Factors Influencing the Serum Osmolar Gap

To the Editor:

The case of isopropyl alcohol intoxication that was published in the "Pulmonary and Critical Care Pearls" section of the July 2001 issue of CHEST® made for interesting reading. However, there were a few comments that may need clarification. The authors stated that "an elevated serum osmolar gap (i.e., > 10 mOsm/L) represents decreased serum water or the presence of low-molecular-weight solutes." While the latter part of the statement is undisputed, the former cannot be made as a general statement. Hypernatremia caused by reduced water content can increase serum osmolarity but does not cause an increased osmolar gap, since the calculated and measured osmolarity are both high. The increased level of Na⁺ increases serum osmolality but does not result in a widened serum osmolar gap. As for the second point, we acknowledge that nonketotic hyperglycemia results in an increased serum osmolarity, without a widened osmolar gap. With respect to the statement that starvation ketosis is associated with a normal anion gap, this statement is not entirely correct. There is a mild anion gap acidosis with starvation, but it is not as profound as that observed with diabetic ketoacidosis. During starvation ketosis, lipids become a significant source of fuel following ≥3 days of starvation. In addition, low insulin levels and increased serum glucagon concentrations result in the hepatic generation of ketones, and consequently incomplete oxidation of fatty acids and accumulation of ketones results in a mild metabolic acidosis.1,2 During a prolonged fast, the degree of ketosis is limited, likely due to a rise in insulin secretion that is secondary to ketonemia, thereby limiting the availability of free fatty acids.

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REFERENCES

Behçet Disease, Adamantiades-Behçet Disease, or Hippocrates-Adamantiades-Behçet Disease?

To the Editor:

Tirilomis (December 2001)1 was correct in stating that the first description in contemporary times of Behçet disease was made by the Greek ophthalmologist Benedict Adamantiades in 1931, and that the disease was described for the second time, independently, by the Turkish dermatologist Hulusi Behçet in 1937. Actually, according to Kaklamani et al.2 before publishing in

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