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THE PATHOPHYSIOLOGY OF POST-RADICAL PROSTATECTOMY INCONTINENCE: A CLINICAL AND VIDEO URODYNAMIC STUDY

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ABSTRACT

Purpose: We examine various mechanisms of post-radical prostatectomy incontinence.

Materials and Methods: A total of 83 consecutive men (mean age 68 ± 6.6 years) referred for evaluation of persistent post-radical prostatectomy incontinence were enrolled in the study. All patients underwent clinical and urodynamic evaluation. Final diagnosis was based on clinical judgment considering patient history, pad test, voiding diary, free (unintubated) uroflow measurements, video urodynamics and linear passive urethral resistance relation curves. We compared free uroflow and pressure flow obtained with a 7Fr urethral catheter in place, and empirically defined low urethral compliance as at least 10 ml. per second difference between these measurements.

Results: Sphincteric incontinence was the most common urodynamic finding, occurring in 73 patients (88%). Detrusor instability was identified in 28 patients (33.7%) and in 6 (7.2%) was the main cause of incontinence. In 2 other patients bladder outlet obstruction (1.2%) or impaired detrusor contractility (1.2%) was the only urodynamic finding. Impaired detrusor contractility was diagnosed by linear passive urethral resistance relation in 82% of cases but considered to be clinically relevant in only a third. In 25 cases (30.1%) low urethral compliance was noted, which we consider nearly synonymous with urethral scarring.

Conclusions: Sphincteric incontinence is the most common urodynamic finding in patients with post-radical prostatectomy incontinence, although other findings may coexist. The most accurate diagnosis is attained when all objective measures are put in perspective with the clinical setting.

KEY WORDS: urinary incontinence, prostate, prostatectomy, urodynamics

Persistent urinary incontinence after radical prostatectomy is common, occurring in 5% to 20% of patients.[1–4] Conceptually, post-prostatectomy incontinence may be caused by sphincter malfunction and/or bladder dysfunction.[5] Bladder dysfunction includes involuntary detrusor contractions, impaired or absent detrusor contractility and low bladder compliance. It may be the result of bladder wall injury following long standing outflow obstruction or arise de novo after surgery.[6] Sphincteric incontinence is the most common cause of post-prostatectomy incontinence, although it often coexists with detrusor instability.[7]

There has been intense research concerning the relative contribution of sphincteric incontinence, detrusor instability and bladder compliance in the development of post-radical prostatectomy incontinence.[8–16] However, data on the role of impaired detrusor contractility, bladder outlet obstruction and urethral scarring are scarce. Furthermore, in patients with a scarred, constricted urethra the use of a transurethral catheter during detrusor pressure uroflow measurements may potentially cause test induced bladder outlet obstruction. These patients may be considered as having a low compliant urethra.

We examined all possible mechanisms of persistent post-radical prostatectomy incontinence, and evaluated the correlation of various objective measurements and clinical interpretation of these findings. We compared maximum uroflow rates with and without a 7Fr transurethral catheter in place, and defined low urethral compliance as a clinically significant difference between these 2 measurements.

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MATERIALS AND METHODS

A urodynamic database of 83 consecutive men referred for evaluation of persistent urinary incontinence after radical prostatectomy was reviewed. Average interval between surgery and urodynamic evaluation was 2.7 years (range 1 to 10). All patients underwent clinical evaluation, consisting of a complete history, physical examination, validated voiding questionnaire, voiding diary, pad test, noninvasive (free) uroflowmetry, post-void residual urine volume and video urodynamics.

Before examination all patients voided in private and free uroflow was recorded. Residual urine volume was measured by ultrasound immediately after bladder emptying. Multichannel video urodynamics were performed according to recommendations of the International Continence Society[17] except for cystometry. Contrary to these recommendations, patients were not instructed to try to inhibit voiding during the filling phase but rather to report sensations to the examiner. Cystometrography was performed using radiographic contrast medium and a 7Fr double lumen catheter via constant infusion at a medium filling rate, with rectal pressure monitoring. Vesical leak point pressure was evaluated at a volume of 150 ml. and defined as the lowest intravesical pressure induced by cough or Valsalva's maneuver necessary to effect any degree of visible stress incontinence. If no leakage occurred, filling continued and leak point pressure was

tested periodically. If there was no leakage with the urethral catheter in place, it was removed and abdominal leak point pressure was defined as the lowest abdominal pressure necessary to effect any degree of visible stress incontinence. At bladder capacity patients were asked to void, and pressure flow studies with simultaneous video fluoroscopy of the bladder outlet and surface electromyography were performed.

Linear passive urethral resistance relation (linPURR) curves were obtained to quantitate urethral obstruction and detrusor contractility.[18] The linPURR is a nomogram based on uroflow rates and detrusor pressures during voiding. A straight line is marked between the point of maximum flow rate and detrusor pressure at maximum flow rate, and the point of lowest pressure at which voiding occurs. Bladder outlet obstruction is categorized according to the curves as no obstruction (grade 0 or 1), equivocal or mild obstruction (grade 2) and increasing severity of obstruction (grades 3 to 6). Similarly, detrusor contractility is classified according to the curves as normal or impaired.

Urodynamic pressure flow tracings were visually inspected and clinically reclassified by 1 examiner (J. G. B.) who was blinded to linPURR results. Obstruction and contractility were defined as bladder outlet obstruction—maximum flow rate less than 12 ml. per second associated with sustained detrusor contraction (detrusor pressure at maximum flow rate) greater than 40 cm. water, impaired detrusor contractility—less than 12 ml. per second associated with less than 30 cm. water, respectively, and indeterminate—less than 12 ml. per second associated with 30 to 40 cm. water, respectively.

Further analysis was performed comparing free uroflow and invasive pressure flow measurements to determine whether the 7Fr urethral catheter had any significant effect. We then empirically defined low urethral compliance as at least 10 ml. per second difference between maximum uroflow measurements with (maximum flow rate) and without (free flow) a 7Fr urethral catheter in place. Final diagnosis was based on clinical judgment considering patient history, pad test, voiding diary, free uroflow measurements and urodynamics. For example, a case classified as obstructed by linPURR but with normal free uroflow was clinically reclassified as unobstructed. Results were analyzed statistically by the Student t and chi-square tests,[19] with $p < 0.05$ considered significant. Data are presented as mean plus or minus standard deviation or percentage according to variables.

RESULTS

Patient characteristics and urodynamic diagnoses (table 1). Mean patient age was 68 ± 6.6 years (range 54 to 88). All patients were evaluated at least 1 year postoperatively (range 1 to 10). Intrinsic sphincter deficiency was the most common urodynamic finding, occurring in 73 patients (88%). In 27 of these patients (32.5%) it was the only cause of incontinence and 46 had concomitant urodynamic findings.

Detrusor instability was identified in 28 patients (33.7%) and was the sole urodynamic finding in only 3 (3.6%). Impaired detrusor contractility was diagnosed in 24 patients (28.9%) and was the only urodynamic finding in only 1. Similarly, bladder outlet obstruction was diagnosed in 17 patients (20.5%), including only 1 (1.2%) with no other evidence of sphincteric or bladder malfunction. From a clinical perspective based on patient history, urodynamic study, diary and pad test, post-radical prostatectomy incontinence was mainly due to intrinsic sphincteric deficiency in 73 cases (88%) and detrusor instability in 6 (7.2%).

Correlation of linPURR and overall clinical assessment. Complete data for 50 patients were available for comparison of linPURR curves and overall clinical assessment (table 2). In all other cases voided volumes or flow rates were too low to allow reliable comparison. Impaired detrusor contractility was found by linPURR in 41 cases (82%) but considered to have clinical implications in only 16 (32%). Similarly, bladder outlet obstruction was diagnosed by linPURR in 8 cases (16%) but considered to have clinical implications in only 3 (6%).

Urethral compliance. In 25 patients (30.1%) the difference between maximum noninvasive (free) and invasive (pressure flow) uroflow was greater than 10 ml. per second. These patients were considered to have low urethral compliance, and were subdivided into urodynamically obstructed and unobstructed groups (table 3). Of the 25 cases 6 (24%) with low urethral compliance were obstructed by urodynamic criteria. Maximum free flow and invasive maximum flow rates were significantly lower in obstructed versus unobstructed cases. Similar analysis was performed in cases with normal urethral compliance, of which 11 (19%) were obstructed by urodynamic criteria. A clear trend of lower maximum flow rate and higher detrusor pressure at maximum flow rate values was evident in obstructed cases with low versus normal urethral compliance. However, due to the relative small number of obstructed cases statistical significance was not established.

DISCUSSION

Post-prostatectomy incontinence continues to be a major morbidity following radical prostatectomy. Published prevalence rates vary from 2% to 87% depending on the definitions, diagnostic modalities and interval from surgery.[1–7] In the majority of patients post-prostatectomy urinary incontinence resolves within several months.[3,16] However, in 5% to 10% of patients symptoms persist beyond the first postoperative year. Earlier studies regarding the pathophysiology of post-prostatectomy incontinence stressed the importance of detrusor instability and low bladder compliance.[8,10] However, recent studies indicate that sphincteric incontinence is the most common cause of post-radical prostatectomy incontinence, occurring as the sole cause in about two-thirds of patients, while isolated bladder dysfunction is uncommon, occurring in only 3%.[12–14] Our results confirm these data. Intrinsic sphincter deficiency was the most common urodynamic finding, occurring in 73 of our patients (88%), and was considered to be the dominant cause of post-radical prosta-

TABLE 1. *Urodynamic diagnoses*

	No. Pts. (%)				
	Intrinsic Sphincter Deficiency	Detrusor Instability	Bladder Outlet Obstruction	Impaired Detrusor Contractility	Normal
Main diagnosis	73 (88)	6 (7.2)	1 (1.2)	1 (1.2)	2 (2.4)
Sole diagnosis	27 (32.5)	3 (3.6)	1 (1.2)	1 (1.2)	2 (2.4)
Secondary diagnoses:					
Detrusor instability	10 (12)				
Bladder outlet obstruction	14 (16.9)	2 (2.4)			
Detrusor instability + bladder outlet obstruction	6 (7.2)				
Impaired detrusor contractility	22 (26.5)	1 (1.2)			
Detrusor instability + impaired detrusor contractility	6 (7.2)				

TABLE 2. *Correlation of linPURR and overall clinical assessment*

	No. linPURR (%)	No. Overall Clinical Assessment (%)
Bladder outlet:		
Obstruction	8 (16)	3 (6)
Equivocal	5 (10)	1 (2)
Normal	37 (64)	46 (92)
Detrusor contractility:		
Impaired	41 (82)	16 (32)
Normal	9 (18)	34 (68)

TABLE 3. *Comparison of low versus normal compliant urethra according to the urodynamic criteria of bladder outlet obstruction*

	Low Compliance		Normal Compliance	
	Obstruction	No Obstruction	Obstruction	No Obstruction
No. pts.	6	19	11	47
Mean free flow rate ± SD (ml./sec.)	18.8 ± 3.67	26.6 ± 6.9	11.5 ± 6.5	13.4 ± 8.1
Mean max. flow rate ± SD (ml./sec.)*	3.2 ± 1.3	9.4 ± 4.9	5.4 ± 4.5	11.1 ± 7.1
Mean detrusor pressure at max. flow rate ± SD (cm. H_2O)	53.0 ± 4.2	28.9 ± 13.6	58.3 ± 14.6	27.3 ± 14.2

* During pressure flow study with 7Fr urethral pressure catheter in place.

tectomy incontinence in all of them. In 27 of these patients (32.5% of the study population) it was the sole and only urodynamic finding. Detrusor instability was identified in 28 patients (33.7% of the study population), and was the main cause of incontinence in only 6 (7.2%).

We evaluated other urodynamic findings of impaired detrusor contractility, bladder outlet obstruction and urethral compliance in these patients. Data on the relative prevalence of these abnormalities as well as their clinical relevance are scarce or almost absent. Chao and Mayo studied 74 men with persistent post-radical prostatectomy incontinence, of whom 26% had anastomotic strictures, but no data were provided concerning pressure flow measurements.[9] Furthermore, 48% of patients with sphincteric incontinence alone voided by straining without demonstrable detrusor contraction and 38% of patients with detrusor instability also voided by straining. The authors suggested that in patients with detrusor instability impaired contractility may be a manifestation of the "detrusor hyperreflexia-impaired contractility" phenomenon described previously in the elderly, while in those with sphincteric incontinence it may be the result of neurological injury during surgery. Alternatively, they suggested that it may be learned behavior, as patients with decreased sphincteric resistance may find it easier and faster to void by straining. Impaired detrusor contractility was diagnosed in 24 of our patients (28.9% of the study population), most of whom (26.5%) had concomitant sphincteric incontinence. Impaired detrusor contractility defined by linPURR was clinically relevant in only a third of our patients. We believe this disparity is most likely due to the fact that in most patients incontinence is so bad that the urethra offers little resistance to flow, and even impaired detrusor contractility is sufficient to achieve normal flow.

Several studies have partially addressed the issue of anastomotic strictures in patients with post-radical prostatectomy incontinence. Chao and Mayo found anastomotic strictures in 26% of their incontinent patients and suggested that excess scarring at the anastomotic site may extend down into the sphincter and impair the closure mechanism.[9] Desautel et al diagnosed urethral scarring and anastomotic strictures in 67% of their patients, all of whom had sphincteric incontinence.[12] None of these studies provided any data on pressure flow parameters. Recently, Ficazzola and Nitti reported the results of 60 incontinent patients evaluated with multichannel video urodynamics.[14] Fluoroscopic evidence of anastomotic strictures was seen in 16 men (27%), of whom 14 had intrinsic sphincteric deficiency. We used pressure flow studies to define bladder outlet obstruction as well as linPURR curves to grade its severity. Although according to the linPURR curves 16% of cases were obstructed, by overall clinical judgment obstruction was clinically relevant in only 6%.

This discordance of urodynamic versus clinical obstruction is likely due to the obstructive effect of the 7Fr pressure catheter inside the urethra. Normally, there is little disparity in uroflow obtained with and without a 7Fr catheter in the urethra.[20] If flow is reduced with the urethral catheter in place, possible explanations include dysfunctional voiding (that is the patient contracts the sphincter because of discomfort), lower voided volume (also due to discomfort) or bladder outlet obstruction caused by the urethral catheter. If a 7Fr urethral catheter causes obstruction, the implication is that urethral compliance is decreased. A low compliant urethra may be the result of anastomotic stricture, fibrosis or bladder neck contracture. We defined low urethral compliance arbitrarily as at least a 10 ml. per second difference between maximum uroflow measurements with (maximum flow rate) and without (free flow) a 7Fr urethral catheter in place. To our knowledge no previously published study has addressed this issue. Some noted an increase in urethral obstruction severity when transurethral pressure flow studies were performed with 10Fr catheters.[21,22] Others found that an 8F urethral catheter does not exert a significant obstructive effect.[20] None of these studies evaluated the effect of the urethral catheter in a low compliant urethra. Furthermore, Schafer described 2 types of urethral obstruction as constrictive and compressive.[23] With constrictive obstruction the cross-section area of the urethra is reduced. We believe that, although an 8Fr or less urethral catheter has no clinically significant effect in compressive obstruction (benign prostatic hyperplasia), it may cause significant additional obstruction in an already constricted urethra (urethral stricture). For this reason after prostatectomy we consider diminished urethral compliance as nearly synonymous with an anastomotic stricture or scar. Furthermore, if lower cutoffs were used, the incidence of low urethral compliance would be much higher. These findings suggest that anastomotic scarring is a common cause of sphincteric incontinence after radical prostatectomy. The implications are that if surgical techniques to reduce scarring can be developed, the incidence of post-prostatectomy incontinence might be reduced.

CONCLUSIONS

The pathophysiology of post-radical prostatectomy incontinence seems to be more complicated than previously suggested. Although sphincteric incontinence is the most common urodynamic finding, detrusor instability, impaired detrusor contractility, bladder outlet obstruction or low urethral compliance may coexist. Disparity between urodynamic findings and overall clinical evaluation emphasizes the need for careful clinical evaluation of data. The most accurate diagnosis is attained when all objective measures are put in perspective with the clinical setting.

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EDITORIAL COMMENT

The authors provide intriguing insight into the possible pathogenesis of intrinsic sphincter deficiency following radical retropubic prostatectomy. The study confirmed that the majority of patients with post-prostatectomy incontinence suffer from intrinsic sphincter deficiency rather than detrusor instability as once thought. This study suggests that bladder and urethral impairment may be more extensive following surgery than previously recognized. An amazing 82% of patients had impaired detrusor contractility defined by urodynamics but the authors inferred that this reduced contractility was clinically relevant in only 29% of men. However, impaired contractility may exist because of reduced leak point pressure, negating the perception of straining to void, and incontinence may allow patients to appear relatively asymptomatic with regard to hypocontractility. It is unclear in this report what criteria were used to make the clinical judgment of "clinically significant" impaired contractility or obstruction. However, normal free uroflows were assumed to indicate the absence of obstruction. Certainly a normal uroflow, if elevated detrusor pressure exists, fails to preclude obstruction.

The major finding is that of impaired urethral function. Low urethral compliance was defined as a 10 ml. per second difference in free uroflow with and without a 7Fr urethral catheter. This catheter does not create obstruction in the normally functioning urethra. The authors used a 10 ml. per second value based on their extensive clinical experience rather than a biomechanical basis. Only 50 patients had voided volumes or flow rates of sufficient magnitude to allow comparison. Thus, it is unclear whether a difference in 10 ml. per second was only based on these patients or the entire population, some of whom had inadequate volume for flow measurements. While 10 ml. per second may be scientifically meaningful, further evidence is needed. Where is the statistical evidence that this change in flow discriminates between normal and impaired compliance? Did video urodynamics show impaired filling of the urethra or different degrees of distensibility on imaging, which could act as a surrogate estimate for reduced compliance? Similar to the mysterious leak point pressure of 60 cm. water, this value is arbitrary and, in reality, a range of abnormal values probably exists. Unfortunately, a limitation of this study is that a large number of patients who were continent after prostatectomy were not evaluated. To conclude that reduced compliance contributes to intrinsic sphincter deficiency and urinary incontinence it must first be shown what percent of all men after prostatectomy have a similar value.

Physiological measurements in animals and humans are necessary to examine the mechanism whereby surgery could create reduced compliance. Recent work in animals suggests that outlet obstruction can cause bladder ischemia. Surgery or preoperative obstruction could result in ischemia producing impaired contractility of the bladder and urethra. Additional assessment of the urethral function would also be valuable in this context. For example, is urethral contractility impaired as well as relaxation? Following administration of an agent, such as an α-agonist or antagonist, measurement of urethral pressures would give insight whether contraction/relaxation of the urethra is impaired.

Before urologists at large can conclude that fibrosis of the outlet is the principal cause of incontinence following retropubic prostatectomy, which I believe is correct, additional data with rigorous testing of the parameters must be provided. Those of us who evaluate a large number of patients with post-prostatectomy incontinence will probably agree with the conclusions of this report. One merely has to perform collagen injection in many of these men to realize the fibrotic nature of the bladder neck and proximal urethra. The findings of this study could have important implications for surgical technique, such as preservation of vascular supply to these tissues, limiting duration of an indwelling catheter or preoperative recognition of obstruction.

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