Literature Briefs

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Briefs were submitted by Drs. C. M. Ballinger, Peter P. Bosomworth, M. T. Clarke, R. B. Clarke, H. S. Davis, Deryck Duncalf, James E. Eckenhoff, Martin Helrich, G. Hohmann (Germany), J. J. Jacoby, F. C. McPartland, W. H. Mannheimer, Alan D. Randall, Norman Rosenbaum, P. H. Sechzer, W. H. Ring, H. S. Rottenstein, E. A. Talmage. Briefs appearing elsewhere in this issue are a part of this column.

HYPERCAPNIA Hypercapnia was induced in 35 dogs by exposing them to 12 per cent carbon dioxide in an environmental chamber for periods ranging from one-half hour to 5 days. In the first 24 hours the bicarbonate concentration in spinal fluid rose to a value approximately 13 mEq. per liter higher than the control. No further significant change occurred during the subsequent 4 day period of observation. In plasma water the concentration of bicarbonate also rose by approximately 13 mEq. per liter, but the new steady state was not achieved so quickly as in spinal fluid. Hydrogen ion concentration in both cerebrospinal fluid and plasma was increased by approximately 15 nanomoles per liter. Chloride concentration in both compartments decreased by an amount nearly identical to the rise in bicarbonate. Concentrations of sodium and potassium were not significantly different from the control state. Despite changes in concentrations of bicarbonate, chloride, and hydrogen ion, the cerebrospinal fluid plasma ratio of all measured electrolytes in the new steady state was nearly identical to those present in the control state. If some potential difference governs the partition of any of the measured ions, this potential at 5 days was virtually identical to that in the control state. *(Bleich, H. L., Berkman, P. M., and Schwartz, W. B.: Response of Cerebrospinal Fluid Composition to Sustained Hypercapnia, J. Clin. Invest. 43: 11 (Jan.) 1964.)*

CHRONIC CARBON DIOXIDE Acid-base balance and electrolyte changes in 20 subjects exposed to 1 per cent carbon dioxide over a period of 42 days with control periods preceding and subsequent to exposure, indicated a slight compensated respiratory acidosis was present during the first 23 days of exposure followed by a compensated respiratory acidosis. Deacclimitization was incomplete, even after four weeks of recovery on air. Arterial carbon dioxide rose 5 mm. of mercury and remained there during the first nine days of recovery on room air. Sodium increased while potassium showed an equivalent decrease in whole blood. *(Schaeffer, K. E., and others: Acid-Base Balance and Blood and Urine Electrolytes of Man During Acclimatization to CO_2, J. Appl. Physiol. 19: 48 (Jan.) 1964.)*

VOLUNTARY HYPERVENTILATION Voluntary hyperventilation over a period of five minutes decreased the arterial P_{CO_2} to 15 mm. mercury and increased the arterial *p*H to a maximum of 7.76. Both values had not returned to normal ten minutes after termination of forced respiration. After 20 minutes, however, normal values were obtained in most volunteers. The arterial P_{O_2} increased during hyperventilation. Ten minutes after termination of hyperventilation the P_{O_2} was significantly below the control values while 20 minutes later normal values were found in most cases. Of the electrolytes studied (K, Na, Ca) only potassium showed a significant increase during and a decrease after hyperventilation. In some cases tetanic symptoms appeared during hyperventilation, but there was no correlation between their appearance and the extent of the *p*H increase, P_{CO_2} decrease and the other parameters measured. *(Ferlinz, R., Anhagen, H., and Hansen, G.: Effect of Voluntary Hyperventilation on pH, Serum Electrolytes, pO_2 and pCO_2 in Arterial Blood, Thoraxchirurgie 11: 463, 1964.)*

VENTILATION AFTER MORPHINE Compliance is reduced during general anes-

thesia, probably because of alveolar collapse, but is improved by an occasional deep breath. Except in very light anesthesia, spontaneous signs do not occur during general anesthesia. This state of partial alveolar collapse continues into the postoperative period, and is aggravated by the rapid, shallow respirations associated with abdominal pain. Morphine, even in doses which do not grossly depress ventilation and gas exchange, may depress the reflex urge to take occasional deep breaths. In addition to an attempt to reduce morphine dosage during the postoperative period, vigorous encouragement may help patients take a deep breath or cough. Forcible expansion of the lungs with a bag and mask, and regional anesthesia to block pain locally are additional measures to reduce pulmonary complications. Normal respiratory function should not be assumed merely on the basis of normal minute volume or carbon dioxide tension. The presence of periodic deep breaths, active or passive, is a component of normal respiratory function. (*Egbert, L. D., and Bendixen, H. H.: Effect of Morphine on Breathing Pattern, J.A.M.A. 188: 485 (May 11) 1964.*)

A-a OXYGEN DIFFERENCE Since the alveolar-arterial oxygen difference may be due to venous shunting, unequal distribution of alveolar ventilation, or diffusion limitations, measurements to determine the significance of each were carried out utilizing alveolar-arterial oxygen difference measurements during breathing of various concentrations of oxygen. Utilizing 14, 21, and 100 per cent oxygen inhalation, venous shunting was 1 to 5 per cent of the systemic blood flow and unequal distribution of alveolar ventilation contributed a shunt of an additional 3.4 per cent of the systemic blood flow. On breathing room air, the diffusion component of the alveolar-arterial oxygen was insignificant. (*Ayres, M .S., and others: Components of Alveolar-Arterial O_2 Difference in Normal Man, J. Appl. Physiol. 19: 43 (Jan.) 1964.*)

RESPIRATORY INERTANCE Total respiratory compliance, respiratory system natural frequency, and total respiratory inertance were measured in 8 normal and 14 excessively obese subjects. The mean total inertance in the normals was 0.0098 cm. of water per liter per second^2. In the obese subjects it was 0.0253 cm. of water per liter per second^2. This is a highly significant difference. There were significant inverse correlations between total respiratory compliance and body weight and between natural frequency and body weight. There was also a significant positive correlation between total respiratory inertance and body weight. A method for estimating separately the gas and tissue components of total respiratory inertance was applied to three normal and three obese subjects. In the normal subjects, tissue inertance averaged 16 per cent of the total inertance. In the excessively obese subjects, tissue inertance averaged 68 per cent of the total inertance. (*Sharp, J. T.: Total Respiratory Inertance and Its Gas and Tissue Components in Normal and Obese Men, J. Clin. Invest. 43: 503 (Mar.) 1964.*)

PULMONARY METASTASES Pulmonary compliance is reduced in all patients with extensive lymphatic and hematogenous spread. Multiple hematogenous metastases cause hypoxemia because of a shunt-like mechanism while diffuse lymphatic metastases reduce oxygenation by their effect on the gas-exchange surface of the lung. (*Emirgil, E., Zsoldos, S., and Heinemann, H. O.: Pulmonary Metastases and Pulmonary Function, Amer. J. Med. 36: 382 (Mar.) 1964.*)

TRACHEOSTOMY Effects of reduction of dead space with tracheostomy were noted in four subjects with lower airway obstruction and in two subjects with upper airway obstruction. The two groups were distinctly different in that the patients with lower airway obstruction demonstrated decrease in minute ventilation after tracheostomy. Also, alveolar ventilation decreased except when alveolar hypoventilation was present to start with. There was either no change or an increase in the arterial P_{CO_2} in subjects with lower airway obstruction. Small increases occurred in the arterial oxygen saturation with tracheostomy. (*Froeb, H. F., and others: Tracheostomy and Respiratory Dead Space in Emphysema, J. Appl. Physiol. 19: 92 (Jan.) 1964.*)