ing cerebral venous obstruction (superior vena cava) is emphasized by the presentation of six case histories. The EEG is probably the most sensitive and rapid indicator of the onset of venous obstruction. Signs suggestive of cerebral hypoxia suggest superior vena caval obstruction and the surgeon should be so informed by the anesthesiologist so that the obstruction may be corrected. (*Paton, B., Pearcy, W. C., and Swan, H.: Importance of Electroencephalogram during Open Cardiac Surgery with Particular Reference to Superior Vena Caval Obstruction, Surg. Gynec. Obst. 111: 197 (Aug.) 1960.*)

CAROTID BLOOD FLOW Quantitative and directional flow in the carotid systems of man were studied by means of a non-cannulating electromagnetic flowmeter. Following occlusion of the common carotid artery, blood flowed from the external to the internal carotid artery in 50 per cent of the cases, but the reverse was observed in the remaining 50 per cent. Head position and occlusion of the opposite carotid artery were each shown to have a pronounced effect upon the flow through the internal carotid artery. Studies on the effect of contralateral percutaneous carotid compression suggest that in acute situations a compensatory mechanism exists whereby flow through one carotid artery increases when that through the other is diminished. (*Hardesty, W. H., and others: Studies of Carotid-Artery Blood Flow in Man, N. Engl. J. Med. 263: 944 (Nov.) 1960.*)

CEREBRAL VASO-DYNAMICS Observations made through a "window" in the skull have shown that anaemia through blood loss leads to dilatation of the arterial and constriction of the venous bed: ligation of arteries supplying the brain or acute impairment of respiration cause dilatation of both the arteries and veins on the surface of the brain. Stimulation of the sympathetic nerve is accompanied by a fall of intracranial CSF pressure and marked diminution of cerebral volume with simultaneous dilatation of the venous bed. Administration of aminophyllin causes dilatation of the superficial cerebral arteries and intracerebral capillaries, leading to improved blood supply to nerve cells; serpasil causes dilatation of both arteries and veins. Diagnosis of "cerebrovascular spasm" does not correspond to anatomical data since complete occlusion of even one intracerebral artery leads to death of the nerve cells and occlusion of large arteries produces focal necrosis. Transitory neurological signs taken to be the result of "cerebrovascular spasm" are most likely to be caused not by complete occlusion of the lumen but by strong constriction of the cerebral capillaries resulting from disorders of neuronal and humoral vasodilating innervation. (*Klosovskii, B. N.: General Problems of Pathology and Physiology of Cerebral circulation, Vestn. Akad. Med. Nauk SSSR 7: 3, 1959.*)

AORTIC COARCTATION Paradoxical postoperative hypertension immediately following resection for coarctation of the aorta is probably due to reflexes from aortic and carotid pressor receptors which were set at a high level preoperatively, and after surgery, with tension in the aortic wall reduced, may cease to act as buffers. Necrotizing arteritis is seen mainly in arteries below the site of coarctation and seems related to postoperative hemodynamic changes and the not infrequent delayed type of hypertension. (*March, H. W., Hultgren, H., and Gerbode, F.: Immediate and Remote Effects of Resection on Hypertension in Coarctation of Aorta, Brit. Heart J. 22: 361 (June) 1960.*)

LOCALIZED HYPOTHALAMIC STIMULATIONS By use of the Harsley-Clarke stereotaxic technique, a series of cats were subjected to hypothalamic electrical stimulation, chiefly in lateral and posterior positions. Electrocardiographic changes noted were marked alterations in QRS and T waves, bigeminal and trigeminal rhythms, A-V dissociation, extrasystoles, paroxysmal nodal and ventricular tachycardia, and the Wilson-Wolff-Parkinson-White configuration. (*Weinberg, S. J. and Fuster, J. M.: Electrocardiographic Changes Produced by Localized Hypothalamic Stimulations, Ann. Int. Med. 53: 332 (Aug.) 1960.*)

SHOCK With the aid of a newly devised technique employing a photoelectric drop-

meter, it is now possible to measure blood flow continuously and simultaneously in the cerebral, coronary, renal and hepatic circulation of dogs. Data revealed that each organ possesses its own characteristic response to shock and vasopressor drugs, that the circulation of the intestine as well as that of the kidney plays a vital role in circulatory homeostasis during acute shock and that the use of vasopressor drugs in shock increased the circulation to the brain and the heart while, at the same time, the blood flow to the intestine and kidney was decreased. The concepts of "mesenteric vascular insufficiency" and conclusions of clinical significance are discussed in detail. (*Corday, E., and Williams, J. H., Jr.: Effect of Shock and of Vasopressor Drugs on Regional Circulation of Brain, Heart, Kidney and Liver, Amer. J. Med. 29: 228 (Aug.) 1960.*)

ATROPINE The effect of 1.5 mg. of atropine sulfate intramuscularly upon blood pressure, pulse rate, respiration rate, pupil size, power of accommodation and perception was compared with that of the same dose of atropine sulfate given in combination with 5 mg. of metaraminol bitartrate (Aramine). Addition of metaraminol bitartrate resulted in a significantly higher mean systolic blood pressure, a larger pulse pressure and a slower heart rate during the hour following administration. Pupillary dilatation was also less. Visual perception was not significantly influenced. (*Taylor, W., and others: Use of Metaraminol Bitartrate to Reduce Side Effects of Atropine, Canad. M. A. J. 82: 1147 (June 4) 1960.*)

ATROPINE METABOLISM Two men were injected with a single 2-mg. dose of isotope labelled atropine. Between 85 and 88 per cent of the radioactivity was excreted in the urine within the first 24 hours. No activity was found in expired air, and only a trace could be extracted from feces. About half the dose appeared in the urine as intact atropine. More than a third was excreted as unknown metabolites, which appeared to be esters of tropic acid; less than 2 per cent appeared as free tropic acid. Neither hydroxylation of the aromatic ring nor glucuronide formation could be demonstrated. In man the ester bond is largely preserved and most if not all modifications in the molecule occurs in the tropine moiety. (*Gosselin, R. E., Gabourel, J. D., and Willis, J. H.: Fate of Atropine in Man, Clin. Pharmacol. Ther. 1: 597 (Sept.-Oct.) 1960.*)

NEUROMUSCULAR TRANSMISSION There are three types of neuromuscular block: depolarization, non-depolarization and dual. Recovery from dual blocks is slow unless aided by anticholinesterase therapy. When there is apnea at the end of an operation, the diagnosis of the cause can be made by using a peripheral nerve stimulator. Muscular paralysis can be differentiated from depression of the respiratory center. Observation of the hand muscles allows distinction between a depolarization and a dual type of neuromuscular block. (*Churchill-Davidson, H. C.: Review of Neuro-Muscular Transmission, Der Anaesthesist 9: 253 (Aug.) 1960.*)

MUSCLE RELAXANT PAIN The incidence of muscle pain and stiffness in 100 patients given suxethonium and suxemethonium was found to be the same for both drugs, namely, 25 per cent complained of severe stiffness or pain and an additional 45 per cent of mild stiffness. Stiffness was mostly in the neck, abdomen and chest. Onset of symptoms is from 12–24 hours postoperatively. Duration of symptoms is 4–5 days on the average. The incidence and severity of symptoms appears to vary with the dose of relaxant used. (*Parbrook, G. D., and Pierce, G. F. M.: Comparison of Post-Operative Pain and Stiffness After Use of Suxamethonium and Suxethonium Compounds, Brit. Med. J. 2: 579 (Aug. 20) 1960.*)

TUBOCURARE A quantitative study of the antagonism between acetylcholine and tubocurarine was made by taking depolarization of the end-plate skeletal muscle as a measure of drug-action. The results are consistent with the hypothesis that these substances compete on a one to one basis for receptors at the end-plate. Micro-electrodes were employed to show that individual end-plates do not vary greatly in their sensitivity