EVIDENCE-BASED INFORMATION FOR HOSPITALISTS, INTENSIVISTS, AND ACUTE CARE CLINICIANS

HOSPITAL MEDICINE ALERT

AHC Media LLC Home Page—www.ahcpub.com

CME for Physicians—www.cmeweb.com

AHC Media LLC

INSIDE

Why Are We Giving Our Patients Blood?

ABSTRACT & COMMENTARY

By Andrew M. Luks, MD

Pulmonary and Critical Care Medicine, University of Washington, Seattle

Dr. Luks reports no financial relationship to this field of study.

This article originally appeared in the January 2008 issue of Critical Care Alert. It was edited by David J. Pierson, MD, and peer reviewed by William Thompson, MD. Dr. Pierson is Professor, Pulmonary and Critical Care Medicine, Harborview Medical Center, University of Washington, and Dr. Thompson is Staff Pulmonologist, VA Medical Center; Associate Professor of Medicine, University of Washington. Drs. Pierson and Thompson report no financial relationships relevant to this field of study.

Synopsis: *This retrospective analysis of data on patients with acute lung injury shows that transfusion of red blood cells in such patients is associated with increased in-hospital mortality, and that the risk is highest with transfusion of non-leukoreduced blood and transfusion following the onset of acute lung injury.*

Source: Netzer G, et al. Association of RBC transfusion with mortality in patients with acute lung injury. *Chest*. 2007;132:1116-1123.

MULTIPLE, RECENT STUDIES HAVE DEMONSTRATED THAT RED blood cell (RBC) transfusions may be deleterious to critically-ill patients, as they have been found to be associated with increased mortality following coronary artery bypass surgery, increased rates of ventilator-associated pneumonia, and worse outcomes in patients with burn injury and trauma. Netzer and colleagues sought to build on this literature and determine if RBC transfusion had an effect on in-hospital mortality in patients with acute lung injury (ALI). They performed a retrospective analysis on previously collected data from a cohort of 248 patients with ALI or acute respiratory distress syndrome (ARDS) at a single center between 1999 and 2002. All patients over the age of 13 admitted to the medical or surgical ICU at this center were enrolled if they met American European Consensus criteria for ALI/ARDS. They did not include patients with heart failure, respiratory diseases such as diffuse alveolar hemorrhage-mimicking ARDS, significant burns, or transplant recipients.

Financial Disclosure

Hospital Medicine Alert's physician editor, Kenneth P. Steinberg, MD, has no relevant financial relationship related to the material presented in this issue.

Hospital Medicine Alert's physician editor, Kenneth P. Steinberg, MD, selected and reviewed the articles contained within this issue on January 10, 2008.

VOLUME 2 • NUMBER 12 • FEBRUARY 2008 • PAGES 89-96

NOW AVAILABLE ONLINE
www.ahcmedia.com

RBC transfusion was the primary exposure variable, and this was evaluated dichotomously (any transfusion), as well as linearly (total number of RBC units transfused). The primary outcome was in-hospital mortality. Because mechanical ventilation strategies changed over the course of the study due to the results of the ARDSNet trial, and because the use of leukoreduced blood increased over the same time period, appropriate adjustments were made in the statistical analysis to account for these changes in practice. Netzer et al also investigated whether the timing of transfusion, relative to the onset of ALI, had any effect on mortality.

Overall mortality among the 248 patients analyzed in the study was 39%, consistent with that seen in the placebo group of the ARDSNet trial. Transfusion was associated with an increased risk of mortality. Transfusion of any RBC was associated with an unadjusted odds ratio (OR) for mortality of 2.90, a value that remained significant when adjusted for various factors such as age, gender, and APACHE III score. The mortality risk per unit of RBC transfused, adjusted for length of stay, was 1.06. Put in other terms, a patient who received 4 units of blood over the course of admission had a 24% increase in mortality risk. With regard to timing of the transfusions, transfusion of blood after the onset of ALI was associated with an OR of mortality of 1.13 per unit transfused, while administration prior to ALI onset was not a risk factor for mortality. The risk of mortality was higher, however, with non-leukoreduced blood; the OR for non-leukoreduced RBC per unit transfused was 1.14 compared to only 1.04 for the leukoreduced products. Finally, when adjusted for RBC transfusion, platelet administration was not associated with a higher risk of mortality.

COMMENTARY

The study by Netzer et al is yet another entry in a growing list of studies that leaves one wondering why it is that we are transfusing RBC in critically ill patients. Basic cardiopulmonary physiology teaches us that increasing a patient's hemoglobin levels improves oxygen delivery, a key goal in the critically ill. Yet, studies on a wide variety of critically ill patients, such as post-cardiac surgery, burn, trauma, and now ALI patients, demonstrates that RBC transfusion may have deleterious effects, such as increased mortality, increased ventilator-associated pneumonia, and decreased organ function. The etiology of these effects has not been definitively established, but may relate to transfusion-related amplification of lung injury or immunosuppressive effects.

It should be noted that there are some methodological issues of concern in the paper, such as the fact that Netzer et al were not able to separate cases of Transfusion-Related Acute Lung Injury (TRALI) from pure ALI, as well as the fact that it was a retrospective analysis, but in light of all the previous studies with similar conclusions, this paper's results warrant consideration.

The Transfusion Requirements in Critical Care (TRICC) trial[1] has previously demonstrated that using a restrictive transfusion strategy, in which patients are only transfused when their hemoglobin values fall below 7 mg/dL, is not associated with increased morbidity or mortality when compared to a more liberal transfusion threshold. Given the large number of studies noted above demonstrating potential deleterious effects of RBC transfusion, increased attention should be devoted to stricter transfusion practices along the lines of those used in this important, prospective, randomized trial. There are situations in which RBC transfusion is likely to be beneficial, warranting deviation from strict thresholds — acute coronary syndrome and early goal-directed therapy[2] are two good examples — but in general, the growing evidence now indicates that we need to be more careful about our use of RBC transfusions in the ICU. ■

References

1. Hebert PC, et al. A multicenter, randomized, controlled clinical trial of transfusion requirements in critical care. *N Engl J Med.* 1999;340:409-417.
2. Rivers E, et al. Early goal-directed therapy in the treatment of severe sepsis and septic shock. *N Engl J Med.* 2001;345:1368-1377.

Hospital Medicine Alert, ISSN 1931-9037, is published monthly by AHC Media LLC, 3525 Piedmont Rd., NE, Bldg. 6, Suite 400, Atlanta, GA 30305.

SENIOR VICE PRESIDENT/GROUP PUBLISHER: Brenda Mooney.
ASSOCIATE PUBLISHER: Lee Landenberger.
MANAGING EDITOR: Leslie Hamlin.
MARKETING PRODUCT MANAGER: Shawn DeMario.

GST Registration Number: R128870672.

Periodicals postage paid at Atlanta, GA.

POSTMASTER: Send address changes to ***Hospital Medicine Alert***, P.O. Box 740059, Atlanta, GA 30374.

Back issues: $42. Missing issues will be fulfilled by Customer Service free of charge when contacted within one month of the missing issue's date.

Subscriber Information

Customer Service: 1-800-688-2421
Customer Service E-Mail: customerservice@ahcmedia.com
Editorial E-Mail: leslie.hamlin@ahcmedia.com

Subscription Prices

United States
1 year with Free *AMA PRA Category 1 Credits™*: $249
Add $17.95 for shipping & handling.
(Student/Resident rate: $125).

Multiple Copies
Discounts are available for group subscriptions. For pricing information, please call Tria Kreutzer at (404) 262-5482.

Canada
Add GST and $30 shipping.

Elsewhere
Add $30 shipping.

Accreditation

AHC Media LLC is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education for physicians.

AHC Media LLC designates this educational activity for a maximum of *20 AMA PRA Category 1 Credits™*. Physicians should only claim credit commensurate with the extent of their participation in the activity.

This CME activity is intended for hospitalists, intensivists and acute care clinicians. It is in effect for 36 months from the date of the publication.

Questions & Comments

Please call **Leslie Hamlin**, Managing Editor, at (404) 262-5416 or e-mail at leslie.hamlin@ahcmedia.com between 8:30 a.m. and 4:30 p.m. ET, Monday-Friday.

Reversible Cerebral Vasoconstriction Syndrome

ABSTRACT & COMMENTARY

By John J. Caronna, MD
Vice-Chairman, Department of Neurology, Cornell University Medical Center; Professor of Clinical Neurology, NewYork-Presbyterian Hospital

Dr. Caronna reports no financial relationship relevant to this field of study. This article originally appeared in the January 2008 issue of Neurology Alert. It was edited by Matthew Fink, MD, and peer reviewed by M. Flint Beal, MD. Dr. Fink is Vice Chairman, Professor of Critical Care Neurology, NewYork-Presbyterian Hospital, and Dr. Beal is Professor and Chairman, Department of Neurology, Cornell University Medical College. Drs. Fink and Beal report no financial relationships relevant to this field of study.

Synopsis: *RCVS occurs in a variety of clinical settings, and should be considered and investigated in any person with sudden severe headache that is unexplained by other disorders.*

Source: Ducros A, et al. The clinical and radiological spectrum of reversible cerebral vasoconstriction syndrome. A prospective series of 67 patients. *Brain*. 2007;130:3091-3101.

Ducros and colleagues have reported the largest series of cases of the so-called "reversible cerebral vasoconstriction syndrome" (RCVS). RCVS is characterized by the association of severe or "thunderclap" headaches with or without neurological deficits, and a string of beads appearance of cerebral arteries that resolves spontaneously in 1-3 months. Ducros et al prospectively followed 67 consecutive patients, diagnosed at Lariboisiere Hospital in Paris, with angiographically confirmed RCVS. There were 43 women and 24 men, with a mean age of 42 years (range, 19-70 years). RCVS was spontaneous in one-third and secondary in two-thirds of patients. More than one-half of the patients (n = 37) reported the previous use of vasoactive substances (cocaine, cannabis, nasal decongestants, serotonin reuptake inhibitors, interferon, nicotine patches), which in some cases was combined with binge drinking. Five patients (12%) were just postpartum; only one had received bromocriptine after delivery to inhibit lactation.

Severe headache, by definition, was the presenting symptom in all patients, and was the only symptom of RCVS in 51 patients (76%). Sixty-three patients (94%) had multiple thunderclap headaches (mean number, 4.5; range, 2-18) that recurred over a mean period of 7.4 days (range, 1-21). In 16 patients with complications, cortical subarachnoid hemorrhage (SAH) (22%), intracerebral hemorrhage (ICH) (6%), seizures (3%), and reversible posterior leukoencephalopathy (RPLS) (9%) were early complications that occurred mainly during the first week. Ischemic events, including TIA (16%) and stroke (4%), occurred mainly during the second week.

Ducros et al, therefore, hypothesize that the different time course and evolution of thunderclap headaches, SAH, and strokes indicate that the vasospastic disorder starts in small distal arteries and progresses to involve medium and large arteries.

No relapses were observed during the mean follow-up of 16 months. Ducros et al concluded from their data that RCVS is more frequent than commonly thought, and is, more often than not, secondary to exposure to vasoactive substances.

COMMENTARY

RCVS is a term that encompasses a group of disorders sharing angiographic and clinical features, namely reversible segmental and multifocal vasoconstriction of cerebral arteries, and severe headaches with or without focal neurological deficits. The names for this syndrome, among many others, include: benign cerebral angiopathy or vasculitis,[1] Call-Fleming syndrome,[2] and thunderclap headache with reversible vasospasm.[3] The pathophysiology of RCVS is not understood, and the cause probably is multifactorial, given the numerous and heterogeneous precipitating events that may or may not be causally related to the syndrome.

In an editorial accompanying this report, van Gijn doubts that RCVS is a nosological entity because of the overlap with other syndromes such as migraine and the multitude of precipitating factors, as well as inter-observer variation in what constitutes arterial narrowing on cerebral angiograms.[4] Nevertheless, Ducros et al have done clinicians a great service by reporting their large series of RCVS patients in useful detail, thereby stimulating further research into this sometimes not-so-benign entity.

References

1. Snyder BD, McClelland RR. Isolated benign cerebral vasculitis. *Arch Neurol* 1978;35:612-614.
2. Call GK, et al. Reversible cerebral segmental vasoconstriction. *Stroke* 1988;19:1159-1170.
3. Dodick DW, et al. Thunderclap headache associated with reversible vasospasm and posterior leukoencephalopathy syndrome. *Cephalalgia* 2003;23:994-997.

4. van Gijn J. Cerebral vasoconstriction, headache and sometimes stroke: One syndrome or many? *Brain.* 2007;130(Pt 12):3060-3062.

Steroids for COPD Exacerbations: Oral or IV?

ABSTRACT & COMMENTARY

By David J. Pierson, MD

Synopsis: *This study of oral versus intravenous prednisolone in patients hospitalized with exacerbations of COPD showed no differences in any outcome variable between the two forms of administration.*

Source: de Jong YP, et al. Oral or IV prednisolone in the treatment of COPD exacerbations: A randomized, controlled, double-blind study. *Chest*. 2007;132:1741-1747.

De Jong and colleagues, in the Netherlands, conducted a prospective, randomized, double-blind, double-dummy, placebo-controlled, parallel-group clinical study of intravenous vs oral corticosteroids in the treatment of patients hospitalized because of an exacerbation of chronic obstructive pulmonary disease (COPD). All patients with known COPD who were admitted to a single institution during the 2-year study period were considered for inclusion. Patients were excluded if they had asthma, significant comorbidity, poor compliance, or previous study enrollment, and also if the exacerbation was severe, as defined by arterial pH 7.25 or less or PCO_2 70 mm Hg or more. Patients received 5 days of either intravenous or oral prednisolone 60 mg as the active medication; they also received nebulized ipratropium and albuterol 4 times daily and oral amoxicillin-clavulanate. Prednisolone was given orally to all patients after the first 5 days, with the daily dose tapered to zero over the next week.

Treatment failure, the primary outcome, was defined as death, admission to ICU, readmission for COPD, or intensification of pharmacological therapy during a 90-day follow-up. The study was designed as a non-inferiority trial, with sample sizes calculated to detect a difference of 15% in the primary outcome. Secondary outcomes included spirometry, length of hospital stay, health status, as measured by the St George's Respiratory Questionnaire, and a measure of health-related quality of life.

During the study period, 435 potentially eligible patients were admitted 581 times. After exclusions, 210 patients (48%) were included, 107 of whom received prednisolone intravenously and 103 of whom received it by mouth. Final data analysis included 99 and 94 of these patients, respectively. The patients were demographically and clinically similar: three-fourths were men, mean age 70 years, and mean FEV1 approximately 1.0 L (37% of predicted, or GOLD stage 3). Most of them had received oral corticosteroids in the month prior to admission, although only about 10% took them chronically. About 12% were on long-term oxygen therapy at home.

Mean duration of hospitalization was about 11 days; 7 patients died. Treatment failure, the primary study outcome, occurred in 62% of the patients who received steroids intravenously, as compared to 56% in those who received them orally, with no differences in early vs late treatment failure. No differences in secondary outcome variables — including changes in FEV1, health status, quality of life, or duration of hospitalization — achieved or approached statistical significance. De Jong et al concluded that orally-administered prednisolone is not inferior to its intravenous counterpart, and that the former is preferable because of its lower cost and greater convenience.

COMMENTARY

As confirmed by a Cochrane review of numerous published, placebo-controlled studies,[1] the systemic administration of corticosteroids to patients with COPD exacerbations hastens physiologic recovery, reduces morbidity, and improves other outcomes, both in ambulatory patients and those ill enough to be admitted to the hospital. In exacerbations, steroids also decrease the incidence of treatment failure and the likelihood of relapse in the succeeding 1 to 3 months. Thus, a short course of systemic steroids is the current standard of care for this condition.

Although once-daily prednisone (or prednisolone, as used in this study) is generally used in treating exacerbations in outpatients, it has been common practice to administer steroids intravenously when patients are sick enough to be admitted to the hospital — despite the fact that they are virtually 100% bioavailable when taken by mouth. Nearly all studies of corticosteroids in hospitalized COPD patients have used intravenous administration, and such patients typically receive the drug in 2 or 4 divided daily doses.

Hospitalization for exacerbations accounts for the largest component of health care costs for COPD. In

the United States, some 750,000 hospitalizations for COPD exacerbations occur each year. Thus, if the findings of this study are valid, oral rather than intravenous administration could have important implications in terms of the use of monetary and other resources.

This study's results agree with my own personal bias that hospitalized patients with COPD exacerbations can be treated as effectively with oral as with intravenous steroids, as long as they can take medication by mouth and are not actively vomiting. However, the present study falls short of being the final word on the subject. De Jong et al acknowledge that their own a priori criteria for the non-inferiority of oral administration were not met because of insufficient patient enrollment during the study period; they achieved only 74% power (vs the planned 80%) to assure non-inferiority of oral prednisolone. And they excluded patients with severe hypercapnia or acidemia, which is the population of greatest interest to intensivists. In addition, although there are no good data to suggest that higher steroid doses are more effective in this condition, the 60 mg daily prednisolone dose is lower than that initially used by many American clinicians in treating hospitalized patients.

As a side note, I know of no data supporting the use of inhaled steroids in COPD exacerbations. Given the lower overall potency of these agents, as compared to usual doses of systemically-given preparations for patients already receiving inhaled steroids prior to the exacerbation, it would make sense to discontinue them until the daily oral prednisone dose is reduced below about 15-20 mg. It has also become commonplace in some intensive care units to administer inhaled steroids to intubated patients who have COPD, whether exacerbated or not. To my knowledge, this practice, which is both expensive and time-consuming for staff, is supported by no evidence at all. Given the inefficiency of aerosol delivery via endotracheal tube (which reduces drug delivery by a factor of 5 to 10 in comparison with optimal oral technique), it is most unlikely that the administered agent reaches the patient's lungs in any biologically significant amount. ■

Reference

1. Wood-Baker R, et al. Oral corticosteroids for acute exacerbations of chronic obstructive pulmonary disease (Cochrane Review). The Cochrane Library. Oxford: Update Software 2003; Issue 3.

Use of Proton Pump Inhibitors in Nursing Facility Patients

ABSTRACT & COMMENTARY

By Malcolm Robinson, MD, FACP, FACG

Emeritus Clinical Professor of Medicine, University of Oklahoma College of Medicine, Oklahoma City

Dr. Robinson reports no financial relationship to this field of study.

This article originally appeared in the December 29, 2007 issue of Internal Medicine Alert. It was edited by Stephen Brunton, MD, and peer reviewed by Gerald Roberts, MD. Dr. Brunton is Clinical Professor, University of California, Irvine, and Dr. Roberts is Clinical Professor of Medicine, Albert Einstein College of Medicine. Dr. Brunton is a consultant for Sanofi-Aventis, Ortho-McNeil, McNeil, Abbott, Novo Norkisk, Eli Lilly, Endo, EXACT Sciences, and AstraZeneca, and serves on the speaker's bureau for McNeil, Sanofi-Aventis, and Ortho-McNeil. Dr. Roberts reports no financial relationship relevant to this field of study.

Synopsis: *Many nursing home patients are admitted with prescriptions for proton pump inhibitors or H2-receptor antagonists without any obvious indication.*

Source: Glew C, Rentler RJ. Use of proton pump inhibitors and other acid suppressive medications in newly admitted nursing facility patients. *J Am Med Dir Assoc*. 2007;8:607-609.

Hospitalized patients are often started on acid suppressive therapy for prophylaxis of stress ulceration and for other even less well-defined reasons. The authors of this paper note that inpatient stress ulcer prevention with acid suppression is rarely, if ever, indicated outside the intensive care setting, and they list several citations that corroborate the lack of evidence-based criteria for use of such therapy in a wide range of hospital settings. Worse yet, according to these authors and their review of pertinent literature, many patients who improperly receive PPIs and H2 receptor antagonists while hospitalized are also prescribed these drugs at discharge (including transfers to skilled nursing facilities). The present study involved a chart review of 98 admissions to a non-profit nursing home facility in Pennsylvania during the last half of 2006.

All available patient records for these admissions were reviewed, including hospital histories, physical examinations, medication lists, operative procedures, laboratory and radiological data, along with hospital discharge summaries. Appropriate indications for

acid suppression were defined (admittedly arbitrarily) as GERD, UGI bleeding, peptic ulcer disease, and empirical treatment for any other unexplained GI bleeding. Since no patient identifiers were collected, no institutional review board approval was sought. Included in the record review were 63 women and 35 men. Only 9% of patients were younger than 74. Of the 98 patients, 61% were admitted with transfer orders, including a PPI; 3 other patients were on an H2 receptor antagonist (total of 64.3% on acid suppression). Only 50% of patients had an appropriate supporting diagnosis for such therapy (mostly GERD), and 3.1% of patients with the diagnosis of GERD were on no acid suppression. The authors comment that many of the accepted diagnoses could have been entirely inactive or even incorrect, and they suggest that the overall rate of unnecessary acid suppression is probably much higher than 50%. They state that even very "safe" drugs like PPIs sometimes lead to adverse events such a headache, diarrhea, and abdominal pain in some patients — not to mention potential drug-drug interactions. It was concluded that discontinuation of acid suppression might be the most appropriate management for many if not most of these nursing home admissions.

COMMENTARY

Most physicians never see articles in journals like the *Journal of the American Medical Directors Association*, and they certainly don't seem to include the same level of peer review we see in most of the journals selected for *Hospital Medicine Alert*. The present study seems to exhibit a number of defects. It is quite small, and it could be argued that even this kind of chart review should have been submitted to an institutional review board for consideration. Furthermore, we all could verify that the discharge summaries and similar materials available for review at the time of nursing home admission may be grossly incomplete in terms of potentially meaningful historical detail. Nevertheless, it is the opinion of this reviewer that the conclusions of this brief report are fully justified. As they point out, the over-prescription of acid suppression in hospital settings and the continuation of such therapy post-discharge have been amply documented. It would be surprising if nursing home admissions did not share in the gross over-prescription of PPIs and H2RAs. These products have been heavily promoted, and they are perceived to be unusually safe. Physicians seem amazingly unconcerned with issues of cost containment, and many of them don't hesitate to prescribe expensive products (like PPIs) even for rather flimsy indications. Insurance companies and other payers would like to eliminate prescription of medications without some evidence-based supporting data. Physicians in general don't like challenges to their decisions, and they are particularly unwilling to provide detailed supportive data for every order written. Nevertheless, our society cannot afford endless increases in medical costs. If we don't regulate ourselves, it is certain that we will ultimately be subject to external regulation. Acid suppression probably is relatively safe, but it should only be prescribed for clear indications that can stand peer review. Unfortunately, once a patient has received a prescription for medication such as the acid suppressing agents, it is often unlikely that the prescription will be discontinued (particularly in the nursing home setting where physicians may have less historical connection with the patients under their care). ■

Should We Be Using Oral Decontamination with Ventilated Patients?

ABSTRACT & COMMENTARY

By Andrew M. Luks, MD

Synopsis: *This systematic review and meta-analysis demonstrates that oral decontamination with antiseptic preparations decreases the risk of ventilator-associated pneumonia but has no effect on mortality, duration of mechanical ventilation or length of stay in the ICU.*

Source: Chan EY, et al. Oral decontamination for prevention of pneumonia in mechanically ventilated adults: Systematic review and meta-analysis. *BMJ*. 2007;334:889.

PREVIOUS WORK DEMONSTRATES THAT SELECTIVE GUT decontamination decreases the risk of ventilator-associated pneumonia (VAP), but the practice has been limited by concerns about promoting antibiotic resistance. Oral decontamination alone has been proposed as an alternative, but the data on its efficacy have been mixed. Chan and colleagues conducted a

meta-analysis to further investigate this issue and to determine whether oral decontamination with antibiotic or antiseptic preparations decreases the risk of VAP.

Two reviewers searched the literature for published and unpublished randomized trials testing the effects of oral decontamination on the incidence of pneumonia and evaluated the quality of these trials. Studies were grouped into two broad categories including those that tested oral antibiotics vs placebo and those that tested oral antiseptics against placebo. No trials compared oral antibiotics with oral antiseptics or selective gut decontamination. The primary outcome measures in their meta-analysis were the incidence of VAP and mortality. They used each included study's definition of pneumonia rather than applying their own standard definition across the included studies. Secondary outcomes included the duration of mechanical ventilation and duration of stay in the intensive care unit.

Eleven trials including a total of 3242 patients were included in the meta-analysis. Four trials (1098 patients) examined oral antibiotics while the remaining seven trials (2144 patients) looked at oral antiseptic solutions. While the oral antibiotic solutions had no effect on the risk of VAP, oral antiseptic solution was, in fact, associated with decreased risk (RR = 0.56, 95% confidence interval 0.39-0.81). When the 11 trials were combined in a single analysis, the relative risk of pneumonia with oral decontamination was 0.61 (95% confidence interval 0.45-0.82) and the number needed to treat to prevent one case of pneumonia was 14 (95% confidence interval 10-31). Oral decontamination with either antibiotics or antiseptics had no effect on mortality, duration of mechanical ventilation or duration of stay in the intensive care unit.

COMMENTARY

Given the high morbidity and mortality associated with VAP, identifying effective strategies for preventing this complication should be a high priority. The study by Chan and colleagues suggests that oral decontamination with antiseptic solution may be effective for this purpose but does not provide strong enough evidence of benefit or low risk to justify widespread adoption of the practice.

There are several issues that prevent broad application of these results. First, there were important methodological differences between the trials included in the meta-analysis, such as the manner in which they defined VAP and the manner in which they did or did not control for important variables, which may make comparison between the included studies difficult. Second, although the meta-analysis examines a large number of patients and points to a benefit for oral antiseptic solutions, important clinical questions remain unanswered. For example, with the exception of one trial that examined the use of povidone iodine, the remaining trials used chlorhexidine, but in varying concentrations. As a result, we have no idea which is the best antiseptic preparation or its optimum concentration for use in decontamination.

More importantly, we still lack information about perhaps the most important question of all regarding this practice: what role, if any, will it have on rates of antibiotic resistance in the ICU? We may prevent VAP, but if we end up concurrently creating resistant bacteria that will make ICU care more problematic in the future, have we really done ourselves or our patients any favors? Until this issue is resolved, we should focus on other proven techniques for preventing VAP, such as elevating the head of the bed, that do not pose this risk. ■

CME Questions

13. Based on the study by Glew et al, approximately 64% of patients admitted to a skilled nursing facility were on acid suppression therapy. Based on prospectively defined criteria, what fraction of those patients were on that therapy without an indication?

a. < 5%
b. 10%
c. 50%
d. 90%

14. According to the study by de Jong et al, comparing oral to intravenous corticosteroids for the treatment of acute COPD exacerbations, which of the following findings was observed?

a. There was no significant difference between oral and intravenous corticosteroids in clinically relevant outcomes.
b. Intravenous corticosteroids led to more rapid recovery of lung function compared to oral corticosteroids.
c. Intravenous corticosteroids led to a shorter hospital length of stay compared to oral corticosteroids.
d. Oral corticosteroids led to a significantly higher rate of treatment failure than intravenous corticosteroids.

15. In the systematic review and meta-analysis by Chan et al, the only clinically relevant outcome associated with the use of oral decontamination of the gastrointestinal tract with antiseptic solution was:

a. improved ICU and in-hospital survival.
b. decreased ICU and hospital length of stay.
c. decreased duration of mechanical ventilation
d. decreased risk of ventilator-associated pneumonia.

Answers: 13. (c); 14. (a); 15. (d)

CME Objectives

The objectives of Hospital Medicine Alert are to:

• review pertinent safety, infection control, and quality improvement practices;

• discuss diagnosis and treatment of acute illness in the hospital setting; and

• review current data on diagnostic and therapeutic modalities for common inpatient problems. ■

CME Instructions

Physicians participate in this CME program by reading the issue, using the references for research, and studying the questions. Participants should select what they believe to be the correct answers, then refer to the answer key to test their knowledge. To clarify confusion on any questions answered incorrectly, consult the source material. ■