



## Pudendal Neuropathy: Current Views

W20, 29 August 2011 14:00 - 18:00

Start	End	Topic	Speakers
14:00	14:05	INTRODUCTION	<ul style="list-style-type: none"> <li>• Francesco Pesce</li> </ul>
14:05	14:30	ANATOMY & PHYSIOLOGY	<ul style="list-style-type: none"> <li>• Jacques Beco</li> </ul>
14:30	14:50	CLINICAL PRESENTATIONS	<ul style="list-style-type: none"> <li>• Francesco Pesce</li> </ul>
14:50	15:10	Diagnosis	<ul style="list-style-type: none"> <li>• Stanley Antolak</li> </ul>
15:10	15:30	Pharmacotherapy	<ul style="list-style-type: none"> <li>• Francesco Pesce</li> </ul>
15:30	16:00	Break	None
16:00	16:30	Manual Theraphy	<ul style="list-style-type: none"> <li>• Jerome Weiss</li> </ul>
16:30	16:50	Physical Theraphy	<ul style="list-style-type: none"> <li>• Cristina Shupe</li> </ul>
16:50	17:00	Pudendal block injections	<ul style="list-style-type: none"> <li>• Stanley Antolak</li> </ul>
17:00	17:15	Surgery: Open	<ul style="list-style-type: none"> <li>• Stanley Antolak</li> </ul>
17:15	17:30	Surgery: endoscopic	<ul style="list-style-type: none"> <li>• Jacques Beco</li> </ul>
17:30	18:00	Case Presentation & Discussion	All

### Aims of course/workshop

Pudendal Neuropathy (PN) is a relatively recent acquisition. Although well described in the literature, the majority of professionals fail to recognize it. AIM of the workshop is to elucidate the role of PN as the common feature of syndromes like DYSFUNCTIONAL VOIDING, NON OBSTRUCTIVE URINARY RETENTION, CHRONIC PELVIC PAIN SYNDROMES (including Vulvodynia, Chronic Prostatitis Type IIIA, Interstitial Cystitis/Painful Bladder Syndrome, Pelvic Floor Tension Myalgia), URINARY AND FECAL INCONTINENCE, ERECTILE DYSFUNCTION. The Agenda will cover: ANATOMY, ETIOLOGY, CLINICAL PRESENTATIONS. ASSESSMENT, MANAGEMENT, CASE PRESENTATIONS.

Participants will be able to recognize, diagnose and successfully treat this difficult condition.

### Educational Objectives

Pudendal Neuropathy is the number one leader in the list of unrecognized causes of pelvic pain. The common causes of this problem, estimated to occur in 20 of pelvic pain pts, are trauma from bicycle seats, sitting on hard chairs for prolonged periods of time, falls, pelvic surgical procedures, or from stretch injuries to the nerve such as chronic straining for bowel movements, childbirth, and exercises that require repetitive and/or vigorous squatting. The pudendal nerve can also become sensitive by receiving chronic pain signals from tender muscles and skin. The degree of diagnostic complexity is the reason that it may take years to uncover the underlying problems. For example, the average time to diagnosis of pudendal nerve entrapment is 4 years and only after seeing 10 physicians. The majority of patients actually make their own diagnosis using resources on the internet. There is a strong need to teach specialised MDs and PTs as there is much scepticism and a refusal to pursue the neuropathic basis of pelvic pain.

The ICS educational efforts have so far rarely focused on this important topic, and this Workshop will cover the gap with the help of some of the leading experts in the field.

# Pudendal Neuropathy: Current Views

## RELEVANT ANATOMY AND ETIOLOGICAL FACTORS

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### Anatomy

According to the literature and our dissections the anatomy of the pudendal nerve could be described as follow. The pudendal nerve is a mixed nerve carrying motor and sensory fibers. Its fibers are derived from the sacral roots S2, S3 and S4 (1,2). Once the roots traverse the sacral foramen, they divide into autonomic branches forming the pelvic plexus (parasympathetic supply of the pelvic organs) and somatic branches merging to form the pudendal nerve travelling under the piriformis muscle. Near its formation point it gives a levator branch running on the inner (upper) surface of the levator plate and providing the innervation of this muscle (1). For Barber et al (3), this levator nerve originated directly from the S3, S4 or S5 roots. Some somatic fibers coming from S2 and S3 run close to the pelvic plexus to innervate the levator ani and the urethral sphincter (1). Caudally, the pudendal nerve enters a small space ("clamp") between the sacro-spinosus and sacro-tuberous ligaments very near the ischial spine. Before the entrance in the clamp, we found in our last dissection a branch of the nerve which runs parallel to the great sciatic nerve in the posterolateral aspect of the thigh. Just below the ischial spine, the nerve gives a terminal branch, the dorsal nerve of the penis (1) or the clitoridal nerve (7). These nerves are separated from the main trunk by the pudendal vein and artery. Then it enters the Alcock's canal formed by a division of the obturator muscle aponeurosis. In the canal the nerve cross the sharp edge of the sacro-tuberous ligament (falciform process) (4,5). Caudally, at the level of the anus, the nerve gives medially the inferior rectal nerves (usually two branches) which innervate the anal sphincter (and probably the pubo-rectalis) and the skin of the posterior perineum and anterolaterally the transversus perinei branch (for this muscle and for the ischiocavernosus muscle) (1, 7). The remaining part of the nerve is usually called the perineal nerve. This nerve gives a bulbocavernosus branch and finally divides into a sphincteric branch (innervation of the urethra) and a branch which innervate the skin of the anterior perineum (6). Some aspects of this anatomy are still controversial and will be discussed during the lecture.

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## ETIOLOGY

The cause of the PCS is not always clear but it is often possible to find a compression (biking, long time sitting, haematoma...) or a stretching (descending perineum, surgery, delivery....) of the pudendal nerve in the Alcock's canal [1-10] in the history of the patient. A change in the shape or orientation of the ischial spine induced by some athletic activities during the youth could also explain some cases [11]. Pudendal neuralgia is a functional entrapment where pain occurs during a compression or stretch maneuver. The neuropathy worsens due to repetitive microtrauma resulting in persistent pain and dysfunctional complaints (13). The pudendal nerve is compressed during sitting and cycling and is especially damaged during motocross (14–16). Perineal pressure from an orthopedic fracture table causes pudendal neuropathy including impotence (17–19). Stretch of the nerve by straining with constipation and childbirth causes pudendal neuropathy measurable in the anal and urethral sphincters (20–24). Fitness exercises, machines, weight lifting with squats, lunges, and leg presses or karate with kick boxing and rollerblading are all etiologic factors.. Driving over rough roads or farm fields (26) causes vibration trauma. Falls onto the buttocks cause pudendal neuralgia (27). Iatrogenic neuropathy includes trauma during vaginal surgery and suture entrapment during colpopexy using the sacrospinous ligament (28–30). Radiation neuropathy following treatment of carcinoma of the prostate may relate to vascular impairment, inflammation within the nerve, or perineural desmoplastic reaction (31). We found very serious pudendal neuralgia in four patients after urine leakage had complicated hysterectomy or radical retropubic prostatectomy. Central sensitization plays an important role in aggravation and maintenance of symptoms in many patients.

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## CLINICAL PRESENTATIONS

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### HISTORY

Symptom history is paramount for diagnosis. Pain aggravated by sitting/driving/ exercise, reduced by recumbence or standing and relieved by sitting on a toilet, is pathognomonic. Symptoms of interstitial cystitis occur in both genders.

The quality of neuropathic pain varies and may be described as burning, stabbing, ache, or pressure. Pain may occur anywhere in the pudendal territory. Primarily this includes the perineum, scrotum, and penis/urethra but extends to suprapubic, inguinal, crural, anal, coccygeal regions, and the upper medial thighs. Scrotalgia and vulvodynia occur with pudendal neuropathy. Pain may be induced by voiding, defecating, ejaculation, vaginal penetration, orgasm, or simply with an urge to void or defecate. Stress and changes in the menstrual cycle aggravate pain. Urinary urgency, frequency, and slowing of the stream occur. Erectile dysfunction, ejaculatory impairment, and painful ejaculation occur. Females may suffer reduced clitoral sensation, pain at vaginal penetration, reduced lubrication, and anorgasmia. Obstructed defecation, narrow stools, and changes in consistency occur. Stress urinary incontinence, pelvic floor prolapse, and fecal incontinence are associated with pudendal neuropathy). Central sensitization (spinal cord wind-up) is apparent in some patients where aggravation of pelvic pain follows sexual arousal. Foreign body sensation in the rectum, vagina, urethra, or perineum is frequent. This may be the golf ball that is common in men with prostatitis-like pains. However, dramatic objects may be a red-hot bowling ball, a pine cone, a fist, or even a stovepipe. The size of the object changes with intensity of the pain. The objects are eliminated with successful treatment of neuropathy. Sacral cord neuroplasticity causes pains in the calf and the dorsum, arch, and toes of the feet that are aggravated during pain flares and eliminated after treatment.

### ASSESSMENT

#### *Physical Examination*

Physical examination focuses on a simple pudendal neurological evaluation. Pinprick sensation is tested at each branch bilaterally: dorsal nerve (clitoris and glans penis), perineal nerve (posterior labia and posterior scrotum), and inferior anal nerve (posterior perianal skin). Hyperalgesia is more common than hypoalgesia. Normal sensation to pinprick may occur even when quantitative sensory testing is abnormal. Pressure is placed on the nerve at the Alcock canal and medial to the ischial spine attempting to reproduce pain, bladder, or rectal symptoms—the Valleix phenomenon.

We evaluate the anal canal, sphincter, pelvic floor tone, and tenderness of the pelvic floor muscles. The parasacral area is examined for a back mouse (episacroiliac lipoma). We check for ilioinguinal and iliohypogastric neuropathies (vide infra). Concurrent involvement of these nerves will complicate the diagnosis, treatment, and control of symptoms. Inflammatory prostatitis must be excluded in male patients. Autonomic stimulation causes skin changes at the natal cleft, including cutis anserina and livedo reticularis, signs of the complex regional pain syndrome. Neurogenic inflammation produces peau d'orange. Glanular cyanosis or labial erythema occurs. The scrotum may be tight with the appearance of a tennis ball. Unilateral labial contraction may occur.

### ***Neurophysiologic studies***

Several tests can measure pudendal neuropathy including biothesiometry, sacral latency test, sensory-evoked potentials, motor-evoked potentials, and motor latency tests. Electromyography of the external urethral and anal sphincters and the bulbocavernosus and ischiocavernosus muscles may show denervation and reinnervation.

Anal and perineal pudendal nerve terminal motor latency test (PNTML) is rather controversial as several authors do not consider it a reliable and accurate diagnostic test. It is performed using the St Marks electrode to stimulate the pudendal nerve by the rectal route just under the ischial spine. For anal PNTML the electrical potentials induced in the striated anal sphincter are collected using the ring of this electrode. For the perineal PNTML the electrical potentials are collected with a concentric needle in the two bulbocavernosus muscles. Normal values in most laboratory are less than 2.5 msec for the anal PNTML and less than 5 msec for the perineal PNTML.

Non-invasive means of testing the sensation of the critical area innervated by the pudendal nerve are the warm detection threshold (WDT), a quantitative sensory test based on the threshold (in degrees of temperature) of detection of heat applied to the skin, and the Current Perception Thresholds (CPT) obtained with dedicated equipment (Neurometer), both very sensitive test for pudendal neuropathy.

### ***Imaging***

Magnetic resonance imaging (MRI) of the lumbosacral spine and plexus evaluate the spinal cord and nerve roots. Abnormalities are rare, including primary or metastatic tumors of unknown origin in the sacral canal, pelvic floor hernia, and local recurrence of carcinoma of the rectum anterior to the sacrum. Tarlov cysts usually are not the basis of patients' complaints. Judet views of the hips provide excellent images of the ischial spines. Magnetic resonance neurography is used by some practitioners to assist diagnosis. This technique awaits further study.

**PHARMACOTHERAPY**  
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First-line Medications. The efficacy of gabapentin, the 5% lidocaine patch, opioid analgesics, tramadol hydrochloride, and tricyclic antidepressants (TCAs) has been consistently demonstrated in multiple randomized controlled trials. Each one can be used as an initial treatment for neuropathic pain in certain clinical circumstances. Opioid analgesics and TCAs generally require greater caution than the other options.

**Gabapentin.** There are at least 8 published double-blind, placebo-controlled randomized clinical trials of gabapentin for chronic neuropathic pain. Gabapentin at dosages up to 3600 mg/d significantly reduced pain compared with placebo; improvements in sleep, mood, and quality of life were also demonstrated in some trials.

The adverse effects of gabapentin include somnolence and dizziness and, less commonly, gastrointestinal symptoms and mild peripheral edema. All of these effects require monitoring and dosage adjustment but usually not discontinuation of the drug. Gabapentin may cause or exacerbate gait and balance problems as well as cognitive impairment in elderly patients, and dosage adjustment is necessary in patients with renal insufficiency. However, its generally excellent tolerability, safety, and lack of drug interactions distinguish gabapentin from most other oral medications used for the treatment of chronic neuropathic pain.

To decrease adverse effects and increase patient adherence to treatment, gabapentin should be initiated at low dosages —100 to 300 mg in a single dose at bedtime or 100 to 300 mg 3 times daily—and then titrated every 1 to 7 days by 100 to 300 mg as tolerated. Although 3 times daily is the target dosage, more rapid titration may be accomplished if most of the daily dose is initially given at bedtime to limit daytime sedation. Target dosages that demonstrated benefits of gabapentin treatment for neuropathic pain ranged from 1800 mg/d to 3600 mg/d. If only partial relief of pain occurs at 1800 mg/d, titration can be continued up to 3600 mg/d (1200 mg 3 times daily) as tolerated. The final dosage should be determined either by achieving complete pain relief or by the development of unacceptable adverse effects that do not resolve promptly. An adequate trial of gabapentin would include 3 to 8 weeks for titration to allow the development of tolerance to adverse effects, plus 1 to 2 weeks at the maximum tolerated dosage.

**Pregabalin.** The anticonvulsant pregabalin has well established anti-neuropathic pain activity. The recommended dose is 150-600 mg daily (twice a day, titrated). Discontinuation has to be gradual (in one week). Common side-effects include dizziness, somnolence, mental confusion and cognitive impairment.

**Oxcarbazepine.** A recently introduced antiepileptic drug (AED), oxcarbazepine was found to possess antineuralgic properties in animal models of neuropathic pain. Several double-blind, placebo-controlled trials have evaluated oxcarbazepine in painful diabetic neuropathy and trigeminal neuralgia. There is good evidence that oxcarbazepine is effective in relieving the pain associated with trigeminal neuralgia and that it may be effective in treating neuropathic pain refractory to other AEDs, such as carbamazepine and gabapentin. The recommended dose is 300-1200 mg daily (titrated). All dosing should be given in a twice-a-day (BID) regimen. The most commonly observed ( $\geq 5\%$ ) adverse experiences seen in association with oxcarbazepine were

dizziness, somnolence, diplopia, fatigue, nausea, vomiting, ataxia, abnormal vision, abdominal pain, tremor, dyspepsia, abnormal gait. Approximately 23% of adult patients discontinued treatment because of an adverse experience in randomised controlled studies. The use of oxcarbazepine may interact with oral contraceptives and cyclosporine resulting in a lower plasma concentration of these drugs.

**Tramadol.** Tramadol is a norepinephrine and serotonin reuptake inhibitor with a major metabolite that is a  $\mu$  opioid agonist. There are 2 published double-blind, placebo-controlled randomized clinical trials of tramadol for neuropathic pain. In both trials, tramadol hydrochloride titrated to a maximum dosage of 400 mg/d significantly relieved pain compared with placebo. Beneficial effects of tramadol treatment on allodynia and quality of life were also reported.

The adverse effects of tramadol include dizziness, nausea, constipation, somnolence, and orthostatic hypotension. These occur more frequently when the dosage is escalated rapidly and with concurrent administration of other drugs that have similar adverse-effect profiles. There is an increased risk of seizures in patients treated with tramadol who have a history of seizures or who are also receiving antidepressants, opioids, neuroleptics, or other drugs that can reduce the seizure threshold. Serotonin syndrome may occur if tramadol is used concurrently with other serotonergic medications, especially selective serotonin reuptake inhibitors (SSRIs) and monoamine oxidase inhibitors. Tramadol may cause or exacerbate cognitive impairment in elderly patients, and dosage adjustment is necessary in patients with renal or hepatic disease. Abuse of tramadol is considered rare but has been observed.

To decrease the likelihood of adverse effects and increase patient adherence to treatment, tramadol should be initiated at low dosages—50 mg once or twice daily—and then titrated every 3 to 7 days by 50 to 100 mg/d in divided doses as tolerated. The maximum dosage of tramadol hydrochloride is 100 mg 4 times daily (in patients older than 75 years, 300 mg/d in divided doses), and an adequate trial requires 4 weeks.

**Tricyclic Antidepressants.** The first medication category that proved effective for neuropathic pain in placebo-controlled trials was TCAs. The primary problem with the use of TCAs is their adverse-effect profile; TCAs must be used cautiously in patients with a history of cardiovascular disease, glaucoma, urinary retention, or autonomic neuropathy. Almost 20% of patients treated with nortriptyline after a myocardial infarction developed adverse cardiac events in a recent study. Consequently, a screening electrocardiogram to check for cardiac conduction abnormalities is recommended before beginning treatment with TCAs, especially in patients older than 40 years. As with opioid analgesics, TCAs must be used cautiously when there is a risk of suicide or accidental death from overdose.

They may block the effects of certain antihypertensive drugs (eg, clonidine or guanethidine), and they interact with drugs metabolized by cytochrome P4502D6 (eg, cimetidine, phenothiazines, and class 1C antiarrhythmics). All SSRIs inhibit cytochrome P4502D6, and to prevent toxic concentrations of TCAs in the plasma, caution must be exercised in the concomitant administration of TCAs and SSRIs and when switching from one drug class to the other. In elderly patients, TCAs may cause balance problems and cognitive impairment. Milder adverse effects of TCAs include sedation, anticholinergic effects (eg, dry mouth or constipation), postural hypotension, and weight gain. Although most clinical trials of TCAs for neuropathic pain have examined amitriptyline, this drug is not recommended in elderly patients because of the risk of significant adverse events. Nortriptyline and desipramine hydrochloride have fewer adverse effects and are generally better tolerated than amitriptyline. In a recent randomized double-blind trial, nortriptyline was found to provide equivalent analgesic benefits in patients with



post-herpetic neuralgia when directly compared with amitriptyline but was better tolerated. Patients must understand that TCAs have an analgesic effect that has been demonstrated to be independent of their antidepressant effect. To decrease adverse effects and increase patient adherence to treatment, TCAs should be initiated at low dosages—10 to 25 mg in a single dose at bedtime—and then titrated every 3 to 7 days by 10 to 25 mg/d as tolerated. Although the analgesic effect of TCAs has been thought to occur at lower dosages than the antidepressant effect, there is no systematic evidence of this. However, some data are consistent with a dose-response relationship; TCAs should be titrated to dosages of 75 to 150 mg/d as tolerated. An adequate trial of a TCA would last 6 to 8 weeks with at least 1 to 2 weeks at the maximum tolerated dosage.

***Other antidepressants.***

**Duloxetine.** It is a Serotonin and Noradrenalin Reuptake Inhibitor (SNRI) with antidepressant and analgesic activity on neuropathic pain. The recommended dose is 60 mg once a day (better if titrated starting from 30 mg). More frequently reported side-effects are nausea, somnolence, dizziness, constipation and fatigue. It must not be taken in association with ciprofloxacin. Its use is contraindicated in patients with uncontrolled blood hypertension, liver insufficiency and convulsions. Abrupt discontinuation of the drug must be avoided.

**Venlafaxine.** The recommended dose is 37,5 mg twice a day (less in patients with liver or renal insufficiency). Abrupt discontinuation of the drug must be avoided.

**MANUAL THERAPY**  
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Direct trauma and severe or chronic stretch of the pudendal nerve are well known causes of pudendal neuralgia. However, beneath the surface, in the pelvic floor and sidewalls, another lesser-considered cause lurks. Anatomically, the pudendal nerve is closely associated with both the piriformis and obturator internus muscles. When these muscles become dysfunctional from injuries or biomechanical abnormalities, they can compress the pudendal nerve leading to neuralgia, inflammation, and/or increased vulnerability of the nerve to future minor traumas. The interconnections between the pudendal nerve and these muscles will be reviewed.

**How do biomechanical abnormalities affect the pudendal nerve?**

The pelvis is the anatomical center of the body and therefore can be influenced by upper body, local pelvic or lower body dysfunctions.

**In the upper body**, compression of the nerves that innervate the piriformis, L5 and S1, can cause tightening of that muscle and compression of the S2-S4 nerve branches that run through the piriformis muscle.

**In the pelvis** itself, the piriformis muscle can be injured with certain activities, hip pathology, or sacroiliac joint dysfunction.

**In the lower body**, it is important to check the feet, since abnormalities such as severe pronation can stress the hip rotators and thus compress the pudendal nerve. In addition, when the pelvic or core muscles are weakened by these lower extremity problems, there may be a tightening of the trunk, neck and jaw to maintain core strength.

**Muscle Dysfunction** can duplicate the symptoms of PN. One example is the iliococcygeus, the posterior segment of the levator ani muscle. It is innervated by S3-4 and the inferior rectal branch of the pudendal nerve, and therefore may cause many of the same symptoms as PN. They can be anal, coccygeal, and pubic pain, and urinary urgency/frequency.

Both may require treatment if their mutual symptoms do not resolve. This muscle should not be overlooked in the evaluation and treatment process.

**The pelvic floor muscle pudendal nerve “merry go round”**

Even though muscle dysfunction can cause pudendal neuropathy, pudendal neuropathy can cause muscle dysfunction. It is important to determine what came first to prevent recurrence, but it is also important to treat both. A basic principal is to treat the muscle first to find out if the extreme tenderness of the nerve diminishes. If so, continue with manual therapy of the surrounding muscles.

**The treatment** of pelvic floor myofascial trigger points centers around increasing blood flow to the ischemic muscle fibers.

The techniques are manual myofascial release, vapocoolant spray and stretch, dry needling, trigger point injections, and occasionally Botox injections.

When palpating muscles internally or externally, myofascial trigger points are identified as tight tender bands of tissue that are extremely painful. Compression may cause the patient to jump, the muscles to twitch, and the pain to refer to other areas.

The internal pelvic floor treatment combines stretching and compression, as well as mobilization of the pudendal nerve.

External muscles are treated in a similar manner, but if resistant may benefit from dry acupuncture needling or trigger point injections.

**Pudendal neuropathy can become a systemic disease.**

Therefore, all of the "fall out" needs to be treated for a successful outcome. The nerve pain spreads and intensifies as the surrounding muscles and fascia become stressed from protective movements, the subcutaneous tissue tightens and becomes tender from sympathetic over activity, and the central nervous system becomes sensitized. If all of these changes are not treated, it is a prescription for continued pain even after surgery.

**ABNORMAL BIOMECHANICS OF THE LOWER EXTREMITIES & PUDENDAL NEURALGIA**

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The extent to which musculoskeletal dysfunction contributes to Pudendal Neuropathy is an ongoing area of research. Current research supports our clinical findings that a number of musculoskeletal conditions may correlate with Pudendal Neuropathy. While it is unlikely that musculoskeletal impairments are the sole cause of pudendal neuralgia, their contribution to abnormal neurodynamics and/or myofascial pain that can result in neural irritation must be considered.

Finely tuned coordination of voluntary and reflexive muscle function attenuates stresses in the soft tissues (including neurovascular) and articular structures of the entire lumbo-pelvic-hip complex. Abnormal lower quarter biomechanics can interfere with this synchronized process and place undue stress on sensitive structures. Impairments in the lumbar spine, sacroiliac joints, as well as the piriformis, obturator internus and pelvic floor muscles have been implicated and continue to be investigated as a potential cause of pudendal nerve irritation. What has been less investigated is the role of abnormal hip and foot biomechanics as a primary source of the above-mentioned problems. This aim here is to describe and to encourage further investigation of several biomechanical abnormalities that are commonly seen in patients with diagnosed pudendal neuralgia.

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## **SELF –CARE, PUDENDAL NERVE PERINEURAL INJECTIONS AND SURGICAL THERAPY**

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The classic description of pudendal neuropathy is perineal pain aggravated by sitting and relieved sitting on a toilet seat. The pudendal nerve has been described as the social nerve because it affects bowel bladder and sexual function in addition to pelvic sensation. Thus the practitioner must be aware that any combination of dysfunctions and pain or sensory variations can indicate pudendal neuropathy.

Doctors in different specialties will see different subsets of pudendal neuropathy. Dr. Jacques Beco of Liege, Belgium has an entirely different patient “symptom population” than attends my clinic. He sees many pelvic floor issues such as prolapse and fecal incontinence – I see these infrequently. He sees more stress urinary incontinence than in my practice. Obviously the colorectal surgeon will see the chronic constipation, fecal incontinence and proctitis fugax in addition to proctalgia/perineal pain.

The requirements for diagnosis of pudendal neuropathy are:

1. Acceptance that pudendal neuropathy exists as a clinical entity.
2. Understanding that pudendal neuropathy causes many complaints in many combinations.

### **3. Examination for pudendal neuropathy.**

Pinprick examination of the three pudendal branches bilaterally: dorsal nerve (penis or clitoris; perineal nerve; inferior rectal nerve).

Observation of skin over natal (gluteal) cleft for changes of sympathetic overload.

Observation of labia for contraction - changes of sympathetic overload. (In males, contracted scrotum or penis).

Place pressure over pudendal nerve medial to ischial spine, during DRE or vaginal examination, to attempt pain reproduction. This is the Valleix phenomenon, similar to Tinel’s sign.

Evaluate pelvic floor for spasm and tenderness.

### **Evaluation for pudendal neuropathy.**

Imaging studies do not aid in diagnosis of pudendal neuropathy and are not recommended routinely. Blood tests do not aid in diagnosis of pudendal neuropathy and are not recommended routinely.

Neurophysiologic tests provide objective confirmation of pudendal neuropathy. Several tests are available. Two tests that I perform can be accomplished rapidly with low cost. The tests confirm pudendal neuropathy in 100% of my patient. I prefer a quantitative sensory test, the warm threshold detection. I also perform the pudendal nerve terminal motor latency test.

#### ***Warm Threshold Detection Test***

This test uses a small thermode placed on the skin surface in each pudendal branch. Beginning at a neutral temperature (usually 31.5 °C), the temperature is raised incrementally. The patient indicates when warmth is first perceived – the “threshold”. The test is repeated at 2°C and 1°C. Neuropathic responses are warmth perceived at >39.5 °.

Evidence of a nervous system in disarray are the pathologic responses that many sufferers notice such as tingling, hot, pain, pinprick, cold, etc that indicate central sensitization. Dramatic sensory reports during the WDT include pain/buzzing on foot or toes, warmth or pain in other pudendal sites, and warmth in the bladder or an urge to void. Some patients have persistence of the warm

signal after the warm stimulus is returned to 31.5 ° C or removed from the test site. This is called hyperpathia or metesthesia and is another indicator of central sensitization.

The test WDT takes about 10 minutes and is abnormal in 60 to 92% of my patients.

#### ***Pudendal Nerve Terminal Motor Latency Test (PNTMLT)***

This test measures the speed of an electrical stimulus along the pudendal nerve from the ischial spine to the sphincter. It requires DRE or vaginal exam and uses the St. Mark's Hospital technique. The stimulus and neural response are measured by computer (Sofomor Dantec/Medtronic). A prolonged PNTMLT indicates motor neuropathy. However the test stimuli often reproduce pudendal complaints. This is another indicator of neuropathy and central sensitization. The test takes about five minutes and is abnormal in 40 to 80% of my patients.

#### **TREATMENT OF PUDENDAL NEUROPATHY**

(Please be aware that multiple peripheral neuropathies cause pelvic pain – see the end of this paper). (I measure treatment results using symptom scores).

Three elements of neurophysiology must be considered during several months of treatment: 1. The nerve fibers; 2. The neurons of those fibers; 3. the central sensitization. Combinations of nerve protection (self care), medications, therapeutic nerve blockade and decompression surgery will be utilized in addition to medications.

#### **SELF CARE (nerve protection)**

About 10% of my patients note pain control with self care plus medications.

1. Stop sitting
2. Use a perineal suspension pad with the center removed to support the body on the ischial tuberosities.
3. Stop hip flexion exercises such as sit ups, Elliptical, jogging, weight lifting.
4. Stop cycling.

Permitted activities include walking, push ups, pull ups and swimming.

Some practitioners use physical therapy extensively in this group. I use this modality if the pelvic floor is in spasm.

#### **PUDENDAL NERVE PERINEURAL INJECTIONS (PNPI).**

A series of three PNPI at four week intervals provides pain control in about 60 % of my patients. The number of blocks and the intervals for successful responses are empirical observations over many years in Nantes, France and since 2000 in my practice. While at Mayo Clinic we did blocks at 2 and 4 and 6 week intervals that demonstrated the superiority of the 4 week interval for progressive pain control (unpublished data).

I infiltrate the perineural tissues with 6 mL of bupivacaine 0.25% and 1 mL of methylprednisolone or betamethasone.

Pudendal nerve blockade usually provides immediate, short duration (6 to 36 hours) relief from action of bupivacaine. Steroid responses begin at about 2-3 days post block and are maximal 2 to 4 weeks after blockade. In most patients a series of three PNPI provides a slow reduction of pain and improvement in bladder, bowel, or sexual dysfunctions. Complete pain control occasionally occurs after the first PNPI.

Non surgical interventions relieve complaints in about 70% of my patients.

I do not believe that PNPI should be considered a diagnostic test, rather they are therapeutic. Parenthetically, the symptoms during the PNPI and the changes in symptoms after each PNPI provide remarkable insight into the power of the pudendal nerve – the French call it the “King of the Pelvis”.

In most patients, pain control occurs several weeks earlier than improvement in bladder symptoms.

Examination two hours after each block is important. Pinprick test of pudendal sensation at six sites will identify the quality of anesthetic response. All blocks are not perfect and steroids will be ineffective if the anesthesia response is poor.

### **SURGICAL DECOMPRESSION OF THE PUDENDAL NERVE**

I use the transgluteal approach developed by Professor Roger Robert. I give each patient 50 mg of ketamine IV at the onset of the procedure.

Exposure of the pudendal nerves requires incision over each nerve. The gluteus muscle is split to expose the sacrotuberous ligament. This ligament is opened longitudinally in its midline so that its role in pelvic strength is not compromised. The nerve is identified anterior to the sacrotuberous ligament. Often the falciform process, an extension of that S-T ligament, requires division and removal as it may be a compressing structure.

Usually the nerve is compressed between the sacrospinous ligament (anterior) and the S-T ligament (posterior). Transection of the sacrospinous ligament divides the "lobster claw" or "clamp" compressing the nerve. Often additional "rogue" or anomalous fibrous bands compress the nerve and require division. Transection of sacrospinous fibers may need to proceed along the superior border of the ischial spine in order to transpose the nerve medially.

The dissection then proceeds distally to the trifurcation of the nerve. The pudendal canal is opened.

I leave a drain for 24 hours. After closing the incision, I place 10mL of 0.5% bupivacaine and 1 mL of methylprednisolone into the perineural space via the drain.

Surgical success rates throughout the world range from 65% to 75%. The patients on whom I operate have failed about 4-6 months of conservative treatments. I always identify nerve compression (see photos in podium presentation). The Nantes group identified slow pain relief postoperatively, often requiring 9 to 24 months. We identified that same response in patients cohorts operated in 2004 and 2005. Persistent post operative pain can be treated using medications, PNPI, IV ketamine, epidural blockade, sacral transforaminal blocks.

### **Additional pelvic peripheral neuropathies:**

Caveat: persistent pain after pudendal decompression surgery may be due to concurrent pain generators, chiefly the posterior ramus syndrome (Maigne syndrome) or ilioinguinal and iliohypogastric neuropathies. Patients may become pain free when these treated. Middle cluneal neuropathies and abdominal cutaneous neuropathy must be sought. A few post op patients with persistent perineal pain required blockade of the perineal branch of the posterior femoral cutaneous nerves. Less frequently I identify neuropathies of the obturator nerve, the genitofemoral nerves, the inferior cluneal nerves and the long perineal nerve.

### **MEDICATIONS**

At consultation I usually start amitriptyline and gabapentin. Side effects often require changing medication. Other helpful medications: clonazepam; Pregabalin; baclofen; clonidine; hydroxyzine; Flomax; Tegretol

### **OTHER TREATMENTS**

I occasionally use hypogastric plexus block for sympathetically maintained pain. I may give IV ketamine 20 mg over 20 minutes for severe central sensitization. I usually use this in postoperative patients but use it at consultation if allostatic overload is noted, for example, profuse diaphoresis,

vomiting, etc. from the intense neuropathic stimulation and high pain levels.

Postural correction exercises provide great relief for the posterior ramus syndrome (Maigne s.).

## **PUDENDOSCOPY: USE OF A RESECTOSCOPE WITH SALINE IRRIGATION DURING TRANSPERINEAL PUDENDAL NERVE DECOMPRESSION**

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The pudendal canal syndrome (PCS) and its surgical treatment have been described by Shafik in 1991 [1].

The three main symptoms of PCS are: - perineodynia (vulvodynia, perineal pain, proctalgia) - anal incontinence- urinary incontinence.

The two neurophysiological tests: - electromyography (EMG) of the anal sphincter and of the bulbocavernosus muscles. - pudendal nerve terminal motor latencies (PNTML) of the anal and perineal branches.

The three clinical signs of the PCS: -Abnormal anal or vulvar sensibility (tested with a needle comparing the left and the right sides of the vulva and of the skin 2 cm lateral to the anus, rated 0 = total anaesthesia, 1 = reduced sensibility, 2 = normal sensibility, 3 = hypersensibility. 0, 1 and 3 are considered as "abnormal sensibility". -Painful Alcock's canal on rectal examination. The pain induced by the palpation of the pudendal canal by rectal examination is evaluated using a seven levels ordinal scale : 0 = no pain, 1 = mild pain, 2 = mild pain with Tinel sign (irradiation of the pain), 3 = moderate pain, 4 = moderate pain with Tinel sign, 5 = severe pain, 6 = severe pain with Tinel sign. The Alcock's canal is considered "painful" if the pain is 4 or more. -Painful "skin rolling test": beginning from 5 cm behind the level of anus the skin is pinched and then rolled to the front until the skin fold is at the level of the clitoris. The skin rolling test is considered "painful" if it induced a severe pain at least at one level.

### **Minimal criteria for surgery**

At least one of the 3 following symptoms resistant to conservative treatments (physiotherapy, drugs, infiltrations, modification of diet or behaviour): a. Anal incontinence b. Perineodynia c. Urinary incontinence Associated with at least two of the five following criteria: a. increased anal or perineal PNTML b. pathological EMG of the anal sphincter or bulbocavernosus muscles (neurogenic trace, reduced activity: richness "poor" or "simple"). c. painful Alcock's canal on rectal examination (at least on one side) d. abnormal perineal sensibility (at least at one level) e. painful "skin rolling test" (at least on one side).

### ***Surgical procedure***

#### **Introduction**

In 1991, Professor Ahmed Shafik introduced transperineal pudendal nerve decompression for patients suffering of anal pain independent of defecation and anal incontinence. At this study, he opens the Alcock's canal and cuts the sacro-spinal ligament (and coccygeal muscle) under finger control [1]. After this first publication, he stopped severing the sacro-spinal ligament because he thought that the opening of the Alcock's canal is enough to suppress stretching on the nerve during defecation [2-6].

In 1997, he uses laparoscopy with a balloon dilator and CO2 to help his dissection but was not convinced by this approach and abandon it [7].

By only opening the Alcock's canal, we have obtained similar results as Professor Shafik on perineodynia, anal and urinary incontinences [8-9]. We have tried to improve the results of our procedure by adding the opening of the *fascia lunata* located between the sacro-spinal and sacro-

tuberous ligament but the results were the same [10-11]. More recently, Professor Shafik decided to come back to his first work and to cut again the sacro-spinal ligament in case of failure of his classical surgery [12] or in case of interstitial cystitis [13].

Since January 2009, the use of a bipolar resectoscope under saline infusion has changed completely my surgical approach [14]. With this approach I propose to call "Pudendoscopy", it is possible to visualize and to test the different levels of entrapment of the nerve during surgery. For example, a compression test that simulated sitting position has showed a new level of compression: direct compression of the nerve on the sacro-spinal ligament just above the "clamp". Because the section of the sacro-spinal ligament (most dangerous part of the surgery and probably indispensable in case of pain while sitting) is done under visual control, the small branches of the nerve that pass through the ligament and the vessels are better protected.

### Method

A classical resectoscope (usually used for endometrial or prostate resection) is introduced in the ischio-rectal fossa during transperineal pudendal nerve decompression. The patient is in gynaecological position. Warm saline irrigation is necessary to obtain a good visualisation of the nerve during the different steps of the surgery.

Here are the main steps of the procedure:

1. The rectal nerve is hooked with the finger.
2. By following this branch the surgeon reach the distal opening of the Alcock's canal. The stretching of the rectal nerve on the internal border of this opening during perineal descent is clearly visible.
3. After the opening of the canal, it is possible to follow and to free the perineal branch until it reaches the superficial perineum. This step removes the angle of the perineal branch that can be a source of pain and enabled a better mobilisation of the nerve.
4. It is also possible to see the nerve entrapped in the *fascia lunata* that closes the space between the sacro-spinal and the sacro-tuberous. Another level of possible entrapment.
5. After the opening of the *fascia lunata* with the finger, sometimes helped with the tip of Mayo scissors, the pudendal nerve runs around the sacro-spinal ligament or the ischial spine. The hole created by the finger between the 2 ligaments (usually 1 cm large) is clearly visible. Sometimes, the main trunk or a branch is included in the sacro-tuberous ligament.
6. A compression test that simulates sitting position can be done at this time to see how the pudendal nerve is compressed on the sacro-spinal ligament (or the ischial spine) by the sacro-tuberous ligament (in the "clamp") or more frequently above this ligament by the soft tissues.
7. The sacro-spinal ligament is cut with the bipolar resectoscope. At this time it is very important to be very careful to avoid cutting small branches of the pudendal nerve that are enclosed in the sacro-spinal ligament. The vessels are usually more laterally located but must be checked also.
8. After the cut of the sacro-spinal ligament the nerve is transposed medially to the ischial spine.
9. At the end of the procedure the nerve is free from the sacral roots to the superficial perineum.

### Results

The short term results of this new approach will be presented during the workshop.

### Conclusion

The use of a bipolar resectoscope with saline irrigation enables the surgeon to check the different steps of his decompression, to show the levels of entrapment and to cut the sacro-spinal ligament under visual control. Transperineal pudendal nerve decompression is not more a blind procedure.



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