Estrogen Use and Postmenopausal Women: A Basis for Informed Decisions

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A panel of experts convened by the National Institute on Aging, National Institutes of Health, reached the following consensus regarding estrogen use and postmenopausal women. The use of estrogens alleviates vasomotor symptoms and atrophy of the vaginal epithelium and might aid in preventing osteoporosis. However, it increases the incidence of endometrial cancer. The addition of progestins might prevent this complication, but potential risks have not been adequately evaluated. Convincing evidence that postmenopausal estrogen use influences the occurrence of cardiovascular disease and breast cancer does not currently exist. Many aspects of the menopause and its management require further research. Any candidate for postmenopausal estrogen use should be given as much information as possible about both benefits and risks and then, with her physician, reach an individualized decision regarding whether to receive estrogens.

The postmenopausal use of estrogens, a topic of considerable public health importance, presents a complex array of benefits, risks, and unknowns. In an attempt to reach the best conclusions and recommendations possible at present, the National Institute on Aging, National Institutes of Health (NIH), held a Consensus Development Conference entitled "Estrogen Use and Postmenopausal Women" on September 13 and 14, 1979. As an NIH consensus activity, it brought together practicing physicians, biomedical researchers, and members of the public.

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Chaired by Kenneth J. Ryan, MD, Chief of Staff, Boston Hospital for Women, the conference began with three background papers: "Indications for Estrogen Replacement Therapy" by Isaac Schiff, MD, Boston Hospital for Women; "Effects of Exogenous Estrogen on Postmenopausal Women: The Epidemiologic Evidence" by Barbara S. Hulka, MD, MPH, University of North Carolina; and "Estrogen Use in Postmenopausal Women: Costs, Risks, and Benefits" by Milton C. Weinstein, PhD, Harvard University. Next, the conference panel, consisting of nominees of ten organizations with relevant fields of interest (Table 1), and the audience discussed the evidence and issues. The panelists and other invited participants then prepared and presented to the audience a preliminary statement of consensus, which summarized the current state of knowledge and concluded that the decision whether to use estrogens must be an individual, informed choice for

Table 1. Panelists at Consensus Development Conference on Estrogen Use and Postmenopausal Women

Panelist	Nominating Organization
Elizabeth Barrett-Connor, MD	American College of Preventive Medicine
Daniel D. Federman, MD	American College of Physicians
Gerald A. Glowacki, MD	American Geriatrics Society
Saul B. Gusberg, MD	American Cancer Society, Inc.
Elizabeth D. Jones, MSW, CSW	Women's Hormone Information Service
Weldon G. Kolb, MD	American Academy of Family Physicians
Howard L. Judd, MD	American College of Obstetricians & Gynecologists
Stanley G. Korenman, MD	Endocrine Society
Anne M. Seiden, MD	American Psychiatric Association
Noel S. Weiss, MD	Society for Epidemiologic Research

each woman. Members of the audience expressed their support and provided suggestions, which were used in revising the document.

The report that follows summarizes the currently available information on postmenopausal estrogen use, as presented by the invited speakers, panelists, and members of the audience. Each section consists of an excerpt from the consensus statement, followed by commentary. More detailed discussion is available in the three background papers, which have now been published.[1-3]

Relief of Vasomotor Flushes

In approaching the topic, the group first reviewed the evidence for the efficacy of estrogens in treating specific conditions associated with the menopause. It was accepted that estrogens are more effective than placebo in decreasing the frequency and/or severity of vasomotor symptoms (hot flashes and sweating). The questions that remain to be answered include whether vasomotor symptoms represent a homogeneous entity with a single cause and why some patients require much larger doses than average to control symptoms. There was general agreement that the decision of whether to initiate therapy should depend on the severity of the symptoms and the patient's perceived need for relief and that the lowest effective dose should be utilized. The occurrence of hot flashes naturally declines over a period of time, and unnecessary prolongation of therapy should be avoided.

Vasomotor flushes ("hot flashes" or "hot flushes") and sweats affect many, but not all, women. These poorly understood symptoms generally begin around the time of menopause, continue for varying periods of time thereafter, and eventually subside spontaneously. The amounts of discomfort and interference with daily life vary considerably among individuals and cultures. Although some physicians have stated that these symptoms may predispose women to respiratory infection or precipitate bronchospasm in asthmatics, vasomotor flushes are not generally thought to be harmful, and thus treatment is not considered necessary for maintenance of physical health.

Research[4] confirms the popular impression that estrogens alleviate vasomotor flushes but indicates that a placebo effect, although smaller, also exists. For unknown reasons, the dose of estrogen needed to control these symptoms varies greatly among women and can be relatively high. Vasomotor flushes tend to recur when estrogen use is

discontinued. Because the risk of adverse effects increases with duration and possibly also dose, therapy should consist of the lowest effective dose for the shortest possible period of time.

Topics for further investigation include the epidemiology and mechanism(s) of hot flashes, as well as the items cited in the consensus statement.

Treatment of Genitourinary Changes

It is recognized that estrogens are effective in overcoming the atrophy of the vaginal epithelium (wall) and the associated symptoms, which may include dryness, burning, itching, and pain on intercourse. It was suggested that the possible relationship of urinary tract symptoms to estrogen lack be more thoroughly investigated. Attempts to avoid systemic effects by treating vaginal symptoms with local application of estrogen-containing creams have been common. However, evidence now exists that the estrogens in these creams may be absorbed rapidly into the bloodstream. The biological consequences of this absorption are undetermined and require study.

Following the menopause, atrophy of the vaginal epithelium can produce the symptoms noted above. Relief of these complaints generally occurs during estrogen use[5] and can persist for varying lengths of time afterward. Estrogens do not, however, appear to be effective in treating vaginal relaxation.[6]

Postmenopausal women sometimes experience dysuria and urinary frequency in the absence of positive urine cultures. These symptoms might result from estrogen deficiency, as the vagina and the urethra possess a common embryonic origin. This hypothesis and the possible role of estrogens in treating urinary tract symptoms deserve investigation.

At one time thought to have only local effects, intravaginally applied estrogens have been prescribed to women in whom systemic estrogens are contraindicated. However, estrogens administered by this route appear to enter the bloodstream rapidly and escape initial metabolism by the liver.[7] Furthermore, the possibility of local effects on the endometrium is of concern. Intravaginal estrogen administration requires further research before recommendations can be made about its use.

Psychological and Cosmetic Effects

There is no evidence at present to justify the use of estrogens in treatment of primary psychological problems. Surveys have shown no established specific or temporal association of sleep patterns, mental performance, mood, or psychological state with menopause or estrogen deprivation. On the other hand, in preliminary intervention studies comparing estrogens to placebo, effects on sleep latency and REM sleep have been noted. Some improvement in mental well-being in women receiving estrogens may be secondary to alleviation of physical symptoms.

The psychological characteristics of the menopausal stage are incompletely defined, but this period of life does not, as once thought, appear to be characterized by a high incidence of new and distinctive mental illness. Nevertheless, the menopause is a psychological as well as physical milestone in the aging process. Such times of transition typically are stressful and require that appropriate support be available.

Although estrogens are not indicated in the treatment of primary psychological problems, the possibility exists that estrogens may have psychological benefits in some women. Studies of the emotional and cognitive effects of estrogen administration have differed in populations examined, variables considered, and methods used. Results of these studies also have varied, and thus no conclusion can be drawn regarding which, if any, psychological functions may be altered by estrogen administration. One study's finding that estrogen use produced more improvement of psychological state in women with severe hot flashes than in those with milder symptoms[8] suggests that an increased sense of emotional well-being may be secondary to alleviation of physical discomfort associated with the menopause.

Preliminary findings suggest that estrogen use might improve the quality of sleep. In a double-blind crossover study,[9] estrogen use was correlated with decreased sleep latency (a shorter time between lights out and sleep onset), more rapid eye movement (REM) sleep, and a greater percentage of time spent in REM sleep.

At one time, it was hoped that estrogens would retard the development of wrinkled skin, gray hair, sagging breasts, and other physical signs of aging. However, it now appears unlikely that they have any such cosmetic benefits.

The psychological characteristics of the menopause, as well as the effects of estrogens on emotional state, cognitive function, and sleep, deserve further research.

Effects on Bone

The group acknowledged the validity of three randomized trials indicating that exogenous estrogens can retard bone loss if given around the time of the menopause. Except for dietary calcium, which appears to decrease bone loss to a lesser extent, other substances have not been shown to have such an effect. It is inferred but not proven that this retardation of bone loss will prevent the ultimate development of osteoporosis and attendant fractures. Case control studies not yet published but discussed at the meeting report an association of estrogen use with a decreased risk of osteoporosis related fractures. However, more data are definitely needed before the efficacy of estrogens in preventing fractures can be established. An inconsistency was noted: namely, in the randomized trials, accelerated bone loss following discontinuation of estrogen use resulted in loss of any favorable effect on bone mass, whereas in the case control studies, use of estrogens at any time in the past conferred some protection against fractures. Identification of patients at increased risk of osteoporosis would be desirable because of the strong possibility of successful prophylaxis. One high-risk group in which to investigate possible benefits of estrogens consists of patients who already have developed osteoporosis and sustained fractures. Estrogen administration represents a promising approach to prevention of the widespread problem of hip fracture.

Following the menopause, loss of bone mass, which begins previously, accelerates, and the incidence of fractures—especially those of the proximal femur (hip), vertebrae, and radius—increases markedly. An estimated 200,000 Americans, most of them older women, suffer hip fractures each year, at a probable cost of over $1 billion. Thus, measures to prevent, arrest, reduce, or reverse bone loss—and thus presumably decrease the risk of fractures—are of considerable clinical and public health importance.

Lack of estrogen hastens the loss of bone and thus the onset of osteoporosis. However, several other factors—including immobilization, white race (and, in particular, fair coloring), slenderness, heavy drinking, low calcium intake, and cigarette smoking—may also increase the risk of osteoporosis. Thus, even if generally effective, estrogen use may not always be sufficient to prevent fractures, and potential exists for decreasing the risk of osteoporosis by means other than estrogen administration.

Research[10-12] indicates that low doses of estrogens can arrest or retard bone loss if given for several years shortly after the menopause, but the effects of estrogen administration on bone mass over longer periods of time require study. Preliminary evidence, including a case control study[13] that was in press at the time of the conference, suggests that estrogens do, as predicted, decrease the incidence of fractures.

Topics for further research include the effects of dose, duration, and recency of estrogen use on bone mass and on risk of fractures, as well as alternative ways to maintain the quantity and quality of bone.

Cardiovascular Effects

There is no convincing evidence that estrogens in customary doses increase the risk of thromboembolic phenomena, stroke, or heart disease in women who have undergone natural menopause. Although it was once hoped that estrogens would protect against heart disease in aging women, this effect has not yet been demonstrated. One promising approach would be to devise a more physiological mechanism for estrogen replacement. Because oral therapy results in the delivery of supraphysiological concentrations of estrogens to the liver, it can exert an exaggerated effect on lipoprotein metabolism, blood coagulation, and other important processes.

The cardiovascular effects of postmenopausal estrogen use remain incompletely defined.

Because the ratio of deaths from myocardial infarction among women to those among men increases sharply beginning between ages 55 and 65, it was initially inferred that the risk of cardiovascular disease suddenly increases after the menopause. However, more careful examination of the data shows that these death rates in women increase at a constant rate throughout life, and that the abrupt change in risk ratio reflects a decrease in the rate of increase in mortality for men. Thus, the epidemiologic support for the hypothesis that estrogens protect against cardiovascular disease is not as strong as was once believed.

Because of this alleged protective effect, the Coronary Drug Project, designed to test the effectiveness of various drugs in preventing death and further illness in men aged 30 to 64 years who had suffered one or more previous myocardial infarctions, included a study of the effects of estrogens. With an average of 18 months of follow-up, the group receiving 5 mg of conjugated estrogens per day suffered an excess number of non-fatal myocardial infarctions, pulmonary emboli, and episodes of thrombophlebitis; a dose of 2.5 mg per day for the same period of time did not affect outcome.[14] Generalization of these findings to postmenopausal estrogen users—who are women rather than men, generally lack previous history of myocardial infarction, and normally receive doses of 0.625 or 1.25 mg per day—is not likely to be valid.

Several studies have examined the relationship of postmenopausal estrogen use to various cardiovascular disorders. In contrast to findings with oral contraceptives, case control studies of two populations[15,16] show no association of myocardial infarction and exogenous estrogen administration. Two studies of a retirement community[17,18] found that estrogens were positively associated with hypertension, but, after controlling for blood pressure, not with stroke; but a third study of the same population[19] reported no such relationship. A review of cases of idiopathic venous thromboembolism[20] suggests that postmenopausal use of estrogens, unlike the use of oral contraceptives, does not increase the risk of this condition. Two follow-up studies[21,22] suggest that estrogen administration might decrease the incidence of and mortality from cardiovascular disease, but methodological problems limit the conclusions that can be drawn.

Postmenopausal estrogen use increases serum levels of high-density lipoprotein cholesterol,[23] which are inversely related to risk of myocardial infarction, but does not restore a premenopausal lipid profile.[24] Non-oral routes of estrogen administration, which avoid delivery of supraphysiological doses to the liver, may exert more physiological effects.

Topics for further research include the effects of postmenopausal estrogen use on blood pressure, on serum lipid levels, and on risk of myocardial infarction, stroke, and thromboembolic phenomena; and the effects of estrogens administered by non-oral routes.

The Risk of Endometrial Cancer

The group then reviewed the evidence for adverse effects associated with postmenopausal estrogen use. In the absence of exogenous estrogens, the incidence rate of endometrial cancer is approximately 1 per 1,000 postmenopausal women per year. It was recognized that this rate increases several-fold beginning after approximately two to four years of use of 0.625 or 1.25 mg of conjugated estrogens per day. Evidence was presented that the risk of endometrial cancer increases with the duration of use and declines after discontinuation. Estrogen use is most strongly associated with lesions of the lowest grade and earliest stage. Of interest is the temporal relationship of the number of estrogen prescriptions and the incidence of carcinoma of the endometrium: both rising steadily until 1976 and then declining in parallel. Although the incidence of carcinoma of the endometrium rose, mortality from the disease did not increase. A considerable part of this discrepancy may be attributable to early detection and the high cure rate.

Cystic hyperplasia of the endometrium, which is considered a premalignant condition, has been associated with unopposed estrogen, whether endogenous (as in anovulatory states) or exogenous. The cost effectiveness of sampling the endometrium in order to screen for endometrial hyperplasia and cancer in completely asymptomatic women currently or potentially receiving estrogens is uncertain at present. It was agreed that suction curettage is effective in evaluating the endometri-

Table 2. Case Control Studies of Estrogen Use and Endometrial Cancer*

First Author	Site of Study	Relative Risk(s) Reported in Abstract
Smith[25]	Seattle	4.5
Ziel[26]	Los Angeles	7.6
Mack[27]	Los Angeles	8.0
McDonald[28]	Rochester	4.9
Antunes[29]	Baltimore	6.0
Hulka[30]	North Carolina	3.6, 4.1

*Adapted from the paper presented by Dr. Hulka

um and that the cause of any bleeding must be determined. A report indicating that uterine bleeding may sometimes be absent early in the course of endometrial cancer was presented at the meeting. Hence, prudence would suggest that, even in the absence of bleeding, the endometrium should be sampled before and during estrogen therapy (on a yearly basis).

The use of progestins for several days of each estrogen treatment cycle has been demonstrated to decrease the occurrence of endometrial hyperplasia and might also reduce the associated risk of developing cancer of the endometrium. Before the application of combined therapy becomes established, risks of the various progestins must be adequately evaluated.

Endometrial cancer is the main recognized risk of postmenopausal estrogen use. As summarized in Table 2, several case control studies of different populations[25-30] support this association. Horwitz and Feinstein[31] have challenged the methodology and conclusions of such studies, but their objections do not appear to have been widely accepted. Of course, women who have undergone hysterectomy cannot develop endometrial cancer.

Risk increases with duration of estrogen use, but its relationship to dose remains uncertain. The temporal association of estrogen use and endometrial cancer suggests that estrogens may function as promotional agents rather than carcinogenic initiators.

In postmenopausal women taking estrogens, the administration of progestins for several days per cycle has been reported to reduce the prevalence of endometrial hyperplasia.[32] Thus, it has been suggested that the addition of these substances might decrease the risk of endometrial carcinoma in this population. However, the possible risks of administering progestins to postmenopausal women are largely unexplored.

Topics for further research include the relationship of estrogen dose to the risk of endometrial cancer, the favorable and unfavorable effects of the addition of progestins, and the yield and cost effectiveness of screening potential and current estrogen users for endometrial hyperplasia and asymptomatic endometrial cancer.

The Possible Risk of Breast Cancer

The association of estrogens and breast cancer in experimental animals is well known. Careful review of several well-conducted case control studies has not revealed such a relationship in humans. In two follow-up studies of estrogen users, varying associations were encountered. Compared to data on the general population, one showed an excess of cases in years 5 to 9, but the other only after 15 years, of estrogen use. Incidence rates of breast cancer have not changed in parallel with those of estrogen use, as have those of endometrial carcinoma. Because of the high incidence and relatively poor prognosis of breast

Table 3. Case Control Studies of Estrogen Use and Breast Cancer*

First Author	Number of Cases	Number of Controls	Relative Risk
Boston Collaborative Drug Surveillance Program[20]	51	774	1.0
Henderson[33]	308	308	0.8
Craig[34]	134	260	1.0
Mack[35]	111	444	1.6
Casagrande[36]	47	31	3.1
Sartwell[37]	284	367	0.8
Wynder[38]	785	2231	1.1
Brinton[39]	405	1156	1.0

*Adapted from the paper presented by Dr. Hulka

cancer, any possible association with estrogen use remains a concern.

As summarized in Table 3, multiple well-designed case control studies[20,33-39] have shown no significant association between estrogen use and breast cancer. For several reasons, however, the possibility that estrogen use increases the risk of developing this condition deserves further attention. Estrogens can induce breast tumors in animals. In addition, the latency period in humans may be so long that an increase has not yet been detected. Furthermore, estrogen administration might produce breast cancer in some groups of women or might stimulate incipient tumors. Two practice based follow-up studies have found some increased risk—one during the first ten years of estrogen use,[40] the other after approximately 15 years thereof[41]; but methodological problems limit the inferences that can be drawn. A case control study that appeared several months after the conference suggests that in women with intact ovaries, estrogen replacement therapy in large cumulative doses may increase the risk of breast cancer.[42]

Additional research and surveillance are needed. Major studies of the possible relationship of postmenopausal estrogen use and breast cancer are now in progress.

Other Possible Risks

There are experimental data indicating that estrogens can induce the production of lithogenic bile, and in one study a 2.5-fold relative risk for the development of surgically confirmed gallbladder disease was observed.

The possible association of postmenopausal estrogen use and gallbladder disease[20] requires further investigation.

The effect, if any, of estrogen use on the risk of ovarian cancer has received relatively little research. However, one well-designed case control study[43] showed no association between the two.

Estrogen Use After Premature Menopause

Some concern was expressed about women who have undergone menopause many years in advance of the normally expected age. Although most of the participants felt intuitively that approximation of the normal physiological state through hormone replacement therapy would be best, there are no carefully controlled studies comparing the risks and benefits in these circumstances. Support was voiced for conserving the ovaries of young women when possible.

Those women in whom surgical or other factors result in loss of ovarian function many years before the normal time of menopause present special considerations. Such individuals can experience vasomotor symptoms and can develop vaginal epithelial atrophy while relatively young. In addition, they lose bone prematurely and thus may be at risk of more and earlier fractures than usually observed. Furthermore, early loss of ovarian function may hasten the development of coronary vascular disease.[44,45] However, early castration also can decrease the risk of breast cancer. Oophorectomy often is accompanied by hysterectomy, which eliminates the possibility of developing endometrial cancer. More data are needed before a definitive statement can be made.

General Conclusions

One area of general agreement was that the patient should be given as much information as possible about the evidence for the effectiveness of estrogens in treating specific menopausal conditions and the risks that their use may entail. Patients must be kept continually informed of new findings as they arise. Given the current state of knowledge, no general recommendation, applicable to all postmenopausal women, can be made.

General consensus was reached on the current state of knowledge, as described. It is clear, however, that much additional information is needed. Specifically, systematic knowledge is needed of the natural course of the menopause in the absence of hormonal therapy, of alternatives to estrogen use, of the optimal way to provide estrogens if they are to be used, and of all aspects of their beneficial and adverse effects. Special attention should be directed toward studies that can determine the proper use of estrogens in young women having undergone oophorectomy ten or more years before the natural time of menopause. No general formulation regarding therapy can be given. Rather, each individual patient must base her decision on the relative values that she assigns to relief of symptoms, to expectations for optimizing health and well-being, and to various risks sustained in the process. Socially and culturally based attitudes toward the menopause may influence these values and require further definition.

Alternatives to Estrogen Use

Considerable interest exists in the use of measures other than estrogens to prevent and manage specific menopausal and postmenopausal conditions. Among the non-estrogenic substances that have been found useful in controlling hot flashes are clonidine[46] and medroxyprogesterone acetate.[47] In women with symptomatic vaginal epithelial atrophy, water-soluble lubricants can aid in preventing dyspareunia. An increased intake of calcium, the absorption of which becomes less efficient after the menopause, appears to retard bone loss[11,12] and thus might reduce the risk of fractures; other means of preventing osteoporosis have been proposed but not well tested.

Good nutrition and exercise have been popularly promoted for general well-being in the postmenopausal years. In addition, educating women about what to expect around and after the time of menopause may help them to cope effectively with various changes.

Types of Estrogens

Various estrogens bind to the same receptors and thus when administered in comparable doses have similar effects, both favorable and unfavorable. However, estrogens administered by different routes can undergo different patterns of metabolism and thus vary in their relative effects on specific organs.

The estrogen preparation that has been most widely prescribed in the United States is a mixture of conjugated estrogens that is obtained from the urine of pregnant mares and of which sodium estrone sulfate seems to be the component present in largest quantity. At present, the standard dose appears to be 0.625 mg per day. When administered orally, these estrogens pass through, affect, and are largely inactivated by the liver before entering the systemic circulation. Thus, they tend to alter serum levels of compounds associated with the liver, such as sex hormone binding globulin and cortisol binding globulin, more than those of such substances as FSH and LH.

Estradiol is the predominant estrogen produced premenopausally and thus might be the most physiologic compound for replacement therapy. However, when taken orally, it is extensively metabolized and appears largely as estrone in the blood.[48] When placed in the vagina,[7] injected, or

implanted, estradiol enters the bloodstream. Ethinyl estradiol and mestranol are stable when taken orally but might be hepatotoxic, as young women taking oral contraceptives containing these substances are at increased risk of hepatic adenomas.

Estriol was once hypothesized to be the safest form of estrogen. However, at doses high enough to provide the benefits of estrogen use, the risks are likely to be the same as those of other estrogens.

Question often arises regarding the relative compositions of estrogen preparations used postmenopausally and the "birth control pill." In general, postmenopausal women receive considerably lower doses of estrogens than are present in oral contraceptives. Furthermore, the latter contain both estrogens and progestins.

Contraindications to Postmenopausal Estrogen Use

Among the conditions commonly considered contraindications to the use of estrogens for the relief of menopausal symptoms are: previous cancer of the breast or endometrium; strong family history of breast cancer; and history of stroke, myocardial infarction, thromboembolic phenomena, and other cardiovascular conditions. In addition, women who have received diethylstilbestrol during pregnancy or while in utero have been advised not to receive exogenous estrogens.

Weighing the Benefits, Risks, Unknowns, and Costs

A woman's decision as to whether to use estrogens involves weighing the benefits, risks, and unknowns. In addition, the costs involved are of personal and social importance.

At the conference, Weinstein approached this problem through cost effectiveness analysis. Costs induced by the use of estrogens include not only that of the medication itself but also those amassed in the treatment of complications. However, estrogens might also reduce costs by preventing fractures. Likewise, estrogen use can increase mortality by causing endometrial cancer and necessitating surgery, but it also might reduce the number of deaths from hip fractures.

Because much remains unknown, the net effects on cost and life expectancy cannot be precisely determined. Furthermore, these values vary depending on the criteria used to select patients for treatment. Nevertheless, these effects appear to be small. Thus, based on the assumptions used, the costs induced and those saved by estrogen administration approximately counterbalance each other. Likewise, years of life lost and gained seem similar in number. These conclusions apply only to the general population of estrogen users, and any individual woman might incur a large net gain or loss in life expectancy or cost.

Quality of life is a major consideration. Estrogen use can enhance the quality of life by relieving menopausal symptoms and perhaps by preventing fractures, but it also can diminish it by producing complications such as endometrial cancer and by resulting in worry about possible adverse effects. Even if quality of life is assigned modest quantitative importance in Weinstein's model, it has a much greater effect than life expectancy on the cost effectiveness—and, in personal terms, on the desirability—of treatment. This finding implies that the decision whether to use estrogens should be an individual one, based on the relative values that each woman attributes to relief of symptoms, possible long-term benefits, and potential risks.

Similarly, the conference participants agreed that no uniform recommendation, applicable to all women, can be made regarding whether estrogens should be used. Rather, they noted, the decision must be a personal one. To make this choice intelligently, women must be given as much information as possible about both the desirable and undesirable effects of estrogen use. In addition, women must be kept up to date as new findings become available. The development of more effective means of educating women about estrogens and aiding them in making satisfying decisions deserves attention.

The decision whether to use estrogens is not an isolated event. Rather, it occurs in the context of a woman's personal history and many other, as yet incompletely understood, medical, psychological, social, and cultural factors relating to the menopause and aging. Thus, the woman and her physician should confer openly and arrive together at the total approach that, whether it includes estrogen use or not, is most likely to promote her general well-being during the postmenopausal years.

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