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Effects of Medical or Surgical Castration on Erectile Function in an Animal Model

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The goal of this study was to investigate the effects of medical castration (luteinizing hormone-releasing hormone [LH-RH] agonist treatment) or surgical castration on erectile function in an animal model. New Zealand White male rabbits were either kept intact (control); surgically orchietomized; or treated for 2, 4, or 8 weeks with the LH-RH agonist leuprolide acetate (107 µg/kg/mo). At 2 weeks, plasma testosterone levels of orchietomized and leuprolide acetate-treated animals were 12.8% and 57.4% of intact control animals, respectively. Erectile function was assessed by continuously recording systemic arterial pressure (SAP) and intracavernosal blood pressure (ICP) and determining the ICP:SAP ratios in response to electrical stimulation of the pelvic nerve at varying frequencies (2.5–32 Hz). Androgen deprivation by surgical (orchietomy) or medical (leuprolide acetate) castration reduced ICP at all frequencies tested but did not alter SAP. Administration of the phosphodiesterase type 5 inhibitor vardenafil (10 µg/kg) did not enhance ICP in surgically orchietomized or leuprolide acetate-treated animals. Nitric oxide synthase and arginase activities in the corpus cavernosum were not significantly altered by surgical or medical castration. Further, Masson trichrome staining of erectile tissue from androgen-ablated animals showed a reduction in smooth muscle content. These data demonstrate that androgen deprivation achieved by surgical or medical castration adversely affects penile hemodynamics and erectile function without producing significant changes in the activities of nitric oxide synthase or arginase. We conclude that androgen deprivation produces structural alterations in the corpus cavernosum leading to corporal veno-occlusive dysfunction.

Key words: Androgens, trabecular smooth muscle, corpus cavernosum, veno-occlusion, vardenafil, leuprolide acetate

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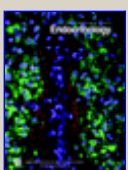
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