Literature Briefs

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Briefs were submitted by Drs. Vagn Askrog (Denmark), C. M. Ballinger, M. T. Clarke, H. S. Davis, Deryck Duncalf, G. Hohmann (Germany), Martin Helrich, J. J. Jacoby, H. Landesman, W. H. Mannheimer, R. E. Ponath, Alan D. Randall, H. S. Roe, Norman Rosenbaum, P. H. Sechzer and W. Stocker. Briefs appearing elsewhere in this issue are a part of this column.

NOREPINEPHRINE Constriction of the ascending aorta in the guinea pig resulted in ventricular hypertrophy and congestive heart failure. In animals with heart failure which were killed 5 to 40 days after constriction, the norepinephrine stores in both ventricles were strikingly reduced; the extent of reduction was related to the severity of the constriction. (*Spann, J. F., Jr., Chidsey, C. A., and Braunwald, E.: Reduction of Cardiac Stores of Norepinephrine in Experimental Heart Failure, Science 145: 1439, 1964.*)

SYNAPTIC INHIBITION An impulse causes some of the synaptic vesicles which are in close contact with the synaptic membrane to eject their contained transmitter substance into the synaptic cleft. Some of the transmitter becomes momentarily attached to the specific receptor sites on the postsynaptic membrane, with the consequence that fine channels open across this membrane. The subsynaptic membrane momentarily assumes a sieve-like character. Chloride and potassium ions move across the membrane thousands of times more readily than normally, and this intense ionic flux gives the current that produces the inhibitory postsynaptic potential and that counteracts the depolarizing action of excitatory synapses, so effecting inhibition. (*Eccles, J. C.: Ionic Mechanism of Postsynaptic Inhibition, Science 145: 1140, 1964.*)

DIMENHYDRINATE Dimenhydrinate was administered intravenously to patients in spontaneous labor in order to evaluate its effect on the length of labor, its analgesic property, its potentiation of the effects of standard analgesics and its overall safety. No evidence was provided that labor was shortened or that uterine activity was increased in those receiving intravenous dimenhydrinate, nor did this drug decrease the need for conventional analgesics. There were no maternal or fetal complications. Intravenous administration of dimenhydrinate has no advantage over analgesics administered by other routes and has a limited use in current obstetrical practice. (*Harkins, J. L., and others: A Clinical Evaluation of Intravenous Dimenhydrinate in Labour, Canad. Med. Ass. J. 91: 164 (July 25) 1964.*)

NEOSTIGMINE TOXICITY Following thymectomy for myesthenia gravis, neostigmine toxicity has been found to be a cause of respiratory failure. A curious state of intolerance to certain effects of the cholinergic agent often develops in the early postoperative period, characterized by restlessness, apprehension, perspiration, tachycardia, dyspnea, excessive respiratory tract secretions, weakness, and hypotension. Repeated observations have shown that the progression of untoward effects can be halted by reduction or omission of neostigmine, provided respiratory failure has not developed. However, reduction of drug dosage to nontoxic levels may result in such generalized muscular weakness that respiratory efforts are inadequate to support ventilation. Even though a larger drug dose will improve muscle strength, it will also evoke copious secretions and bronchospasm. When this occurs, tracheostomy and ventilatory assistance are mandatory and unavoidable. Sensitivity to cholinergic drugs usually persists for 3 to 5 days after operation, occasionally longer. Thereafter, drug dosage can be again increased with relative impunity as the need to improve muscle strength indi-