

Guidelines on Neurogenic Lower Urinary Tract Dysfunction

M. Stöhrer, D. Castro-Diaz, E. Chartier-Kastler, G. Del Popolo,
G. Kramer, J. Pannek, P. Radziszewski, J-J. Wyndaele

TABLE OF CONTENTS

PAGE

1.	AIM AND STATUS OF THESE GUIDELINES	4
1.1	Purpose	4
1.2	Standardization	4
1.3	References	4
2.	BACKGROUND	4
2.1	Risk factors and epidemiology	4
2.1.1	Brain tumours	4
2.1.2	Dementia	4
2.1.3	Mental retardation	4
2.1.4	Cerebral palsy	5
2.1.5	Normal pressure hydrocephalus	5
2.1.6	Basal ganglia pathology (Parkinson's disease, Huntington's disease, Shy-Drager syndrome, etc)	5
2.1.7	Cerebrovascular (CVA) pathology	5
2.1.8	Demyelination	5
2.1.9	Spinal cord lesions	5
2.1.10	Disc disease	5
2.1.11	Spinal stenosis and spine surgery	5
2.1.12	Peripheral neuropathy	6
2.1.13	Other conditions (SLE)	6
2.1.14	HIV	6
2.1.15	Regional spinal anaesthesia	6
2.1.16	Iatrogenic	6
2.2	Standardization of terminology	6
2.2.1	Introduction	6
2.2.2	Definitions	7
2.3	Classification	9
2.3.1	Recommendation for clinical practice	10
2.4	Timing of diagnosis and treatment	10
2.5	References	10
3.	DIAGNOSIS	17
3.1	Introduction	17
3.2	History	17
3.2.1	General history	17
3.2.2	Specific history	18
3.2.3	Guidelines for history taking	18
3.3	Physical examination	18
3.3.1	General physical examination	18
3.3.2	Neuro-urological examination	19
3.3.3	Essential investigations	21
3.3.4	Guidelines for physical examination	21
3.4	Urodynamics	21
3.4.1	Introduction	21
3.4.2	Urodynamic tests	21
3.4.3	Specific uro-neurophysiological tests	22
3.4.4	Guidelines for urodynamics and uro-neurophysiology	23
3.5	Typical manifestations of NLUTD	23
3.6	References	23
4.	TREATMENT	25
4.1	Introduction	25
4.2	Non-invasive conservative treatment	26
4.2.1	Assisted bladder emptying	26
4.2.2	Lower urinary tract rehabilitation	26
4.2.3	Drug treatment	26

4.2.4	Electrical neuromodulation	27
4.2.5	External appliances	27
4.2.6	Guidelines for non-invasive conservative treatment	27
4.3	Minimal invasive treatment	28
4.3.1	Catheterization	28
4.3.2	Guidelines for catheterization	28
4.3.3	Intravesical drug treatment	28
4.3.4	Intravesical electrostimulation	28
4.3.5	Botulinum toxin injections in the bladder	28
4.3.6	Bladder neck and urethral procedures	29
4.3.7	Guidelines for minimal invasive treatment	29
4.4	Surgical treatment	29
4.4.1	Urethral and bladder neck procedures	29
4.4.2	Detrusor myectomy (auto-augmentation)	30
4.4.3	Denervation, deafferentation, neurostimulation, neuromodulation	30
4.4.4	Bladder covering by striated muscle	30
4.4.5	Bladder augmentation or substitution	30
4.4.6	Urinary diversion	31
4.5	Guidelines for surgical treatment	31
4.6	References	32
5.	TREATMENT OF VESICO-URETERAL REFLUX	46
5.1	Treatment options	46
5.2	References	46
6.	QUALITY OF LIFE	47
6.1	Introduction	47
6.2	Conclusions and recommendations	48
6.3	References	48
7.	FOLLOW UP	48
7.1	Introduction	48
7.2	Guidelines for follow-up	48
7.3	References	49
8.	CONCLUSIONS	50
9.	ABBREVIATIONS USED IN THE TEXT	51

1. AIM AND STATUS OF THESE GUIDELINES

1.1 Purpose

The purpose of these clinical guidelines is to provide useful information for clinical practitioners on the incidence, definitions, diagnosis, therapy, and follow-up observation of the condition of neurogenic lower urinary tract dysfunction (NLUTD). These guidelines reflect the current opinion of the experts in this specific pathology and thus represent a state-of-the-art reference for all clinicians, as of the date of its presentation to the European Association of Urology.

1.2 Standardization

The terminology used and the diagnostic procedures advised throughout these guidelines follow the recommendations for investigations on the lower urinary tract (LUT) as published by the International Continence Society (ICS) (1-3).

1.3 REFERENCES

1. Stohrer M, Goepel M, Kondo A, Kramer G, Madersbacher H, Millard R, Rossier A, Wyndaele JJ. The standardization of terminology in neurogenic lower urinary tract dysfunction with suggestions for diagnostic procedures. *Neurourol Urodyn* 1999;18(2):139-58.
<http://www.ncbi.nlm.nih.gov/pubmed/10081953>
2. Abrams P, Cardozo L, Fall M, Griffiths D, Rosier P, Ulmsten U, van Kerrebroeck P, Victor A, Wein A. The standardisation of terminology of lower urinary tract function: Report from the Standardisation Subcommittee of the International Continence Society. *Neurourol Urodyn* 2002;21(2):167-78.
<http://www.ncbi.nlm.nih.gov/pubmed/11857671>
3. Schafer W, Abrams P, Liao L, Mattiasson A, Pesce F, Spangberg A, Sterling AM, Zinner NR, van Kerrebroeck P. Good urodynamic practices: uroflowmetry, filling cystometry, and pressure-flow Studies. *Neurourol Urodyn* 2002;21(3):261-74.
<http://www.ncbi.nlm.nih.gov/pubmed/11948720>

2. BACKGROUND

2.1 Risk factors and epidemiology

NLUTD may be caused by various diseases and events affecting the nervous systems controlling the LUT. The resulting lower urinary tract dysfunction (LUTD) depends grossly on the location and the extent of the neurological lesion (see also Section 2.3).

There are no figures on the overall prevalence of NLUTD in the general population, but data are available on the prevalence of the underlying conditions and the relative risk of those for the development of NLUTD. It is important to realize that most of these data show a very wide range of prevalence figures because of the low level of evidence in most published data and smaller sample sizes.

2.1.1 Brain tumours

Brain tumours can cause LUTD in 24% of patients (1). More recently, mostly case reports to small series have been published (2-3). In a series of patients with brain tumours, voiding difficulty was reported in 46/152 (30%) of patients with tumours in the posterior fossa, while urinary incontinence occurred in only three (1.9%) patients (4).

Urinary retention was found in 12/17 (71%) children with pontine glioma (5).

2.1.2 Dementia

It is not easy to distinguish dementia-associated LUTD from LUTD caused by age-related changes of the bladder and other concomitant diseases and therefore the true incidence of incontinence caused by dementia is not known. However, it has been shown that incontinence is much more frequent in geriatric patients with dementia than in patients without dementia (6, 7).

Alzheimer, Lewy body dementia, Binswanger, Nasu-Hakola and Pick diseases frequently cause non-specific NLUTD (8-13). The occurrence of incontinence is reported to be between 23% and 48% (14, 15) in patients with Alzheimer's disease. The onset of incontinence usually correlates with disease progression (16). A male-to-female ratio of dementia-related incontinence was found to be 1:15.

2.1.3 Mental retardation

In mental retardation, depending on the grade of the disorder, 12-65% of LUTD was described (17, 18).

2.1.4 Cerebral palsy

LUTD has been described in about 30-40% (19, 20).

2.1.5 Normal pressure hydrocephalus

There have only been case reports of LUTD (21-23).

2.1.6 Basal ganglia pathology (Parkinson's disease, Huntington's disease, Shy-Drager syndrome, etc)

Parkinson's disease is accompanied by NLUTD in 37.9-70% (24-25).

In the rare Shy-Drager syndrome, almost all patients have NLUTD (26), with incontinence found in 73% (27).

Hattori et al. (28) reported that 60% of Parkinson patients had urinary symptoms. However, Gray et al. (29) reported that functional disturbances of the LUT in Parkinson's disease were not disease-specific and were correlated only with age. Recent, control-based studies have given the prevalence of LUT symptoms as 27-63.9% using validated questionnaires (30-32), or 53% in men and 63% in women using a non-validated questionnaire, which included a urinary incontinence category (32), with all these values being significantly higher than in healthy controls. In most patients, the onset of the bladder dysfunction occurred after the motor disorder had appeared.

2.1.7 Cerebrovascular (CVA) pathology

Cerebrovascular pathology causes hemiplegia with remnant incontinence NLUTD in 20-50% of patients (33-34), with decreasing prevalence in the post-insult period (35). In 1996, 53% of patients with CVA pathology had significant urinary complaints at 3 months (36). Without proper treatment, at 6 months after the CVA, 20-30% of patients still suffered from urinary incontinence (37). The commonest cystometric finding was detrusor overactivity (38-43).

In 39 patients who had brainstem strokes, urinary symptoms were present in almost 50%, nocturia and voiding difficulty in 28%, urinary retention in 21%, and urinary incontinence in 8%. Several case histories have been published presenting difficulties with micturition in the presence of various brainstem pathologies (45-46).

2.1.8 Demyelination

Multiple sclerosis causes NLUTD in 50-90% of the patients (47-49).

The reported incidence of voiding dysfunction in multiple sclerosis is 33-52% in patients sampled consecutively, regardless of urinary symptoms. This incidence is related to the disability status of the patient (50). There is almost a 100% chance of having LUT dysfunction once these patients experience difficulties with walking. NLUTD is the presenting symptom in 2-12% of patients, with this finding being as high as 34% in some studies (51). LUT dysfunction appears mostly during the 10 years following the diagnosis (52).

2.1.9 Spinal cord lesions

Spinal cord lesions can be traumatic, vascular, medical or congenital. An incidence of 30-40 new cases per million population is the accepted average for the USA. Most of these patients will develop NLUTD (53). The prevalence of spina bifida and other congenital nerve tube defects in the UK is 8-9 per 10,000 aged 10-69 years, with the greatest prevalence in the age group 25-29 years (54), and in the USA 1 per 1,000 births (55). The incidence of urethrovaginal dysfunction in myelomeningocele is not completely known, but most studies suggest it is very high at 90-97% (56). About 50% of these children will have detrusor sphincter dyssynergia (DSD) (57, 58).

2.1.10 Disc disease

This is reported to cause NLUTD in 28-87% of the patients (<20%) (59-60). The incidence of cauda equina syndrome due to central lumbar disc prolapse is relatively rare and is about 1-5% of all prolapsed lumbar discs (60-63, 64-67). There have been case reports of NLUT without cauda equine syndrome (69).

2.1.11 Spinal stenosis and spine surgery

About 50% of patients seeking help for intractable leg pain due to spinal stenosis report symptoms of LUTD, such as a sense of incomplete bladder emptying, urinary hesitancy, incontinence, nocturia or urinary tract infections (UTIs) (70). These symptoms may be overlooked or attributed to primary urological disorders, with 61-62% affected by LUTD (71, 72). The prevalence of neurological bladder is more significantly associated with the anteroposterior diameter of the dural sac than with its cross-sectional area.

Spinal surgery is related to LUTD in 38-60% of patients (73, 74).

2.1.12 *Peripheral neuropathy*

Diabetes: This common metabolic disorder has a prevalence of about 2.5% in the American population, but the disease may be subclinical for many years. No specific criteria exist for secondary neuropathy in this condition, but it is generally accepted that 50% of patients will develop somatic neuropathy, with 75-100% of these patients developing NLUTD (75, 76). Diabetic patients suffer from various polyneuropathies, with 'diabetic cystopathy' reported in 43-87% of insulin-dependent diabetics without gender or age differences. It is also described in about 25% of type 2 diabetic patients on oral hypoglycaemic treatment (77).

Alcohol abuse will eventually cause peripheral neuropathy. This has a reported prevalence that varies widely from 5-15% (78) to 64% (79). NLUTD is probably more likely to be present in patients with liver cirrhosis. The parasympathetic nervous system is attacked more than the sympathetic nervous system (79).

Less prevalent peripheral neuropathies include the following:

- Porphyria: bladder dilatation occurs in up to 12% of patients (80).
- Sarcoidosis: NLUTD is rare (81).
- Lumbosacral zone and genital herpes: incidence of LUT dysfunction is as high as 28% when only lumbosacral dermatome-involved patients are considered. The overall incidence is 4% (82, 83). NLUTD is transient in most patients.
- Guillain Barré syndrome: the prevalence of micturition disorders varies from 25% to more than 80% (84,85), but is regressive in most cases (86). The true incidence is uncertain because, during the acute phase, patients are usually managed by indwelling catheter.

2.1.13 *Other conditions (SLE)*

Nervous system involvement occurs in about half of patients with systemic lupus erythematosus (SLE). Symptoms of LUT dysfunction can occur, but data on prevalence are rare and give an incidence of 1% (87, 88).

2.1.14 *HIV*

Voiding problems have been described in 12% of HIV-infected patients, mostly in advanced stages of the disease (89, 90).

2.1.15 *Regional spinal anaesthesia*

This may cause NLUTD but no prevalence figures have been found (91, 92).

2.1.16 *Iatrogenic*

Abdominoperineal resection of rectum has been described as causing NLUTD in up to 50% of patients (93, 94). One study has reported that NLUTD remains a long-term problem in only 10% (95); however, the study was not clear whether this was because the neurological lesion was cured or bladder rehabilitation was successful. Surgical prevention with nerve preservation was shown to be important (96, 97).

NLUTD has been reported following simple hysterectomy (98) and in 8-57% of patients following radical hysterectomy or pelvic irradiation for cervical cancer (99-102). Surgical prevention can be used to prevent it (103). Neurological dysfunction of the pelvic floor has been demonstrated following radical prostatectomy (104).

2.2 **Standardization of terminology**

2.2.1 *Introduction*

Several national or international guidelines have already been published for the care of patients with NLUTD (105-108). The guidelines will evolve as time goes by. The guidelines include definitions of important terms and procedures. The ICS NLUTD standardization report (106) deals specifically with the standardization of terminology and urodynamic investigation in patients with NLUTD. Other relevant definitions are found in the general ICS standardization report (109).

Section 2.2.2 lists the definitions from these references, partly adapted, and other definitions considered useful for clinical practice in NLUTD (Tables 1 and 2). For specific definitions relating to urodynamic investigation, the reader is referred to the appropriate ICS report (106).

2.2.2 Definitions

Table 1: Definitions useful in clinical practice

Acontractility, detrusor	See below under voiding phase
Acontractility, urethral sphincter	See below under storage phase
Autonomic dysreflexia	Increase of sympathetic reflex due to noxious stimuli with symptoms or signs of headache, hypertension, flushing face and perspiration
Capacity	See below under storage phase
Catheterization, indwelling	Emptying of the bladder by a catheter that is introduced (semi-) permanently
Catheterization, intermittent (IC)	Emptying of the bladder by a catheter that is removed after the procedure, mostly at regular intervals
<ul style="list-style-type: none"> • Aseptic IC 	The catheters remain sterile, the genitals are disinfected, and disinfecting lubricant is used
<ul style="list-style-type: none"> • Clean IC 	Disposable or cleansed re-usable catheters, genitals washed
<ul style="list-style-type: none"> • Sterile IC 	Complete sterile setting, including sterile gloves, forceps, gown and mask
<ul style="list-style-type: none"> • Intermittent self-catheterization (ISC) 	IC performed by the patient
Compliance, detrusor	See below under storage phase
Condition	Evidence of relevant pathological processes
Diary, urinary	Record of times of micturitions and voided volumes, incontinence episodes, pad usage, and other relevant information
<ul style="list-style-type: none"> • Frequency volume chart (FVC) 	Times of micturitions and voided volumes only
<ul style="list-style-type: none"> • Micturition time chart (MTC) 	Times of micturitions only
Filling rate, physiological	Below the predicted maximum: body weight (kg)/ 4 in mL/s (109, 110)
Hesitancy	Difficulty in initiating micturition; delay in the onset of micturition after the individual is ready to pass urine
Intermittency	Urine flow stops and starts on one or more occasions during voiding
Leak point pressure (LPP)	See below under storage phase
Lower motor neuron lesion (LMNL)	Lesion at or below the S1-S2 spinal cord level
Neurogenic lower urinary tract dysfunction (NLUTD)	Lower urinary tract dysfunction secondary to confirmed pathology of the nervous supply
Observation, specific	Observation made during specific diagnostic procedure
Overactivity, bladder	See below under symptom syndrome
Overactivity, detrusor	See below under storage phase
Rehabilitation, LUT	Non-surgical non-pharmacological treatment for LUT dysfunction
Sign	To verify symptoms and classify them
Sphincter, urethral, non-relaxing	See below under voiding phase
Symptom	Subjective indicator of a disease or change in condition, as perceived by the patient, carer, or partner that may lead the patient to seek help from healthcare professionals
Upper motor neuron lesion (UMNL)	Lesion above the S1-S2 spinal cord level
Voiding, balanced: In patients with NLUTD (<80 mL or <20% of bladder volume)	Voiding with physiological detrusor pressure and low residual
Voiding, triggered	Voiding initiated by manoeuvres to elicit reflex detrusor contraction by exteroceptive stimuli
Volume, overactivity	See below under storage phase

Table 2: Further definitions useful in clinical practice

Storage phase	
Maximum anaesthetic bladder capacity	Maximum bladder filling volume under deep general or spinal anaesthesia
Increased daytime frequency	Self-explanatory; the normal frequency can be estimated at about 8 times per day (111)
Nocturia	Waking at night one or more times to void
Urgency	The symptom of a sudden compelling desire to pass urine that is difficult to defer
Urinary incontinence	Any involuntary leakage of urine
<ul style="list-style-type: none"> Stress urinary incontinence 	On effort or exertion, or on sneezing or coughing
<ul style="list-style-type: none"> Urge urinary incontinence 	Accompanied by or immediately preceded by urgency
<ul style="list-style-type: none"> Mixed urinary incontinence 	Associated with urgency and also exertion, effort, sneezing, or coughing
<ul style="list-style-type: none"> Continuous urinary incontinence 	
Bladder sensation	
<i>Normal</i>	
<ul style="list-style-type: none"> Symptom and history 	Awareness of bladder filling and increasing sensation up to a strong desire to void
<ul style="list-style-type: none"> Urodynamics 	First sensation of bladder filling, first desire to void, and strong desire to void at realistic bladder volumes
<i>Increased</i>	
<ul style="list-style-type: none"> Symptom and history 	An early and persistent desire to void
<ul style="list-style-type: none"> Urodynamics 	Any of the three urodynamic parameters mentioned under 'normal' persistently at low bladder volume
<i>Reduced</i>	
<ul style="list-style-type: none"> Symptom and history 	Awareness of bladder filling but no definite desire to void
<ul style="list-style-type: none"> Urodynamics 	Diminished sensation throughout bladder filling
<i>Absent</i> No sensation of bladder filling or desire to void	
Non-specific	Perception of bladder filling as abdominal fullness, vegetative symptoms, or spasticity
<i>Definitions valid after urodynamic confirmation only</i>	
Cystometric capacity	
<ul style="list-style-type: none"> Maximum cystometric capacity 	Bladder volume at strong desire to void
<ul style="list-style-type: none"> High-capacity bladder 	Bladder volume at cystometric capacity far over the mean voided volume, estimated from the bladder diary, with no significant increase in detrusor pressure under non-anaesthetized condition
Normal detrusor function	Little or no pressure increase during filling; no involuntary phasic contractions despite provocation
Detrusor overactivity	
<ul style="list-style-type: none"> Phasic detrusor overactivity 	Characteristic phasic contraction
<ul style="list-style-type: none"> Terminal detrusor overactivity 	A single contraction at cystometric capacity
<ul style="list-style-type: none"> High pressure detrusor overactivity 	Maximal detrusor pressure > 40 cm H ₂ O (106, 112)
<ul style="list-style-type: none"> Overactivity volume 	Bladder volume at first occurrence of detrusor overactivity
<ul style="list-style-type: none"> Detrusor overactivity incontinence 	Self-explanatory
Leak point pressure	
<ul style="list-style-type: none"> Detrusor leak point pressure (DLPP) 	Lowest value of detrusor pressure at which leakage is observed in the absence of abdominal strain or detrusor contraction

<ul style="list-style-type: none"> Abdominal leak point pressure 	Lowest value of intentionally increased intravesical pressure that provokes leakage in the absence of a detrusor contraction
Detrusor compliance	Relationship between change in bladder volume (ΔV) and change in detrusor pressure (Δp_{det}): $C = \Delta V / \Delta p_{det}$ (ml/cm H ₂ O)
<ul style="list-style-type: none"> Low detrusor compliance 	$C = \Delta V / \Delta p_{det} < 20$ mL/cm H ₂ O (106)
Break volume	Bladder volume after which a sudden significant decrease in detrusor compliance is observed
Urethral sphincter acontractility	No evidence of sphincter contraction during filling, particularly at higher bladder volumes, or during abdominal pressure increase
Voiding phase	
<ul style="list-style-type: none"> Slow stream 	Reduced urine flow rate
<ul style="list-style-type: none"> Intermittent stream (intermittency) 	Stopping and starting of urine flow during micturition
<ul style="list-style-type: none"> Hesitancy 	Difficulty in initiating micturition
<ul style="list-style-type: none"> Straining 	Muscular effort to initiate, maintain, or improve urinary stream
<ul style="list-style-type: none"> Terminal dribble 	Prolonged final part of micturition when the flow has slowed to a trickle/dribble
<i>Definitions valid after urodynamic confirmation only</i>	
Normal detrusor function	Voluntarily initiated detrusor contraction that causes complete bladder emptying within a normal time span
Detrusor underactivity	Contraction of reduced strength and/or duration
Acontractile detrusor	Absent contraction
Non-relaxing urethral sphincter	Self-explanatory
Detrusor sphincter dyssynergia (DSD)	Detrusor contraction concurrent with an involuntary contraction of the urethra and/or periurethral striated musculature
Post-micturition phase	
Feeling of incomplete emptying (symptom only)	
Post-micturition dribble: involuntary leakage of urine shortly after finishing the micturition	
Pain, discomfort or pressure sensation in the lower urinary tract and genitalia that may be related to bladder filling or voiding, may be felt after micturition, or be continuous	
Symptom syndrome: combination of symptoms	
<ul style="list-style-type: none"> Overactive bladder syndrome: urgency with or without urge incontinence, usually with frequency and nocturia Synonyms: Urge syndrome, urgency-frequency syndrome This syndrome is suggestive for LUTD 	

2.3 Classification

The classification of NLUTD helps to facilitate the understanding and management of NLUTD and to provide a standardized terminology of the disease processes. The normal LUT function depends on neural integration at, and between, the peripheral, spinal cord, and central nervous systems. The gross type of NLUTD is dependent on the location and the extent of the lesion: suprapontine or pontine, suprasacral spinal cord, or subsacral and peripheral (53, 107).

The classification systems for NLUTD are based on either the neurological substrate (type and location of the neurological lesion) (113), the neuro-urological substrate (neurological lesion and LUTD) (114-116), the type of LUTD (117, 118), or are strictly functional (107, 109, 119-122). Many descriptive terms were derived from these classification systems. However, they are standardized only within any specific system, have little meaning outside the system, and can sometimes be confusing.

A perfect classification system does not exist. Neurological classification systems, by nature, cannot describe the LUTD completely and vice versa. Individual variations exist in the NLUTD caused by a specific neurological lesion, so that the description of the NLUTD should be individualized for any particular patient.

Madersbacher (107, 122) presented a very simple classification, which basically focused on the therapeutic consequences (Figure 2.1). It is based on the clinical concept that the important differentiation in the diagnosis exists between the situations of high and low detrusor pressure during the filling phase and

urethral sphincter relaxation and non- relaxation or DSD during the voiding phase. A non-relaxed sphincter or DSD will cause high detrusor pressure during the voiding phase. This classification is the easiest one for general use in the clinical diagnosis of NLUTD.

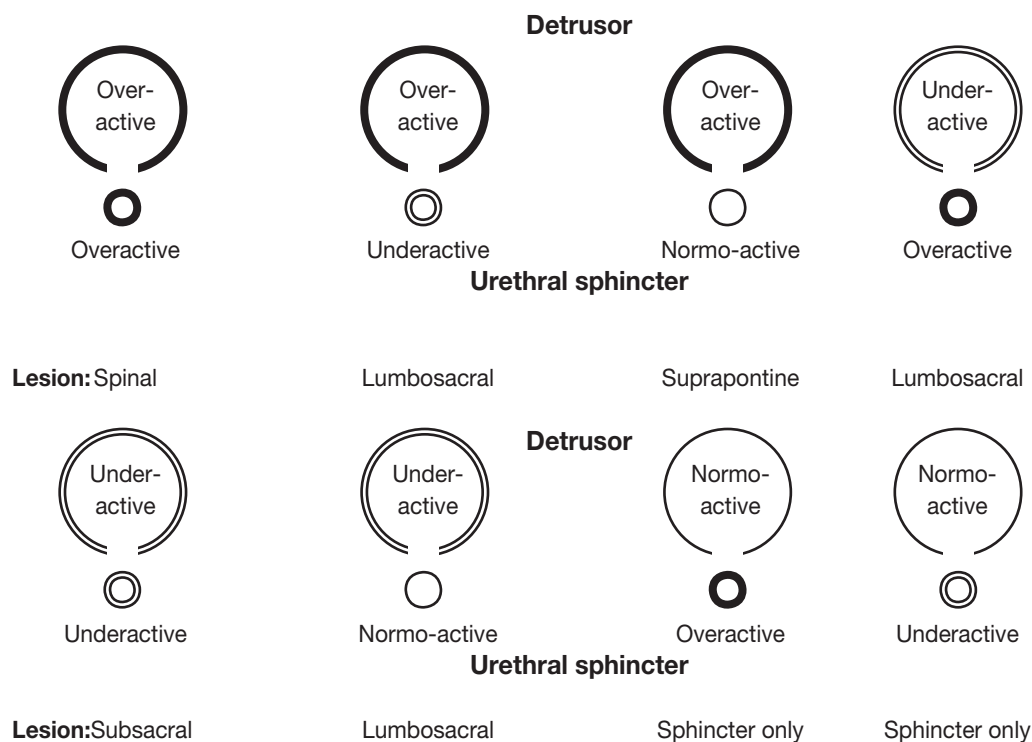


Figure 2.1. Madersbacher classification system (107, 122) with typical neurogenic lesions

2.3.1 Recommendation for clinical practice

The Madersbacher classification system (107, 122) (Figure 2.1) is recommended for clinical practice (Grade of recommendation: B).

2.4 Timing of diagnosis and treatment

Both in congenital and in acquired NLUTD, early diagnosis and treatment is essential as irreversible changes may occur, especially in children with myelomeningocele (123-128), but also in patients with traumatic spinal cord injury (129-131), even if the related neuropathological signs may be normal (132).

It must also be remembered that LUTD by itself may be the presenting symptom for neurological pathology (50, 133).

2.5 REFERENCES

- Andrew J, Nathan PW. Lesions of the anterior frontal lobes and disturbances of micturition and defecation. *Brain* 1964;87:233-62.
<http://www.ncbi.nlm.nih.gov/pubmed/14188274>
- Maurice-Williams RS. Micturition symptoms in frontal tumours. *J Neurol Neurosurg Psychiatry*. 1974;37(4):431-6.
<http://www.ncbi.nlm.nih.gov/pubmed/4365244>
- Lang EW, Chesnut RM, Hennerici M. Urinary retention and space-occupying lesions of the frontal cortex. *Eur Neurol* 1996;36(1):43-7.
<http://www.ncbi.nlm.nih.gov/pubmed/8719650>
- Ueki K. Disturbances of micturition observed in some patients with brain tumor. *Neurol Med Chir* 1960;2:25-33.
- Renier WO, Gabreels FJ. Evaluation of diagnosis and non-surgical therapy in 24 children with a pontine tumour. *Neuropediatrics* 1980;11(3):262-73.
<http://www.ncbi.nlm.nih.gov/pubmed/6252517>
- Toba K, Ouchi Y, Orimo H, Imura O, Sasaki H, Nakamura Y, Takasaki M, Kuzuya F, Sekimoto H, Yoshioka H, Ogiwara T, Kimura I, Ozawa T, Fujishima M. Urinary incontinence in elderly inpatients in Japan: a comparison between general and geriatric hospitals. *Aging (Milano)* 1996;8(1):47-54.
<http://www.ncbi.nlm.nih.gov/pubmed/8695676>

7. Campbell AJ, Reinken J, McCosh L. Incontinence in the elderly: prevalence and prognosis. *Age Ageing* 1985;14(2):65-70.
<http://www.ncbi.nlm.nih.gov/pubmed/4003185>
8. Horimoto Y, Matsumoto M, Akatsu H, Ikari H, Kojima K, Yamamoto T, Otsuka Y, Ojika K, Ueda R, Kosaka K. Autonomic dysfunctions in dementia with Lewy bodies. *J Neurol* 2003;250(5):530-3.
<http://www.ncbi.nlm.nih.gov/pubmed/12736730>
9. Sugiyama T, Hashimoto K, Kiwamoto H, Ohnishi N, Esa A, Park YC, Kurita T. Urinary incontinence in senile dementia of the Alzheimer type (SDAT). *Int J Urol* 1994;1(4):337-40.
<http://www.ncbi.nlm.nih.gov/pubmed/7614397>
10. McGrother C, Resnick M, Yalla SV, Kirschner-Hermanns R, Broseta E, Muller C, Welz-Barth A, Fischer GC, Mattelaer J, McGuire EJ. Epidemiology and etiology of urinary incontinence in the elderly. *World J Urol* 1998;16(Suppl 1):S3-S9.
<http://www.ncbi.nlm.nih.gov/pubmed/9775412>
11. Madersbacher H, Awad S, Fall M, Janknegt RA, Stohrer M, Weisner B. Urge incontinence in the elderly-supraspinal reflex incontinence. *World J Urol* 1998;16 (Suppl 1):S35-S43.
<http://www.ncbi.nlm.nih.gov/pubmed/9775414>
12. Olsen CG, Clasen ME. Senile dementia of the Binswanger's type. *Am Fam Physician* 1998;58(9):2068-74.
<http://www.ncbi.nlm.nih.gov/pubmed/9861880>
13. Honig LS, Mayeux R. Natural history of Alzheimer's disease. *Aging (Milano)* 2001;13:171-82.
14. Burns A, Jacoby R, Levy R. Psychiatric phenomena in Alzheimer's disease. IV: Disorders of behaviour. *Br J Psychiatry* 1990;157:86-94.
<http://www.ncbi.nlm.nih.gov/pubmed/2397368>
15. Cacabelos R, Rodríguez B, Carrera C, Caamaño J, Beyer K, Lao JI, Sellers MA . APOE-related frequency of cognitive and noncognitive symptoms in dementia. *Methods Find Exp Clin Pharmacol* 1996;18(10):693-706.
<http://www.ncbi.nlm.nih.gov/pubmed/9121226>
16. Leung KS, Ng MF, Pang FC, Au SY. Urinary incontinence: an ignored problem in elderly patients. *Hong Kong Med J* 1997;3(1):27-33.
<http://www.ncbi.nlm.nih.gov/pubmed/11847353>
17. Mitchell SJ, Woodthorpe J. Young mentally handicapped adults in three London boroughs: prevalence and degree of disability. *J Epidemiol Community Health* 1981;35(1):59-64.
<http://www.ncbi.nlm.nih.gov/pubmed/7264535>
18. Reid AH, Ballinger BR, Heather BB. Behavioural syndromes identified by cluster analysis in a sample of 100 severely and profoundly retarded adults. *Psychol Med* 1978;8(3):399-412.
<http://www.ncbi.nlm.nih.gov/pubmed/704707>
19. McNeal DM, Hawtrey CE, Wolraich ML, Mapel JR. Symptomatic neurologic bladder in a cerebralpalsied population. *Dev Med Child Neurol* 1983;25(5):612-6.
<http://www.ncbi.nlm.nih.gov/pubmed/6354799>
20. Decter RM, Bauer SB, Khoshbin S, Dyro FM, Krarup C, Colodny AH, Retik AB. Urodynamic assessment of children with cerebral palsy. *J Urol* 1987;138(4 Pt 2):1110-2.
<http://www.ncbi.nlm.nih.gov/pubmed/3656569>
21. Jonas S, Brown J. Neurologic bladder in normal pressure hydrocephalus. *Urology* 1975;5(1):44-50.
<http://www.ncbi.nlm.nih.gov/pubmed/1114545>
22. Black PM. Idiopathic normal-pressure hydrocephalus. Results of shunting in 62 patients. *J Neurosurg* 1980;52(3):371-7.
<http://www.ncbi.nlm.nih.gov/pubmed/7359191>
23. Mulrow CD, Feussner JR, Williams BC, Vokaty KA. The value of clinical findings in the detection of normal pressure hydrocephalus. *J Gerontol* 1987;42(3):277-9.
<http://www.ncbi.nlm.nih.gov/pubmed/3571862>
24. Murnaghan GF. Neurogenic disorders of the bladder in Parkinsonism. *Br J Urol* 1961;33:403-9.
<http://www.ncbi.nlm.nih.gov/pubmed/14477379>
25. Campos-Sousa RN, Quagliato E, da Silva BB, de Carvalho RM Jr, Ribeiro SC, de Carvalho DF. Urinary symptoms in Parkinson's disease: prevalence and associated factors. *Arq Neuropsiquiatr* 2003;61:359-63.
<http://www.ncbi.nlm.nih.gov/pubmed/12894267>
26. Salinas JM, Berger Y, De La Rocha RE, Blaivas JG. Urological evaluation in the Shy Drager syndrome. *J Urol* 1986;135(4):741-3.
<http://www.ncbi.nlm.nih.gov/pubmed/3959195>

27. Chandiramani VA, Palace J, Fowler CJ. How to recognize patients with parkinsonism who should not have urological surgery. *Br J Urol* 1997;80(1):100-4.
<http://www.ncbi.nlm.nih.gov/pubmed/9240187>
28. Hattori T, Yasuda K, Kita K, Hirayama K. Voiding dysfunction in Parkinson's disease. *Jpn J Psychiatry Neurol* 1992;46(1):181-6.
<http://www.ncbi.nlm.nih.gov/pubmed/1635308>
29. Gray R, Stern G, Malone-Lee J. Lower urinary tract dysfunction in Parkinson's disease: changes relate to age and not disease. *Age Ageing* 1995;24(6):499-504.
<http://www.ncbi.nlm.nih.gov/pubmed/8588540>
30. Araki I, Kuno S. Assessment of voiding dysfunction in Parkinson's disease by the international prostate symptom score. *J Neurol Neurosurg Psychiatry* 2000;68(4):429-33.
<http://www.ncbi.nlm.nih.gov/pubmed/10727477>
31. Lemack GE, Dewey RB, Roehrborn CG, O'Suilleabhain PE, Zimmern PE. Questionnaire-based assessment of bladder dysfunction in patients with mild to moderate Parkinson's disease. *Urology* 2000; 56(2):250-4.
<http://www.ncbi.nlm.nih.gov/pubmed/10925088>
32. Sakakibara R, Shinotoh H, Uchiyama T, Sakuma M, Kashiwado M, Yoshiyama M, Hattori T. Questionnaire-based assessment of pelvic organ dysfunction in Parkinson's disease. *Auton Neurosci* 2001;92(1-2):76-85.
<http://www.ncbi.nlm.nih.gov/pubmed/11570707>
33. Currie CT. Urinary incontinence after stroke. *BMJ (Clin Res Ed)* 1986;293(6558):1322-3.
<http://www.ncbi.nlm.nih.gov/pubmed/3790967>
34. Codine PH, Pellissier J, Manderscheidt JC, Costa P, Enjalbert M, Perrigot M. Les troubles urinaires au cours des hémiplegies vasculaires. In: *Hémiplegie vasculaire et médecine de rééducation*. Pellissier J, ed. Paris, Masson, 1988, pp. 261-269.
35. Barer DH. Continence after stroke: useful predictor or goal of therapy? *Age Ageing* 1989;18(3):183-91.
<http://www.ncbi.nlm.nih.gov/pubmed/2782216>
36. Sakakibara R, Hattori T, Yasuda K, Yamanishi T. Micturitional disturbance after acute hemispheric stroke: analysis of the lesion site by CT and MRI. *J Neurol Sci* 1996;137(1):47-56.
<http://www.ncbi.nlm.nih.gov/pubmed/9120487>
37. Nakayama H, Jørgensen HS, Pedersen PM, Raaschou HO, Olsen TS. Prevalence and risk factors of incontinence after stroke. The Copenhagen Stroke Study. 1997;28(1):58-62.
<http://www.ncbi.nlm.nih.gov/pubmed/8996489>
38. Khan Z, Hertanu J, Yang WC, Melman A, Leiter E. Predictive correlation of urodynamic dysfunction and brain injury after cerebrovascular accident. *J Urol* 1981;126(1):86-8.
<http://www.ncbi.nlm.nih.gov/pubmed/7253085>
39. Tsuchida S, Noto H, Yamaguchi O, Itoh M. Urodynamic studies on hemiplegic patients after cerebrovascular accident. *Urology* 1983;21(3):315-8.
<http://www.ncbi.nlm.nih.gov/pubmed/6836813>
40. Kuroiwa Y, Tohgi H, Ono S, Itoh M. Frequency and urgency of micturition in hemiplegic patients; relationship to hemisphere laterality of lesions. *J Neurol* 1987;234(2):100-2.
<http://www.ncbi.nlm.nih.gov/pubmed/3559632>
41. Khan Z, Starer P, Yang WC, Bhole A. Analysis of voiding disorders in patients with cerebrovascular accidents. *Urology* 1990;35(3):265-70.
<http://www.ncbi.nlm.nih.gov/pubmed/3559632>
42. Taub NA, Wolfe CD, Richardson E, Burney PG. Predicting the disability of first-time stroke sufferers at 1 year. 12-month follow-up of a population-based cohort in southeast England. *Stroke* 1994;25(2):352-7.
<http://www.ncbi.nlm.nih.gov/pubmed/8303744>
43. Borrie MJ, Campbell AJ, Caradoc-Davies TH, Spears GF. Urinary incontinence after stroke: a prospective study. *Age Ageing* 1986;15(3):177-81.
<http://www.ncbi.nlm.nih.gov/pubmed/3739856>
44. Sakakibara R, Hattori T, Yasuda K, Yamanishi T. Micturitional disturbance and the pontine tegmental lesion: urodynamic and MRI analyses of vascular cases. *J Neurol Sci* 1996;141(1-2):105-10.
<http://www.ncbi.nlm.nih.gov/pubmed/8880701>
45. Betts CD, Kapoor R, Fowler CJ. Pontine pathology and voiding dysfunction. *Br J Urol* 1992;70(1): 100-2.
<http://www.ncbi.nlm.nih.gov/pubmed/1638364>

46. Manente G, Melchionda D, Uncini A. Urinary retention in bilateral pontine tumour: evidence for a pontine micturition centre in humans. *J Neurol Neurosurg Psychiatry* 1996;61(5):528-9.
<http://www.ncbi.nlm.nih.gov/pubmed/8937354>
47. Litwiller SE, Frohman EM, Zimmern PE. Multiple sclerosis and the urologist. *J Urol*;1999;161(3):743-57.
<http://www.ncbi.nlm.nih.gov/pubmed/10022678>
48. Giannantoni A, Scivoletto G, Di Stasi SM, Grasso MG, Finazzi Agrò E, Collura G, Vespasiani G. LUT dysfunction and disability status in patients with multiple sclerosis. *Arch Phys Med Rehabil* 1999;80(4):437-41.
<http://www.ncbi.nlm.nih.gov/pubmed/10206607>
49. Hinson JL, Boone TB. Urodynamics and multiple sclerosis. *Urol Clin North Am* 1996;23(3):475-81.
<http://www.ncbi.nlm.nih.gov/pubmed/8701560>
50. Bemelmans BL, Hommes OR, Van Kerrebroeck PE, Lemmens WA, Doesburg WH, Debruyne FM. Evidence for early lower urinary tract dysfunction in clinically silent multiple sclerosis. *J Urol* 1991;145(6):1219-24.
<http://www.ncbi.nlm.nih.gov/pubmed/2033697>
51. DasGupta R, Fowler CJ. Sexual and urological dysfunction in multiple sclerosis: better understanding and improved therapies. *Curr Opin Neurol* 2002;15(3):271-8.
<http://www.ncbi.nlm.nih.gov/pubmed/12045724>
52. Perrigot M, Richard F, Veaux-Renault V, Chatelain C, Kuss R. [Bladder sphincter disorders in multiple sclerosis: symptomatology and evolution. 100 cases.] *Sem Hop* 1982;58(45):2543-6. [article in French]
<http://www.ncbi.nlm.nih.gov/pubmed/6297048>
53. Burns AS, Rivas DA, Ditunno JF. The management of neurogenic bladder and sexual dysfunction after spinal cord injury. *Spine* 2001;26(24):S129-S136.
<http://www.ncbi.nlm.nih.gov/pubmed/11805620>
54. Lawrenson R, Wyndaele JJ, Vlachonikolis I, Farmer C, Glickman S. A UK general practice database study of prevalence and mortality of people with neural tube defects. *Clin Rehabil* 2000;14(6):627-30.
<http://www.ncbi.nlm.nih.gov/pubmed/11128738>
55. Selzman AA, Elder JS, Mapstone TB. Urologic consequences of myelodysplasia and other congenital abnormalities of the spinal cord. *Urol Clin North Am* 1993;20(3):485-504.
<http://www.ncbi.nlm.nih.gov/pubmed/8351774>
56. Smith E. *Spina bifida and the total care of spinal myelomeningocele*. Springfield, IL: CC Thomas, ed, 1965; pp. 92-123.
57. van Gool JD, Dik P, de Jong TP. Bladder-sphincter dysfunction in myelomeningocele. *Eur J Pediatr* 2001;160(7):414-20.
<http://www.ncbi.nlm.nih.gov/pubmed/11475578>
58. Wyndaele JJ, De Sy W. Correlation between the findings of a clinical neurological examination and the urodynamic dysfunction in children with myelodysplasia. *J Urol* 1985;133(4):638-40.
<http://www.ncbi.nlm.nih.gov/pubmed/3981715>
59. Bartolin Z, Gilja I, Bedalov G, Savic I. Bladder function in patients with lumbar intervertebral disc protrusion. *J Urol* 1998;159(3):969-71.
<http://www.ncbi.nlm.nih.gov/pubmed/9474195>
60. O'Flynn KJ, Murphy R, Thomas DG. Neurologic bladder dysfunction in lumbar intervertebral disc prolapse. *Br J Urol* 1992;69(1):38-40.
<http://www.ncbi.nlm.nih.gov/pubmed/1737251>
61. Jennett WB. A study of 25 cases of compression of the cauda equina by prolapsed intervertebral discs. *J Neurol Neurosurg Psychiatry* 1956;19(2):109-16.
<http://www.ncbi.nlm.nih.gov/pubmed/13346384>
62. Tay ECK, Chacha PB. Midline prolapse of a lumbar intervertebral disc with compression of the cauda equina. *J Bone Joint Surg Br* 1979;61(1):43-6.
<http://www.ncbi.nlm.nih.gov/pubmed/154521>
63. Nielsen B, de Nully M, Schmidt K, Hansen RI. A urodynamic study of cauda equina syndrome due to lumbar disc herniation. *Urol Int* 1980;35(3):167-70.
<http://www.ncbi.nlm.nih.gov/pubmed/7385464>
64. Bartels RH, de Vries J. Hemi-cauda equina syndrome from herniated lumbar disc: a neurosurgical emergency? *Can J Neurol Sci* 1996;23(4):296-9.
<http://www.ncbi.nlm.nih.gov/pubmed/8951209>
65. Goldman HB, Appell RA. Voiding dysfunction in women with lumbar disc prolapse. *Int Urogynecol J Pelvic Floor Dysfunct* 1999;10(2):134-8.
<http://www.ncbi.nlm.nih.gov/pubmed/10384977>

66. Ahn UM, Ahn NU, Buchowski JM, Garrett ES, Sieber AN, Kostuik JP. Cauda equina syndrome secondary to lumbar disc herniation: a meta-analysis of surgical outcomes. *Spine* 2000;25(12): 1515-22.
<http://www.ncbi.nlm.nih.gov/pubmed/10851100>
67. Shapiro S. Medical realities of cauda equina syndrome secondary to lumbar disc herniation. *Spine* 2000;25(3):348-51.
<http://www.ncbi.nlm.nih.gov/pubmed/10703108>
68. Emmett JL, Love JG. Urinary retention in women caused by asymptomatic protruded lumbar disc: report of 5 cases. *J Urol* 1968;99:597-606.
69. Rosomoff HL, Johnston JD, Gallo AE, Ludmer M, Givens FT, Carney FT, Kuehn CA. Cystometry in the evaluation of nerve root compression in the lumbar spine. *Surg Gynecol Obstet* 1963;117:263-70.
<http://www.ncbi.nlm.nih.gov/pubmed/14080336>
70. Kawaguchi Y, Kanamori M, Ishihara H, Ohmori K, Fujiuchi Y, Matsui H, Kimura T. Clinical symptoms and surgical outcome in lumbar spinal stenosis patients with neurologic bladder. *J Spinal Disord* 2001;14:404-10.
<http://www.ncbi.nlm.nih.gov/pubmed/11586140>
71. Tammela TL, Heiskari MJ, Lukkarinen OA. Voiding dysfunction and urodynamic findings in patients with cervical spondylotic spinal stenosis compared with severity of the disease. *Br J Urol*. 1992;70(2):144-8.
<http://www.ncbi.nlm.nih.gov/pubmed/1393436>
72. Inui Y, Doita M, Ouchi K, Tsukuda M, Fujita N, Kurosaka M. Clinical and radiological features of lumbar spinal stenosis and disc herniation with neurologic bladder. *Spine* 2004;29(8):869-73.
<http://www.ncbi.nlm.nih.gov/pubmed/15082986>
73. Boulis NM, Mian FS, Rodriguez D, Cho E, Hoff JT. Urinary retention following routine neurosurgical spine procedures. *Surg Neurol* 2001;55(1):23-7.
<http://www.ncbi.nlm.nih.gov/pubmed/11248301>
74. Brooks ME, Moreno M, Sidi A, Braf ZF. Urologic complications after surgery on lumbosacral spine. *Urology* 1985;26:202-4.
<http://www.ncbi.nlm.nih.gov/pubmed/4024418>
75. Ellenberg M. Development of urinary bladder dysfunction in diabetes mellitus. *Ann Intern Med* 1980;92(2 Pt 2):321-3.
<http://www.ncbi.nlm.nih.gov/pubmed/7356222>
76. Frimodt-Moller C. Diabetic cystopathy: epidemiology and related disorders. *Ann Intern Med* 1980; 92:318-21.
<http://www.ncbi.nlm.nih.gov/pubmed/7356221>
77. Bradley WE. Diagnosis of urinary bladder dysfunction in diabetes mellitus. *Ann Intern Med* 1980;92(2 Pt 2):323-6.
<http://www.ncbi.nlm.nih.gov/pubmed/7188844>
78. Barter F, Tanner AR. Autonomic neuropathy in an alcoholic population. *Postgrad Med J*. 1987;63(746): 1033-6.
<http://www.ncbi.nlm.nih.gov/pubmed/3451229>
79. Anonymous. Autonomic neuropathy in liver disease. *Lancet* 1989;2(8665):721-2.
<http://www.ncbi.nlm.nih.gov/pubmed/2570966>
80. Bloomer JR, Bonkovsky HL. The porphyrias. *Dis Mon* 1989;35(1):1-54.
<http://www.ncbi.nlm.nih.gov/pubmed/2645098>
81. Chapelon C, Ziza JM, Piette JC, Levy Y, Raguin G, Wechsler B, Bitker MO, Blety O, Laplane D, Bousser MG, et al. Neurosarcoidosis: signs, course and treatment in 35 confirmed cases. *Medicine (Baltimore)*; 1990;69(5):261-76.
<http://www.ncbi.nlm.nih.gov/pubmed/2205782>
82. Chen PH, Hsueh HF, Hong CZ. Herpes zoster-associated voiding dysfunction: a retrospective study and literature review. *Arch Phys Med Rehabil* 2002;83(11):1624-8.
<http://www.ncbi.nlm.nih.gov/pubmed/12422336>
83. Greenstein A, Matzkin H, Kaver I, Braf Z. Acute urinary retention in herpes genitalis infection. Urodynamic evaluation. *Urology* 1988;31(5):453-6.
<http://www.ncbi.nlm.nih.gov/pubmed/3363783>
84. Grbavac Z, Gilja I, Gubarev N, Bozicevic D. [Neurologic and urodynamic characteristics of patients with Guillain-Barré syndrome]. *Lijec Vjesn* 1989;111(1-2):17-20. [article in Croatian]
<http://www.ncbi.nlm.nih.gov/pubmed/2739495>

85. Sakakibara R, Hattori T, Kuwabara S, Yamanishi T, Yasuda K. Micturitional disturbance in patients with Guillain-Barré syndrome. *J Neurol Neurosurg Psychiatry* 1997;63(5):649-53.
<http://www.ncbi.nlm.nih.gov/pubmed/9408108>
86. Lichtenfeld P. Autonomic dysfunction in the Guillain-Barré syndrome. *Am J Med* 1971;50(6):772-80.
<http://www.ncbi.nlm.nih.gov/pubmed/5089852>
87. Sakakibara R, Uchiyama T, Yoshiyama M, Yamanishi T, Hattori T. Urinary dysfunction in patients with systemic lupus erythematosus. *Neurourol Urodyn* 2003;22(6):593-6.
<http://www.ncbi.nlm.nih.gov/pubmed/12951670>
88. Min JK, Byun JY, Lee SH, Hong YS, Park SH, Cho CS, Kim HY. Urinary bladder involvement in patients with systemic lupus erythematosus: with review of the literature. *Korean J Intern Med* 2000;15(1):42-50.
<http://www.ncbi.nlm.nih.gov/pubmed/10714091>
89. Gyrtup HJ, Kristiansen VB, Zachariae CO, Krosgaard K, Colstrup H, Jensen KM. Voiding problems in patients with HIV infection and AIDS. *Scand J Urol Nephrol* 1995;29(3):295-8.
<http://www.ncbi.nlm.nih.gov/pubmed/8578272>
90. Khan Z, Singh VK, Yang WC. Neurologic bladder in acquired immune deficiency syndrome (AIDS). *Urology* 1992;40(3):289-91.
<http://www.ncbi.nlm.nih.gov/pubmed/1523760>
91. Mardirosoff C, Dumont L. Bowel and bladder dysfunction after spinal bupivacaine. *Anesthesiology* 2001; 95(5):1306.
<http://www.ncbi.nlm.nih.gov/pubmed/11685017>
92. Auroy Y, Benhamou D, Barges L, Ecoffey C, Falissard B, Mercier FJ, Bouaziz H, Samii K. Major complications of regional anesthesia in France: The SOS Regional Anesthesia Hotline Service. *Anesthesiology* 2002;97(5):1274-80.
<http://www.ncbi.nlm.nih.gov/pubmed/12411815>
93. Hollabaugh RS Jr, Steiner MS, Sellers KD, Sann BJ, Dmochowski RR. Neuroanatomy of the pelvis: implications for colonic and rectal resection. *Dis Colon Rectum* 2000;43(10):1390-7.
<http://www.ncbi.nlm.nih.gov/pubmed/11052516>
94. Baumgarner GT, Miller HC. Genitourinary complications of abdominoperineal resection. *South Med J* 1976;69(7):875-7.
<http://www.ncbi.nlm.nih.gov/pubmed/941055>
95. Eickenberg HU, Amin M, Klompus W, Lich R Jr. Urologic complications following abdominoperineal resection. *J Urol* 1976;115(2):180-2.
<http://www.ncbi.nlm.nih.gov/pubmed/1249871>
96. Pocard M, Zinzindohoue F, Haab F, Caplin S, Parc R, Turet E. A prospective study of sexual and urinary function before and after total mesorectal excision with autonomic nerve preservation for rectal cancer. *Surgery* 2002;131:368-72.
<http://www.ncbi.nlm.nih.gov/pubmed/11935125>
97. Kim NK, Aahn TW, Park JK, Lee KY, Lee WH, Sohn SK, Min JS. Assessment of sexual and voiding function after total mesorectal excision with pelvic autonomic nerve preservation in males with rectal cancer. *Dis Colon Rectum* 2002;45(9):1178-85.
<http://www.ncbi.nlm.nih.gov/pubmed/12352233>
98. Parys BT, Woolfenden KA, Parsons KF. Bladder dysfunction after simple hysterectomy: urodynamic and neurological evaluation. *Eur Urol* 1990;17(2):129-33.
<http://www.ncbi.nlm.nih.gov/pubmed/2311638>
99. Sekido N, Kawai K, Akaza H. Lower urinary tract dysfunction as persistent complication of radical hysterectomy. *Int J Urol* 1997;4(3):259-64.
<http://www.ncbi.nlm.nih.gov/pubmed/9255663>
100. Zanolla R, Monzeglio C, Campo B, Ordesi G, Balzarini A, Martino G. Bladder and urethral dysfunction after radical abdominal hysterectomy: rehabilitative treatment. *J Surg Oncol* 1985;28(3):190-4.
<http://www.ncbi.nlm.nih.gov/pubmed/3974245>
101. Seski JC, Diokno AC. Bladder dysfunction after radical abdominal hysterectomy. *Am J Obstet Gynecol* 1977;128(6):643-51.
<http://www.ncbi.nlm.nih.gov/pubmed/18009>
102. Lin HH, Sheu BC, Lo MC, Huand SC. Abnormal urodynamic findings after radical hysterectomy or pelvic irradiation for cervical cancer. *Int J Gynaecol Obstet* 1998;63(2):169-74.
<http://www.ncbi.nlm.nih.gov/pubmed/9856324>
103. Kuwabara Y, Suzuki M, Hashimoto M, Furugen Y, Yoshida K, Mitsunashi N. New method to prevent bladder dysfunction after radical hysterectomy for uterine cervical cancer. *J Obstet Gynaecol Res* 2000;26(1):1-8.
<http://www.ncbi.nlm.nih.gov/pubmed/10761323>

104. Zermann DH, Ishigooka M, Wunderlich H, Reichelt O, Schubert J. A study of pelvic floor function pre and postradical prostatectomy using clinical neurourological investigations, urodynamics and electromyography. *Eur Urol* 2000;37(1):72-8.
<http://www.ncbi.nlm.nih.gov/pubmed/10671789>
105. Burgdörfer H, Heidler H, Madersbacher H, Melchior H, Palmtag H, Richter R, Richter-Reichhelm M, Rist M, Rübben H, Sauerwein D, Schalkhäuser K, Stöhrer M. Leitlinien zur urologischen Betreuung Querschnittgelähmter. *Urologe A* 1998;37:222-8. [article in German] [Guidelines for the urological management of paraplegic patients].
106. Stöhrer M, Goepel M, Kondo A, Kramer G, Madersbacher H, Millard R, Rossier A, Wyndaele JJ. The standardization of terminology in neurogenic lower urinary tract dysfunction with suggestions for diagnostic procedures, *NeuroUrol Urodyn* 1999;18(2):139-58.
<http://www.ncbi.nlm.nih.gov/pubmed/10081953>
107. Wyndaele JJ, Castro D, Madersbacher H, Chartier-Kastler E, Igawa Y, Kovindha A, Radziszewski P, Stone A, Wiesel P. Neurologic urinary and faecal incontinence. In: Abrams P, Cardozo L, Khoury S, Wein A, eds. *Incontinence*. Plymouth: Health Publications, 2005:1061-2.
108. Consortium for Spinal Cord Medicine. Bladder management for adults with spinal cord injury: a clinical practice guideline for health-care providers. *J Spinal Cord Med* 2006;29(5):527-3.
<http://www.ncbi.nlm.nih.gov/pubmed/17274492>
109. Abrams P, Cardozo L, Fall M, Griffiths D, Rosier P, Ulmsten U, van Kerrebroeck P, Victor A, Wein A. The standardisation of terminology of lower urinary tract function: Report from the Standardisation Subcommittee of the International Continence Society. *NeuroUrol Urodyn* 2002;21(2):167-78.
<http://www.ncbi.nlm.nih.gov/pubmed/11857671>
110. Klevmark B. Natural pressure-volume curves and conventional cystometry. *Scand J Urol Nephrol Suppl* 1999;201:1-4.
<http://www.ncbi.nlm.nih.gov/pubmed/10573769>
111. Homma Y, Ando T, Yoshida M, Kageyama S, Takei M, Kimoto K, Ishizuka O, Gotoh M, Hashimoto T. Voiding and incontinence frequencies: variability of diary data and required diary length. *NeuroUrol Urodyn* 2002;21(3):204-9.
<http://www.ncbi.nlm.nih.gov/pubmed/10573769>
112. McGuire EJ, Cespedes RD, O'Connell HE. Leak-point pressures. *Urol Clin North Am* 1996;23(2):253-62.
<http://www.ncbi.nlm.nih.gov/pubmed/8659025>
113. Bradley WE, Timm GW, Scott FB. Innervation of the detrusor muscle and urethra. *Urol Clin North Am* 1974;1(1):3-27.
<http://www.ncbi.nlm.nih.gov/pubmed/4372763>
114. Bors E, Comarr AE. *Neurological urology*. Basel, Karger, 1971:75;144-5.
115. Hald T, Bradley WE. *The neurogenic bladder*. Baltimore, Williams and Wilkins, 1982.
116. Stöhrer M, Kramer G, Löchner-Ernst D, Goepel M, Noll F, Rübben H. Diagnosis and treatment of bladder dysfunction in spinal cord injury patients. *Eur Urol Update Series* 1994;3:170-5.
117. Lapedes J. Neuromuscular vesical and urethral dysfunction. In: Campbell MF, Harrison JH, eds. *Urology*. 3rd edn. Philadelphia, WB Saunders, 1970, pp. 1343-1379.
118. Krane RJ, Siroky MB. Classification of neuro-urologic disorders. In: Krane RJ, Siroky MB, eds. *Clinical neuro-urology*. Boston: Little Brown, 1979: 143-58.
119. Quesada EM, Scott FB, Cardus D. Functional classification of neurogenic bladder dysfunction. *Arch Phys Med Rehabil* 1968;49(12):692-7.
<http://www.ncbi.nlm.nih.gov/pubmed/5703244>
120. Wein AJ. Pathophysiology and categorization of voiding dysfunction. In: Walsh PC, Retik AB, Vaughan Jr ED, Wein AJ, eds. *Campbell's urology*. 7th edn. Philadelphia: WB Saunders, 1998, pp. 917-926.
121. Fall M, Ohlsson BL, Carlsson CA. The neurogenic overactive bladder. Classification based on urodynamics. *Br J Urol* 1989;64(4):368-73.
<http://www.ncbi.nlm.nih.gov/pubmed/2819387>
122. Madersbacher H. The various types of neurogenic bladder dysfunction: an update of current therapeutic concepts. *Paraplegia* 1990;28(4):217-29.
<http://www.ncbi.nlm.nih.gov/pubmed/2235029>
123. Cass AS, Luxenberg M, Johnson CF, Gleich P. Incidence of urinary tract complications with myelomeningocele. *Urology* 1985;25(4):374-8.
<http://www.ncbi.nlm.nih.gov/pubmed/3984125>
124. Fernandes ET, Reinberg Y, Vernier R, Gonzalez R. Neurogenic bladder dysfunction in children: review of pathophysiology and current management. *J Pediatr* 1994;124(1):1-7.
<http://www.ncbi.nlm.nih.gov/pubmed/8283355>

125. Stone AR. Neurourologic evaluation and urologic management of spinal dysraphism. *Neurosurg Clin N Am* 1995;6(2):269-77.
<http://www.ncbi.nlm.nih.gov/pubmed/7620353>
126. Satar N, Bauer SB, Shefner J, Kelly MD, Darbey MM. The effects of delayed diagnosis and treatment in patients with an occult spinal dysraphism. *J Urol* 1995;154(2 Pt 2):754-8.
<http://www.ncbi.nlm.nih.gov/pubmed/7609171>
127. Pontari MA, Keating M, Kelly M, Dyro F, Bauer SB. Retained sacral function in children with high level myelodysplasia. *J Urol* 1995;154(2 Pt 2):775-7.
<http://www.ncbi.nlm.nih.gov/pubmed/7609177>
128. Kaefer M, Pabby A, Kelly M, Darbey M, Bauer SB. Improved bladder function after prophylactic treatment of the high risk neurogenic bladder in newborns with myelomeningocele. *J Urol* 1999;162(3 Pt 2):1068-71.
<http://www.ncbi.nlm.nih.gov/pubmed/10458433>
129. Wyndaele JJ. Development and evaluation of the management of the neuropathic bladder. *Paraplegia* 1995;33(6):305-7.
<http://www.ncbi.nlm.nih.gov/pubmed/7644254>
130. Cardenas DD, Mayo ME, Turner LR. Lower urinary changes over time in suprasacral spinal cord injury. *Paraplegia* 1995;33(6):326-9.
<http://www.ncbi.nlm.nih.gov/pubmed/7644258>
131. Amarenco G. [Vesico-sphincter disorders of nervous origin.] *Rev Prat* 1995;45(3):331-5. [article in French]
<http://www.ncbi.nlm.nih.gov/pubmed/7725038>
132. Watanabe T, Vaccaro AR, Kumon H, Welch WC, Rivas DA, Chancellor MB. High incidence of occult neurogenic bladder dysfunction in neurologically intact patients with thoracolumbar spinal injuries. *J Urol* 1998;159(3):965-8.
<http://www.ncbi.nlm.nih.gov/pubmed/9474194>
133. Ahlberg J, Edlund C, Wikkelso C, Rosengren L, Fall M. Neurological signs are common in patients with urodynamically verified 'idiopathic' bladder overactivity. *Neurorol Urodyn* 2002;21(1):65-70.
<http://www.ncbi.nlm.nih.gov/pubmed/11835426>

3. DIAGNOSIS

3.1 Introduction

Before any functional investigation is planned, an extensive general and specific diagnosis should be performed. Part of this diagnosis is specific for neurogenic pathology and its possible sequelae. The clinical assessment of patients with NLUTD includes and extends that for other LUTD. The latter should consist of a detailed history, bladder diary and a physical examination. In urinary incontinence, leakage should be demonstrated objectively.

These data are indispensable for reliable interpretation of the findings in diagnostic investigations performed subsequently in NLUTD.

3.2 History

3.2.1 General history

The general history should include relevant questions about neurological and congenital abnormalities, social factors and the patient's motivation, any previous occurrences and frequency of urinary infections, and relevant surgery. Information must be obtained on medication with known or possible effects on the lower urinary tract (1-3). The general history should also include the assessment of menstrual, sexual and bowel function, and obstetric history (3).

Hereditary or familial risk factors should be recorded. Symptoms of any metabolic disorder or neurological disease that may induce NLUTD must be checked particularly. Specific signs, such as pain, infection, haematuria, fever, etc, may justify further particular diagnosis.

Items of particular importance include:

- Congenital anomalies with possible neurological impact
- Metabolic disorders with possible neurological impact
- Preceding therapy, including surgical interventions
- Present medication
- Lifestyle factors, such as smoking, alcohol, or addictive drug use

- Infections of the urinary tract
- Quality of life
- Life expectancy.

3.2.2 Specific history

Urinary history: This consists of symptoms related to both the storage and the evacuation functions of the lower urinary tract. The onset and the nature of the NLUTD (acute or insidious) should be determined. Specific symptoms and signs must be assessed in NLUTD and if appropriate should be compared with the patient's condition before the NLUTD developed. The separate diagnostic field items should be diagnosed in as much detail as possible (3):

- LUTS
- Voiding pattern
- Urinary incontinence
- Bladder sensation
- Mode and type of voiding (catheterization).

The urinary (bladder) diary gives (semi-)objective information about the number of voidings, daytime and night-time voiding frequency, volumes voided, and incontinence and urge episodes.

Bowel history: Patients with NLUTD may suffer from a related neurogenic condition of the lower gastrointestinal tract. The bowel history must also address symptoms related to the storage and evacuation functions. Specific symptoms and signs must be compared with the patient's condition before the neurogenic dysfunction developed. Again, the diagnostic items should be detailed (3):

- Ano-rectal symptoms
- Defecation pattern
- Fecal incontinence
- Rectal sensation
- Mode and type of defecation.

Sexual history: Sexual function may also be impaired because of the neurogenic condition. The details of the history will differ of course between men and women (3):

- Genital or sexual dysfunction symptoms
- Sexual function
- Sensation in genital area and for sexual functions
- Erection or arousal
- Orgasm
- Ejaculation.

Neurological history: This should concentrate on the following information:

- Acquired or congenital neurological condition
- Neurological symptoms (somatic and sensory), with onset, evolution, and performed therapy
- Spasticity or autonomic dysreflexia (lesion level above Th6).

Individuals with spinal cord injury (SCI) are often not accurate at knowing whether they have had a urinary tract infection based on their symptoms (4).

3.2.3 GUIDELINES FOR HISTORY TAKING

1. An extensive general history is mandatory, concentrating on past and present symptoms and conditions for urinary, bowel, sexual, and neurological functions, and on general conditions that might impair any of these.
2. Special attention should be paid to the possible existence of alarm signs, such as pain, infection, haematuria, fever, etc, that warrant further specific diagnosis.
3. A specific history should be taken for each of the four mentioned functions.

3.3 Physical examination

3.3.1 General physical examination

Attention should be paid to the patient's physical and possible mental handicaps. Problems may be caused by impaired mobility, particularly in the hips, or extreme spasticity.

Patients with very high neurological lesions may suffer from a significant drop in blood pressure when moved in a sitting or standing position. Subjective indications of bladder filling sensations may be impossible in

mentally impaired patients.

Inspection of the abdominal wall, prostate palpation or observation of pelvic organ prolapse is mandatory.

3.3.2 Neuro-urological examination

General neurological examination: This investigates the motor and sensory functions of the body, the limbs and hand functions (Figure 3.1). A suprapubic globe should be looked for and the skin condition in the genital and perineal regions should be assessed.

Figure 3.1. Dermatomes of spinal cord levels L2-S4

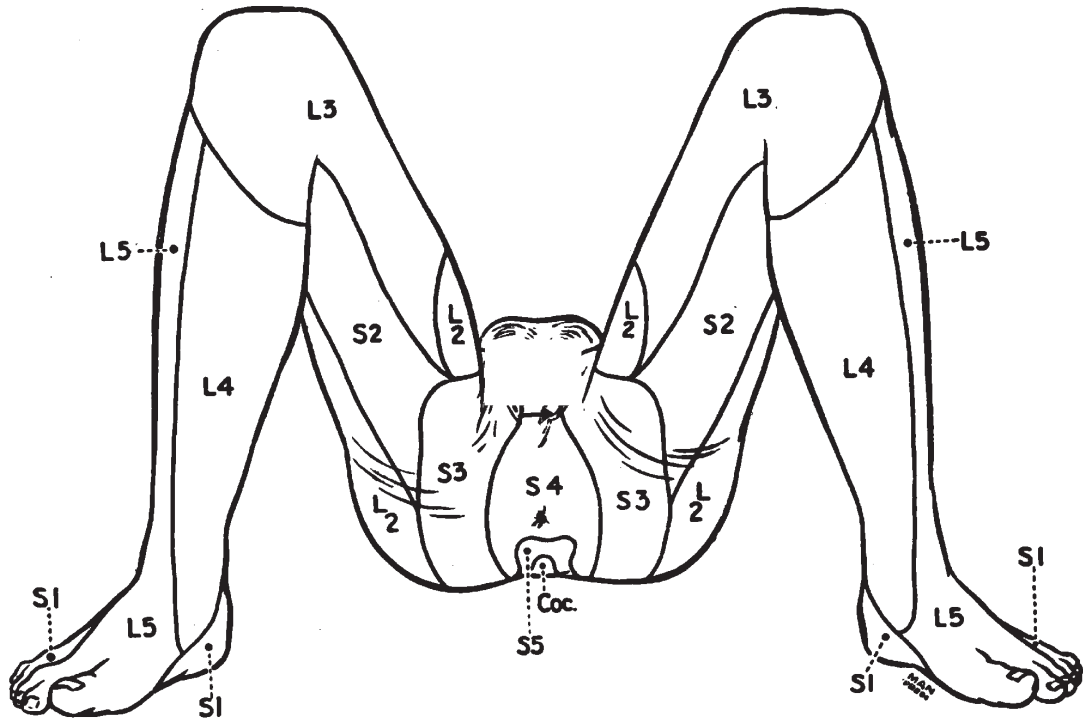


Figure 3.2. Urogenital and other reflexes in lower spinal cord

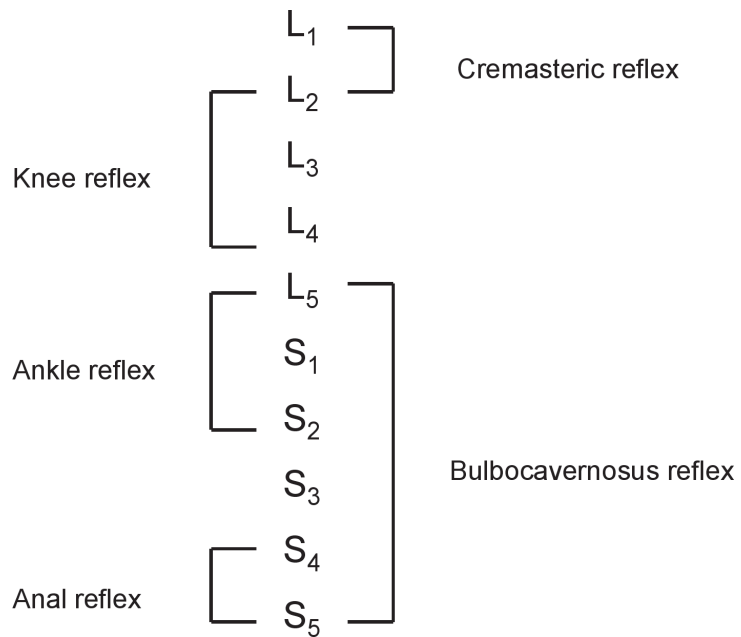


Figure 3.3. Innervation overview of bladder and bowel. Afferent and efferent nerves in the different peripheral nerves and central nervous system are shown

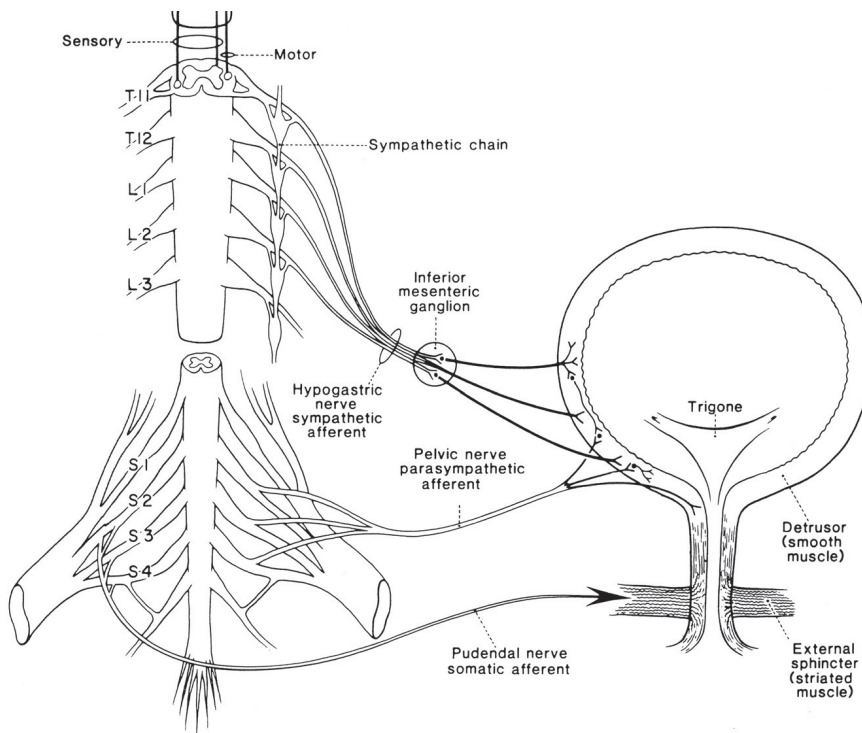
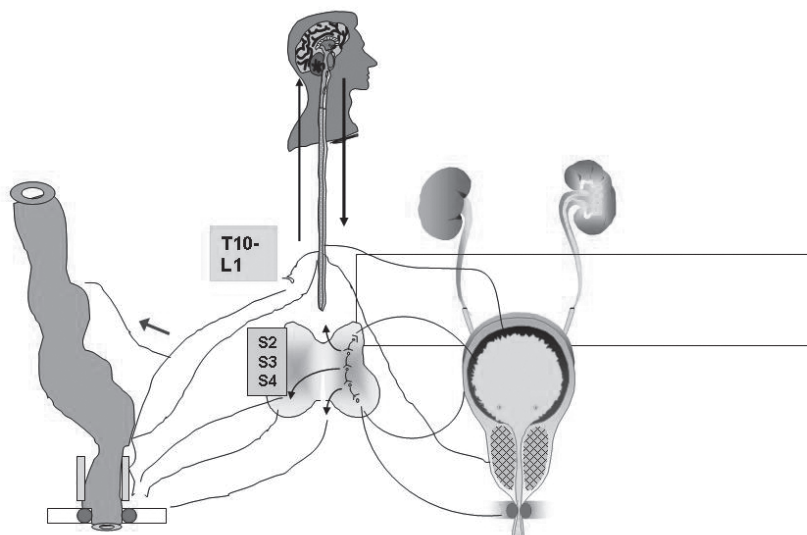


Figure 3.4. Overview of peripheral nerves innervating the lower urinary tract

Innervation lower urinary and bowel tract



Specific neuro-urological examination: This investigation is necessary in patients with NLUTD. It includes several tests for sacral reflex activity and an evaluation of the sensation in the perineal area. Figure 3.1 shows the different dermatomes and Figure 3.2 the associated reflexes in this area.

Specified information should become available on:

- Sensation S₂-S₅ on both sides of the body
- Reflexes
- Anal sphincter tone
- Volitional contraction of anal sphincter and pelvic floor.

A high correlation exists between the clinical neurological findings and NLUTD in some types of neuropathy, but less so in other types (5-10). The correspondence is low, for instance, in myelomeningocele patients (7)

and in combined traumatic spinal cord lesions, but high in single-level traumatic spinal cord lesions (10). In SCI patients with thoracolumbar fractures, neurogenic voiding dysfunction cannot be predicted by the sensory evaluation (11). In SCI patients with thoracolumbar fractures, the presence of a voluntary contraction of the plantar flexors of the toes correlates with active contraction of the external anal/urethral sphincter, but does not enable differentiation of the types of neuropathic voiding disorders (12). A congenital association of lower urinary tract and bowel dysfunction with an abnormal facial expression is named the urofacial (Ochoa) syndrome. (13).

Individuals with SCI were frequently not accurate at predicting whether they had a UTI based on their symptoms (4).

3.3.3 Essential investigations

Essential investigations include (3):

- Urinalysis
- Blood chemistry
- Voiding diary
- Assessment of residual urine, if possible with free flowmetry. Because of natural variations, several assessments (at least 2-3) are necessary (3, 10, 11).
- Quantification of urine loss by pad testing if appropriate
- Urinary tract imaging studies.

3.3.4 GUIDELINES FOR PHYSICAL EXAMINATION

1. Individual patient handicaps should be acknowledged in planning further investigations.
2. The neurological status should be described as completely as possible. Sensations and reflexes in the urogenital area must all be tested.
3. The anal sphincter and pelvic floor functions must be tested extensively.
4. Urinalysis, blood chemistry, voiding diary, residual and free flowmetry, incontinence quantification and urinary tract imaging should be performed.

3.4 Urodynamics

3.4.1 Introduction

Urodynamic investigation is the only method that can objectively assess the (dys-)function of the LUT. It is essential to describe the LUT status in patients with NLUTD.

In these patients, particularly when detrusor overactivity might be present, the invasive urodynamic investigation is even more provocative than in other patients. Any technical source of artefacts must be critically considered. The quality of the urodynamic recording and its interpretation must be ensured (12).

In patients at risk for autonomic dysreflexia, it is advisable to measure blood pressure during the urodynamic study.

In many patients with NLUTD, it may be helpful to assess the maximum anaesthetic bladder capacity. The rectal ampulla should be empty of stool before the start of the investigation. Drugs that influence the lower urinary tract function should be stopped at least 48 hours before the investigation (if feasible) or otherwise be considered when interpreting the data obtained.

All urodynamic findings must be reported in detail and performed according to the ICS technical recommendations and standards (3, 12, 13).

3.4.2 Urodynamic tests

A *bladder diary* is a semi-objective qualification of the LUT. It is a highly advisable diagnostic tool. For reliable interpretation, it should be recorded over at least 2-3 days (3, 14). Possible pathological findings: high voiding frequency, very low or very high voided volumes, nocturnal voidings, urgency, incontinence.

Free uroflowmetry and assessment of residual urine gives a first impression of the voiding function. It is mandatory before planning any invasive urodynamics. For reliable information, it should be repeated at least 2-3 times (3, 10, 11). Possible pathological findings: low flow rate, low voided volume, intermittent flow, hesitancy, residual urine.

Care must be taken when assessing the results in patients who are not able to void in a normal position. Both the flow pattern and the flow rate may be modified by inappropriate positions and by any constructions to divert the flow.

Filling cystometry: The only method to quantify the filling function has limited significance as a solitary procedure. It is much more powerful if combined with bladder pressure measurement during micturition and

even more in video-urodynamics. This investigation is necessary to document the status of the LUT function during the filling phase. The bladder should be empty at the start of filling. A physiological filling rate should be used with body-warm saline, as fast filling and room-temperature saline are provocative (3).

Possible pathological findings include detrusor overactivity, low detrusor compliance, abnormal bladder and other sensations, incontinence, incompetent or relaxing urethra.

Detrusor leak point pressure (DLPP): This specific investigation may estimate the risk for the upper urinary tract or for secondary bladder damage (3, 15). The DLPP is a screening test only, because it gives no impression of the duration of the high pressure during the filling phase, which can be expected to have even more impact on the upper urinary tract (16). A high DLPP thus warrants further testing by video-urodynamics.

Pressure flow study: This measurement reflects the co-ordination between detrusor and urethra or pelvic floor during the voiding phase. It is even more powerful in combination with filling cystometry and with video urodynamics. It is necessary to document the function of the lower urinary tract function during the voiding phase. Possible pathological findings: Detrusor underactivity/acontractility, DSD, non-relaxing urethra, residual urine.

Most types of obstruction caused by NLUTD are due to DSD (17, 18), non-relaxing urethra, or non-relaxing bladder neck (3, 19, 20). Pressure-flow analysis mostly assesses the amount of mechanical obstruction caused by the urethra's inherent mechanical and anatomical properties and has limited value in patients with NLUTD.

Electromyography (EMG): Registration of the activity of the external urethral sphincter, the peri-urethral striated musculature, the anal sphincter, or the striated pelvic floor muscles. The correct interpretation may be difficult due to artefacts introduced by other equipment used. 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: Inadequate recruitment on specific stimuli (bladder filling, hyperreflexive contractions, onset of voiding, coughing, Valsalva, etc.). More detailed analysis (motor unit potentials, single-fibre EMG) is only possible as part of a neurophysiological investigation.

Urethral pressure measurement: This investigation has only a very limited place in NLUTD. There exists no basic consensus on parameters indicating pathological findings (21).

Video-urodynamics: This combination of filling cystometry and pressure flow study with imaging is the gold standard for urodynamic investigation in NLUTD (3, 22, 23). Possible pathological findings: All as described under cystometry and pressure flow study, plus morphological pathology of the LUT and the upper urinary tract.

Ambulatory urodynamics: Functional investigation of the urinary tract utilizing predominantly natural filling of the urinary tract and reproducing normal subject activity (24).

This type of study should be considered when office urodynamics do not reproduce the patient's symptoms and complaints. Possible pathological findings include those found under filling cystometry and pressure flow study, provided the flow is measured also. It should be kept in mind that during this study the actual bladder volume is unknown.

Provocative tests during urodynamics: The LUT function can be provoked by coughing, triggered voiding, or anal stretch.

Fast-filling cystometry with cooled saline (the 'ice water test') is considered a discriminative test between upper motor neuron lesion (UMNL) and lower motor neuron lesion (LMNL) (25-30). Patients with UMNL will develop a detrusor contraction if the detrusor muscle is intact, while patients with lower lesions will not. The test gives false-positive results in young children (27) and does not seem to be fully discriminative in other patients (28, 29).

It was thought that a positive bethanechol test (31) (detrusor contraction > 25 cm H₂O) provided proof of a detrusor denervation hypersensitivity and the muscular integrity of an acontractile detrusor; however, in practice, the test has given equivocal results. Recently, a variation of this method was reported using intravesical electromotive administration of the bethanechol (32); this test turned out to be both selective and predictive for successful oral bethanechol treatment.

3.4.3 Specific uro-neurophysiological tests

These tests are advised as part of the neurological work-up of the patient. They comprise:

- EMG (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 may be asked for specific conditions that became obvious during patient work-up and urodynamic investigations. Possible pathological findings are dependent on the type of the test.

3.4.4 GUIDELINES FOR URODYNAMICS AND URO-NEUROPHYSIOLOGY

1. Urodynamic investigation is necessary to document the (dys-)function of the LUT (Grade of recommendation: A).
2. The recording of a bladder diary is advisable (Grade of recommendation: B).
3. Non-invasive testing is mandatory before invasive urodynamics is planned (Grade of recommendation: A).
4. Video-urodynamics is the gold standard for invasive urodynamics in patients with NLUTD. If this is available, then a filling cystometry continuing into a pressure flow study should be performed (Grade of recommendation: A).
5. A physiological filling rate and body-warm saline must be used (Grade of recommendation: A).
6. Specific uro-neurophysiological tests are elective procedures (Grade of recommendation: C).

3.5 Typical manifestations of NLUTD

Typical findings in NLUTD are listed below:

Filling phase

- Hyposensitivity or hypersensitivity
- Vegetative sensations
- Low compliance
- High capacity bladder
- Detrusor overactivity, spontaneous or provoked
- Sphincter acontractility.

Voiding phase

- Detrusor acontractility
- DSD
- Non-relaxing urethra
- Non-relaxing bladder neck.

These signs warrant further neurological evaluation, as LUTD may be the presenting symptom of NLUTD (33-37).

3.6 REFERENCES

1. Bors E, Turner RD. History and physical examination in neurological urology. J Urol 1960;83:759-67. <http://www.ncbi.nlm.nih.gov/pubmed/13802958>
2. Thüroff JW, Chartier-Kastler E, Corcus J, Humke J, Jonas U, Palmtag H, Tanagho EA. Medical treatment and medical side effects in urinary incontinence in the elderly. World J Urol 1998;16(Suppl 1):S48-S61. <http://www.ncbi.nlm.nih.gov/pubmed/13802958>
3. Stöhrer M, Goepel M, Kondo A, Kramer G, Madersbacher H, Millard R, Rossier A, Wyndaele JJ. The standardization of terminology in neurogenic lower urinary tract dysfunction with suggestions for diagnostic procedures. International Continence Society Standardization Committee. Neurorol Urodyn 1999;18(2):139-58. <http://www.ncbi.nlm.nih.gov/pubmed/10081953>
4. Linsenmeyer TA, Oakley A. Accuracy of individuals with spinal cord injury at predicting urinary tract infections based on their symptoms. J Spinal Cord Med 2003;26(4):352-7. <http://www.ncbi.nlm.nih.gov/pubmed/14992336>
5. Wyndaele JJ, De Sy WA. Correlation between the findings of a clinical neurological examination and the urodynamic dysfunction in children with myelodysplasia. J Urol 1985;133(4):638-40. <http://www.ncbi.nlm.nih.gov/pubmed/3981715>
6. Wyndaele JJ. Correlation between clinical neurological data and urodynamic function in spinal cord injured patients. Spinal Cord 1997;35(4):213-6. <http://www.ncbi.nlm.nih.gov/pubmed/9143082>

7. Keshtgar AS, Rickwood AM. Urological consequences of incomplete cord lesions in patients with myelomeningocele. *Br J Urol* 1998;82(2):258-60.
<http://www.ncbi.nlm.nih.gov/pubmed/9722763>
8. Wyndaele JJ. Is impaired perception of bladder filling during cystometry a sign of neuropathy? *Br J Urol* 1993;71(3):270-3.
<http://www.ncbi.nlm.nih.gov/pubmed/8477312>
9. Kirchof K, Fowler CJ. The value of the Kurtzke Functional Systems Scales in predicting incomplete bladder emptying. *Spinal Cord* 2000;38(7):409-13.
<http://www.ncbi.nlm.nih.gov/pubmed/10962600>
10. Weld KJ, Dmochowski RR. Association of level of injury and bladder behavior in patients with posttraumatic spinal cord injury. *Urology* 2000;55(4):490-4.
<http://www.ncbi.nlm.nih.gov/pubmed/10736489>
11. Schurch B, Schmid DM, Kaegi K. Value of sensory examination in predicting bladder function in patients with T12-L1 fractures and spinal cord injury. *Arch Phys Med Rehabil* 2003;84(1):83-9.
<http://www.ncbi.nlm.nih.gov/pubmed/12589626>
12. Schurch B. The predictive value of plantar flexion of the toes in the assessment of neuropathic voiding disorders in patients with spine lesions at the thoracolumbar level. *Arch Phys Med Rehabil* 1999;80(6):681-6.
<http://www.ncbi.nlm.nih.gov/pubmed/10378495>
13. Ochoa B. Can a congenital dysfunctional bladder be diagnosed from a smile?. The Ochoa syndrome updated. *Pediatr Nephrol* 2004;19(1):6-12.
<http://www.ncbi.nlm.nih.gov/pubmed/14648341>
14. Reynard JM, Peters TJ, Lim C, Abrams P. The value of multiple free-flow studies in men with lower urinary tract symptoms. *Br J Urol* 1996;77(6):813-8.
<http://www.ncbi.nlm.nih.gov/pubmed/8705213>
15. Sonke GS, Kiemeny LA, Verbeek AL, Kortmann BB, Debruyne FM, de la Rosette JJ. Low reproducibility of maximum urinary flow rate determined by portable flowmetry. *Neurourol Urodyn* 1999;18(3):183-91.
<http://www.ncbi.nlm.nih.gov/pubmed/10338438>
16. Schafer W, Abrams P, Liao L, Mattiasson A, Pesce F, Spangberg A, Sterling AM, Zinner NR, van Kerrebroeck P; International Continence Society. Good urodynamic practices: uroflowmetry, filling cystometry, and pressure-flow studies. *Neurourol Urodyn* 2002;21(3):261-74.
<http://www.ncbi.nlm.nih.gov/pubmed/11948720>
17. Abrams P, Cardozo L, Fall M, Griffiths D, Rosier P, Ulmsten U, van Kerrebroeck P, Victor A, Wein A. The standardisation of terminology of lower urinary tract function: report from the Standardisation Subcommittee of the International Continence Society. *Neurourol Urodyn* 2002;21(2):167-78.
<http://www.ncbi.nlm.nih.gov/pubmed/11857671>
18. Homma Y, Ando T, Yoshida M, Kageyama S, Takei M, Kimoto K, Ishizuka O, Gotoh M, Hashimoto T. Voiding and incontinence frequencies: variability of diary data and required diary length. *Neurourol Urodyn* 2002;21(3):204-9.
<http://www.ncbi.nlm.nih.gov/pubmed/11948713>
19. McGuire EJ, Cespedes RD, O'Connell HE. Leak-point pressures. *Urol Clin North Am* 1996;23(2):253-62.
<http://www.ncbi.nlm.nih.gov/pubmed/8659025>
20. Linsenmeyer TA, Bagaria SP, Gendron B. The impact of urodynamic parameters on the upper tracts of spinal cord injured men who void reflexly. *J Spinal Cord Med* 1998;21(1):15-20.
<http://www.ncbi.nlm.nih.gov/pubmed/9541882>
21. Krongrad A, Sotolongo JR Jr. Bladder neck dysynergia in spinal cord injury. *Am J Phys Med Rehabil* 1996;75(3):204-7.
<http://www.ncbi.nlm.nih.gov/pubmed/8663928>
22. Weld KJ, Graney MJ, Dmochowski RR. Clinical significance of detrusor sphincter dyssynergia type in patients with post-traumatic spinal cord injury. *Urology* 2000;56(4):565-8.
<http://www.ncbi.nlm.nih.gov/pubmed/11018603>
23. Rossier AB, Fam BA. 5-microtransducer catheter in evaluation of neurogenic bladder function. *Urology* 1986;27(4):371-8.
<http://www.ncbi.nlm.nih.gov/pubmed/3962062>
24. Al-Ali M, Haddad L. 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(1):34-8.
<http://www.ncbi.nlm.nih.gov/pubmed/8848321>

25. Lose G, Griffiths D, Hosker G, Kulseng-Hanssen S, Perucchini D, Schafer W, Thind P, Versi E. Standardisation Sub-Committee, International Continence Society. Standardisation of urethral pressure measurement: report from the Standardisation Sub-Committee of the International Continence Society. *Neurourol Urodyn* 2002;21(3):258-60.
<http://www.ncbi.nlm.nih.gov/pubmed/11948719>
26. Rivas DA, Chancellor MB. Neurogenic vesical dysfunction. *Urol Clin North Am* 1995;22(3):579-91.
<http://www.ncbi.nlm.nih.gov/pubmed/7645158>
27. Madersbacher HG. Neurogenic bladder dysfunction. *Curr Opin Urol* 1999;9(4):303-7.
<http://www.ncbi.nlm.nih.gov/pubmed/10459465>
28. van Waalwijk van Doorn E, Anders K, Khullar V, Kulseng-Hanssen S, Pesce F, Robertson A, Rosario D, Schafer W. Standardisation of ambulatory urodynamic monitoring: report of the Standardisation Sub-Committee of the International Continence Society for Ambulatory Urodynamic Studies. *Neurourol Urodyn* 2000;19(2):113-25.
<http://www.ncbi.nlm.nih.gov/pubmed/10679828>
29. Geirsson G, Fall M, Lindstrom S. The ice-water test—a simple and valuable supplement to routine cystometry. *Br J Urol* 1993;71(6):681-5.
<http://www.ncbi.nlm.nih.gov/pubmed/8343894>
30. Geirsson G, Lindstrom S, Fall M. Pressure, volume and infusion speed criteria for the ice-water test. *Br J Urol* 1994;73(5):498-503.
<http://www.ncbi.nlm.nih.gov/pubmed/8012770>
31. Geirsson G, Lindstrom S, Fall M, Gladh G, Hermansson G, Hjalmas K. Positive bladder cooling test in neurologically normal young children. *J Urol* 1994;151(2):446-8.
<http://www.ncbi.nlm.nih.gov/pubmed/8283555>
32. Petersen T, Chandiramani V, Fowler CJ. The ice-water test in detrusor hyper-reflexia and bladder instability. *Br J Urol* 1997;79(2):163-7.
<http://www.ncbi.nlm.nih.gov/pubmed/9052463>
33. Chancellor MB, Lavelle J, Ozawa H, Jung SY, Watanabe T, Kumon H. Ice-water test in the urodynamic evaluation of spinal cord injured patients. *Tech Urol* 1998;4(2):87-91.
<http://www.ncbi.nlm.nih.gov/pubmed/9623622>
34. Ronzoni G, Menchinelli P, Manca A, De Giovanni L. The ice-water test in the diagnosis and treatment of the neurogenic bladder. *Br J Urol* 1997;79(5):698-701.
<http://www.ncbi.nlm.nih.gov/pubmed/9158504>
35. Lapidus J. Neurogenic bladder. Principles of treatment. *Urol Clin North Am* 1974;1(1):81-97.
<http://www.ncbi.nlm.nih.gov/pubmed/4428540>
36. Riedl CR, Stephen RL, Daha LK, Knoll M, Plas E, Pfluger H. Electromotive administration of intravesical bethanechol and the clinical impact on acontractile detrusor management: introduction of a new test. *J Urol* 2000;164(6):2108-11.
<http://www.ncbi.nlm.nih.gov/pubmed/11061937>
37. Bemelmans BL, Hommes OR, Van Kerrebroeck PE, Lemmens WA, Doesburg WH, Debruyne FM. Evidence for early lower urinary tract dysfunction in clinically silent multiple sclerosis. *J Urol* 1991;145(6):1219-24.
<http://www.ncbi.nlm.nih.gov/pubmed/2033697>

4. TREATMENT

4.1 Introduction

The primary aims for treatment of NLUTD and their priorities are (1-4):

1. Protection of the upper urinary tract
2. Improvement of urinary continence
3. Restoration of (parts of) the LUT function
4. Improvement of the patient's quality of life.

Further considerations are the patient's disability, cost-effectiveness, technical complexity and possible complications (4).

Preservation of the upper tract function is of paramount importance (1-7). Renal failure was the main factor for mortality in the spinal cord-injured patient surviving the trauma (5-7). This has led to the golden rule in treatment of NLUTD: ensure that the detrusor pressure remains within safe limits during both the filling phase and the voiding phase (1-4). This approach has indeed significantly reduced the mortality from urological

causes in this patient group (8).

The therapy of urinary incontinence is important for social rehabilitation of the patient and thus contributes substantially to the quality of life. It is also pivotal in preventing urinary tract infection (6, 7). If complete continence cannot be achieved, methods to attain a socially acceptable control of incontinence can be used.

The patient's quality of life is an essential part of any treatment decision.

In patients with high detrusor pressure during the filling phase (detrusor overactivity, low detrusor compliance) or during the voiding phase (DSD, other causes of bladder outlet obstruction), treatment is aimed primarily at 'conversion of an active, aggressive high-pressure bladder into a passive low-pressure reservoir' despite the resulting residual urine (1).

4.2 Non-invasive conservative treatment

4.2.1 Assisted bladder emptying

Incomplete bladder emptying is a serious risk factor for UTI, developing a high intravesical pressure during the filling phase, and incontinence. Methods to improve the voiding process are practised in patients with NLUTD.

Third party bladder expression (Credé): Regretfully, this method is still applied, foremost in infants and young children with myelomeningocele and sometimes in tetraplegics. Because of the high pressures that may be created during this procedure, it is potentially hazardous for the urinary tract (9).

Voiding by abdominal straining (Valsalva): The considerations mentioned under Credé above also apply to the Valsalva manoeuvre (1, 9-11). For both methods of emptying, long-term complications are hardly avoidable (9, 10) and the already weak pelvic floor function may be further impaired, thus exacerbating the existing incontinence (11).

Triggered reflex voiding: Stimulation of the sacral or lumbar dermatomes in patients with UMNL can elicit reflex contraction of the detrusor (1, 11). Morbidity occurs more often during the first decades of treatment (12-16). Strict urodynamic control is therefore required (1, 11).

4.2.2 Lower urinary tract rehabilitation

Behavioural modification techniques: These are used to improve continence and include prompted voiding, timed voiding (bladder training), and lifestyle modification (17-20).

Pelvic floor muscle exercises: These aim to improve continence. They may be helpful in selected patients with NLUTD (21).

Pelvic floor electrostimulation: This technique may help to improve the effect of pelvic floor muscle exercises, or to teach the patient how to contract the pelvic floor, or to improve the patient's compliance with the exercises (11, 22, 23).

Biofeedback: This method can be used for supporting the voiding pattern modification (24, 25).

4.2.3 Drug treatment

A single optimal medical therapy for NLUTD is not yet available. A combination of therapies is currently the best way to maximize outcomes (26-32) (Level of evidence: 1a, Grade of recommendation: A).

Anticholinergic agents are the most useful medications available for neurogenic detrusor overactivity (NDO) (32-36) (Level of evidence: 1a, Grade of recommendation: A). As these drugs bind to muscarinic receptors, they are also termed muscarinic receptor antagonists. Anticholinergic agents are used to reduce detrusor overactivity and to improve bladder compliance (32) (Level of evidence: 1a, Grade of recommendation: A).

Neurogenic patients may need a higher dose of anticholinergics than patients with idiopathic detrusor overactivity (32, 33, 37-39) (Level of evidence: 1b, Grade of recommendation: A). However, this may lead to early discontinuation of therapy because of adverse events (19, 21, 38, 40, 41) (Level of evidence: 1b, Grade of recommendation: A).

Oxybutynin (32) (Level of evidence: 1a, Grade of recommendation: A) (33-36, 39, 40, 42), trospium chloride (32, 37, 38), tolterodine (43) (Level of evidence: 1a, Grade of recommendation: A) and propiverine (32, 40, 44) are established effective medical treatments. These drugs are well tolerated and safe, even during long-term treatment. They have diverse tolerance profiles so that a different anticholinergic agent may be prescribed if a patient experiences adverse effects or if the therapeutic effect is not sufficient.

Recently, darifenacin and solifenacin have been introduced, but as yet no clinical experience with

these drugs in neurogenic bladder overactivity has been published.

Phosphodiesterase inhibitors demonstrated significant effects upon detrusor overactivity in pilot studies and may become a future alternative or adjunct to anticholinergic treatment (45). Additional treatment with desmopressin might improve the efficacy of the treatment (46, 47).

Detrusor underactivity: Cholinergic drugs, such as bethanechol chloride and distigmine bromide, have been considered to enhance detrusor contractility and promote bladder emptying, but are not routinely used in clinical practice. The available studies do not support the use of parasympathomimetics, specifically when frequent and/or serious possible side effects are taken into account (48) (Level of evidence: 1a, Grade of recommendation: A). Combination therapy with a cholinergic drug and an alpha-blocker appears to be more useful than monotherapy (49). In conclusion, there is no drug with evidence of efficacy for underactive detrusor (11, 50-53) (Level of evidence: 2a, Grade of recommendation: B).

Decreasing bladder outlet resistance: Alpha-blockers (non-selective and selective) have been partially successful for decreasing bladder outlet resistance, residual urine and autonomic dysreflexia (11, 54-58) (Level of evidence: 2a, Grade of recommendation: B).

Increasing bladder outlet resistance: several drugs have shown efficacy in the treatment of selected cases of mild stress urinary incontinence, but there are hardly any publications in patients with NLUTD (11, 59).

Conclusions:

- Long-term efficacy and safety of anticholinergic therapy for NDO is well documented (Level of evidence: 1a, Grade of recommendation: A).
- A combination of therapies is often considered to maximize outcomes for NDO (Level of evidence: 1a, Grade of recommendation: A).
- There is no drug with evidence of efficacy for underactive detrusor (Level of evidence: 2a, Grade of recommendation: B).
- Alpha-blockers have been partly successful for decreasing bladder outlet resistance and autonomic dysreflexia prophylaxis in SCI patients (Level of evidence: 2a, Grade of recommendation: B).
- There is a lack of prospective randomized controlled studies in the medical management of NLUTD.

4.2.4 Electrical neuromodulation

A strong contraction of the urethral sphincter and/or pelvic floor, but also anal dilatation, manipulation of the genital region, and physical activity reflexly inhibit the micturition (11, 60). Whereas the first mechanism is affected by activation of efferent fibres, the latter ones are produced by activation of afferents (14). Electrical stimulation of the pudendal nerve afferents produces a strong inhibition of the micturition reflex and of the detrusor contraction (61). This stimulation might then support the restoration of the balance between excitatory and inhibitory inputs at the spinal or supraspinal level (11, 62, 63). It might also imply that patients with incomplete lesions will benefit (11, 63, 64), but patients with complete lesions will not (65).

Although electrical stimulation of the posterior tibial nerve afferents has been used for neurogenic patients (66), there is no current evidence suggesting this therapy has any benefit in NLUTD patients.

4.2.5 External appliances

As an ultimate remedy, social continence may be achieved by collecting urine during incontinence (1, 11). Condom catheters with urine collection devices are a practical method for men. Otherwise, incontinence pads may offer a reliable solution. In both cases, the infection risk must be closely observed (11). Because of the risk of developing high intravesical pressure, the penile clamp is absolutely contraindicated.

4.2.6 GUIDELINES FOR NON-INVASIVE CONSERVATIVE TREATMENT

1. The first aim of any therapy is the protection of the upper urinary tract.
2. The mainstay of treatment for overactive detrusor is anticholinergic drug therapy (Level of evidence: 1, Grade of recommendation: A).
3. Lower urinary tract rehabilitation may be effective in selected cases.
4. A condom catheter or pads may reduce urinary incontinence to a socially acceptable situation.
5. Any method of assisted bladder emptying should be used with the greatest caution (Grade of recommendation: A).

4.3 Minimal invasive treatment

4.3.1 Catheterization

Intermittent self- or third-party catheterization (67, 68) is the gold standard for the management of NLUTD (1, 11). It is effective in patients with:

- Detrusor underactivity or acontractility (1)
- With detrusor overactivity, provided the overactivity can be controlled (1, 11, 26-31).

Sterile IC, as originally proposed by Guttman and Frankel (67), significantly reduces the risk of UTI and/or bacteriuria (1, 11, 69, 70), compared with clean IC introduced by Lapedes et al. (68). However, it cannot be considered a routine procedure (11, 70). Aseptic IC is an alternative (1, 71), which provides a significant benefit in reducing the potential for external contamination of an intermittent urinary catheter (72). Insufficient patient education and the inherent greater risk of UTI in patients with NLUTD are contributing factors (11, 73-77). The average frequency of catheterizations per day is 4-6 times and the catheter size should be 12-14 Fr.

Less frequent catheterization results in higher catheterization volumes and a higher risk of UTI (1, 73-76). More frequent catheterization increases the risk of cross-infections and other complications (1, 73-76). Bladder volume at catheterization should be lower than 400 mL.

The prevalence of complications can be limited by adequate patient education, use of non-traumatizing techniques and adequate precautions to prevent infections (11, 77).

Indwelling transurethral catheterization and, to a lesser extent, suprapubic cystostomy are significant and early risk factors for UTI and other complications (11, 16, 78-87). Silicone catheters are preferred because they are less susceptible to encrustation and because of the high incidence of latex allergy in the NLUTD population.

4.3.2 GUIDELINES FOR CATHETERIZATION

1. Intermittent catheterization is the standard treatment for patients who are unable to empty their bladder (Level of evidence: 2, Grade of recommendation: A).
2. Patients should be well instructed in the technique and risks of IC.
3. Aseptic IC is the method of choice (Level of evidence: 2, Grade of recommendation: B).
4. The catheter size should be 12-14 Fr (Grade of recommendation: B).
5. The frequency of IC is 4-6 times per day (Grade of recommendation: B).
6. The bladder volume should remain below 400 mL (Grade of recommendation: B).
7. Indwelling transurethral and suprapubic catheterization should be used only exceptionally, under close control, and the catheter should be changed frequently. Silicone catheters are preferred and should be changed every 2-4 weeks, while (coated) latex catheters need to be changed every 1-2 weeks. (Grade of recommendation: A).

4.3.3 Intravesical drug treatment

To reduce detrusor overactivity, anticholinergics can also be applied intravesically (88-94). This approach may reduce adverse effects because the anticholinergic drug is metabolized differently (92) and a greater amount is sequestered in the bladder, even more than with electromotive administration (93, 94).

The vanilloids, capsaicin and resiniferatoxin, desensitize the C-fibres and thereby decrease detrusor overactivity for a period of a few months until the sensation of these fibres has been restored (95-100).

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 patients refractory to capsaicin. Clinical studies have shown that resiniferatoxin has limited clinical efficacy compared to botulinum toxin A injections in the detrusor (100).

4.3.4 Intravesical electrostimulation

Intravesical electrostimulation (101) enhances the sensation for bladder filling and urge to void and may restore the volitional control of the detrusor (11, 102, 103). Daily stimulation sessions of 90 minutes with 10 mA pulses of 2 ms duration at a frequency of 20 Hz (103, 104) are used for at least 1 week (104). It appears that patients with peripheral lesions are the best candidates, that the detrusor muscle must be intact, and that at least some afferent connection between the detrusor and the brain must still be present (11, 103, 104). Also, the positioning of the stimulating electrodes and bladder filling are important parameters (105). With these precautions, the results in the literature are still not unequivocal: both positive (102, 104, 106, 107) and negative (Level of evidence: 3, Grade of recommendation: C) (108, 109) results have been reported.

4.3.5 Botulinum toxin injections in the bladder

Botulinum toxin causes a long-lasting but reversible chemical denervation that lasts for about 9 months (110-116). 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 a randomized placebo-controlled trial in NLUT (117).

Repeated injections seem to be possible without loss of efficacy (116, 118, 119). Generalized

muscular weakness is an occasional adverse effect (114, 116, 119). Histological studies have not found ultrastructural changes after injection (120).

4.3.6 Bladder neck and urethral procedures

Reduction of the bladder outlet resistance may be necessary to protect the upper urinary tract. This can be achieved by surgical interventions (bladder neck or sphincter incision or urethral stent) or by chemical denervation of the sphincter. Incontinence may result and can be managed by external devices (see Section 4.2.5).

Botulinum toxin sphincter injection can be used to treat detrusor sphincter dyssynergia effectively by injection in a dosage that depends on the preparation used. The dyssynergia is abolished for a few months, necessitating repeat injections. The efficacy of this treatment is high and there are few adverse effects (121-123).

Balloon dilatation: although favourable immediate results were reported (124), no further reports since 1994 have been found. Consequently, 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 (1, 11, 117). The laser technique appears to be advantageous (1, 126).

Sphincterotomy also needs to be repeated at regular intervals in a substantial proportion of patients (127), but is efficient and without severe adverse effects (1, 9, 124-128). Secondary narrowing of the bladder neck may occur, for which combined bladder neck incision might be considered (1, 129).

Bladder neck incision: This is indicated only for secondary changes at the bladder neck (fibrosis) (1, 9, 125, 129). When the detrusor is hypertrophied and causes thickening of the bladder neck, this procedure makes no sense (1).

Stents: Implantation of urethral stents causes the continence to be dependent on the adequate closure of the bladder neck only (1, 4). Although the results are comparable with sphincterotomy and the stenting procedure has a shorter surgery time and reduced hospital stay (130, 131), the costs (1) and possible complications or re-interventions (130, 132, 133) are limiting factors in its use.

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 NLUTD (4, 16, 134-138).

Urethral inserts: Urethral plugs or valves for management of (female) stress incontinence have not been applied in patients with NLUTD. The experience with active pumping urethral prosthesis for treatment of the underactive or acontractile detrusor was disappointing (139).

4.3.6 GUIDELINES FOR MINIMAL INVASIVE TREATMENT

1. Guidelines for catheterization are listed separately under Section 4.3.2.
2. Botulinum toxin injection in the detrusor is the most effective minimally invasive treatment to reduce neurogenic detrusor overactivity (Level of evidence: 1, Grade of recommendation: A).
3. Sphincterotomy is the standard treatment for DSD (Level of evidence: 2, Grade of recommendation: A).
4. Bladder neck incision is effective in a fibrotic bladder neck (Level of evidence: 3, Grade of recommendation: B).

4.4 Surgical treatment

4.4.1 Urethral and bladder neck procedures

Increasing the bladder outlet resistance has the inherent risk of causing high intravesical pressure during the filling, which may become even higher during the voiding phase. Procedures to treat sphincteric incontinence are suitable only when the detrusor activity is, or can be, controlled, when no significant reflux is present.

Moreover, these procedures require the urethra and bladder neck to be in good condition and mostly result in intermittent catheterization being performed after the procedure (4).

Urethral sling: Various materials have been used for this procedure with enduring positive results (4, 140-153). The procedure is established in women; for men, the artificial sphincter is obviously the first choice (4).

Artificial urinary sphincter: This device has stood the test of time in patients with NLUTD (4). It was introduced

by Light and Scott (154) for this patient group and the need for revisions (154) has decreased significantly with new generations of devices (146, 156-159).

Functional sphincter augmentation: By transposing the gracilis muscle to the bladder neck (160) or to the proximal urethra (161), the possibility exists for creating a functional autologous sphincter by electrical stimulation (160,161). This would open the possibility of restoring control over the urethral closure.

Bladder neck and urethra reconstruction: The classical Young-Dees-Leadbetter (162) procedure for bladder neck reconstruction in children with bladder exstrophy and the Kropp urethral lengthening (163) improved by Salle (164) are established methods to restore continence provided that intermittent catheterization is practised and/or bladder augmentation is performed (146, 155, 163-174).

4.4.2 *Detrusor myectomy (auto-augmentation)*

The idea of enlarging a shrunken bladder by removing lateral detrusor tissue to free the entrapped ureter in a non-functional fibrotic detrusor was put forward by Couvelaire (175). Since its clinical introduction by Cartwright and Snow (176) in children and by Stöhrer (177) in adults, this procedure for reducing detrusor overactivity or improving low detrusor compliance has gained popularity because of its acceptable long-term results, its low surgical burden, its low rate of long-term adverse effects, its positive effect on the patient's quality of life, and because it does not preclude further interventions (1, 4, 176-195).

The procedure is performed extraperitoneally under general anaesthesia and consists of the dissection of about 20% of the detrusor tissue around the umbilicus, leaving the mucosa intact (1, 176, 177). A diverticulum will develop, but this may take 1-2 years in adults (1, 165, 166). A laparoscopic procedure (26, 179, 183, 187), covering of the mucosa at the detrusor defect (transperitoneal) (24, 186, 188, 192), supporting the bladder (176, 192), or simple incision of the detrusor muscle (detrusor myotomy) (194, 195) are proposed variations of the procedure but offer no essential advantages.

4.4.3 *Denervation, deafferentation, neurostimulation, neuromodulation*

Various procedures estimated to destroy the peripheral detrusor innervation have been abandoned because of poor long-term results and severe complications (4). These procedures include bladder distension, cystolysis, transvaginal denervation (Ingelman-Sundberg procedure) and subtrigonal phenol injections.

Sacral rhizotomy, also known as sacral deafferentation (SDAF), has achieved some success in reducing detrusor overactivity (16, 196-200), but it is used nowadays mostly as an adjuvant to sacral anterior root stimulation (201-212). Alternatives for rhizotomy are sought in this treatment combination (213-215).

Sacral anterior root stimulation (SARS) is aimed at producing a detrusor contraction. The technique was developed by Brindley (216) and is applicable only in complete lesions above the implant location because of its stimulation amplitude over the pain threshold. The urethral sphincter efferents are also stimulated, but as the striated muscle relaxes faster than the smooth muscle of the detrusor, a so-called 'post-stimulus voiding' will occur. This approach has been successful in highly selected patients (201-212). By changing the stimulation parameters, this method can also induce defecation or erection.

The sacral nerve stimulation or sacral neuromodulation is based on the research by Schmidt and Tanagho (217). This technique stimulates the afferents and thereby probably restores the correct balance between excitatory and inhibitory impulses from and to the pelvic organs at a sacral and supra-sacral level, thus reducing the detrusor overactivity (62, 218). It is used either as a temporary procedure using foramen electrodes with an external stimulator, with the expectation that the changes will persevere after treatment, or as a chronic procedure with an implanted stimulator. In the latter case, a test procedure, the percutaneous nerve evaluation (PNE), with an external stimulator is performed before the implant to judge the patient's response. This procedure also has considerable success in selected patients (184, 219-223).

On the basis of the successful application of these systems, future developments towards a device that may be more integrated in the body are under research (224).

4.4.4 *Bladder covering by striated muscle*

When the bladder is covered by a (part of) striated muscle that can be stimulated electrically, or ideally could be contracted volitionally, an acontractile bladder could be restored to perform a voiding function. The rectus abdominis (225) and the latissimus dorsi (226) have been used successfully in patients with NLUTD.

4.4.5 *Bladder augmentation or substitution*

Replacing or expanding the bladder by intestine or other passive expandable coverage will reduce detrusor compliance and at least reduce the pressure effect of detrusor overactivity. The inherent complications associated with these procedures include recurrent infection, stone building, perforation or diverticula, possible malignant changes, and for intestine metabolic abnormality, mucus production and impaired bowel function

(4, 227-229). Since the age of the NLUTD patient population, when the surgery is performed, is generally much lower than that of patients with bladder malignancy, who are elected for this surgery, it is important that any possible, very long-term, complications in particular must be appraised. Thus, the procedures should be used with caution in NLUTD patients, but may become necessary if all less-invasive treatment methods have failed.

Bladder augmentation, by procedures such as clam cystoplasty, is a valid option to decrease detrusor pressure and increase bladder capacity, whenever more conservative approaches have failed. A number of different techniques have been published. The results of the various procedures are very good and comparable (182, 184-186, 189-191, 228-231). Bladder substitution to create a low pressure reservoir may be indicated in patients with severely thick and fibrotic bladder wall. Scaffolds, probably of tissue-engineered material for bladder augmentation or substitution or alternative techniques, are promising future options (190, 232-237).

4.4.6 Urinary diversion

When no other therapy has been successful urinary diversion must be considered for the protection of the upper tract and for the patient's quality of life (4, 238).

Continent diversion: This should be the first choice for diversion. In patients for whom indwelling catheterization or suprapubic catheterization is the only feasible treatment option, change to a continent stoma may be a better prospect (4). Some patients with limited dexterity prefer a stoma to using the urethra for catheterization (4). The continent stoma is created following various techniques. All of them, however, do show frequent complications, including leakage or stenosis (4, 239). The short-term continence rates are over 80% and good protection of the upper urinary tract is achieved (4, 13, 237-251). For cosmetic reasons, the umbilicus is often used for the stoma site, but this may have a higher risk of stenosis (242, 244, 249).

Incontinent diversion: If catheterization is impossible, incontinent diversion with a urine collecting device is indicated. Fortunately, nowadays, this indication is seldom because many appropriate alternatives can be offered (4). Ultimately, it could be considered in patients who are wheelchair bound or bed-ridden with intractable and untreatable incontinence, in devastated lower urinary tracts, when the upper urinary tract is severely compromised, and in patients who refuse other therapy (4). An ileal segment is used for the deviation in most cases (4, 252-256). The rather poor long-term results and the expected complications warrant a permanent follow-up (4).

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 the detrusor pressure and the incontinence (4). Also, in young patients, body image may play a role (246). The patient must be carefully counselled and must comply meticulously with the instructions (4). Successful undiversion can then be performed (257).

4.5 GUIDELINES FOR SURGICAL TREATMENT

1. Detrusor

- Overactive

- Detrusor myectomy is an acceptable option for the treatment of overactive bladder when more conservative approaches have failed. It is limited invasive and has minimal morbidity (Level of evidence: 2, Grade of recommendation: B).
- Sacral rhizotomy with SARS in complete lesions and sacral neuromodulation in incomplete lesions are effective treatments in selected patients (Level of evidence: 2, Grade of recommendation: B).
- Bladder augmentation is an acceptable option for decreasing detrusor pressure whenever less invasive procedures have failed. For the treatment of a severely thick or fibrotic bladder wall, a bladder substitution might be considered (Level of evidence: 2, Grade of recommendation: B).

- Underactive

- SARS with rhizotomy and sacral neuromodulation are effective in selected patients (Level of evidence: 2, Grade of recommendation: B).
- Restoration of a functional bladder by covering with striated muscle is still experimental (Level of evidence: 4).

2. Urethra

- Overactive (DSD): refer to guidelines for minimal invasive treatment (see Section 4.3.6)

- Underactive

- The placement of a urethral sling is an established procedure (Level of evidence: 2, Grade of recommendation: B).

- The artificial urinary sphincter is very effective (Level of evidence: 2, Grade of recommendation: B).
- Transposition of the gracilis muscle is still experimental (Level of evidence: 4).

4.6 REFERENCES

1. Stöhrer M, Kramer G, Löchner-Ernst D, Goepel M, Noll F, Rübber H. Diagnosis and treatment of bladder dysfunction in spinal cord injury patients. *Eur Urol Update Series* 1994;3:170-5.
2. Burns AS, Rivas DA, Ditunno JF. The management of neurogenic bladder and sexual dysfunction after spinal cord injury. *Spine* 2001;26 (24 Suppl):S129-S136.
<http://www.ncbi.nlm.nih.gov/pubmed/11805620>
3. Rickwood AM. Assessment and conservative management of the neuropathic bladder. *Semin Pediatr Surg* 2002 May;11(2):108-19.
<http://www.ncbi.nlm.nih.gov/pubmed/11973763>
4. Castro-Diaz D, Barrett D, Grise P, Perkash I, Stohrer M, Stone A, Vale P. Surgery for the neuropathic patient. In: *Incontinence*, 2nd edn. Abrams P, Khoury S, Wein A, eds. Plymouth: Health Publication, 2002; pp. 865-891.
5. Donnelly J, Hackler RH, Bunts RC. Present urologic status of the World War II paraplegic: 25-year follow-up. Comparison with status of the 20-year Korean War paraplegic and 5-year Vietnam paraplegic. *J Urol* 1972;108(4):558-62.
<http://www.ncbi.nlm.nih.gov/pubmed/4651345>
6. Hackler RH. A 25-year prospective mortality study in the spinal cord injured patient: comparison with the long-term living paraplegic. *J Urol* 1977;117(4):486-8.
<http://www.ncbi.nlm.nih.gov/pubmed/850323>
7. Game X, Castel-Lacanal E, Bentaleb Y, Thiry-Escudie I, De Boissezon X, Malavaud B, Marque P, Rischmann P. Botulinum toxin A detrusor injections in patients with neurogenic detrusor overactivity significantly decrease the incidence of symptomatic urinary tract infections. *Eur Urol* 2007;53(3): 613-9.
<http://www.ncbi.nlm.nih.gov/pubmed/17804150>
8. Frankel HL, Coll JR, Charlifue SW, Whiteneck GG, Gardner BP, Jamous MA, Krishnan KR, Nuseibeh I, Savic G, Sett P. Long-term survival in spinal cord injury: a fifty year investigation. *Spinal Cord* 1998;36(4):266-74.
<http://www.ncbi.nlm.nih.gov/pubmed/9589527>
9. Stöhrer M. Alterations in the urinary tract after spinal cord injury—diagnosis, prevention and therapy of late sequelae. *World J Urol* 1990;7:205-11.
10. Barbalias GA, Klauber GT, Blaivas JG. Critical evaluation of the Crede manoeuvre: a urodynamic study of 207 patients. *J Urol* 1983;130(4):720-3.
<http://www.ncbi.nlm.nih.gov/pubmed/6887405>
11. Madersbacher H, Wyndaele JJ, Igawa Y, Chancellor M, Chartier-Kastler E, Kovindha A. Conservative management in neuropathic urinary incontinence. In: *Incontinence*, 2nd edn. Abrams P, Khoury S, Wein A, eds. Plymouth: Health Publication, 2002; pp. 697-754.
12. Van Kerrebroeck PE, Koldewijn EL, Scherpenhuizen S, Debruyne FM. The morbidity due to lower urinary tract function in spinal cord injury patients. *Paraplegia* 1993;31(5):320-9.
<http://www.ncbi.nlm.nih.gov/pubmed/8332378>
13. Sekar P, Wallace DD, Waites KB, DeVivo MJ, Lloyd LK, Stover SL, Dubovsky EV. Comparison of longterm renal function after spinal cord injury using different urinary management methods. *Arch Phys Med Rehabil* 1997;78(9):992-7.
<http://www.ncbi.nlm.nih.gov/pubmed/9305274>
14. Linsenmeyer TA, Bagaria SP, Gendron B. The impact of urodynamic parameters on the upper tracts of spinal cord injured men who void reflexly. *J Spinal Cord Med* 1998;21(1):15-20.
<http://www.ncbi.nlm.nih.gov/pubmed/9541882>
15. McKinley WO, Jackson AB, Cardenas DD, DeVivo MJ. Long-term medical complications after traumatic spinal cord injury: a regional model systems analysis. *Arch Phys Med Rehabil* 1999;80(11):1402-10.
<http://www.ncbi.nlm.nih.gov/pubmed/10569434>
16. Weld KJ, Dmochowski RR. Effect of bladder management on urological complications in spinal cord injured patients. *J Urol* 2000;163(3):768-72.
<http://www.ncbi.nlm.nih.gov/pubmed/10687973>
17. Menon EB, Tan ES. Bladder training in patients with spinal cord injury. *Urology* 1992;40(5):425-9.
<http://www.ncbi.nlm.nih.gov/pubmed/1441039>

18. Nijman RJ. Classification and treatment of functional incontinence in children. *BJU Int* 2000;85(3):37-42.
<http://www.ncbi.nlm.nih.gov/pubmed/11954196>
19. Aslan AR, Kogan BA. Conservative management in neurogenic bladder dysfunction. *Curr Opin Urol* 2002;12(6):473-7.
<http://www.ncbi.nlm.nih.gov/pubmed/12409875>
20. Christ KF, Kornhuber HH. Treatment of neurogenic bladder dysfunction in multiple sclerosis by ultrasound-controlled bladder training. *Arch Psychiatr Nervenkr* 1980;228(3):191-5.
<http://www.ncbi.nlm.nih.gov/pubmed/7416934>
21. De Ridder D, Vermeulen C, Ketelaer P, Van Poppel H, Baert L. Pelvic floor rehabilitation in multiple sclerosis. *Acta Neurol Belg* 1999;99(1):61-4.
<http://www.ncbi.nlm.nih.gov/pubmed/10218095>
22. Ishigooka M, Hashimoto T, Hayami S, Suzuki Y, Nakada T, Handa Y. Electrical pelvic floor stimulation: a possible alternative treatment for reflex urinary incontinence in patients with spinal cord injury. *Spinal Cord* 1996;34(7):411-5.
<http://www.ncbi.nlm.nih.gov/pubmed/8963996>
23. Balcom AH, Wiatrak M, Biefeld T, Rauen K, Langenstroer P. Initial experience with home therapeutic electrical stimulation for continence in the myelomeningocele population. *J Urol* 1997;158(3 Pt 2):1272-6.
<http://www.ncbi.nlm.nih.gov/pubmed/9258193>
24. Chin-Peuckert L, Salle JL. A modified biofeedback program for children with detrusor-sphincter dyssynergia: 5-year experience. *J Urol* 2001;166(4):1470-5.
<http://www.ncbi.nlm.nih.gov/pubmed/11547115>
25. McClurg D, Ashe RG, Marshall K, Lowe-Strong AS. 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(4):337-48.
<http://www.ncbi.nlm.nih.gov/pubmed/16637070>
26. Baskin LS, Kogan BA, Benard F. Treatment of infants with neurogenic bladder dysfunction using anticholinergic drugs and intermittent catheterisation. *Br J Urol* 1990;66(5):532-4.
<http://www.ncbi.nlm.nih.gov/pubmed/2249125>
27. Tanaka H, Kakizaki H, Kobayashi S, Shibata T, Ameda K, Koyanagi T. The relevance of urethral resistance in children with myelodysplasia: its impact on upper urinary tract deterioration and the outcome of conservative management. *J Urol* 1999;161(3):929-32.
<http://www.ncbi.nlm.nih.gov/pubmed/10022727>
28. Stone AR. Neurourologic evaluation and urologic management of spinal dysraphism. *Neurosurg Clin N Am* 1995;6(2):269-77.
<http://www.ncbi.nlm.nih.gov/pubmed/7620353>
29. Edelstein RA, Bauer SB, Kelly MD, Darbey MM, Peters CA, Atala A, Mandell J, Colodny AH, Retik AB. The long-term urological response of neonates with myelodysplasia treated proactively with intermittent catheterization and anticholinergic therapy. *J Urol* 1995;154(4):1500-4.
<http://www.ncbi.nlm.nih.gov/pubmed/7658577>
30. DasGupta R, Fowler CJ. Bladder, bowel and sexual dysfunction in multiple sclerosis: management strategies. *Drugs* 2003;63(2):153-66.
<http://www.ncbi.nlm.nih.gov/pubmed/12515563>
31. Buyse G, Verpoorten C, Vereecken R, Casaer P. Treatment of neurogenic bladder dysfunction in infants and children with neurospinal dysraphism with clean intermittent (self)catheterisation and optimized intravesical oxybutynin hydrochloride therapy. *Eur J Pediatr Surg* 1995;5(1):31-4.
<http://www.ncbi.nlm.nih.gov/pubmed/8770576>
32. Appell RA. Overactive bladder in special patient populations. *Rev Urol* 2003;5(8): S37-S41.
<http://www.ncbi.nlm.nih.gov/pubmed/16985989>
33. Bennett N, O'Leary M, Patel AS, Xavier M, Erickson JR, Chancellor MB. Can higher doses of oxybutynin improve efficacy in neurogenic bladder? *J Urol* 2004;171(2 Pt 1):749-51.
<http://www.ncbi.nlm.nih.gov/pubmed/14713802>
34. Chancellor MB, Anderson RU, Boone TB. Pharmacotherapy for neurogenic detrusor overactivity. *Am J Phys Med Rehabil* 2006;85(6):536-45.
<http://www.ncbi.nlm.nih.gov/pubmed/16715024>
35. Diokno A, Ingber M. Oxybutynin in detrusor overactivity. *Urol Clin N Am* 2006;33(4):439-45.
<http://www.ncbi.nlm.nih.gov/pubmed/17011379>

36. Franco I, Horowitz M, Grady R, Adams RC, de Jong TP, Lindert K, Albrecht D. Efficacy and safety of oxybutynin in children with detrusor hyperreflexia secondary to neurogenic bladder dysfunction. *J Urol* 2005;173(1):221-5.
<http://www.ncbi.nlm.nih.gov/pubmed/15592080>
37. Horstmann M, Schaefer T, Aguilar Y, Stenzl A, Sievert KD. Neurogenic bladder treatment by doubling the recommended antimuscarinic dosage. *Neurourol Urodyn* 2006;25(5):441-5.
<http://www.ncbi.nlm.nih.gov/pubmed/16847942>
38. Menarini M, Del Popolo G, Di Benedetto P, Haselmann J, Bödeker RH, Schwantes U, Madersbacher H; TcP128-Study Group. Trospium chloride in patients with neurogenic detrusor overactivity: is dose titration of benefit to the patients? *Int J Clin Pharmacol Ther* 2006;44(12):623-32.
<http://www.ncbi.nlm.nih.gov/pubmed/17190372>
39. O'Leary M, Erickson JR, Smith CP, McDermott C, Horton J, Chancellor MB. Effect of controlled-release oxybutynin on neurogenic bladder function in spinal cord injury. *J Spinal Cord Med* 2003;26(2):159-62.
<http://www.ncbi.nlm.nih.gov/pubmed/12828295>
40. Stöhrer M, Mürtz G, Kramer G, Schnabel F, Arnold EP, Wyndaele JJ; Propiverine Investigator Group. Propiverine compared to oxybutynin in neurogenic detrusor overactivity--results of a randomized, double-blind, multicenter clinical study. *Eur Urol* 2007;51(1):235-42.
<http://www.ncbi.nlm.nih.gov/pubmed/16698176>
41. Schwantes U, Topfmeier P. Importance of pharmacological and physicochemical properties for tolerance of antimuscarinic drugs in the treatment of detrusor instability and detrusor hyperreflexia: chances for improvement of therapy. *Int J Clin Pharmacol Ther* 1999;37(5):209-18.
<http://www.ncbi.nlm.nih.gov/pubmed/10363619>
42. Saito M, Watanabe T, Tabuchi F, Otsubo K, Satoh K, Miyagawa I. Urodynamic effects and safety of modified intravesical oxybutynin chloride in patients with neurogenic detrusor overactivity: 3 years experience. *Int J Urol* 2004;11(8):592-6.
<http://www.ncbi.nlm.nih.gov/pubmed/15285747>
43. Ethans KD, Nance PW, Bard RJ, Casey AR, Schryvers OI. Efficacy and safety of tolterodine in people with neurogenic detrusor overactivity. *Spinal Cord Med* 2004;27(3):214-8.
<http://www.ncbi.nlm.nih.gov/pubmed/15478523>
44. Grigoleit U, Mürtz G, Laschke S, Schuldt M, Goepel M, Kramer G, Stohrer M. Efficacy, tolerability and safety of propiverine hydrochloride in children and adolescents with congenital or traumatic neurogenic detrusor overactivity: a retrospective study. *Eur Urol* 2006;49(6):1114-21.
<http://www.ncbi.nlm.nih.gov/pubmed/16542772>
45. Gacci M, Del Popolo G, Macchiarella A, Celso M, Vittori G, Lapini A, Serni S, Sandner P, Maggi M, Carini M. Vardenafil improves urodynamic parameters in men with spinal cord injury: results from a single dose, pilot study. *J Urol* 2007;178(5):2040-3; discussion 2044.
<http://www.ncbi.nlm.nih.gov/pubmed/17869296>
46. Chancellor MB, Rivas DA, Staas WE Jr. DDAVP in the urological management of the difficult neurogenic bladder in spinal cord injury: preliminary report. *J Am Paraplegia Soc* 1994;17(4):165-7.
<http://www.ncbi.nlm.nih.gov/pubmed/7869058>
47. Valiquette G, Herbert J, Maede-D'Alisera P. Desmopressin in the management of nocturia in patients with multiple sclerosis. A double-blind, crossover trial. *Arch Neurol* 1996;53(12):1270-5.
<http://www.ncbi.nlm.nih.gov/pubmed/8970454>
48. Barendrecht MM, Oelke M, Laguna MP, Michel MC. Is the use of parasympathomimetics for treating an underactive urinary bladder evidence-based? *BJU Int* 2007;99(4):749-52.
<http://www.ncbi.nlm.nih.gov/pubmed/17233798>
49. Yamanishi T, Yasuda K, Kamai T, Tsujii T, Sakakibara R, Uchiyama T, Yoshida K. Combination of a cholinergic drug and an alpha-blocker is more effective than monotherapy for the treatment of voiding difficulty in patients with underactive detrusor. *Int J Urol* 2004;11(2):88-96.
<http://www.ncbi.nlm.nih.gov/pubmed/14706012>
50. Wheeler JS Jr, Robinson CJ, Culkin DJ, Nemchausky BA. Naloxone efficacy in bladder rehabilitation of spinal cord injury patients. *J Urol* 1987;137(6):1202-5.
<http://www.ncbi.nlm.nih.gov/pubmed/14706012>
51. Komersova K, Rogerson JW, Conway EL, Lim TC, Brown DJ, Krum H, Jackman GP, Murdoch R, Louis WJ. The effect of levromakalim (BRL 38227) on bladder function in patients with high spinal cord lesions. *Br J Clin Pharmacol* 1995;39(2):207-9.
<http://www.ncbi.nlm.nih.gov/pubmed/7742166>

52. Wyndaele JJ, van Kerrebroeck P. The effects of 4 weeks treatment with cisapride on cystometric parameters in spinal cord injury patients. A double-blind, placebo controlled study. *Paraplegia* 1995;33(11):625-7.
<http://www.ncbi.nlm.nih.gov/pubmed/8584295>
53. Costa P, Bressolle F, Sarrazin B, Mosser J, Sabatier R. Dose-related effect of moxislyte on maximal urethral closing pressure in patients with spinal cord injuries. *Clin Pharmacol Ther* 1993;53(4):443-9.
<http://www.ncbi.nlm.nih.gov/pubmed/8477560>
54. Cain MP, Wu SD, Austin PF, Herndon CD, Rink RC. Alpha blocker therapy for children with dysfunctional voiding and urinary retention. *J Urol* 2003;170(4 Pt 2):1514-5.
<http://www.ncbi.nlm.nih.gov/pubmed/14501648>
55. Schulte-Baukloh H, Michael T, Miller K, Knispel HH. Alfuzosin in the treatment of high leak-point pressure in children with neurogenic bladder. *BJU Int* 2002;90(7):716-20.
<http://www.ncbi.nlm.nih.gov/pubmed/12410754>
56. Abrams P, Amarenco G, Bakke A, Buczynski A, Castro-Diaz D, Harrison S, Kramer G, Marsik R, Prajsner A, Stöhrer M, Van Kerrebroeck P, Wyndaele JJ; European Tamsulosin Neurogenic Lower Urinary Tract Dysfunction Study Group. Tamsulosin: efficacy and safety in patients with neurogenic lower urinary tract dysfunction due to suprasacral spinal cord injury. *J Urol* 2003;170(4 Pt 1):1242-51.
<http://www.ncbi.nlm.nih.gov/pubmed/14501734>
57. Yasuda K, Yamanishi T, Kawabe K, Ohshima H, Morita T. The effect of urapidil on neurogenic bladder: a placebo controlled double-blind study. *J Urol* 1996;156(3):1125-30.
<http://www.ncbi.nlm.nih.gov/pubmed/8709324>
58. Al-Ali M, Salman G, Rasheed A, Al-Ani G, Al-Rubaiy S, Alwan A, Al-Shaikli A. Phenoxybenzamine in the management of neuropathic bladder following spinal cord injury. *Aust N Z J Surg* 1999;69(9):660-3.
<http://www.ncbi.nlm.nih.gov/pubmed/10515340>
59. Te AE. A modern rationale for the use of phenoxybenzamine in urinary tract disorders and other conditions. *Clin Ther* 2002;24(6):851-61; discussion 837.
<http://www.ncbi.nlm.nih.gov/pubmed/12117078>
60. Fall M, Lindstrom S. Electrical stimulation. A physiologic approach to the treatment of urinary incontinence. *Urol Clin North Am* 1991;18(2):393-407.
<http://www.ncbi.nlm.nih.gov/pubmed/2017820>
61. Vodusek DB, Light KJ, Libby JM. Detrusor inhibition induced by stimulation of pudendal nerve afferents. *Neurourol Urodyn* 1986;5:381-9.
62. Bemelmans BL, Mundy AR, Craggs MD. Neuromodulation by implant for treating lower urinary tract symptoms and dysfunction. *Eur Urol* 1999;36(2):81-91.
<http://www.ncbi.nlm.nih.gov/pubmed/10420026>
63. Primus G, Kramer G. Maximal external electrical stimulation for treatment of neurogenic or nonneurogenic urgency and/or urge incontinence. *Neurourol Urodyn* 1996;15(3):187-94.
<http://www.ncbi.nlm.nih.gov/pubmed/8732985>
64. Madersbacher H, Kiss G, Mair D. Transcutaneous electrostimulation of the pudendal nerve for treatment of detrusor overactivity. *Neurourol Urodyn* 1995;14:501-2.
65. Prevaire JG, Soler JM, Perrigot M. Is there a place for pudendal nerve maximal electrical stimulation for the treatment of detrusor hyperreflexia in spinal cord injury patients?. *Spinal Cord* 1998;36(2):100-3.
<http://www.ncbi.nlm.nih.gov/pubmed/9494999>
66. Amarenco G, Ismael SS, Even-Schneider A, Raibaut P, Demaille-Wlodyka S, Parratte B, Kerdraon J. Urodynamic effect of acute transcutaneous posterior tibial nerve stimulation in overactive bladder. *J Urol* 2003;169(6):2210-5.
<http://www.ncbi.nlm.nih.gov/pubmed/12771752>
67. Guttmann L, Frankel H. The value of intermittent catheterisation in the early management of traumatic paraplegia and tetraplegia. *Paraplegia* 1966;4(2):63-84.
<http://www.ncbi.nlm.nih.gov/pubmed/5969402>
68. Lapidus J, Diokno AC, Silber SJ, Lowe BS. Clean, intermittent self-catheterization in the treatment of urinary tract disease. *J Urol* 1972;107(3):458-61.
<http://www.ncbi.nlm.nih.gov/pubmed/5010715>
69. Wyndaele JJ. Intermittent catheterization: which is the optimal technique?. *Spinal Cord* 2002;44(9):432-7.
<http://www.ncbi.nlm.nih.gov/pubmed/12185603>

70. Prieto-Fingerhut T, Banovac K, Lynne CM. A study comparing sterile and nonsterile urethral catheterization in patients with spinal cord injury. *Rehabil Nurs* 1997;22(6):299-302.
<http://www.ncbi.nlm.nih.gov/pubmed/9416190>
71. Matsumoto T, Takahashi K, Manabe N, Iwatsubo E, Kawakami Y. Urinary tract infection in neurogenic bladder. *Int J Antimicrob Agents* 2001;17(4):293-7.
<http://www.ncbi.nlm.nih.gov/pubmed/11295411>
72. Hudson E, Murahata RI. The 'no-touch' method of intermittent urinary catheter insertion: can it reduce the risk of bacteria entering the bladder?. *Spinal Cord* 2005;43(10):611-4.
<http://www.ncbi.nlm.nih.gov/pubmed/15852058>
73. Waller L, Jonsson O, Norlen L, Sullivan L. Clean intermittent catheterization in spinal cord injury patients: long-term follow-up of a hydrophilic low friction technique. *J Urol* 1995;153(2):345-8.
<http://www.ncbi.nlm.nih.gov/pubmed/7815579>
74. Bakke A, Digranes A, Hoisaeter PA. Physical predictors of infection in patients treated with clean intermittent catheterization: a prospective 7-year study. *Br J Urol* 1997;79(1):85-90.
<http://www.ncbi.nlm.nih.gov/pubmed/9043503>
75. Gunther M, Lochner-Ernst D, Kramer G, Stohrer M. Auswirkungen des intermittierende aseptischen intermittierenden Katheterismus auf die männliche Harnröhre. *Urologe B* 2001;41:359-361. [article in German] [Effects of aseptic intermittent catheterisation on the male urethra]
76. Wyndaele JJ. Complications of intermittent catheterization: their prevention and treatment. *Spinal Cord* 2002;40(10):536-41.
<http://www.ncbi.nlm.nih.gov/pubmed/12235537>
77. Sauerwein D. Urinary tract infection in patients with neurogenic bladder dysfunction. *Int J Antimicrob Agents* 2002;19(6):592-7.
<http://www.ncbi.nlm.nih.gov/pubmed/12135853>
78. Sullivan LP, Davidson PG, Kloss DA, D'Anna JA Jr. Small-bowel obstruction caused by a long-term indwelling urinary catheter. *Surgery* 1990;107(2):228-30.
<http://www.ncbi.nlm.nih.gov/pubmed/2300902>
79. Chao R, Clowers D, Mayo ME. Fate of upper urinary tracts in patients with indwelling catheters after spinal cord injury. *Urology* 1993;42(3):259-62.
<http://www.ncbi.nlm.nih.gov/pubmed/8379025>
80. Chancellor MB, Erhard MJ, Kiilholma PJ, Karasick S, Rivas DA. Functional urethral closure with pubovaginal sling for destroyed female urethra after long-term urethral catheterization. *Urology* 1994;43(4):499-505.
<http://www.ncbi.nlm.nih.gov/pubmed/8154071>
81. Bennett CJ, Young MN, Adkins RH, Diaz F. Comparison of bladder management complication outcomes in female spinal cord injury patients. *J Urol* 1995;153(5):1458-60.
<http://www.ncbi.nlm.nih.gov/pubmed/7714965>
82. Larsen LD, Chamberlin DA, Khonsari F, Ahlering TE. Retrospective analysis of urologic complications in male patients with spinal cord injury managed with and without indwelling urinary catheters. *Urology* 1997;50(3):418-22.
<http://www.ncbi.nlm.nih.gov/pubmed/9301708>
83. West DA, Cummings JM, Longo WE, Virgo KS, Johnson FE, Parra RO. Role of chronic catheterization in the development of bladder cancer in patients with spinal cord injury. *Urology* 1999;53(2):292-7.
<http://www.ncbi.nlm.nih.gov/pubmed/9933042>
84. Mitsui T, Minami K, Furuno T, Morita H, Koyanagi T. Is suprapubic cystostomy an optimal urinary management in high quadriplegics? A comparative study of suprapubic cystostomy and clean intermittent catheterization. *Eur Urol* 2000;38(4):434-8.
<http://www.ncbi.nlm.nih.gov/pubmed/11025382>
85. Weld KJ, Wall BM, Mangold TA, Steere EL, Dmochowski RR. Influences on renal function in chronic spinal cord injured patients. *J Urol* 2000;164(5):1490-3.
<http://www.ncbi.nlm.nih.gov/pubmed/11025689>
86. Zermann D, Wunderlich H, Derry F, Schroder S, Schubert J. Audit of early bladder management complications after spinal cord injury in first-treating hospitals. *Eur Urol* 2000;37(2):156-60.
<http://www.ncbi.nlm.nih.gov/pubmed/10705193>
87. Park YI, Linsenmeyer TA. A method to minimize indwelling catheter calcification and bladder stones in individuals with spinal cord injury. *J Spinal Cord Med* 2001;24(2):105-8.
<http://www.ncbi.nlm.nih.gov/pubmed/11587416>
88. Glickman S, Tsokkos N, Shah PJ. Intravesical atropine and suppression of detrusor hypercontractility in the neuropathic bladder. A preliminary study. *Paraplegia* 1995;33(1):36-9.
<http://www.ncbi.nlm.nih.gov/pubmed/7715952>

89. Amark P, Bussman G, Eksborg S. Follow-up of long-time treatment with intravesical oxybutynin for neurogenic bladder in children. *Eur Urol* 1998;34(2):148-53.
<http://www.ncbi.nlm.nih.gov/pubmed/9693251>
90. Haferkamp A, Staehler G, Gerner HJ, Dorsam J. Dosage escalation of intravesical oxybutynin in the treatment of neurogenic bladder patients. *Spinal Cord* 2000;38(4):250-4.
<http://www.ncbi.nlm.nih.gov/pubmed/10822396>
91. Pannek J, Sommerfeld HJ, Botel U, Senge T. Combined intravesical and oral oxybutynin chloride in adult patients with spinal cord injury. *Urology* 2000;55(3):358-62.
<http://www.ncbi.nlm.nih.gov/pubmed/10699610>
92. Buyse G, Waldeck K, Verpoorten C, Bjork H, Casaer P, Andersson KE. Intravesical oxybutynin for neurogenic bladder dysfunction: less systemic side effects due to reduced first pass metabolism. *J Urol* 1998;160(3 Pt 1):892-6.
<http://www.ncbi.nlm.nih.gov/pubmed/9720583>
93. Riedl CR, Knoll M, Plas E, Pfluger H. Intravesical electromotive drug administration technique: preliminary results and side effects. *J Urol* 1998;159(6):1851-6.
<http://www.ncbi.nlm.nih.gov/pubmed/9598474>
94. Di Stasi SM, Giannantoni A, Navarra P, Capelli G, Storti L, Porena M, Stephen RL. Intravesical oxybutynin: mode of action assessed by passive diffusion and electromotive administration with pharmacokinetics of oxybutynin and N-desethyl oxybutynin. *J Urol* 2001;166(6):2232-6.
<http://www.ncbi.nlm.nih.gov/pubmed/11696741>
95. Geirsson G, Fall M, Sullivan L. Clinical and urodynamic effects of intravesical capsaicin treatment in patients with chronic traumatic spinal detrusor hyperreflexia. *J Urol* 1995;154(5):1825-9.
<http://www.ncbi.nlm.nih.gov/pubmed/7563356>
96. Cruz F, Guimaraes M, Silva C, Reis M. Suppression of bladder hyperreflexia by intravesical resiniferatoxin. *Lancet* 1997;350(9078):640-1.
<http://www.ncbi.nlm.nih.gov/pubmed/9288055>
97. De Ridder D, Chandiramani V, Dasgupta P, Van Poppel H, Baert L, Fowler CJ. Intravesical capsaicin as a treatment for refractory detrusor hyperreflexia: a dual center study with long-term follow-up. *J Urol* 1997;158(6):2087-92.
<http://www.ncbi.nlm.nih.gov/pubmed/9366318>
98. Wiart L, Joseph PA, Petit H, Dosque JP, de Seze M, Brochet B, Deminiere C, Ferriere JM, Mazaux JM, N'Guyen P, Barat M. The effects of capsaicin on the neurogenic hyperreflexic detrusor. A double blind placebo controlled study in patients with spinal cord disease. Preliminary results. *Spinal Cord* 1998;36(2):95-9.
<http://www.ncbi.nlm.nih.gov/pubmed/9366318>
99. Kim JH, Rivas DA, Shenot PJ, Green B, Kennelly M, Erickson JR, O'Leary M, Yoshimura N, Chancellor MB. Intravesical resiniferatoxin for refractory detrusor hyperreflexia: a multicenter, blinded, randomized, placebo-controlled trial. *J Spinal Cord Med* 2003;26(4):358-63.
<http://www.ncbi.nlm.nih.gov/pubmed/14992337>
100. Giannantoni A, Di Stasi SM, Stephen RL, Bini V, Costantini E, Porena M. Intravesical resiniferatoxin versus botulinum-A toxin injections for neurogenic detrusor overactivity: a prospective randomized study. *J Urol* 2004;172(1):240-3.
<http://www.ncbi.nlm.nih.gov/pubmed/15201783>
101. Katona F, Benyo L, Lang J. [Intraluminal electrotherapy of various paralytic conditions of the gastrointestinal tract with the quadrangular current.] *Zentralbl Chir* 1958; 84(24):929-33. [article in German]
<http://www.ncbi.nlm.nih.gov/pubmed/13676705>
102. Kaplan WE. Intravesical electrical stimulation of the bladder: pro. *Urology* 2000;56(1):2-4.
<http://www.ncbi.nlm.nih.gov/pubmed/10869607>
103. Ebner A, Jiang C, Lindstrom S. Intravesical electrical stimulation—an experimental analysis of the mechanism of action. *J Urol* 1992;148(3):920-4.
<http://www.ncbi.nlm.nih.gov/pubmed/1512860>
104. Primus G, Kramer G, Pummer K. Restoration of micturition in patients with acontractile and hypocontractile detrusor by transurethral electrical bladder stimulation. *Neurourol Urodyn* 1996;15(5):489-97.
<http://www.ncbi.nlm.nih.gov/pubmed/8857617>
105. De Wachter S, Wyndaele JJ. Quest for standardisation of electrical sensory testing in the lower urinary tract: the influence of technique related factors on bladder electrical thresholds. *Neurourol Urodyn* 2003;22(2):118-22.
<http://www.ncbi.nlm.nih.gov/pubmed/12579628>

106. Katona F, Berenyi M. Intravesical transurethral electrotherapy in meningomyelocele patients. *Acta Paediatr Acad Sci Hung* 1975;16(3-4):363-74.
<http://www.ncbi.nlm.nih.gov/pubmed/773096>
107. Hagerty JA, Richards I, Kaplan WE. Intravesical electrotherapy for neurogenic bladder dysfunction: a 22-year experience. *J Urol*. 2007;178(4 Pt 2):1680-3;discussion 1683.
<http://www.ncbi.nlm.nih.gov/pubmed/17707024>
108. Nicholas JL, Eckstein HB. Endovesical electrotherapy in treatment of urinary incontinence in spinabifida patients. *Lancet* 1975;2(7948):1276-7.
<http://www.ncbi.nlm.nih.gov/pubmed/54798>
109. Pugach JL, Salvin L, Steinhardt GF. Intravesical electrostimulation in pediatric patients with spinal cord defects. *J Urol* 2000;164(3 Pt 2):965-8.
<http://www.ncbi.nlm.nih.gov/pubmed/10958718>
110. Stohrer M, Schurch B, Kramer G, Schmid D, Gaul G, Hauri D. Botulinum-A toxin in the treatment of detrusor hyperreflexia in spinal cord injury: a new alternative to medical and surgical procedures?. *Neurourol Urodyn* 1999;18:401-2.
111. Schurch B, Schmid DM, Stohrer M. Treatment of neurogenic incontinence with botulinum toxin A (letter). *N Engl J Med* 2000;342(9):665.
<http://www.ncbi.nlm.nih.gov/pubmed/10702067>
112. Schurch B, Stohrer M, Kramer G, Schmid DM, Gaul G, Hauri D. Botulinum-A toxin for treating detrusor hyperreflexia in spinal cord injured patients: a new alternative to anticholinergic drugs? Preliminary results. *J Urol* 2000;164(3 Pt 1):692-7.
<http://www.ncbi.nlm.nih.gov/pubmed/10953127>
113. Schulte-Baukloh H, Michael T, Schobert J, Stolze T, Knispel HH. Efficacy of botulinum-A toxin in children with detrusor hyperreflexia due to myelomeningocele: preliminary results. *Urology* 2002;59(3):325-7.
<http://www.ncbi.nlm.nih.gov/pubmed/11880062>
114. Wyndaele JJ, Van Dromme SA. Muscular weakness as side effect of botulinum toxin injection for neurogenic detrusor overactivity. *Spinal Cord* 2002;40(11):599-600.
<http://www.ncbi.nlm.nih.gov/pubmed/12411968>
115. Reitz A, Stöhrer M, Kramer G, Del Popolo G, Chartier-Kastler E, Pannek J, Burgdörfer H, Göcking K, Madersbacher H, Schumacher S, Richter R, von Tobel J, Schurch B. 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(4):510-15.
<http://www.ncbi.nlm.nih.gov/pubmed/15041117>
116. Del Popolo G, Filocamo MT, Li Marzi V, Macchiarella A, Cecconi F, Lombardi G, Nicita G. Neurogenic detrusor overactivity treated with English Botulinum Toxin A: 8-year experience of one single centre. *Eur Urol* 2007 53(5):1013-9 .
<http://www.ncbi.nlm.nih.gov/pubmed/17950989>
117. Schurch B, de Sèze M, Denys P, Chartier-Kastler E, Haab F, Everaert K, Plante P, Perrouin-Verbe B, Kumar C, Fraczek S, Brin MF; Botox Detrusor Hyperreflexia Study Team. 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(1):196-200.
<http://www.ncbi.nlm.nih.gov/pubmed/15947626>
118. Akbar M, Abel R, Seyler TM, Bedke J, Haferkamp A, Gerner HJ, Möhring K. Repeated botulinum-A toxin injections in the treatment of myelodysplastic children and patients with spinal cord injuries with neurogenic bladder dysfunction. *BJU Int* 2007;100(3):639-45.
<http://www.ncbi.nlm.nih.gov/pubmed/17532858>
119. Grosse J, Kramer G, Stöhrer M. Success of repeat detrusor injections of botulinum a toxin in patients with severe neurogenic detrusor overactivity and incontinence. *Eur Urol* 2005;47(5):653-9.
<http://www.ncbi.nlm.nih.gov/pubmed/15826758>
120. Haferkamp A, Schurch B, Reitz A, Krengel U, Grosse J, Kramer G, Schumacher S, Bastian PJ, Büttner R, Müller SC, Stöhrer M. Lack of ultrastructural detrusor changes following endoscopic injection of botulinum toxin type a in overactive neurogenic bladder. *Eur Urol* 2004;46(6):784-91.
<http://www.ncbi.nlm.nih.gov/pubmed/15548448>
121. Dykstra DD, Sidi AA. Related treatment of detrusor-sphincter dyssynergia with botulinum A toxin: a double-blind study. *Arch Phys Med Rehabil* 1990;71(1):24-6.
<http://www.ncbi.nlm.nih.gov/pubmed/2297305>

122. Schurch B, Hauri D, Rodic B, Curt A, Meyer M, Rossier AB. Botulinum-A toxin as a treatment of detrusor-sphincter dyssynergia: a prospective study in 24 spinal cord injury patients. *J Urol* 1996;155(3):1023-9.
<http://www.ncbi.nlm.nih.gov/pubmed/8583552>
123. Petit H, Wiart L, Gaujard E, Le Breton F, Ferriere JM, Lagueny A, Joseph PA, Barat M. Botulinum A toxin treatment for detrusor-sphincter dyssynergia in spinal cord disease. *Spinal Cord* 1998;36(2): 91-4.
<http://www.ncbi.nlm.nih.gov/pubmed/9494997>
124. Chancellor MB, Rivas DA, Abdill CK, Karasick S, Ehrlich SM, Staas WE. Prospective comparison of external sphincter balloon dilatation and prosthesis placement with external sphincterotomy in spinal cord injured men. *Arch Phys Med Rehabil* 1994;75(3):297-305.
<http://www.ncbi.nlm.nih.gov/pubmed/8129583>
125. Whitmore WF 3rd, Fam BA, Yalla SV. Experience with anteromedian (12 o'clock) external urethral sphincterotomy in 100 male subjects with neuropathic bladders. *Br J Urol* 1978;50(2):99-101.
<http://www.ncbi.nlm.nih.gov/pubmed/754859>
126. Perakash 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(1):33-8.
<http://www.ncbi.nlm.nih.gov/pubmed/9728128>
127. Noll F, Sauerwein D, Stohrer M. Transurethral sphincterotomy in quadriplegic patients: long-term follow-up. *Neurourol Urodyn* 1995;14(4):351-8.
<http://www.ncbi.nlm.nih.gov/pubmed/7581471>
128. Reynard JM, Vass J, Sullivan ME, Mamas M. Sphincterotomy and the treatment of detrusor-sphincter dyssynergia: current status, future prospects. *Spinal Cord* 2003;41(1):1-11.
<http://www.ncbi.nlm.nih.gov/pubmed/12494314>
129. Derry F, al-Rubeyi S. Audit of bladder neck resection in spinal cord injured patients. *Spinal Cord* 1998;36(5):345-8.
<http://www.ncbi.nlm.nih.gov/pubmed/9601115>
130. Chancellor MB, Gajewski J, Ackman CF, Appell RA, Bennett J, Binard J, Boone TB, Chetner MP, Crewalk JA, Defalco A, Foote J, Green B, Juma S, Jung SY, Linsenmeyer TA, MacMillan R, Mayo M, Ozawa H, Roehrborn CG, Shenot PJ, Stone A, Vazquez A, Killorin W, Rivas DA. Long-term follow-up of the North American multicenter UroLume trial for the treatment of external detrusor-sphincter dyssynergia. *J Urol* 1999;161(5):1545-50.
<http://www.ncbi.nlm.nih.gov/pubmed/10210393>
131. Seoane-Rodríguez S, Sánchez R-Losada J, Montoto-Marqués A, Salvador-de la Barrera S, Ferreiro-Velasco ME, Alvarez-Castelo L, Balsa-Mosquera B, Rodríguez-Sotillo A. Long-term follow-up study of intraurethral stents in spinal cord injured patients with detrusor-sphincter dyssynergia. *Spinal Cord* 2007;45(9):621-6.
<http://www.ncbi.nlm.nih.gov/pubmed/17211463>
132. Gajewski JB, Chancellor MB, Ackman CF, Appell RA, Bennett J, Binard J, Boone TB, Chetner MP, Crewalk JA, Defalco A, Foote J, Green B, Juma S, Jung SY, Linsenmeyer TA, Macaluso JN Jr, Macmillan R, Mayo M, Ozawa H, Roehrborn CG, Schmidt J, Shenot PJ, Stone A, Vazquez A, Killorin W, Rivas DA. Removal of UroLume endoprosthesis: experience of the North American Study Group for detrusor-sphincter dyssynergia application. *J Urol* 2000;163(3):773-6.
<http://www.ncbi.nlm.nih.gov/pubmed/10687974>
133. Wilson TS, Lemack GE, Dmochowski RR. UroLume stents: lessons learned. *J Urol* 2002;167(6): 2477-80.
<http://www.ncbi.nlm.nih.gov/pubmed/11992061>
134. Bennett JK, Green BG, Foote JE, Gray M. Collagen injections for intrinsic sphincter deficiency in the neuropathic urethra. *Paraplegia* 1995;33(12):697-700.
<http://www.ncbi.nlm.nih.gov/pubmed/8927407>
135. Guys JM, Simeoni-Alias J, Fakhro A, Delarue A. Use of polydimethylsiloxane for endoscopic treatment of neurogenic urinary incontinence in children. *J Urol* 1999;162(6):2133-5.
<http://www.ncbi.nlm.nih.gov/pubmed/10569603>
136. Kassouf W, Capolicchio G, Berardinucci G, Corcos J. Collagen injection for treatment of urinary incontinence in children. *J Urol* 2001;165(5):1666-8.
<http://www.ncbi.nlm.nih.gov/pubmed/11342951>
137. Caione P, Capozza N. Endoscopic treatment of urinary incontinence in pediatric patients: 2-year experience with dextranomer/hyaluronic acid copolymer. *J Urol* 2002;168(4 Pt 2):1868-71.
<http://www.ncbi.nlm.nih.gov/pubmed/12352378>

138. Block CA, Cooper CS, Hawtrey CE. Long-term efficacy of periurethral collagen injection for the treatment of urinary incontinence secondary to myelomeningocele. *J Urol* 2003;169(1):327-9.
<http://www.ncbi.nlm.nih.gov/pubmed/12478183>
139. Schurch B, Suter S, Dubs M. Intraurethral sphincter prosthesis to treat hyporeflexic bladders in women: does it work? *BJU Int* 1999;84(7):789-94.
<http://www.ncbi.nlm.nih.gov/pubmed/10532973>
140. Herschorn S, Radomski SB. Fascial slings and bladder neck tapering in the treatment of male neurogenic incontinence. *J Urol* 1992;147(4):1073-5.
<http://www.ncbi.nlm.nih.gov/pubmed/1552586>
141. Gormley EA, Bloom DA, McGuire EJ, Ritchey ML. Pubovaginal slings for the management of urinary incontinence in female adolescents. *J Urol* 1994;152(2 Pt 2):822-5.
<http://www.ncbi.nlm.nih.gov/pubmed/8022024>
142. Kakizaki H, Shibata T, Shinno Y, Kobayashi S, Matsumura K, Koyanagi T. Fascial sling for the management of urinary incontinence due to sphincter incompetence. *J Urol* 1995;153(3 Pt 1):644-7.
<http://www.ncbi.nlm.nih.gov/pubmed/7861504>
143. Gosalbez R, Castellan M. Defining the role of the bladder-neck sling in the surgical treatment of urinary incontinence in children with neurogenic incontinence. *World J Urol* 1998;16(4):285-91.
<http://www.ncbi.nlm.nih.gov/pubmed/9775429>
144. Barthold JS, Rodriguez E, Freedman AL, Fleming PA, Gonzalez R. Results of the rectus fascial sling and wrap procedures for the treatment of neurogenic sphincteric incontinence. *J Urol* 1999;161(1):272-4.
<http://www.ncbi.nlm.nih.gov/pubmed/10037423>
145. Dik P, Van Gool JD, De Jong TP. Urinary continence and erectile function after bladder neck sling suspension in male patients with spinal dysraphism. *BJU Int* 1999;83(9):971-5.
<http://www.ncbi.nlm.nih.gov/pubmed/10368238>
146. Kryger JV, Gonzalez R, Barthold JS. Surgical management of urinary incontinence in children with neurogenic sphincteric incompetence. *J Urol* 2000;163(1):256-63.
<http://www.ncbi.nlm.nih.gov/pubmed/10604371>
147. Walker RD, Erhard M, Starling J. Long-term evaluation of rectus fascial wrap in patients with spina bifida. *J Urol* 2000;164(2):485-6.
<http://www.ncbi.nlm.nih.gov/pubmed/10893629>
148. Kapoor R, Dubey D, Kumar A, Zaman W. Modified bulbar urethral sling procedure for the treatment of male sphincteric incontinence. *J Endourol* 2001;15(5):545-9.
<http://www.ncbi.nlm.nih.gov/pubmed/11465337>
149. Nguyen HT, Bauer SB, Diamond DA, Retik AB. Rectus fascial sling for the treatment of neurogenic sphincteric incontinence in boys: is it safe and effective?. *J Urol* 2001;166(2):658-61.
<http://www.ncbi.nlm.nih.gov/pubmed/11458113>
150. Austin PF, Westney OL, Leng WW, McGuire EJ, Ritchey ML. Advantages of rectus fascial slings for urinary incontinence in children with neuropathic bladders. *J Urol* 2001;165(6 Pt 2):2369-71.
<http://www.ncbi.nlm.nih.gov/pubmed/11398778>
151. Mingin GC, Youngren K, Stock JA, Hanna MK. The rectus myofascial wrap in the management of urethral sphincter incompetence. *BJU Int* 2002;90(6):550-3.
<http://www.ncbi.nlm.nih.gov/pubmed/12230615>
152. Colvert JR 3rd, Kropp BP, Cheng EY, Pope JC 4th, Brock JW 3rd, Adams MC, Austin P, Furness PD 3rd, Koyle MA. The use of small intestinal submucosa as an off-the-shelf urethral sling material for pediatric urinary incontinence. *J Urol* 2002;168(4 Pt 2):1872-5.
<http://www.ncbi.nlm.nih.gov/pubmed/12352379>
153. Daneshmand S, Ginsberg DA, Bennet JK, Foote J, Killorin W, Rozas KP, Green BG. Puboprostatic sling repair for treatment of urethral incompetence in adult neurogenic incontinence. *J Urol* 2003;169(1):199-202.
<http://www.ncbi.nlm.nih.gov/pubmed/12478135>
154. Light JK, Scott FB. Use of the artificial urinary sphincter in spinal cord injury patients. *J Urol* 1983;130(6):1127-9.
<http://www.ncbi.nlm.nih.gov/pubmed/6644893>
155. Sidi AA, Reinberg Y, Gonzalez R. Comparison of artificial sphincter implantation and bladder neck reconstruction in patients with neurogenic urinary incontinence. *J Urol* 1987;138(4 Pt 2):1120-2.
<http://www.ncbi.nlm.nih.gov/pubmed/3656572>
156. Fulford SC, Sutton C, Bales G, Hickling M, Stephenson TP. The fate of the 'modern' artificial urinary sphincter with a follow-up of more than 10 years. *Br J Urol* 1997;79(5):713-6.
<http://www.ncbi.nlm.nih.gov/pubmed/9158507>

157. Elliott DS, Barrett DM. Mayo Clinic long-term analysis of the functional durability of the AMS 800 artificial urinary sphincter: a review of 323 cases. *J Urol* 1998;159(4):1206-8.
<http://www.ncbi.nlm.nih.gov/pubmed/9507835>
158. Castera R, Podesta ML, Ruarte A, Herrera M, Medel R. 10-Year experience with artificial urinary sphincter in children and adolescents. *J Urol* 2001;165(6 Pt 2):2373-6.
<http://www.ncbi.nlm.nih.gov/pubmed/11371980>
159. Kryger JV, Levenson G, Gonzalez R. Long-term results of artificial urinary sphincters in children are independent of age at implantation. *J Urol* 2001;165(6 Pt 2):2377-9.
<http://www.ncbi.nlm.nih.gov/pubmed/11371981>
160. Janknegt RA, Baeten CG, Weil EH, Spaans F. Electrically stimulated gracilis sphincter for treatment of bladder sphincter incontinence. *Lancet* 1992;340(8828):1129-30.
<http://www.ncbi.nlm.nih.gov/pubmed/1359213>
161. Chancellor MB, Heesakkers JP, Janknegt RA. Gracilis muscle transposition with electrical stimulation for sphincteric incontinence: a new approach. *World J Urol* 1997;15(5):320-8.
<http://www.ncbi.nlm.nih.gov/pubmed/9372585>
162. Donnhoo KK, Rink RC, Cain MP, Casale AJ. The Young-Dees-Leadbetter bladder neck repair for neurogenic incontinence. *J Urol* 1999;161(6):1946-9.
<http://www.ncbi.nlm.nih.gov/pubmed/10332478>
163. Kropp KA, Angwafo FF. Urethral lengthening and reimplantation for neurogenic incontinence in children. *J Urol* 1986;135(3):533-6.
<http://www.ncbi.nlm.nih.gov/pubmed/3944902>
164. Salle JL, McLorie GA, Bagli DJ, Khoury AE. Urethral lengthening with anterior bladder wall flap (Pippi Salle procedure): modifications and extended indications of the technique. *J Urol* 1997;158(2):585-90.
<http://www.ncbi.nlm.nih.gov/pubmed/9224369>
165. Mollard P, Mouriquand P, Joubert P. Urethral lengthening for neurogenic urinary incontinence (Kropp's procedure): results of 16 cases. *J Urol* 1990;143(1):95-7.
<http://www.ncbi.nlm.nih.gov/pubmed/2294274>
166. Nill TG, Peller PA, Kropp KA. Management of urinary incontinence by bladder tube urethral lengthening and submucosal reimplantation. *J Urol* 1990;144(2 Pt 2):559-61.
<http://www.ncbi.nlm.nih.gov/pubmed/2374240>
167. Rink RC, Adams MC, Keating MA. The flip-flap technique to lengthen the urethra (Salle procedure) for treatment of neurogenic urinary incontinence. *J Urol* 1994;152(2 Pt 2):799-802.
<http://www.ncbi.nlm.nih.gov/pubmed/8022018>
168. Waters PR, Chehade NC, Kropp KA. Urethral lengthening and reimplantation: incidence and management of catheterization problems. *J Urol* 1997;158(3 Pt 2):1053-6.
<http://www.ncbi.nlm.nih.gov/pubmed/9258141>
169. Diamond DA, Bauer SB, Dinlenc C, Hendren WH, Peters CA, Atala A, Kelly M, Retik AB. Normal urodynamics in patients with bladder exstrophy: are they achievable?. *J Urol* 1999;162(3 Pt 1):841-4.
<http://www.ncbi.nlm.nih.gov/pubmed/10458392>
170. Hayes MC, Bulusu A, Terry T, Mouriquand PD, Malone PS. The Pippi Salle urethral lengthening procedure; experience and outcome from three United Kingdom centres. *BJU Int* 1999;84(6):701-5.
<http://www.ncbi.nlm.nih.gov/pubmed/10510119>
171. Yerkes EB, Adams MC, Rink RC, Pope JC IV, Brock JW 3rd. How well do patients with exstrophy actually void? *J Urol* 2000;164(3 Pt 2):1044-7.
<http://www.ncbi.nlm.nih.gov/pubmed/10958737>
172. Surer I, Baker LA, Jeffs RD, Gearhart JP. Modified Young-Dees-Leadbetter bladder neck reconstruction in patients with successful primary bladder closure elsewhere: a single institution experience. *J Urol* 2001;165(6 Pt 2):2438-40.
<http://www.ncbi.nlm.nih.gov/pubmed/11371993>
173. Chan DY, Jeffs RD, Gearhart JP. Determinants of continence in the bladder exstrophy population: predictors of success? *Urology* 2001;57(4):774-7.
<http://www.ncbi.nlm.nih.gov/pubmed/11306402>
174. Ferrer FA, Tadros YE, Gearhart J. Modified Young-Dees-Leadbetter bladder neck reconstruction: new concepts about old ideas. *Urology* 2001;58(5):791-6.
<http://www.ncbi.nlm.nih.gov/pubmed/11711366>
175. Couvelaire R. [Bladder surgery]. Paris: Masson, 1955. [article in French]
176. Cartwright PC, Snow BW. Bladder autoaugmentation: early clinical experience. *J Urol* 1989;142(2 Pt 2):505-8.
<http://www.ncbi.nlm.nih.gov/pubmed/2746767>

177. Stohrer M, Kramer A, Goepel M, Lochner-Ernst D, Kruse D, Rubben H. Bladder auto-augmentation - an alternative for enterocystoplasty: preliminary results. *Neurourol Urodyn* 1995;14(1):11-23.
<http://www.ncbi.nlm.nih.gov/pubmed/7742844>
178. Elder JS. Autoaugmentation gastrocystoplasty: early clinical results. *J Urol* 1995;154(1):322-3.
<http://www.ncbi.nlm.nih.gov/pubmed/7776450>
179. Poppas DP, Uzzo RG, Britanisky RG, Mininberg DT. Laparoscopic laser assisted auto-augmentation of the pediatric neurogenic bladder: early experience with urodynamic follow-up. *J Urol* 1996;155(5):1057-60.
<http://www.ncbi.nlm.nih.gov/pubmed/8583564>
180. Snow BW, Cartwright PC. Bladder autoaugmentation. *Urol Clin North Am* 1996;23(2):323-31.
<http://www.ncbi.nlm.nih.gov/pubmed/8659030>
181. Stohrer M, Kramer G, Goepel M, Lochner-Ernst D, Kruse D, Rubben H. Bladder autoaugmentation in adult patients with neurogenic voiding dysfunction. *Spinal Cord* 1997;35(7):456-62.
<http://www.ncbi.nlm.nih.gov/pubmed/9232751>
182. Duel BP, Gonzalez R, Barthold JS. Alternative techniques for augmentation cystoplasty. *J Urol* 1998;159(3):998-1005.
<http://www.ncbi.nlm.nih.gov/pubmed/9474216>
183. Braren V, Bishop MR. Laparoscopic bladder autoaugmentation in children. *Urol Clin North Am* 1998;25(3):533-40.
<http://www.ncbi.nlm.nih.gov/pubmed/9728222>
184. Chapple CR, Bryan NP. Surgery for detrusor overactivity. *World J Urol* 1998;16(4):268-73.
<http://www.ncbi.nlm.nih.gov/pubmed/9775426>
185. Leng WW, Blalock HJ, Fredriksson WH, English SF, McGuire EJ. Enterocystoplasty or detrusor myectomy? Comparison of indications and outcomes for bladder augmentation. *J Urol* 1999;161(3):758-63.
<http://www.ncbi.nlm.nih.gov/pubmed/10022679>
186. Comer MT, Thomas DF, Trejosiewicz LK, Southgate J. Reconstruction of the urinary bladder by autoaugmentation, enterocystoplasty, and composite enterocystoplasty. *Adv Exp Med Biol* 1999; 462:43-7.
<http://www.ncbi.nlm.nih.gov/pubmed/10599412>
187. Siracusano S, Trombetta C, Liguori G, De Giorgi G, d'Aloia G, Di Benedetto P, Belgrano E. Laparoscopic bladder auto-augmentation in an incomplete traumatic spinal cord injury. *Spinal Cord* 2000;38(1):59-61.
<http://www.ncbi.nlm.nih.gov/pubmed/10762200>
188. Oge O, Tekgul S, Ergen A, Kendi S. Urothelium-preserving augmentation cystoplasty covered with a peritoneal flap. *BJU Int* 2000;85(7):802-5.
<http://www.ncbi.nlm.nih.gov/pubmed/10792156>
189. Cranidis A, Nestoridis G. Bladder augmentation. *Int Urogynecol J Pelvic Floor Dysfunct* 2000; 11(1):33-40.
<http://www.ncbi.nlm.nih.gov/pubmed/10738932>
190. Niknejad KG, Atala A. Bladder augmentation techniques in women. *Int Urogynecol J Pelvic Floor Dysfunct* 2000;11(3):156-69.
<http://www.ncbi.nlm.nih.gov/pubmed/11484743>
191. Westney OL, McGuire EJ. Surgical procedures for the treatment of urge incontinence. *Tech Urol* 2001;7(2):126-32.
<http://www.ncbi.nlm.nih.gov/pubmed/11383990>
192. Perovic SV, Djordjevic ML, Kekic ZK, Vukadinovic VM. Bladder autoaugmentation with rectus muscle backing. *J Urol* 2002;168(4 Pt 2):1877-80.
<http://www.ncbi.nlm.nih.gov/pubmed/12352380>
193. Marte A, Di Meglio D, Cotrufo AM, Di Iorio G, De Pasquale M, Vessella A. A long-term follow-up of autoaugmentation in myelodysplastic children. *BJU Int* 2002;89(9):928-31.
<http://www.ncbi.nlm.nih.gov/pubmed/12010242>
194. Ter Meulen PH, Heesakkers JP, Janknegt RA. A study on the feasibility of vesicomyotomy in patients with motor urge incontinence. *Eur Urol* 1997;32(2):166-9.
<http://www.ncbi.nlm.nih.gov/pubmed/9286647>
195. Potter JM, Duffy PG, Gordon EM, Malone PR. Detrusor myotomy: a 5-year review in unstable and non-compliant bladders. *BJU Int* 2002;89(9):932-5.
<http://www.ncbi.nlm.nih.gov/pubmed/12010243>

196. Nagib A, Leal J, Voris HC. Successful control of selective anterior sacral rhizotomy for treatment of spastic bladder and ureteric reflux in paraplegics. *Med Serv J Can* 1966;22(7):576-81.
<http://www.ncbi.nlm.nih.gov/pubmed/5966992>
197. Young B, Mulcahy JJ. Percutaneous sacral rhizotomy for neurogenic detrusor hyperreflexia. *J Neurosurg* 1980;53(1):85-7.
<http://www.ncbi.nlm.nih.gov/pubmed/7411212>
198. Franco I, Storrs B, Firlit CF, Zebold K, Richards I, Kaplan WE. Selective sacral rhizotomy in children with high pressure neurogenic bladders: preliminary results. *J Urol* 1992;148(2 Pt 2):648-50.
<http://www.ncbi.nlm.nih.gov/pubmed/1640538>
199. Schneidau T, Franco I, Zebold K, Kaplan W. Selective sacral rhizotomy for the management of neurogenic bladders in spina bifida patients: long-term follow-up. *J Urol* 1995;154(2 Pt 2):766-8.
<http://www.ncbi.nlm.nih.gov/pubmed/7609174>
200. Hohenfellner M, Pannek J, Botel U, Dahms S, Pfitzenmaier J, Fichtner J, Hutschenreiter G, Thuroff JW. Sacral bladder denervation for treatment of detrusor hyperreflexia and autonomic dysreflexia. *Urology* 2001;58(1):28-32.
<http://www.ncbi.nlm.nih.gov/pubmed/11445474>
201. MacDonagh RP, Forster DM, Thomas DG. Urinary continence in spinal injury patients following complete sacral posterior rhizotomy. *Br J Urol* 1990;66(6):618-22.
<http://www.ncbi.nlm.nih.gov/pubmed/2265335>
202. Sauerwein D, Ingunza W, Fischer J, Madersbacher H, Polkey CE, Brindley GS, Colombel P, Teddy P. Extradural implantation of sacral anterior root stimulators. *J Neurol Neurosurg Psychiatry* 1990; 53(8):681-4.
<http://www.ncbi.nlm.nih.gov/pubmed/2213045>
203. Koldewijn EL, Van Kerrebroeck PE, Rosier PF, Wijkstra H, Debruyne FM. Bladder compliance after posterior sacral root rhizotomies and anterior sacral root stimulation. *J Urol* 1994;151(4):955-60.
<http://www.ncbi.nlm.nih.gov/pubmed/8126835>
204. Singh G, Thomas DG. Intravesical oxybutinin in patients with posterior rhizotomies and sacral anterior root stimulators. *NeuroUrol Urodyn* 1995;14(1):65-71.
<http://www.ncbi.nlm.nih.gov/pubmed/7742851>
205. Van Kerrebroeck PE, Koldewijn EL, Rosier PF, Wijkstra H, Debruyne FM. 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(4):1378-81.
<http://www.ncbi.nlm.nih.gov/pubmed/8632580>
206. Schurch B, Rodic B, Jeanmonod D. Posterior sacral rhizotomy and intradural anterior sacral root stimulation for treatment of the spastic bladder in spinal cord injured patients. *J Urol* 1997;157(2): 610-4.
<http://www.ncbi.nlm.nih.gov/pubmed/8996369>
207. Van Kerrebroeck EV, van der Aa HE, Bosch JL, Koldewijn EL, Vorsteveld JH, Debruyne FM. Sacral rhizotomies and electrical bladder stimulation in spinal cord injury. Part I: Clinical and urodynamic analysis. Dutch Study Group on Sacral Anterior Root Stimulation. *Eur Urol* 1997;31(3):263-71.
<http://www.ncbi.nlm.nih.gov/pubmed/9129914>
208. Schumacher S, Bross S, Scheepe JR, Alken P, Junemann KP. Restoration of bladder function in spastic neuropathic bladder using sacral deafferentation and different techniques of neurostimulation. *Adv Exp Med Biol* 1999;462:303-9.
<http://www.ncbi.nlm.nih.gov/pubmed/10599434>
209. Van der Aa HE, Alleman E, Nene A, Snoek G. Sacral anterior root stimulation for bladder control: clinical results. *Arch Physiol Biochem* 1999;107(3):248-56.
<http://www.ncbi.nlm.nih.gov/pubmed/10650355>
210. Everaert K, Derie A, Van Laere M, Vandekerckhove T. Bilateral S3 nerve stimulation, a minimally invasive alternative treatment for postoperative stress incontinence after implantation of an anterior root stimulator with posterior rhizotomy: a preliminary observation. *Spinal Cord* 2000;38(4):262-4.
<http://www.ncbi.nlm.nih.gov/pubmed/10822398>
211. Creasey GH, Grill JH, Korsten M, U HS, Betz R, Anderson R, Walter J; Implanted Neuroprosthesis Research Group. An implantable neuroprosthesis for restoring bladder and bowel control to patients with spinal cord injuries: a multicenter trial. *Arch Phys Med Rehabil* 2001;82(11):1512-9.
<http://www.ncbi.nlm.nih.gov/pubmed/11689969>
212. Vignes JR, Liguoro D, Sesay M, Barat M, Guerin J. Dorsal rhizotomy with anterior sacral root stimulation for neurogenic bladder. *Stereotact Funct Neurosurg* 2001;76(3-4):243-5.
<http://www.ncbi.nlm.nih.gov/pubmed/12378103>

213. Schumacher S, Bross S, Scheepe JR, Seif C, Junemann KP, Alken P. Extradural cold block for selective neurostimulation of the bladder: development of a new technique. *J Urol* 1999;161(3): 950-4.
<http://www.ncbi.nlm.nih.gov/pubmed/10022732>
214. Kirkham AP, Knight SL, Craggs MD, Casey AT, Shah PJ. Neuromodulation through sacral nerve roots 2 to 4 with a Finetech-Brindley sacral posterior and anterior root stimulator. *Spinal Cord* 2002;40(6): 272-81.
<http://www.ncbi.nlm.nih.gov/pubmed/12037708>
215. Bhadra N, Grunewald V, Creasey G, Mortimer JT. Selective suppression of sphincter activation during sacral anterior nerve root stimulation. *Neurourol Urodyn* 2002;21(1):55-64.
<http://www.ncbi.nlm.nih.gov/pubmed/11835425>
216. Brindley GS. An implant to empty the bladder or close the urethra. *J Neurol Neurosurg Psychiatry* 1977;40(4):358-69.
<http://www.ncbi.nlm.nih.gov/pubmed/406364>
217. Schmidt RA, Tanagho EA. Feasibility of controlled micturition through electric stimulation. *Urol Int* 1979;34(3):199-230.
<http://www.ncbi.nlm.nih.gov/pubmed/382559>
218. Braun PM, Baezner H, Seif C, Boehler G, Bross S, Eschenfelder CC, Alken P, Hennerici M, Juenemann P. Alterations of cortical electrical activity in patients with sacral neuromodulator. *Eur Urol* 2002;41(5):562-6.
<http://www.ncbi.nlm.nih.gov/pubmed/12074800>
219. Ruud Bosch JL, Groen J. Treatment of refractory urge urinary incontinence with sacral spinal nerve stimulation in multiple sclerosis patients. *Lancet* 1996;348(9029):717-9.
<http://www.ncbi.nlm.nih.gov/pubmed/8806291>
220. Bosch JL, Groen J. Neuromodulation: urodynamic effects of sacral (S3) spinal nerve stimulation in patients with detrusor instability or detrusor hyperreflexia. *Behav Brain Res* 1998;92(2):141-50.
<http://www.ncbi.nlm.nih.gov/pubmed/9638956>
221. Chartier-Kastler EJ, Ruud Bosch JL, Perrigot M, Chancellor MB, Richard F, Denys P. Long-term results of sacral nerve stimulation (S3) for the treatment of neurogenic refractory urge incontinence related to detrusor hyperreflexia. *J Urol* 2000;164(5):1476-80.
<http://www.ncbi.nlm.nih.gov/pubmed/11025686>
222. Groen J, van Mastrigt R, Bosch JL. Computerized assessment of detrusor instability in patients treated with sacral neuromodulation. *J Urol* 2001;165(1):169-73.
<http://www.ncbi.nlm.nih.gov/pubmed/11125389>
223. Hohenfellner M, Humke J, Hampel C, Dahms S, Matzel K, Roth S, Thuroff JW, Schultz-Lampel D. Chronic sacral neuromodulation for treatment of neurogenic bladder dysfunction: long-term results with unilateral implants. *Urology* 2001;58(6):887-92.
<http://www.ncbi.nlm.nih.gov/pubmed/11744452>
224. Haugland M, Sinkjaer T. Interfacing the body's own sensing receptors into neural prosthesis devices. *Technol Health Care* 1999;7(6):393-9.
<http://www.ncbi.nlm.nih.gov/pubmed/10665672>
225. Zhang YH, Shao QA, Wang JM. Enveloping the bladder with displacement of flap of the rectus abdominis muscle for the treatment of neurogenic bladder. *J Urol* 1990;144(5):1194-5.
<http://www.ncbi.nlm.nih.gov/pubmed/2146404>
226. Stenzl A, Ninkovic M, Kolle D, Knapp R, Anderl H, Bartsch G. Restoration of voluntary emptying of the bladder by transplantation of innervated free skeletal muscle. *Lancet* 1998;351(9114):1483-5.
<http://www.ncbi.nlm.nih.gov/pubmed/9605805>
227. Vajda P, Kaiser L, Magyarlaki T, Farkas A, Vastyan AM, Pinter AB. Histological findings after colocolostomy and gastrocystostomy. *J Urol* 2002;168(2):698-701.
<http://www.ncbi.nlm.nih.gov/pubmed/12131353>
228. Greenwell TJ, Venn SN, Mundy AR. Augmentation cystoplasty. *BJU Int* 2001;88(6):511-25.
<http://www.ncbi.nlm.nih.gov/pubmed/11678743>
229. Gough DC. Enterocystoplasty. *BJU Int* 2001;88(7):739-43.
<http://www.ncbi.nlm.nih.gov/pubmed/11890246>
230. Quek ML, Ginsberg DA. Long-term urodynamics followup of bladder augmentation for neurogenic bladder. *J Urol* 2003;169(1):195-8.
<http://www.ncbi.nlm.nih.gov/pubmed/12478134>
231. Chartier-Kastler EJ, Mongiat-Artus P, Bitker MO, Chancellor MB, Richard F, Denys P. Long-term results of augmentation cystoplasty in spinal cord injury patients. *Spinal Cord* 2000;38(8):490-4.
<http://www.ncbi.nlm.nih.gov/pubmed/10962609>

232. Piechota HJ, Dahms SE, Probst M, Gleason CA, Nunes LS, Dahiya R, Lue TF, Tanagho EA. Functional rat bladder regeneration through xenotransplantation of the bladder acellular matrix graft. *Br J Urol* 1998;81(4):548-59.
<http://www.ncbi.nlm.nih.gov/pubmed/9598626>
233. Sievert KD, Tanagho EA. Organ-specific acellular matrix for reconstruction of the urinary tract. *World J Urol* 2000;18(1):19-25.
<http://www.ncbi.nlm.nih.gov/pubmed/10766039>
234. Kropp BP, Cheng EY. Bioengineering organs using small intestinal submucosa scaffolds: in vivo tissue-engineering technology. *J Endourol* 2000;14(1):59-62.
<http://www.ncbi.nlm.nih.gov/pubmed/10735574>
235. Liatsikos EN, Dinlenc CZ, Kapoor R, Bernardo NO, Smith AD. Tissue expansion: a promising trend for reconstruction in urology. *J Endourol* 2000;14(1):93-6.
<http://www.ncbi.nlm.nih.gov/pubmed/10735578>
236. Reddy PP, Barrieras DJ, Wilson G, Bagli DJ, McLorie GA, Khoury AE, Merguerian PA. Regeneration of functional bladder substitutes using large segment acellular matrix allografts in a porcine model. *J Urol* 2000;164(3 Pt 2):936-41.
<http://www.ncbi.nlm.nih.gov/pubmed/10958712>
237. Kawai K, Hattori K, Akaza H. Tissue-engineered artificial urothelium. *World J Surg* 2000;24(10):1160-2.
<http://www.ncbi.nlm.nih.gov/pubmed/11071451>
238. O'Donnell WF. Urological management in the patient with acute spinal cord injury. *Crit Care Clin* 1987;3(3):599-617.
<http://www.ncbi.nlm.nih.gov/pubmed/3332216>
239. Bennett JK, Gray M, Green BG, Foote JE. Continent diversion and bladder augmentation in spinal cord-injured patients. *Semin Urol* 1992;10(2):121-32.
<http://www.ncbi.nlm.nih.gov/pubmed/1636071>
240. Robertson CN, King LR. Bladder substitution in children. *Urol Clin North Am* 1986;13(2):333-44.
<http://www.ncbi.nlm.nih.gov/pubmed/3515729>
241. Duckett JW, Lotfi AH. Appendicovesicostomy (and variations) in bladder reconstruction. *J Urol* 1993;149(3):567-9.
<http://www.ncbi.nlm.nih.gov/pubmed/8437267>
242. Moreno JG, Chancellor MB, Karasick S, King S, Abdill CK, Rivas DA. Improved quality of life and sexuality with continent urinary diversion in quadriplegic women with umbilical stoma. *Arch Phys Med Rehabil* 1995;76(8):758-62.
<http://www.ncbi.nlm.nih.gov/pubmed/7632132>
243. Mollard P, Gauriau L, Bonnet JP, Mure PY. Continent cystostomy (Mitrofanoff's procedure) for neurogenic bladder in children and adolescent (56 cases: long-term results). *Eur J Pediatr Surg* 1997;7(1):34-7.
<http://www.ncbi.nlm.nih.gov/pubmed/9085806>
244. Sylora JA, Gonzalez R, Vaughn M, Reinberg Y. Intermittent self-catheterization by quadriplegic patients via a catheterizable Mitrofanoff channel. *J Urol* 1997;157(1):48-50.
<http://www.ncbi.nlm.nih.gov/pubmed/8976213>
245. Cain MP, Casale AJ, King SJ, Rink RC. Appendicovesicostomy and newer alternatives for the Mitrofanoff procedure: results in the last 100 patients at Riley Children's Hospital. *J Urol* 1999;162(5):1749-52.
<http://www.ncbi.nlm.nih.gov/pubmed/10524929>
246. Stein R, Fisch M, Ermert A, Schwarz M, Black P, Filipas D, Hohenfellner R. Urinary diversion and orthotopic bladder substitution in children and young adults with neurogenic bladder: a safe option for treatment?. *J Urol* 2000;163(2):568-73.
<http://www.ncbi.nlm.nih.gov/pubmed/10647686>
247. Liard A, Segulier-Lipszyc E, Mathiot A, Mitrofanoff P. The Mitrofanoff procedure: 20 years later. *J Urol* 2001;165(6 Pt 2):2394-8.
<http://www.ncbi.nlm.nih.gov/pubmed/11371985>
248. Kajbafzadeh AM, Chubak N. Simultaneous Malone antegrade continent enema and Mitrofanoff principle using the divided appendix: report of a new technique for prevention of stoma complications. *J Urol* 2001;165(6 Pt 2):2404-9.
<http://www.ncbi.nlm.nih.gov/pubmed/11371987>
249. Van Savage JG, Yepuri JN. Transverse retubularized sigmoidovesicostomy continent urinary diversion to the umbilicus. *J Urol* 2001;166(2):644-7.
<http://www.ncbi.nlm.nih.gov/pubmed/11458110>

250. Clark T, Pope JC 4th, Adams C, Wells N, Brock JW 3rd. Factors that influence outcomes of the Mitrofanoff and Malone antegrade continence enema reconstructive procedures in children. *J Urol* 2002;168(4 Pt 1):1537-40.
<http://www.ncbi.nlm.nih.gov/pubmed/12352454>
251. Richter F, Stock JA, Hanna MK. Continent vesicostomy in the absence of the appendix: three methods in 16 children. *Urology* 2002;60(2):329-34.
<http://www.ncbi.nlm.nih.gov/pubmed/12137836>
252. Shapiro SR, Lebowitz R, Colodny AH. Fate of 90 children with ileal conduit urinary diversion a decade later: analysis of complications, pyelography, renal function and bacteriology. *J Urol* 1975;114(2): 289-95.
<http://www.ncbi.nlm.nih.gov/pubmed/1159925>
253. Hald T, Hebjorn S. Vesicostomy - an alternative urine diversion operation. Long term results. *Scand J Urol Nephrol* 1978;12(3):227-31.
<http://www.ncbi.nlm.nih.gov/pubmed/725543>
254. Cass AS, Luxenberg M, Gleich P, Johnson CF. A 22-year follow-up of ileal conduits in children with a neurogenic bladder. *J Urol* 1984;132(2):529-31.
<http://www.ncbi.nlm.nih.gov/pubmed/6471190>
255. Schwartz SL, Kennelly MJ, McGuire EJ, Faerber GJ. Incontinent ileo-vesicostomy urinary diversion in the treatment of lower urinary tract dysfunction. *J Urol* 1994;152(1):99-102.
<http://www.ncbi.nlm.nih.gov/pubmed/8201699>
256. Atan A, Konety BR, Nangia A, Chancellor MB. Advantages and risks of ileovesicostomy for the management of neuropathic bladder. *Urology* 1999;54(4):636-40.
<http://www.ncbi.nlm.nih.gov/pubmed/10510920>
257. Herschorn S, Rangaswamy S, Radomski SB. Urinary undiversion in adults with myelodysplasia: longterm followup. *J Urol* 1994;152(2 Pt 1):329-33.
<http://www.ncbi.nlm.nih.gov/pubmed/8015064>

5. TREATMENT OF VESICO-URETERAL REFLUX

5.1 Treatment options

The treatment options for vesico-ureteral reflux in patients with NLUTD do not differ essentially from those in other reflux patients. They become necessary when the high intravesical pressure during the filling phase or during the voiding phase have been treated successfully, but where the reflux did not resolve (1-4). Subtrigonal injections with bulking agents or ureteral re-implantation are the standard procedures.

Subtrigonal injections of bulking agents: This minimal invasive procedure has a relatively good effect with complete success in about 65% of patients (5-12). It can also be easily repeated if not effective and thereby the success rate can be increased to about 75% after the second or third session.

Ureteral re-implantation: This technique has an immediate and long-lasting result in over 90% of the patients (11-13). In deciding which procedure will be offered to the patient, the relative risks of more invasive surgery and of less successful therapy should be considered.

5.2 REFERENCES

1. Kass EJ, Koff SA, Diokno AC. Fate of vesicoureteral reflux in children with neuropathic bladders managed by intermittent catheterization. *J Urol* 1981;125(1):63-4.
<http://www.ncbi.nlm.nih.gov/pubmed/7463586>
2. Sidi AA, Peng W, Gonzalez R. Vesicoureteral reflux in children with myelodysplasia: natural history and results of treatment. *J Urol* 1986;136(1 Pt 2):329-31.
<http://www.ncbi.nlm.nih.gov/pubmed/3723683>
3. Lopez Pereira P, Martinez Urrutia MJ, Lobato Romera R, Jaureguizar E. Should we treat vesicoureteral reflux in patients who simultaneously undergo bladder augmentation for neuropathic bladder?. *J Urol* 2001;165(6 Pt 2):2259-61.
<http://www.ncbi.nlm.nih.gov/pubmed/11371958>

4. Simforoosh N, Tabibi A, Basiri A, Noorbala MH, Danesh AD, Ijadi A. Is ureteral reimplantation necessary during augmentation cystoplasty in patients with neurogenic bladder and vesicoureteral reflux?. *J Urol* 2002;168(4 Pt 1):1439-41.
<http://www.ncbi.nlm.nih.gov/pubmed/12352413>
5. Diamond T, Boston VE. The natural history of vesicoureteric reflux in children with neuropathic bladder and open neural tube defects. *Z Kinderchir* 1987;42 (Suppl 1):15-6.
<http://www.ncbi.nlm.nih.gov/pubmed/3433968>
6. Chancellor MB, Rivas DA, Liberman SN, Moore J Jr, Staas WE Jr. Cystoscopic autogenous fat injection treatment of vesicoureteral reflux in spinal cord injury. *J Am Paraplegia Soc* 1994;17(2):50-4.
<http://www.ncbi.nlm.nih.gov/pubmed/8064286>
7. Sugiyama T, Hashimoto K, Kiwamoto H, Ohnishi N, Esa A, Park YC, Kurita T, Kohri K. Endoscopic correction of vesicoureteral reflux in patients with neurogenic bladder dysfunction. *Int Urol Nephrol* 1995;27(5):527-31.
<http://www.ncbi.nlm.nih.gov/pubmed/8775034>
8. Misra D, Potts SR, Brown S, Boston VE. Endoscopic treatment of vesico-ureteric reflux in neurogenic bladder—8 years' experience. *J Pediatr Surg* 1996;31(9):1262-4.
<http://www.ncbi.nlm.nih.gov/pubmed/8887097>
9. Haferkamp A, Mohring K, Staehler G, Gerner HJ, Dorsam J. Long-term efficacy of subureteral collagen injection for endoscopic treatment of vesicoureteral reflux in neurogenic bladder cases. *J Urol* 2000;163(1):274-7.
<http://www.ncbi.nlm.nih.gov/pubmed/10604375>
10. Shah N, Kabir MJ, Lane T, Avenell S, Shah PJ. Vesico-ureteric reflux in adults with neuropathic bladders treated with Polydimethylsiloxane (Macroplastique). *Spinal Cord* 2001;39(2):92-6.
<http://www.ncbi.nlm.nih.gov/pubmed/11402365>
11. Engel JD, Palmer LS, Cheng EY, Kaplan WE. Surgical versus endoscopic correction of vesicoureteral reflux in children with neurogenic bladder dysfunction. *J Urol* 1997;157(6):2291-4.
<http://www.ncbi.nlm.nih.gov/pubmed/9146655>
12. Granata C, Buffa P, Di Rovasenda E, Mattioli G, Scarsi PL, Podesta E, Dodero P, Jasonni V. Treatment of vesico-ureteric reflux in children with neuropathic bladder: a comparison of surgical and endoscopic correction. *J Pediatr Surg* 1999;34(12):1836-8.
<http://www.ncbi.nlm.nih.gov/pubmed/10626867>
13. Kaplan WE, Firlit CF. Management of reflux in the myelodysplastic child. *J Urol* 1983;129(6):1195-7.
<http://www.ncbi.nlm.nih.gov/pubmed/6854797>

6. QUALITY OF LIFE

6.1 Introduction

Quality of life (QoL) represents a very important aspect in the global management of neurogenic patients. Restoring QoL as much as possible is one of the aims of therapy. QoL is a reflection of the individual's ability to cope with the new life situation (1). Besides the limitations directly related to the neurological pathology, adequate treatment is possible in most patients and should not interfere with social independence. QoL can be influenced by several factors including family support, adjustment and coping, productivity, self-esteem, financial stability, education, physical and social environment (2) (Level of evidence: 3, Grade of recommendation: B). Age, sex, ethnicity and patient's acceptance of the condition should be taken into consideration (3) (Level of evidence: 3, Grade of recommendation: B).

There are no specific QoL questionnaires for neurogenic bladder dysfunction. The only validated tools are a generic Visual Analogue Scale (VAS) for bother and Qualiveen[®], which is a specific tool for QoL in spinal cord lesion and multiple sclerosis patients. Qualiveen appears to be a discriminative evaluation instrument (4, 5) (Level of evidence: 3, Grade of recommendation: B).

The appropriate therapy should manage symptoms, functional abilities, QoL, and avoid secondary complications (5). Changes in NLUTD appear to be the major determinants of a patient's QoL (6, 7) (Level of evidence: 2a, Grade of recommendation: B).

6.2 Conclusions and recommendations

1. Assess QoL to evaluate LUTS in neurogenic patients and during any type of treatment for neurogenic bowel dysfunction (Level of evidence 2a, Grade of recommendation: B).
2. Available tools are: Qualiveen, a specific tool for spinal cord lesion and multiple sclerosis patients, VAS for bother. However, generic (SF-36) or specific tools for incontinence (I-QOL) questionnaires could be used too. (Level of evidence: 2a, Grade of recommendation: B).
3. There is a lack of disease-specific outcome measures assessing health-related QoL in patients with NLUTD.

6.3 REFERENCES

1. Ku JH, The management of neurogenic bladder and quality of life in spinal cord injury. *BJU Int* 2006;98(4):739-45.
<http://www.ncbi.nlm.nih.gov/pubmed/16978269>
2. Whiteneck G, Meade MA, Dijkers M, Tate DG, Bushnik T, Forchheimer MB. Environmental factors and their role in participation and life satisfaction after spinal cord injury. *Arch Phys Med Rehabil* 2004;85(11):1793-803.
<http://www.ncbi.nlm.nih.gov/pubmed/15520974>
3. Marschall-Kehrel D, Roberts RG, Brubaker L. Patient-reported outcomes in overactive bladder: the influence of perception of condition and expectation for treatment benefit. *Urology* 2006;68(2 Suppl):29-37.
<http://www.ncbi.nlm.nih.gov/pubmed/16908338>
4. Bonniaud V, Jackowski D, Parratte B, Paulseth R, Grad S, Margetts P, Guyatt G. Quality of life in multiple sclerosis patients with urinary disorders: discriminative validation of the English version of Qualiveen. *Qual Life Res* 2005;14(2):425-31.
<http://www.ncbi.nlm.nih.gov/pubmed/15892431>
5. Pappalardo A, Patti F, Reggio A. Management of neuropathic bladder in multiple sclerosis. *Clin Ter* 2004;155(5):183-6.
<http://www.ncbi.nlm.nih.gov/pubmed/15344566>
6. Henze T. Managing specific symptoms in people with multiple sclerosis. *Int MS J* 2005;12(2):60-8.
<http://www.ncbi.nlm.nih.gov/pubmed/16417816>
7. Kalsi V, Apostolidis A, Popat R, Gonzales G, Fowler CJ, Dasgupta P. Quality of life changes in patients with neurogenic versus idiopathic detrusor overactivity after intradetrusor injections of botulinum neurotoxin type A and correlations with lower urinary tract symptoms and urodynamic changes. *Eur Urol* 2006;49(3):528-35.
<http://www.ncbi.nlm.nih.gov/pubmed/16426735>

7 FOLLOW-UP

7.1 Introduction

NLUTD is an unstable condition and can vary considerably, even within a relatively short period. Meticulous follow-up and regular checks are necessary (1-20). Depending on the type of the underlying neurological pathology and on the current stability of the NLUTD, the interval between the detailed investigations should not exceed 1-2 years. In patients with multiple sclerosis and in acute spinal cord injury, this interval is of course much smaller. Urine dip sticks should be available for the patient and urinalysis should be performed at least every second month. The upper urinary tract, the bladder shape, and residual urine should be checked every 6 months. Physical examination and blood and urine laboratory should take place every year. Any sign indicating a risk factor warrants specialized investigation.

7.2 GUIDELINES FOR FOLLOW-UP

1. Possible UTI checked by the patient (dip stick)
2. Urinalysis every second month
3. Upper urinary tract, bladder morphology, and residual urine every 6 months (ultrasound)
4. Physical examination, blood chemistry, and urine laboratory every year
5. Detailed specialistic investigation every 1-2 years and on demand when risk factors emerge. The investigation is specified according to the patient's actual risk profile, but should in any case include a video-urodynamic investigation and should be performed in a leading neuro-urological centre.

6. All of the above should be more frequent if the neurological pathology or the NLUTD status demand this.

7.3 REFERENCES

1. Stohrer M. Alterations in the urinary tract after spinal cord injury—diagnosis, prevention and therapy of late sequelae. *World J Urol* 1990;7:205-11.
2. Perakash I. Long-term urologic management of the patient with spinal cord injury. *Urol Clin North Am* 1993;20(3):423-34.
<http://www.ncbi.nlm.nih.gov/pubmed/8351768>
3. Selzman AA, Elder JS, Mapstone TB. Urologic consequences of myelodysplasia and other congenital abnormalities of the spinal cord. *Urol Clin North Am* 1993;20(3):485-504.
<http://www.ncbi.nlm.nih.gov/pubmed/8351774>
4. Stohrer M, Kramer G, Lochner-Ernst D, Goepel M, Noll F, Rubben H. Diagnosis and treatment of bladder dysfunction in spinal cord injury patients. *Eur Urol Update Series* 1994;3:170-5.
5. Thon WF, Denil J, Stief CG, Jonas U. Urologische Langzeitbetreuung von Patienten mit Meningomyelozele. II. Therapie. *Aktuel Urol* 25:63-76. [article in German] [Long-term care of patients with meningomyelocele. II. Therapy].
6. Waites KB, Canupp KC, DeVivo MJ, Lloyd LK, Dubovsky EV. Compliance with annual urologic evaluations and preservation of renal function in persons with spinal cord injury. *J Spinal Cord Med* 1995;18(4):251-4.
<http://www.ncbi.nlm.nih.gov/pubmed/8591072>
7. Cardenas DD, Mayo ME, Turner LR. Lower urinary changes over time in suprasacral spinal cord injury. *Paraplegia* 1995;33(6):326-9.
<http://www.ncbi.nlm.nih.gov/pubmed/7644258>
8. Capitanucci ML, Iacobelli BD, Silveri M, Mosiello G, De Gennaro M. Long-term urological follow-up of occult spinal dysraphism in children. *Eur J Pediatr Surg* 1996;6(Suppl 1):25-6.
<http://www.ncbi.nlm.nih.gov/pubmed/9008815>
9. Chua HC, Tow A, Tan ES. The neurogenic bladder in spinal cord injury—pattern and management. *Ann Acad Med Singapore* 1996;25(4):553-7.
<http://www.ncbi.nlm.nih.gov/pubmed/8893929>
10. Agarwal SK, Bagli DJ. Neurogenic bladder. *Indian J Pediatr* 1997;64(3):313-26.
<http://www.ncbi.nlm.nih.gov/pubmed/10771853>
11. Rashid TM, Hollander JB. Multiple sclerosis and the neurogenic bladder. *Phys Med Rehabil Clin N Am* 1998;9(3):615-29.
<http://www.ncbi.nlm.nih.gov/pubmed/9894113>
12. Burgdorfer H, Heidler H, Madersbacher H, Melchior H, Palmtag H, Richter R, Richter-Reichhelm M, Rist M, Rubben H, Sauerwein D, Schalkhauser K, Stohrer M. Leitlinien zur urologischen Betreuung Querschnittsgelähmter. *Urologe A* 1998;37:222-8. [article in German] [Guidelines for the urological care of paraplegics]
13. McKinley WO, Jackson AB, Cardenas DD, DeVivo MJ. Long-term medical complications after traumatic spinal cord injury: a regional model systems analysis. *Arch Phys Med Rehabil* 1999; 80(11):1402-10.
<http://www.ncbi.nlm.nih.gov/pubmed/10569434>
14. Atan A, Konety BR, Nangia A, Chancellor MB. Advantages and risks of ileovesicostomy for the management of neuropathic bladder. *Urology* 1999;54(4):636-40.
<http://www.ncbi.nlm.nih.gov/pubmed/10510920>
15. Cranidis A, Nestoridis G. Bladder augmentation. *Int Urogynecol J Pelvic Floor Dysfunct* 2000; 11(1):33-40.
<http://www.ncbi.nlm.nih.gov/pubmed/10738932>
16. Elliott DS, Boone TB. Recent advances in the management of the neurogenic bladder. *Urology* 2000;56 (6 Suppl 1):76-81.
<http://www.ncbi.nlm.nih.gov/pubmed/11114567>
17. Chen Y, DeVivo MJ, Roseman JM. Current trend and risk factors for kidney stones in persons with spinal cord injury: a longitudinal study. *Spinal Cord* 2000;38(6):346-53.
<http://www.ncbi.nlm.nih.gov/pubmed/10889563>
18. Lawrenson R, Wyndaele JJ, Vlachonikolis I, Farmer C, Glickman S. Renal failure in patients with neurogenic lower urinary tract dysfunction. *Neuroepidemiology* 2001;20(2):138-43.
<http://www.ncbi.nlm.nih.gov/pubmed/11359083>

19. Ciancio SJ, Mutchnik SE, Rivera VM, Boone TB. Urodynamic pattern changes in multiple sclerosis. *Urology* 2001;57(2):239-45.
<http://www.ncbi.nlm.nih.gov/pubmed/11182328>
20. Burns AS, Rivas DA, Ditunno JF. The management of neurogenic bladder and sexual dysfunction after spinal cord injury. *Spine* 2001;26 (24 Suppl):S129-S136.
<http://www.ncbi.nlm.nih.gov/pubmed/11805620>

8. CONCLUSIONS

NLUTD is a multi-faceted pathology. It requires an extensive and specific diagnosis before we can embark on an individualized therapy, which takes into account the medical and physical condition of the patient and the patient's expectations about his future social and physical situation with respect to the NLUTD.

The urologist or paediatric urologist can select from a wealth of therapeutical options, each with its own pros and cons. Notwithstanding the success of any therapy embarked upon, a close surveillance is necessary for all the patient's life.

With these guidelines, we offer you expert advice on how to define the patient's NLUTD condition 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 less invasive as possible.

9. ABBREVIATIONS USED IN THE TEXT

This list is not comprehensive for the most common abbreviations

CVA	cerebrovascular
DLPP	detrusor leak point pressure
DSD	detrusor sphincter dyssynergia
EMG	electromyography, electromyogram
FVC	frequency volume chart
IC	intermittent catheterization
ISC	intermittent self-catheterization
ICS	international Continence Society
LPP	leak point pressure
LMNL	lower motor neuron lesion
LUT	lower urinary tract
LUTD	lower urinary tract dysfunction
LUTS	lower urinary tract symptoms
MTC	micturition time chart
NDO	neurogenic detrusor overactivity
NLUTD	neurogenic lower urinary tract dysfunction
PNE	percutaneous nerve evaluation test
QoL	quality of life
SARS	sacral anterior root stimulation
SCI	spinal cord injury
SDAF	sacral deafferentation
SLE	systemic lupus erythematosus
UMNL	upper motor neuron lesion
UTI	urinary tract infection
VAS	visual Analogue Scale

Conflict of interest

All members of the Neurogenic LUTS guidelines writing panel have provided disclosure statements of all relationships which they have and which may be perceived as a potential source of conflict of interest. This information is kept on file in the European Association of Urology Central Office database. 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.

