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-