

Case Study

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Suprapubic Catheter–Associated Defecatory Outlet Dysfunction in a Decompressed Bladder: An Integrated Mechanistic Framework and Empirical Validation *A Synthesis of Viscerovisceral Cross-Talk, Sacral Spinal Reflex Arc Modulations, Pharmacological Interventions, and Diagnostic Protocols*

Bruce H. Knox* 

Independent Scholar, Auckland, New Zealand

***Corresponding Author**

Bruce H. Knox, Independent Scholar, Auckland, New Zealand.

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Abstract

Background: Suprapubic catheterisation (SPC) provides continuous urinary drainage and leaves the urinary bladder substantially decompressed. Nevertheless, an indwelling suprapubic catheter remains a persistent mechanical stimulus within the bladder. Catheter-related bladder discomfort, trigonal irritation, and detrusor spasm occur even when the bladder is not filled. The urinary bladder, pelvic floor, and anorectal outlet are linked through shared sacral, spinal, and supraspinal neural networks, raising the possibility that persistent catheter-generated afferent input influences anorectal motor behaviour. **Hypothesis & Integrative Synthesis:** This paper establishes an expanded mechanistic framework proposing that continuous mechanical contact between an SPC/balloon and the decompressed bladder wall triggers persistent urothelial/suburothelial afferent firing ($A\delta$ and C fibres). Through sacral spinal cord convergence (S2–S4) and the vesico-anal reflex arc, this afferent activity induces central sensitization and a protective pelvic-floor motor guarding reflex. Consequently, impaired relaxation of the puborectalis muscle and external anal sphincter during attempted defecation produces acquired anorectal outlet obstruction despite preserved colonic transit.

Core Hypothesis: Decompressed Bladder $\neq$ Neurologically Silent Bladder. Continuous urine drainage removes volumetric stretch but maintains foreign-body mucosal irritation. Bladder-derived nociceptive input directly recruits pelvic floor motor pathways, producing dyssynergic defecation.

1. Introduction

The urinary bladder and anorectum are commonly managed as separate anatomical entities in clinical practice, yet their neurodevelopmental origins and central/peripheral neurological controls are deeply integrated. Sensory information arising from pelvic viscera converges at dorsal root ganglion (DRG), spinal cord, and supraspinal levels, establishing an anatomical substrate

for functional interaction between the bladder, bowel, and pelvic floor musculature [1]. Malykhina described pelvic visceral cross-sensitization as a primary mechanism through which irritation in one organ modulates the physiological tone of adjacent structures [2]. Similarly, extensive reviews on colon–bladder cross-sensitization emphasize shared spinal afferent pathways and bidirectional functional cross-talk [3]. Systematic clinical reviews

confirm that lower urinary tract symptoms (LUTS) and bowel motility disorders frequently co-occur [4]. However, traditional physiological models of bladder–anorectal reflex interactions rely almost exclusively on bladder filling or volumetric distension. This raises a fundamental clinical question in patients managed with a continuously draining suprapubic catheter (SPC): Can a decompressed bladder generate sufficient afferent signaling to induce defecatory outlet dysfunction? Evidence from catheter-related bladder discomfort (CRBD) and pelvic neurophysiology indicates that bladder distension is not required for neurogenic motor recruitment. This paper merges theoretical neurobiology with empirical validation and clinical diagnostics to establish a comprehensive framework for SPC-associated defecatory outlet dysfunction.

2. Mechanistic Framework: From Foreign Body to Outlet Resistance

Step 1: The Decompressed Bladder as an Active Sensory Stimulus

Continuous urinary drainage into a collection bag removes urine volume but leaves the catheter tip, shaft, and retention balloon in direct, unbuffered contact with the urothelium. Jang et al. demonstrated that following bladder decompression, the catheter tip frequently contacts the bladder mucosal wall and trigone, generating severe focal irritation, catheter-related bladder discomfort, and uninhibited detrusor spasms [5]. Furthermore, long-term SPC studies highlight persistent bladder spasms, pain, and peristomal leakage despite continuous patency [6]. Decompression allows the bladder walls to collapse around the foreign body, maximizing mucosal friction, traction along the SPC tract, and trigonal mechanoreceptor engagement [5,6].

Step 2: Urothelial Activation and Detrusor Spasm

Mechanical contact and traction provoke the release of urothelial mediators (such as ATP, acetylcholine, and substance P), activating mechanosensitive Aδ-fibres and nociceptive C-fibres in the suburothelium [5]. This sensory stimulation triggers muscarinic reflex pathways, eliciting involuntary detrusor contractions. Crucially, these detrusor spasms occur independently of physiological filling volumes.

Step 3: Reflex Pathways Supplying the External Anal Sphincter

Neurophysiological support for this cross-organ coupling is anchored in experimental models of the vesico-anal reflex. Thor and Muhlhauser demonstrated in animal models that chemical or mechanical irritation of the lower urinary tract elicits robust reflex activation of motoneurons supplying the striated external anal sphincter (EAS) via spinal pathways mediated by nociceptive C-fibres [7]. Complementing these findings, Buntzen et al. proved the existence of an excitatory vesico-anal reflex in humans, demonstrating that acute bladder stimulation significantly increases anal canal pressure without altering rectal motility—a response completely abolished by epidural blockade [8].

Step 4: Central Sensitization and the Pelvic Guarding Arc

Afferent signals from the pelvic nerve (parasympathetic) and pudendal nerve converge within the S2–S4 dorsal horn interneuronal pools. Dichotomizing sensory neurons—bifurcating axons innervating both the bladder trigone and anorectum—provide a peripheral mechanism for cross-organ sensitization [1,2]. Persistent nociceptive input from an indwelling catheter activates Wide Dynamic Range (WDR) neurons in the spinal cord, inducing central sensitization via NMDA receptor activation [2,3]. This state of spinal hyper-excitability generates a subconscious motor 'guarding reflex' [9]. When the patient attempts defecation, normal rectal distension or abdominal straining is processed through this sensitized spinal circuit, preventing normal relaxation of the puborectalis muscle and EAS [9,10].

Integrated Sequence of Events

- 1. SPC within decompressed bladder → 2. Persistent catheter/ balloon mucosal & trigonal contact → 3. Urothelial mediator release & detrusor spasm → 4. Aδ and C-fibre afferent signaling → 5. Sacral spinal convergence (S2–S4) & central sensitization → 6. Vesico-anal reflex & motor guarding activation → 7. Incomplete relaxation of puborectalis & EAS → 8. Anorectal outlet obstruction with preserved colonic transit.

3. Empirical Evidence and Validation Domains

To validate the proposed hypothesis beyond theoretical reflex arcs, multi-disciplinary evidence from clinical neuro-urology, pharmacology, and pelvic floor rehabilitation is synthesized below:

Evidence Domain	Key Physiological & Clinical Evidence	Validation Impact on Hypothesis
Pharmacological CRBD Suppression	Antimuscarinics (e.g., solifenacin) and β3-adrenoreceptor agonists (mirabegron) significantly reduce CRBD severity and detrusor spasms [5].	Proves that blunting bladder afferent output reduces downstream pelvic muscle hyperactivity, predicting improved outlet relaxation.
Local Anesthetic Blockade	Intravesical instillation of lidocaine/bupivacaine suppresses C-fibre afferent activity and abolishes detrusor-sphincter hyperreflexia [5,8].	Directly proves that local mucosal sensory input drives hyperactive pelvic floor motor reflexes.
Electromyographic (EMG) Coupling	Needle and surface EMG studies demonstrate real-time co-activation of puborectalis and EAS during uninhibited detrusor contractions [7,9].	Confirms real-time reflex coupling between bladder sensory stimulation and anal sphincter tightening in humans.
Trigonal Chemodenervation	OnabotulinumtoxinA injections into the bladder trigone for overactive bladder lead to secondary reduction in resting pelvic floor tone [9].	Validates that chronic reduction in trigonal afferent/motor activity produces secondary pelvic floor relaxation.

4. Clinical Phenotype and Differential Diagnosis

The predicted clinical phenotype of SPC-associated outlet dysfunction differs fundamentally from primary slow-transit constipation or colonic hypomotility [10,11]. Because colonic propulsion remains relatively intact (as demonstrated by Buntzen et al.), stool arrives normally in the rectal vault [8].

Patients present with characteristic features of dyssynergic defecation:

- Preserved rectal sensation and urge to defecate;
- Prolonged, unsuccessful straining sessions;
- Sensation of anorectal blockage or that the outlet is 'holding shut';
- Incomplete evacuation necessitating manual evacuation, digital assistance, or frequent enemas;
- Temporal onset closely tied to SPC placement or catheter exchange [10,11].

Alternative etiologies such as opioid/anticholinergic medication, severe dehydration, rectocele, anal stenosis, metabolic disorders, and primary slow-transit constipation must be systematically excluded [10-12].

5. Recommended Clinical Diagnostic & Management Suite

To operationalize this framework in clinical practice, a step-wise protocol is recommended:

Phase 1: Catheter Optimization Protocol

Prior to invasive testing, attempt catheter adjustments: 1) Reduce balloon volume from 10 mL to 5 mL (if clinically safe) to minimize trigonal contact; 2) Ensure tension-free catheter securement using specialized leg straps; 3) Verify catheter tip position via bedside ultrasonography [5,6].

Phase 2: Physiological Assessment & Diagnostic Challenge

According to the London Classification guidelines from the International Anorectal Physiology Working Group, definitive diagnosis requires objective physiological testing:

- **High-Resolution Anorectal Manometry (HRAM):** Evaluates resting anal pressure and dyssynergic patterns (paradoxical contraction or failure of EAS/puborectalis relaxation >20% during simulated push) [12].
- **Balloon Expulsion Test (BET):** Assesses functional evacuation capability (failure to expel a 50 mL water-filled balloon within 60 seconds) [11,12].
- **Diagnostic Intravesical Lidocaine Challenge:** Instill 20 mL of 2% plain lidocaine into the catheter/bladder. Re-evaluate HRAM and BET 15–30 minutes post-instillation. A marked reduction in anal sphincter pressure or reversal of dyssynergia directly confirms the afferent-reflex mechanism.

6. Recommended Terminology

To ensure scientific precision, clinicians and researchers should adopt conservative, pathophysiologically accurate nomenclature:

- Preferred Primary Term: Suprapubic Catheter–Associated Defecatory Outlet Dysfunction (SPC-DOD)
- Descriptive Anatomical Term: SPC-Associated Pelvic-Floor Non-Relaxation
- Mechanistic Term: Afferent-Reflex Pelvic Outlet Dysfunction

Following Lower Urinary Tract Catheterisation

7. Conclusion

An indwelling suprapubic catheter in a continuously drained bladder is not biologically inert. Mechanical contact between the catheter tip or balloon and the bladder mucosa provokes sensory afferent signaling, detrusor spasms, and sacral spinal cross-talk. Through the vesico-anal reflex and central sensitization, this lower urinary tract irritation can induce involuntary pelvic floor guarding and dyssynergic defecation. Recognizing this entity prevents inappropriate treatment with aggressive oral laxatives and guides clinicians toward catheter optimization, local neuro-pharmacological modulation, and targeted pelvic floor rehabilitation.

Declarations

Author Contributions

Bruce H. Knox is the sole author and was responsible for conception of the mechanistic framework, literature synthesis, interpretation, manuscript development, critical revision, and final approval of the work.

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Consent for Publication

The author consents to publication.

Data Availability

No research dataset was generated or analysed for this manuscript. The evidence base consists of the published literature cited in the paper together with the author's scholarly synthesis and interpretation.

Medical and Interpretive Disclaimer

This manuscript presents a hypothesis-generating mechanistic framework and should not be interpreted as proof of a causal clinical entity or as individual medical advice. Proposed diagnostic or therapeutic approaches require appropriate clinical assessment, professional oversight, and empirical validation before routine use. Generative AI and Assistive Communication Disclosure ChatGPT (OpenAI) was used as an assistive communication and manuscript-preparation tool to support organisation, sentence construction, spelling, transcription, sequencing, language refinement, and conversion of the author's notes, clinical observations, conceptual material, and literature-based synthesis into academic prose. This use was intended to mitigate substantial dyslexia-related writing

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