kins, D. H., and Klink, E. J.: Orthophasic Post-systolic Myocardial Augmentation, Arch. Surg. 89: 354 (Aug.) 1964.)

HALOTHANE A widespread intense emotional reaction developed following the early reports of liver necrosis following halothane anesthesia. This reaction was similar to that which occurred following the Beecher-Todd report on the adverse effects of muscle relaxants. The "clinical impression" type of reports provoked scientific men and women in the field of anesthesia to undertake a full-scale investigation into the problem. Already the apprehension and emotional reaction is subsiding and some anesthesiologists are returning to their comfortable "ruts" with halothane. This subsidence of concern is just as unfortunate as the emotional convulsion that attended introduction of the problem. *(Cullen, S. C.: Editorial—Halothane, Clin. Pharmacol. Ther. 5: 395 (July-Aug.) 1964.)*

DRUG TERATOGENICITY The placenta behaves toward most drugs as an inert barrier with lipoid properties. Accordingly, any lipid-soluble drug (or one that crosses the blood-brain barrier) will readily penetrate the placental barrier. Morphine, meperidine, hyoscine, chloral hydrate, barbiturates and anticoagulants readily cross the placental membranes but do not seem to cause fetal deformities. Some doubt exists about the effects of meclizine and phenmetrazine. The detection of the teratogenic effect of drugs still remains a challenge. *(Cahen, R. L.: Evaluation of the Teratogenicity of Drugs, Clin. Pharmacol. Ther. 5: 480 (July-Aug.) 1964.)*

CATECHOLAMINES A definite correlation exists between the urinary excretion of catecholamines and the various types of stress. Generally, in forms of mental stress associated with anger, apprehension or exhilaration, norepinephrine excretion is increased. In emotional states characterized by apprehension, discomfort or painful or unpleasant feelings epinephrine excretion is increased. *(von Euler, U. S.: Quantitation of Stress by Catecholamine Analysis, Clin. Pharmacol. Ther. 5: 398 (July-Aug.) 1964.)*

COST OF BREATHING Oxygen cost of breathing increases exponentially with ventilatory efforts. At minute volumes of about 150 liters breathing becomes an end in itself, the whole oxygen uptake being required to satisfy the energy requirements of the respiratory effort. The quantity of oxygen available for work reaches an optimum value at ventilation of between 100–120 liters/minute. Breathing against resistance increases oxygen cost, more so during inspiratory than during expiratory obstruction. With maximal physical effort and in patients with severe restriction to respiration, the energy cost of breathing becomes part of the respiratory regulation in terms of hypoventilation and increasing tolerance to respiratory acidosis. *(Millhahn, H. P., and Eckermann, P.: Energy Consumption of Respiration, Klin. Wschr. 42: 722 (Aug. 1) 1964.)*

CARDIAC CATECHOLAMINES In a cat heart preparation superfusing an isolated segment of rabbit intestine, mephentermine releases a substance into the cardiac perfusate which produces relaxation of the intestine. The intestine-relaxing substance was not released by mephentermine from a heart taken from an animal pretreated with reserpine, but the relaxing substance was released after an infusion of levarterenol through the heart. The relaxing substance was released by mephentermine after pretreatment of the heart with dichloroisoproterenol, but the effects on the heart itself were blocked. The necessity of intact stores of catecholamines is indicated for cardiac action of mephentermine. *(Swaine, C. R., Perlmutter, J. F, and Ellis, S.: Release of Catecholamines from the Isolated Cat Heart by Mephentermine, Naunyn-Schmiedeberg Arch. Exp. Path. 248: 331 (June 22) 1964.)*

CARDIAC METABOLISM During asystole the following metabolic changes occur: glycogen and glucose decrease, alpha-glycerophosphate and lactic acid increase. Phosphocreatin and adenosine triphosphate (ATP) decrease with an associated decrease of the ATP/ADP coefficient. Injections of adenosine and inorganic phosphate, together with epinephrin, norepinephrin and glucose with insulin increased the synthesis of phosphocreatin.

Catecholamines and insulin also increase the rate of ATP synthesis. The quotient of ATP/ADP is increased. (*Isselhard, W., and others: Experiments to Improve Availability of Energy in the Artificially Arrested Heart and in Recovery during Re-perfusion, Klin. Wschr. 42: 757 (Aug. 1) 1964.*)

PERSANTIN Effect of Persantin on the human coronary circulation and oxygen and substrate uptake of the heart was studied in 38 patients undergoing diagnostic heart catheterizations. In a dosage of 0.2 mg./kg., Persantin produces a transient increase of coronary blood flow of about 10 per cent. The effect of the drug is dissipated in about 15 minutes. Oxygen and substrate uptake by the heart is not significantly altered. Persantin does not provide effective therapy in acute disturbances of the coronary circulation. (*Rudolph, W., and others: Effect of Persantin on Coronary Blood Flow, Oxygen and Substrate Uptake of the Human Heart, Klin. Wschr. 42: 667 (July 15) 1964.*)

VAGAL BLOCK Hypertrophic osteoarthropathy can be successfully treated by blocking of the vagus nerve using local analgesics. A new technique for vagus nerve blocking is described. (*Dam, W. H. and Hagelsten, J. O.: Blocking of the Vagus Nerve Relieving Osteoarthropathy in Lung Diseases, Danish Med. Bull. 11: 131, 1964.*)

ELECTRICAL GROUNDING Untreated operating room floors may be placed in the following order in increasing grounding resistance—(1) terrazzo flooring with conducting net, (2) ordinary terrazzo flooring, (3) tiled floors, (4) ordinary vinyl flooring. In all cases treatment with benzolconium chloride resulted in reduction of electrical resistance of charging of personnel. (*Hagelsten, J. O., and Larsen, O. S.: Resistance of Anesthetic Room Flooring and its Significance for the Risk of Explosion Before and After Treatment with Benzalconium Chloride, Ugeskr. Laeg. 125: 1841, 1963.*)

HYPOTHERMIA Patterns of fetal and maternal cardiac activity were observed by continuous electronic monitoring during hypothermia. Marked slowing of maternal and fetal heart rates were found to occur with induction of hypothermia with about a 1:2 ratio being maintained most of the time. The pattern of fetal cardiac response to stress resembled that observed during hypotension in a patient with normal body temperature. A healthy living infant was delivered 23 days following induced maternal hypothermia for repair of a cerebral aneurysm. (*Hess, O. V., and Davis, C. D.: Electronic Evaluation of the Fetal and Maternal Heart Rate During Hypothermia in a Pregnant Woman, Amer. J. Obstet. Gynec. 89: 801 (July 15) 1964.*)

PULMONARY EDEMA Destructive lesions in the preoptic areas in rats leads to fatal pulmonary edema. Bilateral injections of aconite into the preoptic areas of the rat likewise produce fatal pulmonary edema. The edema is due to a mass sympathetic discharge resulting in a marked rise in systemic arterial pressure, shunting of blood into the pulmonary circuit and appreciable rise in pulmonary venous pressure. The left ventricle is then unable to maintain its output against an increased peripheral resistance and leads to back pressure effects on pulmonary venous capillary vessels. These effects are largely blocked by surgical or drug interruption of the sympathetic outflow. (*Wood, C. D., and others: Influence of Autonomic Blockade on Pulmonary Edema, Proc. Soc. Exp. Biol. Med. 116: 809 (July) 1964.*)

CARDIAC CATECHOLAMINES The role played by endogenous catecholamines of the left ventricle in maintaining normal contractility is not established. The effect of guanethidine and reserpine on the isolated hearts of 11 rabbits was studied and a 70 to 94 per cent reduction in catecholamine content of the left ventricle with minimal reduction in contractility was found. Drastic reduction in catecholamine content of the left ventricle of the rabbit does not necessarily result in impairment of its contractility. (*Maxwell, R. A., and others: Catecholamine Depletion and Myocardial Contractility, Proc. Soc. Exp. Biol. Med. 116: 672 (July) 1964.*)