Bone marrow transplantation and intensive care unit admission: What really matters?*

The fundamental principle of critical care is the application of technology and other therapies to support life-threatening organ failure in order for a) time or b) treatment (medical and/or surgical) to reverse the underlying disease process. The decision to provide and/or continue support depends on the extent, severity, and temporal evolution of multiorgan failure and remains the most reliable predictor of mortality regardless of primary disease (1).

It is interesting, therefore, to observe and participate in the discussions concerning bone marrow transplants (BMT) and intensive care unit (ICU) admission. Justifiable questions persist about the appropriate use of ICU resources for a patient population with the stigma of historically poor outcomes. The accompanying report by Dr. Jacobe and colleagues (2) in this issue of Critical Care Medicine reviews the outcomes for BMT patients after pediatric ICU admission in an effort to identify admission characteristics that may accurately predict poor outcomes. None were identified. While all pediatric ICU BMT outcome studies are limited by retrospective data and the majority by small sample sizes, the report by Dr. Jacobe and colleagues reaffirms evolving perspectives on the care of critically ill BMT patients: reported outcomes, while modest, are improving.

Notable conclusions include the improved survival of BMT patients to ICU discharge, including those requiring mechanical ventilation (41.4%), with a lower medium term outcome after ICU discharge as measured by a 12.9% 6-month survival for mechanically ventilated patients. In comparison with other reports, there is a suggestion that a lower acuity of illness in the first 24 hrs of ICU admission (as measured by Pediatric Risk of Mortality Scores) may explain improved outcomes.

The most instructive findings are that BMT preadmission characteristics have no apparent influence on outcome, including underlying diagnosis, age, time of admission post-BMT, type of BMT, conditioning regimen, and graft-versus-host disease. This is consistent with other reports in pediatric BMT patients (3, 4) and pediatric (5) and adult (6) oncology patients. Not unexpectedly, ICU measurements of disease severity, i.e., extent of multiorgan failure, are the dominant influences on short-term outcome. In comparison, the medium and long-term outcomes are primarily influenced by underlying BMT or oncology-related conditions.

These observations confirm that the outcome of BMT patients in the ICU is related to the multiorgan failure rather than the fact that they are BMT patients per se. This is not a minor distinction as the ICU decisions for initiating, continuing, or withdrawing support should primarily depend on each patient’s organ failure characteristics rather than any BMT-related issue. The relevance of being a BMT patient is that they may be more likely to progress to severe organ failure and less likely to have an underlying process of organ failure that is reversible. Evolving therapies by our transplant colleagues may improve upon this in the future. Regardless, decisions around ICU care should be made on organ failure criteria alone.

In addition to being medically complex, burdensome, and resource consuming, BMT patients have the additional stigma of dismal outcomes associated with ICU admission. As a consequence, there remains the risk of a demoralized attitude of ICU care toward this patient group as the label of “BMT in the ICU” consequently triggers the conclusion of “bad outcome.” This report by Jacobe et al. reemphasizes that preadmission characteristics do not influence outcome and multiorgan failure is the most important predictor of survival. Should BMT patients be admitted to ICU? This has become an unproductive question. More important questions are whether early ICU intervention (lower acuity threshold for admission) impacts mortality and how much organ failure is incompatible with survival.

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Difficult to wean chronic obstructive pulmonary disease patients: Avoid heat and moisture exchangers?*

Despite the increasing use of noninvasive ventilation in the management of patients with acute respiratory failure (1), endotracheal intubation and mechanical ventilation are often needed in patients with chronic obstructive pulmonary disease (COPD) and acute respiratory failure. Weaning difficulties are frequent in COPD patients (2). In a recent randomized clinical trial comparing weaning strategies, multivariate analysis performed to search for factors explaining weaning duration showed the etiology of the disease to be significant, with COPD patients being the most difficult to separate from the ventilator (3). The process of discontinuing mechanical ventilation constitutes a major clinical challenge, and it constitutes a large portion of the workload in an intensive care unit.

Over the past decade, we have gained a better understanding of the pathophysiology associated with difficult to wean patients (4, 5). The major mechanism underlying the need for prolonged mechanical ventilation in patients with COPD has been reported to be the association between abnormal lung mechanics, in particular intrinsic positive end-expiratory pressure, lung resistances, and reduced pressure-generating capacity of the inspiratory muscles because of pulmonary hyperinflation. Possible mechanisms of the reported abnormalities of the respiratory muscles include diaphragmatic weakness, atrophy, hyperinflation, and fatigue. In these patients, the magnitude of the inspiratory muscle contraction must exceed the level of intrinsic positive end-expiratory pressure (i.e., the end-expiratory elastic recoil pressure) to create a subatmospheric pressure in the central airway either to generate inspiratory flow and volume or to trigger the ventilator (6, 7). Patients who fail a trial of weaning from mechanical ventilation experience a marked increase in respiratory load and work of breathing (4). With carefully selected settings, mechanical ventilation decreases respiratory work from high levels to the normal range. Lowering patient effort into the normal range must be balanced against the greater likelihood of nontriggerring and complications at high levels of assistance (5). Indeed, insufficient support causes the patient’s inspiratory muscles to continue to contract under load, preventing adequate rest needed to resume spontaneous ventilation (7, 8). On the other hand, high levels of ventilatory support may be associated with ineffective efforts and patient-ventilator dysynchrony. A variety of different mechanisms may give rise to patient-ventilator dysynchrony, such as high levels of intrinsic positive end-expiratory pressure, excessive tidal volume, and reduced expiratory time due to disproportionate inspiratory flow duration with respect to inspiratory effort during assisted modes of mechanical ventilation such as pressure support (PS) ventilation (7, 9). Thus, we have learned to pay particular attention to the close monitoring and to the settings of the assisted modes of mechanical ventilation in difficult to wean patients.

In addition, as demonstrated by Dr. Girault and colleagues (10) in a prospective randomized controlled physiologic study, published in the current issue of Critical Care Medicine, the choice of the type of airways humidification device could also be important in difficult to wean COPD patients. Humidification of inspiratory gases is widely accepted to be necessary in patients studied. Although in many patients the amount of added deadspace with an HME could be trivial and

*See also p. 1306.

Key Words: chronic obstructive pulmonary disease, heat and moisture exchanger; heated humidifier; pressure support ventilation; respiratory failure; weaning

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unlikely to adversely affect weaning trial outcome, this may not be the case in patients with very limited ventilatory reserve such as patients with COPD. Nevertheless, Pelosi et al. (15) evaluated the effect of two commonly used HMEs on respiratory function and gas exchange in 14 non-COPD patients with acute respiratory failure during PS ventilation. They found that the tested HME significantly increased minute ventilation, ventilatory drive, and work of breathing during PS ventilation.

Adverse effects observed with HME use were partially counterbalanced by increasing the PS level by $\geq 8 \text{ cm H}_2\text{O}$ (10) or $5–10 \text{ cm H}_2\text{O}$ (15). In our opinion, this is not a desirable solution for weaning practice. An individual “optimal” PS level is difficult to establish in COPD patients (16). In addition, PS of $6–8 \text{ cm H}_2\text{O}$ is widely used to compensate for the resistance imposed by the endotracheal tube and ventilator circuit. The minimal level of assistance, however, has never been well defined (5). Thus, an imprecise amount of PS may undercompensate, or, on the contrary, may overcompensate and give misleading information about the likelihood that a patient can tolerate extubation.

The discontinuation of mechanical ventilation needs to be carefully timed (5, 17). We have learned how to wean patients more efficiently and to pay particular attention to the settings of the assisted modes of mechanical ventilation and to the monitoring of difficult to wean patients. In addition, HMEs should not be recommended in difficult or potentially difficult to wean COPD patients. HMEs are widely used, and their impact on the weaning process should be evaluated in large populations of patients, including patients without COPD.

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Dollars and sense in the intensive care unit: The costs of ventilator-associated pneumonia*

Ventilator-associated pneumonia (VAP) remains a significant burden in the care of critically ill patients. The incidence of VAP is 8–28% among patients requiring mechanical ventilation (1). Although the rate of VAP varies based on both the patient population and type of intensive care unit (ICU) studied, all critical care physicians regularly face the diagnostic and therapeutic challenges posed by this disease process. More importantly, VAP is associated with significant mortality. Crude mortality rates for those who develop VAP may be as high as 70% (1). The attributable mortality rate of VAP is much less. Nonetheless, most experts concur that 20–30% of subjects diagnosed with VAP die as a result of this infection (1). Putting that into perspective, there are few diseases we treat in the ICU where between one in five and one in three patients die despite our best efforts and technology.

Much attention has focused recently on ways to improve outcomes for patients suspected of having or eventually diagnosed with VAP. Clearly, appropriate antibiotic selection is key (2). To guarantee appropriate initial antibiotic coverage it is imperative that one know the likely pathogens in his or her specific ICU and the resistance patterns of those organisms. The role for bronchoscopy for the diagnosis of VAP is more controversial. Of four prospective trials exploring the impact of invasive diagnostic strategies on survival from VAP, only one showed a decrease in mortality (3–6). Some have criticized the trial by Chastre et al. (3) because patients undergoing bronchoscopy were more likely to be treated with appropriate antibiotics than those randomized to a noninvasive approach. In that trial, however, the overall incidence of inappropriate antibiotic therapy was extremely low. Each of the other three studies in this area failed to show a difference in mortality rate relative to the means by which VAP was diagnosed (4–6). These other projects, however, were small and may have been underpowered to answer this important question.

The diagnosis of VAP is not the main issue. Rather, to make headway against VAP we need to focus our efforts on prevention. Given the morbidity and mortality of VAP, the creation and adoption of preventive strategies will improve outcomes in the ICU. A number of options for prevention exist and are supported by varying degrees of evidence. Some, for example, are relatively uncontroversial. Ensuring elevation of the head of the bed during mechanical ventilation decreases the risk for VAP (7). Similarly, earlier liberation from mechanical ventilation lessens the chances for developing VAP (8). Despite well-conducted studies supporting these concepts, few ICUs have adopted these measures or made them standard protocol. More controversial alternatives include selective digestive decontamination and reliance on endotracheal tubes capable of continuous subglottic suctioning (9, 10). One barrier to the adoption of any approach to prevention in the ICU is the perceived cost of the intervention. For some options, hospitals must allocate additional funds (e.g., endotracheal tubes capable of continuous subglottic suctioning), whereas for others the costs are more intangible. Educating physicians, nurses, and respiratory therapists about the importance of elevating the head of the bed often proves frustrating to those of us who stress these issues. In other words, an investment of time is needed to begin the process of behavior change in our approach to the patient receiving mechanical ventilation.

What might be the return on our investment? In this issue of Critical Care Medicine, Dr. Warren and colleagues (11) provide us with important new data about the cost of VAP. If the potential mortality associated with VAP was not sufficient to motivate us to emphasize prevention, now we have evidence to quantify the financial costs of our inaction. Dr. Warren and colleagues (11) conclude that each case of VAP is associated with a direct cost of nearly $50,000. As with the attributable risk for death, the attributable cost of VAP is less but is not insignificant. Specifically, these authors estimated that costs directly related to VAP totaled approximately $12,000. Their analysis was carefully done. First, unlike prior studies calculating the cost of VAP, these investigators relied on prospective rather than retrospective data. Hence, there is little risk of recall, coding, or other bias that might have confounded the results. Second, they used an appropriate control group for comparisons. Dr. Warren and colleagues (11) limited the control cohort to subjects actually at risk for VAP. Third, their accounting methodology was both rigorous and precise. Fourth, they provided data on the distinction between early-onset and late-onset VAP. The pathogens in early-onset VAP tend to be less virulent than those in late-onset VAP, and early-onset VAP has not conclusively been shown to affect mortality (1). Despite these facts, Dr. Warren and colleagues (11) observed that early-onset VAP was still an expensive event. Total costs for early-onset VAP were more than $10,000 higher than those for patients without VAP.

The results of this study are strikingly similar to those of a recently published assessment of a large, third party-payer database. Rello et al. (12) in a review of outcomes and reimbursement patterns, determined that the development of VAP resulted in nearly $40,000 in excess costs. If there was any doubt about the costs of VAP, these two analyses conclusively show that VAP will continue to be a financial drain on ICUs and on hospitals.

*See also p. 1312.

Key Words: ventilator-associated pneumonia; intensive care unit

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Habit and the hub*

Catheter-related infections should be a major concern to intensivists. The incidence rate among 2201 critically ill adults staying in an intensive care unit for more than 48 hrs has been reported to be 1.8% (39 of 2201 patients) (1). Catheter-related infectious complications include colonization and infection of the catheter site, catheter tip, and blood. Some cases of septic thrombophlebitis, right-sided endocarditis, cellulitis, and death can also be attributable to a catheter (2).

Many measures have been suggested to prevent catheter-related infections. A catheter can become infected either by extraluminal or by endoluminal route. In the former, the catheter-related infection originates from the cutaneous insertion site, and the bacteria migrate along the external surface of the catheter. In the latter, bacteria get access to the endoluminal surface of the catheter through hub contamination. Catheter hubs are frequently manipulated for maintenance purposes, and the bacteria migrate along the external surface of the catheter. Antiseptic—3% iodinated alcohol—was incorporated in the chamber of the new hub model. Monitoring and maintenance were similar. A hub-related sepsis—the primary outcome measure of the trial—was considered to be present if the same organism was recovered from the hub, tip of the catheter, and blood cultures. The rate of catheter-related bloodstream infection (CRBI) originating from the catheter hub was significantly different (Luer-lock vs. new hub, 7% vs. 1.7%, respectively; p < .049; relative risk, 4.1; 95% confidence interval, 0.8–19). Adjustment for APACHE II score, underlying disease, insertion site, and parenteral nutrition did not change significantly the protective effect of the hub. The hub did not cause any adverse event. The conclusion was that the new hub was significantly better than a standard hub to prevent “endoluminal bacterial colonization and catheter-related bloodstream infection from hub contamination in intensive care.”

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sive care unit patients with central venous catheters inserted for ≥6 days.”

There are few weaknesses in this study. For example, it was not blinded, but this was impossible with the maneuver under study. Moreover, it is unclear if cases were consecutive, which would have enhanced the representativeness of the sample of patients collected and improved the generalizability of the results reported. On the other hand, many strengths must be underlined. The clinical trial was randomized and prospective. Only one catheter per patient was studied. Insertion and care of catheter followed standard and appropriate procedures. Screening and monitoring of infections were also systematically done.

This study shows that the new hub, the Segur-Lock, is significantly better than a standard hub to prevent CRBI from hub contamination in intensive care unit patients with central venous catheters inserted for ≥6 days. The efficacy of this new device is clear, but is it cost-effective?

The costs attributable to a CBRI are significant. For example, two studies estimated an excess cost of about 3,000 Euros (about $2,800) per CRBI in critically ill adults (6, 7). In surviving critically ill adults, the average excess length of stay in the intensive care unit attributable to CRBI is 14 to 20 days (1, 7). Singer et al. (8) estimated the cost of one patient day in the intensive care unit to be >$2,000 (1,148 pounds) in 1991. Therefore, the excess cost attributable to a CRBI could be between $3,000 and $40,000 per CRBI if the length of stay in the intensive care unit is indeed prolonged. The latter estimation is supported by Slonim et al. (9) who reported in 2001 that the excess radiology, pharmacy, and laboratory costs attributable to a nosocomial bloodstream infection is $12,211 per CRBI in critically ill children. The new hub can prevent a significant proportion of nosocomial bloodstream infections, which should decrease the clinical costs (morbidity and mortality) and the economic costs attributable to such infections. Besides these economic concerns, another important factor is the fact that the new hub model is user friendly. Care and manipulation of junctions equipped with the Segur-Lock is easy, which should decrease the workload of nurses with respect to proper junctional care. Moreover, the new hub is quite cheap (about $10). Not surprisingly, Dr. León and colleagues (5) estimated that the use of the new hub would have saved 22,580 Euros (about $19,850) in their study. Therefore, we can conclude that the Segur-Lock is cost-effective.

Habit is difficult to change. Our habit was to use the Luer-lock hub; should we change to the new hub model? The Segur-Lock is safe if a patient is not allergic to iodine. Its cost-effectiveness seems to be good. Therefore, the available evidence supports the use of Segur-Lock in critically ill patients who require a central venous catheter for ≥6 days. However, at least another positive clinical trial is required before one can consider that the use of this device is mandatory. Moreover, we need further studies to know if the cost-effectiveness of the new hub is better than the cost-effectiveness of antibiotic-coated or heparin-bonded central venous catheters and if it would be appropriate to use such catheters with the new hub.

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The long and short of pulmonary artery catheter monitoring and catheter-related infections: Is less still best?*

In 1970, Swan and colleagues introduced the pulmonary artery catheter (PAC) for use in patients with cardiovascular disease (1). Since then, it has generated well over 7,866 Medline publications, extensive controversy (2), and concerns about comprehensive guidelines (3) and rigorous outcomes analysis (4, 5). Millions of catheters continue to be used worldwide for hemodynamic monitoring in the critically ill. Intravascular catheter-related bloodstream infections (BSI) account for substantial attributable nosocomial morbidity and mortality (6). It causes 80,000 BSIs and 2,400–20,000 deaths each year in the United States, and the annual cost of caring for patients with central venous catheter (CVC) BSI range from $296 million to $2.3 billion (7).

The recent Centers for Disease Control (CDC) Guidelines for Prevention of Intravascular Catheter-Related Infections (6) extensively address the confounding sources of infection, such as location of insertion site, type of catheter, hand hygiene and aseptic technique, and skin antisepsis. Catheter site dressing regimens, securing devices, impregnation of catheters and cuffs with antimicrobial or antiseptic materials, and systemic antibiotic prophylaxis, ointments, and anticoagulants also are considered (6). Equally important issues include whether catheter sleeve coverage was used and if introducing a transducer were cultured (6). Combined medical-surgical intensive care units in major teaching hospitals have higher associated rates of device-associated BSI compared with all other hospitals with combined medical-surgical units in the National Nosocomial Infection Surveillance System (8, 9).

Numerous risk factors are consistently associated with increased risk for BSI: insertion with less than maximal sterile barriers, placement in the internal jugular or femoral rather than subclavian vein, placement in old site by guidewire exchange, heavy cutaneous colonization of insertion site, contamination of the catheter hub, and duration of CVC placement >7 days (6, 7, 10). Training and experience of the inserter also are associated with risk of BSI (10). Microbial biofilms develop when microorganisms adhere to indwelling catheters (11), and newer technologies may help to decrease the incidence of BSI. Heparin-bonded PAC reduces the risk of catheter thrombosis and microbial adherence to the catheter (6). The relative risk of infection with heparin-bonded PAC is 2.6 per 1,000 catheter days, comparable to that of CVC (2.3 per 1,000 catheter days), whereas nonheparin-bonded PACs have more than twice the risk of catheter-related BSI (5.5 per 1,000 catheter days) (6).

Few studies have systematically examined the mechanisms, risk factors, and incidence of infections associated with PACs, which have similar rates of BSI as CVCs (6, 7, 10, 12, 13). PAC-related colonization varies from 2 to 20% and depends on duration of catheter placement and criteria for the definition of colonization (6, 14, 15). The CDC and the Joint Commission on Accreditation of Healthcare Organizations recommend that the rate of catheter-associated BSIs be expressed as the rate of catheter-associated BSIs per 1,000 CVC days and adjust risk according to the days of catheter use (6). PAC monitoring for >72–96 hrs substantially increases catheter-related BSI (14). Furthermore, the risk increases incrementally with an increased incidence of catheter-related BSI after 5 days (16) and increases substantially if the catheter remains in situ for >7 days (2% before 7 days vs. 16% after 7 days) (15). In a study by Kac et al. (17), PAC-associated colonization and bacteremia occurred in 11.6% and 0.6%, respectively, and the incidence was 17.07 and 0.93 episodes per 1,000 catheterization days, respectively. Greater than 4 days of monitoring was the single variable associated with significant risk of PAC colonization (17).

Does scheduled replacement of PAC and monitoring system at 4 days or 7 days reduce the incidence of infection or colonization? Currently, there are no data to support scheduled replacement of PAC without evidence of colonization or systemic infection (6). The CDC does not recommend routine catheter replacement to prevent catheter-related infection (6). In this issue of Critical Care Medicine, Dr. Chen and colleagues (18) report no significant difference between scheduled PAC replacement at 4 days and 7 days in the rate of associated colonization or infection in a prospective, randomized clinical trial in a subset of 258 patients in a surgical and medical intensive care unit of a teaching hospital. Dr. Chen and colleagues (18) found similar rates of PAC colonization, with 5% and 14% in the 4-day and 7-day groups, respectively, and incidence of bacteremia 1.1 episodes per 1,000 catheter days in the 7-day group and none in the 4-day group.

The majority of patients in this study had acute respiratory failure (18). The authors excluded 29% of their original study patients due to clinical improvement, transfer to the ward, or death before completion of study (18). No significant demographic differences between the two groups were detected, as well as no differences in disease severity measured according to Acute Physiology and Chronic Health Evaluation II scores (18). The non-PAC-associated BSIs noted in this study (18) were 7.1% in the 4-day and 10.5% in the 7-day groups, respectively, consistent with National Nosocomial Infections Surveillance System observations of higher infections in combined medical-surgical intensive care units in major teaching institutions (8).
This elegantly designed trial addressed the necessary procedures for insertion, care of catheters, and monitoring devices for prevention of catheter-related infections and BSI, and the authors obtained most of the essential cultures. This trial suggests that less may not necessarily be the best. As a matter of fact, frequent scheduled PAC changes may pose mechanical complications through new-site replacement, and guidewire exchange is associated with a trend toward increased frequency of colonization, catheter exit-site infection, and catheter-related BSI (13). In addition, Dr. Chen and colleagues (18) highlight potential cost-effectiveness with less frequent catheter changes.

However, there are several limitations intrinsic to their study protocol (18). First, the type of randomization allocation is not reported and may be a source of bias (19). Second, the sample size is small; thus, there may be insufficient statistical power to detect differences between the two groups, potential for Type II errors, and incorrect conclusions (19). Third, the introducers were not cultured, and molecular subtyping was not performed on the organisms cultured; therefore, an underestimation of the true number of PAC infections is a possibility. Last, although the total days each catheter used appears significant (p < .001), the difference between the two groups (4.8 days in the 4-day group and 6.5 days in the 7-day group) may be in fact smaller and is only 1.7 days, instead of the expected 3 days difference. This potentially may account for no observed significant differences in the two groups.

The bottom line is that the PAC should be removed when no longer essential (6), and this study (18) further underscores the CDC guidelines that the PAC need not be changed more frequently than every 7 days, unless clinical indicators suggest otherwise (6). This study by Dr. Chen and colleagues (18) should provide further impetus for additional outcome studies evaluating the differences between 4 days, 5 days, and 7 days replacement in a larger group and cost-benefit analysis. Such studies are needed before a conclusive statement can be made whether less is truly best, in the long and short end of pulmonary artery catheter monitoring.

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Since the initial case series of the use of arginine vasopressin in the treatment of vasodilatory shock not responsive to catecholamines, its use has increased exponentially for this indication (1–8). Recent work on the function of vascular smooth muscle and the pathophysiology of vasodilatory shock has clarified our understanding of the benefit, or lack thereof, of vasopressor therapy including vasopressin (9). Vasopressin has been studied for >70 yrs and used in the past at much higher doses (0.4–0.8 units/min) for the treatment of gastrointestinal hemorrhage. However, its untoward effects of decreasing cardiac output and vasoconstriction restricting gut and coronary flow, along with skin necrosis/gangrene, led to the development and use of other agents such as somatostatin (10–12). With the reintroduction of vasopressin at low doses for vasodilatory catecholamine-resistant hypotension (0.03–0.04 units/min), at least the cardiac and renal complications seem to be minimized (4–8). Because vasopressin, even at low doses, can decrease cardiac output, it should not be used for patients with cardiogenic or hypovolemic shock.

Only two reports suggest that low-dose vasopressin may also result in skin necrosis (3, 13). In this issue of Critical Care Medicine, Dr. Dünser and colleagues (14), using a retrospective, cohort study design, report the incidence of ischemic skin lesions in patients with catecholamine-resistant vasodilatory shock requiring vasopressin. Although the pathophysiology of ischemic skin lesions remains complex, it clearly is associated with circulatory shock and disseminated intravascular coagulation, as demonstrated in the present report (14).

Whereas we all recognize that ischemic skin lesions can occur in patients receiving high doses of vasopressor catecholamines, it may also occur in individuals receiving low-dose catecholamine infusions (15–19). In the present report, Dr. Dünser and colleagues (14) demonstrate that: 1) the prevalence of ischemic skin lesions with vasopressin may be as high as 30%; they also report, for the first time, the development of ischemic lesions of the tongue; 2) none of the 19 patients with ischemic skin lesions required amputation or plastic surgical interventions during their intensive care unit stay; and 3) the presence of septic shock or preexistent peripheral arterial occlusive disease was independently associated with the development of ischemic skin lesions.

Why was the prevalence of ischemic lesions so elevated in the present report? Other series either fail to mention ischemic skin lesions as a significant complication of vasopressin administration or its prevalence is <10% (4–8). Most of the observation periods in these other series were short (i.e., <24 hrs). The median length of vasopressin infusion in this present study was 53 hrs, and ischemia may result with prolonged infusion (14). Although the present report is retrospective, nonetheless, the institution’s strict adherence to documentation of skin changes on a daily basis would increase the probability of reporting this complication. Clearly, other institutions may not be as compulsive and may have under reported the event (4–8). Frankly, the nurses and physicians caring for these patients deserve a major “kudo” for this meticulous attention alone! In addition, the only catecholamine infused in the present study was norepinephrine. In other series, individuals received other catecholamines in addition to norepinephrine (4–8). It is possible that the use of catecholamines or other agents with vasodilatory properties like milrinone, low-dose dopamine, and dobutamine could have decreased the skin complication rate (4). As vasopressin potentiates the effects of norepinephrine (9) and, in patients with ischemic skin lesions, norepinephrine infusion rate could not be decreased to the same extent as those without ischemia (14), and norepinephrine clearly can lead to skin ischemia (16), then the pathophysiology of this complication is understandable and expected. Whereas this study suggests that although skin ischemia occurs, little will need to be done about it, it is not completely clear that this is so. Answers to the following would be beneficial in elucidating the magnitude of the problem. What changes in therapy were made when ischemic skin was detected? What was the ultimate outcome of the skin ischemia in these patients outside the intensive care unit? Did skin loss occur? If so, how extensive? What medical therapy was required?

Thus, the addition of low dose vasopressin to our armamentarium for the treatment of vasodilatory catecholamine-resistant shock at this point would seem reasonable based on small clinical trials. However, it would be prudent to administer this drug through a central catheter and observe for skin ischemia, in particular in those patients with evidence of preexisting peripheral arterial disease and in patients with sepsis. We should all strive for the standard of care of meticulous observation and documentation of skin changes reported in this study, not neglecting to inspect the oral mucosa! It is important to recall that low-dose infusions should be used. Complications of cardiac and renal function and gut ischemia may result if we exceed these doses. Evidence exists that if no effect is seen at a vasopressin infusion rate of 0.04 units/min, increasing the dose further only increases adverse effects and does not increase the effectiveness (5).

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A bioavailability study in the proposed patient population—with much more needed now*

Although the necessity for thromboembolic prophylaxis in critically ill patients (1, 2) is generally acknowledged, the title of the study by Dr. Priglinger and colleagues (3) in this issue of *Critical Care Medicine* is somewhat misleading. The authors did not study prophylaxis at all, but an inferral of such via observation of anti-Xa levels, further documenting the ability of anti-Xa levels after 40 mg of enoxaparin (a low-molecular-weight heparin) delivered subcutaneously, as recently reported (4).

The investigators determined serial anti-Xa levels in critically ill patients, without renal dysfunction (a major finding in critically ill patients and something to be addressed in further studies), after subcutaneous administration and compared these values with those obtained in noncritically ill patients in a medical ward. They did not indicate (see their Table 2) whether either patients had diagnoses related to oncology, trauma, postorthopedic surgery, postmyocardial infarction, multiple organ failure, acute coronary syndrome, or unstable angina—conditions known to be affected positively by enoxaparin (5–9) nor did they report or comment on the prevalence of common and noteworthy adverse reactions (e.g., heparin-induced thrombocytopenia in either cohort).

It is generally agreed that the higher area under the curve of enoxaparin in terms of anti-Xa activity is significant only as another variable, demonstrating that different low-molecular-weight heparins have different pharmacokinetic/dynamic properties—and hence, cannot be used interchangeably (10, 11).

Significant differences in anti-Xa maximal drug concentration and area under the curve (over 24 hrs) were found on days 1–5 in the intensive care patients vs. medical ward patients. They thus performed a basic test of “comparative” bioavailability of enoxaparin in the two hospitalized patient groups using the subcutaneous route of administration. By so doing, they found that enoxaparin was more bioavailable when administered subcutaneously in noncritically ill patients than in critically ill patients, disproved their null hypothesis of anti-Xa equipotence in both groups after subcutaneous administration, and speculate that enoxaparin may not be as preventive against thromboembolic episodes in critically ill patients when administered at the 40 mg subcutaneous daily dose.

Whether subcutaneous enoxaparin administered to critically ill patients is prophylactic against thromboembolic events is the major question that remains to be answered (as previously noted, Dr. Priglinger and colleagues (3) did not determine prophylaxis against thromboembolic events directly, but followed anti-Xa levels as a surrogate markers of enoxaparin activity, “presumably” as a prophylactic agent against thromboembolic epi-

*See also p. 1405.

Key Words: enoxaparine; low-molecular-weight heparins; anti-Xa; thromboembolic prophylaxis; intensive care; surrogate markers; subcutaneous bioavailability

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Intensive care in a field hospital in an urban disaster area: Are we ready?*

The experience presented by Dr. Halpern and colleagues (1) from the Israeli Defense Forces Medical Corps in this issue of Critical Care Medicine is important, because it confirms the preliminary experience of U.S. and European disaster teams with intensive care sections responding to foreign disasters. The original view of reanimatologist experts, such as Dr. Safar, conceptualized rapid response to massive casualties and disasters many years ago, but suggested that the ability to deploy an intensive care unit in the field near the disaster site may not be associated with improved outcomes. Over the years, as critical care medicine was evolving into a new specialty, new experiences maturation to university departmental status, intensivists have become more impulsive and focused on the defined high technology environment in the intensive care unit box. The first expansion of critical care outside the hospital walls, beyond the advance of the critical care ground and air transport, occurred in the 1980s in a scenario strikingly similar to that described in the report by Dr. Halpern and colleagues. A civilian American critical care team was deployed to an earthquake site in Caucuses; very similar severity and impact area, and comparable healthcare impact were experienced. The ubiquitous Israeli Defense Forces team was present at that time also (2).

The first issue to consider is a comparison of military medicine vs. the civilian medicine component. The military

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*s See also p. 1410.

Key Words: intensive care units; disaster medicine; field hospital; critical care; military medicine

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traditionally is able to provide well-defined core components of international disaster response: transportation, including dedicated cargo planes, communication including satellite teleconferencing capabilities, and security for the staff, and resources in an area where societal infrastructure may be under stress. The Israeli Defense Forces team, despite relative geographic proximity and the immediate availability of a military organization, took 3 days to function as an intensive care unit within a military field hospital context. This response is comparable to other experiences (2), and much shorter than that documented by the U.S. National Disaster Medical System Response Teams, which have taken over a week to deploy overseas (3). The time factor is crucial in disaster response and beyond the “golden hour,” the majority of life saving is the function of a motivated and educated public. The second issue to consider in comparing military and civilian response with critical care in a disaster setting is that of triage. There is a major difference in the military and civilian approach to critical care triage, and although one of the first quality articles evaluating intensivist-driven civilian triage was written by Israeli intensivists (4), this is a generic factor to be considered. Finally, the issue of environmental protection, which theoretically should be highly developed in a military organization frequently exposed to the elements, is obviously not perfect as pointed out by the authors. Overall, the argument regarding whether international disaster response should be delegated to national or international military medicine is still not settled (5, 6).

The next issue one needs to consider based on this accumulating experience is that of technology. One of the early advances in the expert telemedical systems has been developed and tested in a disaster setting (7). The intensivists have a tendency to deploy comprehensive resources based on an anticipated volume of syndromes. Disaster medicine is known for producing a high volume of uncommon syndromes in a short period of time, which may require high technology support: toxic inhalations, burns, and crush injuries with renal failure. The Israeli Defense Forces team has accurately pointed out the variable use of technology ranging from ventilators to dialysis, but in all fairness, one must recognize that the Turkish earthquake incident has added significantly to the literature on support of syndromes, to support effective response developments in other events, such as the Kobe earthquake in Japan (8).

In addition to comments regarding infrastructure and technology, specialized skills have also been appropriately pointed out by the authors, and clearly have been documented by other investigators. This in particular concerns the need for a rapidly deployed evaluation team with language skills and political cover, critical care pharmacists experienced in improvising drug dispensing and acquainted with the local alternatives for follow-up care, and the always crucial biomedical engineers who ensure that electricity, water, and oxygen supplies do not become interrupted, thus jeopardizing the patients. In addition, it is important, and is correctly pointed out by the authors, that a highly skilled specialist team may be rapidly required to provide mundane low acuity urban emergency medicine or clinic care. Although discouraging to some, this has a quantitative impact not to be ignored.

The critical care medicine training curriculum delivery is traditionally weak in unconventional skills such as delivery of critical care in emergency medicine and disaster settings. Although it has been clearly shown that critical care can be delivered in both environments (9, 10), much needs to be done. Disaster medicine has grown up, and has scientific forums that include a peer-reviewed journal and an international society. One can only hope that the more recent experiences with urban man-made disasters such as the kamikaze attacks on the World Trade Center and Pentagon will not spread to the extent in which disaster response will become a daily event in the life of the academic intensivist. Integrating experiences such as those presented by the authors in an academic detailing format will improve intensive care. An intensivist should be referred to evolution of this science of disaster medicine (1, 3, 11).

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Is there an “ACE” in the hole for postcoronary artery bypass graft myocardial dysfunction?*

After an initial enthusiastic use of the intravenous angiotensin-converting enzyme (ACE) inhibitor, we were disappointed to find our anecdotal experience with intravenous enalaprilat in hypertensive urgencies to be less than spectacular, and we had largely dismissed this drug as a “pharmaceutical relic.”

However, in this issue of Critical Care Medicine, Dr. Wagner and colleagues (1) present the results of a placebo-controlled, randomized, double-blind protocol of intravenous enalaprilat in postoperative coronary artery bypass graft (CABG) patients with preexisting left ventricular dysfunction (<.35%). The study was simple and elegant in design, with suitable end points and outcomes. Beginning on postoperative day 2, a loading dose of enalaprilat was followed by a 72-hr continuous infusion at 5 mg/24 hrs. Cardiovascular hemodynamics, oxygen delivery variables, and neurohumoral and metabolic variables were trended over this study period. A continuous mixed venous/cardiac output pulmonary artery catheter with periodic pulmonary artery occlusion pressures was used in all patients. Pulmonary artery occlusion pressures were serially recorded.

In the 40 patients randomized, the results were convincing and impressive, with improvement in most of the main outcome variables seen in the study group. The treatment group had early and sustained improvement in hemodynamics and in oxygen delivery. Moreover, it was reassuring to see no detrimental metabolic disturbances or renal dysfunction occur acutely with enalaprilat use in this high-risk setting.

The placebo and treatment groups seemed well matched, with both having moderately severe reductions in left ventricular dysfunction preoperatively with mean left ventricular ejection fractions ranging between 24% and 28%. The pretherapy requirements for intra-aortic balloon pump dobutamine and epinephrine did seem to be greater in the control group but did not meet significance. The vast majority of patients in both groups were on ACE inhibitors chronically and seemed to be compensated at surgery.

Dr. Wagner and colleagues’ (1) findings, and the findings of Ryckwaert et al. (2) in a smaller group of CABG patients treated with intravenous bolus ACE-I, seem to support the efficacy and safety of ACE inhibition before and during bypass surgery. In both studies, cardiodynamics and renal function seemed to improve with little untoward hemodynamic effect of drug delivery. Other investigators have used the postoperative CABG milieu to assess the effect of ACE-I on biochemical and inflammatory response modification (3–5). ACE-I appears to have potent anti-inflammatory activities as well as its cardiodynamic effects. As with most clinical studies, many questions remain to be answered. What is the optimal timing for initiation of ACE-I in the perioperative CABG patient with preexisting left ventricular dysfunction? Is there a dose response effect with enalaprilat? Do the beneficial effects of ACE-I result from the hemodynamic effects, the anti-inflammatory effects, or a combination of the two?

It is truly heartening to see the results of such studies, because a parenteral ACE-I may eventually prove to be the “ace” we need in many urgent situations. We now feel more secure in its use in patients with compromised cardiovascular function and will resurrect our relic again.

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Desperate appliance*

... diseases desperate grown
By desperate appliance are relieved,
Or not at all.
—William Shakespeare, Hamlet, Act IV, scene III

Exsanguinating trauma that has progressed to cardiac arrest is a desperate disease. Few if any patients are successfully resuscitated and survive. Rhee et al. (1) reviewed 24 published studies that included 4,620 cases of emergency department thoracotomy for blunt and penetrating trauma over 25 yrs and reported that, of patients who arrived at the hospital without signs of life, only 2.6% survived; of those without signs of life in the field, only 1.2% survived. These grim survival figures compare with survivals of 26% and 39%, respectively, in patients who were comatose after nontraumatic, out of hospital cardiac arrest (2) or primary ventricular fibrillation (3). In exsanguinating traumatic hemorrhage, unlike cardiac arrest due to intrinsic cardiac disease, the time required not only to achieve systemic resuscitation but also to control hemorrhage and to repair lacerated blood vessels exceeds the ischemic tolerance of the brain. A “desperate appliance” that could improve these dismal statistics in trauma patients would be valuable indeed. At a minimum, an effective intervention would minimize the duration of warm tissue ischemia and preserve vulnerable tissue until completion of definitive surgical treatment and restoration of adequate perfusion pressure. An ideal treatment also would reduce ischemic neuronal injury that had occurred before initiation of preservation.

Peter Safar and colleagues at the Safar Center for Resuscitation Research have been developing for nearly 20 yrs an experimental approach that ultimately could be used to resuscitate patients with exsanguinating cardiac arrest. In this issue of Critical Care Medicine, Dr. Behringer and colleagues (4), from the Safar center, report their most impressive evidence yet of the preclinical feasibility of such an approach. However, before I discuss those data, a brief review of their previous progress is in order. In general, that progress has consisted of developing techniques that can be begun after the onset of cardiac arrest and that have progressively increased the interval of time available for resuscitation and control of hemorrhage. Initially, in dogs that had undergone 30 mins of hemorrhagic shock to 40 mm Hg, cardiopulmonary bypass was used to induce hypothermia to 15°C before inducing circulatory arrest of varying duration; after 60, 90, or 120 mins of hypothermic cardiac arrest, dogs were rewarmed and resuscitated by using cardiopulmonary bypass (5). After 60 and 90 mins of cardiac arrest, dogs showed good neurologic recovery and mild histopathologic damage, whereas those receiving a 60- or 90-min cardiac arrest were killed and their brains were examined 72 hrs after the insult. A 60- or 90-min window of opportunity provides a more reasonable amount of time in which to complete transport, achieve he-

*See also p. 1523.

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1592 Crit Care Med 2003 Vol. 31, No. 5

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mostasis, and restore perfusion. Perhaps that window could be extended further by altering the constituents of the hypothermic aortic flush (9) or by adding drugs to limit ischemic neuronal damage (10).

Can this exciting intervention be extended to the clinical management of trauma patients—civilian or military—with exsanguinating cardiac arrest? Perhaps, but technical and logistic hurdles remain. The most pressing technical hurdle is simply the tiny window of normothermic cardiac arrest that is compatible with successful resuscitation. In these impressive studies, although hypothermia was induced after cardiac arrest, the aortic catheter was placed before hemorrhage. Treatment was initiated after 2 mins of normothermic cardiac arrest. To begin treatment within a similar time frame in exsanguinated trauma patients would require that cardiac arrest occur under direct observation of personnel with appropriate tools and training to place an aortic catheter and begin infusion. Data published in abstract form suggest that the interval between arrest and infusion might be extended to 5 mins (11). However, 5 mins still represents a brief interval in which to accomplish catheterization and initiate treatment. A logistic hurdle is that the volume of 2°C aortic flush necessary to achieve hypothermia at aortic flush temperatures of 15°C or 10°C ranged from 258 to 736 mL/kg. Extrapolating to a 70-kg human requires a volume of 15–50 L. Such volumes of chilled solutions are feasible for a well-equipped emergency department but clearly are less realistic for emergency transport vehicles and are completely impractical for constrained environments such as the battlefield.

Nevertheless, Dr. Behringer and colleagues are to be congratulated for their innovation and persistence in developing techniques that have achieved such impressive results in experimental animals. Their sustained efforts have created the possibility that eventually, perhaps in the not too distant future, trauma patients or battlefield casualties with exsanguinated cardiac arrest will have some hope of survival.

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Do not resuscitate: Ordering nonassault and charting patients’ decisions to forgo cardiopulmonary resuscitation*

In this issue of Critical Care Medicine, Dr. Burns and colleagues (1) provide a definitive and carefully formulated history of 25 yrs of experience with what has so offensively been called a do-not-resuscitate order or DNR. They show how the crude and immoral practice of informally and clandestinely deciding not to resuscitate certain critical and terminal ill patients without bothering to inform them of their fate has been refined into a morally sensitive, humane, carefully institutionalized practice of discussing choices about resuscitation with patients or their valid surrogates and recording the decision in the patients’ records.

How that practice ever got called a do-not-resuscitate order is more an accident of the now-outmoded paternalistic assumptions of medicine as it was practiced a quarter of a century ago. As Dr. Burns and colleagues (1) show, the practice of resuscitation began with the presumption that most people suffering a cardiac arrest would consent to resuscitative efforts if only they were capable of doing so. This meant that once someone called that presumption into question for certain critically ill patients, some policy had to be developed to record the results of the deliberation to determine what constituted appropriate treatment.

Many more traditional analysts tried to convert the question into one of whether resuscitation was medically indicated. They mistakenly believed that the decision whether to attempt resuscitation was a technical one that clinicians could make relying on their traditional skills and authority. When they decided that resuscitation was not indicated, they

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Crit Care Med 2003 Vol. 31, No. 5 1593
ordered the medical care-giving team to refrain from attempting it.

Aside from the fact that charting an order in such cases has an offensively militaristic tone when one is referring to communicating what is really a patient's choice to fellow members of a healthcare team, the concept of a DNR order is, as Dr. Burns and colleagues (1) reveal, a rather muddled, often misunderstood term. It is, in fact, a product of a reassessment of the initial presumption of patient consent. It is determined—preferably through the active participation of the patient or valid surrogate—that the presumption of consent is no longer appropriate. The patient or surrogate has negated that presumption. To treat under these circumstances would be an assault—an illegal and unethical touching. Therefore, a physician writing a DNR order is ordering a nonassault (2).

Although, in principle, it is correct for members of the healthcare team not to assault their patients, it is, on reflection, a rather odd thing to chart. Similarly, one might also chart an order for nonsurgery on medical patients or nonrobbery on all patients. Although all of these would presumably lead to the right behavior, they are inappropriate in style and unnecessarily demeaning. They imply the staff are ethical, legal, and policy questions still unresolved. There are still those who advocate automatic suspension of so-called DNR orders when a patient is in the operating room, but the review and other literature make clear that such a policy is not warranted (3, 4). It confuses the wisdom of reassessing the patient's refusal of CPR, which makes perfectly good sense, with the conclusion that no patient could ever rationally refuse CPR in that setting.

Also makes clear that progress has been made in understanding the difference between goal-directed and procedure-directed discussions about resuscitation. Surely, patients are not normally concerned over exactly which pieces of equipment are used. They are concerned about whether the burdens are justified by the expected benefits, and they now expect that the burdens and benefits are based on the patient's beliefs and values, not those of other participants in the deliberation, including the clinician.

Because decisions to forgo resuscitation necessarily must be based on the beliefs and values of the patient, efforts to develop policies to authorize physicians to give unilateral orders that will make the patient dead against the patient's wishes have created real controversy and have generally been rejected both by the courts and by critical commentators (5). Of course, no physician should ever be placed in a position when he or she must attempt a resuscitation that has literally no chance of succeeding. However, there is a great difference between cases in which the intervention has literally no physiologic effect and those in which there is a low likelihood of success or the clinician has determined that, based on his or her personal values, the outcome is not worth the patient's burden.

One can imagine treatments, including life-prolonging treatments, that are desired by the patient but that society has determined are so costly in comparison with the expected benefit that society is forced to decide to withhold the treatment even against the patient's expressed demand. It is harder, however, to envision a case in which, absent that responsible societal decision to allocate resources, a physician should have the unilateral authority to refrain from resuscitating a patient when that resuscitation has some chance of success and is desired by the patient after that patient understands the burdens involved and the low likelihood of success. To unilaterally order members of the healthcare team to refrain from attempting to save such a patient seems to be the height of hutzpa, the apex of arrogance. In cases in which the physician raises legitimate questions about the presumption that the patient would consent to resuscitation, that physician and the other members of the healthcare team enter the resuscitation no-man's-land: they can no longer presume consent, but they cannot presume refusal either. They face an emergency with an imperative to clarify the patient's consent or refusal. If the patient wants CPR and related resuscitation efforts, and those efforts have some finite probability of success, then, absent administratively and societally placed limits on the use of resources, the patient should be resuscitated. In either case, the patient's decision should be charted and followed. The Society of Critical Care Medicine's Ethics Committee (6) has wisely recognized that the religious or moral views of patients may legitimately provide a basis for delivering such treatments. The 25-yr history that Dr. Burns and colleagues (1) have provided for us is largely a history of the increasing sophistication and rationalization of what started out as an indefensible set of informal practices and gradually became refined, regularized, and morally appropriate. We may still not always think clearly about exactly what is happening. We may still chart orders for nonassault rather than recording the
Physicians and organ procurement organizations—An essential partnership that must not be neglected*

Organ transplantation represents a life-enhancing and, in many cases, life-saving therapy for the transplant recipient. However, the number of potential recipients far outstrips donor availability. Indeed, a lack of donor organs is the major factor limiting the use of transplantation in patients with end-stage disease. The organ donor shortage is strikingly displayed in data from the 2000 Annual Report from the United Network for Organ Sharing which show that the total number of transplant recipients in 1999 was less than one-third the number of patients on the waiting list at the end of the year (1). Thus, efforts truly need to be made to increase organ donation.

An increase in donor organs can come about through extending donor criteria, which to date has been marginally successful. More importantly, we need to assure that all families are offered the option of organ donation and that all imminent deaths are referred in a timely manner. Likewise, we need to increase the conversion of potential organ donors through increased consent rates. To do the latter requires an analysis of factors affecting consent for organ donation, particularly those factors that can be modified. Although the percentage of potential donors in whom the request for organ donation is made has increased to 80%, the goal is 100%. And unfortunately, the percent of families consenting to organ donation remains around 50%. Thus, improving the consent rate would have the largest impact on increasing the number of organ donors.

It was in this context that the Federal Conditions of Participation from the Health Care Financing Administration introduced in 1998 required not only that all families be presented with the option of organ donation but also that trained “designated requestors” make the request (2). This was not an attempt for organ procurement organizations (OPOs) and transplant coordinators (who are trained in requesting organ donation) to take over the consent process, but represented the best means known to increase organ donation. Indeed, studies have shown that specific components in the request for organ donation make a big difference in the consent rate. These factors include not only patient and family characteristics, but modifiable factors such as discussing more topics concerning brain death and the organ donation process, having more conversations about organ donation, having more contact with OPO staff, and experiencing an optimal request pattern (3). An optimal request pattern was defined as having a healthcare provider other than a physician make the initial request followed by discussion with an OPO coordinator; using the optimal request pattern, donor families were almost three times as likely to consent compared with other models.

Unfortunately, the requirement for a trained “designated requestor” was misinterpreted by many to mean that physicians should not be a part of the process, and a resolution was called for at the December 1999 interim meeting of the American Medical Association House of Delegates to oppose and seek revision of the Conditions of Participation because many physicians believed that communication with the donor family was the province of the attending physician. The policy adopted by the American Medical Association House of Delegates in June 2000 led to the writing of “The Physician’s Role in Discussing Organ Donation with Families” which is published in this issue of Critical Care Medicine (4).

The reason the Conditions of Participation required a trained designated requestor is that training in the request process is the most important factor influencing the percentage of successful organ donation requests. Not only are physicians involved in caring for potential organ donors having to deal with end-of-life decisions, an area in which physicians are only recently becoming trained, they also must consider the issue of possible organ donation. In addition, separating the request process from caregivers may decrease the suspicion of the donor family that less than everything possible was done in the care of their loved one because of the potential that he could be an organ donor.

Physicians that wish to be part of the consent process should be trained in how to make the process most effective; indeed, such physicians would be welcomed. This might be most important if the physician has a long-term relationship with the patient and donor family, although this is rarely the case in the intensive care unit situation that results in most organ donors. However,

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5. In the Matter of Baby K1993 WL 343557 (E. D. Va.).

*See also p. 1568.

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many hospital personnel, particularly physicians, have not been trained in requesting organ donations, despite the fact that such training is associated with a higher rate of successful organ donation requests (5). The most likely reason that involvement of OPO staff in the donor request process increases the likelihood of organ donation is the extensive training that OPO personnel undergo regarding the process (6). Indeed, it is acknowledged in the article by Dr. Williams and colleagues (4) that the highest consent rates occur when OPO coordinators and physicians request organ donation together (7). Because of the time and training involved to appropriately make organ donation requests, and the relatively infrequent need for donation requests in the practice of most physicians, it would seem optimum for physicians and OPO personnel to work together in requesting organ donation. The situation truly calls for partnership and not competition.

It is this factor that leads to the major weakness of the article by Dr. Williams and colleagues (4). Although the authors discuss that involvement of OPO coordinators increases the likelihood of a “Yes” response to a request for organ donation, no OPO personnel are included as authors. It is indeed a missed opportunity that those who are most experienced and skilled in making the request for organ donation were not included in the development of such an important document. One of the strongest statements that could have been made in support of the collaborative effort that has been shown to be required for successful organ donation would have been to include an OPO representative as an author. The collaborative efforts to increase organ donation should not only occur at the bedside, but also in preparing a document such as this (4). However, the authors are to be commended for promoting the concept that physicians need to be trained in requesting organ donation if they wish to be part of the process. Indeed, the same group has shown that an experiential training curriculum for those involved in requesting permission for organ donation (physicians, nurses, clergy, and OPO coordinators) can increase the consent rate (8).

The amount and content of the training physicians should obtain to be effective requestors of organ donation have not been defined. However, here too we can learn from the OPOs, because through years of experience and studies they have performed, they have defined essential elements of an effective consent process. Indeed, the training required for physicians to more effectively partner in the organ donation request process involves not only factual information, but also information on when to make the request, how to make the request, and how to most effectively work with our OPO colleagues to make the consent process not only effective, but also the most compassionate possible for donor families in their time of shock and grieving. Physicians, other healthcare providers, and OPOs need to be partners in both identifying potential organ donors and obtaining consent from donor families. Such issues truly represent the difference between life and death for patients on organ transplantation waiting lists. Hopefully, any future documents concerning the organ donation process will more fully acknowledge the essential role of the OPO and organ procurement coordinators by including them in the discussions and resulting authorship of articles on this important topic.

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