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Postmenopausal Hormone Therapy: A Response to the Critics

SPECIAL REPORT

By Leon Speroff, MD

Editor's Note: *In this OB/GYN Clinical Alert Special Report, Dr. Speroff takes a timely look at current controversy involving postmenopausal hormone therapy. Please also notice the inclusion of Dr. Speroff's list of references at the conclusion of the report.*

IT SEEMS LIKE EVERYONE IS JUMPING ON THE BANDWAGON: USE postmenopausal hormone therapy for less than 5 years to treat menopausal symptoms. This position is based upon 3 arguments:

1. The data are insufficient to believe that estrogen will protect against fractures;
2. Recent studies indicate that estrogen will not reduce the risk of coronary heart disease;
3. Postmenopausal hormone therapy used for longer than 5 years will increase the risk of breast cancer.

Let's address each of these issues, and let's bend over backward to be objective, unlike some recent publications by critics of postmenopausal hormone therapy.

Estrogen and Fractures

The argument against estrogen is based upon the contention that conclusions should be based upon randomized clinical trials, and only bisphosphonates have been demonstrated to reduce the risk of fractures.[1-3] The facts are as follows: Estrogen and bisphosphonates are equally potent in their beneficial effect as measured by bone density.[1,4] Case-control and cohort studies indicate that estrogen will reduce the rate of fractures with the same efficacy as bisphosphonates.[5-9] And most importantly, there are 2 randomized trials (always ignored by the critics) documenting a beneficial effect of hormone therapy on fracture incidence. In a Finnish study, a 71% reduction in nonvertebral fractures was documented within 5 years of treatment, a result comparable to that predicted by the case-control and cohort data.[10] In a Danish ongoing randomized trial, there was a 39% reduction in the overall fracture rate after 5 years.[11] In both of these

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randomized trials, the women did not already have osteoporosis at the beginning of treatment. If you want to play the critic's game and be selective in your use of the literature, it would be appropriate to argue that bisphosphonates have been proven to be effective in reducing fractures only in women with documented osteoporosis, whereas hormone therapy has been proven to be effective in the primary prevention of osteoporosis and fractures. Likewise, raloxifene has been demonstrated to reduce fractures only in women with already demonstrated osteoporosis, and the reduction was observed only with spinal fractures, and not with hip or other-site fractures.[12]

Where does that leave us with bone? Hormone therapy prevents bone loss and fractures at all sites, even though long-term treatment is necessary to achieve this effect.[8,13] Bisphosphonates prevent bone loss, probably are effective in the primary prevention of osteoporosis and fractures, and for sure, reduce the fracture risk in women with osteoporosis. Raloxifene prevents bone loss (although it is less potent than estrogen and bisphosphonates[14]) and reduces spinal fracture risk in women with osteoporosis. Add to these conclusions the fact that hormone therapy is the least expensive of the treatment options.

Estrogen and the Cardiovascular System

The evidence of the secondary prevention trials is impressively uniform that hormone therapy will not reduce the risk of a subsequent cardiovascular event in women who already have significant, clinically apparent atherosclerosis.[15-17] It is not apparent whether the "small increase" in cardiovascular events reported in the ongoing Women's Health Initiative was influenced by the women in the study who had atherosclerosis upon entry (it is not a pure primary prevention trial). The American Heart Association recommended that hormone therapy not be initiated in women with atherosclerosis with the expectation that treatment will reduce the risk of a subsequent cardiovascular event.[18] I have no problem with that statement. We know statins are the drug of choice for these women, and furthermore, this group of women represents a very small part of our practice. I do have a problem with the second statement from the American Heart Association that said: no recommendation can be made regarding primary prevention until we have the results of randomized clinical trials. You can't practice medicine based only on randomized clinical trial data! If we based our recommendations only on clinical trial data, we would never tell our patients who smoke to stop smoking. There is an enormous collection of biological and observational data indicating that hormone therapy given to apparently healthy women will reduce the risk of coronary heart disease.[19] A primary prevention trial recently reported progression of atherosclerosis in the placebo group and slight improvement in the estrogen-treated group.[20] It seems to me that there is an emerging theme: it takes a healthy endothelium to respond to estrogen. Animal studies, including the primate, indicate that as the endothelium progressively is involved with atherosclerosis, it loses its ability to respond to estrogen.[21,22] Thus, there are good reasons to believe that estrogen will provide primary prevention of coronary heart disease. The major criticism, the "healthy user effect," will not be answered until we have results from the ongoing large trials. However, the difference in health comparing users and non-users has not been great enough to explain the observed beneficial effect. We also await clinical trial results to answer this important question: are the results in the secondary prevention trials due to a lack of an estrogen effect or is the beneficial effect of new statin treatment obscuring the positive action of estrogen? In the primary prevention trial noted

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above, the beneficial effect of estrogen could be documented only in women not using statins.[20]

In a very recent publication by one of the critics, it was suggested that because the risk of venous thromboembolism increases with age, the venous thrombosis risk with hormone therapy can be expected to be higher in elderly women.[23] However, the risk of venous thrombosis is almost exclusively concentrated in the first two years of use (data from the HERS trial with the critic as senior author[24]), and, therefore, long-term users have little, if any, risk of this side effect as they get older.

Hormone Therapy and Breast Cancer

It is almost fashionable to conclude that hormone therapy for longer than 5 years is associated with a slight increase in the risk of breast cancer, referring either to the collaborative re-analysis of the world's literature published in 1997 or the Nurses' Health Study.[25, 26] Of course, there are no randomized clinical trial data here. When we have only observational data, clinicians want to see uniformity, agreement, and consistency among the studies (like that seen with smoking and lung cancer or oral contraceptives and ovarian cancer). With postmenopausal hormone therapy and breast cancer, we have the opposite situation, a lack of uniformity, agreement, and consistency in over 60 studies. Sometimes clinicians believe that all the recent studies have consistently indicated an increased risk; however, that is not the case. The positive studies (increased risk) have received the publicity; the negative studies (no increased risk) have been ignored (and the negative studies impressively outnumber the positive studies).

In the practice of medicine, we make medical judgments every day. I define a "medical judgment" as making a decision when you don't have all the facts you need to make that decision. That is the art and skill of medicine, of being a clinician. This is one of those situations. Until we have randomized trial data (no earlier than 2006), the clinician must present the confusing picture to the patient. In my view, the evidence is consistent either with a slightly increased risk of breast cancer with long-term treatment or hormonal stimulation of preexisting tumors (an effect that is consistent with the uniformly observed reduced risk of dying of breast cancer in hormone users who develop the disease).

I believe it is appropriate to make the following three statements to patients:

1. The evidence does not indicate a major increase in risk of breast cancer associated with the long-term use of postmenopausal hormone therapy;
2. A small increase in risk of breast cancer or an increase with long duration of use is unlikely;
3. The use of hormone therapy does not increase the risk beyond that already associated with a positive family history of breast cancer (a positive family history is not a contraindication).

Conclusion

There continues to be good reason to believe that long-term use of postmenopausal hormone therapy is associated with preventive health benefits for the bones and the cardiovascular system. Inaccurate and nonobjective reporting in both the lay press and professional journals is unfairly enhancing the appropriate fear of breast cancer in our patients. It is time that some of us speak up to balance the scales. Postmenopausal hormone therapy is the most cost-effective treatment option available and the only one with a broad spectrum of beneficial effects for older women.

Finally, let me share an approach that I have found to be effective with patients. I like to tell patients that there is an enormous amount of research ongoing with postmenopausal women and hormone therapy. My obligation is to review each year the new information. Based on that review, the patient makes a new short-term decision. Thus, repetitive short-term decisions allow the patient to avoid being uncomfortable by the thought of a long-term commitment. I truly believe this approach improves long-term continuation rates. ■

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Dairy Consumption, Obesity, and the Insulin Resistance Syndrome in Young Adults: The CARDIA Study

ABSTRACT & COMMENTARY

Synopsis: *Dairy consumption was inversely associated with the incidence of all components of the insulin resistance syndrome (IRS) among individuals who were overweight (body mass index > 25 kg/m^2). These associations were similar for African Americans and Caucasians and for men and women. Other dietary factors did not explain the association between dairy intake and IRS.*

Source: Pereira MA, et al. *JAMA*. 2002;287: 2081-2089.

THIS STUDY EXAMINED ASSOCIATIONS BETWEEN DAIRY intake and the incidence of insulin resistance syndrome using data generated from the Coronary Artery Risk Development in Young Adults (CARDIA) study, which was a general community sample of 5115 African American and Caucasian adults aged 18-30 years who lived in 1 of 4 major US metropolitan areas. The participants were first seen in 1985-1986 and then again 10 years later. The main outcome variable, insulin resistance syndrome (IRS), is also known as metabolic syndrome or syndrome X. IRS is associated with glucose intolerance, dyslipidemia, hypertension, and impaired fibrinolytic capacity and is a risk factor for cardiovascular disease. The prevalence of IRS among adults has been estimated at 24%. While obesity clearly plays a role in the development of IRS, the role of dietary factors is less well understood. The current recommendation of a low-fat diet has been questioned, in part because its institution has been linked to increased carbohydrate consumption, which may also play a role in the development of IRS. Of the 3157 individuals eligible for participation in this study, 923 had a BMI > 25 kg/m^2. A thorough history of diet and other lifestyle variables was obtained. Waist-hip ratio was determined and blood was obtained after an 8-hour fast. Abnormal glucose homeostasis was defined as a fasting plasma insulin > 20 mU/mL, fasting glucose > 110 mg/dL, or use of medications to control blood glucose. Obesity was defined as a BMI > 30 or a waist-hip ratio > 0.85. An elevated blood pressure was defined as > 130/85 or the use of antihypertensive medications. Dyslipidemia was defined as HDL-C < 35 mg/dL or triglycerides > 200 mg/dL. Insulin resistance was defined as the presence of at least 2 of the following 4 components of the IRS: abnormal glucose homeostasis, obesity, elevated blood pressure, and dyslipidemia.

In general, overweight individuals consumed less dairy products. Notably, dairy consumption was positively associated with whole grain, fruit, vegetable, and saturated fat intake and inversely correlated with soft drink intake. No association was observed between dairy intake and incidence of IRS in normal weight participants. Among overweight individuals, regardless of race or sex, incidence of IRS decreased by more than 50% from lowest to highest categories of dairy consumption. Pereira and associates note that the inverse association between calcium intake and IRS was entirely explained by dairy intake. Pereira et al speculate that the complex mixture of lactose, protein, and fat in dairy foods may enhance satiety and reduce the likelihood of eating high-carbohydrate foods and beverages. Also, there may be other biologically active components in dairy foods. The lack of an association in underweight individuals likely reflects the low likelihood of developing IRS in the absence of obesity.

■ COMMENT BY SARAH L. BERGA, MD

This article struck me as having relevance for 2 populations of women often seen in the office, namely, postmenopausal women and women with polycystic ovary syndrome. Women with these conditions struggle to avoid persistent weight gain, commonly display dyslipidemia and hypertension, and are at risk for IRS. Furthermore, they are often seeking dietary recommendations. For many, weight loss is unlikely. However, it is important to recognize that they can change their diet by adding dairy foods, not lose weight, and still accrue health benefits, including a reduction in the risk of dia-

betes and cardiovascular disease. Another consideration is that dairy products may have health benefits for those who are of normal body weight and it may still be advisable to recommend inclusion of dairy in the diet.

How much dairy consumption is enough? In this study, there was a dose-response curve with no threshold or plateau. The lowest category was 0-10 dairy servings weekly and the highest category was > 35. Interestingly, those with a BMI < 25 average more servings of high-fat dairy products than did overweight individuals. This lends credence to the hypothesis that you need enough fat to turn off appetite.

When queried, most of my patients say they eat the majority of their calories at dinner. They tend to go home from work famished. This places them at risk of overeating at dinner. As glucose levels drop, appetite becomes compelling. As most of us know all too well, appetite does not decline immediately with the first bite. Because of the lag between onset of eating and the sensation of satiety, if one is ravenous at the start of a meal, then one may well overeat. One helpful hint for patients, then, is for them to have a serving of milk in the late afternoon. That way, they go home with their appetite at least somewhat blunted. Of course, dairy products have other benefits as well. They also supply a balanced mineral solution, including calcium, needed for bone accretion. This latter benefit applies also to those of normal weight. ■

Endometrial Thickness as a Test for Endometrial Cancer in Women With Postmenopausal Vaginal Bleeding

ABSTRACT & COMMENTARY

Synopsis: *Measurement of endometrial thickness by transvaginal ultrasonography is not sufficient as a single procedure for determining the presence or absence of endometrial cancer in symptomatic postmenopausal women.*

Source: Tabor A, et al. *Obstet Gynecol*. 2002;99(4): 663-670.

TABOR AND COLLEAGUES SEARCHED THE ENGLISH medical literature for the period January 1991 to September 30, 1997, for articles that discuss the use of vaginal ultrasonography for measurement of the thickness of the endometrium. They identified 48 potential studies, but eventually eliminated 39 of them for 1 or more reasons. The most common reason for exclusion was the inability of the researchers of the published articles to provide data on the median thickness of the endometrium in women in their study who were not affected by endometrial cancer.

The 9 studies that were selected for inclusion in this meta-analysis contained 3483 symptomatic women without endometrial cancer and 330 with cancer. The women were further subdivided into those who were premenopausal, postmenopausal not on HRT, and postmenopausal on HRT. In 6 of the studies, dilation and curettage (D&C) followed vaginal ultrasonography in all cases. In 2 studies, sonography was followed by endometrial biopsy, and 1 study included both a D&C and an endometrial biopsy.

The first (and perhaps most important) finding of the study was that the median thickness of the endometrium among women who did *not* have endometrial cancer varied significantly from 2.0 mm to 6.4 mm ($P < .001$). Two articles contained information about the median thickness of the endometrium among premenopausal women. In these women, the median thickness was 2-3 mm more than symptomatic postmenopausal women, not on HRT. Likewise, among those postmenopausal women who were on HRT (3 studies), the thickness was 2 mm more than postmenopausal women not on HRT.

Because of the differences in the medians, Tabor et al also transformed the data into multiples of the median (MoM). This adjustment did not smooth out the variability among the studies. The MoM with endometrial cancer ranged from 2.1 to 5.9. The average was 3.7 MoM.

Tabor et al then looked at false-positives rates for referral for histologic sampling of 50% and 10%. Overall, if a 10% false-positive rate was chosen, 63% of the women with cancer would have been sampled. At a 50% false-positive rate (no better than a coin-toss), 96% of cancers would have been identified, but 4% would have been missed.

Tabor et al have 2 major conclusions. First, it is critical that each center that performs transvaginal ultrasonography for determination of endometrial thickness develop their institution-specific median endometrial thickness for symptomatic women who are premenopause, postmenopause not on HRT, and postmenopause on HRT. Second, they conclude that vaginal ultrasonography alone is not sufficiently sensitive to rely on it as the only test for endometrial cancer in symptomatic women because of the 4% "false-negative

rate," even with liberal guidelines for referral for D&C.

■ **COMMENT BY KENNETH L. NOLLER, MD**

During the past 30 years, it has been interesting to watch the introduction of new technology into the practice of gynecology. Typically, there will be some skepticism regarding any new technique or test when it is first introduced. Indeed, more than half of all techniques that are introduced never "catch on" and are either disconnected or relegated to use in a few special situations. For those techniques that do happen to catch on, the initial skepticism is often followed by a wave of over-use and over-reliance. Eventually, after the publication of multiple articles, the new technique or test will eventually find its unique place in the day-to-day practice of gynecology.

For some years now, I have felt that we have been experiencing the over-use phase of vaginal ultrasonography for evaluation of postmenopausal women with vaginal bleeding. When it was first introduced, multiple small series applauded the technique as a major advance that nearly eliminated the need for the more painful procedure of endometrial biopsy. Virtually all of these early articles concluded that all one had to do was to perform a vaginal probe ultrasound examination, and if the endometrial lining was less than "x" in thickness, no further evaluation was necessary. There was, of course, an immediate indication that such a simple management scheme was certainly not appropriate since each article used a different "x." Nonetheless, I frequently see menopausal women who have experienced vaginal bleeding and have had no evaluation other than a vaginal ultrasound examination. An unusually large proportion of these women seems to be from specialists who do not usually perform endometrial biopsy and/or D&C.

This article by Tabor et al demonstrates clearly that it is impossible to detect every case of endometrial cancer by use of vaginal sonography. That should come as no surprise since virtually any test will fail occasionally. However, they found that unless at least half of all women who have endometrial thickness scanning are referred for D&C, the failure-to-detect rate is very high. In addition, they found (and I personally believe this is the most important information in the article) that there is no such thing as an "average" endometrial thickness above or below which it is possible to recommend referral for sampling. Rather, each institution must develop its own normal/abnormal thickness. Of course, almost no institution has done this, but rather relies on some isolated article that provided such information. That this is a mistake is clearly shown by Tabor et al. Any article an institution chooses is inappropriate for its population, as scans are read differently at the institution where the article was written and the institution that is using the printed material.

The bottom line is that it is not entirely safe to rely on endometrial thickness for evaluation of postmenopausal bleeding. Of course, we can all recite cases where it is absolutely critical to do so. Occasionally, a woman will be in such poor medical health that even an endometrial biopsy is inadvisable, or perhaps the cervix is entirely scarred and it is not possible to sample the endometrial lining. In such cases, if the lining is extremely thin the clinician can be somewhat reassured. Nonetheless, whenever it is possible to obtain a histologic sample from a postmenopausal woman who is bleeding, that is still the standard of care. ■

Disease Persistence in Patients with Cervical Intraepithelial Neoplasia Undergoing Electrosurgical Conization

ABSTRACT & COMMENTARY

Synopsis: *Following loop electrical excision of CIN when the transformation zone is visible at follow-up, the chance of persistent CIN is low.*

Source: Costa S, et al. *Gynecol Oncol*. 2002;85: 119-124.

Costa and colleagues reviewed their 3-year follow-up data on 699 women who underwent loop electroexcision for treatment of CIN. Nineteen percent of the treated women had an initial diagnosis of CIN 1, 17% CIN 2, and 64% CIN 3. The median width of the excised specimen was 19 mm and the median depth was 15 mm. Twenty seven percent of the specimens had positive margins. Over the study period, a total of 38 women (5.4%) had persistent CIN detected. Most of this was found at the first follow-up visit. Indeed, by the time of the third visit, the prevalence of CIN in the treated group was lower than the prevalence of CIN in the general population in the region of the study.

The presence of a positive margin was not associated with a higher rate of persistence in this study. The only 2 factors that predicted persistence were the pretreatment Pap smear (a high-grade Pap was more highly associated with persistent disease than a low-grade Pap), and a visible squamocolumnar junction at the

time of the follow-up visit.

Costa et al noted that their results—the presence of cone margin involvement did not predict persistent disease—are different from most published studies. They strongly suspect that the reason for this difference is that they conservatively estimated cone margin involvement. In addition, the size of their specimens was considerably larger than that reported by many other authors.

■ **COMMENT BY KENNETH L. NOLLER, MD**

This paper is interesting for only one reason: Costa et al found that if the squamocolumnar junction (really, the entire transformation zone) is visible at the time of follow-up after loop electroexcision for CIN, the chance of persistent disease is small. This finding is somewhat unique but does have great appeal. It will be interesting to see if it is confirmed by other studies, or by reanalysis by previously published work.

I am not terribly surprised that there was no association with disease persistence and specimen margin status since Costa et al removed such large pieces of tissue. The fact that their median depth of excision was 1.5 cm means that they excised far more tissue than is reported in most other papers. Thus, if Costa et al had a positive margin it would be more likely very nearly to include the full extent of the lesion than previous reports that might have removed only the central portion of the lesion and left far more disease behind.

The observation that, after 2 negative postprocedure visits, the prevalence of CIN was actually lower than the general population is interesting. We have probably been following our post-loop excision patients far too closely (and far too expensively). ■

Oncologists' Attitudes Toward and Practices in Giving Bad News: An Exploratory Study

ABSTRACT & COMMENTARY

Synopsis: *There is significant variability in how physicians approach information disclosure to cancer patients.*

Source: Baile WF, et al. *J Clin Oncol*. 2002;20: 2189-2196.

To examine the attitude of oncologists in disclosure of unfavorable medical information to cancer patients, Baile and colleagues administered a questionnaire to a group of physicians who attended the 1999 Annual Meeting of the American Society of Clinical Oncology. The questionnaire assessed demographic and practice-related information and the frequency of patient encounters in which unfavorable cancer-related information was disclosed. Participants were also asked about difficulties they had when approaching stressful discussions and communication strategies used in giving unfavorable information. The questionnaire was completed by 167 oncologists. Sixty-four percent were medical oncologists. Thirty-eight percent practiced in North America, 26% practiced in Europe, 13% practiced in South America, and 13% practiced in Asia. Participants gave bad news to patients an average of 35 times per month. Discussing no further curative treatment and hospice was reported as most difficult. In disclosing the cancer diagnosis and prognosis, physicians from Western countries were less likely to withhold unfavorable information from the patient at the family's request, avoid the discussion entirely, use euphemisms, and give treatments known not to be effective so as not to destroy hope than physicians from other countries. There was significant variability in opinions regarding the best time to discuss resuscitation, with 18% of respondents believing that it should be done close to the end of life. They concluded that there was significant variability in how physicians approach information disclosure to cancer patients. Factors such as geographical region and cultural and family variables may be important influences in this process.

■ **COMMENT BY DAVID M. GERSHENSON, MD**

Baile et al have been among the international leaders in the area of "breaking bad news." They and others have been extremely active in conducting formal courses and workshops and developing guidelines for giving bad news to cancer patients and their families. This paper highlights several important aspects of physician-patient communication. There are still major cultural differences in the information provided to patients and their families between Western and non-Western physicians. Non-Western physicians are more likely to avoid discussing prognosis, to withhold information from the patient at the family's request, and to offer patients futile treatments to preserve hope. However, Western physicians also face several challenges regarding giving unfavorable news. The most problematic areas are providing information about a poor prognosis and the timing of providing counseling about "do not resuscitate" orders, discontinuing therapy, and hospice care. My sense is that most oncologists, including myself, broach the latter set of issues much too late in the course of many patients' care. In addition to cultural differences, this

study explored gender differences. And it is no surprise to me that women were more adept at discussing hospice care and other difficult issues than men. As Baile et al point out, further research is needed in this area. One strategy that is gaining popularity is to query newly diagnosed patients on very specific issues related to how much she wants to know prior to the physician consultation. In this manner, the patient can actively participate in the decision concerning prognostic information, the stickiest issue for most oncologists. ■

CME Questions

21. Which of the following is *not* a component of the insulin resistance syndrome?
a. Dyslipidemia
b. Hypertension
c. Cardiovascular disease
d. Hyperinsulinemia
e. Impaired fibrinolytic capacity

22. In the article by Tabor et al, which one of the following statements is *false*:
a. "Normal" endometrial thickness in a woman with postmenopausal vaginal bleeding is institution-specific.
b. Reporting endometrial thickness as multiples of the median (MoM) rather than actual thickness reliably predicts abnormality.
c. At a false-positive rate of 50% endometrial thickness measurements still fail to identify 4% of endometrial cancers.
d. Among women with postmenopausal vaginal bleeding those on HRT who do not have endometrial cancer have a thicker endometrial lining than similar women not on HRT.

23. In the article by Costa et al, which one of the following factors was associated with the lowest risk of persistence of CIN?
a. Positive specimen margins
b. Multiple (3 or greater) follow-up evaluations
c. Visible squamocolumnar junction
d. None of the above

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