the same conditions, induction of hypothermia to 18° C. produced similar changes but caused myocardial hemorrhage and ventricular fibrillation. Profound hypothermia probably leads to excessive fragility of the capillary bed. (*Hirose, T., and Madden, R. W.: Optimal Coronary Flow in the Bypassed Normothermic and Hypothermic Heart, J. Thor. Cardiov. Surg. 48: 82 (July) 1964.*)

CEREBRAL BLOOD FLOW Cerebral tissue is divided into cylinders of tissue each of which is supplied by one capillary. Since oxygen is being continuously utilized in the tissue cylinder, Po_2 falls toward the outer end of the cylinder. Po_2 also decreases in the course of the capillary from the arterial to the venous end. Therefore, the lowest Po_2 is found at the periphery of the cylinder at the venous end. With a Po_2 of 34 mm. of mercury in the blood at the venous end of the capillary, the tissue Po_2 is 17 mm. of mercury. Hyperventilation, lowering of blood pressure, increase in venous or intracranial pressure reduce cerebral blood flow, whereas hypoventilation increases cerebral blood flow. Hyperventilation was never found to lower the Po_2 to subcritical levels whereas hypoventilation, arterial hypotension or an increase in intracranial pressure easily reduced P_{O_2} to subcritical levels. Apart from these generalized forms of oxygen lack, aggregates consisting mainly of thrombocytes were observed to cause local reduction or stasis of blood flow. (*Hirsch, H.: Blood Flow and P_{O_2} of the Brain, Der Anaesthesist 13: 117 (April) 1964.*)

VASOCONSTRICTOR DRUGS Patients with arterial hypertension have an increased sensitivity to vasoconstrictor drugs. Angiotensin 0.1 μg. in 0.2 ml. saline when given intracutaneously caused a blanched area which persisted 145 minutes in normal patients, 169 minutes in patients with variable hypertension and 197 minutes in patients with fixed hypertension. Similar results occurred after 0.8 μg. noradrenalin. (*Pippig, L.: Angiotensin Skin Test, Z. Kreislaufforsch 53: 587 (June) 1964.*)

HYPERTENSION On the hypothesis that an abnormal rise of blood pressure during general anesthesia is due to an adrenal-sympathetic reaction, phentolamine (Regitine) was administered to 10 patients whose blood pressure had risen markedly or rapidly. In all 10, the blood pressure fell to the preoperative value without the development of cardiac irregularities or tachycardia or any other side effects. (*Sato, M., and Inagaki, M.: Effect of Phentolamine on Hypertension During General Anaesthesia, Japanese J. Anaesth. 12: 347 1964.*)

ACIDOSIS IN SHOCK Metabolic acidosis of early shock is largely due to lactate ion. High blood lactate levels early in shock are indicative of poor prognosis. (*Peretz, D. I., and others: Lacticacidosis: A Clinically Significant Aspect of Shock, Canad. Med. Ass. J. 90: 673 (Mar. 14) 1964.*)

WARMING OF BLOOD High frequency waves warm one unit of blood from refrigerator to body temperature in 3 minutes. Life span of erythrocytes and blood chemistry were shown to be practically unaffected by the warming. Thus the blood is warmed in the shortest possible time preventing waste of blood that had been warmed and then was not needed and also practically eliminating the danger of bacterial growth in the blood. (*Freysz, T., Schwarz, H., and Hossli, G.: New Gadget for Fast Rewarming of Blood Bottles, Der Anaesthesist 13: 174 (May) 1964.*)

RESPIRATORY ACIDOSIS Hemodynamic changes in systemic and pulmonary circulation were examined in dogs in extreme respiratory acidosis and after partial correction by infusion of sodium bicarbonate. Cardiac output remained normal when Pco_2 reached 400 mm. of mercury and *p*H fell to 6.6. Systemic blood pressure remained unchanged but pulmonary blood pressure rose by approximately 100 per cent. Not before carbon dioxide tensions of 600 mm. of mercury and a *p*H of 6.4 was reached did cardiac output fall. Systemic blood pressure decreased by more than 50 per cent and the pulmonary vascular resistance rose by approximately 200 per cent. Infusions of 200 ml. 1 molar sodium bicarbonate resulted in a sustained rise of blood pressure by approximately 33 per cent and an increase of cardiac output by 100 per cent