BRIEFS FROM THE LITERATURE

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Briefs were submitted by Drs. C. M. Ballinger, Lee S. Binder, T. H. Cannard, M. T. Clarke, R. A. Devloo, Cody Eames, D. W. Eastwood, J. E. Eckenhoff, Martin Helrich, S. J. Martin, J. L. McDonnell, Alan Thorogood, R. E. Ponath, R. W. Ridley and H. S. Rottenstein.

CARBON DIOXIDE The changes in blood *p*H and pCO_2 during the recovery phase were studied in a group of children and adults with diabetic acidosis, diarrheal acidosis, and uremic acidosis. Patients recovering from these types of acidosis frequently pass through a phase in which the blood *p*H is normal but pCO_2 is still decreased. This suggests that there is a sustained hyperventilation even though the patient is no longer acidotic. It is also suggested that such patients may have an increased sensitivity of the respiratory center to pCO_2 and/or hydrogen ion concentration accounting for the continuing hyperventilation. (*Winters, R. W., Lowder, J. A., and Ordway, N. K.: Observations on Carbon Dioxide Tension During Recovery from Metabolic Acidosis, J. Clin. Invest. 37: 640 (May) 1958.*)

PULMONARY EMBOLISM Physiologic phenomena associated with pulmonary embolism in the dog, such as pulmonary hypertension, peripheral hypotension, hyperpnoea and bradycardia, appear to be reflex in nature. This reflex is initiated only in vessels less than 25–100 micra in diameter and is mediated through the sympathetic nervous system. The hyperpnoea noted following pulmonary embolism is most likely mediated through the vagus nerve. In cases of massive pulmonary embolism, a mechanical factor is introduced due to the widespread blockage of the pulmonary vascular tree. (*Weidner, M. G., and Light, R. A.: Role of Autonomic Nervous System in Control of Pulmonary Vascular Bed, Ann. Surg. 147: 895 (June) 1958.*)

PULMONARY FUNCTION Vital capacity, residual volume, maximum breathing capacity, arterial blood gases and *p*H, and ventilation during exercise studies were made on a group of 63 paraplegic patients and a group of 17 normals. These were divided into three groups: (1) those with lumbar spinal lesions who had paralysis of the lower limbs and the pelvic floor but no paralysis of the muscles of breathing; (2) those with thoracic spinal lesions with paralysis of the lumbar muscles and lower intercostals; and (3) those with lower cervical spinal lesions with paralysis of all muscles of breathing except the diaphragm and the accessory muscles of respiration. It was found that the vital capacity and maximum breathing capacity of patients with lumbar lesions were normal. The group with lower cervical lesions had a vital capacity approximately two-thirds of normal and a maximum breathing capacity of one-half of normal. The paraplegics with thoracic lesions had vital capacities and maximum breathing capacities between the values for the cervical and lumbar lesions groups. The resting expiratory reserve volume was below the normal percentage only for the group of paraplegics with cervical lesions. For this group the percentage value was one-half normal which was higher than was anticipated for patients with only inspiratory muscle function. Residual volumes of all groups were significantly higher than in the normal. Arterial blood gases of all groups were within normal limits and the ventilatory response to exercise for all groups of paraplegics did not differ from normal. (*Hemingway, A., Bors, E., and Hobby, R. P.: Investigation of Pulmonary Function of Paraplegics, J. Clin. Invest. 37: 773 (May) 1958.*)

DIFFUSING CAPACITY Data from 151 patients have been analyzed to determine the validity of the end tidal sampling method of measuring the mean alveolar carbon monoxide concentration in lung