periodic examinations is perhaps the most equiva-
local of all. Any member of the team may be
chosen; however, most surgeons believe that they
should be the ones to conduct the postoperative
surveillance since they have been responsible for
the major therapeutic intervention in these pa-
tients.7,8 However, this is a local issue that can be
resolved readily. It remains prudent to emphasize
that whoever is chosen, he or she must assume the
responsibility of seeing the patient throughout the
patient’s entire course. The least desirable course
of action is to pass the patient from one team
member to another without continued surveillance
by the primary responsible physician. When this
occurs, the humanistic supportive bond is broken,
and much has been for naught.

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Monitoring Tissue Oxygenation
The Search for the Grail

Generally, it is believed that tissue hypoxia plays a
significant role in the development of organ failure in critically ill patients and is a major factor in
the pathogenesis of the multiorgan dysfunction seen
in the systemic inflammatory response syndrome
(SIRS). This makes intuitive sense because adequate
oxygenation is required for the efficient production
of energy required for the maintenance of cellular
function. Unfortunately, a generally accepted method
of assessing the adequacy of tissue oxygenation has not
been available. Thus, many hypotheses regarding the
putative importance of tissue hypoxia as a cause of
disease, as well as possible benefits of augmenting
tissue oxygen delivery and thus correcting the hypoxia,
have been impossible to test.

Oxygen transport, measured as the product of
cardiac output and the arterial O2 content, is a
commonly evaluated clinical indicator of adequate
tissue oxygenation. However, it should be obvious
that an adequate bulk transport of oxygen by the
cardiovascular system does not guarantee its delivery
to the critical tissues of the body. Factors that
determine the regional distribution of blood flow as
well as events that may alter the normal control of the
microvascular bed prevent any simple translation
of changes in O2 transport into similar quantitative or
even qualitative changes in O2 delivery. Traditional
“global” estimates of tissue hypoxia such as lactate
levels and mixed venous PO2 are nonspecific and
insensitive to regional abnormalities, which, except
in the setting of hypotensive shock, are the most
likely problem. Even direct measurements of indi-
vidual tissue PO2, were they clinically available,
would probably be of little help. Studies suggest that
oxidative phosphorylation can be optimally carried
out at a cellular PO2 in the 2 to 3 mm Hg range.1 This
low operating level would make any change in tissue
PO2 too subtle to use as a clinical indicator of a
correctable problem.

Techniques that directly appraise the state of
tissue energetics would be an optimal approach and
such methods do exist. Magnetic resonance spectro-
copy and near-infrared spectrophotometry can di-
crectly measure the energy charge of living tissue.
However, at the moment, both techniques have far
too many technical limitations to be clinically useful.
Another marker of the onset of significant anaerobic
metabolism is the development of tissue acidosis.
Anaerobic glycolysis results in the accumulation of
H+ ions and a fall in tissue pH. With this in mind,
Grum and coworkers, in search of an early indicator
of bowel ischemia in patients undergoing abdomi-
nal aortic aneurysm resection, found that a rise in the
intraluminal Pco2 mirrored similar changes in the
bowel wall and signaled the onset of tissue acidosis.2
A subsequent modification of this technique, mea-
surement of the intraluminal-arterial PO2 difference,
as used in the paper by Duke and colleagues in
this issue of CHEST (see page 174), appears to
increase the specificity of the measurement. While
this technique monitors only the portion of the GI
tract in which the luminal Pco2 is being assessed,
there is some rationale to choosing the GI tract as a
signal organ. It has a low threshold for changes in O₂ delivery,⁵ and bowel ischemia is thought by some to initiate or at least contribute to the systemic response that results in SIRS.⁴

Using this tonometric approach to assess the interstitial pH of the bowel (pHi), a number of workers have shown that a falling or persistently low pHi indicates a poor prognosis in critically ill patients.⁶⁻⁸ Thus, it is not too surprising that Duke and colleagues found that the intraluminal–arterial Pco₂ difference was better than base deficit, blood lactate, arterial pH, heart rate, and mean arterial pressure in predicting outcome in children receiving extracorporeal life support for various causes of cardiovascular or respiratory failure. This study corroborates prior conclusions regarding the prognostic superiority of a specific marker of organ hypoxia over some “global” index.

Studies demonstrating the prognostic power of this technique unfortunately beg the more important question of whether or not the recognition of tissue hypoxia can be used to alter outcome. Clinical trials of increasing O₂ transport to empirically determined “super-normal” levels in an attempt to improve the survival of many groups of critically ill patients have been attempted. Unfortunately, the results have not been uniform. In some studies the treatment group did better,⁹⁻¹⁴ more often there was no significant difference,¹¹⁻¹⁵⁻¹⁹ and in one study the treatment group did worse.²⁰ The reason for these desperate results is unclear, although a number of possibilities exist. The variable manner in which the studies were carried out, compounded by the possibilities of bias due to the inability to blind the observers, the difficulty of true randomization, and problems of statistical analysis, make interpretation of the results difficult. On the other hand, there may indeed have been true differences in the pathophysiology of the different patient groups or in the efficacy of different approaches to therapy. The onset of physiologic stress is more predictable in the postoperative setting than in the typical mixture of ICU patients. Augmenting cardiac output with fluids may be different than a similar increase engendered by blood, and both may have a different physiologic effect than the use of inotropic or vasoactive agents. Most importantly, there may be subgroups within each population in whom an intermediate level of O₂ transport augmentation was more beneficial to tissue oxygenation than the empirically determined study goal.

All of these complexities emphasize the need for an effective index of adequate tissue oxygenation. Is pHi the answer? The jury remains out. While tantalizing preliminary data exist,¹¹ the need for a well-controlled trial of O₂ transport augmentation guided by pHi is obviously required before we know if the grail has indeed been found.

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