Roles of Sex Steroid Hormones and Nitric Oxide in the Regulation of Sympathetic Nerve Activity in Women

To the Editor:

We read with great interest the article by Dr Carter et al dealing with the relationship between ovarian cycle and sympathoexcitation in premenopausal women. The results of their study demonstrated that the mid luteal phase of the ovarian cycle was characterized by sympathoexcitation, and that the degree of sympathoexcitation might be partially dependent on the degree of sex steroid surges. The authors indicated that increases in estradiol were associated with decreases in muscle sympathetic nerve activity during the mid luteal phase, suggesting that estradiol might be sympathoinhibitory. In addition, the strong inverse association between muscle sympathetic nerve activity and changes in estradiol/progesterone ratio was observed. The authors proposed that a dynamic interaction among estradiol, progesterone, and muscle sympathetic nerve activity may be related to the cardiovascular risk in postmenopausal women.

Evidence indicates that one of the mechanisms underlying the cardiovascular protective effects of estrogen may be the enhancement of NO production. In an in vitro study presented earlier, we demonstrated that 17β-estradiol significantly improved membrane microviscosity of red blood cells through the NO-dependent mechanism in postmenopausal women. It was also shown that hormone replacement therapy ameliorated membrane microviscosity of red blood cells with a concomitant increase in plasma NO-metabolite levels in postmenopausal women. In this context, it is strongly suggested that estrogen-induced NO might have a beneficial effect on the rheological behavior and the microcirculation in postmenopausal women. Vongpatanasin et al have reported that transdermal estrogen replacement therapy decreased muscle sympathetic nerve activity at rest in postmenopausal women. On the contrary, it was demonstrated that acute intravenous administration of NO synthase inhibitors might increase blood pressure and activate sympathetic nerve activity, suggesting that NO might act as a sympathoinhibitory substance. Therefore, we would like to know whether the degree of estradiol and estradiol/progesterone ratio during the ovarian cycle might be correlated with the endothelial function, such as plasma and urinary NO-metabolite levels or flow-mediated dilatation of the brachial artery, in the study of Dr Carter et al. It would be important to assess more precisely the relationships among estrogen, progesterone, and NO, and their role in the regulation of sympathetic nerve activity during sex steroid surges.

Disclosures

None.

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