laboratory estimations of arterial blood and pH and P_{CO_2}. During drug-induced paralysis, it may be desirable to sedate a patient on IPPR, but only the lightest sedation, and sometimes none at all is required to maintain unconsciousness and amnesia. The weaning of a patient from respiratory care may be prolonged; the transition from tracheostomy tube to normal breathing being made with the intermediate use of a fenestrated metal tube to reintroduce gradually the natural deadspace. (*Walsh, R. S.: Management of Patients in a Respiratory Unit, Proc. Roy. Soc. Med. 54: 799 (Sept.) 1961.*)

INFANT RESUSCITATOR A Kreiselman apparatus for resuscitation of the newborn infant was found effective in initiating respiration in some depressed infants when the pressure relief indicator beside the water column was set to a limit of ten millimeters of mercury. A check was made of the pressure flow calibrations of the resuscitators. While the resuscitator was theoretically set at a predetermined pressure limit of 13.6 centimeters of water, the maximum pressures actually varied from 13.6 to 52 centimeters of water. The higher pressures thus applied account for the inflation of the unexpanded infant lung. Emphasis is directed toward limiting the use of high pressures for periods of less than two minutes, in order to avoid rupture of the aerated lung. (*Hustead, R. F., and Avery, M. E.: Observations on Mass Pressure Achieved With the Kreiselman Infant Resuscitator, New Engl. J. Med., 265: 939, (Nov. 9) 1961:*).

PULMONARY EMBOLISM Experimental pulmonary embolism was studied in sheep following the intravenous administration of graded amounts of barium sulfate emulsion. Ventilation, lung mechanics and circulation were measured. The effect of various neuroplegic procedures, oxygen breathing, the administration of antihistaminic and antiserotonin drugs, and continuous epinephrine and isoproterenol infusions was assessed. In addition to hyperventilation, arterial hypoxemia, pulmonary hypertension and bronchoconstriction, a gross fall in lung compliance was shown to occur, unrelated to pulmonary edema. All but one of the procedures were ineffective in altering the onset or severity of these changes. The effect of smaller doses of embolic material was completely prevented by the administration of isoproterenol. It is concluded that postembolic pulmonary hypertension and compliance-fall after a small dose of embolic material are predominantly functional and probably caused by the release of an unknown substance as a response to embolism. (*Halmagyi, D. F. J., and Colebatch, H. J. H.: Cardiorespiratory Effects of Experimental Lung Embolism, J. Clin. Invest. 40: 1785 (Sept.) 1961.*)

PULMONARY EMBOLISM Experimental pulmonary embolization confined to one lung or one lobe of a lung was produced in dogs and the distribution of the embolizing glass beads was verified at autopsy. In all experiments respiratory rate increased and tidal volume decreased following embolization. This response was abolished by cervical vagotomy but not by inhalation of 100 per cent oxygen. It was concluded that the magnitude of the ventilatory response to pulmonary embolization is determined by the number of emboli injected and is independent of the site at which emboli lodge or the degree of their concentration or dispersion. The response is probably not initiated by hemodynamic changes incident to embolization. (*Horres, A. D., and Bernthal, T.: Localized Multiple Minute Pulmonary Embolism and Breathing, J. Appl. Physiol. 16: 842 (Sept.) 1961.*)

PULMONARY EDEMA Hexamethonium 25 mg. was administered intramuscularly to 6 patients with pulmonary edema. In all patients, a pronounced therapeutic effect was noted within 20 to 30 minutes after the injection of the preparation; the improvement consisted of a considerable decrease or complete disappearance of dyspnea, orthopnea and a decrease of moist rales in the lungs. (*Kheideman, K. K.: Hexamethonium in Treatment of Pulmonary Oedema, Klin. Med. (Moskva) 10: 95, 1960.*)

CORONARY THROMBOSIS A counter pulsating pump connected to the arterial sys-

tem is used to minimize ischemic loss of heart muscle during a heart attack. A tube is inserted into one of the patient's leg arteries and passed into the abdominal aorta. The pump, synchronized to the heartbeat with an electrocardiograph, withdraws blood from the aorta during cardiac systole, thus lowering systolic pressure and diminishing the work load of the heart. The blood is then rapidly returned to the aorta during diastole, thus raising the diastolic coronary perfusion pressure as well as maintaining systemic pressure. The pump's action forces open some of the vascular 'detours' in the heart, nourishing the area that is being deprived of blood by the blockage of a main vessel. If started soon enough—within three hours after the patient is stricken—the pump, which has thus far been used satisfactorily on five human patients as well as on animals, can restore a myocardial blood supply before damage becomes irreversible. (*Medical News: Counterpulsating Pump Described at Forty-seventh College of Surgeons Congress, J. A. M. A. 178: 26 (Oct. 28) 1961.*)

MYOCARDIAL AUGMENTATION A new method has been developed in dogs to provide mechanical assistance in the management of central circulatory insufficiency. Postsystolic myocardial pressure was augmented with the aid of an electronic circuit and the QRS complex of the electrocardiogram. Normal circulatory dynamics were approximated by this method in dogs with experimentally induced dilated hearts and circulatory hypotension. (*Watkins, D. H., and Duchesne, E. R.: Postsystolic Myocardial Augmentation: I. Effect Upon an Induced Shock State, Proc. Soc. Exp. Biol. Med. 107: 659 (July) 1961.*)

SHOCK Intravascular coagulation results from the intra-aortic injection of E. coli endotoxin in dogs. This is indicated by (1) a decrease of blood clotting factors, (2) presence of thrombi in tissue sections, (3) finding of focal necrosis and infarcts, and (4) prevention of these findings by preheparinization. The immediate (reversible) fall in blood pressure after endotoxin injection is caused by a decrease in cardiac output. This is due to a decreased venous return to the left heart caused by a cor pulmonale and associated vasospasm following blockage of pulmonary capillaries by thrombi. It is also due to a decreased venous return to the right heart by the damaging effect of thrombi in the portal system. The secondary (irreversible) fall in blood pressure and death appears to be greatly assisted by loss of blood and serum into the bowel lumen, secondary to the necrosis and sloughing of the superficial gastrointestinal mucosa. (*Hardaway, R. M., and others: Endotoxin Shock. A Manifestation of Intravascular Coagulation, Ann. Surg. 154: 791 (Nov.) 1961.*)

SHOCK Serum level serotonin in dogs falls to 22 per cent of normal values during lethal endotoxin shock. Plasma catecholamine concentrations rise after endotoxin administration. Epinephrine returns to normal levels but norepinephrine appears slightly elevated. The release of histamine, serotonin, epinephrine, and norepinephrine cause hemodynamic changes which lead to endotoxin shock. It is believed that a temporary depletion of peripheral catecholamine store occurs resulting in vascular instability, vasodilatation, pooling, decreased cardiac output, and shock. After hemorrhagic hypothermia, peripheral venous levels of endogenous epinephrine increases 90 fold over control values. (*Rosenberg, J. C., and others: Studies on Hemorrhagic and Endotoxin Shock in Relation to Vasomotor Changes and Endogenous Circulating Epinephrine, Norepinephrine and Serotonin, Ann. Surg. 154: 611 (Oct.) 1961.*)

SHOCK Prevention of serious effects from hemorrhage is best accomplished by early and adequate blood transfusion. Experiments on dogs in severe shock from bleeding show that hydrocortisone and adrenergic blocking agents promote survival, but that noradrenaline does not. (*Hakstian, R. W., Hampson, L. G., and Gurd, F. N.: Pharmacological Agents in Experimental Hemorrhagic Shock, A. M. A. Arch. Surg. 83: 335 (Sept.) 1961.*)

ADJUSTMENTS TO HEMORRHAGE By analyzing the volume-flow-pressure relationship in sympathectomized dogs, the various compensatory adjustments to hemorrhage