Gastric Tonometry*

The Role of Mucosal pH Measurement in the Management of Trauma

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Effective management of hemorrhagic shock depends on titration of therapies against reliable resuscitation end points. Conventional clinical and laboratory indexes of shock are often slow to respond to progressive circulatory compromise. GI mucosal ischemia resulting from redistribution of blood flow may, however, precede uncompensated shock and may compound the initial hemorrhagic insult by touching off cascades of inflammatory responses. Trauma patients with evidence of subclinical GI ischemia have been shown to have poor outcomes. Gastric tonometry, by detecting the presence of gastric intramucosal acidosis as a proxy of splanchnic hypoperfusion, may facilitate more timely and rational shock resuscitation. This article reviews the development and validation of gastric tonometry and summarizes the clinical studies that have used this modality to guide the management of shock in trauma patients.

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Abbreviations: D\(\text{O}_2\) = oxygen delivery; pHi = intramucosal pH; V\(\text{O}_2\) = oxygen consumption

In 1872, Gross\(^1\) described shock as a “manifestation of the crude unhinging of the machinery of life.” But more sophisticated insights into the causes and pathophysiology of shock have evolved through the debates of clinicians and basic scientists of the 20th century. Although the determinants of shock are increasingly being recognized as complex, the American College of Surgeons’ Committee on Trauma simply defines their net effect as the presence of inadequate organ perfusion and tissue oxygenation to meet metabolic requirements, resulting from a limiting abnormality of the circulatory system.\(^2\)

Shock is broadly classified as hemorrhagic or non-hemorrhagic—the former being by far more common in trauma situations, and the latter being further subclassified as cardiogenic, obstructive, neurogenic, or resulting from sepsis. Most initiatives in the acute phase of trauma care are based on the premise that the prevention or limitation of impairment of perfusion and oxygenation resulting from hemorrhage is critical to favorable patient outcome. Minimization of transport times from the field to definitive-care centers through the creation of streamlined trauma systems has been emphasized as an important method to reduce the duration of shock. Once patients arrive at the hospital, protocols (which include aggressive oxygenation and hemodynamic interventions), along with expeditious surgery, and ICU support are aimed at restoring adequate metabolic support, as reflected by oxygen delivery (D\(\text{O}_2\)) to both injured tissues, and secondarily affected organ systems. Ideally, these interventions should be titrated against objective measures of tissue perfusion to ensure adequacy of resuscitation, and to prevent complications associated with overresuscitation.

THE PROBLEM OF OCCULT HYPOPERFUSION – COMPENSATED SHOCK

Conventional Methods of Estimation of Hypoperfusion

Purely clinical indexes of shock such as pulse rate, BP, skin temperature and perfusion, and urine output are subject to numerous confounding influences, and are notoriously slow to change as patients adapt to severe stress; their disequilibrium may actually reflect late stages of hypoperfusion.\(^3,4\) Even hemodynamic parameters such as pulmonary capillary wedge pressure and cardiac output, which were extensively studied in the context of shock by Blalock and Harrison,\(^5\) are prone to misinterpretation in different patients and can be slow to change in situations of progressive shock.\(^6\)

Resuscitation of shock patients has relied on more direct global indexes of hypoperfusion such as serum pH, base deficit, and lactate levels. These indexes attempt to measure the magnitude of tissue hypoperfusion as reflected by oxygen debt and ensuing acidosis, and all of them have been found to be predictive of surgical complications, multiple organ failure, and even mortality. Unfortunately, although easily measured, the above indexes are global measures, and may not be sensitive indicators of regional or occult hypoperfusion. Global measures of tissue oxygenation can be normal in the presence of significant splanchnic ischemia; in fact, many, if not most patients with uncompensated shock are found to have normal global indexes of ischemia.\(^6\) Oxygen deprivation in one tissue bed may not be appreciated by the systemically diluted global measures. Furthermore, a profound compromise of tissue perfusion may be required to cause a measurable abnormality in these indexes. Unrecognized hypoperfusion can advance ischemic insult to irreversible levels, and can even potentially touch off secondary effects that may be difficult to rectify unless recognized early, or prevented.
Disturbance of microcirculatory autoregulation. Splanchnic oxygen extraction is impaired through some flow is greater than normal. This is particularly relevant if blood consumption as O₂ delivery was decreased. Between the changes in intramural pH and intestinal O₂ consumption as O₂ delivery was decreased: beyond a critical point in intact dog intestine. Regional acidosis, however, is difficult to measure directly; carbon dioxide generated from the acid-buffering effect of bicarbonate is more readily measurable.

The concept that fluid in a hollow viscus could be used to approximate gas tensions in surrounding tissues was first forwarded by Dawson and colleagues. In the late 1950s, Boda and Muryni were able to measure gastric intramucosal PCO₂ indirectly by measuring carbon dioxide levels in samples of air from latex balloons that were allowed to equilibrate with gastric mucosa. This measurement was intended to be a reflection of arterial PCO₂ and was indeed found to correlate closely with arterial PCO₂ in healthy subjects. The investigators recognized that balloon-measured carbon dioxide levels reflected contributions from both regular oxidative phosphorylation, and from buffering of the acidic products of local anaerobic metabolism, and could therefore make the gastric PCO₂ estimates of arterial PCO₂ “misleadingly high.” Interestingly, it is actually this discrepancy which is exploited in the calculation of the intramucosal-arterial PCO₂ gap, an index of mucosal acidosis and oxygen debt.

Fiddian-Green and colleagues revived the concept of gastric tonometry in the early 1980s. Their experiments involving sampling of saline solution directly from polytetrafluoroethylene balloons placed in the stomachs and small intestines of dogs demonstrated a novel method of measuring gastric intramucosal acidosis. The unit of measurement promoted in these studies was the gastric intramucosal pH (pHi), which was calculated using the measured gastric PCO₂ levels and arterial bicarbonate concentrations in a modification of the Henderson-Hasselbalch equation:

\[ \text{pHi} = 6.1 + \log 10 [\text{HCO}_3^-]/.03 \times \text{PCO}_2 \]

Validation

Follow-up studies demonstrated a high degree of correlation between the calculated pHi and pH microelectrode-calculated mucosal pH (R² = 0.79). Progressive occlusion of the superior mesenteric arteries of dogs was noted to induce a sequential fall in pH measured in the small intestine (R² = 0.68), evidence that measured pH had a strong correlation with intestinal ischemia. Intraduodenal pH has, in fact, also been found to correlate well with histologic degree of ischemic mucosal injury.

Clinical studies have also attempted to validate the tonometry tool by demonstrating associations between pHi measurements and patient outcomes. Critically ill patients with massive blood loss were observed to have significantly lower pHi readings than nonbleeding patients. Gastric mucosal pH was found to be a highly sensitive predictor for complications in 85 patients after elective cardiac surgery.

A 1993 prospective cohort study followed 83 patients with acute circulatory failure in their first 24 h after hospital admission. Of all the indexes of perfusion, gastric mucosal pH was found to be a highly sensitive predictor for complications in 85 patients after elective cardiac surgery.
gastrointestinal mucosal pH, but not by systemic measures, is an important contributor to morbidity and mortality in ICUs.

In 1994 Roumen and colleagues\(^1^0\) undertook a prospective evaluation of pH in 15 trauma patients. During the course of the study, three of eight patients with pH \( < 7.32 \) died. All seven patients with pH \( > 7.32 \) survived. The investigators found no correlation between pH and injury severity score, clinical shock, APACHE (acute physiology and chronic health evaluation) II scores, lactate, or base deficit, suggesting that measurement of gastric intramucosal pH provides independent information from conventional indexes of perfusion. Because of the association of low pH with death, the authors\(^1^0\) concluded that “monitoring gastric pH is useful in severely injured patients admitted to the ICU.”

Interestingly, these findings were challenged by the results of a prospective study of Boyd and colleagues\(^1^9\) of 20 patients. They found that systemic metabolic acidosis as reflected by changes in blood and extracellular fluid base deficits and bicarbonate concentrations significantly correlated with pH through the range of acid-base conditions that their patients represented. A blood base deficit \( > 4.65 \) mmol/L and extracellular fluid base deficit \( > 6.13 \) estimated pH \( < 7.32 \) with a sensitivity of 77%, and specificity of 96%. They concluded that information obtained from gastric tonometry can be more easily obtained from measurement of base deficit. They further argued that, on the basis of this data, significant splanchnic ischemia in the presence of a normal base deficit would be rare. The study of Boyd and colleagues\(^1^9\) suggested that information gleaned from tonometry does not improve on that provided by the usual systemic indicators of hypoperfusion (eg, base deficit).

**Techniques**

pHi is derived from the measurement of carbon dioxide in saline solution that is allowed to equilibrate with the gastric mucosa. Saline solution is instilled into a silicone balloon at the end of a nasogastric tonometer. Such tonometers (TRIP NGS catheter; Tonometric Division, Instrumentarium; Helsinki, Finland) are commercially available or can be synthesized from readily available materials in the ICU. As silicone is permeable to carbon dioxide, this molecule equilibrates between the balloon and the intraluminal fluid in a predictable, time-dependent manner. After the equilibration period (at least 30 min), the saline solution is anaerobically withdrawn from the balloon and its PCO\(_2\) is measured using standard blood gas techniques, and adjusted for equilibration time using a time-dependent correction factor. The resultant “steady-state” PCO\(_2\) is used along with the arterial bicarbonate level to calculate the pH.

Acceptable values for pH have been conventionally considered to be within 2 SDs from mean pH levels noted in healthy, ranitidine-treated volunteers; pH \( < 7.32 \) is considered to represent mucosal acidosis by this criterion.\(^8\) Miller et al,\(^2^0\) on the basis of a prospective study of 114 trauma patients, have suggested that pH \( < 7.25 \) or CO\(_2\) gap \( > 18 \) carries more prognostic significance than other conventionally used values.

**Limitations**

Technical limitations of gastric tonometry have been well described.\(^2^1\) The stomach is conveniently accessible for tonometric instrumentation, but because of its generous blood supply may respond slowly to hypoperfusion states. So far, this has not been found to be the case. Montgomery et al\(^2^2\) found a close correlation of pH at various sites along the GI tract in a pig model of hemorrhagic shock. This finding may in part be explained by the fact that cellular perfusion, and consequently ischemia, primarily depends on the configuration of blood supply at the microvascular level; the arrangement of single third-order capillaries in the GI tract may account for the observed similar patterns of hypoperfusion. pH as measured by tonometric methods has also been shown to correlate well with directly measured pH.\(^1^6\) Still, despite these validations, a few sources of measurement error (in arterial bicarbonate and saline solution sample carbon dioxide measurement) require consideration.

**Bicarbonate**

Administration of bicarbonate can alter the arterial bicarbonate, and falsely elevate the pH. In addition, arterial bicarbonate may not reflect mucosal bicarbonate, which may be lower because of local ischemic lactic acidosis; pH may consequently be overestimated. Because of these factors, arterial bicarbonate can be considered to be only an approximation of mucosal bicarbonate. The calculated pH is therefore only an estimate of the true pH. Despite this, animal studies\(^2^3\) have shown good correlation between tonometric and microelectrode measurements of mucosal pH in both low flow states and endotoxemia.

**Carbon Dioxide**

Equilibration of carbon dioxide between the gastric mucosa and the saline solution in the balloon is time dependent. Although optimum times along the GI tract may vary, in the stomach, equilibration times \( < 90 \) min may be too short. Furthermore, arterial sampling of bicarbonate reflects bicarbonate levels at a single point in time; it is difficult to know when it is best to draw an arterial sample. Results are also not immediately available as both saline solution and arterial samples must be analyzed separately in the laboratory; waiting times can adversely affect implementation of resuscitation measures. In addition, discrepancies in the PCO\(_2\) measurements between analyzers can introduce measurement error.

Duodenogastric reflux and the resulting reaction of pancreatic bicarbonate with gastric acid can generate an external source of carbon dioxide. This carbon dioxide will theoretically cause an elevation of carbon dioxide measured by the tonometer, and thereby result in an overestimation of intramucosal acidosis. To address this problem, numerous authorities have suggested the routine use of an histamine type-2 receptor antagonist to block gastric acid production and thereby limit intraluminal generation of carbon dioxide and artificial reduction of pH.\(^1^6\) Again, the fairly good correlation of tonometer and direct pH measurements, even in the presence of varying concen-
ations of HCl and NaHCO₃ in the stomach, suggests little quantitative importance of this effect. A 1998 randomized controlled trial of ranitidine vs placebo also demonstrated no effect of histamine type-2 blockers on pH measurements.

The idea that shock states could cause stagnation of blood flow and result in accumulation of carbon dioxide from all sources was explored by Schlichtig and Bowles. The accumulation of carbon dioxide from impaired clearance has the theoretical potential of causing an overestimate of mucosal acidosis. Stagnation of blood flow, however, was not found to be a significant contributor to mucosal carbon dioxide levels by these investigators.

Costs

The logistical awkwardness of gastric tonometry has probably been an important factor preventing its more widespread application. Costs from its use are attributable to the physical equipment, as well as to the measurement of saline sample carbon dioxide levels and arterial blood gases. But the maintenance of the tonometer, along with the collection of saline samples using strict protocols, and the follow-up of results with various therapeutic implications is labor intensive; nursing care applied to the collection of samples represents significant time and labor cost. New technological developments, including continuous automated measures of aliquots of air rather than saline solution, may reduce the labor investment required for the acquisition of this data.

TONOMETRY-Directed Therapy: Review of the Evidence

From the above discussion, it is evident that gastric tonometry provides a validated estimate of GI mucosal dysoxia despite a few assumptions and technical impediments. The important questions that remain to be resolved are whether this hypoxia carries pathophysiologic implications in the progression of shock, whether currently available therapeutic modalities specifically directed at intramucosal acidosis can alter intramucosal acidosis, and whether such alteration impacts shock pathophysiology and the prognosis of shock. Numerous investigations have been devoted to the resolution of these controversies.

Interventional studies in which specific efforts were made to alter gastric pH were predated by clinical studies that prospectively documented pH in patients in shock and followed their outcomes while attempting to demonstrate associations. Doglio et al. in a 1991 prospective study of 80 patients, showed that mortality, sepsis, and multiple organ failure were high in the group of patients who presented with a low pH. Persistent depression of pH was associated with higher mortality than when pH was correctable by standard resuscitative measures. Patients whose pH was not correctable had a mortality rate of 86.7%, while those who corrected within the first 12 h had a lower mortality rate (36.4%). Patients with an initially normal pH who subsequently sustained a decrease in pH had a 68.4% mortality rate.

A similar prospective study by Chang and colleagues of 20 critically ill trauma patients noted higher mortality rates in subjects with low initial pH which did not correct within 24 h (50% vs 0%) as well as a higher incidence of organ dysfunction. A poor correlation between pH and measured systemic indexes of acidosis and systemic hemodynamic and oxygen transport variables was observed. Only pH, mixed venous oxygen saturation, and base deficit were noted to be significant predictors of mortality at regression analysis. Of all of the indexes of oxygen delivery, only pH was different between subjects with multiple organ failure at 24 h and those without multiple organ failure. The authors argued that on the basis of their findings, splanchnic perfusion is an important factor in the pathogenesis of multiple organ failure, and that an indicator of gastric ischemia provides useful prognostic information in underperfused patients.

These prospective observational studies imply that correctable pH carries a better prognosis than noncorrectable mucosal acidosis, and that resuscitation titrated to changes in pH might be more beneficial than conventional strategies with their inherent risks of underresuscitation or overresuscitation. This is a plausible hypothesis, but is of value only if an intervention is found that can alter mucosal perfusion in a meaningful way. Systemic measures of increasing oxygen delivery could fail for the same reasons that systemic indexes of perfusion are considered to be inadequate: they are systemic, and they may not address problems of hypoperfusion in important microvascular beds.

A few studies have attempted to define therapeutic measures which may have a specific pH-altering effect, and the randomized trials conducted to date in this area have based their interventions on some of these modalities. Silva et al. conducted a meta-analysis of pH studies, showing that vasoactive agents have a variable effect on pH; dopamine increases pH, as does norepinephrine in septic patients. Epinephrine was found to have detrimental effects on gastric perfusion, while prostacycline possibly increased pH in the analysis of available data. Dopamine was least likely to increase pH, a finding that was supported by a crossover study of dopamine and dobutamine by Neviere et al. There was insufficient evidence for evaluation of nitric oxide donors, pentoxifylline, and N-acetylcysteine, although a randomized trial conducted by Molnar et al. found no benefit from the use of acetylcysteine in shock patients. It is clear that more information on therapeutic modalities is required before the threshold to rational pH-based intervention can be successfully crossed.

One of the first prospective randomized trials on tonometry-directed therapy was published in 1992 by Gutierrez et al. Two hundred sixty medical and surgical patients with APACHE scores of 15 to 20 were randomized to receive tonometry-directed therapy (dobutamine and saline solution infusions) or standard ICU management. Patients with low hospital admission pH (< 7.35) had similar survival rates in both trial arms, whereas patients in the intervention arm with normal hospital admission pH had increased survival (58% vs 42%). Based on the latter finding, the authors concluded that “prompt therapeutic action, when pH falls, to increase systemic oxygen transport could protect patients” from disorders.
such as systemic inflammatory response syndrome and multiple organ failure, both of which could result as a direct consequence of global hypoperfusion or reperfusion, or specifically from underperfusion of the gut. The article argued that tonometry-directed therapy could avoid the potential pitfalls of indiscriminate use of pH\textsubscript{i} optimizing strategies (fluid overload, myocardial work [from use of dobutamine], blood transfusion-related complications) by reserving them for patients with documented intramucosal acidosis.

A prospective trial conducted by Ivatury et al randomized 57 trauma patients to receive therapies aimed at the following: (1) normalization and maintenance of pH\textsubscript{i} (blood, IV fluids, dobutamine), or (2) maintenance of DO\textsubscript{2} index of 600 or VO\textsubscript{2} index of 150. In this trial, whose primary focus was trauma, mortality and single-organ failure differences among the two groups did not reach statistical significance (although the latter outcome trended toward significance). No differences in any end points apart from pH\textsubscript{i} were observed between groups. Subgroup analysis demonstrated that the mortality rate from multiple organ failure was higher in patients whose pH\textsubscript{i} did not correct to 7.3 at 24 h (54% vs 6.8%) and that longer optimization times were predictive of mortality on multiple regression. The multiple organ dysfunction scores were higher in patients whose pH\textsubscript{i} did not correct within 24 h. Persistently low pH\textsubscript{i} was frequently associated with intra-abdominal anastomotic leak, compartment syndrome, abscess formation, or other complications. The authors\textsuperscript{31} concluded that “gastric mucosal pH may be an important marker to assess adequacy of resuscitation.” This tentative conclusion possibly reflects insufficient power of the study to discern survival differences between the trial arms. Alternatively, it could result from insufficient correction of mucosal acidosis due to an inadequate intervention. The possibility that the intervention was found to be of no benefit because of underlying fallacies in the theories supporting gut ischemia as a sentinel of correctable shock might, however, be refuted by proponents of gastric tonometry because of the additional data that this trial provided in the subgroup analyses which support the mucosal acidosis-mortality association.

**Discussion and Future Directions**

**The DO\textsubscript{2} Debate**

Gastric tonometry is appealing since it is an index of regional hypoperfusion. It has been argued that the need for such measurement could be obviated by simply ensuring that shock patients receive supraphysiologic levels of DO\textsubscript{2}. Shoemaker et al\textsuperscript{33} and Moore et al\textsuperscript{34} initially found lower multiple organ dysfunction rates in subjects receiving or responding to supernormal DO\textsubscript{2}. Such global therapy may ignore the divide between capillary bed-rich and bed-poor tissues and may result in potentially toxic over-supply of oxygen to some tissues and continued deprivation in some tissues, while subjecting a hemodynamically unstable environment to inappropriate fluid resuscitation and inotropic support with their associated risks. Sensitive tissue beds such as the GI tract could perpetuate systemic organ failure as barrier function declines and bacterial toxins and cytokines are leaked.

Indiscriminate maximization of oxygen transport in heterogeneous ICU populations has, in fact, met with disappointing results and pharmacologic increases in cardiac output. Although believed to be beneficial by some investigators,\textsuperscript{35} it may actually result in increased mortality.\textsuperscript{36} These findings have formed the central premise for tonometry as an early warning of systemic catastrophe and as a guide for more directed therapy, and have paved the way for its application in the prediction of intestinal ischemia in various settings, including abdominal aortic aneurysm surgery, GI ulceration, weaning from ventilatory support,\textsuperscript{36} trauma outcomes, mortality in the ICU, liver transplant outcome, and surgical complications.

It is interesting that, despite extensive experience with gastric tonometry, no trial has documented better outcomes with tonometry-directed therapy, and that there is not much evidence from randomized studies that intramucosal pH can be reproducibly, favorably influenced relative to placebo. Gomersall et al\textsuperscript{37} speculated that failure of their trial to document a difference in outcome could have been the result of inability to produce a significant change in pH\textsubscript{i} using dobutamine and colloid, or because pH\textsubscript{i} is a marker of disease and itself not a factor in the pathogenesis of multiple organ failure as suggested by proponents of the gut-motor hypothesis.

Unpublished data from a prospective randomized study of trauma patients from our institution suggests that even a comprehensive intervention to correct gastric pH\textsubscript{i} in surgical patients does not significantly reduce mortality rates. In this trial, 151 critically ill trauma patients were randomized in three arms to receive standard therapy, placement of a gastric tonometer with otherwise routine management, or gastric pH\textsubscript{i}-directed therapy with inotropic agents, vasodilators, an infusion of agents designed to limit free-radical damage to the gut mucosa (including mannitol, vitamin C, selenium, and polymyxin B), and supplementation of enteral feeds with glutamine, N-acetylcysteine, and vitamins A and E. No significant difference was observed between the groups in rates of multiple organ failure, or mortality.

It is clear that to date, despite measurable associations of low pH\textsubscript{i} and mortality, currently available interventions designed for correction of regional acidosis do not influence mortality. Better data are needed on the role of intestinal ischemia in the progression of shock to multiple organ failure, and on the modifiable determinants of regional perfusion prior to the more widespread implementation of gastric tonometry technology.

**Refinements in Gastric Tonometry**

New developments in measurement strategies and the tonometry instrument itself may make measurements more precise, and may make it more easy to use. Some investigators\textsuperscript{38} have suggested that the tissue-arterial carbon dioxide partial pressure difference may be a better measure of mucosal acidosis than pH\textsubscript{i} as excess carbon dioxide levels have been shown to primarily correlate with anaerobic metabolism as opposed to blood flow stagnation.
and because calculation of pHi requires the assumption that arterial bicarbonate can be used as an estimate of tissue bicarbonate. Capnometric recirculating gas tonometry, an automated technique that pumps air into a gastric balloon and measures equilibrated carbon dioxide levels using infrared analysis, circumvents some of the criticisms of conventional tonometry by providing continuous data output that is rapidly interpretable.8

Other GI Monitoring Sites

Carbon dioxide is measurable in other tissue beds, and the principles first described for gastric tonometry have been extrapolated to other sites in the GI tract. Again, early detection of oxygen debt in the gut may have implications in the prevention of multiple organ dysfunction syndrome. Walley et al39 after studying the properties of small-bowel tonometry, concluded that gastric tonometry measurements are excessively noisy and inaccurate in the detection of gut ischemia when compared to small-bowel tonometry. Unfortunately, placement of tonometers in the small bowel is more problematic than placement in the stomach. Jacques et al40 studied a more accessible tissue bed, the sigmoid colon. Aortic cross-clamping in pigs predictably resulted in steady gastric pHi and consistent depression of sigmoid pHi, but 65% remained within the baseline range. The authors suggested that wide variation in sigmoid pHi limits the value of individual pHi measurement in the detection of ischemia. The tongue and esophagus have also been used as sites of measurement of regional perfusion.15

New Techniques

Balloonless tonometry and the use of fiberoptic carbon dioxide sensors have been described as methods of measurement of the DO2-Vo2 balance41,42; the inflection point of the delivery-consumption curve may be used as a sensitive indicator of tissue-specific flow-dependent Vo2 and increased prominence of anaerobic metabolism. Interestingly, abnormalities in oxygen supply may not be strictly limited to the GI tract. In a study of 16 septic patients and 10 nonseptic ICU control subjects, Nievie et al43 demonstrated that skeletal muscle microvascular perfusion is reduced in septic patients despite normal or elevated whole-body DO2 (at rest and during reactive hyperemia). The application of near infrared spectroscopy techniques44 to various tissue beds may demonstrate similar findings in other regions. Such microvascular abnormalities are postulated to compromise tissue nutrient flow and to contribute to the development of multiple organ failure in septic patients.

Regardless of the measurement technique used, it is becoming clear that the concept of shock as the presence of inadequate organ perfusion and tissue oxygenation resulting from a limiting abnormality of the circulatory system is applicable at the microvascular level and that detection of microvascular hypoperfusion correlates with increased mortality. Redistribution of blood flow to sustain vital organs necessarily implies that certain tissue beds become deprived. Redistribution of detectable magnitude may signal severity of compensated shock and therefore herald mortality, or it may, through mechanisms such as bacterial translocation or liberation of cytokines, contribute to the progression of shock to multiple organ dysfunction. Rational approaches to these circulatory abnormalities will depend on an accurate understanding of the determinants of regional blood flow and on whether specific treatments to improve regional blood flow can alter shock pathophysiology. These controversies can be resolved by continued work at the cellular level, and through the use of well-designed clinical studies with adequate power to detect outcome differences. The diagnosis and management of shock is at another complex and exciting threshold.

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