



## ESTROGEN SUPPLEMENTS IN MENOPAUSE

■ *To the Editor:* In reading the article by Dr. Booher,<sup>1</sup> several comments and questions arose.

First, the 1983 US Health and Human Services Life Tables indicate a life expectancy of about 31 years for a 50-year-old woman. From what source did Dr. Booher obtain the life expectancy of 36 years for a 50-year-old woman and 39 years for a 52-year-old woman? Next, regarding the expansion of the population age 65 and older, most estimates predict a doubling by the year 2030 or 2050. What is Dr. Booher's source for the quotation of a doubling within the next 10 years? Also, Dr. Booher quoted the prevalence of vasomotor symptoms as 85%, whereas most sources find 50% to 60%. Finally, regarding the recommendation of the use of estrogen in breast cancer patients, does Dr. Booher have any data to support its safety in estrogen-receptor negative patients? Does he use estrogen in such patients in his own practice, and does he recommend it for others?

BRUCE ETTINGER, MD  
Clinical Professor

Department of Internal Medicine  
The Permanente Medical Group, Inc.  
San Francisco

1. Booher DL. Estrogen supplements in menopause. *Cleve Clin J Med* 1990; 57:154-160.

■ *Reply:* Life expectancy figures were obtained from a lecture presented by Dr. Lila Noctigal, New York University School of Medicine, at the menopause symposium conducted by Dr. Wulf Utian of Mount Sinai Medical Center (Cleveland) on September 12, 1986. I realize the error in predicted expansion of the number of women age 65 and older to double by the year 2000; this should have been by the year 2035.

Regarding vasomotor symptoms, my resource was Speroff.<sup>1</sup> I am sure the incidence varies depending on whether subjective or objective criteria are used.

The use of estrogen in women with a history of breast cancer is certainly a thorny issue, and I tend to be extremely conservative in this regard. However, a clinical management issue which has never seemed logical is that we have generally stopped performing

oophorectomy in most breast cancer patients. If endogenous ovarian estrogen is acceptable in breast cancer patients on tamoxifen, then exogenous non-oral ovarian estrogen should be equally acceptable. This point of view is supported by two sources.<sup>2,3</sup>

On rare occasions, I do use estrogen in breast cancer patients with the collaboration and support of the breast cancer surgeon and medical oncologist. To be sure, the overall cost-benefit concept of menopausal management including cardiovascular disease, osteoporosis, and quality of life issues must be carefully evaluated, and meticulous informed consent with shared patient responsibility is essential. In discussing this with my colleagues, I recommend the same conservative approach.

DELBERT L. BOOHER, MD  
Department of Gynecology  
The Cleveland Clinic Foundation

1. Speroff L, Glass RH, Kase NG. Clinical gynecologic endocrinology and infertility. 4th ed. Baltimore: Williams and Wilkins, 1988:137.
2. Notelovitz M. Noncontraceptive hormone therapy and breast cancer: a personal perspective of clinical guidelines. *Menopause Management* 1989; 2(3):5-8.
3. Weinstein L. Hormonal therapy in the patient with surgical menopause. *Obstet Gynecol* 1990; 75(4):47s-50s.

## DIFFERENTIAL DIAGNOSIS OF HYPERSENSITIVITY VASCULITIS

■ *To the Editor:* I enjoyed the Highlight from Medical Grand Rounds article by Dr. Calabrese on hypersensitivity vasculitis.<sup>1</sup> In 1948, a landmark paper by Zeek and coworkers<sup>2</sup> separated periarthritis nodosa (PAN) from hypersensitivity angitis (HA) on the basis of morphological differences between these two angitides. In the ensuing years, this observation has been confirmed by many investigators. However, these criteria were not without criticism. In the early 1940s, Rich had restated the theory first suggested by Gruber, that PAN could occur in human beings as the result of a generalized hypersensitive reaction to some foreign agent, such as serum or sulfonamides. In 1958 Rich noted that Zeek and associates "have sought to differentiate their human cases of PAN that were clearly due to hypersensitivity from those in which no sensitization was apparent and which they term 'primary' PAN. I think I should say that our own experience, as