Volume 46, No. 6
June 1977

THE JOURNAL OF Anesthesiology
THE AMERICAN SOCIETY OF ANESTHESIOLOGISTS, INC.

Editorial Views

Anesthesiology
46:383–384, 1977

Interpretation of Pulmonary-artery Wedge Pressure When PEEP is Used

IN THE EIGHT YEARS that have elapsed since positive end-expiratory pressure (PEEP) was introduced for therapy of acute respiratory failure, articles concerning the clinical importance of hemodynamic responses to PEEP have appeared in this and other journals. When it became apparent that levels of PEEP exceeding 15 cm H_2O were sometimes effective in decreasing pulmonary venous admixture, the hemodynamic consequences became even more critical. One of these consequences is that PEEP alters pulmonary vascular pressures, and there is presently a controversy about how the measured pressure values should be interpreted or corrected. We now recognize that there are difficulties in interpretation of the measured pulmonary-artery wedge pressure (PAWP) for assessment of left ventricular function whenever PEEP is used. PEEP adversely affects accuracy of measurement of PAWP, as performed with a balloon-tipped, pulmonary-artery (Swan-Ganz) catheter, in two ways. First, PEEP alters intrapleural pressure. Second, pressure generated by PEEP may be transmitted to pulmonary microvasculature. In either case, pressures measured with a Swan-Ganz catheter may be altered by PEEP.

When intrapleural pressure is increased, PAWP measured with reference to atmospheric pressure will not accurately assess the pressure gradient across the left atrial wall, because the pressure on the outer wall of the atrium is not atmospheric. Left atrial pressure, which in the absence of mitral stenosis accurately reflects left ventricular filling pressure, is determined by the difference between intrapleural pressure and intra-atrial pressure. To overcome this source of error, various maneuvers are used. Each maneuver introduces its own error into PAWP measurement. When PEEP is briefly discontinued so that its effect on intrathoracic pressure is removed, hemodynamic status may be markedly changed due to the resulting sudden increase in right-heart venous return, pulmonary congestion, and hypoxemia. In this instance, measurement of PAWP may not accurately reflect the condition of the left ventricle before PEEP was discontinued. Formulas derived from measurements of PEEP *vs.* change in PAWP in critically ill patients have been used to extrapolate the effect of PEEP on PAWP. Since lung compliance, which may affect transmission of PEEP to the intrapleural space,[1] will also vary from patient to patient, this type of extrapolation must be viewed with caution. When high levels of PEEP are used, it seems reasonable to measure intrapleural pressure at left atrial level using an esophageal balloon or intrapleural catheter. The difficulties that may be encountered with either of these techniques must be balanced against the value of the information obtained.

The second adverse effect of PEEP on measurement of pulmonary vascular pressures involves interaction of pulmonary alveolar pressure with pulmonary microvasculature.[2] The paper appearing in this issue, by Roy *et al.*, which describes how PEEP may spuriously alter measurement of PAWP, focuses on this interaction. It is well established that airway pressure is transmitted to collapsible portions of the pulmonary microvasculature, creating a "Starling resistor" or "vascular waterfall" effect if certain conditions are met. Such conditions include a pulmonary alveolar pressure that exceeds left atrial or pulmonary venous pressure. When this happens, resistance to pulmonary blood flow becomes dependent upon airway pressure transmitted to pulmonary alveoli. Increasing

alveolar pressure enhances the likelihood of a "Starling resistor" effect, particularly in less dependent portions of lung where venous pressure is lower. When flow is suddenly stopped by occlusion of a pulmonary artery supplying a segment of lung where alveolar pressure exceeds pulmonary venous pressure, collapse of the blood vessels will occur at some point distally. When a Swan-Ganz catheter is wedged in such a lung segment, it will measure alveolar pressure rather than pulmonary venous pressure. Roy *et al.* demonstrate that this phenomenon does occur.

The significance of the observations of Roy *et al.* in healthy dogs is less clear for patients who have abnormal lungs. The mechanical properties of lungs that determine how airway pressures are transmitted to pulmonary vasculature are not yet well quantitated. These properties are altered by factors including pulmonary blood and water content, lung volume, and lung compliance, all of which may change in abnormal lungs. Therefore, the observations must be confirmed in abnormal lungs before their significance can be fully appreciated.

When PEEP is being used, one is left with the feeling that, after all precautionary maneuvers are taken to insure accuracy of measurement of PAWP, such measurements must be viewed with caution, and the clinician will continue to include other indices of circulatory adequacy in making decisions.

It is our current practice to recognize that when PAWP and central venous pressure measurements are unreliable in the presence of PEEP, one must depend on trends and changes in other clinical measurements in response to therapy. No single measurement will suffice. The responses of arterial pressure, central venous pressure, PAWP, partial pressure of oxygen in venous blood, central venous oxygen saturation, central venous oxygen content, urinary output, and peripheral circulation after rapid, timed infusion of a measured increment of colloid or crystalloid will help in assessment of cardiac function and intravascular volume status. Of particular value is the measurement of changes in cardiac output after a challenge with intravascular volume.

After intravascular volume administration, rapid improvement in some of the above measurements without large changes in PAWP or central venous pressure suggests the presence of intravascular volume deficit. Conversely, elevation of central venous pressure and PAWP without improvement in the other measurements implies either heart failure or excessive intravascular volume.

RALPH T. GEER, M.D.
Assistant Professor
Department of Anesthesia
Director of Respiratory Care
Surgical Intensive Care Unit
University of Pennsylvania
Philadelphia, Pennsylvania 19104

References

1. Macklem PT, Murphy B: The forces applied to the lung in health and disease. Am J Med 57:371, 1974
2. Permutt S, Bromberger-Barnea B, Bane HN: Alveolar pressure, pulmonary venous pressure and the vascular waterfall. Med Thorac 19:239, 1962