and prostigmine do not affect the electroencephalogram; however, evidence is presented that *d*-tubocurarine may cause cerebral cortical blockade. During the early administration of ether, 80 per cent nitrous oxide, or 33 per cent cyclopropane, a fast cortical phase is said to develop as a result of an excitatory influence upon the reticular core. Both arousal and recruiting responses are abolished. Evoked midbrain potentials are suppressed earlier than thalamic relay potentials. The comparative efficacies of these anesthetics in suppressing the potentials correspond with clinical impressions of their potency. One effect of small doses is to produce a functional block of ascending impulses in the reticular core. They may even operate on the thalamic relay nuclei. In hypoxic studies, an activation stage precedes the final electrical silence of terminal anoxia. No activation stage occurs, however, after carotid chemoreceptor elimination; therefore, the direct effect of hypoxia on the brain stem is purely depressive. Intense hypercapnia produces prolonged activation which disappears after retromammillary transection but is not influenced by elimination of chemoreceptors or by prebulbar section. Thus hypercapnia seems to activate the ascending reticular activating system directly. (*O'Leary, J. L., and Coben, L. A.: Reticular Core—1957, Physiol. Rev. 38: 213 (April) 1958.*)

WATER REABSORPTION The decrease in urine volume after injection of antidiuretic hormone (ADH) to a hydrated mammal has long been known. One hypothesis derived from clearance studies suggests the following sequence: (a) an active reabsorption of sodium in the proximal tubule with passive reabsorption of water in maintenance of the isosmotic state; (b) further active reabsorption of a fixed amount of sodium and water in the distal tubule, maintaining isosmoticity only in the presence of a maximum dose of ADH but resulting in hypotonic urine with smaller doses, and (c) an active reabsorption of a fixed quantity of water in a more distal segment, possibly the collecting duct.

The counter-current theory involves the concept of a steady state where the fluid entering the descending limb of the loop of Henle is more and more concentrated toward the hairpin bend and rediluted on its way up the ascending limb. These gradients may be brought about by some active cellular transport mechanism either by drawing water from the descending to the ascending limb or transporting solutes in the opposite direction—or both. The collecting ducts, passing through this hypertonic environment lose water from their lumens. ADH is thought to function in the establishment of the counter current system by changing the permeability to water in the descending limb of Henle's loop, the distal convoluted tubules and the collecting tubules. (*Thorn, N. A.: Mammalian Antidiuretic Hormone, Physiol. Rev. 38: 169 (April) 1958.*

UREA EXCRETION The classic mechanism for renal excretion of urea in mammals was thought to consist of glomerular filtration and a passive back diffusion in the tubules. Tubular regulation or secretory mechanisms were not believed to be involved. However, recent evidence suggests that, in man, the urea clearance varies with the dietary protein content. The maximum difference in clearance between a normal and low protein intake is found at low urine flows. The urea clearance can increase rapidly and selectively following nitrogen ingestion during the low protein regime. These variations occur even though the glomerular filtration rate does not change and thus must be due to tubular rather than glomerular regulation. Other observations which lend themselves to the same interpretation are the change in the concentrating power of the kidney when nitrogen intake is altered, and the effect of the pathological reduction in glomerular filtration rate on urea clearance. An explanation of urea transport invoking the counter-current hypothesis is presented. (*Schmidt-Nielsen, B.: Urea Excretion in Mammals, Physiol. Rev. 38: 139 (Apr.) 1958.*)

PULMONARY FUNCTION Studies were made on 23 adult tuberculous patients before and after pulmonary resection. Vital capacity and total capacity were reduced in almost all cases. Average residual volume was unchanged in those having one

or two segments removed and was reduced in those having 3–5 segments removed. Average maximal breathing capacity, per cent of rapid vital capacity expired in one second, the 7 minute alveolar nitrogen and arterial oxygen saturation were essentially unchanged. (*Miller, R. D., and others: Pulmonary Function Before and After Pulmonary Resection in Tuberculous Patients, J. Thoracic Surg. 35: 651 (May) 1958.*)

OXYGEN INTAKE The maximal oxygen intake is dependent on both cardiac output and arteriovenous oxygen difference. The widening of the arteriovenous oxygen difference was due principally to diminution in mixed venous oxygen content. There was no significant change in arterial oxygen tension from rest to heavy work; the slight decrease in oxygen saturation that was observed can be explained by the *p*H change of the blood and the resulting shift in the oxygen dissociation curve. The venous oxygen tension showed no significant change, even though the venous oxygen content and saturation fell appreciably. The end result of the phenomenon is to maintain an adequate oxygen tension gradient from capillary to cell. In ascertaining the physiologic meaning of the maximal oxygen intake, the relative importance of cardiac capacity and increase in arteriovenous oxygen difference must be determined. It is probable that in the normal individual the ability to increase cardiac output is the more important of the two factors. (*Mitchell, J. H., Sproule, B. J., and Chapman, C. B.: Physiological Meaning of Maximal Oxygen Intake Test, J. Clin. Invest. 37: 538 (April) 1958.*)

HYPOXEMIA On hypoventilation with air CO_2 retention develops and hypoxemia progresses at an increasingly rapid rate. However, significant hypoxemia only begins to appear when the arterial CO_2 tension is over 60 mm. of mercury. Hypoxemia of this sort may be caused by increased metabolism without a concomitant increase in ventilation or by diminished alveolar ventilation. The latter may occur from a depressed total ventilation or by an increased dead space ventilation such as might occur with an inefficient anesthetic apparatus. The ventilatory needs of a patient during anesthesia or in a mechanical respirator may be predicted by calculating the alveolar ventilation necessary to maintain the normal CO_2 tension, measuring or assuming a normal dead space, and then calculating the required total ventilation. (*Williams, M. H., Jr.: Quantitative Relationships Between Hypoxemia and Disorder of Pulmonary Function, Yale J. Biol. & Med. 30: 306 (Feb.) 1958.*)

PULMONARY DIFFUSION The function of the lungs depends upon two phenomena: alveolar ventilation and alveolar diffusion. The diffusing capacity of normal resting lung is sufficient to supply about three times the normal resting O_2 uptake, but this capacity must increase in order to deal with the O_2 uptake required by even moderate exercise. Thus the reserve of diffusing capacity for O_2 is small and respiratory failure may occur if diffusing capacity is impaired by disease. By contrast, CO_2 diffuses over twenty times more rapidly than O_2 so that elimination of CO_2 is never limited by diffusion, and CO_2 retention, when it occurs, is due to inefficient ventilation, not to impaired diffusion. Resistance to pulmonary diffusion is provided by the alveolar membrane which consists of the alveolar epithelium, a complex basement membrane and the capillary epithelium. Any increase in thickness of this membrane or reduction in the number of functioning alveoli or capillaries will reduce diffusing capacity. Normally the alveolar membrane offers about 70 per cent of the total resistance to diffusion of O_2 while the resistance of uptake into the red cells accounts for about 30 per cent. Methods for measuring diffusing capacity utilize carbon monoxide gas (D_{CO}), since measurements of D_{O_2} presents a number of technical difficulties. (*Marshall, R.: Methods of Measuring Pulmonary Diffusing Capacity and Their Significance, Proc. Roy. Soc. Med. 51: 101 (Feb.) 1958.*)

RESPIRATORY INSUFFICIENCY Of those who survive an accident involving coma and head injury, the main cause of death is respiratory insufficiency and anoxia. The anoxia is due to central disturbances of the control of respiration, and reduction of compliance of the lungs by