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 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