

See discussions, stats, and author profiles for this publication at: <https://www.researchgate.net/publication/318555632>

# Impact of bladder dysfunction in the management of post radical prostatectomy stress urinary incontinence—a review

Article in *Translational Andrology and Urology* · July 2017

DOI: 10.21037/tau.2017.04.14

CITATIONS

8

READS

87

3 authors:



**Derek B Hennessey**

Mercy University Hospital

158 PUBLICATIONS 1,161 CITATIONS

[SEE PROFILE](#)



**Nathan Hoag**

University of British Columbia - Vancouver

50 PUBLICATIONS 610 CITATIONS

[SEE PROFILE](#)



**Johan Gani**

Austin Health and Western Health

57 PUBLICATIONS 427 CITATIONS

[SEE PROFILE](#)

Some of the authors of this publication are also working on these related projects:



Female pelvic anatomy [View project](#)

# Impact of bladder dysfunction in the management of post radical prostatectomy stress urinary incontinence – a review

Derek B. Hennessey<sup>1</sup>, Nathan Hoag<sup>2</sup>, Johan Gani<sup>1,3</sup>

<sup>1</sup>Department of Urology, Austin Hospital, Heidelberg, Victoria, Australia; <sup>2</sup>Department of Urology, Victoria General Hospital, Victoria, British Columbia, Canada; <sup>3</sup>Department of Urology, Western Health, Footscray, Victoria, Australia

**Contributions:** (I) Conception and design: J Gani, DB Hennessey; (II) Administrative support: DB Hennessey; (III) Provision of study materials or patients: None; (IV) Collection and assembly of data: None; (V) Data analysis and interpretation: None; (VI) Manuscript writing: All authors; (VII) Final approval of manuscript: All authors.

**Correspondence to:** Derek B. Hennessey. Department of Urology, Austin Health, Melbourne, Australia. Email: derek.hennessey@gmail.com.

**Abstract:** Bladder dysfunction is a relatively common urodynamic finding post radical prostatectomy (RP). It can be the sole cause of post prostatectomy incontinence (PPI) or may be found in association with stress urinary incontinence (SUI). The aim of this review is to provide a comprehensive review of the diagnosis and different treatments of post RP bladder dysfunction. A comprehensive literature review using medical search engines was performed. The search included a combination of the following terms, PPI, detrusor overactivity (DO), detrusor underactivity (DU), impaired compliance, anticholinergic, onabotulinumtoxinA (Botox<sup>®</sup>) and sacral neuromodulation (SNM). Definitions, general overview and management options were extracted from the relevant medical literature. DO, DU and impaired compliance are common and may occur alone or in combination with SUI. In some patients the conditions exist pre RP, in others they arise due to denervation and surgical changes. DO can be treated with anticholinergics, Botox<sup>®</sup> and SNM. DO may need to be treated before SUI surgery. DU may be a contraindication to male sling surgery as some patients may go into urinary retention. Severely impaired bladder compliance may be a contraindication to SUI surgery as the upper tracts may be at risk. Each individual dysfunction may affect the outcome of PPI treatments and clinicians should be alert to managing bladder dysfunction in PPI patients.

**Keywords:** Post prostatectomy incontinence (PPI); detrusor overactivity (DO); detrusor underactivity (DU); impaired compliance; anticholinergic; onabotulinumtoxinA; sacral neuromodulation (SNM)

Submitted Jan 03, 2017. Accepted for publication Mar 26, 2017.

doi: 10.21037/tau.2017.04.14

**View this article at:** <http://dx.doi.org/10.21037/tau.2017.04.14>

## Introduction

Radical prostatectomy (RP) is the treatment of choice for patients with localized prostate cancer. Despite advances in pelvic anatomy and surgical technique, the overall incidence of post RP incontinence continues to rise due to the increasing numbers of RP performed (1,2). Currently the prevalence of post-prostatectomy incontinence (PPI) varies from 1% to 87%, depending on the definition, timing of evaluation, surgical approach and who carries out the assessment (3-5). PPI is multi-factorial and is due to intrinsic sphincter deficiency (ISD) and to pre-existing

bladder dysfunction or dysfunction arising *de novo* post RP (6-8). *Table 1* shows studies reporting the cause of PPI. ISD is considered to be the most important and most common contributing factor to PPI; however detrusor overactivity (DO), detrusor underactivity (DU) and poor bladder compliance commonly occur with ISD or in isolation, and are important factors in PPI (12).

The mainstay of treatment of PPI due to ISD is the insertion of an artificial urinary sphincter (AUS), and male slings. There has been some experience with transurethral injection of bulking agent but generally this is offered only to patients with mild ISD. There are no control trials

**Table 1** Etiology of post prostatectomy incontinence (PPI)

Study	Total N	SUI (%)	DO (%)	DU (%)	PC (%)
Hellström <i>et al.</i> , [1989] (9)	19	–	–	19	–
Presti Jr <i>et al.</i> , [1990] (10)	24	25	–	–	–
Foote <i>et al.</i> , [1991] (11)	71	–	–	–	7
	26	33	–	–	–
Groutz A <i>et al.</i> , [2000] (12)	83	32	4	1.2	82
Chao <i>et al.</i> , [1995] (7)	74	–	4	–	42
Goluboff <i>et al.</i> , [1995] (13)	25	8	40	–	8
Leach <i>et al.</i> , [1996] (14)	25	32	4	–	12
Desautel <i>et al.</i> , [1997] (15)	39	–	39	–	39
Hammerer <i>et al.</i> , [1997] (16)	82	–	41	–	–
Winters <i>et al.</i> , [1998] (17)	65	71	3	–	–
Gomha <i>et al.</i> , [2003] (18)	61	100	16.3	–	25.6
Giannantoni <i>et al.</i> , 2004 (19)	49	–	61.2 *(28.6)	38.7 *(18.4)	–
Huckabay <i>et al.</i> , [2005] (20)	60	58	40	–	–
Kielb <i>et al.</i> , [2005] (21)	146	65	2	48	14
Ventimiglia <i>et al.</i> , [2011] (22)	51	–	63	–	–
Matsukawa <i>et al.</i> , [2010] (23)	110	–	33	9	–
Dubbelman <i>et al.</i> , [2012] (24)	66	–	26 *(21.0)	–	–
Majoros <i>et al.</i> , [2006] (25)	63	28.6	3.2	–	–

\*, *de novo*. Total N, number of patients. SUI, stress urinary incontinence; DO, detrusor overactivity; DU, detrusor underactivity; PC, poor compliance.

comparing patients with PPI undergoing urodynamic studies (UDS) *vs.* no UDS prior to AUS insertion. Some papers suggest that the presence of bladder dysfunction does not alter post AUS continence outcomes (26-29). It has also been demonstrated that bladder dysfunction may improve after AUS implantation (30). Nevertheless, AUS placement in those with reduced compliance may lead to upper tract damage (31). Performing UDS has its advantages as it allows the clinician to assess, treat and counsel those with concurrent bladder dysfunction. If severe bladder dysfunction is identified, treatment of presumptive SUI and its potential complications may be avoided. This can lead to improved quality of life and prevention of complications especially when concurrent treatments of bladder dysfunction may potentially compromise each other.

This review seeks to report the etiology, evaluation, and management of non-urethral post-prostatectomy

incontinence. The impact of bladder dysfunction on stress urinary incontinence (SUI) management is also explored. An effort has been made to provide an algorithm to clinicians for appropriate surgical management. The surgical techniques of commonly performed procedures and their outcomes are described.

## Methods

A comprehensive literature inquiry using the following medical search engines were performed; PubMed, Ovid, Science Direct and Google Scholar. The search included a combination of the following terms: post-prostatectomy incontinence, DO, DU, impaired compliance, anticholinergic, onabotulinumtoxinA and sacral neuromodulation (SNM). Search results were assessed for their overall relevance to this review. Definitions, general overview and management options were extracted from the relevant medical literature.

## Pathophysiology of incontinence post RP

The majority of PPI results from ISD which is due to injury to the rhabdosphincter during the apical dissection and denervation of to the neurovascular bundles during RP (6). Bladder dysfunction, such as DO, DU and impaired bladder compliance can be present before RP, or may arise due to the surgery. Mobilization of the bladder can result in partial autonomic and somatic decentralization as well as inflammation, infection, bladder wall alterations and hypoxia (12,31,32).

Pre-operative DO can be due to bladder outlet obstruction (BOO) from an enlarged prostate. *De novo* DO may be secondary to BOO from bladder neck contracture or urethral strictures. It is also postulated that DO results from urethral afferent activity when SUI is present. This is believed to be the basis for the reversibility of bladder dysfunction when SUI is successfully treated. Denervation injury to the bladder is the main cause of DU. As for impaired bladder compliance, pelvic surgery such as RP with or without adjuvant radiation therapy can result in bladder fibrosis and contracture, affecting compliance negatively (32).

## Post RP detrusor overactivity

### Incidence of post prostatectomy DO

DO has been reported to occur at extremely varying rates between 2% and 63% post RP. Kielb *et al.*, found that in patients with PPI, only 2% had DO (21). Similarly, Majoros *et al.*, found DO in 3.2% of 63 patients with PPI and Winters *et al.*, found DO to be the sole cause of PPI in 3.3% (17,25). Huckabay *et al.*, and Groutz *et al.*, reported that PPI was due to DO in 13% and 7.2% of patients respectively (12,20). Differing from these findings, Ventimiglia *et al.*, found DO in 63% with PPI 8–24 months post nerve-sparing RP and considered incontinence to be purely due to DO in 35% of patients (33). Likewise, Leach *et al.*, established that DO contributed to incontinence in 60% of patients (14). DO frequently occurs with other bladder dysfunction post RP. Chao *et al.*, found that only 4% of 74 patients with incontinence after prostatectomy had DO alone, while 39% had mixed bladder and sphincter dysfunction (7). Matsukawa *et al.*, found in patients who underwent UDS before and after laparoscopic RP (LRP), a DO rate of 33% in addition to a DU rate of 9% (23).

Curiously, RP can affect pre-existing DO in different ways. Constantinou *et al.*, showed that in patients with pre-

existing DO, RP did not alter maximum DO pressures (34). Several studies, however, showed that DO can improve post RP. In a study of 66 patients with PPI, Dubbelman *et al.*, found a pre-operative DO rate of 26% which improved to 21% post RP (24). Giannantoni *et al.*, found that 61% of patients had pre-existing DO. After 3 years of follow-up, the post RP DO rate was 56%, some of which were *de novo* (19). Similarly Matsukawa *et al.*, found that DO disappeared in 54% of patients with pre-existing DO post RP, while 21% of patients developed *de novo* DO (23). Comparable results were found by Slova *et al.*, who reported that storage symptoms were significantly improved after open RP (35). Thus, the natural progression of DO post RP can be variable. Some patients with pre-existing DO report an improvement while others stay the same, and some patients develop post RP *de novo* DO.

### Implications of DO in men with SUI

Men with DO appear to have worse continence outcomes after a retroluminal transobturator (AdVance®) sling surgery (36–38). Conversely, the presence of DO does not seem to worsen the continence outcome post AUS surgery (27,28). However, *de novo* or persistent DO related symptoms occur commonly post AUS surgery and a patient needs to be counselled about this (39).

In general, we believe that it is important to treat DO first. This will have the effect of reducing the overall PPI and may make the component of SUI more apparent. Sometimes, the PPI may improve to the point where the patient may not need their SUI treated (14), or be treatable with a male sling rather than an AUS.

### Management of post prostatectomy DO

The management of DO related PPI is determined by its severity and by the presence of ISD, DU and poor compliance (PC). Assessment should comprise of urinalysis, urine culture, 24-hour pad weight, total number of pads, post void residual volume (PVR) and UDS. The subjective impact of PPI may be assessed with a validated questionnaire such as the international consultation on incontinence (ICIQ)—overactive bladder questionnaire.

The three main treatment approaches are similar to non-prostate cancer patients, they are as follows:

- ❖ Behavioral therapy (bladder training, biofeedback, pelvic floor muscle therapy, and restricting fluid intake);

- ❖ Pharmacologic therapy (anticholinergic and  $\beta_3$  agonists);
- ❖ Surgical therapy [Intravesical onabotulinumtoxinA (Botox<sup>®</sup>), SNM, urinary diversion].

There is a relative deficiency of data reporting the use of anticholinergic medications in post RP patients. However Leach *et al.*, demonstrated that anticholinergics significantly decreased pad score in patient with DO prior to AUS insertion (14). Mirabegron, the selective  $\beta_3$  agonist, has similar efficacy to anticholinergics but with less side effects, that may benefit patients with PPI DO. But there is no data reported of its use in the post RP population.

Surgical treatments include intravesical Botox<sup>®</sup>, SNM, and as a last resort, urinary diversion. Intravesical Botox<sup>®</sup> has an efficacy rate of 30–86% (40–42). However, Botox has a limited duration of benefit and repeat treatments are needed. There is also a significant risk of urinary retention (about 5%) and the patient may be required to perform clean intermittent self-catheterization (CISC) (43). Intravesical Botox may be an unattractive treatment if AUS is planned, as repeated cystoscopy or CISC may increase the risk of cuff erosion.

SNM is an alternative to intravesical Botox<sup>®</sup> on theoretical grounds. At this stage there are relatively few data about SNM in post RP population. For DO generally, SNM has a success rate of 53% to 80% (44–46). SNM does not cause retention and may treat the other forms of bladder dysfunction that can be found in association with DO, in particular DU, with success rates of 66.7% to 87.4% (47). Thus, SNM is potentially preferable to intravesical Botox<sup>®</sup> in treating post RP DO.

For severe refractory DO post RP, continued pad use or major open surgery may be the only options remaining. Augmentation cystoplasty is associated with high rates of CISC (75%) (48), and is not recommended, as this increases the risk of device urethral erosion. Creation of a urinary diversion remains another viable option, particularly in patients who might be deemed unsuitable for reconstructive bladder surgery (49). These treatments must be seen as a last resort.

## Post prostatectomy DU

### *Incidence and diagnosis of post prostatectomy DU*

The International Continence Society (ICS) defines DU as “a contraction of reduced strength and/or duration, resulting in prolonged bladder emptying and/or failure to achieve

*complete bladder emptying within a normal time span*” (50). Some patients have pre-existing DU and others develop it *de novo* (7), mainly as a result of denervation injury during the RP. Interestingly Chung *et al.*, postulated that minimally invasive surgery has a higher risk of causing DU as it involves a posterior approach to the dissection of the seminal vesicles where the pelvic nerves are situated. During an open RP, dissection is preformed closer to the seminal vesicles due to traction on the prostate, sparing nerves at the base of the bladder (51). In the community, the prevalence of DU is about 9% to 23% in men less than 50 years, increasing to about 48% in men older than 70 (52). Post RP DU appears to be common; Chung *et al.* reported that 41% of patient post RP had DU (51). Similarly, Porena *et al.*, found DU in 29–61% of patients post RP, of which 47% are *de novo* (32).

Studies reporting the incidence of DU post RP have limitations. Firstly, there is no consensus on which urodynamic method should be used to diagnose DU. Described methods include the Bladder Contractility Index (BCI), the presence of abdominal straining during voiding and arbitrary urodynamic cutoffs such as PdetQmax <20 cmH<sub>2</sub>O or a Qmax <15 mL/sec. PdetQmax in men with ISD may be falsely low due to reduced urethral resistance and formulas based on this may be inaccurate to diagnose DU (53). Isometric detrusor contraction pressure (Piso) may be a more accurate method to diagnose DU. It is measured by gently occluding the penile urethra during voiding, with a Piso of less than 50 cmH<sub>2</sub>O being diagnostic of DU (53). Secondly, not all studies compared pre and post op urodynamics findings, and therefore the true effect of RP on detrusor contractility is not fully appreciated.

### *Implications of DU in men with SUI*

Men with DU often void with abdominal straining due to insufficient detrusor strength. This can also be a learned behavior, as patients with decreased sphincteric resistance may find it easier and faster to void by straining (12). Therefore some concern exists in placing a male sling to treat SUI in men with DU, as the sling is designed to create a fixed resistance and may cause urinary retention. One study tried to alleviate some of these concerns. Han *et al.*, examined 50 patients with DU vs. 42 patients with normal contractility who had sling procedures. They found no significant differences in post sling PVR and Patient Global Impression of Improvement (PGI-I). In addition, there were no differences in those who were valsalva voiders (Pabd

>20 cmH<sub>2</sub>O during voiding) *vs.* normal voiders (54). The authors concluded that placing a male sling in these patients with DU was safe, but we certainly need more similar studies to confirm this.

Regarding the choice of male sling in DU patients, the retroluminal transobturator sling (AdVance sling<sup>®</sup>) offers an advantage over the more compressive quadratic sling (Virtue sling<sup>®</sup>) (55). The AdVance<sup>®</sup> sling acts by relocating the bulbar urethra and causes minimal compression, whereas the compressive quadratic Virtue sling<sup>®</sup> is typically tensioned to a pressure of 60 cmH<sub>2</sub>O. DU patients may not be able to generate this pressure and may not be able to void (56).

Placing an AUS in patients with DU or who are Valsalva voiders post RP appears to be safe and effective. The cuff is cycled open with relief of obstruction during voiding. Studies have demonstrated no increased risk of raised PVR or urinary retention post AUS in these patients (18,57). As DU can present with both voiding and storage symptoms, patients still have to be counselled post SUI surgery about the possibility of persistence of common DU symptoms including urgency, weak stream, straining to void, and nocturia (58).

### Management of post prostatectomy DU

If a patient is able to empty well during voiding after SUI surgery, he can be advised to continue to do double voiding or use abdominal straining. If there is decreased bladder sensation, the patient may have to do timed voiding. In patients who have pre RP DU or in those with an acontractile bladder who are not able to empty the bladder even with abdominal straining, it is anticipated they may have to continue or start doing CISC even after SUI surgery. It is not advisable to perform CISC through a urethral AUS cuff due to the high risk of erosion. A bladder neck AUS cuff is also not recommended as it is considered technically difficult and risky in a post RP patient where the anastomotic area may be scarred.

In this setting, an adjustable transobturator male sling (ATOMS<sup>®</sup>) may be considered although there has been no published data about this. Unlike the AUS which circumferentially compresses the urethra, the ATOMS<sup>®</sup> only compresses the bulbar urethra dorsally, leaving the ventral and lateral blood supply intact. The bulbospongiosus muscle is also left intact and acts as an additional protective layer between the device and the urethra. Hoda *et al.*, reported no case of urethral erosion in their series of 99 patients with

ATOMS<sup>®</sup> with a mean follow-up of 17.8 months, although no patient needed to do CISC (59). In an abstract, Law *et al.* reported on one patient who needed to start CISC three times a day, in their series of eight patients who were implanted with the ATOMS<sup>®</sup>, and there was no device erosion in that patient (60).

SNM is an option for patients with DU. A meta-analysis by Gross *et al.*, found that patients with DU had statistically significant increase in voided volume and a decrease in mean PVR after SNM treatment (61). In the non RP population the place of SNM in DU is well established. SNM is an effective treatment option for DU with excellent success rates. However, there are currently no published data on SNM in patients with post RP DU. Further research may demonstrate the place of SNM in patients with mixed PPI. If the DU can be successfully treated with SNM first, it may obviate the need for CISC, and simplify the subsequent treatment of the SUI.

### Post prostatectomy impaired bladder compliance

#### *Incidence of post prostatectomy impaired bladder compliance*

Bladder compliance is calculated by dividing the volume change ( $\Delta V$ ) by the change in detrusor pressure ( $\Delta P_{det}$ ) during that change in volume (mL/cmH<sub>2</sub>O). The ICS recommends two standard points be used for determination of compliance, firstly the detrusor pressure at the start of bladder filling and secondly, the detrusor pressure at cystometric capacity or before the start of any detrusor contraction (51). Poor bladder compliance, is defined as significant increases in P<sub>det</sub> with small increments in bladder volume and may lead to incontinence and damage of the upper urinary tract (62,63). Various definitions and bladder pressure criterion have been advocated for poor compliance. Chou *et al.*, recommend <10 mL/cmH<sub>2</sub>O (64). Weld *et al.*, reported higher incidences of upper tract damage and vesicoureteral reflux in the spinal cord injury population with bladder compliance of <12.5 mL/cmH<sub>2</sub>O (65), while others suggest <20 mL/cmH<sub>2</sub>O as poor compliance (66).

Several studies have examined for impaired compliance in patients with PPI. Ficazzola *et al.*, found that impaired bladder compliance was present in 5% (6). Conversely, Giannantoni *et al.*, reported that 28.1% of patients demonstrated evidence of impaired bladder compliance which was defined as change in detrusor pressure of 20 mL/cmH<sub>2</sub>O at 3 years post RP (66). Gomha *et al.*, noted approximately 24.6% of patients

demonstrated impaired compliance, of which 9.8% had PC (defined as  $>10$  mL/cmH<sub>2</sub>O) (18). While there seems to be variation in the reported incidence of impaired compliance after RP, it certainly is something that should be borne in mind by the clinician. It must be noted that impaired compliance in severely incontinent patients with ISD may be artefactual, due to supra-physiologic filling of chronically under filled bladders during urodynamics, and may be over reported.

### ***Management of post prostatectomy impaired bladder compliance***

Impaired or poor bladder compliance can be managed with observation and conservative measures such as timed voiding, anticholinergics, beta-3-agonists, or intravesical Botox<sup>®</sup> (28). Anticholinergics medications are effective in increasing bladder capacity, decreasing bladder filling pressure, and improving bladder compliance (67-69). In addition to improving compliance, Watanabe *et al.*, demonstrated improved hydronephrosis and vesicoureteral reflux with anticholinergics (70). Similarly, mirabegron a  $\beta$ 3-adrenoceptor agonist, improves cystometric capacity and bladder compliance, and it lowers vesicoureteral reflux grade in patients with the poorly compliant bladder and is an option for those who are intolerant of anticholinergics (71). Intravesical Botox has been indirectly used to treat PC in several studies examining the effect of Botox<sup>®</sup> on DO. In addition to increasing bladder capacity, Botox<sup>®</sup> has been shown to improve bladder compliance (70,72-74).

In the setting of PPI, when SUI surgery is considered, bladder compliance becomes an important consideration. Any procedure that obstructs the bladder outlet such as male sling or AUS could increase bladder pressure that may be transmitted to the upper tracts, potentially placing the kidneys at risk. Appropriate management of impaired compliance with the goal of reducing bladder pressures should be advocated prior to undertaking SUI surgery. Treatments should be evaluated with repeat urodynamics to assess for treatment success, and to ensure that SUI surgery can proceed without undue risk of upper urinary tract deterioration over the long-term.

### ***Implications of impaired compliance in men with SUI***

There is limited data in the literature about the safety of a male sling in terms of upper tract and renal function preservation in patients with poor bladder compliance.

Logically, a tight compressive sling (quadratic Virtue sling<sup>®</sup>) may be contraindicated and a non-occlusive sling may be safer. Habashy *et al.* reported on 20 patients with PC who had the AdVance<sup>®</sup> sling, and PC is not predictive of worse continence outcome. However, they did not report on the post-sling incidence of renal failure or hydronephrosis (36).

It is still unknown if poor bladder compliance is an absolute contraindication to AUS surgery in the non-neurogenic, non-irradiated patient population. There appears to be a tendency towards worse continence results in those with impaired compliance on pre-operative urodynamic studies (28). Other studies, however, have failed to corroborate these findings (12,27). Preservation of continence status post AUS surgery may also be an ominous sign of potential upper tract damage (28). While no safe cutoff detrusor pressures for the implantation of AUS has been established, patients with mildly impaired bladder compliance may still undergo insertion of an AUS. In these patients, long-term follow-up of the upper tracts with periodic serum creatinine measurement, and renal ultrasound should be employed to screen for upper urinary tract deterioration (75). These patients may also be advised to do timed voiding in order to avoid reaching the threshold bladder volumes that result in high bladder pressures. There remains a subset of patients with persistently elevated detrusor pressures, or cannot be relied upon to do timed voiding, or have evidence of pre-existing renal impairment/hydronephrosis, who ultimately may not be suitable for SUI surgery. These patients should be counseled accordingly, as the PPI may be serving as a “pop-off” mechanism, protecting their upper tracts.

### **Conclusions**

SUI remains the most common cause of PPI, but bladder dysfunction in the form of DU, DO and PC are important causes of PPI that must not be ignored. All can occur pre RP or can arise *de-novo* and can exist alone or in combination with SUI. Bladder dysfunction can affect the outcome of SUI surgery, thus each patient must be treated on an individual basis. Patients with SUI and DO are recommended to have their DO treated first. Patients with DU and SUI must be counselled that they may not be able to void after sling surgery. Patient with PC may need their compliance treated to prevent upper tract damage prior to SUI surgery.

### **Acknowledgements**

None.

## Footnote

*Conflicts of Interest:* The authors have no conflicts of interest to declare.

## References

- Mottet N, Bellmunt J, Bolla M, et al. EAU-ESTRO-SIOG Guidelines on Prostate Cancer. Part 1: Screening, Diagnosis, and Local Treatment with Curative Intent. *Eur Urol* 2017;71:618-29.
- Walsh PC. Anatomic radical prostatectomy: evolution of the surgical technique. *J Urol* 1998;160:2418-24.
- Kielb S, Dunn RL, Rashid MG, et al. Assessment of early continence recovery after radical prostatectomy: patient reported symptoms and impairment. *J Urol* 2001;166:958-61.
- Jønler M, Madsen FA, Rhodes PR, et al. A prospective study of quantification of urinary incontinence and quality of life in patients undergoing radical retropubic prostatectomy. *Urology* 1996;48:433-40.
- Kim JC, Cho KJ. Current trends in the management of post-prostatectomy incontinence. *Korean J Urol* 2012;53:511-8.
- Ficazzola MA, Nitti VW. The etiology of post-radical prostatectomy incontinence and correlation of symptoms with urodynamic findings. *J Urol* 1998;160:1317-20.
- Chao R, Mayo ME. Incontinence after radical prostatectomy: detrusor or sphincter causes. *J Urol* 1995;154:16-8.
- Kleinhans B, Gerharz E, Melekos M, et al. Changes of urodynamic findings after radical retropubic prostatectomy. *Eur Urol* 1999;35:217-21; discussion 221-2.
- Hellström P, Lukkarinen O, Kontturi M. Urodynamics in radical retropubic prostatectomy. *Scand J Urol Nephrol* 1989;23:21-4.
- Presti JC Jr., Schmidt RA, Narayan PA, et al. Pathophysiology of urinary incontinence after radical prostatectomy. *J Urol* 1990;143:975-8.
- Foote J, Yun S, Leach GE. Postprostatectomy incontinence. Pathophysiology, evaluation, and management. *Urol Clin North Am* 1991;18:229-41.
- Groutz A, Blaiwas JG, Chaikin DC, et al. The pathophysiology of post-radical prostatectomy incontinence: a clinical and video urodynamic study. *J Urol* 2000;163:1767-70.
- Goluboff ET, Chang DT, Olsson CA, et al. Urodynamics and the etiology of post-prostatectomy urinary incontinence: the initial Columbia experience. *J Urol* 1995;153:1034-7.
- Leach GE, Trockman B, Wong A, et al. Post-prostatectomy incontinence: urodynamic findings and treatment outcomes. *J Urol* 1996;155:1256-9.
- Desautel MG, Kapoor R, Badlani GH. Sphincteric incontinence: the primary cause of post-prostatectomy incontinence in patients with prostate cancer. *Neurourol Urodyn* 1997;16:153-60.
- Hammerer P, Huland H. Urodynamic evaluation of changes in urinary control after radical retropubic prostatectomy. *J Urol* 1997;157:233-6.
- Winters JC, Appell RA, Rackley RR. Urodynamic findings in postprostatectomy incontinence. *Neurourol Urodyn* 1998;17:493-8.
- Gomha MA, Boone TB. Voiding patterns in patients with post-prostatectomy incontinence: urodynamic and demographic analysis. *J Urol* 2003;169:1766-9.
- Giannantoni A, Mearini E, Di Stasi SM, et al. Assessment of bladder and urethral sphincter function before and after radical retropubic prostatectomy. *J Urol* 2004;171:1563-6.
- Huckabay C, Twiss C, Berger A, et al. A urodynamics protocol to optimally assess men with post-prostatectomy incontinence. *Neurourol Urodyn* 2005;24:622-6.
- Kielb SJ, Clemens JQ. Comprehensive urodynamics evaluation of 146 men with incontinence after radical prostatectomy. *Urology* 2005;66:392-6.
- Ventimiglia B, Tsirgiotis A, Fanzone I, et al. [Urinary incontinence after radical prostatectomy. Neurophysiological and urodynamic diagnosis]. *Urologia* 2011;78:82-5.
- Matsukawa Y, Hattori R, Komatsu T, et al. De novo detrusor underactivity after laparoscopic radical prostatectomy. *Int J Urol* 2010;17:643-8.
- Dubbelman Y, Groen J, Wildhagen M, et al. Quantification of changes in detrusor function and pressure-flow parameters after radical prostatectomy: relation to postoperative continence status and the impact of intensity of pelvic floor muscle exercises. *Neurourol Urodyn* 2012;31:637-41.
- Majoros A, Bach D, Keszthelyi A, et al. Urinary incontinence and voiding dysfunction after radical retropubic prostatectomy (prospective urodynamic study). *Neurourol Urodyn* 2006;25:2-7.
- Pérez LM, Webster GD. Successful outcome of artificial urinary sphincters in men with post-prostatectomy urinary incontinence despite adverse implantation features. *J Urol* 1992;148:1166-70.
- Thiel DD, Young PR, Broderick GA, et al. Do clinical or urodynamic parameters predict artificial urinary sphincter



- outcome in post-radical prostatectomy incontinence? *Urology* 2007;69:315-9.
28. Trigo Rocha F, Gomes CM, Mitre AI, et al. A prospective study evaluating the efficacy of the artificial sphincter AMS 800 for the treatment of postradical prostatectomy urinary incontinence and the correlation between preoperative urodynamic and surgical outcomes. *Urology* 2008;71:85-9.
  29. Afraa TA, Campeau L, Mahfouz W, et al. Urodynamic parameters evolution after artificial urinary sphincter implantation for post-radical prostatectomy incontinence with concomitant bladder dysfunction. *Can J Urol* 2011;18:5695-8.
  30. Lai HH, Hsu EI, Boone TB. Urodynamic testing in evaluation of postradical prostatectomy incontinence before artificial urinary sphincter implantation. *Urology* 2009;73:1264-9.
  31. Steiner MS. Continence-preserving anatomic radical retropubic prostatectomy. *Urology* 2000;55:427-35.
  32. Porena M, Mearini E, Mearini L, et al. Voiding dysfunction after radical retropubic prostatectomy: more than external urethral sphincter deficiency. *Eur Urol* 2007;52:38-45.
  33. Ventimiglia B, Sigona M, Di Dio A, et al. Urinary incontinence and neuropathy after radical prostatectomy: diagnosis and treatment. *Urologia* 2015;82:42-5.
  34. Constantinou CE, Freiha FS. Impact of radical prostatectomy on the characteristics of bladder and urethra. *J Urol* 1992;148:1215-9; discussion 1219-20.
  35. Slova D, Lepor H. The short-term and long-term effects of radical prostatectomy on lower urinary tract symptoms. *J Urol* 2007;178:2397-400; discussion 2400-1.
  36. Habashy D, Losco G, Tse V, et al. Mid-term outcomes of a male retro-urethral, transobturator synthetic sling for treatment of post-prostatectomy incontinence: Impact of radiotherapy and storage dysfunction. *Neurourol Urodyn* 2017;36:1147-50.
  37. Zuckerman JM, Edwards B, Henderson K, et al. Extended outcomes in the treatment of male stress urinary incontinence with a transobturator sling. *Urology* 2014;83:939-45.
  38. Crites MA, Sorial A, Ghoniem GM. Risk factors for male slings: a comparative study of two techniques. *Urology* 2011;78:192-6.
  39. Lai HH, Boone TB. Implantation of artificial urinary sphincter in patients with post-prostatectomy incontinence, and preoperative overactive bladder and mixed symptoms. *J Urol* 2011;185:2254-9.
  40. Rovner E, Kennelly M, Schulte-Baukloh H, et al. Urodynamic results and clinical outcomes with intradetrusor injections of onabotulinumtoxinA in a randomized, placebo-controlled dose-finding study in idiopathic overactive bladder. *Neurourol Urodyn* 2011;30:556-62.
  41. Denys P, Le Normand L, Ghout I, et al. Efficacy and safety of low doses of onabotulinumtoxinA for the treatment of refractory idiopathic overactive bladder: a multicentre, double-blind, randomised, placebo-controlled dose-ranging study. *Eur Urol* 2012;61:520-9.
  42. Schmid DM, Sauermann P, Werner M, et al. Experience with 100 cases treated with botulinum-A toxin injections in the detrusor muscle for idiopathic overactive bladder syndrome refractory to anticholinergics. *J Urol* 2006;176:177-85.
  43. Karsenty G, Denys P, Amarenco G, et al. Botulinum toxin A (Botox) intradetrusor injections in adults with neurogenic detrusor overactivity/neurogenic overactive bladder: a systematic literature review. *Eur Urol* 2008;53:275-87.
  44. Davis T, Makovey I, Guralnick ML, et al. Sacral neuromodulation outcomes for the treatment of refractory idiopathic detrusor overactivity stratified by indication: Lack of anticholinergic efficacy versus intolerance. *Can Urol Assoc J* 2013;7:176-8.
  45. Scheepens WA, van Koeveinge GA, de Bie RA, et al. Urodynamic results of sacral neuromodulation correlate with subjective improvement in patients with an overactive bladder. *Eur Urol* 2003;43:282-7.
  46. Van Voskuilen AC, Oerlemans DJ, Weil EH, et al. Medium-term experience of sacral neuromodulation by tined lead implantation. *BJU Int* 2007;99:107-10.
  47. Saber-Khalaf M, Abtahi B, Gonzales G, et al. Sacral neuromodulation outcomes in male patients with chronic urinary retention. *Neuromodulation* 2015;18:329-34; discussion 334.
  48. Hasan ST, Marshall C, Robson WA, et al. Clinical outcome and quality of life following enterocystoplasty for idiopathic detrusor instability and neurogenic bladder dysfunction. *Br J Urol* 1995;76:551-7.
  49. Singh G, Wilkinson JM, Thomas DG. Supravescical diversion for incontinence: a long-term follow-up. *Br J Urol* 1997;79:348-53.
  50. Abrams P, Cardozo L, Fall M, et al. The standardisation of terminology of lower urinary tract function: report from the Standardisation Sub-committee of the International Continence Society. *Am J Obstet Gynecol* 2002;187:116-26.
  51. Chung DE, Dillon B, Kurta J, et al. Detrusor underactivity is prevalent after radical prostatectomy: A urodynamic study including risk factors. *Can Urol Assoc J* 2013;7:E33-7.
  52. Osman NI, Chapple CR, Abrams P, et al. Detrusor

- underactivity and the underactive bladder: a new clinical entity? A review of current terminology, definitions, epidemiology, aetiology, and diagnosis. *Eur Urol* 2014;65:389-98.
53. Jura YH, Comiter CV. Urodynamics for postprostatectomy incontinence: when are they helpful and how do we use them? *Urol Clin North Am* 2014;41:419-27, viii.
  54. Han JS, Brucker BM, Demirtas A, et al. Treatment of post-prostatectomy incontinence with male slings in patients with impaired detrusor contractility on urodynamics and/or who perform Valsalva voiding. *J Urol* 2011;186:1370-5.
  55. Rehder P, Mitterberger MJ, Pichler R, et al. The 1 year outcome of the transobturator retroluminal repositioning sling in the treatment of male stress urinary incontinence. *BJU Int* 2010;106:1668-72.
  56. Comiter CV, Dobberfuhr AD. The artificial urinary sphincter and male sling for postprostatectomy incontinence: Which patient should get which procedure? *Investig Clin Urol* 2016;57:3-13.
  57. Lai HH, Hsu EI, Teh BS, et al. 13 years of experience with artificial urinary sphincter implantation at Baylor College of Medicine. *J Urol* 2007;177:1021-5.
  58. Hoag N, Gani J. Underactive Bladder: Clinical Features, Urodynamic Parameters, and Treatment. *Int Neurourol J* 2015;19:185-9.
  59. Hoda MR, Primus G, Fischereeder K, et al. Early results of a European multicentre experience with a new self-anchoring adjustable transobturator system for treatment of stress urinary incontinence in men. *BJU Int* 2013;111:296-303.
  60. Law MC, Chan SY, Cheung HY, et al. Adjustable transobturator male system (ATOMS) for male postprostatectomy stress urinary incontinence: initial multicentre experience in Hong Kong. *ICS* 2012:413.
  61. Gross C, Habli M, Lindsell C, et al. Sacral neuromodulation for nonobstructive urinary retention: a meta-analysis. *Female Pelvic Med Reconstr Surg* 2010;16:249-53.
  62. Wyndaele JJ, Gammie A, Bruschini H, et al. Bladder compliance what does it represent: can we measure it, and is it clinically relevant? *Neurourol Urodyn* 2011;30:714-22.
  63. McGuire EJ, Woodside JR, Borden TA, et al. Prognostic value of urodynamic testing in myelodysplastic patients. *J Urol* 1981;126:205-9.
  64. Chou FH, Ho CH, Chir MB, et al. Normal ranges of variability for urodynamic studies of neurogenic bladders in spinal cord injury. *J Spinal Cord Med* 2006;29:26-31.
  65. Weld KJ, Graney MJ, Dmochowski RR. Differences in bladder compliance with time and associations of bladder management with compliance in spinal cord injured patients. *J Urol* 2000;163:1228-33.
  66. Giannantoni A, Mearini E, Zucchi A, et al. Bladder and urethral sphincter function after radical retropubic prostatectomy: a prospective long-term study. *Eur Urol* 2008;54:657-64.
  67. Stöhrer M, Murtz G, Kramer G, et al. Propiverine compared to oxybutynin in neurogenic detrusor overactivity--results of a randomized, double-blind, multicenter clinical study. *Eur Urol* 2007;51:235-42.
  68. Cameron AP, Clemens JQ, Latini JM, et al. Combination drug therapy improves compliance of the neurogenic bladder. *J Urol* 2009;182:1062-7.
  69. Christoph F, Moschkowitsch A, Kempkensteffen C, et al. Long-term efficacy of tolterodine and patient compliance in pediatric patients with neurogenic detrusor overactivity. *Urol Int* 2007;79:55-9.
  70. Watanabe M, Yamanishi T, Honda M, et al. Efficacy of extended-release tolterodine for the treatment of neurogenic detrusor overactivity and/or low-compliance bladder. *Int J Urol* 2010;17:931-6.
  71. Kamei J, Furuta A, Akiyama Y, et al. Video-urodynamic effects of mirabegron, a beta3 -adrenoceptor agonist, in patients with low-compliance bladder. *Int J Urol* 2015;22:956-61.
  72. Karsenty G, Reitz A, Lindemann G, et al. Persistence of therapeutic effect after repeated injections of botulinum toxin type A to treat incontinence due to neurogenic detrusor overactivity. *Urology* 2006;68:1193-7.
  73. Klaphajone J, Kitisomprayoongkul W, Sriplakit S. Botulinum toxin type A injections for treating neurogenic detrusor overactivity combined with low-compliance bladder in patients with spinal cord lesions. *Arch Phys Med Rehabil* 2005;86:2114-8.
  74. Schurch B, Stohrer M, Kramer G, et al. Botulinum-A toxin for treating detrusor hyperreflexia in spinal cord injured patients: a new alternative to anticholinergic drugs? Preliminary results. *J Urol* 2000;164:692-7.
  75. Hussain M, Greenwell TJ, Venn SN, et al. The current role of the artificial urinary sphincter for the treatment of urinary incontinence. *J Urol* 2005;174:418-24.

**Cite this article as:** Hennessey DB, Hoag N, Gani J. Impact of bladder dysfunction in the management of post radical prostatectomy stress urinary incontinence—a review. *Transl Androl Urol* 2017;6(Suppl 2):S103-S111. doi: 10.21037/tau.2017.04.14