

**CEREBRAL BLOOD FLOW** Passive hyperventilation of lightly anesthetized paralyzed human volunteers to a  $P_{aCO_2}$  of 19 mm Hg decreased cerebral blood flow from a control value of 44.1 ml/100 g/min during normocarbica to 25.3 ml/100 g/min, a 43 per cent reduction. Additional reduction of  $P_{aCO_2}$  to 10 mm Hg lowered CBF an additional 17 per cent to 21 ml/100 g/min. Two of six subjects attained minimum CBF at 19 mm Hg and failed to show further decreases on subsequent lowering of  $P_{aCO_2}$ . When  $P_{aCO_2}$  was maintained at 19 mm Hg and sufficient sodium bicarbonate was infused to raise arterial pH from 7.63 to 7.79, CBF increased 17 per cent despite a reduction in cerebral perfusing pressure. Metabolic alkalosis can exert a slight dilating effect on cerebral vessels. (Wollman, H., and others: *Effects of Extremes of Respiratory and Metabolic Alkalosis on Cerebral Blood Flow in Man*, *J. Appl. Physiol.* 24: 60 (Jan.) 1968.)

**MICROTHROMBOSIS** Circulatory collapse in man may result in systemic coagulopathies as manifested by microthrombi in the peripheral circulation. The incidence of disseminated intravascular coagulation after conditions of shock in man is not known. A study of the occurrence of microthrombi was done in 168 deaths, 112 deaths being associated with varying intervals of shock and 56 deaths occurring suddenly without shock and serving as controls. Microthrombosis was demonstrated in the kidneys, liver, lungs and heart at autopsy in 55 per cent of patients who had suffered from shock prior to death. Eighty six per cent of the cases were observed 24 to 48 hours after the onset of shock, but microthrombi could be demonstrated as early as four hours after the onset in 50 per cent of the cases. Patients who came to autopsy eight days after the onset of shock showed an incidence of only 38 per cent. The incidence of microthrombi was highest in patients with intra- and postoperative shock and severe infections and lowest in patients with shock due to myocardial infarction. One case of questionable intravascular thrombosis was observed in the control group. It was concluded that microthrombosis is a valuable criterion for the postmortem diagnosis of shock. (Remmele,

W., and Harms, D.: *The Pathological Anatomy of Circulatory Collapse in Man. I: Microthrombosis in the Peripheral Circulation*, *Klin. Wschr.* 46: 352 (April) 1968.)

**ABTRACTOR'S COMMENT:** Although no therapeutic implications were made, this paper could be quoted as one among many of recent origin that suggest the use of heparin in the treatment of shock and bleeding due to "consumption coagulopathy."

**ATRIAL FIBRILLATION** Thirty patients representing a "pure" group with idiopathic atrial fibrillation were observed. The onset of the arrhythmia often occurred after emotional or physical exhaustion, coughing, vomiting, standing erect, or overindulging in food and alcohol, suggesting that some reflex vagal activity, or an "excitatory factor," or both, may play a causal role. No patients in the series had congestive heart failure, coronary insufficiency, embolic phenomena, or required reversion to sinus rhythm by electrical means. Bouts of atrial fibrillation in these individuals without known heart disease are probably functional and benign. The occurrence of cardiac signs or symptoms out of proportion to those due to the arrhythmia itself suggests that underlying heart disease may be present and that the patient may not have idiopathic atrial fibrillation. (Peter, R. H., Gracey, J. G., and Beach, T. B.: *A Clinical Profile of Idiopathic Atrial Fibrillation*, *Ann. Int. Med.* 68: 1288 (June) 1968.)

**ACIDOSIS AND DYSRHYTHMIAS** The relationship between metabolic acidosis and cardiac dysrhythmias was studied in 21 patients with acute myocardial infarction, established on clinical, electrocardiographic and biochemical evidence. Arterial blood gases and pH were determined on admission, 24 hours later, and at the onset of any cardiac dysrhythmia. Blood pressure measurements were made and noted at corresponding intervals. There was a close association between metabolic acidosis and hypotension. In addition, metabolic acidosis was associated with a poor early prognosis, and its incidence rose with an increasing prognostic index. Therefore, the metabolic acidosis reflected the severity of the

infarct. Patients with metabolic acidosis were predisposed to cardiac dysrhythmias. However, this was considered to be related to the greater severity of their illness rather than the direct result of acidosis, since correction of the acidosis did not correct the dysrhythmia in spite of improved general patient condition. (Anderson, R., and others: *Relation Between Metabolic Acidosis and Cardiac Dysrhythmias in Acute Myocardial Infarction*, *Brit. Heart J.* 30: 493 (July) 1968.)

**INTRAVENOUS LIDOCAINE** In eight patients who were not in shock and did not have complete heart block or significant arrhythmia, but who had had moderate or severe myocardial infarction in the preceding 24 hours (two later died), intravenous injection of 100 mg lidocaine over a five-minute period had no significant effect on cardiac output, heart rate, systemic blood pressure or pulmonary artery pressure. (Stannard, M., and others: *Haemodynamic Effects of Lignocaine in Acute Myocardial Infarction*, *Brit. Med. J.* 1: 468 (May) 1968.)

**LEFT VENTRICULAR FUNCTION** Left ventricular function was studied in seven dogs following establishment of a large systemic arteriovenous fistula by means of a side-to-side infrarenal aortocaval anastomosis. The mechanical properties of left ventricular contraction were evaluated in terms of tension, velocity, length and time, and the results were compared with those obtained in a group of normal dogs studied previously. Both groups were sedated with morphine, promazine and promethazine at the time of study. As would be expected, dogs with A-V fistulas had high left ventricular end-diastolic pressures, high ejection fractions (stroke volume divided by end-diastolic volume), circulatory congestion and marked fluid retention. All dogs developed moderate ventricular hypertrophy even though left ventricular contractility was depressed below the normal range in only one of the seven. Mechanisms for fluid retention resulting in circulatory congestion were activated because of the large hemodynamic burden in spite of normal myocardial contractile properties. (Taylor, R. R., Covell, J. W., and

Ross, J., Jr., *Left Ventricular Function in Experimental Aorto-Caval Fistula with Circulatory Congestion and Fluid Retention*, *J. Clin. Invest.* 47: 1333 (June) 1968.)

#### MYOCARDIAL REVASCULARIZATION

Change in concepts of anesthesia for myocardial revascularization has been a factor in improved surgical results. The change from "light anesthesia" to relatively deep and steady levels of anesthesia using methoxyflurane, coupled with long-acting muscle relaxants, virtually eliminates cardiac arrhythmias and allows the surgeon to manipulate the heart with little effect upon basic cardiac rhythm. As a result, operating room deaths, particularly during the implant procedure, do not occur. Vasodilators (nitrites) are used routinely throughout the operative and postoperative period. Hyperactivity of patients during the critical awakening period from anesthesia predisposes to ventricular fibrillation. Heavy sedation is therefore recommended in the early postoperative period. Respirations are mechanically assisted to prevent hypoxia and hypercarbia. Endotracheal tubes are often left for 24 hours or longer following surgery. A change from an abrupt awakening to a long controlled return to consciousness seems to have reduced appreciably the stressful factors that provoke undesired coronary spasm. (Effler, D. B.: *Anesthesia in Revascularization Surgery—A New Advance* (Editorial), *J. Thorac. Cardio. Surg.* 56: 163 (Aug.) 1968.)

ABSTRACTER'S COMMENT: Though Dr. Effler's statements are based on extensive clinical experience, we know of no objective studies to indicate that light anesthesia, *per se*, predisposes the heart to arrhythmias. Relatively deep anesthesia with methoxyflurane or other potent anesthetic agents has definite, well-documented myocardial depressant effects, particularly in the presence of myocardial disease. The addition of vasodilators like nitrites can precipitate hypotension and compromise coronary circulation. There is some merit to gradual awakening and postoperative assistance of ventilation, though we fail to see why this should be true following revascularization procedures more so than following other types of cardiac surgery.