progression varies from 36% to 72% in the literature (1). In our Institution, the progression in 300 patients was 37% after 5 years of follow-up.

It is important for the urologist the definition and the description of the several kinds of positive surgical margins (2):

a) Positive surgical margins are defined as cancer cells touching the inked surface of the prostate;

b) Iatrogenic surgical margin occurs whenever there is a transection of the intraprostatic tumor. If this occurs, one cannot determine whether there is extraprostatic extension in the region of incision into the prostate as the edge of the prostate has been left in the patient. Unless there is extraprostatic extension in other areas of the surgical specimen, the pathologic stage is called pT2+;

c) Non-iatrogenic surgical positive margin occurs whenever there is an inability to widely excise tumor showing extraprostatic extension.

It is worth mentioning the possibility of positive surgical margins in normal prostatic glands. This is not routinely reported by the pathologist; however, it is very important to report in cases of limited carcinoma in the surgical specimen. In these cases, biochemical (PSA) progression following surgery may be due to normal glands left in the patient. In our Institution, no patient with limited carcinoma in the specimen had biochemical progression, except 3 patients. Reviewing the prostatectomy slides, we found that all 3 patients had frequent and extensive positive surgical margins in normal glands.

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INVESTIGATIVE UROLOGY

Protein oxidation as a novel biomarker of bladder decompensation

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Objective: To measure the degree to which partial bladder outlet obstruction (PBOO) results in oxidative bladder damage, which subcellular components of the bladder are affected and whether these changes correlate with bladder function.

Materials and Methods: In all, 32 rabbits were divided into four groups. Each group underwent PBOO for 1, 2, 4, and 8 weeks, respectively. Bladder tissue from each group was homogenized and separated into subcellular

fractions via differential centrifugation. The carbonyl content within the subcellular fractions, including the nuclear, mitochondrial, and microsomal pellets, was then quantified by dot blot analysis.
Results: Total bladder oxidation increased with duration of obstruction across all subcellular fractions. The largest increase in total oxidation occurred between 4 and 8 weeks. Protein oxidation density in the nuclear and microsomal fractions both showed increases at 2 weeks obstruction, decreases at 4 weeks, and then large increases at 8 weeks. The increase in protein oxidation density between 4 and 8 weeks obstruction was most pronounced in the microsomal fraction.
Conclusions: Overall bladder protein oxidation increased with the duration of obstruction and increased at a greater rate during the transition to decompensation. Furthermore, the subcellular fraction that exhibited the most oxidation was the microsomal pellet. The amount of protein oxidation correlated with the functional changes in the bladder.

Editorial Comment

In this interesting and welcome experimental study, the authors created surgically partial bladder outlet obstruction (PBOO) in 32 rabbits. They were interested to analyze whether oxidative stress measured after PBOO would correlate with the function of the bladder and whether markers of oxidative stress might serve as a biomarker of the progression to bladder decompensation.

The authors presented clear evidence that protein oxidation occurs to a significant degree in the PBOO rabbit bladder. They concluded that overall bladder protein oxidation increases with the duration of obstruction and increases at a greater rate during the transition to full decompensation. They speculated that in the clinical setting, the urologist could obtain tissue from the bladder of a patient with BPH and analyze it specifically for microsomal protein oxidation and determine the degree to which the patient is moving towards decompensation. Of course, theoretically, it could be done, but probably it will be hard to put in clinical use.

The authors are to be congratulated for this elegant study that opens new avenue for the understanding and management of benign prostatic hyperplasia consequences.

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The potential of hormones and selective oestrogen receptor modulators in preventing voiding dysfunction in rats

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Objective: To investigate whether oestrogen, selective oestrogen receptor modulators (SERMs), and growth hormone (GH) can prevent the development of voiding dysfunction in a postpartum postmenopausal rat model of voiding dysfunction.
Materials and Methods: Immediately after spontaneous delivery, nine primiparous Sprague-Dawley rats served as uninjured controls (sham group) and 54 underwent intravaginal balloon dilation. On day 7, the 54 subject rats underwent bilateral ovariectomy. A week later, six treatment groups of nine rats were randomized to receive: normal saline (injured control group), 17beta-oestradiol (E(2)), raloxifene, levormeloxifene, GH, or

GH + E(2). The treatment groups received daily subcutaneous injections for 3 weeks. The effects of hormone treatment were examined by conscious cystometry at the end of the study. Voiding dysfunction was defined to include overactive bladder and sphincter deficiency.
Results: The sham rats had a mean (sd) voiding frequency of 3 (0.87) times in 10 min and a bladder capacity of 0.43 (0.13) mL with smooth cystometry curves. The number of rats in each treatment group (each group contained nine rats) that had voiding dysfunction was as follows: E(2), three; raloxifene, six; levormeloxifene, four; and controls, four ($P > 0.05$ among the groups). Only one rat in the GH-treated group and no rats in the GH + E(2)-treated group had voiding dysfunction, which was significantly less in the GH + E(2)-treated group than in the controls ($P = 0.041$).
Conclusion: This functional data suggest that the development of voiding dysfunction can be prevented by short-term administration of GH and GH + E(2) in our rat model. SERMs and E(2) alone seem to have no therapeutic effect.

Editorial Comment

This is a wished study by Dr. Lue and collaborators that have been working on this topic for the last years. They analyzed if short-term therapy with ultra-low dose of estrogen, selective estrogen receptor modulators (SERMs), and growth hormone (GH) can prevent the development of voiding dysfunction in a postpartum, postmenopausal voiding dysfunction rat model. By using conscious cistometry, developed in its own laboratory, the authors found that short-term therapy with E2, SERMs and GH suggest that, in the dosage and duration used, GH and GH + E2 seem to prevent the development of voiding dysfunction while E2 alone and SERMs do not have significant effects. With this paper, we are able to better understand the effect of these hormones on voiding, with the consequent clinical implications for treating and preventing post-partum and postmenopausal voiding dysfunction.

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RECONSTRUCTIVE UROLOGY

A collagen matrix derived from bladder can be used to engineer smooth muscle tissue

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We have previously demonstrated that a collagen matrix derived from lamina propria, commonly known as bladder submucosa (BSM matrix), is a suitable biomaterial for several urologic applications, including reconstruction of the bladder and urethra in experimental models and clinical trials. In the present study, we evaluated the physical properties of BSM as well as its biocompatibility, cellular interactions, and ability to support the formation of functional tissue in order to determine whether this biomaterial could serve as a matrix for urinary smooth muscle tissue engineering. BSM matrix resembles the extracellular matrix of bladder submucosa in its native structure, composition, and mechanical properties. BSM matrix supported normal