for fifteen minutes at 20 C. During cardiac arrest induced by intracoronary injection of potassium citrate, the safe period of myocardial ischemia could be prolonged to at least one hour at 20 C. (*Gollam, F., and Nelson, I. A.: Anoxic Tolerance of Beating and Resting Heart During Perfusion at Various Temperatures, Proc. Soc. Exper. Biol. & Med. 95: 485 (July) 1957.*)

HYPOTHERMIA The body temperature of six dogs was lowered to 4.7 C. to 7 C. and extracorporeal circulation instituted for two to three hours followed by rewarming. Cardiac asystole occurred at the low temperatures. Easily reversible ventricular fibrillation occurred in 2 of 6 dogs. (*Kenyon, J. R., and Ludbrook, J.: Hypothermia Below 10 C. with Cardiac Recovery on Rewarming, Lancet 2: 171 (July 27) 1957.*)

HYPOTHERMIA In a study of 46 dogs, the effect of hypothermia on renal damage resulting from ischemia produced by unilateral renal arterial occlusion, has been determined by clearance studies. It was found that the renal damage resulting from renal arterial and aortic occlusion for periods of three hours or longer at normal body temperature was greatly reduced under hypothermic conditions. Renal function impairment was less in animals in which the aorta and renal artery were occluded simultaneously for four hours under hypothermic conditions than it was after a period of occlusion of two hours when the body temperature was normal. It appears that hypothermia provides excellent protection to the kidney when renal circulation is occluded for variable periods of time. (*Moyer, J. H., and others: Hypothermia: III. Effect of Hypothermia on Renal Damage Resulting from Ischemia, Ann. Surg. 146: 152 (August) 1957.*)

HYPOTHERMIA Dogs paralyzed with succinylcholine and ventilated by a pressure driven spirometer and 8 patients anesthetized with endotracheal ether and curare and ventilated with a Jefferson ventilator were studied before, during and after immersion hypothermia. Metabolic acidosis occurred with both anesthesia and hypothermia in dogs but in man only after cardiac inflow occlusion. The changes in solubility of carbon dioxide and in lung perfusion very nearly balanced the decrease in metabolism. The partial pressure of carbon dioxide of hypothermic patients could be maintained at the expected normal level for that temperature by ventilation with the same tidal volume and rate as were needed to maintain a partial pressure of 40 mm. Hg at normal body temperatures. (*Severinghaus, J. W., Stupfel, M. A., and Bradley, A. F.: Alveolar Dead Space and Arterial to End-Tidal Carbon Dioxide Differences During Hypothermia in Dog and Man, J. Appl. Physiol. 10: 349 (May) 1957.*)

SALICYLATE INTOXICATION Tetany and convulsions caused by hyperpnea and respiratory acidosis resulting from reflex vagal stimulation of the respiratory center in cases of salicylate overdosage may be controlled by intermittent positive pressure respiration, (*Freier, S., and others: Salicylate Intoxication Treated with Intermittent Positive Pressure Respiration, Brit. M. J. 1: 1333 (June 8) 1957.*)

ANILERIDINE The action of anileridine upon the respiratory and circulatory systems of normal man and the analgesic effects in surgical patients were studied. Anileridine proved less potent than morphine but more potent than meperidine. It was briefer in action, did not appear to liberate histamine and did not seem to have as much circulatory depressant effect as morphine or meperidine. (*Dripps, R. D., Millar, R. A. and Kneale, D. H.: Comparison of Anileridine, Morphine and Meperidine in Man, Surg. Gynec. & Obst. 104: 322 (Sept.) 1957.*)

NALORPHINE This narcotic antagonist was injected into dogs and rats and its distribution in urine, plasma and tissue determined. Following the subcutaneous injection of identical doses of drug (30 mg./kg.) nalorphine was conjugated more rapidly and more completely than morphine. Forty-four to 60 per cent of the nalorphine appeared in the urine in the

free or conjugated form. (*Woods, L. A., and Muehlenbeck, H. E.: Distribution and Fate of Nalorphine in Dog and Rat, J. Pharmacol. & Exper. Therap. 120: 52 (May) 1957.*)

NALORPHINE A comparison of the distribution of morphine and nalorphine in brain tissue was determined in dogs. Nalorphine penetrated into brain tissue much more rapidly and attained a concentration three to four fold greater than that observed with morphine. Nalorphine was dissipated from brain more rapidly than morphine. Nalorphine did not significantly alter the gross brain concentration of morphine. (*Woods, L. A.: Comparative Distribution of Morphine and Nalorphine in Dog Brain, J. Pharmacol. & Exper. Therap. 120: 58 (May) 1957.*)

DIGITALIS TOXICITY A 77-year-old woman developed marked carotid sinus sensitivity so that swallowing cold water or pressure on the carotid sinus produced syncope with cardiac arrest and nodal escape. When the digitalis was discontinued, sensitivity of the carotid sinus disappeared. (*Glotzer, S.: Syncope as Indication of Digitalis Toxicity, Circulation 16: 107 (July) 1957.*)

METABOLIC CHANGES When given at a rate of 0.5 Gm./kg./hour, there is no difference in electrolyte balance between intravenous injection of dextrose or fructose. A patient who does not have unusual extrarenal losses, advanced malnutrition, cardiac, renal, liver or endocrine disease will be in equilibrium on a daily intake of 80–100 mEq. each of sodium, potassium and chloride. (*Abbott, W. E., and others: Metabolic Alterations in Surgical Patients; Sodium, Potassium, Hexose and Water Secretion Following Intravenously Administered Dextrose or Fructose Solutions, Surgery 42: 296 (Aug.) 1957.*)

ACETYLCHOLINE The distribution in the nervous system of acetylcholine and the enzymes which metabolize it are reviewed. In the central nervous system, it seems that acetylcholine production is limited only to a proportion of the central neurons; the remainder must rely upon some other unidentified mechanism for the transmission of their messages. One of the precursors of acetylcholine is choline acetylase. In the discussion of choline acetylase formation and function it is noted that ether, acetone and the cholinesterases do no affect its activity. Although acetylcholine production is decreased by narcotics or local anesthetics in respiring brain slices, this does not seem to be due to inhibition of the choline acetylase mechanisms. Only chloroform may have a slightly inactivating effect. (*Hebb, C. O.: Biochemical Evidence for Neural Function of Acetylcholine, Physiol. Rev. 37: 196 (April) 1957.*)

METABOLISM OF THIOPENTAL Enzyme systems metabolizing thiopental and pentobarbital were studied *in vitro* with rabbit tissue homogenates. Both drugs were oxidized in liver homogenates while thiopental was metabolized to a lesser extent in kidney, brain and other tissues. The oxidation of pentobarbital and thiopental seemed to be carried out by different catalytic systems. (*Cooper, J. R., and Brodie, B. B.: Enzymatic Oxidation of Pentobarbital and Thiopental, J. Pharmacol. & Exper. Therap. 120: 75 (May) 1957.*)

BARBITURATE POTENTIATION The compound *N*-ethyl-piperdyldiphenylacetate hydrochloride (EPDA) is used for its antisecretory and spasmolytic activities. In large doses it is a stimulant to the central nervous system and in even higher doses will produce convulsions. However, pretreatment with the drug causes an increase in the mean sleeping time of mice anesthetized with hexobarbital. It is concluded that EPDA may represent a useful, relatively nontoxic agent for the potentiation of barbiturate hypnosis. (*Fujimoto, J. M.: Enhancement of Hexobarbital Hypnosis, Bull. Tulane Univ. Med. Faculty 16: 167 (Aug.) 1957.*)

FLUOTHANE The clinical experiences of a group of anesthetists from the administration of Fluothane for 2,500 operations was reported. No fatalities occurred and no serious postoperative illnesses were