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Drugs

GLUCAGON Intravenous glucagon (2 $\mu\text{g}/\text{kg}$) restored A-V conduction time to normal in dogs following its increase by prior administration of propranolol. This action of glucagon is not one of beta-adrenergic stimulation. Rather, it appears that the cardiac effects of glucagon are mediated through a pathway similar to that which mediates the effects of catecholamines, *i.e.*, the activation of adenylyl cyclase and consequent formation of cyclic 3',5'-AMP. Unlike the cardiac effects of catecholamines, those of glucagon are not blocked by beta-adrenergic blockade. Glucagon, therefore, may be able to reverse the cardiodepressant effects of propranolol without reversing its effectiveness in controlling arrhythmias. (*Whitsitt, L. S., and others: Effects of Beta-receptor Blockade and Glucagon on the Atrioventricular Transmission System in the Dog, Circ. Research* 23: 585 (Nov.) 1968.)

ANTI-MOTION-SICKNESS The effectiveness of a drug in reducing susceptibility to acute motion sickness is readily determined in a slow-rotation room where the stressful accelerations are under quantitative control. Fifty subjects were used, each serving as his own control, to evaluate 16 representative anti-motion-sickness drugs. Only drugs with sympathomimetic or parasympatholytic actions and some of the antihistamines were notably effective. The summation effect of dextroamphetamine sulfate and 1-scopolamine hydrobromide provided far better protection than any single drug. (*Wood, C. D., and Graybiel, A.: Evaluation of Sixteen Anti-motion Sickness Drugs Under Controlled Laboratory Conditions, Aerospace Med.* 39: 1341 (Dec.) 1968.)

ATROPINE VS. SCOPOLAMINE Central nervous system and peripheral effects of atropine, scopolamine, and eight other drugs were evaluated separately. Scopolamine was less potent than atropine against sarin poisoning in guinea pigs; against the convulsant action of intracerebral carbachol or acceleration of screen climbing in mice; and production of mydriasis in mice and rabbit. Scopolamine was almost twice as potent as atropine in prevention of bradycardia after methacholine. (*Madill, H. D., Stewart, W. C., and Savote, M. L.: Central and Peripheral Anticholinergic Potency of Some Drugs Antagonistic to Anticholinesterase Poisoning, Canad. J. Physiol. Pharmacol.* 46: 559 (July) 1968.)