counts patient and physician biases as "unlikely [to] account for persistent objective improvement in pulmonary function" but does not further address the likelihood that the results could reflect a placebo response. Placebo responses of the magnitude reported by Hahn in the FEV_1 from pre- to posttreatment (12.5%, 95% confidence interval, 4.6% to 20.3%) have been reported in randomized controlled trials of asthma treatment. For example, average improvements of 11% to 18% in serial FEV_1 measurements 1 to 6 hours after administration of a placebo were reported in one study of 12 subjects.[2] An increase of 6% in FEV_1 from baseline, averaged over 12 hourly measures, was noted at 12 weeks of follow-up for 77 placebo controls in another study.[3] The mechanisms by which the well-known placebo effect operates are not known, but in the case of FEV_1, they could include regression to the mean, reduction in anxiety, and improvements in learning and effort. I am concerned that publication of this article will be taken as preliminary evidence of treatment efficacy. The lack of a control group means there is no assurance that the effect being reported is other than a placebo response.

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

I thank Dr Thom for reemphasizing for readers of *The Journal of Family Practice* the limitations of uncontrolled clinical interventions in asthma. I share his concern that my published results could be overinterpreted and that "the danger of promiscuous overuse accompanies any recommendation for empiric antibiotic treatment based solely on uncontrolled clinical observations."[1] An anonymous reviewer from another journal stated that it was a "tragedy" that my study was not a randomized, placebo-controlled, double-blind trial. I remain uncertain how an investigator can attract funding to perform such a trial without first publishing promising preliminary results.

Dr Thom cites pre- and post-bronchodilator FEV_1 results for placebo groups in two studies of bronchodilator treatment for asthma. Since my study[2] reported on changes in pre-bronchodilator results only, its results may be compared more appropriately with equivalent pre-bronchodilator data from randomized controlled trials of chronic inhaled steroid treatment for asthma.[3–6]

The difference between baseline (pretreatment) and follow-up FEV_1 in the control groups can be used as a measure of the natural history of lung function in asthmatics not receiving anti-inflammatory treatment. Pre-bronchodilator FEV_1 declined in control groups treated for 6 weeks,[4] 1 year,[3] 2 years,[5] and 2½ years.[6] This decline in FEV_1 over time in asthmatics who are not receiving inhaled corticosteroids may be (1) due to worsening asthma symptoms, (2) a manifestation of the well-known loss of FEV_1 that occurs in asthma, or (3) both. This decline in pre-bronchodilator FEV_1 must be distinguished from the acute changes following bronchodilator use cited by Dr Thom.

Another interesting exercise is to compare study FEV_1 results (12% improvement)[2] with the improvement in FEV_1 for patient groups treated with inhaled steroids. Improvement in FEV_1 during ongoing inhaled steroid administration of between 6 weeks and 2½ years' duration was 7%,[4] 0%,[3] 4%,[5] and 12%,[6] respectively. Thus, my study results could be due to nonspecific effects (placebo response) that would (1) be equal to or greater than the magnitude of the proven effect of long-term inhaled steroid administration on pre-bronchodilator FEV_1, (2) be associated with delayed clinical improvement consistent with (but not conclusive proof for) resolution of an inflammatory response as shown in Figure 1 of my study,[2] and (3) result in apparent remission of asthma in 15% of the patient group. I believe the expression "rather dramatic" does apply to these results, whether they are due to nonspecific effects, nonantibiotic effects of the administered agents, or the hypothesized antimicrobial effect. Future studies should clarify the situation.

David L Hahn, MD
Arcand Park Clinic
Madison, Wisconsin

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TOBACCO ADVERTISING IN PHARMACIES

To the Editor:

Recently, I went into a pharmacy where some of my patients fill their prescriptions. Next to the pharmacy cash register, a large sign advertising cigarettes proclaimed, "Alive with pleasure." The manager said it was paid advertising and that the tobacco company dictates where each sign is placed, even on the pharmacy counter where medicines are dispensed.

Pharmacies are symbols of healing and health. By placing these advertisements where medicines are dispensed, tobacco companies imply that tobacco products are conducive to health. Advertising can influence those who are sick, many of whom have illnesses directly related to tobacco use and may be attempting to quit smoking. Such advertising may also persuade children that tobacco products are associated with health and well-being. Since most tobacco users begin using tobacco before the age of 18 years, youngsters may be influenced by tobacco advertising.[1,2]

Pharmacies should not allow tobacco advertising. Physicians should encourage their patients, especially those

ho have been advised to quit smoking
d those with children, to purchase their
escriptions at pharmacies in which to-
acco is not advertised. The presence of
bacco advertising in pharmacies is fur-
er evidence that such advertising
ould be regulated by the Food and
rug Administration.

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2. US Department of Health and Human Services. Preventing tobacco use among young people: a report of the surgeon general. Rockville, Md: US Department of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, 1994:5, 65, 88.

WHAT'S IN A NAME?

To the Editor:

When I was young, my friends and I played a game of making up names for people based on their occupations. Given today's multiplicity of specialists, the game seemed like a natural for our profession. Here's what I came up with. Does anyone have other names to contribute?

Addictionist, I. Drinkwater, MD
Anesthesiologist, Bonnie Gasser, MD
Cardiologist, Anne Jinnah, MD
Dermatologist, I. deWart, MD
Diabetologist, P. Sweet, MD
Emergency physician, B. Quick, MD
Endocrinologist, Libby Doe, MD
Family physician, C. A. Lott, MD
Gastroenterologist, Manny Scopes, MD
General surgeon, Kurt Manner, MD
Geneticist, Jean Poole, MD
Geriatrician, Leif Sinding, MD
Gynecologist, Deanne See, MD
Hematologist, Eck E. Moses, MD
Infectious disease specialist, Cole Chivers, MD
Internist, Noah Bunche, MD
Neurologist, E. E. Ghee, MD
Neurosurgeon, A. Burr Hohl, MD
Obstetrician, Kid Cumming, MD
Oncologist, N. Mustarde, MD
Ophthalmologist, Will Seawell, MD
Orthopedic surgeon, Aitken Bach, MD
Otolaryngologist, Addie Noyes, MD
Pathologist, Topsy Dewar, MD
Pediatrician, Bebe Chalmers, MD
Plastic surgeon, Faye Swift, MD
Proctologist, Seymour Bottoms, MD
Psychiatrist, Izzy Batty, MD
Pulmonologist, Les Coffman, MD
Radiologist, A. Katz Canning, MD
Rheumatologist, Daley Payne, MD
Sports medicine specialist, Will Wynne, MD
Urologist, Nita Sample, MD

S. Spence Meighan, MD
Portland, Oregon

ELEVATED ESR IN STROKE

To the Editor:

I read the report by Tharakan et al[1] with keen interest. The authors state that "high ESR, raised IgM, and presence of monoclonal IgM-λ band on electrophoresis established the diagnosis of Waldenström's macroglobulinemia [WM]," and that "bone marrow showed few atypical plasma cells." The bone marrow aspiration is often hypocellular with WM, but the bone marrow biopsy reveals hypercellularity and is extensively infiltrated with lymphoid cells. The number of plasma cells are increased and normal marrow component decreased.[2] Also, 75% to 80% of IgM protein in WM are of κ-light chain, contrary to that seen in the patient in the present report who had IgM-λ chain.[2,3]

Based on the information given in the report, it is impossible to diagnose WM in the patient. The data are indistinguishable from an IgM myeloma. WM and IgM myeloma follow a similar clinical course. WM is often associated with hepatosplenomegaly, while IgM myeloma is associated with lytic bone lesions. The light chain isotope may have an impact on survival. Patients with IgM myeloma secreting λ-light chains, such as the one in the present report, have significantly shorter overall survival than those secreting κ-light chains.[2,3]

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2. Kyle RA. Multiple myeloma and other plasma cell disorders. In: Hoffman R, Benz EJ, Shattil SJ, Furie B, Cohen HJ, Silberstein LE, eds. Hematology: basic principles and practice. 2nd ed. New York, NY: Churchill Livingstone, 1995:1354–74.
3. Longo DL. Plasma cell disorders. In: Wilson JD, Braunwald E, Isselbacher KJ, Petersdorf RG, Martin JB, Fauci AS, Root RK, eds. Principles of internal medicine. 12th ed. New York, NY: McGraw-Hill, 1991:1410–7.

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

This patient has no evidence of osteolytic lesions, and his serum calcium is normal. Bone marrow biopsy showed few plasmacytoid lymphocytes, in addition to atypical plasma cells. His serum viscosity was elevated and he had neurological symptoms and signs. All these features occurring together in a patient with elevated IgM favors the diagnosis of WM more than that of IgM myeloma *(Thomas JK. Macroglobulinemia. In: Earnest B, Marshall AL, Barry S, Thomas JK, eds. Williams hematology. 5th ed. New York, NY: McGraw-Hill, 1995:1127–32.)*. It is well known that the light chain of IgM is constituted by kappa in 75% and lambda in the remaining 25% of WM patients. Therefore, elevation of IgM lambda chain, as seen in this patient, does not argue against the diagnosis of WM.

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