similar to the PRG. Finally, the PRG is well positioned to contribute as active partners in shaping the future priorities and focuses of the PMC.

In conclusion, the PRG offers an innovative, tested, and comprehensive model to appropriately integrate stakeholder and patient perspectives into healthcare research. By including a framework that supports all four facets (researchers, patients, clinicians/clinical leaders, community/stakeholders) of health research throughout the research lifecycle, studies obtain legitimacy and resource support that is not easily found elsewhere. The PRG offers inclusive engagement, external buy-in, and a community-connected perspective that is sure to provide a *Voice for the Voiceless*.

Authors' Contributions

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References

1. PCORI's Timeline. PCORI Patient-Centered Outcomes Research Institute. 2015. Available at: https://www.pcori.org/about-us/pcoris-timeline (accessed January 1, 2021).
2. Sheridan S, Schrandt S, Forsythe L, Hilliard TS, Paez KA; Advisory Panel on Patient Engagement (2013 Inaugural Panel). The PCORI engagement rubric: Promising practices for partnering in research. Ann Fam Med 2017;15(2):165–70.
3. Kerns RD, Brandt CA, Peduzzi P. NIH-DoD-VA pain management collaboratory. Pain Med 2019;20(12):2336–45.
4. Bastian LA, Cohen SP, Katsovich L, et al.; NIH-DOD-VA Pain Management Collaboratory. Stakeholder engagement in pragmatic clinical trials: Emphasizing relationships to improve pain management delivery and outcomes. Pain Med 2020;21(Suppl 2):S13–20.
5. Manafo E, Petermann L, Mason-Lai P, Vandall-Walker V. Patient engagement in Canada: A scoping review of the "how" and "what" of patient engagement in health research. Health Res Policy Syst 2018;16(1):5.Erratum in: Health Res Policy Syst 2018;16(1):24.
6. Forsythe LP, Ellis LE, Edmundson L, et al. Patient and stakeholder engagement in the PCORI pilot projects: Description and lessons learned. J Gen Intern Med 2016;31(1):13–21.
7. Ali J, Davis A, Burgess D, et al. Justice and equity in pragmatic clinical trials: Considerations for pain research in integrated health systems. Learn Health Syst 2021;e10291. doi:10.1002/lrh2.10291.

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Commentary

Spinal Cord Stimulation and Urinary Dysfunction

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Since the first spinal cord electrode implantation in 1967 by Shealy et al., spinal cord stimulation (SCS) has gained an established role in standard pain practice [1]. In SCS, epidural electrodes are used to electrically modulate the circuitry in the spinal cord, thereby altering the functional connectivity between peripheral or central pain generators and pain perception networks. Little has been reported on side effects caused by disrupting the functional connectivity of nonpathological non-pain pathways. In the present commentary article, we report on 11 patients with a normal (nonpathological) spinal cord presenting with stimulation-related autonomic side effects during SCS. We discuss the clinical and pathophysiological aspects and propose diagnostic and therapeutic guidance for clinicians encountering autonomic side effects in patients receiving SCS. In theory, loss of normal bladder function can be attributed to an electrical stimulation–induced alteration of functional connectivity, which induces reversible neurogenic bladder symptoms.

Of 386 patients treated with SCS at the University Hospitals Leuven between 2005 and 2021, three patients (0.8%) with SCS presented with new stimulation-related micturition side effects. Table 1 summarizes patient-specific details and stimulation-relatedness.

A 47-year-old male with complex regional pain syndrome after a crush injury of the right hand received an epidural quadripolar electrode (Pisces Quad Medtronic, Minneapolis, MN, USA) at C6–C7 in 2000, which resulted in significant pain relief. Nine years after the initial implantation, he developed abdominal paresthesia and new-onset urge incontinence (a strong urge to micturate during stimulation ON). Switching the stimulator off could completely and immediately resolve symptoms. The patient had no other medical history or preexisting urinary problems, no recent changes in medication or in SCS use, and no triggering factor that could be withheld. Urodynamic studies in ON–OFF mode revealed a bladder capacity of 140 mL with normal compliance and normal miction phase without residue in the OFF mode. In the ON mode, bladder capacity was limited to 17 mL, and immediate loss of urine occurred because of detrusor overactivity, i.e., an abnormal overactive contraction (Figure 1). Electromyography (including the pelvic floor) revealed bilateral changes in the motor unit potentials of lumbosacral levels L4–S4 in the ON mode (light polyphasic motor unit potentials with irregular firing). Radiography showed no displacement of the electrode, and urinalysis was normal. After multiple unsuccessful reprogramming attempts (abdominal paresthesia disappeared, and urinary incontinence ameliorated somewhat but persisted), a revision with an octopolar electrode (Medtronic, Minneapolis, MN, USA) positioned at a higher cervical level (C4–C7) resulted in good pain control without urinary side effects. Urinary incontinence, however, reappeared after 3 weeks and remained stimulation dependent at follow-up, for which reason the patient still wears urinary pads. Botulinum toxin bladder injections were refused by the patient because of reimbursement issues. Seven years later, turning the stimulation off still completely abolishes incontinence, and during impedance measurement, the patient involuntarily loses urine.

A 68-year-old male with failed back surgery syndrome after right-sided L4–L5 discectomy had been treated with SCS since 2000 for bilateral neuropathic pain in the lower limbs. In his history, only mild obstructive urinary symptoms (which required no further treatment) were relevant. There were no recent changes in medication use. Since placement of a new octopolar electrode (Medtronic, Minneapolis, MN, USA) at the T10–T11 vertebral level in 2020, the patient reported a sudden exacerbation of obstructive urinary symptoms (increased hesitancy with impossible miction in the first minute and increased intermittency), as well as difficulties in defecation. Symptoms were relieved by turning the stimulator off. During a 3-day OFF trial, all symptoms improved and returned to baseline. Electrode radiography, magnetic resonance imaging, and urinalysis were normal (Figure 2). Reprogramming of stimulation parameters (2+ 3– 4– 5 +, 4.15 V, 330 μs, 60 Hz) by decreasing the amplitude to 3.2 V led to improvement of the exacerbated symptoms while maintaining a good effect on neuropathic pain (the preoperatively known mild obstructive symptoms persisted).

In a 41-year-old woman with a newly implanted epidural electrode with the lower tip at the T11–T12 level for failed back surgery syndrome (after left-sided L5–S1 microdiscectomy) with unilateral neuropathic pain, two unprecedented episodes of significant and sudden urinary incontinence occurred in the first 3 months after SCS implantation. Between episodes, she had no urinary symptoms. The electrode was initially inserted at the T9–T10 level, but postoperative radiography revealed accidental displacement of the electrode (T11–12) as compared with intraoperative images. This displacement was accepted, as there was excellent pain relief. There was no relevant history or change in medication use. A direct causal role between SCS and the patient's urinary problems was less clear than in the two previously described patients, as urodynamic studies performed in ON mode were completely normal. The patient is currently in follow-up, and reprogramming will be considered in case of new urinary incontinence.

To compare our findings with international databases, we performed a search in the ongoing international PSR/ISPR database (Medtronic, Minneapolis, MN, USA; Product Surveillance Registry [PSR], 2012–2021 and ongoing; Implantable Systems Performance Registry [ISPR], 2003–2016). This search resulted in eight additional patients with urinary side effects associated with SCS, out of a total of 2,259 patients (0.35%). Five patients presented with irritative urinary symptoms (urgency, frequency, or incontinence), and three presented with obstructive autonomic symptoms. More detailed information on the complaints, the stimulation-

Table 1. Summarized patient-specific details and stimulation-relatedness

	Stimulation Specifics			Micturition Side Effect Specifics		
	Patient Specifics	Primary Indication	Level and Type of Epidural Electrode	Type of Autonomic Adverse Effect	History and Stimulation-Relatedness	Treatment and Effect
1	Male; 47 y/o; 2009	CRPS right hand after crush injury	C6–C7 Quadripolar; Pisces Quad (Medtronic, Minneapolis, MN, USA)	Urge incontinence	Strong urge to micturate and incontinence during stimulation; immediate resolution with stimulation OFF Urodynamic study with stimulation OFF: bladder capacity of 140 cc with normal compliance and miction Urodynamic study with stimulation ON: limited bladder capacity of 17 cc and immediate loss of urine due to detrusor overactivity (abnormal overactive contraction) Medical history: appendectomy and sleep disturbances Medication use: gabapentin, ibuprofen, pantoprazole, amitriptyline, clonazepam, vitamin B12	Reprogramming brought partial relief of symptoms. Revision (implanting a new electrode at a higher cervical level) brought partial relief of symptoms.
2	Male; 68 y/o; 2020	FBSS with bilateral neuropathic pain in lower limbs	T10–T11 Octopolar; (Medtronic, Minneapolis, MN, USA)	Urinary retention Fecal retention	Patient with a history of mild obstructive urinary symptoms; since a revision with placement of a new epidural electrode, important exacerbation of hesitancy and intermittency (obstructive urinary symptoms) Stimulation ON: exacerbation of obstructive symptoms: hesitancy (impossible micturition during the first minute), increased intermittency, and difficulty defecating Stimulation OFF (3-day trial): immediate improvement to baseline Imaging showed correct electrode position; no conus/spinal cord lesion on MRI Medical history: mild obstructive urinary symptoms (untreated), Raynaud's syndrome, gynecomastia (testosterone isocaproate once monthly), arterial hypertension, and cholecystectomy Medication use: testosterone isocaproate and lisinopril-hydrochlorothiazide.	Reprogramming (lowering amplitude) led to resolution of symptoms to baseline within 1 week.
3	Female; 41 y/o; 2020	FBSS with unilateral neuropathic pain in left lower limb	T12–L1	Urinary incontinence	Experienced two unprecedented episodes of sudden urinary incontinence since placement of epidural electrode, with no urinary symptoms in between Normal urodynamic studies in ON mode	Follow-up; reprogramming will be considered in case of new urinary incontinence.

(continued)

Table 1. continued

	Stimulation Specifics			Micturition Side Effect Specifics		
	Patient Specifics	Primary Indication	Level and Type of Epidural Electrode	Type of Autonomic Adverse Effect	History and Stimulation-Relatedness	Treatment and Effect
					Medical history: atopic dermatitis and pleuritis Medication use: ibuprofen, paracetamol, tramadol.	
4	ISPR; 2012			Urinary retention	Experienced urinary retention with stimulation ON and resolution of retention with stimulation OFF	Reprogramming led to resolution of symptoms.
5	ISPR; 2015			Urinary incontinence	Reported new urinary incontinence due to stimulation	Reprogramming led to resolution of symptoms.
6	ISPR; 2018			Urinary retention Fecal retention	New partial urinary and fecal retention since placement of epidural electrode Stimulation ON: urinary and fecal retention Stimulation OFF: resolution of these obstructive symptoms	Reprogramming led to resolution of symptoms.
7	ISPR/ PSR			Urinary frequency	Stimulation ON: Patient feels stimulation of her bladder with new urinary frequency Stimulation OFF: resolution of irritative symptoms	NM
8	ISPR/ PSR			Urinary incontinence Fecal incontinence	Stimulation ON: good effect on pain, but new loss of bladder and bowel control with incontinence Stimulation OFF: resolution of irritative bladder and bowel symptoms	NM
9	ISPR/ PSR			Urinary incontinence	Stimulation ON: urinary incontinence, especially during walking (has to wear a diaper). When impedances were measured during consultation, patient suffered from involuntarily loss of urine Stimulation OFF: resolution of irritative symptoms	NM
10	ISPR/ PSR			Urinary retention Fecal retention	During SCS trial, the patient reported an increase in pain and new difficulties in micturition and defecation (retention)	NM
11	ISPR/ PSR			Urinary urgency	During SCS, the patient reported an increase in pain and new urinary urgency with stimulation ON	NM

y/o = years old; CRPS = complex regional pain syndrome; FBSS = failed back surgery syndrome; MRI = magnetic resonance imaging; ISPR = Implantable Systems Performance Registry; PSR = Product Surveillance Registry; NM = not mentioned.

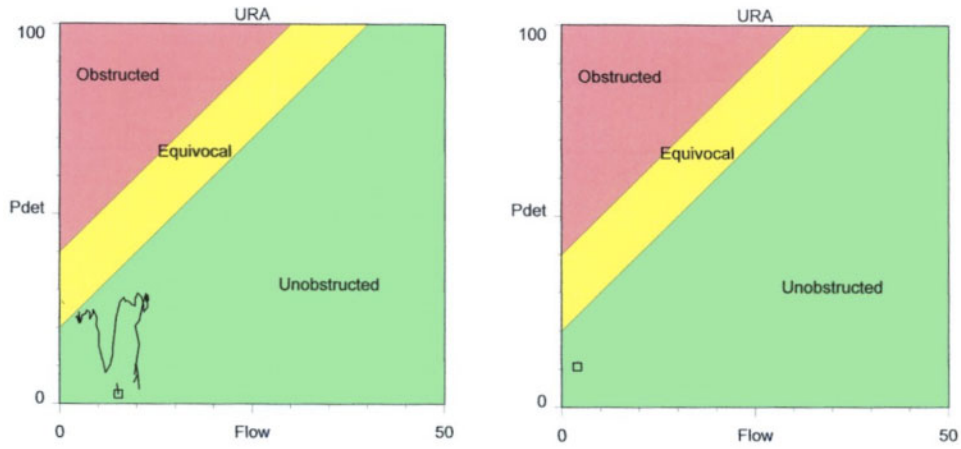

Figure 1. Patient 1—urodynamic studies in ON and OFF mode. Left: Urodynamic study in OFF mode revealed a bladder capacity of 140 mL with normal compliance and normal miction phase without residue. Right: Urodynamic study in ON mode showed bladder capacity limited to 17 mL, and immediate loss of urine occurred because of detrusor overactivity, i.e., an abnormal overactive contraction.

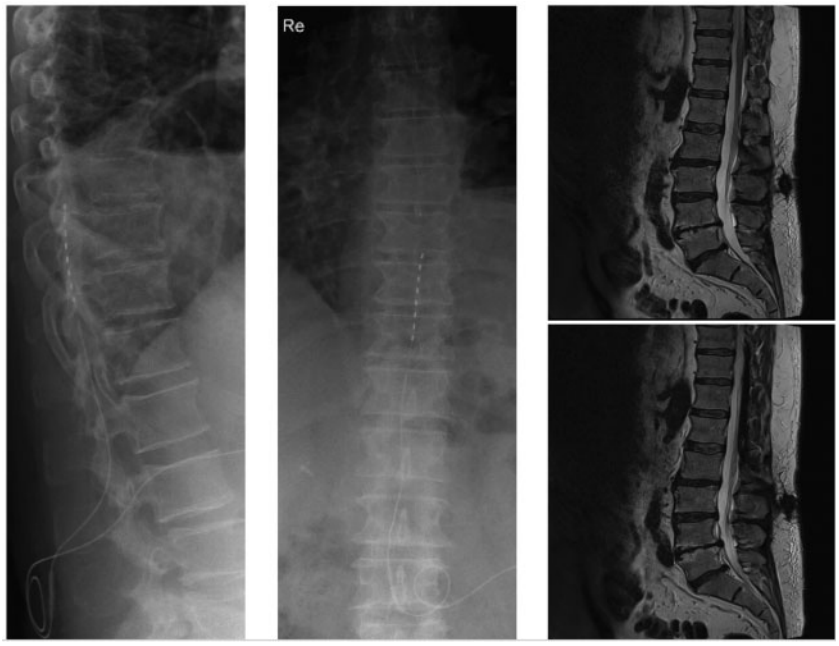

Figure 2. Patient 2—electrode radiography and MRI. Patient 2 had stimulation-related obstructive autonomic symptoms during SCS for failed back surgery syndrome. After placement of a new octopolar electrode in 2020, stimulation-related obstructive urinary and fecal symptoms developed. Radiography revealed correct position of the electrode epidurally at the T10–T11 vertebral level. MRI confirmed correct position of the electrode and ruled out damage to the conus or spinal cord after surgery (MRI-compatible system). MRI= magnetic resonance imaging.

relatedness (by turning the stimulation ON and OFF), and reprogramming effects is presented in Table 1.

The present report aims to increase awareness about possible autonomic side effects of SCS. Two previous case studies discussed micturition inhibition during thoracolumbar SCS in patients with previous spinal cord injury [2, 3]. Urinary adverse effects were categorized as "very rare" in a recent review, based on these reports [4]. According to our report, autonomic bladder dysfunction could occur in an estimated 0.3–0.8% of patients receiving SCS.

Urinary side effects were seen in both cervical and thoracic electrode locations and can encompass the spectrum of both obstructive and irritative bladder and bowel symptoms (obstructive bladder symptoms in 4/11, irritative bladder in 7/11, obstructive bowel in 3/11, and irritative bowel in 1/11 subjects). Furthermore, a direct causal link between neurostimulation and reversible bladder

symptoms is suggested by 1) a urodynamic study in ON–OFF mode in one patient, 2) involuntary urine loss during routine electrode impedance measurement at a clinical visit in two patients, and 3) resolution of symptoms by turning the stimulation OFF in eight patients (not reported in three). Reprogramming of the stimulation parameters resulted in resolution of symptoms in three of five patients (i.e., the five out of eleven subjects that underwent reprogramming. A watchful waiting strategy was deployed in one out of eleven and no information on management in the remaining five out of eleven subjects), one of five showed partial resolution (resolution of the exacerbated symptoms, though baseline prostatism complaints remained), and one of five showed improvement without complete resolution, for which a surgical revision brought partial relief.

Conversely, SCS has been experimentally used to treat urinary dysfunction [5]. A case report described a patient with preexisting urinary incontinence, for which she received sacral nerve stimulation [6]. She concomitantly suffered from failed back surgery syndrome after lumbar surgery, for which an SCS device was implanted (T8–T10). This resulted in excellent pain relief and an unforeseen relief of urinary incontinence, which allowed the sacral neurostimulator to be discontinued [6]. Another article reported on high-frequency SCS in treating neurogenic bladder incontinence in spinal cord injury or neurological disease [7].

We offer a diagnostic and therapeutic guide for clinicians encountering autonomic side effects in SCS in daily practice. As a first step, one should determine a probable causality between stimulation and the side effect: A detailed history of the complaint, ON–OFF stimulation trials (and provoking symptoms during impedance measurement), and urodynamic studies in ON and OFF mode can objectify the impact of stimulation on voiding. Electromyography (including the pelvic floor) might reveal a functional impact on normal motor function. Second, there exists a complex interplay between the neural control of micturition and (chronic and acute) disease, medications, and spinal cord injury. To differentiate, ruling out spinal cord (or conus) damage with magnetic resonance imaging (if the stimulation device allows, and especially if symptoms started after electrode insertion), assessing correct epidural position with electrode radiography, ruling out infection with urinalysis, and reviewing medications (e.g., opioids) and concurrent medical diseases affecting micturition (e.g., benign prostatic hyperplasia, diabetes mellitus, stroke, Parkinson's disease, etc.) are necessary. Consultation with an expert urologist can be useful (Figure 3).

We propose reprogramming stimulation parameters as a first step in an attempt to resolve side effects. If this fails, surgical repositioning of the electrode might alleviate symptoms. Decreasing the amplitude of stimulation will decrease the volume of activated tissue, whereby, hypothetically, the tracts related to micturition will be less affected by SCS. A replacement in a different position can theoretically benefit the patient, as the target tissue activated by stimulation will differ. One could speculate that such stimulation will still result in positive effects on pain while at the same time avoiding the

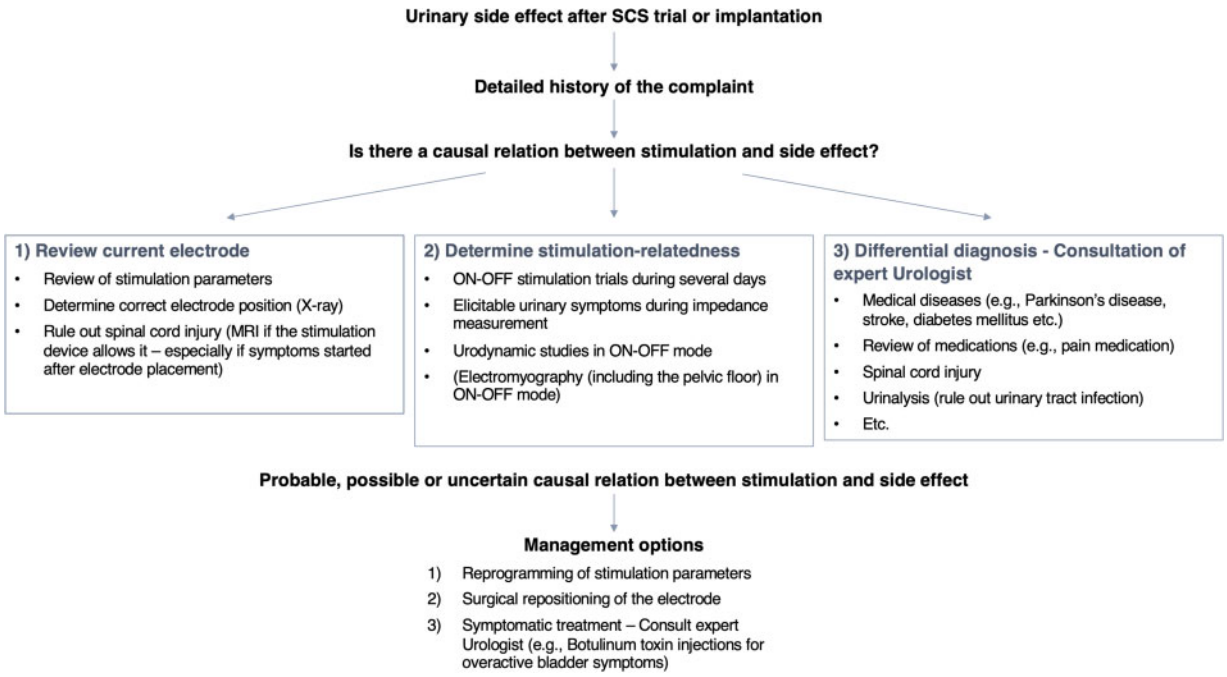

Figure 3. Diagnostic flowchart. Suggested diagnostic measures to guide clinicians in determining a probable, possible, or uncertain causal relation between stimulation and urinary side effects after SCS trial or implantation. X-ray = radiography; MRI = magnetic resonance imaging.

Table 2. Anatomic and functional organization of neural micturition control

Peripheral and Spinal Innervation		
Efferent innervation		
CNS	*Peripheral nerves*	*Function*
Intermediolateral region of sacral spinal cord S2–S4	Autonomic parasympathetic Sacral roots (S2–S4) and pelvic nerves Ganglia in pelvic plexus and bladder wall	Contraction of detrusor (smooth) muscle: voiding Relaxation of urethral (smooth) muscle
Thoracolumbar spinal cord T11–L2	Autonomic sympathetic Hypogastric and pelvic nerves Ganglia in inferior mesenteric plexus and paravertebral sympathetic chain	Relaxation of detrusor (smooth) muscle: storage Contraction of bladder neck and urethra (smooth) muscle: storage
Motor neurons in lateral ventral horns of sacral spinal cord S2–S4 (Onuf's nucleus)	Somatic Pudendal nerves	Contraction of external (striated) urethral sphincter
Afferent innervation		
Peripheral nerve fibers	*CNS*	*Function*
Aδ fibers (myelinated)	Cell bodies located in dorsal root ganglia	Bladder filling: passive distension and active contraction
C fibers (unmyelinated) ("silent" fibers)	Axon travels to dorsal horns of T11–L2 (filling status) and the S2–S4 segments (sensation of bladder neck and sphincter)	Nociception, temperature, pH
Peripheral nerves	*CNS*	*Function*
Pelvic and hypogastric nerves	T11–L2	Sensation of bladder fullness
Pudendal and hypogastric nerves	S2–S4	Sensory information of bladder neck and urethra
Supraspinal Micturition Centers		
Specific micturition regions	*Function*	
PAG (central and lateral PAG)	Important relay station through (often reciprocal) connections between spinal cord, higher micturition regions, and PMC: • Registration of bladder filling sensations: receives and passes bladder afferent input to supraspinal micturition regions (prefrontal cortex, insula, anterior cingulate, thalamus . . .) • Manipulation of the firing of the voiding reflex: receives input from supraspinal micturition regions • Controls the primary input to the PMC: excitates PMC, which facilitates voiding.	
PMC, Barrington's nucleus	Important supraspinal output center Facilitates voiding (e.g., spinobulbospinal pathway)	
Anterior cingulate (medial frontal cortex)	Attributing attention to afferent bladder signals Reacting to afferent bladder signals: decision to void or recruiting mechanisms against voiding (e.g., external sphincter contraction)	
Prefrontal cortex (e.g., inferior frontal gyrus)	Determining social appropriateness to void (reciprocal connections with PAG and connections with hypothalamus and anterior cingulate): exerts tonic suppression of PAG input to the PMC during bladder filling, which prevents incontinence	
Insula	Visceral sensation (desire to void)	
Hypothalamus (caudal and preoptic)	Likely role in permitting voiding when environment is deemed "safe" (through direct control of PMC)	

During bladder filling, a local interneural spinal reflex known as the "guarding reflex" promotes continence. Afferent vesical input (pelvic nerves) stimulates spinal sympathetic output, which leads to contraction of the bladder neck and urethra (hypogastric nerve), inhibits detrusor muscle contraction, and stimulates pudendal output to the external sphincter (contraction). A pontine storage center might also contribute to external urethral sphincter contraction. The spinobulbospinal reflex facilitates voiding. Afferent ascending input passes via the PAG to the PMC, which stimulates parasympathetic output to the detrusor muscle (contraction) and internal sphincter (relaxation) and inhibits sympathetic and pudendal output. Higher cerebral regions are responsible for conscious sensation, integration of the social environment, and the switch between storage and voiding.[8, 9] CNS = central nervous system; T = thoracic; L = lumbar; S = sacral; PAG = periaqueductal gray; PMC = pontine micturition center.

adverse effects due to stimulation of the micturition tracts. No specific stimulation parameters or lead positions are known to cause these adverse effects, nor do optimal programming or placement strategies exist to avoid these symptoms. For refractory cases, the benefits of SCS should be weighed against the burden of micturition dysfunction. Symptomatic treatment (e.g., botulinum toxin injections for overactive bladder) by an expert urologist might alleviate symptoms while keeping stimulation in place.

The exact mechanisms underlying autonomic side effects in SCS remain elusive, and neural control of micturition is a complex system (its components are summarized in Table 2) [8, 9]. SCS might act locally through interference with the local afferent and interneural network in the spinal cord, which is located mainly in the superficial dorsal horn, dorsal commissure, and intermediolateral region (local spinal reflexes). Passing efferent autonomic or somatic fibers could be modulated in the same way. Furthermore, disrupting the functional connectivity between the spinal cord and supraspinal micturition centers by modulating ascending and descending autonomic spinal tracts involved in micturition could be an alternative mechanism. Indeed, as presented, cervical electrode positions can cause these side effects, remote from the local spinal micturition regions (thoracolumbar and sacral spinal segments). Lastly, modulation of supraspinal micturition regions could possibly explain side effects seen in thoracolumbar and cervical electrodes, as SCS is known to cause both functional and morphometric distant brain effects (e.g., through prefrontal regions and the anterior cingulate cortex, which are regions that are also involved in micturition) [10, 11]. Further research is needed to elucidate patient- and stimulation-related factors predisposing to stimulation-induced autonomic side effects, as well as mechanisms of action and subsequent therapeutic interventions.

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References

1. Shealy CN, Mortimer JT, Reswick JB. Electrical inhibition of pain by stimulation of the dorsal columns: Preliminary clinical report. Anesth Analg 1967;46(4):489–91.
2. Loubser PG. Adverse effects of epidural spinal cord stimulation on bladder function in a patient with chronic spinal cord injury pain. J Pain Symptom Manage 1997;13(5):251–2.
3. La Grua M, Michelagnoli G. Rare adverse effect of spinal cord stimulation: Micturition inhibition. Clin J Pain 2010;26 (5):433–4.
4. Bendersky D, Yampolsky C. Is spinal cord stimulation safe? A review of its complications. World Neurosurg 2014;82 (6):1359–68.
5. Li LF, Ka-Kit Leung G, Lui WM. Sacral nerve stimulation for neurogenic bladder. World Neurosurg 2016;90:236–43.
6. Yakovlev AE, Resch BE. Treatment of urinary voiding dysfunction syndromes with spinal cord stimulation. Clin Med Res. 2010;8(1):22–4.
7. Schieferdecker S, Neudorfer C, El Majdoub F, Maarouf M. A retrospective case series of high-frequency spinal cord stimulation (HF10-SCS) in neurogenic bladder incontinence. Oper Neurosurg 2019;17(1):14–20.
8. Fowler CJ, Griffiths D, De Groat WC. The neural control of micturition. Nat Rev Neurosci 2008;9(6): 453–66.
9. Seth JH, Panicker JN, Fowler CJ. The neurological organization of micturition. Handb Clin Neurol 2013;117 :111–7.
10. De Groote S, Goudman L, Van Schuerbeek P, et al. Effects of spinal cord stimulation on voxel-based brain morphometry in patients with failed back surgery syndrome. Clin Neurophysiol 2020;131(11):2578–87.
11. Bentley LD, Duarte RV, Furlong PL, Ashford RL, Raphael JH. Brain activity modifications following spinal cord stimulation for chronic neuropathic pain: A systematic review. Eur J Pain 2016;20(4):499–511.

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Commentary

Sympathetic Blocks for Raynaud's Phenomena in Pediatric Rheumatological Disorders

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