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REPORT OF MEETING

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Report from the meeting: Gastrointestinal Tonometry: State of the Art

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Abstract Gastrointestinal (GI) tonometry, the only clinically available method for the accurate diagnosis of compromised GI blood flow, has been shown to be a sensitive predictor of increased morbidity, mortality and prolonged hospitalization. The recent introduction of the Tonocap, as a means of performing automated air tonometry, has simplified the application of GI tonometry in the clinical setting. Despite this the utility of GI tonometry remains controversial. The GI Tonometry: State of the Art meeting brought together a group of clinicians with a proven track record of research, clinical interest and expertise in this field. The aim of the meeting was to come to a consensus regarding certain issues such as the past and future roles of GI tonometry and standards for its correct usage and interpretation. Finally suggestions as to further research and clinical evaluation were made within a broader discussion regarding the complexities of applying the principles of evidence-based medicine to the introduction of a new piece of medical technology.

Key words Gastrointestinal · Automated Air · Tonometry · Tonocap · Consensus

Introduction

It is difficult to disagree with the hypothesis that maintaining organ blood flow should be a treatment goal for clinicians working in fields such as anaesthesia, high-risk surgery or critical care medicine. The assessment of organ blood flow using physical examination alone maybe incorrect [1]. Monitors, such as the pulmonary artery catheter, thought to help with the assessment of haemodynamic variables, may not only have inherent problems of accuracy and reproducibility [2], they may report global haemodynamic measurements favourably in the presence of ischaemic tissue beds [3, 4]. It would be expected, therefore, that a monitor able to accurately diagnose compromised end organ blood flow, help in the assessment of its treatment and designed to be used in a clinical setting should be welcomed by the clinical community.

The measurement of cellular CO_2 levels has been of clinical interest for many years. Cellular CO_2 levels depend on the balance between CO_2 production via both aerobic and anaerobic metabolism, and CO_2 removal by blood flow from the particular tissue bed and alveolar ventilation [5].

The gastrointestinal (GI) tract is known to receive a disproportionately small portion of the cardiac output

Table 1 General sources of error relating to GI tonometry

Source of artifact	Solution
Correct positioning If the tonometer is placed too close to the pylorus reflux from the duodenum may cause elevation of luminal CO_2 levels.	Catheters have insertion length markers on them at 45, 55, 65 and 75 cm. Manufacturers recommend confirming correct location with an x-ray.
Increased luminal generation of CO_2 The reaction of gastric acid and bicarbonate may cause the luminal generation of CO_2 not related to tissue perfusion. This has been demonstrated to cause marked acute increases in $PgCO_2$ in normal volunteers [42].	The prescription of histamine (H_2) receptor blocking drugs, although traditional, remains controversial. The ability of the critically ill to acidify is often impaired and the response to H_2 blockers may be idiosyncratic and short-lived. It is therefore mandatory to test gastric juice pH and ensure it is greater than 4 for $PgCO_2$ interpretation to be accurate. If standard H_2 receptor blockade cannot achieve this despite increased dosage consider an infusion or proton pump inhibitors.
Enteral feeding Alkaline enteral feed may react with gastric acid and produce CO_2. The presence of food in the stomach may also generate sufficient amounts of CO_2 to cause an apparent increase in $PgCO_2$ [43].	In practice at least 60 minutes should elapse between feeding and reliable $PgCO_2$ measurement.
Suction Nasogastric (NG) suction via the catheter has been suspected as a potential source of error. In healthy volunteers, NG suction has no effect on the results [44].	If in doubt suctioning should be discontinued 30–60 minutes prior to measurement.

when the viability of the organism is threatened e.g., following haemorrhage [6]. Whether one believes that the ensuing gut ischaemia is implicated in the pathogenesis of multiple organ failure or not [7], one can appreciate the logic of measuring GI mucosal cellular CO_2 levels in the diagnosis of GI tract hypoperfusion. The technique of GI tonometry, as a way of indirectly measuring mucosal cellular CO_2 would therefore seem worth pursuing.

The clinical utility of GI tonometry remains controversial [8]. Despite relatively few physicians having extensive clinical experience of GI tonometry, it is common to hear strongly voiced opinions about the subject. The GI Tonometry SOTA meeting was organised by The Centre of Anaesthesia at University College London Hospitals (UCLH). It was funded by UCLH and an unrestricted educational grant from Datex-Ohmeda (Instrumentarium, Helsinki, Finland). A group of clinicians with a proven record of research, peer-reviewed publications and clinical interest in the field of GI tonometry were invited to attend. They represented a variety of medical specialities and a range of European and American centres. (See Appendix A.)

The aim of the meeting, over the course of two days of presentations and discussion, was to come to a consensus view regarding certain issues pertaining to GI tonometry. What is and what is not known about GI tonometry? Should GI tonometry be used to guide therapeutic interventions or to "flag" at risk patients? Is GI tonometry being used in the correct patient groups? What should we call measured variables and is the use of H_2 antagonism mandatory? The concluding discussion was aimed at providing suggestions regarding the future of GI tonometry as a clinical utility and its further clinical evaluation.

What is gastrointestinal tonometry?

Since the early part of this century it has been known that in a hollow viscus, tissue CO_2 diffuses from regional blood vessels into its lumen [9]. GI luminal CO_2 therefore equilibrates with GI mucosal CO_2 which, in turn, depends on the rapid diffusion of intracellular CO_2 across the cell membrane. Intracellular CO_2 will depend on both aerobic metabolism and anaerobic metabolism [5]. When cellular oxygen supply decreases, the production of CO_2 via aerobic metabolism will fall. Below a certain critical level anaerobic metabolism will take place producing lactate and hydrogen ions (H^+) as by-products in the formation of adenosine triphosphate (ATP). If the intracellular H^+ are buffered by bicarbonate CO_2 is produced. Tissue CO_2 is carried to the lungs by venous blood. Hypoperfusion will reduce CO_2 clear-

Table 2 Sources of error specific to automated air tonometry

Temperature A change in temperature by altering the solubility of CO_2 will affect the partial pressure of CO_2 for a given CO_2 content of blood [45]. Automated air tonometry compares $PgCO_2$ measured in the gaseous phase at actual body temperature (which may have deviated from 37 °C) with an arterial blood gas sample corrected by the blood gas analyser (BGA) to 37 °C.	If temperature effects are not accounted for the calculated CO_2 gap or pHi may not be truly representative. Pathological values for CO_2 gap and pHi may remain unrecognised and treatment started for non-significant gaps. $PaCO_2$ must be corrected for body temperature for gap calculations.
Nitrous oxide The molecular weight of nitrous oxide (N_2O) is the same as CO_2, which may result in it being mistaken for CO_2.	Concomitant direct measurement of N_2O via the same infrared technique as CO_2 is performed.
Pressure effects The measurement of CO_2 in the gaseous phase is now affected by the pressure of surrounding anatomical compartments and atmospheric pressure.	A sensor measures absolute pressure in the balloon prior to sampling then measures the CO_2 concentration at a known sensor pressure (slightly less than atmospheric) and finally calculates and displays the partial CO_2 pressure in the balloon according to the following equation: $PgCO_{2\,balloon} = P_{balloon}/P_{sensor} \times PgCO_{2\,sensor}\,(\%) \times P_{ambient}\,(mm\,Hg)$
Equilibration times During the first 2–3 cycles of $PgCO_2$ measurement CO_2 depletion will occur in the gastric balloon, as the air is removed for sampling, resulting in erroneously low values. This effect still occurs despite the sample of air being recycled back to the catheter balloon in order to reduce equilibration times.	A steady state is eventually reached where removal and repletion of sampled gas will not unduly decrease $PgCO_2$ levels. Clinically this takes about 30–40 mins.
$PgCO_2$-$EtCO_2$ gap This index may be used instead of $PgCO_2$-$PaCO_2$. It represents one of the major advantages of automated air tonometry over saline tonometry since no arterial blood gases are required and the monitor is able to display a semicontinuous CO_2 gap trend.	It must be assumed that the $PaCO_2$-$EtCO_2$ difference remains constant throughout measurement. This may not be the case if significant ventilation/perfusion mismatching occurs.

ance causing an accumulation of tissue CO_2 [10]. CO_2 elimination by the lungs will depend on alveolar ventilation and perfusion. This determines how much returns to the cells in arterial blood i.e., the CO_2 content in the afferent vessels of the mucosa. Tissue CO_2 therefore reflects a delicate balance between metabolism, perfusion and lung mechanics.

Tonometry refers to the measurement of the partial pressure of a gas. Hollow visceral tonometry was tested by Bergofsky in the early 1960s by measuring the PO_2 and PCO_2 of saline instilled in the bladder and gallbladder [11]. GI tonometry refers to the same principle but applied to the measurement of the partial pressure of CO_2 within the lumen of the GI tract. The concept of gastric tonometry was in fact proposed even earlier [12]. In the early 1970s the measurement of PO_2 and PCO_2 in subcutaneous tissues of the forearm was further refined by the use of a gas-permeable, saline-filled balloon [13]. This led to the development of the Trip NGS gastric tonometer and sump catheter commonly used when performing gastric saline and, more recently, automated air tonometry. Clinical interest in gastric saline tonometry as a regional monitoring technique was spearheaded by the US surgeon Richard Fiddian Green in the late 1970s [14].

Why measure GI luminal CO_2?

The GI tract is easily accessible in the clinical setting. The GI mucosa is especially vulnerable to hypoperfusion because of the counter-current flow of its microcirculation and the higher critical oxygen requirements of its cells [15]. The arterial and venous flows of an intestinal villus are in opposite directions to the vessels lying in close apposition to one another. It is possible for a large

proportion of blood oxygen to diffuse out of the arterioles directly into the adjacent venules without ever being carried to the tips of the villi [16]. Under normal conditions shunting of oxygen is not harmful to the villi. Occult tissue hypoperfusion, however, may rapidly lead to mucosal ischaemia and injury.

The panel concluded that GI luminal CO_2 represents an index of the balance between metabolism and perfusion in the mucosa of an organ of special interest to the clinician. It was stressed that results obtained should be interpreted in the context of arterial PCO_2.

Should measurement be confined to the stomach?

Having established that the measurement of GI luminal PCO_2 is of potential clinical significance, the stomach has become the natural choice for the performance of GI tonometry because of its ease of access. It is not, however, without potential sources of artefact, in particular, the production of CO_2 from the reaction of gastric acid and refluxed duodenal contents. The mid-gut or sigmoid may provide useful information [17]; the former is difficult to access and the latter technically more challenging than gastric tonometry and not without potential artefact e.g., bacterial production of CO_2.

The measurement of luminal PCO_2 is not restricted to the GI tract but can be performed in other hollow organs such as the bladder or gallbladder [11]. There is little clinical data on the measurement of luminal CO_2 in other sites, probably due to access issues, and certainly no normal values have been established. The investigation of time constants for the accumulation of CO_2 in organs other than the stomach may represent a potential area of research.

The panel concluded that, despite certain potential artefacts, the stomach remains the best site from which to perform the measurement of hollow viscous tonometry.

What is the most clinically applicable method of measuring GI luminal CO_2?

Several methods of measuring GI luminal CO_2 have been used and validated in the past. These include both balloon and balloonless techniques, using both air and saline as equilibration media. Each technique has its own advantages and disadvantages. Balloonless techniques may be cheap but are methodologically difficult to perform. The drawbacks of the balloon saline method have been well documented [18]. The use of the Paratrend 7 sensor (Diametrics Medical, High Wycombe, UK), a sterile single-use device for the continuous measurement of pH, PCO_2, PO_2 and temperature, is a recent development which looks promising. It is comprised of optode-based pH and PCO_2 sensors and a Clark PO_2 electrode. Although trials of the sensor have demonstrated a high level of accuracy, the device is costly, delicate to handle, difficult to position and may require recalibration once in situ [19].

The panel agreed that currently the most viable method of reliably measuring GI luminal CO_2 levels in the clinical setting is by using automated air tonometry (The Tonocap, Datex-Ohmeda, Instrumentarium, Helsinki, Finland); a semi-continuous method of sampling gastric balloon air with infrared measurement of PCO_2. It must not be forgotten that there is no universally accepted gold standard with which to compare automated air tonometry measurements, although most clinical experience and published work has used the balloon saline technique.

How should the GI luminal CO_2 be presented and interpreted?

Literature to date has referred to GI luminal CO_2 in many different ways, making interpretation of data difficult for those not experienced with the technique. The concept of "regional" PCO_2 is not easily understood. The panel felt that identification of an actual measurement site was important. Sampling of gastric luminal PCO_2 should be referred to as $PgCO_2$ and sigmoid luminal CO_2 as $PsCO_2$. Reference to mucosal, interstitial or intramucosal CO_2 ($PmCO_2$, $PimCO_2$) should be avoided as this is easily confusing. Any value for gut luminal CO_2 must be referenced to arterial values, thereby giving CO_2 gap values. The potential use of $PgCO_2 - ETCO_2$ offers an exciting possibility for the online measurement of CO_2 gaps without the need for arterial blood gas analysis. Little clinical data exists on its predictive value. A major concern is the assumption that the difference between arterial and end tidal CO_2 remains constant throughout automated air tonometry use. This may not be the case in those with severe cardiorespiratory compromise or in conditions such as exercise.

The panel felt that, once baseline was established, in the majority of cases one could assume a constant $PaCO_2 - ETCO_2$ difference and therefore resultant $PgCO_2 - ETCO_2$ gaps would prove reliable. Working from first principles, the $PgCO_2 - ETCO_2$ gap would be expected to widen with increasing ventilation/perfusion mismatching due to a greater difference between $PaCO_2$ and ETCO2. The panel speculated whether this combined lung perfusion/gut perfusion signal may in the future be shown to be more predictive than the solely gut perfusion based $PgCO_2 - PaCO_2$ gap, making it in some respects similar to pHi. The panel accepted that no data exists to support such conclusions and therefore agreed that $PgCO_2 - PaCO_2$ remains the gold standard

CO_2 gap which users should be monitoring when using automated air tonometry.

What about pHi?

The impressive past clinical track record of pHi cannot be ignored. One of its major drawbacks is that the pHi calculation is not solely dependent on GI perfusion and oxygenation but is also affected by systemic acid base status, i.e., it combines global and gut specific markers. Indeed this may explain why it has been shown to be such a sensitive predictor of patient outcome. Consequently when researchers are looking at specific GI effects and investigating changes in GI perfusion only $PgCO_2$ and CO_2 gaps should be monitored. Further research needs to be done to assess the prognostic implications of the CO_2 gap alone, which may clarify to what extent the presence of systemic acidosis contributed to the results obtained in previous studies. The panel's conclusion, in agreement with several recent editorials, was that the pHi concept should be abandoned [21, 22].

Potential sources of error when using gastric tonometry

Use of GI tonometry in the clinical setting must be accompanied by a thorough knowledge of potential sources of artefact and error. The majority of errors are now well documented and highlighted by manufacturers' product information. The Tonocap in particular has removed the practical and methodological errors associated with the saline technique, but is subject in turn to a new set of potential artefacts, mostly secondary to the measurement of CO_2 now being in the gaseous phase. Two important sources of error that still apply relate to temperature effects and gastric acid reacting with duodenal contents producing non-metabolic CO_2.

Gastric juice pH must be measured when unexpectedly high $PgCO_2$ values are obtained. Therapy must then be instituted to increase gastric juice pH to a value of greater than 4. An individual's response to H_2 antagonists may be unpredictable so it must not be assumed that gastric juice pH will always change to the same degree following conventional doses of H_2 antagonists. The inclusion of a means of measuring temperature and gastric juice pH on the tonometer may be a useful future development.

The panel concluded that The Tonocap as a means of performing automated air tonometry is probably as reliable as most other pieces of monitoring equipment, such as the pulmonary artery catheter, already accepted in clinical practice. As with all pieces of monitoring equipment, automated air tonometry users must be familiar with its workings, potential faults and sources of error [see Tables 1 and 2].

What about validation?

Since the early part of this century it has been known that $PgCO_2$ reflects gastric intramucosal CO_2 [9]. Automated air tonometry has been proved to reliably measure $PgCO_2$ which reflects the balance between metabolism, perfusion and alveolar ventilation. Certain pathological conditions which may alter the time constant for CO_2 accumulation must be taken into consideration e.g., very poorly perfused mucosa has high CO_2 levels but low levels of production.

What is the normal CO_2 gap?

The value of a normal CO_2 gap is uncertain but derived calculations from past work and, more recently, normal volunteer studies suggests a "normal" $PgCO_2 - PaCO_2$ to be 1 kPa [23]. Assuming a $PaCO_2 - ETCO_2$ gap of 0.5 kPa in the healthy patient, a $PgCO_2 - ETCO_2$ gap of 1.5 kPa may be considered the upper limit of normal. There is still no clinical data evaluating $PgCO_2 - ETCO_2$ gap so users should monitor this with caution. The observation of changing trends in CO_2 gaps may be more informative than looking at their numerical values in isolation. A normal CO_2 gap is no guarantee of tissue normoxia. Negative CO_2 gaps may occur with equipment failure, surplus gastric air from manual ventilation or air swallowing and loss of CO_2 via opened body cavities.

What does an abnormal CO_2 gap mean?

The CO_2 gap can be considered a guide to the adequacy of end organ perfusion. Once artefact has been eliminated an abnormality reflects an imbalance between GI mucosal perfusion and metabolism. The pathogenesis and aetiology of an elevated CO_2 gap cannot be determined simply from its value. The CO_2 gap derived using gastric tonometry has been shown to reflect stagnant hypoxia secondary to hypovolaemia. It will not detect distant isolated areas of stagnant hypoxia such as sigmoid ischaemia following aortic cross clamping during abdominal aortic aneurysm repair or secondary to bowel manipulation and mobilisation during resection. Gastric CO_2 gap has been shown to be relatively insensitive to anaemic hypoxia. Myocardium supplied by a constricted coronary artery will become ischaemic during isovolaemic haemodilution much before the stomach [24]. It is, however, able to detect hypoxia secondary to cytotoxic mechanisms thought to occur in the critically ill population in conditions such as sepsis or following an endotoxin challenge. The CO_2 gap in such cases may not respond to conventional measures aimed at improving end organ perfusion and oxygen delivery.

How does it relate to other surrogates of tissue perfusion?

Many studies that have compared GI tonometry and pHi with other routinely used monitors of cardiorespiratory function have shown them to be more sensitive predictors of morbidity and mortality than global parameters such as blood pressure, cardiac output and urine output [4]. The onset of a low pHi in the ICU may not occur in the setting of cardiovascular instability highlighting the importance of GI tonometry in the early detection of intramucosal acidosis and hypoperfusion [25]. To further demonstrate its value as a warning device, studies in healthy volunteers have shown that hypovolaemia is associated with a widening of CO_2 gap before overt changes in conventional haemodynamic measurements [6].

The panel felt that in daily clinical practice the CO_2 gap should not be considered in isolation, but used in conjunction with information gained from other sources e.g., skin perfusion, urine output, level of consciousness and lactate levels, to build up a balanced clinical picture. The panel appreciated that information gained from these other sources could be normal in the presence of an abnormal CO_2 gap. If faced with such a scenario no panel member would simply ignore the abnormal CO_2 gap, but would respond to it clinically.

The past and possible future roles of GI tonometry

Critical illness and intensive care

In the intensive care unit (ICU) pHi has been found to be a sensitive but poorly specific predictor of mortality and length of stay. In a prospective, randomised, multicentre South American trial of pHi-guided therapy an improvement in survival of over 25 % was reported in patients admitted to the ICU with a normal pHi who were treated according to a pHi-guided protocol [20].

A low pHi has been demonstrated to predict outcome and failure of weaning from mechanical ventilation [26, 27]. This may suggest that withdrawal of ventilatory assistance increases blood flow to the respiratory muscles at the expense of splanchnic flow. The panel was concerned with the low patient numbers and different methodologies of these studies and felt further research was warranted in this area.

Current thinking encourages the early institution of enteral feed in the critically ill population. A low pHi is associated with GI tract dysfunction as judged by failure to acidify the gastric lumen in response to a pentagastrin test [28]. A low pHi has been shown to temporarily occur at the onset of enteral nutrition suggesting (excluding artefact) that some form of perfusional mismatch is taking place. Further studies are required to determine whether GI tonometry has the potential to correctly identify the patient cohort most likely to be successfully enterally fed.

The effect of vasoactive drugs on gut perfusion using GI tonometry has been studied with variable results in the critical care setting. The majority of these have looked at pHi, which may vary in this population due to factors unrelated to GI perfusion affecting systemic acid-base balance. Dopexamine, being an agonist at both dopaminergic and β2- receptors with no α-agonism, is regarded by many authors to be the drug of choice for reversal of low pHi. Several studies have suggested that dopexamine produces significant increases in pHi whereas others have suggested otherwise [29, 30]. Overall, the effects of vasoactive agents on pHi are unpredictable. Among the catecholamines dopamine is the least likely and dobutamine the most likely to increase pHi [31].

The panel identified several areas of potential future development for GI tonometry in the critical care setting. Its introduction into daily ICU practice may allow a reduction in the use of other forms of invasive monitoring. No member of the panel felt it would be necessary to measure cardiac output if blood pressure, urine output, SaO_2, acid base balance, lactate and CO_2 gap were all normal. The observation of $PgCO_2$ trends may provide a more long-term index of changes in perfusion status such as occult hypovolaemia. GI tonometry may help rationalise the choice of vasoactive drugs and the timing of other clinical interventions such as weaning and the commencement of enteral feed. There is currently no specific therapy for tissue hypercarbia secondary to cellular hypoxia thought to be secondary to cytotoxic mechanisms seen in sepsis. The wider clinical usage of GI tonometry in this setting may help us find one.

Trauma patients

GI Tonometry has proved very effective in the field of trauma where young patients with stagnant hypoxia are common and the quick and effective restoration of circulating blood volume is paramount to their care. Splanchnic ischaemia can still exist in trauma victims despite conventional resuscitation endpoints having been attained. Gastric intramucosal pH and global oxygenation variables (delivery and consumption) have been compared as therapeutic indices in the first 24 h following major trauma. Mortality in the group with normalised pHi was significantly lower and those in the pHi guided group tended to show improved survival [32]. As such, GI tonometry variables appear superior to those obtained using a pulmonary artery catheter (PAC) when assessing the adequacy of resuscitation. More recently, the impact of antioxidant and splanchnic directed therapy on persistent uncorrected pHi in a group of critically injured trauma patients was studied

[33]. The protocol included the administration of folate, mannitol and low-dose isoproterenol and the administration of splanchnic sparing inotropic and vasodilatory agents to optimise cardiac output. The "splanchnic therapy" group had fewer organ system failures as well as shortened length of intensive care and hospital stay.

A more significant future role for GI tonometry in the management of trauma patients would seem more probable than in the critical care setting, because the fundamental insult is one of acutely reversible tissue hypoxia secondary to hypovolaemia as opposed to possible cytotoxic mechanisms. Monitoring the CO_2 gap may enable the reduction in the usage of more invasive monitoring, which carries its own inherent risk.

The CO_2 gap may be used both diagnostically and as a therapeutic guide; perhaps in the administration of agents designed to attenuate ischaemia reperfusion injury.

Cardiac surgery

Inadequate perfusion of the GI tract both during and after cardiac operations is well recognised. Hypovolaemia, hypotension, haemodilution and low cardiac output all increase the risk of insufficient blood flow to the gut and liver, all these effects have been studied using GI tonometry [34]. Fluid loading patients undergoing elective cardiac surgery with the aim of maintaining peri-operative gastric mucosal perfusion significantly reduced the incidence of a low pHi, number of complications, days spent in hospital and subsequent cost [35]. The use of extracorporeal circulation and hypothermia are likely to contribute to the inflammatory response induced by cardiac surgery, and the abnormal vasoregulation both during and after surgery and in the immediate postoperative period; GI tonometry may be able to help with the diagnosis and management of both these conditions.

General surgery

GI tonometry is a potentially useful utility in this group if patients are selected carefully. Hypovolaemia is common in the general surgical setting and as such GI tonometry maybe useful in its early diagnosis and management. Studies examining the relationship between pHi and outcome following major surgery have consistently shown that a low pHi (< 7.32) is a predictor of poor outcome [36]. The predictive capacity of GI tonometry may be affected by the site and type of surgery; gastric tonometry will not detect local mucosal effects due to direct handling during major bowel surgery. GI tonometry in the general surgical setting may allow the reduction in use of previously used invasive monitoring and in the guidance of non-invasive goal directed therapy.

Low gastric and sigmoid wall pHi values during abdominal aortic reconstruction have been shown to correlate with the development of ischaemic colitis. They were also highly predictive for the development of major postoperative complications. Cross clamping of the aorta will produce sigmoid ischaemia not recognised by a tonometer placed in the stomach [37, 38]; sigmoid tonometry may be more promising in this field. Unlike the patient group in trauma where stagnant hypoxia secondary to hypovolaemia is the main problem, vascular surgery patients have overwhelming multifactorial pathology that may be difficult to influence by therapeutic interventions made solely in response to automated air tonometry.

Fluid therapy

The administration of fluid is probably the most common intervention performed in response to abnormal tonometry data, as has been evidenced by the studies involving pHi guided therapy.

CO_2 gap and pHi have been shown to reflect acute hypovolaemia much before other haemodynamic parameters and as such could be used for the early diagnosis of an inadequately resuscitated patient.

Gastroenterology

There is a promising role for GI tonometry in this field involving the diagnosis and assessment of abdominal pain caused by occlusive ischaemia secondary to splanchnic stenosis/atherosclerosis. Historically symptomatic occlusive splanchnic ischaemia was thought to be rare due to the abundant collateral circulation present in the gut and the absence of a clinically applicable diagnostic procedure that can separate symptom free from symptomatic ischaemia. The measurement of $PgCO_2$ during and after 10 min of submaximal exercise designed to provoke ischaemia has proved a useful tool in the detection of symptomatic splanchnic ischaemia. It may provide a valuable tool in the investigation of unexplained abdominal complaints and may be used to assess operative results [39].

Can the clinical use of GI tonometry be justified?

The available evidence suggests that the clinical use of GI tonometry may be justified.

The technology now exists to measure $PgCO_2$ accurately in the clinical setting. If the user wants to know this information and knows how to react to it clinically

then they should be using GI tonometry. The user must determine their own comfort level with the technique and subsequently its clinical utility in their own working environment.

When will I see the results of a large prospective randomised controlled trial (PRCT)?

Evidence-based medicine (EBM) is becoming a very important part of clinical practice as health care funding becomes increasingly fixed. Clinicians are being challenged not only by government but also their colleagues to provide the appropriate evidence to justify resources being directed towards a particular technology or treatment, as this will remove funding from other areas.

Both GI tonometry users and non-users frequently refer to the need for a large multicenter PRCT, proving or disproving the effectiveness of CO_2 gap-guided therapy, before actively incorporating its use in the daily practice of their departments. While it may be possible to apply the principles of EBM to the evaluation of a single intervention affecting outcome, applying them to the introduction of a new piece of monitoring is more complex. It is the management driven by and the therapeutic interventions taken in response to a monitor which subsequently affect outcome. Both of these are dependent on many other factors, not simply the numbers displayed on the monitor's screen. Indeed, the panel could think of no therapeutic interventions or pieces of monitoring equipment that had been proved unequivocally to be of benefit. If EBM were to be rigorously imposed many of these treatments would have to be stopped, or monitors withdrawn, presenting a considerable ethical dilemma.

One of the major pieces of work scientifically assessing the clinical impact of a piece of monitoring equipment was the trial conducted by J. Trier Moller et al., "Randomised Evaluation of Pulse Oximetry in 20,802 Patients I&II" [40]. The pulse oximeter had already been proved an accurate and safe monitor. This study was aimed at assessing its clinical efficacy, in particular whether its use decreased the number of major postoperative complications. It was demonstrated, in patient numbers comparable to those in the large multicentre cardiology trials, that pulse oximetry did allow the safe diagnosis and correction of arterial hypoxaemia with significantly more oxygen being used in the monitored group. There was no difference, however, in outcome and complication rate between groups.

The panel thought it interesting, in these days of EBM, that these results showing that pulse oximetry had no effect on patient outcome, had not altered its use by the medical community.

Conclusion

GI tonometry has been shown to be a sensitive monitor of circulatory insufficiency and impaired energy metabolism [41], which heralds complications early on and may be used as a goal for primary resuscitation. Automated air tonometry has been proved safe and accurate. It is the user's response to it that will cause a difference in terms of patient outcome; evaluating this response scientifically is very difficult. This is what is required, however, to declare conclusively the Tonocap (as a means of performing automated air tonometry) a dominant or non-dominant piece of technology. The responsibility remains with those wanting to introduce GI tonometry into their clinical practice to make sure that the operators and decision makers using it have the knowledge to respond appropriately and that these responses and their effects are audited as part of a clinical evaluation programme.

Appendix A: Members of faculty

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