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Guidelines for CAP—Are They Really Worth it?

ABSTRACT & COMMENTARY

Synopsis: *Guidelines for the care of patients hospitalized for community-acquired pneumonia can be helpful if there is variance—they can even save hospital days and money if the physicians are involved. It is important, however, to select practical outcomes indicators and to focus on saving patient lives rather than simply saving money.*

Source: Nathwani D, et al. Do guidelines for community-acquired pneumonia improve the cost-effectiveness of hospital care? *Clin Infect Dis.* 2001;32:728-741.

Community-acquired pneumonia (cap) has become a major focus of investigation and publication because of the large amount of money spent on antibiotics for respiratory infections, the release of new antibiotics, and the changing susceptibility of respiratory pathogens. The British Thoracic Society, the American Thoracic Society, and the Infectious Diseases Society of America have all produced guidelines although they differ somewhat in recommendations. The attempt to bring conformity in decision making by managed care organizations has led to efforts to apply, if not enforce, these guidelines, but the effect they have had is not clear.

Nathwani and associates took on the daunting task of reviewing 76 published studies of the effect of guidelines for patients hospitalized for CAP. They asked 4 questions and found the following answers:

Do guidelines change practices? Yes, they can. Although not all did, studies showed they could lead to earlier hospital discharge through use of the pneumonia severity-of-index scale. Obstacles to change included physician lack of awareness of the guidelines, a lack of trust or disagreement with the guidelines, patient attitudes toward going home, and adequate community services to take responsibility. Successful implementation related to broad physician involvement in developing the guidelines, continuing reinforcement of them, and providing resources to facilitate compliance.

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How could CAP guidelines lead to improved outcome of care? Even if changes in process of care can be accomplished, it may not be possible to demonstrate patient benefit or to actually improve patient outcomes. There are some indicators, however, which do relate to mortality at a statistically significant level. These include obtaining blood cultures, including a macrolide in the initial therapeutic regimen, and the speed of initiation of intravenous antibiotic therapy.

How could CAP guidelines lead to reductions in the cost of care? Better patient care through guidelines has been able to save money through reduced hospital admissions, shortened hospital stays, and shortened courses of intravenous antibiotic therapy, but these have not been shown to improve outcomes in patient care. Cost-effectiveness rather than cost-minimalization studies are needed.

What evidence exists that implementation of CAP guidelines recommendations improves the cost-effectiveness of care? The studies have shown that changes in process can result in cost reduction without loss of quality of care or patient outcomes, but none have shown actual patient benefit.

An assessment of variance in practice before introduction of guidelines may also be worthwhile. If physicians are already compliant, there is little need for change.

Nathwani et al suggest that practice guidelines can be valuable and should be used, but they caution that enhanced patient care rather than simply cost reduction should be the goal. Costs appear to be less when patient outcomes are improved. They also suggest that early physician involvement in developing guidelines is important and that outcomes indicators should be simple, practical, and achievable (ie, how long it takes for a medication to be given after it is ordered, duration of hospital stay, readmission rate, and 30-day mortality). They also note that "the impact of practice guidelines on physicians is never as great as their authors intend" and encourage the development of guidelines for patients to be treated in the community rather than the hospital.

COMMENT BY ALAN D. TICE, MD, FACP

The amount of information being published about the management of respiratory infections has been remarkable. The impetus appears to be the large potential market for antibiotic use (greater than any other infection) that a number of manufacturers are targeting for their new products. With the conflicting influence of growing pressures to reduce the use of antibiotics, the results have been interesting. There is also the increasing demand for accountability from managed care, which has led to an evidence-based medicine approach to decision making. Just how far these endeavors will go and what changes will actually occur is not clear.

At this point in outcomes research, it seems a bit like the proverbial blind men trying to describe an elephant. Outcomes are different in everyone's eyes and there are few quantitative measures of them other than cost, which has clear and tangible features. What else to measure, how to measure it, and its relation to changes in process or patient benefit are uncertain.

Using outpatient intravenous antibiotic therapy (OPAT) instead of hospitalization yields a clear cost advantage, but there are not yet enough cases to know whether there is a compromise in mortality at 30 days. It is also a problem because there may be a number of factors (ie, the lack of a telephone at home, which may prevent OPAT) that are unrelated to the infection being evaluated. The lack of community resources for home care may be a more important factor in deciding on early hospital discharge than practice guidelines.

Another point that emerges is the shortcomings of guidelines compared to physician decision making. It is

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just not possible to incorporate enough factors into a computer program to match the decision-making ability and accuracy of an experienced physician—at least not yet. Even if it were, it would take so long to enter the data on each patient that the physician's services would be well worthwhile in speed as well as accuracy.

In conclusion, this review encourages guideline use for CAP and provides some insight into their development and application. It is best to involve as many physicians as possible in their development so they know what they are and are willing to accept and implement them in their practice. The objective of the guidelines should be for better patient care. Outcomes measures should not simply be monetary but simple, easy to measure, and with a demonstrated benefit in patient care. If they are used and variations are reduced, money should be saved as well. Continuous monitoring should lead to continual changes in the system and improvement in the understanding of what to monitor and use for further improvements to affect a continuous quality improvement system. ❖

Outbreak of *Serratia liquefaciens* Bacteremia in Dialysis Patients due to Contamination of Epoetin Alfa

ABSTRACT & COMMENTARY

Synopsis: *An outbreak of bacteremia due to Serratia liquefaciens in a dialysis center proved to be due to contamination that occurred during pooling of the residual contents of single-dose vials of preservative-free epoetin alfa.*

Source: Grohskopf LA, et al. *Serratia liquefaciens* bloodstream infections from contamination of epoetin alfa at a hemodialysis center. *N Engl J Med*. 2001;344:1491-1497.

Ten episodes of *Serratia liquefaciens* bacteremia and 6 pyrogenic reactions occurred during a 6-week period among patients in a Colorado dialysis center. The state health department invited the CDC to conduct an investigation. In a cohort study, CDC investigators found that 2 risk factors were independently associated with infection or pyrogenic reaction. These were receipt of median doses of epoetin alfa > 4000 U and dialysis during an afternoon or evening session. The epoetin alfa was supplied in single-use, preservative-free vials of 10,000 U. Because there was typically substantial excess medication remaining in the vial after use, the staff routinely entered each vial with a needle for multiple doses, and then withdrew and pooled the residual when the vial was nearly empty.

Cultures of the center's water supply did not yield *S liquefaciens*, and cases occurred after reprocessing of dialyzers was discontinued. *S liquefaciens* was isolated from 61 of 97 empty vials of epoetin alfa, as well as from a soap dispenser and a hand lotion dispenser in the medication room where the epoetin alfa vials were stored. Blood, medication vials, hand lotion, and soap isolates of *S liquefaciens* were identical by pulsed field gel electrophoresis. The outbreak stopped after the practice of re-entering single-dose vials and pooling of epoetin alfa was discontinued.

COMMENT BY ROBERT MUDER, MD

A cluster of bloodstream infections due to certain genera of Gram-negative bacilli, most notably *Klebsiella*, *Enterobacter*, or *Serratia*, should immediately suggest the possibility of a contaminated intravenous fluid or medication, particularly if the patients affected have no obvious primary source of infection.[1,2] Bacteria of these genera can multiply rapidly in a variety of medications and IV fluid preparations once introduced.

The outbreak of *S liquefaciens* bloodstream infections in this hemodialysis center was caused by the practice of re-entering preservative-free single vials of epoetin alfa multiple times, and pooling residual medication. The likely source of the organism was the hand care products in the medication room. Grohskopf and colleagues noted that the staff typically "topped off" the soap or lotion in the dispensers when they were nearly empty, rather than cleaning and refilling them. This practice would tend to encourage multiplication of any contaminating organism to fairly high levels. Once the hands of the staff were contaminated, the vial stoppers could easily become contaminated during handling. The stoppers were cleaned with an alcohol swab prior to each entry; this is not a reliable sterilization method. Once introduced into the vial, the organisms were able to multiply during subsequent storage in the refrigerator.

The impetus for the multiple usage of vials and pooling of the epotein alfa was cost-saving. Epoetin alfa, given to dialysis patients to increase red blood cell mass, is a moderately expensive medication ($10/1000 U, according to Grohskopf et al). The formulation used at this center was supplied in single-dose, preservative-free vials containing 10,000 U. As the average dose given was about 4000-6000 U, there would have been a considerable amount of medication remaining in each vial that would otherwise need to be discarded.

A survey of dialysis centers found that 58 of 71

(69%) used a single-dose preparation of epoetin alfa, and that 45 of 58 (78%) used the vials repeatedly; 9 (16%) routinely pooled residual medication.

The lesson to be learned here is obvious. Disregarding good infection control technique as a cost-saving measure is hazardous. Furthermore, it is likely to be counter productive in the long run, since health care-related infections (bacteremia in particular) are expensive to treat. It is disconcerting that this lesson must be relearned repeatedly. ❖

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Antifungal Prophylaxis in Critical Care Patients

ABSTRACT & COMMENTARY

Synopsis: *Fluconazole (and perhaps other antifungal agents) may provide effective prophylaxis for selected nonneutropenic, critically ill patients, but further studies are required.*

Source: Rex JH, Sobel JD. Prophylactic antifungal therapy in the intensive care unit. *Clin Infect Dis.* 2001;32:1191-1200.

Rex and sobel have examined the rationale of administering antifungal medication to prevent fungal infections in nonneutropenic, critically ill patients. Reasoning that antifungal prophylaxis in patients undergoing bone marrow transplantation reduces the incidence of systemic fungal infections by nearly two-thirds, they critically analyze the theoretical basis for prophylaxis in intensive care unit (ICU) patients, and review the evidence that fluconazole—as the prototypical antifungal agent—may be useful in this context.

Fungal infections, particularly invasive disease due to *Candida* species (especially *C albicans*), occur with increasing frequency in critically ill patients. Diagnosis may be difficult and is often delayed, and treatment may be initiated too late to prevent serious morbidity or even death.

Rex and Sobel begin consideration of prophylaxis by methodically analyzing statistical issues pertinent to randomized, placebo-controlled trials. They stress that for any prophylaxis trial to be achievable, a major reduction in the incidence of infection must occur. Testing an agent that leads to a 2- or 3-fold reduction in infection requires far fewer patients than one in which only a fractional reduction occurs. A second statistical consideration that drives any prophylaxis trial is clinical relevance: reduction of the rate of infection from 1% to 0.5% is clearly less compelling than a study demonstrating a decrease from 20% to 10%, even though the reduction is halved in each case. These 2 elements are of major importance. Several studies on prophylaxis have faltered because they have been inadequately powered to demonstrate statistical significance.

Is there a need for prophylaxis? With surgical precision, Rex and Sobel carefully set forth the critical defining principles: 1) *Candida* infections, because they constitute the vast majority of fungal infections in nonneutropenic, critically ill patients, should be the "target"; 2) prophylaxis, and not early treatment (eg, beginning antifungal therapy in a febrile patient with multiple sites of *Candida* colonization), is the key to reducing morbidity and mortality; and 3) invasive candidiasis must be clearly defined. Although *Candida* in a respiratory tract specimen may not be indicative of pneumonia, there would be little disagreement that repeated isolation of *Candida* in blood cultures represents deep-seated *Candida* infection. *Candida* in urinary tract or wound specimens likewise poses a difficult problem of definition. Any study attempting to define the frequency of invasive candidiasis and the possible role of preventive intervention must address this issue head on.

Rex and Sobel review a number of studies citing rates of invasive *Candida* infections in the ICU ranging from 2% to 35% and conclude that a rate at the lower end of the range is a reasonable estimate (eg, data from the National Nosocomial Infections Surveillance indicate a 2% rate in this patient group). With this low rate, one can easily understand, given the statistical caveats previously mentioned, that only studies using large numbers of patients can be expected to show even the slightest benefit of prophylaxis.

Rex and Sobel critically review published data on antifungal prophylaxis in the ICU setting. Each of 5 studies differs somewhat in inclusion criteria, definition of infection, and specific antifungal prophylaxis. Rex and Sobel find that only 2 trials are designed well enough to provide meaningful data from which the reader may extrapolate to the clinical arena. In one, Garbino and associates administered low-dose fluconazole (100 mg/d) to more than 100 mechanically ventilated patients in a medical/surgical ICU, and they found a reduction in the incidence of invasive candidiasis from 8.9% in

patients receiving placebo to 3.9% in fluconazole recipients.[1] However, Rex and Sobel point out that invasive fungal infection was defined loosely, and the observed differences between groups failed to achieve statistical significance. They find a recent study by Pelz and colleagues to be more compelling.[2] In this study involving 260 patients in the surgical ICU at Johns Hopkins Hospital, administration of fluconazole at a dose of 400 mg/d nearly halved the incidence of invasive candidiasis (from 15.3% in placebo recipients to 8.5% in fluconazole-treated patients). Although the intent-to-treat analysis fell just short of statistical significance ($P = .07$), several manipulations of the data resulted in impressive risk reductions (with $P = .01$). However, no statistically significant difference in mortality was observed between the 2 groups.

The high rate of infection in placebo recipients (15.3%) in the Pelz study, compared with an expected rate of approximately 2% in ICU patients overall, highlights the importance of patient selection. In the study by Pelz et al, patients were ill in a surgical ICU, had high APACHE III scores and multiple underlying medical conditions, and were elderly. But Rex and Sobel explain, ". . . this study makes clear the potential for prophylaxis: if a suitable group can be identified, prophylaxis can and will be of value."

Eggimann and colleagues appear to have identified one such group.[3] Their small study, limited to carefully selected high-risk patients with recurrent gastrointestinal perforations or anastamotic leakages, found that, relative to placebo, fluconazole significantly reduced the incidence of *Candida* perionitis.

Rex and Sobel conclude their provocative and perspicatious review by calling for additional studies of antifungal prophylaxis in the ICU environment, with special emphasis on selection of carefully chosen subsets of patients to determine where the greatest benefit can be achieved. Looking at the data that are available at present, they conclude that it is possible ". . . that the majority of the benefit was received by the minority of the patients." They state that the recently published guidelines of the Infectious Diseases Society of America discourage routine use of antifungal prophylaxis in the ICU[4] (*Reviewer's note: In fact, the guidelines do not directly address the topic*), but they compellingly make the case that proper selection of patients is key. But it is also the rub: Who are these patients? Which ICU patients are most likely to derive maximal benefit? Only additional well-designed studies can provide the answers. Rex and Sobel have provided the spark that may light the way to significant advances in the management of critically ill patients.

COMMENT BY JERRY D. SMILACK, MD

Rex and Sobel place considerable importance on the study by Pelz et al[2] but acknowledge that this trial was reported only in abstract form. The more complete report of the study, recently published in its entirety,[5] provides some missing details. Most bothersome is that a substantial number of patients in both placebo and treatment groups had presumed, but not definite, invasive *Candida* infection. Included in the former category were patients with *Candida* isolation from only urine or vascular catheter tip cultures. Pelz et al indicate, however, that exclusion of these cases from the proven infection totals failed to alter the statistical significance of the results. ❖

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Antibiotic Resistance—Does it Ever go Away?

ABSTRACTS & COMMENTARY

Synopsis: *Reducing the use of an antibiotic may not inevitably bring a return of susceptibility.*

Sources: Enne VI, et al. Persistence of sulphonamide resistance in *Eschericia coli* in the UK despite national prescribing restriction. *Lancet*. 2001;357:1325-1328; Cunha BA. Effective antibiotic-resistance control strategies (editorial). *Lancet*. 2001;357:1307-1308.

Workers at the royal london hospital collected strains of *E coli* in 1991. A total of 359 remained viable for study in 1999 when a comparable

number of strains were collected from similar inpatient and outpatient sources. The organisms were tested for resistance to sulphonamides ("sulfonamides" to Americans) to see the effect of the restrictions of sulfonamide use in the United Kingdom in 1995. This restriction was imposed because of the high incidence of rash with sulfa drugs and the availability of trimethoprim, which seemed as effective as sulfamethoxazole-trimethoprim. The number of prescriptions for sulfa antibiotics fell dramatically from 320,000 in 1991 to 7000 in 1999.

There was no increase in susceptibility to sulfonamides over time. In fact, there was an increase in resistance from 40% to 46% of strains, and those from the community were no better than the hospital. The strains with resistance were also more likely to carry resistance genes to other antibiotics as well. This was particularly true with ampicillin and chloramphenicol. The ampicillin resistance rate rose from 80% to 86% although chloramphenicol fell from 44% to 27%. Upon further analysis, the resistance to sulfa drugs was found to be due to a variety of genes, the most common of which was *sul-I* (16% in 1991 to 18% in 1999) and *sul-II* (27% in 1991 to 37% in 1999). These genes are carried on plasmids in combination with genes to a variety of other antibiotics.

The British attribute the failure to regain sulfonamide susceptibility to the plasmids, which carry a packet of multiple antibiotic-resistant genes with either *sul-I* or *sul-II*, but they note the possibility that the organisms have developed improved survival mechanisms with the resistance mechanisms. It is also possible that continued sulfa use is playing some role in persistence as they still use about 80 tons of sulfonamides per year for animals.

In the related editorial, Burke Cunha takes the issue of antibiotic resistance further. He ascribes the problem with resistance to antibiotics largely to the use of specific drugs that are more likely to select for resistance than others. He thinks it is not an antimicrobial class effect but rather an individual antibiotic defect. He considers the main culprits to be ciprofloxacin (plus nalidixic acid and norfloxacin) among the quinolones, tetracycline among the tetracyclines (not minocycline or doxycycline), cefamandole among the second-generation cephalosporins, ceftazidime among the third-generation cephalosporins, and imipenem among the carbapenems. These drugs can be recognized by the development of resistance during therapy, even in phase III studies. He notes the high-resistance antibiotics are much more likely in the hospital and in the intensive care units, where they may well share genes with other resistant bacteria.

COMMENT BY ALAN D. TICE, MD, FACP

The findings at the London hospital are most interesting. The presumption that reducing antibiotic usage changes the microbial environment and encourages the return of susceptible organisms is called into doubt. It seemed to have worked in Finland with erythromycin, but the result is different in the United Kingdom. Sulfonamides have been around for more than 50 years and have been used in the community as much as in the hospital. There must be a huge reservoir of resistant organisms out there from their use in the 1960s, 1970s, and 1980s, and that reservoir is only contributed to by continuing to add sulfa drugs to animal feed.

In addition, it may be that the resistant organisms that have survived may now offer some selective advantages over the old, antibiotic-susceptible ones. Are we now producing superbugs in addition to antibiotic-resistant ones? It could also be, as postulated, that the genes responsible for resistance have found a survival team to join and are along for the ride. The continued antibiotic pressure has made a variety of armaments necessary for bacteria to survive in the antibiotic-rich hospitals and even the community. The plasmids that carry these teams can defend themselves from a variety of antibiotics including ampicillin, chloramphenicol, tetracycline, and trimethoprim.

What do the *E coli* findings mean? Can we regain what has been lost? Is the cow out of the barn and never to return despite the most prudent use of antimicrobials? It is difficult to know, but the situation we have created may not be reversible for some antibiotics. Which ones? Sulfonamides and *E coli* seem to be a problem but others may not. Will the same thing happen with the cephalosporins and quinolones?

The theories of Cunha are additional food for thought. The idea of antibiotics being different in their ability to develop resistance in bacteria is a relatively new one and threatens the simple concept of less antibiotics are better. The ability of bacteria to produce resistance genes to some antibiotics more easily than others is certainly of concern, but it may not be translated into a general problem. Not all resistant genes escape the hospital and establish themselves in the community. On the other hand, resistance may develop rapidly in some situations such as with staphylococci and the quinolones.

It should be noted that the mechanism for antibiotic resistance may not simply be developing smarter or better-equipped bacteria. It may be from replacing one strain with another—such as *Enterococcus faecium* with *E faecalis* when vancomycin is applied. There is also a note of the importance of infection control when resistance genes appear. This has recently been highlighted by the "Siouxland" study.

Obviously, we need to know more about how and

why antibiotic resistance develops and what can be done to prevent and reduce it. The insight gained into sulfonamide and *E coli* in the United Kingdom brings up many questions that need answers soon. ❖

CME Questions

41. Important obstacles in implementing practice guidelines for patients hospitalized for community-acquired pneumonia include all of the following *except*:
a. physician knowledge of guideline.
b. lack of payment to physicians.
c. patient reluctance to be discharged early.
d. poor home care resources.

42. Practice guidelines for community-acquired pneumonia have been demonstrated to save money by the following ways *except*:
a. reduced cost of antibiotics.
b. shorter duration of hospital stay.
c. less use of equipment and staff.
d. better managed care contracts.

43. Which of the following is the best way to implement CAP guidelines for patient care to general physicians?
a. Continually reinforce compliance.
b. Pick easy and practical outcome indicators.
c. Involve as many people as possible in their development.
d. All of the above

44. Sulfonamide resistance genes are linked to plasmids that carry resistance genes for ampicillin as well.
a. True
b. False

45. Which antibiotics are considered to be *less* likely to produce resistance?
a. Imipenem
b. Doxycycline
c. Ciprofloxacin
d. Ceftazidime

46. Which organism is least likely to cause a cluster of bacteremias resulting from contaminated IV fluid?
a. *S aureus*
b. *K oxytoca*
c. *E cloaca*
d. *S liquefaciens*

47. With regard to antifungal prophylaxis in nonneutropenic critical care patients, which one of the following is correct?
a. Amphotericin B has been demonstrated to be effective.
b. Fluconazole is possibly effective in selected high-risk patients.
c. Fluconazole prophylaxis has been demonstrated to significantly reduce mortality.

In Future Issues:

A Nosocomial Outbreak of Fluoroquinolone-Resistant Salmonella

UPDATES

By Carol A. Kemper, MD, FACP

First Fatality to RIF-PZA for Latent Tuberculosis

Source: CDC. *MMWR Morb Mortal Wkly Rep.* 2001;50:289-291.

This report describes the first fatality due to drug-induced hepatitis in an incarcerated 53-year-old man who received a combination of rifampin (RIF) and pyrazinamide (PZA) for latent tuberculosis (TB). Apparently, a 2-month regimen of RIF-PZA is a standard approach for latent TB without evidence of active disease at this correctional facility. The man was in good health, had no history of hepatitis, and was receiving no other agents with significant hepatotoxicity.

Blood tests performed at 1 month of therapy showed grade 4 hepatotoxicity (bilirubin 4.2 mg/dL and transaminases > 10 × ULN). Two days later, when the results of the laboratory studies became available, both drugs were stopped. Despite persisting symptoms of lassitude and nausea, the patient initially failed to report to the infirmary for evaluation and care. Five days after stopping medications, he was admitted with severe jaundice and altered mental status. He died 3 days later of hepatic necrosis.

A second report describes a 59-year-old previously healthy woman who also received a combination of RIF-PZA for a positive skin test with subsequent life-threatening hepatotoxicity. The patient reported some initial queasiness, but laboratory studies obtained on day 17 of therapy showed only eosinophilia and normal transaminases. She developed progressive anorexia, malaise, and low-grade fever but continued drug therapy until, on the 49th day of treatment, she was admitted with jaundice and altered mental status. Laboratory studies suggested a hypersensitivity reaction with eosinophilia and a positive ANA. She received prednisone, and eventually recovered after a lengthy 4-week hospitalization.

Ironically, the woman elected to receive this particular prophylactic regimen because of concerns regarding potential hepatotoxicity to INH and possible exposure to multidrug resistant TB.

Treatment in both cases was provided for latent TB without evidence of active infection. CXRs in both cases were normal. And, although this report did not indicate whether either patient had converted their skin test, it appears that neither case was considered high risk. It is important to recognize that both cases occurred despite prospective monitoring of laboratory studies at about 2-4 weeks of therapy. In both cases, therapy was continued despite symptoms of possible toxicity, at least until overt evidence of hepatic injury. Patients receiving antituberculous therapy should be warned to stop their therapy should they develop any signs or symptoms of hepatic toxicity. This CDC report serves as a reminder that laboratory screening of these patients is not a substitute for good clinical monitoring. ■

But What About Dogs and *E coli*?

Source: ProMED-mail post; www.promedmail.org, accessed May 8, 2001.

The first documented case of transmission of *E coli* 0157:H7 from a dog to a human has been reported in England. The dog was elderly, incontinent of both urine and stool, and described as "largely immobile." He had been kindly taken in by a family 2 months earlier. Their 3-year-old child developed bloody diarrhea and subsequently required hospitalization with hemolytic-uremic syndrome. Fecal samples from the dog were positive for *E coli* 0157. The case probably would not have come to light except that the family is vegetarian, raising suspicions of an atypical source. Although the report did not allude to this, one wonders if the dog's enteric infection contributed to his overall weakened and incontinent condition.

How the dog acquired *E coli* 0157 is a matter of speculation. During the 2 months he lived with the family, he was primarily fed a commercially-prepared dry dog food, which according to the manufacturer is heated and sterilized. Whether it was fed raw eggs or table scraps, such as undercooked hamburger meat, before being taken in by the family is not known. Dogs fed table scraps or undercooked meat may be at risk for acquisition of *E coli* 0157. ■

Vaginal Douching and PID

Source: Ness RB, et al. *Sex Transm Dis.* 2001;28:240-245.

This large-scale, multicenter evaluation of pelvic inflammatory disease (PID; "cleverly" termed the PEACH Study for PID Evaluation and Clinical Health) examined risk factors in 654 women with and without endometritis and upper genital tract infection (UGTI). Douching—either more than once monthly or within 6 days of the onset of symptoms—was a significant risk factor for UGTI. Even after controlling for various confounding factors, such as ethnicity and presence of bacterial vaginosis, douching remained a significant risk behavior. Although it would not be surprising if douching increased the risk of UGIT in women with STDs, even women with normal vaginal flora in this study who douched more often were at greater risk for UGTI. ■