

the clinical setting for these proposes. Nevertheless, the mechanism of finasteride action in stopping bleeding is still unknown.

The authors of the present paper used male beagle dogs to assess dynamic contrast enhanced magnetic resonance imaging as a biological marker of in vivo changes in microcirculation in the prostatic suburethral region. They found that subjects in the finasteride group had decreased microcirculation, as expressed by lower and slower contrast enhancement, and as quantified by increased Tmax, and decreased A and kep in the prostatic suburethral area. They concluded that finasteride would decrease the prostatic microcirculation and therefore diminish prostatic bleeding in BPH and TURP.

In a recent experimental paper, Canda et al. (1) evaluated the effects of finasteride on the vascular surface density (VSD), number of microvessels (NVES) and vascular endothelial growth factor (VEGF) expression of the rat prostate. After studying 19 adult rats, the authors found that the mean prostatic weights were decreased significantly in rats given finasteride ($p=0.0001$). On the other hand, finasteride does not seem to decrease VSD, NVES and VEGF expression at the level of the rat prostate. The effect of reduction of bleeding in BPH is more likely to be due to its effect on shrinking glandular hyperplasia, which might enhance vessel wall stability, rather than decreasing overall vascularity (1).

From these two papers, we can infer that the exact mechanism of action of finasteride on the prostatic vessels is still open to research and discussion.

Reference

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RECONSTRUCTIVE UROLOGY

Gender Specific Chronological and Morphometric Ssessment of Fetal Bladder Wall Development

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Purpose: To enhance our understanding of sonographically visible alterations in bladder wall thickness, we delineated phenotypic changes occurring in developing smooth muscle cells of the fetal and postnatal bladder with respect to gender specific differences.

Materials and Methods: Bladders of 30 male and 18 female fetuses and 4 stillborn infants were immunostained with an alpha-smooth muscle actin antibody. Morphological and morphometric assessment was performed with the assistance of an image analysis system.

Results: Alpha-smooth muscle actin expression in fetal bladder wall was detectable at 9 weeks of gestation. Bladder wall thickness and mean profile area of smooth muscle bundles increased significantly with advancing gestation, mediated by linear growth patterns. Fetal bladder wall development occurred uniformly, unrelated to gender.

Conclusions: Although the lower urinary tract emerges in a gender specific way, our results suggest that in normal fetal growth detrusor muscle formation proceeds independent of genital sex.

Editorial Comment

The current paper deals with histologic and morphometric assessment of 18 female and 30 male bladder specimens of human fetuses at 9 to 35 weeks of gestation. The findings underline the theory of those favoring a gender independent development of the lower urinary tract. At various times during fetal development no differences between male and female specimens of the muscular structure and configuration of the bladder was seen contrary to previous reports (1). Furthermore, the growth of the muscular bladder wall was linear with gestational age.

This is a very elegant study with nice fetal specimens and reveals several interesting aspects. Apart from the main conclusions outlined above it was also interesting to see that the bladder seems to develop relatively late compared to the gut. At 9 weeks, only immature smooth muscle cells were observed in the bladder whereas the bowel already demonstrated clearly visible inner and outer muscular layers. In addition, smooth muscle cells developed first in the ventral portion of the bladder close to the dome. One may speculate that this has something to do with the umbilical vessels.

Only through the development of the bladder the muscle bundles start to change their shape, direction and intermingling. Unfortunately we do not get any clue from this study when and how neural development starts.

With studies like that we get important information for further tissue engineering of the urinary bladder. We suppose that at the time of in vitro cultivation intermingling and growth may not be our major goal but that we somehow have to have functional and growth stimulations at the time of implantation, which will bring our cultivated smooth muscle cells to a structure, which resembles the native bladder.

Reference

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Miniature Intravesical Urethral Lengthening Procedure for Treatment of Pediatric Neurogenic Urinary Incontinence

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Purpose: Resistance to flow in a fluid conduit is proportional to tube length divided by the radius to the fourth power (Poiseuille's law). We report the results of a miniature intravesical urethral lengthening procedure where outlet resistance is increased by minimizing the diameter of the intravesical urethral tube.

Materials and Methods: Nine pediatric patients with preoperative intractable incontinence underwent the miniature intravesical urethral lengthening procedure along with continent catheterizable stoma (9 patients)

and bladder augmentation (8). The intravesical portion of the urethral lengthening was 3 cm (traditionally 6 cm), and the urethra was tubularized around an 8Fr feeding tube (traditionally a 16Fr catheter). After the tubularized caudal portion was tunneled under the trigone the cephalad part of the urethra was placed as an onlay to the posterior bladder wall without ureteral reimplantation.

Results: At a mean followup of 31 months (range 10 to 47) 8 patients reported dry intervals of 3 hours or more, with minor leak per urethra only if they were overdue on the catheterization schedule. Mean postoperative abdominal leak point pressure was 71 cm H₂O (range 28 to 116). Upper tracts were well preserved in all patients. One patient required bladder neck closure for intractable incontinence.

Conclusions: The miniature intravesical urethral lengthening procedure requires minimal bladder tissue and is easy to perform. It appears to be an effective alternative in bladder neck reconstructive techniques, avoiding the need for ureteral reimplantation due to its small size, while functioning as a pop-off valve when the bladder is overly full. This procedure should be avoided in patients who lack a trigonal bar.

Editorial Comment

The reconstructive surgeon strives to benefit the patient with improved surgical approaches. Urinary incontinence, especially in patients with a neurogenic bladder, presents a significant surgical challenge and requires high level of experience (1). The technique of Kropp further developed by Pippi-Salle demonstrated the step-by-step perfection of the more advanced approach with the presented MIULP technique. This technique refreshed specific aspects of current approaches and further developed thoughts that are reflected in the described modified technique. However, the surgeon's responsibility is continued with the surgeon's legacy and sense of duty through long-term patient follow-up.

On the one hand, the tunneling of the lengthened urethra reduces the chance of fistula development; however, on the other hand the smaller urethral diameter might cause difficulties during catheterization. In our experience, the majority of patients prefer to use a catheterizable stoma. With the improved concept of regular sterile intermittent catheterization, there is a significant reduction in urinary infections and stone occurrence today, which reduces the chance of an endoscopic surgical approach.

The increased leak point pressure meets the patient's request to be dry and the "pop-off" valve makes allowance to limit the bladder pressure. With the introduction of Botulinum toxin, bladder augmentation can be often avoided or at least delayed securing the low-pressure storage (2).

This urethral lengthening technique might be a legitimate technique for the experienced surgeon to improve patient's long-term outcome.

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