**ANGIOTENSIN** Angiotensin II, given intravenously in doses of 40–50 μg./100 Gm. body weight, produces an increase in the capacity of the adrenal to synthesize aldosterone, but increase in the production of corticosterone is not significant. Such changes occur about 24 hours after the last injection of angiotensin II and after the circulatory effect of the drug has ceased. (*Glaz, E., and Sugar, K.: The Effect of Synthetic Angiotensin II on Synthesis of Aldosterone by the Adrenals, J. Endocr. 24: 299 (July) 1962.*)

**VASOPRESSIN** Under physiologic conditions of stress, there is normally an outpouring of antidiuretic substance. In these circumstances, its coronary constricting action is over-ridden by a predominantly sympathomimetic autonomic activity; however, if conditions lead to excessive parasympathomimetic tone, the effects of vasopressin on cardiac function may be deleterious. (*Kramer, J., and others: Role of Vasopressin in Cardiac Arrests, Amer. J. Surg. 104: 664 (June) 1962.*)

**CATECHOL AMINES** During air breathing the levels of circulating norepinephrine had to be increased at least 13 times above control level to duplicate the pulmonary arterial pressor response to acute hypoxia. In intact man the infusion of norepinephrine elicited pulmonary arterial hypertension by a mechanism different from that of acute hypoxia. The pressor response to acute hypoxia arose from an increased pulmonary vascular resistance. The pressor response to norepinephrine originated primarily in back pressure from the left heart without an increase in pulmonary vascular resistance. Levels of circulating norepinephrine that were insufficient to increase the pulmonary wedge pressure did not exaggerate the pulmonary arterial pressor response to acute hypoxia. Depletion of the pulmonary vascular nerve endings of norepinephrine by reserpine in dogs did not prevent the usual pulmonary arterial pressor response to acute hypoxia. No evidence was found for a role of either epinephrine or norepinephrine in the pulmonary arterial response to acute hypoxia of moderate degree. (*Goldring, R. A., and others: Catecholamines in the Pulmonary Arterial Pressor Response to Acute Hypoxia, J. Clin. Invest. 41: 1211 (June) 1962.*)

**CATECHOL AMINES** In the dog chlorpromazine and reserpine act differently, both as to site and mechanism. Using quick freeze techniques, the noradrenalin and adrenalin contents of frontal cortex, hypothalamus, hippocampus and the ascending reticular formation of the midbrain were studied. Small doses of chlorpromazine (less than 5 mg./kg.) raised catechol-amine concentrations in all areas, while larger doses reversed this effect with 25 mg./kg., reducing noradrenalin in all areas except the hypothalamus. The rise in adrenalin was greater than noradrenalin and was particularly high in midbrain and hippocampus. Reserpine, 2 mg./kg., depleted catechol amines in all areas, being greatest for noradrenalin, particularly in the midbrain and frontal cortex. (*Malhotra, C. L., and Prasad, K.: Effect of Chlorpromazine and Reserpine on the Catechol Amine Content of Different Areas of the Central Nervous System of the Dog, Brit. J. Pharmacol. 18: 593 (June) 1962.*)

**RESERPINE** Heart papillary muscle from cats pretreated with 10 mg./kg. body weight of reserpine was studied *in vitro* for the response to subsequently-given ephedrine. The outstanding effect was the inability of the post-reserpine preparation to exhibit the usual spontaneous beat following ephedrine and a diminished positive inotropic action. The peak contraction height and the rate of muscle failure was uninfluenced by reserpine, as was the contractile response to 0.1 μg./ml. of adrenalin. Since reserpine depletes cat heart catechol amines, it is reasoned that the inability of ephedrine to initiate spontaneous beating under these circumstances is due to its action through release of these amines. Because adrenalin causes spontaneous beating in papillary muscles not responsive to ephedrine, blockade of cardiac adrenergic receptors by reserpine was not contributory. (*Cariroli, V. J., Riley, J. F., and Robert, J.: Effect of Reserpine Pre-treatment on the Response of Isolated Papillary Muscle to Ephedrine, Brit. J. Pharmacol. 18: 588 (June) 1962.*)

**NEOSTIGMINE EXCRETION** A method was described for the quantitative estimation of neostigmine and pyridostigmine in urine by colorimetric determination of the respective

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