mortality rates, nevertheless recovery usually occurs if the infants do not die from early respiratory distress and if withdrawal symptoms are recognized early and adequately treated. Proper therapy consists of the administration of sedatives, preferably diminishing doses of barbiturates, oxygen and antibiotics for respiratory distress, and fluid and electrolytes parenterally when indicated in the presence of vomiting, diarrhea and/or dehydration. (*Kunstadter, R. H., and others: Narcotic Withdrawal Symptoms in Newborn Infants, J. A. M. A. 168: 1008 (Oct. 25) 1958.*)

NARCOTICS AND INTESTINAL MOTILITY Effects of morphine, levorphan, alphaprodine, and dihydrocodeine, as well as nalorphine and levallorphan were studied on intestinal motility *in vitro*. Intestinal hypertonicity or spasm was elicited consistently at subsedative and subnauseant dosage for each analgesic studied. Dihydrocodeine and alphaprodine exhibited approximately 1/30 the intestinal spasmogenic potency of morphine. Results of antagonism studies with respect to intestinal tone failed to support the concept of critical and constant analgesic-to-antagonist dose ratios. Instead, the absolute amount of antagonist drug necessary for initial prevention or reversal of analgesic-induced intestinal hypertonicity remained remarkably constant, regardless of the particular analgesic drug employed, dose of analgesic drug administered or order of administration. (*Gray, G. W.: Some Effects of Analgesic and Analgesic-Antagonist Drugs on Intestinal Motility, J. Pharmacol. & Exper. Therap. 124: 165 (Sept.) 1958.*)

INTRACELLULAR POTENTIALS Intracellular recording of action potentials generated by glial cells both *in vivo* and *in vitro* tissue cultures indicate that glial cells produce electrical responses which have a duration of more than 1000 times the length of the action potential of nerve cells. There is also evidence that electrical stimulation of the glial cell evokes a slow mechanical contraction lasting 7 to 16 minutes. These experimental findings raise new problems in the field of brain physiology. (*Tasaki, I., and Chang, J. J.: Electric Response of Glial Cells in Cat Brain, Science 128: 1209 (Nov. 14) 1958.*)

CHOLINESTERASE Pressure applied to a Pacinian corpuscle results in a graded potential inside the sense organ, and within limits this potential is a linear function of applied pressure. It is known that a potential is developed at the non-myelinated nerve ending within the corpuscle, but little is known of the mechanisms that transform the deformation into an action potential. Experimental evidence shows a large concentration of cholinesterase precisely around the nonmyelinated nerve ending and a negligible amount in the outer lamellar layers of the corpuscle. The cholinesterase found is not precisely the same as acetylcholine esterase, however, and the precise nature of the enzyme and its normal substrate remain to be elucidated. (*Loewenstine, W. R., and Molins, D.: Cholinesterase in a Receptor, Science 128: 1284 (Nov. 21) 1958.*)

MESENTERIC REFLEXES The reflex responses to the injections of veratridine, acetylcholine, nicotine, epinephrine, isoproterenol and sodium cyanide into the superior mesenteric artery of dogs anesthetized with morphine and chloralose were studied. The results illustrate that a well developed reflex pattern originates from the stimulation of sensory nerve endings in the mesentery of the small intestine. Veratridine, acetylcholine, and nicotine are capable of eliciting apnea and inhibition of the rhythmic contractions of the small intestine; whereas, epinephrine, isoproterenol and sodium cyanide cannot. The sole afferent pathway to these reflex responses is the splanchnic nerves. The sensory nerve endings being stimulated are apparently the termination of fibers subserving the sensation of pain. (*Riker, W. K.: Reflexes from the Intestinal Mesentery Elicited by Veratridine, Acetylcholine and Nicotine, J. Pharmacol. & Exper. Therap. 124: 120 (Sept.) 1958.*)

ACTH Experiments were carried out on rabbits which were given an intravenous injection of the marked homologous protein. The time required for removal of half the amount of injected protein was used to determine the total vascular permeability in these animals. In control rabbits this period averaged 3.3 hours. Three hours after a subcutaneous injection of 5 U. of ACTH or 5 mg. of cortisone acetate