Answers

(1) Strictly speaking, he did not have an acid-base disturbance because his arterial blood pH and PCO2 were ‘normal’; functionally speaking, he did. This case exemplifies the dictum that one cannot interpret arterial blood gases in the absence of a history. The patient presented with acute congestive heart failure superimposed on underlying pulmonary emphysema. Severe hypoxemia was caused both by a ventilation:perfusion mismatch (shunting) and a diffusion abnormality. Without a mixed venous PC > 2 the initial shunt fraction cannot be calculated exactly, but it is always increased in heart failure, pneumonia, atelectasis, or pulmonary thromboembolism. At sea level, the alveolar PC > 2 (PAO2) may be calculated

\[ \text{PaO}_2 = (760 - 47) \times \text{FiO}_2 - \text{PaCO}_2 / 0.8. \]

In Lubbock, Tex. (altitude 3,241 ft), PB is 680 mm Hg; therefore, his initial PaC’2 calculates to (633 X 0.21) – 40/0.8 = 83 mm Hg and (PAO2-PaO2) = 83-46 = 37 mm Hg (normal < 15 mm Hg, increasing with age) which indicates that alveolar edema caused a severe diffusion abnormality. Through arterial chemoreceptors, a p( > 2 < 60-70 mm Hg stimulates the medullary respiratory center and should induce alveolar hyperventilation and respiratory alkalosis. Respiratory alkalosis is the acid-base disturbance expected with both heart failure and chronic obstructive pulmonary disease. This patient, however, presented with pH 7.40, PCO2 40 mm Hg, and tCO2 24 mmol/l, despite severe hypoxemia (pO2 46 mm Hg). Tachypnea, subjective dyspnea, and labored breathing indicate an intact respiratory drive; therefore, alveolar hypoventilation relative to oxygen demand was likely caused by airway disease (bronchial edema, secretions, mucus plugs, or bronchospasm), and, far from indicating normal acid-base balance, the initial arterial blood gases suggest a severe, life-threatening respiratory disturbance. He should have had respiratory alkalosis, but did not. Administration of 28% O2 by Venturi mask diminished the drive to hyperventilate sufficiently to disclose the underlying alveolar hyperventilation and respiratory acidosis, as the PaCC > 2 rose to 54 mm Hg and the pH fell to 7.30.

On the 4th hospital day, after losing 8 kg of edema fluid, the arterial blood gases reveal a pure metabolic alkalosis (pH 7.55, PCO2 40 mm Hg). In congestive heart failure the effective circulating volume is contracted, the renin-angiotensin II-aldosterone axis is stimulated, the renal proximal reabsorption is increased, and the renal distal delivery is diminished. Hyperaldosteronism alone is insufficient to enhance distal potassium and hydrogen secretion in the absence of adequate distal delivery and does not usually cause either hypokalemia or metabolic alkalosis. Administration of furosemide in this setting blocks sodium, potassium, and chloride reabsorption in the thick ascending limb and increases distal delivery suf-ficently that aldosterone-stimulated Na+ for K+ and H+ exchange in the cortical collecting tubule results in hypokalemic metabolic alkalosis. Potassium depletion lowers the glomerular filtration rate and maintains the alkalosis by reducing the filtered bicarbonate load and directly stimulating renal acidification. Chloride depletion reduces the glomerular filtration rate through tubuloglome-rular feedback, prevents sodium and potassium conservation, and diminishes distal bicarbonate secretion.
Alkalosis decreases tissue oxygen delivery by shifting the Bohr curve to the left and may exacerbate ischemic heart disease and congestive heart failure. Alkalosis, like administration of oxygen, reduces the respiratory drive in patients with chronic obstructive pulmonary disease and may make weaning from a ventilator problematical. Other factors in this setting that may interfere with successful weaning include the following: (a) heart failure and pulmonary congestion interfere with gas exchange and increase the work of breathing; (b) administration of carbohydrate in excess of energy expenditure induces lipogenesis and causes excessive CO2 production (raises the RQ above 0.85); (c) protein-energy malnutrition in chronic obstructive pulmonary disease and decreased hepatic protein synthesis by a congested liver waste weaken respiratory muscles and lower resistance to nosocomial infections; (d) hypokalemia, hypophosphatemia, or hypomagnesemia weaken respiratory muscles; (e) excessive bronchial secretions, mucus plugs, and bronchospasm decrease alveolar ventilation and increase the work of breathing, and (f) a patient whose baseline arterial blood gases reflect chronic respiratory acidosis and hypoxemia and which were maintained at more ‘normal’ values during mechanical ventilation may experience increasing dyspnea, hypoxemia, and respiratory acidosis during weaning.

(4) Metabolic alkalosis is corrected by treating the condition that has prevented renal excretion of excess alkali. In renal failure, dialysis against low-bicarbonate dialysate (22 mmol/l) is usually effective. In primary hyperaldosteronism treatment consists of spironolactone (25-400 mg/day). In congestive heart failure, afterload reduction (by administration of captopril, hydralazine, verapamil, minoxidil, prazosin, labetalol, or nitroprusside), infusion of low-dose dopamine (2-3 ug/kg/min), and administration of digitalis increase renal blood flow, glomerular filtration rate, and filtered bicarbonate load, while reducing proximal reclamation. True volume contraction is corrected with NaCl, potassium depletion with KC1, and chloride depletion with any chloride salt. In this case, stopping the diuretics and administration of sufficient NaCl and KC1 to restore fluid and electrolyte balance might be adequate to reduce renal acidification and restore acid-base balance as long as congestive heart failure did not supervene. However, when the presence of heart failure prohibits saline administration, requires continued administration of diuretics, and prevents restitution of the effective circulating volume, administration of acetazolamide may restore the acid-base balance in patients having sufficient renal function.

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