A Comparison of Pulmonary Artery Occlusion Pressure and Left Ventricular End-diastolic Pressure during Mechanical Ventilation with PEEP in Patients with Severe ARDS

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When positive end-expiratory pressure (PEEP) is applied to normal lungs, the pulmonary artery occlusion pressure (PAOP) may reflect alveolar pressure and not left ventricular end-diastolic pressure (LVEDP). The reliability of PAOP measurements has been questioned when PEEP levels greater than 10 cm H_2O are applied. To verify whether this disparity occurs in patients with severe lung injury, the authors simultaneously measured both PAOP and LVEDP at 0, 10, and 16–20 cm H_2O PEEP in 12 supine patients with severe adult respiratory distress syndrome (ARDS). In all patients, the radiographic location of the PA catheter tip was at or below the level of the posterior border of the left atrium. A close correlation was found between PAOP and LVEDP at each level of PEEP. In only six of 35 simultaneous measurements was the PAOP-LVEDP gradient 2 mmHg or more (2–3 mmHg in four, and 4 mmHg in two). In five patients, the highest PEEP level was 4–9 cm H_2O greater than LVEDP; however, no gradient was measured between LVEDP and PAOP. The authors conclude that, in severe ARDS, a close correspondence between PAOP and LVEDP is maintained despite applying PEEP levels up to 20 cm H_2O, suggesting that, in ARDS, surrounding pathology prevents transmitted alveolar pressure from collapsing adjacent pulmonary vessels. (Key words: Heart: left ventricular end-diastolic pressure. Lung: adult respiratory distress syndrome (ARDS); positive end-expiratory pressure; pulmonary artery occlusion pressure.)

LEFT VENTRICULAR FILLING PRESSURE is an important variable to assess in patients with ARDS because it is a major determinant of both cardiac output and pulmonary microvascular filtration rate. In clinical practice, however, left ventricular end-diastolic pressure (LVEDP) is not routinely measured; instead, a flow-directed pulmonary artery catheter is often used to estimate the pulmonary venous pressure (PVP) by measuring pulmonary artery occlusion pressure (PAOP). PAOP is believed to reflect PVP and LVEDP, provided that patent pulmonary vascular pathways are maintained between the tip of the catheter and the pulmonary veins. Several studies in experimental animals[1–4] and in humans[5,6] have demonstrated discrepancies between PAOP and directly measured left ventricular filling pressure when high levels of PEEP are applied. These discrepancies have usually been ascribed to the fact that PEEP may result in occlusion of the pulmonary capillaries such that, at high levels of PEEP, PAOP reflects alveolar pressure rather than PVP. These considerations led some authors to challenge the validity of PAOP measurements in patients treated by mechanical ventilation with PEEP.[7–10]

In patients with ARDS, a few observations have shown that abruptly discontinuing high PEEP levels did not markedly influence PAOP,[11] suggesting that, in ARDS, microvascular channels in the most severely damaged zones might be protected from alveolar pressure and that patent communications between pulmonary arteries and veins are maintained. We hypothesized that, in patients with severe acute lung injury, the close relationship between PAOP and LVEDP would be preserved, even when high levels of PEEP are used regardless of the position of the catheter tip. We thus compared intravascular and intracavitary pressures at several levels of PEEP in patients with severe ARDS.

Materials and Methods

PATIENTS

Twelve consecutive patients admitted to the Medical Intensive Care Unit of Henri Mondor Hospital for monitoring and for treatment of ARDS were studied. None had major pre-existing cardiac or pulmonary disease. The diagnosis of ARDS was based on the presence of acute bilateral diffuse pulmonary infiltrates, a Pa_{O_2} of 60 mmHg or less at an FI_{O_2} of 0.60 or more, a PAOP of less than 18 mmHg, and a recognized etiology of ARDS. The severity of their pulmonary disease was demonstrated by the high level of venous admixture (42 ± 18% at a mean FI_{O_2} of 0.9 ± 0.2 without PEEP) and the low mean quasi-static thoracopulmonary compliance value of 35 ± 12.0

TABLE 1. Clinical Variables and Outcome of 12 Patients with ARDS

Patient No.	Age (yr)	Sex	Pulmonary Disease	Quasi-static Compliance* (ml/cm H$_2$O)	Venous Admixture at ZEEP (%)	Fi$_{O_2}$	Outcome
1	54	F	Bacterial pneumonia	20	27	1	Death
2	78	F	Bacterial pneumonia	27	23	.6	Death
3	64	M	Bacterial pneumonia	39	44	1	Death
4	76	F	Carcinomatous lymphangitis	35	14	.6	Death
5	31	F	ARDS/Diabetic ketoacidosis	51	43	.6	Survival
6	49	M	Bacterial pneumonia	60	59	1	Death
7	43	M	CMV Pneumonia	45	85	1	Death
8	18	M	Acute interstitial lung disease	24	43	1	Death
9	19	F	Alveolar proteinosis	33	40	1	Survival
10	23	M	Bacterial pneumonia	34	41	1	Death
11	77	F	Bacterial pneumonia	25	50	1	Death
12	59	F	Bacterial pneumonia	26	32	1	Survival
Mean ± SD				35 ± 12.0	42 ± 18		

* Quasi-static compliance is obtained from the airway pressure/ thoracopulmonary volume curve,[13] and measured at 10 ml/kg pulmonary inflation.

ml/cm H$_2$O (table 1). In all patients, mechanical ventilation with PEEP was required to maintain an adequate arterial oxygen tension. Nine patients subsequently died of their disease during ICU therapy. Patients were excluded from this study if they had contraindications to bedside left ventricular catheterization (extensive aortofemoral arteriosclerosis, aortic stenosis, thrombocytopenia, or a coagulopathy). Informed consent was obtained from each patient's family and the protocol was approved by the Committee of Ethics of the Société de Réanimation de Langue Française. During treatment and study, all patients were sedated with a continuous intravenous infusion of flunitrazepam and given intravenous pancuronium bromide.

MEASUREMENTS

Right heart catheterization was performed with a 7F Swan-Ganz catheter (Edwards Laboratories, Santa Ana, CA) inserted percutaneously via an internal jugular vein to measure right atrial pressure (RAP), pulmonary artery pressure (PAP), and pulmonary artery occlusion pressure (PAOP). All values were measured at end-expiration. Patients were studied while supine and zero pressure was taken as atmospheric pressure at the mid-axillary line. PAOP was determined as the mean end-expiratory value measured over five or more cardiac cycles. Criteria of wedge position of the Swan Ganz catheter tip were: 1) phasic wave form (a and v waves) synchronized to the EKG; and 2) mean PAOP value less than PA diastolic pressure. Cardiac output was calculated with an Edwards model 9250 A computer, as the mean of three measurements obtained by injecting 10 ml of a 0° C dextrose solution.

Bedside left heart catheterization was performed with a 7 F pigtail catheter inserted percutaneously via a femoral

artery using the Seldinger technique. LVEDP was measured just prior to the rapid LV systolic pressure rise at the "z" point (i.e., at the end of the "a" wave).[12] An EKG lead II was continuously monitored and systemic blood pressure (BP) was recorded with a radial artery catheter. All pressures were measured via quartz transducers (Hewlett Packard 1290, Waltham, MA), and values recorded on a multichannel recorder (ES 1000, Gould Instruments, Cleveland, OH). Values are reported as intravascular pressures. All pressure catheters were continuously flushed with a saline solution containing heparin.

A two-liter syringe was used to obtain an airway pressure-lung volume curve,[13] and quasi-static thoracopulmonary compliance was calculated as volume/pressure at a 10 ml/kg tidal volume.

A lateral decubitus chest roentgenogram was obtained in each patient to determine the anatomic location of the Swan-Ganz catheter tip relative to the lower border of the left atrium, identified as the confluence of the main pulmonary veins.[14] The vertical distance between these two levels was taken as an estimate of the hydrostatic pressure gradient between the catheter tip and the zero reference level.

STUDY PROTOCOL

Measurements were obtained during mechanical ventilation at three different levels of end-expiratory pressure: zero end-expiratory pressure (ZEEP), 10 cm H$_2$O, and one higher PEEP level (20 cm H$_2$O in ten patients, 18 and 16 cm H$_2$O in two others). In one patient (patient 7), it was deemed unsafe to decrease PEEP to obtain the measurement at ZEEP. Measurements were obtained in order of descending PEEP during a 15-min delay at each level of PEEP, after hemodynamic values had stabilized.

There was no complication of either left ventricular or

TABLE 2. Individual Hemodynamic Data at Each Level of PEEP

Patient No.	PEEP Level cm H_2O	PAOP mmHg	LVEDP mmHg	PAP mmHg	RAP mmHg	BP mmHg	HR beats/ min	CI $l \cdot min \cdot m^{-2}$
1	ZEEP	10.5	9.5	37	8	72	120	3.00
	PEEP 10	14.5	11.5	41	11	80	124	3.33
	PEEP 20	14.5	15	49	12	90	132	3.44
2	ZEEP	17	17	28	11	88	128	3.17
	PEEP 10	13.5	13	25	11	85	130	3.65
	PEEP 20	11	12	24	8	75	132	3.13
3	ZEEP	7.5	7.5	18	2	102	100	3.70
	PEEP 10	11.5	11.8	28	7	82	84	3.50
	PEEP 20	15	13	35	8.5	80	80	3.90
4	ZEEP	11.5	11.5	24	9	110	132	2.55
	PEEP 10	11.5	11.5	23	8	67	128	2.12
	PEEP 16	12.5	12.5	25	9	76	120	2.22
5	ZEEP	11	11	23	9	88	140	3.92
	PEEP 10	13.5	14	27	13	91	135	3.42
	PEEP 20	12.5	12	27	12	73	135	3.14
6	ZEEP	11	15	35	8	85	110	3.50
	PEEP 10	11	14	33	10	79	112	3.40
	PEEP 20	6.5	7	27	8	50	106	2.70
7	ZEEP	—	—	—	—	—	—	—
	PEEP 10	13	12.75	27	13	60	100	5.00
	PEEP 20	15	14.5	31	15	71	104	5.02
8	ZEEP	2.5	4	13	0	67	132	3.60
	PEEP 10	7.5	8	16	6	65	115	4.43
	PEEP 20	9	12	21	8	59	128	3.61
9	ZEEP	11.5	14	23	10	97	105	5.72
	PEEP 10	10	10	25	10	77	110	4.51
	PEEP 20	13.5	13.5	27	13	67	105	3.27
10	ZEEP	11	11	25	8	86	136	2.76
	PEEP 10	12	13	28	10	83	136	2.46
	PEEP 18	13	13	29	11	80	136	2.31
11	ZEEP	10	10	26	8	54	92	3.7
	PEEP 10	11.5	12	28	9	55	95	3.7
	PEEP 20	11.5	12	25	10	51	90	2.9
12	ZEEP	6.2	6.8	17	6	76	94	3.85
	PEEP 10	6.8	8	17	6.5	78	94	3.5
	PEEP 20	8.5	8.8	19	8	71	93	2.95
Mean ± SD	ZEEP	10 ± 3.7	10.7 ± 3.8	24.5 ± 7.2	7.2 ± 3.3	84 ± 16	117 ± 17	3.6 ± 0.8
	PEEP 10	11.4 ± 2.3	11.6 ± 2	26.5 ± 6.6*	9.5 ± 2.3*	75 ± 11	113 ± 17	3.6 ± 0.8
	PEEP 16–20	11.9 ± 2.7	12.1 ± 2.2	28.2 ± 7.8†	10.2 ± 2.4†	70 ± 12†	113 ± 19	3.2 ± 0.7†

* Indicates significant difference ($P < 0.05$) between values at ZEEP and PEEP 10 (ANOVA).

† Significant difference between values at ZEEP and PEEP 16–20.

pulmonary artery catheterization. Arrythmias did not occur during the study. After study, the LV catheter was withdrawn and the femoral artery compressed for 15 min. No femoral hematoma or infection occurred.

STATISTICS

A linear regression analysis was used for comparison between PAOP and LVEDP. Analysis of variance, followed by Tukey's test when appropriate, was used for comparisons of hemodynamic values between successive levels of PEEP. Values of $P < 0.05$ were considered statistically significant.

Results

Individual values of hemodynamic measurements at ZEEP, 10, and 20 cm H_2O PEEP are provided in table 2, in which pressure data are given as intravascular values. The mean BP and CI decreased only at the highest PEEP level (16–20 cm H_2O). However, in four patients (patients 1, 2, 3, and 8), the CI at high PEEP was equal to or higher
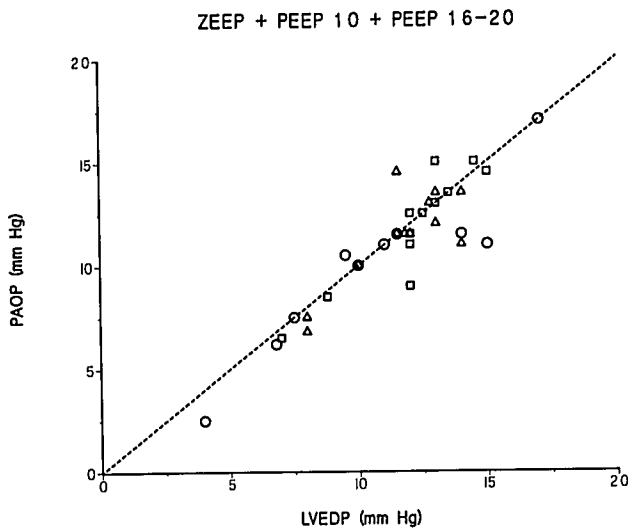
FIG. 1. Illustrations of simultaneous PAOP and LVEDP values for all data points, measured at zero end-expiratory pressure (ZEEP, circles), 10 cm H_2O PEEP (triangles), and 16–20 cm H_2O PEEP (squares). The dashed line of identity is provided. The regression coefficients at each level of end-expiratory pressure are 0.93 (ZEEP), 0.81 (10 cm H_2O PEEP), and 0.91 (16–20 cm H_2O PEEP).

than the value recorded at ZEEP. Mean RAP increased significantly at each level of PEEP, while mean PAOP did not vary significantly (from 10 ± 3.7 mmHg at ZEEP to 11.9 ± 2.7 at PEEP 16–20 cm H_2O). The simultaneously recorded values of mean PAOP and LVEDP usually agreed within 1 or 2 mmHg, and there was a close correlation at all levels of PEEP between PAOP at end-expiration and LVEDP measured at the pre-ejection diastolic plateau (fig. 1); PAOP and LVEDP differed by 2

mmHg or more in six of 35 measurements (2–3 mmHg in four, 4 mmHg in two). These six discrepancies were observed at varying levels of PEEP in several patients; in four of these, LVEDP was higher than PAOP and, in only one, PAOP exceeded LVEDP by 2 mmHg at the highest level of PEEP (patient 3). Simultaneous traces of PAOP and LV pressure at 20 cm H_2O PEEP in patient 9 are presented in figure 2, illustrating the PAOP and LVEDP values, even at peak mechanical inspiration.

In most instances, the tip of the Swan-Ganz catheter was below the left atrial border. The mean vertical distance between the tip of the catheter and the left atrial border, estimated on the lateral chest radiograph, was 0.42 ± 1.05 cm (range −1 to 2 cm). This value was added to the LVEDP at the highest PEEP level in each patient to estimate the pulmonary venous pressure (PVP) at the catheter tip level (table 3). The PEEP value was higher than this estimated PVP value in eight of 12 measurements; in two of these eight, the PEEP-PVP difference was 8.5 and 9 cm H_2O; in three, it was 4 cm H_2O; and in the remaining three, the difference was 1–2.5 cm H_2O. Although PEEP was higher than the estimated PVP in these cases, a close relationship between PAOP and LVEDP was maintained.

Discussion

Our results suggest that, in patients with severe ARDS, mechanical ventilation with PEEP does not impair the near identity of PAOP and LVEDP even when PEEP of 16–20 cm H_2O is applied. These findings contrast with the clinically held view that in patients following cardiac surgery[5] and patients with ARDS,[6] PAOP no longer re-
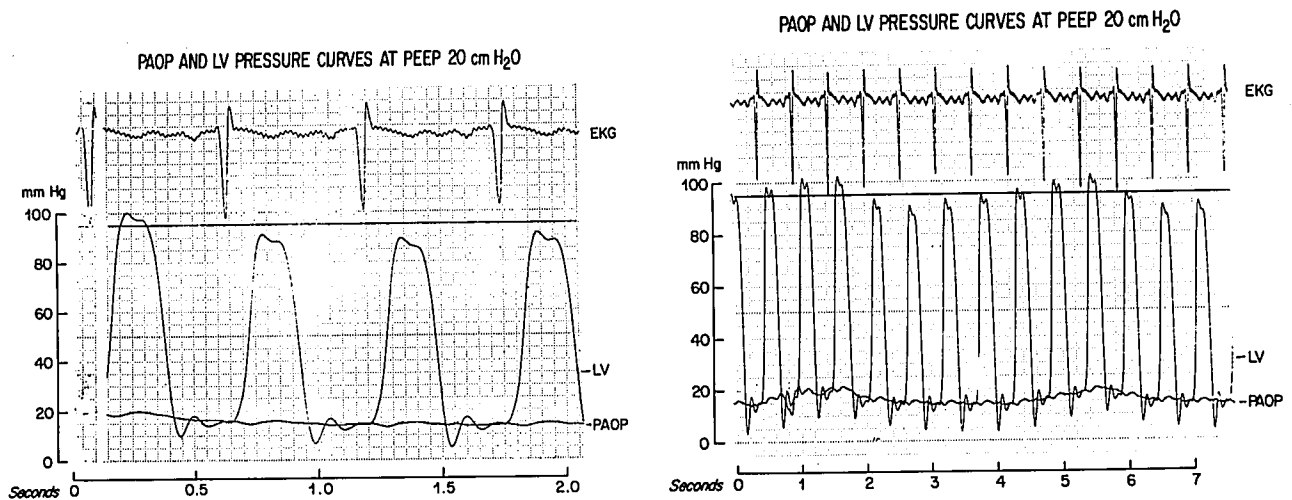

FIG. 2. *A.* A high speed recording of PAOP, LV pressure, and EKG. The close correspondence of LVEDP and PAOP is shown. *B.* A slow speed recording of PAOP, LV pressure, and EKG in another patient. Note the correspondence of LVEDP and PAOP, which is maintained even during the periods of peak mechanical lung inflation.

flects LVEDP at a PEEP level over 10 cm H_2O. Lozman et al. noted a good correlation between PAOP and left atrial pressure (LAP) at low levels of PEEP in five patients after cardiac surgery.[5] However, at 10 and 15 cm H_2O PEEP, a discrepancy was reported between PAOP and LAP in four of 14 measurements, and no statistical correlation was found between PAOP and LAP. Jardin et al. studying patients with ARDS, noted that PAOP closely reflected LVEDP at 10 cm H_2O or less PEEP.[6] At PEEP levels of 15–30 cm H_2O, PAOP was higher than LVEDP. In both of these clinical studies, the catheter location, shunt fraction, and lung compliance were not specified.

A close relationship of PAOP with LAP depends upon the presence of patent vascular channels between the PA wedge catheter and the left atrium. According to West's concept, the catheter reflects PVP only when PVP is higher than alveolar pressure (zone III); the catheter reflects alveolar pressure and not PVP after balloon occlusion in areas where alveolar pressure exceeds both PAP and PVP (zone I) or only PVP (zone II), because alveolar capillaries collapse when extravascular pressure exceeds intraluminal pressure. Therefore, high levels of PEEP, by raising Palv, are expected to produce zone I or II conditions, at least in the normal lung. Cardiac catherization studies in patients without ARDS,[15] or animals with normal lungs or experimental acute lung injury,[1-4,16,17] have emphasized the clinical relevance of this model and stressed the importance of the position of the Swan-Ganz catheter tip with respect to the left atrium to ensure that PAOP will closely reflect LVEDP. When the catheter tip is in a dependant position, PVP is increased at the catheter tip level, and this maintains zone III conditions even if relatively high levels of PEEP are applied. In patients without ARDS, Shasby et al.[15] found a significant discrepancy between PAOP and LAP at PEEP over 5 cm H_2O when the pulmonary artery catheter tip was less than 1 cm below the left atrium and LAP less than 3 mmHg, while near identity was noted with LAP over 5 mmHg at the same position of the PA catheter tip. Therefore, a combination of a low LV filling pressure, and a catheter tip at or higher than the LAP level was able to produce zone I or II conditions in patients with normal lungs, even at relatively low levels of PEEP.

In all of our twelve patients with severe ARDS, PAOP did not differ appreciably from LVEDP. Table 3 shows that the distance between the catheter tip and the left atrium never exceeded 2 cm. Thus, at high PEEP levels, the catheter tip should have been in zone I or II, provided the PVP was lower than the PEEP level. Accordingly, when the hydrostatic pressure gradient between the catheter tip and the left atrium was added to the LVEDP to estimate PVP at the level of the catheter tip (table 3), the highest level of PEEP was notably higher (4–9 cm H_2O) than this hydrostatically adjusted value of LVEDP in five

TABLE 3. Comparison of the Highest PEEP Level Used in Each Patient with the Estimated Pulmonary Venous Pressure (PVP) at the Catheter Tip Level

Patient No.	Highest PEEP Level (cm H_2O)	LVEDP (cm H_2O)	Hydrostatic Gradient (cm)	Estimated PVP* (cm H_2O)	PEEP—Estimated PVP (cm H_2O)
1	20	20.5	2	22.5	-2.5
2	20	16.5	-0.5	16	4
3	20	17.5	1.5	19	1
4	16	17	-0.5	16.5	-0.5
5	20	16.5	-0.5	16	4
6	20	9.5	2	11.5	8.5
7	20	20	1	21	-1
8	20	16.5	1	17.5	2.5
9	20	18.5	0	18.5	1.5
10	18	17.5	0.5	18	0
11	20	16.5	-0.5	16	4
12	20	12	-1	11	9

* The estimated PVP was calculated by adding the hydrostatic gradient to the LVEDP. The hydrostatic gradient was the vertical distance between left atrium and the catheter tip on the lateral decubitus chest radiograph.

of our 12 patients. Therefore, in these patients, the alveolar pressure/PVP ratio was above unity at end-expiration and zone II conditions should have occurred. However, both in these ARDS patients and in the seven others, the measured PAOP and LVEDP were very close, suggesting that West's concept did not apply to our patients with severe ARDS.

Thus, in patients with ARDS, several factors other than the catheter tip position may provide patent vascular channels between the PA wedge catheter and the left atrium despite an alveolar pressure higher than PVP. First, a large intrapulmonary anatomic shunt may provide numerous channels that are not exposed to alveolar pressure. Inert gas elimination studies during ARDS show that the majority of venous admixture is composed of true shunt suggesting gas exchange vessels are shielded from alveolar pressure;[18] second, there may be protection and reinforcement of lung microvessels by interstitial edema, cell infiltration, and remodelling of the vascular wall by smooth muscle cells.[11,19] Thus, the compliance of the lung vessels to hydrostatic compression may be reduced; third, the low thoracopulmonary compliance found in the majority of our 12 patients could decrease the effects of high alveolar pressure on the pulmonary microvasculature. Accordingly, Berryhill and Benumof, in an experimental study, noted that the PAOP-LAP discrepancy with PEEP and the pulmonary artery catheter tip above the left atrium, decreased after reducing lung compliance by oleic acid injection.[4]

The possible protective effect from transmission of high airway pressure to pulmonary microvasculature provided by lung pathology in ARDS would be enhanced if the catheter tip is lodged in a severely injured lung region.

Computed axial tomographic studies show that heterogenous lung injury occurs in patients with ARDS, with most of the opacities usually noted in dependent regions of the lung.[20,21] Since pulmonary artery catheters are usually lodged in dependent regions,[22] vascular protection from high alveolar pressure, whether due to a large shunt, reduced microvascular compliance, or decreased lung compliance, should be enhanced in these areas.

In summary, our data show that in patients with ARDS, PAOP measurements are not influenced by alveolar pressure and adequately reflect LVEDP, even at PEEP levels up to 20 cm H_2O. The anatomic location of the pulmonary artery catheter tip, measured radiographically in each of our patients to be vertically at or below the level of the left atrium, partly accounts for this close correlation between PAOP and LVEDP. Two other factors, specific to patients with ARDS, probably contribute to maintaining this correlation: 1) a large intrapulmonary right-to-left shunt may provide a number of microvessels shielded from alveolar pressure and freely communicating from pulmonary artery to pulmonary vein, and 2) in ARDS, both the vascular and lung compliance may decrease, reducing the transmission of alveolar pressure to the pulmonary microvasculature. Although our data show that PAOP measurements are reliable during PEEP, it is emphasized that the clinical importance of PAOP as reflecting left ventricular filling pressure should be thought of in terms of a distending transmural pressure instead of an absolute intravascular pressure.

References

1. Roy RJ, Powers SR, Feustel PJ, Dutton RE: Pulmonary wedge catheterization during positive end-expiratory pressure ventilation in the dog. ANESTHESIOLOGY 46:385–390, 1977

2. Tooker J, Huseby J, Butler J: The effects of Swan-Ganz catheter height on the wedge pressure-left atrial pressure relationship in edema during positive-pressure ventilation. Am Rev Respir Dis 117:721–725, 1978

3. Cassidy SS, Robertson CM, Pierce AK, Johnson RL: Cardiovascular effects of positive end-expiratory pressure in dogs. J Appl Physiol 44:743–750, 1978

4. Berryhill RE, Benumof JL: PEEP-induced discrepancy between pulmonary arterial wedge pressure and left atrial pressure: The effects of controlled *vs.* spontaneous ventilation and compliant *vs.* noncompliant lungs in the dog. ANESTHESIOLOGY 51:303–308, 1979

5. Lozman J, Powers SR, Older T, Dutton RE, Roy RJ, English M, Marco D, Eckert C: Correlation of pulmonary wedge and left atrial pressure: A study in the patient receiving positive end-expiratory pressure ventilation. Arch Surg 109:270–277, 1974

6. Jardin F, Farcot JC, Boisante L, Curien N, Margairaz A, Bourdarias JP: Influence of positive end-expiratory pressure on left ventricular performance. N Engl J Med 304:387–390, 1981

7. O'Quin R, Marini JJ: Pulmonary artery occlusion pressure: Clinical physiology, measurement, and interpretation. Am Rev Respir Dis 128:319–328, 1983

8. Raper R, Sibbald WJ: Misled by the wedge? The Swan-Ganz catheter and left ventricular preload. Chest 89:427–434, 1986

9. Davison R, Parker M, Harrison RA: The validity of determinations of pulmonary wedge pressure during mechanical ventilation. Chest 73:352–355, 1978

10. Matthay MA: Invasive hemodynamic monitoring in critically ill patients. Clin Chest Med 4:233–249, 1983

11. Zapol WM, Snider MT: Pulmonary hypertension in severe acute respiratory failure. N Engl J Med 296:476–480, 1977

12. Hurst JW, Logue RB, Schlant RC, Wenger NR: The Heart, Arteries, and Veins, 4th edition. New York, McGraw-Hill, 1978, p 487

13. Matamis D, Lemaire F, Harf A, Brun-Buisson C, Ansquer JC, Atlan G: Total respiratory pressure-volume curves in the adult respiratory distress syndrome. Chest 86:58–66, 1984

14. Fraser RG, Paré JAP (eds): An integrated study based on the abnormal roentgenogram, Diagnosis of Diseases of the Chest. Philadelphia, WB Saunders, 1970, p 97

15. Shasby DM, Dauber IM, Pfister S, Anderson JT, Carson SB, Manart F, Hyers TM: Swan-Ganz catheter location and left atrial pressure determine the accuracy of the wedge pressure when positive end-expiratory pressure is used. Chest 80:666–670, 1980

16. Hasan FM, Malanga AL, Braman SS, Corrao WM, Most AS: Lateral position improves wedge-left atrial pressure correlation during positive-pressure ventilation. Crit Care Med 12:960–964, 1984

17. Hotchkiss RS, Katsamouris AN, Lappas DG, Mihelakos PT, Wilson RS, Long M, Coyle J, Brewster P, Greene R: Interpretation of pulmonary artery wedge pressure and pullback blood gas determinations during positive end-expiratory pressure ventilation and after exclusion of the bronchial circulation in the dog. Am Rev Respir Dis 133:1019–1023, 1986

18. Dantzker DR, Brook CJ, de Hart P, Lynch JP, Weg JG: Ventilation-perfusion distributions in the adult respiratory distress syndrome. Am Rev Respir Dis 120:1039–1052, 1979

19. Tomashefski JF, Davies P, Boggis C, Greene R, Zapol WM, Reud LM: The pulmonary vascular lesions of the adult respiratory distress syndrome. Am J Pathol 112:112–126, 1983

20. Maunder RJ, Shuman WP, McHugh JW, Marglin SI, Butler J: Preservation of normal lung regions in the adult respiratory distress syndrome. Analysis by computed tomography. JAMA 55:2463–2465, 1986

21. Gattinoni L, Mascheroni D, Torresin R, Marcolin R, Fumagalli R, Vesconi S, Rossi GP, Rossi F, Baglioni S, Bassi F, Nastri G, Pesenti A: Morphological response to positive end-expiratory pressure in acute respiratory failure. Computerized tomography study. Intensive Care Med 12:137–142, 1986

22. Teboul JL, Besbes M, Axler O, Brun-Buisson C, Andrivet P, Rekik N, Lemaire F. A bedside index for determination of zone III conditions of pulmonary artery (PA) catheter tips during mechanical ventilation (abstract). Am Rev Respir Dis 137:139, 1988