CEREBRAL BLOOD FLOW Passive hyperventilation of lightly anesthetized paralyzed human volunteers to a Pa_{CO_2} of 19 mm Hg decreased cerebral blood flow from a control value of 44.1 ml/100 g/min during normocarbia to 25.3 ml/100 g/min, a 43 per cent reduction. Additional reduction of Pa_{CO_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 Pa_{CO_2}. When Pa_{CO_2} was maintained at 19 mm Hg and sufficient sodium bicarbonate was infused to raise arterial *p*H 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.*)

ABSTRACTOR'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 *p*H 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