he may only be replacing one pathogenic organism with another. Graham et al[6] have implicated isotretinoin in the development of a case of *S aureus* endocarditis in a patient receiving isotretinoin for extensive actinic keratoses.

Problems encountered in the treatment of patients such as these include how to distinguish infection from simple colonization and how to treat recurrent infections. Asymptomatic colonization does not require treatment except in special cases, such as the family member of a patient with recurrent staphylococcal disease or the surgeon whose patients develop frequent infections with his or her own colonizing strain. The symptomatic patient with local or systemic signs of inflammation generally responds to a defined course of specific therapy, but if a history of recurrent disease is present, a culture specimen should be obtained from the patient two to six weeks after completion of antibiotic use to determine his or her carrier status. For such patients, the role of compulsive hand washing cannot be overemphasized, as the organism is spread by direct inoculation. Personal items such as towels and clothes should not be shared and should be washed thoroughly.

Single-drug regimens are frequently inadequate to eliminate the carrier state, and the use of two antistaphylococcal agents, such as rifampin, 300 mg orally twice a day, plus cloxacillin, 250 mg orally four times a day for 14 days, may be required. Daily nasal instillation of neomycin or bacitracin ointments has been used extensively, and limited success has been reported in some patients experimentally recolonized with a less pathogenic strain of staphylococci, such as 502A.[7] None of these therapeutic maneuvers are clearly superior or free from failures, and satisfactory long-term success can be difficult to achieve.

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1. Miles AA, Williams REO, Clayton-Cooper B: The carriage of *Staphylococcus aureus* in man and its relation to wound infection. *J Pathol Bacteriol* 1944;56:513-524.
2. Fekety FR: The epidemiology and prevention of staphylococcal infection. *Medicine* 1964;43:593.
3. Godfrey ME, Smith IM: Hospital hazards of staphylococcic sepsis. *JAMA* 1958;166:1197-1201.
4. Ballou WR, Cross AS, Williams DY, et al: Colonization of newly arrived house staff by virulent staphylococcal phage types endemic to a hospital environment. *J Clin Microbiol* 1986;23:1030-1033.
5. Shalita AR, Cunningham WJ, Leyden JJ: Isotretinoin treatment of acne and related disorders: An update. *J Am Acad Dermatol* 1983;9:629-638.
6. Graham ML, Corey R, Califf R, et al: Isotretinoin and *Staphylococcus aureus* infection: A possible association. *Arch Dermatol* 1986;122:815-817.
7. Drutz DJ, van Way MH, Schaffner W, et al: Bacterial interference in the therapy of recurrent staphylococcal infections: Multiple abscesses due to the implantation of the 502A strain of *Staphylococcus aureus*. *N Engl J Med* 1966;275:1161-1165.

Campylobacter pylori: Diagnosis and Treatment

Q It is taught that barium interferes with the ecology of the gastrointestinal (GI) tract and for that reason, in patients being worked up for a diarrheal illness, culture specimens should be obtained prior to x-ray examination, or perhaps as long as six weeks after, depending on the nature of the illness. How long should one wait to do upper GI tract endoscopy and biopsies in a patient that you suspect as having *Campylobacter pylori* infection but who has just completed an upper GI tract series? Is it worth treating the patient while awaiting endoscopy if all possible alternative diagnoses have been ruled out? Is it justifiable to treat the patient at that time, since the barium may actually have changed the ecology of the upper GI tract flora?

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A The inquiring physician raises some very interesting and controversial points regarding the diagnosis and treatment of *C pylori*. Although no studies have specifically addressed this point, *Campylobacters* are sensitive to heavy metals,[1] so they may also be sensitive to the barium salts used in the upper GI tract series. A number of my patients have reported marked lessening of their symptoms following a barium meal examination, and on many occasions, when they have undergone gastroscopy one week after the x-ray, the ulcers have been much smaller or even healed. It is possible that an upper GI tract series has an ulcer-healing effect due to suppression of *C pylori*. For this reason I recommend that patients wait 48 hours after x-ray before undergoing a biopsy to detect the organism.

At the present time, the frequency of *C pylori* infection in dyspeptic patients has not been determined in the United States. We expect that most persons with a proved duodenal ulcer, 70% of gastric ulcer patients, and about 50% of patients with nonulcer dyspepsia will harbor the organism.[2,3] The infection should always be confirmed by endoscopic gastric antral biopsy before treatment is begun. Without such proof, many patients who do not have *C pylori* will be treated unsuccessfully with antibacterial drugs, and the therapy will fall into disrepute.

Once a firm diagnosis has been established, the end point of therapy is eradication of the organism. Eradication needs to be confirmed by another antral biopsy taken one month after completion of therapy. In patients who do not have an endoscopic lesion, noninvasive diagnosis and follow-up of *C pylori* with serology[4] or a carbon 14 breath test may be a convenient alternative.[5]

I advise physicians to exclude *C pylori* infection in all patients with dyspepsia but not to raise their hopes of cure until the infection has been proved on biopsy. In patients who are content with conventional H_2-receptor antagonist therapy, I do not interfere by changing them to an antibacterial regimen, but I inform them that the bacterium is present and that antibiotic therapy may be a useful future option. In patients who have troublesome ulcer disease that is not effectively controlled by current therapy, I advise antibacterial therapy. I warn the patient that the cure rate for antibiotic treatment may not be more than 50% and that many persons will require two or even three courses of drugs. Preliminary reports state that most patients improve symptomatically once the gastritis has healed,[6] although in some cases this improvement takes place gradually after eradication of the organism.

Both the physician and the patient should be aware that antibacterial therapy for *C pylori* has not been proved to be useful outside the setting of duodenal ulcer disease. There is considerable anecdotal experience, however, and physicians will agree that many patients with dyspepsia and gastritis have no other useful alternatives.

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1. Marshall BJ, Armstrong JA, Francis GJ, et al: The antibacterial action of bismuth in relation to *Campylobacter pyloridis* colonization and gastritis. *Digestion* 1987;37(suppl 2):16-30.
2. Marshall BJ, McGechie DB, Hislop IG, et al: Pyloric campylobacter infection and gastroduodenal disease. *Med J Aust* 1985;149:439-444.
3. Rokkas T, Pursey C, Uzoechina E, et al: *Campylobacter pylori* and non-ulcer dyspepsia. *Am J Gastroenterol* 1987;82:1149-1152.
4. Goodwin CS, Blincow E, Peterson G, et al: Enzyme-linked immunosorbent assay for *Campylobacter pyloridis:* Correlation with presence of *C. pyloridis* in the gastric mucosa. *J Infect Dis* 1987;155:488-494.
5. Marshall BJ, Surveyor I: Carbon-14 urea breath test for the diagnosis of *Campylobacter pylori* associated gastritis. *J Nucl Med* 1988;29:11-16.
6. Borody TJ, Carrick J, Hazell SL: Symptoms improve after the eradication of gastric *Campylobacter pyloridis*. *Med J Aust* 1987;146:450-451.