Gastric Tonometry: The Hemodynamic Monitor of Choice (Pro)

Stephen O. Heard

Chest 2003;123;469-474
DOI: 10.1378/chest.123.5_suppl.469S

Gastric Tonometry*

The Hemodynamic Monitor of Choice (Pro)

Stephen O. Heard, MD, FCCP

Controversy exists as to the best means to monitor the critically ill patient and the appropriate end points of therapy. Use of global hemodynamic or metabolic parameters may be normal in the patient who has not been completely or adequately resuscitated. Decreased perfusion to the gut is not well tolerated and may contribute to the development of the multiple organ dysfunction syndrome. Gastric tonometry is a minimally invasive way to monitor splanchnic perfusion in the critically ill patient. Data suggest that tonometry is useful for outcome prognostication and for detection of early hypovolemia. In addition, use of gastric intramucosal pH or mucosal-arterial CO_2 gap as end points of resuscitation may be superior to other conventional whole-body parameters. For these reasons, gastric tonometry must be considered the hemodynamic monitor of choice. ***(CHEST 2003; 123:469S–474S)***

Key words: gastric tonometry; hemodynamic; hypoperfusion; hypovolemia

Abbreviations: $\dot{D}O_2$ = oxygen delivery; pHi = intramucosal pH

Introduction: Traditional Monitoring

Critically ill patients are most often monitored by measuring vital signs, urine output, indexes of cardiac performance and oxygen transport, and chemical indicators of metabolic activity, such as lactate. These methods are sometimes inadequate for a number of reasons, including the following: (1) BP may be normal despite a low blood volume or cardiac index; (2) heart rate can be affected by multiple variables that are not germane to the adequacy of resuscitation (*eg*, pain); (3) urine output can be confounded by the hormonal milieu of the patient, including antidiuretic hormone and aldosterone; and (4) measurements of central filling pressures, cardiac index, oxygen transport variables, arterial blood gases, and serum lactate assess global perfusion and will not always identify localized peripheral organ hypoperfusion.

A monitor is still needed to identify earlier, and more accurately, those patients at highest risk of ischemic organ failure and death, especially when conventional indicators are normal. Such a monitor should also be able to guide resuscitation and provide better information on those interventions most able to prevent the complications of inadequate perfusion. Gastric tonometry is a minimally invasive means to determine perfusion to the stomach and is the only one of a few clinical organ-specific monitors to help guide resuscitation.

The Theory Behind Gastric Tonometry

The gut is sensitive to ischemia. Periods of hypoperfusion may cause the release of inflammatory cytokines and bacterial translocation, thereby causing damage in remote organs.[1–4] Monitoring perfusion to the gut may help minimize or prevent episodes of mesenteric ischemia and improve the outcome of critically ill patients. The stomach is a relatively easy organ to access and may provide crucial information about perfusion to the rest of the splanchnic bed.

Gastric tonometry attempts to determine the perfusion status of the gastric mucosa using measurements of local P_{CO_2}.[5] CO_2 diffuses from the mucosa into the lumen of the stomach and subsequently into the silicone balloon of the tonometer (Fig 1). The P_{CO_2} within the balloon serves as a proxy for gastric mucosal CO_2 and can be measured by one of two means: (1) saline tonometry, where saline solution is anaerobically injected into the balloon, withdrawn after an equilibration period and measured using a blood gas analyzer; or (2) air tonometry, where air is pumped through the balloon and the P_{CO_2} is determined by an infrared detector on a semicontinuous basis. As blood flow to the stomach decreases, the P_{CO_2} will increase due to a decrease in bulk removal of CO_2 produced by normal respiration. When oxygen delivery ($\dot{D}O_2$) to the mucosa is reduced below metabolic demand (*ie*, anaerobiasis), acidosis ensues. The hydrogen ions that are produced are titrated with bicarbonate, and (by mass action: $H^+ + HCO_3^- \Leftrightarrow H_2CO_3 \Leftrightarrow CO_2 + H_2O$) even more CO_2 will accumulate than would be expected by a reduction in blood flow. By assuming that arterial (art) bicarbonate equals mucosal bicarbonate, intramucosal pH (pHi) can be calculated using the Henderson-Hasselbalch equation:

$$pHi = \log([HCO_3^-]art/0.03(P_{CO_2}muc))$$

where P_{CO_2}muc is gastric mucosal P_{CO_2}.

In addition to many animal investigations, support for the notion that gastric pHi assesses perfusion comes from a study of 17 patients receiving mechanical ventilation.[6] A low gastric pHi in these patients was associated with a lower mucosal blood flow as determined by laser Doppler flowmetry compared to patients with a normal pHi.

Unfortunately, the critical assumption—that arterial bicarbonate equals mucosal bicarbonate—is flawed. Simulations of mesenteric ischemia indicate that use of the arterial bicarbonate will result in errors in the determination of gastric pHi.[7] In addition, respiratory acid/base disturbances will introduce errors in the calculation of pHi.[8] Consequently, pHi has been replaced by the P_{CO_2} or the P_{CO_2} gap (the difference between gastric mucosal and arterial P_{CO_2}) as a better way to determine perfusion to the stomach.[9]

*From the University of Massachusetts Medical School, Worcester, MA.

Dedicated in memory of Robert Schlichtig, MD.

Correspondence to: Stephen O. Heard, MD, FCCP, Department of Anesthesiology, 55 Lake Ave North, University of Massachusetts Medical School, Worcester, MA 01655; e-mail: stephen.heard@umassmed.edu

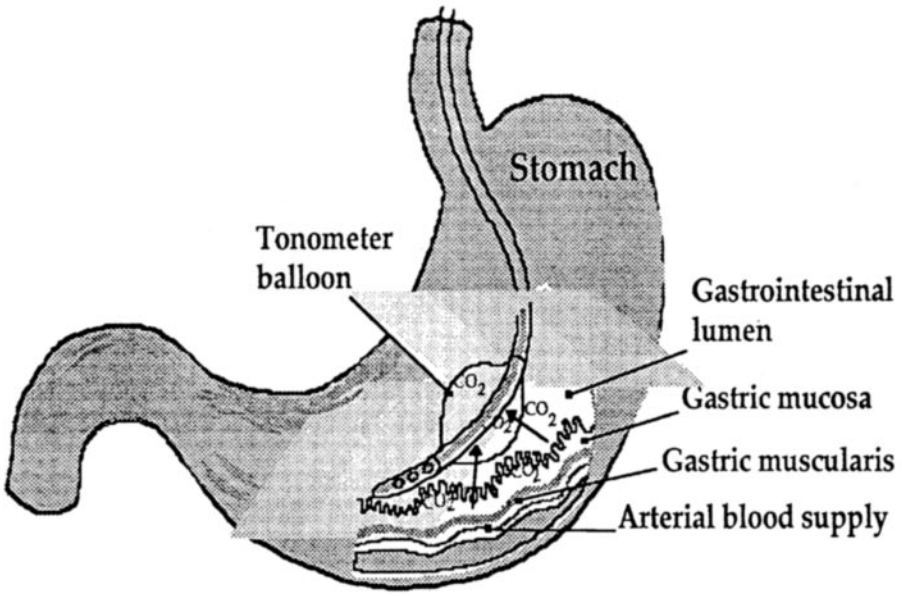

FIGURE 1. Schematic depicting the movement of CO_2 from the mucosa of the stomach into the gastric lumen and tonometer balloon. Reprinted with permission from Mythen et al.[5]

There are a number of factors that may cause errors in the determination of gastric pHi or P_{CO_2}, and these must be taken into account. If saline tonometry is used, some blood gas analyzers will consistently and dramatically underestimate the P_{CO_2} in the saline solution.[10] Use of buffered saline solutions will improve the accuracy of the P_{CO_2} determination, but the time for a steady state to be reached in the tonometer is increased.[11] Gastric acid

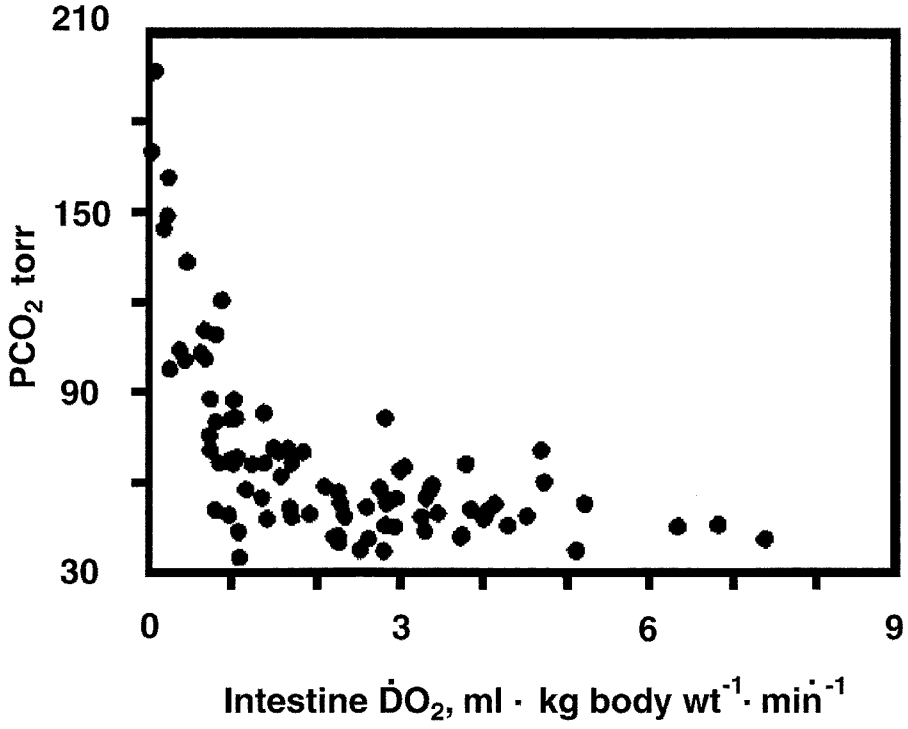

FIGURE 2. Relationship of jejunal mucosal P_{CO_2} and intestinal $\dot{D}_{O_2}$ in a canine model of cardiac tamponade. The estimated critical mucosal P_{CO_2} is 63 to 65 mm Hg. Reprinted with permission from Schlichtig and Bowles.[16]

secretion may also increase CO_2 production by titration of luminal acid with bicarbonate in the gastric mucus or refluxed duodenal contents, thereby introducing additional errors into determination of the P_{CO_2} gap. Use of histamine type-2 receptor antagonists will reduce this error.[12] Sucralfate does not appear to interfere with determination of gastric pHi.[13] Gastric but not duodenal feedings will cause a factitious reduction in gastric pHi.[14,15]

Determination of Critical CO_2 Value

One of the problems that has plagued gastric tonometry is that the value for pHi or P_{CO_2} where dysoxia ($\dot{D}O_2$ is insufficient to meet metabolic demand) occurs is unknown. In a canine model of cardiac tamponade, Schlichtig and Bowles[16] measured intestinal $\dot{D}O_2$, pHi, and tonometric CO_2 in the jejunum and ileum. They determined that dysoxia occurred around a P_{CO_2} value of 65 mm Hg and a P_{CO_2} gap of 25 to 35 mm Hg (Fig 2). These data suggest that the critical P_{CO_2} values currently being used for humans—in the range of 48 mm Hg for P_{CO_2} and 8 mm Hg for the corresponding P_{CO_2} gap—are unnecessarily low.

Indications For the Use of Gastric Tonometry

Since tonometry will provide information about levels of CO_2 (*ie*, blood flow) only in tissue, use of this monitor in shock states where blood flow is normal or elevated may not be particularly helpful. Patients with hypovolemia from any cause (*eg*, hemorrhagic shock or septic shock before fluid resuscitation) or who suffer from cardiac failure will benefit the most from the use of this monitor. The tonometer has been shown to be useful as a prognosticating tool, to detect hypovolemia, and as a guide for therapy.

Prognostic Capability of Gastric Tonometry

In a study of 83 critically ill patients (Figs 3, 4), Maynard and colleagues[17] demonstrated that gastric tonometry can predict outcome with better accuracy than other standard hemodynamic or metabolic variables (arterial pH, serum lactate, base excess, $\dot{D}O_2$ and oxygen consumption, cardiac index, mean arterial BP, and heart rate).

In a study of multiple-trauma patients, Kirton and colleagues[18] demonstrated the superiority of gastric tonometry over other clinical variables in predicting death. Other clinical studies have confirmed these findings,[19] and investigators have found gastric tonometry to be useful as a predictor for the development of multiple organ dysfunction syndrome[20] and successful extubation.[21]

Detection of Hypovolemia

To examine the utility of gastric tonometry in detecting hypovolemia, Hamilton-Davies and colleagues[22] removed and replaced 25% of the blood volume of six volunteers while measuring their gastric pHi and the mucosal-arterial P_{CO_2} gap. Heart rate, BP, base excess, and lactate varied

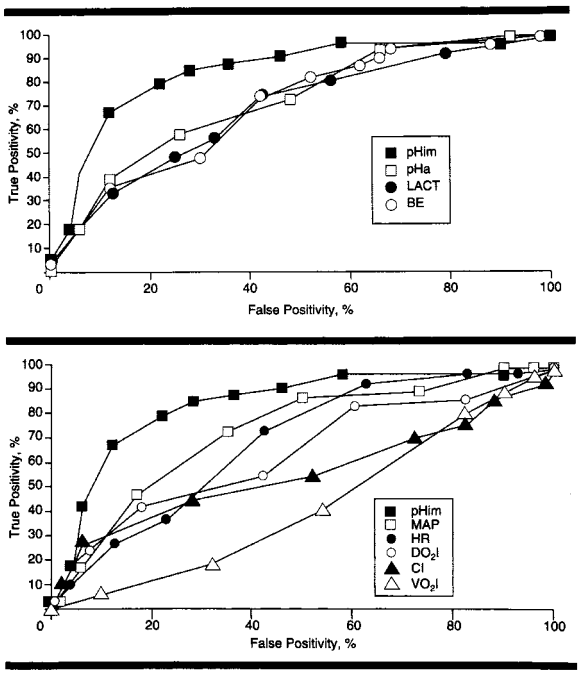

FIGURE 3. Receiver operating characteristic curves for the prediction of death: pHi (pHim), oxygen transport (Do_2I), oxygen consumption ($\dot{V}O_2I$), mean arterial BP (MAP), heart rate (HR), cardiac index (CI), arterial pH (pHa), lactate (LACT), and base excess (BE) derived from 83 critically ill patients. The area under the curve for pHi is greater than the other variables, thereby signifying its utility as a prognostic indicator. Reprinted with permission from Maynard et al.[17]

insignificantly during the experiment, but pHi and the P_{CO_2} gap showed dramatic and significant changes (Fig 5).

Gastric Tonometry as a Guide to Therapy

A number of studies have examined the utility of gastric tonometry as a guide to therapy. Unfortunately, most of these studies did not have the statistical power to detect differences in resuscitation strategies.

In a large, multicenter investigation, Gutierrez and colleagues[23] stratified 260 patients with APACHE (acute physiology and chronic health evaluation) II scores between 15 and 25 according to their hospital admission pHi. Those patients with an initial pHi ≥ 7.35 and whose resuscitation was guided by pHi had a higher 28-day survival compared to those individuals who were resuscitated according to standard protocols (Fig 6). Of interest, there was no difference between groups if the initial pHi was < 7.35.

A small study[24] of major trauma patients compared the utility of resuscitation to a gastric pHi of > 7.3 with resuscitation to global oxygen transport variables ($Do_2 > 600$ mL/min/m^2 or oxygen consumption > 150 mL/min/m^2). There was a statistically insignificant trend ($p = 0.16$) toward increased survival (90% vs 74%) and a reduced incidence of multiple organ dysfunction syndrome (10% vs 26%) in those patients whose treatment end point was pHi. Other small studies[25] with inadequate statistical power also failed to demonstrate a benefit of using pHi as

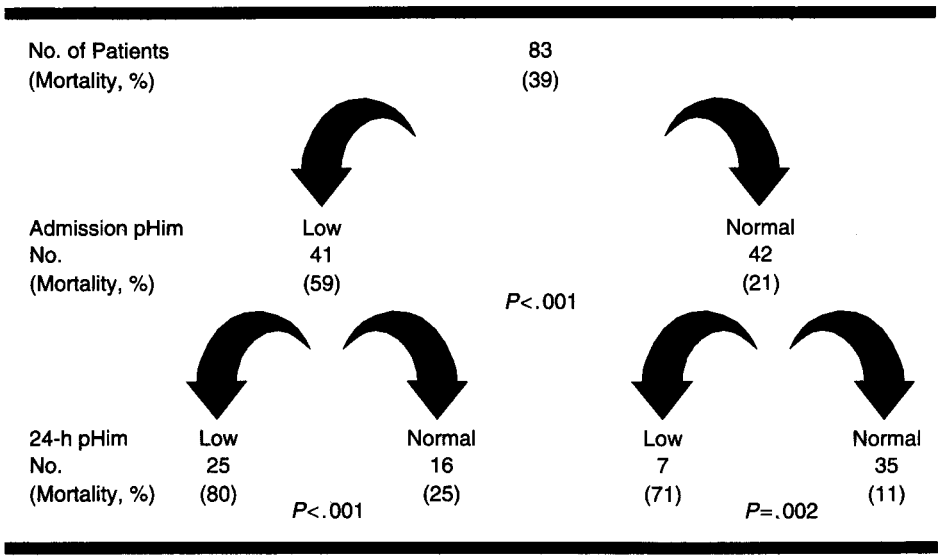

FIGURE 4. Mortality according to pHi on hospital admission and at 24 h. Reprinted with permission from Maynard et al.[17] See Figure 3 legend for expansion of abbreviation.

a therapeutic end point. In addition, a more recent, larger prospective, randomized study[26] of critically ill patients with diverse illnesses did not detect a difference in outcome when resuscitation to a gastric pHi of > 7.35 was compared to a standard resuscitation protocol. The authors recruited 210 patients into the study and hoped to detect a reduction in mortality from 40 to 30%. It appears, however, that this study may also have lacked statistical power as calculations by this author indicate a sample size of > 350 patients per group would be needed to detect such a change in mortality. A consistent observation in all of these studies has been that a low gastric pHi correlates with outcome. Failure to demonstrate an improvement in survival or a decrease in organ dysfunction by guiding therapy to gastric pHi may very well be the result of the failure of the therapeutic intervention protocols to raise gastric pHi.

Gastric tonometry has been shown to be useful in

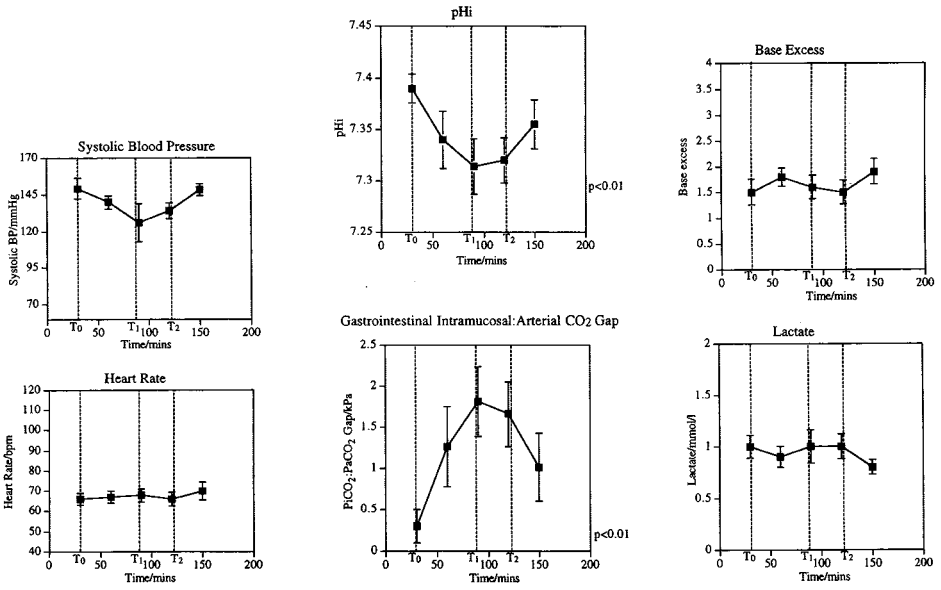

FIGURE 5. Responses to acute hemorrhage in human volunteers. T_0 = baseline; T_1 = end of hemorrhage; T_2 = prior to reinfusion of shed blood. There are significant decreases in gastric intramucosal pH (pHi) and increases in gastric intramucosal:arterial CO_2 gap compared to baseline analysis of variance. There are no significant changes in BP, heart rate, base excess, or lactate. Reprinted with permission from Hamilton-Davies et al.[22]

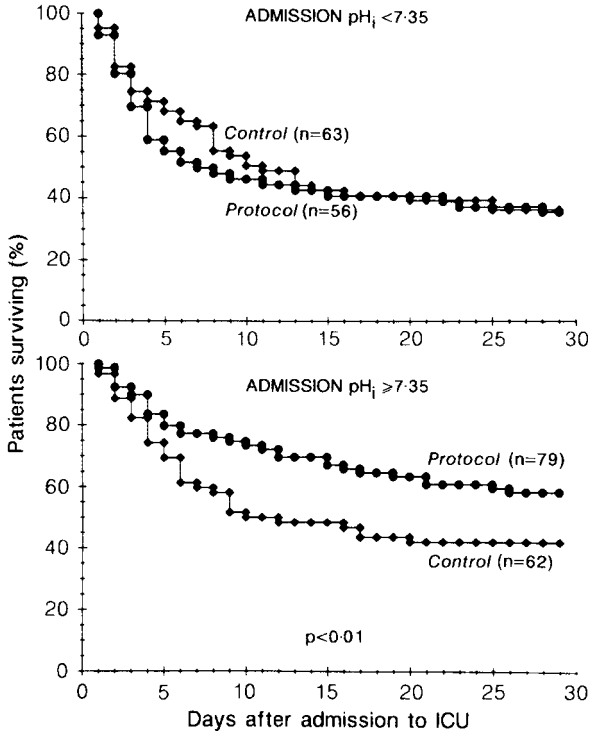

FIGURE 6. Kaplan-Meier hospital survival curves stratified according to admission gastric pHi in 260 critically ill patients. There is a significant difference in survival between those patients resuscitated according to gastric pHi compared to control patients if the hospital admission pHi was ≥ 7.35. There is no difference in mortality if the admission pHi was < 7.35. Reprinted with permission from Gutierrez et al.[23]

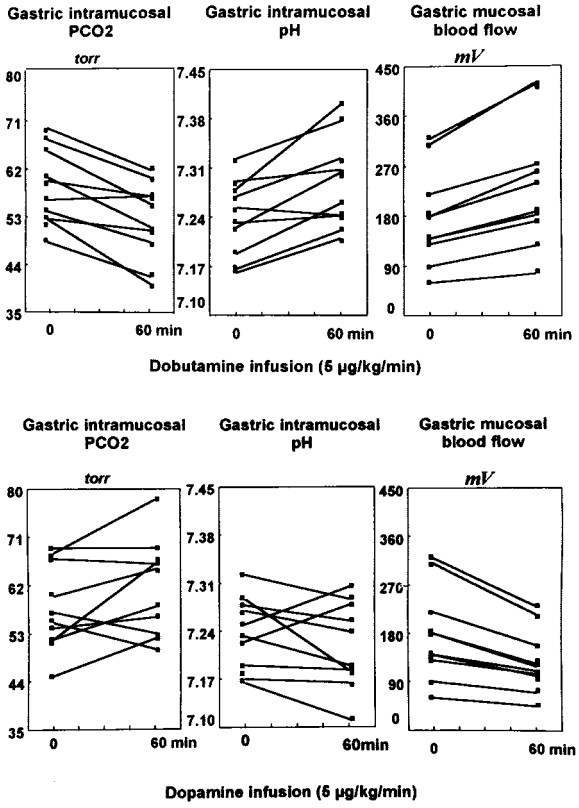

FIGURE 7. The contrasting effects of a 60-min infusion of dobutamine (5 µg/kg/min) [*top*] or dopamine (5 µg/kg/min) [*bottom*] on gastric intramucosal CO_2, intramucosal pH, and mucosal blood flow (as determined by laser Doppler flowmetry) in 10 septic patients. Note there is an inverse relationship between mucosal blood flow and gastric intramucosal P_{CO_2}. Reprinted with permission from Nevière et al.[28]

titrating vasopressor support and determining which vasoactive agent or vasoactive drug combination improves gastric perfusion in critically ill patients.[27–29] Several studies have demonstrated that dobutamine,[28] dobutamine/norepinephrine combinations,[29] or dopexamine[30] will increase gastric pHi or decrease P_{CO_2} gap compared to other agents or placebo in patients with sepsis or septic shock (Fig 7) or high-risk surgical patients.

Limitations of Tonometry

Recent clinical data cast doubt on the validity that gastric tonometry can be used as a proxy for monitoring perfusion to the rest of the hepatosplanchnic bed. Creteur and colleagues[31] measured gastric P_{CO_2} gap, hepatosplanchnic blood flow (via indocyanine green infusion), hepatic venous saturation, and hepatic venoarterial P_{CO_2} gradient in 36 patients with severe sepsis and found that the gastric P_{CO_2} did not correlate with the other indexes of hepatosplanchnic blood flow. Similar findings have been found in cardiac surgery patients treated with dobutamine.[32,33]

Summary

Despite the limitations of gastric tonometry, this minimally invasive monitor remains one of a few organ-specific monitors approved for clinical use. The tonometer remains valuable as a prognostic tool and to detect hypovolemia before it can be identified by global hemodynamic variables. Its use as a guide for therapy remains controversial, but it has fared no worse than other common monitors utilized in the care of critically ill patients.[34,35] Indeed, the use of the tonometer has not been associated with an increase in mortality![36]

Active investigation into other noninvasive monitors continues. Sublingual P_{CO_2} monitoring[37] and near infrared spectroscopy[38] may prove to be more useful than gastric tonometry in the monitoring and treatment of our critically ill patients.

References

1 Mythen MG, Purdy G, Mackie IJ, et al. Postoperative multiple organ dysfunction syndrome associated with gut mucosal hypoperfusion, increased neutrophil degranulation and C1-esterase inhibitor depletion. Br J Anaesth 1993; 71:858–863
2 Soong CV, Blair PH, Halliday MI, et al. Endotoxaemia, the generation of the cytokines and their relationship to intramucosal acidosis of the sigmoid colon in elective abdominal aortic aneurysm repair. Eur J Vasc Surg 1993; 7:534–539
3 Soong CV, Blair PH, Halliday MI, et al. Bowel ischaemia and

organ impairment in elective abdominal aortic aneurysm repair. Br J Surg 1994; 81:965–968
4 Soong CV, Halliday MI, Barclay GR, et al. Intramucosal acidosis and systemic host responses in abdominal aortic aneurysm surgery. Crit Care Med 1997; 25:1472–1479
5 Mythen MG, Woolf R, Noone RB. Gastric mucosal tonometry: towards new methods and applications. Anasthesiol Intensivmed Notfallmed Schmerzther 1998; 33(suppl 2):S85–S90
6 Elizalde JI, Hernandez C, Llach J, et al. Gastric intramucosal acidosis in mechanically ventilated patients: role of mucosal blood flow. Crit Care Med 1998; 26:827–832
7 Morgan TJ, Venkatesh B, Endre ZH. Accuracy of intramucosal pH calculated from arterial bicarbonate and the Henderson-Hasselbalch equation: assessment using simulated ischemia. Crit Care Med 1999; 27:2495–2499
8 Pernat A, Weil MH, Tang W, et al. Effects of hyper- and hypoventilation on gastric and sublingual P_{CO_2}. J Appl Physiol 1999; 87:933–937
9 Schlichtig R, Mehta N, Gayowski TJ. Tissue-arterial P_{CO_2} difference is a better marker of ischemia than intramural pH (pHi) or arterial pH-pHi difference. J Crit Care 1996; 11:51–56
10 Takala J, Parviainen I, Siloaho M, et al. Saline P_{CO_2} is an important source of error in the assessment of gastric intramucosal pH. Crit Care Med 1994; 22:1877–1879
11 Knichwitz G, Kuhmann M, Brodner G, et al. Gastric tonometry: precision and reliability are improved by a phosphate buffered solution. Crit Care Med 1996; 24:512–516
12 Heard SO, Helsmoortel CM, Kent JC, et al. Gastric tonometry in healthy volunteers: effect of ranitidine on calculated intramural pH. Crit Care Med 1991; 19:271–274
13 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
14 Marik PE, Lorenzana A. Effect of tube feedings on the measurement of gastric intramucosal pH. Crit Care Med 1996; 24:1498–1500
15 Levy B, Perrigault PF, Gawalkiewicz P, et al. Gastric versus duodenal feeding and gastric tonometric measurements. Crit Care Med 1998; 26:1991–1994
16 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
17 Maynard N, Bihari D, Beale R, et al. Assessment of splanchnic oxygenation by gastric tonometry in patients with acute circulatory failure. JAMA 1993; 270:1203–1210
18 Kirton OC, Windsor J, Wedderburn R, et al. Failure of splanchnic resuscitation in the acutely injured trauma patient correlates with multiple organ system failure and length of stay in the ICU. Chest 1998; 113:1064–1069
19 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
20 Marik PE. Gastric intramucosal pH: a better predictor of multiorgan dysfunction syndrome and death than oxygen-derived variables in patients with sepsis. Chest 1993; 104: 225–229
21 Uusaro A, Chittock DR, Russell JA, et al. Stress test and gastric-arterial P_{CO_2} measurement improve prediction of successful extubation. Crit Care Med 2000; 28:2313–2319
22 Hamilton-Davies C, Mythen MG, Salmon JB, et al. Comparison of commonly used clinical indicators of hypovolaemia with gastrointestinal tonometry. Intensive Care Med 1997; 23:276–281
23 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
24 Ivatury RR, Simon RJ, Islam S, et al. A prospective randomized study of end points of resuscitation after major trauma: global oxygen transport indices versus organ-specific gastric mucosal pH. J Am Coll Surg 1996; 183:145–154
25 Pargger H, Hampl KF, Christen P, et al. Gastric intramucosal pH-guided therapy in patients after elective repair of infrarenal abdominal aneurysms: is it beneficial? Intensive Care Med 1998; 24:769–776
26 Gomersall CD, Joynt GM, Freebairn RC, et al. Resuscitation of critically ill patients based on the results of gastric tonometry: a prospective, randomized, controlled trial. Crit Care Med 2000; 28:607–614
27 Marik PE, Mohedin M. The contrasting effects of dopamine and norepinephrine on systemic and splanchnic oxygen utilization in hyperdynamic sepsis. JAMA 1994; 272:1354–1357
28 Nevière R, Mathieu D, Chagnon JL, et al. The contrasting effects of dobutamine and dopamine on gastric mucosal perfusion in septic patients. Am J Respir Crit Care Med 1996; 154:1684–1688
29 Duranteau J, Sitbon P, Teboul JL, et al. Effects of epinephrine, norepinephrine, or the combination of norepinephrine and dobutamine on gastric mucosa in septic shock. Crit Care Med 1999; 27:893–900
30 Poeze M, Takala J, Greve JW, et al. Pre-operative tonometry is predictive for mortality and morbidity in high-risk surgical patients. Intensive Care Med 2000; 26:1272–1281
31 Creteur J, De Backer D, Vincent JL. Does gastric tonometry monitor splanchnic perfusion? Crit Care Med 1999; 27:2480–2484
32 Parviainen I, Ruokonen E, Takala J. Dobutamine-induced dissociation between changes in splanchnic blood flow and gastric intramucosal pH after cardiac surgery. Br J Anaesth 1995; 74:277–282
33 Thoren A, Jakob SM, Pradl R, et al. Jejunal and gastric mucosal perfusion versus splanchnic blood flow and metabolism: an observational study on postcardiac surgical patients. Crit Care Med 2000; 28:3649–3654
34 Hayes MA, Timmins AC, Yau EH, et al. Elevation of systemic oxygen delivery in the treatment of critically ill patients. N Engl J Med 1994; 330:1717–1722
35 Gattinoni L, Brazzi L, Pelosi P, et al. A trial of goal-oriented hemodynamic therapy in critically ill patients. SvO_2 Collaborative Group. N Engl J Med 1995; 333:1025–1032
36 Connors AF Jr, Speroff T, Dawson NV, et al. The effectiveness of right heart catheterization in the initial care of critically ill patients: SUPPORT Investigators. JAMA 1996; 276:889–897
37 Marik PE. Sublingual capnography: a clinical validation study. Chest 2001; 120:923–927
38 Soller BR, Heard SO, Cingo NA, et al. Application of fiber optic sensors for the study of hepatic dysoxia in swine hemorrhagic shock. Crit Care Med 2001; 29:1438–1444