CRITICAL CARE

REVIEW

Equipment review: Gastric intramucosal pH measurement

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Introduction

Gastric tonometry has emerged as an attractive, relatively noninvasive technology for assessing gastrointestinal perfusion and oxygenation by detecting acidosis in the gut wall. Several clinical studies have shown that gastric intramucosal acidosis detected by this procedure predicts increased mortality of critcally ill adults in medical and surgical intensive care unit (ICU) settings [1-3], and that it is a better predictor of mortality from critical illness than other mesures of global oxygen delivery and systemic hemodynamics [4]. It has also been suggested that correcting intramucosal acidosis may increase survival in selected critically ill patients [5].

The purpose of this review is to discuss factors influencing in vivo reliability and variability of gastric tonometry, and to analyze the causes of the occasional misinterpretation of its results.

The gastric tonometry technique — causes of misinterpretation of the results

The measurement of gastric mucosal acidosis by gastric tonometry is based on the principle that the fluid in a hollow viscus can be used to estimate gas tensions in the surrounding tissues. The main assumption is that, after a given equilibration time, luminal and mucosal CO_2 partial pressures (PCO_2) will be similar. Consequently, the increased tissue production of CO_2 during hypoxia (from the reaction between hydrogen anions and bicarbonate) can be detected by analyzing the liquid inside the gastric lumen.

Conventional gastric tonometry involves the placement of a modified nasogastric (NG) tube, equipped with a gas-permeable, saline-filled silicone balloon at its tip, into the stomach [6,7] (Fig 1). Allowing enough time for the equilibration of CO_2 between the fluid in the balloon and the gastric lumen (30–90 min), the saline is then aspirated and its PCO_2 determined using a blood gas analyzer. Thus, gastric tonometry can determine intraluminal PCO_2 ($PiCO_2$) which is assumed to be in equilbrium with gastric mucosal PCO_2. Intramucosal pH (pHim) can be calculated by the Henderson-Hasselbach equation, using the $PiCO_2$ value determined by gastric tonometry and the arterial bicarbonate concentration, assuming that the tissue bicarbonate concentration is in the equilibrium with that in the capillaries, which is further assumed to be the same as determined for arterial blood.

Consequently, causes of misleading interpretations of gastric tonometry can be divided as follows:

1. those which 'originate from the patient', and actually confound the logical interpretation based on clinical determinations (mainly, disturbances in systemic acid base balance);
2. local factors in the gastric lumen which can alter the relationship between $PiCO_2$ and mucosal PCO_2, and
3. factors inherent in the technique which can cause erroneous determinations of $PiCO_2$.

Patient factors - from pHim to $DPCO_2$

According to one report [8], gastric tonometry failed to accurately estimate the magnitude of the decrease in tissue pH in conditions of low perfusion (total and partial occlusion of the superior mesenteric artery); direct measurement of pH with microelectrodes was found to be more accurate. One probable explanation for the inaccuracy of gastric tonometry in these low perfusion conditions is the fact that tissue bicarbonate levels are overestimated when determined via arterial concentration. This is because tissue bicarbonate is consumed by buffering protons which are generated by ischemic tissue, therefore reducing the input of fresh bicarbonate. As a clinical example of this phenomenon, Benjamin *et al* [9] reported that calculated pHim became normal with sodium bicarbonate administration in the treatment of a patient with severe systemic acidosis despite laparotomy-proven massive mesenteric

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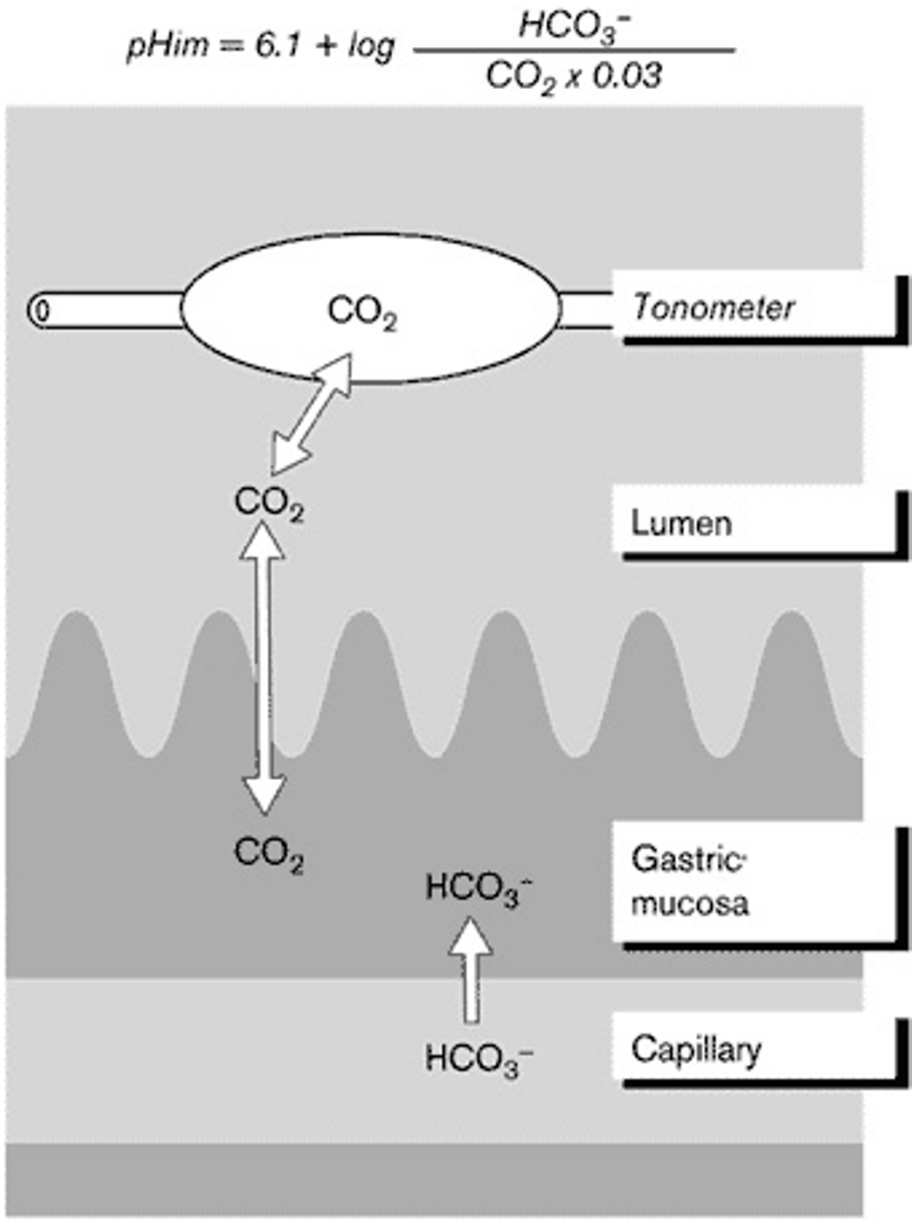

Figure 1 Gastric tonometry determines intraluminal PCO_2 which is assumed to be in equilibrium with PCO_2 in the gastric mucosa. Intramucosal pH (pHim) can be calculated by the Henderson-Hasselbach equation using the PCO_2 value determined by gastric tonometry and the bicarbonate concentration in arterial blood.

ischemia, resulting in a correction of the calculated pHim.

However, an examination of the contribution of the stomach mucosa to its own acid-base status must include an assessment of the arterial blood perfusing that area. If arterial bicarbonate is low due to acidosis arising somewhere other than the gastrointestinal tract, then calculated pHim may be low despite normal $PiCO_2$.

Indeed, studies in critically ill patients have shown a striking correlation between pHim and measurements of metabolic acidosis [4,10]. Therefore, measuring $PiCO_2$ alone is simpler and eliminates arterial bicarbonate as one source of error.

Tissue PCO_2 — the parameter we aim to determine by gastric tonometry — equilbrates almost exactly with capillary PCO_2 and, according to the Fick principle, is related to tissue CO_2 production and arterial CO_2

content, and is inversely related to regional blood flow. Therefore, changes in arterial PCO_2 ($PaCO_2$) should affect tissue PCO_2. It has been shown that respiratory acidosis leads to tissue hypercarbia in animals [11]. This observation is in agreement with clinical studies showing that patients with hypercapnia have a significantly higher $PiCO_2$ than those without, and that their pHim is also signigficantly lower [12]. The relationship between $PaCO_2$ and $PiCO_2$ has also been observed in individual patients whose $PaCO_2$ was modified by changes in dead space [13]. Thus, pHim assesses not only splanchnic oxygenation, but also arterial acid-base status.

The gradient between $PiCO_2$ and $PaCO_2$ ($DPCO_2$), which is solely determined by the ratio between blood flow and CO_2 production in the tissue, should be a better measure of mucosal perfusion. A new question arises from this assertion - how much does $DPCO_2$ have to increase in order to indicate not just low gastric blood flow, but also anaerobic generation of CO_2 [14]? Schlichtig and Bowles [15] recently reported that the onset of intestinal anaerobiosis in normal dogs occurred when $PiCO_2$ had risen to 65 mmHg and $DPCO_2$ had increased to 25–35 mmHg.

The use of the gradient between intramucosal and arterial pH [16] seems to be more cumbersome and less precise than $DPCO_2$ [17].

Finally, we should ask whether gastric mucosal acidosis always indicates tissue hypoperfusion. Like most studies validating gastric tonometry, the aforementioned article by Schlichtig and Bowles [15] used a model of progressive flow reduction. However, there is evidence from animal sepsis models of intestinal mucosal acidosis that is unrelated to tissue hypoxia [18,19]. Therefore, tissue acidosis in sepsis may result from causes other than cellular dysoxia. It has been suggested that a preferential increase in anaerobic glucose utilization at the expense of oxidative glucose metabolism, even in the presence of adequate or even supranormal oxygen levels, could lead to tissue acidosis [20,21]. This concept has important clinical implications - we should take particular care when attending to septic patients with gastric mucosal acidosis because it may not in fact be incontrovertible evidence of tissue hypoperfusion [21].

Local factors that can influence gastric tonometry

The relationship between high $PiCO_2$ and mucosal ischemia in the stomach is invalidated in cases where CO_2 is produced in the lumen. Buffering of gastric acid by bicarbonate, either from an exogeneous source, or from gastric or duodenal secretions, is a major cause of increased intraluminal CO_2.

Studies in human volunteers have shown that administration of ranitidine, an H_2 receptor blocking agent, reduces the error in $PiCO_2$ measurement [22-24]. Consequently, inhibition of acid secretion is now considered to be mandatory for proper assessment of intraluminal PCO_2. However, this recommendation has not been validated in critically ill patients; studies suggest that the use of H_2-blockers in the critically ill has no effect on the assessment of intraluminal PCO_2 [25,26]. Discrepancies between results in healthy volunteers and critically ill patients may be related to a reduced gastric acid secretion in the latter as a result of compromised visceral perfusion [27-29]. However, these studies of critically ill patients were performed with small patient samples and over a short period, without changes in their hemodynamic status. The results may, therefore, not be applicable to hemodynamically unstable patients.

The effect of other treatments commonly administered via an NG tube on the measurement of pHi by gastric tonometry remains unclear. Elsewhere [30] we studied the effect of sucralfate, which is widely used for stress ulcer bleeding prophylaxis because it does not significantly reduce gastric pH and tends to decrease the additional risk of gastric bacterial overgrowth. Our results suggested that enteral administration of sucralfate does not alter the determination of pHim by gastric tonometry in critically ill patients.

Enteral feeding may also affect accurate assessment of $PiCO_2$. Once food enters the stomach it stimulates secretion of gastric juice and bicarbonate ions. This combination, along with the digestion of nutrients, may generate CO_2 inside the gastric lumen. In animals, it has been shown that gastric instraluminal PCO_2 increases after feeding [31]. This effect has also been observed in asymptomatic subjects [32] and in critically ill patients [33]. Consequently, it is currently recommended that enteral feedings be discontinued for about 1–2 h before measuring pHim. This period may need to be longer in patients with delayed gastric emptying.

In normal conditioins, blood flow to each portion of the gastrointestinal tract is proportional to the level of local activity. Blood flow increases after feeding by 100–150% for 3–6 h. Consequently, if the flow cannot increase appropriately, enteral feeding may result in gastrointestinal hypoxia with mucosal acidosis. In fact, the presence of mucosal acidosis after feeding has been used to detect chronic gastric ischemia [32].

Factors related to the technique — from saline to air

Gastric tonometry presents the main sources of problem — the time required from equilibration, the measurement of saline PCO_2, and the potential loss of CO_2 during transport of the sample.

The first of these, the time required for equilibration, is an important factor. Equilibration follows Fick's law

of diffusion. Complete equilibration of the tonometer solution with mucosal PCO_2 requires at least 60–90 min, with shorter times resulting in the measurement becoming significantly more variable.

Measurement of saline PCO_2 is also an important source of error and, as shown by Takala *et al*, depends on both the analyzer used and the actual PCO_2 level [34]. Most analyzers underestimated saline PCO_2 by 5–19%. Notably, the performance of all analyzers markedly improved when a buffer solution was used. Why, then, not use a buffer solution instead of saline? The problem is that due to the higher CO_2-binding capacity of the buffer, more time is required for the equilibration of tissue and sample CO_2, reducing the ability of the intragastric tonometer to respond to changing tissue PCO_2.

Another alternative to the use of saline is air. The use of 'balloonless' air tonometry has been reported in animals, and Salzman et al have demonstrated a good correlation between tonometric PCO_2 measurements obtained simultaneously from samples of air and saline solution [11]. Although, in the above study the air was analyzed by a blood gas analyzer, its use has opened by the possibility of determining intramucosal PCO_2 by capnography.

Capnography is the basis of some new systems for nearly continuous monitoring of intramucosal PCO_2. One recently validated system allows continuous recirculation of gas through the balloon of the tonometer [35]. The new system was compared with a conventional tonometer in an in vivo experiment on dogs with induced hypoxia. The air system showed a higher sensitivity in detecting tissue hypoxia. The probable explanation for the greater sensitivity of the continuous monitoring system was that the recirculating gas was already in equilibrium with $PiCO_2$ immediately before the induction of hypoxia.

An automated tonometric system which also uses capnography with a conventional tonometer is now commercially available [36]. This system allows concomitant determination of end-tidal CO_2 with $PiCO_2$ to estimate $DPCO_2$. This system works by introducing a certain amount of air into the balloon, which is periodically aspirated in order to determine PCO_2. The same air is sent back to the balloon after determining tonometric PCO_2. Therefore, as with the system described above that uses recirculating gas, it increases sensitivity to changes in intramucosal PCO_2, allowing sampling times shorter than 30 min.

In addition to higher sensitivity, the expected advantages of these systems are:

1. shorter sampling times;

2. the selected time for equilibration is always constant, and

3. the fact that there is no need for saline aspiration and transport to a blood gas analyzer, avoiding the risk of CO_2 loss during transport and, thus, reducing the actual number of error sources in the technique.

Conclusion

In summary, gastric tonometry is relatively simple technique, but obtaining reliable results and interpreting them accurately requires a comprehensive knowledge of the technique and careful attention to the smallest detail. Frequency of measurement is limited by the time required and staff intervention involved. The use of air instead of saline, and PCO_2 determination by capnography seem to be promising ways of avoiding some of the problems that the technique presents.

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References

1. Gys T, Hubens A, Neels H, Lauwers LF, Peeters R: **Prognostic value of gastric intramural pH in surgical intensive care patients.** *Crit Care Med* 1988, **16**:1222-1224.
2. Doglio GR, Pusajo JF, Egurrola MA, *et al*: **Gastric mucosal pH as a prognostic Index of mortality in critically ill patients.** *Crit Care Med* 1991, **19**:1037-1040.
3. Gutierrez G, Bismar H, Dantzker Dr, Silva N: **Comparison of gastric intramucosal pH with measures of oxygen transport and consumption in critically ill patients.** *Crit Care Med* 1992, **20**:451-457.
4. Maynard N, Bihari D, Beale R, *et al*: **Assesment of splanchnic oxygenation by gastric tonometry in patients with acute circulatory failure.** *JAMA* 1993, **270**:1203-1210.
5. Gutierrez G, Palizas F, Doglio G, *et al*: **Gastric Intramucosal pH as a therapeutic index of tissue oxygenation in critically ill patients.** *Lancet* 1992, **339**:195-199.
6. Fiddian-Green RG, McGough E, Pittenger G, *et al*: **Predictive value of intramural pH and other risk factors for massive bleeding for stress ulceration.** *Gastroenterology* 1983, **85**:613-620.
7. Fiddian-Green RG, Baker S: **Predictive value of the stomach wall pH for complications after cardiac operations: comparison with other monitoring.** *Crit Care Med* 1987, **15**:153-156.
8. Antonsson JB, Boyle CC III, Kruithoff KL, *et al*: **Validation of tonometric measurement of gut intramural pH during endotoxemia and mesenteric occlusion in pigs.** *Am J Physiol* 1990, **259**:G519-G523.
9. Benjamin E, Polokoff E, Oropello JM, Leibowitz AB, Lberti TJ: **Sodium bicarbonate administration affects the diagnostic accuracy of gastrointestinal tonometry in acute mesenteric ischemia.** *Crit Care Med* 1992, **20**:1181-1183.
10. Boyd O, Mackay CJ, Lamb G, Bland JM, Grounds RM, Bennett ED: **Comparison of clinical information gained from routine blood-gas analysis and from gastric tonometry for intramural pH.** *Lancet* 1993, **341**:142-146.
11. Salzman AL, strong KE, Wang H, Wollert S, Vandermeer TJ, Fink MP: **Intraluminal 'balloonless' air tonometry: a new method for determination gastrointestinal mucosal carbon dioxide tension.** *Crit Care Med* 1994, **22**:126-134.
12. Baigorri F, Mas A, Calvet X, *et al*: **Efecto de la hipercapnia sobre la determinación del pH intramucoso gástrico.** *Med Intensiva* 1995, **19**:239-244.
13. Mas A, Baigorri F, Joseph D, *et al*: **Effect of acute changes of $PaCO_2$ in the assessment of gastric perfusion by tonometry.** *Intensive Care Med* 1996, **22 (suppl 3)**:S437-.
14. Russell JA: **Gastric tonometry: does it work?** *Intensive Care Med* 1997, **23**:3-6.

15. Schlichtig R, Bowles SA: **Distinguishing between aerobic and anaerobic appearance of dissolved CO_2 in intestine during low flow.** *J Appl Physiol* 1994, **76**:2443-2451.
16. Fiddian-Green RG: **Management of gastric intramucosal acidosis.** *In: Yearbook of Intensive Care and Emergency Medicine. Edited by Vincent JL. Berlin: Springer-Verlag,* 1994, 217-224.
17. Schlichtig R, Metha N, Gayowski TJP: **Tissue-arterial PCO_2 difference is a better marker of ischemia than intramural pH (pHi) or arterial pH-pHi difference.** *J Crit Care* 1996, **11**:51-56.
18. Heard SO, Baum TD, Wang HL, Rothschild HR, Fink MP: **Systemic and mesenteric O_2 metabolism in endotoxic pigs:effect of graded hemorrhage.** *Circ Shock* 1991, **35**:44-52.
19. Van der Meer TJ, Wang H, Fink MP: **Endotoxemia causes ileal mucosal acidosis in the absence of mucosal hypoxia in a normodynamic porcine model of septic shock.** *Crit Care Med* 1995, **23**:1217-1226.
20. Russell JA, Baigorri F: **Is serum lactate a clinically useful parameter?** *Crit Care Alert* 1995, **3**:14-16.
21. Fink MP: **Does tissue acidosis in sepsis indicate tissue hypoperfusion?** *Intensive Care Med* 1996, **22**:1144-1146.
22. Heard SO, Helsmoortel CM, Kent JC, Shahnarian A, Fink MP: **Gastric tonometry in healthy volunteers:effect of ranitidine on calculated intramural pH.** *Crit Care Med* 1991, **19**:271-274.
23. Kolkman JJ, Groeneveld ABJ, Meuwissen SGM: **Effect of ranitidine on basal and bicarbonate-enhanced intragastric pCO_2: a tonometric study.** *GUT* 1994, **35**:737-741.
24. Parviainen I, Vaisänen O, Ruokonen E, Takala J: **Effect of nasogastric suction and ranitidine on the calculated gastric intramucosal pH.** *Intensive Care Med* 1996, **22**:319-323.
25. Maynard N, Atkinson S, Mason R, Smithies M, Bihari D: **Influence of intravenous ranitidine on gastric intramucosal pH in critically ill patients.** *Crit Care Med* 1994, **22**:A79.
26. Baigorri F, Calvet X, Duarte M, *et al*: **Effect of ranitidine treatment in gastric mucosal pH determinations in critically ill patients.** *Intensive Care Med* 1994, **20 (suppl 1)**:S3.
27. Stannard A, Hutchinson A, Morris DL, Byrne A: **Gastric exocrine 'failure' in critically ill patients: incidence and associated features.** *Br Med J* 1988, **296**:155-156.
28. Hunt RH: **Acid, bacteria and the mucosal barrier: aspiration or translocation as the cause of pneumonia?** *Eur J Gastroenterol Hepatol* 1992, **4**:881-884.
29. Higgins D, Mythen MG, Webb AR: **Low intramucosal pH is associated with failure to acidify the gastric lumen in response to pentagastrin.** *Intensive Care Med* 1994, **20**:105-108.
30. Calvet X, Baigorri F, Duarte M, *et al*: **Effect of sucralfate on gastric intramucosal pH in critically ill patients.** *Intensive Care Med* 1997, **23**:738-742.
31. Rune SJ, Henriksen FW: **Carbon dioxide tensions in the proximal part of the canine gastrointenstinal tract.** *Gastroentrology* 1969, **56**:758-762.
32. Fiddian-Green RG, Stanley JC, Nostrant T, Phillips D: **Chronic gastric ischemia. A cause of abdominal pain or bleeding identified from the presence of gastric mucosal acidosis.** *J Cardiovasc Surg* 1989, **30**:852-859.
33. Marik PE, Lorenzana A: **Effect of tube feedings on the measurement of gastric intramucosal pH.** *Crit Care Med* 1996, **24**:1498-1500.
34. Takala J, Parviainen I, Siloaho M, Ruokonen E, Hamalainen E: **Saline PCO_2 is an important source of error in the assessment of gastric intramucosal pH.** *Crit Care Med* 1994, **22**:1877-1879.
35. Guzman JA, Kruse JA: **Development and validation of a technique for continuous monitoring of gastric intramucosal pH.** *Am J Respir Crit Care Med* 1996, **153**:694-700.
36. Lahtinen P, Valta P, Takala J: **Gas tonometry - evaluation of a new method and comparison with saline tonometry.** *Intensive Care Med* 1996, **22 (suppl 3)**:S362.

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