DIAGNOSIS OF CANDIDA VULVOVAGINITIS

To the Editor:

Vaginitis is a very common health problem, yet it can also be one of the most frustrating to patients and their physicians. Making a valid diagnosis based on symptoms and physical examination is difficult. Several studies have shown that the diagnosis of *Candida* vulvovaginitis based on clinical criteria is often erroneous compared with diagnosis by culture.[1,2] Since the routine use of vaginal cultures has not become the standard of care, however, diagnostic errors continue.

In the past, the symptom of vaginal itching has been attributed to *Candida* vulvovaginitis; but several studies suggest this complaint alone is not reliable.[3-5] Vaginal itching ranges from minimal discomfort to an overwhelming urge to scratch. After discussing the extent of itching with many patients over the years, we became aware of a subset of patients with severe itching who, in most cases, had culture-proven *Candida* infections. Those patients often expressed the desire to scratch with something (although few ever actually did so). We were eventually able to identify an object of appropriate shape and texture that women with this symptom agreed was what they desired—a picket fence. We termed this clue the "picket fence sign." We therefore designed the following study.

All patients with vaginal complaints were asked if they had itching. If they responded affirmatively, they were then asked if the itching was severe. Those who replied yes to this question were asked the following: "Some patients with vaginal itching have said they itched so bad that they wanted to scratch with a picket fence. Is this how you feel?" If the patient said no or was unsure, she was not entered in the study, and was evaluated and treated routinely in the office. If, however, she emphatically agreed, a vaginal culture was added to the routine evaluation to identify *Candida albicans* as well as *Gardnerella vaginalis* in the vaginal discharge.

Of 11 consecutive patients admitted to the study, 10 were culture-positive for *Candida albicans* (a positive predictive value of 91%). The one patient with a negative culture did not have an obvious etiology by culture but did respond promptly to miconazole intravaginal cream.

The sensitivity of the picket fence sign cannot be determined from this study. Past studies indicate that itching of any degree may only be present in 50% of patients with *Candida* vulvovaginitis.[3,5] Therefore, the presence of *severe* itching would be present in an even lower percentage. Nevertheless, this study suggests that the presence of the picket fence sign in a woman with vaginal symptoms is highly predictive of *Candida* vulvovaginitis, and may obviate the need for culture confirmation prior to a course of presumptive treatment. We suggest further evaluation of this simple, rapid, in-office test for the differentiation of patients with symptomatic vaginitis.

Philip Zazove, MD
West Jordan Medical Center
Salt Lake City, Utah

Barbara D. Reed, MD, MSPH
University of Utah Medical Center
Salt Lake City, Utah

Werner Huck, M(ASCP)
Hucsto, Inc.
Salt Lake City, Utah

Stephen Stahl, MD
West Jordan Medical Center
Salt Lake City, Utah

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OBSTETRICS IN FAMILY PRACTICE

We read with great interest "Present Status of Obstetrics in Family Practice and the Effects of Malpractice Issues" by Bredfeldt, Colliver, and Wesley in the March issue of the *Journal (J Fam Pract 1989; 28:294–297).* We would certainly agree with their premise that obstetrics remains an essential element of family medicine. We have recently conducted a survey of 1987 graduates of all of the family medicine training programs in the state of Georgia.[1]

Our data are in agreement with those of Bredfeldt and colleagues regarding the regional differences in the practice of obstetrics, especially regarding the southern region: only 21% of our responding graduates planned to include obstetrics in their practices.

Our data, however, suggested a significant difference with the conclusions of these authors about underlying causes for rejection of obstetric practice. We may be more in agreement with Klein,[2] who has suggested that malpractice issues are a "scapegoat" for the declining practice of obstetrics within family medicine. Our data clearly suggested that personal and professional lifestyle issues were at least as important as malpractice-litigation issues to our 1987 graduates who decided not to include obstetrics in their practice.

As Bredfeldt and colleagues have written, shrinking obstetric practice within family medicine is a complex issue. Likewise, it is doubtless the case

that malpractice-litigation issues have seriously contributed to the decline. However, and especially because politically active physicians have used declining numbers of physicians who are willing to deliver babies as a lever to influence legislative malpractice reform, we must be careful not to paint a distorted picture. Although recent legislative events have given hope for significant malpractice reform, we would suggest that other issues will need to be addressed by individuals entering the field of family medicine and by family medicine training programs before we see family physicians flocking back to the delivery suite. If and when our malpractice premiums ever decline, family physicians will still be faced with the question of whether they wish to leave their homes at night or a waiting room full of patients during office hours to go deliver a baby.

James L. Fletcher, Jr., MD
Meyer P. Schwartz, MD
Department of Family Medicine
Medical College of Georgia
Augusta

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SMOKING AND GLYCOSYLATED HEMOGLOBIN

To the Editor:

The article by Urberg et al[1] on the effects of cigarette smoking on glycosylated hemoglobin is of great interest. A number of questions come to mind from this study.

First, it would have been helpful to further categorize the subjects of the study in terms of age, sex, weight, and other medical conditions that might lead to elevated glycosylated hemoglobin fractions (eg, iron deficiency anemia, splenectomy, hypertriglyceridemia, or alcoholism).

Second, while the normal fasting blood glucose rules out overt diabetes, it does not preclude the possibility of occult impaired glucose tolerance. Glucose tolerance testing would have shed further light on the significance of their study.

Finally, it would lend further support to their conclusion if increasing fractions of glycosylated hemoglobin were found to correlate with increasing quantity of cigarette consumption. Cigarette consumption could be assayed in a variety of ways.

Until these questions are answered, the significance of this study remains uncertain.

Doug Trotter, MD
Snohomish Family Medical Center
Snohomish, Washington

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1. Urberg M, Shammas R, Rajdev K: The effects of cigarette smoking on glycosylated hemoglobin in nondiabetic individuals. J Fam Pract 1989; 28:529–531

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

Dr. Trotter makes several significant comments about our article relating cigarette smoking to elevations in glycosylated hemoglobin levels in humans.

The subjects in our study were chosen to have normal cholesterol levels and to be different only in that some smoked one pack or more of cigarettes a day and the rest did not smoke at all. This was done to maximize the chances of finding a significant difference between the groups, and that difference was found. To our knowledge, age, sex, and weight have only small effects on glycosylated hemoglobin levels in normoglycemic subjects, and differences between smokers and nonsmokers in our subjects is unlikely to explain the magnitude of the effect seen. The effects of medical conditions on the level of glycosylated hemoglobin in normoglycemic individuals have not yet been systematically studied. Our subjects were in generally good health, however.

Normal fasting blood glucoses cannot rule out impairments in glucose tolerance; however, there is no reason to believe that the normoglycemic smokers had a substantially greater incidence of impaired glucose metabolism than the nonsmokers in our study group. That fact that the smokers in our study had a slightly lower fasting glucose than the nonsmokers supports that conclusion. The glucose tolerance test is not a very sensitive measure of glucose metabolism in normal subjects. The literature on using glucose measurements to document a hyperglycemic effect of smoking was reviewed in the paper and was generally negative.

The problem of quantitating nicotine exposure in smokers is complicated. The number of cigarettes smoked per day is only an approximate measure. Smoking technique varies widely, as does the nicotine content of cigarettes. Nicotine blood levels are available from regional research laboratories, but are expensive. Further, the nicotine level of blood in smokers changes rapidly following smoking. Finally, since blood nicotine levels are very low, one must be scrupulously careful in protecting the blood samples from ambient cigarette smoke, which makes the determination of blood nicotine levels in an actively smoking subject technically difficult. To justify a study of such magnitude and cost, it is first necessary to demonstrate that smoking has a significant effect on blood glucose. This paper was intended to do just that.

Further studies are needed to address Dr. Trotter's questions. I would encourage him and other family physicians to carry out these studies.

Martin Urberg, MD, PhD
Wayne State University
Detroit, Michigan

SERUM CHOLESTEROL AND GLYCOSYLATED HEMOGLOBIN

To the Editor:

This letter is in response to the three letters by Dr. Perez, Dr. Knudson and Dr. Neighbor *(Serum cholesterol and glycosylated hemoglobin. J Fam Pract 1989; 28:731–732),* which ap-

peared in the June issue of the Journal in response to our paper "A Correlation Between Serum Cholesterol and Glycosylated Hemoglobin in Nondiabetic Humans."[1]

Dr. Perez makes several valid observations about the relationship between glucose metabolism and disease. He cites one of Dr. Yudkin's many publications relating sugar intake to atherosclerosis. It is unclear why Dr. Yudkin has fallen into disfavor; however, as evidence relating glucose metabolism to atherosclerosis mounts, his work may be reexamined.

Dr. Knudson makes several editorial comments that reflect the very high standards of academic excellence which are coming to be accepted by the family practice community. These standards will serve us well as we move into the mainstream of academic medical research. His criticisms are, however, rather harsh and do not reflect the realities of the current state of knowledge regarding glycosylated hemoglobin levels in nondiabetic individuals. Subject selection in our study was consistent with the practice in other published studies in similar fields. Our study population was defined by age, sex, and the absence of diabetes or atherosclerotic heart disease. Further, no subject was taking any medications known to affect either glucose metabolism or cholesterol levels. A fasting blood glucose was measured to rule out any unknown diabetes. Sosenko et al[2] did not measure glucose levels at all. The selection of subjects was clearly described and was not random. Subjects were chosen to represent a wide range of cholesterol levels, while at the same time having no other known significant risk factors for atherosclerotic heart disease. This stratification by cholesterol levels was done to maximize the chance of finding a significant correlation if one existed. This strategy was successful, as the paper demonstrated. Studies that use different designs are likely to find smaller correlations between cholesterol and glycosylated hemoglobin.

Significant confounders for a study such as ours are many, and it is difficult to address all of them in a small

continued on page 326

LETTERS TO THE EDITOR

continued from page 241

study with limited resources. Further, it is becoming clear that we do not know which confounders are significant in studies of glycosylated hemoglobin in a nondiabetic population. Barrett-Connor et al[3] found no significant correlation between age and Hb A_1, and this finding has been confirmed in other studies recently. Cigarette smoking, on the other hand, has a profound effect on glycosylated hemoglobin in normoglycemic, normocholesterolemic subjects.[4] Smoking was not addressed by Sosenko et al[2] nor by Barrett-Conner et al.[3] We had three smokers in our study, all of whom had higher glycosylated hemoglobin levels than would be expected from their cholesterol levels. Including significant numbers of smokers in the study group would be expected to decrease the observed correlation between glycosylated hemoglobin and cholesterol. We did not control for obesity, but most of our subjects were normal weight. Obesity did not significantly affect the magnitude of the Hb A_1, cholesterol correlation in the study of Barrett-Connor et al,[3] however.

The paper by Barrett-Connor et al[3] came to my attention after our paper appeared in print. This is unfortunate because their results complement ours, and because Barrett-Connor is one of the most prominent researchers in family medicine. They found significant positive correlations between total cholesterol and Hb A_1 for men ($r = .12$) and for women ($r = .20$) which are consistent with our findings but of much smaller magnitude. There are several differences in the two study designs, which may account for the differences in correlation coefficients found. First, they used Hb A_1 as their measure of glycosylated hemoglobin, while we used the boronate affinity gel technique. While the latter technique is more precise, Hb A_{1c} correlates quite well with average blood glucose. Second, while the number of smokers in our study was small, we do not know the number of smokers in their study. Third, and perhaps most important, we took 5.83 mmol/L (105 mg/dL) as the upper limit of normal for serum glucose, while they used 7.78 mmol/L (140 mg/dL) as the upper limit of normal. Since subjects with fasting glucose levels in the range of 5.83 mmol/L and 7.78 mmol/L are likely to have mild impairments in glucose tolerance, it is not clear that the relationship of glycosylated hemoglobin to total cholesterol for these subjects is the same as in subjects with normal fasting glucose levels. Glycosylated hemoglobin is expected to be high in these subjects, but cholesterol may not be elevated as well.

The most significant findings in the study of Barrett-Connor et al[3] are that with a different study design which asked a different question than our study, they found a significant correlation between glycosylated hemoglobin and cholesterol, which is in the same direction as ours. Further, they were able to document the fact that age and obesity do not substantially detract from that correlation. Their study supports the general conclusions of our report.

Dr. Neighbor calculated the slope of the regression line between cholesterol and glycosylated hemoglobin in our study and found it to be substantially greater than that calculated from that data of Barrett-Connor et al.[3] This is an example of the phenomenon of regression toward the mean,[5] and is due to the fact that our correlation coefficient is greater than theirs.

Martin Urberg, MD, PhD
Wayne State University
Detroit, Michigan

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