

12. Reynolds DV: Surgery in the rat during electrical analgesia induced by focal brain stimulation. *Science* 164:444-445, 1969
13. Akil H, Mayer DJ: Antagonism of stimulation-produced analgesia by p-CPA, a serotonin synthesis inhibitor. *Brain Res* 44:692-697, 1972
14. Black P, Cianci SN, Markowitz RS: Alleviation of pain by hypothalamic stimulation in the monkey. *Confin Neurol* 34:374-381, 1972
15. Schmidek HH, Ervin FR, Sweet WH: Alterations in the pain threshold produced by mesencephalic, thalamic, and limbic stimulation in the awake squirrel monkey (*Saimiri sciureus*). *Fed Proc* 27:518, 1968
16. Valenstein ES, Beer B: Reinforcing brain stimulation in competition with water reward and shock avoidance. *Science* 137:1052-1054, 1962
17. Liebeskind JC, Mayer DJ, Akil H: Central mechanisms of pain inhibition: Studies of analgesia from focal brain stimulation. *Adv Neurol* 4: 261-268, 1974
18. Heath RG, Mickle WA: Evaluation of seven years experience with depth electrode studies in human patients, *Electrical Studies on the Unanesthetized Brain*. Edited by Ramey ER, O'Doherty DS. New York, Hoeber, 1960, pp 214
19. Gol A: Relief of pain by electrical stimulation of the septal area. *J Neurol Sci* 5:115-120, 1967
20. White JC, Sweet WH: Pain and the Neurosurgeon—a Forty-Year Experience. Springfield, Ill., Charles C Thomas, 1969, pp 901-904

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 isoproterenol, 0.1 $\mu\text{g/kg/min}$, 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 respiratory distress. (Berk JL, and others: *Pulmonary insufficiency produced by isoproterenol*. *Surg Gynecol Obstet* 143:725-726, 1976.)