Cauda equina lesions as a complication of spinal surgery

Simon Podnar

Received: 4 September 2008 / Revised: 9 August 2009 / Accepted: 13 September 2009 / Published online: 21 September 2009
© Springer-Verlag 2009

Abstract Although the most common aetiology of cauda equina lesions is lumbar intervertebral disc herniation, iatrogenic lesions may also be the cause. The aim of this study was to identify and present patients in whom cauda equina lesions occurred after spinal surgery. From the author's series of patients with cauda equina lesions, those with the appearance of sacral symptoms after spinal surgery were identified. To demonstrate lesions more objectively, electrodiagnostic studies were performed in addition to history and clinical examination. Imaging studies were also reviewed. Of 69 patients from the series, 11 patients in whom a cauda equina lesion developed after spinal surgery were identified. The aetiology comprised surgery for herniated intervertebral disc in 5 (4 performed by a single surgeon), spinal stenosis surgery in 4, and postoperative lumbar epidural haematoma in 2 patients (each performed by a different surgeon). Proportion of spinal surgeries with this complication varied from 0 to 6.6‰ in different centres. Patients with iatrogenic cauda equina lesion were significantly older ($p < 0.001$), and reported more severe urinary, but similar bowel and sexual symptoms compared to other patients in the series. In conclusion the study identified spinal surgery as the cause of approximately 15% of cauda equina lesions. More than a third of lesions developed after procedures performed by a single surgeon. Most of the remaining lesions could probably be avoided by better surgical technique (e.g. the use of a high-speed drill instead of a Kerrison rongeur in patients with severe spinal stenosis), or prevented by closer postoperative

monitoring (e.g. in patients with postoperative lumbar epidural haematoma).

Keywords Cauda equina · Disc herniation · Spinal stenosis · Spinal surgery · Sacral

Abbreviations

CT	Computer tomography
EAS	External anal sphincter
EMG	Electromyography
MRI	Magnetic resonance imaging
MUP	Motor unit potential

Introduction

Damage to the cauda equina may result in a severe disorder with back pain radiating into the legs, leg weakness, and saddle sensory loss. In addition, bladder, bowel, and sexual functions may also be disturbed [9, 14, 15, 17]. The annual incidence rate of cauda equina lesions has been estimated at 3.4 per million population, and the period prevalence at 8.9 per 100,000 [11]. The most common aetiology is central intervertebral disc herniation (particularly common at L4–5 and L5–S1 levels). Other aetiologies (tumours, haematomas, inflammation, spinal surgery, etc.) are less common.

Although less frequent, cauda equina lesions that occur during spinal surgery are of the utmost importance, both from the clinical and medico-legal perspectives, because they represent potentially avoidable lesions. Such lesions, however, have only rarely been studied previously [4, 7].

The aim of this study was to identify patients in whom cauda equina lesions occurred during spinal surgery,

S. Podnar (✉)
Division of Neurology, Institute of Clinical Neurophysiology,
University Medical Center Ljubljana,
1525 Ljubljana, Slovenia
e-mail: simon.podnar@kclj.si

present the clinical consequences of such lesions, and attempt to elucidate reasons for their occurrence.

Materials and methods

Data from the author's series of patients with cauda equina lesions that occurred in the period 1996–2004 were reviewed [11]. Patients with involvement at the Th12 or L1 spinal levels (i.e. probable conus medullaris lesions) were excluded from the study.

After the development of the cauda equina lesion, in all patients a detailed history of neurologic symptoms, as well as bladder, bowel, and sexual dysfunction was obtained. To score the severity of sacral dysfunctions, Slovene versions of the standard urinary incontinence [3, 5], anal incontinence [6], constipation [1], and male sexual dysfunction [16] questionnaires were used [9, 14, 15]. In all patients clinical examination was performed with emphasis on the lower limbs and sacral segments [13]. Furthermore, all patients underwent concentric needle electromyography (EMG), and electrophysiologic measurement of the sacral reflexes (penilo-/clitro-cavernosus reflex) [10, 18]. First, several muscles belonging to the lumbosacral myotomes were examined qualitatively looking for spontaneous denervation activity, the characteristics of motor unit potentials (MUPs), and the interference pattern. Quantitative EMG of the external anal sphincter (EAS) muscles was then performed bilaterally by multi-MUP analysis, and the EMG abnormalities were graded as possible, probable, or definite [8]. Electrophysiologic measurement of the sacral reflexes was performed using double pulse electrical stimulation [18]. Reproducible responses were looked for, and the latency of this penilo-/clitro-cavernosus reflex [10] was measured [18]. Results were compared to our upper normative value of 36 ms [12].

The diagnosis of the cauda equina lesion was made on the basis of: (1) a history of an appropriate causative event followed by the development of lower limb, bladder, bowel, or sexual dysfunction; (2) clinical examination revealing diminished anal squeeze during rest and voluntary activation, saddle sensory loss or, in men, diminished penilo-cavernosus reflex; (3) EMG signs of denervation (3–10 weeks after the event) or reinnervation (at least 3 months after the event) in sacral myotomes; (4) abnormal results on neurophysiologic measurements of the penilo-/clitro-cavernosus reflex; and (5) in most patients radiologic findings of central lumbar disc herniation, spinal fracture, tumour, or other relevant intraspinal pathology.

In patients with possible iatrogenic lesions, every effort was made to obtain as complete preoperative, operative, and postoperative data files as possible. Reports describing findings on clinical neurologic examination, lumbosacral

electrodiagnostic and imaging studies (X-ray myelography, computer tomography—CT and CT myelography, and magnetic resonance imaging—MRI) were reviewed. Exact descriptions of operative techniques were sought from surgical reports, and personal communications. In addition, a retrospective history of preoperative and postoperative neurologic symptoms and sacral dysfunction was obtained in all these patients. To be included in the group with iatrogenic lesions, patients must have been without symptoms or signs of the neuropathic sacral lesion preoperatively, and have a clear onset of clinical and electrodiagnostic findings compatible with such a lesion after surgical intervention. The intervention also had to be one in which there is a potential risk of causing the cauda equina lesion.

Patients from the whole series were divided into those in whom cauda equina lesions occurred after spinal surgery, and patients with other aetiologies of such lesions. Data obtained on history, clinical examination, and electrodiagnostic studies were compared between the two groups using a Mann–Whitney *U*-test; $p < 0.05$ was regarded as significant.

For period 1996–2004 a proportion of spinal surgeries resulting in cauda equina lesion in three Slovenian centres was calculated. Significance of differences in frequencies was calculated using “Z score for two proportions calculator” [2].

Results

Altogether 69 patients (44 men), aged 20 to 82 years (mean, 48 years), with clinical, electrodiagnostic, and radiologic findings supportive of chronic cauda equina lesions were identified. From these, a subgroup of 11 patients (8 men), aged 41 to 82 years (mean, 61 years) without neuropathic lower sacral lesion symptoms or signs before (Table 1), and with clear clinical and electrodiagnostic findings compatible with such a lesion after spinal surgery (Tables 2 and 3), were identified.

Before spinal surgery (Table 1) patients # 1–5 (all younger than 60 years) had mainly symptoms of single root compression. In contrast, older patients preoperatively mostly had symptoms consistent with spinal stenosis (i.e. neurogenic claudication). Two patients (# 6 and # 7) had Parkinson's disease, and 2 (# 9 and # 11) were treated for hypertension. Mild constipation in patient # 6 with Parkinsonism was the only sacral symptom before surgery in this group. Neurologic signs were not recorded, and electrodiagnostic studies were not performed before surgery in most patients. Spinal imaging studies (Table 4) at that time revealed isolated disc herniations in the youngest 2 patients (# 1 and # 2), disc herniation with sequestration

Table 1 Findings of preoperative clinical, and electrodiagnostic studies in 11 patients with cauda equina lesions after spinal surgery

Patient no.	Gender	Age (years)	Symptoms	Sacral symptoms	Neurologic signs	Electrodiagnostic findings
1	M	41	R S1 radiculopathy	None	Not recorded	Not performed
2	M	43	L S1 radiculopathy	None	Not recorded	Not performed
3	M	49	R leg radicular pain	None	Non-elicitable R ankle jerk	Not performed
4	F	55	L and later R leg radicular pain	None	Not recorded	Not performed
5	M	56	R leg radicular pain, foot tingling	None	R hallux extension weakness, L5 and S1 sensory loss	R L5 myotome denervation
6	M	64	Severe bilateral leg weakness and tingling	Mild constipation	Parkinsonism, Not recorded	Not performed
7	M	65	Radiculopathies L L5 & S1, R S1	None	Not recorded	Bilateral L5 and S1 myotome denervation
8	F	66	Severe pain and tingling during walking	None	Normal	Normal
9	F	73	Severe R leg radicular pain; unable to walk	None	Non-elicitable R knee, and both ankle jerks	Not performed
10	M	79	R thigh pain during standing and walking	None	R knee extension weakness, L4 and S1 sensory loss	Not performed
11	M	82	Bilateral leg electricity-like sensations during standing and walking	None	Not recorded	Not performed

M male, F female, R right, L left, HD disc herniation

in 1 patient (# 4), and spinal stenosis with or without associated disc herniation in the remaining 8 patients.

A number of different surgical procedures were performed (Table 2). Postoperative complications were initially described in only 2 patients with epidural haematoma (# 7 and # 9), and in one patient (# 4) with disc sequester evacuation difficulties and dural tear. However, in all patients intraoperative neuropathic lesions to the lower sacral segments were demonstrated by the onset of sacral (i.e. bladder, bowel, and sexual) and lower limb symptoms and clinical and electrodiagnostic abnormalities that have persisted (Tables 2, 3).

Patients with cauda equina lesions after spinal surgery were significantly older ($p < 0.001$), and reported more severe urinary symptoms than patients with cauda equina lesions of other aetiologies (Table 5). However, no significant differences in gender ($p = 0.79$), bowel, or sexual symptoms, or findings on clinical or electrodiagnostic examinations, were found between the two groups.

In our group of patients with appearance of cauda equina lesion after spinal surgery none was operated in our major neurosurgical centre, 2 patients (#10 and #11) were operated in our major orthopaedic centre, and 5 patients (#1, #2, #3, #5, and #7) were operated in one of regional hospitals. In studied period number of lumbar surgeries for disc herniation and spinal stenosis were 2,070 and 810 in a neurosurgical centre, and 941 and 378 in orthopaedic centre, respectively. In the same period in a regional hospital with 761 spinal surgeries were performed. Proportions of spinal surgery patients with this postoperative

complication were 0% (0/2880), 1.5% (2/1319), and 6.6% (5/761), respectively. Difference in proportions between the neurosurgical centre, and the regional hospital was significant ($Z = 3.42$, $p < 0.01$).

Discussion

The present study demonstrated spinal surgery to be the cause of the cauda equina lesions in about 15% of patients. In our population cauda equina lesions occurred during lumbar disc and spinal stenosis surgery. During study period such lesions occurred in 0%, 1.5%, and 6.6% of spinal surgeries performed in neurosurgical, major orthopaedic, and regional hospital departments in Slovenia, respectively. Neurogenic lesions in these patients were demonstrated by objective methods of clinical neurophysiology in addition to clinical examination (Tables 1, 3). Clinically, all patients, but one (# 11) had clearly impaired touch and pinprick saddle sensation (Table 3). On EMG of the EAS muscles in 5 patients spontaneous denervation activity and in another 3 MUPs reinnervation changes were found. These electrodiagnostic abnormalities pointing to sacral root lesions were substantiated by pathologic measurements of the penile-/clitro-cavernosus reflex in all but 3 patients (Table 3). However, as patients did not report sacral symptoms preoperatively, sacral electrodiagnostic studies were performed only after and not before spinal surgery. The development of neurologic deficits during surgical procedures was therefore reliant on a history of the

Table 2 Surgical procedures and post-surgical neurologic findings in 11 patients with cauda equina lesions after spinal surgery

Patient no.	Surgery	Surgical complications	New somatic symptoms	Later intervention	Early electrodiagnostic findings
1 ^a	Disc evacuation	Not reported	R leg cramps, S1–S4 sensory loss and tingling, plantar flexion weakness	2 years later: resection of epidural fibrosis and L5–S1 HD evacuation	Severe R S1–S5 myotome denervation
2 ^a	Disc evacuation	Not reported	L S2–S4 sensory tingling and loss, plantar flexion weakness	None	Severe L S1–S5 myotome denervation
3 ^a	Disc evacuation, L5 and S1 root adhesiolysis	Not reported	As “wearing tight shoes”, with gradual proximal spread, bil. S2–S4 sensory tingling and loss	None	Moderate bil. L5–S5 myotome denervation
4	Disc material evacuation, partial hemilaminectomy	Difficulties with disc sequester evacuation, dural tear	R leg sensory symptoms, weakness, headache, CSF leak	Bed rest for CSF leak	Severe bil. L5–S5 myotome denervation
5 ^a	Partial L5 hemilaminectomy, disc evacuation	Not reported	R saddle sensory symptoms	None	Moderate R S1–S5 myotome denervation
6	Not described	Not reported	Saddle sensory symptoms	None	Moderate bil. S2–S5 myotome denervation
7 ^a	L2–L4 spinal canal exploration	Postsurgical haematoma	Severe pain, sensory symptoms and transient weakness	12 hours later: evacuation of L2–L4 haematoma	R severe, L mild L5–S5 myotome denervation
8	L5 hemilaminectomy	Not reported	Saddle sensory symptoms	None	R moderate, L mild S2–S5 myotome denervation
9	L4 laminectomy, bil. L4 and L5 decompression	Postsurgical haematoma	Bil. progressive severe pain, sensory symptoms and leg weakness	24 hours later: evacuation of L3–L5 haematoma	Bil. moderate L5–S1 and complete S2–S5 myotome denervation
10	L4 laminectomy	Not reported	Sacral dysfunction	MRT: local oedema, operative revision	Bil. severe S2–S5 denervation
11	R L4 hemilaminectomy, R L3–L4 fenestration, L4–L5 cartilage removal	Not reported	Sacral dysfunction	None	Severe R L5 and S2–S5 myotome denervation

R right, L left, HD disc herniation, CSF cerebrospinal fluid

^a Surgeries performed by a single surgeon

Table 3 Findings of sacral clinical and electrodiagnostic studies and long-term clinical outcomes in 11 patients with cauda equina lesions after spinal surgery

Patient no.	Sacral sensation	Sphincter EMG	Sacral reflex	Laterality of lesion	Bladder function	Bowel function	Sexual function
1	↓	Normal	Non-elicitable	Unilateral	UI, ↓ Emptying	FI, C	↓↓ Erection
2	↓↓	Denervation	Abnormal	Unilateral	UI, ↓ Emptying	FI, C	↓ Erection
3	↓↓	Denervation	Abnormal	Bilateral	UI, ↓ Emptying	FI, C	↓↓ Erection
4	↓	Normal	Abnormal	Bilateral	Ur, ↓ Emptying	C	?
5	↓	Normal	Normal	Unilateral	UI, Ur, ↓ Emptying	AI	Normal
6	↓↓↓	Denervation	Non-elicitable	Bilateral	UI, ↓↓ Emptying	FI, C	↓↓ Erection
7	↓↓	Reinnervation	Abnormal	Bilateral	Normal	AI	Not active
8	↓	Reinnervation	Abnormal	Bilateral	UI, ↓↓ Emptying	C	Not active
9	↓↓↓	Reinnervation	Abnormal	Bilateral	UI, Ur, ↓ Emptying	FI, C	Not active
10	↓	Denervation	Normal	Bilateral	UI, ↓↓ Emptying	C	↓↓↓ Erection
11	Normal	Denervation	Normal	Bilateral	UI, ↓↓ Emptying	C	↓↓↓ Erection

↓, ↓↓, and ↓↓↓ slightly, moderately, and severely reduced; ? unknown

UI urinary incontinence, Ur urgency, AI flatus incontinence, FI faecal incontinence, C constipation

Table 4 Imaging studies in 11 patients with cauda equina lesions after spinal surgery

Patient no.	Imaging method	Spinal level			
		L2/L3	L3/L4	L4/L5	L5/S1
1	Myelography ^a				R dorsolateral HD
2	CT myelography			1.0 cm × 1.6 cm, disc protrusion	1.2 cm × 1.5 cm, L dorsolateral HD
3	Myelography ^a			R dorsolateral HD, skeletal stenosis	
4	CT ^a			HD, fragment behind L4	
5	Myelography ^b			R dorsolateral HD, skeletal stenosis	
6	CT myelography MRI—post operative	1.3 cm × 2.2 cm	1.3 cm × 1.8 cm 1.0 cm × 1.9 cm, granulations with liquid collection	1.0 cm × 1.8 cm Laminectomy L3–L5	1.1 cm × 1.8 cm
7	CT myelography		0.7 cm × 1.6 cm, block of contrast	1.0 × 1.2 cm	1.2 cm × 1.3 cm
8	CT ^a	Severe spinal stenosis	Severe spinal stenosis	Severe spinal stenosis	
9	CT myelography	1.0 cm × 1.8 cm, thickened small joints and ligaments, disc protrusion	1.0 cm × 1.8 cm, thickened small joints and ligaments	1.0 cm × 1.5 cm, thickened small joints and ligaments	1.3 cm × 1.8 cm
	CT myelography—post operative	Dural sac compressed anteriorly at L3	CSF squeezed out	CSF squeezed out, laminectomy L4	CSF squeezed out
10	CT		0.8 cm × 1.5 cm, thickened small joints and ligaments, disc protrusion	0.9 cm × 1.1 cm, thickened small joints and ligaments, R dorsolateral HD	0.9 cm × 1.1 cm, thickened small joints, and ligaments, disc protrusion
	CT myelography—post operative	1.0 cm × 2.0 cm, thickened small joints and ligaments, disc protrusion	0.9 cm × 1.6 cm, thickened small joints and ligaments, disc protrusion	1.6 cm × 2.1 cm, laminectomy	1.6 cm × 2.1 cm, laminectomy
11	CT	1.0 cm × 1.8 cm, thickened small joints, and ligaments, disc protrusion	0.9 cm × 1.5 cm, thickened small joints, and ligaments, flava, disc protrusion	0.5 cm × 1.0 cm, thickened small joints, and ligaments, R dorsolateral HD	
	CT myelography			Block, CSF squeezed out	

The first dimension of the measurements is anterior–posterior, and the second is lateral

R right, L left, HD disc herniation, CSF cerebrospinal fluid

^a Only written report was available

^b Length scale was not provided on a film therefore exact measurements were not possible

onset of sacral neurologic, urinary, bowel, or sexual symptoms, and on a comparison of preoperative neurologic findings (as described in patients' files) with neurologic findings after surgical intervention. Except one patient (# 7) with normal urinary function, all other patients reported urinary incontinence or reduced bladder emptying, and 3 of patients also reported urinary urgency. Majority reported constipation, in most associated by incontinence of faeces, or at least flatus. Most included patients also reported either severe erectile dysfunction or complete sexual abstinence (Table 3).

The severity of neuropathic lesions in our patients with lesions due to spinal surgery was similar to the severity in patients with non-iatrogenic causes, with the exception of urinary symptoms, which were more severe in patients with iatrogenic lesions (Table 5).

In 5 patients with iatrogenic lesions included in this series spinal surgery was performed by a single orthopaedic surgeon from a regional hospital. The cauda equina lesion occurred during lumbar disc surgery in 4 of these patients (# 1, # 2, # 3, and # 5, Tables 2, 4), which are probably preventable. Similarly a case of lumbar discectomy with

Table 5 Comparison of clinical and electrodiagnostic findings in patients with cauda equina lesions after spinal surgery or due to other aetiologies

Feature	Abnormal (%)		<i>p</i> value
	Iatrogenic	Other aetiologies	
Urinary incontinence	92	54	0.02*
Bladder overactivity	33	61	0.001*
Bladder emptying difficulties	78	82	0.04*
Faecal incontinence	45	47	0.70
Constipation	82	51	0.28
Erectile dysfunction	83	66	0.25
Anal tone	88	62	0.09
Anal squeeze	75	71	0.36
Penilo-cavernosus reflex	88	78	0.23
Quantitative EMG	70	67	0.96
Sacral reflex measurements	70	74	0.66

Data were obtained from 11 patients with cauda equina lesions after spinal surgery and 58 patients with cauda equina lesions due to other aetiologies. The Mann–Whitney *U*-test was used for comparison.

* Significant differences ($p < 0.05$)

dural tear during evacuation of sequestered disc material (# 4) which was performed in another regional hospital would be probably preventable. Damage could probably also be reduced by better postoperative surveillance, and prompt re-operation in both patients (# 7 and # 9) with postoperative haematoma occurring after lumbar disc surgery. The remaining 4 cases (# 6, #8, # 10, and # 11) during surgery for spinal stenosis, in which 4 different orthopaedic surgeons were involved, would appear to be more difficult to prevent.

In a retrospective review of 2,842 discectomies, McLaren and Bailey found a stenotic spinal canal (antero-posterior diameter <13 mm) at the level of the disc protrusion in 5 out of 6 cases with consequent cauda equina lesions [7]. In all these cases a keyhole interlaminar approach was used, which did not allow enough space for postoperative swelling. Decompressive laminectomy has been suggested for surgical management of disc protrusion superimposed on spinal stenosis. Similarly, in our series 3 out of 5 patients (# 2, # 3, and # 5) with cauda equina lesion occurring after surgery (performed by the same surgeon) for large dorsolateral lumbar disc herniation also had central spinal stenosis. There were no imaging studies available for one (# 1), and sequestered disc fragment was present in the other (# 4) of remaining two patients. According to the surgical reports, our surgeons did perform decompressive laminectomies, and none of 3,011 discectomies performed in our neurosurgical and orthopaedic centres resulted in cauda equina lesion (difference between their and our proportions was significant; $Z = 1.98$, $p < 0.05$). In one case, from McLaren and Bailey's series

[7], stenosis could not be identified, and the cause of the lesion was unclear. In a similar, more recent study from Sweden, Henriques et al. [4] also noted preexisting narrowing of the spinal canal in all 5 of their patients with this complication. They suggested venous congestion triggered by postoperative oedema as a possible mechanism for the lesion [4].

The exact mechanisms for lesions in our spinal stenosis patients remain unclear. In addition to causes of the cauda equina lesions described above [4, 7], lesions might occur because of excessive pressure or traction exerted on the nerve tissue during surgery. This is supported by the fact that all cauda equina lesions reported in this series occurred with orthopaedic surgeons who used a Kerrison rongeur, even in patients with severe spinal stenosis. In contrast, no cases from the series occurred with surgeons, who in patients with severe spinal stenosis also had the option of using a high-speed drill, and the aid of the operative microscope. Both patients (# 10 and # 11) with postoperative appearance of cauda equina lesion operated in the orthopaedic centre had severe spinal stenosis (antero-posterior diameter <10 mm, Table 4). However, the difference in proportion of cauda equina lesion after spinal surgery for spinal stenosis between neurosurgical and orthopaedic centre did not reach significance ($0/810 = 0\%$ vs. $2/378 = 5.3\%$, $Z = 0.20$, $p = >0.05$). Further studies are needed to reveal the causes of cauda equina lesions during spinal stenosis surgery.

Preoperative imaging modalities varied in our series of 11 patients with cauda equina lesion occurring after spinal surgery; 3 were evaluated by myelography, 4 by CT, and remaining 4 by CT myelography. These spinal imaging modalities were standard in Slovenia during the period 1996–2004. None of these patients were imaged by MRI, as this method was not as widely available as today. Nowadays, the situation changed dramatically, and majority of spinal surgery patients are preoperatively evaluated by MRI. This change may improve results of spinal surgery by allowing the surgeon to better plan the procedure. However, particularly in difficult cases MRI is often still combined with CT myelography.

Although some surgeons frankly discussed the complication with affected patients, others from our series either skipped discussion or denied the complication altogether. Those patients who did not have the opportunity to openly discuss the complication with their surgeon seem to be particularly unhappy; some even declined any further medical assistance. None of our patients with iatrogenic lesions were offered psychological support or financial compensation, which further worsened the situation.

In conclusion, the present study demonstrated that surgery to the lumbar spine is prone to cause lesions to the cauda equina in a low percentage of patients (up to 6.6% in

our series). Such events result in severe disability and may have medico-legal consequences. As most of these complications seem potentially preventable, further prospective studies on surgical techniques and appropriate actions after the complication are needed to reduce this neglected problem.

Acknowledgments The author thanks Prof. Roman Bošnjak (surgery), Prof. David B. Vodusek (neurology), Dr. Gašper Gregorič (radiology), and Dr. Dianne Jones (language) for review of the manuscript. The study was supported by the Republic of Slovenia Research Agency, Grant No. J3 7899.

References

1. Agachan F, Chen T, Pfeifer J, Reissman P, Wexner SD (1996) A constipation scoring system to simplify evaluation and management of constipated patients. *Dis Colon Rectum* 39(6):681–685
2. Dimension Research, Inc. (2005) Z-test for two proportions calculator. <http://www.dimensionresearch.com/index.html>
3. Donovan JL, Abrams P, Peters TJ, Kay HE, Reynard J, Chapple C, De La Rosette JJ, Kondo A (1996) The ICS-‘BPH’ Study: the psychometric validity and reliability of the ICSmale questionnaire. *Br J Urol* 77(4):554–562
4. Henriques T, Olerud C, Petren-Mallmin M, Ahl T (2001) Cauda equina syndrome as a postoperative complication in five patients operated for lumbar disc herniation. *Spine* 26(3):293–297
5. Jackson S, Donovan J, Brookes S, Eckford S, Swithinbank L, Abrams P (1996) The Bristol Female Lower Urinary Tract Symptoms questionnaire: development and psychometric testing. *Br J Urol* 77(6):805–812
6. Jorge JM, Wexner SD (1993) Etiology and management of fecal incontinence. *Dis Colon Rectum* 36(1):77–97
7. McLaren AC, Bailey SI (1986) Cauda equina syndrome: a complication of lumbar discectomy. *Clin Orthop Relat Res* 204:143–149
8. Podnar S (2004) Criteria for neuropathic abnormality in quantitative anal sphincter electromyography. *Muscle Nerve* 30(5):596–601
9. Podnar S (2006) Bowel dysfunction in patients with cauda equina lesions. *Eur J Neurol* 13(10):1112–1117
10. Podnar S (2006) Nomenclature of the electrophysiologically tested sacral reflexes. *Neurourol Urodyn* 25(1):95–97
11. Podnar S (2007) Epidemiology of cauda equina and conus medullaris lesions. *Muscle Nerve* 35(4):529–531
12. Podnar S (2007) Neurophysiologic studies of the penile-cavernosus reflex: normative data. *Neurourol Urodyn* 26(6):864–869
13. Podnar S (2007) Saddle sensation is preserved in a few patients with cauda equina or conus medullaris lesions. *Eur J Neurol* 14(1):48–53
14. Podnar S, Oblak C, Vodusek DB (2002) Sexual function in men with cauda equina lesions: a clinical and electromyographic study. *J Neurol Neurosurg Psychiatry* 73(6):715–720
15. Podnar S, Trsinar B, Vodusek DB (2006) Bladder dysfunction in patients with cauda equina lesions. *Neurourol Urodyn* 25(1):23–31
16. Rosen RC, Riley A, Wagner G, Osterloh IH, Kirkpatrick J, Mishra A (1997) The international index of erectile function (IIEF): a multidimensional scale for assessment of erectile dysfunction. *Urology* 49(6):822–830
17. Shapiro S (1993) Cauda equina syndrome secondary to lumbar disc herniation. *Neurosurgery* 32(5):743–746
18. Vodusek DB, Amarenco G, Batra A, Benson T, Bharucha AE, Podnar S, Yang CC (2005) Clinical neurophysiology. In: Abrams P, Cardozo L, Khoury S, Wein A (eds) *Incontinence*. Health Publication Ltd., Plymouth (UK), pp 675–706