Further Section

Am. J. Nephrol. 1985;5:71-72

Quiz of the Month

Answer to Question 1 & Answer to Question 2

A diagnosis of pure chronic respiratory alkalosis might be made in this patient with a blood pH of 7.5, since the level of plasma bicarbonate is appropriate for the level of PCO2 ($\Delta$ bicarbonate = 0.5 $\Delta$ pCC½). However, there is an increased anion gap which suggests the presence of metabolic acidosis as well.

The combination of respiratory alkalosis and high anion gap metabolic acidosis is seen in aspirin intoxication, in patients with sepsis, and in those with liver disease and lactic acidosis. This patient had ingested large quantities of analgesics, most likely containing salicylate, since she had elevated blood levels of salicylate.

Salicylates stimulate the respiratory center and also cause uncoupling of oxidative phosphorylation and induce an increment in lactic acid and keto acid production. Early studies had suggested that respiratory alkalosis was common in adults, and metabolic acidosis was common in children. More recent studies have indicated that the combination of respiratory alkalosis and metabolic acidosis is actually seen in 50% of adults.

The elevated blood pCC½ and bicarbonate and reduced blood pH are consistent with respiratory acidosis. Moreover, the level of plasma bicarbonate concentration is within the range expected for an individual with pure chronic hypercapnia ($\Delta$ HCO3 = 0.35 $\Delta$ pCC½). The history of several days of respiratory distress is very helpful documenting the chronicity of the acid-base disturbance.

The increase in plasma bicarbonate concentration noted in response to chronic elevations in pCC½ occurs in two steps. Initially, a small rise in plasma bicarbonate of a few milliequivalents per liter occurs as a result of titration of nonbicarbonate buffers by H2CO3 arising from the CO2. The largest increment in plasma bicarbonate is generated as the elevated pCC½ stimulates both acid excretion (thereby adding a substantial quantity of bicarbonate to body fluids) and renal bicarbonate reabsorption thus sustaining the elevated plasma bicarbonate concentration. Studies in animals exposed to graded degrees of hypercapnia indicated that 3–5 days were required for a steady state to emerge. At that time, it was found that the plasma bicarbonate concentration had risen by an average of 3.5 mEq/l and the hydrogen ion concentration by 1.7 nEq/l for every 10 mm Hg rise in pCC½. Studies in humans exposed to acute hypercapnia for several hours have confirmed the similarity of response to animals. Examination of acid-base parameters in patients with chronic hypercapnia as a result of chronic lung disease has generally demonstrated a qualitatively similar response.

After therapy both blood pCC¼ and pH were decreased significantly, but blood bicarbonate remained elevated, suggesting the development of metabolic alkalosis. Some degree of metabolic alkalosis commonly complicates the course of chronic respiratory acidosis for the following reasons: (1) The rate of mobilization and

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excretion of carbon dioxide by the lung is much more rapid than the rate of mobilization and excretion of bicarbonate by the kidney. Therefore, metabolic alkalosis will be seen in all patients, if ventilation is rapidly improved as in this case. This type of metabolic alkalosis is transient, and acid-base parameters will return to normal, as bicarbonate is excreted in the urine. (2) During the adaptation to chronic hypercapnia, there are increased losses of chloride, sodium, and potassium in the urine. If sufficient dietary chloride is not given to the individual, when PCO2 has returned to normal, excretion of bicarbonate by the kidney will be curtailed just as it is in other forms of chloride-responsive metabolic alkalosis. The regimen of dietary sodium restriction contributed to the metabolic alkalosis in this patient.

References
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Announcements
2nd International Milano Meeting of Nephrology
Milan, Italy, September 30 to October 1, 1985
6th Asian Colloquium in Nephrology

The topic of the Meeting will be: Antiglobulins, Cryoglobulins and Glomerulonephritis. Special sessions will be devoted to physiology and pathology of antiglobulins, role of rheumatoid factors in experimental glomerulonephritis, classification of cryoglobulins and biochemical factors inducing cryoprecipitation, immunological aspects of essential mixed cryoglobulinaemia, clinical and morphological findings and role of different therapeutic regimens in essential mixed cryoglobulinaemia.
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This meeting will be held during November 14–17, 1985. On November 18, 1985, there will also be a seminar on renal transplantation. A postgraduate course in nephrology will be given during November 11–21, 1985. All three meetings will take place in Kuala Lumpur, Malaysia. For further information contact: