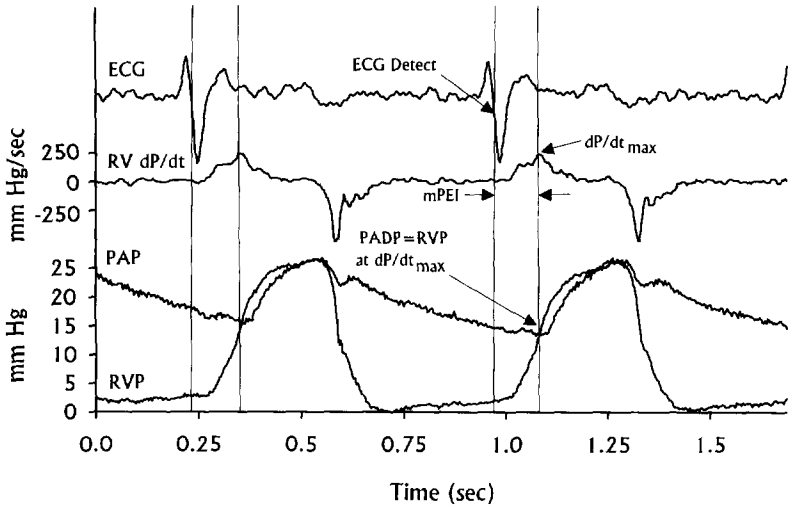

Figure 2. Schematic illustration of computer detection of the QRS complex (ECG Detect), maximal first derivative of right ventricular (RV) pressure (dP/dt_{max}) and modified preejection interval (mPEI) and the hypothesis that pulmonary artery diastolic pressure (PADP) equals right ventricular pressure at maximal dP/dt. Other abbreviations as in Figure 1.

patients had obvious pulmonary valve disease, and because we did not document the hydrostatic pressure differences between the transducers in the pulmonary artery and right ventricle, we assumed that we should have observed no systematic differences in pulmonary artery and right ventricular systolic pressures. However, because this source of systematic uncertainty was known to be possible in our signals, and because we saw pulmonary artery systolic pressures systematically 3.8 mm Hg greater than right ventricular systolic pressures in 9 of 10 patients, we applied a correction factor of −1.9 mm Hg to all pulmonary pressure recordings and a correction factor of +1.9 mm Hg to all right ventricular pressure signals before data analysis for these patients. Despite this approach, it is likely that the right ventricular transducer is at a "higher" position than the pulmonary artery transducer and that a portion of this "offset" is related to this anatomic relation.

Baseline pressure recordings. Baseline pressures were recorded for several minutes with patients in a supine position at quiet rest. No significant problems were encountered in making recordings for any of the patients.

Isometric exercise stress. Patients were instructed to conduct static (isometric) arm exercise, without breath holding, to voluntary end points while the ECG and pulmonary artery and right ventricular pressures from the micromanometers were continuously recorded on an analog tape recorder. The isometric work consisted of resisted unilateral flexion at the elbow. Although the duration of isometric stresses was carefully noted in the data recordings, the strength of the patient's individual work levels was not quantified. Further, the recorded data suggest that Valsalva maneuvers were performed by some patients during the isometric work.

Valsalva maneuvers. Patients were also instructed to conduct brief Valsalva maneuvers for 10 to 20 s while the ECG and pulmonary artery and right ventricular pressures from the micromanometers were continuously recorded. The duration of the strain phase of the maneuver was carefully noted in the data recordings, but the strength of the strain phase of the maneuvers was not quantified.

Data processing. Tape-recorded signals (625-Hz frequency response) were played into a custom personal computer–based data analysis system. This system performed analog-to-digital conversion on each signal at 200 Hz, detected each QRS complex from the surface ECG signal to mark the onset of each beat and then processed the pulmonary artery and right ventricular pressure signals on a beat-by-beat basis (Fig. 2). Pulmonary artery systolic and diastolic pressure values were determined for each beat. Simultaneously, right ventricular systolic and minimal diastolic pressures were measured. In addition, from each cardiac cycle, we determined the peak positive value of the first derivative of the right ventricular pressure waveform (maximal dP/dt). The time from the computer detection of the QRS complex to occurrence of maximal dP/dt is defined as the modified preejection interval, because maximal dP/dt occurs at the end of the isovolumetric contraction period (8–11). Additionally, the value of right ventricular pressure at the time of occurrence of maximal dP/dt was determined and, according to the hypothesis of the present study, presumed to be an estimate or index of actual pulmonary artery diastolic pressure because it happens at pulmonary valve opening (8,9).

Statistical analysis. Steady state data comparisons were made using two-tailed paired comparisons (Student *t* test), and differences were considered significant at $p \leq 0.05$. Dynamic data evaluation was done using simple linear regression of serial, beat-by-beat data over selected time periods. Where the estimated and actual pulmonary diastolic pressures were analyzed using linear regression, validity of the correlation coefficients for these two time-varying measurements was confirmed by cross-correlation analysis. Results are reported as mean value ± SD unless otherwise noted. A Bland-Altman plot of data was also developed.

Table 2. Baseline Data

	Pulmonary Artery (mean ± SD)				Right Ventricle (mean ± SD)				
Pt No.	Systolic Pressure (mm Hg)	Pulse Pressure (mm Hg)	Mean Pressure (mm Hg)	Diastolic Pressure (mm Hg)	Systolic Pressure (mm Hg)	Diastolic Pressure (mm Hg)	ePADP (mm Hg)	Pos dP/dt (mm Hg/s)	mPEI (ms)
1	35 ± 4	17 ± 4	23 ± 3	18 ± 4	38 ± 4	2.8 ± 2.9	22 ± 4	374 ± 67	54 ± 20
2	32 ± 3	14 ± 4	23 ± 2	17 ± 2	32 ± 3	1.2 ± 0.8	16 ± 3	298 ± 48	75 ± 13
3	50 ± 5	16 ± 5	42 ± 5	33 ± 5	45 ± 6	7.3 ± 3.4	33 ± 5	206 ± 49	82 ± 26
4	57 ± 3	39 ± 3	29 ± 2	17 ± 3	58 ± 3	0.4 ± 1.8	18 ± 2	335 ± 24	72 ± 7
5	32 ± 8	17 ± 10	22 ± 7	15 ± 9	32 ± 9	−1.4 ± 6.4	16 ± 6	260 ± 47	115 ± 9
6	20 ± 1	11 ± 1	13 ± 1	10 ± 2	21 ± 1	−0.6 ± 1.1	12 ± 1	260 ± 15	62 ± 5
7	49 ± 5	28 ± 5	33 ± 4	21 ± 5	53 ± 4	−4.9 ± 3.2	21 ± 4	747 ± 146	85 ± 8
8	35 ± 5	19 ± 4	24 ± 5	16 ± 6	32 ± 5	0.0 ± 4.9	14 ± 5	375 ± 48	69 ± 6
9	28 ± 1	16 ± 2	18 ± 1	11 ± 2	26 ± 1	0.5 ± 0.7	16 ± 1	262 ± 21	91 ± 5
10	29 ± 2	19 ± 1	19 ± 2	10 ± 2	26 ± 2	−7.0 ± 1.8	8 ± 3	460 ± 62	65 ± 4
All pts	37 ± 12	20 ± 8	25 ± 8	17 ± 7	36 ± 12	−0.2 ± 3.9	18 ± 7	358 ± 156	77 ± 17

ePADP = estimated pulmonary artery end-diastolic pressure; mPEI = modified preejection interval; Pos dP/dt = maximal positive first derivative of right ventricular pressure; Pt (pts) = patient(s).

Results

Baseline pressures. Values for baseline pulmonary artery and right ventricular pressures are summarized in Table 2. Pulmonary artery and right ventricular systolic pressures were 36.7 ± 11.6 and 36.3 ± 12.2 mm Hg, respectively. Pulmonary artery and right ventricular minimal diastolic pressures were 16.7 ± 6.7 and −0.2 ± 3.9 mm Hg, respectively. These values for systolic and diastolic pressures yielded a pulmonary artery pulse pressure of 19.7 ± 8.3 mm Hg. Mean pulmonary artery pressure was 24.6 ± 8.3 mm Hg.

Waveform analysis indicated that maximal dP/dt in the right ventricle occurred an average of 76.8 ± 17.3 ms after detection of the QRS complex in surface lead II of the ECG. Right ventricular pressure at the time of maximal positive dP/dt, determined beat by beat, was 17.7 ± 6.6 mm Hg, which represents the estimated pulmonary artery diastolic pressure and was not significantly different from the actual pulmonary artery diastolic pressure of 16.7 ± 6.7 mm Hg.

Pressure changes during stress. Figure 3 illustrates typical examples of the actual and estimated pulmonary artery diastolic pressure values recorded during an isometric exercise maneuver in one patient (Fig. 3, top) and during a Valsalva maneuver in another (Fig. 3, bottom). For isometric work (Fig. 3, top), actual and estimated pulmonary artery end-diastolic pressures were 9.7 ± 1.9 and 8.4 ± 2.5 mm Hg, respectively, over the 15-s period before, and then increased to 34.9 ± 3.3 and 30.3 ± 3.6 mm Hg, respectively, during, the strain phase of the maneuver. In the eight patients who attempted isometric exercise maneuvers, actual pulmonary artery diastolic pressures increased from 14.2 ± 3.6 to 28.4 ± 10.1 mm Hg (Table 3). Estimated pulmonary artery diastolic pressures measured from within the right ventricle increased from 15.4 ± 4.0 to 30.4 ± 12.7 mm Hg (p = NS for actual versus estimated pulmonary artery end-diastolic pressure changes). Although we focused on pulmonary artery diastolic pressure (as a reflection of left ventricular filling pressure), we also observed

Figure 3. Illustration of beat-by-beat values of actual (**solid squares**) and estimated pulmonary artery diastolic (**open squares**) pressures and the error of or difference between estimated and actual pulmonary artery diastolic pressures (**solid circles**) during isometric exercise stress in one patient (**top**) and Valsalva maneuver in another patient (**bottom**). The estimated pressure is the value of right ventricular pressure at maximal dP/dt. (The single elevated pressure values at 25 s into the record represents a pressure increase after the isometric stress caused by a patient's cough.)

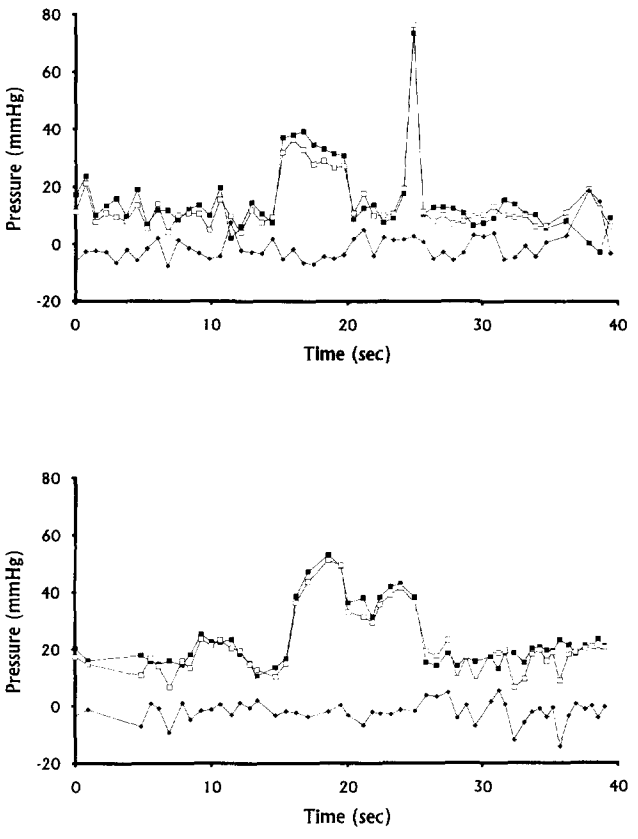

Table 3. Isometric Exercise and Valsalva Data

	Pulmonary Artery (mean ± SD)				Right Ventricle (mean ± SD)				
Pt No.	Systolic Pressure (mm Hg)	Pulse Pressure (mm Hg)	Mean Pressure (mm Hg)	Diastolic Pressure (mm Hg)	Systolic Pressure (mm Hg)	Diastolic Pressure (mm Hg)	ePADP (mm Hg)	Pos dP/dt (mm Hg/s)	mPEI (ms)
Isometric									
1	48 ± 5	23 ± 5	33 ± 4	26 ± 6	48 ± 7	5 ± 3	31 ± 8	401 ± 76	53 ± 28
2	33 ± 4	13 ± 6	26 ± 3	19 ± 3	33 ± 5	2 ± 2	18 ± 3	332 ± 52	73 ± 19
4	63 ± 8	40 ± 4	34 ± 7	23 ± 7	64 ± 8	6 ± 7	24 ± 6	357 ± 50	69 ± 8
5	63 ± 13	19 ± 2	52 ± 12	43 ± 12	64 ± 13	26 ± 11	52 ± 13	343 ± 12	107 ± 6
6	25 ± 3	10 ± 2	18 ± 4	15 ± 3	26 ± 3	5 ± 5	19 ± 3	246 ± 56	64 ± 9
8	43 ± 8	18 ± 6	33 ± 8	25 ± 8	39 ± 9	6 ± 8	22 ± 7	417 ± 35	70 ± 4
9	59 ± 2	18 ± 3	49 ± 2	41 ± 4	59 ± 2	28 ± 4	47 ± 3	337 ± 24	82 ± 3
10	51 ± 7	16 ± 4	40 ± 10	35 ± 3	45 ± 7	14 ± 10	30 ± 4	389 ± 30	71 ± 5
All pts	48 ± 14	20 ± 9	35 ± 11	28 ± 10	47 ± 14	11 ± 10	30 ± 13	353 ± 53	73 ± 16
Control (mm Hg)	33 ± 11	19 ± 9	21 ± 5	14 ± 4	33 ± 11	0 ± 3	15 ± 4	328 ± 72	75 ± 19
Change (mm Hg)	15 ± 12	0 ± 3	14 ± 12	14 ± 12	14 ± 12	12 ± 11	15 ± 13	25 ± 49	2 ± 5
Valsalva									
1	51 ± 5	12 ± 5	43 ± 3	39 ± 3	48 ± 5	26 ± 3	40 ± 4	246 ± 51	36 ± 8
2	51 ± 8	9 ± 6	45 ± 8	41 ± 6	50 ± 8	23 ± 8	39 ± 7	260 ± 83	103 ± 19
6	69 ± 13	7 ± 3	61 ± 10	63 ± 10	70 ± 13	49 ± 6	63 ± 10	160 ± 77	73 ± 22
8	41 ± 8	20 ± 7	30 ± 8	21 ± 8	36 ± 10	2.5 ± 7.6	17 ± 8	378 ± 92	68 ± 10
10	40 ± 7	14 ± 1	30 ± 6	26 ± 8	33 ± 7	10.1 ± 3.7	23 ± 6	333 ± 17	80 ± 6
All pts	50 ± 12	12 ± 5	42 ± 13	38 ± 16	47 ± 15	22 ± 18	36 ± 18	275 ± 84	72 ± 24
Control (mm Hg)	30 ± 6	16 ± 4	20 ± 5	14 ± 4	30 ± 6	−1 ± 4	15 ± 5	353 ± 78	65 ± 8
Change (mm Hg)	20 ± 17	−4 ± 3	21 ± 16	24 ± 18	18 ± 18	23 ± 17	22 ± 18	−78 ± 58	7 ± 17

Abbreviations as in Table 2.

that pulmonary artery systolic pressure increased from 33.4 ± 10.6 to 48.1 ± 13.7 mm Hg during isometric stress. Neither maximal positive dP/dt in the right ventricle (328 ± 72 mm Hg/s) nor the time from the QRS complex at which maximal dP/dt occurred (75.2 ± 19.2 ms) changed significantly during the brief isometric stresses.

For the Valsalva maneuver (Fig. 3, bottom), actual and estimated pulmonary artery end-diastolic pressures were 17.2 ± 2.2 and 16.0 ± 2.9 mm Hg, respectively, over the 15 s before, and increased to 41.4 ± 6.3 and 38.9 ± 7.1 mm Hg, respectively, during, the strain phase of the maneuver. In the five patients who attempted Valsalva maneuvers, actual pulmonary artery diastolic pressures increased from 14.1 ± 4.1 to 38.0 ± 16.1 mm Hg (Table 3). Estimated pulmonary artery diastolic pressures measured from within the right ventricle increased from 14.6 ± 5.0 to 36.5 ± 17.8 mm Hg (p = NS for actual versus estimated pulmonary artery end-diastolic pressure changes). Pulmonary artery systolic pressure increased from 30.2 ± 6.2 to 50.4 ± 11.8 mm Hg during Valsalva maneuvers. The Valsalva maneuvers resulted in small decreases in maximal positive dP/dt in the right ventricle (353 ± 78 to 275 ± 84 mm Hg/s, $p = 0.040$). There was a small but insignificant increase in the time from the QRS complex to maximal dP/dt (64.9 ± 7.9 vs. 71.8 ± 23.9 ms).

Dynamic response to stress. In addition to evaluating the steady state change in pressures during stress, we also evaluated the dynamic behavior of these pressure variables by observing the beat-by-beat responses. Using beat-by-beat data ~10 to 15 s before, 10 to 15 s during and 10 to 15 s after stress (isometric and Valsalva maneuvers), we determined the linear regression of the estimated versus actual pulmonary artery diastolic pressure (both stresses combined). A linear regression slope of 1.00 would have indicated that estimated pulmonary artery end-diastolic pressure changed exactly proportionally to actual pulmonary artery end-diastolic pressure, but the slope for the data from all patients combined (Fig. 4, top) was 0.88, suggesting that on average, the estimated pulmonary artery end-diastolic pressure changed somewhat less than the actual pulmonary artery end-diastolic pressure over the whole range of pressures observed when beat-by-beat data were used. The intercept value for the linear regression was 2.53 mm Hg for the pooled data from all patients. This positive intercept value suggests that the absolute values of the estimated pulmonary artery end-diastolic pressure tended to be somewhat higher than those for pulmonary artery end-diastolic pressure. Either the estimated pulmonary artery end-diastolic pressure was actually systematically higher than the actual pulmonary artery end-diastolic pressure, or the correction for systematic pressure differences in our data set based on systolic pressure was not perfect (see Methods), or the assumption of a linear relation between the variables was not perfect. Nevertheless, the standard error of the fit of the regression line for the pooled patient data was 4.32 mm Hg, and the multiple correlation coefficient (r^2) for the regression was 0.814. A Bland-Altman plot of the same data is also provided (Fig. 4, bottom).

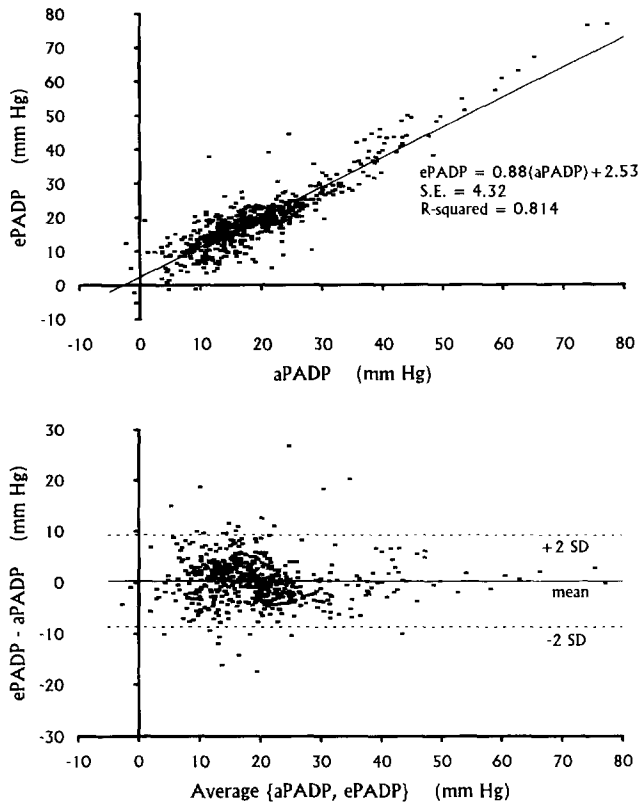

Figure 4. Top, Plot of linear regression relation between actual (aPADP) and estimated (ePADP) pulmonary artery diastolic pressures for all patient responses to isometric exercise stress and Valsalva maneuvers. Data include beat-by-beat pulmonary artery diastolic pressure values from ~40 s of values, including baseline values before and after, as well as during, each stress. The regression equation, standard error of the estimate and multiple correlation coefficient are shown. **Bottom,** Bland-Altman plot of difference between estimated and actual pulmonary artery diastolic pressures versus the mean value of their sum. Same data included as in regression plot.

Discussion

Background. There has been recent interest in using hemodynamic measurements for more objective tailoring of medical therapy in patients with clinically significant congestive heart failure (11–13) and such problems as chronic lung disease (10). Preliminary reports of applications of implantable hemodynamic sensors in patient management have appeared (5,6,14–18).

Concept of estimated pulmonary artery diastolic pressure. The potential obstacles to long-term implantation of measurement devices in the pulmonary artery are not insignificant. In contrast, implantation of long-term pressure sensors in the right ventricle as part of a permanent pacing lead has been reported without significant complication (5,6,9,14,15), although substantial effort will be necessary to ensure long-term stability of implanted transducers, and the lack of such stability could be an important limitation. Because right ventricular pacing lead placement is a highly successful medical intervention, the ability to obtain a useful estimation of pulmonary artery pressure, especially as a reflection of left ventricular filling pressure, from within the right ventricle could be advantageous. The intermittency of ventricular outflow provides this opportunity. The point in the cardiac cycle where the pressure in the right ventricle equals the pressure in the pulmonary artery, just before the transition to a positive forward pressure gradient, occurs at the end of the isovolumetric contraction phase of the ventricle (8–10). Also, because flow into the pulmonary artery begins at this point, the pulmonary artery pressure is at its end-diastolic level. Thus, determination of right ventricular pressure at the end of the isovolumetric contraction phase could serve as a measure of pulmonary artery diastolic pressure (2).

With normal pulmonary and tricuspid valve function, the dynamics of right ventricular contraction dictate that the maximal rate of pressure development (maximal dP/dt) occurs during isovolumetric contraction (8–10). As the valve opens and ejection begins, the volume in the chamber is no longer fixed, and the rate of pressure development diminishes as emptying occurs. Because maximal dP/dt occurs at the end of isovolumetric contraction (8,9), the occurrence of maximal dP/dt marks the time at which the pressure in the right ventricle equals the diastolic pressure of the pulmonary artery. We therefore hypothesized that the right ventricular pressure at maximal dP/dt could serve as a continuous (beat-by-beat) measure of pulmonary artery end-diastolic pressure.

However, because the rate of pressure change within the right ventricle is at its maximum at the specific time point when this pressure measurement is to be taken, small errors in determining time to maximal dP/dt could result in large errors in estimating pulmonary diastolic pressure. In the present data, maximal dP/dt was 358 mm Hg/s. This means that an error of 3 to 4 mm Hg could occur simply by our being off by 10 ms in determining of the occurrence of end-diastole. If maximal dP/dt were larger, then this error would be larger, and it is known that maximal dP/dt increases during many physiologic and pathophysiologic stresses (14,15,18). The objective of this preliminary study was to determine whether practical measurement methods would allow reliable determination of pulmonary artery diastolic pressure from the right ventricle using these concepts in baseline conditions, and whether this method would also allow tracking of changes in pulmonary artery diastolic pressures from the right ventricle during brief, voluntary physiologic stresses in the catheterization laboratory.

Potential limitations. The systematic assessment of conditions under which pulmonary artery diastolic or pulmonary artery wedge pressure is not a reliable measure of left ventricular filling pressure (19), such as mitral stenosis, which was present in two of our patients, was outside the scope of the present study. It also seems likely that conditions that primarily affect the right ventricle could lead to erroneous estimates of pulmonary artery diastolic pressure from the right ventricle. For example, significant tricuspid insufficiency could result in a blunted or diminished isovolumetric contraction phase with a poorly defined maximal dP/dt and pressure at maximal dP/dt. Indeed, it is in these patients in whom these measurements

may be most valuable and in whom this relation must be carefully elucidated. Other conditions may cause similar uncertainties, and delineation of such circumstances will be necessary.

The present study was conducted in supine patients using limited physiologic stresses. It remains unknown whether our findings can be extrapolated to ambulatory patients during other physiologic stresses. However, it is encouraging that the correlation between actual and estimated values was maintained over a range of dP/dt and preejection interval values.

The substantial requirements for accuracy and precision of pressure measurement needed for studies such as the present one are not trivial and can pose limitations. Pressure measurement fidelity greater than that usually accomplished in catheterization laboratories is required to determine dP/dt in the right ventricle and the occurrence of maximal dP/dt. Technologic uncertainties are compounded (and effects are cumulative) when multiple pressure-recording sites (right ventricle and pulmonary artery) are utilized. Technical issues and uncertainties noted in the present study (see Methods) exemplify these challenges.

Conclusions. Although questions remain for future studies, we interpret the findings of the present study to support our hypothesis that continuous estimates of pulmonary artery diastolic pressure can be made from pressure measurements within the right ventricle. The present concept and methodology may have important implications for future hemodynamic monitoring technologies, particularly for long-term implantable systems.

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