N E W S L E T T E R

ASCCA INTERCHANGE

AMERICAN SOCIETY OF CRITICAL CARE ANESTHESIOLOGISTS

Volume 16 Number 1 2004 www.ascca.org

President's Message

Something's Changed

By Clifford S. Deutschman, M.D.
Philadelphia, Pennsylvania

Can you feel it? Can you smell it in the air? Does the water you drink (assuming it comes from the tap and not out of an overpriced bottle) taste a little different? Does the food from your hospital cafeteria (ugh) seem a bit better? Do your colleagues, especially those who are not intensivists, look at you a little differently? Are you no longer the oddball who would rather practice somewhere besides the operating room? Something has changed. The world we live in is different. I have noticed. Have you?

Sometime within the last two years, the leaders of American anesthesiology have changed their attitudes about the practice of critical care medicine and its importance to the specialty as a whole. In the past, we were a small, "interesting" subgroup of primarily academic practitioners thought to be out on the fringe.

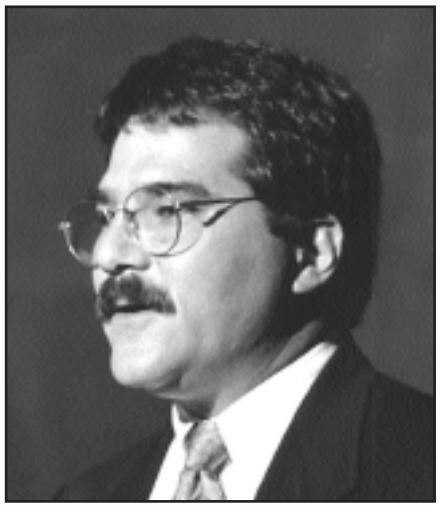
A New Image

This year critical care has taken on a new image. The change is obvious in many ways. It became clear to me some time ago. The American Society of Anesthesiologists (ASA) leadership routinely invites leaders of subspecialty organizations to their March Board of Directors meeting. I attended two years ago. It was clear that then-ASA President James E. Cottrell, M.D., and other ASA leaders were profoundly interested in what I had to say. This is not to imply that they were the first to care, just that the attitude was different than I had expected. This was not just lip service; these individuals seemed to regard ASCCA in a way that differed from their predecessors. Something had changed. Perhaps we were better able to articulate our message, perhaps the changing nature of the specialty influenced the leadership to view critical care more favorably, but the altered perspective was palpable.

Subsequent events confirm my impressions, including the February 2004 issue of the *ASA NEWSLETTER*, which is devoted to critical care, and in the content of the ASA's 2004 Doctors Day kit, also highlighting critical care medicine. Both of these products indicate that ASA leadership regards our subspecialty much differently than in the past. There are other indicators that merit discussion.

Task Force Formed

At last year's ASA Annual Meeting, then-President-Elect Roger W. Litwiller, M.D., asked Gerald A. Maccioli, M.D. (ASCCA Secretary, North Carolina Society President, North Carolina alternate delegate and chair of the ASA Committee on Critical Care Medicine and Trauma Medicine) and me to head a task force to examine the role of critical care within anesthesiology and to provide suggestions on how critical care might become a more integral part of anesthesiology. Dr. Litwiller appointed to this task force prominent individuals not only from general critical care medicine (Todd Dorman, M.D.; Neal H. Cohen, M.D., ASCCA Immediate Past-President; C. William Hanson, M.D., past ASCCA President; and Alex S. Evers, M.D., Chair of



Clifford S. Deutschman, M.D.

Anesthesiology at Washington University in St. Louis and a past president of the Association of University Anesthesiologists) but also leaders from anesthesiology subspecialties where critical care plays an important role (including James A. Ramsey, M.D., a cardiac anesthesiologist; Jayant K. Deshpande, M.D., a pediatric anesthesiologist; and W. Andrew Kofke, M.D., a neuroanesthesiologist). Our charge is to detail what needs

Continued on page 3

Sometime within the last two years, the leaders of American anesthesiology have changed their attitudes about the practice of critical care medicine and its importance to the specialty as a whole.

CONTENTS

MEMBERSHIP INFORMATION

E-mail

You may e-mail inquiries to ASCCA at the following addresses:
General inquiries:
ascca@ASAhq.org
Meeting information:
asccameetings@ASAhq.org
Membership information:
asccamembership@ASAhq.org

Membership

Membership in ASCCA is open to all anesthesiologists and residents in approved anesthesiology programs. Membership applications may be obtained by writing to **ASCCA**, 520 N. Northwest Highway, Park Ridge, IL 60068-2573 or through the ASCCA Web site at <www.ascca.org/membership.html>.

Web Page

You may visit the ASCCA World Wide Web site at:
www.ascca.org

ASCCA Dues

Dues are $150 for active and associate members; $100 for international members and $50 for residents/fellows. Dues may be paid online at <www.ascca.org/cart.html> by credit card or by mailing payment to the ASCCA office. Remember, payment of your dues allows you to enjoy the full privileges of ASCCA membership.

EDITORIAL NOTES

Editorial Policy

The opinions presented are those of the authors only, not of ASCCA. Drug dosages, accuracy and completeness of content are not guaranteed by ASCCA.

The ASCCA *Interchange* is published by the American Society of Critical Care Anesthesiologists, 520 N. Northwest Highway, Park Ridge, IL 60068-2573; (847) 825-5586. Deadlines are six weeks prior to the month of publication (e.g., January 15 for an issue scheduled for March).

Editorial

Tomorrow

By Michael L. Ault, M.D.
Editor


Michael L. Ault, M.D.

With the arrival of summer, new anesthesiology residents across the country enter their respective training programs. It is at this time that I wonder whether these residents will become the physicians who will cure our ailing health care system. We now have a system that is overburdened with malpractice insurance premiums, cost, technology and a paucity of time. Most physicians have significant concerns about cuts in reimbursement and salaries while the demands of an ever-growing health care system eat away at physician autonomy, time spent with patients and job satisfaction. No longer physicians, we are now *employees who practice medicine*.

We now have a system that is overburdened with malpractice insurance premiums, cost, technology and a paucity of time.

How do we find our way back to being physicians? I certainly do not have the answer to this question, but I hope that within the new class of anesthesiology trainees, someone will find the answer. Several years ago, many physicians began to aggressively seek business administration education and training to help extricate medicine from the overwhelming bureaucracy in which it found

Continued on page 10

Something's Changed

Continued from page 1

to be done and how it might be accomplished. We have made excellent progress. Dr. Maccioli presented a preliminary report to the ASA leadership at the 2004 Board of Directors meeting last March (I was attending a meeting in Munich, Germany, which is another story worth telling, but not here and now). The reception was, by report, overwhelmingly positive. Dr. Maccioli and I are scheduled to present our final report and recommendations to Dr. Litwiller and other prominent ASA leaders in the near future. The timing is such that it may be possible to introduce appropriate resolutions to the ASA House of Delegates at our 2004 Annual Meeting on October 22 in Las Vegas, Nevada. My final "President's Message" will detail the results of our deliberations and, hopefully, the response of ASA's leadership.

Fine-Tuning Training

Among the changes our task force will likely recommend is an alteration in the overall curriculum for anesthesiology training programs. It seems, however, that those charged with providing the blueprint for how and what future generations of anesthesiologists will learn have begun already. The Residency Review Committee for Anesthesiology has initiated steps to modify critical care medicine training within the anesthesiology curriculum. In the process, it appears that six months of critical care will be required. This represents an increase from the current two-month requirement and is more consistent with the approach taken in Canada, Europe, South America and Australasia. The details of the differences between U.S. practices and practices in the rest of the world are detailed nicely by Heidi B. Kummer, M.D., Ph.D., ASCCA board member and chair of the Committee on Outreach, in the previously mentioned February 2004 *ASA NEWSLETTER*. That issue also contains a blueprint for improving the length, scope and quality of anesthesiology residents' exposure to the intensive care unit and may provide the fundamentals for additional recommendations from the task force regarding the educational requirements for anesthesiology residency programs. ASCCA Past President Robert N. Sladen, M.B., Ch.B., has advocated this approach for quite some time, and the details of his plan represent an ideal way to begin the process.

Another sign of changing attitudes toward critical care can be found in some initial changes made to the ASA Annual Meeting format. This year, for the first time, ASA plans two subspecialty tracks within the meeting.

Things really have changed, but more is needed. I encourage each of you to add what you can and recruit others who can help. This is a volunteer organization, and it only works with the support and effort of you, the volunteers.

On the Right Track

Another sign of changing attitudes toward critical care can be found in some initial changes made to the ASA Annual Meeting format. This year, for the first time, ASA plans two subspecialty tracks within the meeting. One track will emphasize critical care medicine and include panels, Refresher Course Lectures, point-counterpoint debates, scientific sessions and a plenary lecture. (The other track will focus on obstetrical anesthesia.) The critical care track will take place on Saturday, October 23, and Sunday, October 24, 2004, and will provide an outstanding extension of our own meeting to be held on Friday, October 22. Details can be found in a short article by Dr. Cohen in the February 2004 *ASA NEWSLETTER*. The program is very exciting. In fact, those who have worked on reformatting the ASA Annual Meeting are so pleased with the educational programs that the track will be included in the 2005 meeting as well. (Can you smell the difference?) Please make a point of attending the ASCCA and the ASA annual meetings; they will be educationally worthwhile, and it is important that we have a large turnout and enthusiastic support.

Reimbursement

Yet another area of focus for the task force involves reimbursement for critical care services. This is a contentious area that has required the attention of practitioners from across the critical care spectrum. We seek to utilize ASA contacts to lobby for a more reasonable reimbursement methodology. The response from ASA has been quite positive. Dr. Dorman and Dr. Cohen have been intimately involved in this process for a number of years and will spearhead our efforts to involve ASA.

Specialty Melting Pot

An emphasis on critical care medicine has included many anesthesiologists from other subspecialties. Cardiac anesthesiologists have become more involved in the postoperative care of their patients; the same is true for neuroanesthesiologists. Pediatric anesthesiologists have long been intimately involved in the care of critically ill children. Many of these practitioners are seeking an educational and political "home" where their needs and concerns can be voiced and acted upon. ASCCA hopes to provide this, and our Committee on Membership, chaired by Eugene Y. Cheng, M.D., is making a concerted effort to attract these individuals. For those of you who practice in these areas, please help to recruit your colleagues and aid the ASCCA leadership in defining what issues are

Continued on page 11

ASCCA Annual Meeting

October 22, 2004

Las Vegas, Nevada

7 a.m. *Registration and Continental Breakfast*

7:55 a.m. *Welcome*
William E. Hurford, M.D.
Michael F. O'Connor, M.D.

Scientific Session

8 a.m. *Oral Abstracts*
Moderator:
Michael H. Wall, M.D.
Scientific Paper Chair

8 a.m.	Abstract 1
8:15 a.m.	Abstract 2
8:30 a.m.	Abstract 3
8:45 a.m.	Abstract 4

9 a.m. *Young Investigator Award*
Presented by:
Michael H. Wall, M.D.
Scientific Paper Chair

9:30 a.m. *Lifetime Achievement Award Presentation and Lecture*
Presenter: Neal H. Cohen, M.D.

10:20 a.m. *Coffee Break and Poster Viewing*

Leadership Session

10:40 a.m. *Introduction of Research Award*
Presenter: Robert N. Sladen, M.B.

10:50 a.m. *Regional Effects of Alveolar Recruiting Strategies in Acute Lung Injury*
Guido Musch, M.D.

11:20 a.m. *Joint ASCCA/SCCM Anesthesia Section "Burchardi Award"*
Presenter: Heidi B. Kummer, M.D., Ph.D.
Recipient: Douglas B. Coursin, M.D.

11:30 a.m. *Address by ASA President-Elect*
Eugene P. Sinclair, M.D.

12 noon Luncheon (sponsored by Hospira)
Epidemiology and Pathophysiology of Delirium
Gerald A. Maccioli, M.D.

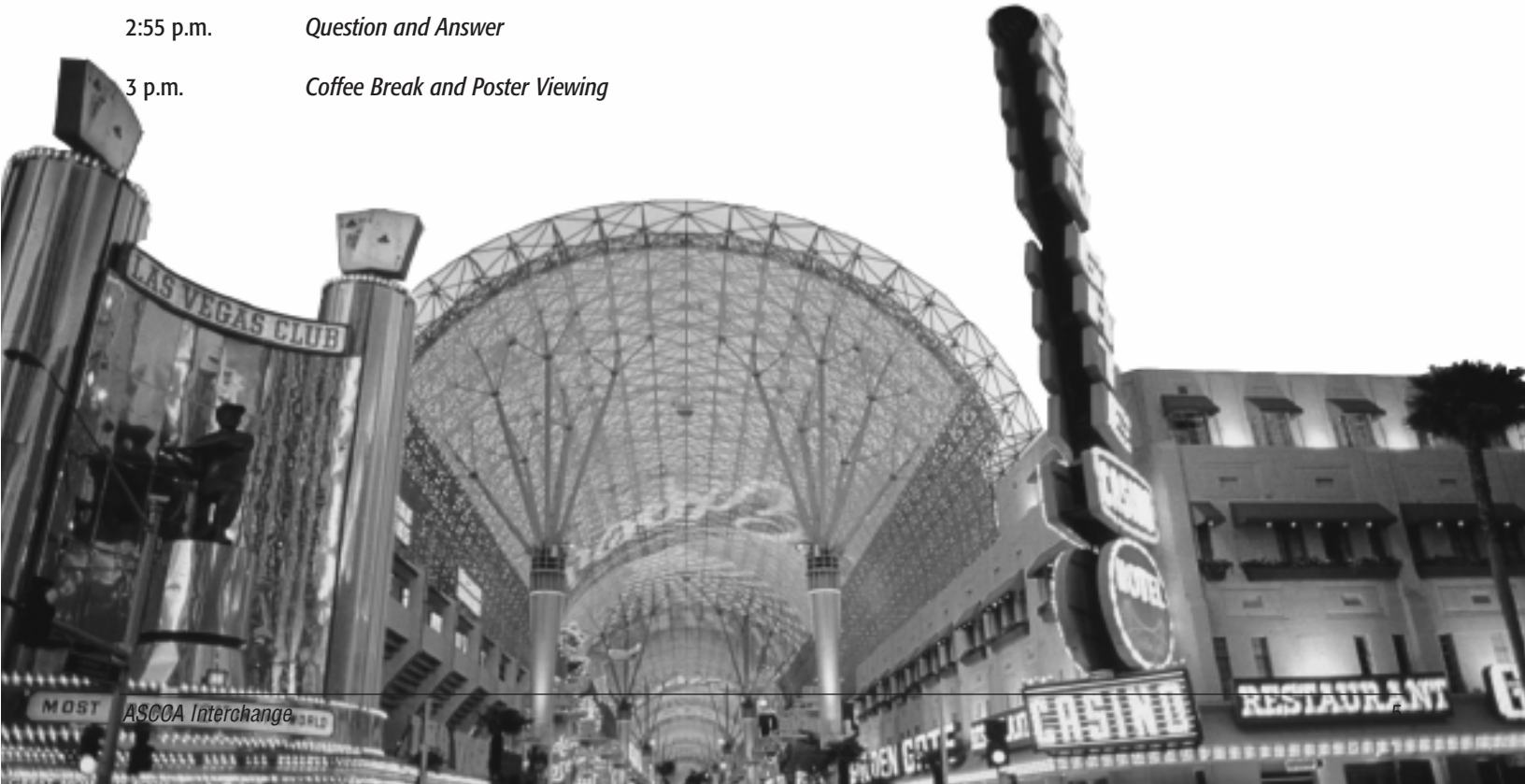
Prevention and Treatment of Delirium
Robert N. Sladen, M.B.

State of the Art

1 p.m.	*Tight Glycemic Control* Aristides P. Koutrouvelis, M.D.
1:15 p.m.	*Question and Answer*
1:20 p.m.	*The PA Catheter: Evidence Based?* Avery Tung, M.D.
1:35 p.m.	*Question and Answer*
1:40 p.m.	*Replacement Doses of Steroids in Sepsis: Who? When?* Joel B. Zivot, M.D.
1:55 p.m.	*Question and Answer*
2 p.m.	*Intentional Hypothermia Following In-Hospital CPR: What Should We Be Doing?* Brenda G. Fahy, M.D.
2:15 p.m.	*Question and Answer*
2:20 p.m.	*Protein C - Who Should Get It? Who Shouldn't?* Michael S. Avidan, M.B.,B.Ch.
2:35 p.m.	*Question and Answer*
2:40 p.m.	*Beta Blockers – How Do They Really Work?* Andrew J. Patterson, M.D.
2:55 p.m.	*Question and Answer*
3 p.m.	*Coffee Break and Poster Viewing*

Future Shocks Alternatively

3:20 p.m.	*Private Practice Critical Care by Anesthesiologists* Gerald A. Maccioli, M.D.
3:50 p.m.	*Ventricular Assist Devices* Andrew D. Rosenberg, M.D.
4:20 p.m.	*Infusion Devices and Safety* Mark E. Nunnally, M.D.
4:50 p.m.	*Reacting to Accidents in the ICU: Trying to Learn While Trying to Recover* Richard I. Cook, M.D.
5:20 p.m.	*ASCCA Business Meeting*
7 p.m.	*Reception*



Total Enteral Nutrition in the Critically Ill: A Review of the Controversies

Robert M. Pousman, D.O.,
Assistant Director of Critical Care Services
Assistant Professor
Department of Anesthesiology
Division of Critical Care Medicine
Vanderbilt University Medical Center
Nashville, Tennessee

Critical illness and injury invoke enormous physiologic stress upon the patient. Cuthbertson described the metabolic response as biphasic involving an early, short-lived hypometabolic ebb phase followed by a prolonged hypermetabolic flow phase.[1] Homeostasis is restored if the initial injury or insult is short-lived and uncomplicated by infection. A systemic inflammatory response ensues, however, and involves unrelated organs from the initial site of insult. This prolonged maladaptive response, when left uncorrected, renders the patient at risk of infectious morbidity that may run the spectrum from multiple-organ dysfunction syndrome (MODS) to multiple-organ failure (MOF) to death.[2]

This entity is known as the systemic inflammatory response syndrome (SIRS). A component of the response to this stress is catabolism promoted in part by inflammatory mediators such as the cytokines tumor necrosing factor (TNF), interleukin-1 (IL-1) and interleukin-6 (IL-6), which act as immunomodulators and hormonal stimulators that cause cachexia, anorexia, pyrexia, proteolysis and endothelial liberation of nitric oxide, thus causing hypotension via vasodilatory effects on vascular smooth muscle.[3-6]

Other substances liberated during inflammatory stress are acute-phase reactants, which are generated by the liver and include C-reactive protein (CRP), procalcitonin (PCT), fibronectin, fibrinogen, transferrin, 1-acid glycoprotein, ceruloplasmin, alpha1-proteinase inhibitor and alpha1-antitrypsin.[3,4] They act as opsonins, protease inhibitors, transporters and hemostatic compounds. The reticuloendothelial system (RES), which is responsible primarily for opsonization of bacteria, is depressed and is thought to be due in large part by a decrease in fibronectin initially after injury/inflammation. Impaired function of the RES plays a role in subsequent development of infection.

To counter-balance the inciting inflammatory response, the body responds with an anti-inflammatory cascade. Agents participating in this anti-inflammatory response identified include IL-4, IL-10 and IL-13. These interleukins inhibit monocyte, B-cell and T-cell function and downregulate proinflammatory mediators such as IL-1, IL-6 and IL-8 as well as TNF.[3,7] This equally opposite response to the proinflammatory event can suppress immune function and swing the continuum toward anergy, which occurs as a result of the concurrent dichotomous response that Bone termed "compensatory anti-inflammatory response syndrome" (CARS).[7]

Critical illness can provoke a heterogeneous reaction to the stress with some patients displaying an inappropriate hypometabolic response where generally a hypermetabolic response is expected.[5] A definitive hypometabolic response may be a predictor of mortality. The typical hypermetabolic stressed state such as in SIRS alters nutrient metabolism and utilization. Carbohydrates and fats are utilized initially as the primary substrate for energy but last for only 24 to 48 hours despite increased gluconeogenesis. Depending on the degree and duration of stress, proteins are utilized to provide amino acids for healing wounds, immune function and acute-phase proteins. Protein catabolism primarily from skeletal muscle produces a negative nitrogen balance. The liver redirects protein synthesis from the formation of visceral stores to production of acute-phase proteins and true protein catabolism/malnutrition ensues.

Robert M. Pousman, D.O.

While the majority of the attention is directed at identifying, correcting and treating the source of the ongoing inflammation/infection, much interest also has been focused on providing adequate nutrition to critically ill patients and recognizing those who will need and benefit from nutritional supplementation during this period of stress. Strong evidence exists that total enteral nutrition (TEN) is beneficial and that the gut should be the primary means utilized in providing caloric needs, when feasible.[8-13]

The gut has been suggested to be a major organ contributing to infectious morbidity and MOF. Villous integrity is disrupted during low-flow states, and with reperfusion, mucosa is further disrupted allowing bacterial translocation across the bloodstream; moreover, the involution of gut-associated lymphoid tissue

While the majority of the attention is directed at identifying, correcting and treating the source of the ongoing inflammation/infection, much interest also has been focused on providing adequate nutrition to critically ill patients and recognizing those who will need and benefit from nutritional supplementation during this period of stress.

(GALT) that occurs may have a more profound effect on morbidity. Immunologic defense is provided by the GALT and is reportedly responsible for up to 70 percent of the body's total immunity.[10] The gut also hosts neutrophils and is capable of priming them after a primary inflammatory or ischemic event to respond systemically to a second insult. These neutrophils act as chemoattractants and promote extraintestinal organ dysfunction.[14] Delivering nutrition by way of the gut has been shown clinically and experimentally to attenuate the inflammatory response and protect gut mucosal integrity.[11-13,15]

Indeed, in critically ill/injured trauma patients, benefits have been shown not only in utilizing TEN but also in its early implementation. Compared to parenteral nutrition, TEN may reduce inflammatory cytokines and improve immune function, promoting fewer septic complications and improved wound healing.[9,10,12,13] These benefits may be greatest in the more severely injured patients. Reproduction of these benefits, however, has not been as robust in well-nourished or mildly malnourished surgical or medical patients.[16,17] Nonetheless a recent study in medical intensive care unit patients showed a higher incidence of catheter-related bloodstream infections in patients receiving < 25 percent of their recommended caloric goal.[18] Therefore identifying those patients who will benefit most from aggressive nutritional support is paramount.

In general a consensus exists that, when feasible, TEN is the preferred method to provide nutrition; however, obstacles in reaching the nutritional goal for a patient still remain. TEN has been delivered safely in patients previously considered to have contraindications, e.g., patients on vasopressor or receiving prone ventilation.[19,20] Although generally considered safe, TEN can create devastating complications, such as nonocclusive bowel necrosis, when the potential for such complications is not adequately appreciated.[21]

A lack of consensus with regard to such variables as acceptable gastric residual volume (GRV), how one defines intolerance to feeding, the best route (i.e., gastric or small-bowel feedings) to deliver nutrition and discontinuation during or prior to certain routine intensive care unit (ICU) or operative procedures hinders the rapidity at reaching the caloric goal and maintenance of that goal.

Measuring and monitoring response to therapy ranges from physical anthropometric evaluation such as triceps skin-fold measurement to hand-grip strength to laboratory assessments of serum albumin, prealbumin, transferrin and retinol binding protein and nitrogen balance via urinary excretion of urea.

With regard to GRV, common thresholds to define intolerance and withhold further TEN administration have been between 100 ml to 250 ml. Little, if any, evidence supports this practice, which is a major impediment to reaching and maintaining nutritional goals of critically ill patients. The rationale is that when a large volume of fluid is contained in the stomach, it will "overflow" and place the patient at risk of aspiration. Physiologically this is not valid; such a rationale assumes that GRVs accurately reflect the true volume in the stomach but neglects the usual production of saliva and gastric secretions as well as gastric function. The arbitrary use of gastric residual volumes has been found to be a major impediment to advancement of TEN in the critically ill.[22-25] While aspiration of gastric contents is certainly a valid concern and is considered a major etiology in the development of ventilator-associated pneumonia (VAP), identifying those patients most at risk (oversedated patients or those with head injuries) while providing protective measures and maintaining vigilance is most important.

The route of TEN, gastric versus small bowel feedings, is a frequent impediment to patients reaching their nutritional goals. Patients fed intragastrically will frequently achieve their goals more quickly compared to those patients fed into the small bowel. Clearly one of the most common reasons that intragastic-fed patients do not maintain their nutritional goals is the cessation of feeding due to GRVs.[23,26,27] The argument for utilizing small-bowel feedings frequently parallels the concern with regard to "high" GRVs, namely a perceived higher risk of aspiration. This risk has not been proven in the literature; instead, identification of those patients who will benefit from small-bowel feeds, such as severe pancreatitis, diabetic gastroparesis or post-gastric resection, is preferable. Providing gastric feeding to patients with a propensity for intolerance (such as in burn patients or those with head injuries) has been performed with no increase in complications plus improvements not only in caloric delivery but in outcomes realized.[28,29]

Critically ill ICU patients, especially trauma/burn/surgical patients, frequently undergo procedures either at the bedside or in the operating suite. A common practice that also hinders the delivery of adequate nutrition is cessation of TEN for a standard fasting interval as used in elective surgical cases, namely an eight-hour fast. When small-bowel feeds are utilized and the patient has a "protected" airway (an endotracheal or tracheostomy tube in place), the necessity of this practice is questionable. When gastric feeds are being delivered, the risk of aspiration may be greater, but an eight-hour fast may still not be indicated. Invariably procedures are delayed or postponed that leave the patient without nutrition for far greater than the scheduled fast. Again, as noted above, TEN has been safely administered to burn patients during operative procedures with a resulting significant reduction in caloric deficit when compared to unfed controls who underwent standard fasts.

Recent interest in specialty formulas that provide additives such as arginine, glutamine

Continued on page 8

Total Enteral Nutrition in the Critically Ill: A Review of the Controversies

Continued from page 7

and omega-3 fatty acids — so-called immune-enhancing formulas (IEFs) — has created greater confusion to the provision of TEN. Indeed this topic warrants a separate discussion unto itself; a brief overview will be discussed, but recent reviews can be found elsewhere.[30-33] The broad consensus is that IEF may reduce infectious morbidity, decrease hospital length of stay, reduce ventilator days and possibly reduce mortality in a select patient population.[34] The subsets of patients who appear to benefit the most from IEFs are those with injury severity scores greater than 17, burn patients and major head and neck or gastrointestinal surgical patients. Providing these formulas, specifically arginine, to overtly septic patients has been shown to increase mortality as the inducible form of nitric oxide is expressed and worsens shock.[35]

Another controversy that lacks consensus concerns the best method to estimate nutritional needs, monitor adequacy of delivery and measure response to nutritional therapy. Indirect calorimetry is utilized exclusively in some centers but is expensive and operator-dependent. Estimating caloric needs by way of established equations has been shown to generally correlate well with indirect calorimetry. Measuring and monitoring response to therapy ranges from physical anthropometric evaluation such as triceps skin-fold measurement to hand-grip strength to laboratory assessments of serum albumin, prealbumin, transferrin and retinol binding protein and nitrogen balance via urinary excretion of urea. Acute-phase protein measures such as albumin, prealbumin, transferrin and retinol binding protein frequently underestimate the adequacy of nutritional delivery and utilization as their synthesis is reduced during the acute phase of the inflammatory response.[4,6] Serum CRP and PCT trends have been utilized to assist in interpreting the degree of stress and ongoing inflammation as well as efficacy of therapy.[36-40] It is important to ascertain whether a decline in albumin or prealbumin reflects active inflammatory response or a need for additional protein.

Helping to establish a standard protocol of providing nutritional supplementation to critically ill patients may reduce the obstacles that hamper adequate delivery and may also reduce not only the complications of the underlying disease process but the cost of nutritional supplementation as well. By defining an acceptable practice and criteria that would warrant deviation from proposed guidelines, the obstacles that frequently impede nutrition delivery can be overcome. Existing literature demonstrates that the adoption and adherence to evidence-based enteral feeding guidelines improves caloric delivery, decreases hospital stay and improves outcomes such as mortality.[41-46]

Clinical studies that have examined the effects of TEN protocols in critically ill patients suggest that, compared to control (nonprotocol) patients, a greater amount of TEN volumes is delivered per day as well as a higher percentage of prescribed caloric intake. These studies attribute this discrepancy not only on standardizing the delivery of nutrition with definitive criteria of those patients most likely to benefit but also defining criteria in which TEN should be withheld and the inclusion of nursing and dietitian participation with regard to protocol compliance. A recent study by Martin et al.[44] showed that by adopting and implementing an algorithm for TEN delivery in critically ill patients in whom a statistically insignificant increase in caloric and protein energy was delivered, the primary outcomes of hospital mortality and hospital stay were significantly decreased. The authors attribute their results not only to improved delivery of TEN but also to improved patient care that resulted from proper physician and nurse education as well.

A recent study by Martin et al.[44] showed that by adopting and implementing an algorithm for TEN delivery in critically ill patients in whom a statistically insignificant increase in caloric and protein energy was delivered, the primary outcomes of hospital mortality and hospital stay were significantly decreased.

In conclusion, prolonged critical illness invokes a marked hypermetabolic response that can create counterproductive effects. The basis of care lies in reducing secondary inflammatory/infectious insults, identifying the source of ongoing inflammation and providing appropriate therapy. Nutritional support can augment the stress response, protect against infectious morbidity and possibly reduce morbidity, mortality and length of stay. Developing evidence-based protocols can assist with identifying patients at risk of malnutrition-related morbidity and TEN-associated complications. Protocols also may assist in ensuring that adequate caloric intake is provided and unwarranted cessation of nutrition is minimized.

References:

1. Cuthbertson D. Postshock metabolic response. *Lancet*. 1942; 1:433-437.
2. Sauaia A, Moore FA, Moore EE. Postinjury multiple-organ failure. In: Moore EE, Mattox KL, Feliciano DV, eds. *Trauma*. 4th ed. New York: McGraw-Hill; 2000:1427-1460.
3. Kim PK, Deutschman CS. Inflammatory responses and mediators. *Surg Clin N Am*. 2000; 80(3):885-894.
4. Martindale RG, Nishikawa R, Siepler JK. The metabolic response to stress and alterations in nutrient metabolism. In: Shikora SA, Schwaitzberg SD, eds. *Nutritional Considerations in the Intensive Care Unit — Science, Rationale and Practice*. Dubuque: Kendall/Hunt; 2002:11-20.

5. McClave SA. Understanding the metabolic response to critical illness: Factors that cause patients to deviate from the expected pattern of hypermetabolism. *New Horizons*. 1994; 2(2):139-146.
6. Kudsk KA. Nutritional support. In: Moore EE, Mattox KL, Feliciano DV, eds. *Trauma*. 4th ed. New York: McGraw-Hill; 2000:1369-1407.
7. Bone RC. Sir Issac Newton, sepsis, SIRS and CARS. *Crit Care Med*. 1996; 24(7):1125-1128.
8. Windsor AC, Kanwar S, Li AG, et al. Compared with parenteral nutrition, enteral feeding attenuates the acute phase response and improves disease severity in acute pancreatitis. *Gut*. 1998; 42(3):431-435.
9. Kudsk KA, Croce MA, Fabian TC, et al. Enteral versus parenteral feeding. Effects on septic morbidity after blunt and penetrating abdominal trauma. *Ann Surg*. 1992; 215(5):503-511; discussion 511-513.
10. Kudsk KA. *Parenteral vs. Enteral Nutrition*. Dubuque: Kendall/Hunt; 2002.
11. Li J, Gocinski B, Dent D, et al. Effects of parenteral and enteral nutrition on gut-associated lymphoid tissue. *Journal of Trauma Injury, Infection, and Critical Care*. 1995; 39(1):44-52.
12. Moore FA, Feliciano DV, Andrassy RJ, et al. Early enteral feeding, compared with parenteral, reduces postoperative septic complications. The results of a meta-analysis. *Ann Surg*. 1992; 216(2):172-183.
13. Moore FA, Moore EE, Jones TN, et al. TEN versus TPN following major abdominal trauma reduced septic morbidity. *J Trauma*. 1989; 29:916-923.
14. Moore EE, Moore FA, Franciose RJ, et al. The postischemic gut serves as a priming bed for circulating neutrophils that provoke multiple organ failure. *J Trauma*. 1994; 37(6):881-887.
15. King BK, Li J, Kudsk KA. A temporal study of TPN-induced changes in gut-associated lymphoid tissue and mucosal immunity. *Arch Surg*. 1997; 132(12):1303-1309.
16. The Veteran Affairs Total Parenteral Nutrition Cooperative Study Group. Perioperative total parenteral nutrition in surgical patients. *N Engl J Med*. 1991; 325:525-532.
17. Brennan MF, Pisters PW, Posner M, et al. A prospective randomized trial of total parenteral nutrition after major pancreatic resection for malignancy. *Ann Surg*. 1994; 220(4):436-441; discussion 441-444.
18. Rubinson L, Diette GB, Song X, et al. Low caloric intake is associated with nosocomial bloodstream infections in patients in the medical intensive care unit. *Crit Care Med*. 2004; 32(2):350-357.
19. McClave SA. Feeding the hypotensive patient: Does enteral feeding precipitate or protect against ischemic bowel? *Nutr Clin Pract*. 2003; 18(4):279-284.
20. Zaloga GP RP, Marik P. Feeding the hemodynamically unstable patient: A critical evaluation of the evidence. *Nutr Clin Pract*. 2003; 18(4):285-293.
21. Marvin RG, McKinley BA, McQuiggan M, et al. Nonocclusive bowel necrosis occurring in critically ill trauma patients receiving enteral nutrition manifests no reliable clinical signs for early detection. *Am J Surg*. 2000; 179(1):7-12.
22. McClave SA, Snider HL, Lowen CC, et al. Use of residual volume as a marker for enteral feeding intolerance: Prospective blinded comparison with physical examination and radiographic findings. *J Parenter Enteral Nutr*. 1992;16(2):99-105.
23. McClave SA, Sexton LK, Spain DA, et al. Enteral tube feeding in the intensive care unit: Factors impeding adequate delivery. *Crit Care Med*. 1999; 27(7):1252-1256.
24. McClave SA, Snider HL. Clinical use of gastric residual volumes as a monitor for patients on enteral tube feeding. *J Parenter Enteral Nutr*. 2002; 26(6 suppl):S43-48; discussion S49-50.
25. Mentec H, Dupont H, Bocchetti M, et al. Upper digestive intolerance during enteral nutrition in critically ill patients: Frequency, risk factors, and complications. *Crit Care Med*. 2001; 29(10):1955-1961.
26. Heyland DK, Konopad E, Alberda C, et al. How well do critically ill patients tolerate early, intragastric enteral feeding? Results of a prospective, multicenter trial. *Nutr Clin Pract*. 1999; 14(1):23-28.
27. Heyland DK, Drover JW, Dhaliwal R, Greenwood J. Optimizing the benefits and minimizing the risks of enteral nutrition in the critically ill: Role of small bowel feeding. *J Parenter Enteral Nutr*. 2002; 26(6 suppl):S51-55; discussion S56-57.
28. Taylor SJ, Fettes SB, Jewkes C, Nelson RJ. Prospective, randomized, controlled trial to determine the effect of early enhanced enteral nutrition on clinical outcome in mechanically ventilated patients suffering head injury. *Crit Care Med*. 1999; 27(11):2525-2531.
29. Jenkins ME, Gottschlich MM, Warden GD. Enteral feeding during operative procedures in thermal injuries. *J Burn Care Rehabil*. 1994; 15(2):199-205.
30. Beale RJ, Bryg DJ, Bihari DJ. Immunonutrition in the critically ill: A systematic review of clinical outcome. *Crit Care Med*. 1999; 27(12):2799-2805.
31. Heys SD, Walker LG, Smith I, Eremin O. Enteral nutritional supplementation with key nutrients in patients with critical illness and cancer: A meta-analysis of randomized controlled clinical trials. *Ann Surg*. 1999; 229(4):467-477.
32. Heyland DK, Novak F, Drover JW, et al. Should immunonutrition become routine in critically ill patients? A systematic review of the evidence. *JAMA*. 2001; 286(8):944-953.
33. McCowen KC, Bistrian BR. Immunonutrition: Problematic or problem solving? *Am J Clin Nutr*. 2003; 77(4):764-770.
34. Proceedings from summit on immune-enhancing enteral therapy. *J Parenter Enteral Nutr*. 2001; 25(suppl):S61-S63.
35. Bansal V, Ochoa JB. Arginine availability, arginase, and the immune response. *Curr Opin Clin Nutr Metab Care*. 2003; 6(2):223-228.
36. Wanner GA, Keel M, Steckholzer U, et al. Relationship between procalcitonin plasma levels and severity of injury, sepsis, organ failure, and mortality in injured patients. *Crit Care Med*. 2000; 28(4):950-957.
37. Luis DD, Izaola O, Cuellar L, et al. Effect of c-reactive protein and interleukin blood levels in postsurgery arginine-enhanced enteral nutrition in head and neck cancer patients. *Eur J Clin Nutr*. 2003; 57(1):96-99.
38. Braga M, Gianatti L, Cestari A, et al. Gut

Continued on page 10

Total Enteral Nutrition in the Critically Ill: A Review of the Controversies

Continued from page 9

function and immune and inflammatory responses in patients perioperatively fed with supplemented enteral formulas. *Arch Surg*. 1996; 131(12):1257-1264.
39. Reinhart K, Karzai W, Meisner M. Procalcitonin as a marker of the systemic inflammatory response to infection. *Intensive Care Med*. 2000; 26(9):1193-1200.
40. Brown RO, Alexander E, Jr, Hanes SD, et al. Procalcitonin and enteral nutrition tolerance in critically ill patients. *J Parenter Enteral Nutr*. 2003; 27(1):84-88.
41. Adam S, Batson S. A study of problems associated with the delivery of enteral feed in critically ill patients in five ICUs in the UK. *Intensive Care Med*. 1997; 23(3):261-266.
42. Heyland D, Cook DJ, Winder B, et al. Enteral nutrition in the critically ill patient: A prospective survey. *Crit Care Med*. 1995; 23(6):1055-1060.
43. Heyland DK, Dhaliwal R, Drover JW, et al. Canadian clinical practice guidelines for nutrition support in mechanically ventilated, critically ill adult patients. *J Parenter Enteral Nutr*. 2003; 27(5):355-373.
44. Martin CM, Heyland DK, Morrison T, Sibbald WJ. Multicenter, cluster-randomized clinical trial of algorithms for critical-care enteral and parenteral therapy. *Can Med Assoc J*. 2004; 170(2):197-204.
45. Spain DA, McClave SA, Sexton LK, et al. Infusion protocol improves delivery of enteral tube feeding in the critical care unit. *J Parenter Enteral Nutr*. 1999; 23(5):288-292.
46. Barr J, Hecht M, Flavin KE, et al. Outcomes in critically ill patients before and after the implementation of an evidence-based nutritional management protocol. *Chest*. 2004; 125:1446-1457.

Tomorrow

Continued from page 2

itself. Now many of these individuals barely have the time to help their departments sort through the mountains of paper work, federal regulations, third-party payers and negotiations with the hospital administration, let alone rescue a health care system from its own suicide. We have created a monster that we no longer can control.

It is rare for me to attend a hospital-based meeting where patient care is directly addressed. There are many discussions about quality management, budgetary constraints, plans for expansion and consolidation of services; but there is little, if any, discussion about direct patient care. It is evident that we have become professionals at "working a health care system," not professionals working *within* a health care system. We must at some point pull ourselves away from the regulations, the evaluations, the proposals and the objectives and find those whom we are here to serve: our patients.

It is rare for me to attend a hospital-based meeting where patient care is directly addressed.

When working with new residents, I am reminded of their focus on the patient. It is with remorse that I see this primary concern slowly overshadowed by a need to master completion of paperwork, access online technology and comply with ill-conceived regulations from outside agencies. In short, they become more and more a distraction from patient care. It is inevitable, but nonetheless disturbing.

My hope remains, however, that somewhere within this group of eager, new anesthesiologists-to-be, there is an individual who will possess the insight and judgment to help fix a health care system gone awry, and with this thought, I am reminded of the importance of being open and honest with those who are here to learn from us. It is important to let them see our attributes and flaws so that they can realize the limitations of the current health care system. Today's residents remain our hope of a better health care system for tomorrow.

AMERICAN SOCIETY OF
CRITICAL CARE
ANESTHESIOLOGISTS

520 N. Northwest Highway
Park Ridge, IL 60068-2573

Something's Changed

Continued from page 3

most important to these colleagues and ways in which ASCCA can help.

Hard Science

Critical care anesthesiologists continue to make important contributions to medical knowledge, both in basic science research and clinical care. ASCCA has proudly awarded the first ASCCA/FAER/Abbott Research Fellowship Award to Guido Musch, M.D., of Massachusetts General Hospital. We are grateful to Alan D. Sessler, M.D., President of the Foundation for Anesthesia Education and Research (FAER); ASCCA Board of Directors member Jeffrey R. Balser, M.D., Ph.D.; Yvonne Harter of Abbott Laboratories; and particularly ASCCA past president Dr. Sladen for their roles in making this and future grants possible. Others continue to make outstanding contributions to research; such work has been encouraged by the support of the journal *Anesthesiology*. This year the journal will sponsor a session at the ASA Annual Meeting that will focus on research in critical care. Details on the individuals responsible for the ongoing success and those who hold the key to the future of critical care can be found in the February 2004 *ASA NEWSLETTER* <www.ASAhq.org/Newsletters/2004/02-04/TOC02_04.html>.

Moving on Up

What once was an academic subspecialty appears to be moving into the mainstream. More and more of our members practice outside of the "ivory tower." With the advent of the Leapfrog Group's recommendations and as a result of landmark investigations by Sean M. Berenholtz, M.D., Peter J. Pronovost, M.D., Ph.D., and Dr. Dorman, all at Johns Hopkins University, the value of intensivists in any practice setting has become clear. We wish to provide these individuals with a forum for discussion and a blueprint for practice growth. Therefore I have charged Aryeh Shender, M.D., to head a subcommittee dedicated to determining the interests of private practitioners, devising strategies by which ASCCA can aid these dedicated intensivists and identifying approaches by which the numbers of anesthesiologist/intensivists in the private setting can be increased. One approach is outlined by Dr. Maccioli and Dr. Cohen in the February 2004 *ASA NEWSLETTER*.

Things really have changed, but more is needed. I encourage each of you to add what you can and recruit others who can help. This is a volunteer organization, and it only works with the support and effort of you, the volunteers. We have made progress, but in this case, surgical thinking holds — more is better. We need to take advantage of this unique opportunity now, while we have the chance. Something has changed. Help to make even more things change. ✧