Intrathoracic Pressure, Pulmonary Vascular Pressures and Gas Exchange during Pulmonary Lavage

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Intrathoracic pressure, blood pressures of intrathoracic vascular structures (central venous, mean pulmonary arterial, pulmonary "capillary" (wedge) and left ventricular end-diastolic pressures) and \( P_{aw} \) were measured as a function of pulmonary lavage in a patient, three dogs and a calf. Instillation of liquid in a single lung leads to an increase in intrathoracic pressure generated by the hydrostatic pressure of the liquid column used for instilling the liquid. The blood pressures of intrathoracically located vessels increase as a result of the increased intrathoracic pressure and as a result of an acute mediastinal shift away from the liquid-filled lung. There is loss of perfusion to liquid-filled alveoli because of increased alveolar pressure. As a result, \( P_{aw} \) is increased, since pulmonary blood flow is shifted to non-liquid-filled (ventilated) alveoli. Drainage of the lung causes a reduction of \( P_{aw} \) because of restoration of perfusion to nonventilated (collapsed) alveoli.

(Key words: Pulmonary lavage; Gas exchange; Pulmonary circulation; Pulmonary vascular pressures.)

The therapeutic instillation of large volumes of liquid in the lung (pulmonary lavage) is used in the treatment of various disorders such as pulmonary alveolar proteinosis and asthma.‡ During pulmonary lavage in a patient with pulmonary alveolar proteinosis we found marked increases in central venous pressure and \( P_{aw} \) after saline solution had been instilled in one lung. When the lung was drained central venous pressure and \( P_{aw} \) rapidly (less than 30 seconds) fell. It seemed of interest to document these changes and to consider their mechanisms. In addition to observations made during five therapeutic pulmonary lavages in this patient, three mongrel dogs and a calf were studied.§

Methods

MAX

The technique was essentially that described by J. Ramírez-R, in which a Carlsen catheter is inserted with the patient under topical anesthesia. Ventilation was maintained spontaneously throughout the procedure. The patient breathed approximately 100 per cent \( O_2 \).

A polyethylene catheter (PE 160) for monitoring central venous pressure was inserted through an antecubital vein for approximately 60 cm. A polyethylene catheter (PE 90) inserted percutaneously into a femoral artery was used for blood-gas sampling. Lead 2 of the EKG monitored continuously throughout the procedure.

The lavage liquid consisted of 0.9 per cent saline solution containing heparin, 7.5 units/ml, buffered to a pH of approximately 7.5 with sodium bicarbonate. Before use, the saline solution was warmed to 37°C.

After 20 minutes of inhalation of oxygen (to permit denitrogenation) the office of the Carlsen catheter from the lung to be lavaged was clamped. "Degassing" was continued for five minutes. The lung was then lavaged with the saline solution, the height of the saline column being maintained 25–29 cm above the mid-portion level. The volumes of saline solution used during individual cycles varied between 1,200 and 1,500 ml. Continuous auscultation of the ventilated lung was carried out to detect major spillover. Approximately

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The dog studies were performed to circumvent extensive studies of a severely ill patient. The calf was selected for study because the technical aspects of left ventricular pressure measurements are relatively simple in this species.
three minutes were required for the introduction of saline solution into the lung, which was then quickly drained by gravity.

The lavage was repeated for six to eight cycles. At the end of the last cycle, the patient was placed in the Trendelenburg position to encourage drainage and the lung was suctioned free of the remaining saline solution. Approximately 90 per cent of the total liquid used in the sequential lavages was recovered. Ventilation was then assisted (intermittent positive-pressure breathing) with 100 per cent oxygen for one to two hours, depending upon the blood-gas values.

Central venous pressure and blood-gas measurements were monitored throughout the procedure, with particular emphasis at the following times: 1) after intubation while the patient was breathing 100 per cent oxygen; 2) after “degassing”; 3) during lavage cycles with the lung containing saline solution and after emptying; 4) during the 2-hour post-lavage period, before and after extubation.

**Animal Studies**

The technique was similar to that described for man. All animals were anesthetized with intravenous pentobarbital. Correct placement of the Carlen catheter was verified as described above. Controlled ventilation with approximately 100 per cent oxygen using a Harvard piston respirator was maintained during the entire study.

In each dog, a catheter was inserted into the pulmonary artery and its location confirmed by fluoroscopy and pressure contours. In the calf, left ventricular pressures were obtained by retrograde catheterization through the femoral artery. Determinations of mean pulmonary arterial, pulmonary wedge, and left ventricular end-diastolic pressures permitted consideration of representative pulmonary vascular pressures on both right and left sides of the pulmonary circulation. A standard Latex esophageal balloon was placed in the middle third of the esophagus for monitoring intraesophageal pressures. Pressure measurements were recorded by appropriate pressure transducers on an Electronics for Medicine recorder. Blood-gas measurements were made on an Instrumentation Laboratory trielectrode unit. Changes in mediastinal position with one lung filled with liquid were determined by roentgenograms of the chest.

**Results**

**MAN**

Changes in Central Venous Pressure. Figure 1 shows results with respect to central venous pressure. Each point represents the mean value of four observations. On degassing, central venous pressure fell from a postintubation control level of 7 to 6 cm H$_2$O. When saline solution was instilled in one lung, the central venous pressures rose to levels higher than controls, i.e., 10, 14, 14, and 16 cm H$_2$O. When the lung was drained, the central venous pressures fell to control levels. This cycle,
a rise in central venous pressure when the lung contained a large volume of liquid and a fall in central venous pressure when the lung was drained, occurred invariably.

Changes in Arterial Oxygen Tension and Degree of Shunting. Figure 2 summarizes data concerning $P_{aO_2}$ and percentage of cardiac output not exposed to alveolar air. Each point represents the mean of five observations. Following “degassing” (atelectasis) there was the expected fall in $P_{aO_2}$ and increase in pulmonary right-to-left shunting. ** When liquid was instilled in the lung there was a substantial rise in $P_{aO_2}$ (to more than 100 mm Hg) and a decrease in the percentage of shunting. This suggests that nonventilated segments of the lung lost perfusion during filling with liquid. When the lung was drained, perfusion was restored and $P_{aO_2}$ fell significantly as pulmonary right-to-left shunting increased.

ANIMALS

Changes in Intraseophageal, Pulmonary Arterial and Pulmonary Capillary Pressure in Dogs. Figure 3 summarizes the changes in intraseophageal (intrathoracic) pressure, mean pulmonary arterial pressure and pulmonary “capillary” (wedge) pressure during cycles of instillation and drainage of liquid. Each point represents the mean of three observations. It may be noted that the basal intraseophageal pressures were positive, a finding related to the use of the positive-pressure respirator. When the lung contained liquid, there were substantial increases in all pressures, which returned toward control values when the lungs were drained. It should be noted that the changes in mean pulmonary arterial pressure and wedge pressure when the lung contained liquid and following drainage were similar. Moreover, these pressure changes paralleled

** The percentage of shunting was calculated in conventional fashion, assuming a constant arterial-venous $O_2$ content difference.
changes in intrathoracic (intraesophageal) pressure.

Changes in Intrathoracic and Left Ventricular End-diastolic Pressure in a Calf (fig. 4). Left ventricular end-diastolic pressure varied with intraesophageal pressure with instillation and drainage of liquid, as was true of central venous, mean pulmonary arterial and pulmonary "capillary" pressure. There were marked increases in left ventricular end-diastolic and intraesophageal pressures following instillation. When the lung was drained, these pressures returned toward control values.

In animals, roentgenograms of the chests taken with the lungs filled with liquid showed that the mediastinum acutely shifted away from the liquid-filled side (fig. 5), returning to the normal position once the lung was drained. During "degassing" the mediastinum shifted toward the atelectatic side.

Discussion

The instillation of large volumes of liquid in a single lung by hydrostatic pressure results in four important changes: 1) an increase in intrathoracic pressure; 2) a generalized increase of intrathoracic intravascular pressures; 3) an acute shift of mediastinal structures away from the liquid-filled lung; 4) loss of pulmonary capillary perfusion to liquid-filled alveoli. All these changes must reflect acute mechanical effects produced by filling the lung with liquid, since rapid reversal occurs when the liquid is drained from the lung.

The increased intrathoracic pressure is re-
lated to hydrostatic pressure generated by the column of liquid involved in filling the lung and transmitted to other intrathoracic structures. This differs from other circumstances associated with intra-alveolar liquid accumulation. In intra-alveolar pulmonary edema of cardiogenic origin, intrathoracic pressure is not profoundly affected, since liquid filling occurs diffusely in both lungs and intrathoracic pressure generally reflects non-liquid-filled thoracic structures. In drowning, there is usually more or less symmetrical filling of the lungs with liquid. Intrathoracic pressure is elevated to the same extent as ambient external pressures, so the difference between intrathoracic and extrathoracic pressure is not affected. The level of intrathoracic pressure depends primarily on the depth at which drowning occurs.

The generalized increase in intrathoracic intravascular pressures reflects transmission of the elevated intrathoracic pressure, as well as compression of major vessels by the mediastinal shift. This view is supported by the fact that the changes in pressure (Δ pressure) are essentially equal in all segments of the pulmonary vascular bed and are quantitatively related to the increase in intracapillary pressure. As a result, pressure gradients, e.g., pulmonary artery minus left ventricular end-diastolic pressure, are unchanged. The mediastinal shift is produced by the localized increase in intrapulmonary pressure produced by the instillation of liquid in one lung.

The changes in PaO₂ reflect a complex series of events, as follows. Demineralization of a lung followed by occlusion results in acute atelectasis, as indicated in our studies by direct roentgenographic demonstration. This confirms previous reports that lobes filled with 100 per cent oxygen show complete atelectasis within ten minutes. Acute pulmonary atelectasis produced by endobronchial occlusion results in increased venous admixture related to continued perfusion of nonventilated alveoli, demonstrated both by increased pulmonary right-to-left shunting and by histologic observations indicating that the collapsed lung has a markedly dilated capillary bed supplying collapsed alveoli.6

During filling of alveoli with liquid there must be an increase of intra-alveolar pressure as a result of increased intrapulmonary pressure. The role of alveolar pressure in modifying regional pulmonary blood flow has been defined by the work of Rodbard,7 Banister and Torrance,8 Permutt and co-workers,9 and West.10 These workers showed that pulmonary capillaries are compressible vessels and are exposed to alveolar pressure. In areas where alveolar pressure exceeds mean pulmonary arterial pressure, pulmonary vessels are collapsed and blood flow ceases (Zone 1). It may be postulated that following instillation of liquid alveolar pressure in liquid-filled segments exceeds regional pulmonary arterial pressure and blood flow ceases. As a result, these segments are not only nonventilated, but also nonperfused. Pulmonary blood flow is shifted to the non-liquid-filled segments of lung which, being ventilated, function more normally in gas exchange. As a result, PaO₂ increases. When the lung is emptied, the lung returns to its previous atelectatic state, perfusion is restored to nonventilated alveoli, and PaO₂ drops.

References