

Bedside Measurement of Pulmonary Capillary Pressure in Patients with Acute Respiratory Failure

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In this report, the authors present the results of 34 estimates of pulmonary capillary pressure (Pcap) in 15 adult patients receiving intensive care for acute respiratory failure (ARF). Within the pulmonary artery pressure profile during transient balloon occlusion, the authors identified two exponential decay components—the slower one representing the discharge of the pulmonary capillary pressure through the pulmonary venous resistance. By extrapolating this exponential to its origin at the moment of pulmonary artery occlusion, a pressure within the pulmonary vascular bed which approximates pulmonary capillary pressure (Pcap) was identified. Pcap, and not the pulmonary artery occlusion pressure (PAOP), is the major driving pressure forcing fluid from the pulmonary microvasculature. The results indicate that a discrete value for pulmonary capillary pressure can be reproducibly measured in paralyzed ventilated patients. The data report that mean pulmonary artery pressure, pulmonary capillary pressure, and total pulmonary vascular resistance (PVR) are increased in acute respiratory failure, but there is considerable variation in the distribution of pulmonary vascular resistance between the arterial and venous beds. The data suggest that there is unequal and variable partitioning of the increased PVR during acute respiratory failure. Bedside pressure profile Pcap measurements will allow optimum reduction of Pcap during ARF by infusing vasoactive agents to modify the distribution of PVR or reducing the PAOP. (Key words: Complications: Adult Respiratory Distress syndrome. Lungs: pulmonary artery wedge pressure; pulmonary capillary pressure; pulmonary hypertension. Measurement techniques: pulmonary artery catheter.)

THERE IS CONSIDERABLE clinical interest in measuring pulmonary microvascular filtration pressure in patients with acute respiratory failure. Pulmonary capillary pressure (Pcap) influences the rate of edema formation in the injured lung, and an understanding of how Pcap is altered by vasoactive drug infusions or variations of wedge pressure is important to improve our treatment of acute respiratory failure. Practical methods of measuring Pcap, however, have been reported only in laboratory animals.¹ Holloway *et al.* reported a method of estimating Pcap in intact canine lungs by analyzing the pulmonary artery pressure trace during occlusion.² They demonstrated a close correlation between Pcap estimated with this method, and both the isogravimetric measurement³ and

Gaar's mathematical estimate of pulmonary microvascular pressure (Pgaar): $P_{gaar} = PAOP + 0.4 (MPAP - PAOP)$.⁴ We applied the method of Holloway *et al.*² to determine pulmonary capillary pressure in 15 patients with acute respiratory failure undergoing intensive care. We identified a pressure (Pcap) within the pulmonary vascular bed which can be repeatedly and reproducibly measured, thereby enabling estimation of mean capillary filtration pressure in the critically ill patient.

Methods

A continuous plot of the pulmonary artery pressure was recorded for 10 seconds following inflation of the balloon of a pulmonary artery balloon-flotation catheter in each of 15 adult patients receiving intensive care for acute respiratory failure. Swan-Ganz balloon occlusion catheters manufactured by the American Edwards Laboratories (Model 93A-831H-7.5F) had been inserted in these patients for routine clinical management.

The distal port of each catheter was connected to a disposable Gould (T4812AD) pressure transducer complete with continuous irrigation device, the mid-axillary line serving as the zero reference point in supine patients. The resultant electric signal was transmitted to a Hewlett Packard HP8805B preamplifier and the pressure trace was recorded on a two-channel recorder (Hewlett-Packard 7702B) with a simultaneous electrocardiogram. Conversion of the electrical pressure trace to a digital format was performed by a DEC LSI-11/23 with an analog to digital converter sampling at a rate of 200 points per second, and the data were recorded onto floppy disks.

The shortest exponential time constant we measured in the pressure decay from Pcap to PAOP was 0.77 s, thereby requiring accurate reproduction of pressure wave forms up to a frequency of only 2 Hz to ensure high fidelity tracings. In 14 studies, the performance of our pressure monitoring system was tested by the "pop off" technique described by Gardner,⁵ demonstrating a resonant frequency on the order of 7-10 Hz (mean 8.8 ± 1.9 Hz) with a damping coefficient range of 0.2-0.4 (mean 0.33 ± 0.07). The catheter-transducer system therefore provided a low fidelity, under-damped system for reproduction of the pulmonary artery waveform,^{5,6} and the rates of exponential pressure decay following pulmonary arterial occlusion were undistorted.

All patients included in the study were undergoing mechanical ventilation, and were paralyzed and sedated

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TABLE 1. Mean Data from Patients Grouped According to ARF Severity*

ARF Severity	Number of Studies	Age (Yr)	MPAP	PAOP	Pcap	Pgar	F _{io₂}	PEEP	Q _s /Q _t , %	C.O. (l/min)	PVR (mm Hg/l/min)	HR	Ven. R./PVR
Normal lungs	7	24	19 ± 1.3	12 ± 1.3	/	15	0.21	0	/	6.2	0.7	67 ± 4	/
At Risk	4	75 ± 5	32 ± 7	16 ± 4	23 ± 7	22 ± 5	0.36 ± 0.05	6 ± 3	20 ± 12	5.9 ± 1.8	2.2 ± 0.9	98 ± 12	0.41 ± 0.13
Mild	6	73 ± 7	38 ± 12	17 ± 7	25 ± 4	25 ± 9	0.51 ± 0.11	9 ± 4	24 ± 9	5.9 ± 2.0	3.3 ± 2.1	94 ± 14	0.43 ± 0.20
Moderate	8	47 ± 24	48 ± 15	24 ± 11	32 ± 7	34 ± 12	0.61 ± 0.13	15 ± 4	29 ± 14	5.9 ± 2.4	3.7 ± 1.9	91 ± 22	0.47 ± 0.14
Severe	6	40 ± 21	49 ± 10	27 ± 7	33 ± 1	36 ± 8	0.63 ± 0.12	19 ± 7	28 ± 9	6.9 ± 3.0	2.7 ± 1.2	93 ± 8	0.42 ± 0.08

Mean values and standard deviations are presented with patients grouped according to ARF severity. Normal values are from Naieji *et*

*al.*¹⁰ All pressures are in cm H₂O.

* ARF severity classified using the criteria in Reference 8.

without spontaneous respiratory effort, thus permitting a 15-s interruption of positive pressure ventilation during data collection. This maneuver minimized artifact caused by respiratory variations of intra-thoracic pressure.⁷ To ensure relative hemodynamic stability during the collection of the data, no study was performed within 30 min of either a change in ventilator setting or chest physiotherapy. All traces were obtained at end-expiration with positive end-expiratory pressure (PEEP) maintained and the patient supine. PEEP varied from 5–25 cm H₂O and, in all but three studies, the measured PAOP was greater than the level of PEEP at which the study was performed. Several of the patients were receiving intravenous infusions of vasoactive agents. Details of respiratory and cardiovascular function are summarized in table 1, together with a gradation of the severity of their acute respiratory failure based upon gas exchange and requirements for mechanical ventilation⁸ at the time of the study.

ANALYSIS OF PRESSURE TRACES

The moment of balloon occlusion of the pulmonary artery was identified by superimposing an unwedged PAP trace upon the wedged trace. Occlusion was assumed to have occurred when the wedged pressure deviated sharply below the unwedged tracing, as indicated in figure 1. There were occasional difficulties in accurately identifying the moment of pulmonary artery occlusion due to slight non-uniformity of pulmonary artery wave forms. The PA catheter balloon took approximately 0.2 s to inflate fully, and fluoroscopy demonstrated significant movement of the catheter as it floated into an occlusive position. There was often a period of 0.15–0.25 s during which the wedging pressure trace was distorted by artifacts. Nevertheless, a moment of occlusion was clearly identified in about 70% of the traces collected. In the discarded 30% of traces, exponentials were observed by pressure-recording artefacts caused by patient movement, movements of the catheter during balloon inflation, or other catheter whip. The mean PAOP was subtracted from the values of PAP as it decreased following balloon inflation, and an exponential fit was plotted (fig. 1). To accomplish this, the

values of PAP-PAOP lying between the inflection point of the PA trace where the rapid first exponential meets the slower second exponential, and the time when PAP first equals PAOP, were plotted on a semi-logarithmic scale: Log₁₀(PAP-PAOP) versus time. The best straight line was fitted to these data by a least squares regression analysis.⁹ In all cases, a good fit was observed when the exponential was superimposed upon the wedging trace of PAP.

Pulmonary capillary pressure (Pcap) was identified as the origin of the pressure decay exponential at the moment of balloon occlusion of the pulmonary artery. This analysis was performed for each of the traces, and a mean value of Pcap was calculated. Others have employed a graphic technique for visually determining the inflection point between the rapid and slow exponential components of the pulmonary arterial pressure trace after the balloon is inflated.² We did not compare the visual technique with

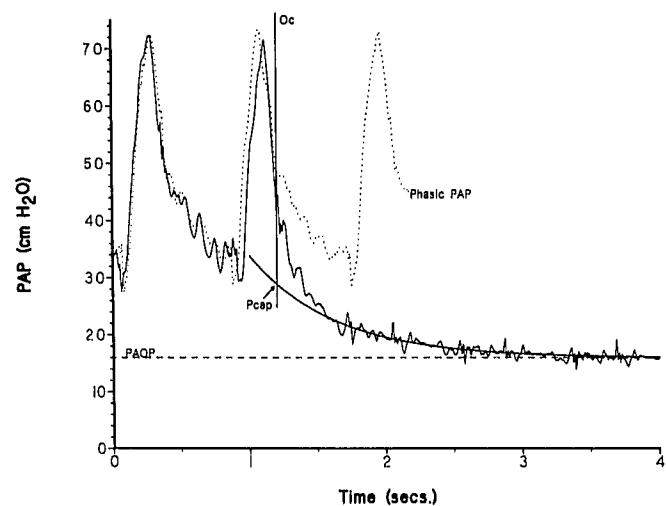


FIG. 1. The phasic pulmonary artery pressure trace (dotted line) is superimposed upon the pulmonary artery pressure trace during pulmonary artery occlusion (solid line). The time of pulmonary artery occlusion can then be identified (Oc) when the two traces sharply diverge. Pcap is estimated as the pressure at which the exponential approximation to the occluded trace (see text) intersects the vertical line drawn at the moment of occlusion (Oc).

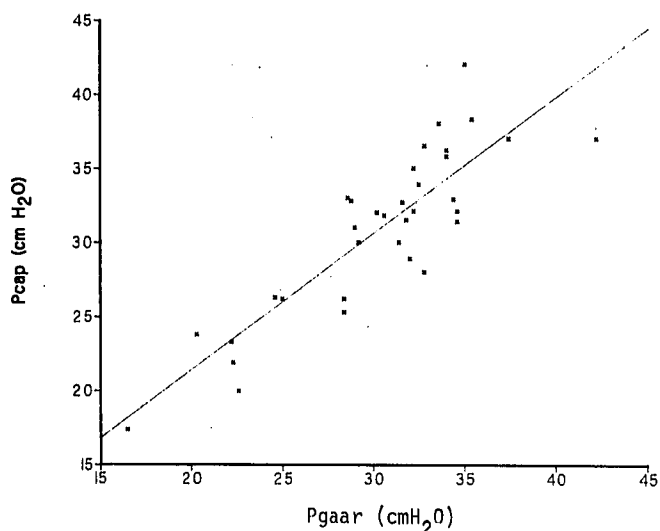


FIG. 2. Mean values of P_{cap} are compared with Gaar's estimates⁴ of pulmonary capillary pressure (P_{gaar}): $P_{gaar} = PAOP + 0.4$ (MPAP-PAOP). A linear regression analysis of the data yields the equation:

$$y = 3.0 + 0.92x; r = 0.87 (n = 34).$$

the computer determined P_{cap} ; thus, we cannot compare the methods.

Concomitant measurements of pulse rate, systemic blood pressure, central venous pressure, and cardiac output (the mean of three determinations by thermodilution) were made, and the pulmonary vascular resistance and stroke volume were calculated. PVR was calculated by dividing PAP-PAOP by the cardiac output. The chest radiograph was graded according to the degree and distribution of radio-opacity,⁸ and the following indices of respiratory function were recorded: Fi_{O_2} , arterial and mixed venous blood gas tensions, venous admixture, level of positive end-expiratory pressure, and the quasi-static total pulmonary compliance.

Results

In 15 patients, we obtained 34 estimations of mean pulmonary capillary pressure by averaging a total of 169 traces of pulmonary artery pressure during occlusion with a balloon flotation catheter. An analysis of variance⁹ performed on a random sample of 20 studies yielded a mean error of the residual mean square equal to 4.4 cm H₂O, thereby indicating an overall reproducibility of our P_{cap} measurements of ± 2.1 cm H₂O. In table 1, the mean P_{cap} is reported along with the mean pulmonary artery pressure, pulmonary artery occlusion pressure, and the microvascular pressure calculated with Gaar's equation.⁴

Average values from serial studies of any patient at any single severity grade are included in the calculation of group means. The normal values are taken from a study of 23 healthy volunteers conducted by Naeije *et al.*¹⁰

To compare estimates of P_{cap} by occlusion pressure trace analysis with those obtained by using Gaar's equation, P_{cap} is plotted against P_{gar} (fig. 2). A least squares regression analysis of these data yields the equation:

$$P_{cap} = 3.0 + (0.92 \times P_{gaar}); r = 0.87$$

There is a small but important variation about the regression line. This becomes apparent if the distribution of pulmonary vascular resistance between arterial and venous beds is expressed quantitatively. The pressure drop across the venous bed (P_{cap} -PAOP) can be expressed as a fraction of the total pressure drop across the pulmonary vascular tree (MPAP-PAOP). Thus, if pulmonary arterial resistance exceeds the venous component, the ratio of P_{cap} -PAOP/MPAP-PAOP will fall, since P_{cap} will approach PAOP. Conversely, if venous resistance predominates the ratio of P_{cap} -PAOP/MPAP-PAOP approaches unity, since P_{cap} will be much larger than PAOP, and nearly equal to MPAP. When our results are expressed in this manner, a wide range of values from 0.2-0.7 reflect the venous fraction of pulmonary vascular resistance. There is no consistent relationship between the distribution of resistance and severity of ARF or the PVR (table 1, figs. 3, 4).

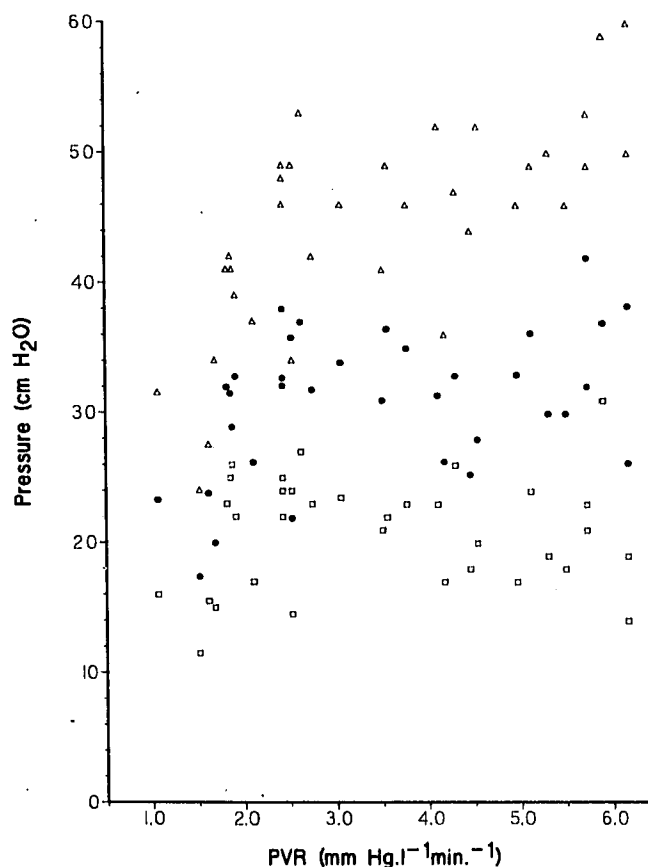


FIG. 3. MPAP (Δ), P_{cap} (\bullet), and PAOP (\square) graphed against total pulmonary vascular resistance.

During ARF, the PVR rises and becomes markedly elevated with severe lung injury.¹¹ In figure 3, MPAP, Pcap, and PAOP are plotted against PVR. The variability of Pcap with increasing MPAP suggests that, in the population studied, changes in the distribution of pulmonary vascular resistance during acute lung injury appear largely independent of the absolute value of pulmonary vascular resistance. However, for PVR values > 2.0 cm H₂O · m/l, we observe that the Pcap does not appear to rise with increasing PVR, while, as expected, the PAP does increase. This suggests that the elevated arteriolar resistance may partially shield the perfused pulmonary microvasculature from the elevated pulmonary artery pressure. In six patients, sequential studies of Pcap were performed before and after the clinically indicated administration of intravenous vasopressor infusions. The results of one of these vasopressor studies is presented in figure 5. In this patient, L-norepinephrine was continuously infused into the pulmonary artery catheter. Despite increasing the L-norepinephrine dose from 1 to 5 to 10 μg/min, there was no alteration of the distribution of resistance between the arterial and venous beds. Later reductions of the L-norepinephrine infusion to 5 and 1 μg/min did not alter this distribution. Cardiac output remained constant throughout the 3-h period of study at 7.3 L/min. Student's *t* test confirmed that values of MPAP, Pcap, and PAOP at 1 μg/min (n = 7) were significantly different (*P* < 0.001). However, the distribution of PVR was not significantly altered by L-norepinephrine infusion at the

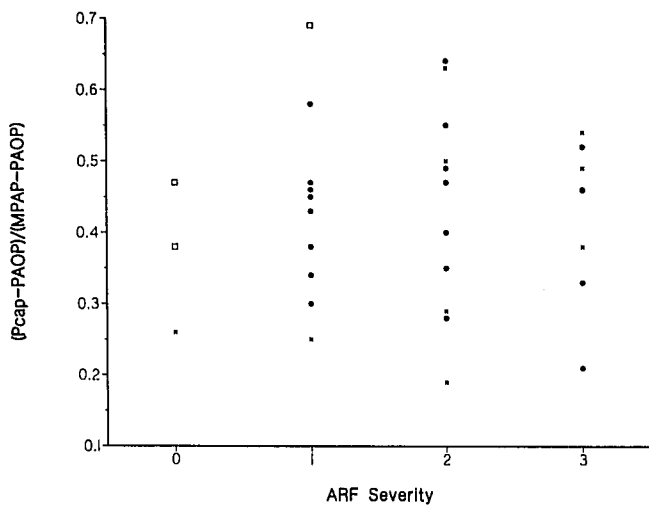


FIG. 4. The ratio of pulmonary venous resistance to total pulmonary vascular resistance (Pcap-PAOP)/MPAP-PAOP is plotted against the severity of acute respiratory failure classified according to Reference 8. 0 = at risk; 1 = mild; 2 = moderate; 3 = severe. Patients are reported along with a key to the intravenous vasoactive agent infusions they were receiving at the time of study: X = none or dopamine (5 μg/kg · min) alone; ● = norepinephrine/epinephrine/phenylephrine; □ = sodium nitroprusside/glyceryl trinitrate.

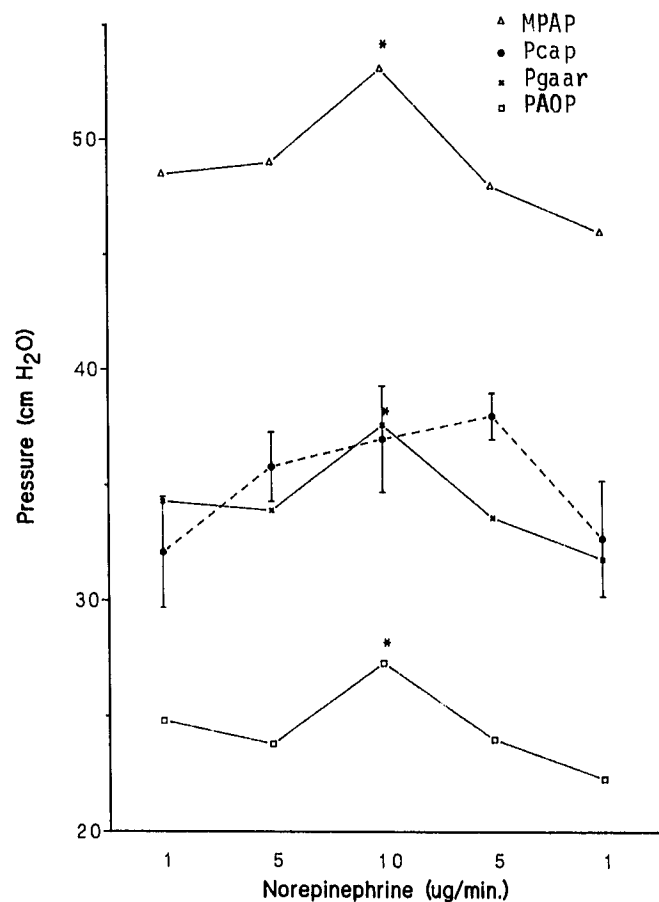


FIG. 5. Mean values of MPAP (Δ), Pcap (●), Pgaar (×), and PAOP (□) in patient 15 during a therapeutic trial of L-norepinephrine infusion via the PA catheter. These studies were performed over a period of 3 h. Cardiac output remained constant throughout the study at 7.3 l/min. Student's *t* test confirmed that values of MPAP, Pcap, and PAOP at 1 μg/min (n = 11) and 10 μg/min (n = 7) were significantly different (*) (*P* < 0.001). The distribution of PVR was not significantly altered by norepinephrine infusion at the higher dose (*P* = 0.59). The patient received an intravenous infusion of dopamine (6.5 μg/kg · min) throughout this study.

higher dose (*P* = 0.59). This patient was also receiving an intravenous infusion of dopamine (6.5 μg/kg/min) throughout this study.

In one patient with mild diffuse acute lung injury, traces were collected before and immediately after removing 15 cm H₂O PEEP. As previously reported,¹¹ during severe ARF there was minimal change in the measured MPAP and PAOP upon discontinuing PEEP. The wedging pressure profile of this patient with PAOP 23 cm H₂O was unaltered (fig. 6) despite removing 15 cm H₂O PEEP.

Discussion

Clinical studies by Zapol and Snider¹¹ reported mild pulmonary hypertension without a markedly increased

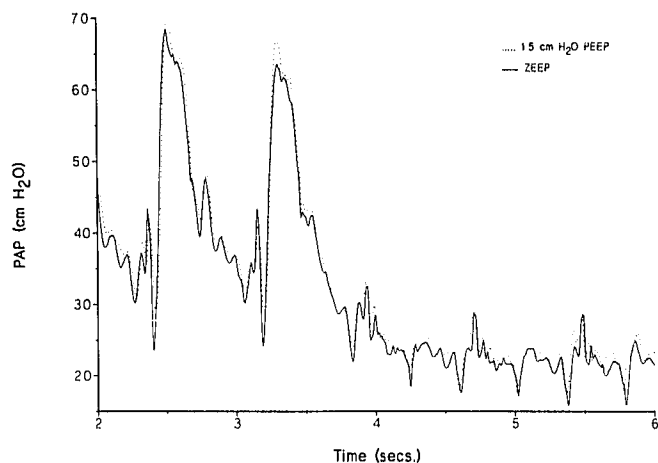


FIG. 6. Tracings of pulmonary artery pressure in patient 22 with mild ARF during PA occlusion with a Swan-Ganz catheter. The traces were collected during end-expiration at 15 cm H₂O PEEP (dotted line) and zero PEEP (ZEEP-solid line). Note that the PAOP was higher than the 15 cm H₂O PEEP level.

PAOP to be a common hallmark of moderate and severe adult respiratory distress syndrome (ARDS), regardless of the etiology of the syndrome. Whether this occurs as a primary or secondary event remains uncertain; however, it is clear that an elevated PVR is intimately related to the pathogenesis of ARDS. The distribution of the PVR in ARDS between arterial and venous beds is uncertain.

The theoretical basis of a technique of P_{cap} estimation is described by Hakim *et al.*,^{13,14} Lineham *et al.*,^{15,16} and Dawson *et al.*,^{1,17} who propose a simple resistance-capacitance model of the pulmonary vascular bed, noting that the majority of the normal pulmonary vascular compliance lies in the capillary bed, and that the pulmonary vascular resistance is distributed on either side of this, as displayed in the electrical analog representation (fig. 7A). A biexponential drop of pulmonary artery pressure following sudden pulmonary artery occlusion has been shown. The slower exponential, which decays to the pulmonary artery occlusion pressure (PAOP), represents the pulmonary capillary bed pressure discharging through the pulmonary venous resistance. P_{cap} can, therefore, be estimated by extrapolating this second exponential back to its original pressure at the moment of pulmonary artery occlusion (fig. 7B).

Dawson modified this model to include a significant resistance within the microvascular compartment, pointing out that the estimated P_{cap} represents an average pressure for a microvascular segment lying between arterial and venous beds.¹⁷ He also subdivided the vascular compliance into two components on either side of the capillary resistance. Successful application of the model depends upon the exponential pressure drop from pulmonary artery pressure (PAP) to P_{cap} having a time con-

stant considerably smaller than that of the exponential from P_{cap} to PAOP; otherwise, the two exponentials do not join (fig. 7B). The rates of decline of these exponentials are governed by the resistance of the pulmonary arterial and venous beds (R_a and R_v, respectively) and by the capacitance of the pulmonary arterial and capillary beds (C_a and C_c, respectively).

Conceivably, with the marked disturbances of pulmonary vascular architecture which occur in ARDS,¹² the resistance-capacitance model (fig. 7A) which appears to accurately mimic the normal canine lung might no longer directly apply. If the value of the product of arterial resistance and capacitance (R_aC_a) approaches the value of venous resistance multiplied by capillary capacitance

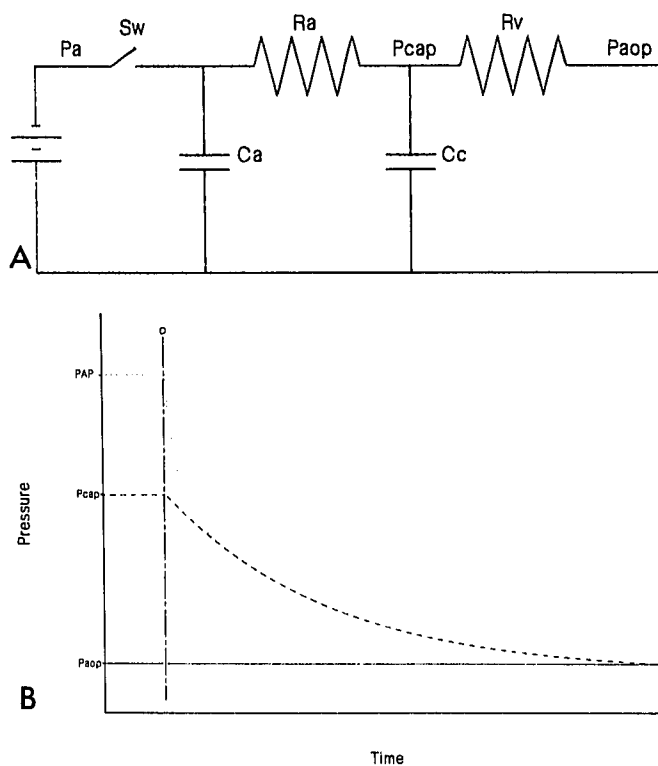


FIG. 7. A. The pulmonary vascular bed is represented by a simplified electric analog model. Pa, P_{cap}, and PAOP are the pulmonary artery, pulmonary capillary, and pulmonary artery occlusion pressures respectively. Resistance to flow exists on the upstream (arterial) side and the downstream (venous) side as indicated R_a and R_v. Most of the vascular capacitance lies in the pulmonary capillary bed (C_c), with the arterial bed capacitance (C_a) being significantly smaller. Occlusion of the pulmonary artery is equivalent to opening the switch (Sw). B. Estimating the pulmonary capillary pressure (P_{cap}) from the pressure decay curve following pulmonary artery occlusion. On occlusion of the pulmonary artery (O), pulmonary artery pressure (PAP) falls rapidly to pulmonary capillary pressure (P_{cap}). The subsequent decay of pressure occurs more slowly as the capillary bed capacitor (C_c in A) discharges through the venous resistance. Extrapolation of this exponential drop of pressure back to the moment of occlusion (O) yields the pulmonary capillary pressure (P_{cap}).

(RvCc), then the pulmonary artery pressure decay curve following pulmonary artery occlusion will cease to be biexponential. In such circumstances, the Pcap cannot be estimated using pulmonary artery pressure decay curve analysis. Our results suggest that the pulmonary vasculature does continue to fit the resistance-capacitance model of figure 7A, even during severe acute lung injury. In our estimations of Pcap during ARF, two exponential components of the decay of pulmonary artery pressure from the moment of PA balloon occlusion to PAOP have been identified. Extrapolation of the second (slower) exponential back to its original pressure at the moment of balloon occlusion yields a reproducible value which is a close approximation to the pulmonary capillary filtration pressure.

Gaar's equation assumes a static distribution of pulmonary vascular resistance, the arterial bed accounting for 60% and the venous bed for 40% of the total resistance.⁴ We found a varying distribution of PVR between the arterial and venous beds in patients with acute lung injury (fig. 4). This variation is consistent with the work of Cope *et al.*,¹⁸ who recently reported significant variations in the distribution of pulmonary vascular resistance in patients during cardiothoracic surgery. During acute lung injury, the distribution of pulmonary vascular resistance between the arterial and venous beds (which determines the pulmonary microvascular pressure) may be significantly altered by a wide variety of stimuli. Important factors include physical determinants, such as lung volume or alveolar hypoxia, humoral mediators of pulmonary vascular tone, and neural reflexes, although many of these systems have yet to be fully characterized. There is, therefore, good reason to expect the ratio of precapillary to postcapillary resistance to vary markedly from the normal in pulmonary disease states. It is probably these factors that contribute to the variability of the distribution of pulmonary vascular resistance we measured during acute lung injury (fig. 4). The small number of patients we studied serially during ARF does not allow general conclusions about the causes or sequence of the variation in the distribution of pulmonary vascular resistance. Correlating the data from all 15 patients, we found no relationship between the ratio of Pcap-PAOP/MPAP-PAOP and the severity of lung injury (table 1, fig. 4) or the levels of quasi-static pulmonary compliance, PEEP, venous admixture, PaO₂, or density grading of the chest radiograph. However, by analyzing all our patient data for PVR values above 2.0 cm H₂O · m/l, we observed that the Pcap does not appear to rise with increasing PVR, while, as expected, the PAP does increase with mounting PVR. This suggests that vascular resistance is partitioned to a greater extent in the precapillary vasculature at higher values of PVR.

We found that ARF patients with an acutely elevated pulmonary artery pressure have an increased pulmonary

capillary pressure (table 1). Note that the arterial resistance would need to be increased to two or three times the venous resistance to shield the Pcap from an elevated PAP. A simple adaptation of Gaar's equation⁴ will predict the pulmonary capillary pressure in such circumstances, for example: 1) Elevated PAP (MPAP = 60 cm H₂O, PAOP = 15 cm H₂O) without any change of the distribution of PVR (*i.e.*, Ra = 1.5 Rv):

$$P_{cap} = 15 + 0.4(60-15) = 33 \text{ cm H}_2\text{O}$$

2) Elevated PAP (MPAP = 60 cm H₂O, PAOP = 15 cm H₂O) with an increased arterial component of PVR such that Ra = 2 Rv:

$$P_{cap} = 15 + 0.33(60-15) = 30 \text{ cm H}_2\text{O}$$

3) Elevated PAP (MPAP = 60 cm H₂O, PAOP = 15 cm H₂O) with an increased arterial component of PVR such that Ra = 3 Rv:

$$P_{cap} = 15 + 0.25(60-15) = 26 \text{ cm H}_2\text{O}.$$

In the last example of an increased arteriolar resistance, the Pcap would be reduced by 7 cm H₂O when compared with the normal arterial:venous resistance ratio. With the overall reproducibility of Pcap estimates at ± 2.1 cm H₂O, such resistance changes should be detected with this clinical measurement technique. In the study by Cope *et al.* of eight patients during cardiothoracic surgery,¹⁸ preoperative values of the ratio of pulmonary arterial to pulmonary venous resistance were near 1.5–2.0. We found a wider variation of the ratio of pulmonary artery to venous resistance ratio during ARDS, our values (fig. 4) correspond to arterial venous resistance ratios of the order of 0.8 to 3.0, evidencing a marked variation of the distribution of PVR during acute lung injury.

The mean values of PAOP were high in moderate (24 cm H₂O) and severe ARDS (27 cm H₂O) (table 1). These elevated values probably reflect concomitant left ventricular failure documented in several of the patients by scintiscanning. In these patients, acute inflammatory respiratory failure was aggravated by the high PAOP.

Several problems with the technique of Pcap estimation remain to be resolved. The region of the pulmonary vascular tree (1–3 segments) sampled by the catheter can vary, and these segments may not represent the average pathology within the lung. The PAOP may not equal the left atrial pressure in mild ARF or moderate ARDS when PAOP is lower than the PEEP level. The interruption of ventilation necessary during pulmonary artery pressure trace collection may cause circulatory transients, possibly with a degree of desaturation which could affect the pulmonary vascular tone. However, we never observed cyanosis or tachycardia during the 15-s period of arrested ventilation. In patients with mild lung disease subjected to high inflation pressures, the pulmonary capillary bed

may not drain freely into the pulmonary veins, since the catheter may be lodged in a zone 1 or 2 region.¹⁹ Reassuringly, the PAOP has been reported to closely approximate left ventricular end-diastolic pressure in severe ARDS despite applying high levels of PEEP.¹¹ In 34 of the 37 reported measurements, the PAOP level was greater than the level of PEEP; thus, we can assume PAOP assessed the pulmonary venous pressure and not alveolar pressure; that is, we were in zone 3. This is convincingly demonstrated by figure 6.

Unfortunately, one cannot measure Pcap directly in either closed chest experimental animals or humans. Iso-gravimetric techniques, venous occlusion, and capillary micropuncture laboratory studies support the validity of the method we have employed, but cannot be performed clinically to provide an *in vivo* calibration of our study.

Measuring Pcap will elucidate the factors influencing the distribution of pulmonary vascular resistance during ARF, and will allow clinicians to learn if vasoactive agents infused in the management of acute respiratory failure can reduce the microvascular filtration pressure. In this manner, clinicians may directly reduce the rate of edema formation in the acutely injured lung.

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