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### Publication Date

2021-07-01

### DOI

10.1016/j.urology.2021.04.016

Peer reviewed

## Association Between Ejaculatory Dysfunction and Post-Void Dribbling After Urethroplasty



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### OBJECTIVE

To determine whether ejaculatory dysfunction (EjD) and post-void dribbling (PVD) after urethroplasty are associated, providing evidence for a common etiology.

### METHODS

We reviewed a prospectively maintained database for first-time, anterior urethroplasties. One item from the Male Sexual Health Questionnaire (MSHQ) assessed EjD: "How would you rate the strength or force of your ejaculation". One item from the Urethral Stricture Surgery Patient-Reported Outcome Measure (USS-PROM) assessed PVD: "How often have you had slight wetting of your pants after you had finished urinating?". The frequency of symptoms was compared after penile vs. bulbar repairs, and anastomotic versus augmentation bulbar repairs. Associations were assessed with chi-square.

### RESULTS

A total of 728 men were included. Overall, postoperative EjD and PVD were common; 67% and 66%, respectively. There was a significant association between EjD and PVD for the whole cohort ( $p < 0.0001$ ); this association remained significant after penile repairs ( $p = 0.01$ ), bulbar repairs ( $p < 0.0007$ ), and bulbar anastomotic repairs ( $p = 0.002$ ), but not after bulbar augmentation repairs ( $p = 0.052$ ). EjD and PVD occurred at similar rates after penile and bulbar urethroplasty. The rate of EjD was similar after bulbar augmentation and bulbar anastomotic urethroplasties, but PVD was more common after bulbar augmentation (70% vs. 52%) ( $p = 0.0001$ ).

### CONCLUSION

EjD and PVD after anterior urethroplasty are significantly associated with one another, supporting the theory of a common etiology. High rates after penile repairs argue against a bulbospongiosus muscle damage etiology, and high rates after anastomotic repairs argue against graft sacculation. More work is needed to better understand and prevent symptoms. UROLOGY 153: 320–326, 2021. © 2021 Elsevier Inc.

Trauma and Urologic Reconstruction Network of Surgeons

Financial Support for Study: None

Declaration of Interests: None

Funding: Alex J Vanni – Fellowship Educational Funds & PI on AUSCO study for Boston Scientific; NIH Funding-R21DK115945-91 and U01DK097772-07; Bradley A Erickson – Fellowship Educational Funds & PI on AUSCO study for Boston Scientific; PI for clinical trial for Urotronic; NIH Funding-R21DK115945-91, U01DK097772-07, U01DK082344-11; Benjamin N Breyer – Alafi Foundation Funding; NIH Funding R21DK115945-01; Jeremy B. Myers – Fellowship Educational Funds Boston Scientific; Consultant Cooper Surgical; Department of Defence SCI Grant-SCI150071; NIH Funding-R21DK115945-91; NIH Funding-R21DK124733-01; Neilsen Foundation Grant; Bryan Voelzke – None; Joshua A Broghammer – Consultant Boston Scientific; Clinical Events Committee for AUSCO study for Boston Scientific Keith F Rourke; Andrew C Peterson – Fellowship Educational Funds Boston Scientific; PI on AUSCO study Boston Scientific; Scientific Advisory Board Boston Scientific; Movember Foundation Grant; NIH Funding-U01DK097776-01; Sean Elliott – Consultant & investigator for clinical trial for Boston Scientific; PI for clinical trial for Urotronic; Shareholder in Percutis; NIH Funding-R21DK115945-01

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Submitted: February 11, 2021, accepted (with revisions): April 12, 2021

Since the 1990s urologists have been cautioned about the potential for sexual side effects following urethroplasty.<sup>1</sup> Data showed that despite objective success, several patients experienced bothersome erectile dysfunction and ejaculatory dysfunction (EjD) after urethroplasty.<sup>1–3</sup> Further, patients frequently complained of post-void dribbling (PVD) which was attributed to a similar etiology as EjD. Modern series also report a high prevalence of ejaculatory dysfunction<sup>4</sup> and post-void dribbling<sup>5</sup> after urethroplasty, and these symptoms are a major source of bother to patients.<sup>6</sup> Theories proposed for the development of EjD and PVD after urethroplasty include: bulbospongiosus muscle damage, urethral sacculation from graft or flap augmentation, neuropraxia of the perineal nerve, and loss of normal expansive and contractive characteristics of the spongiosum after augmentation. To date, the data dedicated to this phenomenon has been inconclusive and the etiology of EjD and PVD remains elusive.<sup>5,7–11</sup>

There is increased awareness in the urologic community of the importance of investigating and mitigating these

side effects after urethroplasty. Various surgical approaches have been developed to eliminate post-operative EjD and PVD<sup>12,13</sup>, however none to date have resulted in consistent or definitive improvement. While it is presumed that these symptoms are the result of the same etiology, no study to date has proven an association between EjD and PVD; either in frequency or severity. If truly associated, then we can be more confident they share a common etiology, and thus, could expect an improvement in both symptoms with a single targeted intervention.

Our primary objective is to determine whether complaints of EjD and PVD after urethroplasty are associated with one another; we hypothesize that they are. A secondary objective is to add to the literature about possible causes of EjD and PVD by comparing the frequency of symptoms across repair types. We hypothesize that both EjD and PVD are the result of bulbospongiosus muscle damage and thus are more likely after bulbar urethroplasty than penile urethroplasty. We also hypothesize that graft/flap sacculation contribute to these symptoms, and thus expect that EjD and PVD will be more common after bulbar augmentation vs. bulbar anastomotic repairs.

## MATERIALS AND METHODS

**Source of data:** The Trauma and Urologic Reconstructive Network of Surgeons (TURNS) is a multi-institutional research group that collects data on urologic diseases that are traumatic and reconstructive in nature. All members are fellowship-trained reconstructive surgeons. All data are collected prospectively by the surgeons and stored in a single, web-based, institutional review board-approved data repository. The database enrolls men  $\geq 18$  years of age undergoing reconstructive procedures.

**Study population:** We included men who underwent a first-time anterior urethroplasty. We excluded hypospadias, posterior urethroplasties, fistula repairs, diverticula repairs, perineal urethrostomy, extended meatotomy, first or second stage procedures, and those with incomplete data. All men had at least one follow-up visit 2 to 12 months after surgery with completed post-operative questionnaires for EjD and PVD. We documented demographic and clinical information including age, body mass index (BMI), history of diabetes, length and location of stricture, number of prior endoscopic interventions, and repair type (augmentation vs. anastomotic).

**Study outcomes:** We assessed post-operative EjD with a single item from the Male Sexual Health Questionnaire (MSHQ).<sup>14</sup> The question asked "In the last month, how would you rate the strength or force of your ejaculation?" with responses ranging from "could not ejaculate" (score = 0) to "as strong as it always was" (score = 6) on a 6-item Likert scale. Similarly, we utilized a single item from the USS-PROM questionnaire<sup>15</sup> to assess post-operative PVD. Specifically, "how often do you have slight wetting of your pants a few minutes after you had finished urinating and had dressed yourself?". Responses lie on a 5-item Likert scale from "never" (score = 0) to "all the time" (score = 5). Of note, the scales go in opposite directions; so, a more symptomatic man has a low EjD score but a high PVD score.

The first responses to the questionnaires in the 2 to 12-month postoperative window were used in order to capture more symptomatic events and thus increase the power of our analysis.

## Statistical analysis

**Primary objective - Association of EjD and PVD:** The frequency distribution of responses to both questions were compared across the entire cohort using chi-square. This initial test of association included men treated for any anterior stricture. Subset analyses were then performed to assess the strength of association between EjD and PVD after (1) penile, (2) bulbar, (3) bulbar anastomotic, and (4) bulbar augmentation urethroplasties. We did not include men with strictures spanning the penile and bulbar urethra in any further analyses because our goal was to assess the association between symptoms in distinct urethral locations.

Patients were included in the bulbar anastomotic cohort if they underwent excision and primary anastomosis (EPA), Heineke-Mikulicz repair, or non-transsecting EPA. The augmentation cohort included ventral and dorsal grafts, augmented-anastomotic repairs, and fasciocutaneous flaps. We could not compare symptoms after dorsal versus ventral augmentations due to few ventral augmentations.

Approximately 30% of men answered that their ejaculation was *as strong as ever* (a score of 5/5); so we dichotomized the EjD question as "EjD Absent" if they responded *as strong as ever*, and "EjD present" if they selected any other response. Similarly, approximately 30% of men answered that dribbling occurred *never* (a score of 0/4); so, we dichotomized the PVD question as "PVD Absent" for men who responded *never* and "PVD Present" if they selected any other response. The presence of EjD and PVD within each repair-type cohort was then compared with chi-square test.

**Secondary objective - Etiology of EjD and PVD.** After testing for an association between EjD and PVD *within* urethroplasty types, we proceeded with our second objective: to compare the frequency of EjD and PVD across different urethroplasty types to gain understanding of their potential etiology. We compared the frequency of symptoms between penile versus bulbar urethroplasties; a proxy for evaluating the role of bulbospongiosus muscle damage in symptom development. We then compared the frequency of EjD and PVD between anastomotic vs. augmentation urethroplasty in the bulbar urethra; a proxy for evaluating the role of graft/flap sacculation in symptom development (controlling for bulbospongiosus muscle transection).

The statistical analyses were conducted with the entire cohort of men and sub-analysis was performed after excluding all patients who had evidence of stricture recurrence on cystoscopy. All analyses were carried out using SAS software (version 9.4; SAS Institute Inc., Cary, NC) and a  $P < 0.05$  was considered significant.

## RESULTS

A total of 728 patients underwent anterior urethroplasty from 2006 – 2019 and met inclusion criteria. The cohort's mean ( $\pm$  standard deviation) age was 47 years ( $\pm 15.3$ ), BMI was 30 kg/m<sup>2</sup> ( $\pm 11$ ), and stricture length was 3.8cm ( $\pm 3$ ). Only 52 patients (7%) had pre-existing diabetes. The majority of patients underwent at least 1 prior endoscopic procedure prior to urethroplasty, while 39% underwent  $\geq 2$  procedures. Demographic information by location of stricture is shown in Table 1. Bulbar strictures occurred in 445 (61%) patients, penile strictures in

**Table 1.** Demographic data by stricture location

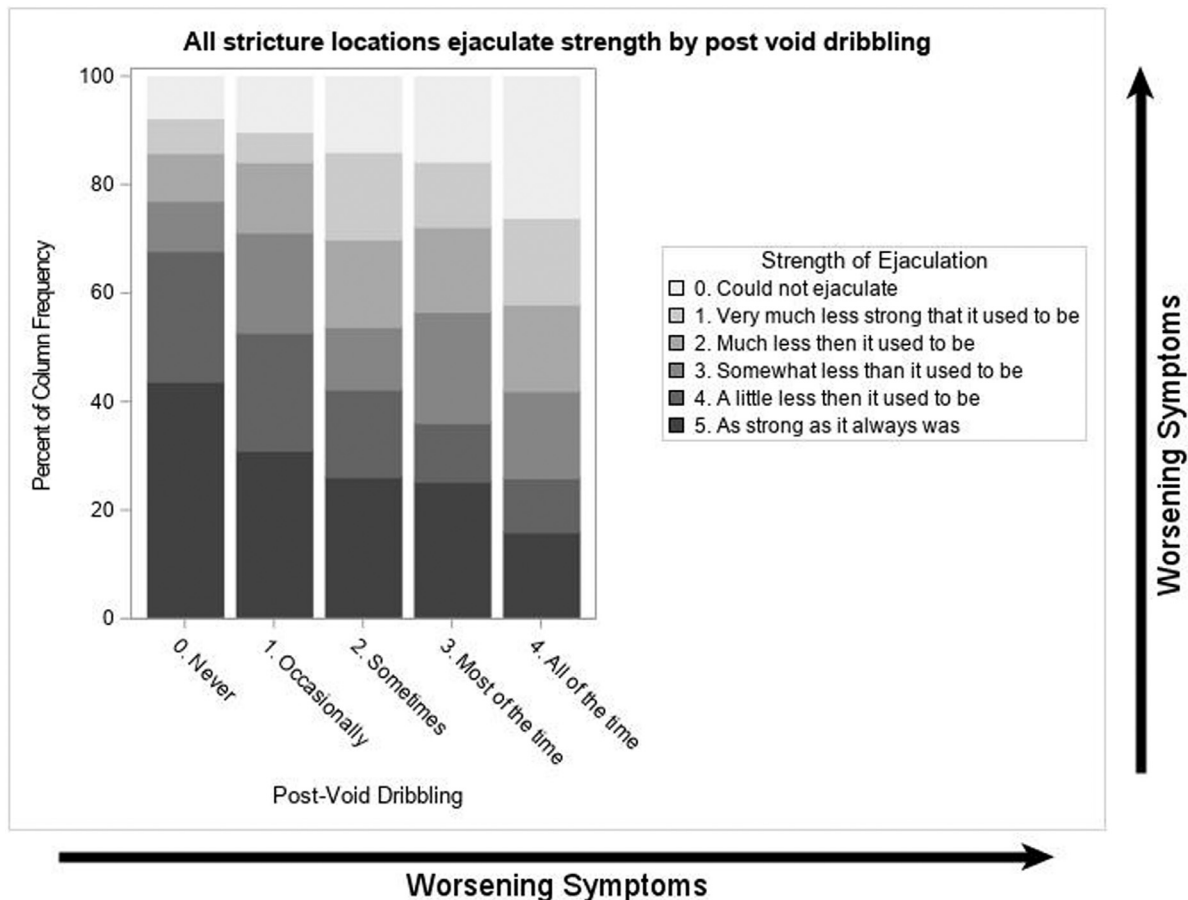
	Stricture Location				P value
	All	Bulbar Only	Bulbar and Penile	Penile Only	
Number	728	445	108	175	
Age mean (SD)	46.9 (15.3)	43.5 (14.8)	49.4 (14.4)	53.1 (14.9)	< 0.0001
BMI mean (SD)	30.6 (11.0)	30.3 (13.0)	32.4 (7.8)	30.0 (6.5)	0.2039
Stricture Length (SD)	3.8 (3.0)	2.7 (1.5)	7.3 (4.2)	4.5 (3.2)	< 0.0001
Comorbidities					<.0001
Diabetes N (%)	52 (7.1)	16 (3.6)	17 (15.7)	19 (10.9)	
Prior Endoscopic Treatments					0.0059
0 N (%)	256 (35.2)	154 (34.6)	27 (25.0)	75 (42.9)	
1 N (%)	186 (25.6)	117 (26.3)	24 (22.2)	45 (25.7)	
2 or more N (%)	286 (39.2)	174 (39.1)	57 (52.8)	55 (31.4)	
Repair Type					< 0.0001
Augmentation Urethroplasty N (%)	480 (65.9)	227 (51.0)	101 (93.5)	152 (86.9)	
Anastomotic Urethroplasty N (%)	248 (34.1)	218 (49.0)	7 (6.5)	23 (13.1)	

Percentages in a group may not add up to 100 due to rounding. SD – standard deviation, N – number, BMI – body mass index.

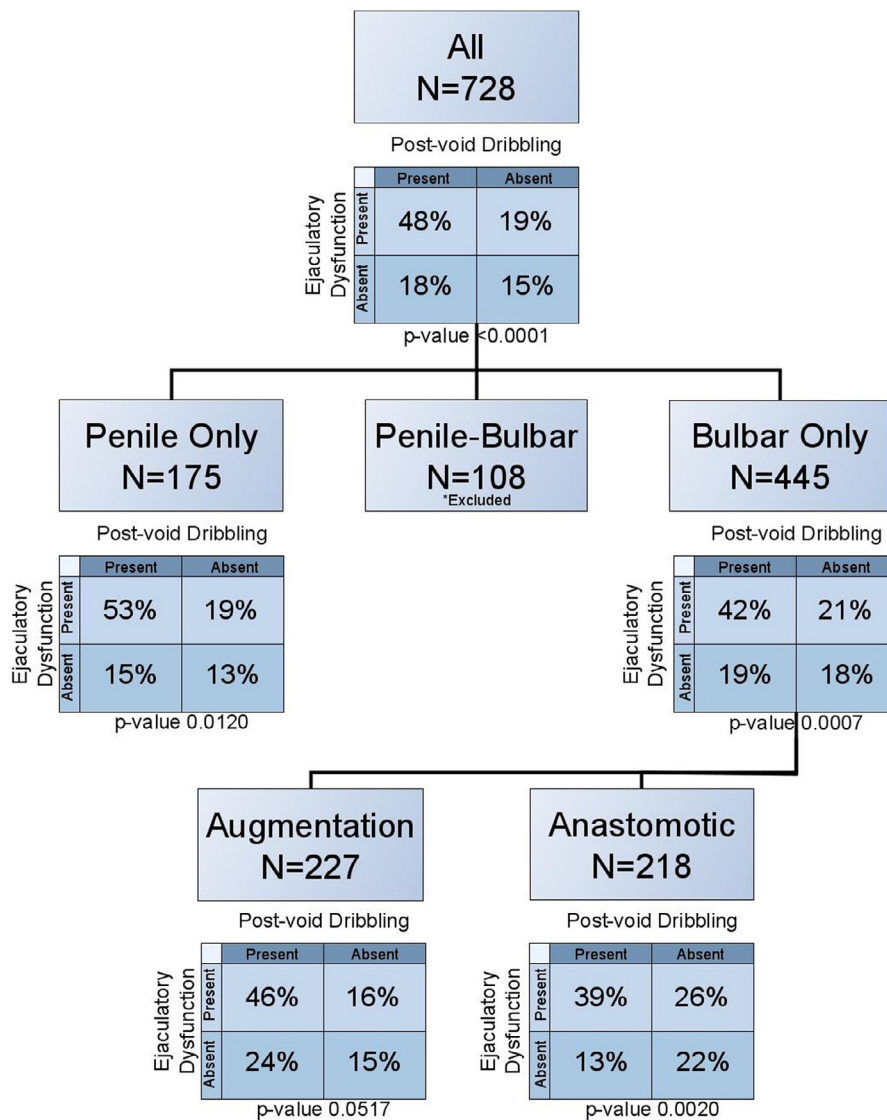
175 (24%), and penile + bulbar strictures in 108 (15%). Men with penile strictures were older, and less likely to have prior endoscopic interventions. Men with penile + bulbar strictures had longer strictures, and more frequently underwent  $\geq 2$  prior endoscopic interventions. Patients with bulbar strictures underwent an equal proportion of anastomotic and augmentation repairs, while the other groups underwent more augmentation repairs.

### Primary Objective - Association of EjD and PVD

Across all repair types, there was a significant association between the severity of EjD and PVD ( $P < 0.001$ ) (Fig. 1); patients that reported more severe EjD symptoms reported more severe PVD symptoms. This association persisted across subsets of stricture location (penile and bulbar) as well as across some subsets of stricture repair types (bulbar anastomotic and bulbar augmentation) (Fig. 2). Specifically, EjD and PVD were



**Fig. 1.** Stacked bar chart demonstrating the frequency of post-void dribbling and ejaculatory dysfunction. Worse symptoms on both scales are in the upper right-hand corner of the chart. The severity of symptoms are associated with each other ( $p < 0.001$ ).



**Fig 2.** Associations between EjD and PVD by stricture location and use of graft during urethroplasty. (Color version available online.)

significantly associated with each other after penile urethroplasty ( $P = 0.012$ ) and after bulbar urethroplasty ( $P = 0.001$ ). Further, within the bulbar urethroplasty subset, EjD and PVD were significantly associated after anastomotic urethroplasty ( $P = 0.002$ ) but the relationship was not significant after bulbar augmentation urethroplasty ( $P = 0.052$ ). Sub-analysis after removing men with evidence of stricture recurrence did not change the direction of the results.

### Secondary Objective - Etiology of EjD and PVD

*Testing a bulbospongiosus muscle injury hypothesis.* We compared the frequency of EjD across different repair types, then the frequency of PVD across different repair types (Figure 3). There were high rates (>50%) of EjD and PVD across all cohorts. The rates of EjD and PVD were not higher following bulbar repairs compared to penile repairs, causing us to reject our hypothesis. In fact, there was a higher rate of EjD in patients undergoing penile urethroplasty (71% vs. 63% for penile and bulbar urethroplasty, respectively) but this did not reach statistical significance ( $P = 0.051$ ). Similarly, there was a higher rate of PVD after penile urethroplasty (68% vs. 61% for penile and bulbar

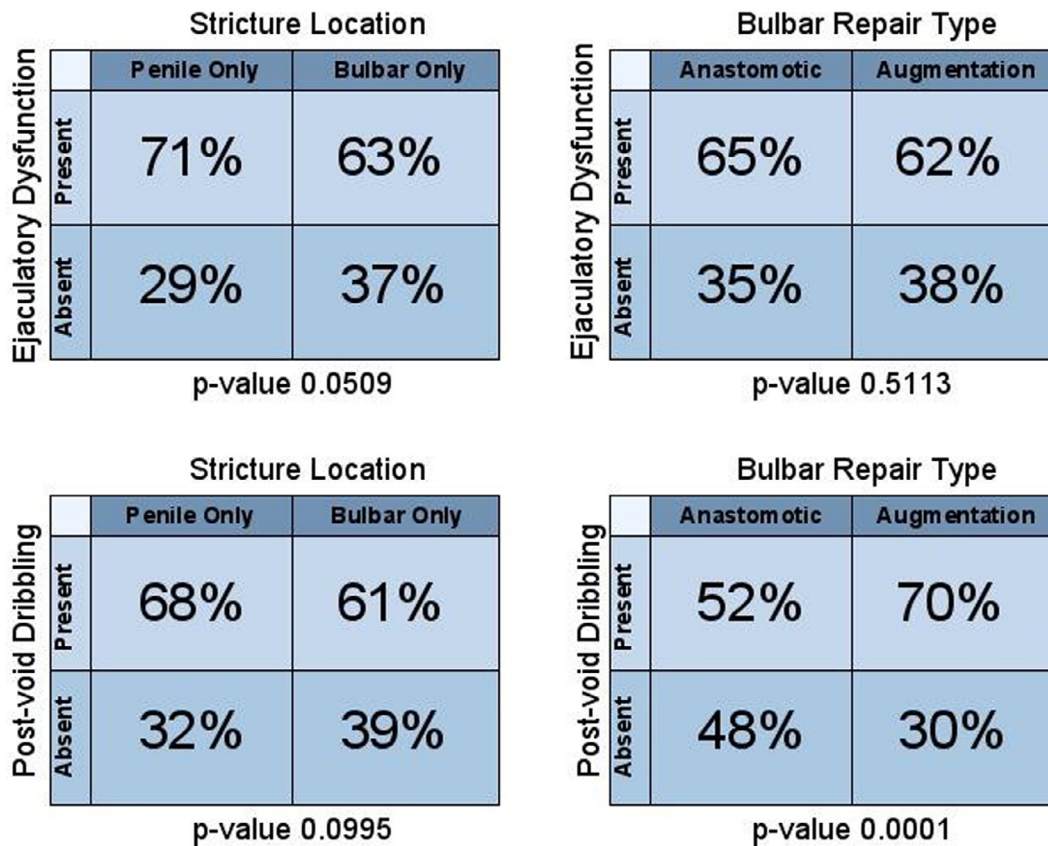
urethroplasty, respectively), but this also did not reach statistical significance ( $P = 0.09$ ).

*Testing a graft/flap sacculation hypothesis:* When focusing on the subset of men undergoing bulbar urethroplasty, there was no difference in EjD rates between augmentation vs. anastomotic urethroplasty (62% vs 65%, respectively;  $P = 0.51$ ) (Fig. 3). However, PVD was more common after augmentation (70%) vs. anastomotic urethroplasty (52%) ( $P < 0.0001$ ). Repeat sub-analysis after removing men with evidence of stricture recurrence on cystoscopy did not change the direction of our secondary outcome measures.

## DISCUSSION

Herein we have shown that symptoms of EjD and PVD after anterior urethroplasty are strongly associated in frequency and severity. This association exists across different locations (penile and bulbar urethroplasties) and different repair types in the same location (anastomotic and augmentation bulbar urethroplasties). These findings





**Fig. 3.** Frequency of EjD by stricture location and use of graft during urethroplasty. Frequency of PVD by stricture location and use of graft during urethroplasty. (Color version available online.)

argue for a common etiology behind symptom development. However, our findings do not support bulbospongiosus muscle damage or graft/flap sacculation as a unifying etiology. Thus, although EjD and PVD are strongly associated, arguing for a common etiology, the exact etiology remains unknown. Our study is best understood in the context of the previous literature dedicated to this topic and the previously proposed theories behind symptom development.

**Bulbospongiosus Muscle Injury Theory:** The role of the bulbospongiosus muscle in ejaculation was first documented in the 1960s<sup>16</sup>. Its potential role in micturition is proposed to be via a bulbocavernosal reflex.<sup>17</sup> Sparing the bulbospongiosus muscle during urethroplasty was first proposed by Barbagli.<sup>13</sup> This series of 12 patients undergoing muscle-sparing urethroplasty reported no EjD or PVD at 6 or 12 months after surgery using a non-validated telephone survey.<sup>13</sup> This was further evaluated in a retrospective, multi-institutional, matched case-control study comparing muscle-sparing to non-muscle-sparing bulbar urethroplasty.<sup>18</sup> This study assessed pre- and post-operative ejaculatory dysfunction and, contrary to the initial report, found no benefit to muscle-sparing for preventing EjD.

The muscle-sparing approach recently became the focus of a randomized controlled trial.<sup>19</sup> Elkady randomized men to muscle sparing or non-muscle sparing ventral graft bulbar urethroplasty. Ten patients (40%) in the non-muscle sparing and 2 (8%) in the muscle-sparing group reported

EjD. In addition, 9 (36%) in the non-muscle sparing and 1 (4%) in the muscle-sparing group reported PVD. While these findings support that bulbospongiosus muscle injury could be responsible for EjD and PVD after urethroplasty, other studies refute this finding. A number of series published show no effect of repair type on the development of EjD<sup>10,11,20</sup> or PVD.<sup>5,20</sup> Further, these symptoms have been reported in patients undergoing isolated penile urethroplasties.<sup>10,11,21</sup> Our study also shows high rates of symptoms after penile urethroplasty. If bulbospongiosus muscle injury is the main driver of EjD and PVD, we would expect these conditions to be more common in bulbar but not penile urethroplasties.

**Graft Sacculaton Theory:** One of the earliest theories for EjD and PVD after urethroplasty is that skin flaps and grafts may result in sacculaton of the urethra and create a pseudodiverticulum. This, in turn, results in pooling of semen and urine.<sup>2, 3</sup> As a result, it was recommended to perform EPA whenever possible and keep graft sizes small if necessary.<sup>3</sup> In response to this concern, Barbagli developed the dorsal free-graft urethroplasty.<sup>12</sup> They reported on 25 men with a follow-up of 36 months. They found no patient reporting EjD or PVD after dorsal grafting. Shortly thereafter, Dubey<sup>21</sup> published retrospective data also in favor of dorsal grafting. In their series of 109 patients, EjD and PVD were more common after ventral augmentation; EjD was reported in 20% vs. 5%, and PVD in 39% vs. 23% of ventral vs. dorsal grafts, respectively. However, the symptoms

still developed in a significant number of dorsal repairs arguing against graft sacculcation as the sole etiology.

One of the biggest counters to the graft sacculcation theory is the finding that these symptoms are common after anastomotic repairs. Barbagli showed that 24% of men undergoing EPA in the bulbar urethra reported EjD<sup>9</sup>. Our data also show that EjD and PVD are commonly reported after anastomotic repairs.

### **New Theory: Different Etiologies for Different Locations**

As mentioned above, we found that EjD and PVD occur just as often after penile urethroplasty as bulbar urethroplasty. They also occur at near similar rates after bulbar anastomotic vs. bulbar augmentation repairs. These findings argue against either of the previously proposed theories as the sole reason behind EjD and PVD development. However, our data does show a strong association between EjD and PVD across stricture location and repair type. Therefore, our findings challenge the notion that there is one central etiology for symptom development. On the contrary, it supports different etiologies driving symptom development depending on stricture location and repair type.

One further consideration that could explain some of the conflicting data in the literature is that the tools we use to assess symptoms are inaccurate. Questionnaires rely on patient ability to subjectively report what surgeons are objectively defining. Perhaps, any operation on the penis can drive men to report changes in their urinary or sexual function even if it is not specific to the outcome being assessed. The solution involves development of objective tests for ejaculatory function (e.g. measurement of semen volume, force of ejaculation, urethral pressure profiles during ejaculation) and post-void dribbling (e.g. pad weights, condom catheter collections, post-voiding cystourethrogram films for residual contrast in the urethra). Objective measures are critical if we hope to understand patient experiences and actual changes in ejaculatory and urinary function.

Limitations of this study include its retrospective design and the potential for bias of our study population since men could only be included if they completed post-operative questionnaires. In addition, the augmentation urethroplasty group included a variety of repair types: ventral and dorsal graft, augmented-anastomotic repairs, and skin flaps. Therefore, the detrimental effects of any one repair type was not able to be determined. Further, we did not analyze pre-operative EjD or PVD; we have previously shown these to be common in this group.<sup>5,18</sup> It can be assumed that pre-operative symptoms are due to the stricture and post-operative symptoms are due to the surgery; we were interested in the impact of surgery. Some men may have had persistent, rather than new EjD or PVD; however, with the stricture corrected the main determinant of their persistent symptoms is most likely surgery. Similarly, while these lower urinary tract symptoms could also be the result of benign prostatic enlargement (BPE),

we argue this to be unlikely since this is a young patient population. We noted high rates of EjD and PVD (> 50% across all cohorts). This could be because of our definition of EjD and PVD (i.e., any dysfunction on the questionnaire) and because we used the first available questionnaire after surgery. It is assumed that these symptoms improve over time after surgery, however, our goal was test for an association between the two symptoms to help identify a possible etiology; our goal was not to characterize the long-term persistence of symptoms.

### **CONCLUSION**

EjD and PVD after urethroplasty are associated with each other and the severity of symptoms presents in a co-linear fashion. The association persists across stricture location (penile vs. bulbar strictures) and repair type (augmentation vs. anastomotic), which challenges the presence of one central etiology for symptom development. Thus, while the association of EjD and PVD suggests a common etiology, it is unlikely to be due to bulbospongiosus muscle dissection or graft sacculcation. Further work is needed to better understand the pathophysiology of symptoms and develop strategies to prevent their occurrence.

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