ASSESSMENT OF BLADDER AND URETHRAL SPHINCTER FUNCTION BEFORE AND AFTER RADICAL RETROPUBIC PROSTATECTOMY

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ABSTRACT

Purpose: In a prospective study we analyzed the effects of radical retropubic prostatectomy (RRP) on detrusor and urethral sphincter function by comparing urodynamic status preoperatively with that during longitudinal followup.

Materials and Methods: A total of 49 consecutive patients underwent urodynamics with pressure flow studies and Valsalva leak point pressure measurements 3 to 7 days before RRP (baseline), and then 1 and 8 months after surgery. We assessed bladder compliance, detrusor overactivity, detrusor contractility and intrinsic sphincter deficiency (ISD).

Results: There was no significant change in detrusor overactivity at 1 and 8 months of followup. Decreased bladder compliance was observed in 20.4% of patients at baseline, and in 38.7% and 30.6% at 1 and 8 months, respectively. De novo decreased compliance was detected in 18.4% and 10.2% of patients at the same points. Impaired bladder compliance was comparable to that before surgery in 20% of cases. Impaired detrusor contractility was detected in 42.8% of patients at baseline, and in 61.2% (p <0.05) and 42.8% at 1 and 8 months, respectively. De novo hypocontractility was observed in 28.6% and 10.2% of patients at 1 and 8 months, respectively. A strong association between detrusor overactivity and ISD was observed at 1 and 8 months (p <0.01).

Conclusions: Following RRP detrusor hypontractility and decreased bladder compliance represent de novo transient dysfunction probably due to bladder denervation and an established condition not influenced by the operation. The strong association between overactivity and ISD suggests that stress urinary incontinence increases urethral afferent nerve activity and induces involuntary detrusor contractions.

KEY WORDS: urethra, prostatectomy, urodynamics, bladder

There are a number of reports describing continence status after radical retropubic prostatectomy (RRP) but there is little information on bladder and urethral sphincter function followed with time.[1] Recently it was reported that following RRP a number of patients were affected by impaired detrusor contractility,[2] which may coexist with sphincter weakness, bladder overactivity and/or decreased compliance.[3] However, to our knowledge the exact roles of impaired detrusor contractility, overactivity and decreased compliance after RRP have not been clarified. These abnormalities may occur de novo as a consequence of bladder denervation during surgery, they may be induced by long-term outlet obstruction or they can be conditions related to an aging bladder.[4] Furthermore, it was suggested that after RRP many patients void with minimal detrusor contraction and/or abdominal straining because of acquired behavior adapted to voiding through a low resistance urethra.[2,4] A comparison between detrusor and sphincter function before and after prostatectomy would help clarify this issue. In this prospective study we analyzed the effects of RRP on detrusor and urethral sphincter function early and late following surgery. We also investigated the issue of de novo bladder dysfunction by comparing urodynamic status preoperatively and during longitudinal followup.

PATIENTS AND METHODS

Enrollment and clinical evaluation. We included 49 consecutive patients with clinically localized prostate cancer (cT1c to cT2b) undergoing radical retropubic prostatectomy. All patients were informed about the scientific nature of the investigations and provided written informed consent. Anatomical RRP was performed by a single surgeon with nerve sparing and bladder neck preserving techniques based on clinical stage, cancer location, patient age and preoperative sexual function.[5] Following history and physical examination we performed urodynamic evaluation and Valsalva leak point pressure (VLPP) measurements 3 to 7 days before RRP (baseline evaluation), and then 1 and 8 months after surgery. In incontinent patients we recorded the number of pads (0 to 1 vs greater than 1 daily) and urgency/urge incontinence.

Urodynamics. Urodynamic assessment was done according to International Continence Society Standards[6] and involved water cystometry with 37C normal saline solution at a filling rate of 50 ml per minute. A 6Fr double lumen Nelaton transurethral catheter was used for infusion and for recording intravesical pressure, while a 10Fr intrarectal balloon catheter was used for recording abdominal pressure. We assessed detrusor overactivity, maximum bladder capacity and bladder compliance, defined as normal—greater than 20, impaired—10 to 20 and poor—less than 10 ml/cm water.[2] At capacity patients were asked to void, and the maximum flow rate (Qmax), detrusor pressure at maximum flow rate (pdetQmax) and post-void residual volume were recorded. Bladder outlet obstruction (BOO) and detrusor contractility were assessed by pressure flow studies using Schafer's

nomogram.[7] Grades 0 to II bladder outlet conditions were considered unobstructed, while grades III to VI were considered obstructed. The nomogram was also used to classify detrusor strength as normal, weak or very weak. During the pressure flow study voiding by straining or by detrusor contraction was recorded.[2]

VLPP was evaluated with the patient upright at a volume of 250 ml after removing the urethral catheter. Defined as the lowest abdominal pressure induced by cough or Valsalva maneuver causing visible stress incontinence, it provided an assessment of intrinsic sphincter deficiency (ISD).[8]

Data analysis. Statistical analysis was performed using tests for repeated nonparametric data, namely the Friedman and Cochran Q tests. The Bonferroni correction was applied to the Wilcoxon and McNemar tests for post hoc multiple comparisons. The chi-square test was applied for trend. The level of statistical significance was set at $p < 0.05$. All data analyses were performed using SPSS, release 10.1.1 for Windows (SPSS, Inc., Chicago, Illinois).

RESULTS

Demographics. Mean patient age $\pm$ SD was 65 ± 5 years. RRP involved bilateral, unilateral and nonnerve sparing techniques in 28, 12 and 9 patients, respectively. Bladder neck preservation was performed in all cases.

Baseline evaluation. Seven patients reported urgency and urge incontinence but none used pads or collecting devices. Table 1 lists urodynamic results. BOO was observed in 28 patients (57.1%) and detrusor overactivity was noted in 27 (55.1%). Ten patients (20.4%) with BOO showed impaired or poor compliance. Impaired detrusor contractility was detected in 21 cases (42.8%) and in 7 it was associated with detrusor overactivity. Table 2 lists mean bladder compliance, pdetQmax and Qmax. The association of impaired detrusor contractility with BOO was identified in 8 patients, while the association of detrusor overactivity with BOO and with impaired detrusor contractility was noted in 17 and in 7 cases, respectively. On VLPP examination no patients showed ISD.

Followup at 1 month. A total of 48 patients complained of stress urinary incontinence, of whom 9 had urge urinary incontinence and 15 used more than 1 pad daily. Table 1 shows urodynamic results. BOO was observed in 4 cases (8.1%) due to an anastomotic stricture. Two of these patients complained of persistent obstructive symptoms. The mean flow rate was 4.3 ± 0.5 ml per second, which was lower than in patients without stricture. Detrusor overactivity was detected in 33 cases (67.3%). Mean bladder compliance was significantly decreased compared to baseline with 19 patients (38.7%) showing impaired or poor compliance (p

TABLE 2. *Mean bladder compliance, pdetQmax and Qmax in 49 patients preoperatively, and 1 and 8 months following RRP*

|	Mean Baseline $\pm$ SD	Mean 1 Mo $\pm$ SD	Mean 8 Mos $\pm$ SD
Bladder compliance (ml/cm water)	37.1 ± 14.9	26.9 ± 12.7	29.2 ± 12.8
pdetQmax (cm water)	62.4 ± 23.8	32.6 ± 14.5	34.2 ± 13.7
Qmax (ml)	8.6 ± 3.3	12.6 ± 6.8	13.6 ± 5.7

Baseline vs 1 and 8 months $p < 0.05$.

< 0.001, table 2). Nine of these patients (18.4%) showed de novo decreased compliance. Impaired detrusor contractility was diagnosed in 30 patients (61.2%), which was a significant increase compared to the preoperative number ($p < 0.05$). In 16 patients (32.6%) detrusor hypocontractility grades were comparable to those before surgery, while 14 (28.6%) showed de novo hypocontractility. Five patients (10.2%) with a diagnosis of impaired detrusor contractility before surgery had normal detrusor strength. There was a significant decrease in mean pdetQmax and a significant increase in mean Qmax compared to baseline (table 2). The figure shows pressure flow studies, as assessed by Schafer's nomogram at baseline, and at 1 and 8 months of followup. There was no significant difference in the number of strain voiders compared to baseline (table 1). We did not find any significant relationship between voiding by straining and BOO or detrusor hypocontractility. VLPP was determined in 40 patients (81.6%) with a mean value of 54.4 ± 18.2 cm water. The association of impaired detrusor contractility with overactivity, which was detected in 18 patients (36.7%), was significantly increased compared to baseline ($p < 0.05$). The association of detrusor hypocontractility with ISD was observed in 28 patients (57.1%), and the association of detrusor overactivity and ISD was found in 32 (65.3%, $p < 0.01$).

Followup at 8 months. A total of 29 patients (59.1%) complained of stress urinary incontinence, including 3 with urge urinary incontinence. Nine patients required more than 1 pad daily. Anastomotic strictures were treated and BOO disappeared in all 4 cases. Table 1 shows urodynamic results. Detrusor overactivity persisted in 32 patients (65.3%). Bladder compliance was significantly worse compared to baseline with 15 patients (30.6%) showing impaired or poor compliance ($p < 0.001$, table 2). Impaired detrusor contractility was diagnosed in 21 patients (42.8%), of whom 16 had preexisting hypocontractility and an additional 5 with weak detrusor strength diagnosed at 1 month had persistent hypocontractility. Nine patients with weak detrusor contractility at 1 month returned to normal detrusor strength at 8 months. A significant decrease in mean pdetQmax and a significant

TABLE 1. *Urodynamic dysfunction in 49 patients observed preoperatively, and 1 and 8 months following RRP*

Urodynamic Dysfunction	No. Baseline (%)	No. 1 Mo (%)		No. 8 Mos (%)	
		Preexisting	De Novo	Preexisting	De Novo
Bladder outlet:					
Normal	21 (42.9)	21 (42.9)	45 (91.8)	21 (42.9)	49 (100)
Obstructed	28 (57.1)	28 (57.1)	4 (8.2)	28 (57.1)	0
Detrusor:					
Normal	22 (44.9)	16 (32.7)	0	17 (34.7)	0
Hyperactive	27 (55.1)	27 (55.1)	6 (12.2)	27 (55.1)	5 (10.2)
Bladder compliance:					
Normal	39 (79.6)	30 (61.2)	0	34 (69.4)	0
Impaired or poor	10 (20.4)	10 (20.4)	9 (18.4)	10 (20.4)	5 (10.2)
Detrusor contractility:					
Strong	2 (4.1)	0	0	0	0
Normal	26 (53.1)	14 (28.6)	5 (10.2)	23 (47)	5 (10.2)
Weak	21 (42.8)	16 (32.6)	9 (18.4)	16 (32.6)	5 (10.2)
Very weak	0	0	5 (10.2)	0	0
Voiding:					
Without straining	40 (81.6)	38 (77.5)	0	34 (69.4)	0
With straining	9 (18.4)	9 (18.4)	2 (4.1)	9 (18.4)	6 (12.2)
ISD	0	0	40 (81.6)	0	32 (65.3)

De novo dysfunction at 8 months is relative to baseline.
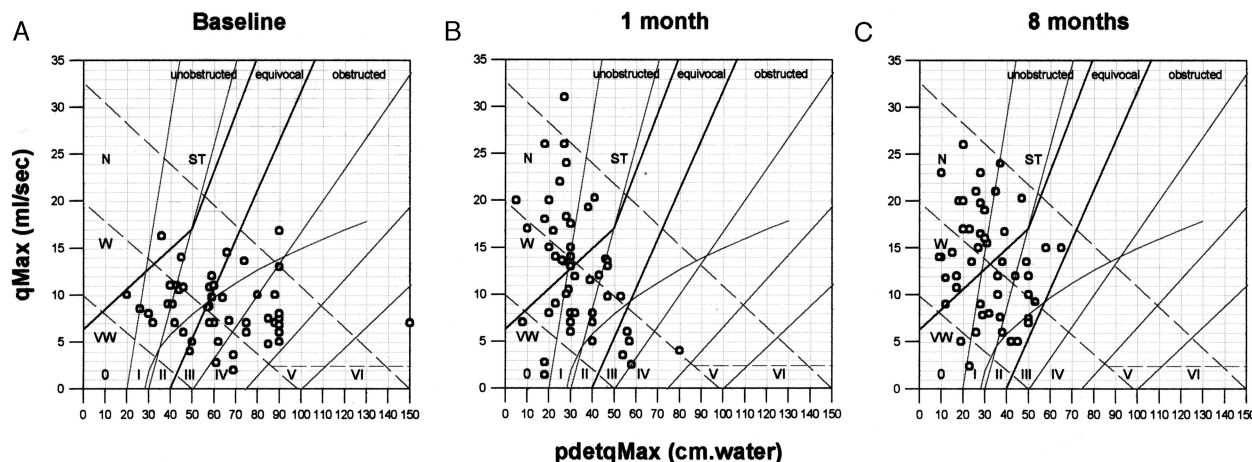
Detrusor pressure at pdetQmax and Qmax, as assessed by Schafer's nomogram in 49 patients observed preoperatively, and 1 and 8 months following RRP. Baseline pdetQmax and Qmax vs 1 and 8 months p <0.05. *n*, normal. *ST*, strong. *W*, weak. *VW*, very weak.

increase in mean Qmax persisted compared to baseline (table 2, see figure). We did not find any significant difference in the number of strain voiders compared to baseline and 1 month of followup (table 2).

VLPP was determined in 32 patients (65.3%) with a mean value of 66.7 ± 21.4 cm water. The association of impaired detrusor contractility with overactivity was detected in 11 patients (22.4%) with no significant difference compared to baseline. Detrusor hypocontractility with ISD was observed in 15 patients (30.6%), and the association of detrusor overactivity and ISD was found in 26 (53%) (vs baseline, p <0.01). Finally, when stratifying detrusor overactivity, bladder compliance and ISD by nerve sparing status, no significant linear by linear association was detected at 1 or 8 months of followup.

DISCUSSION

The pathophysiology of urinary incontinence following RRP is relevant because it is the most distressing problem in the majority of patients.[3] Wide anatomical dissection around the prostate during surgery may disrupt afferent and efferent innervation of the trigone, neobladder neck and posterior urethra, causing outlet incompetence and partial denervation of the detrusor muscle. Therefore, in addition to any preoperative pathological conditions, a substantial proportion of patients are subject to various postoperative abnormalities, including detrusor overactivity, impaired detrusor contractility and voiding by straining, sphincteric weakness and incontinence.[2–4]

To our knowledge there are no consistent data available on how detrusor and sphincter function are affected by the operation. An explanation could be that it is difficult to perform a complete evaluation of lower urinary tract with a routine pressure flow study. Although uroflowmetry can help diagnose some dysfunction, ie BOO due to anastomotic strictures, a pressure flow study remains the most important investigation for assessing lower urinary tract function after RRP.

There are some obvious limitations to the current study. Urodynamic followup was limited to 8 months, whereas it is known that changes in urinary function continue for 12 months or longer.[4] However, the 8-month period of close observation combined with periodic urodynamic evaluation established some definite trends, which continued for at least 12 months and received some confirmation from repeat clinical evaluation. Although eventual patient outcome, recurrence, progression, metastases, other therapies and disease specific mortality were important, they were not the objectives of the current study.

Hellstrom et al analyzed urodynamic measurements preop-

eratively and 6 months later in 19 patients, noting a significant decrease in bladder wall compliance, and a significant increase in vesical and abdominal pressure during voiding, implying that the 2 conditions are related to partial surgical decentralization of the bladder.[9] Hammerer and Huland examined vesical-sphincter function in 82 patients before RRP, and 6 to 8 weeks and 6 months (9 cases) subsequently.[10] However neither of these studies provided data on pressure flow studies and detrusor contractility grades. Kleinhans et al reported a significant decrease in maximum detrusor pressure and a significant increase in the maximum flow rate after prostatectomy[11] but it remains unclear if the condition was related to obstruction relief or to changes induced by bladder denervation.

At 1 month after surgery 28.6% of patients in our series showed a worsening of detrusor contractility grade compared to baseline. In these patients de novo hypocontractility may have been the result of transient bladder denervation, which persisted in 10% at 8 months. None of these patients showed a post-void residual volume of greater than 100 ml. By comparing urodynamic status preoperatively and during longitudinal followup we observed that detrusor hypocontractility was an established condition in about 32% of patients, which may be related to long standing BOO,[12] an age related phenomenon[13] or a urodynamic pattern of detrusor hyperreflexia with impaired detrusor contractility.[14] However, at baseline only 7 patients with hypocontractility in this series showed concomitant detrusor overactivity, which was associated with BOO in all. These results are consistent with those of Ameda et al who reported a 31% rate of impaired detrusor contractility in symptomatic male patients with nonobstructive voiding dysfunction.[15]

In our series we detected a high rate (55%) of detrusor overactivity preoperatively with a minor increase at 1 and 8 months of followup. This finding is in contrast to previously reported 14% and 41% rates of detrusor overactivity before and after RRP, respectively, suggesting that the condition is related to bladder denervation during surgery.[10] Leach et al reported a 56% rate of bladder dysfunction in men with post-prostatectomy incontinence but they did not perform a comparison to bladder function before surgery.[16]

It has been assumed that patients with BOO can have detrusor overactivity because of the enhancement or recruitment of new spinal circuits following obstruction.[17] The condition has been shown to reverse with obstruction relief in 62% of typical prostate cases,[12] although it may be less common in the elderly population.[18] In our series the persistence of detrusor overactivity can be partially explained by bladder denervation during surgery or by sustained neuroplasticity. More likely the strong association of detrusor overactivity

with ISD suggests that detrusor overactivity can be sustained by a deficient urethral sphincter with urine leakage into the urethra stimulating urethral afferents that induce involuntary voiding reflexes.[19]

Finally, in our series we detected a significant decrease in bladder compliance that was possibly related to bladder denervation. De novo impaired or poor compliance occurred in a limited number of patients (18.4% and 10.2% at 1 and 8 months of followup, respectively), while impaired bladder compliance was comparable to that before surgery in 20%. This finding may be explained by the observation that decreased compliance correlates with the decreased bladder perfusion induced by long-term elevated intravesical pressure.[20] In our series the persistence of decreased compliance was observed only in patients with BOO.

CONCLUSIONS

Bladder and urethral sphincter function can be adequately assessed by comparing urodynamic status before RRP and during longitudinal followup. De novo detrusor hypocontractility and impaired or poor compliance occur in a limited proportion of patients as a consequence of bladder denervation and they resolve in the majority within 8 months. Detrusor hypocontractility and decreased bladder compliance also represent preexisting conditions in about 30% and 20% of patients, and they do not appear to be influenced by RRP. Persistent detrusor overactivity after obstruction relief is probably related to concomitant sphincter deficiency and stress urinary incontinence, which increase afferent nerve activity of the proximal urethra and induce involuntary detrusor contractions.

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