

Pitfalls, pearls, and practicalities in the diagnosis of *Helicobacter pylori* infection

Dr. Gary Falk: In light of the overviews that Drs. Fennerty, Vakil, and Fendrick have presented on the clinical relevance of *Helicobacter pylori* and the noninvasive testing options for *H pylori* in 2005, let's start our roundtable by considering a case study that touches on additional issues that primary care physicians might grapple with when considering *H pylori* infection in their daily practice.

■ CASE STUDY

Dr. Falk: A 39-year-old otherwise healthy male patient that you have been seeing for several years comes to your office and reports epigastric burning discomfort without heartburn or acid regurgitation. It is the first time he has reported these symptoms. He has no weight loss, nausea, vomiting, or other alarm symptoms. David, as a community family practitioner, would *H pylori* be on your radar with a patient like this?

Dr. David Wyatt: Definitely. For a patient with dyspepsia symptoms like these, *H pylori* would certainly be among the things I'd consider, and this type of presentation is very common in my practice.

Dr. Falk: How would you approach this patient from a diagnostic perspective?

Dr. Wyatt: I would begin by assessing the likelihood that his symptoms, which are dyspeptic symptoms, are due to peptic ulcer. Since peptic ulcer seems like a possibility, I would assess his

risk of *H pylori* infection by considering his race and ethnicity, whether he is an immigrant and his country of origin, where he lives—questions that get at the epidemiologic risk factors for *H pylori* infection. If his symptoms did not suggest peptic ulcer disease and he was not a member of a demographic group with an elevated risk for *H pylori* (see **Table 2** on page S11)—say, a middle-class white male whose family had been in the United States for generations—I might be less likely to test for *H pylori* right away, particularly if there were no family history of gastric cancer.

Dr. Falk: Let's say his symptoms are consistent with uncomplicated dyspepsia and the probability of *H pylori* infection seems reasonable, so that testing is indicated. Which testing method are you going to use?

Dr. Wyatt: I would go straight for one of the tests for active infection, either the urea breath test or the stool antigen test, for all the reasons spelled out in the overview presentations that preceded this. A serologic test would not be an option because it tests only for antibody to *H pylori*. A positive result with serology does not tell me whether the patient has current infection or had a past infection that is now cured; I would still be left with no idea whether to treat or not.

With one of the active tests, a positive result would give me a high level of confidence that treatment to eradicate *H pylori* was appropriate. Because the urea breath test and the stool antigen test are essentially equal in performance, my decision between them would be based on their availability in my area, their availability and cost under the patient's health plan, and, if both were available, the patient's preference after hearing about each test and how it is performed.

Dr. Falk: Mark, you also see patients in the primary care setting. Let's say you've tested this patient with an active test, got a positive result, and prescribed treatment for *H pylori* eradication. How do you manage him from that point?

Dr. Mark Fendrick: I like to say that the active tests should be sold in pairs—one for making

A positive result with serologic testing still leaves me with no idea whether to treat or not.
—Dr. David Wyatt

DISCLOSURES: Gary Falk, MD, has served as a consultant to AstraZeneca Pharmaceuticals and Meridian Bioscience. Brian Fennerty, MD, has served as a consultant to AstraZeneca Pharmaceuticals, Eisai, Meridian Bioscience, Santarus, and TAP Pharmaceutical Products. Ben Gold, MD, has served as a consultant to AstraZeneca Pharmaceuticals, Meridian Bioscience, Meretek Diagnostics, and TAP Pharmaceutical Products. Nimish Vakil, MD, has received grant/research support from AstraZeneca Pharmaceuticals, Medtronic, Novartis, and Pfizer; has served as a consultant to AstraZeneca, Medtronic, Meridian Bioscience, and Novartis; and is on the speakers' bureaus of AstraZeneca, Novartis, and TAP Pharmaceutical Products. Leonard Ehrlich, MD, A. Mark Fendrick, MD, Derek van Amerongen, MD, and David Wyatt, MD, have served as consultants to Meridian Bioscience.

the initial diagnosis and, if positive, the other for testing to confirm eradication after treatment. That way retesting wouldn't even be left to choice. Seriously, though, this patient should be retested after treatment to verify whether the infection has been cured. We've seen from studies that patients demand confirmation of eradication and are willing to pay for it,¹ and there is real clinical value to retesting because of the lack of a guaranteed cure even with optimal therapy. And of course there are some patients in whom retesting is a no-brainer, like those with a family history of gastric cancer or a prior symptomatic documented ulcer.

Dr. Falk: Or patients with a bleeding ulcer. Yet post-treatment testing is still often not done. Nimish, what do practice guidelines say on this score?

Dr. Nimish Vakil: The Maastricht 2 Consensus Report from 2000² recommends that retesting should be offered to all patients after *H pylori* eradication therapy, and the updated Maastricht report that will be issued in 2005 is likely to recommend post-treatment testing for all patients even more strongly. At the time you prescribe therapy, you should tell the patient, "We will bring you back after the treatment is completed and test you again to make sure the infection is gone," and you should schedule the patient's post-treatment test at that time.

Dr. Falk: And it goes without saying that the post-treatment test should not be serology. At what point should you do the post-treatment test?

Dr. Vakil: It should be done 4 weeks out from the completion of therapy, and when the patient has been off proton pump inhibitor (PPI) therapy for at least 2 weeks, since recent PPI use can cause false-negative results with either active test. Histamine₂-receptor blockers can be used as antisecretory salvage therapy during this time if necessary, but ideally you should have the patient off all acid suppressants.

Dr. Brian Fennerty: The beauty of that approach is that taking them off all acid suppressants might address the question of whether you are really treating reflux disease.

Dr. Fendrick: One more key point is that there is no guarantee that this patient's symp-

toms will go away even if his infection is cured. *H pylori* eradication does not necessarily equal symptom resolution. Patients must clearly understand that up front and understand that there is value in eradicating the bacterium even without symptom resolution. Eradication with persistence of symptoms is not a failed outcome.

■ WHY CARE ABOUT FALSE POSITIVES?

Dr. Falk: Let me back up and focus on David's rationale for choosing active testing over serology. The rationale was essentially that serology is inferior to active testing in sensitivity and especially specificity, which produces a lot of false positives in a low-prevalence setting. If I'm the average clinician, how much is that going to resonate with me? After all, if I have a test with 80% sensitivity and 80% specificity, I'm apt to think that's not too bad, especially if the biggest concern is getting false positives. My biggest worry is about missing a diagnosis—getting a false negative—so why should I care about false positives?

Dr. Fendrick: On the false-negative question, the specter of false negatives should loom somewhat less large as *H pylori* prevalence declines, and most US physicians now practice in fairly low-prevalence settings.

But the real reason to care about false positives is because of the patients whom I call TPNI—"true positive for antibody, not infected." With an antibody test like serology, the "false-positive" results include both actual false positives for active infection and these TPNI results. When people hear "false positive," they think a test is not doing what it is supposed to do. But in actuality, when serology produces TPNI results it is doing exactly what it is supposed to do—detect antibodies. So even when serology is doing its job well, it is not doing well for the patient.

The result, as we've discussed, is that you end up treating a lot of people who aren't actively infected, which wastes a lot of resources, inconveniences a lot of patients, and contributes to antibiotic resistance.

Dr. Fennerty: I think a helpful analogy is to hepatitis B—we would never get a hepatitis B surface antibody and assume that it is an active hepatitis infection. Physicians understand that.

Schedule the post-treatment test to confirm *H pylori* cure at the same time you prescribe eradication therapy.

—Dr. Nimish Vakil

■ INDIVIDUAL PATIENTS TRANSCEND PREVALENCE

Dr. Vakil: When we speak of low-prevalence settings, as we have just now, it's important not to give the impression that *H pylori* prevalence is a yes/no phenomenon. Given the melting pot that we have in the United States, there are very few physicians who have a truly low-prevalence situation in all patients at all times.

In Wisconsin, where I practice, we have a low-prevalence population overall, and for our white middle-class patients born in suburban or rural parts of the state, you can argue that *H pylori* is a low-prevalence matter to the point of being almost a nonissue. But no physician in Wisconsin sees only patients like this, except in remote areas. In all of Wisconsin's cities we have large Hmong populations, we have immigrants from Mexico and South America, we have immigrants from Eastern Europe and Russia. The *H pylori* prevalence in these groups is 80% to 90%. So while there are some portions of the community for whom *H pylori* is almost a nonissue, there are other portions for whom it is a huge issue and whose needs are not being met. If a patient who moved here from Mexico walks into your office, you cannot apply the dynamics of a low-prevalence population to him.

Dr. Falk: You are absolutely right—everyone is being lumped together when the risk is quite different in different populations. Physicians need to realize how much their assessment of the likelihood of infection must be individualized (see **Table 2** on page S11).

Dr. Vakil: Many physicians recognize that Hispanics are at increased risk, but they don't realize the degree of risk for many other groups of recent immigrants. For example, the older epidemiologic studies wouldn't suggest elevated risk for Eastern Europeans, but recent immigrants from Eastern Europe have quite a high prevalence, and they are the largest group of immigrants coming to certain parts of the United States.

Dr. Ben Gold: That type of demography-based risk information can be a valuable tool for a primary care physician who is evaluating a particular patient, especially as it relates to disease phenotype. I consistently see children adopted from Eastern Europe who have 90% infection rates by

5 years of age and typically have multi-drug-resistant strains of *H pylori*. The infection is associated with a lot of morbidity in these children. It's important for primary care physicians to realize that there are these epidemiologic pockets.

Dr. Vakil: Yes, and this idea applies generally to gastric cancer as well. For instance, South Americans who are infected with *H pylori* have about five to ten times the risk of developing gastric cancer as an *H pylori*-infected native white Wisconsinite does. And while I do not disagree with the point in your presentation, Brian, about the gaps in the current data on the *H pylori*/gastric cancer connection, if I were a South American who was infected with *H pylori*, I'd be a bit worried. So I think it's reasonable to test for the organism in patients with a family history of gastric cancer, particularly if they belong to a high-risk population like this or they request testing.

Dr. Fennerty: I agree. The problem is that in an adult population there is no evidence that we can change the risk of gastric cancer even if *H pylori* is detected and treated. We just need to be careful not to encourage widespread testing to look for gastric cancer risk in patients without these high-risk factors.

■ IMPLICATIONS OF INCORRECT DIAGNOSTIC STRATEGIES

Dr. Falk: Before we go into some more focused areas of discussion, let's wrap up this discussion of *H pylori* testing in general by reviewing the specific implications of an incorrect diagnostic strategy. David, would you comment on the clinical implications of an incorrect strategy?

Dr. Wyatt: The most fundamental one is that you will inappropriately treat more patients who are not infected, which means putting patients through needless regimens of PPIs or histamine₂ blockers plus antibiotics. Because there is no infection for these treatments to address, the patient won't get symptom resolution, which will likely lead you to throw additional treatments at them or to order additional, and probably costly, diagnostic studies. Additional office visits will undoubtedly result. The cost inefficiencies of all of this are obvious. But there are also big costs in terms of patient inconvenience, patient dissatisfaction, and lost time—the wasting of all parties'

Patients must be told that *H pylori* eradication does not necessarily mean symptom resolution.

—Dr. Mark Fendrick

time and also a delay during which the patients' symptoms are not being properly addressed.

Dr. Derek van Amerongen: The effects on the patient are key. It seems clear to me, from a managed care perspective, that *H pylori* is not going to have a large cost impact on health care systems as a whole, but we need to be sensitive to the cost impact on the individual patient, in terms of out-of-pocket costs. As the patient works his or her way through the unnecessary treatment and then the additional testing to clarify what's really going on, the cost impact can be significant for a family trying to stretch its health care dollar. There also are emotional impacts for patients from being treated and not getting better, which undermines the credibility of the medical system, and we know that's already shaky these days.

Dr. Falk: Ben, would you summarize the biologic implications of an inappropriate diagnostic strategy and the resulting inappropriate therapy?

Dr. Gold: The most obvious implication is a burgeoning of antibiotic resistance, including multi-drug-resistant organisms, from the inappropriate and nonjudicious use of antibiotics. And it's not just among *H pylori*—other pathogens are being affected. We are seeing an increase over time in multi-drug-resistant and monotherapy-resistant strains of organisms in the United States, Canada, and Europe. Clarithromycin resistance has almost doubled in the last 7 years. Amoxicillin resistance is now being described. Tetracycline resistance, which 5 years ago was unreported, is now being described. Of course, there has been a strong push lately among many medical specialty groups to reduce inappropriate antibiotic use for these reasons.

The second implication is reduced efficacy of our treatments for *H pylori* infection, which is obviously related to the first. Eradication rates following therapy are no longer the proverbial 90%+ rates first reported but have fallen to around 75% or lower, owing to the increase in antibiotic resistance. This has implications on disease outcome as well as on transmission of the organism to yet-uninfected people.

Dr. Vakil: Plus, remember that most of the treatment studies are at least 5 or 6 years old, and resistance rates have risen since then. So the rates you mentioned are probably overoptimistic.

Dr. Leonard Ehrlich: From my experience in community office settings, I think there is a disconnect on the outpatient side, where there are many physicians who think that the treatment is still very effective and do not retest to check for cure. Despite our earlier discussion of this issue, the message about the need to retest and the high rate of therapeutic failure has not been well disseminated.

■ SHOULD WE TEST FOR *H PYLORI* PRIOR TO NSAID THERAPY?

Dr. Falk: Let's change gears and consider a controversy that comes up a lot—whether we should test patients for *H pylori* prior to therapy with a nonsteroidal anti-inflammatory drug (NSAID). This question appears to involve two different settings—the NSAID-naïve patient and the chronic NSAID user. You touched on this in your presentation, Brian, but let's explore it a little more in this forum. Can you get us started?

Dr. Fennerty: There are three main issues. First, there are no conclusions to be drawn from US data, and the Asian and European data do not always correlate with US outcomes. Second, many of the global societies' recommendations that will be coming out soon will recommend testing and treating for *H pylori* in the naïve NSAID user who is starting therapy, based on the Asian and European data. That is not, however, the position of the American College of Gastroenterology or the American Gastroenterological Association. Third, there is no evidence that testing for *H pylori* is justified in chronic NSAID users, which raises the specter of inevitable confusion if you advocate testing for naïve NSAID users but not for chronic users. Moreover, few patients are actually starting NSAIDs for the first time but are rather switching from one NSAID to another or from a COX-2 inhibitor to an NSAID, which breeds further confusion.

Dr. Fendrick: *H pylori* and NSAID use are clearly independent risk factors for ulcer. One can argue that, as in other diseases, you should intervene on as many levels, via as many mechanisms, as you can, at least in patients who are at highest risk for ulcer. From that standpoint, testing for *H pylori* before starting NSAID therapy in such patients would probably be good practice, but I agree with Brian

You cannot apply the dynamics of a low-prevalence population to a patient who moved here from Mexico.

—Dr. Nimish Vakil

that the evidence is simply not conclusive.

Dr. Vakil: This is a difficult question because if you make a blanket recommendation for it, then you are looking at mass testing of all the geriatric patients in their 80s and 90s who are taking NSAIDs, and the data are too weak to recommend that. At the same time, I agree with Mark that it would seem to be a no-brainer in patients at high risk for ulcer, although that tends to be geriatric patients. Further clarification is awaited, and eradication should be considered on a case-by-case basis.

Dr. Falk: The life of a primary care physician is complicated enough, and since we all agree that data are currently insufficient to make a clear, evidence-based recommendation, I don't think anyone should be hunting for *H pylori* before they give an NSAID or before they suggest prophylaxis with aspirin. Exceptions would be if the patient has a history of ulcer disease or any other clear-cut risk for *H pylori* infection, such as family history of gastric cancer.

Dr. Fennerty: One thing that we can separate out is that there are no data suggesting that testing for *H pylori* is an effective strategy in the aspirin user. That is not even controversial at this point.

Dr. Falk: I agree, but for NSAID users I think it is safe to say that right now we just don't know what to do in these patients. This is an evolving area, however, and clearer data are sure to emerge.

■ H PYLORI IN PEDIATRIC PATIENTS

Dr. Falk: Ben, you specialize in pediatric gastroenterology. Can you give us an overview of considerations for the diagnosis of *H pylori* infection in pediatric patients?

Dr. Gold: The epidemiology of *H pylori* infection and the diseases with which it seems to be associated are broadly similar in children and adults. Because most people acquire the organism in childhood, the epidemiology in children is especially important. It's notable that the prevalence of the organism in US children 6 to 19 years of age fell from 25% in the 1988–1991 National Health and Nutrition Examination Survey (NHANES) to 11% in the 1999–2000 NHANES. The prevalence among non-

Hispanic white children in the 1999–2000 survey was a mere 5%.³

Three consensus guidelines have been published on *H pylori* infection in children and adolescents,^{4–6} and the conclusions I will share are drawn from these guidelines along with the Maastricht 2 Consensus Report.² There currently are no recommended indications for noninvasive testing for *H pylori* infection in pediatric patients in the primary care setting, at least in the United States (in Europe, the recommended approach may differ). Specifically, testing is not recommended in asymptomatic children or in children without documented ulcer. Testing should be reserved for children with endoscopically documented duodenal or gastric ulcers and therefore can be done with an invasive test in conjunction with endoscopy. Recurrent abdominal pain or nonulcer dyspepsia is not a sufficient indication for screening children at this time.

Dr. Fendrick: So you're saying that primary care physicians shouldn't test for *H pylori* in children, that testing shouldn't be considered until the child has reached a point where he or she will have been referred to a gastroenterologist?

Dr. Gold: Based on the best available evidence, that's correct. At the specialist level, there are other indications for testing in children, such as following treatment of documented infection or if there is pathologic evidence of mucosa-associated lymphoid tissue (MALT) lymphoma. Screening also can be considered in children with a family history of gastric cancer in a first-degree relative or in children with recurrent peptic ulcer disease, although these uses are not yet endorsed by guidelines. The noninvasive tests for active infection (urea breath test and stool antigen test) may be considered in these settings and for confirmatory testing after treatment, as their characteristics are similar in children and adults. However, use of these noninvasive tests is not universally recommended by current pediatric guidelines. As in adults, current serologic tests are unreliable and not recommended for use in children.

■ THE ECONOMICS OF INCORRECT DIAGNOSTIC STRATEGIES: DO PAYERS GET IT?

Dr. Falk: We've touched on some of the economic implications of doing things right versus doing things wrong. I'd like to explore the economic implications of testing strategies a

Treating patients without active infection promotes antibiotic resistance among both *H pylori* and other pathogens.

—Dr. Ben Gold

little more fully. Derek, from your vantage point as chief medical officer of a large health plan, how do payers look at testing for a condition like *H pylori* infection?

Dr. van Amerongen: Payers see the costs of testing as far more than the cost of the test itself. There's the cost at the site of service—is the test done in the primary care physician's office, as noninvasive tests can be, or in a specialist's office? There's also the cost of false negatives, which is tremendously important because it means missing the disease, which can lead down false diagnostic paths, carries additional costs, and represents a missed opportunity for avoiding worse disease later. From an employer perspective, false negatives represent a missed opportunity to improve the patient's functionality and productivity.

Then there is the cost of false positives, which in the case of *H pylori* includes the cost of unneeded and inappropriate antisecretory and antibiotic therapy, as well as the cost of repeat testing to confirm eradication of an organism that wasn't there to begin with. The payer is also sensitive to out-of-pocket costs to the patient, which can be substantial in the case of false-positive or false-negative results, as I said earlier.

Dr. Falk: What are some general principles by which payers evaluate testing strategies?

Dr. van Amerongen: First, testing should adhere strictly to guidelines and reflect the best science. Second, the test should be readily accessible; it does no good to endorse a test that no one can get. Third, the implications of the test must stand up to the increasing patient scrutiny that comes with growing patient cost-sharing. Patients are increasingly going to ask, "What is the cost of this test?" and "What will we do next after this test is done?" The latter question leads to the final principle, which is that the test must be clearly integrated with effective therapy. If a test is not going to potentially change what you do, you should never use it.

Based on our discussion today, active testing for *H pylori* satisfies all of these principles rather well, with the possible exception of ready accessibility, which I assume we will be discussing later.

Dr. Vakil: Derek, you represent one health plan. How do you respond to concerns that,

despite all we have discussed, some managed care organizations might still look at \$10 for serology and \$100 for an active test and say, "We're going to use the \$10 test, at least up front"? I've encountered managed care plans that still put out algorithms on test and treat that recommend a sequential testing strategy that calls for low-cost serologic testing up front.

Dr. van Amerongen: That represents a misunderstanding of what managed care is all about. From day one, managed care has promoted adherence to national expert protocols and a rational approach to care. Payers need to focus on the ultimate goal, which is to diagnose people accurately and get them the test or therapy that is most effective for their problem. It's short-sighted to say, "Let's use the cheapest test even though it doesn't work." The smartest thing is to cut to the chase, go to the first-line approach first, which is why they call it first-line, and use the test or therapy that is most likely to get the best outcome. If you tell employers or consumers or payers that this test costs X but it leads to the best outcomes, the response typically will be, "Then that's what it costs."

Dr. Fendrick: The most expensive test is the one that doesn't work.

Dr. Ehrlich: I agree, and my experience is consistent with Derek's. In the managed care companies where I worked, it was always "the most appropriate test, the most appropriate setting," and so on. When we did continuous quality improvement initiatives at those companies, we would bring in experts to present to us, and then we would develop a guideline based on that expert opinion and disseminate it to our providers.

I think that's the approach that's needed now for *H pylori* testing. Wherever sequential testing with serology first is still being done, it has to be abandoned once and for all. For companies that manage active testing under prior authorization, they need to be reeducated to buy into active testing more fully. Based on what I've heard today, I think the evidence is solid and the message is pretty simple—specificity is not good enough with serology, patients are being misdiagnosed, drug resistance is rising. These messages will resonate with the employers Derek mentioned who are asking about both value and quality.

We don't yet know what to recommend about testing for *H pylori* in NSAID users, but clearer data will emerge.
—Dr. Gary Falk

Dr. Vakil: I agree that you can build a clear case that managed care organizations should reevaluate their dyspepsia management strategies. The timing is good, because an updated dyspepsia guideline from the American Gastroenterological Association is coming out later in 2005, and it can serve to bolster efforts to convince health plans and educate their providers.

Dr. Fennerty: Before we leave this economic discussion, I'd like to say that I'm glad it didn't get too caught up in the issue of cost-effectiveness or cost savings. Ultimately, active testing for *H pylori* is not an issue of cost; it's an issue of best medicine. It's a matter of the accurate diagnosis of an infectious disease so that you can employ a specific treatment for that disease. And testing for active infection blows away serologic testing—that's the real issue.

■ HOW DO WE BREAK THE SEROLOGY REFLEX?

Dr. Falk: Nimish, you've been writing and speaking on this subject for years. Why is it such a huge challenge to break physicians, be they primary care doctors or gastroenterologists, of the serologic testing reflex? It seems like it shouldn't be so hard, since we are not talking about huge expenses here or a highly controversial or emotion-laden clinical issue.

Dr. Vakil: Two things happened at the same time. Just as we began to change our thinking about testing and treatment, general interest in the whole *H pylori* issue started to ebb and the number of conferences, meetings, and discussions suddenly dropped off. And so the last message that primary care physicians were left with was test and treat, based on Mark's original work, which correctly told them that this is what you should be doing. And at that time, which was about 10 years ago, serology was the noninvasive testing option. That was before the noninvasive active tests were marketed and before the prevalence had changed so much.

Dr. Falk: So what can be done now to break the serology habit?

Dr. Vakil: I think there are two simultaneous components. One is education, and the second is access, and they are interwoven because when you educate physicians about this, if they don't have immediate access to one of the active tests, they can't implement what they have learned.

And neither the urea breath test nor the stool antigen test is readily available right now to the average physician in most parts of the country.

The reason physicians tend to incorporate new drugs into their practice is that as soon as they get educated about a drug, they get samples of the drug that they can try. If a physician hears some expert speak at a conference about these highly accurate active tests for *H pylori*, she is likely to go back home, call her lab, and inquire about the active tests. If she's told that they don't have the tests, her interest and openness is likely to end there.

Of course, this is the responsibility of the manufacturers of the tests for active infection. There have been different roadblocks to access in the past, including contracting issues with health plans, reimbursement rates, and diagnostic coding and paperwork issues. In different regions of the country there are different access hurdles. For example, in Wisconsin, where I practice, it is very difficult to get reimbursed for the urea breath test. But by increasing the availability of both tests, you solve that problem. There are signs that the manufacturers' efforts to do this may be increasing. We can hope so, because without good access, education is really undermined.

Dr. Falk: Let's just suppose access is no longer a problem. How do you educate physicians in today's climate of declining educational dollars and the lack of a therapeutic interest that's pushing this issue? Let's go around the table with this.

Dr. Vakil: No single event ever changes physicians' practices. The education needs to be a repetitive, iterative process in which physicians are reached by different modalities saying the same thing.

Dr. Gold: I agree, and the education needs to be data-driven to make them rethink their process, and ideally supported by guidelines in which a medical society's imprimatur provides further validation that this is the way to go.

Dr. Vakil: Fortunately, that will be coming later in 2005 in the form of the updated American Gastroenterological Association guideline on dyspepsia and an American College of Gastroenterology guideline on the same subject.

Dr. Wyatt: And physicians should be hearing the same thing from their managed care organizations and health networks as well,

Any health plans that manage active testing under prior authorization need to rethink that practice.

—Dr. Leonard Ehrlich

telling them, “Look, we no longer support the serologic testing strategy.” It’s absolutely got to be a multiphase, repetitive effort.

Dr. van Amerongen: Disseminating guidelines is important, but the literature on their success is disheartening. I think one way to get change to happen more quickly is to empower consumers, to get them to engage in a much more meaningful discussion with their physicians about their health and about testing and treatment that they may or may not need. When a physician has even a handful of patients come and ask about a particular issue, that physician will begin to change his or her practice. And studies show that when patients actively engage their physicians, care decisions change—invariably for the better.

Dr. Ehrlich: I think the key points have been covered. I would add that I’d like to see what the gastroenterologists are doing about this issue nationally. Frankly, in the hospitals with which I have been associated I have not seen active testing being done by gastroenterologists, let alone by primary care physicians. Also, I think it would be interesting to try to convince managed care organizations to require prior authorization before blood serology could be ordered for *H pylori* testing, and to make the tests for active infection available without prior authorization.

Dr. Fennerty: While I love that idea, Len, it would cost more to implement that plan than

you’d gain from any cost savings, so this will always fall below the radar screen of managed care decision-makers.

Dr. Falk: Mark, you get the last word.

Dr. Fendrick: Actually, the way to get managed care organizations and primary care physicians to adopt a practice is to make it a HEDIS (Health Plan Employer Data and Information Set) measure for National Committee for Quality Assurance accreditation. Unfortunately, given the smaller scale of this issue and the declining prevalence of *H pylori*, I’m not sure we’ll ever get managed care medical directors to devote attention to manipulating the utilization of these tests in an important way. If physicians want active tests enough, they’ll find a way to get them to be more available and accepted.

On the question of the serology reflex, I am somewhat optimistic that if the suggestions that have been offered here come to pass, we can break this thing. The literature on the “dis-adoption” of bad practices is unbelievably poor with the rare exception of when you have something that’s better to take the place of the practice you want to disadopt. In this case we clearly do. But this is where the question of access comes in, because Nimish’s point about physicians’ ability to try out the active tests soon after learning about them is crucial. Let’s hope for the best on the access front, because I am optimistic that we will build on this effort and do the right things on the education front.

**Ultimately,
active testing for
H pylori is not
an issue of cost
but an issue of
best medicine.**

—Dr. Brian Fennerty

■ REFERENCES

1. Fendrick AM, Chey WD, Margaret N, Palaniappan J, Fennerty MB. Symptom status and the desire for *Helicobacter pylori* confirmatory testing after eradication therapy in patients with peptic ulcer disease. *Am J Med* 1999; 107:133–136.
2. Malfertheiner P, Megraud F, O’Morain C, et al. Current concepts in the management of *Helicobacter pylori* infection: the Maastricht 2-2000 Consensus Report. *Aliment Pharmacol Ther* 2002; 16:167–180.
3. Gold BD, Kruszon-Moran D, Sobel L, McQuillan GM, Everhart J. Decreasing seroprevalence of *Helicobacter pylori* infection in U.S. children ages 6–19 years [abstract]. *J Pediatr Gastroenterol Nutr* 2003; 37:360. Abstract 99.
4. Sherman P, Hassall E, Hunt RH, et al. Canadian *Helicobacter* Study Group Consensus Conference on the approach to *Helicobacter pylori* infection in children and adolescents. *Can J Gastroenterol* 1999; 13:553–559.
5. Drumm B, Koletzko S, Oderda G, European Paediatric Task Force on *Helicobacter pylori*. *Helicobacter pylori* infection in children: a consensus statement. *J Paediatr Gastroenterol Nutr* 2000; 30:207–213.
6. Gold BD, Colletti RB, Abbott M, et al; North American Society for Pediatric Gastroenterology and Nutrition. *Helicobacter pylori* infection in children: recommendations for diagnosis and treatment. *J Pediatr Gastroenterol Nutr* 2000; 31:490–497.