Somatomotor and sensory urethral control of micturition in female rats

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1In rats, axons of external urethral sphincter (EUS) motoneurons travel through the anastomotic branch of the pudendal nerve (ABPD) and anastomatic branch of the lumbosacral trunk (ABLT) and converge in the motor branch of the sacral plexus (MBSP). The aim of the present study was to determine in female rats the contribution of these somatomotor pathways and urethral sensory innervation from the dorsal nerve of the clitoris on urinary continence and voiding. EUS electromyographic (EMG) activity during cystometry, leak point pressure (LPP), and voiding efficiency (VE) were recorded before and after transection of the pudendal nerve (PD nerve), which innervates the penis/clitoris (8, 32, 35). In females, this nerve is known as the dorsal nerve of the clitoris (DNC), and some of its axons provide afferent innervation of the distal urethra (37). Sensory input from the urethra is carried in the pudendal (PD), lateral (LPP), and some of its axons provide afferent innervation of the distal urethra. Sensory input from the urethra is carried in the pudendal (PD), lateral (LPP), and voiding efficiency (VE) were assessed in anesthetized virgin Sprague-Dawley female rats before and after transection of the above named nerves. Transsection of the MBSP eliminated EUS EMG, decreased LPP by 50%, and significantly reduced bladder contraction duration, peak pressure, intercontraction interval, and VE. Transsection of the ABPD or ABLT decreases EUS EMG discharge and LPP by 25% but did not affect VE. Transsection of the dorsal nerve of the clitoris did not affect LPP but reduced contraction duration, peak pressure, intercontraction interval, and VE. We conclude that somatomotor control of micturition is provided by the MBSP with axons travelling through the ABPD and ABLT. Partial somatomotor urethral denervation induces mild urinary incontinence, whereas partial afferent denervation induces voiding dysfunction. ABPD and ABLT pathways could represent a safeguard ensuring innervation to the EUS in case of upper nerve damage. Detailed knowledge of neuroanatomy and functional innervation of the urethra will enable more accurate animal models of neural development, disease, and dysfunction in the future.

MATERIALS AND METHODS

Sixty-seven adult virgin Sprague-Dawley female rats underwent investigation (body weight: 250–300 g, Harlan). Animals were anesthetized with an intraperitoneal injection of urethane (ethyl carbamate, 1.2 g/kg, Sigma-Aldrich, St. Louis, MO). The experimental protocol was approved by the Institutional Animal Care and Use Committee of the Cleveland Clinic.

Experimental design. In experiment 1 (n = 15), EUS EMG activity was recorded during induced micturition by cystometry (CMG) before and after bilateral transection of the MBSP, ABLT, and ABPD (5 rats/nerve) to test innervation of the EUS by different neural pathways. Nerves were localized before electrodes were inserted in the EUS, before recordings were made in the intact condition, to avoid changing the site of EMG recording after nerve transection.

In experiment 2 (n = 28, 7 rats/nerve), CMG and leak point pressure (LPP) were recorded before and after bilateral transection of each nerve to determine the contribution of motor (MBSP, ABLT, and ABPD) or sensory (DNC) innervation on urinary continence. In experiment 3 (n = 24, 6 rats/nerve), voiding volume (VV) and residual volume (RV) were recorded before and after bilateral transection of the MBSP, ABLT, ABPD, and DNC to determine the role of each nerve in voiding efficiency.

EUS EMG activity measurements. The pubis was removed, and the urethra was legalized. Using a stereomicroscope, the tip of a pair of platinum wire electrodes (0.05 mm in diameter and insulated except for 2 mm at the tip) were inserted in the EUS of the pelvic urethra, 6–7 mm below the bladder neck (asterisk in Fig. 1A) to record EMG signals. The electrodes were connected to an amplifier (model PS11 AC Amplifier, Astromed, Providence, RI; band-pass filtered at 10 Hz.
micturition were transected, and the threshold volume to induce micturition was considered as LPP. After measurement of three LPPs, nerves were quickly withdrawn (3). Peak bladder pressure at the moment of leakage occurred at the urethral meatus, at which point the external pressure over the bladder region, using a cotton swab, until leakage was taken as LPP. The bladder was emptied and refilled as follows. The bladder was refilled, and LPP was measured at half bladder capacity by slowly and manually increasing the abdominal pressure necessary to remove the muscle covering the ilium and a portion of this bone to see the plexus. Nerves were covered with wet gauze to avoid drying until these were bilaterally transected according to the experimental design described above. Sites of the bilateral neurectomies in the present study were bilateral. Arrows indicate sites of nerve transection. The bladder was exposed by a midline abdominal incision, and a silastic cannula (outer diameter: 1.5 mm) was secured in the bladder dome with a purse string suture as a suprapubic catheter to infuse saline and measure pressure during CMG. The suprapubic catheter was attached via a stopcock to a pressure transducer (model P300, Grass Instruments, West Warwick, RI) and a syringe pump (model 200, KD Scientific, New Hope, PA). Pressure data were recorded by the same physiological recording system used for EMG recordings. The abdominal incision was then closed in two layers. During continuous infusion (5 ml/h), repeated voiding episodes were observed, and, after 20–25 min, the following parameters were determined during three micturition cycles: intercontraction interval (ICI; the period between two successive voiding contractions; Fig. 2A), peak pressure (PP; maximal pressure during voiding; Fig. 2A), and contraction duration (CD; the period between when the intravesical pressure begins to rise for voiding and when this pressure returned to baseline; Fig. 2A). Mean values of each parameter from three micturition cycles in each rat were calculated and used to calculate group means before and after nerve transection.

RV measurements. After 30 min of continuous saline infusion, capacity was measured as follows: immediately after voiding with the pump turned off, residual saline in the bladder was withdrawn through the intravesical catheter connected to the three-way stopcock by manually pressing the abdominal wall to facilitate emptying. The volume collected was the RV and was added to the volume threshold to induce micturition to calculate capacity.

LPP measurements. LPP was then measured during bladder filling as follows. The bladder was refilled, and LPP was measured at half bladder capacity by slowly and manually increasing the abdominal pressure over the bladder region, using a cotton swab, until leakage occurred at the urethral meatus, at which point the external pressure was quickly withdrawn (3). Peak bladder pressure at the moment of leakage was taken as LPP. The bladder was emptied and refilled between LPPs. Only leakage in the absence of a bladder contraction was considered as LPP. After measurement of three LPPs, nerves were transected, and the threshold volume to induce micturition was measured again. LPP was tested three times after each nerve transection. The mean value of the three trials was used for group comparisons (intact vs. neurectomized).

**Voiding efficiency measurements.** Rats were placed in the supine position. The bladder was canulated, and pressure data were recorded as described above. Nerves were localized, and the pump was turned on. After 30 min of continuous saline infusion, three CMGs were recorded by emptying the bladder as described above. The volume expelled during a voiding contraction was measured as VV, and the infusion pump was stopped immediately after each voiding contraction. The bladder was drained as above by withdrawing through the intravesical catheter connected to the three-way stopcock to obtain the RV. This was done three times to obtain mean VV and RV for each rat. Each nerve (MBSP, ABLT, ABPD, or DNC, 6 rats/nerve) was bilaterally transected, and saline was continuously infused for 10 min, followed by recording three additional bladder fill/empty cycles and determination of VV and RV. Bladder capacity and voiding efficiency were calculated as VV + RV and [VV × 100%/VV + RV], respectively. The mean value of the three trials per condition was used for group comparisons.

**Neurectomies.** With urethane-anesthetized rats in the supine position, the DNC and MBSP were localized after removal of a portion of the ischium (Fig. 1A). In a lateromedial direction, both nerves run parallel, with the PD vein between. Near the ischiatic arch, the MBSP branches and small branches run rostrally to innervate the EUS, whereas others run caudally to innervate the external anal sphincter. The sacral plexus and its anastomotic branches (ABLT and ABPD) were localized medial to the head of the femur (Fig. 1). It was necessary to remove the muscle covering the ilium and a portion of this bone to see the plexus. Nerves were covered with wet gauze to avoid drying until these were bilaterally transected according to the experimental design described above. Sites of the bilateral neurectomies are shown in Fig. 1, A and B.

**Data analysis.** EMG data before and after nerve transection were analyzed by selecting a 1-s sample during filling (basal condition taken at the middle of the ICI, when the EUS shows a low level of activity, as shown in Fig. 2A), voiding (when high-frequency oscillations related to urine flow are present; Fig. 2B) as well as after voiding (immediately after high-frequency oscillations end; Fig. 2B) using AstroVIEW (Astro-Med).

Quantitative assessment of EMG signals was performed by determining the mean rectified amplitude and frequency of firing activity (42). Briefly, segmented raw data were smoothed using a triangular Bartlett window. The resultant signal was subtracted from the original signal to 3 kHz and an electrophysiological recording system (DASH 8X, Astromed, 10-kHz sampling rate).

**CMG measurements.** The bladder was exposed by a midline abdominal incision, and a silastic cannula (outer diameter: 1.5 mm) was secured in the bladder dome with a purse string suture as a suprapubic catheter to infuse saline and measure pressure during CMG. The suprapubic catheter was attached via a stopcock to a pressure transducer (model P300, Grass Instruments, West Warwick, RI) and a syringe pump (model 200, KD Scientific, New Hope, PA). Pressure data were recorded by the same physiological recording system used for EMG recordings. The abdominal incision was then closed in two layers. During continuous infusion (5 ml/h), repeated voiding episodes were observed, and, after 20–25 min, the following parameters were determined during three micturition cycles: intercontraction interval (ICI; the period between two successive voiding contractions; Fig. 2A), peak pressure (PP; maximal pressure during voiding; Fig. 2A), and contraction duration (CD; the period between when the intravesical pressure begins to rise for voiding and when this pressure returned to baseline; Fig. 2A). Mean values of each parameter from three micturition cycles in each rat were calculated and used to calculate group means before and after nerve transection.

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signal to create a corrected signal, which represented the original signal with the wandering baseline removed, which was then notch filtered for 60- and 120-Hz noise. A threshold for differentiating noise was set at 15 μV and used to calculate the mean firing frequency (number of peaks above the threshold per second) and mean amplitude of the signal (average of the amplitudes of each peak that crossed threshold). A mean of three values of each EMG in each condition was calculated for each animal. Using LabChart (AD Instruments) tools, the root mean square (RMS) value was calculated for the second shown in Fig. 2 in basal, void, and postvoid periods. To compare RMS EMGs, arithmetic amplitude and firing frequency at basal, void, and postvoid time points in the intact condition, values of the 15 rats were analyzed using ANOVA for repeated measures (GraphPad InStat 3.1, GraphPad Software, San Diego, CA). A Tukey post hoc test was used to compare between specific time points. Two-way ANOVA was used to determine significant differences between groups (MBSP, ABLST, and ABPDN) and the intact versus neurectomized condition, with the Holm-Sidak method for pairwise multiple comparison. P < 0.05 indicated a statistically significant difference for all tests.

In experiments 2 and 3, a mean of three values of each CMG, LPP, and voiding efficiency outcome variable (ICI, PP, CD, and LPP values) was calculated for each animal. Mean values of each variable for each animal were used to calculate a mean and SE in each condition and are presented as means ± SE. Two-way ANOVA was used to determine significant differences between groups and intact and neurectomized conditions with the Holm-Sidak method for pairwise multiple comparison. P < 0.05 indicated a statistically significant difference.

RESULTS

Experiment 1: EUS innervation. EUS EMG activity was low during filling (basal condition), increased as a bladder contraction began (Fig. 2A), and remained high during voiding, when high-frequency oscillations were present, and after voiding was complete (Fig. 2A). EMG bursting activity appeared during voiding, concurrent with high-frequency oscillations in bladder pressure (Fig. 2B). EUS EMG activity decreased slowly as bladder pressure decreased, reaching basal levels of tonic activity ~20 s after the bladder contraction was finished (Fig. 2, A and B). Compared with the basal condition, EUS EMG amplitude (P < 0.001) and frequency of firing activity (P < 0.001) were significantly increased during voiding and postvoiding (Figs. 2C and 3). RMS values were significantly low at the basal condition compared with void (P < 0.001) and postvoid (P < 0.001) periods (Fig. 3).

Bilateral neurectomy of the MBSP eliminated EUS EMG activity in all three states [basal (P < 0.01), voiding (P < 0.01), and postvoiding (P < 0.01); Figs. 4 and 5]. Bilateral neurectomy of the ABLT significantly decreased amplitude and firing frequency of the EUS EMG during basal (P < 0.01), voiding (P < 0.01), and postvoiding (P < 0.01) segments (Figs. 4 and 5) and eliminated bursting activity during voiding (Figs. 4 and 5). Bilateral neurectomy of the ABPD significantly decreased both amplitude and firing fre-
frequency of the EUS EMG in all three states but to a lesser extent ($P < 0.05$) than the other two neurectomies (Fig. 5), since some EUS bursting activity was preserved during voiding after transection of the ABPD (Fig. 4).

**Experiment 2: effect of bilateral neurectomies on CMG and LPP.** Neurectomy of the MBSP and ABLT decreased ICI, PP, and CD values significantly ($P < 0.001$; Table 1). Neurectomy of the ABPD significantly decreased PP and CD ($P < 0.05$). Denervation of the EUS by ABPD neurectomy decreased ICI only in five of seven rats. Neurectomy of the DNC dramatically decreased ICI ($P < 0.001$) but had a less dramatic but still significant effect on PP and CD ($P < 0.05$; Table 1). The effect of neurectomies on PP varied significantly between groups and between conditions (factor $B$, before neurectomy vs. after neurectomy; factor $A$: $P = 0.02$ and factor $B$: $P < 0.01$), with the values of MBSP- and ABLT-neurectomized animals re-

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**Fig. 3.** Example of EUS EMG activity during CMG of 1-s segments of the basal void and postvoid condition. RMS, root mean square value. ***$P < 0.001$ vs. the basal condition. Vertical scale bar = 50 μV. All three examples are from the same animal and are presented on the same scale.

**Fig. 4.** Example EUS EMG activity (top trace) and simultaneous bladder pressure (bottom trace) during CMG before and after bilateral neurectomy of the MBSP, ABLT, and ABPD. Arrows indicate the timing of the neurectomy.
duced to a greater extent than those with ABPD-neurectomized animals (Table 1).

Transection of the MBSP significantly decreased (P < 0.001) LPP by 50%. In contrast, transection of the ABPD or ABLT had a lesser but still significant effect (P < 0.05), decreasing LPP by ~25% (Fig. 6). Transection of the DNC did not significantly affect LPP values (Fig. 6). LPP varied between groups after MBSP neurectomy (factor A: P = 0.005, factor B: P < 0.001, and factor A × factor B: P = 0.11; Fig. 6) and was significantly reduced compared with DNC neurectomy (P < 0.01) and ABPD neurectomy (P < 0.05).

Experiment 3: voiding efficiency. Bilateral ABLT or ABPD transection did not significantly modify bladder capacity, VV, RV, or voiding efficiency. In contrast, bilateral neurectomy of the MBSP did not significantly affect these parameters, whereas neurectomy of the ABLT or ABPD significantly reduced voiding efficiency (P < 0.01 and P < 0.05, respectively).

### Table 1. Urinary parameters recorded during cystometrograms

<table>
<thead>
<tr>
<th>Groups</th>
<th>Intercontraction Interval, s</th>
<th>Peak Pressure, cmH₂O</th>
<th>Contraction Duration, s</th>
<th>Voiding Volume, ml</th>
<th>Residual Volume, ml</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before Nx</td>
<td>After Nx</td>
<td>Before Nx</td>
<td>After Nx</td>
<td>Before Nx</td>
</tr>
<tr>
<td>Motor branch of the sacral plexus</td>
<td>115 ± 10†</td>
<td>77 ± 5.0</td>
<td>36 ± 2.0†</td>
<td>20 ± 1.5‡</td>
<td>19 ± 0.9‡</td>
</tr>
<tr>
<td>Anastomotic branch of the lumbosacral trunk</td>
<td>104 ± 3.0*</td>
<td>82 ± 4.0</td>
<td>33 ± 1.8†</td>
<td>25 ± 1.2‡</td>
<td>17.5 ± 0.1*</td>
</tr>
<tr>
<td>Anastomotic branch of the pudendal nerve</td>
<td>95 ± 5.0</td>
<td>85 ± 5.0</td>
<td>36 ± 2.3†</td>
<td>27 ± 2.8</td>
<td>17.8 ± 1*</td>
</tr>
<tr>
<td>Dorsal nerve of the clitoris</td>
<td>117 ± 6.0†</td>
<td>59 ± 2.8</td>
<td>38 ± 1.7*</td>
<td>31 ± 2.2</td>
<td>18 ± 0.6*</td>
</tr>
</tbody>
</table>

Values are means ± SE. Nx, neurectomy. *P < 0.05 and †P < 0.01, significant difference between conditions; ‡P < 0.05, significant difference vs. the dorsal nerve of the clitoris group after Nx.
DISCUSSION

Neural control of the lower urinary tract is crucial to maintain normal urinary continence and voiding, and damage to its neural pathways results in overactive or underactive bladder, bladder sphincter dyssynergia, or urinary incontinence (10, 27, 46, 48). In the present study, we determined the effect of somatomotor and sensory urethral impairment on urinary continence and voiding in female rats by bilateral transection of specific nerves. With EMG recordings, we measured the multiple motor units of the EUS and analyzed the EMG in terms of amplitude and frequency of firing (number of spikes above a determinate threshold per second) during induced micturition to enable quantitative comparison of EUS EMG activity at different states during CMG and to determine the contribution of motor units whose axons travel through different nerves to generate the EUS EMG signal using nerve transection. While not ideal, these measures suffice to demonstrate a change in EMG activity with the selective neurectomies performed in this study.

EUS-EMG activity markedly increase during bladder contractions (9, 38). Bursting EUS activity during voiding is the causal factor of high-frequency oscillations observed in bladder pressure (6, 30). Urine flow takes place during the short silent periods of EUS bursting, when the bladder pressure oscillation is at the valley (43, 44). Elimination of EUS activity decreases voiding efficiency, indicating that this muscle activity during voiding is physiologically relevant (7, 30). Our method of EUS EMG analysis does not quantify bursting duration or intraburst interval but suffices to indicate that decreased EMG activity occurs after neurectomy, particularly after transection of the MBSP, which also significantly reduce void efficiency.

Our results confirm those of previous studies demonstrating that the EUS is innervated by the MBSP, as indicated by the total depletion of EUS EMG activity after bilateral MBSP neurectomy, whose axons travel through two anastomotic branches, the ABPD and ABLT (37). Compared with MBSP neurectomy, transection of the anastomotic branches partially eliminates EMG activity but did not dramatically decrease LPP or significantly affect voiding efficiency, indicating that partial denervation of the EUS induces mild symptoms of stress urinary incontinence (SUI), in contrast to complete EUS denervation by MBSP transection, which dramatically decreased LPP, indicative of severe SUI.

The existence of two peripheral neural pathways innervating the EUS suggests complementary innervation that could represent a safeguard, ensuring that innervation to the EUS remains intact in case of upper nerve damage, since, as our results indicate, half innervation (intact ABPD or ABLT) may partially support urinary continence and without voiding dysfunction. Dual neural pathways innervating pelvic organs is not uncommon, considering that afferent axons to the bladder and uterus travel through both PV and HG nerves (1, 13). Likewise, the receptive field of two nerves overlap in the neural circuit of the perineal skin (8, 35). Since EUS innervation is somatic, bladder and uterus innervation is autonomic, and perineal skin innervation is sensory, it appears that complementary innervation may be consistent across different types of neural circuitry. However, this remains to be proven definitively.

The EUS motoneuron pool is located in the dorsolateral nucleus of the L6 to S1 spinal segments (24, 32). The ABLT originates from the lumbosacral trunk, emerging from L4–L5 spinal spinal cord segments and increasing the possibility that some EUS motoneurons are localized rostral to L6. It is also plausible that axons of motoneurons located at L6 travel through the ABPD and ABLT (Fig. 7). This proposal is supported by the anatomic connection described between L6 and the lumbosacral trunk in ~80% of male and female rats (35, 37).

Neuromuscular circuits are established when the EUS fibers develop and mature at late embryonic and early postnatal periods (2, 23). Axons of lumbosacral motoneurons traveling through the ABLT branch at the sacral plexus, instead of continuing in the sciatic nerve, and join the MBSP to innervate the EUS, suggesting that motoneuron-target specificity is established at an early embryonic stage and the axons follow their predetermined target pathways presumably by chemical mediation (19, 25, 29).

Denervation of the EUS in experimental animals has been used in urological research to determine the contribution of the EUS to the control of urinary continence (17, 41). However, our findings suggest that, depending on the site of neurectomy, the result might be a partial EUS denervation. PD neurectomy distal to branching of the PV nerve, which is the classic localization of the PD nerve through a lower midline abdominal incision near the internal iliac vessels (14, 36), will likely only partially denervate the EUS (through the axons traveling through the ABPD). In addition, this procedure will transect other PD branches, such as those innervating the clitoris/penis, coccygeus, and perineal skin (8). When the PD and sacral plexus are localized from a dorsal midline skin incision at the level of the ischiorectal fossa, the EUS can be partially or fully denervated depending on the site of transection: partially if nerve transection is performed proximal to the sacral plexus and fully denervated if the nerve is transected distal to the location of branching from sacral plexus (MBSP in Fig. 1).

The relationship of the sacral plexus to the sciatic nerve has also been misused as an anatomic marker to indicate the origin...
of innervation of the EUS. This is the case where it is reported that the “PD was localized and cut distal to the origin of the pudendal nerve from the sciatic nerve” (28). Previous studies have localized and damaged or transected the nerve innervating the EUS in the ischiorectal fossa, as it travels dorsolateral to internal PD vessels (26, 32, 38). In the terminology we use, we believe that they cut the MBSP and thus fully denervated the EUS. At this level, clear notation should be made to indicate if the sensory branch of the PD nerve, located ventromedial to the vessels, is also injured or not. This anatomic information is important when comparing results between different studies.

The urethra participates in both continence and expulsive urinary function, particularly in rats, since the EUS is currently activated with bladder contraction during micturition, which facilitates voiding efficiency (7). The results of our MBSP neurectomy experiments indicated that full urethral somatomotor denervation affects both of these functions. Similarly, previous studies have demonstrated that both SUI and voiding dysfunction can be induced by impairment of urethral closure (14, 20, 21).

The fact that transection of the DNC did not significantly affect LPP but decreased CMG parameters and voiding efficiency suggests that deafferentation of the distal urethra does not induce urinary continence impairment but does induce voiding dysfunction, probably by mechanisms related to urethral obstruction, since neurectomy of the sensory branch of the PD nerve reduces EUS bursting activity during voiding (11, 39). Considering that the DNC only innervates the distal region of the urethra (37), significant effects of transection on voiding likely result from a partial sensory denervation of the urethra, at least in anesthetized rats.

Impairment of sensory and/or motor lower urinary tract innervation may occur by traumatic events or neurogenic pathologies, such as spinal cord injury or multiple sclerosis, resulting in voiding dysfunction (5, 31). In spinal cord-injured patients, high intravesical pressure associated with bladder sphincter dysynergia is reduced by performing a sphincterotomy (40, 45). In humans, the EUS is innervated by the perineal branch of the PD nerve and dorsal nerve of the penis (34). If partial motor denervation is sufficient to decrease EUS activity without collateral urinary incontinence has not been determined.

The pathology most commonly reported after pelvic surgeries or vaginal delivery is SUI, with damage of the EUS innervation as a factor (4, 15, 16). Whether urethral sensory nerves are damaged during childbirth is unknown. Urethral afferents activate the detrusor, and pathological activation of these afferent fibers may be involved in detrusor instability (23). However, additional studies are necessary to determine the clinical role of sensory innervation in SUI and other pathologies.

A limitation of the present study is the possible effect of the estrous cycle on EUS activity. Although it is an important issue to be analyzed, we consider that our results are validated by the fact that the effect of the neurectomy was measured within subjects, each individual was their own control, and that neither micturition of intact rats as well as induction of SUI in mice are affected by the estrous cycle (18, 22).

In conclusion, somatomotor control of micturition is provided by the MBSP with axons travelling through the ABPD and ABLT, the latter with a major contribution. PN transection only partially denervates the EUS. Partial somatomotor urethral denervation induces signs of mild SUI but not significant voiding dysfunction. Partial afferent denervation of the urethra induces signs of voiding dysfunction but does not affect urinary continence.

Peripheral nerves may be impaired not only with illness or spinal cord injury but also with aging or during reproductive processes such as parturition. Detailed knowledge of the neuroanatomy and functional innervation of the urethra will enable more accurate animal models of neural development, disease, and dysfunction in the future.

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

AUTHOR CONTRIBUTIONS

Author contributions: Y.C. conception and design of research; Y.C., C.F.P., and P.J.Z. performed experiments; Y.C., C.F.P., and B.M.B. analyzed data; Y.C. and M.S.D. interpreted results of experiments; Y.C. and B.M.B. prepared

Fig. 7. Diagram of the sacral plexus in rats. The dark dashed lines indicate the possible neural pathways followed by axons of the EUS motoneurons to reach the MBSP, passing by the ABPD or ABLT. The axons, represented by light dashed lines, have their cell bodies at the L5 spinal cord segment. The oval represents the column of the EUS motoneuron pool. SBPD, sensory branch of the pudendal nerve; LST, lumbosacral trunk.


