Further Section


Quiz of the Month

Answers

It should have been obvious that this patient was in critical condition when he arrived in the Emergency Room. A PaCO₂ of 32 mm Hg indicates hyperventilation which should have raised his PaO₂ to 68 mm Hg or more (100 mm Hg – 72 his age). The PaC > 2 of 45 mm Hg indicated severe V/Q mismatch. That his PaCC > 2 was not lower (20 mm Hg or less), despite this degree of hypoxemia, indicated that his ability to hyperventilate had been impaired by fatigue, medications, or impending respiratory failure. The initial blood gases misled the physicians to suppose the patient had a metabolic acidosis. Attempting to interpret the blood gas data without correlating them with his history and physical examination may have cost this patient his life.

The patient’s serum electrolytes showed normal sodium, hyperchloremia, hypocarbia, and a normal anion gap (15.6 mEq/l). This electrolyte pattern could have been compatible with a normal anion gap (hyperchlor-emic) metabolic acidosis, a chronic compensated respiratory alkalosis, or a mixed acid-base disturbance. His urinary electrolytes indicated avid sodium chloride reab-sorption, bespeaking diminished distal sodium delivery. There was no history of diarrhea, urinary tract obstruction, or kidney stones. His urine pH was appropriately acidic, his serum potassium was normal, and his renal function was unimpaired. Thus, there was no reason for him to have had a normal anion gap metabolic acidosis, and, in fact, he did not have one, despite electrolyte and blood gas values which suggested that he did.

This elderly smoker had a background of chronic obstructive pulmonary disease/emphysema, hypertension, coronary artery disease, and congestive heart failure. Because CO₂ is some 20 times more diffusible than O₂, chronic obstructive pulmonary disease is most often associated with hypoxemia and chronic respiratory alkalosis until late in the course, when respiratory failure is accompanied by alveolar hypoventilation and respiratory acidosis. That this patient had chronic respiratory alkalosis was later confirmed by blood gases, obtained 2 months previously in the outpatient clinic, which showed a compensated respiratory alkalosis (pH 7.42, PaCO₂ 20mmHg, PaO₂ 60mmHg, and HC03 13 mEq/l). On arrival in the Emergency Room his arterial blood gases showed overcompensated respiratory alkalosis, not metabolic acidosis.

Stopping the digitalis and diuretic worsened the heart failure and further contracted the effective circulating volume. This induced avid proximal sodium reabsorp-tion and decreased distal sodium delivery. Reduced so-dium-for-hydrogen exchange in the cortical collecting tubule further lowered bicarbonate regeneration. A rising PaCO₂ could either reflect an improvement in respiratory alkalosis with lessening of the hypoxic drive, or it could mean that his condition was worsening because he was no longer able to hyperventilate to the same degree as he had, despite hypoxemia. The low level of PaC > 2 in the face of a relatively high PaCO₂ makes it apparent that his condition was worsening, not improving. When he had stopped taking theophylline, alveolar ventilation began to decline, and, although he was still hyperventilating, the PaCC > 2 rose from a baseline of 20 mm Hg to the admission level of 30 mm Hg. Because congestive heart failure had decreased distal sodium delivery, he was no longer able to compensate for a rising PaCC > 2 through distal HCO₃ regeneration, and the serum [HCO₅] rose only 2 mEq/l, while the pH fell from 7.42 to 7.32.
It is helpful, when analyzing acid-base disturbances, to plot the patient’s data on an acid-base map. The Davenport diagram shown in figure 1 graphically portrays the interrelationships of arterial blood pH, PaCC > 2, and [HCO3]. Point 1 indicates the patient’s usual state of hypoxia-induced respiratory alkalosis with metabolic compensation, at the intersection of pH 7.42 and the PaCC > 2 =
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Fig. 1. Davenport diagram (see text for explanations).
(i.e., distal acidification) not been compromised by heart failure.
It is doubtful that increasing the patient’s inspired oxygen significantly improved his condition, since raising the PaC > 2 would have suppressed his respiratory drive, induced a further rise in PaCO2, and lowered the arterial pH. Continuing or worsening hypoxemia and a falling pH may have reduced myocardial contractility and the inotropic response to catecholamines, aggravated the heart failure, reduced tissue perfusion, and further lowered the pH through lactic acidosis. This positive feedback loop might have produced the respiratory arrest. If he had been admitted directly to the Medical Intensive Care Unit, promptly intubated, and adequately ventilated, this patient might have survived.

20 mm Hg isobar. Point 2 is a plot of his acid-base profile on admission, at the intersection of pH 7.32 and the PaCCh = 30 mm Hg isobar. The arrow connecting point 1 (compensated respiratory alkalosis) with point 2 (over-compensated respiratory alkalosis) graphically portrays the effect produced by reduction of alveolar ventilation. The dotted arrow connecting point 1 with point 3 is the path his acid-base profile would have followed as alveolar ventilation declined, had metabolic compensation
References

Announcement
Second International Conference on Systemic Lupus Erythematosus
Singapore, November 26-30, 1989
This meeting will combine clinical and scientific plenary sessions, seminars, and free papers. For further details from the organizers, please contact: