cardiac sympathetic and vagal nerve stimulation on atrial and ventricular dynamics, Circ. Res. 8: 871, 1960.
16. Donald, E. E., and Shepherd, J. T.: Initial cardiovascular adjustment to exercise in dogs with chronic denervation, Ann. J. Physiol. 207: 1325, 1964.
17. Warner, H. R., and Toronto, A. F.: Regulation of cardiac output through stroke volume, Circ. Res. 8: 549, 1960.
18. Guyton, A. C.: Regulation of cardiac output, ANESTHESIOLOGY 29: 314, 1968.
19. Gernandt, B., Liljgestrand, G., and Zotterman, Y.: Efferent impulses in the splanchnic nerve, Acta. Physiol. Scand. 11: 230, 1945.
20. Weidinger, H., Fedina, L., Schaffer, H., and Kehrel, H.: Ueber die Lokalisation das "Bulbaren Sympathischen Zentrums" und Seine Beeinflussung Durch Atung und Blatdruck, Z. Kreislaufforsch 50: 229, 1961.

## Drugs

**GLUCAGON** Intravenous glucagon (2 μg/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 adenyl 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 l-scopolamine hydrobromide provided far better protection than any single drug. (*Wood, C. D., and Graybiel, A.: Evaluation of Sixteen Antimotion 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 Savoie, M. L.: Central and Peripheral Anticholinergic Potency of Some Drugs Antagonistic to Anticholinesterase Poisoning, Canad. J. Physiol. Pharmacol. 46: 559 (July) 1968.*)