Communications to the editor

Gastric Intramural PCO₂ in Peritonitis and Shock

To the Editor:

As experienced users of the tonometric technique for the estimation of gastric intramural pH, both in humans and the laboratory setting, we read with great interest the article regarding gastric intramural CO₂ measurements in a rat peritonitis model by Desai et al.,¹ which appeared in the October 1993 issue of Chest. While we agree with the overall sentiment of the report, that is, the suggestion that gastric mural hypercarbia is a far more sensitive indicator of hypovolemia than of sepsis, we do believe many of the conclusions are unduly harsh on the tonometric technique for the estimation of gastric intramural pH (pHi). We have outlined these below.

First, as pointed out in the text, the bacterial inoculum produced by caecal ligation was uncontrolled. Therefore, although the common end point of death was reproducible, it does not seem that many of the measured physiologic changes leading up to death were reproducible. The data are presented as means and standard deviations, and the statistical tests used are suitable for normally distributed data. This is a very reasonable assumption for the control rats but not for many of the changes seen in the septic rats. For example, the gastric intramural PCO₂ recorded at 2 h gave a mean of 55 mm Hg with a range of two standard deviations about the mean of 11 to 99 mm Hg; likewise, the blood pressure at 4 h showed a mean value that was exactly twice the standard deviation. This makes the observed changes reported and the statistical significance of them more difficult to interpret.

Second, the directly measured pH (calculated from the [H⁺]) was higher than the arterial pH at baseline. No mention is made of this in the discussion. This is of great importance in interpreting the discrepancies between the bicarbonate calculations shown in Figure 4. Was this a real effect, as we might expect if the rats’ stomachs are still producing acid, or an artifact as the direct measurement of [H⁺] in the stomach was less reproducible. The coefficient of variation was about four times greater for the direct measurements at baseline compared with the arterial measurements.

Third, no information is provided on calibration of the various pH and PCO₂ electrodes. The two different bicarbonate calculations given in Figure 4, one for the gastric wall and one for the arterial blood, are made using completely separate sets of electrodes. All of these systems have some bias and imprecision. Unless we know that the two separate CO₂ electrodes, for example, were calibrated using solutions of a known CO₂ tension, then these potentially fascinating results are difficult to interpret.

Fourth, over the course of the experiment, the septic rats hyperventilate and thus maintain their mean arterial pH. This has a direct influence on the absolute level of the gastric intramural CO₂. This blunting effect can be resolved by observing over time the changes in gradients between arterial PCO₂ and gastric PCO₂. A similar technique has been used for the venoarterial PCO₂ gradient but not for the gastric-arterial CO₂ gradient, which was of a greater magnitude on the 2 and 4 h figures shown (26 vs 5 mm Hg and 75 vs 10 mm Hg respectively). It would have been interesting to know if these changes were significant over the course of the experiment and how they compared with the other end points, as this might have put a different complexion on the conclusions drawn.

Finally, although not the purpose of the study, it would have been most helpful to know how the intramural pH calculated from the directly measured PCO₂ and arterial bicarbonate compared with the directly measured gastric intramural pH and to know how the calculated pH changed with time. This would seem a reasonable thing to report as the abstract conclusion casts doubt over the validity of this now commonly used calculation. From the figures available in the paper, the two values, by our calculations, seem to be quite similar and even lie within a clinically acceptable range, considering the number of potential errors in their respective calculations. As clinicians who, like so many others, have been very impressed with the tonometer as a clinical tool yet share the authors’ reservations over the concept of calculating pH₁ rather than just measuring the gastric PCO₂, we are obliged to say that this comes as a pleasant surprise.

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REFERENCE


To the Editor:

We are pleased to respond to the letter of Drs. Mythen and Salmon. Increases in gastric intramural PCO₂ and H⁺ were observed during experimentally induced peritonitis as the cause of circulatory shock. We concluded that the prognostic value of intramural H⁺ was better than that of PCO₂ in this setting and suggested, on the basis of earlier studies with hemorrhagic and anaphylactic shock, that gastric acid base changes were in part contingent on the cause of the low visceral blood flow state. More specifically, predictability of outcome based on intramural PCO₂ measurements was unimpressive in this setting of septic shock when contrasted with that of hemorrhagic shock in which decreases in arterial pressure were coincident with the onset of hypercarbic acidosis of the stomach wall.

The standard student’s t test requires both normally distributed data and equal variances. Gastric PCO₂ measurements were normally distributed but variances were unequal. We viewed nonparametric tests in this setting as unnecessarily conservative. We,