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: 185 (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