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# HOSPITAL MEDICINE ALERT

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## INSIDE



# Continuing Low-dose Aspirin with Peptic Ulcer Bleeding

ABSTRACT & COMMENTARY

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*Dr. Ferris reports no financial relationship to this field of study.*

*This article originally appeared in the January 29, 2009 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 Nordisk, Eli Lilly, Endo, EXACT Sciences, and AstraZeneca, and serves on the speaker's bureau for McNeil, Sanofi-Aventis, and Ortho McNeil, and Dr. Roberts reports no financial relationships relevant to this field of study.*

***Synopsis:*** *Recurrent peptic ulcer bleeding was increased in patients with known cardiovascular or cerebrovascular disease when daily low-dose aspirin was continued along with proton-pump inhibitors, but overall mortality was significantly less during the 8-week follow-up.*

**Source:** Sung JJ, et al. Continuation of low-dose aspirin in peptic ulcer bleeding. *Ann Intern Med* 2010;152:1-9.

After successful treatment of acute peptic ulcer bleeding with endoscopic control and intravenous pantoprazole, patients who had previously been taking low-dose aspirin for established cardiovascular or cerebrovascular disease were randomized to either continue it or receive placebo; all continued oral pantoprazole for eight weeks. The study, extended over three years, enrolled 156 patients of 246 identified with aspirin-related bleeding from a total of 3,412 treated for GI bleeding in a tertiary university endoscopy center.

Recurrent GI bleeding occurred in 10.3% (eight patients) of the aspirin group, compared to 5.4% (four patients) in the placebo group (a 50% increase), although this would have been a smaller increase if two additional patients in the placebo group who died from GI bleeding had not been excluded according to the strict study criteria. Transfusion rates were similar between the two groups, suggesting similar severity of bleeding episodes. All-

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cause mortality at 30 days was lower in the aspirin group at 1.3%, compared to 12.9% for the placebo group; mortality from cardiovascular, cerebrovascular, and GI complications was 1.3% for the aspirin group, compared to 10.3% for the placebo group. Within the eight-week follow-up, only one death occurred in the aspirin group from congestive heart failure; 10 deaths in the placebo group included five from cardiovascular events, three from GI complications, and two from pneumonia.

## ■ COMMENTARY

Despite the known benefits of prophylactic daily low-dose aspirin in cardiovascular and cerebrovascular disease, it does increase peptic ulcer bleeding, and its use requires a risk/benefit analysis between the different organ systems.[1] When patients develop acute peptic ulcer bleeding and the aspirin is stopped, clinicians are faced with the dilemma of whether to resume the aspirin prophylaxis, as well as when that should occur.

This small study supports resumption of low-dose aspirin as soon as the cardiovascular risks outweigh the GI risks. An accompanying editorial makes a case for resumption within seven days of the treated bleeding episode, along with continuation of proton-pump inhibitors for at least eight weeks, based on the observation that thrombotic events often occur as soon as 7-10 days after aspirin withdrawal.[2] This study was stopped after eight weeks and, thus, did not provide further information beyond that time.

Given the devastating consequences of acute thrombosis to the heart and brain, clinicians are left with limited alternatives to resuming low-dose aspirin even though we know it can cause life-threatening GI hemorrhage. Other antiplatelet agents have not been shown to be any better, and may possibly cause even more bleeding.[3] As always, we need to consider the "whole patient" and not focus on one part of the body while ignoring the other organs; current information suggests that the risks of bleeding from low-dose aspirin are less than the risks of thrombosis without it. ■

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# Prevention of Surgical-site Infections

ABSTRACT & COMMENTARY


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*Dr. Winslow serves as a consultant for Siemens Diagnostics, and is on the speaker's bureau for GSK and Cubist.*



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***Synopsis:*** *In total, 6,771 patients underwent screening for* Staphylococcus aureus *nasal colonization using real-time polymerase chain reaction (PCR). Of those, 1,251 patients were positive, 917 were enrolled in the trial, and 808 subsequently underwent a surgical procedure. The enrolled patients were treated with nasal mupirocin ointment plus body chlorhexidine*



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**Questions & Comments**

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*baths (or placebo). Rates of* S. aureus *infection were 3.4% in the mupirocin-chlorhexidine group vs. 7.7% in the placebo group.*

**Source:** Bode LGM, et al. Preventing surgical-site infections in nasal carriers of *Staphylococcus aureus*. *N Engl J Med*. 2010;362:9-17.

In this prospective, randomized, placebo-controlled trial from the Netherlands, 6,771 patients were screened on admission to the hospital for the presence of *Staphylococcus aureus* nasal colonization using real-time PCR. Of those, 1,251 patients were positive for *S. aureus* and 917 were included in the intent-to-treat analysis. A total of 808 underwent a surgical procedure. Staph strains identified in this study were all methicillin and mupirocin-susceptible. Patients were randomized to twice daily intranasal mupirocin (vs. placebo) plus daily baths with chlorhexidine scrub (vs. placebo soap) for five days, beginning shortly after admission. The rate of *S. aureus* infections was 3.4% in the mupirocin- chlorhexidine group and 7.7% in the control group. The beneficial effect of decolonization was most pronounced for deep surgical-site infections (4 vs. 16). No difference in overall mortality was seen between the groups; however, three patients who underwent cardiothoracic surgery and developed Staph infections died. Hospital length of stay was significantly shorter in the intervention vs. control group (12.2 vs. 14 days). Molecular typing of isolates obtained from wound infections was performed, and the results were compared to the pre-treatment nasal isolates from the same cases to determine whether the infection was of endogenous or exogenous origin. Twelve endogenous infections were seen in the decolonization group vs. 25 in the control group. Four vs. six exogenous infections were seen in the decolonization vs. control groups, respectively.

■ **COMMENTARY**

This is an important study that demonstrates that rapid screening using PCR can identify a population of patients where relatively simple and benign interventions can have a significant impact on a clinical outcome. The use of both intranasal mupirocin plus chlorhexidine scrubs was important for the simultaneous elimination of both nasal and extranasal carriage. The study nicely demonstrates positive results in the context of a carefully controlled experimental design. However, it is likely that, in the real world, results might not be as positive for several reasons. These include the difficulty of performing five consecutive days of decolonization immediately prior to surgery in all cases, particularly when surgery is urgently performed. It is not known whether shorter periods of decolonization would be as successful, since only a small number of patients in this study had surgery performed during the first five days of hospitalization. The authors also mention that in patients who had prolonged hospitalizations, repeat decolonizations might be necessary. Another concern is that the increasing prevalence of mupirocin resistance in *S. aureus*, which has been shown to limit the effectiveness of mupirocin decolonization, might make this intervention less effective in the United States. One last caveat is that this study looked at short-term decolonization and its effect on reducing surgical-site infections. Larger studies looking at decolonization in targeted populations of surgical patients which incorporate cost effectiveness analysis would be of interest.

A much more common problem infectious disease specialists see in the clinic is the patient referred because of recurrent furuncles, usually due to MRSA. I have been very disappointed over the years with the efficacy of decolonization (using combinations of systemic antibiotics such as rifampin ± trimethoprim/sulfamethoxazole, antiseptic scrubs, and intranasal mupirocin) in reducing the frequency of recurrences. I suspect that this is due to fairly rapid recolonization with pathogenic Staphylococci following completion of the decolonization regimen.

Another paper evaluated chlorhexidine-alcohol vs. povidone-iodine for perioperative surgical-site anti- sepsis.[1] This prospective, randomized trial of 849 patients demonstrated that the rate of surgical-site infection was significantly lower in the chlorhexidine-alcohol group (9.5%) vs. povidone-iodine (16.1%). This study represents important information which can be immediately applied to improve clinical practice. ■

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# Lending a (Clean) Hand: Improving Hand-hygiene Practices

ABSTRACT & COMMENTARY

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*Dr. Kleinpell reports no financial relationship to this field of study.*

*This article originally appeared in the February 2010 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, Seattle, and Dr. Thompson is Staff Pulmonologist, VA Medical Center; Associate Professor of Medicine, University of Washington; they report no financial relationships relevant to this field of study.*

***Synopsis:*** *When ICU practitioners worked under senior clinicians who modeled good hand-hygiene practices, their own behavior with respect to hand hygiene improved.*

**Source:** Schneider J, et al. Hand hygiene adherence is influenced by the behavior of role models. *Pediatr Crit Care Med*. 2009;10:360-363.

Infection prevention is a priority area of focus for critical-care units worldwide. As hospital-acquired infections remain the most common cause of preventable morbidity and mortality, efforts to improve hand-hygiene compliance have great significance for improving health care outcomes for hospitalized patients.

This prospective, observational study examined the impact of hand-hygiene adherence of two critical-care fellows and four nurse orientees in a pediatric and cardiac intensive care unit of a tertiary care children's hospital who were paired with physician and nurse preceptors who maintained strict hand-hygiene adherence. The results revealed that hand-hygiene adherence improved from a baseline of 22% of 222 hand-hygiene opportunities prior to introduction of a senior supervising clinician who modeled this behavior to 56% of 234 opportunities after the supervising clinician was introduced. The study results indicate that hand-hygiene practices of senior practitioners have an impact on the hand-hygiene compliance of other health care team members.

## ■ COMMENTARY

It is well established that one of the most important measures for preventing the spread of pathogens is effective hand washing.[1] Hand-hygiene measures are an essential component of preventing health care-associated infections. Normal human skin is colonized by bacteria, including both resident flora and microorganisms that reside under the superficial cells of the stratum corneum, and transient flora, which colonize the superficial layers of the skin. Handwashing or hand antisepsis is effective at reducing the transmission of health care-associated pathogens, as well as the incidence of health care-associated infections.[2] A number of studies have demonstrated the impact of hand hygiene on health care-associated infection rates or the reduction in cross transmission of antimicrobial resistant pathogens.[3-5] As a result, a number of evidence-based guidelines have been published addressing effective hand hygiene.[1,3,6] In addition, several organizational initiatives have focused on promoting best practices for hand hygiene to improve infection-prevention measures, including the Institute for Healthcare Improvement,[7] the Hand Hygiene Resource Center,[8] and ongoing campaigns such as the World Health Organization's "Save Lives: Clean Your Hands" initiative, which has designated May 5, 2010, as a global day call to action for hand hygiene to health care workers worldwide.[2]

As the transmission of health care-associated pathogens has been attributed most often to the contaminated hands of health care personnel,[7] focusing on improving hand-hygiene compliance rates is beneficial in targeting the prevention of health care-associated infections. The study by Schneider et al demonstrated a significant improvement in hand-hygiene adherence based on the role modeling of proper hand-hygiene practices by senior practitioners. This study adds to the body of literature that has focused on improving the hand-hygiene behaviors of health care workers. Yet, as a single-site study involving a small sample size of health care practitioners, the generalizability of the results is limited. Other studies have demonstrated the benefit of multidisciplinary hospital-wide educational programs, easy access to hand-rub solutions, dispensing systems, and the impact of hospital redesign on hand-hygiene compliance rates.[9-12] Yet the impact of interventions on sustaining the rates of adherence to hand-hygiene measures has been limited.[3] As hand-hygiene practices involve a complex interaction of factors, additional research on the impact of role modeling to improve hand-hygiene compliance is indicated to focus on further improving a culture of patient safety and proper hand hygiene in critical care. ■

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# Intensive Lipid Lowering in ACS Patients Undergoing PCI

ABSTRACT & COMMENTARY

***By Andrew J. Boyle, MBBS, PhD***
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*Dr. Boyle reports no financial relationships relevant to this field of study. This article originally appeared in the February 2010 issue of* Clinical Cardiology Alert. *It was edited by Michael H. Crawford, MD, and peer reviewed by Ethan Weiss, MD. Dr. Crawford is Professor of Medicine, Chief of Cardiology, University of California, San Francisco, and Dr. Weiss is Assistant Professor of Medicine, Division of Cardiology and CVRI, University of California, San Francisco. Dr. Crawford is on the speaker's bureau for Pfizer, and Dr. Weiss reports no financial relationships relevant to this field of study.*

**Source:** Gibson CM, et al. Effect of intensive statin therapy on clinical outcomes among patients undergoing percutaneous coronary intervention for acute coronary syndrome PCI-PROVE IT: A PROVE IT-TIMI 22 (Pravastatin or Atorvastatin Evaluation and Infection Therapy-Thrombolysis In Myocardial Infarction 22) Substudy. *J Am Coll Cardiol*. 2009;54:2290-2295.

INTENSIVE LIPID LOWERING WITH STATIN THERAPY (ATORvastatin 80 mg) in patients presenting with acute coronary syndromes (ACS) resulted in improved outcomes compared to treatment with moderate lipid lowering (pravastatin 40 mg) in the PROVE-IT TIMI-22 study (Cannon et al. *N Engl J Med*. 2004;350:1495-1504), which included patients treated conservatively, as well as those treated with percutaneous coronary intervention (PCI). Whether this effect holds true in patients treated with PCI is unknown. Therefore, Gibson et al performed a post-hoc analysis of the PROVE-IT TIMI 22 study and analyzed the outcomes of those patients who had PCI during the index hospitalization for ACS.

Patients presenting with ACS (myocardial infarction [MI] or high-risk unstable angina) were screened for total cholesterol 240 mg/dL if statin-naïve or 200 mg/dL if they were already taking statins. Within 10 days of presentation, they were randomized 1:1 in a double-blind fashion to receive atorvastatin 80 mg daily or pravastatin 40 mg daily. If low-density lipoprotein (LDL) remained 125 mg/dL, the pravastatin could be increased to 80 mg daily. The main outcome measure of this analysis was time to the occurrence of one of the components of the primary endpoint of PROVE-IT TIMI 22: death, MI, unstable angina requiring hospitalization and revascularization at least 30 days after randomization, and stroke. Additional outcome measures included target vessel revascularization (TVR) and non-target vessel revascularization (non-TVR). Patients were followed for two years.

**Results:** Of the 4,162 patients enrolled in the trial, 2,868 underwent PCI during the initial hospitalization and were eligible for this analysis. Among this PCI cohort, the baseline characteristics between those randomized to atorvastatin 80 mg and those randomized to

pravastatin 40 mg were similar, with the exception of peripheral arterial disease being more prevalent in the pravastatin group. Baseline LDL was 106 mg/dL in both groups. This dropped to 89 mg/dL in the pravastatin group and 57 mg/dL in the atorvastatin group at 30 days ($p < 0.001$). Baseline C-reactive protein (CRP) was 13.2 mg/dL, and dropped to 2.14 mg/dL in the pravastatin group and 1.55 in the atorvastatin group ($p < 0.001$). Patients treated with intensive lipid-lowering therapy exhibited significant reductions in the primary composite endpoint (death, MI, stroke or unstable angina requiring hospitalization, and revascularization) compared to patients treated with moderate lipid-lowering therapy (21.5% vs. 26.5% in the atorvastatin 80 mg and the pravastatin 40 mg groups, respectively; $p = 0.001$). Several secondary endpoints were also significantly reduced in the intensive lipid-lowering group: recurrent ischemia (13.0% vs. 17.1%; $p < 0.001$), rehospitalization for unstable angina (3.3% vs. 4.7%; $p < 0.001$), and revascularization 30 days after randomization (16.6% vs. 21.0%; $p = 0.002$) in the atorvastatin 80 mg and pravastatin 40 mg groups, respectively. In addition, there were non-significant trends favoring the intensive lipid-lowering group in all-cause mortality (1.5% vs. 2.5%, $p = 0.057$) and MI (5.8% vs. 7.7%, $p = 0.052$). Intensive lipid-lowering therapy resulted in lower rates of TVR (11.4% vs. 15.4%, $p < 0.001$) and non-TVR (8.0% vs. 10.5%, $p < 0.001$) than moderate lipid-lowering therapy. However, after adjustment for on-treatment LDL and CRP levels, only the reduction in TVR remained significant. Interestingly, the cohort from the original study that was treated medically, instead of with PCI, did not appear to benefit from intensive over moderate lipid-lowering therapy. Gibson et al conclude that among patients with ACS who undergo PCI, intensive statin therapy reduces major adverse cardiac events compared with moderate-dose statin therapy.

### ■ COMMENTARY

Intensive statin therapy improves outcomes following hospitalization for ACS. Gibson et al asked whether this held true in patients who had PCI during their ACS hospitalization and found, somewhat unexpectedly, that this only held true in the PCI patients. Patients treated medically had no statistically significant improvement in the primary outcome in this post-hoc analysis of PROVE-IT TIMI 22 (24.5% vs. 25.2%, $p = 0.779$) when treated with intensive lipid-lowering therapy. Their data are consistent with several recent studies demonstrating the benefits of high-dose statin therapy immediately prior to PCI. In this study, statin therapy was commenced after the PCI procedure, suggesting there may be incremental benefit in high-dose statins before PCI and then continued for two years thereafter. Importantly, their data were collected during the time of bare-metal stents and may, therefore, not apply to patients receiving drug-eluting stents and prolonged dual anti-platelet therapy. It is not clear if the achieved LDL level or the change in LDL correlated with the occurrence of the primary outcome in this substudy. Thus, we do not know if the observed effects are due to more effective lipid lowering or to the pleiotropic effects of statins. Regardless, this study reinforces the need for aggressive lipid-lowering therapy in patients undergoing PCI for ACS. ■

# Pharmacokinetics of IV Immunoglobulins and Outcome in GBS

ABSTRACT & COMMENTARY

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*This article originally appeared in the February 2010 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 Neurology, Weill Cornell Medical College; Chief, Division of Stroke and 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:*** *Fixed dose of IVIg may not be effective in all patients with GBS.*

**Sources:** Kuitwaard K, et al. Pharmacokinetics of intravenous immunoglobulin and outcome in Guillain-Barré syndrome. *Ann Neurol* 2009;66:597-603. Cornblath DR, Hughes RAC. Treatment for Guillain-Barré syndrome. *Ann Neurol* 2009;66:569-570.

KUITWAARD ET AL REVIEWED DATA FROM 174 PATIENTS with Guillain-Barré Syndrome (GBS) who had previously participated in two randomized clinical trials using intravenous gamma globulins (IVIg). All patients were unable to walk unaided and received a

standard dose of IVIg. The data analysis revealed considerable variation in the increase in serum IgG (ΔIgG) at two weeks after IVIg treatment, but the patients with a low ΔIgG recovered significantly more slowly, and fewer reached the ability to walk unaided at six months. In a multivariant analysis adjusted for other known prognostic factors, a low ΔIgG was independently associated with poor outcome. The authors suggest that patients with a small increase in serum IgG level may benefit from a higher dosage or second course of IVIg.

In an accompanying editorial, Cornblath and Hughes stress the need for improvement in the management of GBS, as at one year, 2%-3% of those with severe GBS will have died and 15%-18% will still have significant disability. They note the absence of studies to determine the optimal dose of IVIG in GBS, and voice support for additional clinical trials with higher or repeat doses.

## ■ COMMENTARY

The preferred treatment for GBS is IVIg, although questions remain regarding the dose, timing, repeat treatment, and mechanism of action.

The findings by Kuirwaard et al point to the importance of identifying biomarkers that predict response to treatment. Differences can be due to underlying disease mechanisms, or to differences in the interaction of the IVIg with the host immune system. In chronic inflammatory demyelinating polyradiculoneuropathy (CIDP), for example, recent studies show that the response to IVIg is influenced by polymorphism in TAG-1,[1] and may be associated with upregulation of the expression of Fcgamma receptor IIB on B cells.[2] Following ΔIgG in patients with GBS treated with IVIG might be such a biomarker that would help guide treatment.

Another important observation is that not all patients with GBS are the same, which puts current guidelines into question.[3] This study, as an example, provides a rationale for giving patients an additional dose of IVIg if there is a relapse or no response to the initial treatment. Restricting the treatment recommendations to patients who are no longer ambulatory, as only such patients were included in clinical trials, without comparing other time points, may also be inappropriate given our inability to identify those patients who will progress to severe disability. ■

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# CME Questions

**19. In the prospective, randomized trial by Sung et al, continuation of low-dose aspirin, in addition to eight weeks of pantoprazole in patients admitted with acute peptic ulcer bleeding, led to which of the following outcomes compared to patients in whom aspirin was discontinued?**

a. Increased recurrent GI bleeding in patients in whom low-dose aspirin was continued
b. No significant difference in transfusion rates
c. Decreased all-cause, 30-day mortality in patients in patients in whom low-dose aspirin was continued
d. All of the above

**20. According to the randomized, placebo-controlled trial reported by Bode et al, which of the following interventions successfully decreased *Staphylococcus aureus* surgical-site infections in hospitalized patients with methicillin-sensitive *S. aureus* nasal colonization?**

a. Prophylactic perioperative administration of intravenous cefazolin for 48 hours
b. Twice daily intranasal mupirocin plus daily baths with chlorhexidine scrub for five days prior to surgery
c. Once-daily intranasal mupirocin for three days prior to surgery
d. Povidine-iodine scrubs for two days prior to surgery

**21. In recent studies of Guillain-Barre syndrome:**

a. IV corticosteroids are the current treatment of choice.
b. Interferon beta-1a has emerged as the treatment of choice.
c. A small increase in serum IgG levels after a course of IV immunoglobulin treatment was independently associated with a poor outcome compared to patients who had a more substantive rise in IgG levels.
d. Treatment with IV immunoglobulin (IVIg) is uniformly successful.

**Answers:** 19. (d); 20. (b); 21. (c)

# CME Objectives

Upon completion of this educational activity, participants should be able to:

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

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

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


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

# HOSPITAL MEDICINE ALERT

**HMED030110TM**

**CE/CME Evaluation**

Please take a moment to answer the following questions to let us know your thoughts on the CE/CME program. Fill in the appropriate space and return this page in the envelope provided. You must return this evaluation to receive your certificate. Thank you.

**CORRECT** ● **INCORRECT** ⊘ ✓ ✗ ⋀⋀

1. In which program do you participate? ○ CE ○ CME

2. If you are claiming physician credits, please indicate the appropriate credential: ○ MD ○ DO ○ Other _____

3. If you are claiming nursing contact hours, please indicate your highest credential: ○ RN ○ NP ○ Other _____

| | Strongly Disagree | Disagree | Slightly Disagree | Slightly Agree | Agree | Strongly Agree |
|---|---|---|---|---|---|---|
| **After participating in this program, I am able to:** | | | | | | |
| 4. Review pertinent safety, infection control, and quality improvement practices; | ○ | ○ | ○ | ○ | ○ | ○ |
| 5. Discuss diagnosis and treatment of acute illness improvement practices; in the hospital setting; and | ○ | ○ | ○ | ○ | ○ | ○ |
| 6. Review current data on diagnostic and therapeutic modalities for common inpatient problems. | ○ | ○ | ○ | ○ | ○ | ○ |
| 7. The test questions were clear and appropriate. | ○ | ○ | ○ | ○ | ○ | ○ |
| 8. I am satisfied with customer service for the CE/CME program. | ○ | ○ | ○ | ○ | ○ | ○ |
| 9. I detected no commercial bias in this activity. | ○ | ○ | ○ | ○ | ○ | ○ |
| 10. This activity reaffirmed my clinical practice. | ○ | ○ | ○ | ○ | ○ | ○ |
| 11. This activity has changed my clinical practice. | ○ | ○ | ○ | ○ | ○ | ○ |

If so, how? _____

12. How many minutes do you estimate it took you to complete this entire semester (6 issues) activity? Please include time for reading, reviewing, answering the questions, and comparing your answers to the correct ones listed. _____ minutes.

13. Do you have any general comments about the effectiveness of this CE/CME program?

_____

**I have completed the requirements for this activity.**

**Name (printed)** _____ **Signature** _____

Please make label address corrections here or PRINT address information to receive a certificate.

PLEASE NOTE: If your correct name and address do not appear below, please complete the section at left.

Account # _____

Name: _____

Company: _____

Address: _____

City: _____ State: _____ Zip _____

Fax: _____ Phone: _____

E-mail: _____