



Hormone Replacement Therapy by William Rawls, Jr., M.D., FACOG

HRT: Based on well done studies done over the past twenty years on primates and humans, I have been informing patients of the risks and benefits of hormone replacement therapy. Interestingly, the most recent study that has caused so much controversy still supports all the things I know to be true about estrogen replacement and has not changed my counseling. This may come as a bit of surprise to many individuals, as the WHI study caused such a stir. When making decisions using scientific information clinical judgment should never be based solely on a single study, especially if other information is available. Also, all scientific studies should be accompanied by a healthy dose of common sense when making decisions that affect people's lives.

To understand estrogen we have to look at what it does in the body. Here I use the term estrogen generically, recognizing that there are several estrogens and many compounds that act like estrogen, all with different potencies. Estrogen is basically a messenger, like so many other hormones in the body. It, along with progesterone and other reproductive hormones informs the rest of the body that a cyclic reproductive cycle is occurring. It does so by being carried from the ovaries to all the other cells of the body via the blood stream. Once arriving, it touches a receptor inside the cell and completes a message. This continuous and cyclic message comes to a fairly abrupt end at menopause when the ovaries lose the capacity to produce estrogen and progesterone.

In many individuals this fairly sudden change throws the body into something of a tail-spin. All of the cells that make up the tissues of the body are affected by no longer receiving this constant message. Hot flashes, sleeplessness, mood changes, changes in skin and vaginal tissues, accelerated bone loss, and increased risk of heart disease occur as a result. In most individuals these changes are transient and we refer to menopause as a period of transition. With time the cells of the body gradually remove or down-regulate estrogen and progesterone receptors and become less sensitive. Symptoms resolve and bone loss slows. A significant amount of damage, however, can occur in this 2 to 7 year transition. The primary strategy of hormone replacement therapy has always been to alleviate symptoms and limit damage during this transition. Standard menopausal hormone replacement often includes not only estrogen, but also progesterone and sometimes testosterone.

In my practice I have targeted women having significant symptoms who are within two years of menopause as the ideal candidates for hormone replacement therapy. I consider that HRT should ideally be continued for 5 to 7 years and then tapered gradually. Early on I recognized that symptoms of breast tenderness were commonly associated with the standard product containing conjugated estrogens derived from horse urine. Research of the product revealed that about 25% of these equine estrogens were completely foreign to the human body. I would suspect that slow metabolism of these estrogens results in increased breast tissue stimulation and therefore more breast tenderness. It was no surprise to me when studies started linking an increased risk of postmenopausal breast cancer to use of these hormones. By then I had begun switching patients to bio-identical hormone replacement. Patients had less breast tenderness and limited studies in Germany and Japan were reassuring in that they did not show a link with this type of HRT to breast cancer.

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Bio-identical hormone replacement refers to using hormones that are normally metabolized by the human body. Not to say they are natural, because giving any reproductive hormone after the age of menopause cannot be considered natural, but the combinations of hormones commonly used are well thought out and are well tolerated by most individuals. Another advantage of bio-identical HRT is ease of administration. Compounding pharmacies are able to provide not only oral routes of administration, but also sublingual (under the tongue), topical, and vaginal. In addition, the dosages of each hormone can be catered to individual needs.

Back to all the controversy about HRT and heart disease. We know from earlier primate studies that estrogen does have a protective effect on the vessels of the heart and we also know that estrogen has a favorable effect on cholesterol. Estrogen has the somewhat opposing effect of making the blood more coagulable (causing blood to clot). In most newly menopausal patients with relatively clean heart vessels, this opposing effect does not seem to be of great concern. Recommend a mild blood thinner, such as a baby aspirin or fish oil, and it is even less of a problem. On the other hand, select a patient far removed from menopause that already has established plaque formation in the heart vessels, and increasing the clotting capacity of their blood becomes a very real concern.

Amazingly, that is exactly what they did in this controversial study. The study was large, involving thousands of patients. It was controlled and double blinded. That means that half the group was on placebo and the other half was on the study drug, with no one knowing who was on what. Ordinarily this is the best way to conduct a clinical study, except early on they recognized that women having hot flashes in the placebo group would give things away. Therefore, all women having any symptoms of menopause were excluded from the study. Unbelievable! They excluded the entire population of menopausal patients that have been targeted for HRT over the past 20 years. The average age in the study was 64. That means they were placing women in their 60's and 70's on estrogen for the first time in their lives. It should be no surprise that if you place individuals who already have established vessel disease on a drug that adversely affects their coagulation that you will see an increased incidence of heart attacks!

The study used synthetic conjugated equine estrogens and a synthetic progesterone called medroxyprogesterone. There was an increased incidence of breast cancer, but again making firm deductions is difficult because of the age of the population. It could be that at some point we will find that use of *any* estrogen during the postmenopausal period will be found to be associated with an increased risk of breast cancer, but I do not believe we have enough information at present to make that determination. In the mean time, I believe it does make sense to use hormones that are naturally metabolized by the human body rather than the opposite when considering HRT. I also believe that for many symptomatic individuals, the benefits of hormone replacement during the menopausal transition far outweigh the risks.

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