verine and Morphine Synergism in Pain Relief in Man, Science, 128: 84 (July 11) 1958.)

PLASMA PROTEINS AND CURARE The levels of *d*-tubocurarine chloride and its distribution in plasma was studied in 7 normal and 2 refractory human subjects. In the two refractory patients, considerably higher plasma levels of *d*-tubocurarine chloride were found than in those of normal controls. This finding suggests that *d*-tubocurarine is found in excessive quantities in the plasma of refractory patients, and is unable to diffuse out of the vascular system in effective concentration to exert its action at the neuromuscular junction. *(Aladjemoff, L., Dikstein, S., and Shafrir, E.: Binding of d-Tubocurarine Chloride to Plasma Proteins, J. Pharmacol. & Exper. Therap. 123: 43 (May) 1958.)*

MEPHENTERMINE The sympathomimetic amine, mephentermine sulphate (Wyamine) increases ventricular function in the isolated dog heart and in the dog with an open chest with a complete circulation. It has little effect on total peripheral vascular resistance. It increases myocardial oxygen consumption and decreases efficiency in the nondilated heart; but, the reverse was found when filling pressure was high in the heart. This is in agreement with LaPlace's Law concerning the relationship between the total tension developed by the myocardium and its oxygen utilization. *(Welch, G. H., and others: Effect of Mephentermine Sulphate on Myocardial Consumption, Myocardial Efficiency and Peripheral Vascular Resistance, Am. J. Med. 24: 871 (June) 1958.)*

VASOCONSTRICTOR DRUGS A study was conducted in dogs of the effects of epinephrine, *l*-norepinephrine, methoxamine and mephentermine on the excitability, refractory period, rhythmic abnormalities, conduction times and action potential of the heart. Methoxamine proved to be depressant in that it prolonged the action potential and the absolute refractory period, while raising the threshold to stimulation and slowing A-V conduction. The other drugs induced ectopic pacemaker action, abnormal spontaneous beats, and multiple responses to test stimuli. All drugs exhibited some degree of tachyphylaxis. *(Gilbert, J. L., and others: Effects of Vasoconstrictor Agents on Cardiac Irritability, J. Pharmacol. & Exper. Therap. 123: 9 (May) 1958.)*

POLYPHARMACY Methonium compounds, steroids and tranquilizers are only a few of the drugs introduced in the last few years which influence the response of patients to anesthesia. Side actions cannot be predicted by pharmacologists but become known only by clinical use over the years. *(Dundee, J. W.: Iatrogenic Disease and Anesthesia, Brit. M. J. 1: 1433 (June 21) 1958.)*

VITAMIN ANESTHESIA SCTZ, a derivative of the thiozole fraction of vitamin B_1, is being introduced in France as a sedative and hypnotic. *(Laborit, H., and others: SCTZ, A Depression of the Cerebral Cortex, J. Internat. Coll. Surgeons 29: 573 (May) 1958.)*

EEG AND ETHER Encephalogram desynchronization during ether anesthesia is dependent upon connection between cerebral cortex and the reticular formation. A microelectrode technique employed in cats showed continuing cortical activity where a section of cortex was disconnected from the reticular formation, while spontaneous firing of cortical cells still connected was deeply depressed. No over-all increase or decrease in activity of the reticular formation occurred though there was a change in rate of individual cellular discharges in response to peripheral stimulation. Cortical response to cortical stimulation was affected only under deep ether. Chlorpromazine prevented the EEG desynchronization of ether while Dibenamine did not. *(Schlag, J., and Brand, H.: Analysis of Electrophysiological Events in Cerebral Structures During Ether Anesthesia, Electroencephalog. & Clin. Neurophysiol. 10: 305 (May) 1958.)*

EEG A high per cent of children with cyanotic congenital heart disease had abnormal electroencephalograms as compared with those having acyanotic congenital heart disease. The abnormal patterns of the electroencephalograms were similar to changes produced by experimental hy-

poxia. (*Shev, E. E., and Robinson, S. J.: Electroencephalographic Findings Associated with Congenital Heart Disease, Electroencephalog. & Clin. Neurophysiol. 10: 253 (May) 1958.*)

XYLOCAINE Xylocaine is a member of a new group of anaesthetic agents. Its active principle gramine was isolated from grasses growing in central Asia. In 1946 xylocaine was synthesized by the Swedish chemists Lofgren and Lundquist. In the body it undergoes slow decomposition, mainly in the liver. It shows a greater affinity towards sensory nerve fibers than to motor or sympathetic nerves. Xylocaine lowers the arterial pressure and is an effective local anesthetic. Its main advantage over procaine is that anesthesia commences sooner and lasts longer. The maximum permissible dose of xylocaine is 0.2 Gm. In case of overdose oxygen should be administered and artificial respiration performed. To increase the arterial pressure, intravenous ephedrine is advised. The use of xylocaine as a local anesthetic by the method of creeping infiltration produces excellent analgesia. (*Bantsekina, M. M., and Kudryavtseva, A. M.: Anesthetic Properties of Xylocaine, Eksper. Khir. 5: 32, 1956.*)

SUBARACHNOID BLOCK Careful selection of patients with intractable pain, rigid technique of needle placement and neurological evaluation before and after blocks make subarachnoid alcohol blocks useful in malignant disease. Repeat blocks are necessary if the first block fails to eliminate the pain, if the pain is bilateral or if the block wears off. It is especially useful in patients with a short life expectancy and should be supplemented with tranquilizers or sedatives. (*Perese, D.: Subarachnoid Alcohol Block in Management of Pain of Malignant Disease, A. M. A. Arch. of Surg. 76: 347 (Mar.) 1958.*)

HEPARINIZED BLOOD The severe and occasionally fatal shock syndrome observed when citrated blood is used for exchange transfusion does not occur when heparinized blood is used straight from the donor. (*Valentine, G. H.: Exchange Transfusion in Newborn Using Heparinized Blood, Canad. M. A. J. 78: 977 (June 15) 1958.*)

OVERTRANSFUSION Circulatory overloading is now probably the most common cause of death from blood transfusion when proper methods are employed to prevent incompatibilities. Overtransfusion can be clinically noted by the appearance within an hour of dyspnea, orthopnea, intense cyanosis, blood-tinged frothy sputum, venous engorgement, sibilant and sonorous râles and often acute auricular fibrillation. Prophylaxis is the best treatment. Active treatment should be prompt and consist of (1) use of extremity tourniquets, (2) phlebotomy, (3) positive pressure oxygen therapy, (4) restriction of fluid intake. (*Downs, J. W.: Problem of Overtransfusion in Massive Hemorrhage, Ann. Surg. 148: 73 (July) 1958.*)

MASSIVE AIR EMBOLISM Massive air embolism (approximately 300 cc. within a few seconds) occurred in a patient momentarily left unattended during the administration of blood under positive pressure. Cardiac and respiratory arrest occurred within 15 seconds. Essential steps in the treatment of this catastrophe are: (1) Withdraw the infusion needle and discontinue the source of the air embolism. (2) Place the patient in Trendelenburg position to prevent air from entering the cerebral circulation in the event that arterial embolism may have occurred. (3) If time permits, place the patient in the left lateral position so that the air block (of the pulmonary valve) may be released. (4) If cardiac arrest occurs or the air embolism is too massive for the previous maneuver to succeed, perform emergency thoracotomy with needle aspiration of the right ventricle followed by cardiac massage. (5) Maintain a clear airway and provide effective artificial respiration with oxygen. (*Shires, T., and O'Banion, J.: Successful Treatment of Massive Air Embolism Producing Cardiac Arrest, J. A. M. A. 167: 1483 (July 19) 1958.*)

TRANSFUSION REACTION A total of 191 transfusions of 15–20 ml. of incompatible blood was administered. To 123 of these a ganglion-blocking agent was