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Compensation for metabolic alkalosis is also respiratory and renal. In some patients there is diminished ventilation, with some increase in P_{CO_2} , particularly when the CO_2 content of the plasma rises above 35 mEq/L. However, respiratory compensation is small and cannot be detected in the majority of patients.

Golding *et al.*³⁹ studied normal volunteers made alkalotic under controlled conditions by administration of buffers, diuretics, and aldosterone. Alkalosis induced by sodium bicarbonate, THAM, and ethacrynic acid was associated with high values of arterial P_{CO_2} (P_{CO_2} values of 46 to 48 mm Hg) while alkalosis induced by thiazide diuretics and aldosterone was associated with normal arterial blood P_{CO_2} values despite comparable increase in HCO_3^- values to a range of 30 to 44 mEq/L.

Figure 7-10 indicates that some patients have an increase in P_{CO_2} values at 35 mEq total CO_2 to compensate in this range. However, Figure 7-11, plotted from Elkin-

compensation for rising HCO_3^- by inhibition of CO_2 loss through hypoventilation.

Initial renal compensation is the loss of chloride ion and sodium bicarbonate in the urine. Unless a potassium-sparing diuretic is available, this loss of potassium is inevitable. Metabolic alkalosis develops, renal excretion of potassium is decreased, and absorption of this ion is increased to the paradoxical effect of helping to perpetuate the alkalosis.

Treatment of metabolic alkalosis is by sodium retention (administration) will be of little value. Alkalosis caused by potassium loss responds to the administration of chloride and potassium. Both ions are required, and potassium chloride is the drug of choice. Isotonic saline solution also provides additional chloride.

the release of oxygen from hemoglobin and decreasing arterial PO_2 , with the production of excess lactic acid. In addition, depression of the ionization of calcium may lead to cardiac arrhythmias and digitalis intoxication. With circulatory failure, there is rapid conversion to a severe and often lethal metabolic acidosis.

Compensation for respiratory alkalosis is sodium bicarbonate. However, sodium retention following trauma may block this compensation, an alkaline urine.

Respiratory alkalosis depends on a partial degree of respiratory depression. In a large percentage of postoperative patients upon whom blood-gas analysis is performed. In general, these patients have pulmonary, and renal function.



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