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Pulmonary Physiology

ISOPROTERENOL AND PULMONARY SHUNTING Twelve mongrel dogs weighing 15–18 kg were anesthetized with pentobarbital and their lungs ventilated mechanically following tracheal intubation. The femoral vein and aorta were cannulated, and a Swan-Ganz catheter was placed in the pulmonary artery. Control values for pulmonary shunting, cardiac output, pulmonary arterial pressure, pulmonary-artery wedge pressure, and pulmonary vascular resistance were obtained following establishment of a steady state. An intravenous infusion of iosproterenol, 0.1 μg/kg/min, was given for two hours. Measurements were made 5, 15, 30, 60, 90, and 120 minutes following the start of the infusion. Pulmonary shunting, cardiac output, and pulmonary arterial pressure significantly increased at all measurement times compared with control values, with maximum changes obtained at 30, 15, and 5 minutes, respectively. There was no significant change in pulmonary-artery wedge pressure. Pulmonary vascular resistance decreased at all measurement times. These decreases were significant only at 15, 30, and 60 minutes. The increase in pulmonary shunting was assumed to be due to ventilation–perfusion inequalities secondary to increased pulmonary blood flow and pulmonary arteriolar vasodilation. Use of isoproterenol in critically ill patients could lead to repiratory distress. (*Berk JL, and others: Pulmonary insufficiency produced by isoproterenol. Surg Gynecol Obstet 143:725–726, 1976.*)