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Testosterone facilitates the baroreceptor control of reflex bradycardia: role of cardiac sympathetic and parasympathetic components.

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Reported clinical and experimental findings have shown that baroreflex control of heart rate is attenuated in women compared with men. This study investigated whether the sexual dimorphism in baroreflex function relates to the ability of the male hormone testosterone to facilitate baroreflex responsiveness. Relative contributions of the vagal and sympathetic autonomic components to testosterone modulation of baroreflex function were also investigated. Baroreflex curves relating changes in heart rate to increases or decreases in blood pressure evoked by phenylephrine and sodium nitroprusside, respectively, were constructed in sham-operated rats and castrated rats with and without testosterone replacement. Slope of the curves was taken as an index of baroreflex sensitivity (BRS PE and BRS NP). Castration (for 10 days) significantly reduced plasma testosterone levels and attenuated reflex bradycardia, as indicated by significantly smaller BRS PE in castrated rats compared with values in sham-operated rats (-0.85 +/- 0.07 vs. -1.51 +/- 0.10 beats/min per mm Hg). Testosterone replacement in castrated rats restored plasma testosterone and BRS PE to levels similar to those of sham-operated rats. Muscarinic blockade by atropine caused 55% reduction in BRS PE in sham-operated rats, an effect that was significantly ($p < 0.05$) attenuated in castrated rats and restored to intact levels after testosterone supplementation. beta-Adrenergic blockade by propranolol caused slight and insignificant decreases in BRS PE. Castration and testosterone supplementation had no effect on BRS NP, ruling out a modulatory effect of testosterone on reflex tachycardia. These data provide the first experimental evidence of a favorable role for testosterone in baroreceptor control of reflex bradycardia. Further, baroreflex modulation by testosterone appears to be autonomically mediated and involves an enhancement of cardiomotor vagal activity.

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