DIAZOXIDE Diazoxide is a congener of chlorothiazide which has antihypertensive properties, but lacks ability to increase sodium excretion. Patients given 150 mg. of diazoxide twice daily showed highly significant decreases in systolic and diastolic blood pressure, but, unless salt and fluids were restricted, developed pitting edema. Simultaneously urine volume and renal sodium excretion fell while total blood volume increased. (*Hutcheon, D. E., and Barthalmus, K. S.: Antihypertensive Action of Diazoxide, Brit. Med. J. 2: 159 (July 21) 1962.*)

METHYLDOPA This drug is thought to produce its antihypertensive effect by inhibiting the decarboxylation of dopa and other amino-acids, thus lessening the production of noradrenaline and serotonin. Fall in blood pressure is proportional to the dose in a given patient. Though side effects such as slight drowsiness are present there are no serious toxic effects. Tolerance does occur. (*Dailey, D., and Evans, B.: Another Hypotensive Agent–Methyldopa, Brit. Med. J. 2: 156 (July 21) 1962.*)

ATYPICAL PSEUDOCHOLINESTERASE Four of six children in one family were found to have a deficiency of plasma-cholinesterase, and three of them have had lengthy episodes of apnea under general anesthesia. Their plasma-cholinesterase was estimated both by spectrometry and by means of "Acholest" test papers. Not all cases of apnea after administration of methonium compounds are due to low plasma-cholinesterase. Even should a low plasma-cholinesterase be responsible for such apnea it may only start this condition and then other factors may take over. Simple plasma-cholinesterase deficiency alone cannot cause more than twenty minutes apnea. Other factors include acidosis due to the accumulation of carbon dioxide, electrolyte imbalance, insecticide poisoning, and accumulation of succinylmonocholine from the breakdown of the dicholine. (*Ruddell, J. S.: Apnoea Due to Suxamethonium Associated with Familial Pseudocholinesterase Deficiency, Lancet 1: 832 (April 21) 1962.*)

RESPIRATORY STIMULANT Vanillic acid diethylamide (Ethamivan) produced respiratory stimulation which was most marked when the associated alveolar P_{CO_2} was low. As alveolar P_{CO_2} increased above 39 mm. of mercury, the drug effect disappeared. These results would limit the usefulness of the drug inasmuch as clinical situations usually involve elevated P_{CO_2}. However, larger doses, which were associated with severe side effects in conscious subjects, might be tolerated in clinical situations by less conscious patients with more effective respiratory stimulation. (*Anderson, J. L., and others: Effect of Ethamivan (Vanillic Acid Diethylamide) on the Respiratory Response of Healthy Young Men to Carbon Dioxide, in the Absence of Hypoxia, Brit. J. Pharmacol. Chem. 19: 142 (Aug.) 1962.*)

BARBITURATE POISONING A 2.3 per cent mortality rate was reported in 605 cases of acute barbiturate poisoning treated between 1950 and 1959. Fluid balance was emphasized and forced diuresis was routinely employed. A urine flow from an indwelling catheter of 15 to 20 drops per minute was held to indicate that the circulation and kidneys were functioning normally. Mercurial diuretics were administered intravenously, and the speed of intravenous infusion ('Macrodex,' Ringer's solution, or 5 per cent glucose solution) was increased to that of the urine flow. Where necessary, diuretics were given again 17 to 20 hours later. On an average, 7.2 liters of fluid (no blood) was given over twenty-four hours, and this resulted in a urinary output of 4.8 liters per twenty-four hours. The following advantages are claimed: more rapid recovery of consciousness; reduced need for drugs to raise the blood pressure; absence of renal complications; absence of hyperthermia; and diminished tendency for crusts to form in the respiratory passages. (*Ohlsson, W. T. L., and Fristedt, B. I.: Blood Lavage in Acute Barbiturate Poisoning, Lancet 2: 12 (July 7) 1962.*)

PULMONARY HEMODYNAMICS Pulmonary carbon monoxide diffusing capacity and pulmonary capillary blood volume were