RESPIRATORY ACIDOSIS The pattern of renal and cellular responses of electrolytes to chronic respiratory acidosis was studied in rats. Urinary acid excretion increases transiently for the first day or two and then returns to control values despite continued severe respiratory acidosis. A rise in urine *p*H appears to be chiefly responsible for the rapid return of urinary ammonia to control values. This is presumably due to increased bicarbonate excretion as serum bicarbonate reaches maximal values. Renal glutaminase is not activated during respiratory acidosis. This is strong evidence against the role of intracellular or extracellular *p*H as regulatory factors in the adaptation of renal amomnia-producing enzymes. Adaptation of renal carbonic anhydrase does not occur in spite of increased tubular bicarbonate reabsorption. Chloride excretion is greatly elevated the first day of respiratory acidosis but thereafter returns to control values. Potassium excretion is also markedly elevated the first day and continues to be excreted at a slightly increased rate. Muscle sodium and potassium are slightly decreased after eleven days of respiratory acidosis. The most striking change in bone electrolytes is a fall in magnesium content. (*Carter, N. W., Seldin, D. W., and Teng, H. C.: Tissue and Renal Response to Chronic Respiratory Acidosis, J. Clin. Invest. 38: 949 (June) 1959.*)

RESPIRATORY OBSTRUCTION Endotracheal anesthesia was administered to a patient for Cesarean section. On connecting the corrugated tubing to the endotracheal tube, it was impossible to inflate the patient's lung, and cyanosis developed. The cause could not be ascertained. The tube was finally removed and another inserted, following which there were no difficulties. The first tube was examined. The lumen contained a broken cleaning brush causing complete occlusion. (*Jenkins, A. V.: Unexpected Hazard of Anaesthesia, Lancet 1: 761 (April 11) 1959.*)

PULMONARY FUNCTION The means by which exercise increases the pulmonary diffusing capacity for carbon monoxide in normal subjects has been studied using both the steady state and breath holding techniques. Increase in cardiac output produced by means other than exercise caused no significant change in the diffusing capacity for carbon monoxide as measured by either of these techniques. Steady state diffusing capacity for carbon monoxide was found to be very sensitive to the ventilation rate, and by hyperventilation alone was increased as much as by exercise at the same minute ventilation. The breath-holding diffusing capacity was not affected by hyperventilation preceding the experiment. The effect of increased ventilation can explain the increase in the steady-state diffusing capacity with exercise, but an adequate explanation for the increase in breath-holding diffusing capacity which occurs during exercise is not obtained. (*Ross, J. C., Frayser, R., and Hickam, J. B.: Study of Mechanism by Which Exercise Increases the Pulmonary Diffusing Capacity for Carbon Monoxide, J. Clin. Invest. 38: 916 (June) 1959.*)

RENAL FUNCTION The effect of continuous pressure breathing (postive or negative) upon renal function and water clearance was studied in 12 normal volunteers. Positive pressure breathing produces a decrease in urine flow resulting primarily from a decrease in free water clearance, with decrease in glomerular filtration rate as a contributing factor. Negative pressure breathing is associated with an increase in free water clearance with resultant increase in the rate of urine flow without changes on glomerular filtration rate. Administration of alcohol during water diuresis causes partial or complete inhibition of the antidiuretic effect of positive pressure breathing even though the change in glomerular filtration rate is similar to the group with water diuresis. During osmotic diuresis there is no antidiuresis during or following positive pressure breathing, although there were decreases in glomerular filtration rate. The administration of vasopressin prevents the diuresis in response to negative pressure breathing. Alterations in electrolyte excretion and renal hemodynamics occur during positive pressure breathing, but are not of the magnitude or always in the direction of the changes in water clearance and rate of urine flow. On the other hand, electrolyte excretion and renal hemo-

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dynamics are not significantly changed with the diuresis of negative pressure breathing. It is concluded that altered antidiuretic hormone activity is the primary mechanism by which continuous pressure breathing changes the rate of urine flow. (*Murdaugh, H. V., Sieker, H. O., and Manfredi, F.: Effect of Altered Intrathoracic Pressure on Renal Hemodynamics, Electrolyte Excretion and Water Clearance, J. Clin. Invest. 38: 834 (May) 1959.*)

PULMONARY EDEMA Pulmonary vascular congestion was produced in spontaneously breathing anesthetized dogs by partial aortic constriction and intravenous infusion. Brief periods of congestion were associated with small changes in the lung compliance compared with the progressive and striking compliance reduction (minus 78 per cent) noted with prolonged congestion. Findings suggested that surface phenomena were responsible for the mechanical behavior of edematous lungs rather than vascular congestion, *per se*, or intrinsic tissue changes. (*Cook, C. D., and others: Pulmonary Mechanics During Induced Pulmonary Edema in Anesthetized Dogs, J. Appl. Physiol. 14: 177 (March) 1959.*)

HYPERVENTILATION Twenty patients with hyperventilation syndrome, in whom organic heart disease had been ruled out, were studied to determine changes in electrocardiogram, blood *p*H, serum electrolytes, and blood gases during voluntary hyperventilation with room air and again with 6 per cent carbon dioxide. Transient depression of ST segments and T wave inversion were seen most commonly. These were eliminated by infusion of K^+ ion, and/or inhalation of 6 per cent carbon dioxide. It is suggested that electrocardiographic changes during hyperventilation may be due to combined effects of respiratory alkalosis and increased sympathetic tone secondary to release of epinephrine, which in turn may cause a migration of intracellular potassium and alterations in membrane potentials across myocardial cell membranes. Hypervagotonia produced by respiratory alkalosis may also contribute to electrocardiographic changes. Emphasis is placed on importance of interpretation of electrocardiographic changes and distinguishing clinically between symptoms due to hyperventilation syndrome and organic heart disease. (*Yu, P. N., Yim, B. J. B., and Stanfield, A.: Hyperventilation Syndrome, Arch. Int. Med. 103: 902 (April) 1959.*)

PULMONARY REFLEXES In mongrel dogs the left lung was isolated except for the nerve supply. Increasing the pressure in the isolated left pulmonary veins resulted in pulmonary hypertension and arteriolar constriction in the opposite intact lung. Thus evidence is submitted that elevation in pulmonary venous and capillary pressures produces reflex arteriolar vasospasm in the lungs and this reflex aids in the regulation of the lesser circulation. (*Sanger, P. W., and others: Observations on Pulmonary Vasomotor Reflexes, J. Thoracic Surg. 37: 774 (June) 1959.*)

PULMONARY VASCULAR RESISTANCE In 30 patients with atrial septal defects and pulmonary artery pressure over 60 mm. Hg, breathing 100 per cent oxygen reduced the systolic, diastolic and mean pressures in the pulmonary artery. Pulmonary blood flow increased, and total pulmonary resistance fell. The increase in flow and fall in pressure indicate a decline in pulmonary vascular resistance. The mechanism of the fall in pressure and increase in flow with breathing 100 per cent oxygen is not known. Patients with atrial septal defect and in whom breathing of oxygen produced the greater fall in resistance had a higher operative survival rate. (*Swan, H. J. C., and others: Effect of Oxygen on Pulmonary Vascular Resistance in Patients with Pulmonary Hypertension Associated with Atrial Septal Defect, Circulation 20: 66 (July) 1959.*)

HYPOXEMIA After one minute of succinylcholine apnea, with gas flow disconnected, arterial oxygen saturation dropped 15 per cent. Apnea with endotracheal suction produced similar results. The effects of apnea and apnea and suction combined were practically nullified by the concomitant insufflation of oxygen at 4 liters per minute. Obviously oxygen insufflation protects not only against apnea, but also against suctioning of the apneic patient. (*Boba, A., and others: Effects of Apnea, Endo-*

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