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Abstract Reproduction Form B-1

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Title (type in CAPITAL LETTERS): EFFECT OF ESTROGEN ON NORADRENALINE RELEASE IN THE FEMALE RABBIT URETHRA

Aims of Study

It has been reported that estrogen replacement therapy increases urethral pressure and improves stress incontinence in postmenopausal women (1). Furthermore, estrogen has been shown to influence morphology and function of the lower urinary tract (2, 3). Several studies have shown that estrogen treatment decreases muscarinic acetylcholine receptor density of the rabbit urinary tract (4), and that estrogen causes an increase in number of post-junctional alpha-2 adrenoceptors, which contributes to the increased contractile response of alpha-2 adrenergic nerve mediated stimulation in the rabbit urethra (5). We have also reported that estrogen treatment reduce NO synthase activity, and inhibit the relaxation induced by nitrergic nerve stimulation in rabbit urethral smooth muscle (6). However, there is no information yet available on the effect of estrogen on noradrenaline release from urethra. Therefore, using microdialysis and high-performance liquid chromatography with electro-chemical detection (HPLC-ECD) (7), we evaluated the effects of estrogen on noradrenaline release and contractile response induced by electrical field stimulation (EFS) in rabbit urethral smooth muscles.

Methods

Female New Zealand white rabbits weighing 3.5 kg were divided into three groups: control group; sham operated, estrogen group; ovariectomized and treated with 0.1 mg/kg/day eatradiol polyphosphate, and ovariectomized group; ovariectomized and untreated. After 2 weeks, rabbits were killed by exsanguination after intravenous administration of sodium pentobarbital and urethral strips from all groups were mounted in thermostatically controlled organ baths filled with Krebs-Henseleit solution, and were attached to two L-shaped metal specimen holders. Atropine (1 μM) and indomethacine (1 μM) were present in the muscle baths throughout the experiment. One end of each strip was connected to a force-displacement transducer and isometric forces were recorded and monitored on a pen writing recorder.

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The microdialysis probe (O-P-100-10, Eicom, Kyoto, Japan) was inserted through the muscle strip and the inlet cannula of the probe was connected to a microinfusion syringe pump. In order to minimize the degree of breakdown of noradrenaline, Ringer solution (pH 7.4) containing 0.05 mM ascorbic acid was continuously perfused at a rate of 2 μl/min. The dialysate during EFS (supramaximum voltage, pulse duration 0.5 ms, frequency 5-80 Hz and train of pulse 2 s, interval between stimulations 1 min; ten muscle contractions were induced by shocks.) was collected and a volume of 10 μl of the dialysate of each sample was injected into the noradrenaline determination system. The amount of noradrenaline released in the dialysate was calculated by reference to the peak area of the standard noradrenaline solution (0.1 pmol) by a chromatogram recorder.

Results

The detection limit of noradrenaline was 0.01 pmol/injection. EFS caused frequency-dependent contractions and noradrenaline releases in all groups, and pretreatment with tetrodotoxin (1 μM) and guanethidine (50 μM) almost completely inhibited the contractions and noradrenaline releases. The contraction and noradrenaline release induced by EFS in ovariectomized group were significantly lower than those in the control. Estrogen replacement returned the values to the control levels. There were not significant differences in the contraction and noradrenaline release between the estrogen and control groups.

Conclusions

The data suggest that ovariectomy caused decreases in the contraction and noradrenaline release induced by EFS in the rabbit urethra, and that estrogen replacement returned them to the control values. These results may support the usefulness of estrogen replacement therapy for urinary incontinence in postmenopausal women.

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