Time	Time	Topic	Speaker
9:00	9:15	Relevant anatomy and etiological factors	Jacques Beco
9:15	9:30	Clinical presentations and assessment	Francesco Pesce
9:30	9:45	MANAGEMENT: - Pharmacotherapy	Mike Siroky
9:45	10:15	-Manual Therapy	Jerome Weiss
10:15	10:30	-Self-Care and Pudendal Blocks	Stanley Antolak
10:30	11:00	Break	
11:00	11:30	-Surgical Therapy	Jacques Beco & Stanley Antolak
11:30	11:40	Case Presentation	All
11:40	12:00	Discussion	All

Aims of 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.

Educational Objectives

AUDIENCE: Urologists, Urogynaecologists, Neurologists, Physical Therapists.

Participants in the Workshop will be able to recognize, diagnose and successfully treat this frequently overlooked condition and related syndromes.

Definition

Pudendal neuralgia is a peripheral neuropathy of the pudendal nerve that occurs in one or both sides in both genders. Pudendal neuralgia is characterized by perineal (and other pelvic) pain that is aggravated by sitting, generally decreased by standing and recumbence and typically reduced or relieved by sitting on a toilet seat. The syndrome may include suprapubic, inguinal, genital, and perineal pain, vulvodynia, coccygodynia, and proctalgia. Bladder, bowel, and sexual dysfunction occur. Synonyms include pudendal nerve entrapment or the syndrome of the pudendal canal (Alcock canal). Clinicians rarely associate these symptoms with pudendal neuropathy.

Conventional diagnoses include "prostatitis" or "prostatodynia" in males or endometriosis or vulvodynia in females.

RELEVANT ANATOMY AND ETIOLOGICAL FACTORS

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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 pyriformis 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 & ASSESSMENT

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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 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.

MANAGEMENT

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 gm 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 gm daily (titrated). All dosing should be given in a twice-a-day (BID) regimen. The most commonly observed (≥ 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 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 µ 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 allodynia48 and quality of life47 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, **n**ortriptyline 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. It's use is contraindicated in patients with uncontrolled blood hypertension, liver insufficiency and convulsions. Abrupt discontinuation of the drug must be avoided.

Venlaflaxine. 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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As in this syndrome the pudendal nerve becomes fixed and inflamed, therefore, one important treatment should be directed toward nerve mobilization with manual therapy to

decrease the nerve fixation and tension which can contribute to repetitive trauma. Since the pudendal nerve can be entrapped by the sacrospinous and sacrotuberous ligaments or the obturator internus and piriformis muscles, attention is directed to correcting dysfunction in these areas. I palpate the nerve to assess the areas of maximal tenderness, and then check all of the pelvic floor muscles for spasm and trigger points. I generally begin with the piriformis since its release has a profound effect on reducing spasm in the other muscle groups. When this is done internally, I insert my finger in the rectum or vagina while the patient is in the lithotomy position and grasping and pulling the knee of the involved side towards the opposite shoulder. The internal finger then sandwiches and compresses the muscle against the fingers of the opposite hand which is placed externally on the buttock. The compression continues intermittently and covers the entire muscle until it softens.
Once this is accomplished an internal active release technique is performed on the obturator internus. Active release technique, invented by Dr. Lahey, a chiropractor in Colorado Springs, is a method in which the patient actively moves a muscle group while the practitioner holds and stabilizes another. In the case of the obturator internus, the internal finger stabilizes the pudendal nerve in Alcock's canal while the patient grasps his or her ankle with the knee bent at a right angle. The lower leg is then used to rotate the thigh internally, thus stretching and separating the obturator internus muscle from the nerve.
Another important aspect of the physical therapy approach is to determine if muscle dysfunction created by the PNE is contributing to a significant portion of post surgical pain. Overlooking this possibility can lead to assumption of surgical failure. We have found that this surgical "failure" may not be a failure of surgery at all, but rather pain that has eveloped in muscles that have been subjected to long periods of nerve dysfunction. As a consequence, we have been able to help patients who have continued to have pain after surgery.

SELF –CARE

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Self-care is a program of nerve protection based on Robert's observation that sitting on a toilet seat relieves pains of pudendal neuralgia. Patients avoid sitting. When sitting is necessary, they use a "perineal suspension pad" fashioned quite inexpensively from a gardener's kneeling pad by cutting out the center (Fig. 2). The ischial tuberosities support the patient. The perineum is suspended. The ischioanal fat body descends, reducing pressure of the nerve against the falciform process of the sacrotuberous ligament. Activities that cause and aggravate pudendal neuralgia are discontinued, such as cycling, hip flexion activities including leg presses, Stairmaster®, ab crunches, jogging, and rollerblading. Walking on a flat surface and doing pushups are permitted.

Climbing stairs backward, using a "pick-up" tool, and wearing slip-on shoes are helpful in cases of extreme pain. Mothers must avoid squatting to lift children.

We advice amitriptyline 10 mg at h.s., increasing every 5 days to a maximum of 50 mg, adjusting the dosage for side effects. Narcotics generally do not control neuropathic pain. Pregabalin is effective in only a few of our patients and often has bothersome side effects. Monitoring symptom scores demonstrates that pain and bladder function may improve dramatically using self-care. Cure for more than 5 years has followed self-care. Occasionally, patients return to jogging and cycling, using a hornless saddle.

PUDENDAL NERVE PERINEURAL INJECTIONS (PNPI)

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Because pudendal neuropathy is a tunnel syndrome it can be treated in a manner analogous to the carpal tunnel syndrome; i.e. splinting, therapeutic nerve blockade, and surgical decompression.

Treatment of pudendal neuropathy should be sequential and dependent upon responses to simple measures. About 4 to 6 months are needed before a decision about surgical nerve decompression:

1. Self-care is a nerve protection program that prevents compression and stretching injury.
a. Avoid sitting
b. Use a pad with the center removed to prevent pressure on the perineum.
c. Avoid bending, squatting, lifting.
d. Stop cycling and hip flexion exercises such as jogging and exercise machines..
e. Medications such as amitriptyline 10 to 50 mg at hs.
2. Pudendal nerve perineural injections (PNPI) using bupivacaine and a corticosteroid.
a. A series of three blocks at one month intervals can be curative.
i. Two at ischial spine (in the interligamentary space).
ii. One into pudendal canal (Alcock canal).
3. Surgical decompression via transgluteal approach.

Results: 9% of males treated in 2005 responded to self-care. Another 56% had pain control following PNPI. Approximately 35% required decompression surgery. Pain control was achieved about 65% of these men without post operative interventions. Postoperative pain may require additional PNPI, epidural infusion of bupivacaine and/or intravenous infusion of ketamine.

Other pelvic neuropathies must be diagnosed and treated during the entire pudendal sequential therapies. The Maigne syndrome a major problem because of the suprapubic

pains and voiding complaints is causes. Ilioinguinal and iliohypogastric neuropathies may cause vexatious aggravation of suprapubic and crural pains. Neuralgias of the middle cluneal nerves, the posterior subcutaneous rami of S 2-3-4, may exacerbate pudendal symptoms

Transgluteal Pudendal Nerve Blocks: Technique
A simple, rapid, and effective fluoroscopic approach was originally introduced by Dr. Maurice Bensignor in Nantes, France. Two injections are given at the ischial spine at one month intervals.
A third is given into the Alcock canal by an interventional radiologist using CT guidance. I find this very effective. Ultrasound guided blocks or stimulus guided blocks are possible,
The fluoroscopic procedure is performed without sedation.
• The patient is placed in the prone position and a foam wedge is used to elevate the ipsilateral hip at about 35 degrees. Using fluoroscopy, the tip of the ischial spine is identified and skin is marked. The nerve is typically found immediately medial to tip of the ischial spine. Its position is variable.
• After skin preparation lidocaine 1% is infiltrated into the skin and subcutaneous fat. We use a 22 gauge 4-1/2 inch needle and slowly advance until the bone of the ischial spine is softly touched. Because the pudendal nerve is always found posterior to the ischial spine, this is the maximum extent of the needle. This depth is marked by fixing the ring finger to the needle at the skin level. The needle is retracted 2-3 cm and guided medially.
• The sacrotuberous ligament is posterior to the interligamentary space. The ligament may be soft or very firm and 3 to 12 mm thick. Needle passage through the ligament may be difficult and slightly painful. At this point the patient should experience rectal, perineal, or scrotal pain. The distance of the ring finger on the needle from the skin is the best means of gauging when the nerve will be stimulated.
• Retract the needle 1-2mm immediately when pudendal sensations are stimulated. Attach the syringe with 6 ml of bupivacaine 0.25% without epinephrine and the corticosteroid and aspirate. If there is no bleeding, the mixture is slowly infiltrated while the needle is slowly advanced. If bleeding occurs, a second site is used about 2 cm distal to the first. Extreme caution is necessary to avoid intravascular injection of the medications.
• Examine the patient two hours after the PNPI.
o Ask about changes in pain levels before and after the block (0 to 10 scale).
o Adequacy of the block is measured by analgesia or hypalgesia during pinprick examination of the three pudendal nerve branches bilaterally.
o Review symptom scores. The National Institutes of Health Chronic Prostatitis Symptom Index (NIH-CPSI) is a validated score available in many languages. It is simple, rapidly completed, and can be used weekly to monitor symptoms. Voiding symptoms are monitored using the American Urological Association Symptom Index (AUASI). Results demonstrate a mix of success or failure of treatment efforts.

Patients who have inadequate pain control after a series of PNPI are advised to undergo surgical decompression of the pudendal nerve. At surgery, those patients who fail pain control with PNPI typically have severe nerve compression, often proximal to the typical injection site at the ischial spine. Occasionally, at surgery, the nerve may be atretic, pale, or discolored suggesting ischemia.

Complications:
• Spread of local anesthetic to the sciatic nerve will cause weakness of the leg for one to two hours. Wheelchair support is recommended.
• Penetration of the nerve by the injection needle occurs in < 0.1% of injections at our facility. It is suggested by an immediate, sharp pain as the needle nears the expected pathway of the nerve. Several weeks may pass before the new pain is reduced.
• Pain "flares" or exacerbation of neuropathic symptoms occurs in about 5% of our patients. The onset is typically one or two days following the block. Pain flares may last a few days or one to two weeks.
• Intravascular injection will cause perioral paresthesias and oily taste.
• Rarely are new symptoms reported. Urinary incontinence may occur in female for a few hours. It is not common in males except after radical prostatectomy where the internal sphincter is typically not functional.

Results:
• Symptoms due to exercise trauma and sitting compression may respond well to the selfcare, nerve protection program. The longest response is >8 years.
• Pudendal nerve blocks must be given consistently at four week intervals in order to maximize therapeutic benefits. Three blocks were standard in the Nantes, FR program and I have found this very effective. My personal longest absolute cure is >8 years. I occasionally add o fourth PNPI for better symptom control.
• Therapeutic benefit of consistent PNPI at four week intervals is cumulative over time. Delayed symptom relief may occur 4-6 weeks after the third PNPI.
• Organ dysfunction such as voiding complaints, reversal of impotence, or normalization of defecation can also be transient with each block but complete responses generally take longer time than pain control.
• Second series of pudendal blocks are effective only when there is significant therapeutic effect of the initial series (months to years). The longest "cure" after a second series is over 6 years, measured by symptom scores. In some patients, sporadic "interval PNPI" may suffice to control symptom exacerbations.
• The severity of pudendal neuropathy cannot be gauged by clinical methods and it is not possible to predict responses to treatments. Duration of pain and severity of pain have some correlation with treatment results. Pain that persists overnight or in the recumbent position generally suggests entrapment. However, complete response to PNPI is possible

in these patients.

• There is no practical imaging that can guide the likelihood of treatment responses. A prolonged PNTMLT suggests more severe nerve damage. Paresthesias or dysesthesias during the warm detection threshold test suggest spinal cord windup.

• Transient pain control after a PNPI is called a "diagnostic response". I do not use PNPI for diagnosis. Diagnosis is clinical, confirmed with neurophysiologic testing. Failure of therapeutic effect is the indication to proceed to pudendal nerve decompression surgery.

• An entrapped nerve is identifiable only at surgery.

• The impact of central sensitization and windup require long term management with medications that may include IV ketamine, clonidine in addition to antieleptics.

Long term pain relief using PNPI is possible in those patients who failed multiple previous interventional techniques such as: physical therapy, caudal blocks, facet blocks, sacroiliac joint blocks, "trigger point" injections, ganglion impars blocks, hypogastric plexus blocks and nerve root stimulator. Misdiagnosis leads to use of these interventions. Definitive diagnosis of pudendal neuropathy is possible prior to any interventions. The clinician must also evaluate for:

• the Maigne syndrome or thoracolumbar junction syndrome

• Ilioinguinal and iliohypogastric neuropathies

• Abdominal cutaneous nerve entrapment.

• Middle cluneal neuropathy

I have rarely seen neuropathy of the perineal branch of the posterior femoral cutaneous nerve.

SURGICAL THERAPY

Pudendal Nerve Decompression Surgery: Perineal Approach [1]

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The pudendal canal syndrome (PCS) and its surgical treatment have been described by Shafik in 1991 [2]. 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 iss 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

Surgical procedure as described by Shafik in 1991 [2]. The operations were done under spinal or general anaesthesia. The patients were installed in the gynaecological position. The different steps of the procedure are:

- Vertical incision of the skin between the anus and the ischial tuberosity.

- Opening of the ischio-rectal fossa with scissors.

- The inferior rectal nerve is hooked under the finger and followed to the entrance of the Alcock's canal.

- Opening of this canal (without opening the clamp between the sacro-spinal and sacro-tuberous ligament).

- Control of the haemostasis.

- Self draining closing of the skin with nylon.

Comments
The pudendal nerve decompression by the perineal route is a blind procedure. The search for the inferior rectal nerve and the opening of the Alcock's canal are done under finger control. In our experience it is not easier with retractors. Therefore it is necessary to have a clear anatomical vision of this area before performing the operation. Maybe the use of a laparoscope would help [3] but the procedure will become more expensive and time consuming. To suppress the blind character of the procedure the transgluteal approach proposed by Robert [4] or the more recent transvaginal approach from Bautrant [5] could be other ways to treat the PCS. Until now the results on pain are the same as those obtained by the Shafik's approach but with the concurrent sections of one or two ligaments of the pelvis (sacro-spinal and/or sacro-tuberous ligaments).
However, we should be aware that the long term effects of these sections on the stability of the pelvic region are until yet unknown. Therefore, if the "clamp" must be open efforts should be done to open it without cutting a ligament. Up to now no data are available about a potential effect of the transgluteal or transvaginal procedures on urinary or anal incontinence.
Despite the blind character of the procedure we only had one heavy bleeding probably coming from the pudendal artery. One patient still presents with a mild intermittent clitoridal pain and a worsening of anal incontinence. Because the nerve of the clitoris leaves the pudendal nerve just before the entrance into the Alcock's canal this problem is probably the result of a too large dissection in the upper part of this canal. The two cases of anal incontinence worsening (gas incontinence becoming liquid incontinence), including the aforementioned patient, are difficult to explain. Maybe the neuropathy increased with the stretching involved in the procedure, the scarring process or a too large dissection. It could also be the result of the changes in the posterior level anatomy induced by concomitant procedures (easier expulsion of gas or faeces).
For the 2 patients the EMG data and the clinical examination after the operation did not improve therefore showing that the neuropathy was not healing.

Conclusions
Pudendal neuropathy is probably a frequent "defect" in perineology. Pudendal nerve decompression seems to be the defect specific procedure indicated in such a problem.

In fact it can treat perineodynia, anal and probably urinary incontinence. Anal incontinence can be cured by pudendal nerve decompression alone even in the presence of a clear disruption of the anal sphincter on anal ultrasound. Anal richness on EMG increases and PNTML decrease significantly after surgery proving an objective effect on the nerve. The

frequency of abnormal puncture sensibility, painful Alcock's canal and painful "skin rolling test" are significantly reduced by the operation. The three clinical tests could be used in practice to confirm or suspect the diagnosis of pudendal neuropathy in case of pain, urinary and/or anal incontinence.

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Pudendal Nerve Decompression Surgery: Transgluteal Approach

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I personally treat each step in the sequential treatment of pudendal neuropathy. Thus, only definite failures of self-care (nerve protection) and three (or four) pudendal nerve perineural injections (PNPI) are advised to undergo decompression surgery. The number varies in nnual reviews but is approximately 30-35% of consultations. The necessity for surgical intervention is consistently demonstrated by the severity of compression of the nerve. It is obvious that such compressed nerves would not respond to other than a decompression procedure.

The pathophysiology the surgical anatomy of pudendal nerve compression can be demonstrated through the transgluteal approach quite well. The nerve can be exposed from the fascicles of the lumbosacral plexus through the trifurcation of the nerve in the Alcock canal. A major advantage is the frequent surgical observation of anatomical variations that may be inaccessible via perineal/vaginal approach. Anomalies include a broad or sharp sacrospinous ligament and falciform process; abnormal nerve pathways penetrating through the sacrotuberous or sacrospinous ligaments and "tethering" of the nerve along the supralateral margin of the ischial spine. The transgluteal approach also permits visual access distally as the Alcock Canal is opened.

The procedure is performed in the prone position with the table flexed to "stretch" the buttocks. In Nantes they are moving to a near-kneeling position. The skin is marked at the approximate middle of the sacrotuberous ligament. An oblique incision is made between the sacral margin and the lateral aspect of the ischial tuberosity. Gluteal fascia is opened. Muscle bundles are separated to expose the sacrotuberous ligament. Occasionally a small potion of the gluteus muscle may need to be transected for exposure. The ligament is opened axially. I do not remove the ligament.

A retractor (Omni Tract, Minnesota Scientific, Inc., St. Paul, MN) retracts the edges of the sacrospinous ligament. The nerve is identified medial to the obturator internus muscle caudal to the ischial spine. It is elevated using a broad vessel loop. Dissection proceeds cranially, identifying and transecting any fascial structures compressing the nerve. The sacrospinous ligament is identified and it is transected. This releases the nerve and permits transposition anteriorly and medially. Perineural varices may develop after relief of compression similar to observations during carpal tunnel surgery. The coccygeous muscle must be separated from the ischial spine to permit transposition of the nerve medially and anteriorly to the ischial spine. The Alcock Canal is opened. Any adhesions or perineural fibrosis are released. An adhesion barrier is placed to minimize postoperative perineural scarring. A drain is brought through a separate stab wound. At the end of the procedure bupivacaine and corticosteroids are instilled through the drain an the drain remains off of suction for one hour. The sacrotuberous ligaments and gluteus fascia are approximated.

The patient stands on the evening of surgery and ambulates the following day. Typically, hospitalization requires two days. We advise a postoperative "gliding exercise". The hip is flexed and rotated laterally and medially, twice, bilaterally. Exercise is repeated twice daily for one to two years. Patients should not sit for one month except during meals. They continue to use the perineal suspension pad. Return to work varies from 10 days to three months. Some patients remain permanently disabled.

OBSERVATIONS DURING SURGERIES: Bony imaging and surgical observation provide the best anatomical information regarding pudendal neuropathy. Fresh cadaver dissection is better than prepared anatomic specimens.

Fluoroscopic views during PNPI occasionally demonstrate an elongated ischial spine that may need osteotomy (about 1% in my experience). Posterior and medial re-positioning of

the ischial spine by youthful athletics remodels the pelvis and places the sacrospinous and sacrotuberous ligaments in close proximity.

At surgery, the sacrotuberous ligament may be thick and indurated. The nerve may be tethered to its anterior surface. The falciform process may be prominent and compressive. Careful dissection is needed to open this structure and prevent damage to the nerve immediately anteriorly.

The sacrospinous ligament is quite variable. It may be a broad sheet over which the pudendal nerve stretches during flexion motions of the hip. It may have sharp, firm superior edge that impinges the nerve or there may be a firm ridge at its apex compressing the nerve against the anterior surface of the sacrotuberous ligament. Coalescence of fibers of the sacrotuberous and sacrospinous ligaments cranially may form an inverted funnel at the ischial spine. The aperture of this funnel may be extremely small, constricting the nerve. Tethering of the nerve at this site would cause stretching of the nerve during hip flexion. Perineural scar tissue has been identified fixing the nerve to the sacrospinous ligament.

He surgeon must be alert to other fibrous bands 1 to 10 mm wide along the nerve trunk that may compress the nerve against the anterior surface of the sacrotuberous ligament. Occasionally these bands penetrate and divide the nerve. Coccygeus muscle bundles may interdigitate with radiating bands of the sacrospinous ligament and 'bind' the pudendal nerve. Distally, fibrosis within the Alcock Canal may restrict the neural gliding mechanisms. Radiation fibrosis of the pudendal nerve has been identified with nerve compression. Discoloration of the nerve suggests devascularization. Small diameter of the pudendal nerve following previous pelvic surgeries may reflect infarction or atrophy due to interruption of the vasonervorum. The inferior rectal nerve is separate from the main trunk in about 10% of patients. It may be compressed as it penetrates through sacrospinous ligament.

Surgical success and "failure":

Defining surgical success or failure is challenging because of central sensitization or spinal cord windup that require months/years of management to obtain pain relief. The surgeon is responsible only for decompression of the nerve. Diligent patient nerve protection and "tincture of time" are the best post operative therapists. Early return to overenthusiastic exercise will hinder pain control. Surgeons indicate that 30% to 40% of patients fail to have significant relief following surgical intervention. Prolonged duration and severity of pre-operative symptoms are associated with poor results. Patients with severely compressed nerves are slow to achieve pain control.

Many of my "pelvic" pain patients have multiple neuropathies causing pelvis region pain.

• The Maigne syndrome, or thoracolumbar junction syndrome is present is about 40% of my patients with pudendal neuropathy. The suprapubic pains and bladder dysfunction from this problem respond only very slowly to postural correction exercises and infiltration of abdominal wall cellulalgia.

• Ilioinguinal and iliohypogastric neuropathies enter the pudendal territory and complicate pain relief after pudendal decompression surgery. Occasionally separate ilioinguinal and

iliohypogastric neurectomies are necessary.

• Middle cluneal neuropathy (posterior rami of S 2-3-4) may produce symptoms comparable to pudendal neuropathy. These nerves are a vexatious problem, easily overlooked except by a consistent physical examination at consultation.

• Abdominal cutaneous nerve entrapment may be a concurrent neuropathic pain generator.

• About 50% of patients choosing unilateral surgery may subsequently a later contralateral procedure to control symptoms.

Surgeons generally report reduction of pain in 60 to 75% of operated patients. Bladder, bowel, and sexual dysfunctions improve. In a controlled study, Robert reported durable improvement at four years in nine of 12 cases. Unoperated controls were unimproved at one year.

Data from the *Pudendal Neuralgia Foundation* (unpublished) indicate improvement continuing as long as three years after decompression surgery (measured by validated symptom scores). Post operative results from 2004 indicate that average pain and voiding scores were normal at 18 months post op. Data from males seen for consultation in 2005 indicate that averages of pain scores were normal by 24 months post op.

Other post operative observations:

• Urine retention in hospital is seen in about 5% of men and women and requires catheterization once or twice after the indwelling catheter is removed. Only one male was discharged with an indwelling catheter.

• Wound complications in over 500 nerve decompressions are limited to one hematoma and one case of lobular, subcutaneous fat necrosis and one superficial wound infection.

• Four patients developed pneumonia.

• Neuropraxia occurs and requires several days to several weeks to resolve.

• Pelvic instability occurred in a woman operated in 2004 following transection of the sacrotuberous ligament. Since that time I open the ligament along its midline vertical axis and retain the integrity of the ligament.

• Scar tissue can develop and re-tether or compress the nerve. This has also occurred after use of cadaver fascia to replace the sacrotuberous ligament.

Treatment of Postoperative Failures

Always examine for concurrent pelvic neuropathic pain generators in patients with persistent post operative pain (vide supra). Bensignor in Nantes, France, treated persistent pain by repeating pudendal nerve blocks of steroid and bupivacaine as soon as two months after surgery.

He treated sympathetically maintained pain for five days using infusions of ketamine and clonazepam via epidural catheter.

We treat failures from several surgical venues using perineural blocks of bupivacaine and heparin 4000 units with 0.8 ml of NaHCO3. Six weekly injections are given followed by gradual increase of the interval. Dense scar tissue may be identified by the needle and require significant

pressure to infiltrate medications. After repeated injections softening occurs and infiltration of heparin solution meets less resistance. Conversely, a complete series may be unsuccessful.

Adjuvant intravenous ketamine (20 mg in 5 mg bolus over 20 minutes) can provide significant pain control when used is a progressive program over many weeks. It is safe in an outpatient setting.

Re-operation has been performed by some surgeons identifying scar tissue or inadequate transection of ligaments (unpublished). One patient had a foreign body granuloma from chemical adhesion barrier.

SUMMARY

Pudendal neuropathy is a tunnel syndrome. Decompression surgery to divide the "clamp" between the sacrotuberous and sacrospinous ligaments or to decompress the pudendal is needed in about 30-35% of patients. The transgluteal approach described by Robert et al permits visualization of the nerve from above the ischial spine to the urogenital diaphragm. Many anomalies of the nerve and ligaments occur and demand meticulous dissection. Reported surgical success rates range from 60 to 70%. Pain-free status may require four to five years. Bladder, bowel, and sexual dysfunctions show variable improvement. Persistent symptoms after four to six months require re-evaluation for concurrent neuropathies (Maigne syndrome, ilioinguinal, middle cluneal) and attention to spinal cord windup. Post-operative interventions progress from perineural injections to epidural anesthetic infusions or intravenous ketamine. A spinal cord stimulator completed the pain control in one patient. Sacral nerve root stimulators have not been effective in controlling pudendal neuropathy.