rest consequent to coronary and cerebral embolism. These pressure tracings confirm Kouwenhoven's observation that a pulse is transmitted to the limbs, but it is conceded that a pressure pulse is not necessarily proof of blood flow. That the blood flow was adequate is presumed from the patient's speedy recovery from the cerebral lesion. The need for a prepared mind in the successful treatment of cardiac arrest is re-emphasized. Five phases are listed: (1) Lower the head; raise the legs; thump the precordium. (2) Closed-chest cardiac massage; mouth-to-mouth lung insufflation. (3) Tracheal intubation and artificial respiration from an anesthetic rubber bag. (4) Record the electrocardiogram to determine the nature of the arrest. (5) Apply definitive treatment to the arrested heart. (*Nixon, P. G. F.: The Arterial Pulse in Successful Closed-Chest Cardiac Massage, Lancet 2: 844 (Oct. 14) 1961.*)

EXPIRED AIR RESUSCITATION Clinical observations on 1,500 patients showed that with mouth-to-mouth breathing the stomach was inflated as a rule. With mouth-to-nose breathing this occurred only in rare instances. Since regurgitation of gastric contents would introduce an additional complication during expired air, resuscitation mouth-to-nose breathing is preferred. (*Ruben, A.: Considérations sur la Respiration Artificielle d'Urgence, Acta Anaesth. Belg. 11: 298 (Dec.) 1960.*)

ARTERIAL OCCLUSION Short periods of occlusion of the vascular supply to the forearm in man produced an oxygen debt in both deep and superficial tissues. This oxygen debt was repaid by both increased blood flow and increased extraction of oxygen from blood during the period of reactive hyperemia following resumption of circulation. Results suggested an absence of delicately balanced and efficient checks on mechanisms governing repayment of the oxygen debt incurred during atrial occlusion. (*Abramson, D. I., and others: Effect of Short Periods of Atrial Occlusion on Blood Flow and Oxygen Uptake, J. Appl. Physiol. 16: 851 (Sept.) 1961.*)

MESENTERIC OCCLUSION Superior mesenteric arteries of dogs were clamped. All control dogs died; 25 per cent of dogs given epidural block lived. Late autopsies showed that the blocked dogs developed extensive collateral circulation. Epidural block was thought to relieve vasospasm and promote collateral circulation. (*Liang, H., Bernard, H. R., and Dodd, R. B.: Effect of Epidural Block upon Experimental Mesenteric Occlusion, A. M. A. Arch. Surg. 83: 409 (Sept.) 1961.*)

CEREBRAL CIRCULATION Cerebral blood flow remains remarkably constant in the face of broad changes in arterial blood pressure and cardiac output, and it is only when blood pressure is lowered to half or less of normal value or cardiac output decreased by more than a third that cerebral perfusion becomes inadequate. Within these broad limits cerebral blood flow is then regulated by *intrinsic* factors, principally cerebrovascular tone. This, in turn, depends chiefly on chemical influences, *viz.*, the respiratory gas content of the blood, and only slightly upon neurogenic influences. The most marked dilatation of cerebral vessels results from an increase in blood P_{CO_2}, and sharp vasoconstriction with a reduced cerebral blood flow is caused by a lowered blood P_{CO_2}. Moderate changes in blood P_{O_2} do not affect cerebral blood flow, but markedly lowered blood P_{O_2} will greatly increase cerebral blood flow and increased blood P_{O_2} will moderately reduce cerebral blood flow through vasoconstriction. Only a marked elevation of intracranial pressure will result in reduced cerebral blood flow. (*Shenkin, H. A., and Novack, Paul: Control of the Cerebral Circulation, J. A. M. A. 178: 390 (Oct. 28) 1961.*)

FIBRINOLYSIS The intravenous injection of some pressor amines (epinephrine, norepinephrine, and phenylephrine) in man resulted in fibrinolytic activity as measured by the euglobulin lysis technique and whole plasma lysis time. Such activity could be repeatedly recalled or maintained by constant infusion. (*Genton, E., and others: Fibrinolysis Induced by Pressor Amines, Amer. J. Med. 31: 564 (Oct.) 1961.*)

THROMBOLYSIS PREVENTION Certain operations have been known to produce

thrombolysis or fibrinolysis as a result of increased fibrinolysin activity and consequent hemorrhage, particularly following pulmonary, cardiac, pancreatic, and prostatic surgery. Administration of certain drugs or hormones also increase this tendency—epinephrine, vitamin K, nicotinic acid, PABA, and others. Lysis was determined by dissolution in a test tube of the organized clot over a period of 4 to 8 hours. In five patients who had a prostatectomy, use of fat emulsion intravenously resulted in subsequent control of hemorrhage and reduction of *in vitro* clot lysis. (*Biggs, A. W., and others: The Use of Fat Emulsion IV in Control of Hemorrhage Due to Thrombolysin Activity, South. Med. J. 54: 1252 (Nov.) 1961.*)

ANTICOAGULANT ANTAGONIST Coumarin anticoagulant therapy is frequently accompanied by the administration of other drugs, including barbiturates. Results obtained in guinea pig, dog and man indicate that pretreatment with barbiturates may antagonize the hypo-prothrombinemic effect of coumarin anticoagulants. This effect has been correlated with lower plasma levels of the anticoagulant drugs. In man, the route and timing of dosage significantly influence this barbiturate effect. It may be necessary to pay more attention to the mutual influence of concomitant therapy on the physiological disposition of drugs employed. (*Dayton, P. G., and others: Influence of Barbiturates on Coumarin Plasma Levels and Prothrombin Response, J. Clin. Invest. 40: 1797 (Oct.) 1961.*)

CATECHOLAMINES Catecholamines have a variety of metabolic effects. The one that has received the most emphasis is the stimulation of glycogenolysis as a consequence of the activation of glycogen phosphorylase in liver, muscle, myorcardium, intestinal smooth muscle, adipose tissue, and other tissues. The glycogenolytic effect of epinephrine results in a parallel increase in blood glucose and lactate. Other metabolic effects are hyperkalemia, inhibition of glucose uptake by tissues, mobilization of fat involving an increase in plasma free fatty acids, inhibition of the incorporation of amino acids into muscle protein, and inhibition of cholesterol and fatty acid synthesis. A beta-adrenergic antagonist (dichloroisoproterenol) was used to study the metabolic aspects of adrenergic blockade. DCI has previously been shown to prevent both epinephrine-induced augmentation of contractile force and activation of phosphorylase in the dog heart *in situ*. It was found that DCI almost completely abolishes the increase in blood glucose and free fatty acids produced by epinephrine, norepinephrine, and isoproterenol in the dog. The hyperlactacidemic effect of epinephrine is partly blocked. However, DCI does not block epinephrine-induced hyperglycemia in mice. (*Mayer, S., Moran, N. C., and Fain, J.: Effect of Adrenergic Blocking Agents on Some Metabolic Actions of Catecholamines, J. Pharmacol. Exp. Ther. 134: 18 (Oct.) 1961.*)

THIOBARBITURATES The extent to which the liver of humans or other species can carry out the reaction C=S yields C=O in the barbiturates remains somewhat uncertain. The reaction occurs with thiourea and many of its aryl derivatives. Thiourea is formed in small yield from thiopental metabolism along with a considerable yield of inorganic sulphur. This reaction may be of no more than minor importance in the metabolic disposition of thiobarbital. Barbital is well known to be stable in the animal body and to be excreted practically quantitatively in the urine, so that its quantitative determination is an uncomplicated indicator of the extent of desulfurization of its thioanalogue. It was found that barbital is excreted in the urine of man very slowly and in very small amounts after the intravenous injection of moderate doses of pure thiobarbital. The 5 to 7 per cent barbital yield and the 1 to 5 per cent thiobarbital excreted shows that some other metabolic reactions are responsible for the fate of about 90 per cent of the thio compound. (*Bush, M. T., Mazel, P., and Chambers, J.: Metabolic Fate of Thiobarbiturates: Thiobarbital in Man, J. Pharmacol. Exp. Ther. 134: 110 (Oct.) 1961.*)

ATROPINE Reflex parotid secretion and heart rate were measured in nine volunteers at intervals before and after injection of graded