

BRIEFS FROM THE LITERATURE

JOHN W. PENDER, M.D., *Editor*

Briefs were submitted by Drs. John Adriani, Norman A. Bergman, Peter P. Bosomworth, M. T. Clarke, J. E. Eckenhoff, Martin Helrich, J. R. Householder, J. J. Jacoby, S. J. Martin, S. R. Oechs, R. E. Ponath, William Rabenn, Alan D. Randall, Lawrence Reichmann, Clarence Serfling. Briefs appearing elsewhere in this issue are a part of this column.

PULMONARY BLOOD FLOW Positive pressure inflation of human and canine lungs diminishes pulmonary blood flow in preparations perfused with their own blood. The increasing resistance to flow is explained on the basis of capillary compression. If the lungs are encased and inflated by applying negative intrapleural pressure, pulmonary blood flow increases. Maximal flow occurs with negative pressures of 10–20 cm. of water. A further increase of the negative pressure causes a decline of pulmonary perfusion which is thought to be related to capillary collapse. If the trachea is occluded and negative pressure applied to the surface of the lungs, pulmonary blood flow continues to rise. (*Müller, A., and Debrunner, W.: Pleuraler Sog und endotrachealer Druck im Vergleich zur Lungendurchblutung, Der Anesthetist 9: 344 (Nov.) 1960.*)

PULMONARY HYPERTENSION An evaluation was made of the effect of increasing negative intrapleural pressure with suction catheters in the closed chest on pulmonary artery pressure in dogs following 50 to 85 per cent pneumonectomy. A determination was made of the effect of distention of the lung on pulmonary vascular resistance. Fifteen mongrel dogs were subjected to 50 to 85 per cent resection of the total lung capacity and their chests closed with thoracotomy tubes in place. Intrapleural, endotracheal, femoral and pulmonary artery pressures were then recorded. Negative pressure inflation of the residual lung was carried out by removal of the residual intrapleural air. The animals were permitted to breathe spontaneously with both normal intrapleural pressures and high negative intrapleural pressures so as to over-

distend the lung. The results obtained demonstrated that over-distention of residual lung does not aggravate any pulmonary hypertension produced by large pulmonary resections. The study did not evaluate the effect of over-distention by elevated endotracheal pressures. (*Reimann, A. F. and others: Pulmonary Artery Pressure Studies: Does Over Distention of Lung Cause Hypertension? Dis. Chest 39: 56 (Jan.) 1961.*)

CARDIOPULMONARY BYPASS Prolonged employment of the filming oxygenator is associated with the deposition of considerable amounts of embolic fat within the vascular tree of both humans and experimental animals. In dogs, following two hours of cardiopulmonary bypass, employing a macrobubble type oxygenator at high flow rates, coalesced fat was observed in the kidney, brain, liver and urine. The degree of embolization was similar to that following use of the filming type oxygenator. Following an identical period of bypass with a membrane oxygenator no intravascular fat was observed. The fat emboli observed probably resulted from alterations in the state of emulsification of serum lipids on prolonged direct exposure to gases in the oxygenator. The clinical significance of fat embolization during cardiopulmonary bypass is not known. (*Owens, G., Adams, J., and Scott, H. W.: Embolic Fat as Measure of Adequacy of Various Oxygenators, J. Appl. Physiol. 15: 999 (Nov.) 1960.*)

EXTRACORPOREAL CIRCULATION In a series of 30 cases of cardiopulmonary bypass and an additional 4 cases using hypothermia, anesthesia consisted of thiopental induction, curarization and hyperventilation with