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DISCOVERY SCIENTIFIC SOCIETY

# The impact of diabetic neuropathy on the function of the bladder and the urethral sphincter

**Justyna Gręda[1]*, Bartosz Zieliński[1], Karol Mateusz Wojnarowski[1]**

## ABSTRACT

Introduction: Diabetic neuropathy, its autonomic forms, is a common complication of longstanding diabetes mellitus. It has profound impacts on lower urinary tract function, affecting both the bladder and the urethral sphincter, and frequently results in significant morbidity known as diabetic cystopathy or neurogenic bladder. Results: Patients with diabetic neuropathy commonly experience elevations in postvoid residual urine volumes, in addition to reductions in detrusor muscle contractility and decreased bladder sensation. For example, these alterations can lead to signs such as urinary retention. Other signs include recurrent urinary tract infections plus overflow incontinence. Urgency and frequency may present during the early stages, while a large atonic bladder, unable to empty effectively, reveals itself in later stages. The urethral sphincter acts for itself and comes across as dyssynergia. It also causes muscle weakness, leading to impaired coordination. These signs additionally aggravate micturition problems and incontinence. These dysfunctions are due to neuropathic changes that affect both sensory and motor innervation, as well as reduced nerve growth factor and autonomic dysregulation. Conclusions: Diabetic neuropathy significantly impairs both usual bladder function and urethral sphincter function, as detailed neurogenic processes contribute to lower urinary tract symptoms. Preventing urinary complications as well as preserving quality of life in diabetic patients requires managing by identifying early.

**Keywords:** diabetic neuropathy, bladder dysfunction, urethral sphincter, neurogenic bladder

## 1. INTRODUCTION

Diabetic neuropathy represents a frequent and crippling sequel of diabetes mellitus that features advancing nerve injury caused by both sustained hyperglycemia and its ensuing metabolic and vascular consequences. A diverse range of neuropathic syndromes is included in it. It is fairly thorough. These syndromes primarily affect the outer somatic nervous system. The syndromes also aim at the autonomic nervous system (Strand et al., 2024; Zaino et al., 2023). If the lower urinary tract has dysfunction, it affects the bladder, as diabetic cystopathy primarily impacts the

bladder, affecting both the storage and voiding phases (Powell & Gehring, 2023; Agochukwu-Mmonu et al., 2020).

The bladder and urethral sphincter operate under complex neurophysiological control involving the somatic and autonomic components of the nervous system. Autonomic neuropathy in diabetes compromises both the sympathetic and parasympathetic innervations that are important for sphincter relaxation/contraction and detrusor muscle contractility, while somatic nerve injury also weakens external sphincter control (Kumar et al., 2022; Panicker, 2020). These processes result in lower urinary tract symptoms, such as incontinence and recurrent infections. The symptoms also include increased residual volumes along with urinary retention with neuromuscular functions synchronized, then impaired, which are important for urine storage as well as voiding (Song et al., 2022; Quast et al., 2023).

Many factors contribute to nerve damage in diabetes, as metabolic, oxidative, inflammatory, and ischemic actions are involved, leading to nerve fiber loss and dysregulation of neurotrophic support (Eid et al., 2023; Callaghan et al., 2020). The studies do show us that hyperglycemia-induced oxidative stress promotes mitochondrial dysfunction along with the accumulation of advanced glycation end-products (AGEs). The affected nerves are those that play a critical role in afferent bladder signaling (Strand et al., 2024; Powell & Gehring, 2023), predominantly comprising small-fibre sensory nerves.

From a neuroanatomical perspective, it appears that diabetes primarily affects the nervous system through the pelvic plexus, dorsal root ganglia, and peripheral nerves. The pelvic, hypogastric, and pudendal nerves may each play some role in innervating the lower urinary tract. The pelvic (parasympathetic), hypogastric (sympathetic), and pudendal (somatic) nerves are in this group (Kumar et al., 2022; Sasaki et al., 2020). It has been suggested that damage to the parasympathetic fibres may contribute to a reduction in detrusor contractility. Some studies have indicated that sympathetic dysfunction could be involved in bladder filling by affecting the relaxation of the detrusor muscle and the contraction of the internal sphincter (Agochukwu-Mmonu et al., 2020; Cao et al., 2020). At the same time, neuropathy affecting the pudendal nerve can result in weakness or incoordination of the external urethral sphincter, which can contribute to incontinence or obstructive voiding symptoms (Kumar et al., 2022; Panicker, 2020).

Experimental evidence underscores the role of oxidative stress in the pathogenesis of diabetic bladder dysfunction. Research on diabetic rats has shown that the activity of nitric oxide synthase plays an important role in how nitric oxide helps relax smooth muscle in the urethra. These findings help us understand the molecular pathways that lead to sphincter dysfunction (Cao et al., 2020). Human diabetic cystopathy presents clinical manifestations similar to these neurochemical disruptions. This condition involves sensory afferent loss and motor efferent impairments.

Clinically, diabetic bladder dysfunction has a variable phenotype reflecting the stage and severity of neuropathic injury. Early in the disease course, patients may experience irritative symptoms such as urgency and frequency caused by partial denervation and compensatory detrusor overactivity (Powell & Gehring, 2023). However, a neuropathy progression is a typical pattern characterized by diminished bladder sensation, increased bladder capacity, decreased detrusor contractility, and increased post-void residual volume—an atonic or hypotonic bladder (Eid et al., 2023; Sasaki et al., 2020).

The urethral sphincter is likewise affected during diabetic neuropathy. Smooth muscle, as well as striated muscle, make up the external and internal sphincters. Functional obstruction or incompetence can occur if the internal sphincter relaxes in an impaired way while the external sphincter is weak or uncoordinated. This causes stress incontinence or urinary retention. Kumar et al., (2022) and Panicker (2020) certainly discuss these respective contributions. Symptoms such as hesitancy and diminished urinary flow may manifest. Also, bladder emptying may still be incomplete. This elevates the threat of urinary tract infections and upper urinary tract problems (Agochukwu-Mmonu et al., 2020; Quast et al., 2023).

Notably, autonomic neuropathy is a key driver of these urological complications and is strongly associated with cardiovascular, gastrointestinal, and sudomotor dysfunctions. This emphasizes the systemic nature of diabetic autonomic involvement (Song et al., 2022; Quast et al., 2023). Autonomic neuropathy does indicate a greater chance of severe diabetic cystopathy. Therefore, thorough screening, along with multidisciplinary management, is needed.

Diabetic neuropathy in the lower urinary tract is often overlooked and inadequately treated, despite its significant impact on quality of life and its prevalence (Callaghan et al., 2020; Panicker, 2020). Subtle voiding dysfunctions and early sensory deficits are often overlooked in standard clinical assessments. Recent advances in urodynamic testing and neurophysiological evaluations, combined with biomarker development, offer earlier and more accurate diagnoses (Powell & Gehring, 2023; Panicker, 2020).

Recent research has focused on elucidating all the molecular pathways involved in nerve injury. In diabetic neuropathy, these pathways also contribute to the repair process. This research examined all the roles of inflammation. Inflammation impacts microvascular endothelial dysfunction plus neurotrophic factor signaling (Eid et al., 2023; Strand et al., 2024). Within these perceptions

are therapeutic implications. Prevention or reversal of bladder and sphincter dysfunction suggests that these strategies may be for nerve protection. The present leaders are still helpful (Zaino et al., 2023; Panicker, 2020). It highlights better glycemic control in addition to training the bladder then utilizes pharmacotherapy so sometimes catheterization is necessary. The researchers are looking into some new treatments that have agents for the adjustment of oxidative stress, along with NGF mimetics and regenerative ways that use stem cells (Eid et al., 2023; Rosenberger et al., 2020). To summarize, diabetic neuropathy deeply and multi-facetedly affects bladder and urethral sphincter function as sensory afferents are lost, motor efferents are dysfunctional, and autonomic balance is disrupted.

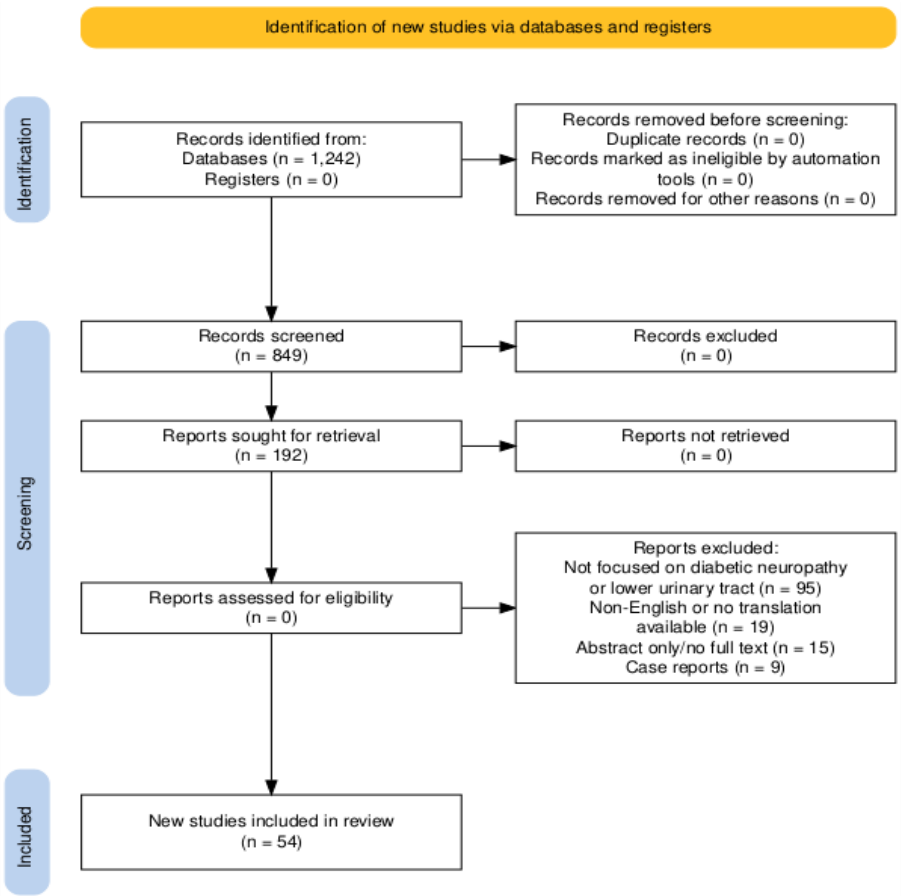


**Figure 1**. PRISMA flowchart

## 2. REVIEW METHODS

This review was conducted using a systematic approach, following the PRISMA guidelines. The PubMed, Google Scholar, Web of Science, and Scopus databases were thoroughly searched to find relevant literature. To provide context from applicable earlier studies when referenced, the search period was set from January 2018 to May 2025, capturing the most recent developments.

### Search Strategy

Databases searched included PubMed, Scopus, Web of Science, and EMBASE. Diabetic neuropathy, bladder dysfunction, urethral sphincter, and neurogenic bladder were keywords. These keywords drove the search strategy. Original research articles, systematic reviews, meta-analyses, and high-quality narrative reviews address the effects of diabetic neuropathy on bladder and urethral sphincter function, as well as related neurogenic mechanisms.

Clinical studies (observational and interventional alike) have human subjects with diabetes mellitus (either type 1 or type 2) experiencing urinary dysfunction since neuropathy causes it. Pathophysiological mechanisms were elucidated via preclinical studies. These mechanisms are relevant for both diabetic bladder and urethral dysfunction. From 2018 onward, articles were published in peer-reviewed journals, complementing earlier seminal studies for a mechanistic context or explanation.

**Exclusion Criteria**

Articles not focused on diabetic neuropathy or lower urinary tract involvement, non-English language publications lacking translation, abstracts without available full text, case reports with fewer than five subjects, and studies lacking clear diagnostic criteria or methodology.

**Screening**

A total of 1,242 references were identified from database searches and relevant grey literature. After automatic and manual deduplication, 849 unique studies remained. Two senior authors independently reviewed titles and abstracts for potential relevance to diabetic neuropathy, bladder, and urethral dysfunction. Of these, 192 full-text papers were reviewed in detail to determine eligibility based on inclusion and exclusion criteria, as well as methodological criteria. Ultimately, 54 high-quality studies were included in the final synthesis, as they represented the most relevant and most substantial evidence addressing the impact of diabetic neuropathy on bladder and urethral sphincter function. Methodological transparency is ensured through the use of the PRISMA flow diagram (Figure 1).

## 3. RESULTS & DISCUSSION

A significant amount of research has been published over the last few years, particularly from January 2018 to May 2025. This research, encompassing both studies and systematic reviews, consistently demonstrates the significant and multifaceted effects of diabetic neuropathy on the bladder and urethral sphincter (Strand et al., 2024; Powell & Gehring, 2023). A substantial body of epidemiological data from large hospital-based cohorts, such as that presented by Wang et al., (2023), indicates a high prevalence of bladder dysfunction in diabetic populations. Risk factors for this condition include advanced age, female sex, chronic kidney disease, subcutaneous insulin therapy, prior urinary tract infections, and high post-void residual urine volumes, all of which are associated with poorer outcomes.

From a mechanistic perspective, advances in molecular and experimental studies have elucidated the critical role of oxidative stress, decreased nerve growth factor (NGF) expression, mitochondrial dysfunction and microvascular ischaemia in the progressive damage to the autonomic and somatic nerves that innervate the lower urinary tract (Eid et al., 2023; Song et al., 2022; Zaino et al., 2023). Animal models of streptozotocin-induced diabetes display damaged nitric oxide pathways plus urethral sphincter complex smooth muscle issues, contributing directly to incontinence and urinary retention (Cao et al., 2020).

Clinically, diabetic bladder issues show LUTS as a varied range of lower urinary tract symptoms. The symptoms change in stages based on just how bad diabetic neuropathy is. How symptoms change is also reliant on how long the diabetic neuropathy lasts. This first stage is believed to cause some denervation in addition to responsive detrusor smooth muscle overactivity, since afferent sensory fibers start to break down, and efferent control gets disrupted (Powell & Gehring, 2023; Agochukwu-Mmonu et al., 2020). The hypothesis that the loss of inhibitory neural input may facilitate uninhibited bladder contractions has been postulated. The irritative symptoms that often prompt clinical evaluation are a consequence of this process.

As the neuropathic damage progresses, there is a transition toward detrusor underactivity, with the bladder evolving into an atonic, poorly contractile organ exhibiting increased capacity and diminished sensation of fullness. Patients in this late stage typically report hesitancy, weak or intermittent urinary stream, sensations of incomplete emptying, and elevated postvoid residual volumes, which predispose to urinary tract infections and upper urinary tract complications (Powell & Gehring, 2023; Agochukwu-Mmonu et al., 2020). That progression reflects the cumulative impairment of parasympathetic efferent fibers that mediate detrusor contraction, along with sensory afferent loss, which blunts bladder sensation and disrupts the standard voiding reflex arc.

Diabetic bladder dysfunction is clinically characterized by a wide range of lower urinary tract symptoms (LUTS) that get worse over time with the severity and duration of diabetic neuropathy. It is clear that at the onset of the disease, patients present with detrusor overactivity. This is characterized by increased urinary urgency, frequency, and, on occasion, urge incontinence. This initial phase is caused by partial denervation and compensatory hyperactivity of the detrusor smooth muscle. This is because afferent sensory fibres begin to degenerate and efferent control becomes dysregulated (Powell & Gehring, 2023; Agochukwu-Mmonu et al., 2020). The hypothesis that the loss of inhibitory neural input may facilitate uninhibited bladder contractions is correct. The irritative symptoms that frequently necessitate clinical evaluation are a consequence of this process.

Neuropathic damage causes a transition towards detrusor underactivity. This results in the bladder becoming an atonic and poorly contractile organ. It exhibits increased capacity and diminished sensation of fullness. In patients presenting with a late stage of the

condition, symptoms include hesitancy, a weak or intermittent urinary stream, straining to void, sensations of incomplete emptying, and elevated postvoid residual volumes. These symptoms increase the risk of urinary tract infections in patients. Upper urinary tract complications can also be caused by these symptoms (Powell & Gehring, 2023; Agochukwu-Mmonu et al., 2020). This progression is clear evidence of cumulative impairment of parasympathetic efferent fibres. These fibres are responsible for mediating detrusor contraction, in conjunction with sensory afferent loss. The latter is known to blunt bladder sensation, thus disrupting the standard voiding reflex arc.

Research examining autonomic neuropathy has produced large evidence because it reveals a link between sensory and motor nerve fibres and how severe lower urinary tract symptoms are in diabetic patients. Studies have shown that the pelvic plexus, pudendal nerve, and dorsal root ganglia influence nerves throughout the body and control various bodily functions. This plainly occurring explains about the fundamental causes that enormously diversify symptoms appearing. Difficulties in urination as well as incontinence may be symptoms. Impaired micturition can also be a symptom (Quast et al., 2023). The links clearly show that issues pertaining to the bladder in individuals diagnosed with diabetes are attributable to complications because both the nervous and vascular systems of the body are involved.

It is necessary to see that signs vary a lot between people. Additional issues, like blindness, kidney disease, and nerve damage, can complicate the situation (Sasaki et al., 2020). An inactive bladder can harm the kidneys, and some patients may have symptoms similar to an overactive bladder. This shows that we should treat each person as an individual and create a personalized plan for diagnosis and treatment. The problem is often not diagnosed properly because there are so many different types of it and it develops slowly. This suggests that patients may be reluctant to share mild or early symptoms, and doctors may not spot the neuropathic cause without specific tests (Powell & Gehring, 2023; Panicker, 2020).

Better ways to diagnose it were developed, such as complex bladder testing along with nerve evaluations, which ease the process of identifying and understanding more instances of diabetic neurogenic bladder. Minor sensory problems and motor dysfunction are spotted by clinicians. They can use the methods before ominous signs show (Panicker, 2020; Powell & Gehring, 2023).

Therapeutically, optimizing glycemic control remains foundational; however, direct interventions targeting lower urinary tract dysfunction have evolved. Pharmacologic treatments, such as antimuscarinics and beta-3 agonists, provide symptom relief for patients with detrusor overactivity; however, clean intermittent catheterization remains the mainstay for individuals with retention (Panicker, 2020; Agochukwu-Mmonu et al., 2020). Small-scale medical studies for complex cases offer hope for new treatments, such as botulinum toxin injections into the detrusor or external urethral sphincter (Lee & Kuo, 2019). These therapies are emerging. Also under early study are antioxidant compounds and nerve-protecting agents that may reduce nerve damage and improve bladder function (Eid et al., 2023; Rosenberger et al., 2020).

Despite significant improvements in understanding how the disease affects the lower urinary tract, there are still important gaps in our knowledge. This means we cannot yet provide the best possible care for patients or develop ways to change the course of the disease. One of the main issues is that good studies of how bladder and urethral sphincter issues grow over time in people who have diabetes are few. Cross-sectional as well as case-control studies provide valuable information regarding the prevalence of common problems, the factors that contribute to their occurrence, and the associations with them. However, studies seldom track individuals during the period when they get nerve problems from diabetes (Wang et al., 2023; Quast et al., 2023). These studies are critical because they help us understand more about neurogenic bladder dysfunction, find ways to measure its progression, and determine the best times to administer treatment.

Longitudinal data prove especially critical to understanding the temporal changes of diabetic cystopathy. Detrusor overactivity can first be shown, later progressing to detrusor underactivity. This condition cuts the bladder's feeling and then makes the bladder empty not all the way. Powell links it to these results, like Gehring (2023). These changes in behavior could be tied to neuropathy or to many items, such as genetics (Eid et al., 2023; Callaghan et al., 2020). A person can learn if that is right through a better understanding over time, along with studying health problems related to blood sugar balance. Longitudinal studies using repeated functional assessments (urodynamic testing, measuring urinary biomarkers, and neurophysiological measures) might better explain how sensory and motor neurodegeneration in the bladder-sphincter complex changes over time. This could help doctors to make a personalized prediction of how the disease will progress and to monitor the patient's condition.

It is important to note that longitudinal data can provide valuable insights into the progression of diabetic cystopathy over time. In the early stages, there is sometimes a slight chance that it may show detrusor overactivity, which can then change into detrusor underactivity. It has been suggested that this is accompanied by impaired bladder sensation and incomplete emptying (Powell &

Gehring, 2023). A more profound understanding of time could reveal whether particular neuropathies associate with these shifts in behavior or if they form a spectrum where an individual's genetics, other health issues, and factors such as blood sugar control may play a role (Eid et al., 2023; Callaghan et al., 2020). It may be the case that longitudinal studies using repeated functional assessments (urodynamic testing, measuring urinary biomarkers, and neurophysiological measures) could potentially offer a more comprehensive explanation of how sensory and motor neurodegeneration in the bladder-sphincter complex changes over time. This could potentially assist doctors in making a personalized prediction of how the disease might progress and in monitoring the patient's condition.

Beyond the clear scientific challenges, in clinical settings, there is no consistent use of joined expert care. Patient outcomes must improve. Dealing with this makes sure they do. Endocrinologists, neurologists, urologists, rehabilitation experts, and healthcare workers must collaborate more closely to effectively manage diabetic neuropathy that affects bladder and urethral sphincter function. To address metabolic issues in systems and control blood sugar, endocrinologists must take action (Callaghan et al., 2020). Many healthcare systems are fragmented and lack standardized referral pathways. This regrettably results in missed opportunities for comprehensive assessment and management (Powell & Gehring, 2023).

It is important to recognize that psychosocial support and patient education are integral components of multidisciplinary care, yet they are often inadequately incorporated. Patients with neurogenic bladder from diabetes may meet key mental health issues that are urinary leakage shame, repeated infections, and effects on sex life with close bonds. Kumar et al., (2022) put forth that coordinated care models may improve adherence to bladder management plans and also improve overall well-being. Diabetes care should include regular testing plans for lower urinary tract problems (Quast et al., 2023).

In the future, it would be beneficial for research to focus on prospective, multicenter cohorts with standardized protocols that evaluate diabetic neurogenic bladder progression, incorporating multimodal biomarkers and patient-reported outcome measures. Additionally, it would be highly beneficial to design clinical trials for promising neuroprotective and regenerative therapies meticulously. These must be stratified based on neuropathy phenotypes and disease stage. We must continue to evaluate models of integrated care delivery, optimise interprofessional collaboration, and reduce barriers to comprehensive management.

Recent literature supports the idea that diabetic bladder and urethral sphincter dysfunction has a complex neurogenic underpinning, highlighting the significance of oxidative stress, autonomic neuropathy, and impairment of neuromuscular coordination (Table 1). Early recognition, thorough evaluation, and personalized treatment strategies are vital in preserving urinary function and enhancing the quality of life in diabetic patients.

**Table 1**. Summarizes key factors influencing lower urinary tract outcomes in diabetic neuropathy from the selected recent studies.

| **Factor** | **Association with Lower Urinary Tract Dysfunction** | **Key Evidence Sources** |
|---|---|---|
| Oxidative stress and mitochondrial dysfunction | Contributes to nerve injury and bladder/sphincter impairment | Song et al., (2022), Eid et al., (2023) |
| Reduced nerve growth factor expression | Impairs nerve regeneration, sensory loss | Powell & Gehring (2023), Strand et al., (2024) |
| Autonomic neuropathy presence | Strongly predicts bladder dysfunction severity | Quast et al., (2023), Agochukwu-Mmonu et al., (2020) |
| High postvoid residual volume | Associated with UTI risk and renal complications | Wang et al., (2023), Panicker (2020) |
| Urethral sphincter dysfunction (external and internal) | Leads to voiding dysfunction, incontinence, retention | Kumar et al., (2022), Cao et al., (2020) |
| Glycemic control | Modulates risk and progression of neuropathic dysfunction | Zaino et al., (2023), Quiroz-Aldave et al., (2023) |
| Pharmacological and interventional therapies | Symptom management and improved quality of life | Lee & Kuo (2019), Panicker (2020) |

This review clearly highlights the very complex impact of diabetic neuropathy on the bladder and also urethral sphincter function, for it stresses the broad spectrum of lower urinary tract dysfunction in patients with diabetes. Diabetic neuropathy affects the nerve pathways for sensation and movement. These pathways are key to aligned bladder hold and release, as recent function and patient studies have shown. This gives rise to symptoms spanning from detrusor overactivity when urgency and frequency happen to an atonic bladder, which is shown by poor sensation, larger capacity, and high remaining volumes (Powell & Gehring, 2023; Agochukwu-Mmonu et al., 2020). This clinical progression clearly demonstrates the dynamic neuropathic injury affecting the autonomic nervous system and the somatic innervation of the external urethral sphincter. Oxidative stress may lead to mitochondrial dysfunction, decrease the availability of neurotrophic support such as nerve growth factor, and contribute to neuronal degeneration in both the pelvic plexus and peripheral nerves (Strand et al., 2024; Song et al., 2022; Eid et al., 2023). Diabetic bladder dysfunction does definitively show, via mechanistic understandings, a manifestation of systemic neuropathic injury with distinct molecular and cellular substrates, not just as some secondary complication.

Clinical studies indicate that lower urinary tract symptoms correlate to the extent that autonomic and somatic neuropathy exert effects upon someone (Quast et al., 2023; Sasaki et al., 2020). During voiding detrusor-sphincter dyssynergia may pose difficulties, though health outcomes may significantly improve through proactive management techniques such as urodynamic testing, nerve function evaluations, as well as biomarker assessments. There are new opportunities for effective care upon embracing these approaches. Nerve injury slowing is possible. Reasonable control over blood sugar is something that enables this. At this time, actions that are direct to the bladder with sphincter trouble are still restricted. For relief from overactive bladder, medications such as antimuscarinics combined with beta-3 adrenergic agonists may be effective, while intermittent catheterization is an option for managing retention (Agochukwu-Mmonu et al., 2020; Panicker, 2020). Some emerging modalities, such as botulinum toxin injections into the detrusor or external urethral sphincter, show promise, especially for cases that do not respond well to other treatments. However, it has been suggested that further validation through randomized controlled trials may be beneficial (Lee & Kuo, 2019). It is encouraging to see that preclinical studies investigating antioxidant and neurotrophic therapies show potential for disease-modifying strategies. However, its translation to clinical use has been gradual, and there is a perception that this approach has not yet fulfilled its potential (Eid et al., 2023; Rosenberger et al., 2020).

Despite these advances, this review identifies important gaps that could be addressed to help improve the care of diabetic patients with neurogenic bladder and sphincter dysfunction (Callaghan et al., 2020; Eid et al., 2023). Furthermore, the heterogeneity of diabetic populations, varying glycemic control, comorbidities, and genetic susceptibilities introduces complexity that demands large-scale, prospective investigations integrating clinical, neurophysiological, and molecular endpoints.

Another important issue to consider is the fragmented delivery of multidisciplinary care. It appears that effective management of diabetic bladder neuropathy may require coordinated efforts among endocrinologists, neurologists, urologists, and rehabilitation specialists. This review comprehensively examines all the current evidence that supports the idea that complex nerve-based actions contribute to the impact of diabetic neuropathy on bladder function and urethral sphincter function. Although easing signs can provide absolute comfort, it is crucial to treat the main nerve damage as well as to offer a care approach focused on the patient. In future research, we must explore ways to enhance the lives of affected individuals. Future research should focus on three key areas: monitoring the condition over time, targeting specific molecules, and integrating diverse healthcare models.

## 4. CONCLUSION

It is thought that diabetic neuropathy can have an impact on the complex neural control mechanisms that govern bladder and urethral sphincter function, which can sometimes result in a range of lower urinary tract dysfunctions. It has been suggested that these may be expressed clinically as varying combinations of storage and voiding symptoms, which may include urgency, frequency, incontinence, hesitancy, and urinary retention. The progressive nature of neuropathic injury involves both autonomic and somatic nerves, which can, unfortunately, lead to sensory loss, impaired detrusor contractility, and sphincteric dysfunction. These can, unfortunately, together impair bladder emptying and continence. Current management is primarily focused on addressing symptoms and complications, underscoring the importance of earlier diagnosis and personalized, multidisciplinary care approaches. It is suggested that integrated strategies involving glycemic control, urological assessment, neurological evaluation, and patient education may be beneficial in mitigating the progression and morbidity of lower urinary tract dysfunction in diabetic populations. In the future, priority will be given to studies that examine the development of diseases over time. This approach will expedite the treatment process and enhance the

patients' qual ty of life. Taking into account the repercussions of diabetic neuropathy on the bladder and urethral sphincter is an important part of managing diabetes.

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**Author contributions**
Justyna Gręda - Conceptualization; writing - rough preparation; supervision
Karol Mateusz Wojnarowski - Writing - rough preparation
Bartosz Zieliński - Writing - rough preparation

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**Conflict of interest**
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**Data and materials availability**
All data sets collected during this study are available upon reasonable request from the corresponding author.

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