

EAU Guidelines on Neuro-Urology

B. Blok (Chair), J. Pannek (Vice-chair) D. Castro-Diaz,
G. Del Popolo, J. Groen, R. Hamid, G. Karsenty, T.M. Kessler
Guidelines Associates: H. Ecclestone, S. Musco,
B. Padilla-Fernández, V. Phé, A. Sartori, L. 't Hoen

TABLE OF CONTENTS

PAGE

1.	INTRODUCTION	4
	1.1 Aim and objectives	4
	1.2 Panel composition	4
	1.3 Available publications	4
	1.4 Publication history	4
	1.5 Background	4
2.	METHODS	4
	2.1 Introduction	4
	2.2 Review	5
3.	THE GUIDELINE	5
	3.1 Epidemiology, aetiology and pathophysiology	5
	3.1.1 Introduction	5
	3.2 Classification systems	7
	3.2.1 Introduction	7
	3.2.2 Definitions	7
	3.3 Diagnostic evaluation	12
	3.3.1 Introduction	12
	3.3.2 Classification systems	12
	3.3.3 Timing of diagnosis and treatment	12
	3.3.4 Patient history	13
	3.3.4.1 Bladder diaries	14
	3.3.5 Patient quality of life questionnaires	14
	3.3.5.1 Questions	14
	3.3.5.2 Evidence	14
	3.3.6 Physical examination	15
	3.3.6.1 Autonomic dysreflexia	15
	3.3.6.2 Summary of evidence and recommendations for history taking and physical examination	17
	3.3.7 Urodynamics	17
	3.3.7.1 Introduction	17
	3.3.7.2 Urodynamic tests	17
	3.3.7.3 Specialist uro-neurophysiological tests	18
	3.3.7.4 Summary of evidence and recommendations for urodynamics and uro-neurophysiology	19
	3.3.8 Renal function	19
	3.4 Disease management	19
	3.4.1 Introduction	19
	3.4.2 Non-invasive conservative treatment	19
	3.4.2.1 Assisted bladder emptying - Credé manoeuvre, Valsalva manoeuvre, triggered reflex voiding	19
	3.4.2.2 Neuro-urological rehabilitation	20
	3.4.2.2.1 Bladder rehabilitation including electrical stimulation	20
	3.4.2.3 Drug treatment	21
	3.4.2.3.1 Drugs for storage symptoms	21
	3.4.2.3.2 Drugs for voiding symptoms	21
	3.4.2.4 Summary of evidence and recommendations for drug treatments	22
	3.4.2.5 Minimally invasive treatment	22
	3.4.2.5.1 Catheterisation	22
	3.4.2.5.2 Summary of evidence and recommendations for catheterisation	22
	3.4.2.5.3 Intravesical drug treatment	22
	3.4.2.5.4 Botulinum toxin injections in the bladder	23
	3.4.2.5.5 Bladder neck and urethral procedures	23
	3.4.2.5.6 Summary of evidence and recommendations for minimal invasive treatment*	24

3.4.3	Surgical treatment	24
3.4.3.1	Bladder neck and urethral procedures	24
3.4.3.2	Denervation, deafferentation, sacral neuromodulation	24
3.4.3.3	Bladder covering by striated muscle	25
3.4.3.4	Bladder augmentation	25
3.4.3.5	Urinary diversion	25
3.4.3.6	Summary of evidence and recommendations for surgical treatment	26
3.5	Urinary tract infection in neuro-urological patients	26
3.5.1	Epidemiology, aetiology and pathophysiology	26
3.5.2	Diagnostic evaluation	26
3.5.3	Disease management	26
3.5.3.1	Recurrent UTI	27
3.5.3.2	Prevention	27
3.5.4	Summary of evidence and recommendations for the treatment of UTI	27
3.6	Sexual (dys)function and fertility	27
3.6.1	Erectile dysfunction (ED)	28
3.6.1.1	Phosphodiesterase type 5 inhibitors (PDE5Is)	28
3.6.1.2	Drug therapy other than PDE5Is	28
3.6.1.3	Mechanical devices	28
3.6.1.4	Intracavernous injections and intraurethral application	28
3.6.1.5	Sacral neuromodulation	28
3.6.1.6	Penile prostheses	29
3.6.1.7	Summary of evidence and recommendations for erectile dysfunction	29
3.6.2	Male fertility	29
3.6.2.1	Sperm quality and motility	29
3.6.2.2	Summary of evidence and recommendations for male fertility	30
3.6.3	Female sexuality	30
3.6.4	Female fertility	30
3.6.4.1	Summary of evidence and recommendation for female sexuality and fertility	31
3.7	Follow-up	31
3.7.1	Introduction	31
3.7.2	Summary of evidence and recommendations for follow-up	31
3.8	Conclusions	32
4.	REFERENCES	32
5.	CONFLICT OF INTEREST	55
6.	CITATION INFORMATION	55

1. INTRODUCTION

1.1 Aim and objectives

The European Association of Urology (EAU) Neuro-Urology Guidelines aim to provide information for clinical practitioners on the incidence, definitions, diagnosis, therapy, and follow-up of neuro-urological disorders. These Guidelines reflect the current opinion of experts in this specific pathology and represent a state-of-the-art reference for all clinicians, as of the publication date.

The terminology used and the diagnostic procedures advised throughout these Guidelines follow the recommendations for investigations of the lower urinary tract (LUT) as published by the International Continence Society (ICS) [1-3]. Readers are advised to consult other EAU Guidelines that may address different aspects of the topics discussed in this document.

It must be emphasised that clinical guidelines present the best evidence available to the experts but following guideline recommendations will not necessarily result in the best outcome. Guidelines can never replace clinical expertise when making treatment decisions for individual patients, but rather help to focus decisions - also taking personal values and preferences/individual circumstances of patients into account. Guidelines are not mandates and do not purport to be a legal standard of care.

1.2 Panel composition

The EAU Neuro-Urology Guidelines Panel consists of an international multidisciplinary group of neuro-urological experts. All experts involved in the production of this document have submitted potential conflict of interest statements which can be viewed on the EAU website: <http://www.uroweb.org/guideline/neuro-urology/>.

1.3 Available publications

A quick reference document, the Pocket Guidelines, is available in print and as an app for iOS and Android devices. These are abridged versions which may require consultation with the full text version. A guideline summary has also been published in European Urology [4]. All are available through the EAU website: <http://www.uroweb.org/guideline/neurourology/>.

1.4 Publication history

The EAU published the first Neuro-Urology Guidelines in 2003 with updates in 2008, 2014, and 2017. This 2018 document represents a limited update of the 2017 publication. The literature was assessed for all chapters.

1.5 Background

The function of the LUT is mainly storage and voiding of urine, which is regulated by the nervous system that co-ordinates the activity of the urinary bladder and bladder outlet. The part of the nervous system that regulates LUT function is disseminated from the peripheral nerves in the pelvis to highly specialised cortical areas. Any disturbance of the nervous system involved, can result in neuro-urological symptoms. The extent and location of the disturbance will determine the type of LUT dysfunction, which can be symptomatic or asymptomatic. Neuro-urological symptoms can cause a variety of long-term complications; the most significant being deterioration of renal function. Since symptoms and long-term complications do not correlate [5], it is important to identify patients with neuro-urological symptoms, and establish if they have a low or high risk of subsequent complications. The risk of developing upper urinary tract (UUT) damage and renal failure is much lower in patients with slowly progressive non-traumatic neurological disorders than in those with spinal cord injury or spina bifida [6]. In summary, treatment and intensity of follow-up examinations are based on the type of neuro-urological disorder and the underlying cause.

2. METHODS

2.1 Introduction

For the 2018 Neuro-Urology Guidelines, new and relevant evidence has been identified, collated and appraised through a structured assessment of the literature. A broad and comprehensive literature search, covering all sections of the Neuro-Urology Guidelines was performed. Databases searched included Medline, EMBASE, and the Cochrane Libraries, covering a time frame between June 30th 2016 and June 1st 2017. A total of 659 unique records were identified, retrieved and screened for relevance. A detailed search strategy is available online: <http://uroweb.org/guideline/neuro-urology/?type=appendices-publications>.

For the 2018 edition of the EAU Guidelines the Guidelines Office have transitioned to a modified GRADE methodology across all 20 guidelines [7, 8]. For each recommendation within the guidelines there is an accompanying online strength rating form which addresses a number of key elements namely:

1. the overall quality of the evidence which exists for the recommendation, references used in this text are graded according to a classification system modified from the Oxford Centre for Evidence-Based Medicine Levels of Evidence [9];
2. the magnitude of the effect (individual or combined effects);
3. the certainty of the results (precision, consistency, heterogeneity and other statistical or study related factors);
4. the balance between desirable and undesirable outcomes;
5. the impact of patient values and preferences on the intervention;
6. the certainty of those patient values and preferences.

These key elements are the basis which panels use to define the strength rating of each recommendation. The strength of each recommendation is represented by the words ‘strong’ or ‘weak’ [10]. The strength of each recommendation is determined by the balance between desirable and undesirable consequences of alternative management strategies, the quality of the evidence (including certainty of estimates), and nature and variability of patient values and preferences. The strength rating forms will be available online.

Additional information can be found in the general Methodology section of this print, and online at the EAU website; <http://www.uroweb.org/guideline/>. A list of associations endorsing the EAU Guidelines can also be viewed online at the above address.

2.2 Review

Publications ensuing from panel led systematic reviews have all been peer-reviewed. The 2015 Neuro-Urology Guidelines were subject to peer review prior to publication.

3. THE GUIDELINE

3.1 Epidemiology, aetiology and pathophysiology

3.1.1 Introduction

Neuro-urological symptoms may be caused by a variety of diseases and events affecting the nervous system controlling the LUT. The resulting neuro-urological symptoms depend predominantly on the location and the extent of the neurological lesion. There are no exact figures on the overall prevalence of neuro-urological disorders in the general population, but data are available on the prevalence of the underlying conditions and the relative risk of these for the development of neuro-urological symptoms. It is important to note that the majority of the data shows a very wide range of prevalence/incidence. This reflects the variability in the cohort (e.g. early or late stage disease) and the frequently small sample sizes, resulting in a low level of evidence in most published data (summarised in Table 1).

Table 1: Epidemiology of Neuro-Urological Disorders

<i>Suprapontine and pontine lesions and diseases</i>		
Neurological Disease	Frequency in General Population	Type and Frequency of Neuro-Urological Symptoms
Cerebrovascular accident (Strokes)	450 cases/100,000/yr (Europe) [11], 10% of cardiovascular mortality.	Nocturia - overactive bladder (OAB) - urgency urinary incontinence (UUI) - detrusor overactivity (DO), other patterns less frequent [12]. 57-83% of neuro-urological symptoms at 1 month post stroke, 71-80% spontaneous recovery at 6 months [13]. Persistence of urinary incontinence (UI) correlates with poor prognosis [14].

Dementias: Alzheimer's disease (80%), Vascular (10%), Other (10%).	6.4% of adults > 65 yrs [15].	OAB - UII – DO 25% of incontinence in Alzheimer's disease, > 25% in other dementias: Lewy body, NPH, Binswanger, Nasu-Hakola, Pick Disease [16]. Incontinence 3 times more frequent in geriatric patients with dementia than without [17].
Parkinsonian syndrome (PS) Idiopathic Parkinson's disease (IPD): 75-80% of PS. Non-IPD: Parkinson's-plus (18%): - Multiple system atrophy (MSA), - Progressive supranuclear palsy, - Corticobasal degeneration, - Dementia with Lewy bodies. Secondary Parkinson's (2%)	2nd most prevalent neurodegenerative disease after Alzheimer's disease. Rising prevalence of IPD with age [18]. MSA is the most frequent non-IPD PS.	LUTS frequency 30% at onset, 70% after 5 yrs. Storage phase symptoms: Nocturia (78%) OAB - UII - DO [19]. OAB and DO at the initial phase, intrinsic sphincter deficiency and impaired contractility appear as the disease progress. Complications of neuro-urological symptoms (infections) account for a major cause of mortality in MSA [20]. Impaired detrusor contractility seems to be the urodynamic finding distinguishing MSA from IPD [21, 22].
Brain tumours	26.8/100,000/yr in adults (> 19 yrs), (17.9 benign, 8.9 malignant) [23].	Incontinence occurs mainly in frontal location (part of frontal syndrome or isolated in frontal location) [24].
Cerebral palsy	Cerebral palsy: 3.1-3.6/1,000 in children aged 8 yrs [25].	62% of women and 58% of men with cerebral palsy suffer from UI [26] 70% DO. Recurrent urinary tract infection (UTI) and radiologic abnormalities in > 10% of cases [25-27].
Traumatic brain injury	235/100,000/yr [28]	44% storage dysfunction. 38% voiding dysfunction, 60% urodynamic abnormalities [29].
Lesions and diseases between caudal brainstem and sacral spinal cord		
Spinal cord injury (SCI)	Prevalence of traumatic SCI in developed countries ranges from 280 to 906/million [30].	Neurogenic detrusor overactivity (NDO) and detrusor sphincter dyssynergia (DSD) (up to 95%) and detrusor underactivity (up to 83%) depending on the level of the lesion [31].
Spina bifida (SB)	Spina bifida 3-4/10,000 Lumbar and lumbosacral form are the most common (60%) [32].	Bladder function is impaired in up to 96% of SB patients [33].
Lesions and diseases of the peripheral nervous system		
Lumbar spine Degenerative disease Disk prolapse Lumbar canal stenosis	Male (5%) and female (3%) > 35 yr have had a lumbosacral episode related to disc prolapse. Incidence: approx. 5/100,000/yr More common in females > 45 yr.	26% difficulty to void and acontractile detrusor [34]. Detrusor underactivity (up to 83%) [31].
Latrogenic pelvic nerve lesions	Rectal cancer. Cervical cancer (multimodal therapy, radiotherapy and surgery). Endometriosis surgery.	After abdomino-perineal resection (APR): 50% urinary retention. After total mesorectal excision (TME): 10-30% voiding dysfunction [35].

Peripheral neuropathy Diabetes Other causes of peripheral neuropathy causing neuro-urological symptoms: alcohol abuse; lumbosacral zona and genital herpes; Guillain Barré syndrome; porphyria; sarcoidosis.	Worldwide, prevalence of pharmacologically treated diabetes 8.3% [36].	Urgency/frequency +/-incontinence [37]. Hyposensitive and detrusor underactivity at later phase [37].
Disseminated central diseases		
Multiple sclerosis (MS)	Prevalence: 83/100,000 in Europe [38].	10% of MS patients present with voiding dysfunction at disease onset, 75% of patients will develop it after 10 yrs of MS [39]. DO: 86% [39]. DSD: 35% [39]. Detrusor underactivity: 25% [39].

3.2 Classification systems

3.2.1 Introduction

Relevant definitions are found in the general ICS standardisation report [1, 2]. Section 3.2.2 lists the definitions from these references, partly adapted, and other definitions considered useful for clinical practice (Tables 2 and 3).

3.2.2 Definitions

Table 2: Definitions useful in clinical practice

Autonomic dysreflexia (AD)	Autonomic dysreflexia is a sudden and exaggerated autonomic response to various stimuli in patients with SCI or spinal dysfunction at or above level Th 6. It is defined as an increase in systolic blood pressure (SBP) > 20 mmHg from baseline [40]. Autonomic dysreflexia may be symptomatic (headache, blurred vision, stuffy nose, piloerection, flushing, sweating above the lesion level (vasodilatation), pale and cold skin (vasoconstriction) below the lesion level or asymptomatic (silent).
Bladder expression	Various manoeuvres aimed at increasing intravesical pressure in order to facilitate bladder emptying (abdominal straining, Valsalva's manoeuvre and Crede's manoeuvre) [3].
Bladder reflex triggering	Various manoeuvres performed by the patient or the therapist in order to elicit reflex detrusor contraction by exteroceptive stimuli (suprapubic tapping, thigh scratching and anal/rectal manipulation) [3].
Bladder sensation, absent	<i>During history taking</i> , the patient reports no sensation of bladder filling or desire to void [3]. <i>During filling cystometry</i> , the patient has no bladder sensation [3].
Bladder sensation, normal	<i>During history taking</i> , the patient is aware of bladder filling and increasing sensation up to a strong desire to void [3].
First sensation of bladder filling	The feeling, during filling cystometry, when the patient first becomes aware of the bladder filling [3]. <i>During filling cystometry</i> , can further be judged by the two following defined points and evaluated in relation to the bladder volume at that moment and in relation to the patient's symptomatic complaints [3].
First desire to void	The feeling, during filling cystometry, that would lead the patient to pass urine at the next convenient moment, but voiding can be delayed if necessary [3].
Strong desire to void	Persistent desire to void, during filling cystometry, without the fear of leakage [3].

Bladder sensation, increased	<i>During history taking</i> , the patient feels an early and persistent desire to void [3]. <i>During filling cystometry</i> , an early first sensation of bladder filling (or an early desire to void) and/or an early strong desire to void, which occurs at low bladder volume and which persists. It is a subjective assessment, not possible to quantify [3].
Bladder sensation, non-specific	<i>During history taking</i> , the patient reports no specific bladder sensation but may perceive bladder filling as abdominal fullness, vegetative symptoms, or spasticity [3]. <i>During filling cystometry</i> , may make the patient aware of bladder filling, for example, abdominal fullness or vegetative symptoms [3].
Bladder sensation, reduced	<i>During history taking</i> , the patient is aware of bladder filling but does not feel a definite desire to void [3]. <i>During filling cystometry</i> , a diminished sensation throughout bladder filling [3].
Catheterisation	Technique for bladder emptying employing a catheter to drain the bladder or a urinary reservoir [3].
Catheterisation, indwelling	An indwelling catheter remains in the bladder, urinary reservoir or urinary conduit for a period of time longer than one emptying [3].
Catheterisation, intermittent (IC)	Drainage or aspiration of the bladder or a urinary reservoir with subsequent removal of the catheter [3]. When not specified “self”, it is performed by an attendant (e.g. doctor, nurse or relative).
Aseptic IC	Use of a sterile technique. This implies genital disinfection and the use of sterile catheters and instruments/gloves [3].
Clean IC	Use of a clean technique. This implies ordinary washing techniques and use of disposable or cleansed reusable catheters [3].
Intermittent self-catheterisation	Performed by the patient him/herself [3].
Daytime frequency, increased	Complaint by the patient who considers that he/she voids too often by day. This term is equivalent to pollakiuria used in many countries [3]. Many population-based studies of OAB have defined frequency as either eight or more voids/day, or eight or more voids/24 hours [41].
Diary, bladder	Records the times of micturitions and voided volumes, incontinence episodes, pad usage and other information such as fluid intake, the degree of urgency and the degree of incontinence [3].
Frequency volume chart (FVC)	Records the volumes voided as well as the time of each micturition, day and night, for at least 24 hours [3].
Micturition time chart	Records only the times of micturitions, day and night, for at least 24 hours [3].
Enuresis	Any involuntary loss of urine. If it is used to denote incontinence during sleep, it should always be qualified with the adjective “nocturnal” [3].
Hesitancy	Difficulty in initiating micturition resulting in a delay in the onset of voiding after the individual is ready to pass urine [3].
Intermittent stream (Intermittency)	Urine flow which stops and starts, on one or more occasions, during micturition [3].
Motor neuron lesion, lower (LMNL)	Lesion resulting from damage to motor neurons of the ventral horns or motor neuron of the cranial nerve nuclei, or resulting from interruption of the final common pathway connecting the neuron via its axon with the muscle fibres it innervates (the motor unit) [3].
Motor neuron lesion, upper (UMNL)	Lesion resulting from damage to cortical neurons that give rise to corticospinal and corticobulbar tracts. It may occur at all levels of the neuraxis from the cerebral cortex to the spinal cord. When rostral to the pyramidal decussation of the caudal medulla, they result in deficits below the lesion, on the contralateral side. When caudal to the pyramidal decussation, they result in deficits below the lesion, on the ipsilateral side [42].

Neurogenic shock	Loss of vascular tone in part of the body deprived of supraspinal control. It commonly occurs during the acute period following SCI and is associated with failure of the sympathetic nervous system. In this condition, systolic blood pressure < 90 mmHg in the supine posture is not the result of low intravascular volume (e.g. blood loss, dehydration, sepsis, cardiac disorders) [40].
Spinal shock	Characterised by marked reductions in spinal reflex activity below the level of injury [40].
Nocturia	The complaint that the individual has to wake at night one or more times to void [3]. Each void is preceded and followed by sleep.
Nocturnal polyuria	It is present when an increased proportion of the 24-hour output occurs at night (normally during the 8 hours whilst the patient is in bed). The night time urine output excludes the last void before sleep but includes the first void of the morning [3].
Neurogenic lower urinary tract dysfunction (NLUTD)	Lower urinary tract dysfunction (LUTD) secondary to confirmed pathology of the nervous supply.
Orthostatic hypotension	Symptomatic (dizziness, headache or neck ache, fatigue) or asymptomatic decrease in blood pressure defined as a drop of at least 20mmHg systolic or 10 mmHg diastolic within 3 minutes of moving from the supine to an upright position [2, 41].
Overactive bladder syndrome (also urge syndrome or urgency-frequency syndrome)	Urgency, with or without urge incontinence, usually with frequency and nocturia [3].
Pain, genital and lower urinary tract	Abnormal sensations felt by the individual as pain, discomfort and pressure. Should be characterised by type, frequency, duration, precipitating and relieving factors and by location [3].
Bladder pain	<i>During history taking</i> , pain that is felt suprapubically or retropubically, and usually increases with bladder filling, it may persist after voiding [3]. <i>During filling cystometry</i> , is an abnormal finding [3].
Pelvic pain	Is less well defined than, for example, bladder, urethral or perineal pain and is less clearly related to the micturition cycle or to bowel function and is not localised to any single pelvic organ [3].
Perineal pain	In females, between the posterior fourchette (posterior lip of the introitus) and the anus. In males, between the scrotum and the anus [3].
Scrotal pain	May or may not be localised, for example to the testis, epididymis, cord structures or scrotal skin [3].
Urethral pain	Pain that is felt in the urethra and the individual indicates the urethra as the site [3].
Vaginal pain	Is felt internally, above the introitus [3].
Vulvar pain	Is felt in and around the external genitalia [3].
Pelvic organ prolapse	Descent of one or more of the anterior vaginal wall, the posterior vaginal wall, and the apex of the vagina (cervix/uterus) or vault (cuff) after hysterectomy. Absence of prolapse is defined as stage 0 support; prolapse can be staged from stage I to stage IV [3].
Slow stream	Perception of reduced urine flow, usually compared to previous performance or in comparison to others [3].
Spinal cord injury	Incomplete: if partial preservation of sensory and/or motor functions is found below the neurological level and includes the lowest sacral segment. Complete: when there is an absence of sensory and motor function in the lowest sacral segment [43].
Cauda equina	Injuries affecting the cauda equina and generally causing an acontractile or lower motor neuron picture affecting the LUT, distal bowel and sexual function [40].
Conal	Injuries affecting the conus medullaris of the spinal cord and often causing a mixed lesion to the LUT, distal bowel and sexual functions with a resultant either overactive or acontractile picture [40].

Supraconal	Injuries occurring above the conus medullaris. In general, supraconal injuries cause an overactive or upper motor neuron pattern of damage affecting the LUT, distal bowel and sexual functions [40].
Straining to void	Muscular effort used to either initiate, maintain or improve the urinary stream [3].
Terminal dribble	Prolonged final part of micturition, when the flow has slowed to a trickle/dribble [3].
Urgency	The complaint of a sudden compelling desire to pass urine which is difficult to defer [3].
Urinary incontinence	Complaint of any involuntary leakage of urine [3].
Stress urinary incontinence (SUI)	Complaint of involuntary leakage on effort or exertion, or on sneezing or coughing [3].
Urge urinary incontinence	Complaint of involuntary leakage accompanied by or immediately preceded by urgency [3].
Mixed urinary incontinence	Complaint of involuntary leakage associated with urgency and also with exertion, effort, sneezing or coughing [3].
Continuous urinary incontinence	Complaint of continuous leakage [3].
Voided volume, maximum	The largest volume of urine voided during a single micturition which is determined either from the frequency/volume chart or bladder diary [3].

Table 3: Definitions useful when interpreting urodynamic studies.

Bladder compliance	Relationship between change in bladder volume and change in detrusor pressure. Compliance is calculated by dividing the volume change (ΔV) by the associated change in detrusor pressure (Δp_{det}) during the change in bladder volume ($C = \Delta V / \Delta p_{det}$). It is expressed in mL/cm H ₂ O [3].
Bladder filling, artificial	Filling the bladder, via a catheter, with a specified liquid at a specified rate [3].
Bladder filling, natural	The bladder is filled by the production of urine rather than by an artificial medium [3].
Bladder outlet obstruction	Generic term for obstruction during voiding, characterised by increased detrusor pressure and reduced urine flow rate. It is usually diagnosed by studying the synchronous values of flow rate and detrusor pressure [42].
Cystometric capacity	The bladder volume at the end of the filling cystometrogram, when "permission to void" is usually given. The volume voided together with any residual urine [3].
Maximum anaesthetic bladder capacity	The volume to which the bladder can be filled under deep general or spinal anaesthetic and should be qualified according to the type of anaesthesia used, the speed, the length of time, and the pressure at which the bladder is filled [3].
Maximum cystometric capacity	In patients with normal sensation, the volume at which the patient feels they can no longer delay micturition (has a strong desire to void) [3].
Detrusor function, normal	Allows bladder filling with little or no change in pressure. No involuntary phasic contractions occur despite provocation [42]. Normal voiding is achieved by a voluntarily initiated continuous detrusor contraction that leads to complete bladder emptying within a normal time span, and in the absence of obstruction. For a given detrusor contraction, the magnitude of the recorded pressure rise will depend on the degree of outlet resistance [3].
Detrusor overactivity	Urodynamic observation characterised by involuntary detrusor contractions during the filling phase which may be spontaneous or provoked [3].
Detrusor overactivity incontinence	Incontinence due to an involuntary detrusor contraction [3].
Idiopathic detrusor overactivity	When there is no defined cause [3].

Phasic detrusor overactivity	Is defined by a characteristic wave form and may or may not lead to UI [3].
Neurogenic detrusor overactivity	When there is a relevant neurological condition present [3].
Terminal detrusor overactivity	A single, involuntary detrusor contraction, occurring at cystometric capacity, which cannot be suppressed and results in incontinence usually resulting in bladder emptying (voiding) [3].
Detrusor sphincter dyssynergia	A detrusor contraction concurrent with an involuntary contraction of the urethral and/or periurethral striated muscle. Occasionally, flow may be prevented altogether [3]. This term is specific to patients with a neurological diagnosis.
Detrusor underactivity	Contraction of reduced strength and/or duration, resulting in prolonged bladder emptying and/or a failure to achieve complete bladder emptying within a normal time span [3].
Acontractile detrusor	Detrusor that cannot be demonstrated to contract during urodynamic studies [3].
Dysfunctional voiding	Intermittent and/or fluctuating flow rate due to involuntary intermittent contractions of the peri-urethral striated muscle during voiding in neurologically normal individuals [3].
Filling cystometry	Method by which the pressure/volume relationship of the bladder is measured during bladder filling [3].
Filling rate, physiological	Filling rate less than the predicted maximum - body weight (kg)/4 in mL/min [3, 44].
Filling rate, non-physiological	Filling rate greater than the predicted maximum filling rate [3, 42].
Leak point pressure, abdominal (ALPP)	The intravesical pressure at which urine leakage occurs due to increased abdominal pressure in the absence of a detrusor contraction [3].
Leak point pressure, detrusor (DLPP)	The lowest detrusor pressure at which urine leakage occurs in the absence of either a detrusor contraction or increased abdominal pressure [3].
Non-relaxing urethral sphincter obstruction	Characterised by a non-relaxing, obstructing urethra resulting in reduced urine flow. Usually occurs in individuals with a neurological lesion [3].
Post void residual (PVR)	The volume of urine left in the bladder at the end of micturition [3].
Pressure flow study	Method by which the relationship between pressure in the bladder and urine flow rate is measured during bladder emptying [3].
Provocative manoeuvres	Techniques used during urodynamics in an effort to provoke detrusor overactivity, for example, rapid filling, use of cooled or acid medium, postural changes and hand washing [3].
Urethral closure mechanism, incompetent	Allows leakage of urine in the absence of a detrusor contraction [3].
Urethral relaxation incontinence	Leakage due to urethral relaxation in the absence of raised abdominal pressure or detrusor overactivity [3].
Urethral closure mechanism, normal	Maintains a positive urethral closure pressure during bladder filling even in the presence of increased abdominal pressure, although it may be overcome by detrusor overactivity.
Urethral pressure	The fluid pressure needed to just open a closed urethra [3].
Urethral pressure, maximum	The maximum pressure of the measured profile [3].
Urethral pressure profile	A graph indicating the intraluminal pressure along the length of the urethra [3].
Urethral closure pressure profile	Is given by the subtraction of intravesical pressure from urethral pressure [3].
Urethral closure pressure, maximum	The maximum difference between the urethral pressure and the intravesical pressure [3].
Urethral functional profile length	The length of the urethra along which the urethral pressure exceeds intravesical pressure in women [3].
Urethral pressure “transmission” ratio	The increment in urethral pressure on stress as a percentage of the simultaneously recorded increment in intravesical pressure [3].
Urodynamic stress incontinence	The involuntary leakage of urine during increased abdominal pressure, in the absence of a detrusor contraction [3].

Urodynamic study, ambulatory	Functional test of the lower urinary tract, utilising natural filling, and reproducing the subject's every day activities [3].
Urodynamic study, conventional	Normally takes place in the urodynamic laboratory and usually involve artificial bladder filling [3].

3.3 Diagnostic evaluation

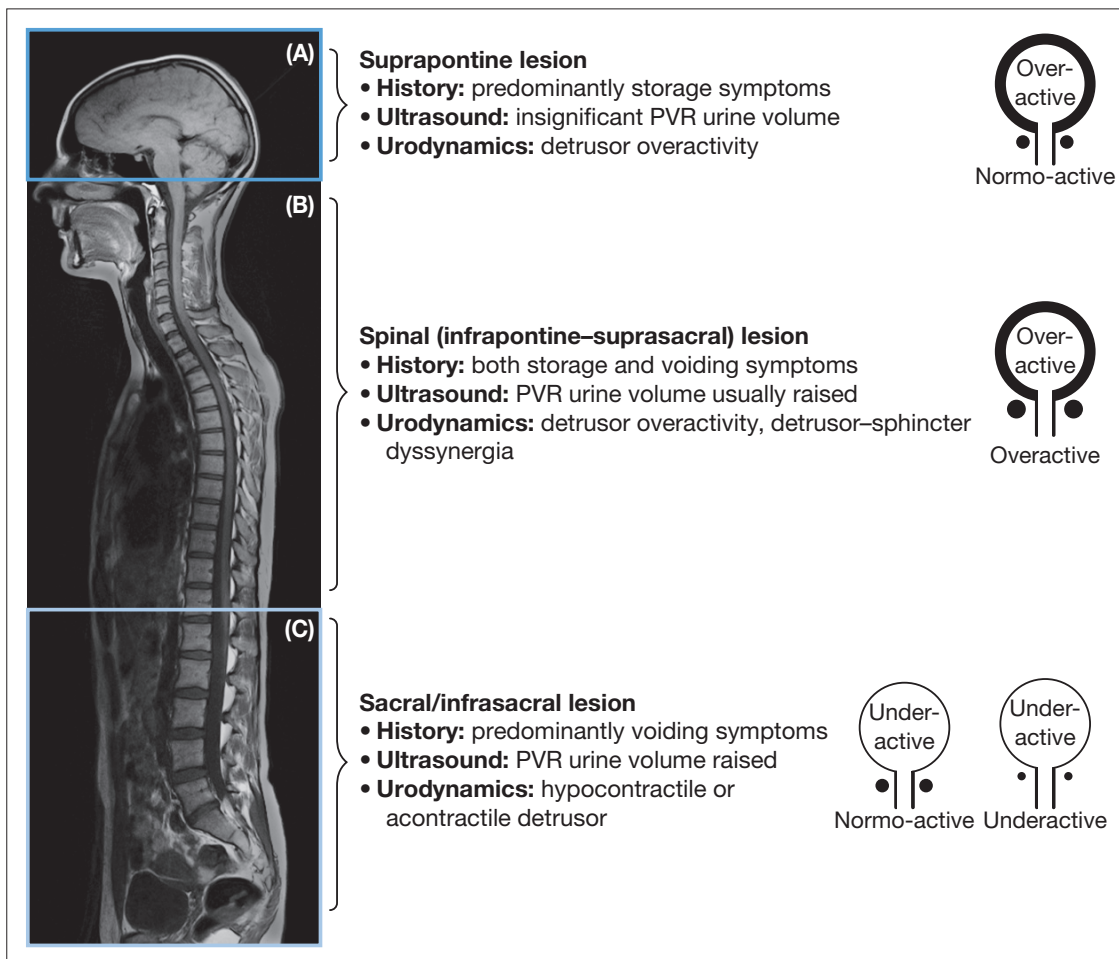
3.3.1 Introduction

The normal physiological function of the LUT depends on an intricate interplay between the sensory and motor nervous systems. When diagnosing neuro-urological symptoms, the aim is to describe the type of dysfunction involved. A thorough medical history, physical examination and bladder diary are mandatory before any additional diagnostic investigations can be planned. Results of the initial evaluation are used to decide the patient's long-term treatment and follow-up.

3.3.2 Classification systems

The pattern of LUT dysfunction following neurological disease is determined by the site and nature of the lesion. A very simple classification system for use in daily clinical practice to decide on the appropriate therapeutic approach is provided in Figure 1 [6].

Figure 1: Patterns of lower urinary tract dysfunction following neurological disease



The pattern of LUT dysfunction following neurological disease is determined by the site and nature of the lesion. Panel (A) denotes the region above the pons, panel (B) the region between the pons and the sacral cord and panel (C) the sacral cord and intrasacral region. Figures on the right show the expected dysfunctional states of the detrusor-sphincter system. Figure adapted from Panicker et al. [6] with permission from Elsevier. PVR = post-void residual.

3.3.3 Timing of diagnosis and treatment

Early diagnosis and treatment are essential in both congenital and acquired neuro-urological disorders [45]. This helps to prevent irreversible changes within the LUT, even in the presence of normal reflexes [46, 47].

Furthermore, urological symptoms can be the presenting feature of neurological pathology [48, 49]. Early intervention can prevent irreversible deterioration of the LUT and UUT [50]. Long term follow up (life-long) is mandatory to assess risk of UUT damage, renal failure and bladder cancer [51-53].

3.3.4 Patient history

History taking should include past and present symptoms and disorders (Table 4). It is the cornerstone of evaluation, as the answers will aid in diagnostic investigations and treatment options.

- In non-traumatic neuro-urological patients with an insidious onset, a detailed history may find that the condition started in childhood or adolescence [54].
- Urinary history consists of symptoms associated with both urine storage and emptying.
- Bowel history is important because patients with neuro-urological symptoms may also have related neurogenic bowel dysfunction [55].
- Sexual function may be impaired because of the neuro-urological condition [56].
- Special attention should be paid to possible warning signs and symptoms (e.g. pain, infection, haematuria and fever) requiring further investigation.
- Patients with SCI usually find it difficult to report UTI-related symptoms accurately [57, 58].
- The presence of urinary, bowel and sexual symptoms without neurological symptoms could be suggestive of an underlying neurological disease or condition.
- Ambulatory status after acute SCI does not predict presence or absence of unfavourable urodynamic parameters [59].

Table 4: History taking in patients with suspected neuro-urological disorder

Past history
Childhood through to adolescence and into adulthood
Hereditary or familial risk factors
Specific female: Menarche (age); this may suggest a metabolic disorder
Obstetric history
History of diabetes
Diseases, e.g. multiple sclerosis, parkinsonism, encephalitis, syphilis
Accidents and operations, especially those involving the spine and central nervous system
Present history
Present medication
Lifestyle (smoking, alcohol and drugs); may influence urinary, sexual and bowel function
Quality of life
Specific urinary history
Onset of urological history
Relief after voiding; to detect the extent of a neurological lesion in the absence of obstructive uropathy
Bladder sensation
Initiation of micturition (normal, precipitate, reflex, strain, Credé)
Interruption of micturition (normal, paradoxical, passive)
Enuresis
Mode and type of voiding (catheterisation)
Frequency, voided volume, incontinence, urgency episodes
Sexual history
Genital or sexual dysfunction symptoms
Sensation in genital area
Specific male: erection, (lack of) orgasm, ejaculation
Specific female: dyspareunia, (lack of) orgasm
Bowel history
Frequency and faecal incontinence
Desire to defecate
Defecation pattern
Rectal sensation
Initiation of defecation (digitation)

Neurological history
Acquired or congenital neurological condition
Mental status and comprehension
Neurological symptoms (somatic and sensory), with onset, evolution and any treatment
Spasticity or autonomic dysreflexia (especially in lesions at or above level Th 6)
Mobility and hand function

3.3.4.1 *Bladder diaries*

Bladder diaries provide data on the number of voids, voided volume, pad weight and incontinence and urgency episodes [60, 61]. Although a 24 hour bladder diary (recording should be done for three consecutive days) is reliable in women with UI [62, 63], no research has been done on bladder diaries in neuro-urological patients. Nevertheless, bladder diaries are considered a valuable diagnostic tool.

3.3.5 **Patient quality of life questionnaires**

An assessment of the patient's present and expected future quality of life (QoL) is important to evaluate the effect of any therapy. Quality of life is an essential aspect of the overall management of neuro-urological patients, for example when evaluating treatment related changes on a patient's QoL [64]. The type of bladder management has been shown to affect health-related QoL (HRQoL) in patients with SCI [65] and MS [66], as does the presence or absence of urinary and faecal incontinence [67]. Other research has also highlighted the importance of urological treatment and its impact on the urodynamic functionality of the neuro-urological patient in determining patient QoL [68].

In recent years a proliferation in the number of questionnaires to evaluate symptoms and QoL has been seen. Condition-specific questionnaires can be used to assess symptom severity and the impact of symptoms on QoL. A patient's overall QoL can be assessed using generic questionnaires. It is important that the questionnaire of choice has been validated in the neuro-urological population, and that it is available in the language that it is to be used in.

3.3.5.1 *Questions*

- Which validated patient questionnaires are available for neuro-urological patients?
- Which questionnaires are the most appropriate for use in neuro-urological patients?

3.3.5.2 *Evidence*

Three condition-specific questionnaires for urinary or bowel dysfunction and QoL have been developed specifically for adult neuro-urological patients [69]. In MS and SCI patients the Qualiveen [70, 71] is validated and can be used for urinary symptoms. A short form of the Qualiveen is available [70, 71] and it has been translated into various languages [72-75]. The Neurogenic Bladder Symptom Score (NBSS) has been validated in neurological patients to measure urinary symptoms and their consequences [76]. The QoL scoring tool related to Bowel Management (QoL-BM) [77] can be used to assess bowel dysfunction in MS and SCI patients.

In addition, sixteen validated questionnaires that evaluate QoL and assess urinary symptoms as a subscale or question in neuro-urological patients have been identified [78] (Table 5). The condition-specific Incontinence-Quality of Life (I-QoL) questionnaire which was initially developed for the non-neurological population has now also been validated for neuro-urological patients [79].

A patient's overall QoL can be assessed by generic HRQoL questionnaires, the most commonly used being the Incontinence Quality of Life Instrument (I-QOL), King's Health Questionnaire (KHQ), or the Short Form 36-item and 12-item Health Survey Questionnaires (SF-36, SF-12) [69]. In addition, the quality-adjusted life year (QALY), quantifies outcomes, by weighing years of life spent in a specified health state, adjusted by a factor representing the value placed by society or patients on their specific health state [80].

Several patient reported outcome measures (PROMs) have been identified to evaluate sexual function in neurologic patients. Strong evidence was found only for the Multiple Sclerosis Intimacy and Sexuality Questionnaire-15 and Multiple Sclerosis Intimacy and Sexuality Questionnaire-19 for patients with MS [362].

No evidence was found for which validated questionnaires are the most appropriate for use, since no quality criteria for validated questionnaires have been assessed [69].

Table 5: Patient questionnaires

Questionnaire	Underlying neurological disorder	Bladder	Bowel	Sexual function
FAMS [81]	MS	X		X
FILMS [82]	MS	X	X	
HAQUAMS [83]	MS	X	X	X
IQOL [79]	MS, SCI	X		X
MDS [84]	MS	X	X	
MSISQ-15 / MSISQ-19 [85, 86]	MS	X	X	X
MSQLI [87]	MS	X	X	X
MSQoL-54 [88]	MS	X	X	X
MSWDQ [89]	MS	X	X	
NBSS [90]	MS, SCI, Congenital neurogenic bladder	X		
QoL-BM [77]	SCI		X	
Qualiveen/SF-Qualiveen [71, 91]	MS, SCI	X		X
RAYS [92]	MS	X		X
RHSCIR [93]	SCI	X	X	X
Fransceschini [92]	SCI	X	X	X

3.3.6 *Physical examination*

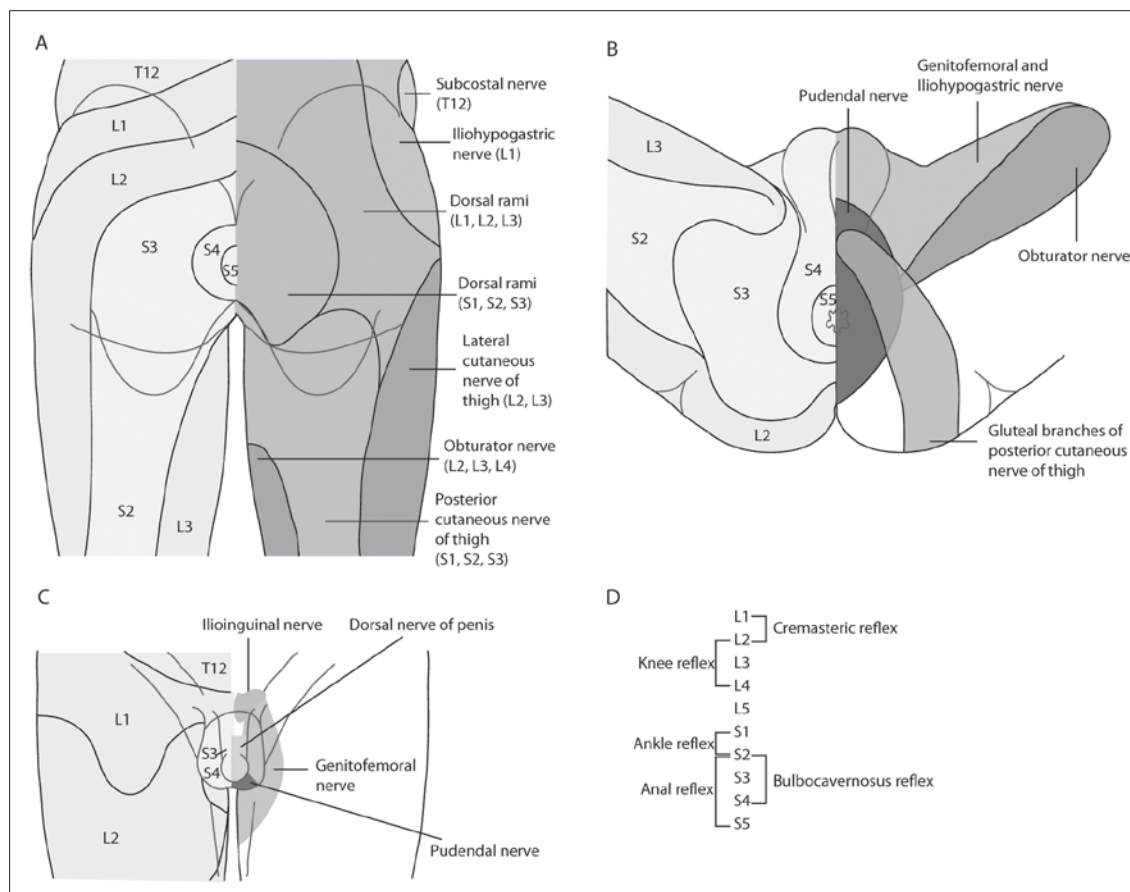
In addition to a detailed patient history, attention should be paid to possible physical and intellectual disabilities with respect to the planned investigations [94, 95]. Neuro-urological status should be described as completely as possible (Figure 2) [6]. Patients with a high spinal cord lesion or supraspinal neurological lesions may suffer from a significant drop in blood pressure when moved into a sitting or standing position. All sensations and reflexes in the urogenital area must be tested [6]. Furthermore, detailed testing of the anal sphincter and pelvic floor functions must be performed (Figure 2) [6, 96]. It is essential to have this clinical information to reliably interpret later diagnostic investigations.

Additionally, urinalysis, blood chemistry, ultrasonography, residual and free flowmetry and incontinence quantification should be performed as part of the routine assessment of neuro-urological patients [6, 97].

3.3.6.1 *Autonomic dysreflexia*

Autonomic dysreflexia is a sudden and exaggerated autonomic response to various stimuli in patients with SCI or spinal dysfunction. It generally manifests at or above level Th 6. The stimulus can be distended bladder or bowel. For example, iatrogenic stimuli during cystoscopy or urodynamics can trigger AD [98]. It can also be secondary to sexual stimulation or a noxious stimulus, e.g. infected toe nail or pressure sore. Autonomic dysreflexia is defined by an increase in systolic blood pressure > 20 mmHg from baseline [40] and can have life-threatening consequences if not properly managed.

Figure 2: Lumbosacral dermatomes, cutaneous nerves, and reflexes



The physical examination includes testing sensations and reflexes mediated through the lower spinal cord. Abnormal findings would suggest a lesion affecting the lumbosacral segments; mapping out distinct areas of sensory impairment helps to further localise the site of the lesion. Distribution of dermatomes (areas of skin mainly supplied by a single spinal nerve) and cutaneous nerves over the perianal region and back of the upper thigh (A), the perineum [99] (B), male external genitalia [100] (C) and root values of lower spinal cord reflexes (D). Figure adapted from Panicker et al. [6] with parts A-C adapted from Standring [101], both with permission from Elsevier.

Table 6: Neurological items to be specified

Sensation S2-S5 (both sides)
Presence (increased/normal/reduced/absent)
Type (light touch/pin prick)
Affected dermatomes
Reflexes (increased/normal/reduced/absent)
Bulbocavernosus reflex
Perianal/anal reflex
Knee and ankle reflexes
Plantar responses (Babinski)
Anal sphincter tone
Presence (increased/normal/reduced/absent)
Voluntary contractions of anal sphincter and pelvic muscles (increased/normal/reduced/absent)
Prostate palpation
Descensus (prolapse) of pelvic organs

3.3.6.2 Summary of evidence and recommendations for history taking and physical examination

Summary of Evidence	LE
Early diagnosis and treatment are essential in both congenital and acquired neuro-urological disorders to prevent irreversible changes within the LUT.	4
An extensive general history is the basis of evaluation focusing on past and present symptoms including urinary, sexual, bowel and neurological functions.	4
Assessment of present and expected future QoL is an essential aspect of the overall management of neuro-urological patients and is important to evaluate the effect of any therapy.	2a
Quality of life assessment should be completed with validated QoL questionnaires for neuro-urological patients.	1a
Bladder diaries provide data on the number of voids, voided volume, pad weight and incontinence and urgency episodes.	3

Recommendations	Strength rating
History taking	
Take an extensive general history, concentrating on past and present symptoms.	Strong
Take a specific history for each of the four mentioned functions - urinary, bowel, sexual and neurological.	Strong
Pay special attention to the possible existence of alarm signs (e.g. pain, infection, haematuria, fever) that warrant further specific diagnosis.	Strong
Assess quality of life when evaluating and treating the neuro-urological patient.	Strong
Use available validated tools including the Qualiveen and I-QoL for urinary symptoms and the QoL-BM for bowel dysfunction in multiple sclerosis and spinal cord injury patients. In addition, generic (SF-36 or KHQ) questionnaires can be used.	Strong
Use MSISQ-15 and MSISQ-19 to evaluate sexual function in multiple sclerosis patients.	Strong
Physical examination	
Acknowledge individual patient disabilities when planning further investigations.	Strong
Describe the neurological status as completely as possible, sensations and reflexes in the urogenital area must all be tested.	Strong
Test the anal sphincter and pelvic floor functions.	Strong
Perform urinalysis, blood chemistry, bladder diary, residual and free flowmetry, incontinence quantification and urinary tract imaging.	Strong

I-QoL = Incontinence Quality of Life Instrument; QoL-BM = Quality of Life Bowel Management scoring tool; KHQ = King's Health Questionnaire; SF-36 = Short Form 36-item Health Survey Questionnaires; MSISQ 15/19 = Multiple Sclerosis Intimacy and Sexuality Questionnaire 15/19 question version.

3.3.7 Urodynamics

3.3.7.1 Introduction

Urodynamic investigation is the only method that can objectively assess the function and dysfunction of the LUT. In neuro-urological patients, invasive urodynamic investigation is even more challenging than in general patients. Any technical source of artefacts must be critically considered. It is essential to maintain the quality of the urodynamic recording and its interpretation [1]. Same session repeat urodynamic investigations are crucial in clinical decision making, since repeat measurements may yield completely different results [102].

In patients at risk for AD, it is advisable to measure blood pressure during the urodynamic study [103]. The rectal ampulla should be empty of stool before the start of the investigation. All urodynamic findings must be reported in detail and performed, according to the ICS technical recommendations and standards [1, 104].

3.3.7.2 Urodynamic tests

Free uroflowmetry and assessment of residual urine: Provides a first impression of the voiding function and is compulsory prior to planning any invasive urodynamics in patients able to void. For reliable information, it should be repeated at least two to three times [1]. Possible pathological findings include a low flow rate, low voided volume, intermittent flow, hesitancy and residual urine. Care must be taken when assessing the results in patients unable to void in a normal position, as both flow pattern and rate may be modified by inappropriate positions.

Filling cystometry: This test is the only method for quantifying the patient's filling function. The status of LUT function must be documented during the filling phase. However, this technique has limited use as a solitary

procedure. It is much more effective combined with bladder pressure measurement during micturition and is even more effective in video-urodynamics.

The bladder should be empty at the start of filling. A physiological filling rate should be used with body-warm saline. Possible pathological findings include DO, low bladder compliance, abnormal bladder sensations, incontinence, and an incompetent or relaxing urethra. There is some evidence that a bladder capacity < 200 mL and detrusor pressures over 75 cm H₂O are independent risk factors for UUT damage in patients with SCI [51].

Detrusor leak point pressure (DLPP) [105]: Appears to have no use as a diagnostic tool. Some positive findings have been reported [106, 107], but sensitivity is too low to estimate the risk to the UUT or for secondary bladder damage [108, 109].

Pressure flow study: Reflects the co-ordination between detrusor and urethra or pelvic floor during the voiding phase. It is even more powerful if combined with filling cystometry and video-urodynamics. Lower urinary tract function must be recorded during the voiding phase. Possible pathological findings include detrusor underactivity, bladder outlet obstruction (BOO), DSD, a high urethral resistance, and residual urine.

Most types of obstruction caused by neuro-urological disorders are due to DSD [110, 111], non-relaxing urethra, or non-relaxing bladder neck [112, 113]. Pressure-flow analysis mainly assesses the amount of mechanical obstruction caused by the urethra's inherent mechanical and anatomical properties and has limited value in patients with neuro-urological disorders.

Electromyography (EMG): Reflects the activity of the external urethral sphincter, the peri-urethral striated musculature, the anal sphincter and the striated pelvic floor muscles. Correct interpretation may be difficult due to artefacts introduced by other equipment. In the urodynamic setting, an EMG is useful as a gross indication of the patient's ability to control the pelvic floor. Possible pathological findings include inadequate recruitment upon specific stimuli (e.g. bladder filling, involuntary detrusor contractions, onset of voiding, coughing, Valsalva manoeuvre) suggesting a diagnosis of DSD [114].

Urethral pressure measurement: Has a very limited role in neuro-urological disorders. There is no consensus on parameters indicating pathological findings [115].

Video-urodynamics: Is the combination of filling cystometry and pressure flow studies with imaging. It is the optimum procedure for urodynamic investigation in neuro-urological disorders [5]. Possible pathological findings include all those described in the cystometry and the pressure flow study sections, and any morphological pathology of the LUT and reflux to the UUT [116].

Ambulatory urodynamics: This is the functional investigation of the urinary tract, which predominantly uses the natural filling of the urinary tract to reproduce the patient's normal activity. Although this type of study might be considered when conventional urodynamics does not reproduce the patient's symptoms, its role in the neuro-urological patient still needs to be determined [117, 118].

Triggered tests during urodynamics: Lower urinary tract function can be provoked by coughing, triggered voiding, or anal stretch. Fast-filling cystometry with cooled saline (the 'ice water test') will discriminate between upper and lower motor neuron lesions [119, 120]. Patients with UMNL develop a detrusor contraction if the detrusor is intact, while patients with LMNL do not. However, the test does not seem to be fully discriminative in other types of patients [121].

Previously, a positive bethanechol test [122] (detrusor contraction > 25 cm H₂O) was thought to indicate detrusor denervation hypersensitivity and the muscular integrity of an acontractile detrusor. However, in practice, the test has given equivocal results. A variation of this method was reported using intravesical electromotive administration of the bethanechol [123], but there was no published follow-up. Currently, there is no indication for this test.

3.3.7.3 Specialist uro-neurophysiological tests

The following tests are advised as part of the neurological work-up [124]:

- electromyography (in a neurophysiological setting) of pelvic floor muscles, urethral sphincter and/or anal sphincter;
- nerve conduction studies of pudendal nerve;
- reflex latency measurements of bulbocavernosus and anal reflex arcs;
- evoked responses from clitoris or glans penis;
- sensory testing on bladder and urethra.

Other elective tests, for specific conditions, may become obvious during the work-up and urodynamic investigations.

3.3.7.4 Summary of evidence and recommendations for urodynamics and uro-neurophysiology

Summary of Evidence	LE
Urodynamic investigation is the only method that can objectively assess the (dys-)function of the LUT.	2a
Video-urodynamics is the optimum procedure for urodynamic investigation in neuro-urological disorders.	4
Specific uro-neurophysiological tests are elective procedures and should only be carried out in specialised settings.	4

Recommendations	Strength rating
Perform a urodynamic investigation to detect and specify lower urinary tract (dys-)function, use same session repeat measurement as it is crucial in clinical decision making.	Strong
Non-invasive testing is mandatory before invasive urodynamics is planned.	Strong
Use video-urodynamics for invasive urodynamics in neuro-urological patients. If this is not available, then perform a filling cystometry continuing into a pressure flow study.	Strong
Use a physiological filling rate and body-warm saline.	Strong

3.3.8 Renal function

In many patients with neuro-urological disorders, the UUT is at risk, particularly in patients who develop high detrusor pressure during the filling phase. Although effective treatment can reduce this risk, there is still a relatively high incidence of renal morbidity [125]. Patients with SCI or SB have a higher risk of developing renal failure compared with patients with slowly progressive non-traumatic neurological disorders, such as MS and Parkinson's disease (PD) [126].

Caregivers must be informed of this risk and instructed to watch carefully for any signs or symptoms of a possible deterioration in the patient's renal function. In patients with poor muscle mass cystatin C based GFR is more accurate in detecting chronic kidney disease than serum creatinine estimated GFR [127, 128]. There are no high level evidence publications available which show the optimal management to preserve renal function in these patients [129].

3.4 Disease management

3.4.1 Introduction

The primary aims for treatment of neuro-urological symptoms, and their priorities, are [130, 131]:

- protection of the UUT;
- achievement (or maintenance) of urinary continence;
- restoration of the LUT function;
- improvement of the patient's QoL.

Further considerations are the patient's disability, cost-effectiveness, technical complexity and possible complications [131].

Renal failure is the main mortality factor in SCI patients who survive the trauma [132, 133]. Keeping the detrusor pressure during both the filling and voiding phases within safe limits significantly reduces the mortality from urological causes in these patients [134-136] and has consequently become the top priority in the treatment of patients with neuro-urological symptoms [130, 131].

In patients with high detrusor pressure during the filling phase (DO, low bladder compliance), treatment is aimed primarily at conversion of an overactive, high-pressure bladder into a low-pressure reservoir despite the resulting residual urine [130]. Reduction of the detrusor pressure contributes to urinary continence, and consequently to social rehabilitation and QoL. It is also pivotal in preventing UTIs [137, 138]. Complete continence, however, cannot always be obtained.

3.4.2 Non-invasive conservative treatment

3.4.2.1 Assisted bladder emptying - Credé manoeuvre, Valsalva manoeuvre, triggered reflex voiding

Incomplete bladder emptying is a serious risk factor for UTI, high intravesical pressure during the filling phase, and incontinence. Methods to improve the voiding process are therefore practiced.

Bladder expression (Credé manoeuvre) and voiding by abdominal straining (Valsalva manoeuvre): The downwards movement of the lower abdomen by suprapubic compression (Credé) or by abdominal straining (Valsalva) leads to an increase in intravesical pressure, and generally also causes a reflex sphincter contraction [139, 140]. The latter may increase bladder outlet resistance and lead to inefficient emptying. The high pressures created during these procedures are hazardous for the urinary tract [141, 142]. Therefore, their use should be discouraged unless urodynamics show that the intravesical pressure remains within safe limits [131].

Long-term complications are unavoidable for both methods of bladder emptying [140]. The already weak pelvic floor function may be further impaired, thus introducing or exacerbating already existing SUI [142].

Triggered reflex voiding: Stimulation of the sacral or lumbar dermatomes in patients with UMNL can elicit a reflex detrusor contraction [142]. The risk of high pressure voiding is present and interventions to decrease outlet resistance may be necessary [143]. Triggering can induce AD, especially in patients with high level SCI (at or above Th 6) [144]. All assisted bladder emptying techniques require low outlet resistance. Even then, high detrusor pressures may still be present. Hence, patients need dedicated education and close urodynamic and urological surveillance [142, 145-147].

Note: In the literature, including some of the references cited here, the concept “reflex voiding” is sometimes used to cover all three assisted voiding techniques described in this section.

External appliances: Social continence may be achieved by collecting urine during incontinence, for instance using pads [131]. Condom catheters with urine collection devices are a practical method for men [131]. The infection risk must be closely observed [131]. The penile clamp is absolutely contraindicated in case of DO or low bladder compliance because of the risk of developing high intravesical pressure and pressure sores/necrosis in cases of altered/absent sensations.

3.4.2.2 *Neuro-urological rehabilitation*

3.4.2.2.1 *Bladder rehabilitation including electrical stimulation*

The term bladder rehabilitation summarises treatment options that aim to re-establish bladder function in patients with neuro-urological symptoms. Strong contraction of the urethral sphincter and/or pelvic floor, as well as anal dilatation, manipulation of the genital region, and physical activity inhibit micturition in a reflex manner [131, 148]. The first mechanism is affected by activation of efferent nerve fibres, and the latter ones are produced by activation of afferent fibres [108]. Electrical stimulation of the pudendal nerve afferents strongly inhibits the micturition reflex and detrusor contraction [149]. This stimulation might then support the restoration of the balance between excitatory and inhibitory inputs at the spinal or supraspinal level [131, 150]. Evidence for bladder rehabilitation using electrical stimulation in neurological patients is mainly based on small non-comparative studies with high risk of bias.

Peripheral temporary electrostimulation: Tibial nerve stimulation and transcutaneous electrical nerve stimulation (TENS) might be effective and safe for treating neurogenic lower urinary tract dysfunction, but more reliable evidence from well-designed randomised controlled trials (RCTs) is required to reach definitive conclusions [150, 151]. In post-stroke patients TENS has been shown to effectively improve urodynamic findings and QoL [152].

Peripheral temporary electrostimulation combined with pelvic floor muscle training and biofeedback: In MS patients, combining active neuromuscular electrical stimulation with pelvic floor muscle training and EMG biofeedback can achieve a substantial reduction of neuro-urological symptoms [153]. This treatment combination seems to be more effective than either therapy alone [154, 155].

Intravesical electrostimulation: Intravesical electrostimulation can increase bladder capacity and improve bladder compliance and bladder filling sensation in patients with incomplete SCI or myelomeningocele (MMC) [156]. In patients with neurogenic detrusor underactivity, intravesical electrostimulation may also improve voiding and reduce residual volume [157, 158].

Repetitive transcranial magnetic stimulation: Although improvement of neuro-urological symptoms has been described in PD and MS patients, this technique is still under investigation [159, 160].

Summary: To date, bladder rehabilitation techniques are mainly based on electrical or magnetic stimulation. However, there is a lack of well-designed studies.

3.4.2.3 Drug treatment

A single, optimal, medical therapy for neuro-urological symptoms is not yet available. Commonly, a combination of different therapies (e.g. IC and antimuscarinic drugs) is advised to prevent urinary tract damage and improve long-term outcomes, particularly in patients with a suprasacral SCI or MS [142, 161-163].

3.4.2.3.1 Drugs for storage symptoms

Antimuscarinic drugs: They are the first-line choice for treating NDO, increasing bladder capacity and reducing episodes of UI secondary to NDO by the inhibition of parasympathetic pathways [131, 164-170]. Antimuscarinic drugs have been used for many years to treat patients with NDO [168, 169, 171], and the responses of individual patients to antimuscarinic treatment are variable. Despite a meta-analysis confirming the clinical and urodynamic efficacy of antimuscarinic therapy compared to placebo in adult NDO, a more recent integrative review has indicated that the information provided is still too limited for clinicians to be able to match trial data to the needs of individual patients with SCI mainly due to the lack of use of standardised clinical evaluation tools such as the American Spinal Injury Association, bladder diary and validated symptoms score [169, 172].

Higher doses or a combination of antimuscarinic agents may be an option to maximise outcomes in neurological patients [165, 166, 173-176]. However, these drugs have a high incidence of adverse events, which may lead to early discontinuation of therapy. Despite this, NDO patients have generally shown better treatment adherence compared to idiopathic DO patients [177].

Choice of antimuscarinic agent: Oxybutynin [131, 165, 166, 168, 169, 178], trospium [169, 175, 179], tolterodine [180] and propiverine [169, 181] are established, effective and well tolerated treatments even in long-term use [168, 169, 182, 183]. Darifenacin [184, 185] and solifenacin [186] have been evaluated in NDO secondary to SCI and MS [169, 184-186] with results similar to other antimuscarinic drugs. A pilot study using solifenacin in NDO due to PD showed an improvement in UI [187]. The relatively new drug, fesoterodine, an active metabolite of tolterodine, has also been introduced, even though to date there has been no published clinical evidence of its use in the treatment of neuro-urological disorders. Favourable results with the new drug imidafenacin have been reported in suprapontine as well as SCI patients [188, 189].

Side effects: Controlled-release antimuscarinics have some minor side effects, e.g. dry mouth [190]. It has been suggested that different ways of administration may help to reduce side effects [191]. A significant reduction in adverse events was observed for 30 mL 0.1% intravesical oxybutynine compared to 15 mg oral administration, per day [191]. Moreover, imidafenacin has been safely used in neurological patients with no worsening of cognitive function [188].

Other agents

Beta-3-adrenergic receptor agonists have recently been introduced and evaluated in OAB, but clinical experience in neuro-urological patients is limited [192]. Studies on safety and effectiveness in NDO are ongoing [193]. Depending on the results of these studies, combined therapy with antimuscarinics may be an attractive option [194]. In preliminary studies, improvements in daily incontinence rates, nocturia, daytime and 24 hour voids, as well as the low risk of adverse events, suggest that cannabinoids may be effective and safe in MS patients [195].

3.4.2.3.2 Drugs for voiding symptoms

Detrusor underactivity: Cholinergic drugs, such as bethanechol and distigmine, have been considered to enhance detrusor contractility and promote bladder emptying, but are not frequently used in clinical practice [196]. Only preclinical studies have documented the potential benefits of cannabinoid agonists on improving detrusor contractility when administered intravesically [197, 198].

Decreasing bladder outlet resistance: α -blockers (e.g. tamsulosin, naftopidil and silodosin) seem to be effective for decreasing bladder outlet resistance, post-void residual and AD [199-201].

Increasing bladder outlet resistance: Several drugs have shown efficacy in selected cases of mild SUI, but there are no high-level evidence studies in neurological patients [131].

3.4.2.4 Summary of evidence and recommendations for drug treatments

Summary of Evidence	LE
Long-term efficacy and safety of antimuscarinic therapy for neurogenic detrusor overactivity is well documented.	1a
A significant reduction in adverse events was observed for intravesical administration of oxybutynine compared to oral administration.	1a
Maximise outcomes for neurogenic detrusor overactivity by considering a combination of antimuscarinic agents.	3

Recommendations	Strength rating
Use antimuscarinic therapy as the first-line medical treatment for neurogenic detrusor overactivity.	Strong
Offer intravesical oxybutynin to neurogenic detrusor overactivity patients with poor tolerance to the oral route.	Strong
Prescribe α -blockers to decrease bladder outlet resistance.	Strong
Do not prescribe parasympathomimetics for underactive detrusor.	Strong

3.4.2.5 Minimally invasive treatment

3.4.2.5.1 Catheterisation

Intermittent self- or third-party catheterisation [202, 203] is the preferred management for neuro-urological patients who cannot effectively empty their bladders [131]. Sterile IC, as originally proposed by Guttman and Frankel [202], significantly reduces the risk of UTI and bacteriuria [131, 204, 205], compared with clean IC introduced by Lapidus *et al.* [203]. However, it has not yet been established whether or not the incidence of UTI, other complications and user satisfaction are affected by either sterile or clean IC, coated or uncoated catheters or by any other strategy.

Sterile IC cannot be considered a routine procedure [131, 205]. Aseptic IC is an alternative to sterile IC [206].

Contributing factors to contamination are insufficient patient education and the inherently greater risk of UTI in neuro-urological patients [131, 207-211]. The average frequency of catheterisations per day is four to six times [212] and the catheter size most often used is between 12-16 Fr. In aseptic IC, an optimum frequency of five times showed a reduction of UTI [212]. Ideally, bladder volume at catheterisation should, as a rule, not exceed 400-500 mL.

Indwelling transurethral catheterisation and, to a lesser extent, suprapubic cystostomy are associated with a range of complications as well as an enhanced risk for UTI [131, 213-220]. Therefore, both procedures should be avoided, when possible. Silicone catheters are preferred as they are less susceptible to encrustation and because of the high incidence of latex allergy in the neuro-urological patient population [221].

3.4.2.5.2 Summary of evidence and recommendations for catheterisation

Summary of Evidence	LE
Intermittent catheterisation is the standard treatment for patients who are unable to empty their bladder.	3
Indwelling transurethral catheterisation and suprapubic cystostomy are associated with a range of complications as well as an enhanced risk for UTI.	3

Recommendations	Strength rating
Use intermittent catheterisation, whenever possible aseptic technique, as a standard treatment for patients who are unable to empty their bladder.	Strong
Thoroughly instruct patients in the technique and risks of intermittent catheterisation.	Strong
Avoid indwelling transurethral and suprapubic catheterisation whenever possible.	Strong

3.4.2.5.3 Intravesical drug treatment

To reduce DO, antimuscarinics can also be administered intravesically [191, 222-225]. The efficacy, safety and tolerability of intravesical administration of 0.1% oxybutynin hydrochloride compared to its oral administration for treatment of NDO has been demonstrated in a recent randomised controlled study [191]. This approach

may reduce adverse effects due to the fact that the antimuscarinic drug is metabolised differently [222] and a greater amount is sequestered in the bladder, even more than with electromotive administration [223].

The vanilloids, capsaicin and resiniferatoxin, desensitise the C-fibres and thereby decrease DO, for a period of a few months, until the sensation of these fibres has been restored [226-228]. The dosage is 1-2 mMol capsaicin in 100 mL 30% alcohol, or 10-100 nMol resiniferatoxin in 100 mL 10% alcohol for 30 minutes. Resiniferatoxin has about a 1,000-fold potency compared to capsaicin, with less pain during the instillation, and is effective in a patient refractory to capsaicin. Clinical studies have shown that resiniferatoxin has limited clinical efficacy compared to botulinum toxin A (BTX-A) injections in the detrusor [227]. Currently, there is no indication for the use of these substances, which are not licensed for intravesical treatment.

3.4.2.5.4 Botulinum toxin injections in the bladder

Botulinum toxin A causes a long-lasting but reversible chemical denervation that lasts for about nine months [229, 230]. The toxin injections are mapped over the detrusor in a dosage that depends on the preparation used. Botulinum toxin A has been proven effective in patients with neuro-urological disorders due to MS, SCI and Parkinson's disease in multiple RCTs and meta-analyses [231-233]. Repeated injections seem to be possible without loss of efficacy, even after initial low response rates, based on years of follow up [229, 234-236]. A switch between different toxin variations can improve responsiveness [237]. The most frequent side effects are UTIs and elevated post void residual [234, 238]. Intermittent catheterisation may become necessary. Rare but severe adverse events include AD and respiratory problems. Generalised muscular weakness may occur [229, 235, 238]. Current research focuses on different delivery approaches to injection such as liposome encapsulated Botulinum toxin to decrease side effects [239].

3.4.2.5.5 Bladder neck and urethral procedures

Reduction of the bladder outlet resistance may be necessary to protect the UUT. This can be achieved by chemical denervation of the sphincter or by surgical interventions (bladder neck or sphincter incision or urethral stent). Incontinence may result and can be managed by external devices (see Section 3.4.2.1).

Botulinum toxin A: This can be used to treat DSD effectively by injection at a dose that depends on the preparation used. The dyssynergia is abolished for a few months, necessitating repeat injections. The efficacy of this treatment has been reported to be high and with few adverse effects [240-242]. However, a recent Cochrane report concluded that, because of limited evidence, future RCTs assessing the effectiveness of BTX-A injections also need to address the uncertainty about the optimal dose and mode of injection [243]. In addition, this therapy is not licensed.

Balloon dilatation: Favourable immediate results were reported [244], but there have been no further reports since 1994 therefore, this method is no longer recommended.

Sphincterotomy: By staged incision, bladder outlet resistance can be reduced without completely losing the closure function of the urethra [130, 131, 245]. Different techniques are used, and laser treatment appears to be advantageous [246, 247]. Sphincterotomy needs to be repeated at regular intervals in many patients [248], but it is efficient and does not cause severe adverse effects [130, 244]. Secondary narrowing of the bladder neck may occur, for which combined bladder neck incision might be considered [249].

Bladder neck incision: This is indicated only for secondary changes at the bladder neck (fibrosis) [130, 246]. This procedure is not recommended in patients with detrusor hypertrophy, which causes thickening of the bladder neck [130].

Stents: Implantation of urethral stents results in continence being dependent on adequate closure of the bladder neck [131]. The results are comparable with sphincterotomy and the stenting procedure has a shorter duration of surgery and hospital stay [250, 251]. However, the costs [130], possible complications and re-interventions [252, 253] are limiting factors in their use [254-257].

Increasing bladder outlet resistance: This can improve the continence condition. Despite early positive results with urethral bulking agents, a relative early loss of continence is reported in patients with neuro-urological disorders [131, 258, 259].

Urethral inserts: Urethral plugs or valves for the management of (female) stress incontinence have not been applied in neuro-urological patients. The experience with active pumping urethral prosthesis for treatment of the underactive or acontractile detrusor were disappointing [260].

3.4.2.5.6 Summary of evidence and recommendations for minimal invasive treatment

Summary of Evidence	LE
Botulinum toxin A has been proven effective in patients with neuro-urological disorders due to MS or SCI in multiple RCTs and meta-analyses.	1a
Bladder neck incision is indicated only for secondary changes (fibrosis) at the bladder neck.	4

Recommendations	Strength rating
Use botulinum toxin injection in the detrusor to reduce neurogenic detrusor overactivity in multiple sclerosis or spinal cord injury patients if antimuscarinic therapy is ineffective.	Strong
Bladder neck incision is effective in a fibrotic bladder neck.	Strong

3.4.3 **Surgical treatment**

3.4.3.1 *Bladder neck and urethral procedures*

Increasing the bladder outlet resistance has the inherent risk of causing high intravesical pressure. Procedures to treat sphincteric incontinence are therefore suitable only when the detrusor activity can be controlled and when no significant reflux is present. A simultaneous bladder augmentation and IC may be necessary [131].

Urethral sling: Various materials have been used for this procedure with enduring positive results. The procedure is established in women with the ability to self-catheterise [131, 261-264]. There is growing evidence that synthetic slings can be used effectively with acceptable medium to long-term results and minimal morbidity in neurological patients [265, 266]. Besides the pubovaginal sling, which has been considered the procedure of choice in this subgroup of patients, recent reports suggest that both the transobturator and the retropubic approaches may also be considered, with similar failure rates and a reduction in the need for IC. However, for both approaches a higher incidence of *de novo* urgency was reported [266, 267]. In men, both autologous and synthetic slings may also be an alternative [268-272].

Artificial urinary sphincter: This device was introduced by Light and Scott for patients with neuro-urological disorders [273]. It has stood the test of time and acceptable long-term outcomes can be obtained [274]. However, the complication rates and re-operation rates are higher than in non-neurogenic patient groups (up to 60%), so it is advisable that patients are conscientiously informed about the success rates as well as the complications that may occur after the procedure [275, 276]. Re-interventions are commonly due to infection, urethral atrophy or erosion and mechanical failure.

Adjustable continence device - ProACT/ACT®: The efficacy of this device has been reported mainly in post-prostatectomy incontinence. A small retrospective study including sixteen patients with neurogenic stress urinary incontinence of differing origins (SCI, MMC, ischemic myelopathy, cauda equine syndrome, laminectomy), reported a marginally lower cure rate in neurological patients when compared to non-neurological patients [277].

Functional sphincter augmentation: Transposing the gracilis muscle to the bladder neck [278] or proximal urethra [279], can enable the possible creation of a functional autologous sphincter by electrical stimulation [278-280]. Therefore, raising the prospect of restoring control over the urethral closure.

Bladder neck and urethra reconstruction: The classical Young-Dees-Leadbetter procedure [281] for bladder neck reconstruction in children with bladder exstrophy, and Kropp urethra lengthening [282] improved by Salle [283], are established methods to restore continence provided that IC is practiced and/or bladder augmentation is performed [131, 284].

Urethral inserts: See section 3.4.2.5.5.

3.4.3.2 *Denervation, deafferentation, sacral neuromodulation*

Sacral rhizotomy, also known as sacral deafferentation, has achieved some success in reducing DO [285-287], but nowadays, it is used mostly as an adjuvant to sacral anterior root stimulation (SARS) [288-292]. Alternatives to rhizotomy are sought in this treatment combination [293-295].

Sacral anterior root stimulation is aimed at producing detrusor contraction. The technique was developed by Brindley [296] and is only applicable to complete lesions above the implant location, as its stimulation amplitude is over the pain threshold. The urethral sphincter efferents are also stimulated, but

because the striated muscle relaxes faster than the smooth muscle of the detrusor, so-called “post-stimulus voiding” occurs. This approach has been successful in highly selected patients [289, 297, 298]. By changing the stimulation parameters, this method can also induce defecation or erection. A recent study reports that Charcot spinal arthropathy should be considered as a potential long-term complication of SARS, leading to spinal instability and to SARS dysfunction [299].

Sacral neuromodulation [300] might be effective and safe for treating neuro-urological symptoms, but there is a lack of RCTs and it is unclear which neurological patients are most suitable [301-304].

3.4.3.3 *Bladder covering by striated muscle*

When the bladder is covered by striated muscle, that can be stimulated electrically, or ideally that can be contracted voluntarily, voiding function can be restored to an acontractile bladder. The rectus abdominis [305] and latissimus dorsi [306] have been used successfully in patients with neuro-urological symptoms [307, 308].

3.4.3.4 *Bladder augmentation*

The aim of auto-augmentation (detrusor myectomy) is to reduce DO or improve low bladder compliance. The advantages are: low surgical burden, low rate of long-term adverse effects, positive effect on patient QoL, and it does not preclude further interventions [130, 131, 309-312].

Replacing or expanding the bladder by intestine or other passive expandable coverage will improve bladder compliance and at least reduce the pressure effect of DO [313, 314]. Improved QoL and stable renal function has been reported during long-term follow-up [315]. Long term complications included bladder perforation (1.9%), mucus production (12.5%), metabolic abnormalities (3.35%), bowel dysfunction (15%), and stone formation (10%) [315].

The procedure should be used with caution in patients with neuro-urological symptoms, but may become necessary if all less-invasive treatment methods have failed. Special attention should be paid to patients with pre-operative renal scars since metabolic acidosis can develop [316]. Bladder substitution, even by performing a supratrigonal cystectomy [317], to create a low-pressure reservoir is indicated in patients with a severely thick and fibrotic bladder wall [131]. Intermittent catheterisation may become necessary after this procedure. The long-term scientific evidence shows that bladder augmentation is a highly successful procedure that stabilises renal function and prevents anatomical deterioration; however, lifelong follow-up is essential in this patient group given the significant morbidity associated with this procedure [315].

3.4.3.5 *Urinary diversion*

When no other therapy is successful, urinary diversion must be considered for the protection of the UUT and for the patient's QoL [131].

Continent diversion: This should be the first choice for urinary diversion. Patients with limited dexterity may prefer a stoma instead of using the urethra for catheterisation. For cosmetic reasons, the umbilicus is often used for the stoma site [318-323]. A systematic review of the literature concluded that continent catheterisable tubes/stomas are an effective treatment option in neuro-urological patients unable to perform intermittent self-catheterisation through the urethra [324]. However, the complication rates were significant with 85/213 post-operative events requiring re-operation [324]. Tube stenosis occurred in 4-32% of the cases. Complications related to concomitant procedures (augmentation cystoplasty, pouch) included neovesicocutaneous fistulae (3.4%), bladder stones (20-25%), and bladder perforations (40%). In addition, data comparing HRQoL before and after surgery were not reported [324].

Incontinent diversion: If catheterisation is impossible, incontinent diversion with a urine-collecting device is indicated. Ultimately, it could be considered in patients who are wheelchair bound or bed-ridden with intractable and untreatable incontinence, in patients with LUT destruction, when the UUT is severely compromised, and in patients who refuse other therapy [131]. An ileal segment is used for the deviation in most cases [131, 325-328]. Patients gain better functional status and QoL after surgery [329].

Undiversion: Long-standing diversions may be successfully undiverted or an incontinent diversion changed to a continent one with the emergence of new and better techniques for control of detrusor pressure and incontinence [131]. The patient must be carefully counselled and must comply meticulously with the instructions [131]. Successful undiversion can then be performed [330].

3.4.3.6 Summary of evidence and recommendations for surgical treatment

Summary of Evidence	LE
Bladder augmentation is an effective option to decrease detrusor pressure and increase bladder capacity, when all less-invasive treatment methods have failed.	3
Urethral sling placement is an established procedure, with acceptable medium to long-term results, in women with the ability to self-catheterise.	3
Artificial urinary sphincter insertion is a viable option, with acceptable long-term outcomes, in males. The complication and re-operation rates are higher in neuro-urological patients therefore; patients must be adequately informed regarding the success rates as well as the complications that may occur following the procedure.	3

Recommendations	Strength rating
Perform bladder augmentation in order to treat refractory neurogenic detrusor overactivity.	Strong
Place an autologous urethral sling in female patients with neurogenic stress urinary incontinence who are able to self-catheterise.	Strong
Insert an artificial urinary sphincter in male patients with neurogenic stress urinary incontinence.	Strong

3.5 Urinary tract infection in neuro-urological patients

3.5.1 Epidemiology, aetiology and pathophysiology

Urinary tract infection is the onset of signs and/or symptoms accompanied by laboratory findings of a UTI (bacteriuria, leukocyturia and positive urine culture) [319]. There are no evidence-based cut-off values for the quantification of these findings. The published consensus is that a significant bacteriuria in persons performing IC is present with $> 10^2$ cfu/mL, $> 10^4$ cfu/mL in clean-void specimens and any detectable concentration in suprapubic aspirates. Regarding leukocyturia, 10 or more leukocytes in centrifuged urine samples per microscopic field (400x) are regarded as significant [319].

The pathogenesis of UTI in neuro-urological patients is multifactorial. Male gender seems to be a risk factor for febrile UTI [331]. Several etiological factors have been described: altered intrinsic defence mechanisms, impaired washout and catheterisation [332]. Poor glycemic control has been established as a risk factor for UTI in women with type 1 diabetes [333]. The exact working mechanisms, however, still remain unknown. The presence of asymptomatic bacteriuria in SCI patients is higher than in the general population, and varies depending on bladder management. Prevalence of bacteriuria in those performing clean IC varies from 23-89% [334]. Sphincterotomy and condom catheter drainage has a 57% prevalence [335]. Asymptomatic bacteria should not be routinely screened for in this population [336].

Individuals with neuro-urological symptoms, especially those with SCI, may have other signs and symptoms in addition to or instead of traditional signs and symptoms of a UTI in able-bodied individuals. Other problems, such as AD, may develop or worsen due to a UTI [337]. The most common signs and symptoms suspicious of a UTI in those with neuro-urological disorders are fever, new onset or increase in incontinence, including leaking around an indwelling catheter, increased spasticity, malaise, lethargy or sense of unease, cloudy urine with increased urine odour, discomfort or pain over the kidney or bladder, dysuria, or AD [337, 338].

3.5.2 Diagnostic evaluation

The gold standard for diagnosis is urine culture and urinalysis. A dipstick test may be more useful to exclude than to prove UTI [339, 340]. As bacterial strains and resistance patterns in persons with neuro-urological disorders may differ from those of able-bodied patients, microbiologic testing is mandatory [341].

3.5.3 Disease management

Bacteriuria in patients with neuro-urological disorders should not be treated. Treatment of asymptomatic bacteriuria results in significantly more resistant bacterial strains without improving the outcome [342]. Urinary tract infections in persons with neuro-urological disorders are by definition a complicated UTI; therefore, single-dose treatment is not advised. There is no consensus in the literature about the duration of treatment as it depends on the severity of the UTI and the involvement of kidneys and the prostate. Generally, a five to seven day course of antibiotic treatment is advised, which can be extended up to fourteen days according to the extent of the infection [342]. The choice of antibiotic therapy should be based on the results of the microbiologic testing. If immediate treatment is mandatory (e.g. fever, septicaemia, intolerable clinical

symptoms, extensive AD), the choice of treatment should be based on local and individual resistance profiles [343].

3.5.3.1 Recurrent UTI

Recurrent UTI in patients with neuro-urological disorders may indicate suboptimal management of the underlying functional problem, e.g. high bladder pressure during storage and voiding, incomplete voiding or bladder stones. The improvement of bladder function, by treating DO by BTX-A injection in the detrusor [344], and the removal of bladder stones or other direct supporting factors, especially indwelling catheters, as early as possible, are mandatory [341].

3.5.3.2 Prevention

If the improvement of bladder function and removal of foreign bodies/stones is not successful, additional UTI prevention strategies should be utilised. The use of hydrophilic catheters was associated with a lower rate of UTI in a recent meta-analysis [345]. Bladder irrigation has not been proven effective [346].

Various medical approaches have been tested for UTI prophylaxis in patients with neuro-urological disorders. The benefit of cranberry juice for the prevention of UTI could not be demonstrated in RCTs [347]. Methenamine hippurate is not effective in individuals with neuro-urological symptoms [348]. There is no sufficient evidence to support the use of L-methionine for urine acidification to prevent recurrent UTI [349]. There is only weak evidence that oral immunotherapy reduces bacteriuria in patients with SCI, and no evidence that recurrent UTIs are reduced [350]. Low-dose, long-term, antibiotic prophylaxis cannot reduce UTI frequency, but increases bacterial resistance and is therefore not recommended [342].

An application scheme of antibiotic substances for antibiotic prophylaxis provided long-term positive results, but the results of this trial need to be confirmed in further studies [351]. Another possible future option, the inoculation of apathogenic *Escherichia coli* strains into the bladder, has provided positive results in initial studies, but because of the paucity of data [352], cannot be recommended as a treatment option.

In summary, based on the criteria of evidence-based medicine, there is currently no preventive measure for recurrent UTI in patients with neuro-urological disorders that can be recommended without limitations. Therefore, individualised concepts should be taken into consideration, including immunostimulation, phytotherapy and complementary medicine [353]. Prophylaxis in patients with neuro-urological disorders is important to pursue, but since there are no data favouring one approach over another, prophylaxis is essentially a trial and error approach.

3.5.4 Summary of evidence and recommendations for the treatment of UTI

Summary of Evidence	LE
Treatment of asymptomatic bacteriuria results in significantly more resistant bacterial strains without improving patient outcome.	1a
Low-dose, long-term, antibiotic prophylaxis does not reduce UTI frequency, but increases bacterial resistance.	2a
Recurrent UTI in patients with neuro-urological disorders may indicate suboptimal management of the underlying functional problem. Improvement of bladder function as early as possible is mandatory.	3
There is currently no preventive measure for recurrent UTI in patients with neuro-urological disorders that can be recommended without limitations.	3

Recommendations	Strength rating
Do not screen for or treat asymptomatic bacteriuria in patients with neuro-urological disorders.	Strong
Avoid the use of long-term antibiotics for recurrent urinary tract infections (UTIs).	Strong
In patients with recurrent UTI, optimise treatment of neuro-urological symptoms and remove foreign bodies (e.g. stones, indwelling catheters) from the urinary tract.	Strong
Individualise UTI prophylaxis in patients with neuro-urological disorders as there is no optimal prophylactic measure available.	Strong

3.6 Sexual (dys)function and fertility

These Guidelines specifically focus on sexual dysfunction and infertility in patients with a neurological disease [354, 355]. Non-neurogenic, male sexual dysfunction and infertility are covered in separate EAU Guidelines

[356, 357]. In neuro-urological patients sexual problems can be identified at three levels: primary (direct neurological damage), secondary (general physical disabilities) and tertiary (psychosocial and emotional issues) sexual dysfunction [358]. Adopting a systematic approach, such as the PLISSIT model (Permission, Limited Information, Specific Suggestions and Intensive Therapy) [359], provides a framework for counselling and treatment involving a stepwise approach to the management of neurogenic sexual dysfunction. Sexual dysfunction is associated with neurogenic LUT dysfunction in patients with MS [360] and spina bifida [361]. Although various PROMs are available to evaluate sexual function, the evidence for good PROMs is limited and studies with high methodological quality are needed [362].

3.6.1 **Erectile dysfunction (ED)**

3.6.1.1 *Phosphodiesterase type 5 inhibitors (PDE5Is)*

Questions:

- What is the effectiveness of the various PDE5Is in the different neuro-urological patient groups?
- What common side-effects are described?

Evidence:

Phosphodiesterase type 5 inhibitors (PDE5Is) are recommended as first-line treatment in neurogenic ED [354, 355]. In SCI patients, tadalafil, vardenafil and sildenafil have all improved retrograde ejaculation and improved erectile function and satisfaction on IIEF-15. Tadalafil 10 mg was shown to be more effective than sildenafil 50 mg. All currently available PDE5Is appear to be effective and safe, although there are no high level evidence studies in neuro-urological patients investigating the efficacy and side effects across different PDE5Is, dosages and formulations [363].

For MS patients two studies reported significant improvement in ED when using sildenafil and tadalafil. One study, however, showed no improvement in ED with sildenafil.

In PD normal erectile function was described in over half of the patients using sildenafil 100 mg and a significant improvement in IIEF-15 score was found compared to placebo. While most neuro-urological patients require long-term therapy for ED some have a low compliance rate or stop therapy because of side effects [364, 365], most commonly headache and flushing [355]. In addition, PDE5Is may induce relevant hypotension in patients with tetraplegia/high-level paraplegia and multiple system atrophy [364, 365]. As a prerequisite for successful PDE5I-therapy, some residual nerve function is required to induce erection. Since many patients with SCI use on-demand nitrates for the treatment of AD, they must be counselled that PDE5Is are contraindicated when using nitrate medication.

3.6.1.2 *Drug therapy other than PDE5Is*

Fampridine to treat neurogenic spasticity has been shown to be beneficial in improving ED in two domains of the IIEF-15 in SCI and MS patients, however, with a significant discontinuation rate due to severe adverse events [366]. Sublingual apomorphine was shown to have poor results on ED in SCI patients and side-effects in half of the patients [367]. In PD pergolide mesylate showed a significant improvement in IIEF-15 scores up to twelve months follow-up [368].

3.6.1.3 *Mechanical devices*

Mechanical devices (vacuum tumescence devices and penile rings) may be effective but are less popular [369-373].

3.6.1.4 *Intracavernous injections and intraurethral application*

Patients not responding to oral drugs may be offered intracavernous injections (alprostadil, papaverine and phentolamine) that have been shown to be effective in a number of neurological conditions, including SCI, MS, and diabetes mellitus [374-380] but their use requires careful dose titration and some precautions. Complications of intracavernous drugs include pain, priapism and corpora cavernosa fibrosis.

Intracavernous vasoactive drug injection is the first-line therapeutic option in patients taking nitrate medications, as well as those with concerns about drug interactions with PDE5Is, or in whom PDE5Is are ineffective. The impact of intracavernous injections on ejaculation and orgasmic function, their early use for increasing the recovery rate of a spontaneous erection, and their effectiveness and tolerability in the long-term are unclear [364]. Intra-urethral alprostadil application is an alternative but a less effective route of administration [376, 381].

3.6.1.5 *Sacral neuromodulation*

Sacral neuromodulation for LUT dysfunction may improve sexual function but high level evidence studies are lacking [372, 355].

3.6.1.6 Penile prostheses

Penile prostheses may be considered for treatment of neurogenic ED when all conservative treatments have failed. At a mean follow-up of seven years 83.7% of patients with SCI were able to have sexual intercourse [355]. Serious complications, including infection and prosthesis perforation, may occur in about 10% of patients, depending on implant type [382-384].

3.6.1.7 Summary of evidence and recommendations for erectile dysfunction

Summary of Evidence	LE
The long-term efficacy and safety of oral PDE5Is for the treatment of ED is well documented.	1b
Intracavernous vasoactive drug injections have been shown to be effective in a number of neurological conditions, including SCI and MS; however, their use requires careful dose titration and precautions.	3
Mechanical devices (vacuum tumescence devices and penile rings) may be effective but are less popular.	3
Reserve penile prostheses for selected patients, those in which all conservative treatments have failed, with neurogenic ED.	4

Recommendations	Strength rating
Prescribe oral phosphodiesterase type 5 inhibitors as first-line medical treatment in neurogenic erectile dysfunction.	Strong
Give intracavernous injections of vasoactive drugs (alone or in combination) as second-line medical treatment in neurogenic erectile dysfunction.	Strong
Offer mechanical devices such as vacuum devices and rings to patients with neurogenic erectile dysfunction.	Strong

3.6.2 Male fertility

Male fertility can be compromised in the neurological patient by ED, ejaculation disorder, impaired sperm quality or various combinations of these three disorders. Among the major conditions contributing to neurogenic infertility are pelvic and retroperitoneal surgery, diabetes mellitus, SB, MS and SCI [385]. Erectile dysfunction is managed as described previously. Retrograde ejaculation may be reversed by sympathomimetic agents contracting the bladder neck, including imipramine, ephedrine, pseudoephedrine, and phenylpropanolamine [385]. The use of a balloon catheter to obstruct the bladder neck may be effective in obtaining antegrade ejaculation [386]. If antegrade ejaculation is not achieved, the harvest of semen from the urine may be considered [387].

Prostatic massage is safe and easy to use for obtaining semen in men with lesions above Th 10 [388]. In several patients, vibrostimulation or transrectal electroejaculation are needed for sperm retrieval [385, 389, 390]. Semen retrieval is more likely with vibrostimulation in men with lesions above Th 10 [391-393]. In men with SCI, especially at or above Th 6, AD might occur during sexual activity and ejaculation [394, 395]; patients at risk and fertility clinics must be informed and aware of this potentially life-threatening condition. In SCI patients the use of oral midodrine can improve sperm retrieval at vibrostimulation [396].

In men with MS, use of disease modifying drugs during the conception phase, has not been associated with altered pregnancy outcomes [397]. Surgical procedures, such as, microsurgical epididymal sperm aspiration (MESA) or testicular sperm extraction (TESE), may be used if vibrostimulation and electroejaculation are not successful [398, 399]. Pregnancy rates in patients with SCI are lower than in the general population, but since the introduction of intracytoplasmic sperm injection (ICSI), men with SCI now have a good chance of becoming biological fathers [400-402].

3.6.2.1 Sperm quality and motility

The following has been reported on sperm quality and motility:

- bladder management with clean IC may improve semen quality compared to indwelling catheterisation, reflex voiding or bladder expression [403];
- in SCI patients sperm quality decreases at the early post traumatic phase demonstrating lower spermatozoid vitality (necrospermia), reduced motility (asthenospermia) and leucospermia [398];
- long-term valproate treatment for epilepsy negatively influences sperm count and motility [404];
- vibrostimulation produces samples with better sperm motility than electrostimulation [405, 406];
- electroejaculation with interrupted current produces better sperm motility than continuous current [407];
- freezing of sperm is unlikely to improve fertility rates in men with SCI [408].

3.6.2.2 Summary of evidence and recommendations for male fertility

Summary of Evidence	LE
Vibrostimulation and transrectal electroejaculation have been shown to be effective for sperm retrieval in neuro-urological patients.	1b
Surgical procedures, such as, microsurgical epididymal sperm aspiration or testicular sperm extraction, may be used if vibrostimulation and electroejaculation are not successful.	3
In men with SCI at or above Th 6, AD might occur during sexual activity and ejaculation.	3

Recommendations	Strength rating
Perform vibrostimulation and transrectal electroejaculation for sperm retrieval in men with spinal cord injury.	Strong
Perform microsurgical epididymal sperm aspiration, testicular sperm extraction and intracytoplasmic sperm injection after failed vibrostimulation and/or transrectal electroejaculation in men with spinal cord injury.	Strong
Counsel men with spinal cord injury at or above Th 6 and fertility clinics about the potentially life-threatening condition of autonomic dysreflexia.	Strong

3.6.3 Female sexuality

The most relevant publications on neurogenic female sexual dysfunction are in women with SCI and MS. After SCI, about 65-80% of women continue to be sexually active, but to a much lesser extent than before the injury, and about 25% report a decreased satisfaction with their sexual life [409-411]. Although sexual dysfunction is very common in women with MS, it is still often overlooked by medical professionals [412, 413].

The greatest physical barrier to sexual activity is UI. A correlation has been found between the urodynamic outcomes of low bladder capacity, compliance and high maximum detrusor pressure and sexual dysfunction in MS patients. Problems with positioning and spasticity affect mainly tetraplegic patients. Peer support may help to optimise the sexual adjustment of women with SCI in achieving a more positive self-image, self-esteem and feelings of being attractive to themselves and others [409, 414-416].

The use of specific drugs for sexual dysfunction is indicated to treat inadequate lubrication. Data on sildenafil for treating female sexual dysfunction are poor and controversial [355]. Although good evidence exists that psychological interventions are effective in the treatment of female hypoactive sexual desire disorder and female orgasmic disorder [417], there is a lack of high-level evidence studies in the neurological population.

Neurophysiological studies have shown that women with the ability to perceive Th 11-L2 pin-prick sensations may have psychogenic genital vasocongestion. Reflex lubrication and orgasm is more prevalent in women with SCI who have preserved the sacral reflex arc (S2-S5), even when it has not been shown in an individual woman that a specific level and degree of lesion is the cause of a particular sexual dysfunction. In SCI women with a complete lesion of the sacral reflex, arousal and orgasm may be evoked through stimulation of other erogenous zones above the level of lesions [418-420].

Sacral neuromodulation for LUT dysfunction may improve sexual function but high-level evidence studies are lacking [355].

Women with SCI reported dissatisfaction with the quality and quantity of sexuality-related rehabilitation services and were less likely to receive sexual information than men [418, 421, 422].

3.6.4 Female fertility

There are few studies on female fertility in neurological patients. More than a third (38%) of women with epilepsy had infertility and the relevant predictors were exposure to multiple (three or more) antiepileptic drugs, older age and lower education [423].

Although it seems that the reproductive capacity of women with SCI is only temporarily affected by SCI with cessation of menstruation for approximately six months after SCI [424], there are no high-level evidence studies. About 70% of sexually active women use some form of contraception after injury, but fewer women use the birth control pill compared to before their injury [425].

Women with SCI are more likely to suffer complications during pregnancy, labour and delivery compared to able-bodied women. Complications of labour and delivery include bladder problems, spasticity, pressure sores, anaemia, and AD [426-430]. Obstetric outcomes include higher rates of Caesarean sections and an increased incidence of low birth-weight babies [425, 428-430].

Epidural anaesthesia is chosen and effective for most patients with AD during labour and delivery [431, 432].

There is very little published data on women's experience of the menopause following SCI [433]. Women with MS who plan a pregnancy should evaluate their current drug treatment with their treating physician [434-436]. Clinical management should be individualised to optimise both the mother's reproductive outcomes and MS course [435-437].

3.6.4.1 Summary of evidence and recommendation for female sexuality and fertility

Summary of Evidence	LE
Data on specific drugs for treating female sexual dysfunction are poor and controversial.	4
There are limited numbers of studies on female fertility in neurological patients, clinical management should be individualised to optimise both the mother's reproductive outcomes and medical condition.	4

Recommendations	Strength rating
Do not offer medical therapy for the treatment of neurogenic sexual dysfunction in women.	Strong
Take a multidisciplinary approach, tailored to individual patient's needs and preferences, in the management of fertility, pregnancy and delivery in women with neurological diseases.	Strong

3.7 Follow-up

3.7.1 Introduction

Neuro-urological disorders are often unstable and the symptoms may vary considerably, even within a relatively short period. Regular follow-up is therefore necessary [129].

Depending on the type of the underlying neurological pathology and the current stability of the neuro-urological symptoms, the interval between initial investigations and control diagnostics may vary and in many cases should not exceed one to two years. In high-risk neuro-urological patients this interval should be much shorter. Urinalysis should be performed regularly; the frequency to be guided by patient symptoms. The UUT should be checked by ultrasonography at regular intervals in high-risk patients; about once every six months [6, 438]. In these patients, physical examination and urine laboratory should take place every year [6, 438]. Any significant clinical change warrants further, specialised, investigation [6, 438]. However, there is a complete lack of high level evidence studies on this topic and every recommendation must be viewed critically in each individual neuro-urological patient [129]. A urodynamic investigation should be performed as a diagnostic baseline, and repeated during follow-up, more frequently in high-risk patients [6, 438].

In addition, bladder wall thickness can be measured on ultrasonography as an additional risk assessment for upper tract damage [439], although a 'safe' cut-off threshold for this has not been agreed [440]. The utility of DMSA for follow-up of neuro-urological patients has not been fully evaluated [441].

Adolescent patients with neurological pathology are at risk of being lost to follow-up during the transition to adulthood. It is important that a standardised approach during this transition is adopted to improve follow-up and specific treatment during adult life [442].

3.7.2 Summary of evidence and recommendations for follow-up

Summary of Evidence	LE
Neuro-urological disorders are often unstable and the symptoms may vary considerably, therefore, regular follow-up is necessary.	4

Recommendations	Strength rating
Assess the upper urinary tract at regular intervals in high risk patients.	Strong
Perform a physical examination and urine laboratory every year in high risk patients.	Strong
Any significant clinical changes should instigate further, specialised, investigation.	Strong
Perform urodynamic investigation as a mandatory baseline diagnostic intervention in high-risk patients at regular intervals.	Strong

3.8 Conclusions

Neuro-urological disorders have a multi-faceted pathology. They require an extensive and specific diagnosis before one can embark on an individualised therapy, which takes into account the medical and physical condition of the patient and the patient's expectations about their future. The urologist can select from a wealth of therapeutic options, each with its own pros and cons. Notwithstanding the success of any therapy embarked upon, close surveillance is necessary for the patient's entire life.

These Guidelines offer you expert advice on how to define the patient's neuro-urological symptoms as precisely as possible and how to select, together with the patient, the appropriate therapy. This last choice, as always, is governed by the golden rule: as effective as needed, as non-invasive as possible.

4. REFERENCES

1. Schafer, W., *et al.* Good urodynamic practices: uroflowmetry, filling cystometry, and pressure-flow studies. *Neurourol Urodyn*, 2002. 21: 261.
<https://www.ncbi.nlm.nih.gov/pubmed/11948720>
2. Abrams, P., *et al.* Reviewing the ICS 2002 terminology report: the ongoing debate. *Neurourol Urodyn*, 2009. 28: 287.
<https://www.ncbi.nlm.nih.gov/pubmed/19350662>
3. Abrams, P., *et al.* The standardisation of terminology of lower urinary tract function: report from the Standardisation Sub-committee of the International Continence Society. *Neurourol Urodyn*, 2002. 21: 167.
<https://www.ncbi.nlm.nih.gov/pubmed/11857671>
4. Groen, J., *et al.* Summary of European Association of Urology (EAU) Guidelines on Neuro-Urology. *Eur Urol*, 2015.
<https://www.ncbi.nlm.nih.gov/pubmed/26304502>
5. Nosseir, M., *et al.* Clinical usefulness of urodynamic assessment for maintenance of bladder function in patients with spinal cord injury. *Neurourol Urodyn*, 2007. 26: 228.
<https://www.ncbi.nlm.nih.gov/pubmed/16998859>
6. Panicker, J.N., *et al.* Lower urinary tract dysfunction in the neurological patient: clinical assessment and management. *Lancet Neurol*, 2015. 14: 720.
<https://www.ncbi.nlm.nih.gov/pubmed/26067125>
7. Guyatt, G.H., *et al.* GRADE: an emerging consensus on rating quality of evidence and strength of recommendations. *BMJ*, 2008. 336: 924.
<https://www.ncbi.nlm.nih.gov/pubmed/18436948>
8. Guyatt, G.H., *et al.* What is "quality of evidence" and why is it important to clinicians? *BMJ*, 2008. 336: 995.
<https://www.ncbi.nlm.nih.gov/pubmed/18456631>
9. Phillips B, *et al.* Oxford Centre for Evidence-based Medicine Levels of Evidence. Updated by Jeremy Howick March 2009. 1998.
<http://www.cebm.net/blog/2009/06/11/oxford-centre-evidence-based-medicine-levels-evidence-march-2009/>
10. Guyatt, G.H., *et al.* Going from evidence to recommendations. *BMJ*, 2008. 336: 1049.
<https://www.ncbi.nlm.nih.gov/pubmed/18467413>
11. Townsend, N., *et al.* Cardiovascular disease in Europe - epidemiological update 2015. *Eur Heart J*, 2015.
<https://www.ncbi.nlm.nih.gov/pubmed/26306399>
12. Tibaek, S., *et al.* Prevalence of lower urinary tract symptoms (LUTS) in stroke patients: a cross-sectional, clinical survey. *Neurourol Urodyn*, 2008. 27: 763.
<https://www.ncbi.nlm.nih.gov/pubmed/18551565>
13. Marinkovic, S.P., *et al.* Voiding and sexual dysfunction after cerebrovascular accidents. *J Urol*, 2001. 165: 359.
<https://www.ncbi.nlm.nih.gov/pubmed/11176374>
14. Rotar, M., *et al.* Stroke patients who regain urinary continence in the first week after acute first-ever stroke have better prognosis than patients with persistent lower urinary tract dysfunction. *Neurourol Urodyn*, 2011. 30: 1315.
<https://www.ncbi.nlm.nih.gov/pubmed/21488096>

15. Lobo, A., *et al.* Prevalence of dementia and major subtypes in Europe: A collaborative study of population-based cohorts. Neurologic Diseases in the Elderly Research Group. *Neurology*, 2000. 54: S4.
<https://www.ncbi.nlm.nih.gov/pubmed/10854354>
16. Na, H.R., *et al.* Urinary incontinence in Alzheimer's disease is associated with Clinical Dementia Rating-Sum of Boxes and Barthel Activities of Daily Living. *Asia Pac Psychiatry*, 2015. 7: 113.
<https://www.ncbi.nlm.nih.gov/pubmed/23857871>
17. Grant, R.L., *et al.* First diagnosis and management of incontinence in older people with and without dementia in primary care: a cohort study using The Health Improvement Network primary care database. *PLoS Med*, 2013. 10: e1001505.
<https://www.ncbi.nlm.nih.gov/pubmed/24015113>
18. Pringsheim, T., *et al.* The prevalence of Parkinson's disease: a systematic review and meta-analysis. *Mov Disord*, 2014. 29: 1583.
<https://www.ncbi.nlm.nih.gov/pubmed/24976103>
19. Ragab, M.M., *et al.* Idiopathic Parkinson's disease patients at the urologic clinic. *Neurourol Urodyn*, 2011. 30: 1258.
<https://www.ncbi.nlm.nih.gov/pubmed/21404318>
20. Papatsoris, A.G., *et al.* Urinary and erectile dysfunction in multiple system atrophy (MSA). *Neurourol Urodyn*, 2008. 27: 22.
<https://www.ncbi.nlm.nih.gov/pubmed/17563111>
21. Kim, M., *et al.* Impaired detrusor contractility is the pathognomonic urodynamic finding of multiple system atrophy compared to idiopathic Parkinson's disease. *Parkinsonism Relat Disord*, 2015. 21: 205.
<https://www.ncbi.nlm.nih.gov/pubmed/25534084>
22. Sakakibara, R., *et al.* A guideline for the management of bladder dysfunction in Parkinson's disease and other gait disorders. *Neurourol Urodyn*, 2015.
<https://www.ncbi.nlm.nih.gov/pubmed/25810035>
23. Dolecek, T.A., *et al.* CBTRUS statistical report: primary brain and central nervous system tumors diagnosed in the United States in 2005-2009. *Neuro Oncol*, 2012. 14 Suppl 5: v1.
<https://www.ncbi.nlm.nih.gov/pubmed/23095881>
24. Maurice-Williams, R.S. Micturition symptoms in frontal tumours. *J Neurol Neurosurg Psychiatry*, 1974. 37: 431.
<https://www.ncbi.nlm.nih.gov/pubmed/4365244>
25. Christensen, D., *et al.* Prevalence of cerebral palsy, co-occurring autism spectrum disorders, and motor functioning - Autism and Developmental Disabilities Monitoring Network, USA, 2008. *Dev Med Child Neurol*, 2014. 56: 59.
<https://www.ncbi.nlm.nih.gov/pubmed/24117446>
26. Marciniak, C., *et al.* Urinary incontinence in adults with cerebral palsy: prevalence, type, and effects on participation. *PM R*, 2014. 6: 110.
<https://www.ncbi.nlm.nih.gov/pubmed/23978464>
27. Yildiz, N., *et al.* Cross-sectional study of urinary problems in adults with cerebral palsy: awareness and impact on the quality of life. *Neurological Sciences*, 2017: 1.
<https://www.ncbi.nlm.nih.gov/pubmed/28389939>
28. Tagliaferri, F., *et al.* A systematic review of brain injury epidemiology in Europe. *Acta Neurochir (Wien)*, 2006. 148: 255.
<https://www.ncbi.nlm.nih.gov/pubmed/16311842>
29. Kulakli, F., *et al.* Relationship between urinary dysfunction and clinical factors in patients with traumatic brain injury. *Brain Inj*, 2014. 28: 323.
<https://www.ncbi.nlm.nih.gov/pubmed/24377376>
30. Singh, A., *et al.* Global prevalence and incidence of traumatic spinal cord injury. *Clin Epidemiol*, 2014. 6: 309.
<https://www.ncbi.nlm.nih.gov/pubmed/25278785>
31. Weld, K.J., *et al.* Association of level of injury and bladder behavior in patients with post-traumatic spinal cord injury. *Urology*, 2000. 55: 490.
<https://www.ncbi.nlm.nih.gov/pubmed/10736489>
32. Kondo, A., *et al.* Neural tube defects: prevalence, etiology and prevention. *Int J Urol*, 2009. 16: 49.
<https://www.ncbi.nlm.nih.gov/pubmed/19120526>
33. Sawin, K.J., *et al.* The National Spina Bifida Patient Registry: profile of a large cohort of participants from the first 10 clinics. *J Pediatr*, 2015. 166: 444.
<https://www.ncbi.nlm.nih.gov/pubmed/25444012>

34. Bartolin, Z., *et al.* Relationship between clinical data and urodynamic findings in patients with lumbar intervertebral disk protrusion. *Urol Res*, 2002. 30: 219.
<https://www.ncbi.nlm.nih.gov/pubmed/12202938>
35. Lange, M.M., *et al.* Urinary and sexual dysfunction after rectal cancer treatment. *Nat Rev Urol*, 2011. 8: 51.
<https://www.ncbi.nlm.nih.gov/pubmed/21135876>
36. Int. Diabetes Federation, IDF Diabetes Atlas, 6th edn. 2013, International Diabetes Federation: Brussels, Belgium.
<https://www.idf.org/e-library/epidemiology-research/diabetes-atlas/19-atlas-6th-edition.html>
37. Yuan, Z., *et al.* Diabetic cystopathy: A review. *J Diabetes*, 2015. 7: 442.
<https://www.ncbi.nlm.nih.gov/pubmed/25619174>
38. Pugliatti, M., *et al.* The epidemiology of multiple sclerosis in Europe. *Eur J Neurol*, 2006. 13: 700.
<https://www.ncbi.nlm.nih.gov/pubmed/16834700>
39. de Seze, M., *et al.* The neurogenic bladder in multiple sclerosis: review of the literature and proposal of management guidelines. *Mult Scler*, 2007. 13: 915.
<https://www.ncbi.nlm.nih.gov/pubmed/17881401>
40. Krassioukov, A., *et al.* International standards to document remaining autonomic function after spinal cord injury. *J Spinal Cord Med*, 2012. 35: 201.
<https://www.ncbi.nlm.nih.gov/pubmed/22925746>
41. Irwin, D.E., *et al.* Understanding the elements of overactive bladder: questions raised by the EPIC study. *BJU Int*, 2008. 101: 1381.
<https://www.ncbi.nlm.nih.gov/pubmed/18336602>
42. Fix, J.D., *Neuroanatomy*. 4th ed. 2008, Philadelphia, Pennsylvania, USA.
43. Maynard, F.M., Jr., *et al.* International Standards for Neurological and Functional Classification of Spinal Cord Injury. American Spinal Injury Association. *Spinal Cord*, 1997. 35: 266.
<https://www.ncbi.nlm.nih.gov/pubmed/9160449>
44. Klevmark, B. Natural pressure-volume curves and conventional cystometry. *Scand J Urol Nephrol Suppl*, 1999. 201: 1.
<https://www.ncbi.nlm.nih.gov/pubmed/10573769>
45. Del Popolo, G., *et al.* Diagnosis and therapy for neurogenic bladder dysfunctions in multiple sclerosis patients. *Neurol Sci*, 2008. 29 Suppl 4: S352.
<https://www.ncbi.nlm.nih.gov/pubmed/19089675>
46. Satar, N., *et al.* The effects of delayed diagnosis and treatment in patients with an occult spinal dysraphism. *J Urol*, 1995. 154: 754.
<https://www.ncbi.nlm.nih.gov/pubmed/7609171>
47. Watanabe, T., *et al.* High incidence of occult neurogenic bladder dysfunction in neurologically intact patients with thoracolumbar spinal injuries. *J Urol*, 1998. 159: 965.
<https://www.ncbi.nlm.nih.gov/pubmed/9474194>
48. Ahlberg, J., *et al.* Neurological signs are common in patients with urodynamically verified "idiopathic" bladder overactivity. *Neurourol Urodyn*, 2002. 21: 65.
<https://www.ncbi.nlm.nih.gov/pubmed/11835426>
49. Bemelmans, B.L., *et al.* Evidence for early lower urinary tract dysfunction in clinically silent multiple sclerosis. *J Urol*, 1991. 145: 1219.
<https://www.ncbi.nlm.nih.gov/pubmed/2033697>
50. Klausner, A.P., *et al.* The neurogenic bladder: an update with management strategies for primary care physicians. *Med Clin North Am*, 2011. 95: 111.
<https://www.ncbi.nlm.nih.gov/pubmed/21095415>
51. Çetinel, B., *et al.* Risk factors predicting upper urinary tract deterioration in patients with spinal cord injury: A retrospective study. *Neurourol Urodyn*, 2017. 36: 653.
<https://www.ncbi.nlm.nih.gov/pubmed/26934371>
52. Elmelund, M., *et al.* Renal deterioration after spinal cord injury is associated with length of detrusor contractions during cystometry-A study with a median of 41 years follow-up. *Neurourol Urodyn*, 2016.
<https://www.ncbi.nlm.nih.gov/pubmed/27813141>
53. Ineichen, B.V., *et al.* High EDSS can predict risk for upper urinary tract damage in patients with multiple sclerosis. *Mult Scler*, 2017. 1352458517703801.
<https://www.ncbi.nlm.nih.gov/pubmed/28367674>
54. Bors, E., *et al.* History and physical examination in neurological urology. *J Urol*, 1960. 83: 759.
<https://www.ncbi.nlm.nih.gov/pubmed/13802958>

55. Cameron, A.P., *et al.* The Severity of Bowel Dysfunction in Patients with Neurogenic Bladder. *J Urol*, 2015.
<https://www.ncbi.nlm.nih.gov/pubmed/25956470>
56. Vodusek, D.B. Lower urinary tract and sexual dysfunction in neurological patients. *Eur Neurol*, 2014. 72: 109.
<https://www.ncbi.nlm.nih.gov/pubmed/24993182>
57. Linsenmeyer, T.A., *et al.* Accuracy of individuals with spinal cord injury at predicting urinary tract infections based on their symptoms. *J Spinal Cord Med*, 2003. 26: 352.
<https://www.ncbi.nlm.nih.gov/pubmed/14992336>
58. Massa, L.M., *et al.* Validity, accuracy, and predictive value of urinary tract infection signs and symptoms in individuals with spinal cord injury on intermittent catheterization. *J Spinal Cord Med*, 2009. 32: 568.
<https://www.ncbi.nlm.nih.gov/pubmed/20025153>
59. Bellucci, C.H.S., *et al.* Acute spinal cord injury - Do ambulatory patients need urodynamic investigations? *J Urol*, 2013. 189: 1369.
<https://www.ncbi.nlm.nih.gov/pubmed/23069382>
60. Abrams, P., *et al.* The standardisation of terminology of lower urinary tract function: report from the Standardisation Sub-committee of the International Continence Society. *Neurourol Urodyn*, 2002. 21: 167.
<https://www.ncbi.nlm.nih.gov/pubmed/11857671>
61. Kessler, T.M. Diagnosis of urinary incontinence. *JAMA*, 2008. 300: 283; author reply 283.
<https://www.ncbi.nlm.nih.gov/pubmed/18632541>
62. Honjo, H., *et al.* Impact of convenience void in a bladder diary with urinary perception grade to assess overactive bladder symptoms: a community-based study. *Neurourol Urodyn*, 2010. 29: 1286.
<https://www.ncbi.nlm.nih.gov/pubmed/20878998>
63. Naoemova, I., *et al.* Reliability of the 24-h sensation-related bladder diary in women with urinary incontinence. *Int Urogynecol J Pelvic Floor Dysfunct*, 2008. 19: 955.
<https://www.ncbi.nlm.nih.gov/pubmed/18235981>
64. Henze, T. Managing specific symptoms in people with multiple sclerosis. *Int MS J*, 2005. 12: 60.
<https://www.ncbi.nlm.nih.gov/pubmed/16417816>
65. Liu, C.W., *et al.* The relationship between bladder management and health-related quality of life in patients with spinal cord injury in the UK. *Spinal Cord*, 2010. 48: 319.
<https://www.ncbi.nlm.nih.gov/pubmed/19841636>
66. Khalaf, K.M., *et al.* The impact of lower urinary tract symptoms on health-related quality of life among patients with multiple sclerosis. *Neurourol Urodyn*, 2016. 35: 48.
<https://www.ncbi.nlm.nih.gov/pubmed/25327401>
67. Szymanski, K.M., *et al.* All Incontinence is Not Created Equal: Impact of Urinary and Fecal Incontinence on Quality of Life in Adults with Spina Bifida. *J Urol*, 2017. Part 2. 197: 885.
<https://www.ncbi.nlm.nih.gov/pubmed/28131501>
68. Pannek, J., *et al.* Does optimizing bladder management equal optimizing quality of life? Correlation between health-related quality of life and urodynamic parameters in patients with spinal cord lesions. *Urology*, 2009. 74: 263.
<https://www.ncbi.nlm.nih.gov/pubmed/19428089>
69. Patel, D.P., *et al.* Patient reported outcomes measures in neurogenic bladder and bowel: A systematic review of the current literature. *Neurourol Urodyn*, 2016. 35: 8.
<https://www.ncbi.nlm.nih.gov/pubmed/25327455>
70. Bonniaud, V., *et al.* Qualiveen, a urinary-disorder specific instrument: 0.5 corresponds to the minimal important difference. *J Clin Epidemiol*, 2008. 61: 505.
<https://www.ncbi.nlm.nih.gov/pubmed/18394545>
71. Bonniaud, V., *et al.* Development and validation of the short form of a urinary quality of life questionnaire: SF-Qualiveen. *J Urol*, 2008. 180: 2592.
<https://www.ncbi.nlm.nih.gov/pubmed/18950816>
72. Bonniaud, V., *et al.* Italian version of Qualiveen-30: cultural adaptation of a neurogenic urinary disorder-specific instrument. *Neurourol Urodyn*, 2011. 30: 354.
<https://www.ncbi.nlm.nih.gov/pubmed/21305589>
73. Ciudin, A., *et al.* Quality of life of multiple sclerosis patients: translation and validation of the Spanish version of Qualiveen. *Neurourol Urodyn*, 2012. 31: 517.
<https://www.ncbi.nlm.nih.gov/pubmed/22396437>

74. D'Ancona, C.A., *et al.* Quality of life of neurogenic patients: translation and validation of the Portuguese version of Qualiveen. *Int Urol Nephrol*, 2009. 41: 29.
<https://www.ncbi.nlm.nih.gov/pubmed/22396437>
75. Pannek, J., *et al.* [Quality of life in German-speaking patients with spinal cord injuries and bladder dysfunctions. Validation of the German version of the Qualiveen questionnaire]. *Urologe A*, 2007. 46: 1416.
<https://www.ncbi.nlm.nih.gov/pubmed/17605119>
76. Welk, B., *et al.* The conceptualization and development of a patient-reported neurogenic bladder symptom score. *Res Rep Urol*, 2013. 5: 129.
<https://www.ncbi.nlm.nih.gov/pubmed/24400244>
77. Gulick, E.E. Bowel management related quality of life in people with multiple sclerosis: psychometric evaluation of the QoL-BM measure. *Int J Nurs Stud*, 2011. 48: 1066.
<https://www.ncbi.nlm.nih.gov/pubmed/21377677>
78. Tsang, B., *et al.* A systematic review and comparison of questionnaires in the management of spinal cord injury, multiple sclerosis and the neurogenic bladder. *Neurourol Urodyn*, 2015.
<https://www.ncbi.nlm.nih.gov/pubmed/25620137>
79. Schurch, B., *et al.* Reliability and validity of the Incontinence Quality of Life questionnaire in patients with neurogenic urinary incontinence. *Arch Phys Med Rehabil*, 2007. 88: 646.
<https://www.ncbi.nlm.nih.gov/pubmed/17466735>
80. Hollingworth, W., *et al.* Exploring the impact of changes in neurogenic urinary incontinence frequency and condition-specific quality of life on preference-based outcomes. *Qual Life Res*, 2010. 19: 323.
<https://www.ncbi.nlm.nih.gov/pubmed/20094804>
81. Cella, D.F., *et al.* Validation of the functional assessment of multiple sclerosis quality of life instrument. *Neurology*, 1996. 47: 129.
<https://www.ncbi.nlm.nih.gov/pubmed/8710066>
82. Wesson, J.M., *et al.* The functional index for living with multiple sclerosis: development and validation of a new quality of life questionnaire. *Mult Scler*, 2009. 15: 1239.
<https://www.ncbi.nlm.nih.gov/pubmed/19737850>
83. Gold, S.M., *et al.* Disease specific quality of life instruments in multiple sclerosis: validation of the Hamburg Quality of Life Questionnaire in Multiple Sclerosis (HAQUAMS). *Mult Scler*, 2001. 7: 119.
<https://www.ncbi.nlm.nih.gov/pubmed/11424632>
84. Goodin, D.S. A questionnaire to assess neurological impairment in multiple sclerosis. *Mult Scler*, 1998. 4: 444.
<https://www.ncbi.nlm.nih.gov/pubmed/9839306>
85. Foley, F.W., *et al.* The Multiple Sclerosis Intimacy and Sexuality Questionnaire -- re-validation and development of a 15-item version with a large US sample. *Mult Scler*, 2013. 19: 1197.
<https://www.ncbi.nlm.nih.gov/pubmed/23369892>
86. Sanders, A.S., *et al.* The Multiple Sclerosis Intimacy and Sexuality Questionnaire-19 (MSISQ-19). *Sex Disabil*, 2000. 18: 3.
<https://link.springer.com/article/10.1023/A:1005421627154>
87. Marrie, R.A., *et al.* Validity and reliability of the MSQLI in cognitively impaired patients with multiple sclerosis. *Mult Scler*, 2003. 9: 621.
<https://www.ncbi.nlm.nih.gov/pubmed/14664477>
88. Vickrey, B.G., *et al.* A health-related quality of life measure for multiple sclerosis. *Qual Life Res*, 1995. 4: 187.
<https://www.ncbi.nlm.nih.gov/pubmed/7613530>
89. Honan, C.A., *et al.* The multiple sclerosis work difficulties questionnaire (MSWDQ): development of a shortened scale. *Disabil Rehabil*, 2014. 36: 635.
<https://www.ncbi.nlm.nih.gov/pubmed/23786346>
90. Welk, B., *et al.* The validity and reliability of the neurogenic bladder symptom score. *J Urol*, 2014. 192: 452.
<https://www.ncbi.nlm.nih.gov/pubmed/24518764>
91. Bonniaud, V., *et al.* Measuring quality of life in multiple sclerosis patients with urinary disorders using the Qualiveen questionnaire. *Arch Phys Med Rehabil*, 2004. 85: 1317.
<https://www.ncbi.nlm.nih.gov/pubmed/15295759>
92. Franceschini, M., *et al.* Follow-up in persons with traumatic spinal cord injury: questionnaire reliability. *Eura Medicophys*, 2006. 42: 211.
<https://www.ncbi.nlm.nih.gov/pubmed/17039217>

93. Noreau, L., *et al.* Development and assessment of a community follow-up questionnaire for the Rick Hansen spinal cord injury registry. *Arch Phys Med Rehabil*, 2013. 94: 1753.
<https://www.ncbi.nlm.nih.gov/pubmed/23529142>
94. Husmann, D.A. Mortality following augmentation cystoplasty: A transitional urologist's viewpoint. *J Pediatr Urol*, 2017.
<https://www.ncbi.nlm.nih.gov/pubmed/28645552>
95. Yang, C.C., *et al.* Bladder management in women with neurologic disabilities. *Phys Med Rehabil Clin N Am*, 2001. 12: 91.
<https://www.ncbi.nlm.nih.gov/pubmed/11853041>
96. Podnar, S., *et al.* Protocol for clinical neurophysiologic examination of the pelvic floor. *Neurourol Urodyn*, 2001. 20: 669.
<https://www.ncbi.nlm.nih.gov/pubmed/11746548>
97. Harrison, S., *et al.* Urinary incontinence in neurological disease: assessment and management. NICE Clinical Guideline 2012. [CG148].
<https://www.nice.org.uk/guidance/cg148>
98. Liu, N., *et al.* Autonomic dysreflexia severity during urodynamics and cystoscopy in individuals with spinal cord injury. *Spinal Cord*, 2013. 51: 863.
<https://www.ncbi.nlm.nih.gov/pubmed/24060768>
99. Labat, J.J., *et al.* Diagnostic criteria for pudendal neuralgia by pudendal nerve entrapment (Nantes criteria). *Neurourol Urodyn*, 2008. 27: 306.
<https://www.ncbi.nlm.nih.gov/pubmed/17828787>
100. Brown, D., *Atlas of regional anesthesia*. 3rd. ed. 2006, Philadelphia
101. Standring, S., *Gray's anatomy*, . 40th ed. 2008.
102. Bellucci, C.H., *et al.* Neurogenic lower urinary tract dysfunction--do we need same session repeat urodynamic investigations? *J Urol*, 2012. 187: 1318.
<https://www.ncbi.nlm.nih.gov/pubmed/22341264>
103. Walter, M., *et al.* Autonomic dysreflexia and repeatability of cardiovascular changes during same session repeat urodynamic investigation in women with spinal cord injury. *World J Urol*, 2015.
<https://www.ncbi.nlm.nih.gov/pubmed/26055644>
104. Gammie, A., *et al.* International Continence Society guidelines on urodynamic equipment performance. *Neurourol Urodyn*, 2014. 33: 370.
<https://www.ncbi.nlm.nih.gov/pubmed/24390971>
105. McGuire, E.J., *et al.* Leak-point pressures. *Urol Clin North Am*, 1996. 23: 253.
<https://www.ncbi.nlm.nih.gov/pubmed/8659025>
106. Ozkan, B., *et al.* Which factors predict upper urinary tract deterioration in overactive neurogenic bladder dysfunction? *Urology*, 2005. 66: 99.
<https://www.ncbi.nlm.nih.gov/pubmed/15992868>
107. Wang, Q.W., *et al.* Is it possible to use urodynamic variables to predict upper urinary tract dilatation in children with neurogenic bladder-sphincter dysfunction? *BJU Int*, 2006. 98: 1295.
<https://www.ncbi.nlm.nih.gov/pubmed/17034510>
108. Linsenmeyer, T.A., *et al.* The impact of urodynamic parameters on the upper tracts of spinal cord injured men who void reflexly. *J Spinal Cord Med*, 1998. 21: 15.
<https://www.ncbi.nlm.nih.gov/pubmed/9541882>
109. McGuire, E.J., *et al.* Prognostic value of urodynamic testing in myelodysplastic patients. *J Urol*, 1981. 126: 205.
<https://www.ncbi.nlm.nih.gov/pubmed/7196460>
110. Krongrad, A., *et al.* Bladder neck dysynergia in spinal cord injury. *Am J Phys Med Rehabil*, 1996. 75: 204.
<https://www.ncbi.nlm.nih.gov/pubmed/8663928>
111. Weld, K.J., *et al.* Clinical significance of detrusor sphincter dyssynergia type in patients with post-traumatic spinal cord injury. *Urology*, 2000. 56: 565.
<https://www.ncbi.nlm.nih.gov/pubmed/11018603>
112. Rossier, A.B., *et al.* 5-microtransducer catheter in evaluation of neurogenic bladder function. *Urology*, 1986. 27: 371.
<https://www.ncbi.nlm.nih.gov/pubmed/3962062>
113. Al-Ali, M., *et al.* A 10 year review of the endoscopic treatment of 125 spinal cord injured patients with vesical outlet obstruction: does bladder neck dyssynergia exist? *Paraplegia*, 1996. 34: 34.
<https://www.ncbi.nlm.nih.gov/pubmed/8848321>

114. Bacsu, C.D., *et al.* Diagnosing detrusor sphincter dyssynergia in the neurological patient. *BJU Int*, 2012. 109 Suppl 3: 31.
<https://www.ncbi.nlm.nih.gov/pubmed/22458490>
115. Lose, G., *et al.* Standardisation of urethral pressure measurement: report from the Standardisation Sub-Committee of the International Continence Society. *Neurourol Urodyn*, 2002. 21: 258.
<https://www.ncbi.nlm.nih.gov/pubmed/>
116. Marks, B.K., *et al.* Videourodynamics: indications and technique. *Urol Clin North Am*, 2014. 41: 383.
<https://www.ncbi.nlm.nih.gov/pubmed/25063594>
117. Virseda, M., *et al.* Reliability of ambulatory urodynamics in patients with spinal cord injuries. *Neurourol Urodyn*, 2013. 32: 387.
<https://www.ncbi.nlm.nih.gov/pubmed/>
118. Virseda-Chamorro, M., *et al.* Comparison of ambulatory versus video urodynamics in patients with spinal cord injury. *Spinal Cord*, 2014. 52: 551.
<https://www.ncbi.nlm.nih.gov/pubmed/24663000>
119. Geirsson, G., *et al.* The ice-water test--a simple and valuable supplement to routine cystometry. *Br J Urol*, 1993. 71: 681.
<https://www.ncbi.nlm.nih.gov/pubmed/8343894>
120. Geirsson, G., *et al.* Pressure, volume and infusion speed criteria for the ice-water test. *Br J Urol*, 1994. 73: 498.
<https://www.ncbi.nlm.nih.gov/pubmed/8012770>
121. Al-Hayek, S., *et al.* The 50-year history of the ice water test in urology. *J Urol*, 2010. 183: 1686.
<https://www.ncbi.nlm.nih.gov/pubmed/20299050>
122. Lapedes, J. Neurogenic bladder. Principles of treatment. *Urol Clin North Am*, 1974. 1: 81.
<https://www.ncbi.nlm.nih.gov/pubmed/4428540>
123. Riedl, C.R., *et al.* Electromotive administration of intravesical bethanechol and the clinical impact on acontractile detrusor management: introduction of a new test. *J Urol*, 2000. 164: 2108.
<https://www.ncbi.nlm.nih.gov/pubmed/11061937>
124. Podnar, S., *et al.* Lower urinary tract dysfunction in patients with peripheral nervous system lesions. *Handb Clin Neurol*, 2015. 130: 203.
<https://www.ncbi.nlm.nih.gov/pubmed/26003246>
125. Ouyang, L., *et al.* Characteristics and survival of patients with end stage renal disease and spina bifida in the United States renal data system. *J Urol*, 2015. 193: 558.
<https://www.ncbi.nlm.nih.gov/pubmed/25167993>
126. Lawrenson, R., *et al.* Renal failure in patients with neurogenic lower urinary tract dysfunction. *Neuroepidemiology*, 2001. 20: 138.
<https://www.ncbi.nlm.nih.gov/pubmed/11359083>
127. Dangle, P.P., *et al.* Cystatin C-calculated Glomerular Filtration Rate-A Marker of Early Renal Dysfunction in Patients With Neuropathic Bladder. *Urology*, 2017. 100: 213.
<https://www.ncbi.nlm.nih.gov/pubmed/27542858>
128. Mingat, N., *et al.* Prospective study of methods of renal function evaluation in patients with neurogenic bladder dysfunction. *Urology*, 2013. 82: 1032.
<https://www.ncbi.nlm.nih.gov/pubmed/24001705>
129. Averbek, M.A., *et al.* Follow-up of the neuro-urological patient: a systematic review. *BJU Int*, 2015. 115 Suppl 6: 39.
<https://www.ncbi.nlm.nih.gov/pubmed/25891319>
130. Stöhrer, M., *et al.* Diagnosis and treatment of bladder dysfunction in spinal cord injury patients. *Eur Urol Update Series* 1994. 3: 170. [No abstract available].
131. Drake, M., *et al.*, Conservative management in neuropathic urinary incontinence, in *Incontinence*, P. Abrams, L. Cardozo, S. Khoury & A. Wein, Editors. 2013, Health Publication: Plymouth: , 2013; pp. 827-1000.
132. Chamberlain, J.D., *et al.* Mortality and longevity after a spinal cord injury: systematic review and meta-analysis. *Neuroepidemiology*, 2015. 44: 182.
<https://www.ncbi.nlm.nih.gov/pubmed/25997873>
133. Game, X., *et al.* Botulinum toxin A detrusor injections in patients with neurogenic detrusor overactivity significantly decrease the incidence of symptomatic urinary tract infections. *Eur Urol*, 2008. 53: 613.
<https://www.ncbi.nlm.nih.gov/pubmed/17804150>
134. Frankel, H.L., *et al.* Long-term survival in spinal cord injury: a fifty year investigation. *Spinal Cord*, 1998. 36: 266.
<https://www.ncbi.nlm.nih.gov/pubmed/9589527>

135. Jamil, F. Towards a catheter free status in neurogenic bladder dysfunction: a review of bladder management options in spinal cord injury (SCI). *Spinal Cord*, 2001. 39: 355.
<https://www.ncbi.nlm.nih.gov/pubmed/11464308>
136. Thietje, R., *et al.* Mortality in patients with traumatic spinal cord injury: descriptive analysis of 62 deceased subjects. *J Spinal Cord Med*, 2011. 34: 482.
<https://www.ncbi.nlm.nih.gov/pubmed/22118255>
137. Hackler, R.H. A 25-year prospective mortality study in the spinal cord injured patient: comparison with the long-term living paraplegic. *J Urol*, 1977. 117: 486.
<https://www.ncbi.nlm.nih.gov/pubmed/850323>
138. Rodrigues, P., *et al.* Involuntary detrusor contraction is a frequent finding in patients with recurrent urinary tract infections. *Urol Int*, 2014. 93: 67.
<https://www.ncbi.nlm.nih.gov/pubmed/25011551>
139. Bauer, S.B. Neurogenic bladder: etiology and assessment. *Pediatr Nephrol*, 2008. 23: 541.
<https://www.ncbi.nlm.nih.gov/pubmed/18270749>
140. Barbalias, G.A., *et al.* Critical evaluation of the Crede maneuver: a urodynamic study of 207 patients. *J Urol*, 1983. 130: 720.
<https://www.ncbi.nlm.nih.gov/pubmed/6887405>
141. Reinberg, Y., *et al.* Renal rupture after the Crede maneuver. *J Pediatr*, 1994. 124: 279.
<https://www.ncbi.nlm.nih.gov/pubmed/8301439>
142. Wyndaele, J.J., *et al.* Neurologic urinary incontinence. *Neurourol Urodyn*, 2010. 29: 159.
<https://www.ncbi.nlm.nih.gov/pubmed/20025021>
143. Menon, E.B., *et al.* Bladder training in patients with spinal cord injury. *Urology*, 1992. 40: 425.
<https://www.ncbi.nlm.nih.gov/pubmed/1441039>
144. Furusawa, K., *et al.* Incidence of symptomatic autonomic dysreflexia varies according to the bowel and bladder management techniques in patients with spinal cord injury. *Spinal Cord*, 2011. 49: 49.
<https://www.ncbi.nlm.nih.gov/pubmed/20697419>
145. Outcomes following traumatic spinal cord injury: clinical practice guidelines for health-care professionals. *J Spinal Cord Med*, 2000. 23: 289.
<https://www.ncbi.nlm.nih.gov/pubmed/17536300>
146. El-Masri, W.S., *et al.* Long-term follow-up study of outcomes of bladder management in spinal cord injury patients under the care of the Midlands Centre for Spinal Injuries in Oswestry. *Spinal Cord*, 2012. 50: 14.
<https://www.ncbi.nlm.nih.gov/pubmed/21808256>
147. Singh, R., *et al.* Bladder management methods and urological complications in spinal cord injury patients. *Indian J Orthop*, 2011. 45: 141.
<https://www.ncbi.nlm.nih.gov/pubmed/21430869>
148. Fall, M., *et al.* Electrical stimulation. A physiologic approach to the treatment of urinary incontinence. *Urol Clin North Am*, 1991. 18: 393.
<https://www.ncbi.nlm.nih.gov/pubmed/2017820>
149. Vodusek, D.B., *et al.* Detrusor inhibition induced by stimulation of pudendal nerve afferents. *Neurourol Urodyn*, 1986. 5: 381.
<http://onlinelibrary.wiley.com/doi/10.1002/nau.1930050404/full>
150. Gross, T., *et al.* Transcutaneous Electrical Nerve Stimulation for Treating Neurogenic Lower Urinary Tract Dysfunction: A Systematic Review. *Eur Urol*, 2016. 69: 1102.
<https://www.ncbi.nlm.nih.gov/pubmed/26831506>
151. Schneider, M.P., *et al.* Tibial Nerve Stimulation for Treating Neurogenic Lower Urinary Tract Dysfunction: A Systematic Review. *Eur Urol*, 2015.
<https://www.ncbi.nlm.nih.gov/pubmed/26194043>
152. Liu, Y., *et al.* Effects of Transcutaneous Electrical Nerve Stimulation at Two Frequencies on Urinary Incontinence in Poststroke Patients: A Randomized Controlled Trial. *Am J Phys Med Rehabil*, 2016. 95: 183.
<https://www.ncbi.nlm.nih.gov/pubmed/26259053>
153. McClurg, D., *et al.* Neuromuscular electrical stimulation and the treatment of lower urinary tract dysfunction in multiple sclerosis--a double blind, placebo controlled, randomised clinical trial. *Neurourol Urodyn*, 2008. 27: 231.
<https://www.ncbi.nlm.nih.gov/pubmed/17705160>
154. McClurg, D., *et al.* Comparison of pelvic floor muscle training, electromyography biofeedback, and neuromuscular electrical stimulation for bladder dysfunction in people with multiple sclerosis: a randomized pilot study. *Neurourol Urodyn*, 2006. 25: 337.
<https://www.ncbi.nlm.nih.gov/pubmed/16637070>

155. Ferreira, A.P., *et al.* Impact of a Pelvic Floor Training Program Among Women with Multiple Sclerosis: A Controlled Clinical Trial. *Am J Phys Med Rehabil*, 2016. 95: 1.
<https://www.ncbi.nlm.nih.gov/pubmed/25888662>
156. Hagerty, J.A., *et al.* Intravesical electrotherapy for neurogenic bladder dysfunction: a 22-year experience. *J Urol*, 2007. 178: 1680.
<https://www.ncbi.nlm.nih.gov/pubmed/17707024>
157. Primus, G., *et al.* Restoration of micturition in patients with acontractile and hypocontractile detrusor by transurethral electrical bladder stimulation. *Neurourol Urodyn*, 1996. 15: 489.
<https://www.ncbi.nlm.nih.gov/pubmed/8857617>
158. Lombardi, G., *et al.* Clinical efficacy of intravesical electrostimulation on incomplete spinal cord patients suffering from chronic neurogenic non-obstructive retention: a 15-year single centre retrospective study. *Spinal Cord*, 2013. 51: 232.
<https://www.ncbi.nlm.nih.gov/pubmed/23147136>
159. Brusa, L., *et al.* Effects of inhibitory rTMS on bladder function in Parkinson's disease patients. *Mov Disord*, 2009. 24: 445.
<https://www.ncbi.nlm.nih.gov/pubmed/19133657>
160. Centonze, D., *et al.* Effects of motor cortex rTMS on lower urinary tract dysfunction in multiple sclerosis. *Mult Scler*, 2007. 13: 269.
<https://www.ncbi.nlm.nih.gov/pubmed/17439897>
161. Thomas, L.H., *et al.* Treatment of urinary incontinence after stroke in adults. *Cochrane Database Syst Rev*, 2008: Cd004462.
<https://www.ncbi.nlm.nih.gov/pubmed/18254050>
162. Yeo, L., *et al.* Urinary tract dysfunction in Parkinson's disease: a review. *Int Urol Nephrol*, 2012. 44: 415.
<https://www.ncbi.nlm.nih.gov/pubmed/21553114>
163. Phe, V., *et al.* Management of neurogenic bladder in patients with multiple sclerosis. *Nat Rev Urol*, 2016. 13: 275.
<https://www.ncbi.nlm.nih.gov/pubmed/27030526>
164. Andersson, K.E. Antimuscarinic mechanisms and the overactive detrusor: an update. *Eur Urol*, 2011. 59: 377.
<https://www.ncbi.nlm.nih.gov/pubmed/21168951>
165. Bennett, N., *et al.* Can higher doses of oxybutynin improve efficacy in neurogenic bladder? *J Urol*, 2004. 171: 749.
<https://www.ncbi.nlm.nih.gov/pubmed/14713802>
166. Horstmann, M., *et al.* Neurogenic bladder treatment by doubling the recommended antimuscarinic dosage. *Neurourol Urodyn*, 2006. 25: 441.
<https://www.ncbi.nlm.nih.gov/pubmed/16847942>
167. Kennelly, M.J., *et al.* Overactive bladder: pharmacologic treatments in the neurogenic population. *Rev Urol*, 2008. 10: 182.
<https://www.ncbi.nlm.nih.gov/pubmed/18836537>
168. Madersbacher, H., *et al.* Neurogenic detrusor overactivity in adults: a review on efficacy, tolerability and safety of oral antimuscarinics. *Spinal Cord*, 2013. 51: 432.
<https://www.ncbi.nlm.nih.gov/pubmed/23743498>
169. Madhuvrata, P., *et al.* Anticholinergic drugs for adult neurogenic detrusor overactivity: a systematic review and meta-analysis. *Eur Urol*, 2012. 62: 816.
<https://www.ncbi.nlm.nih.gov/pubmed/22397851>
170. Stohrer, M., *et al.* EAU guidelines on neurogenic lower urinary tract dysfunction. *Eur Urol*, 2009. 56: 81.
<https://www.ncbi.nlm.nih.gov/pubmed/19403235>
171. Mehnert, U., *et al.* The management of urinary incontinence in the male neurological patient. *Curr Opin Urol*, 2014. 24: 586.
<https://www.ncbi.nlm.nih.gov/pubmed/25389549>
172. Stothers, L., *et al.* An integrative review of standardized clinical evaluation tool utilization in anticholinergic drug trials for neurogenic lower urinary tract dysfunction. *Spinal Cord*, 2016. 31: 31.
<https://www.ncbi.nlm.nih.gov/pubmed/27241452>
173. Amend, B., *et al.* Effective treatment of neurogenic detrusor dysfunction by combined high-dosed antimuscarinics without increased side-effects. *Eur Urol*, 2008. 53: 1021.
<https://www.ncbi.nlm.nih.gov/pubmed/18243516>

174. Cameron, A.P. Pharmacologic therapy for the neurogenic bladder. *Urol Clin North Am*, 2010. 37: 495.
<https://www.ncbi.nlm.nih.gov/pubmed/20955901>
175. Menarini, M., *et al.* Tropicium chloride in patients with neurogenic detrusor overactivity: is dose titration of benefit to the patients? *Int J Clin Pharmacol Ther*, 2006. 44: 623.
<https://www.ncbi.nlm.nih.gov/pubmed/17190372>
176. Nardulli, R., *et al.* Combined antimuscarinics for treatment of neurogenic overactive bladder. *Int J Immunopathol Pharmacol*, 2012. 25: 35s.
<https://www.ncbi.nlm.nih.gov/pubmed/22652160>
177. Tijngel, M.J., *et al.* Real life persistence rate with antimuscarinic treatment in patients with idiopathic or neurogenic overactive bladder: a prospective cohort study with solifenacin. *BMC Urology*, 2017. 17: 13.
<https://www.ncbi.nlm.nih.gov/pubmed/28403849>
178. Cameron, A.P., *et al.* Combination drug therapy improves compliance of the neurogenic bladder. *J Urol*, 2009. 182: 1062.
<https://www.ncbi.nlm.nih.gov/pubmed/19616807>
179. Isik, A.T., *et al.* Tropicium and cognition in patients with late onset Alzheimer disease. *J Nutr Health Aging*, 2009. 13: 672.
<https://www.ncbi.nlm.nih.gov/pubmed/19657549>
180. Ethans, K.D., *et al.* Efficacy and safety of tolterodine in people with neurogenic detrusor overactivity. *J Spinal Cord Med*, 2004. 27: 214.
<https://www.ncbi.nlm.nih.gov/pubmed/15478523>
181. McKeage, K. Propiverine: A review of its use in the treatment of adults and children with overactive bladder associated with idiopathic or neurogenic detrusor overactivity, and in men with lower urinary tract symptoms. *Clin Drug Invest*, 2013. 33: 71.
<https://www.ncbi.nlm.nih.gov/pubmed/23288694>
182. Nicholas, R.S., *et al.* Anticholinergics for urinary symptoms in multiple sclerosis. *Cochrane Database Syst Rev*, 2009: Cd004193.
<https://www.ncbi.nlm.nih.gov/pubmed/19160231>
183. van Rey, F., *et al.* Solifenacin in multiple sclerosis patients with overactive bladder: a prospective study. *Adv Urol*, 2011. 2011: 834753.
<https://www.ncbi.nlm.nih.gov/pubmed/21687581>
184. Bycroft, J., *et al.* The effect of darifenacin on neurogenic detrusor overactivity in patients with spinal cord injury. *Neurourol Urodyn* 2003. 22: A190.
<https://pdfs.semanticscholar.org/ba7c/06ce8114149cfabffebf3f8a090ec06d5432.pdf>
185. Carl, S., *et al.* Darifenacin is also effective in neurogenic bladder dysfunction (multiple sclerosis). *Urology*, 2006. 68: 250. [No abstract available].
186. Amarenco, G., *et al.* Solifenacin is effective and well tolerated in patients with neurogenic detrusor overactivity: Results from the double-blind, randomized, active- and placebo-controlled SONIC urodynamic study. *Neurourol Urodyn*, 2015. 29: 29.
<https://www.ncbi.nlm.nih.gov/pubmed/26714009>
187. Zesiewicz, T.A., *et al.* Randomized, controlled pilot trial of solifenacin succinate for overactive bladder in Parkinson's disease. *Parkinsonism Relat Disord*, 2015. 21: 514.
<https://www.ncbi.nlm.nih.gov/pubmed/25814050>
188. Sakakibara, R., *et al.* Imidafenacin on bladder and cognitive function in neurologic OAB patients. *Clin Auton Res*, 2013. 23: 189.
<https://www.ncbi.nlm.nih.gov/pubmed/23820664>
189. Sugiyama, H., *et al.* Effect of imidafenacin on the urodynamic parameters of patients with indwelling bladder catheters due to spinal cord injury. *Spinal Cord*, 2017. 55: 187.
<https://www.ncbi.nlm.nih.gov/pubmed/27897185>
190. Stohrer, M., *et al.* Efficacy and tolerability of propiverine hydrochloride extended-release compared with immediate-release in patients with neurogenic detrusor overactivity. *Spinal Cord*, 2013. 51: 419.
<https://www.ncbi.nlm.nih.gov/pubmed/23338657>
191. Schroder, A., *et al.* Efficacy, safety, and tolerability of intravesically administered 0.1% oxybutynin hydrochloride solution in adult patients with neurogenic bladder: A randomized, prospective, controlled multi-center trial. *Neurourol Urodyn*, 2016. 35: 582.
<https://www.ncbi.nlm.nih.gov/pubmed/25754454>
192. Wollner, J., *et al.* Initial experience with the treatment of neurogenic detrusor overactivity with a new beta-3 agonist (mirabegron) in patients with spinal cord injury. *Spinal Cord*, 2015.
<https://www.ncbi.nlm.nih.gov/pubmed/>

193. Welk, B. Urodynamic and Clinical Efficacy of Mirabegron for Neurogenic Bladder Patients, Ongoing study: Clinical Trials. Gov Identifier NCT02044510.
<https://clinicaltrials.gov/ct2/show/NCT02044510>
194. Abrams, P., *et al.* Combination treatment with mirabegron and solifenacin in patients with overactive bladder: efficacy and safety results from a randomised, double-blind, dose-ranging, phase 2 study (Symphony). *Eur Urol*, 2015. 67: 577.
<https://www.ncbi.nlm.nih.gov/pubmed/24612659>
195. Abo Youssef, N., *et al.* Cannabinoids for treating neurogenic lower urinary tract dysfunction in patients with multiple sclerosis: a systematic review and meta-analysis. *BJU Int*, 2017. 119: 515.
<https://www.ncbi.nlm.nih.gov/pubmed/28058780>
196. Barendrecht, M.M., *et al.* Is the use of parasympathomimetics for treating an underactive urinary bladder evidence-based? *BJU Int*, 2007. 99: 749.
<https://www.ncbi.nlm.nih.gov/pubmed/17233798>
197. Apostolidis, A. Taming the cannabinoids: new potential in the pharmacologic control of lower urinary tract dysfunction. *Eur Urol*, 2012. 61: 107.
<https://www.ncbi.nlm.nih.gov/pubmed/21996529>
198. Gratzke, C., *et al.* Effects of cannabimor, a novel selective cannabinoid 2 receptor agonist, on bladder function in normal rats. *Eur Urol*, 2010. 57: 1093.
<https://www.ncbi.nlm.nih.gov/pubmed/20207474>
199. Abrams, P., *et al.* Tamsulosin: efficacy and safety in patients with neurogenic lower urinary tract dysfunction due to suprasacral spinal cord injury. *J Urol*, 2003. 170: 1242.
<https://www.ncbi.nlm.nih.gov/pubmed/14501734>
200. Gomes, C.M., *et al.* Neurological status predicts response to alpha-blockers in men with voiding dysfunction and Parkinson's disease. *Clinics*, 2014. 69: 817.
<https://www.ncbi.nlm.nih.gov/pubmed/25627993>
201. Moon, K.H., *et al.* A 12-week, open label, multi-center study to evaluate the clinical efficacy and safety of silodosin on voiding dysfunction in patients with neurogenic bladder. *LUTS: Lower Urinary Tract Symptoms*, 2015. 7: 27.
<https://www.ncbi.nlm.nih.gov/pubmed/26663648>
202. Guttmann, L., *et al.* The value of intermittent catheterisation in the early management of traumatic paraplegia and tetraplegia. *Paraplegia*, 1966. 4: 63.
<https://www.ncbi.nlm.nih.gov/pubmed/5969402>
203. Lapedes, J., *et al.* Clean, intermittent self-catheterization in the treatment of urinary tract disease. *J Urol*, 1972. 107: 458.
<https://www.ncbi.nlm.nih.gov/pubmed/5010715>
204. Wyndaele, J.J. Intermittent catheterization: which is the optimal technique? *Spinal Cord*, 2002. 40: 432.
<https://www.ncbi.nlm.nih.gov/pubmed/12185603>
205. Prieto-Fingerhut, T., *et al.* A study comparing sterile and nonsterile urethral catheterization in patients with spinal cord injury. *Rehabil Nurs*, 1997. 22: 299.
<https://www.ncbi.nlm.nih.gov/pubmed/9416190>
206. Kiddoo, D., *et al.* Randomized Crossover Trial of Single Use Hydrophilic Coated vs Multiple Use Polyvinylchloride Catheters for Intermittent Catheterization to Determine Incidence of Urinary Infection. *J Urol*, 2015. 194: 174.
<https://www.ncbi.nlm.nih.gov/pubmed/25584995>
207. Bakke, A., *et al.* Physical predictors of infection in patients treated with clean intermittent catheterization: a prospective 7-year study. *Br J Urol*, 1997. 79: 85.
<https://www.ncbi.nlm.nih.gov/pubmed/9043503>
208. Günther, M., *et al.* Auswirkungen des aseptischen intermittierenden Katheterismus auf die männliche Harnröhre. [German] *Der Urologe B*, 2001. 41: 359. [no abstract available].
209. Kurze, I., *et al.* Intermittent Catheterisation and Prevention of Urinary Tract Infections in Patients with Neurogenic Lower Urinary Tract Dysfunction - Best Practice An Overview. [German]. *Aktuelle Neurologie*, 2015. 42: 515. [no abstract available].
210. Waller, L., *et al.* Clean intermittent catheterization in spinal cord injury patients: long-term followup of a hydrophilic low friction technique. *J Urol*, 1995. 153: 345.
<https://www.ncbi.nlm.nih.gov/pubmed/7815579>
211. Wyndaele, J.J. Complications of intermittent catheterization: their prevention and treatment. *Spinal Cord*, 2002. 40: 536.
<https://www.ncbi.nlm.nih.gov/pubmed/12235537>

212. Woodbury, M.G., *et al.* Intermittent catheterization practices following spinal cord injury: a national survey. *Can J Urol*, 2008. 15: 4065.
<https://www.ncbi.nlm.nih.gov/pubmed/18570710>
213. Bennett, C.J., *et al.* Comparison of bladder management complication outcomes in female spinal cord injury patients. *J Urol*, 1995. 153: 1458.
<https://www.ncbi.nlm.nih.gov/pubmed/7714965>
214. Chao, R., *et al.* Fate of upper urinary tracts in patients with indwelling catheters after spinal cord injury. *Urology*, 1993. 42: 259.
<https://www.ncbi.nlm.nih.gov/pubmed/8379025>
215. Larsen, L.D., *et al.* Retrospective analysis of urologic complications in male patients with spinal cord injury managed with and without indwelling urinary catheters. *Urology*, 1997. 50: 418.
<https://www.ncbi.nlm.nih.gov/pubmed/9301708>
216. Mitsui, T., *et al.* Is suprapubic cystostomy an optimal urinary management in high quadriplegics?. A comparative study of suprapubic cystostomy and clean intermittent catheterization. *Eur Urol*, 2000. 38: 434.
<https://www.ncbi.nlm.nih.gov/pubmed/11025382>
217. Weld, K.J., *et al.* Effect of bladder management on urological complications in spinal cord injured patients. *J Urol*, 2000. 163: 768.
<https://www.ncbi.nlm.nih.gov/pubmed/10687973>
218. Weld, K.J., *et al.* Influences on renal function in chronic spinal cord injured patients. *J Urol*, 2000. 164: 1490.
<https://www.ncbi.nlm.nih.gov/pubmed/11025689>
219. West, D.A., *et al.* Role of chronic catheterization in the development of bladder cancer in patients with spinal cord injury. *Urology*, 1999. 53: 292.
<https://www.ncbi.nlm.nih.gov/pubmed/9933042>
220. Lavelle, R.S., *et al.* Quality of life after suprapubic catheter placement in patients with neurogenic bladder conditions. *Neurourol Urodyn*, 2016. 35: 831.
<https://www.ncbi.nlm.nih.gov/pubmed/26197729>
221. Hollingsworth, J.M., *et al.* Determining the noninfectious complications of indwelling urethral catheters: a systematic review and meta-analysis. *Ann Intern Med*, 2013. 159: 401.
<https://www.ncbi.nlm.nih.gov/pubmed/24042368>
222. Buysse, G., *et al.* Intravesical oxybutynin for neurogenic bladder dysfunction: less systemic side effects due to reduced first pass metabolism. *J Urol*, 1998. 160: 892.
<https://www.ncbi.nlm.nih.gov/pubmed/9720583>
223. Di Stasi, S.M., *et al.* Intravesical oxybutynin: mode of action assessed by passive diffusion and electromotive administration with pharmacokinetics of oxybutynin and N-desethyl oxybutynin. *J Urol*, 2001. 166: 2232.
<https://www.ncbi.nlm.nih.gov/pubmed/11696741>
224. Haferkamp, A., *et al.* Dosage escalation of intravesical oxybutynin in the treatment of neurogenic bladder patients. *Spinal Cord*, 2000. 38: 250.
<https://www.ncbi.nlm.nih.gov/pubmed/10822396>
225. Pannek, J., *et al.* Combined intravesical and oral oxybutynin chloride in adult patients with spinal cord injury. *Urology*, 2000. 55: 358.
<https://www.ncbi.nlm.nih.gov/pubmed/10699610>
226. Geirsson, G., *et al.* Clinical and urodynamic effects of intravesical capsaicin treatment in patients with chronic traumatic spinal detrusor hyperreflexia. *J Urol*, 1995. 154: 1825.
<https://www.ncbi.nlm.nih.gov/pubmed/7563356>
227. Giannantoni, A., *et al.* Intravesical resiniferatoxin versus botulinum-A toxin injections for neurogenic detrusor overactivity: a prospective randomized study. *J Urol*, 2004. 172: 240.
<https://www.ncbi.nlm.nih.gov/pubmed/15201783>
228. Kim, J.H., *et al.* Intravesical resiniferatoxin for refractory detrusor hyperreflexia: a multicenter, blinded, randomized, placebo-controlled trial. *J Spinal Cord Med*, 2003. 26: 358.
<https://www.ncbi.nlm.nih.gov/pubmed/14992337>
229. Del Popolo, G., *et al.* Neurogenic detrusor overactivity treated with english botulinum toxin a: 8-year experience of one single centre. *Eur Urol*, 2008. 53: 1013.
<https://www.ncbi.nlm.nih.gov/pubmed/17950989>
230. Reitz, A., *et al.* European experience of 200 cases treated with botulinum-A toxin injections into the detrusor muscle for urinary incontinence due to neurogenic detrusor overactivity. *Eur Urol*, 2004. 45: 510.
<https://www.ncbi.nlm.nih.gov/pubmed/15041117>

231. Yuan, H., *et al.* Efficacy and Adverse Events Associated With Use of OnabotulinumtoxinA for Treatment of Neurogenic Detrusor Overactivity: A Meta-Analysis. *Int Neurourol J*, 2017. 21: 53.
<https://www.ncbi.nlm.nih.gov/pubmed/28361515>
232. Cheng, T., *et al.* Efficacy and Safety of OnabotulinumtoxinA in Patients with Neurogenic Detrusor Overactivity: A Systematic Review and Meta-Analysis of Randomized Controlled Trials. *PLoS One*, 2016. 11: e0159307.
<https://www.ncbi.nlm.nih.gov/pubmed/27463810>
233. Wagle Shukla, A., *et al.* Botulinum Toxin Therapy for Parkinson's Disease. *Seminars in Neurology*, 2017. 37: 193.
<https://www.ncbi.nlm.nih.gov/pubmed/28511260>
234. Ginsberg, D., *et al.* Phase 3 efficacy and tolerability study of onabotulinumtoxinA for urinary incontinence from neurogenic detrusor overactivity. *J Urol*, 2012. 187: 2131.
<https://www.ncbi.nlm.nih.gov/pubmed/22503020>
235. Grosse, J., *et al.* Success of repeat detrusor injections of botulinum a toxin in patients with severe neurogenic detrusor overactivity and incontinence. *Eur Urol*, 2005. 47: 653.
<https://www.ncbi.nlm.nih.gov/pubmed/15826758>
236. Rovner, E., *et al.* Long-Term Efficacy and Safety of OnabotulinumtoxinA in Patients with Neurogenic Detrusor Overactivity Who Completed 4 Years of Treatment. *J Urol.*, 2016.
<https://www.ncbi.nlm.nih.gov/pubmed/27091236>
237. Bottet, F., *et al.* Switch to Abobotulinum toxin A may be useful in the treatment of neurogenic detrusor overactivity when intradetrusor injections of Onabotulinum toxin A failed. *Neurourol Urodyn*, 2017. 21: 21.
<https://www.ncbi.nlm.nih.gov/pubmed/28431196>
238. Cruz, F., *et al.* Efficacy and safety of onabotulinumtoxinA in patients with urinary incontinence due to neurogenic detrusor overactivity: a randomised, double-blind, placebo-controlled trial. *Eur Urol*, 2011. 60: 742.
<https://www.ncbi.nlm.nih.gov/pubmed/21798658>
239. Tyagi, P., *et al.* Past, Present and Future of Chemodenervation with Botulinum Toxin in the Treatment of Overactive Bladder. *J Urol*, 2017. 197: 982.
<https://www.ncbi.nlm.nih.gov/pubmed/27871929>
240. Dykstra, D.D., *et al.* Treatment of detrusor-sphincter dyssynergia with botulinum A toxin: a double-blind study. *Arch Phys Med Rehabil*, 1990. 71: 24.
<https://www.ncbi.nlm.nih.gov/pubmed/2297305>
241. Schurch, B., *et al.* Botulinum-A toxin as a treatment of detrusor-sphincter dyssynergia: a prospective study in 24 spinal cord injury patients. *J Urol*, 1996. 155: 1023.
<https://www.ncbi.nlm.nih.gov/pubmed/8583552>
242. Huang, M., *et al.* Effects of botulinum toxin A injections in spinal cord injury patients with detrusor overactivity and detrusor sphincter dyssynergia. *J Rehab Med*, 2016. 48: 683.
<https://www.ncbi.nlm.nih.gov/pubmed/27563834>
243. Utomo, E., *et al.* Surgical management of functional bladder outlet obstruction in adults with neurogenic bladder dysfunction. *Cochrane Database Syst Rev*, 2014. 5: Cd004927.
<https://www.ncbi.nlm.nih.gov/pubmed/24859260>
244. Chancellor, M.B., *et al.* Prospective comparison of external sphincter balloon dilatation and prosthesis placement with external sphincterotomy in spinal cord injured men. *Arch Phys Med Rehabil*, 1994. 75: 297.
<https://www.ncbi.nlm.nih.gov/pubmed/8129583>
245. Schurch, B., *et al.* Botulinum toxin type a is a safe and effective treatment for neurogenic urinary incontinence: results of a single treatment, randomized, placebo controlled 6-month study. *J Urol*, 2005. 174: 196.
<https://www.ncbi.nlm.nih.gov/pubmed/15947626>
246. Perlash, I. Use of contact laser crystal tip firing Nd:YAG to relieve urinary outflow obstruction in male neurogenic bladder patients. *J Clin Laser Med Surg*, 1998. 16: 33.
<https://www.ncbi.nlm.nih.gov/pubmed/9728128>
247. Reynard, J.M., *et al.* Sphincterotomy and the treatment of detrusor-sphincter dyssynergia: current status, future prospects. *Spinal Cord*, 2003. 41: 1.
<https://www.ncbi.nlm.nih.gov/pubmed/12494314>
248. Noll, F., *et al.* Transurethral sphincterotomy in quadriplegic patients: long-term-follow-up. *Neurourol Urodyn*, 1995. 14: 351.
<https://www.ncbi.nlm.nih.gov/pubmed/7581471>

249. Derry, F., *et al.* Audit of bladder neck resection in spinal cord injured patients. *Spinal Cord*, 1998. 36: 345.
<https://www.ncbi.nlm.nih.gov/pubmed/9601115>
250. Chancellor, M.B., *et al.* Long-term followup of the North American multicenter UroLume trial for the treatment of external detrusor-sphincter dyssynergia. *J Urol*, 1999. 161: 1545.
<https://www.ncbi.nlm.nih.gov/pubmed/10210393>
251. Seoane-Rodriguez, S., *et al.* Long-term follow-up study of intraurethral stents in spinal cord injured patients with detrusor-sphincter dyssynergia. *Spinal Cord*, 2007. 45: 621.
<https://www.ncbi.nlm.nih.gov/pubmed/17211463>
252. Gajewski, J.B., *et al.* Removal of UroLume endoprosthesis: experience of the North American Study Group for detrusor-sphincter dyssynergia application. *J Urol*, 2000. 163: 773.
<https://www.ncbi.nlm.nih.gov/pubmed/10687974>
253. Wilson, T.S., *et al.* UroLume stents: lessons learned. *J Urol*, 2002. 167: 2477.
<https://www.ncbi.nlm.nih.gov/pubmed/11992061>
254. Abdul-Rahman, A., *et al.* A 20-year follow-up of the mesh wallstent in the treatment of detrusor external sphincter dyssynergia in patients with spinal cord injury. *BJU Int*, 2010. 106: 1510.
<https://www.ncbi.nlm.nih.gov/pubmed/20500511>
255. Pannek, J., *et al.* Clinical usefulness of the memokath stent as a second-line procedure after sphincterotomy failure. *J Endourol*, 2011. 25: 335.
<https://www.ncbi.nlm.nih.gov/pubmed/20977372>
256. Polguer, T., *et al.* [Treatment of detrusor-striated sphincter dyssynergia with permanent nitinol urethral stent: results after a minimum follow-up of 2 years]. *Prog Urol*, 2012. 22: 1058.
<https://www.ncbi.nlm.nih.gov/pubmed/23182120>
257. van der Merwe, A., *et al.* Outcome of dual flange metallic urethral stents in the treatment of neuropathic bladder dysfunction after spinal cord injury. *J Endourol*, 2012. 26: 1210.
<https://www.ncbi.nlm.nih.gov/pubmed/22519741>
258. Bennett, J.K., *et al.* Collagen injections for intrinsic sphincter deficiency in the neuropathic urethra. *Paraplegia*, 1995. 33: 697.
<https://www.ncbi.nlm.nih.gov/pubmed/8927407>
259. Block, C.A., *et al.* Long-term efficacy of periurethral collagen injection for the treatment of urinary incontinence secondary to myelomeningocele. *J Urol*, 2003. 169: 327.
<https://www.ncbi.nlm.nih.gov/pubmed/12478183>
260. Schurch, B., *et al.* Intraurethral sphincter prosthesis to treat hyporeflexic bladders in women: does it work? *BJU Int*, 1999. 84: 789.
<https://www.ncbi.nlm.nih.gov/pubmed/10532973>
261. Barthold, J.S., *et al.* Results of the rectus fascial sling and wrap procedures for the treatment of neurogenic sphincteric incontinence. *J Urol*, 1999. 161: 272.
<https://www.ncbi.nlm.nih.gov/pubmed/10037423>
262. Gormley, E.A., *et al.* Pubovaginal slings for the management of urinary incontinence in female adolescents. *J Urol*, 1994. 152: 822.
<https://www.ncbi.nlm.nih.gov/pubmed/8022024>
263. Kakizaki, H., *et al.* Fascial sling for the management of urinary incontinence due to sphincter incompetence. *J Urol*, 1995. 153: 644.
<https://www.ncbi.nlm.nih.gov/pubmed/7861504>
264. Mingin, G.C., *et al.* The rectus myofascial wrap in the management of urethral sphincter incompetence. *BJU Int*, 2002. 90: 550.
<https://www.ncbi.nlm.nih.gov/pubmed/12230615>
265. Abdul-Rahman, A., *et al.* Long-term outcome of tension-free vaginal tape for treating stress incontinence in women with neuropathic bladders. *BJU Int*, 2010. 106: 827.
<https://www.ncbi.nlm.nih.gov/pubmed/20132201>
266. Losco, G.S., *et al.* Long-term outcome of transobturator tape (TOT) for treatment of stress urinary incontinence in females with neuropathic bladders. *Spinal Cord*, 2015. 53: 544.
<https://www.ncbi.nlm.nih.gov/pubmed/25917951>
267. El-Azab, A.S., *et al.* Midurethral slings versus the standard pubovaginal slings for women with neurogenic stress urinary incontinence. *Int Urogynecol J*, 2015. 26: 427.
<https://www.ncbi.nlm.nih.gov/pubmed/25315169>
268. Athanasopoulos, A., *et al.* Treating stress urinary incontinence in female patients with neuropathic bladder: the value of the autologous fascia rectus sling. *Int Urol Nephrol*, 2012. 44: 1363.
<https://www.ncbi.nlm.nih.gov/pubmed/22821050>

269. Groen, L.A., *et al.* The AdVance male sling as a minimally invasive treatment for intrinsic sphincter deficiency in patients with neurogenic bladder sphincter dysfunction: a pilot study. *Neurourol Urodyn*, 2012. 31: 1284.
<https://www.ncbi.nlm.nih.gov/pubmed/22847896>
270. Mehnert, U., *et al.* Treatment of neurogenic stress urinary incontinence using an adjustable continence device: 4-year followup. *J Urol*, 2012. 188: 2274.
<https://www.ncbi.nlm.nih.gov/pubmed/23083648>
271. Daneshmand, S., *et al.* Puboprostatic sling repair for treatment of urethral incompetence in adult neurogenic incontinence. *J Urol*, 2003. 169: 199.
<https://www.ncbi.nlm.nih.gov/pubmed/12478135>
272. Herschorn, S., *et al.* Fascial slings and bladder neck tapering in the treatment of male neurogenic incontinence. *J Urol*, 1992. 147: 1073.
<https://www.ncbi.nlm.nih.gov/pubmed/1552586>
273. Light, J.K., *et al.* Use of the artificial urinary sphincter in spinal cord injury patients. *J Urol*, 1983. 130: 1127.
<https://www.ncbi.nlm.nih.gov/pubmed/6644893>
274. Farag, F., *et al.* Surgical treatment of neurogenic stress urinary incontinence: A systematic review of quality assessment and surgical outcomes. *Neurourol Urodyn*, 2016. 35: 21.
<https://www.ncbi.nlm.nih.gov/pubmed/25327633>
275. Kim, S.P., *et al.* Long-term durability and functional outcomes among patients with artificial urinary sphincters: a 10-year retrospective review from the University of Michigan. *J Urol*, 2008. 179: 1912.
<https://www.ncbi.nlm.nih.gov/pubmed/18353376>
276. Wang, R., *et al.* Long-term outcomes after primary failures of artificial urinary sphincter implantation. *Urology*, 2012. 79: 922.
<https://www.ncbi.nlm.nih.gov/pubmed/22305763>
277. Ammirati, E., *et al.* Management of male and female neurogenic stress urinary incontinence in spinal cord injured (SCI) patients using adjustable continence therapy. *Urologia*, 2017. 0: 16.
<https://www.ncbi.nlm.nih.gov/pubmed/28525663>
278. Janknegt, R.A., *et al.* Electrically stimulated gracilis sphincter for treatment of bladder sphincter incontinence. *Lancet*, 1992. 340: 1129.
<https://www.ncbi.nlm.nih.gov/pubmed/1359213>
279. Chancellor, M.B., *et al.* Gracilis muscle transposition with electrical stimulation for sphincteric incontinence: a new approach. *World J Urol*, 1997. 15: 320.
<https://www.ncbi.nlm.nih.gov/pubmed/9372585>
280. Chancellor, M.B., *et al.* Gracilis urethromyoplasty--an autologous urinary sphincter for neurologically impaired patients with stress incontinence. *Spinal Cord*, 1997. 35: 546.
<https://www.ncbi.nlm.nih.gov/pubmed/9267922>
281. Donahoo, K.K., *et al.* The Young-Dees-Leadbetter bladder neck repair for neurogenic incontinence. *J Urol*, 1999. 161: 1946.
<https://www.ncbi.nlm.nih.gov/pubmed/10332478>
282. Kropp, K.A., *et al.* Urethral lengthening and reimplantation for neurogenic incontinence in children. *J Urol*, 1986. 135: 533.
<https://www.ncbi.nlm.nih.gov/pubmed/3944902>
283. Salle, J.L., *et al.* Urethral lengthening with anterior bladder wall flap (Pippi Salle procedure): modifications and extended indications of the technique. *J Urol*, 1997. 158: 585.
<https://www.ncbi.nlm.nih.gov/pubmed/9224369>
284. Rawashdeh, Y.F., *et al.* International Children's Continence Society's recommendations for therapeutic intervention in congenital neuropathic bladder and bowel dysfunction in children. *Neurourol Urodyn*, 2012. 31: 615.
<https://www.ncbi.nlm.nih.gov/pubmed/22532368>
285. Nagib, A., *et al.* Successful control of selective anterior sacral rhizotomy for treatment of spastic bladder and ureteric reflux in paraplegics. *Med Serv J Can*, 1966. 22: 576.
<https://www.ncbi.nlm.nih.gov/pubmed/5966992>
286. Schneidau, T., *et al.* Selective sacral rhizotomy for the management of neurogenic bladders in spina bifida patients: long-term followup. *J Urol*, 1995. 154: 766.
<https://www.ncbi.nlm.nih.gov/pubmed/7609174>
287. Young, B., *et al.* Percutaneous sacral rhizotomy for neurogenic detrusor hyperreflexia. *J Neurosurg*, 1980. 53: 85.
<https://www.ncbi.nlm.nih.gov/pubmed/7411212>

288. Koldewijn, E.L., *et al.* Bladder compliance after posterior sacral root rhizotomies and anterior sacral root stimulation. *J Urol*, 1994. 151: 955.
<https://www.ncbi.nlm.nih.gov/pubmed/8126835>
289. Krasmik, D., *et al.* Urodynamic results, clinical efficacy, and complication rates of sacral intradural deafferentation and sacral anterior root stimulation in patients with neurogenic lower urinary tract dysfunction resulting from complete spinal cord injury. *Neurourol Urodyn*, 2014. 33: 1202.
<https://www.ncbi.nlm.nih.gov/pubmed/24038405>
290. Singh, G., *et al.* Intravesical oxybutynin in patients with posterior rhizotomies and sacral anterior root stimulators. *Neurourol Urodyn*, 1995. 14: 65.
<https://www.ncbi.nlm.nih.gov/pubmed/7742851>
291. Van Kerrebroeck, P.E., *et al.* Results of the treatment of neurogenic bladder dysfunction in spinal cord injury by sacral posterior root rhizotomy and anterior sacral root stimulation. *J Urol*, 1996. 155: 1378.
<https://www.ncbi.nlm.nih.gov/pubmed/8632580>
292. Kutzenberger, J.S. Surgical therapy of neurogenic detrusor overactivity (hyperreflexia) in paraplegic patients by sacral deafferentation and implant driven micturition by sacral anterior root stimulation: methods, indications, results, complications, and future prospects. *Acta Neurochir*, 2007. 97: 333.
<https://www.ncbi.nlm.nih.gov/pubmed/17691394>
293. Bhadra, N., *et al.* Selective suppression of sphincter activation during sacral anterior nerve root stimulation. *Neurourol Urodyn*, 2002. 21: 55.
<https://www.ncbi.nlm.nih.gov/pubmed/11835425>
294. Kirkham, A.P., *et al.* Neuromodulation through sacral nerve roots 2 to 4 with a Finetech-Brindley sacral posterior and anterior root stimulator. *Spinal Cord*, 2002. 40: 272.
<https://www.ncbi.nlm.nih.gov/pubmed/12037708>
295. Schumacher, S., *et al.* Extradural cold block for selective neurostimulation of the bladder: development of a new technique. *J Urol*, 1999. 161: 950.
<https://www.ncbi.nlm.nih.gov/pubmed/10022732>
296. Brindley, G.S. An implant to empty the bladder or close the urethra. *J Neurol Neurosurg Psychiatry*, 1977. 40: 358.
<https://www.ncbi.nlm.nih.gov/pubmed/406364>
297. Benard, A., *et al.* Comparative cost-effectiveness analysis of sacral anterior root stimulation for rehabilitation of bladder dysfunction in spinal cord injured patients. *Neurosurgery*, 2013. 73: 600.
<https://www.ncbi.nlm.nih.gov/pubmed/23787880>
298. Martens, F.M., *et al.* Quality of life in complete spinal cord injury patients with a Brindley bladder stimulator compared to a matched control group. *Neurourol Urodyn*, 2011. 30: 551.
<https://www.ncbi.nlm.nih.gov/pubmed/21328472>
299. Krebs, J., *et al.* Charcot arthropathy of the spine in spinal cord injured individuals with sacral deafferentation and anterior root stimulator implantation. *Neurourol Urodyn*, 2016. 35: 241.
<https://www.ncbi.nlm.nih.gov/pubmed/25524388>
300. Wollner, J., *et al.* Surgery Illustrated - surgical atlas sacral neuromodulation. *BJU Int*, 2012. 110: 146.
<https://www.ncbi.nlm.nih.gov/pubmed/22691023>
301. Kessler, T.M., *et al.* Sacral neuromodulation for neurogenic lower urinary tract dysfunction: systematic review and meta-analysis. *Eur Urol*, 2010. 58: 865.
<https://www.ncbi.nlm.nih.gov/pubmed/20934242>
302. Lombardi, G., *et al.* Sacral neuromodulation for neurogenic non-obstructive urinary retention in incomplete spinal cord patients: a ten-year follow-up single-centre experience. *Spinal Cord*, 2014. 52: 241.
<https://www.ncbi.nlm.nih.gov/pubmed/24394604>
303. Lay, A.H., *et al.* The role of neuromodulation in patients with neurogenic overactive bladder. *Curr Urol Rep*, 2012. 13: 343.
<https://www.ncbi.nlm.nih.gov/pubmed/22865208>
304. Puccini, F., *et al.* Sacral neuromodulation: an effective treatment for lower urinary tract symptoms in multiple sclerosis. *Int Urogynecol J*, 2016. 27: 347.
<https://www.ncbi.nlm.nih.gov/pubmed/26156206>
305. Zhang, Y.H., *et al.* Enveloping the bladder with displacement of flap of the rectus abdominis muscle for the treatment of neurogenic bladder. *J Urol*, 1990. 144: 1194.
<https://www.ncbi.nlm.nih.gov/pubmed/2146404>
306. Stenzl, A., *et al.* Restoration of voluntary emptying of the bladder by transplantation of innervated free skeletal muscle. *Lancet*, 1998. 351: 1483.
<https://www.ncbi.nlm.nih.gov/pubmed/9605805>

307. Gakis, G., *et al.* Functional detrusor myoplasty for bladder acontractility: long-term results. *J Urol*, 2011. 185: 593.
<https://www.ncbi.nlm.nih.gov/pubmed/21168866>
308. Ninkovic, M., *et al.* The latissimus dorsi detrusor myoplasty for functional treatment of bladder acontractility. *Clin Plast Surg*, 2012. 39: 507.
<https://www.ncbi.nlm.nih.gov/pubmed/23036300>
309. Duel, B.P., *et al.* Alternative techniques for augmentation cystoplasty. *J Urol*, 1998. 159: 998.
<https://www.ncbi.nlm.nih.gov/pubmed/9474216>
310. Snow, B.W., *et al.* Bladder autoaugmentation. *Urol Clin North Am*, 1996. 23: 323.
<https://www.ncbi.nlm.nih.gov/pubmed/8659030>
311. Stohrer, M., *et al.* Bladder auto-augmentation--an alternative for enterocystoplasty: preliminary results. *Neurourol Urodyn*, 1995. 14: 11.
<https://www.ncbi.nlm.nih.gov/pubmed/7742844>
312. Stohrer, M., *et al.* Bladder autoaugmentation in adult patients with neurogenic voiding dysfunction. *Spinal Cord*, 1997. 35: 456.
<https://www.ncbi.nlm.nih.gov/pubmed/9232751>
313. Vainrib, M., *et al.* Differences in urodynamic study variables in adult patients with neurogenic bladder and myelomeningocele before and after augmentation enterocystoplasty. *Neurourol Urodyn*, 2013. 32: 250.
<https://www.ncbi.nlm.nih.gov/pubmed/22965686>
314. Krebs, J., *et al.* Functional outcome of supratrigonal cystectomy and augmentation ileocystoplasty in adult patients with refractory neurogenic lower urinary tract dysfunction. *Neurourol Urodyn*, 2014.
<http://onlinelibrary.wiley.com/doi/10.1002/nau.22709/abstract>
315. Hoen, L., *et al.* Long-term effectiveness and complication rates of bladder augmentation in patients with neurogenic bladder dysfunction: A systematic review. *Neurourol Urodyn*, 2017. 07: 07.
<https://www.ncbi.nlm.nih.gov/pubmed/28169459>
316. Mitsui, T., *et al.* Preoperative renal scar as a risk factor of postoperative metabolic acidosis following ileocystoplasty in patients with neurogenic bladder. *Spinal Cord*, 2014. 52: 292.
<https://www.ncbi.nlm.nih.gov/pubmed/24469144>
317. Krebs, J., *et al.* Functional outcome of supratrigonal cystectomy and augmentation ileocystoplasty in adult patients with refractory neurogenic lower urinary tract dysfunction. *Neurourol Urodyn*, 2016. 35: 260.
<https://www.ncbi.nlm.nih.gov/pubmed/25524480>
318. Moreno, J.G., *et al.* Improved quality of life and sexuality with continent urinary diversion in quadriplegic women with umbilical stoma. *Arch Phys Med Rehabil*, 1995. 76: 758.
<https://www.ncbi.nlm.nih.gov/pubmed/7632132>
319. Peterson, A.C., *et al.* Urinary diversion in patients with spinal cord injury in the United States. *Urology*, 2012. 80: 1247.
<https://www.ncbi.nlm.nih.gov/pubmed/23206770>
320. Sylora, J.A., *et al.* Intermittent self-catheterization by quadriplegic patients via a catheterizable Mitrofanoff channel. *J Urol*, 1997. 157: 48.
<https://www.ncbi.nlm.nih.gov/pubmed/8976213>
321. Van Savage, J.G., *et al.* Transverse retubularized sigmoidovesicostomy continent urinary diversion to the umbilicus. *J Urol*, 2001. 166: 644.
<https://www.ncbi.nlm.nih.gov/pubmed/11458110>
322. Vanni, A.J., *et al.* Ileovesicostomy for the neurogenic bladder patient: outcome and cost comparison of open and robotic assisted techniques. *Urology*, 2011. 77: 1375.
<https://www.ncbi.nlm.nih.gov/pubmed/21146864>
323. Wiener, J.S., *et al.* Bladder augmentation versus urinary diversion in patients with spina bifida in the United States. *J Urol*, 2011. 186: 161.
<https://www.ncbi.nlm.nih.gov/pubmed/21575969>
324. Phe, V., *et al.* Continent catheterizable tubes/stomas in adult neuro-urological patients: A systematic review. *Neurourol Urodyn*, 2017.
<https://www.ncbi.nlm.nih.gov/pubmed/28139848>
325. Atan, A., *et al.* Advantages and risks of ileovesicostomy for the management of neuropathic bladder. *Urology*, 1999. 54: 636.
<https://www.ncbi.nlm.nih.gov/pubmed/10510920>
326. Cass, A.S., *et al.* A 22-year followup of ileal conduits in children with a neurogenic bladder. *J Urol*, 1984. 132: 529.
<https://www.ncbi.nlm.nih.gov/pubmed/6471190>

327. Hald, T., *et al.* Vesicostomy--an alternative urine diversion operation. Long term results. *Scand J Urol Nephrol*, 1978. 12: 227.
<https://www.ncbi.nlm.nih.gov/pubmed/725543>
328. Schwartz, S.L., *et al.* Incontinent ileo-vesicostomy urinary diversion in the treatment of lower urinary tract dysfunction. *J Urol*, 1994. 152: 99.
<https://www.ncbi.nlm.nih.gov/pubmed/8201699>
329. Sakhri, R., *et al.* [Laparoscopic cystectomy and ileal conduit urinary diversion for neurogenic bladders and related conditions. Morbidity and better quality of life]. *Prog Urol*, 2015. 25: 342.
<https://www.ncbi.nlm.nih.gov/pubmed/25726693>
330. Herschorn, S., *et al.* Urinary undiversion in adults with myelodysplasia: long-term followup. *J Urol*, 1994. 152: 329.
<https://www.ncbi.nlm.nih.gov/pubmed/8015064>
331. Mukai, S., *et al.* Retrospective study for risk factors for febrile UTI in spinal cord injury patients with routine concomitant intermittent catheterization in outpatient settings. *Spinal Cord*, 2016. 54: 69.
<https://www.ncbi.nlm.nih.gov/pubmed/26458969>
332. Vasudeva, P., *et al.* Factors implicated in pathogenesis of urinary tract infections in neurogenic bladders: some revered, few forgotten, others ignored. *Neurourol Urodyn*, 2014. 33: 95.
<https://www.ncbi.nlm.nih.gov/pubmed/23460489>
333. Lenherr, S.M., *et al.* Glycemic Control and Urinary Tract Infections in Women with Type 1 Diabetes: Results from the DCCT/EDIC. *J Urol*, 2016. 196: 1129.
<https://www.ncbi.nlm.nih.gov/pubmed/27131462>
334. Bakke, A., *et al.* Bacteriuria in patients treated with clean intermittent catheterization. *Scand J Infect Dis*, 1991. 23: 577.
<https://www.ncbi.nlm.nih.gov/pubmed/9043503>
335. Waites, K.B., *et al.* Epidemiology and risk factors for urinary tract infection following spinal cord injury. *Arch Phys Med Rehabil*, 1993. 74: 691.
<https://www.ncbi.nlm.nih.gov/pubmed/8328888>
336. Nicolle, L.E., *et al.* Infectious Diseases Society of America guidelines for the diagnosis and treatment of asymptomatic bacteriuria in adults. *Clin Infect Dis*, 2005. 40: 643.
<https://www.ncbi.nlm.nih.gov/pubmed/15714408>
337. Goetz, L.L., *et al.* International Spinal Cord Injury Urinary Tract Infection Basic Data Set. *Spinal Cord*, 2013. 51: 700.
<https://www.ncbi.nlm.nih.gov/pubmed/23896666>
338. Pannek, J. Treatment of urinary tract infection in persons with spinal cord injury: guidelines, evidence, and clinical practice. A questionnaire-based survey and review of the literature. *J Spinal Cord Med*, 2011. 34: 11.
<https://www.ncbi.nlm.nih.gov/pubmed/21528621>
339. Deville, W.L., *et al.* The urine dipstick test useful to rule out infections. A meta-analysis of the accuracy. *BMC Urol*, 2004. 4: 4.
<https://www.ncbi.nlm.nih.gov/pubmed/15175113>
340. Hoffman, J.M., *et al.* Nitrite and leukocyte dipstick testing for urinary tract infection in individuals with spinal cord injury. *J Spinal Cord Med*, 2004. 27: 128.
<https://www.ncbi.nlm.nih.gov/pubmed/15162883>
341. Biering-Sorensen, F., *et al.* Urinary tract infections in patients with spinal cord lesions: treatment and prevention. *Drugs*, 2001. 61: 1275.
<https://www.ncbi.nlm.nih.gov/pubmed/11511022>
342. Everaert, K., *et al.* Urinary tract infections in spinal cord injury: prevention and treatment guidelines. *Acta Clin Belg*, 2009. 64: 335.
<https://www.ncbi.nlm.nih.gov/pubmed/19810421>
343. D'Hondt, F., *et al.* Urinary tract infections in patients with spinal cord injuries. *Curr Infect Dis Rep*, 2011. 13: 544.
<https://www.ncbi.nlm.nih.gov/pubmed/21853416>
344. Jia, C., *et al.* Detrusor botulinum toxin A injection significantly decreased urinary tract infection in patients with traumatic spinal cord injury. *Spinal Cord*, 2013. 51: 487.
<https://www.ncbi.nlm.nih.gov/pubmed/23357928>
345. Li, L., *et al.* Impact of hydrophilic catheters on urinary tract infections in people with spinal cord injury: systematic review and meta-analysis of randomized controlled trials. *Arch Phys Med Rehabil*, 2013. 94: 782.
<https://www.ncbi.nlm.nih.gov/pubmed/23168400>

346. Waites, K.B., *et al.* Evaluation of 3 methods of bladder irrigation to treat bacteriuria in persons with neurogenic bladder. *J Spinal Cord Med*, 2006. 29: 217.
<https://www.ncbi.nlm.nih.gov/pubmed/16859225>
347. Gallien, P., *et al.* Cranberry versus placebo in the prevention of urinary infections in multiple sclerosis: a multicenter, randomized, placebo-controlled, double-blind trial. *Mult Scler*, 2014. 20: 1252.
<https://www.ncbi.nlm.nih.gov/pubmed/24402038>
348. Lee, B.S., *et al.* Methenamine hippurate for preventing urinary tract infections. *Cochrane Database Syst Rev*, 2012. 10: Cd003265.
<https://www.ncbi.nlm.nih.gov/pubmed/23076896>
349. Günther, M., *et al.* Harnwegsinfektprophylaxe. Urinansäuerung mittels L-Methionin bei neurogener Blasenfunktionsstörung. [German]. *Urologe B*, 2002. 42: 218. [No abstract available].
<https://www.ncbi.nlm.nih.gov/pubmed/>
350. Hachen, H.J. Oral immunotherapy in paraplegic patients with chronic urinary tract infections: a double-blind, placebo-controlled trial. *J Urol*, 1990. 143: 759.
<https://www.ncbi.nlm.nih.gov/pubmed/2179584>
351. Poirier, C., *et al.* Prevention of urinary tract infections by antibiotic cycling in spinal cord injury patients and low emergence of multidrug resistant bacteria. *Medecine et Maladies Infectieuses*, 2016. 16: 16.
<https://www.ncbi.nlm.nih.gov/pubmed/27321478>
352. Darouiche, R.O., *et al.* Multicenter randomized controlled trial of bacterial interference for prevention of urinary tract infection in patients with neurogenic bladder. *Urology*, 2011. 78: 341.
<https://www.ncbi.nlm.nih.gov/pubmed/21683991>
353. Pannek, J., *et al.* Usefulness of classical homoeopathy for the prevention of urinary tract infections in patients with neurogenic bladder dysfunction: A case series. *Indian J Res Homoeopathy*, 2014. 8: 31.
<http://www.ijrh.org/article.asp?issn=0974-7168;year=2014;volume=8;issue=1;spage=31;epage=36;aulast=Pannek>
354. Rees, P.M., *et al.* Sexual function in men and women with neurological disorders. *Lancet*, 2007. 369: 512.
<https://www.ncbi.nlm.nih.gov/pubmed/17292771>
355. Lombardi, G., *et al.* Treatments for erectile dysfunction in spinal cord patients: alternatives to phosphodiesterase type 5 inhibitors? A review study. *Spinal Cord*, 2015.
<https://www.ncbi.nlm.nih.gov/pubmed/26193811>
356. Jungwirth, A., *et al.*, EAU Guidelines on Male Infertility, in Presented at the 30th Annual Congress in Madrid. 2015.
357. Hatzimouratidis, K., *et al.*, EAU guidelines on Male Sexual Dysfunction and Premature Ejaculation., in Presented at the 30th Annual Congress in Madrid. 2014.
358. Foley, F.W., Sexuality, in *Multiple Sclerosis: A Guide for Families* K. RC., Editor. 2006, Demos Medical Publishing: New York, USA.
359. Annon, J.S., PLISSIT Therapy in *Handbook of Innovative Psychotherapies.* , R. Corsini, Editor. 1981, Wiley & Sons: New York.
360. Fragala, E., *et al.* Relationship between urodynamic findings and sexual function in multiple sclerosis patients with lower urinary tract dysfunction. *Eur J Neurol*, 2015. 22: 485.
<https://www.ncbi.nlm.nih.gov/pubmed/25410608>
361. Game, X., *et al.* Sexual function of young women with myelomeningocele. *J Pediatr Urol*, 2014. 10: 418.
<https://www.ncbi.nlm.nih.gov/pubmed/23992838>
362. 't Hoen, A., *et al.* A Quality Assessment of Patient-Reported Outcome Measures for Sexual Function in Neurologic Patients Using the Consensus-based Standards for the Selection of Health Measurement Instruments Checklist: A Systematic Review. *Eur Urol Focus*, 2016.
<https://www.ncbi.nlm.nih.gov/pubmed/28753768>
363. Chen, L., *et al.* Phosphodiesterase 5 Inhibitors for the Treatment of Erectile Dysfunction: A Trade-off Network Meta-analysis. *Eur Urol*, 2015. 68: 674.
<https://www.ncbi.nlm.nih.gov/pubmed/25817916>
364. Lombardi, G., *et al.* Ten years of phosphodiesterase type 5 inhibitors in spinal cord injured patients. *J Sex Med*, 2009. 6: 1248.
<https://www.ncbi.nlm.nih.gov/pubmed/19210710>

365. Lombardi, G., *et al.* Treating erectile dysfunction and central neurological diseases with oral phosphodiesterase type 5 inhibitors. Review of the literature. *J Sex Med*, 2012. 9: 970.
<https://www.ncbi.nlm.nih.gov/pubmed/22304626>
366. Cardenas, D.D., *et al.* Two phase 3, multicenter, randomized, placebo-controlled clinical trials of fampridine-SR for treatment of spasticity in chronic spinal cord injury. *Spinal Cord*, 2014. 52: 70.
<https://www.ncbi.nlm.nih.gov/pubmed/24216616>
367. Strebel, R.T., *et al.* Apomorphine sublingual as primary or secondary treatment for erectile dysfunction in patients with spinal cord injury. *BJU Int*, 2004. 93: 100.
<https://www.ncbi.nlm.nih.gov/pubmed/14678378>
368. Pohanka, M., *et al.* The long-lasting improvement of sexual dysfunction in patients with advanced, fluctuating Parkinson's disease induced by pergolide: evidence from the results of an open, prospective, one-year trial. *Parkinsonism Relat Disord*, 2005. 11: 509.
<https://www.ncbi.nlm.nih.gov/pubmed/15994112>
369. Chancellor, M.B., *et al.* Prospective comparison of topical minoxidil to vacuum constriction device and intracorporeal papaverine injection in treatment of erectile dysfunction due to spinal cord injury. *Urology*, 1994. 43: 365.
<https://www.ncbi.nlm.nih.gov/pubmed/8134992>
370. Cookson, M.S., *et al.* Long-term results with vacuum constriction device. *J Urol*, 1993. 149: 290.
<https://www.ncbi.nlm.nih.gov/pubmed/8426404>
371. Denil, J., *et al.* Vacuum erection device in spinal cord injured men: patient and partner satisfaction. *Arch Phys Med Rehabil*, 1996. 77: 750.
<https://www.ncbi.nlm.nih.gov/pubmed/8702367>
372. Levine, L.A. External devices for treatment of erectile dysfunction. *Endocrine*, 2004. 23: 157.
<https://www.ncbi.nlm.nih.gov/pubmed/>
373. Levine, L.A., *et al.* Vacuum constriction and external erection devices in erectile dysfunction. *Urol Clin North Am*, 2001. 28: 335.
<https://www.ncbi.nlm.nih.gov/pubmed/15146095>
374. Bella, A.J., *et al.* Intracavernous pharmacotherapy for erectile dysfunction. *Endocrine*, 2004. 23: 149.
<https://www.ncbi.nlm.nih.gov/pubmed/15146094>
375. Bodner, D.R., *et al.* The application of intracavernous injection of vasoactive medications for erection in men with spinal cord injury. *J Urol*, 1987. 138: 310.
<https://www.ncbi.nlm.nih.gov/pubmed/3599245>
376. Deforge, D., *et al.* Male erectile dysfunction following spinal cord injury: a systematic review. *Spinal Cord*, 2006. 44: 465.
<https://www.ncbi.nlm.nih.gov/pubmed/16317419>
377. Dinsmore, W.W., *et al.* Treating men with predominantly nonpsychogenic erectile dysfunction with intracavernosal vasoactive intestinal polypeptide and phentolamine mesylate in a novel auto-injector system: a multicentre double-blind placebo-controlled study. *BJU Int*, 1999. 83: 274.
<https://www.ncbi.nlm.nih.gov/pubmed/10233493>
378. Hirsch, I.H., *et al.* Use of intracavernous injection of prostaglandin E1 for neuropathic erectile dysfunction. *Paraplegia*, 1994. 32: 661.
<https://www.ncbi.nlm.nih.gov/pubmed/7831071>
379. Kapoor, V.K., *et al.* Intracavernous papaverine for impotence in spinal cord injured patients. *Paraplegia*, 1993. 31: 675.
<https://www.ncbi.nlm.nih.gov/pubmed/8259331>
380. Vidal, J., *et al.* Intracavernous pharmacotherapy for management of erectile dysfunction in multiple sclerosis patients. *Rev Neurol*, 1995. 23: 269.
<https://www.ncbi.nlm.nih.gov/pubmed/7497173>
381. Bodner, D.R., *et al.* Intraurethral alprostadil for treatment of erectile dysfunction in patients with spinal cord injury. *Urology*, 1999. 53: 199.
<https://www.ncbi.nlm.nih.gov/pubmed/9886612>
382. Gross, A.J., *et al.* Penile prostheses in paraplegic men. *Br J Urol*, 1996. 78: 262.
<https://www.ncbi.nlm.nih.gov/pubmed/8813925>
383. Kimoto, Y., *et al.* Penile prostheses for the management of the neuropathic bladder and sexual dysfunction in spinal cord injury patients: long term follow up. *Paraplegia*, 1994. 32: 336.
<https://www.ncbi.nlm.nih.gov/pubmed/8058351>
384. Zermann, D.H., *et al.* Penile prosthetic surgery in neurologically impaired patients: long-term followup. *J Urol*, 2006. 175: 1041.
<https://www.ncbi.nlm.nih.gov/pubmed/16469612>

385. Fode, M., *et al.* Male sexual dysfunction and infertility associated with neurological disorders. *Asian J Androl*, 2012. 14: 61.
<https://www.ncbi.nlm.nih.gov/pubmed/22138899>
386. Lim, T.C., *et al.* A simple technique to prevent retrograde ejaculation during assisted ejaculation. *Paraplegia*, 1994. 32: 142.
<https://www.ncbi.nlm.nih.gov/pubmed/8008416>
387. Philippon, M., *et al.* Successful pregnancies and healthy live births using frozen-thawed sperm retrieved by a new modified Hotchkiss procedure in males with retrograde ejaculation: first case series. *Basic Clin Androl*, 2015. 25: 5.
<https://www.ncbi.nlm.nih.gov/pubmed/26034605>
388. Arafa, M.M., *et al.* Prostatic massage: a simple method of semen retrieval in men with spinal cord injury. *Int J Androl*, 2007. 30: 170.
<https://www.ncbi.nlm.nih.gov/pubmed/17298549>
389. Kolettis, P.N., *et al.* Fertility outcomes after electroejaculation in men with spinal cord injury. *Fertil Steril*, 2002. 78: 429.
<https://www.ncbi.nlm.nih.gov/pubmed/12137889>
390. Chehensse, C., *et al.* The spinal control of ejaculation revisited: a systematic review and meta-analysis of anejaculation in spinal cord injured patients. *Hum Reprod Update*, 2013. 19: 507.
<https://www.ncbi.nlm.nih.gov/pubmed/23820516>
391. Beretta, G., *et al.* Reproductive aspects in spinal cord injured males. *Paraplegia*, 1989. 27: 113.
<https://www.ncbi.nlm.nih.gov/pubmed/2717193>
392. Brackett, N.L., *et al.* Application of 2 vibrators salvages ejaculatory failures to 1 vibrator during penile vibratory stimulation in men with spinal cord injuries. *J Urol*, 2007. 177: 660.
<https://www.ncbi.nlm.nih.gov/pubmed/17222653>
393. Sonksen, J., *et al.* Ejaculation induced by penile vibratory stimulation in men with spinal cord injuries. The importance of the vibratory amplitude. *Paraplegia*, 1994. 32: 651.
<https://www.ncbi.nlm.nih.gov/pubmed/7831070>
394. Claydon, V.E., *et al.* Cardiovascular responses to vibrostimulation for sperm retrieval in men with spinal cord injury. *J Spinal Cord Med*, 2006. 29: 207.
<https://www.ncbi.nlm.nih.gov/pubmed/16859224>
395. Eklund, M.B., *et al.* Incidence of autonomic dysreflexia and silent autonomic dysreflexia in men with spinal cord injury undergoing sperm retrieval: implications for clinical practice. *J Spinal Cord Med*, 2008. 31: 33.
<https://www.ncbi.nlm.nih.gov/pubmed/18533409>
396. Soler, J.M., *et al.* Midodrine improves ejaculation in spinal cord injured men. *J Urol*, 2007. 178: 2082.
<https://www.ncbi.nlm.nih.gov/pubmed/17869290>
397. Pecori, C., *et al.* Paternal therapy with disease modifying drugs in multiple sclerosis and pregnancy outcomes: a prospective observational multicentric study. *BMC Neurol*, 2014. 14: 114.
<https://www.ncbi.nlm.nih.gov/pubmed/24884599>
398. Brackett, N.L., *et al.* Treatment of infertility in men with spinal cord injury. *Nat Rev Urol*, 2010. 7: 162.
<https://www.ncbi.nlm.nih.gov/pubmed/20157304>
399. Raviv, G., *et al.* Testicular sperm retrieval and intra cytoplasmic sperm injection provide favorable outcome in spinal cord injury patients, failing conservative reproductive treatment. *Spinal Cord*, 2013. 51: 642.
<https://www.ncbi.nlm.nih.gov/pubmed/23689394>
400. Schatte, E.C., *et al.* Treatment of infertility due to anejaculation in the male with electroejaculation and intracytoplasmic sperm injection. *J Urol*, 2000. 163: 1717.
<https://www.ncbi.nlm.nih.gov/pubmed/10799167>
401. Shieh, J.Y., *et al.* A protocol of electroejaculation and systematic assisted reproductive technology achieved high efficiency and efficacy for pregnancy for anejaculatory men with spinal cord injury. *Arch Phys Med Rehabil*, 2003. 84: 535.
<https://www.ncbi.nlm.nih.gov/pubmed/12690592>
402. Taylor, Z., *et al.* Contribution of the assisted reproductive technologies to fertility in males suffering spinal cord injury. *Aust N Z J Obstet Gynaecol*, 1999. 39: 84.
<https://www.ncbi.nlm.nih.gov/pubmed/10099757>
403. Rutkowski, S.B., *et al.* The influence of bladder management on fertility in spinal cord injured males. *Paraplegia*, 1995. 33: 263.
<https://www.ncbi.nlm.nih.gov/pubmed/7630651>

404. Hamed, S.A., *et al.* Seminal fluid analysis and testicular volume in adults with epilepsy receiving valproate. *J Clin Neurosci*, 2015. 22: 508.
<https://www.ncbi.nlm.nih.gov/pubmed/25636832>
405. Ohl, D.A., *et al.* Electroejaculation versus vibratory stimulation in spinal cord injured men: sperm quality and patient preference. *J Urol*, 1997. 157: 2147.
<https://www.ncbi.nlm.nih.gov/pubmed/9146603>
406. Brackett, N.L., *et al.* Semen quality of spinal cord injured men is better when obtained by vibratory stimulation versus electroejaculation. *J Urol*, 1997. 157: 151.
<https://www.ncbi.nlm.nih.gov/pubmed/8976239>
407. Brackett, N.L., *et al.* Semen retrieval in men with spinal cord injury is improved by interrupting current delivery during electroejaculation. *J Urol*, 2002. 167: 201.
<https://www.ncbi.nlm.nih.gov/pubmed/11743305>
408. DeForge, D., *et al.* Fertility following spinal cord injury: a systematic review. *Spinal Cord*, 2005. 43: 693.
<https://www.ncbi.nlm.nih.gov/pubmed/15951744>
409. Ferreiro-Velasco, M.E., *et al.* Sexual issues in a sample of women with spinal cord injury. *Spinal Cord*, 2005. 43: 51.
<https://www.ncbi.nlm.nih.gov/pubmed/15303115>
410. Kreuter, M., *et al.* Sexuality and sexual life in women with spinal cord injury: a controlled study. *J Rehabil Med*, 2008. 40: 61.
<https://www.ncbi.nlm.nih.gov/pubmed/18176739>
411. Kreuter, M., *et al.* Sexual adjustment and quality of relationship in spinal paraplegia: a controlled study. *Arch Phys Med Rehabil*, 1996. 77: 541.
<https://www.ncbi.nlm.nih.gov/pubmed/8831469>
412. Kessler, T.M., *et al.* Sexual dysfunction in multiple sclerosis. *Expert Rev Neurother*, 2009. 9: 341.
<https://www.ncbi.nlm.nih.gov/pubmed/19271943>
413. Lew-Starowicz, M., *et al.* Prevalence of Sexual Dysfunctions Among Women with Multiple Sclerosis. *Sex Disabil*, 2013. 31: 141.
<https://www.ncbi.nlm.nih.gov/pubmed/23704801>
414. Reitz, A., *et al.* Impact of spinal cord injury on sexual health and quality of life. *Int J Impot Res*, 2004. 16: 167.
<https://www.ncbi.nlm.nih.gov/pubmed/14973522>
415. Harrison, J., *et al.* Factors associated with sexual functioning in women following spinal cord injury. *Paraplegia*, 1995. 33: 687.
<https://www.ncbi.nlm.nih.gov/pubmed/8927405>
416. Westgren, N., *et al.* Sexuality in women with traumatic spinal cord injury. *Acta Obstet Gynecol Scand*, 1997. 76: 977.
<https://www.ncbi.nlm.nih.gov/pubmed/9435740>
417. Fruhauf, S., *et al.* Efficacy of psychological interventions for sexual dysfunction: a systematic review and meta-analysis. *Arch Sex Behav*, 2013. 42: 915.
<https://www.ncbi.nlm.nih.gov/pubmed/23559141>
418. Alexander, M., *et al.* Spinal cord injuries and orgasm: a review. *J Sex Marital Ther*, 2008. 34: 308.
<https://www.ncbi.nlm.nih.gov/pubmed/18576233>
419. Sipski, M.L., *et al.* Physiologic parameters associated with sexual arousal in women with incomplete spinal cord injuries. *Arch Phys Med Rehabil*, 1997. 78: 305.
<https://www.ncbi.nlm.nih.gov/pubmed/9084355>
420. Sipski, M.L., *et al.* Sexual arousal and orgasm in women: effects of spinal cord injury. *Ann Neurol*, 2001. 49: 35.
<https://www.ncbi.nlm.nih.gov/pubmed/11198294>
421. McAlonan, S. Improving sexual rehabilitation services: the patient's perspective. *Am J Occup Ther*, 1996. 50: 826.
<https://www.ncbi.nlm.nih.gov/pubmed/8947375>
422. Schopp, L.H., *et al.* Impact of comprehensive gynecologic services on health maintenance behaviours among women with spinal cord injury. *Disabil Rehabil*, 2002. 24: 899.
<https://www.ncbi.nlm.nih.gov/pubmed/12519485>
423. Sukumaran, S.C., *et al.* Polytherapy increases the risk of infertility in women with epilepsy. *Neurology*, 2010. 75: 1351.
<https://www.ncbi.nlm.nih.gov/pubmed/20938026>

424. Axel, S.J. Spinal cord injured women's concerns: menstruation and pregnancy. *Rehabil Nurs*, 1982. 7: 10.
<https://www.ncbi.nlm.nih.gov/pubmed/6921826>
425. Jackson, A.B., *et al.* A multicenter study of women's self-reported reproductive health after spinal cord injury. *Arch Phys Med Rehabil*, 1999. 80: 1420.
<https://www.ncbi.nlm.nih.gov/pubmed/10569436>
426. Baker, E.R., *et al.* Pregnancy in spinal cord injured women. *Arch Phys Med Rehabil*, 1996. 77: 501.
<https://www.ncbi.nlm.nih.gov/pubmed/8629929>
427. Baker, E.R., *et al.* Risks associated with pregnancy in spinal cord-injured women. *Obstet Gynecol*, 1992. 80: 425.
<https://www.ncbi.nlm.nih.gov/pubmed/1495699>
428. Bertschy, S., *et al.* Delivering care under uncertainty: Swiss providers' experiences in caring for women with spinal cord injury during pregnancy and childbirth - an expert interview study. *BMC Pregnancy Childbirth*, 2016. 16: 181.
<https://www.ncbi.nlm.nih.gov/pubmed/27443838>
429. Le Liepvre, H., *et al.* Pregnancy in spinal cord-injured women, a cohort study of 37 pregnancies in 25 women. *Spinal Cord*, 2017. 55: 167.
<https://www.ncbi.nlm.nih.gov/pubmed/27670808>
430. Skowronski, E., *et al.* Obstetric management following traumatic tetraplegia: case series and literature review. *Aust N Z J Obstet Gynaecol*, 2008. 48: 485.
<https://www.ncbi.nlm.nih.gov/pubmed/19032665>
431. Cross, L.L., *et al.* Pregnancy, labor and delivery post spinal cord injury. *Paraplegia*, 1992. 30: 890.
<https://www.ncbi.nlm.nih.gov/pubmed/1287543>
432. Hughes, S.J., *et al.* Management of the pregnant woman with spinal cord injuries. *Br J Obstet Gynaecol*, 1991. 98: 513.
<https://www.ncbi.nlm.nih.gov/pubmed/1873238>
433. Dannels, A., *et al.* The perimenopause experience for women with spinal cord injuries. *SCI Nurs*, 2004. 21: 9.
<https://www.ncbi.nlm.nih.gov/pubmed/15176344>
434. Vukusic, S., *et al.* Multiple sclerosis and pregnancy in the 'treatment era'. *Nat Rev Neurol*, 2015. 11: 280.
<https://www.ncbi.nlm.nih.gov/pubmed/25896084>
435. Amato, M.P., *et al.* Management of pregnancy-related issues in multiple sclerosis patients: the need for an interdisciplinary approach. *Neurol Sci*, 2017. 38: 1849.
<https://www.ncbi.nlm.nih.gov/pubmed/28770366>
436. Delaney, K.E., *et al.* Multiple sclerosis and sexual dysfunction: A need for further education and interdisciplinary care. *NeuroRehabilitation*, 2017. 41: 317.
<https://www.ncbi.nlm.nih.gov/pubmed/29036844>
437. Bove, R., *et al.* Management of multiple sclerosis during pregnancy and the reproductive years: a systematic review. *Obstet Gynecol*, 2014. 124: 1157.
<https://www.ncbi.nlm.nih.gov/pubmed/25415167>
438. Abrams, P., *et al.* A proposed guideline for the urological management of patients with spinal cord injury. *BJU Int*, 2008. 101: 989.
<https://www.ncbi.nlm.nih.gov/pubmed/18279449>
439. Pannek, J., *et al.* Clinical usefulness of ultrasound assessment of detrusor wall thickness in patients with neurogenic lower urinary tract dysfunction due to spinal cord injury: Urodynamics made easy? *World J Urol*, 2013. 31: 659.
<https://www.ncbi.nlm.nih.gov/pubmed/23073657>
440. Silva, J.A., *et al.* Association between the bladder wall thickness and urodynamic findings in patients with spinal cord injury. *World J Urol*, 2015. 33: 131.
<https://www.ncbi.nlm.nih.gov/pubmed/24573904>
441. Veenboer, P.W., *et al.* Diagnostic accuracy of Tc-99m DMSA scintigraphy and renal ultrasonography for detecting renal scarring and relative function in patients with spinal dysraphism. *Neurourol Urodyn*, 2015. 34: 513.
<https://www.ncbi.nlm.nih.gov/pubmed/24706504>
442. Lewis, J., *et al.* A framework for transitioning patients from pediatric to adult health settings for patients with neurogenic bladder. *Neurourol Urodyn*, 2017. 36: 973.
<https://www.ncbi.nlm.nih.gov/pubmed/27276694>

5. CONFLICT OF INTEREST

All members of the Neuro-urology Guidelines working group have provided disclosure statements of all relationships that they have that might be perceived as a potential source of a conflict of interest. This information is publically accessible through the European Association of Urology website: <http://uroweb.org/guideline>. This guidelines document was developed with the financial support of the European Association of Urology. No external sources of funding and support have been involved. The EAU is a non-profit organisation and funding is limited to administrative assistance and travel and meeting expenses. No honoraria or other reimbursements have been provided.

6. CITATION INFORMATION

The format in which to cite the EAU Guidelines will vary depending on the style guide of the journal in which the citation appears. Accordingly, the number of authors or whether, for instance, to include the publisher, location, or an ISBN number may vary.

The compilation of the complete Guidelines should be referenced as:

EAU Guidelines. Edn. presented at the EAU Annual Congress Copenhagen 2018. ISBN 978-94-92671-01-1.

If a publisher and/or location is required, include:

EAU Guidelines Office, Arnhem, The Netherlands. <http://uroweb.org/guidelines/compilations-of-all-guidelines/>

References to individual guidelines should be structured in the following way:

Contributors' names. Title of resource. Publication type. ISBN. Publisher and publisher location, year.