anesthetic periods. Ventilation of one area of the lung decreases if the carbon dioxide concentration in that area is allowed to decrease by occluding its pulmonary blood supply. This may be a homeostatic mechanism to control the distribution of ventilation to those areas of the lung where blood flow is going. The cerebral blood flow is regulated, in part, by the carbon dioxide concentration in arterial blood. Inspiration of seven per cent carbon dioxide doubles the cerebral blood flow and hyperventilation may reduce the cerebral blood flow to 60 per cent of normal. The symptoms of hyperventilation are in part due to hypoxia of the brain, resulting from cerebral vasoconstriction. Carbon dioxide inhalation causes hyperpnea by an elevation of the P_{CO_2} in the respiratory center.

The electrotlyte composition of the cerebral spinal fluid in the fourth ventricle plays a role in the control of respiration, slightly acid solutions stimulating and slightly alkaline solutions depressing ventilation. Carbon dioxide present in the brain contributes to the narcosis produced by nitrous oxide inhalation. The average nitrous oxide concentration required to produce loss of consciousness varied from 30 to 60 per cent when alveolar carbon dioxide was varied downward from nine to three per cent. *(Severinghaus, J.: Carbon Dioxide Tension and Perfusion in Tissue, Der Anaesthesist 9: 50 (Feb.) 1960.)*

CARBON DIOXIDE Experiments are shown which demonstrate the respiratory driving action of hydrogen ions in cerebrospinal fluid. A specific action of carbon dioxide at this location can be excluded. The action of cerebrospinal fluid hydrogen ions is not direct effect on centers but an influence on intracranial efferents to the centers. It can be eliminated by procaine introduced into the cerebrospinal fluid. *(Loeschcke, H. H.: Relationship Between Carbon Dioxide and Respiration, Der Anaesthesist 9: 38 (Feb.) 1960.)*

CARBON DIOXIDE Adjustments of body stores of carbon dioxide were studied during voluntary hyperventilation for one hour at a constant rate by trained subjects. Carbon dioxide was eliminated at an average of 161 ml. per mm. Hg decrease in mixed venous tension. Increasing the respiratory minute volume by about 50 per cent for one hour resulted in elimination of 1.5 to 2.5 liters of carbon dioxide in excess of the metabolic production. *(Vance, J. W., and Fowler, W. S.: Adjustment of Stores of Carbon Dioxide during Voluntary Hyperventilation, Dis. Chest. 37: 304 (March) 1960.)*

HYPERCAPNIA Progesterone is capable of lowering the arterial P_{CO_2} in patients with emphysema and hypercapnia. This hormone will aslo lower the alveolar P_{CO_2} in normal subjects. If one assumes that the action of progesterone causes a small but definite degree of hyperventilation, then it is of interest that this increase in ventilation is sufficient to cause a fall in arterial P_{CO_2} in patients with severe obstructive diseases and hypercapnia. Voluntary hyperventilation is incapable of lowering the arterial P_{CO_2} in patients with emphysema. The effect of progesterone on ventilation is not solely the effect of progestational activity. The ethinyl group inactivates the respiratory effects seen with progesterone itself and alterations of the molecular structure at other sites can abolish the effect. Conclusions on the mode of action of progesterone are not possible from this study. It lowers the arterial P_{CO_2} without altering the ventilatory response to carbon dioxide. Other areas of the brain, possibly the hypothalamus, may represents the site of action. *(Tyler, J. M.: The Effect of Progesterone on the Respiration of Patients with Emphysema and Hypercapnia, J. Clin. Invest. 39: 34 (Jan.) 1960.)*

HYPERCAPNIC ACIDOSIS Experiments were designed to investigate the effects of tris-hydroxymethyl-amino methane (T.H.A.M.) on hypercapnic acidosis in dogs. High degrees of hypercapnic acidosis were induced in the animals using the technique of earlier diffusion respiration experiments. Treated dogs were administered .34 mM. of T.H.A.M./kg./min. during a 60-minute period of apneic oxygenation. In the untreated dog, the *p*H decreased from 7.41 to 6.45; arterial P_{CO_2} increased from 38 to 346 mm. Hg; arterial oxygen saturation dropped from 100 to 54 per cent; total catecholamines increased from 1 to 44 micrograms per liter. In the dogs