Case reports

Chiropractic care of interstitial cystitis/painful bladder syndrome associated with pelvic lumbar spine dysfunction: a case series

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Received 23 June 2011; received in revised form 22 September 2011; accepted 28 October 2011

Key indexing terms:

Manipulation;
Chiropractic;
Cystitis;
Interstitial;
Urinary tract
physiological
phenomena

Abstract

Objective: The purpose of this case series is to describe findings for patients with bladder pain syndrome (BPS) or interstitial cystitis (IC) who responded positively under chiropractic care.

Clinical Features: Eight cases were selected retrospectively reviewed from 2 independent chiropractic clinics in Scotland. Cases were selected if patients reported bladder dysfunction problems and responded positively to chiropractic care. The cases in this report describe the range of patients affected by this condition. Each patient was treated using chiropractic methods that were specific to the individual case.

Intervention and Outcomes: The patients selected for this case series showed positive response to chiropractic care over various lengths of time and numbers of treatments. Some of the chiropractic patients who had chronic spinal conditions had reoccurrence of bladder symptoms during an exacerbation of mechanical spinal problems.

Conclusion: This case series highlights that bladder and urinary problems may be associated with spinal dysfunction for some patients.

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Introduction

Interstitial cystitis (IC) is a common condition that affects both men and women, but more frequently women. There are varying estimates of the exact frequency of the condition, with some authors suggesting

an incidence ratio of 1:12,¹ whereas others have reported sex-specific incidence of 1 in 20 in men and of 1 in 4 in women.² It presents with a very similar symptom pattern to urinary tract infections; however, cytology investigations classically do not find any pathogens. The condition's etiology is one of an alteration of the normal physiological and biochemical processes in the epithelial linings of the bladder and urethra.³ These include the presence of antiproliferative factor in the urine, and there can be widespread

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glomerulations and Hunner ulcers in 10% of IC cases.⁴ After the meeting of the European Society for the Study of Interstitial Cystitis in 2007, it was suggested that the condition should be termed *bladder pain syndrome* (BPS). This was to reflect the complex nature of the condition and the enormous range of symptoms, many of which do not strictly fall into the classic diagnosis of IC.⁵ However, the term *IC* continues to be used widely. We have therefore opted to use the 2 terms together.

The pathogenesis of IC/BPS is now better understood; and as well as epithelial inflammatory histopathology and chemistry,^{6–8} the presence of aberrant peripheral and central nerve function in this condition is well documented.^{9–12} However, a complete understanding of the autonomic control of the human bladder is still elusive.^{8,13} The divergent nature of the autonomic nervous system in the pelvic region creates a complex pattern of altered neurophysiology, and evidence has been documented to show the presence of cross-system viscera-visceral interactions, that is, bladder inflammation having a direct influence on uterine or colon contractions/inflammations, and vice versa.¹⁴ This helps to partially explain the multifactorial nature of IC/BPS and the frequent comorbidity of it with irritable bowel syndrome and others. Historically, this relationship was reported by Pottenger¹⁵ as early as 1925.

This condition has reportedly been treated by an array of allopathic medicines and herbal remedies, as well as sacral nerve stimulation, physical therapy, and abdominal ischemic compression.^{16–19} We theorized that IC/BPS may have an association with low back pain; and for a number of patients, the condition may subside when their low back condition improves. It is unknown if there is a casual relationship, but the potential impact of IC/BPS on patients' lives suggests that we consider a potential relationship.

Both intervertebral disk prolapse and cervical spondylosis have been reported as etiological factors for urinary tract dysfunction.²⁰ A previous international study of chiropractic patients had observed that 6% of patients with low back pain experienced an improvement in urinary system symptoms after treatment.²¹ Another article reports that 9 out of 10 IC/BPS patients have reductions in symptoms following surgery for compression of the spinal nerves by prolapsed disk material at the L5/S1 intervertebral foramina.²² Browning²³ described similar improvement in a patient receiving chiropractic care for an L5/S1 disk protrusion with subsequent S2/3 nerve root impairment. Improvement in a patient with total loss of both bowel and bladder control has also been documented.²⁴

Although we are aware of another case study reporting chiropractic care of incontinence in the elderly population,²⁵ we wished to evaluate the occurrence of urinary problems in a wider age group of chiropractic patients. Therefore, the purpose of this case series is to describe BPS findings for patients with IC/BPS who responded positively under chiropractic care. This report also proposes an etiology for the IC/BPS and a rationale for including spinal manipulation as part of treatment of these conditions.

Case reports

Case files were drawn from 2 separate chiropractic clinics in Scotland. Cases were selected if they had urinary problems that appeared to worsen when they had mechanical spinal problems and if they responded positively to chiropractic care. We tried to include the range of patients affected by this condition and so included different age and sex as well as differences in signs and symptoms when selecting the patient cases. All patients gave their permission for their anonymous data to be included in the study (Table 1).

Case 1

This 57-year-old man presented with low grade back pain since his teens and with sporadic bouts of severe pain. These episodes of pain were accompanied by increased frequency of urination and nocturia. Exacerbating factors for his low back pain were heavy lifting/manual work, and he reported morning low back stiffness. Previous treatment had included physiotherapy and manipulation. His general health was good apart from his need to take medication for hypertension. He reported having had no prior major trauma.

On examination, right FABERE test caused sacroiliac pain, straight leg raise caused pain bilaterally at 60°, and hamstrings were tight bilaterally. Spinal palpation findings indicated altered motion at the right SI joint and the L4/5 and L5/S1 motor units. Treatment included diversified spinal manipulative therapy (SMT), high-velocity drop techniques, and soft tissue treatment. After 6 treatments over a 2-month period, his low back condition was stable and his urinary problems resolved. Five months later, he exacerbated his low back condition while erecting fencing posts, which resulted in the return of his urinary problems. Further chiropractic care appeared to resolve his low back and urinary complaints in 4 weeks.

Table 1 Summary of cases

Case	Age	Sex	Duration of LBP (y)	Duration IC/PBS (y)	Initiating trauma	Areas of spinal dysfunction	Timing of IC/PBS symptoms relative to LBP episodes	Bladder symptoms
1	57	M	40	40	None reported	L4/5/S1 right SI	After low back	Nocturia and frequency
2	49	M	15	2	Very heavy lifting	T10/11/12 L4/5/S1 bilateral SI	After low back	Dysuria
3	57	F	30	38	Major fall as child	Occ/C1/2 L4/5/S1 bilateral SI	Before or with low back	Generalized cystitis
4	26	F	6	2	Childbirth under epidural	Bilateral SI	Concurrent with LBP	No bladder sensation
5	52	F	1/12	1	None reported	Occ/C1/2 C5/6 T4/5/6 L4/5/S1 Left SI	Precedes LBP	Incontinence
6	76	F	50	1	Fall	L3/4 L5/S1	Comes together but bladder improves first	Urgency
7	55	F	21	28	None	Right SI	Improves first	Generalized cystitis
8	45	F	7	3	None	L2-S1 Bilateral SI	Precedes LBP	Generalized cystitis

Case 2

A 49-year-old man presented with a 15-year history of low back pain. His complaint was initiated by very heavy work. Following his initial recovery, he had regular episodes of low back pain with increasing frequency, but not severity. Symptoms of difficulty urinating developed 2 years after his initial spinal problem; however, the results of all urological investigations were normal. His general health was good, although his blood pressure was slightly elevated and he was taking no medication at the time of initial consultation. On examination there was a decrease in cervical rotation bilaterally, together with decreased motion of both SI joints and the L4/5/S1 and T10/11/12 motor units.

After chiropractic care consisting of SMT, bladder symptoms resolved within 24 hours of the low back pain easing. However, over the following 14 years, with approximately 3 episodes of back pain per year, his urinary symptoms changed from occurring whenever back pain was present to every fourth or fifth episode. A similar picture of changing symptomatology of chronic low back pain has been reported.²⁶

Case 3

A 57-year-old woman presented with a 30-year history of spinal pain and headaches and a 38-year history of cystitis. She had a fall at the age of 8, which was before she had any musculoskeletal symptoms. There had been no prior treatment of the spinal problems, although she had been seen by her family

doctor and had low back radiographs taken and she had received courses of antibiotics for the cystitis. Her general health was good other than recurrent sore throats, and she was taking no regular medication.

Examination showed anterior head carriage, limitation of the cervical ranges of motion in bilateral rotation and extension, and right lateral flexion of the lumbar spine. Spinal palpation revealed restricted motion of the Occ/C1/C2 and L4/5/S1 motor units and both SI joints.

Treatment included SMT over 2 months. The results were improved ranges of motion throughout the spine, and the pain and headaches were reduced. Bladder symptoms disappeared, and the patient became asymptomatic. However, over the last 9 years, she had frequent bouts of spinal pain that coincided with the return of the urinary problems. Urinary symptoms would resolve within 24 hours of chiropractic care for this patient.

Case 4

A 26-year-old woman presented with a 6-year history of low back pain. Two years ago, the pain worsened when she had an epidural injection during the birth of her first child. At that time, she lost bladder sensation. This resulted in losing the stimulus to urinate; and she reported that, "after the birth she passed three times the normal volume of urine." Since then, she had little bladder sensation and was told her bladder had been overstretched, resulting in a need to visit the toilet very frequently. Her general health was good, and she was taking no regular medication.

On physical examination, deep tendon reflexes appeared to be heightened bilaterally with some mild

clonus. Cerebellar balance tests were positive, and the patient was referred back to her medical practitioner for further tests. Following inconclusive investigations, she returned for chiropractic treatment. Spinal examination showed hypermobility throughout the thoraco/lumbar junction and restricted motion of both SI joints.

Treatment included SMT and trigger point therapy to paravertebral muscles. Spinal function improved; low back pain was reduced; and she reported a return to normal bladder sensation after the second treatment, which was maintained.

Case 5

A 52-year-old woman presented with a history of thoracic pain and a 1-month history of low back/anterior thighs pain and heaviness in her legs. She concurrently had a 1-year history of intermittent morning bladder incontinence. Her medical history included celiac disease diagnosed 13 years ago and left facial neuralgia with noticeable ptosis of 10 years' duration. She had self-managed her conditions with lifestyle and diet alterations, and she was taking no regular medication other than over-the-counter analgesics.

Gait examination showed a plodding gait, but cerebellar test results were normal. All reflexes were normal. Quadriceps muscles were weak bilaterally (grade 4), and the left femoral stretch was restricted but not painful. Spinal palpation showed altered motion at Occ/C1/2, C5/6, T4/5/6, L4/5/S1, and left SI joint.

Chiropractic care consisted of SMT, soft tissue work, ergonomic advice, and exercises. Response to treatment was slow but sustained over 3 years. Bladder improvement followed the increase in spinal function. For this patient, incontinence occurs as a precursor to her low back or leg symptoms. Presently, symptoms begin to reoccur after about 3 months without chiropractic care.

Case 6

A 76-year-old woman presented with a 50-year history of episodic low back pain. After a fall onto her coccyx, she developed bilateral anterior leg pain. She received physiotherapy for this and reported that, after one treatment, where she heard an articular release, she had felt the need to urinate. Her general health was good, and she was taking no regular medication.

Gross movement in the low back and pelvis was limited by pain and muscle spasm. Spinal palpation indicated restricted motion at L3/4 and L5/S1 motor units.

Treatment included using SMT and soft tissue therapy with advice to take regular short walks each day. The bladder symptoms improved within 1 week of initiating treatment, which was 2 weeks before improvement in leg pain.

Case 7

A 55-year-old woman presented with a 28-year history of vulva/perineum symptoms (increased tension and sensitivity, altered bladder and rectal function) that began after an episiotomy and 2 subsequent perineal repairs. Following a twisting injury, she developed right buttock pain; and her vulva/perineal symptoms were exacerbated. Her general health was good, and a nasal spray for allergic rhinitis was her only medication.

Examination demonstrated normal ranges of motion throughout the spine, but the pelvis articulations were restricted. Pelvic palpation showed the sacrum and right SI joint dysfunction right piriformis and gluteal muscles were hypertonic.

Treatment included SMT, sacral adjusting, and abdominal visceral mobilization weekly for 1 month. After 1 month, she noticed improvement in bladder and rectal function and an easing of tension in the right buttock. Nine treatments over the following 11 months resulted in resolution of all symptoms and improved pelvic function.

Case 8

A 45-year-old woman presented with a 15-year history of neck pain that developed after a thyroid operation to remove a malignant tumor. She reported that her neck pain responded well to chiropractic care. Seven years prior, she developed low back and right leg pain, with episodic exacerbations and paresthesia in her feet. Three years prior, she developed bladder problems. Treatment included courses of antibiotics; cystoscopy, the result of which was reported as normal; and ultrasound scanning of the kidneys and bladder. A small stone was detected in the left kidney, but thought not to be relevant to her bladder symptoms. Two further ultrasound scans were performed for recurrence of bladder symptoms, but nothing additional was detected.

Chiropractic examination and motion palpation of the lumbo/pelvic area showed restricted motion in the sacroiliac joints and all the lumbar motor units except L1/2.

Treatment included diversified SMT and drop techniques and slow stretch (SacroOccipital Technique) blocking. The patient reported resolution of both somatic and visceral symptoms after 8 treatments. However, they reoccurred on 3 occasions but seem to reduce with chiropractic treatment.

Discussion

Although recent advances suggest that IC/BPS may be a form of neurogenic inflammation,²⁷ the present understanding of the neurology involved in bladder function is complex and still incomplete.⁸ It is known that the detrusor muscle is innervated by parasympathetic nerves that come from the pelvic plexus and have preganglionic fibers originating from the S2 to 4 sacral roots. There is little direct evidence of sympathetic fibers in the detrusor muscle; but the bladder base and the urethral smooth muscle receive such innervation, and it is thought that inhibition does occur at the pelvic and hypogastric plexus by nerves that originate at the T10 to L2 level (these sympathetic inputs are responsible for bladder relaxation reflexes that occur when there is either bladder or anorectal distension).⁸ Also there is strong evidence that the urothelium, which is highly innervated, acts as a moderating influence on bladder control and that its barrier properties change with neuronal stimuli.²⁸ We believe that it is possible to cause irritation to the sacral nerve roots at or around the lumbosacral junction leading to an alteration of the parasympathetic supply to the bladder and urethra. This in turn may lead to an alteration of the normal physiological and biochemical processes in the tissues of the area. This irritation may remain localized to the bladder, or it may help to cause and explain the comorbidity of other pelvic problems through its interference with the divergent autonomic nervous system in the pelvic region.^{29,30}

Modern laboratory techniques have resulted in studies of the somatosensory nervous systems in recent years. The neurons innervating the detrusor muscle are located in the dorsal columns (ascending neurons) and the lateral columns (descending neurons). However, the divergent nature of these somato-visceral sensory pathways has become a cornerstone for the understanding of the complexity of IC/BPS. In primates, for example, less than 10% of ascending spinal fibers terminate in the thalamus. Anterolateral fibers synapse not only in many parts of the thalamus but also throughout the brainstem and cerebellum, and

a similar pattern is reflected for the fibers from the dorsal column and solitary nucleus. These systems are extensively interconnected, and the interconnections are reflected for all rostral sensory neurons.¹⁴ This means that the effect of sensory irritation in the bladder is projected to many areas of the brain³¹; and because these areas of the brain also receive input from all other pelvic viscera, the pelvic organs are inherently linked neurologically.¹⁴

This divergent nature of sensory input leads to a convergence of sensory inputs from various viscera in localized areas of the higher centers. In short, sensory stimulation of the bladder has an effect on the threshold levels of many structures in the brain; and conversely, these same structures are influenced by more than one visceral organ. Functional brain studies have demonstrated clear differences in the central nervous system control of the bladder in incontinent patients.³²

It is this convergent/divergent property of the visceral nervous system that mimics the recent concept of central sensitization of pain, which means that pain is not perceived by stimulation of one pathway but by the summation of the inputs of many pathways by the central nervous system: "in this situation a noxious event in the peripheral bodily tissues triggers ... a long lasting sensitisation of recipient spinal neurones that can continue long after the initial peripheral injury or pathology has healed."¹⁴ The pain of painful bladder syndrome may be of a similar nature; and in some cases, an irritation of the sacral nerve roots in the cauda equina may be a contributing factor to the recurrent nature of the condition.

Modern urology has acknowledged the role of the altered nervous system in urinary dysfunction for some time now. Snooks et al³³ describe how traction neuropathy of the pelvic nerves has caused urethral incompetence; and Torrens in his review of "Disorders of Micturition" goes as far as to state, "There is a neuropathic element in almost every form of urinary dysfunction."³⁴

One form of urinary dysfunction is contractile overactivity of the bladder, which causes spontaneous contractions during filling, which the patient cannot inhibit. If there is evidence of neuropathy, it is called *detrusor hyperreflexia*; but without evidence of neuropathy, it is simply labelled *unstable*. Bladder denervation has been used for many years to control such hypercontractility.³⁴ And in a similar way, epidural electrode stimulation of the spinal cord has also been shown to decrease bladder contractility.³⁵ These treatments highlight the link between spinal nerves

and bladder function, and we believe that it is possible that altered lumbosacral spinal mechanics may influence bladder/urethra function by a similar mechanism. Therefore, we propose the possibility that spinal adjusting may help correct altered neurological function in some IC/BPS patients.

Limitations

The limitations of this retrospective series include selection and reporting bias when reviewing case histories from our own clinical setting. Also, there is a risk of overattributing a causal relationship between 2 separate clinical entities. Without further studies, it is impossible to confirm or rule out the proposed relationship.

It would be interesting to undertake further studies into the incidence of bladder dysfunction in confirmed cases of disk protrusion and, similarly, the incidence of spinal dysfunction in cases of IC/BPS. Also, it would be appropriate to do a pilot study on painful bladder syndrome patients who were evaluated and treated for spinal dysfunction.

Conclusion

This case series highlights that a common condition, IC/BPS, may be associated with spinal dysfunction. Some of the chiropractic patients who had chronic spinal conditions had reoccurrence of bladder symptoms during an exacerbation of mechanical spinal problems. This suggests that there may be an association between the symptoms and spinal dysfunction.

Funding sources and potential conflicts of interest

No funding sources or conflicts of interest were reported for this study.

References

1. Clemens JQ, Joyce GF. Interstitial cystitis and painful bladder syndrome. In: Litwin MS, Saigal CS, editors. *Urologic diseases in America*. National Institute of Diabetes and Digestive and Kidney Diseases, Dept of Health and Human Services, Public Health Service. Washington, DC: U.S. Govt Printing Office; 2007.
2. Parsons JK, Kurth K. Epidemiologic issues in interstitial cystitis. *Urology* 2007;69(4):s5-8.
3. Lowell Parsons C. The role of the urinary epithelium in the pathogenesis of interstitial cystitis/prostatitis/urethritis. *Urology* 2007;69(4):s9-s16.
4. Interstitial cystitis/painful bladder syndrome. National institute of Diabetes and Digestive and Kidney Diseases, Dept of Health and Human Services. Bethesda: National Kidney and Urologic Diseases Information Clearinghouse; 2008.
5. van de Merwe JP, Nordling J. European Society for the study of Interstitial Cystitis (ESSIC). *Eur Urol* 2008;53(1):60-7.
6. Lowe EM, Anand P, Terenghi G, Williams-Chestnut RE, Sinicropi DV, Osborne JL. Increased nerve growth factor levels in the urinary bladder of women with idiopathic sensory urgency and interstitial cystitis. *Br J Urol* 1997;79: 572-7.
7. Pang X, Marchand J, Sant GR, Kream RM, Theoharides TC. Increased number of substance P positive nerve fibres in interstitial cystitis. *Br J Urol* 1995;75:744-50.
8. Griffiths DJ, Apostolidis A. Neurological control of the bladder in health and disease. In: Fowler CJ, Panicker JN, Emmanuel A, editors. *Pelvic organ dysfunction in neurological disease*. Cambridge: Cambridge press; 2010. p. 1-24.
9. Bon K. Involvement of the dorsal paratrigeminal nucleus in visceral pain-related phenomena. *CR Acad Sci* 1997;320(8): 607-13.
10. Steers WD, Tuttle JB. Mechanisms of disease: the role of nerve growth factor in the pathophysiology of bladder disorders. *Nat Clin Pract Urol* 2006;3(2):101-10.
11. Ustinova EE, Fraser MO, Pezzone MA. Colonic irritation in the rat sensitizes urinary bladder afferents to mechanical and chemical stimuli: an afferent origin of pelvic organ cross-sensitization. *Am J Physiol Renal Physiol* 2006;290(6): 1478-87.
12. Coderre TJ, Katz J. Contribution of central neuroplasticity to pathological pain: review of clinical and experimental evidence. *Pain* 1993;52:259-85.
13. Bradley WE, Yang C. Autonomic regulation of urinary bladder. In: Low P, editor. *Clinical autonomic disorders*. 2nd ed. Philadelphia: Lippincott-Raven; 1997. p. 117-26.
14. Berkley KJ. A life of pelvic pain. *Physiol Behav* 2005;86(3): 272-80.
15. Pottenger FM. The urogenital tract. In: *Symptoms of visceral disease*. 3rd edition. London: Henry Kimpton; 1925. p. 359-60.
16. Herbison P, Hay-Smith J, Ellis G, Moore K. Effectiveness of anticholinergic drugs compared with placebo in the treatment of overactive bladder: systematic review. *BMJ* 2003;326: 841-4.
17. Chapple CR, Khullar V, Gabriel Z, Dooley JA. The effects of antimuscarinic treatments in overactive bladder: a systematic review and meta-analysis. *Eur Urol* 2008;54:543-62.
18. Shafik A, Shafik IA. Overactive bladder inhibition in response to pelvic floor muscle exercises. *World J Urol* 2003;20:374-7.
19. Hains G, Hains F, Descarreaux M, Bussieres A. Urinary incontinence in women treated by ischemic compression over the bladder area: a pilot study. *J Chiropr Med* 2007;6(4): 132-40.
20. Norris JP, Staskin DR. History physical examination and classification of neurogenic voiding dysfunction. *Urol Clin North Am* 1996;23:337-43.

21. Leboeuf-Yde C, Pedersen E, Bryner P, Cosman D, Hayek R, Meeker W, et al. Self-reported nonmusculoskeletal responses to chiropractic intervention: a multinational study. *J Manipulative Physiol Ther* 2005;28(5):294-302.
22. Gillespie L, Bray R, Levin N, Delamarter R. Lumbar nerve root compression and interstitial cystitis—response to decompressive surgery. *Br J Urol* 1991;68(4):361-4.
23. Browning JE. Mechanically induced pelvic pain and organic dysfunction in a patient without low back pain. *J Manipulative Physiol Ther* 1990;13(7):406-11.
24. Kamrath KR. Chiropractic management of a 5 yr old boy with urinary and bowel incontinence. *J Chiropr Med* 2010;9(1):28-31.
25. Zhang J, Haselden P, Tepe R. A case series of reduced urinary incontinence in elderly patients following chiropractic manipulation. *J Chiropr Med* 2006;5(3):88-91.
26. Papageorgiou AC, Croft PR, Thomas E, Ferry S, Jayson MIV, Silman AJ. Influence of previous pain experience on the episode incidence of low back pain: results from the South Manchester Back Pain Study. *Pain* 1996;66:181-5.
27. Jasmin L, Janni G. Experimental neurogenic cystitis. *Adv Exp Med Biol* 2003;539:319-35.
28. Birder LA, De Groat WC. Mechanisms of disease: involvement of the urothelium in bladder dysfunction. *Nat Clin Pract Urol* 2007;4(1):46-54.
29. Winnard KP, Dmitrieva N, Berkley KJ. Cross-organ interactions between reproductive, gastrointestinal and urinary tracts: modulation by estrous stage and involvement of the hypogastric nerve. *Am J Physiol Regul Integr Comp Physiol* 2006;291(6):R1592-601.
30. Marinkovic S, Moldwin R, Gillen LM, Stanton SL. The management of interstitial cystitis or painful bladder syndrome in women. *BMJ* 2009;339:337-9.
31. Kuhtz-Buschbeck JP, Gilster R, Van der Horst C, Hamann M, Wolff S, Jansen O. Control of the bladder sensations: an fMRI study of brain activity and effective connectivity. *Neuroimage* 2009;47:18-27.
32. Griffiths D, Tadic SD. Bladder control, urgency, and urge incontinence: evidence from functional brain imaging. *Neurourol Urodyn* 2008;27(6):466-74.
33. Snooks SJ, Barney PRH, Swash N. Damage to the innervation of the voluntary anal and periurethral striated sphincter musculature in incontinence: an electrophysiological study. *J Neurol Neurosurg Psychiatry* 1984;47:1269-73.
34. Torrens MJ. Disorders of micturition. In: Swash M, Oxbury J, editors. *Clinical neurology*, vol. 1. London: Churchill-Livingstone; 1991. p. 602-14.
35. Illis LS, Sedgwick EM, Glanville HJ. Stimulation procedures. *Rehabilitation of the neurological patient*. Oxford Blackwell; 1982. p. 384-410.