Avoidance of Aggravated Hypoxemia During Measurement of Mean Pulmonary Artery Wedge Pressure in ARDS*

Matthew B. Divertie, M.D., F.C.C.P.;† John C. McMichan, M.B.B.S., F.C.C.P.;‡ Luc Michel, M.D., F.C.C.P.;§ Kenneth P. Offord, M.S.;‖ and Anders B. Ness ¶

A marked drop occurs in mixed venous oxygen saturation during temporary interruption of mechanical ventilation. To avoid this potentially dangerous problem and eliminate possible errors induced by associated hemodynamic changes, a simple electronic circuit was constructed for measurement of mean pulmonary artery wedge pressure without separation of the patient from the ventilator. Its 12-second time constant was sufficient to cover two to four respiratory cycles. In 50 ventilator-supported patients with the adult respiratory distress syndrome, it was shown that the value obtained from the circuit was not different from the instantaneous value obtained at the end of expiration during temporary cessation of mechanical ventilation, but was different from the pulmonary artery diastolic pressure. The circuit value was not affected by positive end-expiratory pressures up to 10 cm H_2O. The method is sufficiently accurate for its intended purpose, improves the care of these severely-ill patients, and can be safely used without a physician being present.

Changes in intra-alveolar pressure due to mechanical ventilation are transmitted to the pulmonary circulation and are reflected in measurements obtained through a pulmonary artery catheter. Since these augmented respiratory cycles produce variable and biased interpretation of changes in pulmonary artery pressure, it has become a common practice to temporarily discontinue mechanical ventilation during the period in which the latter is recorded. This practice is undesirable because the values obtained are not representative of what is taking place during ventilatory support. It is also dangerous since temporary cessation of mechanical ventilation, especially in association with positive end-expiratory pressure,[1] produces a rapid onset of significant hypoxemia in an already compromised patient. Mixed venous oxygen saturation also falls, reflecting an increase in tissue oxygen extraction, a fall in cardiac output, or both (Fig 1). Frequently, these procedures must be carried out when a physician is not present.

The pulmonary artery wedge pressure, represented as an arithmetic mean, can be obtained by planimetry of the inscribed pressure pulse, or more conveniently and directly, from an electronic averaging circuit. Several such circuits are available commercially, but they have the disadvantage of using a short time constant which often results in processing the pressure

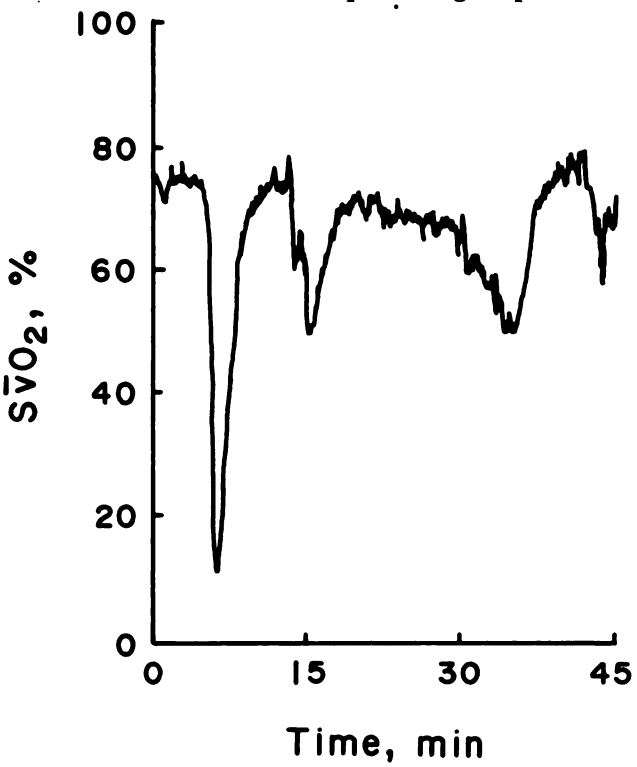


FIGURE 1. Marked reduction in mixed venous oxygen saturation ($S\bar{v}O_2$) during and following 15 to 30 second separation from mechanical ventilation (MV) in ARDS. The first drop in $S\bar{v}O_2$ occurred when PAWP was measured and the other two when the tracheobronchial tree was suctioned. The duration of each fall substantially exceeded the cessation of MV, and preceded reduction in the level of arterial oxygen.

*From the Division of Thoracic Diseases, Department of Internal Medicine; Critical Care/Respiratory Section, Department of Anesthesiology; Department of Medical Statistics and Epidemiology; and Section of Engineering, Mayo Clinic and Mayo Foundation, Rochester, MN.
†Professor of Medicine.
‡Assistant Professor of Anesthesiology.
§Staff Surgeon, Unité de Chirurgie, Cliniques Universitaires de Mont-Godinne, Yvoir, Belgium.
‖Consultant, Medical Research Statistics.
¶Section of Engineering.
Manuscript received June 21; revision accepted June 30
Reprint requests: Dr. Divertie, Mayo Clinic, Rochester, Minnesota 55901

signal over less than one respiratory cycle during mechanical ventilation. Because of the value of such a circuit in patient care, a simple system was devised with the ability to measure mean pressure during several respiratory cycles. The accuracy of the results was examined, as well as the influence of positive end-expiratory pressure and the agreement between wedge pressure and pulmonary artery diastolic pressure during mechanical ventilation. The purpose of this paper is to consider whether the system is sufficiently accurate to justify its advantages in patient care.

Methods

The 50 patients studied had severe acute respiratory failure due to nonhydrostatic pulmonary edema of the adult respiratory distress syndrome (ARDS). In each instance, there was an alveolar-arterial oxygen tension difference ($P[A\text{-}a]O_2$) greater than 300 mm Hg at an $FIO_2 = 1.0$, with or without an arterial carbon dioxide tension ($PaCO_2$) greater than 50 mm Hg and a pH less than 7.35. All required mechanical ventilation using a volume-limited ventilator with supplemental oxygen at an FIO_2 greater than 0.50 to maintain an arterial oxygen tension (PaO_2) greater than 60 mm Hg. Positive end-expiratory pressure (PEEP) did not exceed 25 cm H_2O. The majority had reduced static compliance of the respiratory system at some time in their clinical course, measured from expired tidal volume and peak airway pressure at a point of zero airflow during supported ventilation.

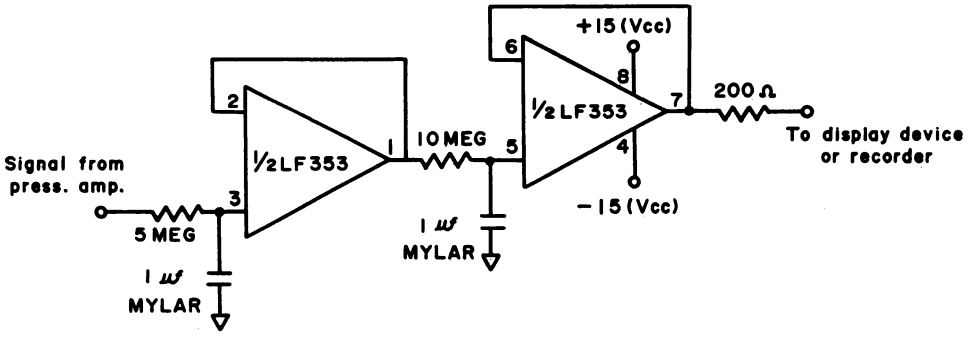
Hemodynamic monitoring was carried out using a balloon-tipped flow-directed pulmonary artery catheter. In 16 patients reported elsewhere,[2] two fiberoptic channels were incorporated in the catheter (Opticath), permitting continuous photometric measurement of mixed venous oxygen saturation ($S\bar{v}O_2$). Each catheter was advanced by a pressure-monitored technique and the position of its tip in a dependent branch of the pulmonary artery was confirmed by chest roentgenography. Calibration of the monitoring equipment was performed before each set of measurements was made using a static system with a mercury manometer. Electronically damped vascular and airway pressures obtained from pressure transducers and an ECG tracing were inscribed on suitable paper by a multichannel recorder. Mean vascular pressures were obtained through an averaging circuit and numbers were read from a digital display. With 15 second disconnection of mechanical ventilation and oxygen supply, the electronic smoothing of the filter system was excluded, and pressures were measured from the graphic representation. The phases of the respiratory cycle were identified from the airway pressure tracing and the P wave of the ECG coming immediately before inspiration was arbitrarily chosen to define the end-point of expiration. The vascular pressure at the end of expiration which followed temporary balloon occlusion of a branch of the pulmonary artery will be referred to as the instantaneous pulmonary artery wedge pressure ($PAWP_{INST}$). This was identified by its configuration on a simultaneous recording of the pressure pulse.

The pressure monitoring system used in our intensive care units involves amplification of venous pressure measurements. The dynamic response of the catheter-strain gauge transducer system is not as significant in the measurement of pressure pulses in the pulmonary circulation as it is with the high pressures of the systemic arteries. The electronic circuit for averaging pulmonary artery wedge pressure consisted of two electronic filters with buffers connected in series (Fig 2). Its 12-second time constant allowed mean pressures to be obtained over two to four respiratory cycles. The circuit was placed between the strain gauge amplifier and a visual display unit or recorder. The occlusion pressure thus recorded will be designated the electronic mean pulmonary artery wedge pressure ($PAWP_{EM}$). When the mean pressure was not required, the circuit was excluded by a switch. In this way, no more than 1 to 2 seconds separated the 12-second recording strips from which comparisons were made. No measurement was made of intra-esophageal or intrapleural pressure since calculation of ventricular compliance has not been found to have significant influence on clinical management of ARDS.

In 17 of the 50 patients, over an identical time period, $PAWP_{INST}$ was compared to $PAWP_{EM}$ during mechanical ventilation without PEEP from records obtained using a four-channel recorder with a suitable response time. In addition, $PAWP_{INST}$ and pulmonary artery diastolic pressure (PADP) were compared at the end point of expiration during mechanical ventilation.

The remaining 33 patients were treated with different values of positive end-expiratory pressure (PEEP) during mechanical ventilation. For these patients, $PAWP_{EM}$ was compared to the $PAWP_{EM}$ value obtained during a 15-second cessation of mechanical ventilation and PEEP. Eleven patients were treated with only one value of PEEP.

The data were statistically evaluated using linear regression, rank sum tests, and paired *t* tests. Although ideally the analysis of the relationship between $PAWP_{EM}$ and PEEP should be done using the within patient differences between different PEEP levels, only a few patients had measurements at more than two levels of PEEP. Hence, two sample comparisons were made. Where more than one set of measurements were obtained on a patient under the same conditions, the averages was used. The methods employed in this study were in accord with the standards of the Mayo Clinic Human Studies Committee.



LF353 DUAL F.E.T. OP. AMP. – NATIONAL SEMICONDUCTOR

FIGURE 2. Diagram of electronic circuit for measuring mean pressure.

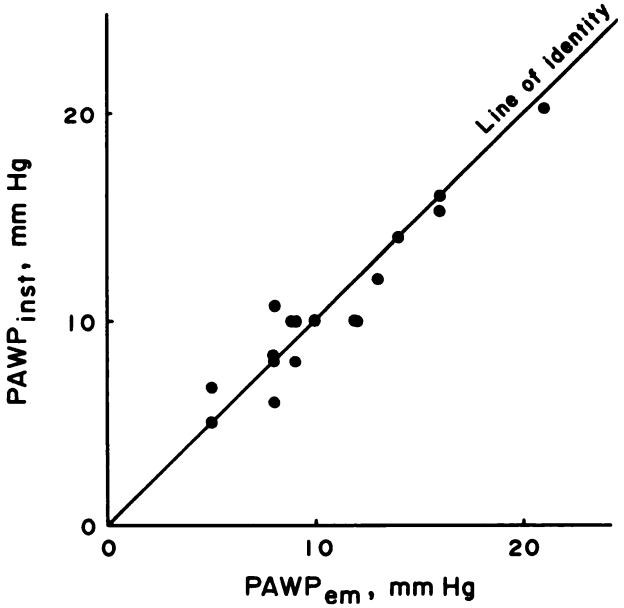


FIGURE 3. Comparison of values of electronically-obtained mean pulmonary wedge pressure ($PAWP_{EM}$) during mechanical ventilation with those obtained from pressure tracings made at the end of expiration ($PAWP_{INST}$).

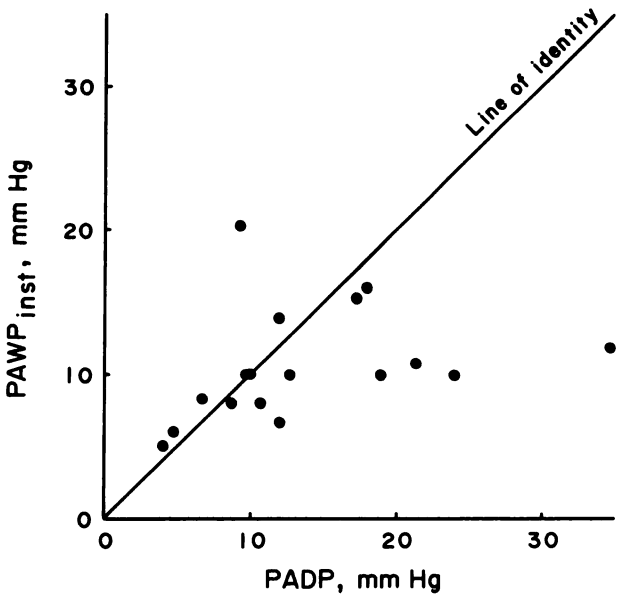


FIGURE 4. Comparison of values of pulmonary wedge pressure ($PAWP_{INST}$) with pulmonary artery diastolic pressure (PADP), both obtained from pressure tracings made at the end of expiration.

RESULTS

The system for reflection spectrophotometry has proved accurate and dependable.[2] There was a prompt, severe fall in $S\bar{v}O_2$ which persisted considerably beyond the period of separation from mechanical ventilation for measurement of PAWP (Fig 1). These results in 16 ventilator-dependent patients have been reported previously elsewhere.[2]

For the 17 patients receiving mechanical ventilation without PEEP, the $PAWP_{EM}$ was compared with the coincident $PAWP_{INST}$. Triplicate measurements were made. For $PAWP_{INST}$, the estimate of the standard deviation among the three replicates pooled across the 17 subjects was ±0.52 mm Hg. The difference between the highest and lowest replicate ranged from 0 to 2 mm Hg. Triplicate determinations on a given patient were then averaged. The mean (±SD) $PAWP_{EM}$ was 10.8 (±4.2), ranging from 5 to 21 mm Hg. For $PAWP_{INST}$, the mean was 10.6 (±3.9), ranging from 5 to 20 mm Hg. The mean paired differences 0.2 (±1.3) were not significantly different from zero as assessed by the paired *t* test. The paired differences ranged from −2.7 to +2.0 (Fig 3).

Coincident measurement of $PAWP_{INST}$ and PADP were obtained from 17 patients during mechanical ventilation without PEEP. For PADP, the standard deviation among the three replicates, pooled across the 17 subjects, was ±1.07 mm Hg. The range in the difference between the highest and lowest replicate was 0 to 6 mm Hg. Using the averages for each patient, the mean of the paired differences between $PAWP_{INST}$ and PADP was not found to differ significantly from zero by paired *t* test. The means were 10.6 (±3.9), 13.8 (±7.8), and −3.2 (±7.5) for $PAWP_{INST}$, PADP, and the paired differences ($PAWP_{INST}$–PADP), respectively. Ranges for these three variables were, respectively, 5 to 20, 4 to 35, and −23 to +11 mm Hg. Although the mean difference was not significantly different from zero, there was marked variability in the differences (SD = ±7.5 mm Hg). Furthermore, the linear correlation between $PAWP_{INST}$ and PADP of 0.32 was not significantly different from zero (Fig 4).

Figure 5 depicts the changes in 33 patients in the measurement of $PAWP_{EM}$ produced by different levels of PEEP. The variable of interest is the difference between the value under PEEP with mechanical ventilation and the value obtained during a 15-second cessation of both. When the end-expiratory pressure was zero, removal of mechanical ventilation for 15 seconds produced a small but consistent change in $PAWP_{EM}$ in most patients. Only slight variations in $PAWP_{EM}$ were seen between PEEP of 0 and 10 cm H_2O. For PEEP greater than 10 cm H_2O, the resultant change in $PAWP_{EM}$ was significantly ($p = 0.003$) greater when compared to a PEEP of zero. The five patients with PEEP greater than 10 cm H_2O had resultant changes in $PAWP_{EM}$ of 5, 6, 7, 8, and 12 mm Hg and PEEP of 15, 15, 12.5, 12.5, and 20 cm H_2O, respectively.

DISCUSSION

Proper management of ARDS requires the use of hemodynamic monitoring to ensure that adequate left ventricular function is maintained during support of the patient by mechanical ventilation. Left ventricular pre-load is reflected in the pulmonary artery wedge pressure. This is conventionally obtained during tem-

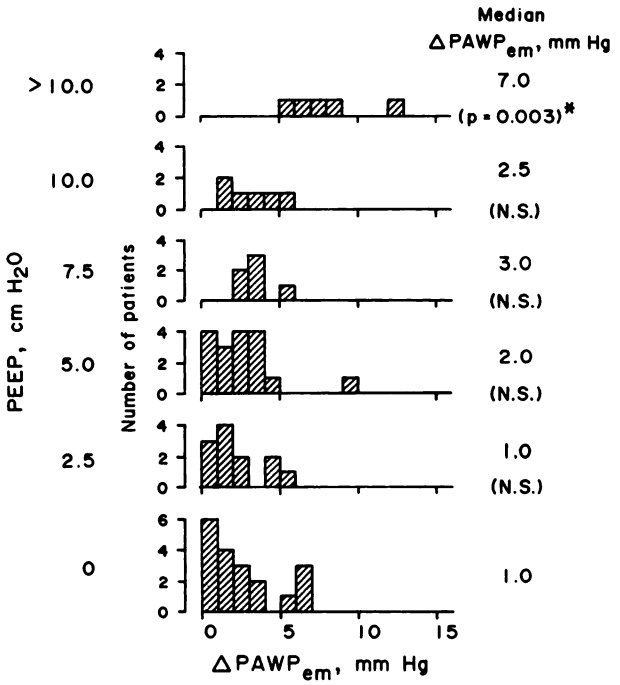


FIGURE 5. Effect of positive end-expiratory pressure (PEEP) on electronically determined mean pulmonary artery wedge pressure ($PAWP_{EM}$) during mechanical ventilation for ARDS. The effect becomes significant above 10 cm H_2O PEEP. $\Delta PAWP_{EM}$ is the difference obtained at various levels of PEEP between $PAWP_{EM}$ and zero obtained during 15 seconds of cessation of mechanical ventilation and PEEP. NS is not significant, and asterisk indicated rank sum test compared to PEEP = 0.

porary occlusion by the inflated balloon tip of a flow-directed pulmonary artery catheter. Separation from mechanical ventilation is commonly used during measurement to exclude ventilator-induced artifact and variation in the pulmonary artery and wedge pressures. This procedure is usually entrusted to allied health personnel and may occupy 15 to 30 seconds until a satisfactory trace is obtained. In spite of the fact that this is routinely done, it is contrary to good medical practice to remove profoundly hypoxemic and unstable patients from high levels of FIO_2 and PEEP, and have them spontaneously breathe room air or oxygen. Continuous monitoring of $S\bar{v}O_2$ has shown the rapidity and severity with which deterioration can develop in these circumstances[2] (Fig 1). In our experience and that of others, its occurrence often precedes other evidence of hemodynamic instability.[2,3] For these reasons, a system was developed in which cessation of mechanical ventilation is unnecessary.

Electronic analysis of hemodynamic pressure pulses can be accomplished in a brief time interval because of the relatively high heart rate. Thus, a three-second time constant will provide three or more pulses for analysis. The high pressure peripheral arterial circulation is little affected by respiratory movements. In contrast, the low intravascular pressures of the pulmonary circulation are significantly altered by the changes in intrathoracic pressure which accompany breathing. This is also the case in the venae cavae and atria. Pressure in these channels is affected by changes in positive airway and intrathoracic pressures at a frequency determined by spontaneous or mechanical ventilatory rates. The use of equipment with a time constant suitable for peripheral pressure pulse survey clearly will not answer the problem presented by slower intrathoracic pressure variations superimposed on pressure pulse movements. Therefore, it was decided that at least two full respiratory cycles must be incorporated in any analysis of pulmonary artery pressures, specifically for determination of mean pulmonary artery wedge pressure. Consequently, a 12-second time constant was utilized in a simple circuit, ensuring that the appropriate survey time would be achieved for at least 12 pressure pulses and two respiratory cycles. A longer period was not considered desirable because of management problems inherent in movement of the patient or catheter tip, and the artifacts that would result.

During spontaneous breathing, left ventricular end-diastolic pressure is reflected directly in left atrial pressure and indirectly in the pulmonary artery wedge pressure.[4] While mechanical ventilation has some influence on these values,[5-8] the changes produced do not appear to seriously influence these relationships in the presence of severe acute respiratory failure[8] or when PEEP is used at less than 10 cm H_2O.[9] In another study at similar low levels of PEEP, the mean pulmonary artery wedge pressure was unaffected by temporary interference with mechanical ventilation, but spontaneous respiratory efforts had been suppressed by the use of sedatives and muscle relaxants.[10] This is only infrequently necessary when supported ventilation is required and did not arise in any of the patients in the present series. During the brief time of measurement of $PAWP_{EM}$, $PAWP_{INST}$, and PADP in the present study, significant changes in ventricular compliance would not be expected, and consequently, no effort was made to plot ventricular function curves. In addition to a constant hemodynamic state, the extent and distribution of pulmonary parenchymal disease and the position of the catheter tip did not change.

This study demonstrates 17 patients with ARDS where there is an accurate relationship in these circumstances between the values obtained for the mean pulmonary artery wedge pressure using a simple electronic averaging circuit and those instantaneous values obtained at the end point of expiration. This point in the respiratory cycle was chosen for comparison because changes in airway pressure during mechanical ventilation are at a minimum at that time. It was concluded, therefore, that a circuit of this type with a time constant of sufficient length to cover several respiratory cycles could be used to obtain an accurate value of the mean pulmonary artery wedge pressure

during mechanical ventilation.

The effect of PEEP on pulmonary vascular pressures was also demonstrated in 33 patients in this study. In all of these, discontinuance of PEEP resulted in a fall in $PAWP_{EM}$. The extent of this decrease was not marked until PEEP exceeded 10 cm H_2O. The alteration in $PAWP_{EM}$ above 10 cm H_2O was significantly greater than at a PEEP of 0 cm H_2O. Beyond 10 cm H_2O, $PAWP_{EM}$ should not be used as an indicator of left ventricular function in patients with this condition. This level is consistent with the results of a recent study investigating the effects of PEEP on left ventricular function.[11] Consequently, high levels of PEEP should be temporarily lowered to not more than 10 cm H_2O for electronic measurement of the mean wedge pressure as an alternative to the more hazardous practice of temporarily disconnecting the patient from the ventilator. These observations warrant further studies at higher levels of PEEP combined with direct measurement of left atrial pressure and also in patients in whom compliance is not reduced.

In the absence of pulmonary vascular disease and pulmonary arterial hypertension, the PADP measured at the end of expiration and $PAWP_{INST}$ bear a close relationship to each other, and the difference between them is normally less than 5 mm Hg. In these patients with respiratory failure, a significant linear correlation between these variables was not detected, while the mean of the paired differences was not significantly different from zero. The paired differences displayed large variability. The standard deviation of these paired differences was ± 7.5 mm Hg. It must be concluded that PADP cannot be accurately substituted for PAWP in these circumstances. This is increasingly important as the possibility of hydrostatic pulmonary edema becomes clinically significant. Whether regional changes in vasoconstriction are responsible for this discrepancy cannot be determined from these data. The necessity for this substitution is most commonly due to rupture of the balloon at the tip of the catheter preventing an occlusion or wedge pressure from being obtained or failure of the balloon to occlude a branch of the pulmonary artery. Data indicate that in this form of severe respiratory failure it is essential to obtain a proper pulmonary artery wedge pressure for accurate interpretation of left ventricular function.

The mortality rate from ARDS is high, but in the recovery phase, a pulmonary artery catheter is almost always removed before mechanical ventilation is discontinued. The present system is accurate during full support by a mechanical ventilator in the control or assist-control mode. Possible inaccuracies during intermittent mandatory or demand ventilation, or with spontaneous breathing are not relevant since the principal reason for its use is the profound hypoxemia of the acute phase. The method is inexpensive and within the limitations described, it is safe and accurate, and can be used without direct supervision by a physician.

ACKNOWLEDGMENT: The authors would like to acknowledge with gratitude the constructive criticism of Dr. Kai Rehder and the secretarial help of Carol Mickow.

REFERENCES

1 Rose DM, Downs JB, Heenen TJ. Temporal response of functional residual capacity and oxygen tension to changes in positive end-expiratory pressure. Crit Care Med 1981; 9:79-82
2 Baele PL, McMichan JC, Marsh HM, Sill JC, Southorn PA. Continuous monitoring of mixed venous oxygen saturation in critically ill patients. Anesth Analg 1982; 6:513-17
3 Krauss XH, Verdouw PD, Hugenholtz PG, Nanta J. On-line monitoring of mixed venous oxygen saturation after cardiothoracic surgery. Thorax 1975; 30:636-43
4 Humphrey CB, Oury JH, Virgilio RW, Gibbons JA, Folkerth TL, Shapiro AR, et al. An analysis of direct and indirect measurements of left atrial filling pressure. J Thorac Cardiovasc Surg 1976; 71:643-47
5 Qvist J, Pontoppidan H, Wilson RS, Lowenstein E, Laver MB. Hemodynamic responses to mechanical ventilation with PEEP: the effect of hypervolemia. Anesthesiology 1975; 42:45-55
6 Roy RJ, Powers SR Jr, Dutton RE. Pulmonary wedge catheterization during positive end-expiratory pressure (PEEP) ventilation, abstracted. Physiologist 1974; 17:323
7 Rosenbaum RW, Hayes MF, Morello DC, Matsumoto T. The importance of pulmonary artery pressure monitoring. Surg Gynecol Obstet 1973; 136:261-64
8 Zapol WN, Snider MT. Pulmonary hypertension in severe acute respiratory failure. N Engl J Med 1977; 296:476-80
9 Lozman J, Powers SR, Older T, Dutton R, Roy RJ, English M, et al. Correlation of pulmonary wedge and left atrial pressures. Arch Surg 1974; 109:270-76
10 Davison R, Parker M, Harrison RA. The validity of determinations of pulmonary wedge pressure during mechanical ventilation. Chest 1978; 73:352-55
11 Jardin F, Farcot J-C, Boisante L, Curren N, Margairaz A, Bourdarias JP. Influence of positive end-expiratory pressure on left ventricular performance. N Engl J Med 1981; 304:387-92