Letters to the Editor

The Journal welcomes letters to the editor. If found suitable, they will be published as space allows. Letters should be typed double-spaced, should not exceed 400 words, and are subject to abridgment and other editorial changes in accordance with Journal style. All letters that reference a recently published Journal article are sent to the original authors for their reply. If no reply is published, the authors have not responded by date of publication. Send letters to Paul M. Fischer, MD, Editor, The Journal of Family Practice, 519 Pleasant Home Rd, Suite A-3, Augusta, GA 30907-3500, or Fax (706) 855-1107. E-mail: jfampract@aol.com

CHRONIC INFECTION AND ASTHMA

To the Editor:

We read with interest the recent *Journal* article concerning the possible relationship between *Chlamydia pneumoniae* and asthma.[1] While the author raises some intriguing questions regarding a relationship between *C pneumoniae* infection and asthma, he has failed to account for some clinical issues that make his interpretation of the data suspect.

First, many of the subjects in this study may have had chronic sinusitis, a frequently neglected diagnosis[2] and cause of asthma exacerbations.[3–5] As a result of intermittent aspiration of chronic, purulent postnasal drainage, this entity may cause airway inflammation and bronchospasm in susceptible individuals. Treatment of the infection requires a 3- to 6-week course of antibiotics, in contrast to acute sinusitis, which generally responds to a 7- to 10-day course of therapy. In cases refractory to medical therapy, surgical treatment may be required to drain obstructed sinuses. Following adequate treatment, elimination of the postnasal drainage results in significantly improved asthma symptoms and a decreased requirement for asthma pharmacotherapy.

In the absence of any pretreatment sinus evaluation, it is difficult to conclude that antibacterial therapy has provided any additional effect beyond the treatment of chronic sinusitis. Of note, *C pneumoniae* is not among the common causative agents of chronic sinusitis.

Second, this study utilized a polyvalent serologic assay for *C pneumoniae*. Epidemiologic studies have shown *C pneumoniae* seropositivity in up to 63% of individuals.[6] Following acute infection or reinfection, patients may have seroreactivity to *C pneumoniae* for years.[7] The serologic titers for the patients in this study did not change significantly, suggesting remote infection, making this an incidental finding with questionable relationship to asthma.

Bruce Sherman, MD
Byschelle Jesberger, RN
Division of Pulmonary and Critical Care Medicine
Mt Sinai Medical Center
Cleveland, Ohio

References

1. Hahn DL. Treatment of *Chlamydia pneumoniae* infection in adult asthma: a before-after trial. J Fam Pract 1995; 41:345–51.
2. Chester AC. Chronic sinusitis and the internist. Arch Intern Med 1994; 154:133–5.
3. Schwartz HJ, Thompson JS, Sher TH, Ross RJ. Occult sinus abnormalities in the asthmatic patient. Arch Intern Med 1987; 147: 2194–6.
4. Rachelefsky GS, Katz RM, Siegel SC. Chronic sinus disease with associated reactive airway disease in children. Pediatrics 1984; 73:526–9.
5. Herrera AM, deShazo RD. Sinusitis: its association with asthma. Postgrad Med 1990; 87:153–64.
6. Hahn DL, Dodge RW, Golubjatnikov R. Association of *Chlamydia pneumoniae* (strain TWAR) infection with wheezing, asthmatic bronchitis, and adult-onset asthma. JAMA 1991; 266:225–30.
7. Aldous MB, Grayston JT, Wang S. Seroepidemiology of *Chlamydia pneumoniae* TWAR infection in Seattle families, 1966–1979. J Infect Dis 1992; 166:646–9.

The preceding letter was referred to Dr Hahn, who responds as follows:

Sherman and Jesberger raise two important points regarding the interpretation of a possible antibiotic effect against asthma.

They are correct to point out that sinusitis is often associated with asthma, and that selected patients who have concurrent sinusitis and asthma can show improvement in asthma after aggressive antibiotic and/or surgical management of sinusitis. However, the majority of study responders having radiologic evaluation had no evidence for chronic sinusitis. Sinus radiographs were not routinely obtained but were performed as clinically indicated for symptoms or for screening in selected individuals. None of the 25 responders had clinical sinusitis, although one responder who did not have a pretreatment sinus radiograph had a prior history of chronic sinusitis that was asymptomatic at the time of treatment. Five responders had normal sinus radiographs and four had abnormal radiographs. One abnormality was attributable to prior sinus surgery, one showed minimal mucosal thickening of uncertain clinical significance, and two were positive for sinusitis. No patient in this series had evidence of otitis media or other pyogenic bacterial infection. Furthermore, 16 of the 25 responders had pretreatment chest radiographs, all of which were negative for an acute infiltrate. It seems unlikely, therefore, that asthma improvement was due to an antibacterial effect against conventional pyogenic organisms causing disease in the sinuses or elsewhere in the respiratory tract.

Sherman and Jesberger state that *C pneumoniae* is not among the common causative agents of chronic sinusitis. This is a misleading statement, since *C pneumoniae* has not been evaluated adequately as a cause for chronic sinusitis. *C pneumoniae* is a recognized cause of acute sinusitis[1] and is capable of producing chronic respiratory infection.[2] A role for chronic *C pneumoniae* infection in chronic sinusitis is plausible but has not yet been investigated extensively. Since chronic chlamydial infections seldom yield cultivable organisms, such studies will require the use of nucleotide detection techniques such as the polymerase chain reaction (PCR) test. My colleagues (Robert Gilbert, Lee Ann Campbell and Pekka Saikku) and I recently evaluated a patient with chronic relapsing sinusitis who was positive for *C pneumoniae* by PCR and who also was seroreactive against *C pneumoniae*-specific IgA (Hahn DL, Gilbert R, Campbell LA, Saikku P. Unpublished observations). Seroreactivity against *C pneumoniae*-specific IgA is a probable serologic marker for chronic infection.[3] It would appear, therefore, that *C pneumoniae* is capable of causing chronic as well as acute sinusitis, but its quantitative contribution remains to be determined.

Regarding their second point, Sherman and Jesberger are correct to point out that polyvalent seroreactivity cannot distinguish previous infection from persistent chronic infection. They are incorrect, however, to state that unchanging titers suggest remote infection, since persisting titer levels over many years are more likely to indicate chronic infection or boosting from repeated reinfections. My colleagues (Roberta McDonald and Pekka Saikku) and I have developed evidence that asthma-associated polyvalent seroreactivity is actually due to seroreactivity against IgA, suggesting chronic infection.[4,5] However, further study will be

necessary to determine whether IgA will prove to be a useful clinical tool in diagnosis or treatment of asthma.

David L. Hahn, MD
Arcand Park Clinic
Madison, Wisconsin

References

1. Grayston JT, Aldous M, Easton A, Wang S-p, Kuo C-c, Campbell LA, Altman J. Evidence that *Chlamydia pneumoniae* causes pneumonia and bronchitis. J Infect Dis 1993; 168:1231–5.
2. Hammerschlag MR, Chirgwin K, Roblin PM, Gelling M, Dumornay W, Mandel L, Schachter J. Persistent infection with *Chlamydia pneumoniae* following acute respiratory illness. Clin Infect Dis 1992; 14:178–82.
3. Saikku P, Leinonen M, Tenkanen L, Linnanmäki E, Ekman M-R, Manninen V, Mänttäri M, Frick MH, Huttunen JK. Chronic *Chlamydia pneumoniae* infection as a risk factor for coronary heart disease in the Helsinki Heart Study. Ann Intern Med 1992; 116:273–8.
4. Hahn DL. Evidence for *Chlamydia pneumoniae* infection in asthma. In: Allegra L, Blasi F, eds. *Chlamydia pneumoniae* infection. Milan, Italy: Springer-Verlag, 1995: 65–75.
5. Hahn DL, Saikku P. Serologic evidence for *Chlamydia pneumoniae* infection in recently symptomatic asthma: a pilot case-control study. Am J Respir Crit Care Med 1995; 151(Pt 2 of 2):A470.

MMA TEST TO DETECT VITAMIN B_{12} DEFICIENCY

To the Editor:

Dr Swain's interesting review of vitamin B_{12} deficiency[1] fails to mention the availability of the most accurate test for identifying cobalamin deficiency: the spot urinary methylmalonic acid (MMA) test by gas chromatograph mass spectrometry (GC/MS).[2–4] In a clinical setting, this test has been reported to have a sensitivity of 100% and a specificity of 99%.[4]

For many years, our laboratory performed the urinary MMA assay by GC/MS using a 1-mL random-spot specimen that was mailed unrefrigerated in mailing tubes containing preservative. Creatinine levels are also measured, and MMA results are normalized to urine creatinine. The current cost is $50. Obtaining a 24-hour collection is inconvenient and MMA results may be unreliable, since it is difficult to guarantee that all urine is collected. In addition, hospital laboratories may analyze MMA using a colorimetric assay and not by GC/MS. The colorimetric assay lacks the sensitivity and specificity needed to differentiate slightly elevated MMA from normal MMA.

I concur with Dr Swain's recommendations to screen at-risk populations. However, careful thought must be given to selection of the screening test. A satisfactory screening test must be specific, sensitive, and acceptable to the population being screened. Using the serum cobalamin (Cbl) assay as a screening test will identify high numbers of nondeficient subjects that require additional expensive confirmatory testing. For example, the serum Cbl test has a positive predictive value of only 22.2%: nearly 4 of 5 values may be falsely positive.[5] Furthermore, the number of false positives will be inordinately high if the cutoff level for follow-up is increased to 350 pmol/L to increase the sensitivity of the serum Cbl assay. Thus, in evaluating the currently available means for screening for Cbl deficiency, the spot urinary MMA test by GC/MS is the preferred choice because of its cost, accuracy, convenience, and noninvasiveness.[6]

Eric J. Norman, PhD
Norman Clinical Laboratory, Inc.
Cincinnati, Ohio

References

1. Swain R. An Update of vitamin B_{12} metabolism and deficiency states. J Fam Pract 1995; 41:595–600.
2. Norman EJ. Gas chromatography mass spectrometry screening of urinary methylmalonic acid: early detection of vitamin B12 (cobalamin) deficiency to prevent permanent neurologic disability. GC-MS News 1984; 12:120–9.
3. Norman EJ, Morrison JA. Screening elderly populations for cobalamin (vitamin B12) deficiency using the urinary methylmalonic acid assay by gas chromatograph mass spectrometry. Am J Med 1993; 94: 589–94.
4. Matchar DB, Feussner JR, Millington DS, et al. Isotope-dilution assay for urinary methylmalonic acid in the diagnosis of vitamin B12 deficiency. Ann Intern Med 1987; 106:707–10.
5. Matchar DB, McCrory DC, Millington DS, Feussner JR. Performance of the serum cobalamin assay for diagnosis of cobalamin deficiency. Am J Med Sci 1994; 308:276–83.
6. Norman EJ, Cronin C. Urinary MMA for detection of cobalamin deficiency. Neurology 1996. In press.

The previous letter was referred to Dr Swain, who responds as follows:

I do agree that a spot urine would be much more satisfactory and feasible to obtain from a patient than a 24-hour collection. I do not agree, however, with Dr Norman's point that this should become the standard of screening. Cobalamin-deficient patients may have normal levels of MMA *(Savage DG, Lindenbaum J, Stabler SP, Allen RH. Sensitivity of methylmalonic acid and total homocysteine determinations for diagnosing cobalamin and folate deficiencies. Am J Hematol 1990; 34:90–8)*. Homocysteine levels are also needed to diagnose B_{12}-deficient patients. I suggest that B_{12} levels may be used for screening because this test is readily available and relatively inexpensive ($39). Usually you can rule out deficiency with this commonly available test.

Randall A. Swain, MD
Robert C. Byrd Health Sciences Center
West Virginia University
Charleston, West Virginia

TREATMENT OF BACTERIAL VAGINOSIS

To the Editor:

I found the study by Ferris and colleagues on the treatment of bacterial vaginosis[1] interesting and timely in confirming the efficacy of topical preparations for treatment of this disorder. I was puzzled, however, by the inclusion of use of a DNA probe for *Gardnerella vaginalis* as a diagnostic criterion. The study reported by Amsel et al[2] that resulted in widespread use of Amsel's criteria for diagnosis of bacterial vaginosis showed that *G vaginalis* was not invariably found in women with the clinical syndrome of bacterial vaginosis. In addition, *G vaginalis* has been cultured from asymptomatic women with no evidence for bacterial vaginosis.[3]

The discussion suggests that the results were similar in looking separately at groups identified by clinical criteria and by DNA probe and pH. Was this the case?

In looking at women with recurrent bacterial vaginosis, Cook et al[4] found residual *G vaginalis* in less than 20% of patients with recurrent bacterial vaginosis. They suggest that a persistence of total obligate anaerobes was more predictive of recurrence, as opposed to the presence of *G vaginalis* alone. It would be interesting to follow the women he has identified

with residual *G vaginalis* to see whether they do, in fact, have a higher recurrence rate.

Barbara A. Majeroni, MD
School of Medicine
and Biomedical Sciences
State University of New York at Buffalo
Buffalo, New York

References

1. Ferris D, Litaker MS, Woodward L, Mathis D, Hendrich J. Treatment of bacterial vaginosis: a comparison of oral metronidazole, metronidazole vaginal gel, and clindamycin vaginal cream. J Fam Pract 1995; 41:443–9.
2. Amsel R, Totten PA, Spiegel CA, Chen KC, Eschenbach D, Holmes KK. Nonspecific vaginitis: diagnostic criteria and microbial and epidemiological associations. Am J Med 1983; 74:14–22.
3. Eschenbach DA, Hillier S, Critchlow C, Stevens C, DeRouen T, Holmes KK. Diagnosis and clinical manifestations of bacterial vaginosis. Am J Obstet Gynecol 1988; 158: 819–28.
4. Cook RL, Redondo-Lopez V, Schmitt C, Meriwether C, Sobel J. Clinical, microbial, and biochemical factors in recurrent bacterial vaginosis. J Clin Microbiol 1992; 30: 870–7.

The preceding letter was referred to Dr Ferris, who responds as follows:

The DNA probe was added to the study for several reasons. First, there were limited published data available on its clinical performance. According to the manufacturer, the *G vaginalis* DNA positive test threshold was supposedly targeted at a high level to reliably discriminate between true infection indicative of bacterial vaginosis and simply colonization of *G vaginalis* in asymptomatic women. Second, the DNA probe assay also included tests for *Candida* sp and *Trichomonas vaginalis*, of which the former test was helpful for establishing test-of-cure-visit treatment complications. Finally, we fully anticipated poorer results with the *G vaginalis* DNA probe for the reasons mentioned by Dr Majeroni. The presence of *G vaginalis* alone is not a sufficient criterion for a reliable diagnosis of bacterial vaginosis, nor does its presence necessarily indicate treatment failure. We had hoped that the combination of the DNA results with a pH determination would improve test performance, but this was not the case.

Cure rates using the DNA probe for *G vaginalis* plus pH were approximately 20% lower than cure rates reported using Amsel's criteria. This lower cure rate is partially explained by the continued presence of *G vaginalis* at levels insufficient to produce an abnormal vaginal ecosystem. Because of funding limitations, we were unable to follow women with residual *G vaginalis* to examine for bacterial vaginosis recurrence. Further research is necessary to ascertain why the vast majority of women with bacterial vaginosis experience troublesome recurrences.

Daron G. Ferris, MD
Medical College of Georgia
Augusta, Georgia

CAROTIDYNIA RURALLY REVISITED

To the Editor:

This is a belated report of appreciation for the article about carotidynia *(Hill LM, Hastings G. Carotidynia: a pain syndrome. J Fam Pract 1994; 39:71–5)*. I had read and saved the article at that time, but secretly felt that in my rural setting, I would likely never see the syndrome myself. As if in response to that sentiment, a 42-year-old man presented with a 2-week history of neck pain. He stated that he had had a "viral cold" approximately 2½ weeks before presentation, and that as the "cold" receded, he experienced right anterior neck pain, which would often progress to a mild, diffuse headache. He had no neurological changes on review but did state that he had had two episodes of similar symptoms within the past year, which resolved over weeks without specific therapy.

The screening neurologic examination was within normal limits, as was the temporomandibular joint, thyroid, and overall HEENT evaluation. The patient's symptoms were reproducible by direct palpation of the right carotid impulse, just inferior to the bifurcation. The diagnosis of carotidynia was made, and the patient was sent for an ESR (normal at 6 mm/h). The patient was then instructed to go home and apply local heat and take oral ibuprofen 800 mg three times daily. He has since done very well, with full resolution of his pain.

It was wonderful to be able to review with the patient such a comprehensive and "user-friendly" article at the time of diagnosis, especially in light of the paucity of information on carotidynia in standard texts. Thank you for publishing this article and others similar to it; perhaps syndromes presently considered "uncommon" will become more widely recognized, as readable articles describing them become *more* mmon.

J.W. Hinterberger, MD
Dundee Primary Care Center
Dundee, New York

PSYCHOLOGICAL MANAGEMENT IN FAMILY PRACTICE

To the Editor:

I read with amusement and concern the articles in the December 1995 issue of the *Journal* having to do with the primary physician's treatment of psychological illness.[1,2] I think that much more counseling and pharmacologic treatment are being done than are being reported.

In my area, claims are automatically rejected by most of the insurance carriers if a diagnosis of depression or anxiety is listed on the claim form. We are told that only psychiatrists may treat these conditions. I work closely with a clinical psychologist in my community, and he refers patients to me for medical management when he feels the patient would benefit from medications. After evaluating the patient myself, I determine whether to start medications, and which ones would be most effective. I see the patient for follow-up on a regular basis, but have to code the visits as if I were seeing them for their secondary (somatic) complaints rather than for the primary (psychologic) disorder. Any statistical evaluation of treatment rendered, therefore, would suggest that I am "missing" the diagnosis of depression when, in fact, I am treating it.

We need to educate third-party payers that family physicians, internists, and pediatricians are, indeed, trained and qualified to treat these conditions in a cost-effective manner, so we do not have to keep playing games with the ICD-9.

Allan C. Edson, DO
Wauwatosa, Wisconsin

References

1. Olfson M, Weissman MM, Leon AC, et al. Psychological management by family physicians. J Fam Pract 1995; 41:543–50.
2. Rowe MG, Fleming MF, Barry KL, et al. Correlates of depression in primary care. J Fam Pract 1995; 6:551–8.

The preceding letter was referred to Dr Olfson, who responds as follows:

The practice that Dr Edson describes of intentionally misdiagnosing psychiatric disorders as general medical conditions appears to be widespread. A recent survey of primary care physicians revealed that fully one half (50.3%) had substituted another diagnosis for major depression during a 2-week period.[1] In this study, concern over reimbursement was one of the most commonly cited reasons for deliberately substituting other diagnoses.[1]

In our study, the data were collected on research questionnaires that were not related to billing forms or medical record entries.[2] For this reason, I suspect, but cannot prove, that deliberate misdiagnosis did not significantly alter our findings.

It is unfortunate that so many primary care physicians feel compelled to employ deceptive coding practices. Physicians who deliberately misdiagnose their patients may succeed in securing short-term financial savings for themselves and their patients. Reform, however, will occur only if discriminatory reimbursement policies are openly challenged. The current system too often rewards patients of physicians who deliberately misdiagnose psychiatric disorders and penalizes patients of physicians who accurately record these disorders. I urge Dr Edson to reconsider how he addresses this issue.

Mark Olfson, MD, MPH
Department of Psychiatry
College of Physicians & Surgeons of Columbia University
New York, NY

References

1. Rost K, Smith R, Matthews DB, Guise B. The deliberate misdiagnosis of major depression in primary care. Arch Fam Med 1994; 3:333–7.
2. Olfson M, Weissman MM, Leon AC, Higgins ES, Barrett JE, Blacklow RS. Psychological management by family physicians. J Fam Pract 1995; 41:543–50.

LATIN REDUX

To the Editor:

In a recent issue of *The Journal of Family Practice*, Matthew M. Eschelbach, DO, MS, authored an article entitled "Grandson of the Heimlich Maneuver" *(Eschelbach MM. Grandson of the Heimlich maneuver. J Fam Pract 1995; 41:505–6)*, which he concludes with, "Do no harm, but first laugh. I wonder how you say that in Latin."

After consulting a Latin and English dictionary from my high school days (I attended Regis, a Jesuit high school that required 3 years of Latin), I realized that "do no harm" is *non nocere*, and "but" is *sed*. For the word "first," I prefer *imprimis*, as it distinguishes this new phrase from the original, better-known one. "Laugh" is *ridere*. Therefore, "do no harm, but first laugh" is: *Non nocere, sed imprimis ridere.*

Should you have any questions or further consults in Latin, please feel free to contact me.

Len Scarpinato, DO
St Mary's Hospital Family Practice Center
Family Practice Residency Program
Medical College of Wisconsin
Milwaukee, Wisconsin

"WINDOW OF OPPORTUNITY" IN MANAGED CARE

To the Editor:

With the recently changing environment for the practice of medicine, there has been much written about how physicians will weather the "storms of change." I would like to discuss with other primary care physicians a "window of opportunity" that has recently become evident to me.

My wife and I are family physicians. In our community, managed care is just starting. We have recently purchased a home from an anesthesiologist who has decided to move to another community to practice. We bought our new home at an extremely reasonable price, and it is in a nice neighborhood that houses no other "primary care physicians." Our neighbors are somewhat surprised that we work weekends, go back to the hospital after hours, and have a listed phone number. Yet, in general, they have accepted us without too much furor.

It might be appropriate for family physicians to begin the process of home shopping by contacting their realtor and asking specifically to see the homes of cardiology, anesthesia, and radiology colleagues. A little anticipatory shopping might offer wonderful opportunities for primary care physicians in this realm of managed care. I have never envisioned myself in a Maserati . . . who would have ever dreamed . . . ?

J.M. Pontious, MD
OU/Enid Family Medicine
Enid, Oklahoma

HUMOROUS ANECDOTES

To the Editor:

As a family physician in private practice, I have a continuous supply of humorous anecdotes.

I recently performed a rectal examination on one of my patients. In an effort to reduce the anxiety associated with this procedure, I told the patient how truly wondrous the anal sphincter is. "Here is a muscle that can distinguish between solid, liquid, and gas, selectively letting one out, while with remarkable consistency, containing the others," I said.

At this point, the patient turned her head and said, "So the next time someone calls me an asshole, I guess I should take it as a compliment."

Clearly, humor is present in all aspects of the human condition.

Ira G. Warshaw, MD
Palm Beach Gardens, Florida

Erratum

In the article "Appetite Suppressants as Adjuncts in the Treatment of Obesity" *(Elks ML. J Fam Pract 1995; 42:287–92)*, the scheduling status of Prozac was incorrectly stated on the table on page 288. Prozac is *not* a schedule IV drug but an unscheduled drug. US classification schedules indicate potential for abuse on a scale of I (highest potential) to V (lowest potential). The *Journal* regrets the error.