Optimizing sexual function outcomes after radical prostatectomy

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A paradigm shift in the management of erectile dysfunction (ED) secondary to prostate cancer surgery occurred with the introduction of cavernous nerve–sparing radical prostatectomy by Walsh[1] and the widespread availability of effective, safe and well-tolerated pharmacotherapies for ED.[2,3] Despite these advances and evolving surgical techniques, radical prostatectomy remains associated with a significant incidence of ED and decreased quality of life.

Al Shaiji and colleagues[4] have reported a well-balanced contemporary review of "penile rehabilitation" concepts and contemporary strategies to optimize recovery of sexual function. However, it is clear there remains a significant gap between pathophysiology and treatment. Options available to the clinician remain "reactive" to the damage that has occurred at surgery or the subsequent downstream changes that result from compromise of cavernous nerve signaling due to intraoperative injury (for nerve-sparing procedures). A rapid growth in basic science understanding, specifically the response of tissues to injury on a molecular level, promises to fuel further advances. The treatment goal remains optimization of the cavernous nerve response to injury at surgery (with preoperative optimization of endogenous response) to promote nerve regeneration and neuroprotection. In this way, the collateral damage of surgery could be minimized, including smooth muscle and endothelial compromise, and therapy could be initiated in the surgical suite. It is likely that these future treatments will require a combination of strategies to counteract primary and secondary injury, including enhancing axonal regrowth, inhibition of apoptosis, and promotion of synaptic plasticity via neurotrophic factors, adult-tissue derived stem cells, or neuro/smooth muscle protectants.[5] For non–nerve-sparing procedures, interventions are more limited, but may include the introduction of seeded nerve "bridges" or "scaffolds" to counter the significant physical gaps following cavernous nerve excision.

Where are we now? As surgeons, new anatomical knowledge of cavernous nerve distribution may further advances in surgical technique.[6] Research continues concerning pathophysiology and potential treatment. Clinically, the data are incomplete but compelling with ample basic science evidence for beneficial effects of phosphodiesterase type 5 (PDE5) inhibitors and intracavernous injections in this population. Al Shaiji and colleagues[4] have correctly emphasized the need to identify patient goals and provide an overview of the ever-evolving literature and its limitations. If considering penile rehabilitation, early initiation is *likely* best, given the time-dependent penile changes that occur after surgery. Montorsi and colleagues[7] must be commended for the REINVENT trial, but care must be taken in extrapolating results to all men undergoing radical prostatectomy as this was a select, highly functioning population preoperatively and there are methodological limitations to the study as well. The shift to on-demand PDE5 inhibitor use *exclusively* is not supported for all patients; rather, on-demand use is an evidence-based option with excellent results for the appropriate patient.[8] It is identifying the correct patient for on-demand versus chronic PDE5 inhibitor (with its attendant benefits), injection therapy or combination approaches that is most difficult. Importantly, attempts at intercourse even without medication (penile physiotherapy) once or twice weekly may benefit recovery and this may be incorporated as part of any sexual rehabilitation program.[7]

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