counts patient and physician biases as “unlikely to account for persistent ob-
jective 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
FEV1 from pre- to post-treatment (12.5%,
95% confidence interval, 4.6% to 20.8%)
have been reported in randomized con-
trolled trials of asthma treatment. For
example, average improvements of 11% to
18% in serial FEV1 measurements 1 to 6
hours after administration of a placebo
were reported in one study of 12
subjects.3 An increase of 6% in FEV1 from
baseline, averaged over 12 hourly mea-
sures, was noted at 12 weeks of follow-up
for 77 placebo controls in another study.4
The mechanisms by which the wel-
known placebo effect operates are not
known, but in the case of FEV1, they
would 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 effi-
cacy. The lack of a control group means
there is no assurance that the effect being
reported is other than a placebo response.

David Thom, MD, PhD
Stanford University
Palo Alto, California

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2. Pinnas JL, Schachet BP, Chen TM, Rose-
berry HR, Thoden WR. Inhaled epineph-
rine and oral theophylline in the treat-
ment of asthma. J Clin Pharmacol 1991;
31:242-7.
3. Pearlman DS, Chevinsky P, LaForce C, et
al. A comparison of salmeterol with albuterol
in the treatment of mild-to-moderate

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

I thank Dr Thom for reemphasizing for
readers of The Journal of Family Practice
the limitations of uncontrolled clinical inter-
ventions in asthma. I share his concern that
my published results could be over-
interpreted and that “the danger of pro-
miscuous overuse accompanies any rec-
ommendation for empiric antibiotic treat-
ment based solely on uncontrolled clinical
observations.”5 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 in-
vestigator can attract funding to perform
such a trial without first publishing prom-
ising preliminary results.

Dr Thom cites pre- and post-brocho-
dilator FEV1 results for placebo groups in
two studies of bronchodilator treatment
for asthma. Since my study2 reported on
changes in pre-brochodilator results only,
its results may be compared more
appropriately with equivalent pre-
bronchodilator data from randomized con-
trolled trials of chronic inhaled steroid
treatment for asthma.3-6

The difference between baseline (pre-
treatment) and follow-up FEV1 in the
group controls can be used as a measure
of the magnitude of improvement in
asthma not receiving anti-inflammatory
treatment. Pre-brochodilator FEV1 declined in control groups treated for
6 weeks,1 1 year,3 2 years,6 and 2 1/2 years.6
This decline in FEV1 over time in asthmatics
who are not receiving inhaled cortico-
oids may be (1) due to worsening asthma
symptoms, (2) a manifestation of the wel-
known loss of FEV1 that occurs in asthma,
or (3) both. This decline in pre-brocho-
dilator FEV1 must be distinguished from
the acute changes following bronchodila-
tor use cited by Dr Thom.

Another interesting exercise is to com-
pare study FEV1 results (12% improve-
ment)2 with the improvement in FEV1
for patient groups treated with inhaled
steroids. Improvement in FEV1 during
ongoing inhaled steroid administration of
between 6 weeks and 2 1/2 years’ duration
was 7%,4 9%,3 4%,5 and 12%,6 respec-
tively. Thus, my study results could be
due to nonspecific effects (placebo re-
sponse) that would (1) be equal to or
greater than the magnitude of the proven
effect of long-term inhaled steroid admin-
istration on pre-brochodilator FEV1,
(2) be associated with delayed clinical im-
provement consistent with (but not con-
clusive proof for) resolution of an inflam-
natory response as shown in Figure 1 of
my study,2 and (3) result in apparent re-
mission 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 ef-
fects, nonantibiotic effects of the admin-
istered agents, or the hypothesized anti-
microbial effect. Future studies should
clarify the situation.

David L Haim, MD
Arcad Park Clinic
Madison, Wisconsin

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1 new twist to an old problem. J Fam Pract
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3. Juniper EF, Kline PF, Vanziegelema GH,
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of long-term treatment with inhaled
corticoestroid (budesonide) on airway
hyperresponsiveness and clinical asthma in
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4. Vathenen AS, Knox AJ, Wiesneck AT, Tit-
tersfield AE. Time course of change in
bronchial reactivity with an inhaled corti-
coestroid in asthma. Am Rev Respir Dis 1991;
143:1317-21.
5. Haathela T, Järvinen M, Kava T, Kiviranta
K, Koskinen S, Lehtonen K, et al. Compar-
ison of a β2-agonist, terbutaline, with an
inhaled corticoestroid, budesonide, in a
6. Kerstjens HM, Brand PLP, Hughes MD,
Robinson NJ, Postma DS, Slater A, et al.
A comparison of bronchodilator therapy
with or without inhaled corticoestroid ther-
apy for obstructive airways disease. N Engl J

TOBACCO ADVERTISING IN PHARMACIES

To the Editor:

Recently, I went into a pharmacy where
some of my patients fill their pre-
scriptions. Next to the pharmacy cash-
register, a large sign advertising cigarettes
proclaimed, “Alive with pleasure.” The man-
ger said it was paid advertising and that the tobacco company
dictates where each sign is placed, even
on the pharmacy counter where medi-
cines are dispensed.

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

Pharmacies should not allow to-
bacco advertising. Physicians should en-
courage their patients, especially those
who have been and those with pre-existing
asthma or diabetes, to refuse tobacco ad-
vertising. Other evidence, if available,
should be required.

Drug Adm

1. Pierce JP, Rosbrook DF. Does tobacco
2. US Department of Health and Human
Services: Preventing tobacco use among
young people: a report of the Surgeon
Printing Office on behalf of the United
Preventing tobacco use: 1981-1991; Vol II;
Health consequences of smoking; Vol III;
Economic consequences of smoking.

WHAT’S NEW IN MEDICINE

Letters to the Editor


Letters to the Editor

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, L. Drinkwater, MD
Anesthesiologist, Bonnie Gasser, MD
Cardiologist, Anne Jinnah, MD
Dermatologist, I. deWart, MD
Dietologist, 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
Gyneciatrician, 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 Cummings, MD
Oncologist, N. Mustarde, MD
Ophthalmologist, Will Seawell, MD
Orthopedic surgeon, Aitken Bach, MD
Ortolaryngologist, Addie Noyes, MD
Pathologist, Topsy Dewar, MD
Pediatrician, Bebe Chalmers, MD
Plastic surgeon, Faye Swift, MD
Psychologist, 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 with keen interest. The authors state that "high ESR, raised IgM, and presence of monoclonal IgM-λ band on electrophoresis established the diagnosis of Waldenstrom’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. Also, 75% to 80% of IgM protein in WM are of κ-light chain, contrary to that seen in the report in the present report who had IgM-λ.

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 hepatitis.

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.

John K. J. Tharakan, MD
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Sultanate of Oman

References


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


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