Neostigmine for Controlled Cardioplegia, J. Thoracic Surg. 37: 655 (May) 1959.)

EXTRACORPOREAL PROBLEMS Many problems occur in extracorporeal circulation relating to venous cannulation and drainage, many of which have been satisfactorily resolved. Siphon caval drainage is preferred to fixed venous pumping. Hepatic venous drainage should not be interfered with. Venous catheters should be accurately and securely placed with attached metal tips. The vena cavae should not be intubated until just before the start of the perfusion to avoid interference with cardiac output. Lethal air embolism in the right atrium may occur. Its mechanism and prevention is outlined. Oxygen consumption in experimental studies is increased slightly by elevation of the venous pressure at flow rates of 50–100 cc./kgm. Modifications in venous cannulation necessitated by anomalies of the cavae system are described. *(Bosher, L. H., Jr.: Problems in Extracorporeal Circulation Relating to Venous Cannulation and Drainage, Ann. Surg., 149: 652 (May) 1959.)*

GAS ANALYSIS By combining a vacuum extraction method with gas chromotography it is possible to make rapid, accurate, and reproducible determinations of gases in biological fluids (*e.g.*, plasma). Determination of oxygen tension in one milliliter of human plasma is possible. *(Ramsey, L. H.: Analysis of Gas in Biological Fluids by Gas Chromotography, Science, 129: 900 (April 3) 1959.)*

CARBON DIOXIDE ANALYZER An apparatus consisting of a bridge-type continuous-flow carbon dioxide analyzer is described which repeatedly samples end expiratory air for use in patients receiving artificial respiration, either by intermittent positive pressure or in a tank respirator. The results of the analysis are rapidly obtainable. A comparison of results obtained with this and other methods is presented. *(Smith, A. C., Schuster, E., and Spalding, J. M. K.: An End-Tidal Air Sampler for Use During Artificial Respiration, Lancet 1: 277 (Feb. 7) 1959.)*

CARBON DIOXIDE STUDIES Under certain conditions, the difference between arterial and venous *p*H and pCO_2 is negligible in the arm. This occurs when patients are at rest in bed with the skin warm, and the temperature of the skin over the dorsum of the hand is at least 35 C. It also occurs in patients under general anesthesia, or upon heating the hand and arm for 15 minutes with electric pads. Observations made from venous blood under these conditions simplified the assessment of alveolar ventilation by blood studies. *(Brooks, D., and Wynn, V.: Use of Venous Blood for pH and Carbon-Dioxide Studies, Lancet 1: 227 (Jan. 31) 1959.)*

PULMONARY FUNCTION After irradiation to the chests of dogs, pulmonary diffusing capacity, lung compliance and functional residual volume decreased progressively, but pulmonary vascular resistance remained normal for a period of five months. A decrease in compliance before six months suggests that an increase in fibrous tissue was present even though it was not demonstrated by pulmonary vascular resistance studies. Little pathologic change was observed after a single dose of irradiation except for capillary dilatation. Four to five months after fractional irradiation, the histologic findings demonstrated focal atelectasis with some fibrosis and hyperemia of the interstitial areas. These changes were not evident on x-ray examination of the chest. In the animals studied after longer periods, there was obvious interstitial fibrosis with a paucity of cellular elements and capillaries. There was some evidence of narrowing due to endothelial proliferation, focal necrosis of the walls and a few areas of acute hemorrhage. *(Sweany, S. K., Moss, W. T., and Haddy, F. J.: The Effects of Chest Irradiation on Pulmonary Function, J. Clin. Invest. 38: 587 (March) 1959.)*

HYPOVENTILATION SYNDROME The fifth of a series of cases of "primary hypoventilation syndrome" is described along special studies which have served to clarify the etiology concerned. It is believed that in "primary hypoventilation syndrome," the essential defect is in the respiratory regulatory mechanism. There is no underlying disturbance in the lungs or musculoskeletal apparatus of the chest. In the special studies performed, there was a totally absent response to hypoxia and

lobeline and a slightly diminished response to carbon dioxide. A paradoxical increase in minute ventilation followed the administration of oxygen which was explained by a supposed sensitivity of the respiratory center to carbon dioxide with improved oxygenation. (*Rodman, T., and Close, H. P.: The Primary Hypoventilation Syndrome, Am. J. Med. 26: 808 (May) 1959.*)

HYPOVENTILATION Arterial blood gas tensions, blood volumes and oxygen cost of breathing were studied in a series of obese subjects. For the most part there was no evidence of gross lung disease as revealed by clinical history and ventilatory function measurements. Twelve of eighteen subjects who had measurements of arterial blood gas tensions had hypoxia. Four had associated hypercapnia which apparently was due to reduced tidal volume. Red cell mass per square meter of body surface was increased in both male and female subjects. Plasma volume was increased only in the female subjects. The oxygen cost of breathing was increased in all the obese subjects. It is suggested that this was due to an increase in elastic resistance of the thorax. There appeared to be a relationship between the oxygen cost of breathing and the arterial carbon dioxide tension in obese subjects. This is in accordance with the hypothesis that respiratory acidosis is an adaptive mechanism sparing oxygen for nonventilatory work, a rise in carbon dioxide tension being tolerated when the work of breathing is increased. The data also indicate that, in the obese individual, further increments in ventilation could result in a disproportionate increase in metabolic work of breathing which would be exaggerated if he developed bronchitis or other lung disease. Conversely, individuals with chronic lung disease and increased work of breathing would get into further difficulty with the development of obesity. (*Kaufman, B. J., Ferguson, M. H., and Cherniack, R. M.: Hypoventilation in Obesity, J. Clin. Invest. 38: 500 (March) 1959.*)

OXYGEN CONSUMPTION The oxygen consumption of the respiratory muscles was measured in normal and emphysematous subjects. The oxygen cost of increased ventilation was considerably higher in the emphysematous subjects and rose even further with slight increases in ventilation. Efficiency of the respiratory muscles was considerably lower in patients with emphysema than in normal subjects. The total mechanical work performed on the lung and thorax tends to be less in the emphysematous than in the normal individual at low ventilation. This might be expected since about 63 per cent of the work of breathing is performed in over-coming elastic resistance and a substantial loss of lung elasticity occurs in emphysema. The oxygen cost of breathing is four to five times greater because of the marked reduced efficiency of the respiratory muscles. Increases in ventilation result in a disproportionate increase in oxygen consumption of the respiratory muscles in emphysema. This may explain the disability present in pulmonary emphysema and the inability of the severely emphysematous patient to meet the increased energy demands of exercise and infection. (*Cherniack, R. M.: The Oxygen Consumption and Efficiency of the Respiratory Muscles in Health and Emphysema, J. Clin. Invest. 38: 494 (March) 1959.*)

PULMONARY EDEMA In this review article outlining a rational approach to the treatment of all types of pulmonary edema, emphasis is placed on relief of anoxia. Some caution regarding continuous inhalation of 60 to 100 per cent oxygen for several hours is emphasized, but the danger of pulmonary edema from hyperoxyia is remote in the treatment of patients with pulmonary edema. In addition to the conventional means of reducing circulating blood volume the use of ganglion blocking drugs is suggested. While atropine is most useful in the treatment of poisoning by cholinergic and anticholinesterase agents, it is not very useful in the treatment of pulmonary edema due to congestive heart disease. A new synthetic compound (#45–50) has been used in the treatment of edema associated with burns of the respiratory tract. (*Aviado, D. M., Jr., and Schmidt, C. F.: Physiologic Basis for the Treatment of Pulmonary Edema, J. Chronic Dis. 9: 495 (May) 1959.*)

PULMONARY EDEMA Experimental toxic lung edema in white mice was induced