may be due to a decrease in pulmonary capillary volume. As healing occurs, ventilation, edema, and perfusion shifts become of less importance and the change in diffusion is dependent on lasting physiologic changes, such as loss of lung volume or change in pulmonary hemodynamics brought about by the operation. (*Howatt, W. F., and others: Pulmonary Function Changes Following Repair of Heart Lesions with the Aid of Extracorporeal Circulation, J. Thor. Cardiov. Surg. 43: 649 (May) 1962.*)

PULMONARY FUNCTION Expiratory reserve volume, vital capacity, and functional residual capacity were diminished in a group of postpoliomyelitis patients exhibiting chronic flaccid respiratory paralysis. There was no significant difference in residual volume between the patient and the control groups. These findings negate contentions of other workers that residual volume is increased in postpoliomyelitis patients due to passive acquisition of a portion of the expiratory reserve volume. Diminution of lung and chest wall compliance greater than would be expected for the observed decrease in functional residual capacity suggests some loss of elasticity in the lungs and/or the chest wall as a residual of poliomyelitis. (*Faerber, I., Liebert, P. B., and Suskind, M.: Loss of Functional Residual Capacity in Poliomyelitis, J. Appl. Physiol. 17: 289 (Mar.) 1962.*)

VENTILATION VOLUME The effect of ventilation volume during artificial ventilation on subsequent spontaneous ventilation was studied in two paretic patients. Spontaneous ventilation can be influenced by a factor other than P_{CO_2} and P_{O_2}. This factor is related to minute volume or tidal volume which has previously prevailed. The mechanism for this may be dependent on an afferent pathway originating in the receptors in the lungs and/or chest wall. (*Smith, A. C., Spalding, J. M. K., and Watson, W. E.: Ventilation Volume as a Stimulus to Spontaneous Ventilation after Prolonged Artificial Ventilation, J. Physiol. 160: 22 (Jan.) 1962.*)

CARBON DIOXIDE The effect of carbon dioxide on ventilation was studied in cross-circulation dogs. The recipient dog's head was perfused exclusively by arterial blood from the donor dog. Ventilation and arterial P_{CO_2} of both dogs were determined during inhalation of carbon dioxide by the donor dog. Although the blood of the recipient dog's body was hypocapnic when its head was receiving hypercapnic blood, its ventilatory response was similar to that of the donor dog. It is inferred that the central carbon dioxide chemoreceptors can account for all the ventilatory response to carbon dioxide inhalation. (*Kao, F. F., and others: Respiratory Sensitivity to Carbon Dioxide in Cross-Circulated Dogs, Amer. J. Physiol. 202: 1024 (May) 1962.*)

CARBON DIOXIDE The response to hypoxia is much reduced one hour after a period of breathing carbon dioxide in air mixtures. Measurements were made when breathing 9.5 per cent oxygen and nitrogen: (1) at successive intervals for periods up to four hours after breathing a carbon dioxide in air mixture; (2) at time intervals on different days after breathing the same carbon dioxide in air mixtures. The ventilatory response to hypoxia was initially depressed after carbon dioxide breathing. The magnitude and duration of this depression varied with changes in concentration of carbon dioxide in the mixture and the time for which it was breathed. There was no constant relationship between the response to hypoxia and the immediately preceding level of end-tidal carbon dioxide tension. It appears that some factor other than variation in end-tidal carbon dioxide tension is responsible for these changes in the ventilatory response to hypoxia following carbon dioxide breathing. (*Penman, R. W. B., and Singh, H. D.: Changes in Ventilatory Response to Hypoxia after Breathing Carbon Dioxide Mixtures, J. Physiol. 160: 17P (Feb.) 1962.*)

DYSPNEA Slight to severe dyspnea was produced in a patient with nearly complete respiratory paralysis by inhalation of 7 per cent carbon dioxide-in-air in a respirator. Intensive stimulation of the respiratory center is sufficient alone to produce dyspnea. (*Patterson, J. L., and others: Carbon Dioxide-Induced Dyspnea in a Patient with Respiratory Muscle Paralysis, Amer. J. Med. 32: 811 (May) 1962.*)