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Gastric Intramucosal pH Predicts Outcome After Surgery for Ruptured Abdominal Aortic Aneurysm

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Objective: *The mortality associated with repair of ruptured abdominal aortic aneurysms (RAAA) remains obstinately high and many deaths result from multiple organ failure which is likely to be related to splanchnic ischaemia. The aim of this study is to investigate the importance of splanchnic ischaemia in determining outcome from RAAA by comparing gastric intramucosal pH with other methods of assessing the adequacy of splanchnic oxygenation.*
Design and setting: *Prospective cohort of patients following surgery for RAAA admitted to the Intensive Care Unit of Guy's Hospital, London.*
Outcome measures: *Gastric intramucosal pH (pHim) and global haemodynamic, oxygen transport and metabolic variables were measured on admission, at 12 h and at 24 h after admission. Results were compared between survivors and non-survivors and Receiver Operating Characteristic (ROC) curves were constructed to assess the ability of each measurement to predict outcome.*
Results: *The median 24 h APACHE II was 18 and the ICU mortality 45.5%. Gastric pHim was significantly higher in survivors than non-survivors at 24 h (7.42 vs. 7.24, $p < 0.01$). In survivors who had a low intramucosal pH (pHim) on admission there was a significant improvement over the first 24 h (7.26 to 7.40, $p < 0.05$), whereas in patients who subsequently died, and had a normal pHim on admission, there was a significant fall in pHim (7.35 to 7.16, $p < 0.05$). ROC curves showed that gastric pHim was the most sensitive measurement for predicting outcome in these patients.*
Conclusions: *Gastric intramucosal pH is the most reliable indicator of adequacy of tissue oxygenation in patients with RAAA, suggesting that splanchnic ischaemia may have played an important role in determining survival.*

Key Words: Splanchnic circulation - physiology; Aortic aneurysm, abdominal; Aortic rupture; Critical illness; Ischaemia.

Introduction

Since the first successful repair of an abdominal aortic aneurysm in 1951,[1] improvements in surgical and anaesthetic techniques and graft materials have led to operative mortality rates of only 1–3%[2] for elective operations. In contrast, the operative mortality rate for ruptured abdominal aortic aneurysms has changed very little and remains obstinately high with figures of 40–50% being commonplace.[3] Many who survive the initial haemodynamic insult will develop multiple organ failure (MOF), and, despite advances in renal, cardiac and respiratory support, this syndrome continues to be associated with a mortality of more than 70%.[4]

Ischaemia of the colon is a potentially lethal complication of aortic surgery[5–9] and usually results from interruption of the local blood supply via the inferior mesenteric artery. Overt intestinal ischaemia following abdominal aortic surgery remains rare, however, but it has been suggested that a relative, non-occlusive intestinal ischaemia frequently occurs in low-flow states such as ruptured abdominal aortic aneurysms.[10] This is thought to lead to disruption of the enteric mucosal barrier and the translocation of endotoxin and micro-organisms into the circulation,[10,11] thence initiating a sequence of events that culminates in the clinical picture of MOF.

It has been suggested that one means of assessment of the adequacy of oxygenation in a segment of the upper gastrointestinal tract[12–17] is by the measurement of gastric intramucosal pH using tonometry.[10] Inadequate oxygenation of the gastrointestinal tract identified in this way has been associated with an extremely poor outcome in the critically ill.[12,16] This prospective study was designed to compare measurements of gastric pHim with other systemic measurements of

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Table 1. Patient data

22 Patients	3 Female 19 Male	
Age 71 (55 – 88) years		
24 h APACHE II score	All Non-survivors Survivors	18 (12–34) 21 (15–34) 17 (12–24)
Median daily TISS	37 points (28–57)	
ICU mortality 45.5% S.M.R. 1.30		

S.M.R. = Standardised Mortality Ratio; T.I.S.S. = Therapeutic Intervention Scoring System. Values for age, APACHE II and TISS scores are medians, with ranges in brackets.

tissue oxygenation in patients following surgery for ruptured abdominal aortic aneurysm, and, in particular, their relative abilities to predict death.

Patients and Methods

(i) Study population

Twenty two patients (19 males, 3 females; median age 71 years, range 55–88 years) admitted to the Intensive Care Unit (ICU) of Guy's Hospital following surgery for ruptured abdominal aortic aneurysms were studied during the period from April 1991 to June 1992 (Table 1). Three patients were transfers from other hospitals immediately following surgery and consequently five separate surgeons operated on the patients. All patients were receiving mechanical ventilation at the time of study and were resuscitated with the help of a pulmonary artery flotation catheter. Low-dose dopamine (2.5 μg/kg/min) was routinely administered to all cases. Of note, H_2 antagonists were not used routinely. The study was approved by Guy's Hospital Ethics Committee, and informed consent was obtained from the next of kin of each patient.

Using a pulmonary artery flotation catheter and a radial arterial cannula central venous, pulmonary artery occlusion and mean arterial pressures and cardiac index (by thermodilution in triplicate) were measured, and oxygen delivery and consumption indices calculated in the standard fashion. Arterial and mixed venous blood were sampled and arterial pH, standard base excess, and mixed venous lactate concentration measured.

A gastric tonometer ("TRIP" TGS catheter, Tonometrics Inc., Bethesda, Maryland, U.S.A.) was inserted via the nasogastric route into all patients immediately after ICU admission, and its position confirmed by Xray. Gastric pHim was measured as previously described[16] — the tonometer balloon is filled with saline, and the carbon dioxide equilibrates between the saline and the gastric mucosa. The tonometric pCO_2 and arterial bicarbonate concentration are placed in the Henderson-Hasselbalch equation to estimate intramucosal pH. A value of 7.32 or more was considered normal.[17–19]

All measurements were taken on admission, at 12 h and at 24 h after admission. The clinicians were aware of the measurements of pHim but they were not used to alter patient management. For the purposes of statistical analysis, mortality has been defined as death in the Intensive Care Unit. All statistical analysis was carried out using a commercially available statistics software package (CSTAT, Cherwell Scientific, Oxford, U.K.). Data were non-parametric, and are given as medians and ranges. The Mann-Whitney U-test was used to test for differences in measured variables between different groups, and paired data were compared using the Wilcoxon signed rank paired differences test. Chi-square analysis (or the two-tailed Fisher's exact test for small numbers) was used to test for differences in mortality rate between different groups. Correlations were performed using the Spearman rank correlation test. The accuracy of the various measurements at predicting outcome was determined by examining the areas under their receiver operating characteristic (ROC) curves.[20,21] A p value of 0.05 or less has been considered to be significant.

Results

The demographic characteristics of the patients studied are shown in Table 1. The ICU mortality rate was 45.5%, and the standardised mortality ratio (SMR - actual hospital mortality : expected mortality, calculated from the APACHE II scores in relation to specific diagnostic category and the presence or absence of emergency surgery) was 1.30. There were no complications associated with either the pulmonary artery catheters or the tonometers.

Measurements of systemic haemodynamics and oxygen transport

Table 2 gives these data. Pulmonary artery occlusion pressure, cardiac index and oxygen delivery index all improved significantly over the first 24 h (13 to 18mmHg, $p < 0.05$; 3.3 to 3.9 l/min/m^2, $p < 0.01$; and 451 to 560 ml/min/m^2, $p < 0.02$ respectively).

Table 2. Haemodynamic and oxygen transport data

		Survivors (n = 12)	Non-survivors (n = 10)
HR	Admission	100 (60–123)	107 (50–118)
	12 h	94 (50–126)	107 (70–170)
	24 h	96 (65–113)	112 (78–124)*
MAP	Admission	92 (68–180)	71 (61–149)
	12 h	92 (60–125)	90 (57–145)
	24 h	98 (58–120)	77 (55–108)
CVP	Admission	8.5 (6–14)	10.5 (8–14)
	12 h	10.5 (6–15)	14 (7–21)
	24 h	11 (4–19)	13 (10–19)
PAOP	Admission	11 (5–18)	15.5 (12–19)
	12 h	14.5 (8–19)	17.5 (14–32)
	24 h	17 (13–21)	18 (14–21)
CI	Admission	3.4 (2.3–4.8)	3.2 (2–5.8)
	12 h	3.6 (2.6–4.9)	4.1 (2.6–4.5)
	24 h	4.1 (2.8–5.9)	3.8 (3.2–5.4)
DO_2I	Admission	490 (293–763)	411 (223–665)
	12 h	511 (206–943)	592 (248–695)
	24 h	573 (327–925)	547 (386–730)
VO_2I	Admission	124 (79–147)	113 (76–149)
	12 h	112 (55–178)	108 (68–140)
	24 h	122 (108–196)	125 (102–159)

HR = heart rate (beats per minute); MAP = mean arterial pressure (mmHg); CVP = central venous pressure (mmHg); PAOP = pulmonary artery occlusion pressure (mmHg); CI = cardiac index ($l/min/m^2$); DO_2I = oxygen delivery index ($ml/min/m^2$); VO_2I = oxygen consumption index ($ml/min/m^2$); Values are medians, with ranges in brackets.
*=$p<0.05$ – difference between survivors and non-survivors.

Measurements of arterial pH, standard base excess and mixed venous lactate concentration

The metabolic data are presented in Table 3. Arterial pH increased significantly over the first 24 h in both survivors and non-survivors. There was no significant change in lactate concentrations over this time in either survivors or non-survivors. There was a significant improvement in standard base excess in non-survivors over the first 24 h — this was not seen in survivors.

Table 3. Gastric intramucosal pH and metabolic data

		Survivors (n = 12)	Non-survivors (n = 10)
pHart	Admission	7.33 (7.25–7.50)	7.28 (7.17–7.51)
	12 h	7.32 (7.20–7.48)	7.33 (7.24–7.48)
	24 h	7.39 (7.30–7.53)	7.38 (7.29–7.46)
SBE	Admission	–3 (–7 to +3.1)	–6.5 (–16 to –1)*
	12 h	–3.5 (–10 to +5)	–3 (–10 to +2)
	24 h	–2 (–7 to +4)	+1 (–6 to +3)
LACT	Admission	2.0 (0.7–3.6)	2.9 (0.6–11.7)
	12 h	2.1 (0.6–4.1)	2.6 (1–6.4)
	24 h	1.9 (0.3–4.4)	2.4 (1.6–4.2)
pHim	Admission	7.35 (7.13–7.55)	7.31 (6.92–7.49)
	12 h	7.39 (7.11–7.77)	7.32 (7.12–7.55)
	24 h	7.42 (7.16–7.58)	7.24 (7.13–7.42)**

pHart = arterial pH; SBE = arterial standard base excess; LACT = mixed venous lactate concentration (mmol/l); pHim = gastric intramucosal pH. Values are medians, with ranges in brackets.
*=$p<0.02$; **=$p<0.01$ – differences between survivors and non-survivors.

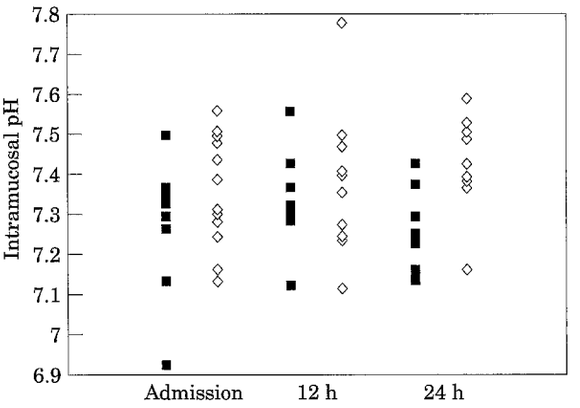

Fig. 1. Gastric intramucosal pH in first 24 h following admission to the Intensive Care Unit according to outcome. (■) non-survivors; (◇) survivors.

Measurements of gastric intramucosal pH (Table 3, Fig. 1)

Eleven (50%) patients had a low gastric pHim (< 7.32) on admission to the ICU and eleven (50%) patients had a normal gastric pHim. Mortality was 45% in both groups. In those patients with a low pHim on admission and who subsequently survived, there was a significant improvement in gastric pHim over the first 24 h (7.26 to 7.40, $p < 0.05$), which was not seen in patients who died. Conversely, in patients with a normal gastric pHim on admission there was a significant fall in gastric pHim in non-survivors (7.35 to 7.16, $p < 0.05$), which did not occur in survivors. Neither absolute values of gastric pHim, nor changes in pHim were correlated with any other measurement.

Discrimination between survivors and patients who died

Figure 2 demonstrates the receiver operating characteristic curves for intramucosal pH and the other measurements at 24 h. The greatest area is that under the pHim curve, showing that gastric intramucosala pH is the most discriminatory with regard to death and survival. At a cut-off point of 7.32, gastric pHim gives a sensitivity of 80% and specificity of 92% for

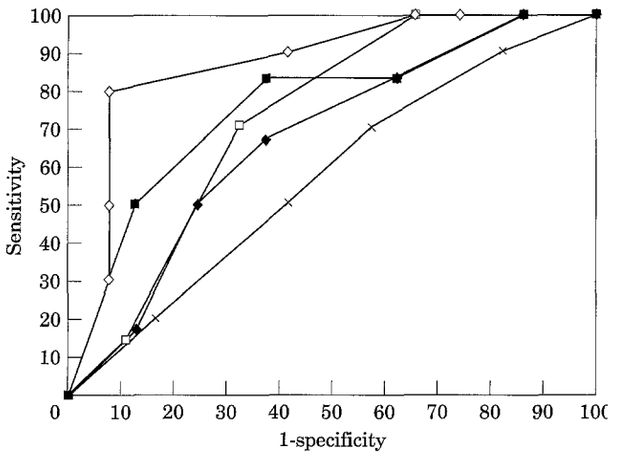

Fig. 2. Receiver Operating Characteristic Curves. Measurements which showed no ability to discriminate between death and survival (i.e. areas less than 50% of maximum) are omitted. (◇) intramucosal pH; (■) heart rate; (◆) mean arterial pressure; (*) arterial pH; (□) mixed venous lactate concentration.

predicting death. Figure 3 shows a tree diagram demonstrating mortality when admission and 24 h values of gastric pHim are considered. The patients have been stratified into four groups depending on these values. If gastric pHim was low on admission the mortality was 45%, and if the pHim stayed low at 24 h despite resuscitation, mortality increased to 80%. In contrast, if the gastric pHim corrected to normal by 24 h, mortality was reduced to 16% ($p < 0.05$). If gastric pHim was normal on admission mortality was also 45%, increasing to 100% if pHim subsequently deteriorated over the next 24 h; this was in marked contrast to a 14% mortality in those in whom the gastric pHim remained normal ($p < 0.01$).

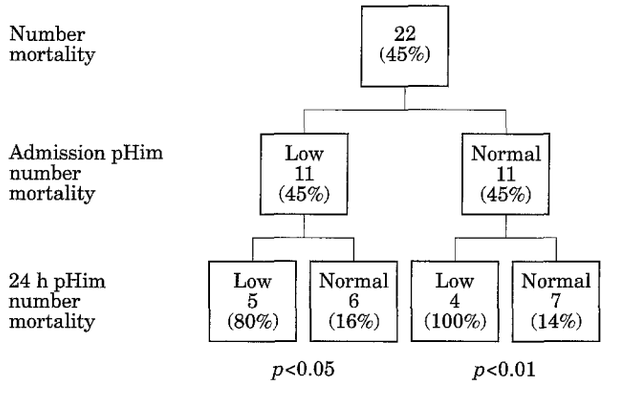

Fig. 3. Mortality rates according to gastric intramucosal pH on admission and at 24 h.

Discussion

Rupture of an abdominal aortic aneurysm is a catastrophic event, often leading to multiple organ failure and death, and this study suggests that gastric intramucosal pH may provide the best means of assessing adequacy of resuscitation in these high risk patients. Much of the organ damage seen in these patients can be attributed to the development of tissue hypoxia.[22] Systemic measurements of the adequacy of tissue oxygenation take no account of regional variations in oxygen delivery and uptake, and there may be significant regional ischaemia present despite indications from traditional measurements that the patient is fully resuscitated.[23]

Disproportionate splanchnic vasoconstriction is an early response to hypovolaemia as blood is diverted towards the heart and brain,[24,25] and the countercurrent system of arterioles and venules in the gastrointestinal villi renders the mucosa particularly susceptible to states of reduced oxygen delivery.[26] An intramucosal acidosis is an early indication of a mismatch between oxygen demand and supply, and may be detected by gastrointestinal tonometry.[10] In animal studies there is good correlation between tonometric and microelectrode intramucosal pH measurements,[27–29] and pHim correlates closely with intestinal oxygen consumption (VO_2).[30] Intramucosal pH is also linearly related to levels of hepatic venous lactate, pO_2 and O_2 saturation,[13] which also reflect the adequacy of splanchnic oxygenation. Thus in clinical practice it would seem that a low pHim indicates inadequate splanchnic oxygenation.

Sigmoid tonometers have been used to detect colonic ischaemia following aortic surgery[31–36] and it has been shown that the development of a sigmoid intramucosal acidosis predicts the subsequent development of ischaemic colitis, major complications and death from MOF. Furthermore, a sigmoid intramucosal acidosis is associated with a significantly higher concentration of endotoxin[37] and cytokines (tumour necrosis factor and Interleukin-6)[38] in the blood.

Several groups have studied changes in gastric intramucosal pH both perioperatively[13,18,35–39] and on the Intensive Care Unit.[12,14–16,28,40] A gastric intramucosal acidosis is common in the critically ill and measurements of pHim seem to be highly predictive of outcome.[12,15–17] In this study we used gastric rather than sigmoid tonometers since gastric tonometry is less likely to be affected by vascular occlusion related to the aortic surgery itself — a fall in sigmoid pHim perhaps being only an indicator of ischaemic colitis secondary to interruption in local blood supply. We

have shown that measurements of pHim in the first 24 h following surgery for ruptured abdominal aortic aneurysms were highly predictive of outcome. The pHim at 24 h was most predictive of outcome because it presumably distinguished those who responded well to resuscitation from those who did not. These results are very similar to those from a larger study in a more heterogeneous group of critically ill patients in our department.[16] The ROC curves in Fig. 2 graphically represent the superior ability of gastric intramucosal pH to discriminate between survivors and patients who died.

We were unable to report any consistent differences in the various haemodynamic and oxygen transport measurements between survivors and non-survivors. Cardiac filling pressures, oxygen delivery index and cardiac index all improved, however, over the time of study, regardless of the outcome, indicating that aggressive resuscitation following surgery led to an improvement in these global variables. Similarly, arterial pH improved over the first 24 h, although there was no difference between survivors and non-survivors. Unlike previous work,[16] we could not show any correlation between arterial pH and pHim in this relatively small group of patients but the base deficit on admission was greater in patients who died. This improved however, with resuscitation and there was no difference at 24 h.

In this study we measured pHim only after the patients had arrived on the Intensive Care Unit following their aortic surgery. This was often several hours since their aortic rupture and the commencement of resuscitation. Patients with a low pHim at 24 h after admission to the ICU had an extremely high mortality of 89%, and it may be that a normal gastric pHim should be an important goal of the resuscitation of patients with a leaking abdominal aortic aneurysm. If this is the case then early monitoring of gastric intramucosal pH is essential. Mythen *et al.*[18] measured gastric pHim during major surgical procedures in 51 patients and found that patients who developed a gastric intramucosal acidosis during surgery spent longer on the Intensive Care Unit, had more complications, were more likely to die and cost more money. Furthermore, the same group[19] have reported that the incidence of perioperative gut mucosal hypoperfusion can be reduced from 50% to less than 10% by the administration of colloid immediately after induction of anaesthesia with the aim of achieving maximum stroke volume.

The measurement of gastric intramucosal pH appears to reveal a state of compensated shock that is not detected by global haemodynamic and oxygen transport measurements, but which compromises the integrity of the gut mucosal barrier; this may lead to the development of distant organ dysfunction and MOF. The tonometer is easy to use and the early deployment of this technique in patients with ruptured abdominal aortic aneurysm might allow the identification of a high risk group at an early enough stage to reverse the intramucosal acidosis. It remains to be seen whether or not treatment with fluid (colloid and blood) and inotropes alone is sufficient to correct the perfusion deficit or whether other more experimental regimens designed to increase splanchnic blood flow (e.g. dopexamine[41]) are required, but there is already some evidence that therapy guided by gastric intramucosal pH might improve outcome.[14]

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