W15: Neuromodulation in complex pelvic floor disorders

Workshop Chair: Sohier Elneil, United Kingdom

20 October 2014 14:00 - 18:00

Start	End	Topic	Speakers
14:00	14:20	Overview of pelvic floor diosrders	• Sohier Elneil
14:20	14:50	Neuromodulation: Proof of Concept	• Michele Spinelle
14:50	15:10	PTNS: Managing Complex Pelvic Floor Disorders	• Alex Digesu
15:10	15:30	PTNS: Managing the Neurological and Non-neurological Patient with Bladder Dysfunction	• Jalesh Panicker
15:30	16:00	Break	None
16:00	16:25	Sacral Neuromodulation: Managing Intractable Bladder Dysfunction and Chronic Pelvic Pain Syndrome	• Daniel Engeler
16:25	16:50	Pudendal Neuromodulation: Managing the Patient who did not respond to Sacral Neuromodulation	• Michele Spinelle
16:50	17:15	Sacral Neuromodulation: Managing Bowel and Sexual Disorders	• Sohier Elneil
17:15	17:45	"Hands On" Neuromodulation using Models and Videos	All
17:45	18:00	Discussion	All

Aims of course/workshop

The aim is to explore the neurological basis of complex pelvic floor disorders and to provide guidance for management and treatment using different types of neuromodulation. The objectives are: 1. To understand the neurology of the pelvic floor in complex pelvic floor disorders like intractable OAB/DO, chronic pelvic pain and faecal incontinence. 2. To comprehend how neuromodulation works. 3. To look at the role of Posterior Tibial Nerve Stimulation (PTNS), Sacral Neurmodulation (SNM) and Pudendal Neuromodulation (PNM) in the management of complex pelvic floor disorders. 4. To practice PTNS, SNM and PNM techniques on provided models.

WORKSHOP 15

NEUROMODULATION IN COMPLEX PELVIC FLOOR DIORDERS

Chairperson: Miss Sohier Elneil

Introduction

The pelvic floor is highly complex structure made up of skeletal and striated muscle, support and suspensory ligaments, fascial coverings and an intricate neural network. Its dual role is to provide support for the pelvic viscera (bladder, bowel and uterus) and maintain functional integrity of these organs. In order to maintain good pelvic floor function, this elaborate system must work in a highly integrated manner. When this is system if damaged, either directly or as a consequence of an underlying neurological condition, pelvic floor failure ensues along with organ dysfunction.

The aetiology is inevitably multi-factorial, and seldom as a consequence of a single aetiological factor. It can affect one or all three compartments of the pelvic floor, often resulting in prolapse and functional disturbance of the bladder (urinary incontinence and voiding dysfunction), rectum (faecal incontinence), vagina and/or uterus (sexual dysfunction). This compartmentalisation of the pelvic floor has resulted in the partitioning of patients into urology, gynaecology, colo-rectal surgery or neurology, depending on the patients presenting symptoms. In complete pelvic floor failure, all three compartments are inevitably damaged resulting in apical prolapse, with associated organ dysfunction. It is clear that in this state, the patient needs the clinical input of at least two of the three pelvic floor clinical specialities. Whilst the primary clinical aim is to correct the anatomy, it must also be to preserve or restore pelvic floor function. As a consequence, these patients need careful clinical assessment, appropriate investigations, and counselling before embarking on a well-defined management pathway. The latter includes behavioural and lifestyle changes, conservative treatments, pharmacotherapy, minimally invasive surgery, and radical specialised surgery.

It is not surprising that in this complex group of patients, a multidisciplinary approach is not only necessary, but critical, if good clinical care and governance is to be ensured. But it is of significant import that one has a good understanding of the neurology of the pelvis and its organs.

Aims and Objectives of the Workshop

- Current concepts relating to the neurological control of the bladder and the pelvic floor.
- Innovative therapies in treating neurogenic bladder and pelvic floor disorders: Indications and limitations of PTNS
- Innovative therapies in treating neurogenic bladder and pelvic floor disorders: Indications and limitations of sacral/pudendal neuromodulation

Overview of Neurology of the Pelvic Floor

Voluntary control over the uro-genital system is critical to our social existence. Since its peripheral innervation derives from the most distal segments of the spinal cord, integrity of the long tracts of the central nervous system for physiological function is immediately

apparent. In a survey of the site of the underlying neurological disease affecting a sample of patients referred to the department with bladder symptoms, spinal cord involvement of various pathologies was found to be the commonest cause of bladder symptoms.
Because of the commonality of innervation shared by the bladder and genital organs, it might be expected that abnormalities of these two systems inevitably occur together. This however is not the case because although the organs share the same root innervation and have common peripheral nerves within the pelvis, each is controlled by its own unique set of central nervous system reflexes.

Voluntary control of micturition is based on a complex neural circuitry highly distributed on different levels of the nervous system. A variety of neurotransmitters are involved in signalling of neural control. Understanding the pathways involved at the level of the brain, the spinal cord and the peripheral nervous system as well as the peripheral organ is important for the physician diagnosing and treating patients with neurogenic bladder and pelvic floor dysfunction. Diseases or injuries to this complex system may lead to abnormal function of the end organs, i.e. leading to pathologic storage or release of urine. Disruption of the normal neural pathways has different specific functional consequences in the lower urinary tract as well as the pelvic floor. Cerebral lesions, multiple sclerosis, Parkinson's disease and trauma to the nervous system at different levels, such as the brain, spinal cord, or cauda equina are therefore followed by a variety of functional disturbances, which can be derived from the pathways involved. Both, current concepts relating to the normal neurological control of the bladder and the pelvic floor, as well as disease or trauma specific pathologies are discussed here.

In this workshop, a brief account of the neurophysiological control of the bladder and pelvic is given initially, followed by a description of the effect that neurological disease at different levels of the nervous system may have and finally the management of those conditions.

Complex Pelvic Floor Disorders in Urogynaecology

In Urogynaecology and Female Urology clinicians tend to focus on urinary incontinence (both stress and urge) and pelvic organ prolapse. But in a significant proportion of patients these conditions become intractable and difficult to treat because of failure to respond to standard therapies, failure to respond to standard surgical techniques and other associated conditions, such as diabetes, neurological pathology and radical surgery which may impact directly on the condition.

The peripheral innervation of the pelvic organs can be damaged by extirpative pelvic surgery such as resection of rectal carcinoma, radical prostatectomy in men, or radical hysterectomy. The dissection necessary for rectal cancer is likely to damage the parasympathetic innervation to the bladder and genitalia, as the pelvic nerves take a medio-lateral course through the pelvis either side of the rectum and the apex of the prostate. The nerves may either be removed together with the fascia which covers the lower rectum or may be damaged by a traction injury as the rectum is mobilized prior to excision.

Urinary incontinence following radical hysterectomy which includes the upper part of the vagina, is probably also due to damage to the parasympathetic innervation of the detrusor and in the case of a radical prostatectomy, there may be additional direct damage to the innervation of the striated urethral sphincter Therapies to manage these conditions

depend on a multi-disciplinary approach. This workshop will help guide practitioners on how to maximise the therapeutic options for their patients.

Neuromodulation: Proof of Principle including Anatomy and Neurology of the Sacrum

Please see attached PDF from Dr Michele Spinelli

Neuromodulation (PTNS) in the Intractable Bladder and Pelvic Floor Dysfunction

Pelvic floor disorders such as lower urinary tract symptoms (LUTS), anal incontinence and sexual dysfunctions are common disorders. Urgency represents the most bothersome LUTS and severely affects the quality of life (QOL). Neurogenic detrusor overactivity, detrusor sphincter dyssynergia and/or detrusor underactivity are the most common cause of LUTS in neurogenic patients. These bladder abnormalities tend to become more severe with the progression of the disease leading to voiding difficulties, urinary retention, recurrent urinary tract infections and need of clean intermittent self-catheterization. Drugs, surgery and repeated intradetrusor injections of botulinum toxin have been suggested as therapeutic options. However, neurological and non-neurological patients can fail to respond to drug therapy, report intolerable side effects and/or are reluctant to invasive surgical treatment.

Neuromodulation is a mechanism by which the nervous system regulates electrical impulses flowing through neural tissues. Percutaneous tibial nerve stimulation (PTNS), a minimally invasive neuromodulation technique, is able to modify the lower urinary tract behaviour by inhibiting involuntary detrusor contractions in patients with both neurogenic and idiopathic detrusor overactivity in an outpatient setting.

PTNS has been demonstrated to be an effective, safe and well tolerated treatment in both neurogenic and non-neurogenic patients affected by LUTS and unresponsive to anticholinergic drugs. Both subjective and objective improvement has been reported. A statistically significant improvement of patient perception of bladder condition, overactive bladder (OAB) symptoms, mean voided volume per micturition, post micturition residual and QOL parameters have been reported.

The mechanism of action of PTNS is not completely understood yet. Long-latency somatosensory evoked potentials (LL-SEP) are well known to reflect information processing in the brain after stimulation of peripheral somatosensory system. Some authors found a modification of brain activity after PTNS and speculated that its efficacy is mediated by sacral and suprasacral centres of stimulus elaboration involving cortical associative areas.

Considering its high safety, ease of use, lack of side effects and office-based convenience, PTNS could be consider as an ideal alternative treatment for neurogenic patients suffering from LUTS, especially taking into account the lack of scientific evidence of anticholinergic efficacy in this group of patients.

PTNS has been also demonstrated to be clinically effective in the treatment of other pelvic floor disorders such as anal incontinence and sexual dysfunction, but it has not been fully evaluated. The main limitation of PTNS remains its longevity of action, the need for

dedicated personnel and the need for dedicated facilities. A new version of this device is likely to have a more long-term impact.
Please see attached PDF from Dr Alex Digesu and Dr Jalesh Panicker

Neuromodulation (SNM and PNM) in Intractable Pelvic Floor and Bladder Dysfunction

Elneil: Electrical neuromodulation of the lower urinary tract began over a century ago, but it was the pioneering work of Tanagho and Schmidt at the University of California in the late 1980s that demonstrated electrical activation of efferent fibres to the striated urethral sphincter inhibited detrusor contractions [1]. Stimulation of the third sacral root (S3) has been shown to be effective in stimulating the urethral sphincter [2]. It became evident that sacral neuromodulation may thus restore voiding in women with chronic urinary retention [3], by resetting brainstem function [4]. This was first described in the mid-1990s.

Though the mechanism of action of SNM remains indeterminate, there are various theories based on careful observations. Two components have been identified (i) activation of efferent fibres to the urethral sphincter with negative feedback to the bladder (pro-continence reflex) and (ii) activation of sacral spinal afferents resulting in inhibitory reflex efferent activity to the bladder. Reflex pathways at the spinal cord and supra spinal levels are thought to be modulated to achieve these effects [5, 6]. The prolonged beneficial effects of the stimulator, after it is switched off, support this observation. In urinary retention, SNM is postulated to interfere with the inhibitory afferent activity arising from the urinary sphincter and thus restoring the sensation of bladder filling and the ability to void [7].

In patients with overactivity of the bladder, at a central level, decreases in regional cerebral blood flow measured by PET scanning was demonstrated in the cingulate gyrus, orbitofrontal cortex, midbrain and adjacent midline thalamus in chronically implanted patients with urge incontinence [5]. SNM appears to restore activity associated with brainstem auto regulation and attenuation of cingulate activity [6, 8], critical to bladder function. Therefore, paradoxically SNM can be a treatment for both intractable incontinence and retention.

SNM is not without its complications and need for revision surgery. Therefore, it is important that patients are counselled regarding failure of the procedure (25%), the significant revision rate (15-50%), and the risk of box site pain, sciatica and nerve injury.

The most important determinant of success in bladder dysfunction and other pelvic floor symptoms (including pelvic pain syndromes, sexual dysfunction and bowel dysfunction) is the careful selection of the patient. This includes a urological and gynaecological history, pelvic examination to rule out surgical correctable causes and urine assessment to rule out infection and haematuria. We advocate the use of frequency-volume charts, urodynamic evaluation where indicated, post void residuals if they are able to void at all and quality of life questionnaires to qualify the degree of improvement before and after the procedure.

In the last decade there has been a plethora of innovative neuromodulation devices for treatment of lower urinary tract symptoms and pelvic floor dysfunction, though sacral neuromodulation remains the most widely used form of peripheral neuromodulation. In this lecture, a review of the role of pudendal neuromodulation and sacral dermal neuromodulation devices will also be considered. Their place in an algorithm of bladder and pelvic floor management will be rationalised.

Engeler: Chronic neuromodulation using the sacral route via S3 and S4 (SNM) is part of most routine treatment algorithms for refractory lower urinary tract dysfunction (LUTD), including overactive bladder syndrome (OAB), and non-obstructive urinary retention. In this

regard, SNM is a minimally invasive, reversible therapy with the potential of restoring normal lower urinary function and may be considered after failure of conservative treatment options.

Although the mechanism of action of SNM is still not well understood, it is now widely accepted, that it involves modulation of spinal cord reflexes and brain pathways by peripheral afferents rather than direct stimulation of motor responses of the bladder or urethral sphincter. Therefore, a partially intact afferent and efferent nervous system is necessary for the treatment success. In patients with non-obstructive urinary retention, SNM has been postulated to inhibit inappropriate activation of the “guarding reflex” facilitating voiding by interruption of the excitatory outflow of the urethral sphincter. However, in patients with a primary disorder of urethral sphincter relaxation (Fowler’s syndrome) it probably has a more important effect on detrusor contractility than on the non-relaxing sphincter, which is still overactive under SNM. In contrast, in patients with urgency-frequency syndrome resulting from detrusor overactivity, SNM is thought to inhibit detrusor activity, probably at the level of the spinal cord.

Most often SNM is performed in two phases including an evaluation with acute and subchronic neuromodulation and a treatment period with permanent implant based neuromodulation. The test phase can either be performed using temporary electrodes followed by a single-stage implantation or by sequential two-stage implantation of quadripolar electrodes and impulse generator. The use of the two-stage implantation technique has been shown to improve treatment response [1]. Reported long-term success rates vary from 50 to 80% after positive testing [2]. SNM for OAB and non-obstructive urinary retention is also effective over the long term [3].

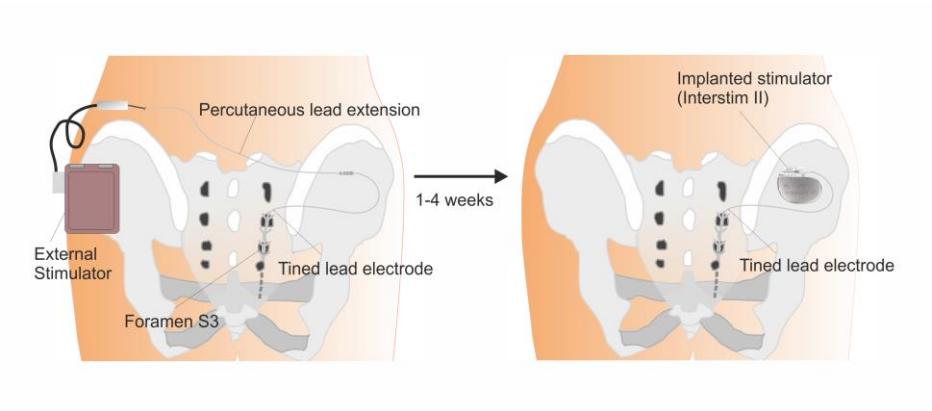

Figure 1. Two stage implantation

Despite lack of randomized trial showing the efficacy for neurogenic lower urinary tract dysfunction, SNM also might be effective in neurogenic LUTD. In a recent systematic review and meta-analysis, we found a pooled success rate of 68% for the test phase and of 92% for the permanent SNM for all neurogenic conditions [4]. Although these results must be interpreted with caution because of the lack of randomized-controlled trials, it is suggesting a potential success rate in the range of non-neurogenic indications.

SNM may also be an attractive treatment option for urinary urgency incontinence combined with faecal incontinence (FI) - so called “double incontinence”. Over the last years,

SNM treatment of FI has been widely used and has a reported success rate of 80% after permanent implant at 7 years [5]. In some cases, both conditions can be treated with one implant. Although, many patients with FI will search help mainly for this problem, the prevalence of urinary incontinence in this population is very high [6].

For chronic pelvic pain syndrome (CPPS), SNM may be considered as part of a broader management plan. It is thought to modulate central nociceptive pathways. At the moment, only limited recommendations can be given for the use of SNM for CPP because of the lack of high evidence studies [7]. Despite this, in experienced hands and selected patients it may be a useful treatment option especially for a “urology” phenotype of CPP including relevant LUTS.

References:

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Please see attached PDF from Dr Michele Spinelli

Take Home Message

-Neurological basis of bladder and pelvic floor dysfunction is essential to all practitioners

-In complex pelvic floor disorders in patients, practitioners should investigate all aspects of bladder and pelvic floor dysfunction
-Different therapeutic options should be made available and discussed with all patients

References

1. Tanagho, E.A. and R.A. Schmidt, *Electrical stimulation in the clinical management of the neurogenic bladder.* J Urol, 1988. **140**(6): p. 1331-9.
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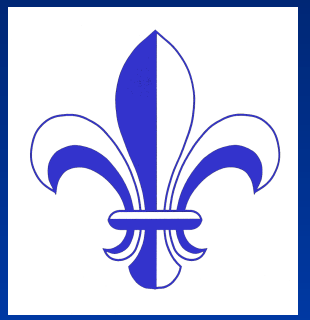

Complex pelvic floor dysfunction

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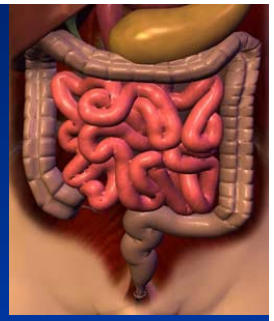
Complex Pelvic Floor dysfunction

- Bowel Disorders
- Urogenital pain
- Non relaxing pelvic floor dysfunction/ Levator myalgia
- Sexual Dysfunction

Fecal incontinence



- Uncontrolled loss of faeces (liquid or solid) from the bowel
- It may occur passively (without the person being aware of passing faeces) or be preceded by urgency

1. damage of the anal sphincter - trauma
2. damage of nerve supply – neurological diseases
3. no sphincter causes – dementia, laxatives

ASSESSMENT

BOWEL HABIT DIARY

Please record all of your bowel movements by completing the form below:-

NAME _____

Day/Date And Time	Consistency e.g. Pellets/soft/hard or watery	Did you reach the toilet on time?		Did you use a lot of toilet paper?		Did you mark your underwear or pad?		Any other comments e.g. blood/ mucus
		Yes	No	Yes	No	Yes	No	

KI Bowel Habit Diary/ 2001

Bristol Stool Chart

Type 1		Separate hard lumps, like nuts (hard to pass)
Type 2		Sausage-shaped but lumpy
Type 3		Like a sausage but with cracks on the surface
Type 4		Like a sausage or snake, smooth and soft
Type 5		Soft blobs with clear-cut edges
Type 6		Fluffy pieces with ragged edges, a mushy stool
Type 7		Watery, no solid pieces. **Entirely Liquid**

NBD SCORE

3. Wexner Fecal Incontinence Score: Please check the appropriate box in each row as honestly as possible regarding your bowel movement habits & your bowel control.

Total Score (0-20): _____

	Never (0)	Less than once per month (1)	Less than once/ week & greater than once/month (2)	Less than once/day & greater than once/month (3)	Once a day or more than once a day (4)
How often do you have accidents to solid, well-formed stool?					
How often do you have accidents to liquid stool/ diarrhea?					
How often does the gas escape without your knowledge or control?					
How often do you wear a pad/ depends or change underwear?					
How much do the above answers alter your lifestyle or activities?					

The number of points for each possible answer is given in parenthesis

(1) Frequency of defecation — Points ____
Daily ☐(0) 2–6 times every week ☐(1) Less than once a week ☐(6)

(2) Time used for each defecation ____
0–30 min ☐(0) 31–60 min ☐(3) More than one hour ☐(7)

(3) Uneasiness, headache or perspiration during defecation ____
No ☐(0) Yes ☐(2)

(4) Regular use of tablets against constipation ____
No ☐(0) Yes ☐(2)

(5) Regular use of drops against constipation ____
No ☐(0) Yes ☐(2)

(6) Digital stimulation or evacuation of the anorectum ____
Less than once every week ☐(0) Once or more every week ☐(6)

(7) Frequency of faecal incontinence
Less than once every month ☐(0) 1–4 times every month ☐(6)
1–6 times every week ☐(7) Daily ☐(13) ____

(8) Medication against faecal incontinence ____
No ☐(0) Yes ☐(4)

(9) Flatus incontinence ____
No ☐(0) Yes ☐(2)

(10) Perianal skin problems ____
No ☐(0) Yes ☐(3)

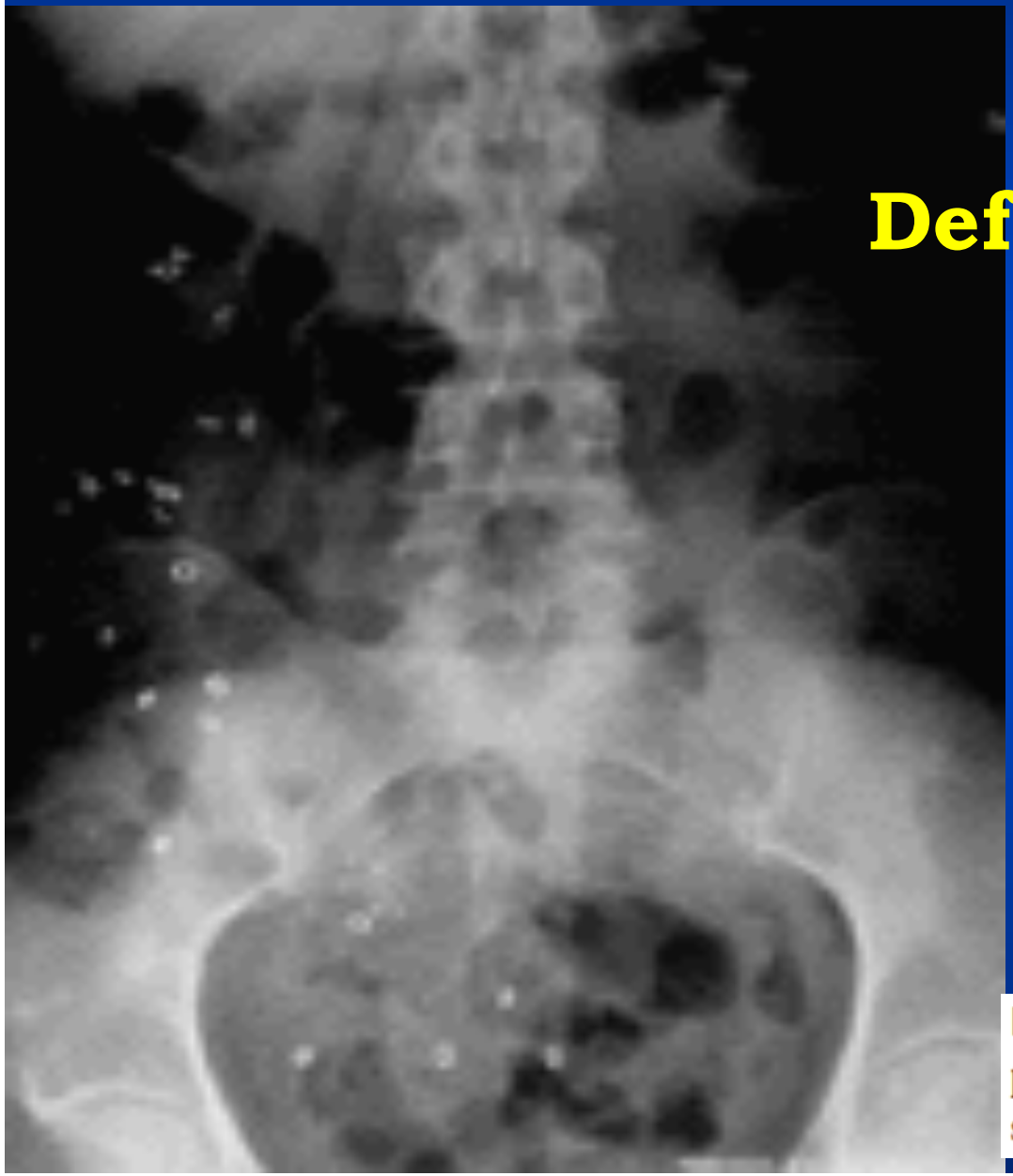
Total NBD score (range 0–47) ____

NBD score	Bowel dysfunction
0–6	Very minor
7–9	Minor
10–13	Moderate
14 or more	Severe

Physical Examination

- **Perianal ispection to assess:**
  - **Rectal prolapse**
  - **Anal fissures**
  - **Hemorrhoids**
  - **Soiling**
- **Anorectal digitation to assess:**
  - **Anorectal tone**
  - **Voluntary contraction**
- **Colonoscopy is recommended over 40 yo if blood in stools, abdominal pain, weight loss**

Colorectal Transit Time



Defecating proctogram

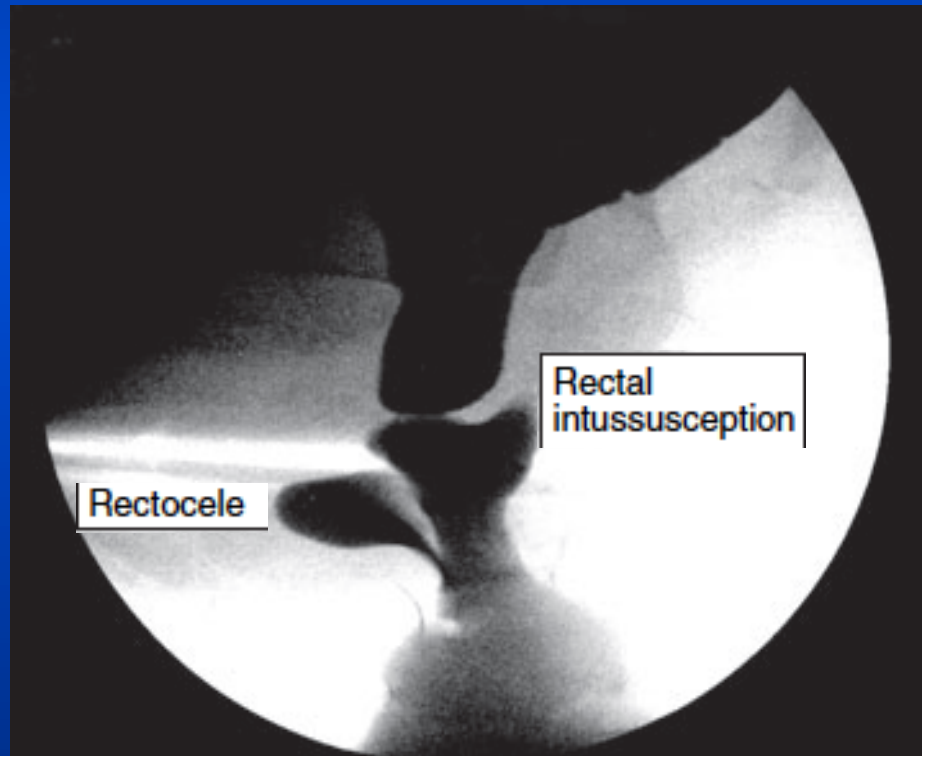


Figure 1 Defaecating proctogram demonstrating a rectocele protruding into the vagina with a concomitant rectal intussusception.

Anorectal tests

1. Anal manometry: with determination of anal resting pressure (reflecting the function of the internal anal sphincter muscle) and anal squeeze pressure (reflecting the function of the external anal sphincter muscle)
2. Rectal balloon distension: to evaluate rectal sensations and rectal wall compliance
3. Electrophysiology test: to determine pudendal nerve terminal motor latency
4. Electrical stimulation: anal mucosa stimulation to evaluate anal sensibility
5. Transanal ultrasonography: to detect tears of the internal cr anal sphincter muscle
6. Defecography: radiography after rectal nstallation of contrast may be performed in select patients

Fecal Incontinence

Conservative

- Pads
- Anal plugs
- Dietary advices
- Antidiarrhoeal drugs
- PFE & biofeedback

Surgical

- Anal sphincter repair
- Graciloplasty
- Artificial sphincter
- Colostomy/ileostomy

Constipation

- Establishment of a routine
- Diet (fiber, fruit, whole grain food, cereals)
- 1.5 – 2 L fluid/day
- Abdominal massage (clockwise direction)
- Digital evacuation of feces
- Laxatives/enema/Transanal irrigation (Peristeen)
- Bowel resection or stoma

Sacral nerve stimulation for faecal incontinence and constipation in adults (Review)

Mowatt G, Glazener CMA, Jarrett M

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Analysis 1.1. Comparison 1 Faecal Incontinence, Outcome 1 Patients cured and improved on treatment.

Patients cured and improved on treatment

Study	Cured	%	Improved	%
Leroi 2005	5/19	26	17/19	89
Vaizey 2000	1/2	50	2/2	100

Episodes of faecal incontinence per week

Study		Measure	Patients	Baseline	Three months
Leroi 2005	Group of 19 who chose 'on' following the crossover period	Median (range)	Baseline: 16 3 months: 16 'Off' period: 19 'On' period: 19 Follow-up: 18	3.5 (0 to 16)	0.3 (0 to 3)
Leroi 2005	Group of 5 who chose 'off' following the crossover period	Median (range)	Baseline: 5 3 months: 4 'Off' period: 5 'On' period: 5 Follow-	7 (0 to 11)	1.9 (1 to 10)

Sacral nerve stimulation for faecal incontinence and constipation in adults (Review)

Mowatt G, Glazener CMA, Jarrett M

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Analysis 2.1. Comparison 2 Constipation, Outcome 1 Bowel movements per week.

Bowel movements per week

Study	Measure	Patients	Baseline	One year	'Off' period	'On' period	Change (%)	Notes
Kenefick 2002	Mean (range)	2	2 (1 and 3)	8 (8 and 9)	2 (1 and 2)	5 (4 and 5)	3 (150%)	

Analysis 2.3. Comparison 2 Constipation, Outcome 3 Wexner Constipation Score.

Wexner Constipation Score

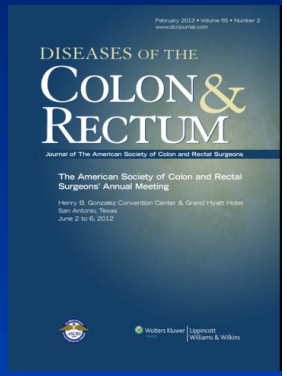
Study	Measure	Patients	Baseline	One year	'Off' period	'On' period	Change (%)	Notes
Kenefick 2002	Mean (range)	2	21 (20 to 22)	5 (4 to 6)	14 (13 to 15)	9 (5 to 13)	-5 (-36%)	The score ranges from 0 (normal) to 30 (severe constipation)

Analysis 2.4. Comparison 2 Constipation, Outcome 4 Symptom Analogue Score.

Symptom Analogue Score

Study	Measure	Patients	Baseline	One year	'Off' period	'On' period	Change (%)	Notes
Kenefick 2002	Mean (range)	2	30 (28 and 32)	89 (84 and 94)	32 (30 and 33)	74 (60 and 88)	42 (131%)	The score ranges from a best score of 100 to a worst score of 0

SNM & chronic FI



- Multicenter, prospective study
- 120 patients and 5 years follow up
- Patients with chronic refractory FI
- FI episodes/ week decreased from a mean of **9.1 to 1.7**
- **89%** had ≥ 50% improvement and **36%** complete continence

Hull 2013

Conclusions: FI & Constipation

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- Limited evidence that SNM can improve:

FI

- episodes of FI/week
- ability to defer defecation/ urgency
- incontinence scores
- QOL
- anorectal manometry parameters

Constipation

- bowel movements/week
- abdominal pain and bloating
- Wexner constipation score

Mowatt 2007

EUROPEAN UROLOGY 53 (2008) 60–67

Diagnostic Criteria, Classification, and Nomenclature for Painful Bladder Syndrome/Interstitial Cystitis: An ESSIC Proposal

- IC/BPS defined as:
  - CPP (>6 mo)
  - Bladder pressure/discomfort
  - at least one urinary symptom (ie.urgency, frequency).

- 3.4 million of women affected in US (prevalence of 2.7%)

- Associated symptoms due to similar embriological origin
  -vaginitis
  - vestibulodynia
  - pelvic floor dysfunction

Table 1 – Confusable diseases for bladder pain syndrome

Confusable disease	Excluded or diagnosed by[a]
Carcinoma and carcinoma in situ	Cystoscopy and biopsy
Infection with	
Common intestinal bacteria	Routine bacterial culture
Chlamydia trachomatis, Ureaplasma urealyticum	Special cultures
Mycoplasma hominis, Mycoplasma genitalium	
Corynebacterium urealyticum, Candida species	
Mycobacterium tuberculosis	Dipstick; if "sterile" pyuria culture for *M. tuberculosis*
Herpes simplex and human papilloma virus	Physical examination
Radiation	Medical history
Chemotherapy, including immunotherapy with cyclophosphamide	Medical history
Anti-inflammatory therapy with tiaprofenic acid	Medical history
Bladder-neck obstruction and neurogenic outlet obstruction	Uroflowmetry and ultrasound
Bladder stone	Imaging or cystoscopy
Lower ureteric stone	Medical history and/or hematuria: upper urinary tract imaging such CT or IVP
Urethral diverticulum	Medical history and physical examination
Urogenital prolapse	Medical history and physical examination
Endometriosis	Medical history and physical examination
Vaginal candidiasis	Medical history and physical examination
Cervical, uterine, and ovarian cancer	Physical examination
Incomplete bladder emptying (retention)	Postvoid residual urine volume measured by ultrasound scanning
Overactive bladder	Medical history and urodynamics
Prostate cancer	Physical examination and PSA
Benign prostatic obstruction	Uroflowmetry and pressure-flow studies
Chronic bacterial prostatitis	Medical history, physical examination, culture
Chronic non-bacterial prostatitis	Medical history, physical examination, culture
Pudendal nerve entrapment	Medical history, physical examination, nerve block may prove diagnosis
Pelvic floor muscle-related pain	Medical history, physical examination

CT = computed tomography; IVP = intravenous pyelogram; PSA = prostate-specific antigen.

[a] The diagnosis of a confusable disease does not necessarily exclude a diagnosis of BPS.

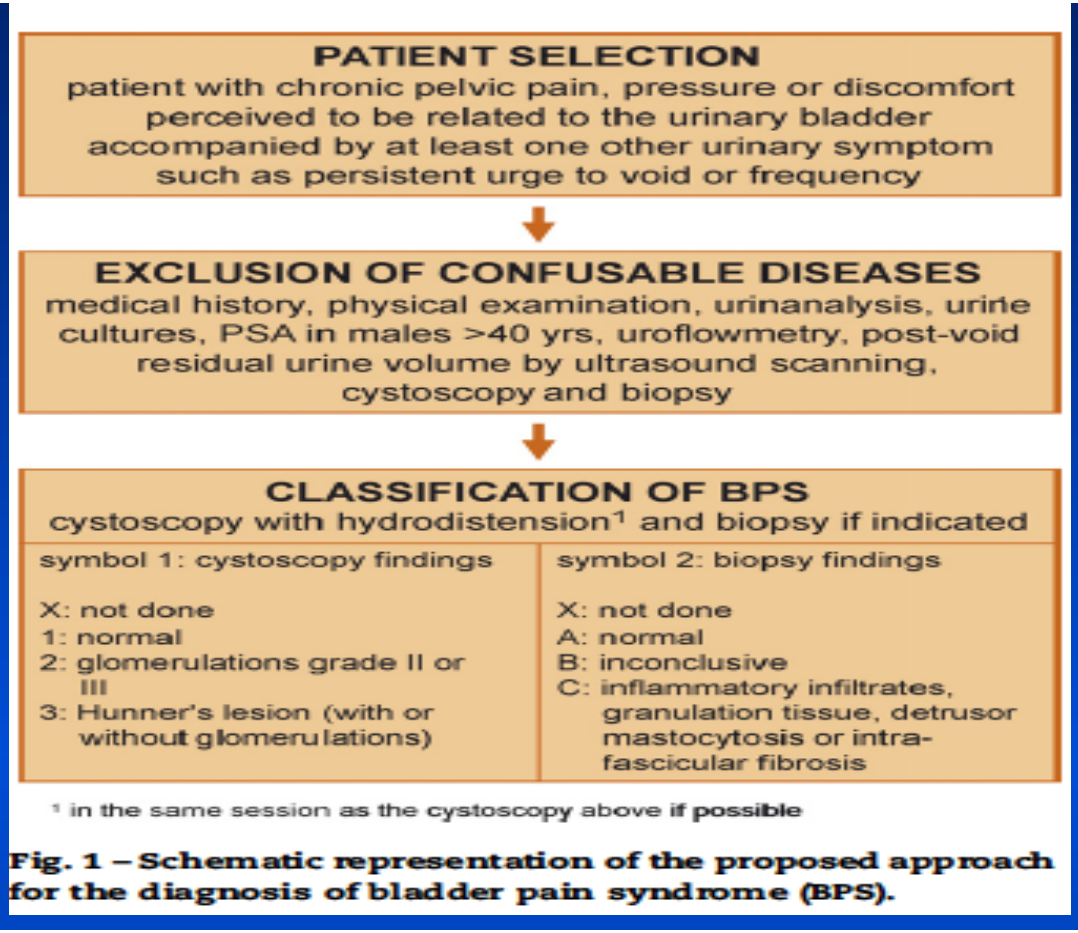


Fig. 1 – Schematic representation of the proposed approach for the diagnosis of bladder pain syndrome (BPS).

Table 2 – Classification of types of bladder pain syndrome on the basis of findings at cystoscopy with hydrodistention and of biopsies

	Cystoscopy with hydrodistention			
	Not done	Normal	Glomerulations[a]	Hunner's lesion[b]
Biopsy				
Not done	XX	1X	2X	3X
Normal	XA	1A	2A	3A
Inconclusive	XB	1B	2B	3B
Positive[c]	XC	1C	2C	3C

[a] Cystoscopy: glomerulations grade 2–3.
[b] With or without glomerulations.
[c] Histology showing inflammatory infiltrates and/or detrusor mastocytosis and/or granulation tissue and/or intrafascicular fibrosis.

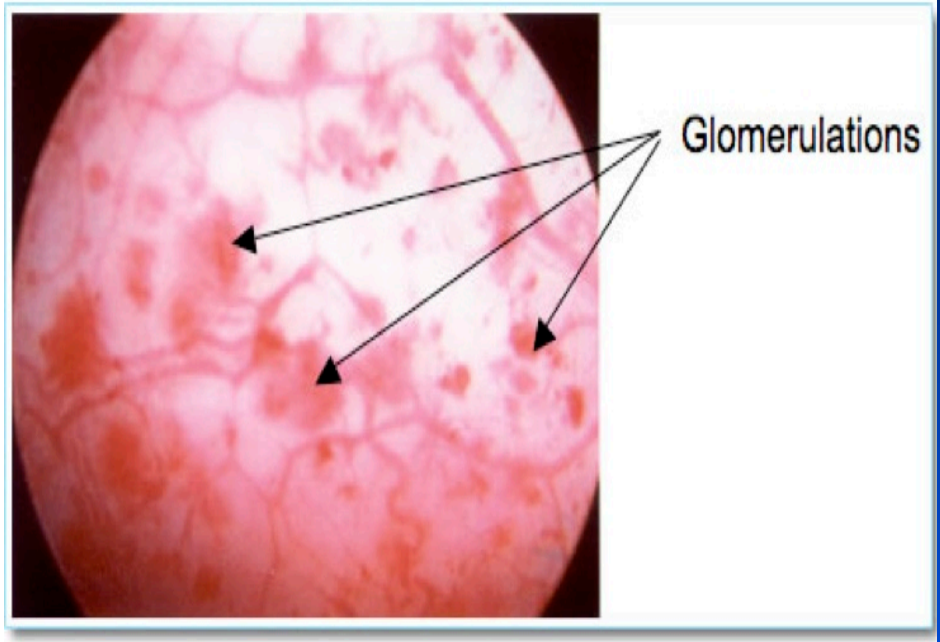
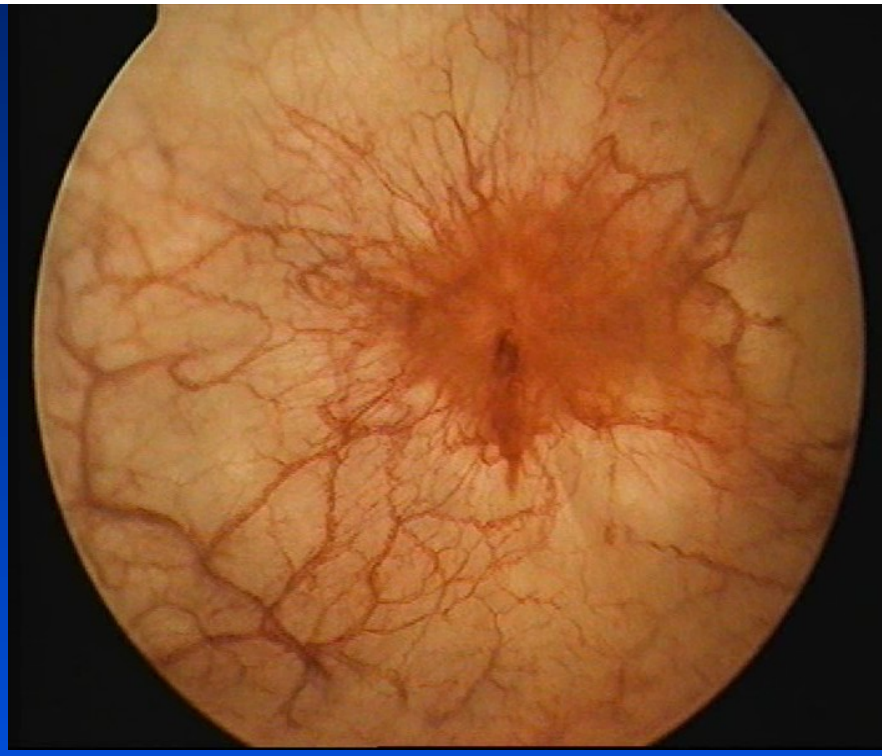


Figure 4. Interstitial cystitis → Glomerulation



- Cystoscopy and hydrodistension are prerequisite

- Positive signs of BPS are:
- Glomerulations grade 2–3 or Hunner's lesions or both
- inflammatory infiltrates and/or granulation tissue and/ or detrusor mastocytosis and/or intrafascicular fibrosis

BPS/IC: etiology

Defect in the urothelial lining or glycosamminoglycan layer

Mast cells activation within bladder wall → Influx of K+ iones

↓

Upregulation of afferent nerves

↓

Activation of more mast cells

↓

Chronic neurogenic inflamation

↓

Afferent nerves overactivity

↓

Central sensitization → Chronic pelvic pain

Rosenberg 2007

SNM & refractory IC/BPS

- **87%** patients reported a 50% decrease in pain

  Comiter 2003, Whitemore 2003, Maher 2001

- **36%** decrease in narcotic (morphine) use

- **25%** remained narcotic free at 15 months

  Peters 2004

- Normalization of antiproliferative factors and epidermal growth factors after SNM

  Chai 2008

- **48%** reduction of efficacy at 2 years

  Rockley 2005

SNM & refractory IC/BPS: Conclusions

- SNM seems to be efficacious in treating IC/BPS
- Studies still small and limited
- Immediate pain relief in responders
- SNM success declines over time
- Further research is needed

SNM & Urogenital pain

- **Coccygodynia:** painful condition in or around the coccyx, typically worsened with sitting, often stemming from trauma, infection, tumor, osteoarthritis of the sacrococcygeal joint, spasm of the pelvic floor, obesity.

- **Anorectal pain:** idiopathic or secondary (inflammation, tumor, pelvic floor muscle spasms) is a diagnosis of exclusion.

Vulvodynia

- Vulvar discomfort (sharp pain, burning) in the absence of physical exam findings or a neurological disorder.
- Constant, intermittent, or only provoked with contact ie. wearing tight clothing, inserting tampons (provoked vestibulodynia).
- 15% of women
- Etiology unknown
  - muscular hypothesis (perineal muscle spasm)
  - chronic inflamation of nerve (biopsy studies)

Non-relaxing pelvic floor dysfunction

- The contribution of pelvic floor muscle tenderness to CPP is well established in the literature.
- Several terms used (levator myalgia, piriformis syndrome, levator ani syndrome, pelvic floor muscle spasms).
- 24% of women attending urogynecological clinics in US & 16 per 100000 person/year in Minesota

Adams 2013

- 60% of women with CPP have LA myalgia.

Mathias 1996, Fitzgerald 2011

Potential mechanisms

1. Dysfunctional voiding/defecation
   (voluntary holding of urine/stool)

2. Pain (dyspareunia due to atrophic vaginitis, vulvodynia)
   if intercourse is continued despite the pain can lead to persistent contraction of the PFM

3. Injury of the pelvic floor from surgery/trauma
   mesh/permanent suture in muscle ⟶ pain ⟶ spasm

4. Neural "cross-talk" between pelvic organs
   Visceral pain (IC/PBS, IBS)

5. Postural abnormalities ⟶ overcompensation of PF

6. Sexual abuse

Visual inspection

Cotton swab testing

Speculum

Digital palpation:

External (urogenital triangle)

Internal (LA & obturator internus)

Rectal examination

(LA/sphincter/coccyx

TABLE. Recognizing and Managing Nonrelaxing Pelvic Floor Dysfunction

Inquire about symptoms of bowel, bladder, and sexual dysfunction and pain
Bowel function: bloating, constipation, difficulty evacuating stool, straining with bowel movement, splinting the posterior vagina, anal digitation, incomplete evacuation, sense of anal blockage during defecation
Urinary function: frequency, hesitancy, urgency, dysuria, bladder pain, urge incontinence
Sexual function: insertional or deep dyspareunia, pelvic ache after intercourse
Pain: low back pain radiating to thighs or groin, pelvic pain unrelated to intercourse, lower abdominal wall pain
Perform a focused physical examination
Vulvar, vaginal, and rectal examination
External palpation of the urogenital triangle
Internal palpation of deep pelvic floor muscles (may reveal tension and tenderness)
Consider diagnostic testing, as dictated by symptoms
Pelvic ultrasonography for pelvic pressure, pain, bloating
Anorectal manometry and rectal balloon expulsion for defecatory symptoms
Voiding diary, urinalysis, and possibly urodynamic study for voiding symptoms
Provide education about pelvic floor muscles and function
Refer for pelvic floor physical therapy (a cornerstone of management)
Refer to subspecialists (gastroenterology, gynecology, physical medicine, sexual medicine, and urology) when symptoms and examination findings are complex

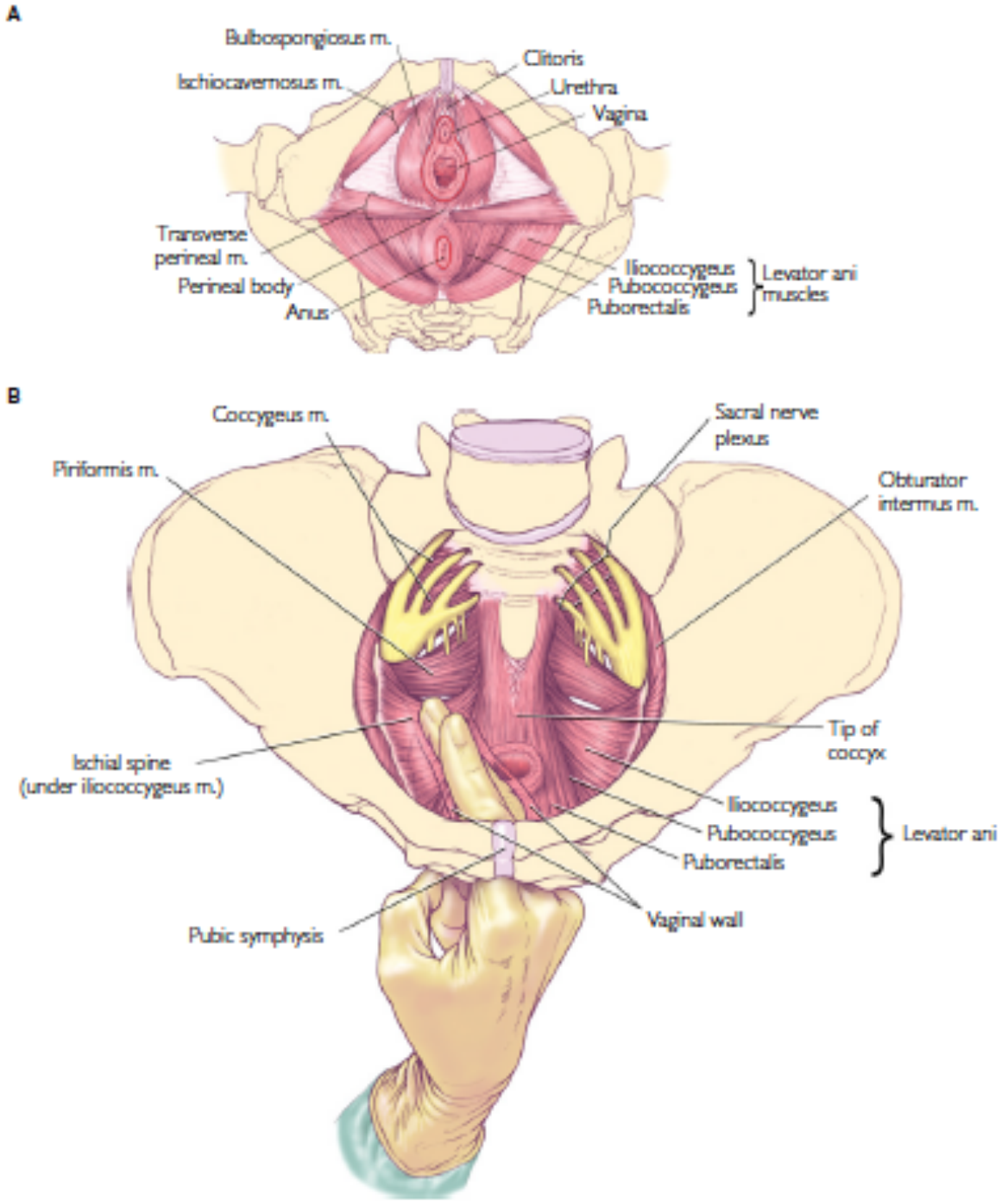


FIGURE. A, Muscles of the pelvic floor. B, Digital palpation of deep pelvic floor muscles. m = muscle.

Physical therapy:

Trigger point massage

PFE

Biofeeback

To avoid penetrative sexual activity till PFM are rehabilitated

TABLE. Recognizing and Managing Nonrelaxing Pelvic Floor Dysfunction

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Provide education about pelvic floor muscles and function
Refer for pelvic floor physical therapy (a cornerstone of management)
Refer to subspecialists (gastroenterology, gynecology, physical medicine, sexual medicine, and urology) when symptoms and examination findings are complex

Other treatments

- Neuropathic pain modulators (amithriptyline, gabapentin, pregabalin….)
- Local anaesthetics and corticosteroids
- Botox of trigger points
- Neuromodulation: PTNS, SNM.

SNM & Urogenital pain

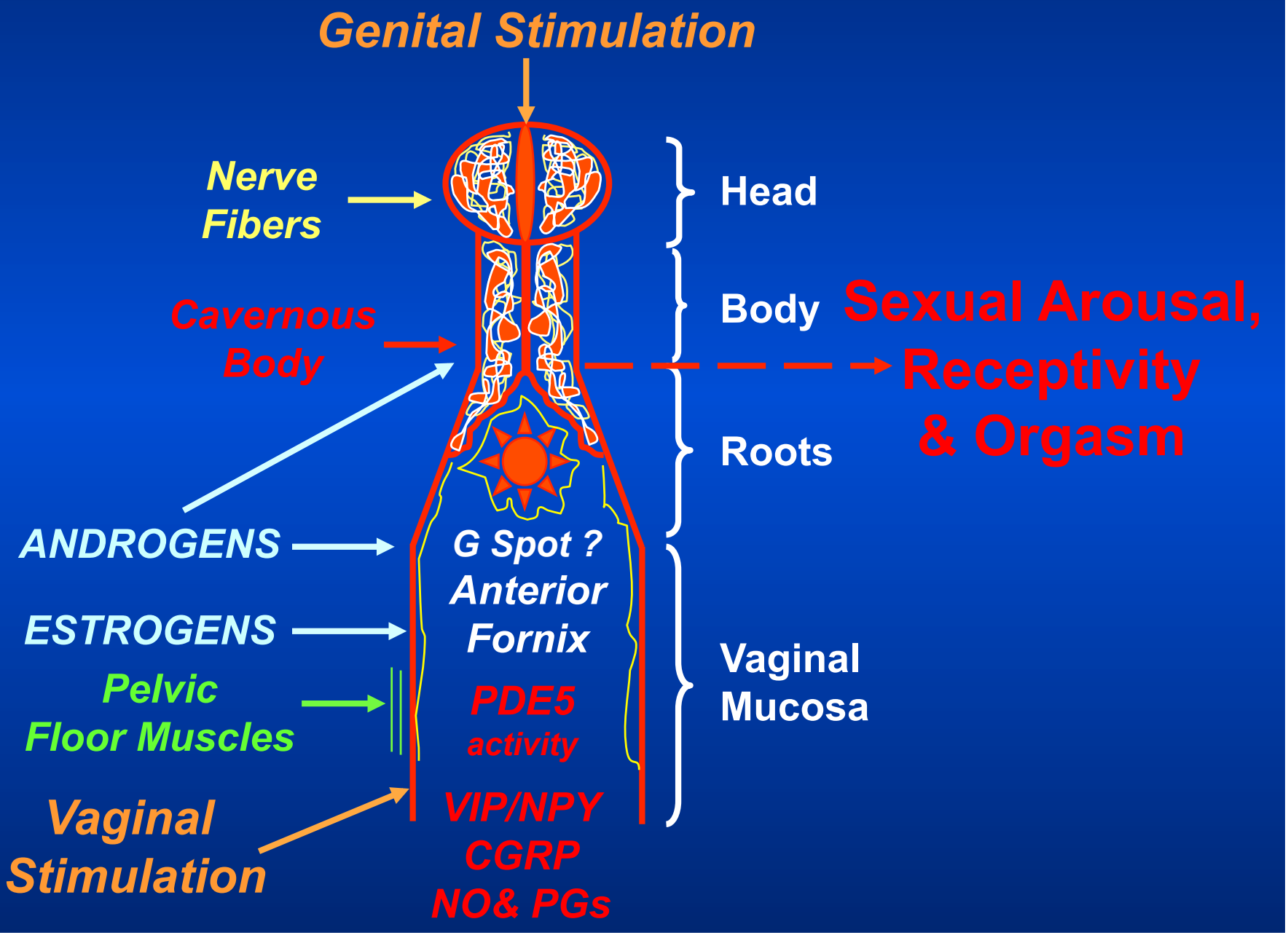
Table 3 Percentage of success and mean improvement of the VAS score in previously published study

Author	Tested patients/ successful patients	%	Kind of pain	Preoperative VAS	Postoperative VAS
Siegel et al. [17]	Not stated/10		Pelvic/urogenital	9.7	4.4
Everaert et al. [16]	26/11	42	Pelvic/urogenital	NS	NS
Falletto et al. [13]	27/12	44	Anorectal	8.2	2.2
Govaert et al. [12]	9/4	44	Anorectal	8.0	1.0
Martellucci et al. [18]	17/8	47	After pelvic surgery	8.2	1.9
Present series	27/16	59	Pelvic/urogenital/ anorectal	8.1	2.1

NS not significant

- A positive response to gabapentin or pregabalin or Stage I are predictors of a successful outcome.
- Multiple localizations of pelvic pain and pain occurred after surgery seem to be negative factors for the success of the treatment.
- The mechanism of action and who may benefit from the treatment are still unclear

CLITORIS AND VAGINA ARE "THE QUEENS" OF SEX



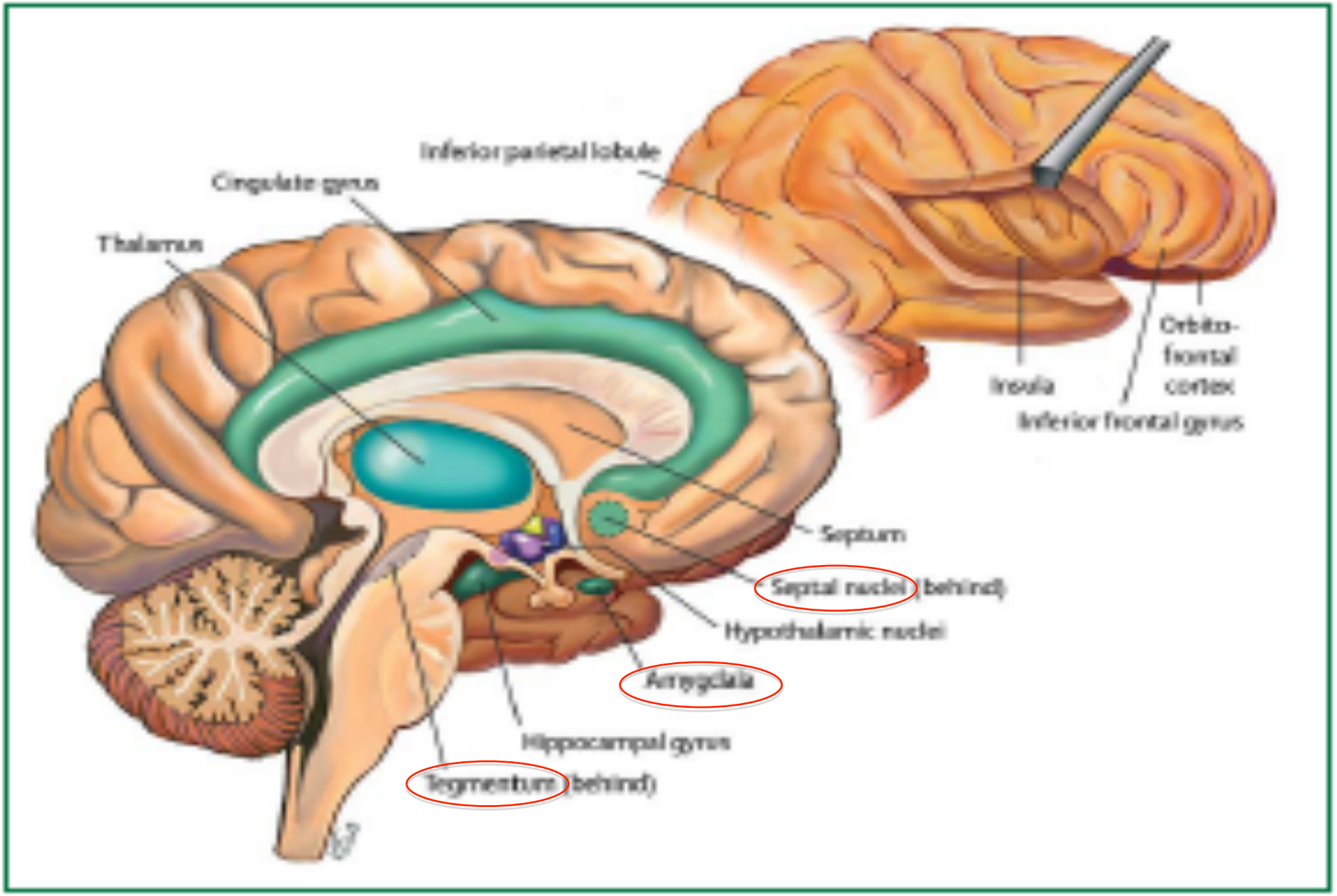


Figure 2: Regions of sexual activation in the brain

The limbic lobe is shown in green. Paralimbic regions include the posterior orbitofrontal cortex and much of the insula.

Neurophysiology & neuroanatomy of sexuality

- Since 2000, PET and MRI have confirmed that these and other regions of the brain are activated during sexual arousal.

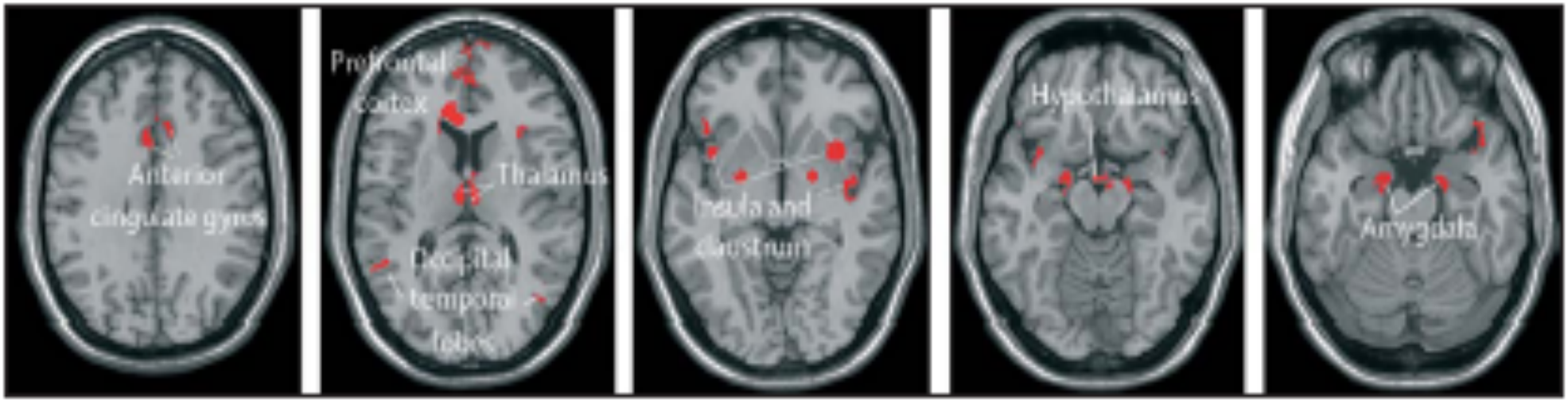


Figure 2: MRI images of regions of the brain activated during sexual arousal

Regions activated while patients viewed erotic videos.

Redoutè 2000, Holstege 2003

SEXUAL DYSFUNCTION

- Brain injuries
- Stroke
- Epilepsy
- Spinal cord injuries
- Parkinson, MS, Peripheral neuropathy

7. Surgical Disruption of the genital autonomic nerve supply

- Radical surgery for cancer of the cervix, bladder and rectum, damage autonomic nerves and vessels of vaginal wall important for lubrification causing sexual dysfunction.

	Standard treatment	Typical surgical nerve damage	Site of nerve damage
Cancer of the cervix	Radical hysterectomy with lymphadenectomy with or without postoperative radiotherapy	Hypogastric nerves Pelvic plexus and pelvic splanchnic nerves Pelvic plexus	Sacrouterine ligaments* Cardinal ligaments† Vesicouterine ligaments‡
Prostate cancer	Radical prostatectomy, external beam radiotherapy, or radioactive seed implants	Pelvic plexus	Neurovascular bundle dorsolateral to the prostate
Bladder cancer	Radical cystectomy	Pelvic plexus	Neurovascular bundle dorsolateral to the prostate or lateral to the vagina
Cancer of the rectum	Total mesorectal excision with or without preoperative radiotherapy	Superior hypogastric plexus and hypogastric nerves Pelvic plexus and pelvic splanchnic nerves	Presacral mesorectal manipulation Lateral ligament of the rectum or lateral mesorectal manipulation

Nomenclature for ligaments relevant to radical hysterectomy varies in published work: *Uterosacral ligaments or rectouterine ligaments. †Parametrium or transverse cervical ligaments. ‡Pubocervical ligaments or vesicovaginal ligaments.

Table 4: Nerve disruption during conventional radical pelvic surgery

- The benefit of nerve sparing has yet to be proved

Sipski 2001

7. Surgical Disruption of the genital autonomic nerve supply

	Instrument	Treatment period (years)	Follow-up (years)	Outcome	Study (n/total)	Controls (n/total)	Prevalence in study % (OR; 95% CI)
Cervix							
Cross-sectional retrospective study[110]*	Validated questionnaire, constructed for study	1991–1992	4–6	Insufficient lubrication	46/177	27/248	26 (2·5; 1·6–3·8)
				Short vagina	52/197	8/240	26 (8·1; 4·4–14·9)
				Non-elastic vagina	45/195	9/246	23 (6·7; 3·6–12·5)
				Dyspareunia	31/196	5/246	16 (8·5; 3·5–18·6)
				Distress due to changes	62/243	25/332	26 (3·4; 2·2–2·5)
Prospective observational study;[111] no baseline*	Validated questionnaire, constructed for study	1992–1995	2	Lack of sexual interest	69/121	148/317	57 (1·2; 1·0–1·5)
				Lack of lubrication	8/83	8/239	10 (2·9; 1·1–7·4)
Prospective observational study[112]*	Non-validated questionnaire constructed for study	1998–2003	2	Less lubrication	9/64	6/213	14 (5·0; 1·9–14)
				Narrow/short vagina	14/55	4/196	25 (13; 4·3–36)
				Dyspareunia	12/55	3/196	18 (12; 3·4–42)
				Sexual dissatisfaction	13/58	6/202	22 (7·6; 3·0–19)
Prostate							
Prospective observational study[113]*	Non-validated questionnaire, constructed for study	1994–1995	5	Erectile dysfunction	693/901	182/286	79 (2·5; 1·6–3·8)
Cross-sectional retrospective study[114]	26-item prostate cancer index composite	1995–1999	6	Erectile dysfunction	709 questionnaires		71
Bladder							
Prospective observational study[115]	10-item Index of Female Sexual Function (FSFI)	1997–2002	2	Difficult orgasm	12/27		45
				Less lubrication	11/27		41
				Dyspareunia	6/27		22
				Intercourse impossible	14/27		52
				Decreased desire	10/27		37
				Dissatisfaction	14/27		52
Prospective observational study[116]	5-item International Index of Erectile Function	1995–2002	4	Eerectile dysfunction	42/49		86
Retrospective study[117]*	Non-validated questionnaire constructed for study	NA	NA	Erectile dysfunction	61/90	7/42	68 (p<0·05)
				Partial erectile dysfunction	72/90	7/42	80 (p<0·05)
				Vaginal dysfunction	3/18	2/12	17 (NS)
Rectum							
Cross-sectional, retrospective study[118]*	Validated questionnaire[119]	1996–2002	2–6	Dyspareunia	4/22	0/19	18 (p<0·05)
				Short/less elastic vagina	5/22		
Cross-sectional retrospective study[120]	Female Sexual Function Index (FSFI)	1980–2003	4–5 (median)	Libido	7/25		41
				Arousal	5/25		29
				Less lubrication	14/25		56
				Difficult orgasm	6/25		24
				Dyspareunia	9/25		36
				Surgery made sex worse	19/81		29

Int Urogynecol J (2007) 18:391–395
DOI 10.1007/s00192-006-0168-9

ORIGINAL ARTICLE

Effects of sacral neuromodulation on female sexual function

Rachel N. Pauls · Serge P. Marinkovic · W. Andre Silva · Christopher M. Rooney · Steven D. Kleeman · Mickey M. Karram

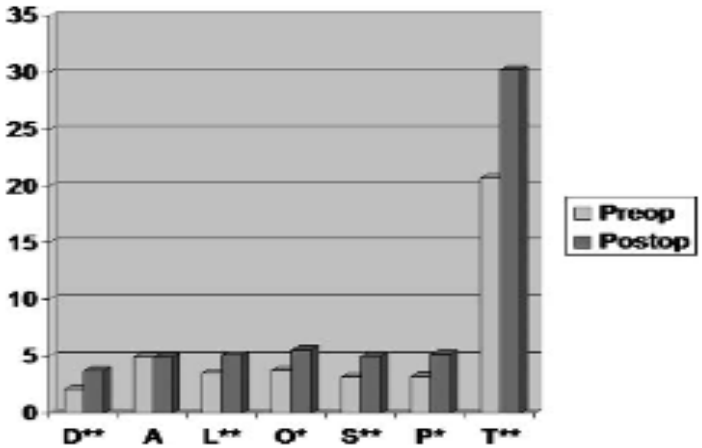


Fig. 1 Mean FSFI scores pre- and postoperatively. *D* Desire, *A* arousal, *L* lubrication, *O* orgasm, *S* satisfaction, *P* pain, *T* total. An *asterisk* indicates significance at the 0.05 level. *Two asterisks* indicate significance at the 0.01 level

Int Urogynecol J (2008) 19:697–700
DOI 10.1007/s00192-007-0504-8

ORIGINAL ARTICLE

The effects of bilateral caudal epidural S2–4 neuromodulation on female sexual function

Nasim Zabihi · Arthur Mourtzinos · Mary Grey Maher · Shlomo Raz · Larissa V. Rodríguez

	Total	Desire	Arousal	Lubrication	Orgasm	Satisfaction	Pain
Pre-op	12.0	2.7	1.8	1.7	1.7	2.1	2.1
Post-op	18.2	2.5	3.1	3.3	3.1	3.2	3.1
% Improvement	52	−7	75	88	79	54	51
P-value	0.05	0.35	0.03	0.03	0.04	0.06	0.11

International Journal of Impotence Research (2006) 18, 470–476

0955-9930/06 $30.00
www.nature.com/ijir

ORIGINAL ARTICLE

Sexual functioning in patients with lower urinary tract dysfunction improves after percutaneous tibial nerve stimulation

MR van Balken[1], H Verguns[2] and BLH Bemelmans[3]

[1]*Rijnstate Hospital, Arnhem, The Netherlands;* [2]*Canisius-Wilhelmina Hospital, Nijmegen, The Netherlands and* [3]*Free University Medical Center, Amsterdam, The Netherlands*

Table 2 Change in dissatisfaction with current sexual life

Patient group			*Prior to PTNS*	*After PTNS*	P-value
	Number	*P-value overall satisfaction whole (sub) group*	*Pts (very) dissatisfied/respondents*	*Pts still (very) dissatisfied/ pts that felt so before PTNS*	
All	$N=121$	NS	40/103	20/35	$P=0.002$
Women	$N=76$	$P=0.024$	22/60	9/19	$P=0.003$
Men	$N=45$	NS	18/43	11/16	NS

Complex Pelvic Floor dysfunctions

- Focus on global symptoms complex rather than a individual symptoms
- Focused physical examination and exclusion of other conditions
- Education of the patient
- Initial conservative treatment
- Referral to other specialists may be appropriate for multidisciplinary care for women whose symptoms do not respond to initial therapy

Complex Pelvic Floor dysfunctions

- Multidisciplinary assessment & management:
  - urogynaecologist
  - urologist
  - gastroenterologist
  - neurologist
  - colorectal surgeon
  - pain medicine physician
  - physiotherapist
  - specialist nurse
  - psychologist

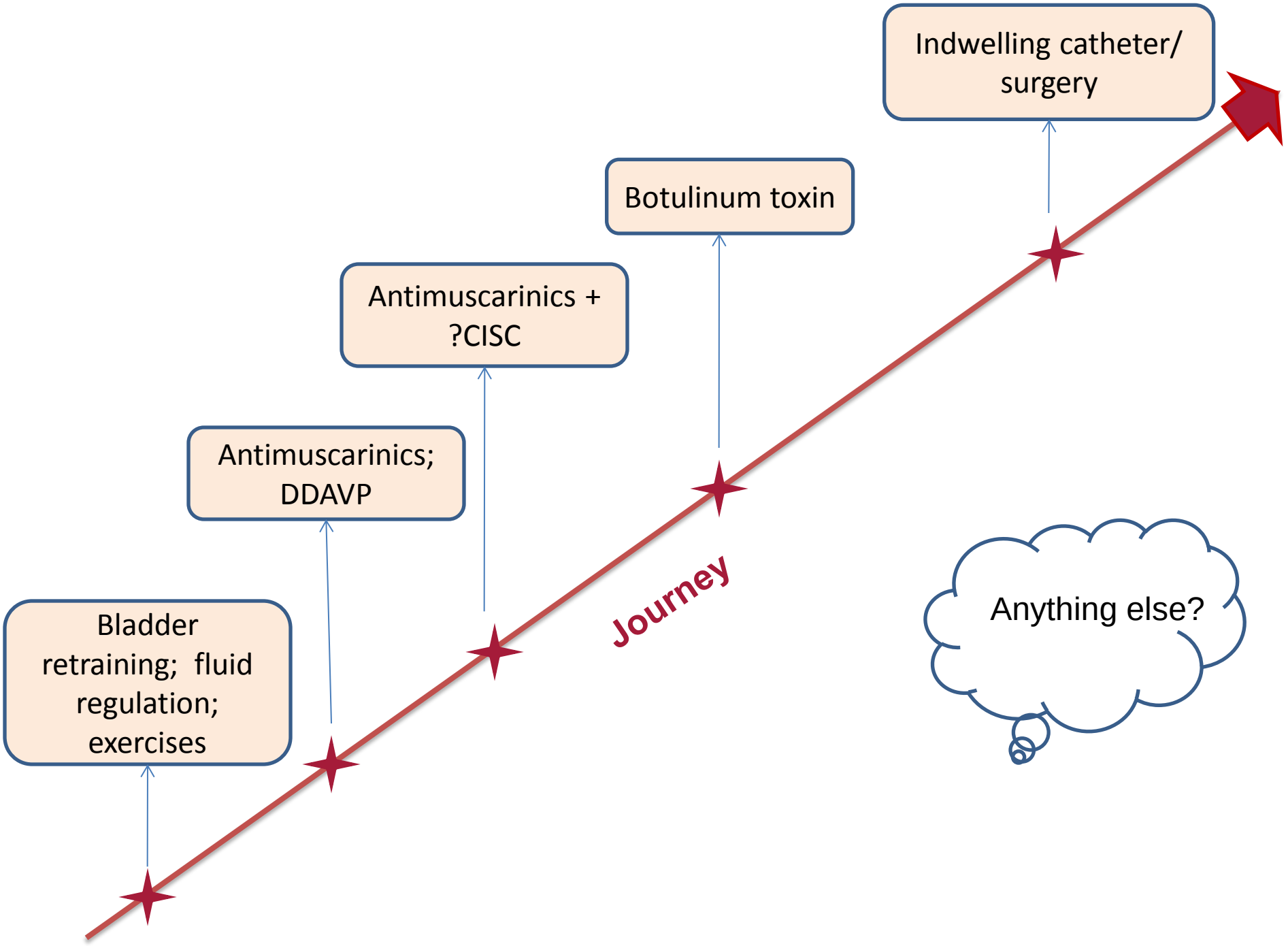
PTNS for the overactive bladder syndrome

Jalesh N. Panicker
Consultant Neurologist
Department of Uro-Neurology
National Hospital for Neurology and Neurosurgery
and
Honorary Senior Lecturer, UCL Institute of Neurology
Queen Square, London

ICS Barcelona August 2013

Case scenario

- 48 year old lady
- Urinary urgency, frequency, nocturia and urge incontinence
- Tried two antimuscarinics- dry mouth and constipation
- Reluctant to try Botulinum toxin because of concerns about ISC
- How to manage?


Indwelling catheter/ surgery
Botulinum toxin
Antimuscarinics + ?CISC
Antimuscarinics; DDAVP
Bladder retraining; fluid regulation; exercises
Journey
Anything else?

Tibial nerve stimulation

- *Percutaneous* vs. *Transcutaneous*
- Stoller afferent Nerve Stimulation
- Recent exponential increase in publications

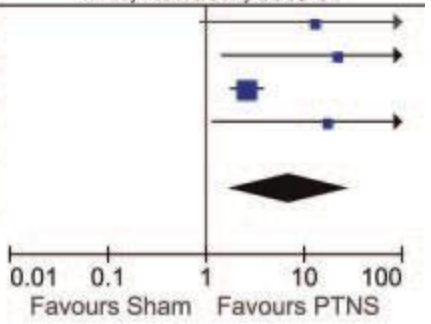
The evidence

- 16 studies
- 4 RCTs

Study or Subgroup	PTNS Events	PTNS Total	Sham Events	Sham Total	Weight	Risk Ratio M-H, Random, 95% CI
Finazzi Agro 2005	6	8	0	8	17.6%	13.00 [0.85, 198.14]
Finazzi Agro 2010	12	17	0	15	17.4%	22.22 [1.43, 345.99]
SuMIT 2010	60	110	23	110	47.4%	2.61 [1.75, 3.90]
Vohra 2002	9	11	0	10	17.6%	17.42 [1.14, 265.34]
Total (95% CI)		**146**		**143**	**100.0%**	**7.02 [1.69, 29.17]**
Total events	87		23			

Heterogeneity: $Tau^2 = 1.07$; $Chi^2 = 6.18$, df = 3 (P = 0.10); $I^2 = 51\%$

Test for overall effect: Z = 2.68 (P = 0.007)



Burton et al. 2012

Percutaneous Tibial Nerve Stimulation Effects on Detrusor Overactivity Incontinence are Not Due to a Placebo Effect: A Randomized, Double-Blind, Placebo Controlled Trial

Enrico Finazzi-Agrò,*,† Filomena Petta, Francesco Sciobica, Patrizio Pasqualetti, Stefania Musco and Pierluigi Bove

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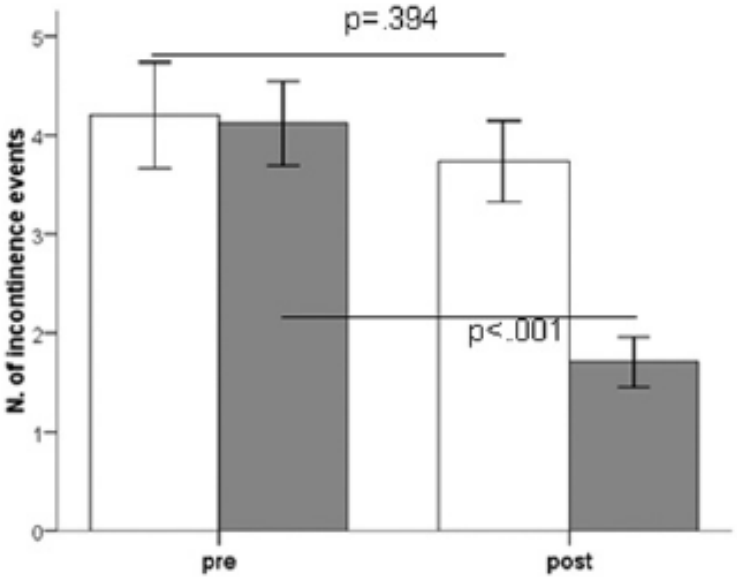
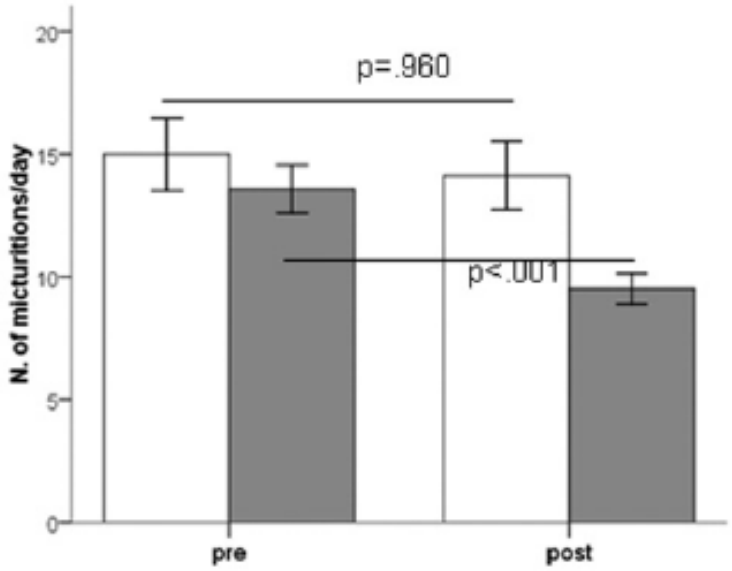
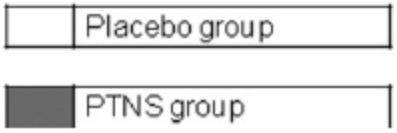
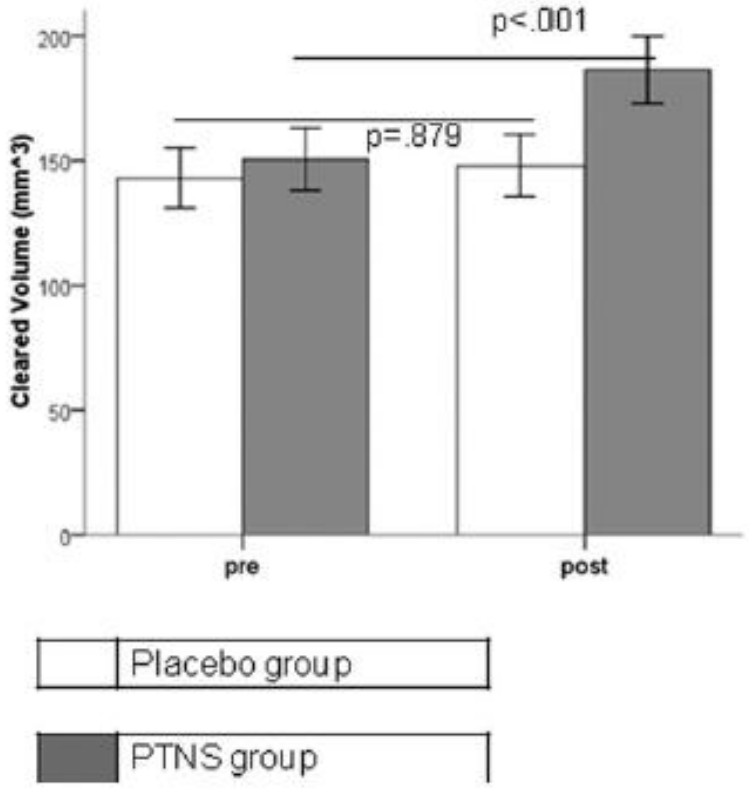
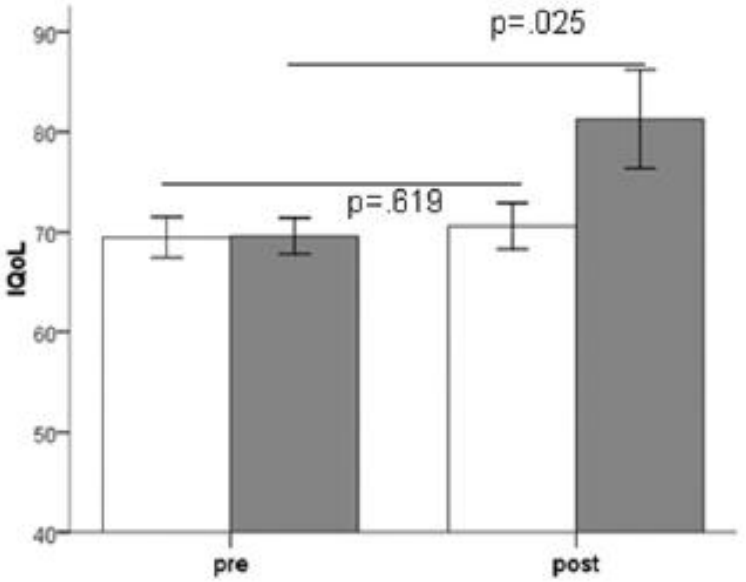

p=.394
p<.001
N. of incontinence events
0
1
2
3
4
5
pre
post


p=.960
p<.001
N. of micturitions/day
0
5
10
15
20
pre
post


Placebo group
PTNS group


p<.001
p=.879
Cleared Volume (mm^3)
200
150
100
50
0
pre
post
Placebo group
PTNS group


p=.025
p=.619
IQoL
90
80
70
60
50
40
pre
post

Randomized Trial of Percutaneous Tibial Nerve Stimulation Versus Sham Efficacy in the Treatment of Overactive Bladder Syndrome: Results From the SUmiT Trial

Kenneth M. Peters,*,† Donna J. Carrico, Ramon A. Perez-Marrero,‡ Ansar U. Khan, Leslie S. Wooldridge,§ Gregory L. Davis‖ and Scott A. MacDiarmid¶

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The evidence (2)

- SuMIT trial: pivotal multicenter, double-blind, randomized, sham controlled trial
- Level I evidence that PTNS is safe and effective in treating overactive bladder symptoms
- 54.5% reported moderately or markedly improved responses vs. 20.9% sham subjects

Peters KM, Carrico DJ, Perez-Marrero RA, Khan AU, Wooldridge LS, Davis GL, et al. Randomized trial of percutaneous tibial nerve stimulation versus Sham efficacy in the treatment of overactive bladder syndrome: results from the SUmiT trial. *J Urol* 2010;183(4):1438-43.

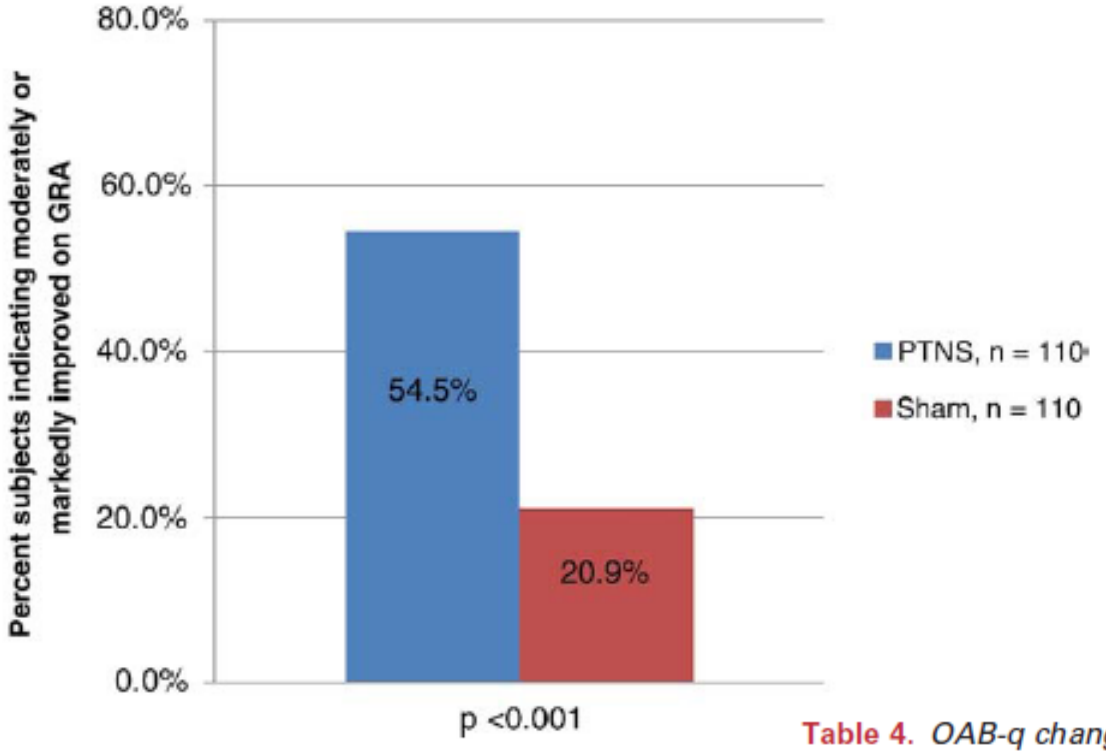


Table 4. *OAB-q change from baseline at 13 weeks*

	Symptom Severity Score*	Health Related Quality of Life Score†
PTNS:		
No.	101	103
Mean ± SD change	−36.7 ± 21.5	34.2 ± 21.3
Sham:		
No.	102	105
Mean ± SD change	−29.2 ± 20.0	20.6 ± 20.6
Difference (PTNS − sham)	−7.5 ± 20.7	8.2 ± 21.0
p Value	0.01	0.006

* Lower score is better score.
† Higher score is better score.

Follow up?

- STEP
- FDA approval 2000
- NICE interventional procedure guidance 362: October 2010

Approvals

- FDA approval 2000
- NICE interventional procedure guidance 362: October 2010

NHS
National Institute for
Health and Clinical Excellence

Neurourology and Urodynamics 28:964–968

The Clinical and Urodynamic Results of a 3-Month Percutaneous Posterior Tibial Nerve Stimulation Treatment in Patients With Multiple Sclerosis-Related Neurogenic Bladder Dysfunction

Sahin Kabay,[1*,†] Sibel Canbaz Kabay,[2†] Mehmet Yucel,[1†] Hilmi Ozden,[3‡] Zahide Yilmaz,[4§] Ozgen Aras,[5†] and Bahar Aras[5†]

TABLE II. The Effects of PTNS on Urodynamic Variables for the Comparison of Baseline and After PTNS Data in MS Patients

Urodynamic variables	Baseline value, mean ± SD (range)	PTNS, mean ± SD	*P*-value
First involuntary detrusor contraction			
At volume (ml)	124.2 ± 37.6 (60–185)	217.5 ± 66.4 (94–347)	0.000
P_{detmax} (cmH_2O)	43.7 ± 20.2 (14–97)	29.7 ± 10.2 (13–51)	0.005
Maximum cytometric capacity			
At volume (ml)	199.7 ± 29.3 (128–263)	266.8 ± 36.9 (198–342)	0.000
P_{detmax} (cmH_2O)	48.8 ± 21.4 (18–98)	35.8 ± 10.5 (21–59)	0.001
$P_{det}Q_{max}$	35.8 ± 8.8 (21–53)	24.7 ± 7.6 (10–37)	0.002
Q_{max} (cmH_2O)	11.6 ± 2.1 (7–15)	13.2 ± 3.5 (7–22)	0.003
PVR (ml)	82.9 ± 72.5 (0–276)	48 ± 26.6 (0–107)	0.006

Kabay et al

Percutaneous posterior tibial nerve stimulation as an effective treatment of refractory lower urinary tract symptoms in patients with multiple sclerosis: preliminary data from a multicentre, prospective, open label trial

C Gobbi[1], GA Digesu[1,2,3,4], V Khullar[3], S El Neil[4], G Caccia[2] and C Zecca[1]

Table 2. Study outcomes

Assessment	Pre-PTNS	Post-PTNS	*p*-value
Daytime frequency*	9 (6–11)	6 (5–10)	0.04
Nocturia*	3 (2–4)	1 (0–3)	0.002
Voided volume**	182 ml (±50)	225 ml (±50)	0.003
Post-micturition residual**	98 ml (±124)	43 ml (±45)	0.02
PPBC*	5 (5;6)	2 (2;3)	0.003
PPIUS*	4 (3;4)	2 (1;3)	0.005
UB-VAS (cm)*	10 (8;10)	6 (4;8)	0.005

Neurourology and Urodynamics 28

Acute Urodynamic Effects of Percutaneous Posterior Tibial Nerve Stimulation on Neurogenic Detrusor Overactivity in Patients With Parkinson's Disease

Sibel Canbaz Kabay, Sahin Kabay,* Mehmet Yucel, and Hilmi Ozden

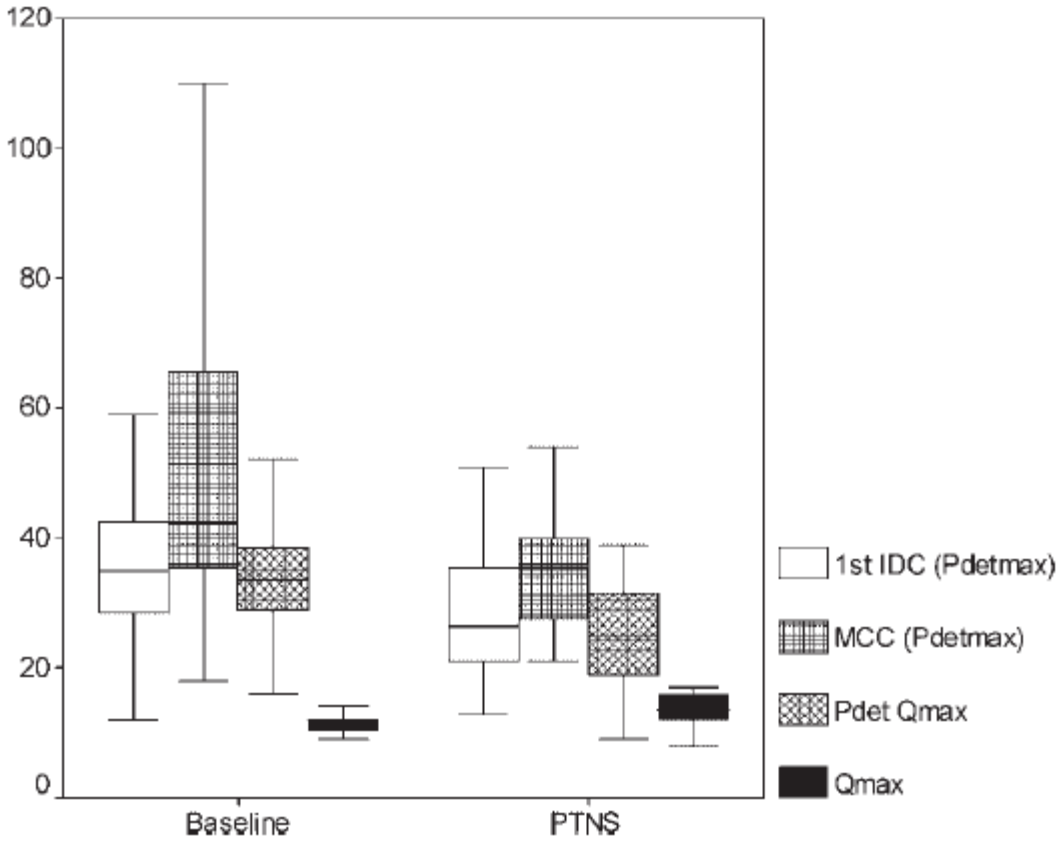
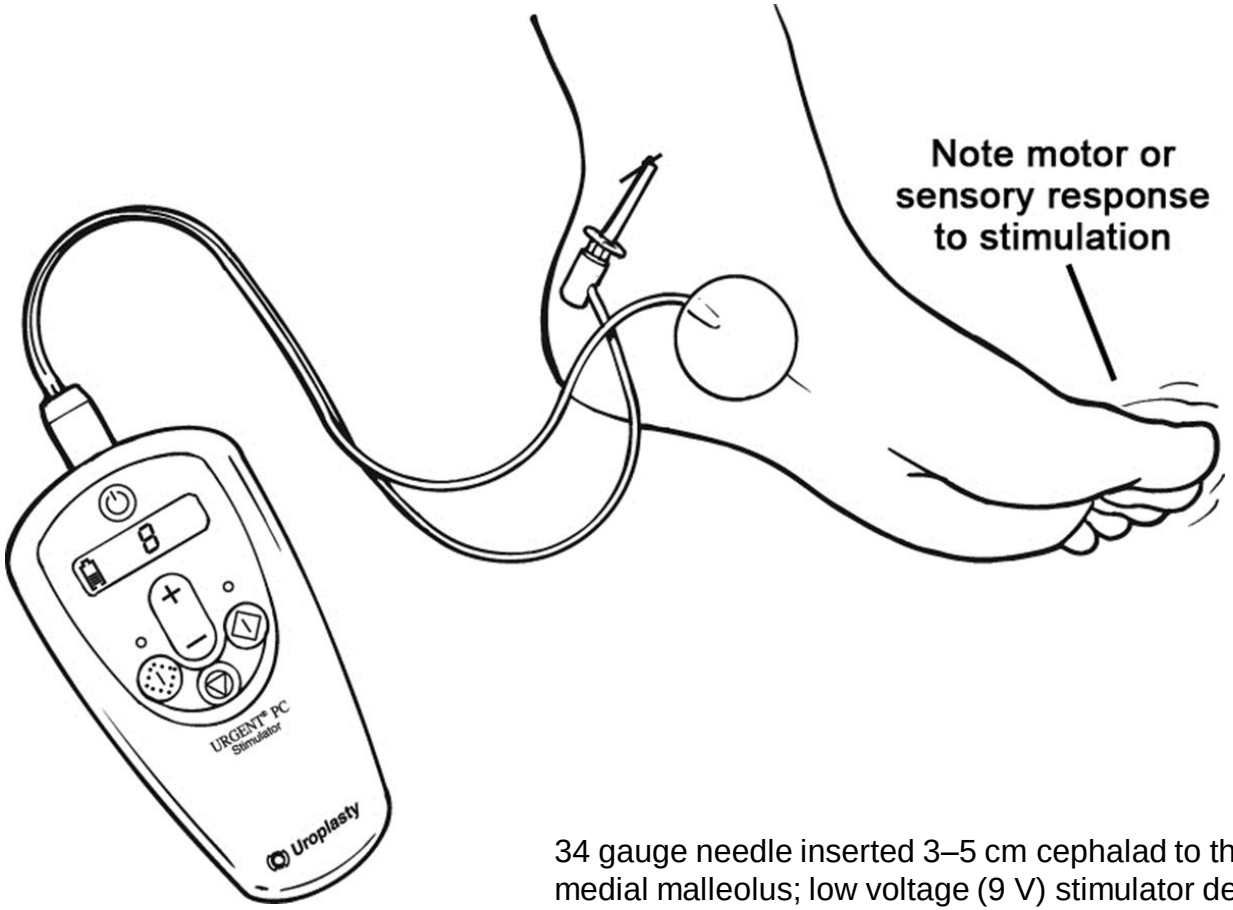
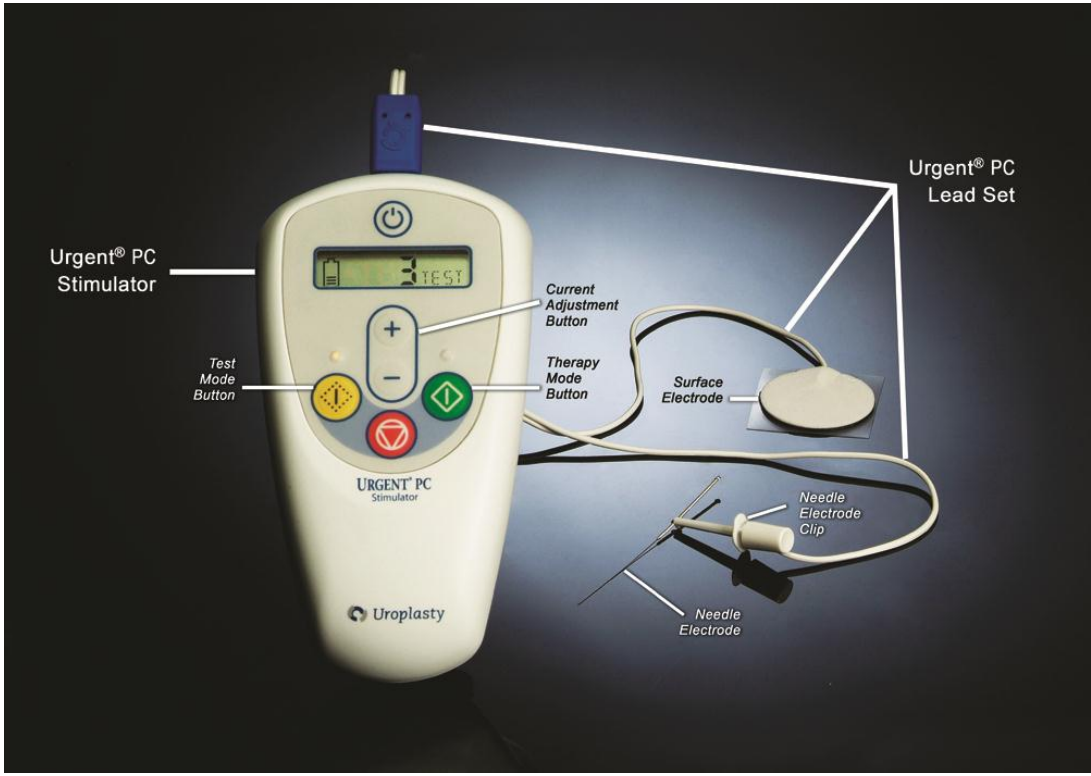


Fig. 2. Graphics of the effects of PTNS on urodynamic variables for the comparison of baseline and after PTNS findings in PD patients.

PTNS
(Percutaneous Tibial Nerve Stimulation)



34 gauge needle inserted 3–5 cm cephalad to the medial malleolus; low voltage (9 V) stimulator device, 0–10 mA, 20 Hz frequency, pulse width 200 ms


Urgent® PC
Lead Set
Urgent® PC
Stimulator
Current
Adjustment
Button
Test
Mode
Button
Therapy
Mode
Button
Surface
Electrode
Needle
Electrode
Clip
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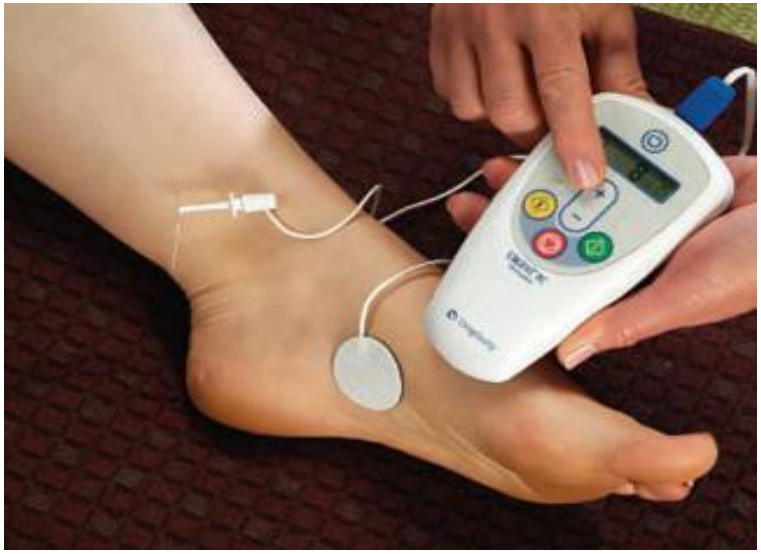
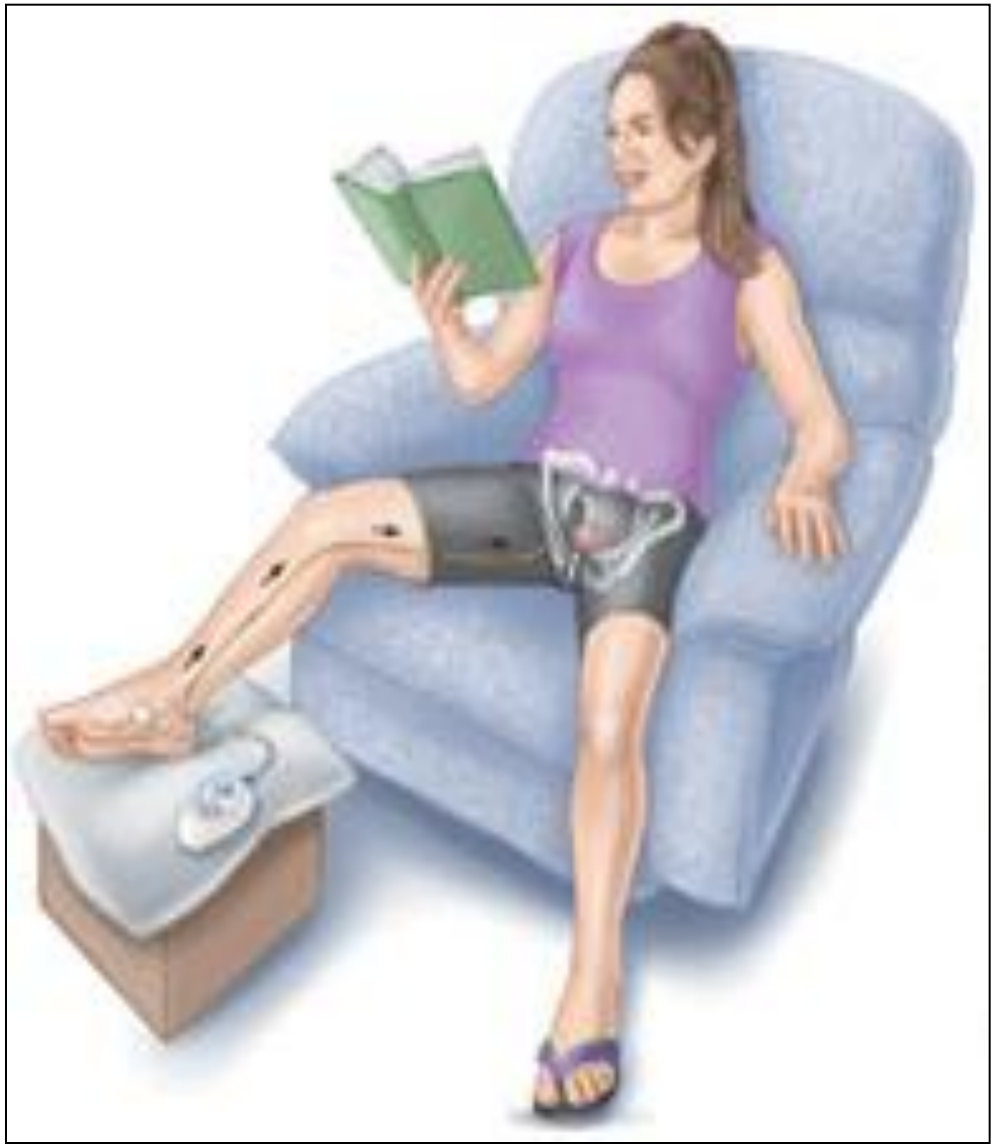
URGENT® PC
Stimulator
Uroplasty





Advantages PTNS

As a treatment option	As a service
• Minimally invasive procedure	• No additional resources required to establish the service in the department
• Impressive results in patients who have failed medications	• Nurse-delivered
• No major side effects. Specifically, no risk for requiring catheterisation or for urinary tract infections	• Simultaneous treatment of several patients possible
	• Cost benefit ratio > 1

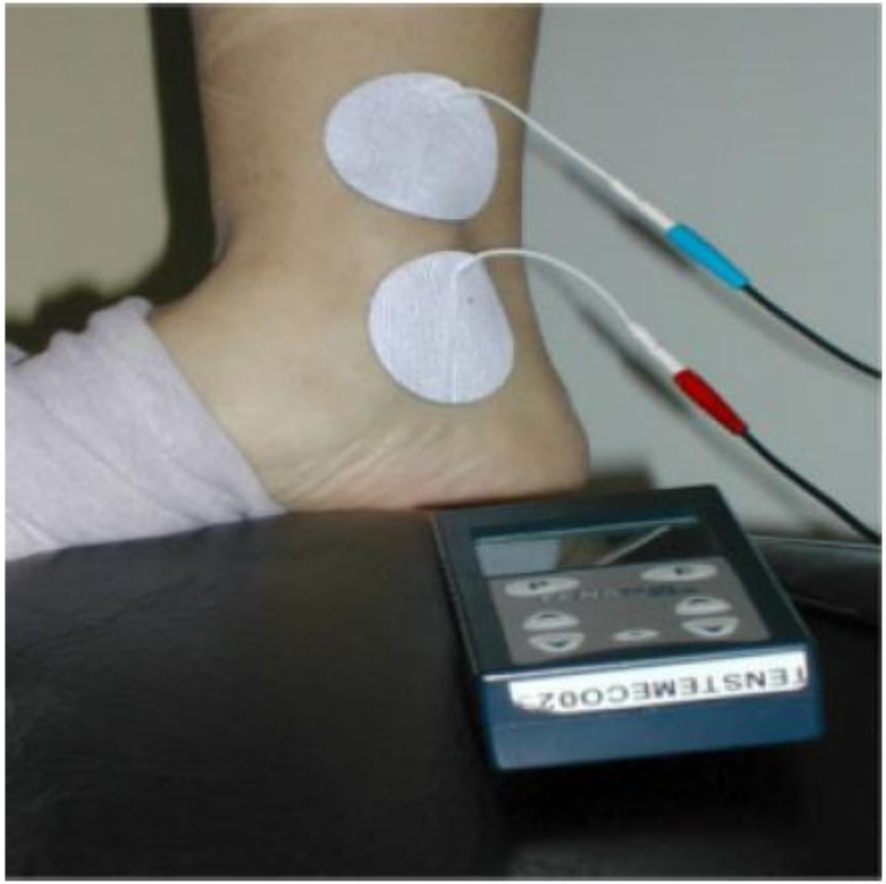
Caution

- Exclusion: pregnancy
- Caution: arrhythmias, pacemaker/ICD, excessive bleeding tendency

Neurourology and Urodynamics 30:306–311

Transcutaneous Posterior Tibial Nerve Stimulation for Treatment of the Overactive Bladder Syndrome in Multiple Sclerosis: Results of a Multicenter Prospective Study

Marianne de Sèze, M.D., Ph.D.[1,*] Patrick Raibaut,[2] Philippe Gallien,[3] Alexia Even-Schneider,[4] Pierre Denys,[4] Veronique Bonniaud,[5] Xavier Gamé,[6] and Gérard Amarenco[2]


TENSTEMECO02

Setting up a PTNS service?

- Age/Diagnosis
- Symptoms:
  - Bladder: frequency (day/night), urgency, incontinence, voiding symptoms and UTIs
  - Bowel: constipation or urgency/incontinence
- Post void residual
- Whether catheterising
- Co-morbidities- fibromylagia
- Other treatments tried for OAB and reasons for discontinuing : lack of efficacy, side effects, suitability

Setting up a PTNS service? (2)

- Sessions/stimulation parameter used
- Parameters to assess:
  - Bladder diary
  - Questionnaires, eg. ICIQ-OAB & ICIQ-LUTSqol
- Any adverse events: intolerable pain, infection, bleeding, miscellaneous
- Follow up: further treatments and intervals

Conclusion

- Successful treatment
- Level 1 evidence
- Useful for the patient with mild to moderate OAB