Pathophysiology, monitoring, and therapy of shock with organ failure

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Introduction:
The conventional approach to resuscitation and management of critically ill patients

Half of the two million persons in the United States who die annually die acutely of circulatory problems, shock and its sequelae, organ failure. Since shock is the commonest complication of high risk surgery, trauma, and acute illnesses, its early recognition and correction are key to its prevention. Thus, it is extremely important to recognize shock as early as possible and to vigorously treat it while it is still readily reversible.

In the late stage, it is easy to diagnose shock by hypotension, oliguria and collapse, but at that stage, therapy is often ineffective. The more important question is, „How can we recognize shock in its earliest stage when therapy is most effective?" Unfortunately, the conventional approach could not be worse. We routinely recognize shock by subjective symptoms and imprecise signs: cold clammy skin, weak thready pulse, unstable vital signs, pallor, and altered mental status. These criteria are subjective, imprecise, and observer-dependent; they are remote from physiologic evidence that could objectively characterize nonsurvivors'patterns.

The first question to ask in high risk patients is, „How do you make early practical therapeutic decisions at the bedside?" Traditionally these decisions are often inadequate because we have developed too many simple solutions to complex problems without considering the underlying pathophysiology; for example, „If the B.P. goes down, give fluids; if the fluids don't work give dopamine."

In the traditional approach „one-at-a-time" approach, each abnormality is identified, documented and then corrected. As soon as the first defect is corrected, the next deficit is sought, measured, evaluated, and then corrected; the process is repeated as long as abnormalities are found. However, this may result in uncoordinated, disorganized and sometimes contradictory therapeutic plans.

The most commonly monitored variables in hospitalized patients are the mean arterial pressure, heart rate, central venous pressure, and cardiac output. According to the conventional therapeutic paradigm, the aim of therapy is to return these values to their normal range. In several thousand monitored values of patients who died, we were able to restore 76% of them to the normal range; nevertheless, these patients still went on to die. Then, survivors were found to have essentially the same survival percentages in these variables. Obviously, something clearly is wrong with the conventional approach to monitoring and treatment of acutely ill patients. First, the wrong variables may be monitored. Second, normal values may be appropriate for normal healthy people, but not appropriate for critically ill postoperative patients (1-4).

The conventional etiological approach characterizes hemorrhagic, cardiogenic, traumatic, neurologic, and septic shock in terms of: a) signs and symptoms, b) laboratory findings, c) pathophysiology, and d) therapy. This conventional approach is widely accepted,

simple, straight forward, easy to understand, but wrong! It is wrong for three reasons; first, the signs and symptoms are the same for each etiology; they all have oliguria, tachycardia, and collapse. Second, the laboratory is not at all diagnostic. Third and worst of all, it has a one-dimension description of the pathophysiology. This one-dimension approach precludes understanding of the interactions of the three circulatory components: cardiac function, pulmonary function and tissue perfusion. Tissue perfusion represents the important overall purpose of the circulation, to supply body metabolism.

Physiologic approach to invasive monitoring of high risk patients

The alternative is to observe surviving patient's responses to hemorrhage, high risk surgery, trauma, and shock. It was found that survivors developed a pattern of supranormal cardiac index (CI), O_2 delivery (DO_2), and O_2 consumption (VO_2). The hypothesis is that if we prophylactically drive high risk patients to the survival pattern with early aggressive therapy that optimizes cardiac output, oxygen delivery and consumption values, outcome may be improved.

Recognition of shock in the earliest period often depends on subjective symptoms, rather than precise physiologic measurements. Because of this uncertain definition of shock, hemodynamic and oxygen transport variables were monitored in 708 high risk, surgical patients preoperatively, during surgery, and in the immediate post-operative period (5). The survivors' values were compared with the values of those who died to describe the early temporal patterns. The first and most dramatic differences occurred in CI, DO2 and VO2; nonsurvivors were found to have relatively normal values, while survivors had markedly increased values for these variables (1-17).

Invasive monitoring of oxygen transport to evaluate the pathophysiologic pattern of shock

The essential pathophysiology of the shock state after high risk surgery may be described in terms of oxygen metabolism and oxygen balance. The delivery of oxygen is the most clearly defined direct measurement of circulatory function and, more importantly, the capacity of the circulation to compensate for exercise, stress, trauma, sepsis, and other acute illnesses. Oxygen consumption is the most direct measurement of metabolic activity. However, it measures what the patient is actually consuming, but not necessarily what the patient may need. Based on these key variables which were found to be early outcome predictors. Now it may be hypothesized that inadequate delivery of oxygen in the face of increased metabolic demands (for example, recovery from trauma, surgical operations, blood loss, infection, etc.) leaves the patient with tissue hypoxia, organ dysfunction, organ failure and death.

The first problem is to identify the appropriate patients to monitor. In the U.S., most patients survive major surgery, but 1.5 to 2% die; since there are about 35 million major operations per years this means that over half a million patients die postoperatively every year. If we look at only the high risk group of patients, about 7% of patients subjected to surgery are high risk and about 20 to 30% of them die in the immediate postoperative period. High risk criteria consist of prior cardiac infarction or stroke, extensive ablative cancer surgery, severe multiple trauma, massive hemorrhage, septic shock, respiratory failure, renal failure, acute abdominal catastrophes (4); these 7% high risk group contributed to 82% of deaths on our surgical service (1).

Prospective clinical trial

Using the survivors oxygen transport values as goals of therapy, we studied the effects of

optimizing these values prospectively, first by allocating patients of each of three surgical services to the protocol or control groups. In the first five years, we found that patients who entered the study on the second, third, or fourth postoperative day had successively less and less effect from optimizing CI, DO_2, and VO_2. In the last year we prospectively performed a pre-operatively randomized controlled trial in the following manner. After each high risk patient was identified preoperatively, and signed an „informed consent," a sealed opaque envelop was opened to allocate the patient to one of three groups: a) the central venous catheter, b) the Swan-Ganz pulmonary artery (PA) catheter with normal values as the goals of therapy, and c) the PA-protocol group which had a PA catheter with goals of therapy as the supranormal values determined empirically from a previous survivor group as CI > 4.5 L/min/m², DO2 as > 600 mL/min/m², and VO2 > 700 mL/min/m² (1).

The results of the study are shown in Table 2 and Table 5. The central venous catheter was as good as the PA catheter-control group, if all that was done with the catheter was to achieve normal values. However the PA-protocol group had a significant reduction in the mortality from 32% to 4% compared with the normal values of the PA-control group. Also the PA-protocol group had a significant 67% reduction in the ventilator days, a 30% reduction in ICU days and hospital days and a 25 % reduction in the hospital costs (1).

Of interest was a group of high risk patients whose doctors didn't think they were sick enough to need invasive monitoring; this „non-randomized group" had the highest mortality and highest percentage of organ failures; ironically, sixty percent of these had a PA Catheter placed postoperatively after they developed a life-threatening post-operative cardiorespiratory event. However, the PA Catheter at this time did not improve the overall group mortality. The data showed that the PA catheter can prevent organ failure if oxygen transport is optimized in the first 8 to 12 hours, but is not able to reverse lethal organ failure after it occurs.

Table 1: Normal values for invasively monitored hemodynamic variables

Variable	Formula	Normal Value	Units
Cardiac index	CI = cardiac output/BSA	3.2 ± 0.2	L/min¹ m²
Systemic vascular resistance index	SVRI = 79.92 X (MAP-CVP)/CI	2180 ± 210	dyne s/cm⁵ m²
Pulmonary vascular resistance index	PVRI = 79.92 X (MPAP - WP)/CI	270 ± 15	dyne s/cm⁵ m²
Mean transit time	Direct measurement	15 ± 1.4	seconds
Central blood volume	CBV = MTT X CI X 16.7	830 ± 86	mL/m²
Stroke index	SI = CI/HR	46 ± 5	mL/m²
Left ventricular stroke work	LVSW = SI X MAP X 0.144	56 ± 6	g m/m²
Right ventricular stroke work	RVSW = SI X MPAP X .0144	8.8 ± 0.9	g m/m²
Left cardiac work	LCW = CI X MAP X .0144	3.8 ± 0.4	kg m/m²
Right cardiac work	RCW = CI X MPAP X .0144	0.6 ± 0.06	kg m/m²

*.0144 and 79.92 are conversion terms.
Abbreviations: BSA = body surface area; MPAP = mean pulmonary artery pressure; WP (or PAOP) = pulmonary artery occlusion pressure (or wedge pressure); MTT = mean transit time; HR = heart rate.

Table 2: Oxygen transport variables

Variable	Formula	Normal Value	Unit
Arterial Hgb O_2 saturation	Direct measurement	96 ± 1	%
Mixed venous Hgb saturation	Direct measurement	75 ± 1	%
Arterial oxygen content	$CaO2 = SaO2 \ X \ 1.36 \ X \ Hgb* \ + (.0031 \ X \ PaO_2)$	19 ± 1	mL/dL
Mixed venous O_2 content	$CvO2 = SvO2 \ X \ 1.36 \ Hgb* + (.0031 \ X \ PvO_2)$	14 ± 1	mL/dL
Oxygen delivery	$DO2 = CI \ X \ CaO_2$	520 ± 16	mL/min m^2
Oxygen consumption	$VO_2 = CI \ (CaO_2 - CvO_2)$	131 ± 2	mL/min m^2
Oxygen extraction	$O_2 \ ext = DO_2/VO_2$	26 ± 1	%

*Hgb = hemoglobin

Hemodynamic values of the protocol and control groups were described for the preoperative, intraoperative, and postoperative periods. The cardiac index values of the protocol group reached optimal values within 8 to 12 hour postoperatively, while the control values remained at the preoperative levels. There were no significant differences in the other hemodynamic variables. The oxygen transport values of the control group were maintained at their preoperative normal values, while the DO2 and VO2 values of the protocol group reached their optimal goals in the first 12 hours postoperatively. There were no significant differences in blood gases, hematocrit, or other oxygen transport variables. The striking difference between the groups was the incidence of organ failure. The protocol group had only one patient with ARDS, while the control groups had significantly more patients with organ failures (Table 2). There were no significant differences between the groups in the incidence of complications not due to organ failure. That is, the oxygen transport optimization protocol did not protect against local mechanical or anatomic complications such as wound infection, dehiscence, postoperative bleeding, nosocomial infections, and drug or transfusion reactions (1-4).

Measurement of oxygen debt

These observations lead on to a corollary hypothesis that states that the basic underlying problem in all shock is tissue hypoxia and this can be measured as the net cumulative oxygen debt. If this oxygen debt is prevented or corrected early on, there will be a reduction in organ failure and death. Oxygen debt can be measured directly in surgical patients by measuring oxygen consumption preoperatively and then, correcting for the effects of anesthesia and temperature, extrapolating this value to the intra-operative and immediate postoperative periods. Integrating the area under the curve gives us the net oxygen excess or debt at any specified time. Oxygen debt was measured in 253 consecutively monitored patients with measurements taken pre-operatively, intra-operatively and postoperatively (17). This is easy to document in surgical patients, where pre-illness baseline values can be obtained, and the times of the beginning and end of the operation are easily determined. The patients, who survived without organ failures had the smallest oxygen debt which lasted an average of 12 hours. By contrast, the nonsurvivors, all of whom died with organ failures, had a continuing oxygen debt during the 48-hour period

of observation. Intermediate between these two groups were the data of survivors with organ failures who had oxygen debts lasting about 24 hours. In the prospective controlled trial described above, the protocol patients had less oxygen debt intraoperatively, and the oxygen debt became an excess more rapidly than did the control patients who had normal values as therapeutic goals (4).

The common denominator in all forms of shock is oxygen debt and is limited by reduced oxygen consumption (VO_2) to levels less than the body needs. Secondly, oxygen debt from reduced VO_2 is also the major determinant of outcome. The concept was described over 35 years ago by Guyton et al (18,19) who anesthetized and bled dogs; those that accumulated an oxygen debt of 100 ml/kg all survived, while dogs that accumulated a debt of 140 ml/kg all died; the 50% mortality occurred at 120 ml/kg. Our studies on high risk surgical patients (as compared with hemorrhage in dogs) corroborated the conclusions of Guyton (18,19).

Therapeutic goals of invasive monitoring

We conclude from these studies that at least two-thirds of the patients who die postoperatively do not die of anatomic reasons, they die of physiological problems that can be described, predicted and prevented. The second important conclusion is that values observed in critically ill high risk patients who survive ought to be considered as the goals of therapy. Third, if these goals are achieved early, i.e., in the first 8 to 12 hours postoperatively, there will be a reduction in morbidity and mortality. This is not only the case in the surgical patients but also in a wide range of medical problems (6-16).

Branch chain decision tree

A Branch Chain Decision Tree or Clinical Algorithm for management of high risk patients shows the physiologic criteria for each major decision node (20). The upper loop describes criteria for fluid therapy; the second describes criteria for inotropic therapy; the third for vasodilators, and the lower for vasopressors.

The goals of therapy are different in various types of illness (Table 3). In postoperative patients, the mean cardiac index of survivors was observed to be 4.5 L/min/m^2, in trauma patients 5.0, in sepsis 5.5, and in cardiogenic shock from acute myocardial infarction 2.5 L/min/m^2. These values are appreciably affected by age, co-morbid clinical conditions, degree of blood loss, length of time in shock, and the reserve compensatory capacity of each vital organ.

When oxygen consumption values are plotted against their corresponding value of O_2 delivery, there is an initial sloping line showing increasing VO_2 as the DO_2 increases sharply, indicating supply-dependant VO_2. There is a critical point beyond which further increase in delivery of oxygen does not increase the oxygen consumption, indicating supply-independent VO_2 above this point. We use the mean values of each etiologic category of shock as a first approximation to the true goals. Then additional fluids or inotropic agents are given to increase the DO_2 in order to determine if VO_2 also increases (as in supply-dependent VO_2). Optimization occurs when further increases in DO_2 no longer increase VO_2 (supply-independent VO_2).

Relative effectiveness of crystalloids and colloids in patients with ARDS

Invasive monitoring is extremely useful to evaluate objectively and scientifically the relative efficacy of alternative therapies in various clinical conditions. For example, it is said almost as a party-line dictum that albumin should never be given in adult respiratory distress syndrome (ARDS) patients, because colloids leak into the lung tissues through the pulmonary capillaries and drag a lot of water

Table 3: Invasive and noninvasive hemodynamic values for survivors and nonsurvivors

Variable, unit	Optimal Value	Survivors (N=103) Mean ± SEM	Nonsurvivors (N=48) Mean ± SEM	P value
CI, L/min/m^2	4.0	4.14 ± 0.02	3.57 ± 0.03	0.001
MAP, mmHg	85	88 ± 0.37	83 ± 0.69	0.08
SapO$_2$, %	98	99 ± 0.05	96 ± 0.26	0.001
O$_2$ delivery	600	624 ± 39	520 ± 47	0.01
O$_2$ consumption	140	170 ± 76	127 ± 67	0.01
PtcO$_2$/FiO$_2$, torr	200	206 ± 2.9	93 ± 2.6	0.001

CI cardiac index, MAP mean arterial pressure, SapO$_2$ arterial hemoglobin saturation by pulse oximetry, PtcO$_2$/FiO$_2$ transcutaneous oxygen tension indexed to FiO$_2$

into the lungs with it. However, this concept had not been experimentally verified and, therefore, we tested it by a prospective, random-order, cross-over designed study of patients in early ARDS (defined as within 24 to 48 hours after the diagnosis was established) given 1.0 liters of Ringer's lactate (RL) and 100 mL of 25% albumin (ALB); each agent was given to each patient, half of the patients receiving RL first and then crossed over to ALB, the other half receiving ALB first and then RL. These data show that 1000 ml of RL increased blood volume only 200 mL at the end of the infusion, which was the maximum volume effect, while 100 ml of 25% ALB increased blood volume an average of 450 mL. In essence, ALB didn't leak, rather it dragged 350 mL of interstitial water back into the plasma volume and with this increase in plasma volume there was increased cardiac index, colloidal osmotic pressure, and O$_2$ consumption without worsening the P(A-a)O$_2$ gradient or pulmonary shunt. Albumin as well as starch significantly improved both DO$_2$ and VO$_2$, while RL only transiently increased DO$_2$ and actually decreased VO$_2$ probably because of increases in the diffusion pathway and the diffusion time.

We subsequently expanded this series to over 400 studies in 212 patients in early ARDS (24 to 48 hours after diagnostic criteria were met) given 500 ml of 5% plasma protein fraction, 100 ml 25% albumin, 500 ml of 10% Dextran-40, 1000 ml Ringer's lactate, 500 ml of whole blood (l unit), or 500 ml of packed red blood cells according to appropriate indications. Data have shown that colloids increase cardiac index, but RL did not. The colloids, whole blood and packed red cells all improved DO$_2$ and VO$_2$, but Ringer's lactate (RL) did not significantly change either of these variables. However, in the late stage of ARDS and septic shock, capillary leak does occur, and there were minimal improvements in response to fluid interventions.

When the changes in VO$_2$ plotted against the corresponding changes in DO$_2$, there are increases in both DO$_2$ and VO$_2$ after the colloids and blood, but not after crystalloids. Since O$_2$ can not be stored, increases in DO$_2$ that increase VO$_2$ indicate that there had been preexisting oxygen debts which are partially restored

Future directions for noninvasive monitoring of acutely ill emergency and critically ill patients

Finally, we would like to briefly outline the future direction of both critical care and emergency care in noninvasive circulatory monitoring because these are easier, cheaper and more feasible to use. Although previous

bioimpedance instruments have been unreliable, new high tech hardware and software innovations from defense industries have greatly improved bioimpedance technology. These newer technologies provide more reliable data that allow us to titrate therapy more closely according to physiological criteria. We compared the new bioimpedance method (Noninvasive Medical Technologies, Auburn MI) to the standard thermodilution technique in 2192 paired measurements; there was a close correlation (r=0.86). These data demonstrated satisfactory agreement between the two methods and the ability of this bioimpedance method to track and trend thermodilution changes.

Most importantly, the new impedance cardiac output system may be combined with other noninvasive monitoring systems including pulse oximetry to assess pulmonary function and transcutaneous oximetry to assess tissue oxygenation. These noninvasive monitors evaluate the interactions of the three circulatory components to identify, diagnose, and recommend treatment in early stages when circulatory problems are easily reversible.

Noninvasive monitoring of emergency patients

The advantages of these noninvasive monitoring systems are that they provide continuous on-line real-time display of cardiac function, pulmonary function, and tissue perfusion that can be used anywhere in the hospital including the emergency department, prehospital area or physician's offices. Irrespective of the precipitating event in shock, the cardiac, pulmonary and tissue perfusion functions should be evaluated in terms of the primary problem, pathophysiology, compensation mechanisms, decompensations, and therapy (Table 4). This allows a more comprehensive approach to acute circulatory problems irrespective of the precipitating etiological event, ie, cardiogenic, hemorrhagic, traumatic, septic, or post-surgical. This offers an integrated physiologic approach for treatment of the critically ill patient.

The temporal patterns of noninvasive circulatory variables of the survivors and nonsurvivors were described in a series of 139 blunt trauma patients beginning with the initial measurements after admission to the ED. The nonsurvivors' mean arterial pressure (MAP) was higher than normal and higher than those of the survivors in the first hour after admission consistent with greater adrenomedullary stress responses. The nonsurvivors' MAP fell abruptly about the 3rd or 4th hour after admission. Cardiac index values were initially higher in the survivors indicating lesser blood volume deficits or better physiologic compensations. Nonsurvivors' $SapO_2$ were significantly lower than the survivors' values. The nonsurvivors' transcutaneous oxygen tensions indexed to the FiO_2, $PtcO_2/FiO_2$, were markedly lower than the survivors' values and lower than normal throughout the observation period.

Table 4: Mean net cumulative deficits or excesses of monitored values of survivors and nonsurvivors throughout the period of observation

Variable	SURVIVORS		NONSURVIVORS		P-Value
	Mean	SEM	Mean	SEM	
CI, L/m²	+81	52	-232	138	0.007
MAP, mmHg.h	-10	13	-57	24	0.078
$SapO_2$, %.h	-1	0.3	-8	2.6	0.006
$PtcO_2/FiO_2$, torr.h	+313	87	-793	175	0.001

Table 5: Outcome of various groups treated to normal and supranormal goals of PA (1). (W. Shoemaker et al. Prospective trial of supranormal values of survivors as therapeutic goals in high-risk surgical patients).

Groups	Nonrandomized	CVP-Control	PA-control	PA-protocol
No.patients	(N=45)	(N=30)	(N=30)	(N=28)
Years	56.9 ± 2.5	55.2 ± 3.0	53.4 ± 2.5	56.4 ± 3.1
Males/females (%)	45/55	64/36	39/61	75/25
Hospital days	21.9 ± 1.7	22.2 ± 2.8	25.2 ± 3.4	19.3 ± 2.4
ICU days	14.0 ± 1.7	11.5 ± 1.7	15.9 ± 3.1	10.2 ± 1.6*
Ventilator days	6.5 ± 1.3	4.6 ± 1.4	9.4 ± 3.4	2.3 ± 0.5*
Intraop.death	0	0	1	0
Postop.deaths No.(%)	17 (23%)	7 (23%)	10 (33%)	1 (4%) +

Net cumulative amount of deficit (or excess) of monitored variables and outcome prediction

The circulatory measurements were used to quantify the amount of deficits in cardiac, pulmonary, and tissue perfusion functions in a series of 151 high risk emergency patients on admission to the ED; the net cumulative deficit or excess of each monitored variable of each patient was calculated as the area between the continuously monitored values and the normal or optimal values. Table 5 lists differences in the net cumulative deficits or excesses in survivors and nonsurvivors for cardiac index, MAP, pulse oximetry, and transcutaneous O_2 during the period of monitoring. These differences evaluated by discriminant analysis to quantify the capacity of each variable to affect outcome; 95 % of the survivors and 62% of the nonsurvivors were correctly classified in the early post-admission resuscitation period.

Importance of time relationships for improved outcome

Invasive PA catheters have been used for definitive monitoring of critically ill ICU patients, but recent studies showed no advantage of the PA catheter in cardiac and other medical conditions nor in postoperative patients admitted to the ICU after organ failures had developed (17-22). Two recent consensus conferences found insufficient evidence to determine whether PA catheter-guided therapy significantly alters outcome; they did not consider time factors, but mixed early and late studies together, and arrived at ambiguous conclusions (22,23). However, an incisive meta-analysis by Boyd and Bennett (24) found significant outcome improvement in seven prospectively randomized studies when PA catheter-directed therapy was given early or prophylactically, but they found no outcome improvements in seven other randomized studies of patients who entered the ICU after organ failure or sepsis had already occurred. Monitoring and therapy is often ineffective in the late stages of shock after organ failure has occurred because no amount of oxygen will restore irreversible oxygen debts, failed organs, or dead cells. Optimal therapeutic goals must be achieved in the first 8 to 12 hours after surgery or trauma to be effective (1,2,15,24,35-38,42,43).

Summary

Early or prophylactic therapy is a useful approach to improving outcome in acute illness, trauma, and high risk surgery. Noninvasive monitoring provides a way to recognize circulatory deficiencies earlier, to evaluate the evolving temporal patterns in acute illnesses, and to quantify deficits of monitored variables representing cardiac, pulmonary, and tissue perfusion functions, and to titrate therapy to predetermined physiolgic end points (25,26,34-38,42,43). Our present approach quantitatively compares continuous displays of monitored data with normal values, calculates their net cumulative deficits or excesses, and uses of discriminant analysis to predict outcome.

Noninvasive monitoring systems were found to be feasible early in acute emergency conditions in patients with potential or suspected circulatory alterations and significant risk of death or organ failures. Noninvasive systems are able to identify the time course of hemodynamic patterns and to provide the means to calculate quantitatively the accumulated amount of deficit or excess of each monitored variable. For example, the cardiac index of severely traumatized patients who lived pumped an average of 81 liters/m^2 more than would have been provided by the optimal value of 4.0 L/min/m^2; this was equivalent to 140 liters of cardiac output per patient. Those who died pumped an average of 232 liters/m^2 or 402 liters per patient less than optimal, a difference of 542 liters.

Noninvasive monitoring provides a platform to develop a coherent seamless therapeutic plan based on physiologic criteria as the patient proceeds from the ED to the OR, and to the ICU. Linear discriminant function predicted outcome correctly in 95% of the survivors and 62% of the nonsurvivors in the early period after admission. This is probably as much as should be expected for nonsurvivors since many patients develop late lethal complications rather such as resistant nosicomial infections, drug reactions, wound disruptions, postoperative bleeding, and anastomotic breakdowns late in their hospital course that could not be suspected in the early resuscitation period.

The conceptual basis of this approach was that if these methods could be implemented in emergency trauma patients entering the ED in a large inner city county hospital as a worst case scenario, they could be applied more widely in the ED, OR, and throughout the hospital. The major assumption in this approach is that early circulatory deficiencies that ultimately lead to shock, organ failure, and death can be identified shortly after onset of trauma, hemorrhage, or surgery. Semi-quantitative estimates of the amount of deficits may be calculated from the area between normal and the continuously monitored data; discriminant analysis provides the mathematical basis for prediction of outcome. When identified early, these deficiencies may be more effectively treated than after occurrence of multiple organ failures from irreversible oxygen debt. In essence, noninvasive monitoring begun in the ED was easier, cheaper, faster, safer, and equally sensitive to the invasive PA catheter (25,26, 42,43).

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