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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