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Molecular mechanisms of vacuum therapy in penile rehabilitation: a novel animal study.

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BACKGROUND: Penile rehabilitation (PR) is widely applied after radical prostatectomy. Vacuum erectile device (VED) therapy is the one of three PR methods used in the clinical setting that improve erectile function (EF) and is the only PR method which may preserve penile length. However, its unknown mechanism hampered doctors' recommendations and patients' compliance.

OBJECTIVES: To assess the effects of VED therapy on erectile dysfunction (ED) in a rat model of bilateral cavernous nerve crush (BCNC) and to investigate the molecular mechanism of VED in postprostatectomy ED.

DESIGN, SETTING, AND PARTICIPANTS: This was an experimental study using Sprague-Dawley rats in three groups: sham, BCNC, and BCNC plus VED.

INTERVENTION: Intervention included BCNC, electrical stimulation of the cavernous nerve (CNS), and VED therapy.

MEASUREMENTS: At the end of a 4-wk period, CNS was used to assess EF by maximum intracavernosal pressure (ICP)/mean arterial pressure (MAP) ratio and duration (area under the curve [AUC]). For the structural analyses, whole rat penis was harvested. Terminal deoxynucleotidyl transferase biotin-dUTP nick end labeling assay was used for the assessment of apoptotic indices (AI). Immunohistochemistry was performed for endothelial nitric oxide synthase (eNOS), α -smooth muscle actin (ASMA), transforming growth factor beta 1 (TGF- β 1), and hypoxia inducible factor-1 α (HIF-1 α). Staining for Masson's trichrome was utilized to calculate the smooth muscle/collagen ratios.

RESULTS AND LIMITATIONS: EF was improved with VED therapy measured by ICP/MAP ratios and AUC. VED therapy reduced HIF-1 α expression and AI significantly compared with control. Animals exposed to VED therapy had decreased TGF- β 1 expression, increased smooth muscle/collagen ratios, and preserved ASMA and eNOS expression.

CONCLUSIONS: To our knowledge, this is the first scientific study to suggest that VED therapy in the BCNC rat model preserves EF through antihypoxic, antiapoptotic, and antifibrotic mechanisms.

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