model of peritonitis and septic shock accurately underscores the pervasive misunderstanding of tonometric CO₂ values. The authors simultaneously recorded stomach wall intramucosal PCO₂ and hydrogen ion concentration (H⁺) from which mucosal bicarbonate values were calculated. The number of animals used in each group was either not reported or quite low.

When considered in the form of pH, the mucosal H⁺ data confirms the accuracy of estimating intramucosal pH with mucosal PCO₂ and arterial bicarbonate. We calculated intramucosal pH from the reported values of arterial pH, PCO₂, and mucosal PCO₂ using the Henderson-Hasseloch equation. This information is directly compared with measured pH from the H⁺ data of Desai et al in Figure 1. The work of Desai et al, far from detracting from the significance of gastric mucosal pH measurements, provides experimental evidence of the validity of the tonometric estimation of gastric mucosal pH from mucosal PCO₂.

Tissue and venous PCO₂ normally should vary with arterial PCO₂. The authors noted that the gradient between arterial and venous PCO₂ increased significantly at 60 min reflecting that the normally small gradient may widen in shock. This useful gradient is extended as an organ specific indicator of ischemia when the tissue PCO₂ from a tonometric measurement is compared with the arterial PCO₂. The authors note that 2 h after the onset of sepsis and peritonitis, the intramucosal PCO₂ rose on the average of 10 mm Hg while the arterial PCO₂ fell 16 mm Hg and atrial PCO₂ fell 10 mm Hg compared with baseline. The rise in intramucosal PCO₂ did not obtain statistical significance until 4 h after the onset of peritonitis and sepsis. The difference between the average arterial PCO₂ fell 10 mm Hg and gastric intramucosal PCO₂ at 2 h was 26 mm Hg which, even with a small number of animals, maybe statistically significant (Fig 2). Even in the control animals, however, the intramucosal PCO₂ rose 5 mm Hg over the course of the experiment while arterial PCO₂ fell 8 mm Hg, which indicates that the gradient between arterial and mucosal PCO₂ rose from 4 mm Hg at baseline to 13 mm Hg at 4 h. Perhaps the experimental preparation was not stable.

The authors have corroborated the utility of tonometric monitoring and current method of estimating intramucosal pH. We applaud their effort but disagree with their interpretation of the data.

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REFERENCE


To the Editor:

Our article, "Gastric Intramural PCO₂ During Peritonitis and Shock" (Chest 1993; 104:1254-58), addressed increases in gastric intramural CO₂ and H⁺ in experimentally induced peritonitis with circulatory shock. It concluded, "acid-base measurements on the stomach wall are indicative of the severity of visceral ischemia and therefore of perfusion failure. However, the prognostic value of intramural H⁺ was better than that of PCO₂. The rate of change of these parameters differs contingent on the mechanism underlying the low visceral blood flow state. Predictability of intramural H⁺ and PCO₂ measurements with respect to outcome was earlier with hemorrhagic than with septic shock. During peritonitis, arterial pressure and arterial blood lactate levels were earlier indicators of the severity and outcome than the gastric intramural acid base measurements."

Your letter addresses "the accuracy of estimating gastric wall pH with the mucosal PCO₂ and arterial bicarbonate." Accordingly, it addressed only our coincidental observation that there were differences between the bicarbonate computed directly from the measured gastric intramural pH and PCO₂ and the bicarbonate computed from arterial blood as shown in Figure 4. As stated in the Abstract and shown in Figure 3, there were five experimental and five control animals. It is true that the number of animals in each group was low but the variances of bicarbonate measure-
ment were modest; and therefore, there were significant differences in gastric wall and arterial blood bicarbonate in the first 120 min of peritonitis, and this is what we found and what we reported. Though we have some difficulty with the focus of your letter, it remains that this paper does not assail the use of tonometry but presents only direct measurements.

Our data are in no way contradictory of your comments regarding gastric mucosal pH and your graphics. In fact, we have gone one step further and calculated the gastric mucosal pH as directly measured, as computed from tissue bicarbonate, and computed from arterial bicarbonate (enclosed). These are to some extent artifactual, of course, since tissue bicarbonate was derived from measured gastric wall pH and Pco2. Nevertheless, neither graph proves other than that in the first 2 h there are differences related to the estimate of bicarbonate. Whether these differences are of clinical importance is entirely contingent on the clinical circumstance. To this extent, we believe you are in error in assuming that tissue and venous Pco2 would predictably vary with arterial Pco2. That applies if the animal or the patient is breathing spontaneously and when the ventilation/perfusion ratio is decreased because cardiac output and pulmonary blood flow are decreased. It is not obtained when the patient fails to ventilate, and there is a progressive increase in arterial Pco2 or in settings of septic shock when cardiac output is increased or normal. Therefore, I'm not sure that as a general rule you would want to always rely on the gradient between stomach wall and arterial Pco2 to refine tonometry measurements.

Both septic rats and control rats were anesthetized for intervals exceeding 4 h. By definition, such controls are used because of the very fact that there are issues of stability of the preparation that included minor reductions in cardiac output and therefore minor increases in arterial blood lactate and decreases in PaCO₂. The paper supported the concepts underlying tonometry, especially with respect to PaCO₂.

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Hemoptysis and Munchausen Syndrome

To the Editor:

We read with great interest the article by Baktari and colleagues¹ that appeared in the March 1994 issue of Chest in which they presented a fascinating case report and excellent review of the literature regarding factitious hemoptysis.

We would, however, like to call attention to our recently published report² on a 19-year-old woman who presented to our institution with a history of acute hemoptysis. She stated that she had been diagnosed with cystic fibrosis (CF) at age 2 and had a long history of CF-related pulmonary complications, including recurrent hemoptysis severe enough to necessitate blood transfusions and bronchial artery embolization. Medical history was also remarkable for placement of a permanent cardiac pacemaker and an appendectomy. Physical examination was remarkable for mild obesity, shallow respirations, a median sternotomy scar, a few scattered lung crackles, and absence of digital clubbing. Laboratory data were consistent with iron deficiency anemia. Chest radiograph showed clear lung fields without hyperinflation.

After admission, the patient continued to complain of hemoptysis and though none was ever witnessed by medical personnel, her hemoglobin concentration fell. Bizarre behavior, inconsistencies in her stories, a relatively benign physical examination, and laboratory data inconsistent with a diagnosis of cystic fibrosis (including sweat chloride values) made us doubt whether indeed she had CF. After psychiatric consultation, a diagnosis of Munchausen syndrome was made. Records from other hospitals eventually revealed that during one admission for hemoptysis, a dark substance the patient claimed to have expectorated was found to be a mixture of iodine solution and shampoo. Actual hemoptysis was never documented.

Our case differs from that of Baktari et al in that our patient did indeed have laboratory evidence of iron deficiency anemia. However, we believe it was not secondary to chronic hemoptysis, but rather self-phlebotomy, a speculation supported by the presence of multiple antecubital scars and noniatrogenic ecchymoses. Therefore, in any patient presenting with a history of hemoptysis, the possibility of factitious illness should always be considered, even in the presence of laboratory data suggesting substantial blood loss.

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REFERENCES


To the Editor:

I agree with Dr. Rusakow and his colleagues that their case differs from ours in that their patient had abnormal laboratory values and that this, in fact, does not exclude the diagnosis of factitious hemoptysis. However, there is one aspect of both of our case reports that are very similar and unfortunately rare. Both cases involved the physician investigating and exposing the attempt at subterfuge by the patient. It appears that most physicians for understandable reasons do not wish to play "Sherlock Holmes." Instead, when they finally realize that their patient has a possible factitious illness, the general response in most cases in our review was to quickly discharge the patient from the hospital with no further investigation.

Some physicians appear to want to cut their losses short, ie, too much time wasted on a nonorganic problem, while other physicians believe that they were ethically handcuffed to check with or warn other physicians in their community. Some physicians took their struggle with ethics one step further. After submitting our report for publication, we found that the patient involved in our case was admitted to a county hospital in our region. Our attempt to communicate our findings of 23 previous hospital admissions and 16 previous bronchoscopies appeared not to be welcomed. Apparently, the patient went on to receive a diagnostic workup anyway.

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Communications to the Editor