REVIEW ARTICLE

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Bladder dysfunction after advanced pelvic surgeries: neuropelveological strategies for prevention and management

Atanas Aleksandrov[1,2], Filipa Osorio[3], Shaheen Khazali[4,5], Taner Usta[6], Nucelio Lemos[7,8,9], Benoit Rabischong[10]

[1]Department of Gynaecology, MHAT Heart and Brain, Pleven, Bulgaria
[2]Department of Department of Obstetrics and Gynaecology, Medical University - Varna Prof. Dr. Paraskev Stoyanov, Varna, Bulgaria
[3]Department of Gynecology and Obstetrics, Hospital da Luz, Lisboa, Portugal
[4]London Centre for Endometriosis and Minimally Invasive Gynaecology (CEMIG) at Hospital Corporation of America the Lister Hospital, London, United Kingdom
[5]Royal Holloway - University of London, Egham, Surrey, United Kingdom
[6]Clinic of Obstetrics and Gynecology, Acıbadem Altunizade Hospital, İstanbul, Türkiye
[7]Department of Obstetrics and Gynecology, University of Toronto, Toronto, Canada
[8]Department of Gynecology, Pelvic Neurodysfunctions Division, Federal University of São Paulo, São Paulo, Brazil
[9]Department of Gynecology, Advanced Pelvic Surgery and Neuropelveology Department, Institute for Care and Rehabilitation in Gynecology and Neuropelveology (Increasing), São Paulo, Brazil
[10]Department of Gynecological Surgery, Clermont-Ferrand University Hospital Estaing, Clermont-Ferrand, France

ABSTRACT

Advanced pelvic surgeries, such as radical hysterectomy, deep endometriosis surgery and sacrocolpopexy, pose risks to autonomic pelvic nerves leading to voiding dysfunction and reduced quality of life. This review article evaluates neuropelveological strategies for preventing and managing bladder dysfunction by exploring pelvic neural anatomy, nerve-sparing techniques, and postoperative rehabilitation approaches. Nerve-sparing approaches can reduce postoperative urinary retention and improve recovery of bladder function. Neuromodulation techniques provide additional support in managing persistent voiding dysfunction in selected cases. A multidisciplinary approach integrating detailed knowledge of pelvic neural anatomy, precise surgical techniques and structured postoperative management can minimise bladder dysfunction and optimise patient outcomes.

Keywords: Bladder dysfunction, neuropelveology, nerve-sparing surgery, neuromodulation, postoperative management

Introduction

In extensive gynaecological disorders, pelvic nerve injury can be related not only to the disease itself but also to complex surgical procedures that demand precise dissection for clear margins.[1] Lack of understanding of the intricate anatomy of pelvic neural structures further exacerbates this risk. Damage to the autonomic nerves can result in bladder, bowel, or sexual dysfunction, severely impacting the patient's

Corresponding Author: Atanas Aleksandrov, MD, Department of Gynaecology, MHAT Heart and Brain, Pleven; Department of Department of Obstetrics and Gynaecology, Medical University Pr. Paraskev Stoyanov, Varna, Bulgaria, Medical University of Prof. Dr. Paraskev Stoyanov, Varna, Bulgaria
E-mail: atanas.k.aleksandrov@gmail.com **ORCID ID:** orcid.org/0000-0001-7998-2112
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quality of life, and sometimes leading to irreversible organ dysfunction.[2] Advanced pelvic surgeries, including procedures for deep infiltrating endometriosis, radical surgery for cervical cancer, and sacrocolpopexy for pelvic organ prolapse are at the highest risk for these complications. Studies suggest that bladder dysfunction occurs in approximately 30% to 80% of patients undergoing these surgeries, depending on the type of surgery, emphasising the magnitude of the issue.[3,4]

Managing these pathologies requires a thorough understanding of pelvic anatomy, meticulous preoperative planning, adherence to the principles of neuropelveology, and effective postoperative care.[5] Among the affected organs, the bladder is particularly vulnerable. This article aims to provide an in-depth exploration of neuropelveological strategies to preserve autonomic nerves, with a focus on detailed preventive measures and comprehensive postoperative management tailored to specific surgical interventions.

Pelvic Neuroanatomy and Bladder Innervation

The pelvic autonomic nervous system integrates sympathetic, parasympathetic, and sensory fibres to regulate bladder function and maintain homeostasis of the pelvic organs. Understanding the anatomy and topography of these neural pathways is critical to minimising complications during pelvic surgeries.[6]

The superior hypogastric plexus (SHP) is the origin of sympathetic innervation to the bladder and other pelvic organs. This triangular network is formed by fibres from T11-L2, which converge from the abdominal aortic plexus, lumbar splanchnic nerves, and inferior mesenteric plexus.[7] Situated anterior to the sacral promontory at the L5-S1 level, the SHP is covered by the anterior layer of visceral pelvic fascia. The SHP gives rise to the hypogastric nerves (HNs), which descend bilaterally along the pelvic sidewalls and serve as key conduits for sympathetic fibres (Figure 1). These nerves run medial to the ureters and lateral to the mesorectum, making them vulnerable during rectal and lateral pelvic dissections.[8]

The HNs are critical structures for bladder storage function, as they facilitate detrusor relaxation and internal sphincter contraction.[9] Sensory fibres responsible for transmitting information about bladder fullness, pain, and distension also primarily travel through the HNs.[10] These sensory fibres are crucial for coordinating the storage phase of micturition, enabling the central nervous system to regulate bladder filling.[11] The HNs can be identified transperitoneally along the pelvic sidewalls, medial to the ureters (Figure 2). Due to their close proximity to the uterosacral ligaments and the presacral fascia, the HNs need to be carefully dissected during procedures at higher risk of nerve damage, such as radical hysterectomy, endometriosis excision and sacrocolpopexy.[12,13]

The pelvic splanchnic nerves (PSNs), carrying parasympathetic fibres, originate from S2-S4 spinal segments. These delicate nerves travel through the dorsocaudal pararectal space before merging with the hypogastric nerves to form the inferior hypogastric plexus (IHP) (Figure 3).[2] While their primary role is motor innervation to stimulate detrusor contraction and facilitate bladder emptying, the PSNs also carry visceral afferent fibres that contribute to the sensory pathways of pelvic organs.[15] However, in the context of bladder innervation, sensory signals are predominantly mediated by the hypogastric nerves rather than the parasympathetic system.[9]

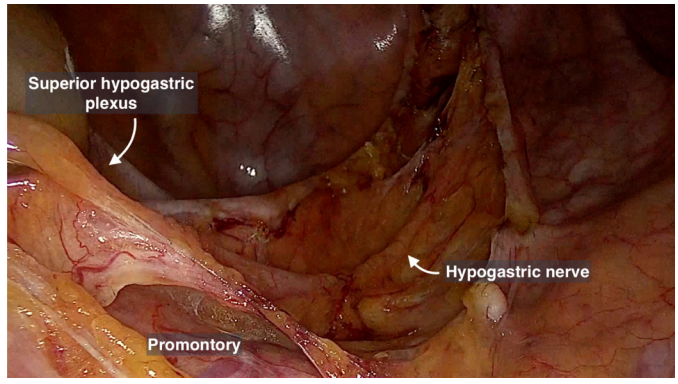

Figure 1. The superior hypogastric plexus at the level of the promontory and the hypogastric nerve completely dissected during a sacrocolpopexy.

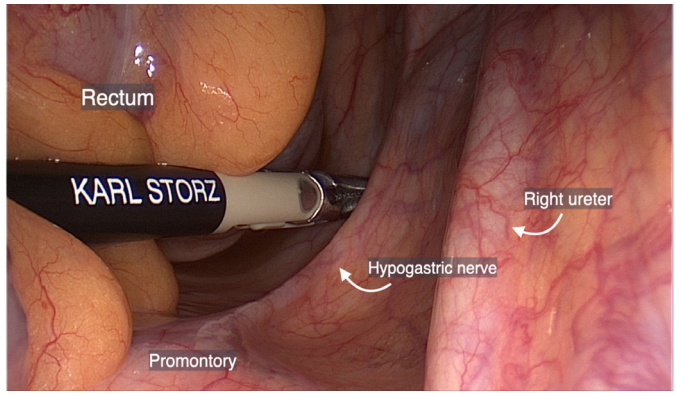

Figure 2. The hypogastric nerve seen transperitoneally at the beginning of the surgery and its relationship to the ureter.

The IHP, also referred to as the pelvic plexus, is the neurological hub for autonomic control of pelvic viscera. Approximately 2 × 2 cm in size, the IHP is located in the visceral pelvic fascia, between the anterolateral surface of the rectum and the posterolateral surface of the vaginal fornix.[16,17] Its branches, categorized as medial, cranial, and anterior efferent bundles, innervate the rectum, uterus, vagina, and bladder.[6] The vesical branches within the anterior bundle supply the bladder and urethra, playing a central role in voiding function. Preservation of the IHP during surgery is essential to avoid severe bladder dysfunction.[18]

The vesical branches of the IHP pass beneath the inferior vesical vein and run parallel to the blood vessels of the paracolpium in close proximity to the vaginal wall before reaching the bladder (Figure 4).[19] These are responsible for motor innervation to the detrusor muscle and coordination of the micturition reflex. Identification of these branches is facilitated by careful dissection of the posterior leaf of the vesicouterine ligament, where meticulous separation of the middle vesical vein and inferior vesical vein allows for the visualization and preservation of the bladder branches.

Sensory signals from the bladder are primarily transmitted via the HNs, with additional minor nociceptive contributions from the PSNs. These fibres relay critical information about bladder fullness and nociceptive stimuli to the central nervous system, enabling proper coordination of the micturition reflex.[10] The integration of sensory and motor inputs ensures the delicate balance required for bladder function. The somatic innervation, mediated by the pudendal nerve, controls the external urethral sphincter, ensuring voluntary control over urination.[9]

These interconnected plexuses and their efferent branches represent a delicate balance of motor, sensory, and autonomic functions, and nerve-sparing approaches and intraoperative neuro-navigation are pivotal in preserving these structures and optimising functional outcomes.[20] Damage to any component of this network can lead to bladder storage and voiding dysfunctions, emphasising the need for meticulous surgical planning and execution.[21]

Radical Hysterectomy (Cervical Cancer Surgery)

Radical hysterectomy is among the most challenging pelvic surgeries and demands meticulous dissection of the parametrial and paracervical regions.[19] While this

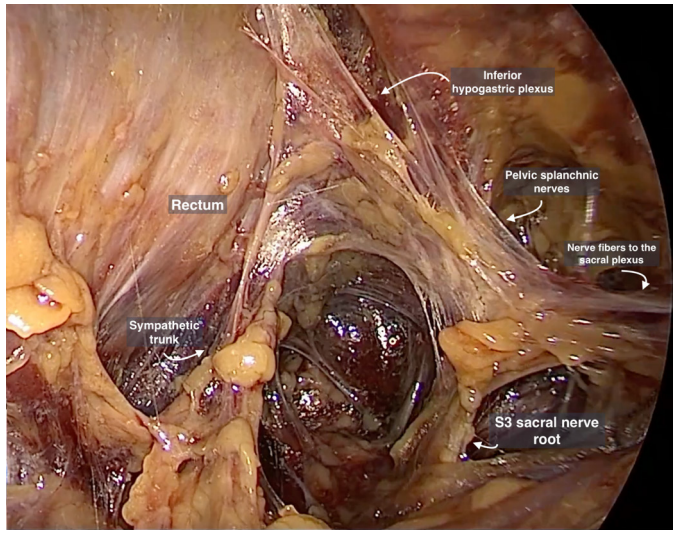

Figure 3. The pelvic splanchnic nerves arise from S2-S2 sacral nerve roots and merge with the hypogastric nerves and the sympathetic trunks on both sides of the rectum to form the inferior hypogastric plexus. Cadaveric dissection, ESGE Annual Congress 2022.

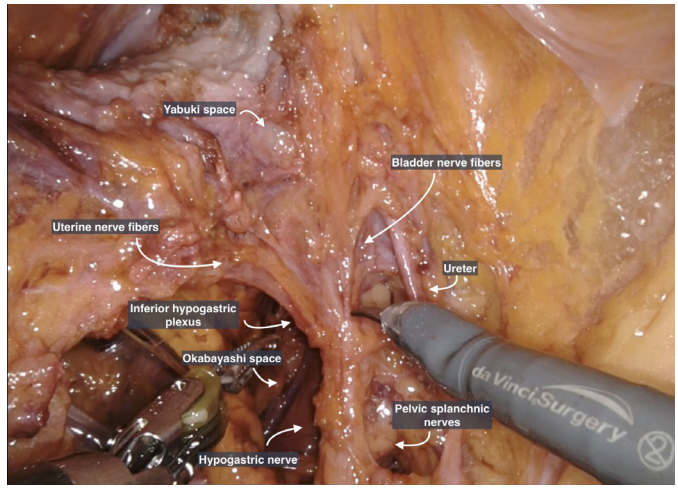

Figure 4. The bladder branches from the inferior hypogastric plexus run in close proximity to the vaginal wall and are prone to injury in extensive vaginal excisions. Cadaveric dissection, ESGE Annual Congress 2023.

procedure is effective in achieving oncological control, it carries significant risks to the autonomic nerves critical for bladder function, with patients presenting voiding dysfunction in up to 80% of cases.[22] The most vulnerable structures during radical hysterectomy include the HNs and the IHP, mainly, its vesical branches which run in the lateral parametrium.[23]

Dissection of the dorsal parametrium poses a particular risk to the HNs, which are essential for bladder storage. These nerves facilitate detrusor relaxation and contribute

to the tone of the internal urethral sphincter. Damage to these nerves can result in stress urinary incontinence due to decreased sphincter tone, as well as bladder overactivity caused by parasympathetic dominance.[24,25] Maneschi et al.[26] have demonstrated that injury to the HNs during parametrial dissection often leads to decreased bladder compliance and altered sensitivity to fullness, resulting in symptoms such as urgency and incomplete voiding.

The vesicouterine ligament is another critical site. This structure houses the vesical branches of the IHP, which provide motor innervation to the detrusor muscle. Resection of this ligament can cause denervation of the bladder, leading to detrusor atony and voiding dysfunction.[19] Plotti et al.[25] emphasize that damage to these branches not only impairs bladder emptying but also increases the risk of chronic urinary retention and associated complications, such as recurrent infections.

Early bladder complications after radical hysterectomy primarily manifest within the first 3-6 months and are characterized by parasympathetic dominance.[23] This results in a hypertonic bladder with low compliance and reduced capacity, affecting up to 85% of patients.[22] Additionally, some patients may experience loss of bladder sensation and urinary retention, with a residual urine volume exceeding normal limits in 10-15% of cases. If unmanaged, this can lead to irreversible changes such as detrusor hypertrophy and myogenic damage.

Late bladder complications, occurring beyond 6 months post-surgery, often stem from incomplete nerve recovery or aberrant nerve regeneration.[18] These include chronic urinary retention, overactive bladder symptoms, and stress urinary incontinence due to loss of internal sphincter tone. Reduced maximal urethral closure pressure, as reported in up to 40% of patients, exacerbates the risk of incontinence.[3] Inadequate management of early complications can predispose patients to long-term sequelae, such as bladder fibrosis and permanent detrusor dysfunction.[27]

Deep Endometriosis Surgery

Deep endometriosis (DE) poses significant challenges, as infiltration around pelvic autonomic nerves is common and leads to severe anatomical distortion. Bladder dysfunction following DE surgery arises through two primary mechanisms: direct infiltration of the nerve fibres by endometriotic lesions and collateral damage caused by surgical dissection or resection.[1,28] These surgeries aim for complete removal of diseased tissue but are fraught with risks to autonomic nerve integrity.

The localization and extent of endometriotic nodules play a critical role in determining the risk of postoperative bladder dysfunction. According to Boulus et al.,[4] urinary retention occurs in up to 30% of patients following DE surgery, with approximately 83% regaining normal voiding function within 18 months, while 17% may suffer from persistent dysfunction due to irreversible nerve damage. The risk strongly correlates with the proximity of lesions to autonomic structures such as the PSNs and the IHP.

Parametrial infiltration significantly increases the likelihood of autonomic nerve injury. Posterior DE nodules involving the rectovaginal septum, uterosacral ligaments, and the parametrium have been associated with a higher incidence of postoperative voiding difficulties.[29,30] Imboden et al.[31] also identified that ENZIAN B lesions larger than 3 cm significantly increase the risk of bladder dysfunction, likely due to their impact on the IHP and PSNs.These findings highlight the necessity of targeted nerve-sparing techniques when excising parametrial disease.

The need for colorectal resection further compounds the risk. Roman et al.[28] and Ballester et al.[32] showed that segmental colorectal resections—especially when combined with bilateral uterosacral ligament excision—lead to higher rates of postoperative neuropathy and prolonged bladder rehabilitation. Similarly, Landi et al.[33] found that even unilateral parametrectomy may cause bladder dysfunction, reinforcing the vulnerability of autonomic pathways. Dubernard et al.[34] demonstrated that extensive resections involving the uterosacral ligaments or ischial spine correlate with increased rates of intermittent self-catheterization (ISC).

Bladder dysfunction after DE surgery typically arises from either reversible neuropraxia or irreversible axonal injury. Neuropraxia, due to inflammation or thermal injury, preserves axonal continuity and often resolves over time. This explains the relatively high recovery rates observed in studies such as those by Boulus et al.[4] and Gabriel et al.[35]. In contrast, direct nerve transection during extensive parametrial or colorectal dissection may lead to permanent dysfunction.[36] Kovoor et al.[37] noted that transient urinary retention usually resolves within 5–7 days, while long-term retention—observed in 4–10% of cases—is more common after bilateral USL excisions or bowel resections.

It is also important to recognize that patients with endometriosis may present with lower urinary tract symptoms that are preexisting and attributable to the disease itself, rather than being solely iatrogenic in origin. Preoperative bladder dysfunction caused by nerve damage and infiltration by the endometriosis strongly predicts postoperative outcomes. According to Boulus et al.,[4] patients with preexisting nerve damage are more likely to experience persistent bladder voiding dysfunction postoperatively. This correlation underscores the importance of thorough preoperative assessments, including urodynamic testing and pelvic imaging, to identify patients at higher risk. Ballester et al.[32] emphasized that preoperative urodynamic testing provides valuable insights into baseline bladder function, helping to anticipate the likelihood of postoperative dysfunction and guide surgical planning. Similarly, Panel et al.[38] found that lower urinary tract symptoms identified preoperatively due to nerve damage caused by the disease in patients with DE strongly correlated with postoperative outcomes, indicating that early recognition of voiding disturbances allows for better perioperative counselling and tailored postoperative management. These findings highlight the need for comprehensive preoperative evaluation to mitigate long-term bladder dysfunction following DE surgery.

Sacrocolpopexy for Pelvic Organ Prolapse

Abdominal and minimally invasive sacrocolpopexy remain the gold standards for managing pelvic organ prolapse.[39] However, these procedures are associated with a significant risk of nerve injury, particularly during promontory dissection and vesicouterine ligament manipulation. Injuries to the SHP, HNs, and vesical branches of the IHP can result in debilitating bladder and bowel dysfunction.

Dissection at the sacral promontory poses a high risk to the SHP and HNs. According to Ercoli et al.,[40] the use of an inverted "L-shaped" peritoneal incision, medial to the right common iliac artery, minimizes nerve injury during promontory dissection. They emphasize that the presacral fascia must be carefully medialised to preserve the HNs and prevent iatrogenic denervation. [40] Cosma et al.[41] further demonstrated that nerve-sparing sacrocolpopexy significantly reduces the incidence of postoperative voiding dysfunction compared to the standard technique.

The vesical branches within the vesicouterine ligament are particularly vulnerable during anterior vaginal wall fixation. Shiozawa et al.[42] highlighted that precise delineation of the vesicouterine ligament and identification of the anterior longitudinal ligament during dissection can mitigate this risk. Preservation of these branches is crucial to maintaining bladder contractility and avoiding postoperative catheter dependency.

Nerve-sparing Techniques and Preventive Strategies

Nerve-sparing techniques have become a cornerstone in minimising autonomic nerve damage during pelvic surgeries. These approaches require a deep understanding of pelvic anatomy and surgical precision, emphasising the preservation of key autonomic structures while ensuring oncological and functional outcomes.

The concept of nerve-sparing was first introduced in oncological surgery, particularly in the context of radical hysterectomy, to reduce the significant morbidity associated with pelvic organ dysfunction.[43,19] The benefits of nerve-sparing approaches have been substantiated by numerous studies. Magrina et al.[44] demonstrated that patients undergoing nerve-sparing radical hysterectomy experienced significantly lower rates of urinary dysfunction without compromising oncological outcomes. Similarly, Kavallaris et al.[45] reported improved bladder function recovery in cases where precise nerve preservation techniques were employed during cervical cancer surgeries. According to Possover et al.,[46] laparoscopic exposure and precise dissection of PSNs markedly improved postoperative bladder function, significantly reducing dysfunction rates. Their findings highlighted the importance of meticulous surgical planning and real-time nerve identification in minimising complications, with reported rates of severe dysfunction being significantly low among patients undergoing radical pelvic surgeries. This approach underscores the critical role of nerve-sparing techniques in optimising both oncological and functional outcomes.

Over time, these principles have been adapted to other complex pelvic surgeries, including procedures for deep infiltrating endometriosis and prolapse surgery, to optimize outcomes and improve quality of life. Volpi et al.[47] was among the first to describe the identification and sparing of the nervous structures during DE surgery, with further advancements by Landi et al.[48] and Ceccaroni et al.,[13] who standardized the nerve-sparing technique for DE

excision. Their findings consistently demonstrated shorter catheterization times, lower rates of urinary retention, and better functional outcomes in nerve-sparing groups compared to standard techniques. Despite the benefits, nerve-sparing may not always be feasible due to disease infiltration, necessitating individualized surgical planning to balance functional preservation with disease control. According to Roman and Darwish,[1] bilateral involvement of critical structures, such as the uterosacral ligaments, significantly increases the likelihood of autonomic nerve damage, particularly to the IHP. This can result in profound bladder dysfunction, including detrusor atony and chronic urinary retention. They emphasized that preoperative imaging and advanced laparoscopic techniques are pivotal in assessing the extent of infiltration and guiding a nerve-sparing approach, even in complex cases. By employing precise dissection within avascular planes and preserving neural pathways where possible, the risk of severe postoperative bladder dysfunction can be minimized.

Emerging technologies such as artificial intelligence and augmented reality are providing new opportunities for enhancing pelvic nerve preservation. According to Kinoshita et al.,[49] Artificial intelligence (AI)-based nerve recognition models have demonstrated significant potential in improving intraoperative nerve identification. Their deep learning algorithm, trained on surgical videos, enhances surgeons' ability to accurately recognize autonomic nerves in real-time, reducing the risk of inadvertent nerve damage. Additionally, AI-assisted surgical education has shown improved learning outcomes for junior surgeons by enhancing nerve recognition skills through visual analysis, further supporting the integration of AI in nerve-sparing strategies.

The fundamental principle of nerve-sparing surgery is to minimize unnecessary nerve dissection, handling, and manipulation to prevent potential nerve damage. These techniques should be employed only when the nerves are directly affected by the disease, ensuring that interventions are justified and beneficial. It is strongly advised against performing unnecessary nerve dissection, as preserving nerve integrity is essential for maintaining postoperative function. Studies have emphasized that systematic full surgical dissection of pelvic nerves is not required unless there is direct nerve entrapment, reinforcing the importance of a tailored approach in complex gynaecological procedures. Research further supports that unnecessary nerve handling can increase the risk of postoperative dysfunction, making precise surgical planning crucial. A simplified approach to nerve-sparing using the HNs as landmarks has been proposed[50] to reduce the amount of nerve dissection while still providing the advantage for the direct visualization proposed by Possover.[46] These principles align with the article of Aleksandrov et al.[5] and the Strasbourg consensus by Wattiez et al.,[51] which stress that full nerve dissection should only be undertaken when absolutely necessary to preserve function and optimize patient outcomes.

The successful application of nerve-sparing strategies in pelvic surgeries relies on a combination of advanced anatomical knowledge, precise surgical techniques, and the integration of innovative tools such as neuro-navigation and artificial intelligence. These approaches facilitate real-time nerve identification, minimising intraoperative damage and optimising functional recovery. By prioritising nerve preservation, these methods contribute to improved patient outcomes, reduced postoperative complications, and an overall better quality of life.

Postoperative Management of Voiding Dysfunction

The management of voiding dysfunction following neurological damage during pelvic surgeries is crucial to restoring normal voiding function and preventing long-term bladder complications. Early identification and management of voiding dysfunction is critical. Clear, structured protocols for catheter removal and trial without catheter (TWOC) should be implemented postoperatively. While often implied, this step must be explicitly recognized as one of the most effective and straightforward strategies to ensure timely diagnosis and prevent long-term complications.

TWOC is a standardized protocol used to assess a patient's ability to void spontaneously after catheter removal. It typically involves removing the indwelling catheter 24–72 hours postoperatively, depending on the extent of nerve dissection, followed by close monitoring of spontaneous urination and post-void residual (PVR) volume measured with bladder ultrasound or catheterization. A PVR less than 100 mL is generally considered a successful trial. Higher residuals may necessitate ISC or reinsertion of the catheter. The implementation of a TWOC protocol enables early detection of voiding impairment, prevention of bladder overdistension, and timely initiation of rehabilitative strategies.

Patients with a post-void residual volume exceeding 100 mL are typically advised to perform ISC four to six times daily to prevent bladder overdistension and maintain low residual volumes.[4,52] Research, including studies by Boulus et al.[4] and de Resende et al.,[36] demonstrates that short-term bladder dysfunction is often attributed to neuropraxia caused by surgical manipulation, dissection, or thermal injury to the nerves. Neuropraxia generally resolves within 12-18 months when managed appropriately. To minimize unnecessary damage, excessive handling of or dissection near neural structures should be avoided, and neurosurgical principles should guide surgical interventions near delicate nerve pathways.[5]

In addition to ISC, pharmacological interventions such as alpha-adrenergic blockers are effective in reducing hypertonicity of the internal urethral sphincter, facilitating voiding, and alleviating retention.[4] Possover has highlighted the importance of addressing detrusor-sphincter dyssynergia, a common consequence of nerve damage, with pharmacological and supportive therapies.[2]

Electrostimulation therapy targeting the sacral or tibial nerves provides additional support in managing bladder dysfunction. Tibial nerve stimulation offers retrograde activation of sacral plexus pathways governing bladder function.[53] This can be achieved using percutaneous needle electrodes, transcutaneous surface electrodes, or wireless implantable devices. In the study by Boulus et al.,[4] all patients with bladder dysfunction were discharged with a transcutaneous tibial nerve electrostimulation device to stimulate the splanchnic nerves and promote recovery of voiding function. Regular follow-up assessments, including urodynamic studies and bladder ultrasounds, are essential for tracking recovery and tailoring therapeutic strategies. These evaluations guide decisions regarding the continuation or cessation of supportive treatments, ensuring that management aligns with individual patient needs.

Sacral Nerve Stimulation

Sacral nerve stimulation (SNS) involves the implantation of electrodes to deliver mild electrical pulses to the sacral nerves, particularly the S3 segment, to regulate bladder and anal sphincter activity. This procedure comprises a two-stage process: the initial phase involves the percutaneous insertion of a temporary electrode wire through the S3 sacral foramen to evaluate efficacy, followed by the second stage in which the electrode is connected to a permanent implantable pulse generator.[54] SNS has demonstrated success in alleviating symptoms of urinary incontinence, urgency, and retention in up to 70% of treated patients.[55] Aublé et al.'s[56] retrospective analysis of SNS for voiding dysfunction following endometriosis surgery revealed that 60% of patients experienced significant improvements in bladder function and quality of life after a median follow-up of 55 months. More than half of those who required clean ISC (CISC) before SNS were able to discontinue CISC post-treatment. However, complications such as infections, paraesthesia, and electrode migration were noted, highlighting the importance of careful patient selection and follow-up.

Agnello et al.[57] similarly reported that 69.2% of patients undergoing SNS following deep infiltrating endometriosis DE surgery demonstrated significant improvements in symptoms, including bladder sensitivity and emptying.[57] The study emphasized that SNS not only improves voiding efficiency but also reduces the need for daily catheterizations, making it a promising option for managing persistent voiding dysfunction.

The LION procedure, pioneered by Possover, provides a targeted approach by directly stimulating pelvic nerves, such as the SHP or pudendal nerve.[14,58] Unlike SNS, which modulates sacral nerves indirectly, the LION procedure delivers focused neuromodulation through laparoscopically placed electrodes adjacent to the target nerves. The nervous structures that could be reached and stimulated using the LION procedure are the sacral nerve roots S2-S4, which play a role in the treatment of bladder and bowel dysfunction after nerve injury, the SHP, in cases of bladder atonia secondary to radical pelvic surgery the pudendal nerve, stimulation of which might help patients with bladder or faecal incontinence, or the sciatic or lumbar nerves L1-L5, which might play a role in management of chronic pain or motor dysfunction. Possover's studies have demonstrated the efficacy of the LION procedure in treating refractory bladder atonia, showing significant improvements in voiding function and reductions in complications related to urinary retention.[59] This is a more invasive and complex procedure than SNS, which could be considered in select cases where SNS has failed.

Importance of Adequate Follow-up in Preventing Chronic Morbidity

The long-term outcomes of bladder dysfunction are closely tied to the quality of postoperative follow-up.

As detailed by Possover, inadequate follow-up can lead to insidious and irreversible complications.[2] Among the 47 patients evaluated in his study for bladder retention following DE surgeries, those without consistent postoperative monitoring often presented with advanced neurogenic bladder conditions, including chronic atony and severe detrusor dysfunction years after the surgery.

Failure to identify early signs of sensory or motor deficits with flow deterioration on postoperative uroflowmetry or clinical red flags such as reduced urinary frequency or the need for Valsalva manoeuvres to complete emptying, allows for progressive bladder overdistension. This can result in secondary myogenic damage, reduced elasticity, and eventual loss of detrusor contractility. Possover emphasized that regular assessments of post-void residual volumes and bladder sensation, paired with timely interventions like ISC, are critical to preventing such outcomes.

Without proactive management, complications such as recurrent urinary tract infections, overflow incontinence, and even renal impairment due to bilateral ureterohydronephrosis may develop. The findings from Possover's study underline the necessity for structured follow-up protocols focusing not only on voiding function but also on sensory and neurological assessments of the bladder. This approach ensures early detection of dysfunctions, timely interventions, and preservation of long-term bladder health.

Conclusion

Bladder dysfunction following advanced pelvic surgeries remains a significant challenge that can greatly affect a patient's quality of life. A comprehensive approach incorporating meticulous surgical planning, a profound understanding of pelvic neural anatomy, and the implementation of nerve-sparing techniques are essential to minimize the risk of autonomic nerve injury. Avoiding unnecessary nerve dissection and manipulation is paramount, as excessive handling can result in irreversible damage and long-term functional impairment.

A tailored surgical approach, guided by preoperative imaging and intraoperative neuro-navigation, allows for precise dissection while preserving essential nerve structures. When disease infiltration creates the need for nerve resection, selective dissection should be employed to balance oncological control with functional preservation. Emerging technologies such as artificial intelligence and augmented reality further enhance intraoperative nerve identification, reducing the risk of inadvertent damage and improving surgical outcomes.

Beyond intraoperative strategies, postoperative management plays a crucial role in bladder function recovery. Multimodal rehabilitation, including pharmacological therapies, electrostimulation, and neuromodulation techniques, is essential for restoring autonomic control and preventing long-term complications such as detrusor hypotonia or atonia or chronic urinary retention. Early and structured follow-up, incorporating urodynamic and uroflowmetry assessments and targeted interventions, is critical in identifying dysfunction at an early stage and preventing irreversible sequelae.

Ultimately, optimising patient outcomes in pelvic surgery requires a multidisciplinary effort that integrates advanced surgical techniques, technological innovations, and individualized patient care strategies. By prioritising nerve preservation and ensuring meticulous postoperative management, the risk of bladder dysfunction can be significantly mitigated, leading to improved patient recovery, functional outcomes, and overall quality of life.

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