Gastric Tonometry: The Hemodynamic Monitor of Choice (Pro)

Stephen O. Heard

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

The online version of this article, along with updated information and services can be found online on the World Wide Web at: http://chestjournals.org/cgi/content/abstract/123/5_suppl/469S

CHEST is the official journal of the American College of Chest Physicians. It has been published monthly since 1935. Copyright 2007 by the American College of Chest Physicians, 3300 Dundee Road, Northbrook IL 60062. All rights reserved. No part of this article or PDF may be reproduced or distributed without the prior written permission of the copyright holder (http://www.chestjournal.org/misc/reprints.shtml). ISSN: 0012-3692.
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: \( D\dot{O}_2 \) = 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 \( PCO_2 \).\(^5\) \( CO_2 \) diffuses from the mucosa into the lumen of the stomach and subsequently into the silicone balloon of the tonometer (Fig 1). The \( PCO_2 \) 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 \( PCO_2 \) is determined by an infrared detector on a semicontinuous basis. As blood flow to the stomach decreases, the \( PCO_2 \) will increase due to a decrease in bulk removal of \( CO_2 \) produced by normal respiration. When oxygen delivery (\( D\dot{O}_2 \)) 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: \( H^+ + HCO_3^- \rightarrow H_2CO_3 \rightarrow CO_2 + H_2O \)) 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:

\[
\text{pHi} = \log([HCO_3^-]/\text{art}/0.03(\text{PCO}_2\text{muc}))
\]

where \( \text{PCO}_2\text{muc} \) is gastric mucosal \( \text{PCO}_2 \).

In addition to many animal investigations, support for the notion that gastric pH assesses perfusion comes from a study of 17 patients receiving mechanical ventilation.\(^6\) A low gastric pH in these patients was associated with a lower mucosal blood flow as determined by laser Doppler flowmetry compared to patients with a normal pH. 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.\(^7\) Consequently, pH has been replaced by the \( \text{PCO}_2 \) or the \( \text{PCO}_2 \) gap (the difference between gastric mucosal and arterial \( \text{PCO}_2 \)) as a better way to determine perfusion to the stomach.\(^8\)

*From the University of Massachusetts Medical School, Worcester, MA.
Dedicated in memory of Robert Schlichtig, MD.
Reproduction of this article is prohibited without written permission from the American College of Chest Physicians (e-mail: permissions@chestnet.org).
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

www.chestjournal.org

Downloaded from chestjournals.org on March 29, 2007
Copyright © 2003 by American College of Chest Physicians
There are a number of factors that may cause errors in the determination of gastric pH or PCO$_2$, and these must be taken into account. If saline tonometry is used, some blood gas analyzers will consistently and dramatically underestimate the PCO$_2$ in the saline solution. Use of buffered saline solutions will improve the accuracy of the PCO$_2$ determination, but the time for a steady state to be reached in the tonometer is increased. Gastric acid

**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.

**Figure 2.** Relationship of jejunal mucosal PCO$_2$ and intestinal DO$_2$ in a canine model of cardiac tamponade. The estimated critical mucosal PCO$_2$ is 63 to 65 mm Hg. Reprinted with permission from Schlichtig and Bowles.
secretion may also increase CO₂ production by titration of luminal acid with bicarbonate in the gastric mucus or refluxed duodenal contents, thereby introducing additional errors into determination of the Pco₂ gap. Use of histamine type-2 receptor antagonists will reduce this error.¹² Sucralfate does not appear to interfere with determination of gastric pH.¹³ Gastric but not duodenal feedings will cause a factitious reduction in gastric pH.¹⁴,¹⁵

**Detection of Critical CO₂ Value**

One of the problems that has plagued gastric tonometry is that the value for pH or Pco₂ where dysoxia (DO₂ is insufficient to meet metabolic demand) occurs is unknown. In a canine model of cardiac tamponade, Schlichtig and Bowles¹⁰ measured intestinal DO₂, pHᵢ, and tonometric CO₂ in the jejunum and ileum. They determined that dysoxia occurred around a Pco₂ value of 65 mm Hg and a Pco₂ gap of 25 to 35 mm Hg (Fig 2). These data suggest that the critical Pco₂ values currently being used for humans—in the range of 48 mm Hg for Pco₂ and 8 mm Hg for the corresponding Pco₂ gap—are unnecessarily low.

**Indications For the Use of Gastric Tonometry**

Since tonometry will provide information about levels of CO₂ (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 colleagues¹⁷ demonstrated that gastric tonometry can predict outcome with better accuracy than other standard hemodynamic or metabolic variables (arterial pH, serum lactate, base excess, DO₂ and oxygen consumption, cardiac index, mean arterial BP, and heart rate).

In a study of multiple-trauma patients, Kirton and colleagues¹⁸ 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 syndrome²⁰ and successful extubation.²¹

**Detection of Hypovolemia**

To examine the utility of gastric tonometry in detecting hypovolemia, Hamilton-Davies and colleagues²² removed and replaced 25% of the blood volume of six volunteers while measuring their gastric pH and the mucosal-arterial Pco₂ gap. Heart rate, BP, base excess, and lactate varied insignificantly during the experiment, but pHᵢ and the Pco₂ 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 colleagues²³ 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 study²⁴ of major trauma patients compared the utility of resuscitation to a gastric pHᵢ of > 7.3 with resuscitation to global oxygen transport variables (DO₂ > 600 mL/min/m² or oxygen consumption > 150 mL/min/m²). 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 studies²⁵ with inadequate statistical power also failed to demonstrate a benefit of using pHᵢ as
a therapeutic end point. In addition, a more recent, larger prospective, randomized study of critically ill patients with diverse illnesses did not detect a difference in outcome when resuscitation to a gastric pH 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 pH correlates with outcome. Failure to demonstrate an improvement in survival or a decrease in organ dysfunction by guiding therapy to gastric pH may very well be the result of the failure of the therapeutic intervention protocols to raise gastric pH.

Gastric tonometry has been shown to be useful in

![Figure 5](image1)

**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

<table>
<thead>
<tr>
<th>Admission pHim</th>
<th>83 (39)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No.</td>
<td>41 (59)</td>
</tr>
<tr>
<td>(Mortality, %)</td>
<td>42 (21)</td>
</tr>
<tr>
<td></td>
<td>$P&lt;.001$</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>24-h pHim</th>
<th>35 (11)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No.</td>
<td>16 (25)</td>
</tr>
<tr>
<td>(Mortality, %)</td>
<td>7 (71)</td>
</tr>
<tr>
<td></td>
<td>$P=.002$</td>
</tr>
</tbody>
</table>

![Figure 4](image2)

**Figure 4.** Mortality according to pH on hospital admission and at 24 h. Reprinted with permission from Maynard et al.¹⁷ See Figure 3 legend for expansion of abbreviation.
titrating vasopressor support and determining which vasoactive agent or vasoactive drug combination improves gastric perfusion in critically ill patients. Several studies have demonstrated that dobutamine, dobutamine/norepinephrine combinations, or dopexamine will increase gastric P\textsubscript{co}\textsubscript{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 measured gastric P\textsubscript{co}\textsubscript{2} gap, hepatosplanchnic blood flow (via indocyanine green infusion), hepatic venous saturation, and hepatic venaocleral P\textsubscript{co}\textsubscript{2} gradient in 36 patients with severe sepsis and found that the gastric P\textsubscript{co}\textsubscript{2} did not correlate with the other indexes of hepatosplanchnic blood flow. Similar findings have been found in cardiac surgery patients treated with dobutamine.

**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. Indeed, the use of the tonometer has not been associated with an increase in mortality.

Active investigation into other noninvasive monitors continues. Sublingual P\textsubscript{co}\textsubscript{2} monitoring and near infrared spectroscopy may prove to be more useful than gastric tonometry in the monitoring and treatment of our critically ill patients.

**References**

3. Soong CV, Blair PH, Halliday MI, et al. Bowel ischaemia and
9 Schlüchting R, Meltha N, Gayowski TJ. Tissue-arterial Pco2 difference is a better marker of ischemia than intramural pH (pHi) or arterial pH-pHi difference. J Crit Care 1996; 11:51–56
Gastric Tonometry: The Hemodynamic Monitor of Choice (Pro)
Stephen O. Heard
Chest 2003;123:469-474
DOI 10.1378/chest.123.5_suppl.469S

This information is current as of March 29, 2007

<table>
<thead>
<tr>
<th>Updated Information &amp; Services</th>
<th>Updated information and services, including high-resolution figures, can be found at: <a href="http://chestjournals.org/cgi/content/full/123/5_suppl/469S">http://chestjournals.org/cgi/content/full/123/5_suppl/469S</a></th>
</tr>
</thead>
<tbody>
<tr>
<td>References</td>
<td>This article cites 37 articles, 13 of which you can access for free at: <a href="http://chestjournals.org/cgi/content/full/123/5_suppl/469S">http://chestjournals.org/cgi/content/full/123/5_suppl/469S</a> #BIBL</td>
</tr>
<tr>
<td>Citations</td>
<td>This article has been cited by 3 HighWire-hosted articles: <a href="http://chestjournals.org/cgi/content/full/123/5_suppl/469S">http://chestjournals.org/cgi/content/full/123/5_suppl/469S</a></td>
</tr>
<tr>
<td>Permissions &amp; Licensing</td>
<td>Information about reproducing this article in parts (figures, tables) or in its entirety can be found online at: <a href="http://chestjournals.org/misc/reprints.shtml">http://chestjournals.org/misc/reprints.shtml</a></td>
</tr>
<tr>
<td>Reprints</td>
<td>Information about ordering reprints can be found online: <a href="http://chestjournals.org/misc/reprints.shtml">http://chestjournals.org/misc/reprints.shtml</a></td>
</tr>
<tr>
<td>Email alerting service</td>
<td>Receive free email alerts when new articles cite this article sign up in the box at the top right corner of the online article.</td>
</tr>
<tr>
<td>Images in PowerPoint format</td>
<td>Figures that appear in CHEST articles can be downloaded for teaching purposes in PowerPoint slide format. See any online article figure for directions.</td>
</tr>
</tbody>
</table>