The pediatric care crisis in Iraq

To the Editor:

Spinella et al (1) recently described the demographics of injured and critically ill children who continue to flock to U.S. Army hospitals in Iraq and Afghanistan. This report mirrors our experience in the 332nd Expeditionary Medical Group Air Force Theater Hospital (AFTH). Since the authors' deployment in January 2007, the AFTH has received an average of 23 children per month with life-threatening injuries—more than the required number of admissions for level I pediatric trauma centers in the United States.

Our care of these patients exceeds typical combat support doctrine, but out of a sense of moral obligation, we continue to admit, resuscitate, and operate on these children. Numerous case examples show that this approach has effectively won the hearts and minds of the Iraqi people in the community around the AFTH and the surrounding regions (Fig. 1).

After a blast near her home, a 1-year-old girl was struck in the forehead by an 8-cm iron chunk. Her father brought her to us with this fragment protruding like a horn. Our neurosurgeon removed the jagged block from her frontal lobe, and she returned to her family in good health. Another 2-year-old girl was discharged after recovering from a blast to her right neck. We had to emergently ligate her vertebral artery and repair a large laceration in her trachea. A young man on whom we had operated for abdominal trauma brought his infant niece to our gate with a note from an Iraqi physician: "Baby vomiting. Barium study consistent with pyloric stenosis. Please treat." We corrected the child's profound electrolyte derangements and then performed a routine pyloromyotomy. Another 10-month-old baby exposed to vaporized chlorine was intubated and transported to our hospital, where a bronchoscopy showed profound bronchial edema. After 5 days of bronchodilators and steroids, he extubated and was soon discharged. This list goes on, as both Iraqi families and military medics continue to bring children to our facility.

As documented by Spinella et al (1), caring for these children requires a disproportionate investment of time and resources by our combat hospitals, but the Iraqis currently have no viable alternative. According to the Brookings Institute, more than half of Iraqi physicians have fled the country while 2000 have been murdered and another 250 kidnapped (2). A World Health Organization report cites a 70% mortality rate for critically injured patients cared for in the sparsely supplied and minimally staffed Iraqi hospitals (3).

As a transitional solution, the relative security of our bases after the recent surge might be used to encourage nongovernmental organizations (NGOs) to return to Iraq. Allowing NGO healthcare workers to offer services for Iraqi civilians within the walls of our bases would require progressive thinking, representing a landmark in cooperation between the military and the NGOs, but it could serve as an important intermediate step as the Iraqi medical system rebuilds. But for now we must not grow weary of our care for these children. The future of this new democracy and our moral integrity as physicians demand nothing less.

The views expressed are personal opinions of the authors and do not officially represent the U.S. Air Force.

Jeremy W. Cannon, MD, SM, David L. Smith, MD, Wilford Hall Medical Center, Lackland AFB, Texas

REFERENCES

1. Spinella PC, Borgman MA, Azarow KS: Pediatric trauma in an austere combat environment. *Crit Care Med* 2008; 36(7 Suppl): S293–S296
2. O'Hanlon ME, Campbell JH: Iraq Index: Tracking Variables of Reconstruction and Security in Post-Saddam Iraq. Washington, DC, The Brookings Institution, 2007
3. Rosenthal E. Iraq's public health services severely strained, group says. *New York Times* April 18, 2007

DOI: 10.1097/CCM.0b013e3181a9f7db

Norepinephrine in fluid-refractory catecholamine-resistant cold shock: Are we sure?

To the Editor:

I read with great interest the revision of clinical guidelines and practice parameters for hemodynamic support of pediatric and neonatal septic shock (1). These guidelines represent an extraordinary tool for any pediatric intensivist involved in the treatment of septic children and newborns, and I am sincerely grateful to all the authors for achieving this milestone in the field of pediatric intensive care.

However, a significant proposed modification in the algorithm for children with fluid refractory cold shock is, in my opinion, highly questionable and requires further clarification.

Indeed, unlike the 2002 version (2), in the algorithm, the fluid-refractory catecholamine-resistant cold shock has been split into two arms, depending on the presence or the absence of hypotension: in a normovolemic, low blood pressure child with cold shock, after titration of fluids and epinephrine, in presence of $Scvo_2$ >70% and Hb >10 g/dL, norepinephrine is recommended.

At least two questions may be submitted:

1) How many skilled pediatric intensivists may be honestly sure, even after careful re-evaluation, about adequate fluid resuscitation and appropriate epinephrine and steroid dosage in a cold septic shock child with hypotension? The use of a pulmonary artery catheter or new noninvasive techniques may be difficult in small children, and dosage of hydrocortisone is not well established, so the managing physician must continue to rely on clinical goals (3).
2) Cold shock is probably a high systemic vascular resistance condition. In this condition, what is the rationale to indicate a strong vasopressive agent, such as norepinephrine?

Any transposition from adults to pediatric patients regarding the use of norepinephrine as a first-line agent in fluid refractory shock is, in my opinion, extremely dangerous in terms of pseudonormalization of blood pressure despite severe tissue hypoperfusion.

The author has no potential conflicts of interest to disclose.

Paolo Silvani, MD, Department of Cardiothoracic Anesthesia and Intensive Care, University Vita-Salute San Raffaele, Milan, Italy

REFERENCES

1. Brierley J, Carcillo JA, Choong K, et al: Clinical practice parameters for hemodynamic support of pediatric and neonatal septic shock: 2007 update from the American College of Critical Care Medicine. *Crit Care Med* 2009; 37:666–688
2. Carcillo JA, Fields AI; American College of Critical Care Medicine Task Force Committee Members: Clinical practice parameters for hemodynamic support of pediatric and neonatal patients in septic shock. *Crit Care Med* 2002; 30:1365–1378
3. Parker MM: Pediatric sepsis: Time is of the essence. *Crit Care Med* 2009; 37:785–786

DOI: 10.1097/CCM.0b013e3181a9f84c

The author replies:

Your points are well taken indeed and we agree with you completely. In the cold hypotensive septic shock patients, aggressive volume resuscitation and inotropic dose epinephrine directed to normal blood pressure and capillary refill $\leq$2 seconds should be used. Once measurements of superior vena cava/right atrial junction or inferior vena cava/right atrial junction oxygen saturation, and/or cardiac index (CI) are available, then therapies should further be directed to attain a central venous oxygen saturation ($ScvO_2$) $>$70% (with hemoglobin $\geq$10 gm/dL) and a CI between 3.3 and 6.0 $L{\cdot}min^{-1}{\cdot}m^{-2}$. In addition, when an arterial line is in place, therapies should also be directed to normal perfusion pressure for age as indicated by the mean arterial pressure-central venous pressure. If the cold patient has a normal mean arterial pressure-central venous pressure and a low $ScvO_2$/CI, then more volume with afterload reduction is recommended to improve cardiac output. Agents used for this include type IV phosphodiesterase inhibitors, as well as nitrosovasodilators, prostaglandins, and dopexamine. We presume that you agree with this approach.

What about the cold shock patients who after aggressive resuscitation with fluids and inotropic dose epinephrine continue to have hypotension? These are the patients who are most likely to die with refractory shock. We now suggest considering adding norepinephrine (a mixed inotrope/vasopressor) to this patient to restore blood pressure rapidly, because inotropic dose epinephrine and aggressive fluid resuscitation have been unsuccessful. With blood pressure now restored, we then recommend adding dobutamine and/or type IV phosphodiesterase inhibitors with more volume resuscitation to attain an $ScvO_2$ $>$70% and a CI >3.3 $L{\cdot}min^{-1}{\cdot}m^{-2}$ adequate cardiac output and oxygen delivery.

As Dr. Sylvani correctly points out that cold shock is likely to be a high systemic vascular resistance condition, which requires more fluid, not a vasopressor. Management of these near-refractory shock patients is difficult without more information attained with measurements from the pulmonary catheter, pulse contour cardiac output catheter, or some other cardiac output/volume status monitoring. We use the pulmonary artery catheter in our center. Commonly, when the pulmonary artery occlusion pressure and CI numbers are available to us, we establish a milrinone infusion, wean off norepinephrine, give another 80 mL/kg of fluid resuscitation, and find that refractory shock is no longer refractory. We thank Dr. Sylvani for highlighting this important nuance of advanced septic shock management.

The author has not disclosed any potential conflicts of interest.

Joseph A. Carcillo, MD, Department of Critical Care Medicine and Pediatrics, Department of Anesthesiology-Children's Hospital of Pittsburgh, Pittsburgh, PA

DOI: 10.1097/CCM.0b013e3181a9edb7

Gene-expression profiling of peripheral blood mononuclear cells in sepsis

To the Editor:

The article by Tang et al (1) provides important results performed on circulating peripheral blood monocytic cells (PBMC) using microarray technique to assess potential difference in gene expressions (pangenomic microchip) between septic and nonseptic systemic inflammatory response syndrome. If one can adopt easily the results showing differences in genes expression controlling immune function and inflammation processes between sepsis and nonseptic patients, with no difference between Gram-positive and Gram-negative infection profile, some comments can be added to the discussion.

First, it is not true that no data are available on PBMC in septic patients, because many studies have been published on PBMC or in whole blood that include both neutrophils and PBMC (2–4). Second, although the authors mentioned that timing was not the aim of the study, it has to remember that gene expression is changing fast along time evolution (hours). It might be important to mention the time of sampling referring to admission, or infection onset or to organ failure occurrence. Third, some explanations on the impact of the significant difference in incidence of septic shock between the two cohorts may hamper the results (Table 1). This implies a certain degree of heterogeneity within septic patients, which may correspond to different gene expression as previously published (3). Fourth, the potential "contamination" of circulating PBMC by nonmature myeloid cells was not mentioned. These myeloid cells may account for at least 50% of the cells after Ficoll separation within the first 2 days and be differentially present in severe sepsis or in septic shock (3). This may account for common and uncommon gene expression patterns between neutrophils and PBMC as for differences in gene expression between the two groups. Fifth, despite performing a sophisticated statistical process, the validation of microarray for some genes by reverse transcriptase-polymerase chain reaction was not shown and may add credibility to the presented results.

The specific mention of the gene *S100A8* as an important potential target is supported by the recent article by Vogl et al (5). However, little is known in human beings about its role in sepsis, particularly the origin and the level of this protein (3). The demonstrated link between this molecule and TLR-4 receptor pathway might stimulate further human research. In this study, it is not clear if the gene controlling this protein was more expressed in sepsis than in nonseptic systemic inflammatory response syndrome. The expression of this gene as the one encoding for S100A12 has been shown in PBMC to decrease rapidly in patients who survived in comparison with those who died in relation with the protein level (3).

The authors have not disclosed any potential conflicts of interest.

Didier Payen, Anne Claire Lukaszewicz, Department of Anesthesiology and Critical Care, University Paris 7 Denis Diderot, Hopital Lariboisiere AP-HP, Paris, France

REFERENCES

1. Tang BM, McLean AS, Dawes IW, et al: Gene-expression profiling of peripheral blood mononuclear cells in sepsis. *Crit Care Med* 2009; 37:882–888
2. Pachot A, Lepape A, Vey S, et al: Systemic transcriptional analysis in survivor and non-survivor septic shock patients: A preliminary study. *Immunol Lett* 2006; 106:63–71
3. Payen D, Lukaszewicz AC, Belikova I, et al: Gene profiling in human blood leucocytes during recovery from septic shock. *Intensive Care Med* 2008; 34:1371–1376
4. Prucha M, Ruryk A, Boriss H, et al: Expression profiling: Toward an application in sepsis diagnostics. *Shock* 2004; 22:29–33
5. Vogl T, Tenbrock K, Ludwig S, et al: Mrp8 and Mrp14 are endogenous activators of toll-like receptor 4, promoting lethal, endotoxin-induced shock. *Nat Med* 2007; 13:1042–1049

DOI: 10.1097/CCM.0b013e3181aab5a3

The authors reply:

We thank Dr. Payen for his interest in our work. Our response to his question is as follows.

First, the first two studies quoted by Dr. Payen had different focus than our study. Our study investigated the differential gene expression between septic and nonseptic patients, whereas the two above-mentioned studies looked at the relationship between gene expression and survival/recovery in septic patients. The third study did not use purified mononuclear cells, and hence the generalization of its findings to mononuclear cells is limited.

Second, we agree with Dr. Payen that the timing of sample collection is absolutely important. Unfortunately, we did not collect this information in our study.

Third, we agree with the observation that the occurrence rate of septic shock differs between the two cohorts. There are advantages and disadvantages in having such a difference. The advantage is that the external validation is more readily generalized to usual clinical settings. The disadvantage is that there will be an expected decrease in the diagnostic accuracy of the second cohort due to its inherent heterogeneity.

Fourth, we also agree that there was potential for "contamination" of our samples by nonmature myeloid cells. However, this will need to be addressed in future studies.

Fifth, findings from reverse transcriptase-polymerase chain reaction would enhance our manuscript. However, it was not presented due to space limitation.

The expression level of the gene S100A8 can be found both in the figure and the supplementary data of our manuscript (both are available online). The gene is reduced in septic patients compared with control. In the study of Dr. Payen, we noted with interest that the same gene also showed a reduced level of expression during the recovery of septic patients.

We also agree with Dr. Payen that more studies are needed to further explore the large amount of information revealed by gene-expression studies in septic patients.

The authors have not disclosed any potential conflicts of interest.

Benjamin M. P. Tang, Anthony S. McLean, Stephen J. Huang, Department of Intensive Care Medicine, Nepean Hospital and Western Clinical School, University of Sydney, Penrith, Australia; Ian W. Dawes, Ruby C. Y. Lin, Ramaciotti Centre for Gene Function Analysis and School of Biotechnology and Biomolecular Sciences, University of New South Wales, Sydney, Australia

DOI: 10.1097/CCM.0b013e3181a9ef74

Comment on the 2007 American College of Critical Care Medicine clinical guidelines for management of pediatric and neonatal septic shock

To the Editor:

We read with great interest the article by Brierley et al (1). The update of the 2002 American College of Critical Care Medicine clinical practice parameters for hemodynamic support of neonates and children with septic shock has intended to promote improved patient outcomes with the implementation of "best clinical practices." However, some recommendations should be revised to be in line with the published literature and current evidence. We raise five concerns.

First, considering that standardization of diagnostic criteria is crucial to early recognition and timely intervention, and also for the purposes of clinical research trials, the consensus definitions of pediatric sepsis and septic shock (2) should be contemplated.

Second, the authors recommend that the type of fluid for initial resuscitation be at the practitioner's preference. Despite the lack of evidence of better efficacy of any particular type of colloid solution for fluid resuscitation (3), albumin infusion has been associated with less serious adverse effects, such as coagulopathy, renal failure, and anaphylactoid reactions, compared with the synthetic colloids (4, 5). Therefore, on the flow diagram, the generic term *colloid* should be replaced by *albumin* until further information on the safety of synthetic colloids is available.

Third, the recommendation of antibiotic administration over the first 15 minutes of therapy may lead to work overload for the healthcare professionals and less efficient fluid resuscitation. Instead, this recommendation should be placed on the algorithm at 60 minutes after treatment initiation.

Fourth, because children frequently respond well to aggressive volume resuscitation, inotropes and vasopressors should only be used in patients with fluid refractory shock, after hypovolemia has been resolved. This can avoid a bias in the interpretation and misclassification of the hemodynamic status (fluid-responsive or fluid-refractory shock).

Finally, management guidelines must be implemented into daily clinical practice to promote outcome improvement. Despite wide dissemination, the compliance with the American College of Critical Care Medicine guidelines has been poor (6, 7). In the United Kingdom, fluid and inotrope management suggested by the 2002 American College of Critical Care Medicine guidelines was not followed in 62% of children with septic shock (7), and 80% of septic shock pediatric cases had central venous oxygen saturations $<70\%$ at the time of intensive care unit admission (8). Therefore, better implementation strategies are needed to efficiently translate the American College of Critical Care Medicine recommendations into clinical practice.

The authors have not disclosed any potential conflicts of interest.

Werther B. Carvalho, MD, Federal University of São Paulo, São Paulo, Brazil; Ana P. C. P. Carlotti, MD, Fabio Carmona, MD, Eduardo J. Troster, MD, Albert Bousso, MD, Andréa M. C. Ventura, MD, Daniela Carla de Souza, MD, and Ricardo S. Yamaguchi, MD, University of São Paulo, São Paulo, Brazil

REFERENCES

1. Brierley J, Carcillo JA, Choong K, et al: Clinical practice parameters for hemodynamic support of pediatric and neonatal septic shock:

2007 update from the American College of Critical Care Medicine. *Crit Care Med* 2009; 37:666–688
2. Goldstein B, Giroir B, Randolph A: International pediatric sepsis consensus conference: Definitions for sepsis and organ dysfunction in pediatrics. *Pediatr Crit Care Med* 2005; 6:2–8
3. Bunn F, Trivedi D, Ashraf S: Colloid solutions for fluid resuscitation. *Cochrane Database Syst Rev* 2008; 1:CD001319
4. Barron ME, Wilkes MM, Navickis RJ: A systematic review of the comparative safety of colloids. *Arch Surg* 2004; 139:552–563
5. Kheirabadi BS, Crissey JM, Deuzman R, et al: Effects of synthetic versus natural colloid resuscitation on inducing dilutional coagulopathy and increasing hemorrhage in rabbits. *J Trauma* 2008; 64:1218–1229
6. Oliveira CF, Nogueira de Sá FR, Oliveira DS, et al: Time- and fluid-sensitive resuscitation for hemodynamic support of children in septic shock: Barriers to the implementation of the American College of Critical Care Medicine/Pediatric Advanced Life Support guidelines in a pediatric intensive care unit in a developing world. *Pediatr Emerg Care* 2008; 24:810–815
7. Inwald DP, Tasker RC, Peters MJ, et al: Emergency management of children with severe sepsis in the United Kingdom—The results of the Paediatric Intensive Care Society sepsis audit. *Arch Dis Child* 2009; 94:348–353
8. Peters MJ, Brierley J: Back to basics in septic shock. *Intensive Care Med* 2008; 34:991–993

DOI: 10.1097/CCM.0b013e3181aabc7c

The author replies:

I thank the Brazilian Study Group on Pediatric Critical Care Medicine for their comments on the 2007 American College of Critical Care Medicine (ACCM) clinical guidelines for management of pediatric and neonatal septic shock (1). For the most part, I agree with all the recommendations to varying degrees. I will address these recommendations in the order of their appearance.

On the first recommendation, the text of the IPSCC guidelines (2) states that our definitions should be used to initiate therapy in part, because the ACCM definitions recognize shock at earlier stages when the best outcomes can be attained with time-sensitive use of conventional therapy guidelines. The new definitions in the International Pediatric Sepsis Consensus Conference document recognize late shock with a capillary refill >5 seconds and 18% mortality, compared with our definitions that recognize a capillary refill cutoff of >2 seconds and a mortality of 5%. I continue to recommend the use of the ACCM definitions for timely, successful conventional therapy guideline implementation and the International Pediatric Sepsis Consensus Conference definitions for randomized trials of experimental agents, such as activated protein C.

On the second recommendation, we agree that the pediatric literature supports albumin as the best colloid to use. However, in some parts of the world albumin is not available. We have little experience with colloids other than albumin among the members who wrote the guidelines.

On the third recommendation, we agree that antibiotic administration should not slow resuscitation. Antibiotic administration within the first hour is excellent. On the fourth recommendation, if one has a choice to make between fluid administration and inotrope administration in a patient who requires fluid administration, then fluid resuscitation has priority. However, if one has enough access (i.e., two intravenous lines), then both can be given simultaneously. The new change is to not wait for central access to be established before starting the inotrope. Start the inotrope peripherally while you are attaining central access. Inotropes and particularly adrenaline can be given through inhalation or subcutaneously when access is not available.

On the fifth and final recommendation, we agree entirely. There is a consensus among all who have attempted to implement guidelines that our challenge is one of education, organization, and logistics. The Global Pediatric Sepsis Initiative housed at www.pediatricsepsis.org or www.wfpicccs.org provides a bundle registry to help with this task. Here, participants (physicians like us) anonymously enter the data of patients with their individual care characteristics and outcomes into their respective bundles. This quality assurance tool tracks performance and barriers to performance allowing for systematic improvement over time. Together we face the great but rewarding challenge of improving outcomes in these children globally.

The author has not disclosed any potential conflicts of interest.

Joseph A. Carcillo, MD, Department of Critical Care Medicine and Pediatrics, Department of Anesthesiology, Children's Hospital of Pittsburgh, Pittsburgh, PA

REFERENCES

1. Carvalho WB, Carlotti APCP, Carmona F, et al: Comment on the 2007 American College of Critical Care Medicine clinical guidelines for management of pediatric and neonatal septic shock. *Crit Care Med* 2009; 37:2324–2325
2. Goldstein B, Giroir B, Randolph A: International Pediatric Sepsis Consensus Conference: Definitions for sepsis and organ dysfunction in pediatrics. *Pediatric Crit Care Med* 2005; 6:2–8

DOI: 10.1097/CCM.0b013e3181a9ee81

Lipid emulsion as antidotal therapy—Ready to register?

To the Editor:

Two recent editorials (1, 2) endorsing the use of lipid in refractory arrest secondary to any lipophilic cardiotoxin are likely to push lipid in general toxicology off the lab bench and onto the shop floor of our emergency departments and intensive care units. We would add to the chorus of calls to continue posting cases of use on Guy Weinberg's Website www.lipidrescue.org and to join the published cases of use in the peer-reviewed literature (3–6).

It is, however, an unfortunate truth that the plural of anecdote is not data. If, as seems likely, uptake of lipid is about to increase significantly, this gives us an opportunity to gain knowledge about a therapy for which much remains unknown. Refinement of dosage regimens, appropriate timing of use in patient decline, and adverse effects at the doses used need more work if the therapy is to progress. Centralized, systematic collection of data on usage holds much attraction as a means of investigation.

This represents an exciting challenge and choice for interested colleagues in emergency medicine, intensive care, toxicology, and anesthesia—to be either active drivers or passive observers of the evolution of this therapy. It seems to us that the time to start a serious dialogue about registries of use of lipid emulsion as antidote has come.

The authors have not disclosed any potential conflicts of interest.

Grant Cave, FACEM, Departments of Emergency, Anaesthesia and Intensive Care, Hutt Hospital, Lower Hutt, New Zealand; Martyn Harvey, FACEM, Department of Emergency Medicine, Waikato Hospital, Hamilton, New Zealand

REFERENCES

1. Brent J: Poisoned patients are different—Sometimes fat is a good thing. *Crit Care Med* 2009; 37:1157–1158
2. Picard J, Harrop-Griffiths W: Lipid emulsion

to treat drug overdose—Past, present and future. *Anaesthesia* 2009; 64:119–121
3. Sirianni AJ, Osterhoudt KC, Calello DP, et al: Use of lipid emulsion in the resuscitation of a patient with prolonged cardiovascular collapse after overdose of bupropion and lamotrigine. *Ann Emerg Med* 2008; 51:412–415
4. Finn SD, Uncles DR, Willers J, et al: Early treatment of a quetiapine and sertraline overdose with intralipid. *Anaesthesia* 2009; 64: 191–194
5. Doulcourt BA, Aaron CK: Intravenous fat emulsion for refractory veralpamil and atenolol induced shock: A human case report [abstract]. *Clin Toxicol* 2008; 46:119–120
6. Harchelroad FP, Palma A: Efficacy and safety of intravenous lipid therapy in a beta blocker overdose [abstract]. *Clin Toxicol* 2008; 46:634

DOI: 10.1097/CCM.0b013e3181aabcd7

The author replies:

I agree with the remarks of Cave and Harvey (1) regarding my recent editorial article (2). Their letter provides an opportunity to reflect on the question of how best to proceed in the evaluation of the apparently promising use of lipid emulsion therapy (LET) in the treatment of poisoned patents.

My use of the adjective "apparently" is no mere quip. Rather, it describes the current state of scientific wisdom concerning the efficacy of LET. Cave and Harvey articulate the fundamental truth that "the pleural of anecdote is not data." However, as alluded to in my editorial, the justification of LET is not purely anecdotal. There is a substantial body of animal data propelling the widespread interest in the human use of this treatment. However, we can never know if a therapy is efficacious in humans unless it is studied in humans in real clinical scenarios.

Although I agree with Cave and Harvey that the next logical step in the evaluation of LET is to establish a formal registry, it is ironic to note that registry data are still anecdotal and, thus, will never definitively answer the question of the true clinical utility of LET. It is likely that this question would only be unambiguously resolved with a randomized clinical trial. However, the relative rarity of relevant poisonings and the need for on-the-spot immediate entry into a randomized clinical trial dramatically erode the likelihood that such a trial will be performed. Thus, we are left with the registry concept as the next best solution.

For a registry of cases of LET use to be truly informative, it is imperative that it fulfills certain fundamental criteria: 1) it should be prospective, 2) it must capture all cases in participating centers regardless of the outcome, and 3) cases should be cared for at the bedside by specialists familiar with the use of LET and cognizant of the necessary data that should be collected for the registry.

Coincidently, Toxicology Investigators Consortium of the American College of Medical Toxicology is presently developing a registry of the use of LET meeting the stringent criteria outlined earlier. Toxicology Investigators Consortium is a network of medical toxicology practices providing bedside inpatient care for 10,000–20,000 cases in the United States annually. A retrospective series of cases cared for by the Toxicology Investigators Consortium network has already been assembled. The prospective phase is due to begin shortly.

The author has not disclosed any potential conflicts of interest.

Jeffrey Brent, MD, PhD, Toxicology Associates, University of Colorado, Health Sciences Center, Denver, CO

REFERENCES

1. Cave G, Harvey M: Lipid emulsion as antidotal therapy—Ready to register? *Crit Care Med* 2009; 37:2325–2326
2. Brent J: Poisoned patients are different—Sometimes fat is a good thing. *Crit Care Med* 2009; 37:1157–1158

DOI: 10.1097/CCM.0b013e3181a9edf9

Critically ill patients need "FAST HUGS BID" (an updated mnemonic)

To the Editor:

We have recently reviewed the excellent article by Vincent (1) describing a daily "FAST HUG" checklist, initially developed to highlight the key aspects of care for all critically ill patients, to be used by the multidisciplinary care team. We propose an updated mnemonic, "FAST HUGS BID," which includes daily assessment for ventilator separation with a spontaneous breathing trial (S), evaluation and maintenance of appropriate bowel function (B), removal of indwelling catheters (I), and de-escalation (D) of antimicrobial and other pharmacotherapies (Table 1). Although we suggest the use of this checklist at least twice daily (i.e., BID), systematic approaches to patient care should, ideally, be used repeatedly throughout work shifts.

Table 1. Components of "FAST HUGS BID"

F	Feeding
A	Analgesia
S	Sedation
T	Thromboembolic prophylaxis
H	Head of bed elevation
U	Ulcer (stress) prophylaxis
G	Glycemic control
S	Spontaneous breathing trial
B	Bowel regimen
I	Indwelling catheter removal
D	De-escalation of antibiotics

The daily assessment of a spontaneous breathing trial has been shown to be a safe, effective, and highly predictive method for determining which patients will tolerate ventilator separation (2, 3). Prolonged mechanical ventilation has been associated with increased rates of ventilator-associated pneumonia and in-hospital and total mortality. A spontaneous breathing trial should, therefore, be considered at least daily and performed in a highly protocolized fashion by a well-trained team of nurses and respiratory therapists.

Disorders of gastrointestinal motility, including ileus, constipation, and diarrhea, are common in critically ill patients and may contribute to additional disease burden. Diarrhea may result in electrolyte imbalances, dehydration, hemorrhoidal irritation with resultant anemia, and delirium. Constipation, in contrast, may result in significant patient discomfort, feeding intolerance, and delirium. Institutional guidelines and use of standardized definitions of constipation and diarrhea may facilitate bowel dysfunction management (4). Routine assessment and treatment to maintain normal bowel function should be conducted in all critically ill patients.

Indwelling catheters, including urinary, arterial, central venous, pulmonary artery, and dialysis catheters, are commonly used in critically ill patients. Because they penetrate through the body's natural protective mechanisms, they are at high risk for local and systemic infections. Early discontinuation and removal, when these catheters are no longer needed, remains an important strategy to combat catheter-associated infections. Daily (or more frequent) assessment should be performed of the ongoing need for these catheters, and their removal, when not medically necessary.

Following the collection of appropriate specimens for microbial culture, prompt empirical antimicrobial therapy is crucial for the treatment of serious, nosocomial infections. Once a pathogen has been identified and antimicrobial susceptibilities have been reported, the regimen should be converted to the most narrow-spectrum, cost-effective, and pathogen-specific antibiotic. This practice, known as *antibiotic de-escalation* or *streamlining*, minimizes exposure to broad-spectrum antimicrobial therapy (5). The same principle of de-escalation can be applied to other pharmacologic treatments, which should be regularly re-evaluated for appropriate indications to minimize risk of adverse effects and medication errors.

Multidisciplinary protocols and checklists are an essential and evolving component of excellent evidence-based critical care. We suggest giving patients "FAST HUGS BID," as a modification to a widely used mnemonic, which can be used on a routine basis during multidisciplinary rounds to improve the care provided to all critically ill patients.

The authors have not disclosed any potential conflicts of interest.

William R. Vincent III, PharmD, Arnold & Marie Schwartz College of Pharmacy and Health Sciences, Division of Pharmacy Practice, Long Island University, Brooklyn, NY; Kevin W. Hatton, MD, Department of Anesthesiology, Division of Critical Care, University of Kentucky College of Medicine, Lexington, KY

REFERENCES

1. Vincent JL: Give your patient a FAST HUG (at least) once a day. *Crit Care Med* 2005; 33: 1225–1229
2. Girard TD, Ely EW: Protocol-driven ventilator weaning: Reviewing the evidence. *Clin Chest Med* 2008; 29:241–252
3. Robertson TE, Mann HJ, Hyzy R, et al: Multicenter implementation of a consensus-developed, evidence-based, spontaneous breathing trial protocol. *Crit Care Med* 2008; 36:2753–2762
4. Dorman BP, Hill C, McGrath M, et al: Bowel management in the intensive care unit. *Intensive Crit Care Nurs* 2004; 20:320–329
5. Ibrahim EH, Ward S, Sherman G, et al: Experience with a clinical guideline for the treatment of ventilator-associated pneumonia. *Crit Care Med* 2001; 29:1109–1115

DOI: 10.1097/CCM.0b013e3181aabc29

The author replies:

I appreciate the interest expressed by Vincent et al (1) in my article (2). I, too, can think of other elements that could be added to the original mnemonic. For example, we should also pay attention to Electrolytes, Airway, Catheters, Hematology, Hemodynamics, Oral care, Urine analysis, and Relatives; this would give us FAST HUG EACH HOUR, even better than the twice daily suggested by Vincent et al!

In fact, we could continue expanding the mnemonic almost indefinitely, creating long phrases, even poems (!), but this would defeat the original concept underlying the FAST HUG, which was to provide a short and simple mental checklist that can be easily remembered by all staff members, but that includes most important aspects of patient management to be checked whenever attending an intensive care unit patient. A longer mnemonic is less likely to be remembered and hence less likely to be applied.

The author has not disclosed any potential conflicts of interest.

Jean-Louis Vincent, MD, PhD, FCCM, Department of Intensive Care, Erasme Hospital, Université libre de Bruxelles, Brussels, Belgium

REFERENCES

1. Vincent W, Hatton KW: Critically ill patients need "FAST HUGS BID" (an updated mnemonic). *Crit Care Med* 2009; 37:2326–2327
2. Vincent JL: Give your patient a fast hug (at least) once a day. *Crit Care Med* 2005; 33:1225–1229

DOI: 10.1097/CCM.0b013e3181a9eefb