Do Quality Improvement Collaboratives Improve Antimicrobial Prophylaxis in Surgical Patients?

TO THE EDITOR: The conclusions stated in the abstract of the article by Kritchevsky and colleagues (1) do not accurately represent the study’s findings. In fact, there is substantial reason to conclude that the collaborative was of meaningful clinical value. For example, improvement was statistically significant in 4 of the 6 mean performance measures in the intervention group but in only 3 of 6 measures in the feedback-only (control) group. Moreover, improvement in the mean “all-or-none” measure was 15.6% greater in the intervention group than the control group, which exceeds the 15% difference sought in the power calculation for the primary outcome. Although that particular observed difference was not statistically significant, the upper bound of the CI for the all-or-none measure does not exclude a between-group advantage as great as 49% for the intervention group.

Although the article did not include the changes observed in each hospital, the wide CIs for many of the before and after measures (for example, 15.6 percentage points for the primary outcome in the control group, which is 20.8% of the baseline rate) indicate substantial heterogeneity of response to both interventions among individual hospitals, including quite large responses in some hospitals. Heterogeneity of response to either biological or social interventions is not simply “noise” (2); rather, it serves as the starting point for understanding the mechanisms involved in those responses. The real value of this study therefore seems to lie in the discovery of that heterogeneity, and the opportunity for explaining it—why feedback alone or the feedback-plus-collaborative model was effective, for whom, and in what circumstances (3)—as much as in the demonstration of whether, in the aggregate, each intervention “worked.” Numbers are not explanations (4), so it is fortunate that Kritchevsky and colleagues have pinpointed in the discussion several local and external factors that could explain the differential effect of the interventions within each study group (for example, diversity of staff training and experience, variation in total and type of staff participation, and variable involvement of institutional leadership).

Unfortunately, however, the study was not designed to explain how the best individual hospital performers differed from the worst, and why. For example, we have no way of knowing whether the history of successful improvement efforts differed among participating hospitals, or exactly which staff participated in the collaborative and why, and how they were involved in the care processes being changed.

Changing health care work is a complex, context-bound social process. Evaluation of any improvement intervention therefore requires clarity regarding the theory that underlies its selection; recognition of the contributions of individual stakeholders to its effects; awareness of the sequence and execution of its individual steps; recognition of the power relationships among people involved in its implementation; understanding of the multiple realities that affect its execution: timing, culture, resource allocation, staffing, and competing priorities; and awareness that most interventions start by being adapted to fit local circumstances and that the interventions and the contexts in which they are implemented evolve over time in response to the resulting changes. For their findings to be meaningful, future studies of improvement interventions will need to include these elements in their study designs (5).

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References

IN RESPONSE: We thank Dr. Liu and colleagues for their comments regarding our recent article. They made several statements about our results that readers less familiar with the article may find misleading. Although both groups showed improvements in many aspects of antimicrobial prophylaxis, differences in the extent of improvement between the 2 groups on any indicator were not significant. Our conclusion is correct in this regard. Our study was designed to examine the absolute difference in change between the 2 groups, not the relative difference in change; thus, the proper statistic to evaluate the effect of participation in the collaborative group for the “all-or-none” indicator shows a 6.3% (95% CI, −7.3% to 19.8%) greater improvement in the active intervention.

We agree that improvement interventions are complex social processes that should be theory-based and explained in context by using evaluation models drawn from social science (1, 2). However, we disagree with the statement that the real value of our study lies in the discovery of the heterogeneity and the opportunity for explaining it. Participants in all randomized studies, be they patients or organizations, exhibit heterogeneity. One of the greatest values of a randomized trial design is the ability to answer the question of effectiveness in the face of heterogeneity, thereby addressing the expectation of improvement for the next patient (or organization) that adopts the evaluated treatment. We agree that case studies of success cannot be extremely helpful in stimulating quality improvement, but the fact that we do not know which mechanisms worked within which circumstances does not negate the value of the cluster randomized trial as an evaluative methodology.
Much needs to be done to improve the quality of health care delivery, and quality improvement collaboratives have a prominent role. In a recent systematic review of the impact of quality improvement collaboratives, Schouten and colleagues (3) concluded that the evidence of their effectiveness is encouraging but limited; they called for studies with a balance of both process-oriented reports and rigorously controlled designs to understand why some collaboratives succeed while others have little effect on practice. Nevertheless, quality improvement collaboratives can be expensive to implement, and tightening resources require the selection of cost-effective strategies. Data from rigorous evaluations, such as that collected for TRAPE (Trial to Reduce Antimicrobial Prophylaxis Errors), are needed to help organizations select among potential improvement strategies.

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References

Before answering this question, we must consider whether rates of adherence to screening are test-specific, such that differences in adherence may be more important for prediction of the best screening strategy than test-specific sensitivity for neoplasia. This issue can be appreciated by examining Figure 3, in which it is clear that 80% adherence to Hemoccult SENSA (Beckman Coulter, Fullerton, California) provides better outcomes per 1000 persons than 50% adherence to colonoscopy (2). Provision of more-detailed analyses of outcomes relative to test-specific variation in adherence is desirable. For example, at what point of relative adherence does a more-sensitive colonoscopy strategy dominate a less-sensitive fecal immunochromatographic test strategy? More important, if small (even in the range of 10%) advantages in adherence for the less-sensitive strategies are associated with better projected outcomes relative to colonoscopy, then trials that compare both adherence and clinical outcomes after invitation to fecal immunochromatographic testing or Hemoccult SENSA versus colonoscopy are warranted.

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References

IN RESPONSE: We agree that an interesting finding from our analysis (1) for the USPSTF is that all the screening strategies now recommended by the USPSTF are nearly equivalent for life-years gained with screening when all have equivalent 100% adherence. We note that the USPSTF recommendation for the strategies of a highly sensitive guaiac or immunochromatographic fecal occult blood test (FOBT) annually, flexible sigmoidoscopy every 5 years with a sensitive FOBT, and colonoscopy every 10 years are based on the perspective of a program of screening from age 50 to 75 years with the end point of life-years gained rather than for screening at a point in time with the end point of reducing colorectal cancer incidence (2). We also agree that the issue of adherence is a crucial component to the effectiveness of a screening intervention. The assumption of 100% adherence to all aspects of screening was used to provide a comparable assessment of potential efficacy for the different screening strategies and represents the best screening offer for those who adhere to testing, follow-up of positive findings, surveillance, and treatment. However, in clinical practice, adherence is very complex (and con-

Letters
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It will be interesting to see how the importance of these skills is rated by patients, and how much these skills actually influence their clinical course.

TO THE EDITOR: The healing skills identified by Churchill and Schenck (1) are vital to implementing continuity care “personal care” and a “whole-person orientation,” 2 of the original 4 principles of a medical home described by Rogers (2) (the other 2 principles being team-directed medical practice and care that is coordinated and integrated). More recent iterations of the medical home concept, however, have moved from including reimbursement as a piece of the medical home (3) to translating “medical home” almost exclusively into a reimbursement concept. This article provides helpful documentation of the healing skills that must be the foundation of any conceptualization of what it means to build a medical home and a vital reminder to busy clinicians on what needs to be the focus of their day-to-day clinical practice.

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References
Mussel-Associated Azaspiracid Intoxication in the United States

Background: Azaspiracids (AZAs) are a group of structurally novel, heat-stable marine toxins identified in mussels from Ireland following outbreaks of severe vomiting and diarrhea in Europe in 1995 and 1997 (1, 2). Azaspiracids accumulate in mussels and other bivalve mollusks that feed on toxic phytoplankton and vector organisms (3). Shellfish contaminated with AZAs have been documented in several European countries, including Ireland, Norway, and the United Kingdom (3).

Objective: To describe the first recognized cases of azaspiracid poisoning outside of Europe.

Case Report: Mussels harvested in Ireland and marketed in a cooked, frozen product consisting of mussels in a garlic and butter sauce (net weight, 1 lb) were heated and eaten by a husband and wife in Washington in July 2008. The husband ate approximately three quarters of the product, and the wife ate one quarter. Iced tea was the only other food ingested at the time. Five hours after eating the product, the couple experienced abdominal heaviness, vomiting (12 to 15 episodes for the husband, 5 to 10 for the wife), and profuse watery diarrhea for 24 to 30 hours. No medical treatment was sought. Two days later, the husband reported the illnesses to the U.S. Food and Drug Administration (FDA). The FDA purchased 5 packages of the product with the same lot number as that on the package the couple had bought and consumed. Analysis of the mussels for the presence of AZA (4) was conducted at FDA's Gulf Coast Seafood Laboratory in Dauphin Island, Alabama. The presence of AZA was determined by liquid chromatography-mass spectrometry/mass spectrometry. Toxicon. 2005;46:62-71. [PMID: 15922391]

Discussion: Although meal remnants were not available for testing, we believe that AZA caused the couple’s illness because symptoms were consistent with those of previous reports of azaspiracid poisoning and because the toxins were identified in mussels from multiple packages of the same product lot as that ingested. Levels of AZA have been shown to vary significantly among mussels harvested from a given region (5), suggesting the concentration of toxins present in the mussels consumed exceeded the AZA regulatory limit. This report highlights the concern for the safety of foods marketed internationally from areas of disease endemicity.

Conclusion: Shellfish contaminated with AZA may be a cause of food-induced gastroenteritis in the United States.

References
within 18 hours. Electrocardiographic examination was normal. The carboxyhemoglobin level was 1%. The patient’s mental status normalized within 3 hours, and inpatient cardiovascular work-up (including echocardiography) was unremarkable. Computed axial tomography of the head was also unremarkable. The patient was discharged after a 2-day hospitalization.

Discussion: Asphyxia occurs when atmospheric oxygen is less than 15%. This can occur with displacement of oxygen by a carbon dioxide concentration of 2000 ppm. Tokaoka and colleagues (1) describe a similar episode of carbon dioxide–induced asphyxia in a 37-year-old man exposed to 40 kg of dry ice in his car. In that report, the individual experienced seizures, pulmonary edema, and acidosis. We speculate that this patient’s obstructive sleep apnea increased his susceptibility to toxicity from exposure to carbon dioxide and suggest that physicians caution patients with sleep apnea about the dangers of working with dry ice in enclosed environments (2–4).

Conclusion: Dry ice can induce asphyxia in enclosed spaces, an effect that can be more pronounced in patients with preexisting respiratory disease, such as obstructive sleep apnea.

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References

Mothball Mayhem: Relapsing Toxic Leukoencephalopathy due to p-Dichlorobenzene Neurotoxicity

Background: Aromatic hydrocarbons (such as toluene, n-hexane, and methyl butyl ketone) are volatile, lipid-soluble compounds found in various household and commercial products, such as aerosols, cleaning fluids, paints, and fuels. These substances are not identified as recreational drugs, but they are readily available and are commonly abused by sniffing (nasal inhalation) or huffing (oral inhalation). Symptoms due to inhalant abuse are similar to those resulting from central nervous system depressants, such as alcohol.

Because of its much lesser toxicity, p-dichlorobenzene (PDB) has replaced naphthalene as the primary component of mothballs. Both compounds have abuse potential. Inhalation and dermal absorptions are the common exposure routes. Toxicity is generally a consequence of accidental or occupational exposure. Toxicity rarely results from chronic ingestion (sucking or chewing) (1–4). Short-term ingestion of mothballs may be seen in the setting of pica (4). The commonly recognized manifestations of PDB toxicity is hemolysis with methemoglobinemia and jaundice. Neurotoxicity related to PDB is very rare (1–5). Cerebellar, extrapyramidal, cognitive, and pyramidal manifestations have been described. Diffuse leukoencephalopathy with progression to stupor, mutism, and coma has also been reported (4, 5). Frank addiction may occur (1). In some cases, withdrawal rather than use of PDB has been the suspected mechanism (3). In cases of clinical deterioration after abstinence, readministration and gradual taper of PDB may be a therapeutic option (3).

Objective: To report a case of toxic leukoencephalopathy resulting from years of intermittent nasal inhalation and ingestion of mothballs.

Case Report: A 32-year-old woman with a history of bariatric surgery at age 27 years was hospitalized at our institution for a 1-year history of recurrent episodes of neurologic dysfunction characterized by subacute onset of dystartria, ataxia, cognitive decline (at one point to catatonia), and skin scaling. Ninety percent improvement was noted after a past hospitalization, but the underlying diagnosis remained elusive, with no evidence of nutritional deficiencies or toxic exposures. After discharge to home, the patient’s condition deteriorated, prompting hospitalization at our institution. She demonstrated clinically significant deficits: dementia, spastic and hypokinetic dystartria, limb spasticity, bradykinesia, predominantly distal limb weakness, ataxia, and hyperreflexia. Ambulation without assistance was not possible. Magnetic resonance imaging showed diffuse increased T2 signal involving the cerebral white matter (Figure). No cause was determined despite extensive investigations. She improved and was discharged to a local skilled-nursing facility where improvement continued. However, after transfer to another skilled-nursing facility closer to her home, she again deteriorated, prompting return to our institution.

We discovered that the patient had a history of chronic mothball sniffing starting as a child and progressing to ingestion approximately 2 years ago. After each hospitalization and before each episode of deterioration, she admitted resumption of ingestion of at least 1 mothball daily. The plasma PDB level, determined by gas chromatography, was elevated to 0.50 µg/mL (detection threshold, 0.02 µg/mL). The PDB level was rechecked after a subsequent relapse and was markedly elevated to 34 µg/mL.

Discussion: Mothballs and other PDB-containing products (insect repellents, air fresheners, or toilet-bowl or diaper-pail odorizers) are readily available and, as with other aromatic hydrocarbon-containing products, have recognized abuse potential. Toxicity from PDB commonly results in hematologic and dermatologic manifestations that can be additional diagnostic clues (2). Rapid hepatic clearance of PDB and its metabolite 2,5-dichlorophenol is followed by accumulation in adipose tissue. Depletion of adipose tissue stores of these compounds is slow. Delayed deterioration after discontinuation of PDB use or an acute presentation may reflect release of the lipophilic toxin from fat reserves (4). The lipophilic nature is probably responsible for accumulation in and damage to myelin. Magnetic resonance imaging changes due to PDB toxicity may persist despite clinical improvement and reduction in PDB levels (4).

Conclusion: Our case emphasizes that mothball inhalation and ingestion can lead to severe encephalopathy. This should be considered in the differential diagnosis of obscure encephalopathies and cerebellar disorders in all age groups.

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Potential Financial Conflicts of Interest: None disclosed.

References
References

CORRECTIONS

Correction: In The Clinic: Obesity
There is an error on page ITC4-3 of the Obesity issue of In The Clinic (1). The accepted definition of overweight is listed as 25.0 to 25.9 kg/m², which is incorrect. The correct range is 25.0 to 29.9 kg/m². The online version has been corrected.

Reference

Correction: In The Clinic: Hypertension
There is an error on page ITC6-11 of the Hypertension issue of In The Clinic (1). The subheading “ACE inhibitors and ARBs combined with nonhydropyridine calcium-channel blockers” should say “dihydropyridine” instead. The online version has been corrected.

Reference