(where the thorax is in the normal mid inspiratory position at rest), with chronic laryngeal or bronchial obstruction, and with bronchiectasis and other states, many others are not secondary to air flow obstruction, but may be due to the deep penetration of harmful dusts causing air sac dissolution. There is an associated reduction in arterial and arteriolar vessels, advanced cases having a 70 to 80 per cent reduction in normal vascular architecture. Right ventricular hypertrophy occurs, its severity being correlated with the extent of the disease except in gibbous deformities where it may be associated with vascular root distortion rather than with vascular occlusion as a result of the distension-dissolution process. Resistance to air flow in these lungs was from three to ten times normal and the average flow rate about one-half normal. (*Wyatt, J. P., and Sweet, H: The Morphogenesis of Panlobular Emphysema, Amer. Rev. Resp. Dis. 83: 426 (Mar.) 1961.*)

COR PULMONALE The most common cause of cor pulmonale is emphysema, though many cases of tuberculosis and bronchiectasis which would formerly have died of infection now develop it. In pulmonary embolism, myocardial infarction and valvular heart disease, mortality is more than twice as high in patients with emphysema. Cor pulmonale may be secondary to both hypoxia and pulmonary hypertension. The hypertension is much more marked in the latter group, though it is present in the other. Arterial oxygen saturation is always below normal in cor pulmonale, and if below 85 per cent and accompanied by marked polycythemia and carbon dioxide retention, probably indicates a primary pulmonary etiology of right heart failure. Electrocardiogram and roentgenograms may not aid in the diagnosis. During exacerbations of emphysema with bronchitis, renal ischemia also may occur. Long term treatment of emphysematous patients with acetazolamide may be of benefit partly by reducing hypercapnia and partly by aiding diuresis. Digitalis, diuretics, specifically indicated antibiotics, tracheal suction, tracheostomy, aerosolized bronchodilators and the cautious use of oxygen with or without mechanical assistance to ventilation, avoidance of drugs depressant to respiration, and small repeated phlebotomies all can contribute to the treatment of cor pulmonale. (*Muschenhein, C.: The Growing Importance of Pulmonary Heart Disease as a Cause of Congestive Cardiac Failure, Amer. Rev. Resp. Dis. 83: 475. (Apr.) 1961.*)

RESPIRATORY ACIDOSIS Administration of Ringer's lactate solution to dogs during acute respiratory acidosis results in a greater ion secretion by the kidney and, therefore, greater conservation of bicarbonate bound base than after administration of 5 per cent glucose and water. Surgical trauma superimposed on the acute respiratory acidosis resulted in no further changes in this response. (*Hutchin, P., McLaughlin, J. S., and Hayes, M. A.: Renal Response to Acidosis During Anesthesia and Operation: III. Maintenance of Homeostasis in Acute Respiratory Acidosis During Intravenous Infusion of Ringer's Lactate and 5 per cent Glucose in Water, Ann. Surg. 154: 161 (Aug.) 1961.*)

ASPIRATION Prior to induction of ether anesthesia, 150 patients were given 1 ml. of methylene blue and instructed to distribute this evenly within the oral cavity. One hundred patients were given open drop ethyl chloride-ether anesthesia; 50 received ether-nitrous oxide-oxygen anesthesia using endotracheal intubation. Following surgery, bronchoscopic examination was performed. Twenty-seven per cent of the patients who had received open drop ether anesthesia and 16 per cent of those who had their trachea intubated showed aspiration. (*Klimpel, L.: Bronchoscopic Examinations for Aspiration following Narcosis, Der Anaesthesist 10: 310. (Oct.) 1961.*)

RESPIRATORY UNIT Respiratory care is necessary in a variety of illnesses in which respiratory failure is a transient but potentially lethal episode. Tracheostomy should usually be performed early. A cuffed rubber tube is used, care being taken not to over-inflate the cuff. Humidification is necessary. The control of IPPR is based on clinical observation of the patient's condition, helped by measuring the respiratory volume. In cases of doubt it is helpful but not essential to have

laboratory estimations of arterial blood and *p*H 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. J.: 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 counterpulsating pump connected to the arterial sys-