The effect of CO₂ on renal perfusion was studied using a direct cannulation technique to measure renal blood flow. PₐCO₂ was varied from 20 to 120 mm Hg. Measurements were made with PₐCO₂ maintained within narrow ranges. Renal vascular resistance, oxygen consumption, and filtration fraction were measured, as was creatinine and p-aminohippurate clearance, while excretion of sodium and potassium was monitored. No real change in renal perfusion occurred until PₐCO₂ exceeded 70 mm Hg. A further increase in PₐCO₂ was associated with a progressive decrease in renal blood flow, accompanied by similar decreases in glomerular filtration rate, renal plasma flow, and electrolyte excretion. The depressed perfusion which resulted from hypercapnia was preventable by mannitol infusion and by renal nerve blockade. The authors suggest that CO₂ has no direct effect on the intrarenal vasculature, and that the vasococonstrictor effect of hypercapnia is secondary to the interaction between CO₂ and the sympathoadrenal system. (Norman, J. N., and others: Effect of Carbon Dioxide on Renal Blood Flow, Amer. J. Physiol. 219: 672 (Sept.) 1970.)

Respiration

2,3-DPG, pH AND HEMOGLOBIN-OXYGEN AFFINITY The influence of pH on hemoglobin affinity for oxygen (Bohr effect) is well known. On the other hand, we know now that an intermediate of glucose metabolism, 2,3-diphosphoglyceric acid (2,3-DPG) is present in high concentration in erythrocytes, and has been shown to reduce the affinity of hemoglobin for oxygen. In 1942, Guest showed that diabetic acidosis was associated with low erythrocyte 2,3-DPG. A reduction of the concentration of 2,3-DPG leads to a higher affinity of hemoglobin for oxygen, whereas the decrease in pH, mediated through the Bohr effect, leads to a shift of the dissociation curve to the right. The purpose of the present study was to investigate the adaptive response to systemic acidosis or alkalosis of oxyhemoglobin dissociation in normal man to delineate mechanisms involved in this regulation.

Acidosis was induced acutely in four healthy volunteers with intravenous acetazolamide given in a one-week period. The acidosis was then rapidly corrected and alkalesis induced with sodium bicarbonate injected intravenously. Control values for hemoglobin-oxygen affinity, erythrocyte acid-soluble organic phosphates, plasma and erythrocyte pH, blood gases, plasma electrolytes, and hemoglobin were determined, and hematocrit readings were made. Serial values were obtained during acidosis and alkalosis. Arterial blood was obtained for all measurements.

Acute changes in plasma pH produced no alteration in erythrocyte 2,3-DPG content, but there were changes in hemoglobin-oxygen affinity, and these correlated with changes in mean corpuscular hemoglobin concentration (MCHC). When acidosis or alkalosis was maintained (usually for four hours) erythrocyte 2,3-DPG content was affected and correlated with the change in hemoglobin-oxygen affinity. An increase in pH was countered by an increase in 2,3-DPG; conversely, a decrease in pH was associated with diminution of erythrocyte 2,3-DPG. Provided the rate of pH change is not too rapid or too great, 2,3-DPG changes counteract the pH effect and hemoglobin-oxygen affinity in vivo remains unchanged. Approximately 35 per cent of the change in hemoglobin-oxygen affinity which occurs from alteration in erythrocytic 2,3-DPG, can be explained by the effect of 2,3-DPG on erythrocyte pH. (Bellingham, A. J., Detter, J. C., and Lenfant, C.: Regulatory Mechanisms of Hemoglobin Oxygen Affinity in Acidosis and Alkalosis, J. Clin. Invest. 50: 700 (March) 1971.)

Endocrine Function

THYROID ACTIVITY AND CATECHOLAMINE SENSITIVITY This paper reviews the state of our knowledge of thyroid hormone and its relationship to the action of catecholamines. According to the author, studies of the circulatory effects of hyperthyroidism in relation to catecholamine hyperactivity published prior to 1960 are inconclusive, because they failed to utilize dose-response curves or their data were insufficient for statistical analysis.

Subsequent studies (1965–1967) found no difference between the responses of hyperthyroid-