and Rauwolfia tranquilizer therapy. With many of these drugs, severe extrapyramidal symptoms are uncommon, but with those phenothiazines containing a piperazine moiety in the side chain, *e.g.*, trifluoperazine (Stelazine), prochlorperazine (Compazine), perphenazine (Trilafon), thiopropazate (Dartal), such side effects are the rule rather than the exception. Based on the postulate that the syndrome is due to interference with the normal functioning of central catecholamines, particularly dopamine, and of central histamine; dopa (the precursor of the catecholamines) and diphenhydramine (Benadryl) were employed therapeutically. Dopa was only mildly beneficial in a small number of patients, but diphenhydramine completely controlled the reaction in all cases. Although with rare exceptions the syndrome is completely reversible on removal of the offending drug, the development of severe and unexpected extrapyramidal reactions in patients with some underlying physical illness would be potentially serious because of heightened sensitivity to, and slower metabolism of the drug. Parenteral anti-Parkinsonian medication to accompany discontinuance of the phenothiazine would be strongly indicated. Appropriate treatment would be immediate parenteral administration of 25 mg. of diphenhydramine. (*McGeer, P. L., and others: Drug-Induced Extrapyramidal Reactions, J. A. M. A. 177: 665 (Sept. 9) 1961.*)

ORAL ANTISPASMODIC Five drops of a solution containing 50 mg. procaine, 4 mg. pentobarbital, and 4 mg. phenobarbital (Barbicaine) per cubic centimeter gave relief to 4 infants with pyloric stenosis and 64 per cent of 50 infants with infantile colic. (*LaBranche, H. G.: Procaine-Barbiturate Mixture in Infantile Colic, Western Med. 2: 394 (Sept.) 1961.*)

RESPIRATORY STIMULANT A series of 43 patients with respiratory depression from severe barbiturate poisoning, chronic pulmonary emphysema, and miscellaneous causes was treated with ethamivan (vanillic diethylamide, Emivan). In patients severely depressed from barbiturate intoxication, initial doses of 400–500 mg. of ethamivan were given intravenously, followed by continuous infusions containing 1 Gm. in 250 ml. of 5 per cent dextrose in water. Rate of flow was adjusted according to response, close supervision of therapy being required to maintain optimal respiratory stimulation with a minimum of undesirable side effects (generalized pruritus, muscular twitchings, excitation and restlessness). The mildly vasopressor properties of the drug appeared to be valuable in those patients unable to tolerate intermittent positive pressure breathing because of ensuing hypotension. When given in adequate dosage, prompt stimulation of respiration and rousing could be expected, with ventilatory improvement demonstrated by significant decreases in Pa_{CO_2} in the emphysematous subjects. (*Silipo, S., and others: Experiences with Ethamivan, New Respiratory Stimulant and Analeptic Agent, J. A. M. A. 177: 378 (Aug. 12) 1961.*)

SUCCINYLCHOLINE APNEA When easily accessible superficial veins are not available, (*e.g.*, in infants) or in an emergency where rapid endotracheal intubation has to be performed, the intramuscular administration of succinylcholine provides a useful method for the relatively rapid production of muscular relaxation. When it is necessary to use intramuscular succinylcholine for the maintenance of prolonged muscular relaxation, the repeat dose should be one-third to one-half the initial dose, and its administration should be delayed until the patient's respiratory tidal volume is well on its way toward normal. If prolonged apnea develops, controlled ventilation must be maintained until the patient's spontaneous respiration returns, and then respiration must be assisted until its depth becomes adequate. On rare occasions it may be necessary to maintain artificial respiration for many hours. During this time, the patient's cardiovascular system should be supported by intravenous infusions and other measures (vasopressors, digitalis) as necessary. The maintenance of adequate circulation and urinary excretion will facilitate the excretion of succinylcholine. The use of respiratory stimulants or anticholinesterases (*e.g.*, edrophonium or neostigmine) is contraindicated. Beyond careful attention to the patient's circulation and respiration, infinite patience is the only additional measure re-

quired for the successful treatment of prolonged apnea caused by the intramuscular use of succinylcholine. (*Foldes, F. F., and Brown, I. M.: Possible Dangers of Intramuscular Succinylcholine, J. A. M. A. 177: 514 (Aug. 19) 1961.*)

LEVALLORPHAN The effect of levallorphan on intestinal motility following administration of morphine was studied by using a balloon tube in human beings and in dogs. Levallorphan counteracted the effect of morphine on the intestines without, however, interfering with the analgesic effects of morphine. In the average patient, best results were obtained by using 20 mg. of morphine and 1 mg. of levallorphan. In this dosage, undesired increase of the tone of the intestinal wall is prevented but the increase of peristalsis owing to morphine is not reduced. (*Hart, W., and Becker, F.: Clinical and Experimental Investigations Concerning the Effect of Levallorphan Tartrate on Change of Function of Intestines due to Morphine, Der Anaesthesist 10: 230 (Aug.) 1961.*)

ANESTHETIC TIME/DOSE CURVE An equipotent mixture of thiopenal and oxymorphone (470 : 1) was administered intravenously to supplement nitrous oxide anesthesia in 44 cases of major surgical operation. The mean anesthetic time/dose curve indicated the anesthetic action of the thiopental-oxymorphone mixture is additive rather than synergistic. However, the recovery or "inactivation" time was reduced from 200 to 97 minutes. (*Keeri-Szanto, M., Knaff, M., and Rondeau, Y.: Anesthetic Time/Dose Curves, III. The Interaction of Thiopental and Oxymorphone During Surgical Anesthesia, Clin. Pharmacol. Ther. 2: 441 (Jul.-Aug.) 1961.*)

PRESSOR HORMONES The actions of various pressor hormones were studied in the cat anesthetized with chloralose. There were no constant changes in cardiac output after angiotensin and vasopressin, but epinephrine caused a rise. The rise in blood pressure caused by angiotensin and vasopressin was mainly due to increased peripheral resistance. Epinephrine usually caused a biphasic increase in total peripheral resistance so that at different times during the rise in blood pressure this was predominantly due to increased cardiac output or to increased peripheral resistance. Angiotensin constricted the renal and mesenteric vascular beds strongly, the femoral, carotid, pulmonary, and coronary vascular beds probably less strongly. The existence of small vascular beds which could be dilated by angiotensin could not be excluded. Pulmonary vascular bed appeared to be less sensitive to angiotensin than the systemic vascular bed. Lysine-vasopressin constricted the mesenteric and femoral vascular beds, had no significant effect on the pulmonary vascular bed, and caused a profound dilatation in the renal vascular bed. Epinephrine constricted the renal vascular beds. There were variable effects on the mesenteric vessels. The femoral area was both dilated and constricted, the former predominating. Pulmonary arterial pressure was raised much more than by comparable doses of angiotensin, but the pulmonary vascular resistance was reduced. It appears that angiotensin is five to ten times as powerful by weight in raising blood pressure as is epinephrine in the cat. (*Barer, G. R.: Comparison of Effects of Angiotensin, Vasopressin and Adrenaline in Anesthetized Cat, J. Physiol. 156: 49 (Apr.) 1961.*)

STRESS RESPONSE Different anesthetic agents were studied in dogs regarding stress response. Blood sugar, serum sodium and potassium were measured, and eosinophile counts were done. Ethyl chloride, ether and barbiturates caused the greatest stress. Nitrous oxide and hydroxydione seemed to show least stress response. (*Csernohorszky, V., and others: Experimental Investigations Concerning Stressor Effect of Different Narcotics, Der Anaesthesist 10: 234 (Aug.) 1961.*)

HYDROXYCORTICOID LEVELS There is a progressive increase in the 17-hydroxycorticosteroid levels with advancing pregnancy, a further rise during labor and a prompt decline in the immediate postpartum period. This hyperadrenal state during pregnancy has long been recognized, for there is a decreased glucose tolerance and a frequent improvement of rheumatic diseases and asthma. (*Bryans, F. E., and Belither, A.: 17-Hydroxycorticosteroid Levels in Pregnancy, Amer. J. Obstet. Gynec. 82: 52 (July) 1961.*)