arrest in 37 patients with heart disease of whom half had undergone cardiac surgery. Of the 13 patients who left the hospital, the majority were in cardiac arrest for 15 minutes or less and had only a single episode. No patients who were in advanced heart failure before cardiac arrest survived. A high proportion of patients were hypoxemic during and after the cardiac arrest despite artificial ventilation with pure oxygen. There was a wide variation in acid-base status during and after the cardiac arrest which in three cases was corrected satisfactorily by administering a dose of sodium bicarbonate, in milliequivalents, which was equal to the weight of the patient in kilograms multiplied by one-tenth of the duration of the cardiac arrest in minutes. The state of the central nervous system provided no guide to the degree of acid-base disturbance. Experience showed the value of continuing resuscitation for at least an hour when there were satisfactory signs of brain activity. Five main criteria were used to assess the status of the central nervous system: pupil size, blink reflex, respiratory pattern, degree of struggling, and state of muscle tone, especially in the jaw. (*Gilston, A.: Clinical and Biochemical Aspects of Cardiac Resuscitation, Lancet 2: 1039 (Nov.) 1965.*)

PHYSIOLOGIC SHUNT After the administration of 100 per cent oxygen, blood samples simultaneously drawn from the left atrium and the aorta of subjects who had undergone open-heart procedures were analyzed for P_{O_2}, P_{CO_2} and *p*H. Using a modification of the shunt equation, the contribution of the Thebesian veins to the physiologic shunt was found to be 0.12 to 0.43 per cent of the aortic flow. (*Ravin, M. B., Epstein, R., and Malm, J. R.: Contribution of the Thebesian Veins to the Physiologic Shunt in Anesthetized Man, Bull. N. Y. Acad. Med. 42: 328 (April) 1966.*)

PULMONARY SHUNTING Eighteen anesthetized patients undergoing nonthoracic surgical procedures were mechanically hyperventilated with gas mixtures containing 40 or 99 per cent oxygen. When arterial carbon dioxide tension was increased toward normal levels by adding carbon dioxide to inspired gas and then reduced with CO_2 free mixtures, arterial oxygen tension usually increased and decreased concomittantly. Similar changes in carbon dioxide levels were induced in 11 other patients in whom the effect on cardiac output was measured and the extent of the right-left pulmonary shunting was estimated. With initial hypocapnia, shunting exceeded 8 per cent. When cardiac output was unchanged with increased carbon dioxide levels, the increase in arterial oxygen tension reflected decrease of shunting. With change in cardiac output, there was a similarly direct change in shunting, interacting with and at times overriding the apparent "direct" effect of carbon dioxide. In 4 patients, observations at inspired oxygen tensions of 40 per cent were followed by similar observations at 99 per cent. In each case a significant increase in shunting occurred with increase in inhaled oxygen tension without change in carbon dioxide levels or cardiac output. Blood flow through nonventilated alveoli decreases at approximately 1 hour. This is interpreted as a vasocontrictor effect. (*Michenfelder, J. D., Theye, R. A.: CO_2 Levels and Pulmonary Shunting in Anesthetized Man, Fed. Proc. 25: 293 (March) 1966.*)

PULMONARY VASCULAR RESISTANCE Pulmonary vascular resistance in ventilated and collapsed lungs was measured in cats. When a lung was collapsed by occluding the main stem bronchus, blood flow through the collapsed lung was reduced by 35 per cent. This was accompanied by a large increase in pulmonary vascular resistance. These changes occurred whether or not the lung was denervated indicating independence from nervous control. Hypoxia increased pulmonary vascular resistance in both ventilated and collapsed lungs. Catecholamines, 5-HT and histamine produced a similar but less marked increase. Alpha-adrenergic blocking agents markedly reduced the response to a lesser extent. Lysergic acid diethylamide (LSD) and mepyramine blocked the response to 5-hydroxytryptamine and histamine, respectively, but had little effect on the response to hypoxia. These data indicate that a local release of catecholamines (norepinephrine pri-