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 vasoconstrictor effect. (*Michenfelder, J. D., Theye, R. A.: $CO_2$ Levels and Pulmonary Shunting in Anesthetized Man, Fed. Proc. 25: 253 (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 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-

marily) may mediate the increase in pulmonary vascular resistance and the decrease in blood flow which occurs after collapse of a lung secondary to obstruction and hypoxia. (*Barer, G. R.: Reactivity of the Vessels of Collapsed and Ventilated Lungs to Drugs and Hypoxia, Circ. Res. 28: 366 (April) 1966.*)

**CAROTID BODY** The carotid bifurcation of cats was perfused at constant pressures with bicarbonate buffered Ringer's solution containing dextran. The response of the chemoreceptors to abrupt changes in *p*H and/or $P_{CO_2}$ was measured as the change in impulse frequency recorded from Hering's nerve. An increase in $P_{CO_2}$ of 30 mm. of mercury consistently resulted in a carotid body response only when accompanied by a decrease in *p*H. In some instances in which there was a response to an increase in $P_{CO_2}$ at constant *p*H, the receptor discharge returned rapidly to control values despite maintained stimulus of constant intensity. When *p*H was decreased from 7.4 to 7.1 through either a reduction in bicarbonate ion or an increase in $P_{CO_2}$, the responses were of the same magnitude. However, the response to acidification by $CO_2$ occurred more rapidly than that to bicarbonate ion reduction. The data suggest that the receptor is responsive to and in equilibrium with extracellular *p*H but separated from the vascular space by a diffusion barrier more permeable to carbon dioxide than to hydrogen ion. (*Gray, B. A., Munroe, A. B., and Tenney, S. M.: Response of the Perfused Carotid Body to Changes in pH and $P_{CO_2}$, Fed. Proc. 25: 264 (March) 1966.*)

**CEREBRAL BLOOD FLOW** Man living at high altitude, when made normoxic, has normal cerebral blood flow (CBF) and cerebrospinal fluid (CSF) *p*H, despite low $Pa_{CO_2}$ and high *p*H. Alkalemia does not influence CBF and CSF *p*H at constant $Pa_{CO_2}$. Thus, carbon dioxide may control CBF via *p*H in extracellular fluid (ECF) where bicarbonate ion ($HCO_3^-$) similar to that of CSF is subject to the *p*H regulating activity of the blood-CSF barrier. Measurement of CBF during step hyperventilation was made by computing CBF as per cent of control from the change in A-V oxygen saturation, utilizing internal jugular vein blood. Volunteers were taught to reduce their end-tidal $P_{CO_2}$, in 3 seconds to 23 mm. of mercury and to hold it constant for 2 hours. $Pa_{CO_2}$ fell from 41 to 25 in about ½ minute and remained constant while $Pv_{CO_2}$, taken as an index of tissue $P_{CO_2}$, fell slowly from 50 to 36. It is concluded that $Pa_{CO_2}$ controls CBF by means of its effect on *p*H of arteriolar smooth muscle ECF, and that this ECF *p*H, like CSF, can be reset to normal in chronic hypocapnia by transport across the blood cerebrospinal fluid barrier. (*Severinghaus, J. W.: Site and Mode of Action of $CO_2$ on Cerebral Blood Flow in Man, Fed. Proc. 25: 461 (March) 1966.*)

**CEREBRAL BLOOD FLOW** Experimental review led to the following conclusions concerning cerebral blood flow: (1) Cervical sympathectomy produces no significant increase of cerebral blood flow in normal dogs or in dogs with bilateral ligation of the internal carotid arteries. (2) In dogs, and in humans with obstructed carotid arteries, inhalation of 5 per cent carbon dioxide increases cerebral blood flow about 40 per cent during the period of inhalation and for about 30 minutes thereafter. (3) Administration of papaverine increases cerebral blood flow significantly in dogs, but to a far lesser degree than carbon dioxide inhalation. (4) Hypothermia decreases cerebral blood flow markedly by chemical changes resulting from reduced metabolism. (5) Circulation of blood in small cerebral vessels is under chemical rather than neurogenic control. These findings suggest the careful trial of carbon dioxide inhalation in the treatment of patients with strokes caused by partial obstruction of the cerebral arterial blood supply, providing that the patient is observed closely for evidence of carbon dioxide intoxication. Intermittent inhalations of carbon dioxide in air may beneficially supplement extracranial cerebral arterial surgery or, if operation is impossible, may have a beneficial effect when used alone. (*Shackelford, R., and Hegedus, S.: Factors Affecting Cerebral Blood Flow, Experimental Review: Sympathectomy, Hypothermia, $CO_2$ Inhala-*