SHOCK A strain gauge sewed to the heart gives an accurate and reproducible measurement of contractile force and reflects a summation of the influences on cardiac physiology. During hemorrhagic shock, contractile force is a more stable parameter than blood pressure. When contractile force decreases markedly, death is imminent and treatment is ineffectual. Partial replacement of blood volume was not as effective in treatment as partial blood replacement plus levarterenol. (*Cooley, J. C., and McIntosh, C. L.: Myocardial Contractile Force in Experimental Hemorrhagic Shock, Arch. Surg. 87: 330 (Aug.) 1963.*)

SHOCK Tissue hypoxia and metabolic acidosis play an important part in the development of cardiac and circulatory insufficiency of burns and shock. Sodium bicarbonate and THAM given early cause elevation of blood pressure, return of effectiveness of pressor amines, decrease in pyruvic and lactic acid, and increased output of urine. Base excess should be raised to 5 or 7 mEq./liter but not over 10. Irreversible shock develops when oxygen debt reaches 100 ml./kg. Patients with burns were treated with fluids as follows: one-fourth Rheomacrodex, one-fourth sodium bicarbonate, one-fourth electrolyte and glucose and one-fourth plasma with THAM and Trasylol. (*Krauss, H., Koslowski, L., and Zimmerman, W. E.: New Results in Metabolic Changes in Burns and in Shock, Langenbecks Arch. Klin. Chir. 303: 23 (June) 1963.*)

PULMONARY CIRCULATION Atropine increases cardiac index, decreases right atrial pressure and pulmonary artery pressure in normal supine subjects. Changes in pulmonary blood volume, estimated by external I^{131} counting technique, suggest that atropine causes a redistribution of the blood volume away from the lung. With the decrease in pulmonary vascular pressures and volume, pulmonary diffusing capacity and pulmonary capillary volume decrease, whereas lung compliance increases. These changes are not the result of altered distribution of ventilation or changes in lung volume. Acute pulmonary vascular engorgement produced by the G-suit inflation increases right atrial pressure, pulmonary artery pressure, pulmonary diffusing capacity and pulmonary capillary volume and decreases lung compliance. Similar, though smaller, changes are produced by G-suit inflation after atropine. Continuous positive-pressure breathing, procedure known to move blood out of the chest, decreases pulmonary diffusing capacity and pulmonary capillary volume. These findings suggest that atropine causes a shift of blood out of the lungs into an area where it is not effectively mobilized by G-suit inflation. A specific effect of atropine on pulmonary vasculature cannot be excluded. The study demonstrates the dependence of pulmonary diffusing capacity and pulmonary capillary volume on pulmonary vascular pressures, or volume, or both, and their relative independence of cardiac output. (*Daly, W. J., Ross, J. C., and Behnke, R. H.: Effect of Changes in the Pulmonary Vascular Bed Produced by Atropine, Pulmonary Engorgement, and Positive-Pressure Breathing on Diffusing and Mechanical Properties of the Lung, J. Clin. Invest. 42: 1083 (July) 1963.*)

PULMONARY VENTILATION In 1,317 men, aged 40–65 years, in apparent good health the forced expiratory volume, the one second forced expiratory volume and the maximal mid-expiratory volume and the maximal mid-expiratory flow were obtained. All three measurements fell with age and were adversely affected by bronchitis and cigarette smoking while only the one second forced expiratory volume was significantly lowered in patients with silicosis. (*Brinkman, G. L., and Coates, E. O.: One Effect of Bronchitis, Smoking and Occupation on Ventilation, Amer. Rev. Resp. Dis. 87: 684 (May) 1963.*)

ATELECTASIS Saline extractions of atelectatic portion of human lungs as compared to those from normal portions of lungs showed a decrease in surface activity as determined by the use of a modified Wilhelmy balance. Experimental atelectasis in dogs, followed by saline extractions and determination of surface activity confirmed the clinical findings. Adult human atelectasis is basically a mechanical phenomenon with secondary deficiency of an "anti-atelectasis" surfactant. (*Sutnick, A. I., and Soloff, L. A.: Surface Tension Reducing Activity in the Normal and Atelec-*