Gastrointestinal luminal $P_{CO_2}$ tonometry: an update on physiology, methodology and clinical applications

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Gastrointestinal mucosal pH (pHi), calculated from tonometrically measured $P_{CO_2}$ in the gastrointestinal lumen and blood bicarbonate content using the Henderson–Hasselbalch equation, has been suggested to constitute an index of the adequacy of splanchnic mucosal perfusion. This may relate to the prognosis of critically ill patients, as bowel wall hypoperfusion may result in tissue injury, increased permeability, endotoxin–bacterial translocation and a harmful inflammatory (cytokine) response. The theory is that hypoperfusion below a critical level causes tissue (mucosal) carbon dioxide accumulation and acidosis. As carbon dioxide diffuses easily across membranes, the $P_{CO_2}$ in the gut lumen also increases, leading to widening of the tonometer–blood $P_{CO_2}$ gradient. In fact, pHi has been used successfully to guide treatment and to improve the outcome of critically ill patients. Nevertheless, tonometry has not yet become a routine intensive care monitoring technique. This may relate to uncertainties regarding its physiological background, methodology and clinical usefulness. This review will therefore update current thoughts on these aspects.

Physiological background

Gastrointestinal hypoperfusion

Several conditions may lead to altered gut perfusion (Table 1). Haemorrhagic hypotension, cardiac tamponade, cardiac bypass or vasopressin infusion for example, may lead to mucosal hypoperfusion along the entire gastrointestinal tract, and this may be assessed with the help of simultaneous tonometric measurements in various gastrointestinal segments. In fact, the bowel $P_{CO_2}$ gradient may increase and pHi decrease during hypoperfusion states, similar to changes in gastrically determined tonometric variables. This may also apply to sepsis and shock, even if the oxygen demands by the bowel wall increase, and to cardiopulmonary bypass surgery, during which bowel wall oxygen demands may decrease following hypothermia and increase during rewarming. Moreover, shock may result in early selective splanchnic vasoconstriction so that gastric tonometry may reveal an early indicator of general hypoperfusion. The gastric tonometric $P_{CO_2}$ gradient proved an early, sensitive indicator of hypovolaemia during haemorrhage in healthy volunteers. Experimental studies using vascular occlusion and reperfusion, induction of shock or pharmacological splanchnic vasoconstriction have revealed that changes in blood flow to the gut wall, as measured by microspheres, laser Doppler, electromagnetic or ultrasonic flow probes, or reflectance spectrophotometry, were paralleled by concordant changes in tonometric variables. A decrease in blood flow to less than 50% of baseline during incremental hypoperfusion leads to an increased tonometric $P_{CO_2}$ relative to supplying (and draining) blood values. This results in a decrease in pHi, in parallel with the decreasing blood flow and decrease in tissue $P_O_2$ and oxygen consumption. In critically ill, septic and mechanically ventilated patients, laser Doppler (and reflectance spectroscopy) measurement of gastric mucosal blood flow, hepatosplanchnic blood flow measured by indocyanine green, or hepatic breakdown of injected lidocaine to monoethylglycine xylidide, were lower in
patients with an increased $P_{\text{CO}_2}$ gradient and subnormal pH
to demand ratio. Changes in splanchnic blood flow, as
as assessed by indocyanine green clearance or laser Doppler
techniques, are not necessarily accompanied by changes in
tonic variables and vice versa, particularly when
demand changes, for example with the variation in body
temperature associated with cardiopulmonary bypass
surgery and sepsis. During hypothermic cardiopulmonary
bypass surgery, gastric mucosal (Doppler) blood flow may
decrease, but neither the gastric luminal to blood $P_{\text{CO}_2}$
gradient nor pH may change.\(^{3, 101, 102, 127}\) The generally observed
decrease in pH during rewarming and in the postoperative
phase may not concord with splanchnic blood flow changes
and oxygen consumption, which may even increase, and
this discrepancy suggests either increased oxygen demand
or insufficient ability of pH to reflect the balance between
oxygen supply and demand.\(^{53, 106, 109, 110, 114, 129}\) Even during
normothermic cardiopulmonary bypass surgery, bowel
demands may increase and may be insufficiently met by an
increase in blood flow, thereby lowering pH.\(^{53}\) Disconcord-
ant changes in cardiac output and gut blood flow on the
one hand and tonometric variables on the other during
treatment with catecholamines, with an increase in the
former but no improvement in the latter, may relate in part
to redistribution of blood flow within the stomach or
gut wall, away from the mucosa.\(^{11, 110, 111, 127, 129}\) During
endotoxaemic–septic shock, gut blood flow may not
decrease and gut $P_{\text{O}_2}$ and oxygen consumption may increase,
but this may not prevent a subnormal gastrointestinal
pH developing, suggesting redistribution of blood flow,
impaired oxygen use, or both.\(^{131}\) Finally, it remains to be
seen if deterioration in tissue oxygenation and increased
anaerobic metabolism through severe hypoxaemia or anaem-
A study of the pHi changes in the stomach and small bowel, respectively. This may not occur
with tonometry in the oesophagus or sublingually as these areas are not supplied by splanchnic blood vessels. Thus
the sensitivity of tonometry for early detection of general hypoperfusion may be inferior to that for detecting hypo-
perfusion in the stomach or small bowel, where selective vasoconstriction may occur.\(^{33}\) During severe haemorrhage
or sepsis in animals, sublingual, oesophageal and gastric $P_{\text{CO}_2}$, nevertheless increased in parallel and simultaneously
with the decrease in blood flow and increase in blood lactate
concentration.\(^{52, 64, 100, 118, 124}\) With regard to the value of small bowel vs gastric tonometry, widening of the tono-
metric–blood $P_{\text{CO}_2}$ gradient in pigs may be earlier in the
small bowel than in the stomach during haemorrhagic
shock,\(^{101}\) but not during cardiac tamponade.\(^{3}\) The increase
in jejunal $P_{\text{CO}_2}$ during endotoxaemia in horses was less and
occurred later than that of gastric $P_{\text{CO}_2}$,\(^{123}\) Walley and
colleagues,\(^{134}\) however, showed that during haemorrhagic
hypotension, widening of the tonometer–blood $P_{\text{CO}_2}$ gradient
was less and less variable in the jejunum than in the
stomach, largely because of greater measurement error in
the latter.

### Sublingual, oesophageal, gastric and bowel $P_{\text{CO}_2}$
tonometry

Luminal production of carbon dioxide during buffering of
gastric acid by bicarbonate or bacterial fermentation may
limit the specificity of luminal $P_{\text{CO}_2}$ and thus pH as
indicators of the adequacy of mucosal perfusion in the
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### $P_{\text{CO}_2}$ gradient vs pH as an indicator of mucosal
hypoperfusion

It has been argued repeatedly that pH is a composite
variable, consisting of a systemic and locally derived
variable, and should be replaced by the tonometer–blood
$P_{\text{CO}_2}$ (or perhaps pH) gradient. This is especially so if
tonometry is being done to yield a sensitive and specific
measure of the adequacy of gastrointestinal mucosal perfu-
sion, independent of systemic metabolic and respiratory
alterations.\(^{26, 42, 46, 51, 55, 61, 73, 87, 120, 134}\) Indeed, the major
pitfall in the calculation of pH from gastric $P_{\text{CO}_2}$ and
arterial bicarbonate content is the assumption that the latter
equals mucosal content. In humans, the mucous layer of
the acid-secreting stomach also contains bicarbonate, which
is secreted by non-parietal cells and helps to protect the
underlying mucosa from the gastric acid secreted by parietal
cells.\(^{69}\) Mild hypovolaemia in human volunteers decreases
gastric bicarbonate secretion more than acid secretion, and
this may be associated with alterations in tissue bicarbonate
concentration.\(^{69}\) Hence the bicarbonate content of the
mucosa may normally be higher than in blood, while

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<th>Table 1 Conditions in which tonometry is of potential clinical value</th>
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<td>Perioperative monitoring in major surgery, including cardiopulmonary bypass, major (emergency) vascular surgery and liver transplantation</td>
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hypoperfusion may result in a dissimilar decrease in blood and mucosal bicarbonate content, depending on the balance between general and regional hypoperfusion, diminished bicarbonate secretion and the degree of buffering of lactic acid in the hypoperfused stomach wall. Conversely, changes in arterial blood bicarbonate content may lead to alterations in calculated pH, independent of changes in the degree of mucosal hypoperfusion and the increase in $P_{CO_2}$ gradient. I.v. bicarbonate administration to a patient with mesenteric thrombosis resulted in an increase in gastric tonometer pH to normal values, despite a necrotic bowel found at laparotomy, but the stomach may not have represented the small bowel. Conversely, in cardiac surgery patients, a rapid decrease in arterial bicarbonate content by volume loading during surgery may contribute to a lowered gastric pH, independent of luminal $P_{CO_2}$.

Moreover, gastric $P_{CO_2}$ closely parallels local arterial (and draining venous) blood $P_{CO_2}$ values during changes in alveolar ventilation, as demonstrated in animals and human volunteers with (presumably) normal perfusion, so that changes in pH may result from changes in alveolar ventilation, independent of mucosal hypoperfusion. The dependency of pH on arterial blood bicarbonate content and $P_{CO_2}$ may underlie the observed concurrent changes or correlations between calculated pH on the one hand and arterial blood pH and acid–base variables, including lactate concentration, on the other.

It is still unclear as to the threshold for anaerobic metabolism, but the upper limit of a normal gradient would be approximately 1.2 kPa. The increased luminal $P_{CO_2}$ during hypoperfusion is assumed to stem from two sources. With a moderately gradual reduction in perfusion, mucosal $P_{CO_2}$ accumulates after a reduced washout and an increase in tissue and venous $P_{CO_2}$. A further decrease in blood flow results in a steep increase in tissue (luminal) and to a lesser extent venous $P_{CO_2}$, and this may be caused by buffering of anaerobically produced lactate and protons by mucosal bicarbonate. Hence a gradient between tissue and draining venous $P_{CO_2}$, in spite of allegedly high diffusibility of carbon dioxide, may be a marker of anaerobic tissue metabolism. It seems that a decrease in tissue oxygen consumption and tension and development of anaerobic metabolism and production of lactic acid occurs at a $P_{CO_2}$ gradient of 3.3 kPa or greater, a value that can be regarded as the critical gradient. A lower gradient, however, does not exclude a focal oxygen deficit in the mucosa. The baseline $P_{CO_2}$ tonometer–blood gradient in patients with gastric erosions or ulcerations and stenotic or occluded coeliac and mesenteric vessels at angiography was approximately 0.4 kPa and increased to 2.5 kPa during submaximal exercise, whereas no such increase was observed in patients with normal angiography. Thus some patients had lesions in the stomach attributable to hypoperfusion in spite of a resting $P_{CO_2}$ gradient presumably below the critical value of approximately 3.3 kPa. The lesions may relate to intermittent or patchy hypoperfusion rendering the mucosa susceptible to damage by other factors.

**Methodological considerations**

**Comparison of tonometric with directly measured variables**

In the control state, tonometric pH may agree with directly measured tissue pH in the stomach and bowel. Some authors have shown that directly measured $P_{CO_2}$ is higher than tonometrically derived $P_{CO_2}$, and that the response time of the latter is slower. This may result in tonometric pH overestimating directly measured tissue pH during sudden hypoperfusion. The latter may also relate to the erroneous bicarbonate assumption discussed above.

**Normal values**

Normal values for gastric tonometry have been defined, taking *in vivo* determined correction factors and blood-gas analyser bias (see below) into account. The upper limit of normal values for $P_{CO_2}$ was 6.5 kPa and for the tonometer–blood $P_{CO_2}$ gradient, 1.2 kPa, so that tonometric $P_{CO_2}$ is normally a few kPa higher than that in the blood supply. The lower limit of normal for pH is 7.33 and for the pH gradient, −0.06. This agrees in part with other reports, where the upper limit of normal pH varied between 7.32 and 7.35. Data on normal $P_{CO_2}$ or the $P_{CO_2}$ gradient in the human gut are lacking. Bass and colleagues noted that the control intestinal (jejunal or ileal) mass spectrometer $P_{CO_2}$ was 11.9 kPa in dogs, but arterial values were not given. The normal fibreoptic/tonometric ileum to blood $P_{CO_2}$ gradient may, as in the stomach, amount to 1.3–2.7 kPa in pigs and dogs. The normal oesophagus to blood $P_{CO_2}$ gradient is also in that range. There is no information on the normal tonometer balloon equilibration characteristics from the oesophagus to the large bowel, where changes in luminal contents and diffusion conditions may have an effect.

**Gastric $P_{CO_2}$ as a measure of gastrointestinal mucosal $P_{CO_2}$**

It is generally assumed that rapid diffusion results in luminal $P_{CO_2}$ being identical to mucosal $P_{CO_2}$. Nevertheless, the increase in tonometric $P_{CO_2}$ may underestimate that of directly measured $P_{CO_2}$. Carbon dioxide diffuses rapidly from mucosa into the lumen with complete equilibration in 60 min, while diffusion in the opposite direction is about four times slower. Even though carbonic anhydrase, present in the gastric mucosa, may contribute to the difference in diffusion rate, inhibition of carbonic anhydrase activity did not decrease the rate of $P_{CO_2}$ build-up in the tonometer.
balloon. This suggests that the balloon $P_{\text{CO}_2}$ arises from gastric contents as opposed to the gastric mucosa. Indeed, gastric carbon dioxide content may decrease by 6% and $P_{\text{CO}_2}$ may decrease by 0.3 kPa during equilibration of 1.5 ml of tonometer balloon saline, in the absence of transmucosal diffusion. Gastric balloon rather than transmucosal carbon dioxide diffusion may be the rate limiting step during gastric $P_{\text{CO}_2}$ tonometry.

**Gastric secretion of acid**

The close approximation of luminal and mucosal $P_{\text{CO}_2}$ may be lost in the stomach when carbon dioxide is produced by buffering of gastric acid, contributing to a relatively poor reproducibility of gastric pH. Indeed, in the normally perfused upper gastrointestinal tract, luminal $P_{\text{CO}_2}$ may directly relate to the amount of acid secreted and buffered in the stomach, and has even been used as a measure of gastric acid secretion. This luminally produced carbon dioxide stems from the buffering of gastric acid by bicarbonate secreted by the gastric mucosa or by bicarbonate entering the stomach through duodenal reflux or from saliva via the oesophagus. The increase in $P_{\text{CO}_2}$ resulting from buffering can be prevented by inhibition of acid secretion. Hence it has been recommended to perform tonometry after H2 block of gastric acid secretion. In fact, gastric $P_{\text{CO}_2}$ in human volunteers is 1.3–2.7 kPa above blood levels and decreases towards arterial levels with less interindividual variation after inhibition of acid secretion by the H2 blocker ranitidine, so that calculated pH is higher in the presence of prior H2 block. When duodenogastric reflux is simulated by oral administration of bicarbonate in an amount approximating the hourly pancreaticoduodenal production, gastric $P_{\text{CO}_2}$ increases to approximately 10.7 kPa above blood levels but only in the absence and not in the presence of prior H2 block. Removal of gastric acid by suction may not decrease gastric $P_{\text{CO}_2}$ towards blood values. This can be explained by the fact that gastric acid, which is produced in the deep pits of the gastric mucosa, is buffered by gastric bicarbonate in the mucus layer covering these pits. Buffering of gastric acid by administration of non-carbon dioxide releasing antacids such as aluminium oxide–magnesium hydroxide also releases large amounts of carbon dioxide in vitro and is therefore not useful.

It can be questioned whether data in healthy subjects would apply to critically ill patients as H2 blockers, which would be expected to lower $P_{\text{CO}_2}$, had no effect on gastric $P_{\text{CO}_2}$ in the seriously ill. Together with the concept that inhibition of acid secretion may predispose to gastric bacterial colonization and nosocomial infections, this may be the reason that many authors do not use H2 block before tonometry in critically ill patients. Similarly, administration of sucralfate, which has acid-buffering capacities and increases gastric bicarbonate secretion, and could therefore be expected to increase $P_{\text{CO}_2}$ in the presence of acid, had no effect on $P_{\text{CO}_2}$ in critically ill patients. This may be explained by a reduction in gastric acid secretion, as shown by a relatively high gastric juice pH that fails to decrease after pentagastrin infusion in many critically ill patients after operation. During the first 24 h after cardiopulmonary bypass surgery, gastric fluid may be acidic (pH <4.0) in a minority of patients. Simulation of duodenogastric reflux by gastric bicarbonate administration had no effect on gastric $P_{\text{CO}_2}$ immediately after surgery, indicating lack of gastric acid secretion but increased gastric $P_{\text{CO}_2}$ on the first postoperative day by 2.7 kPa in six of 10 subjects. The observations suggest postoperative recovery occurs after reduced gastric acid secretion caused by transient hypoperfusion.

Indeed, gastric acid secretion is an active, energy-demanding process. During haemorrhagic hypotension, gastric mucosal blood flow and gastric acid (and bicarbonate) secretion diminish, but less so in pentagastrin- or histamine-stimulated conditions than in animals pretreated with H2 blockers. This suggests that gastric acid secretion in shock is blood flow dependent and that administration of H2 blockers (or proton pump inhibitors) may further diminish energy-demanding gastric acid secretion and blood flow and may protect against the development of stress ulcers related to hypoperfusion. In fact, a low pH may be associated with failure to acidify the gastric lumen in response to pentagastrin. The combination of a reduced demand and an increased perfusion reserve during inhibition of acid secretion probably results in lowered sensitivity of tonometry as an indicator of early perfusion failure or gastric mucosal hypoperfusion. A patient who develops an increased $P_{\text{CO}_2}$ gradient might either have mucosal hypoperfusion or recurrence of gastric acid secretion after amelioration of hypoperfusion. Inhibition of acid secretion reduces the incidence of this dilemma, thereby increasing the validity of gastric tonometry, and simple measurement of gastric juice pH, preferably by continuous monitoring, could solve the problem. An aspirated juice pH above 4.0–5.0, checked with the help of litmus paper, would suggest minimum or no buffering effects, so that changes in gastric $P_{\text{CO}_2}$ would probably result from changes in mucosal perfusion only.

However, H2 blockers such as ranitidine may fail to control gastric juice pH, even in critically ill patients. The efficacy of the first dose after i.v. administration is reduced within 24 h, and after 48 h, the gastric juice pH is again less than 4.0 for 60% of the time. The proton pump inhibitor omeprazole increases gastric juice pH more efficiently and continuously than H2 antagonists, even 24 h after administration. While abolishing acid and inhibiting bicarbonate secretion, the drug may maintain mucosal perfusion, in contrast with H2 blockers that decrease both acid secretion and mucosal blood flow. Preserved blood flow after proton pump inhibitors may contribute to preventing stress ulcer bleeding during haemorrhagic hypotension.
Tonometry in fed or fasting state?

Eating a meal might serve as a stress test for mucosal vasodilator reserve as the meal stimulates gastric acid secretion, and small bowel secretions, and active resorption of nutrients. The increased energy expenditure of the gut causes an increase in blood flow to both the stomach and small gut mucosa. Indeed, luminal $P_{CO_2}$ may increase to 53.5 kPa in the normal stomach and duodenum after gastric feeding, as a result of buffering of the increased acid by the increased bicarbonate output. As the increased postprandial $P_{CO_2}$ is caused by acid buffering, it might be expected that acid suppression could circumvent this increase. However, although $H_2$ blockers can effectively block basal or fasting acid secretion, suppression of meal-stimulated gastric acid secretion may be incomplete, even in critically ill patients. This may also occur in some individuals where acid secretion has been inhibited completely by a combination of ranitidine and the muscarinic receptor antagonist pirenzepine. The cause of this increase proved to be the increased bicarbonate output. As the increased feeding, as a result of buffering of the increased acid secretion has been inhibited completely by a combination of ranitidine and the muscarinic receptor antagonist representing catheter deadspace, (3) withdrawing and discarding the last 1 ml from the balloon resides in this catheter deadspace, (2) waiting a fixed dwell time to allow for (partial) equilibration with surrounding luminal $P_{CO_2}$, (in most studies 30 min), (4) aspiration of the gastric emptying rate determine this dilutional effect. Finally, when the reserve in cardiac output is limited, a meal may increase blood flow in the proximal gastrointestinal tract while diminishing blood flow and pH in the distal gastrointestinal tract. Hence normal tonometry variables after a meal do not exclude gastrointestinal hypoperfusion. Taken together, it is recommended to perform gastric tonometry in the fasting state as this may increase specificity, even though it may decrease the sensitivity of the method. Alternatively, duodenal feeding may affect gastric tonometry less than gastric feeding.

Confounding factors

In the colon, carbon dioxide production by bacteria may lead to a lower normal pH than in the stomach. Indeed, normal colonic $P_{CO_2}$ may amount to 40.0 kPa, depending on the diet. In horses, who have a large bacterial flora and eat almost exclusively carbohydrates, the normal colonic $P_{CO_2}$ was >26.7 kPa. These observations should prompt collection of data for normal values of colonic $P_{CO_2}$ or the effects of bowel preparation, laxatives or antibiotics, as sigmoid tonometry has been reported to be of value in aortic surgery patients.

Entry of environmental air into the stomach may transiently lower gastric $P_{CO_2}$. Hence a negative gradient may indicate a measurement artefact. This occurs particularly in non-intubated, spontaneously breathing patients.

Sources of error during manual fluid tonometry

Manual fluid (usually saline) $P_{CO_2}$ tonometry is laborious and involves seven steps, each carrying a risk of error so that reproducibility may be relatively poor (Table 2). The steps are: (1) infusion of exactly 2.5 ml in the catheter, (2) waiting a fixed dwell time to allow for (partial) equilibration with surrounding luminal $P_{CO_2}$, (in most studies 30 min), (3) withdrawing and discarding the first 1.0 ml representing catheter deadspace, (4) aspiration of the final 1.5 ml into a syringe, capping it and storage on ice, (5) sending the capped syringe to the laboratory for analysis, (6) calculation of steady-state $P_{CO_2}$ using a correction factor for each dwell time that is less than that needed for full equilibration (Fig. 1). With the help of $P_{CO_2}$ arterial blood bicarbonate content and the Henderson–Hasselbalch equation, a regional, mucosal pH (pHi) can be calculated. It is likely that the sources of error involved in manual fluid tonometry have limited its widescale clinical application.

Catheter deadspace

With each tonometer measurement, the balloon fluid is aspirated and measured in a blood-gas analyser. However, as the volume of the tonometer tube itself is approximately 1.0 ml, the last 1 ml from the balloon resides in this deadspace. With a new measurement, this 1.0 ml deadspace volume is then pushed into the balloon and mixed with freshly infused fluid. Thus the $P_{CO_2}$ in the balloon at the start of the dwell time is variable and depends partly on the last measured $P_{CO_2}$. We found that with dwell times of 10 and 20 min, this phenomenon may cause errors in measured $P_{CO_2}$ of 10% and 6%, respectively, at a deadspace $P_{CO_2}$ of 4.0 kPa. At longer dwell times this error becomes negligible. The deadspace error can be avoided either by a calculation based on the last measured $P_{CO_2}$ and the dwell time or by flushing the tonometer four times with fresh saline to remove all remaining, carbon dioxide-containing saline in the catheter deadspace, thus ensuring a $P_{CO_2}$ of 0 kPa in the tonometer balloon before each subsequent measurement. Errors associated with an unrecognized deadspace effect may have influenced the manufacturer’s correction factors for short dwell times.
Blood-gas analyser bias and dwell time-dependent correction factors

It has become apparent that blood-gas analysers, calibrated for blood or calibration fluid, may underestimate $P_{CO_2}$ measured in saline (or air) by 5–50%.

The magnitude of the error depends on the fluid and blood-gas analyser used, and potentially on the absolute $P_{CO_2}$.

The mean error among analysers (at $P_{CO_2}$ 40 torr) is 12%, ranging from 5% to 29% (Table 3). Consequently, other fluids have been evaluated and used, including succinylated gelatin, and phosphate–bicarbonate buffer.

However, buffered solutions increase the time needed for $P_{CO_2}$ to equilibrate by a factor of 2–5.

Non-bicarbonate-based buffered solutions could share some of the advantages without these disadvantages. Indeed, saline and phosphate had similar $P_{CO_2}$ build-up kinetics and similar propensity for accidental errors, while the bias for phosphate was smaller.

Nevertheless, saline and phosphate may be equally suitable as a tonometer fluid provided strict anaerobic techniques are used and the blood-gas analyser bias is known and taken into account.

It is hard to assess the manufacturer’s correction factors as $in vitro$ studies from which they have been derived have not been published and the role of the deadspace effect and blood-gas analyser bias has not been taken into account. Blood-gas analyser bias has evoked debates in the literature on the issue of whether a correction for the bias should be superimposed on the correction for incomplete equilibration provided in the manufacturer’s correction factors.

Compared with the manufacturer’s correction factors, the correction factors for short dwell times were considerably larger because we avoided the deadspace effect by repeated flushing of the tonometer.

However, the difference at long dwell times cannot be explained by these effects and must be attributed to blood-gas analyser bias.

### Table 2 Sources of error associated with manual fluid tonometry

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<th>Result</th>
<th>Solution</th>
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<td>Error, dependent on fluid and analyser</td>
<td>Evaluate and correct</td>
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<td>Measurement error</td>
<td><em>In vitro</em> 3%, <em>in vivo</em> 6–8%</td>
<td>–</td>
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<tr>
<td>Poor anaerobic technique</td>
<td>Decrease in $P_{CO_2}$ of 8%</td>
<td>Careful anaerobic technique</td>
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<tr>
<td>Improper storage of syringe</td>
<td>Decrease in $P_{CO_2}$ after 60 min of at least 8%</td>
<td>No delay before analysis</td>
</tr>
<tr>
<td>Imprecise dwell time</td>
<td>Error</td>
<td>Instructions and training, or using dwell times &gt;60 min</td>
</tr>
<tr>
<td>Deadspace contamination</td>
<td>Error of 6–10% at 10 or 20 min dwell times</td>
<td>Rinse tonometer four times before fluid installation</td>
</tr>
<tr>
<td>Incorrect correction factors</td>
<td>Error</td>
<td>Use correct correction factors</td>
</tr>
</tbody>
</table>

### Table 3 Blood-gas analyser bias for $P_{CO_2}$ measurements in saline. Bias = Percentage difference from true $P_{CO_2}$ at a $P_{CO_2}$ of approximately 5.3 kPa

<table>
<thead>
<tr>
<th>Manufacturer</th>
<th>First author and source</th>
<th>Bias</th>
</tr>
</thead>
<tbody>
<tr>
<td>Radiometer</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ABL2</td>
<td>Knichwitz, Anaesthesia 1995</td>
<td>–10</td>
</tr>
<tr>
<td>ABL3</td>
<td>Temmesfeld, Intensive Care Medicine 1997</td>
<td>–12</td>
</tr>
<tr>
<td>ABL130</td>
<td>Knichwitz, Anaesthesia 1995</td>
<td>–12</td>
</tr>
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<td>ABL300</td>
<td>Riddington, Critical Care Medicine 1994</td>
<td>–18</td>
</tr>
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<td>ABL330</td>
<td>Temmesfeld, Intensive Care Medicine 1997</td>
<td>–7</td>
</tr>
<tr>
<td>ABL505</td>
<td>Knichwitz, Anaesthesia 1995</td>
<td>–6</td>
</tr>
<tr>
<td>ABL520</td>
<td>Takala, Critical Care Medicine 1994</td>
<td>–6</td>
</tr>
<tr>
<td>ABL620</td>
<td>Venkatesh, Anaesthesia and Intensive Care 1998</td>
<td>–20</td>
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<tr>
<td>Ciba-Corning</td>
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<tr>
<td>Ciba238</td>
<td>Takala, Critical Care Medicine 1994</td>
<td>–5</td>
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<td>Ciba278</td>
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<td>–15</td>
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<tr>
<td>Ciba288</td>
<td>Knichwitz, Anaesthesia 1995</td>
<td>–29</td>
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<td>Ciba840</td>
<td>Kolkman, Intensive Care Medicine 1997</td>
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<tr>
<td>Instrumentation Laboratories</td>
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<td>IL1302</td>
<td>Takala, Critical Care Medicine 1994</td>
<td>–8</td>
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<td>Novostat</td>
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<td>Nova4</td>
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<td>Nova9</td>
<td>Knichwitz, Anaesthesia 1995</td>
<td>–57</td>
</tr>
<tr>
<td>Eschweiler</td>
<td>Knichwitz, Anaesthesia 1995</td>
<td>–51</td>
</tr>
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</table>
incorporated in the manufacturer’s correction factors.\textsuperscript{74} \textsuperscript{79} Indeed, the manufacturer’s correction factors for dwell times beyond 90 min are large. But we have shown that after a 120 min dwell time, the tonometer \( P_{CO_2} \) is 96\% of the surrounding value (Fig. 1) with a corresponding correction factor of 1.03 compared with 1.17 advised by the manufacturer.\textsuperscript{79} That this 1.17 does not relate to incomplete diffusion can be appreciated from a study by Fiddian-Green and colleagues documenting the same \( P_{CO_2} \) after dwell times of 60 and 360 min.\textsuperscript{37} In fact, correction for both incomplete equilibration using the factors supplied by the manufacturer and blood-gas analyser bias may result in overestimations of \( P_{CO_2} \) while incorrect correction may result in underestimations.\textsuperscript{58} \textsuperscript{63} \textsuperscript{79}

In vitro corrections factors have been assumed to apply to the in vivo situation.\textsuperscript{27} However, in comparing \( P_{CO_2} \) equilibration kinetics of tonometers placed in a saline bath and in the stomach of healthy volunteers, the rate of \( P_{CO_2} \) equilibration in the latter proved 31\% slower.\textsuperscript{79} This may relate to gastric juice, gastric folds and mucus covering the tonometer balloon in vivo. Finally, the rate of \( P_{CO_2} \) build-up depends on the size and diameter of the catheter, because of the sink effect of the deadspace.\textsuperscript{124} Hence equilibration may be a few percent slower when using a long, sigmoid-type catheter instead of the shorter gastric type. This is relevant at 10-min dwell times only.\textsuperscript{124} When the sources of error are taken into account, manual measurements are not only reproducible but accurate.\textsuperscript{79} The response of the fluid technique, however, remains relatively slow.

Temperature corrections
As with blood \( P_{CO_2} \) and acid–base balance, it is unclear if tonometer fluid \( P_{CO_2} \) and \( pH_\text{i} \) should be corrected for body temperature if it is not 37°C, for example during hypothermia or fever, as the correction on the blood-gas machine may yield unpredictable results.\textsuperscript{8} \textsuperscript{29} \textsuperscript{63} Nevertheless, it seems prudent to calculate the \( P_{CO_2} \) blood–tonometer fluid gradient from measurements made at the same temperature.

Air and other types of tonometry
From animal and human (post-cardiac surgery) studies,\textsuperscript{117} it has been suggested that \( P_{CO_2} \) determination in gastric air or juice would represent mucosal \( P_{CO_2} \) and could replace the balloon tonometry technique. The balloon technique facilitates and standardizes gastric sampling, and air as opposed to saline tonometry allows more rapid diffusion and thus a shorter response time.\textsuperscript{128}

The recently introduced semi-continuous automated air tonometry involves a pump which automatically inflates (and deflates) via an airtight circuit, 6–8 ml of air into the tonometer balloon every 5–60 min (standard 10 min). This measures \( P_{CO_2} \) in the aspirated air using a modified infrared capnograph and may eliminate some of the sources of measurement error of manual fluid tonometry, thereby increasing the clinical applicability of the technique.\textsuperscript{8} \textsuperscript{27} \textsuperscript{58} \textsuperscript{63} \textsuperscript{80} \textsuperscript{126} \textsuperscript{128} The device is available commercially (Tonocap, Datex, Finland). The response time of the Tonocap, defined as the time needed to reach a 95\% change in \( P_{CO_2} \) at dwell times of 5–10 min, was 10–18 min. Bias was +1\% to −3\% and precision about 1\%.\textsuperscript{27} \textsuperscript{58} \textsuperscript{80} \textsuperscript{128} The relatively long response time, particularly at a high \( P_{CO_2} \), and in comparison with the dwell time, may relate to a deadspace effect. \( P_{CO_2} \) measurements by the air tonometry technique may underestimate (high) saline tonometric \( P_{CO_2} \), even if they are highly correlated.\textsuperscript{27} \textsuperscript{58} \textsuperscript{63} \textsuperscript{79} \textsuperscript{117} This may relate to ‘overcorrection’ of the latter. Air tonometry may also overestimate saline tonometry, possibly as a consequence of the use of wrong correction factors for the saline technique.\textsuperscript{8} \textsuperscript{27} The techniques may have some methodological problems and sources of error in common. This may relate, in part, to the probable need for fasting and inhibition of gastric acid secretion and the value of \( pH_\text{i} \) vs the tonometer–blood \( P_{CO_2} \) gradient. The accuracy of \( P_{CO_2} \) measured by air tonometry is not influenced by temperature corrections.\textsuperscript{8}

Capnometric recirculating gas tonometry involves a tonometer with two lumina, connected to a circulation pump and an infrared sensor, yielding \( P_{CO_2} \) measurements which correlate highly with those obtained by the manual saline technique.\textsuperscript{50–52} Compared with the latter, recirculating gas tonometry has a more rapid response time (5 min), allows online measurements and is more sensitive during haemorrhage and endotoxaemia.\textsuperscript{50–52} A new method involves a fibreoptic \( P_{CO_2} \) sensor which also allows monitoring of \( pH \) and \( P_{O_2} \).\textsuperscript{73} The sensor proved faster and more accurate than saline tonometry, both in vitro and in vivo. The potential for using this probe nasogastrically needs further study. Other techniques that may become available for direct mucosal \( P_{CO_2} \) measurements include the ion-sensitive field effect transistor sensor.\textsuperscript{100} \textsuperscript{103} \textsuperscript{118} \textsuperscript{125} Finally, the tonometry can also be used to measure gastrointestinal luminal nitric oxide concentrations.\textsuperscript{1}

Clinical applications
Diagnostic aid in symptomatic coeliac and mesenteric vascular disease
Some patients with otherwise unexplained abdominal signs of dyspepsia and pain (abdominal angina) may have chronic gastrointestinal vascular disease, but it is hard to establish a relation between signs and symptoms on the one hand and objective indicators of gastrointestinal hypoperfusion, such as abnormal angiographic findings, on the other.\textsuperscript{80} The management of these patients and the effect of reconstructive vascular surgery may be hard to predict. A diagnostic test for gastrointestinal hypoperfusion that also predicts the success of surgery is therefore needed.\textsuperscript{80} \textsuperscript{84} It has been suggested that gastric tonometry during a test meal may be used.\textsuperscript{16} \textsuperscript{39} However, feeding may confound tonometry in the stomach and may thereby yield negative results, even in patients with otherwise proven splanchnic hypoper-
fusión. Alternatively, during exercise, perfusion of the splanchnic organs is at risk because of the ‘steal’ effect of the increased blood flow to skeletal muscle. Hence vascular disease in the splanchnic region and diminished vasodilator reserve could increase the risk of hypoperfusion during exercise. Indeed, many patients with presumed gastrointestinal vascular disease may have abdominal complaints on exercise. We therefore assessed the value of exercise gastric tonometry in managing patients with suspected gastrointestinal hypoperfusion. When the patients were divided on the basis of normal angiograms and those with stenotic lesions in the coeliac or superior mesenteric artery, the $P_{\text{CO}_2}$ gradient during exercise did not increase in the former but increased to about 2.7 kPa in the latter group, with only a minority of patients exhibiting a supranormal gradient at rest. Furthermore, the exercise tonometric findings correlated with a symptom score, the extent of angiographic abnormalities and the presence of discrete mucosal lesions on gastroscopy, consistent with mucosal hypoperfusion and damage. In some patients re-examined after reconstructive surgery, the exercise tonometry test had normalized. Gastric tonometry could help to diagnose acute mesenteric vascular disease resulting in bowel hypoperfusion and necrosis, provided that the stomach is also partly involved and some tissue metabolism remains.

**Estimating prognosis from pH$i$ or the $P_{\text{CO}_2}$ gradient and pH$i$ in critically ill patients**

The conditions wherein (gastric) tonometric variables proved prognostically significant for the development of multiple organ failure and subsequent death are: orthotopic liver transplantation; acute pancreatitis; cardiopulmonary bypass and other types of major, emergency vascular surgery; sepsis; trauma; mechanical ventilation; and $P_{\text{CO}_2}$ gradients, proved prognostically unfavourable in critically ill patients, it can be questioned if tonometry is of additive predictive value. Although splanchnic hypoperfusion and lactate production may influence the systemic indicators, tonometric variables may nevertheless correlate only poorly with systemic factors.

As hyperlactataemia-associated metabolic acidosis is prognostically unfavourable in critically ill patients, it can be questioned if tonometry is of additive predictive value. A low (subnormal) pH$i$ or increased $P_{\text{CO}_2}$ gradient may be superior or have additive value in the prediction of morbidity, that is multiple organ failure and mortality, to global haemodynamic and metabolic variables, including lactate concentration and acid–base variables in the systemic blood of critically ill patients. A subnormal gastric pH$i$ may predict circulating markers of an inflammatory response and a poor outcome with postoperative complications and multiple organ failure after major cardiac, vascular and abdominal surgery. After cardiopulmonary bypass surgery, however, neither pH$i$ nor the $P_{\text{CO}_2}$ gradient in the stomach may constitute an early predictor of increased permeability, endotoxaemia and death in the ICU, whereas global haemodynamics and systemic lactate concentration may predict a worsening disease course.

Even though $P_{\text{CO}_2}$ or pH gradients may be more specific indicators of gastric hypoperfusion than pH$i$, it may be questioned if the former are equally useful prognostic variables as pH$i$, which incorporates a systemic variable. In recent studies in critically ill patients, pH$i$, but not pH or $P_{\text{CO}_2}$ gradients, proved prognostic indicators, suggesting an additive value of systemic acid–base disorders in predicting outcome. In addition, a low pH$i$ may predict occurrence of stress ulcer bleeding and ischaemic colitis when measured in the stomach and sigmoid colon of critically ill patients. Hence, pH$i$ may be superior to the $P_{\text{CO}_2}$ gradient in predicting stress ulcer (bleeding) in the stomach, as the development of stress ulcers may not only relate to local factors but also to systemic acidosis. In fact, lowering of the blood bicarbonate content is a risk factor and systemic alkalisation protects against stress ulcer development during haemorrhagic shock and mucosal hypoperfusion in animals. Taken together, it emerges that the components of pH$i$, $P_{\text{CO}_2}$ as a measure of alveolar ventilation and gastric hypoperfusion, and bicarbonate concentration, as a measure of general circulatory status, are of varying importance in different patients and disease stages.

**Children**

In a study in paediatric patients with sepsis, lactate content, but not pH$i$ or $P_{\text{CO}_2}$ gradients, predicted outcome, suggesting the superiority of systemic over regional abnormalities. In contrast, in many other studies in critically ill (septic) children, pH$i$ and $P_{\text{CO}_2}$ (or pH) gradients were better predictors than systemic haemodynamic and metabolic variables, including blood pH, bicarbonate and lactate concentrations, for haemodynamic complications, multiple organ failure and survival.

**Sigmoid colon**

Even though the appropriate methodology of sigmoid tonometry has still to be established, studies have suggested that the technique may be helpful in predicting tissue hypoxia and injury leading to ischaemic colitis, the main cause of morbidity and mortality after major abdominal vascular surgery. Colonic hypoperfusion detected by tonometry may be associated with endotoxaemia and cytokine release that might contribute to mortality.
Gastric tonometry may also be of predictive value after (emergency) major vascular surgery. 12 88 94

Effect of interventions
As a low gastric pH or increased $P_{CO_2}$ gradient may be prognostically important, studies have examined the effect of various interventions on these tonometric variables, but the beneficial effect on morbidity and mortality of ‘pHi guided’ treatment is still controversial. 7 49 104 108 A variety of treatment procedures consisting of fluid and drug therapy, such as dopamine, dopexamine, epinephrine and norepinephrine, aimed at restoration of splanchnic blood flow, have been evaluated after cardiopulmonary bypass surgery. 11 42 99 110 111 127 Trauma and sepsis. 15 48 49 68 86 87 101 102 107 121

Colloid fluids may be superior to crystalloids but different types may differ in their ability to increase global oxygen delivery and uptake, and to ameliorate a low gastric pH in septic and postoperative patients. 15 91 Some catecholamine treatments in haemodynamically compromised patients may benefit the splanchnic circulation and others may not, as judged by a decrease in gastric $P_{CO_2}$, an increase in pH or both, in a variety of conditions. 7 11 42 48 86 87 91 102 107 110 127 129 For example, norepinephrine and dobutamine decreased the (elevated) gastric $P_{CO_2}$ gradient and increased (subnormal) pH. Dopamine, dopexamine or epinephrine treatment had no effect or tended to further decrease pH in the treatment of sepsis and shock, even if systemic haemodynamics were unchanged or improved. 48 86 87 91 102 127 Dopexamine or dobutamine treatment may not prevent a decrease in pH during and after cardiopulmonary bypass surgery, even if they increase systemic and hepato-splanchnic blood flow and oxygen delivery. 11 42 110 129 In other critically ill patients, dopexamine appeared to improve splanchnic blood flow and a subnormal pH, independent of systemic haemodynamics, suggesting selective splanchnic vasodilatation, while dopamine had no such effect. 121

Vasodilator therapy by infusion or inhalation of prostacyclin may improve hepatosplanchnic blood flow and decrease the tonometric gastric $P_{CO_2}$ gradient during septic shock. 74 Administration of nitroprusside or angiotensin converting enzyme (ACE) inhibitors did not change the gastric $P_{CO_2}$ gradient or pH in septic shock patients and this argues against a role for angiotensin in splanchnic vasoconstriction, although in this study the baseline $P_{CO_2}$ gradient was not markedly increased. 109 111 ACE inhibition may ameliorate an increased $P_{CO_2}$ gradient and a low pH in trauma, however, even if systemic haemodynamic variables do not change. 67 Pulsatile blood flow may better preserve gastric mucosal perfusion adequacy than non-pulsatile flow during cardiopulmonary bypass. 71 Inducing muscle paralysis in mechanically ventilated patients may increase a reduced pH, suggesting redistribution of blood flow from respiratory to splanchnic organs. 89 Treatment of sepsis and trauma patients with antioxidants may prevent a decrease in oxygen extraction abilities and gastric pH, particularly during transient hyperoxia. 7 During large intestinal surgery, gastric $P_{CO_2}$ increased relative to blood values, independent of global haemodynamics; adjunctive clonidine administration did not attenuate this increase. 133 Abdominal decompression in trauma-related intra-abdominal hypertension may normalize a low pH, particularly in patients who survive. 62

Conclusion
Gastrointestinal tonometry of the luminal to blood $P_{CO_2}$ gradient can be used to assess the adequacy of mucosal perfusion provided that, if applied to the empty stomach, acid buffering and carbon dioxide generation are avoided. Appropriate use may improve the accuracy of manual fluid tonometry until the semi-continuous automated air tonometry technique, which may eliminate some sources of error inherent to the manual technique, becomes widely available. This may broaden the clinical applicability of gastrointestinal luminal tonometry as a monitoring tool in a variety of conditions.

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