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Initiation of CPB can cause acidosis dependent on prime fluids

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Keywords

Acid base physiology, anion gap, hypoalbuminaemia, strong ion gap

Comments

This interesting paper highlights the complexity of the area of acid base balance and possible iatrogenic manipulations. The use of cardiopulmonary bypass (CPB) as a model allows rapid infusion and assessment before homeostatic mechanisms can compensate. Both groups studied developed an acidosis within 2 min of prime fluid delivery and the authors judged it reasonable to attribute the acidosis to the prime fluids. Acid base balance, as based on the work of Stewart (see Additional information), describes three independent factors that induce changes in pH; these are partial pressure of carbon dioxide, the strong ion difference and the total concentration of weak acids. As the strong ion difference becomes smaller the pH decreases. On this basis the acidosis in group one can be calculated as being due to serum chloride whereas plasma acetate and gluconate were responsible for the acidosis in group two. The rapid liver uptake of acetate and gluconate then returned the base excess to baseline levels by the end of the study. Although the changes in base deficit are relatively small (decrease of <1.5 mEq/l in group one at the end of the study), it may be enough to provoke concern. This result is convincing as it was consistently and significantly shown in the sample group of 22 patients. The recognition of acid base disturbance may be especially important when the base deficit is considered to be related to hypovolaemia and further infusions of chloride-bearing solutions are given. This work provides insight into the nature of the problem and should prompt more investigation into iatrogenic changes in acid base balance in the ICU.

Introduction

The development of an acidosis shortly after commencing CPB is common, but the mechanism for this is poorly understood. It may be due to an increase in serum lactate or chloride or simply a decrease in bicarbonate due to dilution (an increase in plasma volume of around 50% occurs on initiating CPB). The study aimed to test the hypothesis that the pump prime fluids were responsible for the acidosis and that the characteristics of the acidosis would vary if the prime was altered.

Methods

- A randomised double blind study
- A total of 22 patients undergoing coronary revascularisation were studied. Exclusion criteria included renal dysfunction (elevated creatinine) or pre-existing acid base disturbance
- Group one received 500 ml polygeline and 1000 ml Ringers injection as prime. Ionic concentration sodium 146 mEq/l, chloride 151 mEq/l, potassium 4.4 mEq/l and calcium 6.8 mEq/l. Group two received 1500 ml plasmalyte 148 as prime. Ionic concentration sodium 140 mEq/l, chloride 98 mEq/l, potassium 5 mEq/l, magnesium 3 mEq/l, acetate 27 mEq/l and gluconate 23 mEq/l
- Samples were collected immediately before bypass, 2 min after CPB at full flow and at the end of the procedure
- Arterial blood gas samples were analysed and serum sodium, potassium, calcium, magnesium, chloride, phosphate, albumin and lactate measured

Results

Marked hyperchloraemia, hypercalcaemia and hypoalbuminaemia were observed in group one. These changes caused a non-anion gap hyperchloraemic acidosis, which was only partially compensated by the hypoalbuminaemia induced alkalosis. There was a significant decrease in base excess which returned towards normal during the study. However, homeostatic mechanisms did not completely reverse the acidosis and all patients were admitted to the ICU with residual metabolic acidosis. Lowered serum albumin and an anion gap acidosis which brought about a decrease in base excess were observed in group two. This anion gap acidosis was not due to lactate since when this was removed from the calculations the acidosis remained unchanged. By the end of the study homeostatic mechanisms had resolved the acidosis.

Additional information

A concise account of the methodology used is given in the original paper, it is beyond the scope of this report to reproduce it here.

Several other paper reports link to related papers:

Scheingraber S, Rehm M, Schmisch C, Finsterer U: **Rapid saline infusion produces hyperchloremic acidosis in patients undergoing gynecological surgery.** *Anesthesiology* 1999, **90**:1265-1270.

Rehm M, Orth V, Scheingraber S, Kreimeier U, Brechtelsbauer H, Finsterer U: **Acid–base changes caused by 5% albumin versus 6% hydroxyethyl starch solution in patients undergoing acute normovolemic hemodilution. A randomized prospective study.** *Anesthesiology* 2000; **93**:1174-1183.

Stewart PA: **Modern quantitative acid–base chemistry.** *Can J Physiol Pharmacol* 1983, 61:1444-1461

There is also an accompanying Editorial View

Prough DS: **Acidosis associated with perioperative saline administration: Dilution or delusion?** *Anesthesiology* 2000; **93**:1167-1169.

References

1. Liskaser FJ, Bellomo R, Hayhoe M, Story D, Poustie S, Smith B, Letis A, Bennett M: Role of pump prime in the etiology and pathogenesis of cardiopulmonary bypass-associated acidosis. Anesthesiology . 2001, 93: 1170-1173.