such as digitalization, prior administration of a beta-adrenergic blocking agent, or for the patient afflicted with extensive coronary atherosclerosis of a degree which usually precludes anesthesia, much less surgery. This was best illustrated by a recent cooperative analysis of intraoperative deaths occurring during bilateral internal mammary artery implantations. The results of this joint study revealed the importance of constant maintenance of perfusion pressure, normal oxygen saturation and pH, and indicated also the exquisite sensitivity of the circulation in patients with coronary disease to even minor shifts in blood volume away from normal. Some problems were not so readily recognized, such as the sudden circulatory collapse which occurred in occasional patients and appeared to result from potentiation of beta-adrenergic blocking drugs by certain anesthetics. Combined thinking elucidated these problems and led to a significant reduction in intraoperative mortality. Similarly, at many institutions cardiorespiratory therapy in acute care areas is administered by members of the anesthesia service working in close collaboration with their cardiological colleagues.

Thus, the relationship of the anesthesiologist and the cardiologist has been one of mutual education and collaboration. This is best viewed as a healthy and productive symbiosis, the fruits of which have been improved and more rational patient care.

Drugs

EPINEPHRINE SENSITIVITY *In dogs and rabbits, myocardial sensitivity* to epinephrine was enhanced by administration of thiopental or halothane. This increased cardiac irritability was not decreased by prevention of carbon dioxide accumulation; therefore, it was not due merely to hypoventilatory hypercapnia. (*Lipecz, J., and Harbauer, G.: Investigation on the Effect of Halothane and Thiopental on the Epinephrine Tolerance of the Heart, Der Anaesthesist 18: 259 (Aug.) 1969.*)

MAO INHIBITORS AND HALOTHANE Four monoamine oxidase (MAO) inhibitors, tranylcypromine, pheniprazine, pargyline, and nialamide, prevented the 2 to 3.5 C drop in temperature that invariably accompanies halothane anesthesia in cats. The MAO inhibitors themselves produced either no change in temperature or slight hyperthermia. Subsequent administration of halothane produced marked hyperthermia in cats that had no response to MAO inhibition and marked acceleration in temperature elevation in those that had shown only slight temperature elevation after MAO inhibition. In several instances the combination of MAO inhibition and halothane produced lethal hyperpyrexia. Temperature elevation secondary to MAO inhibition is thought to be related to increased levels of 5-hydroxtryptamine in the anterior hypothalamus. Halothane somehow potentiates this effect. (*Summers, R. J.: Effects of Monoamine Oxidase Inhibitors on the Hypothermia Produced in Cats by Halothane, Brit. J. Pharmacol. 37: 400 (Oct.) 1969.*)