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Femoston®, menopause and the cardiovascular system

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Objective: To establish the impact of the new HRT product Femoston® (17β-estradiol sequentially combined with dydrogesterone, a structural isomer of natural progesterone) on risk markers for cardiovascular disease by reviewing published literature.

Study design: Database assembled following a Medline search (September 1995) for 311 papers containing the word 'dydrogesterone' or 'isopregnenone'. In addition, information on current studies was provided by the pharmaceutical company and by other workers in this field.

Results: As with other HRT preparations, there are no clinical studies of cardiovascular disease in women treated with the combination of estradiol and dydrogesterone As yet, there are no data on the effects of such therapies on endothelial function. Trials using surrogate measures of arterial function are in progress.

The available data concern blood pressure and metabolic risk markers. Estradiol/dydrogesterone therapy resembles other contemporary HRT formulations by having no effect on blood pressure. There are limited and conflicting data on the effects of such therapy on coagulation and fibrinolysis. Plasma levels of fibrinogen, a well-established risk marker for female cardiovascular disease, have been reported to be decreased, unchanged or increased by estradiol/dydrogesterone therapy. Levels of antithrombin III are unchanged or, in one study, fall. Protein S activity has been shown to fall by 15–20%. The overall impact on thrombosis is difficult to predict from this limited database. In contrast, there is extensive information on the effects of such therapy on serum lipid and lipoprotein concentrations. Levels of low-density lipoprotein (LDL) are lowered and those of high-density lipoproteins (HDL) increased, giving an 'estrogenic' lipoprotein profile. Triglyceride levels have been reported to be reduced, unchanged or increased, depending on the estrogen used. Serum lipoprotein (a) [Lp(a)] levels are reduced. Many of these actions would be predicted to reduce the risk of cardiovascular disease. Glucose tolerance is not affected by the estradiol/dydrogesterone combination. Striking reductions are seen in fasting insulin levels and in the insulin responses to a glucose load. Such reductions are desirable, especially in women at high risk of diabetes or cardiovascular disease

Conclusions: Overall, dydrogesterone does not appear to oppose the effects of estradiol on the cardiovascular risk profile, consistent with the high progestational specificity and low androgenicity seen with this steroid.

Comparative trials currently in progress are likely to show that, in terms of cardiovascular risk markers, dydrogesterone has distinct advantages over other commonly used progestogens.