

*lowing Chronic Administration of Levorphan and Levorphan-Levallorphan Mixture in Rabbits, J. Pharmacol. & Exper. Therap. 118: 193 (Oct.) 1956.*

**CHLORPROMAZINE** After intravenous administration to dogs, the drug was most concentrated in the brain, where the level was about seventy times that in the plasma. The biologic half life of the drug is probably about six hours. Negligible amounts of the unchanged drug are excreted in the urine. The sulfoxide is probably a major metabolic product which undergoes further metabolism to an unidentified product. (Salzman, N. P., and Brodie, B. B.: *Physiological Disposition and Fate of Chlorpromazine and Method for Its Estimation in Biological Material, J. Pharmacol. & Exper. Therap. 118: 46 (Sept.) 1956.*

**CHLORPROMAZINE** Intravenously administered chlorpromazine frequently reduced cerebral blood flow, apparently as a result of the associated reduction in mean arterial blood pressure. Cerebral oxygen consumption was not depressed. The metabolic effect of chlorpromazine is quite different from that of morphine and barbiturates, which depress cerebral oxygen uptake without affecting cerebral blood flow. (Moyer, J. H., and others: *Effect of Chlorpromazine on Cerebral Hemodynamics and Cerebral Oxygen Metabolism in Man, Circulation 14: 380 (Sept.) 1956.*

**CHLORPROMAZINE** Spinal, epidural or intercostal celiac (splanchnic) block is usually contraindicated for a patient who has recently received chlorpromazine. A general anesthetic or peripheral nerve block is safer. (Moore, D. C., and Bridenbaugh, L. D.: *Chlorpromazine; Report of One Death and Eight Near Fatalities Following Its Use in Conjunction with Spinal, Epidural, and Celiac Plexus Block, Surgery 40: 543 (Sept.) 1956.*

**BLOOD PRESSURE** Pitressin caused sodium and water to move into cells, the shift being correlated with the rise in blood pressure. The rise and fall of plasma potassium also coincided with the rise and fall of blood pressure. Since such "sodium pump mechanisms" are also affected by adrenaline, norepinephrine, and histamine,

it is implicated as a basic mechanism in smooth muscle contraction and the elevation of blood pressure. (Friedman, S. M., Nakashima, M., and Friedman, C. L.: *Extrarenal Effects of Intravenous Pitressin in Nephrectomized Rats, Circulation Res. 4: 557 (Sept.) 1956.*

**CÉREBRAL CIRCULATION** There are no significant adrenergic constrictor receptors in the intracranial arterioles supplied by the internal carotid artery of the dog. (Green, H. D., and Dennison, A. B., Jr.: *Absence of Vasomotor Response to Epinephrine and Arterenol in Isolated Intracranial Circulation, Circulation Res. 4: 565 (Sept.) 1956.*

**CEREBRAL HEMODYNAMICS** In man thiopental was shown to decrease cerebral oxygen consumption with little change in blood flow, and to decrease cerebral resistance and arteriovenous oxygen difference. At reduced body temperatures, cerebral metabolism and blood flow were proportionately reduced, provided shivering was well controlled. In the presence of shivering, cerebral metabolism was increased in spite of reduction of body temperature to 82.6 F. (Stone, H. H., and Frobese, A. S.: *Effect of Lowered Body Temperature on the Cerebral Hemodynamics and Metabolism of Man, Surg., Gynec. & Obst. 103: 313 (Sept.) 1956.*

**PULMONARY EDEMA** Treatment of pulmonary edema is directed toward amelioration of the alveolar ventilation defect and correction of the water storage defect. The first may be facilitated by oxygen at increased pressure, helium-oxygen mixtures, antifoam agents, posture, tracheotomy and/or bronchial suction. If the water storage defect is the result of left ventricular failure, the work load on the left ventricle may be decreased by venesection, positive pressure breathing, venous occlusion at the extremities, sympatholytic agents, spinal anesthesia and diuretic agents. The work output of the heart may be increased by digitalis and, in cases with increased intracranial pressure and bradycardia, by atropine. (Visscher, M. B., and others: *Physiology and Pharmacology of Lung Edema, Pharmacol. Rev. 8: 389 (Sept.) 1956.*