Observational Study

Chronic Pelvic Pain: Neurogenic or Non-Neurogenic? Warm Detection Threshold Testing Supports a Diagnosis of Pudendal Neuropathy

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Background: Chronic pelvic pain (CPP) in men is rarely considered to have a neurogenic (neuropathic) basis. Separation of neurogenic from non-neurogenic pain is possible using clinical examination and neurophysiologic tests. A definite diagnosis of neuropathic pain can be made.

Objectives: We aim to demonstrate that definite pudendal neuropathic abnormalities can be supported by a quantitative sensory test (QST) called the warm temperature threshold detection (WDT) test in men with CPP.

Study Design: This is a retrospective review of 25 consecutive, unrecruited men evaluated in a private clinical practice beginning on January 1, 2010. The techniques of examination and neurophysiological testing have been standard since 2003.

Setting: A private practice that is a referral center because of its focus on CPP of a neuropathic basis.

Methods: Pinprick sensation was evaluated at 6 sites in the pudendal nerve territory (3 branches on each side). A WDT was performed at each nerve branch using a Physitemp NTE-2C Thermoprobe and Controller. This used a stepping algorithm from a neutral baseline of 31.5°C. Quantitative and subjective "qualitative responses" were recorded. Our preferred symptom score to evaluate pain level at consultation is the National Institutes of Health Chronic Prostatitis Symptom Index (NIH-CPSI). The results become the benchmark for comparison of responses following future treatments (not discussed). When possible, microscopy was used to evaluate prostate secretions for inflammatory prostatitis except in 2 men with CPP who had undergone previous radical prostatectomy for cancer. Observations were made of the skin in the pudendal territory. Our specific evaluation for neuropathy also sought evidence of multiple additional neuropathic pelvic pain generators.

Results: The WDT was abnormal in all men (88% quantitative), and pinprick sensation was abnormal in 92% of the men. The combination of tests provided a diagnosis of pudendal neuropathy in all patients, resulting in an accurate and timely explanation of the neurogenic basis of their CPP symptoms. The NIH-CPSI scores ranged from 10 to 35 (median 25). Four of 15 men had inflammatory prostatitis in addition to pudendal neuropathy.

Limitations: There is selection bias because the men were either self-referred, suspecting their diagnosis from internet searches, or were referred by physicians who were aware of the focus of this clinical practice. The warm temperature testing used established normal values for the men. The NIH-CPSI does not evaluate sexual or bowel symptoms. Sensitivity or specificity values for the tests could not be obtained.

Conclusions: A possible neuropathic basis for CPP in men can be suspected from symptoms and history of activities. A probable diagnosis of neuropathy can be determined using a pinprick sensory evaluation in the pudendal territory. A definite diagnosis of pudendal neuropathy can be made using WDT. The combination of these 2 examinations demonstrated pudendal neuropathy in 100% of this cohort.
The institutional review board deemed this study met criteria for exemption.

Key words: Chronic pelvic pain, pudendal neuropathy, quantitative sensory testing, warm temperature detection threshold test, neuropathic pelvic pain, definite diagnosis of neuropathy, chronic prostatitis

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Typically, diagnoses of chronic pelvic pain (CPP) in men include prostatitis, interstitial cystitis, orchalgia, epididymalgia, varicocele, inguinal hernia, and others. The conundrum of CPP includes at least 22 syndromes in both genders (1). None of the syndromes separate neurogenic pain from non-neurogenic pain, although it is possible to distinguish a neurogenic cause of CPP. There are several peripheral neuropathies that may cause CPP, but these are rarely discussed in medical literature (Table 1).

A hierarchy is available to designate the certainty of a neuropathic basis of pain. The levels of certainty include: unlikely, possible, probable, and definite (2). A simple sensory examination of the pudendal territory supported by a quantitative sensory test (QST) can provide a definite diagnosis of pudendal neuropathy in men with CPP. Patients' symptoms and medical history suggest the possibility of a neuropathic origin. A cutaneous pinprick evaluation, when abnormal, supports the probable neuropathic basis of CPP (3). A definite diagnosis of neuropathy can be made using a QST. This progression of simple evaluations can demonstrate pudendal neuropathy in patients with CPP; however, QST is rarely reported in a clinical, private practice setting (4,5). One type of QST, called the warm temperature detection threshold (WDT), can objectively quantify sensory changes in patients with CPP (6). Our experience suggests that many patients with CPP, whether untreated or with persistent pain after previous unsuccessful treatments, have definite pudendal neuropathy by pinprick examination and neurophysiological testing (4). Unfortunately, the methodology sections of most articles regarding CPP include only a perfunctory note that the neurological examination was "normal" or there may be no comment about any neurological evaluation, especially a specific examination for pudendal neuropathy (7). "The need to quantify is central to any scientific process; one cannot make any valid conclusion about disease mechanism, epidemiology, natural history or response to therapy without quantifying the relevant parameters (8)."

Table 1. *Pudendal neuropathy and additional peripheral neuropathic generators of pelvic pain. In descending order of approximate prevalence in patients with CPP.*

Pudendal Neuropathy
Thoracolumbar junction syndrome or posterior ramus syndrome (Maigne syndrome)
Middle cluneal neuropathy
Ilioinguinal-iliohypogastric -bilateral
Ilioinguinal-iliohypogastric -unilateral
Abdominal cutaneous nerve entrapment
T-12 Posterior cutaneous perforating branch
T-12 Posterior ramus
Perineal branch of PFCN
Posterior femoral cutaneous nerve
Genitofemoral nerve
Inferior cluneal nerve

PFCN = posterior femoral cutaneous nerve

Pudendal neuropathy is a polysymptomatic tunnel syndrome and affects both genders from childhood to nonagenerians (9). The pudendal nerve is a mixed nerve affecting sensory, motor, and autonomic functions. Complex symptoms may include pain, bladder, bowel, and sexual dysfunction. These encompass a "pudendal syndrome" (Table 2). Perineal pain is a common complaint (10). Men are often erroneously considered to have "prostatitis" (11). The causes include repetitive microtrauma (lifting, sports, and exercise activities) and nerve compression while sitting. It is considered a cumulative trauma syndrome.

Several neurophysiological tests have been performed during evaluations for a neuropathic basis of CPP (10,12-19) (Table 3). Sensory tests include sensory evoked potentials and sensory conduction velocity of the dorsal nerve of the penis. WDT is ideally suited for use in pain syndromes because it evaluates the small diameter, unmyelinated C-fibers responsible for transmission of neuropathic pain and autonomic signals (20). C-fibers also constitute the major pathway for central sensitization, a problem found in many CPP patients.

Neurologists recognize that testing pinprick sensation is an excellent "bedside" test for identification of peripheral neuropathy (21). WDT results are not identical to bedside testing but are complementary. Bleustein et al (22) established WDT as the best method to identify neuropathy in impotent men. This stimulated our experience beginning in 2003. WDT is not a heat-pain test, which evaluates larger nerve fibers. It is a psychophysical test akin to audiogram or visual field testing and assesses the somatosensory system.

We aim to introduce WDT testing in men with CPP and to demonstrate results of WDT testing in the 6 branches of the pudendal nerve territory for confirmation of pudendal neuropathy in men with symptoms suggesting pudendal neuropathy and/or men with abnormal pinprick testing in the pudendal territory.

Table 2. *Symptoms of pudendal neuropathy confirmed after successful pudendal nerve blocks and/or decompression surgeries, as reported in medical literature (not a complete list).*

Pain Locations	**Perineum, scrotum (testicles), penile -glans or urethra, anus, suprapubic, coccygeal**
Pain Aggravators	Sitting, driving, tying shoes, cycling, exercise, full bladder, defecation
Pain Relievers	Sitting on a toilet seat, standing, recumbence
Bladder Symptoms	Pain with filling, pain relieved by voiding, loss of feeling of fullness or urge to void, painful urination, urethral pain
Bladder Symptoms: Irritable	Frequency, urgency, nocturia, urge incontinence
Bladder Symptoms: Storage	Hesitancy, slow stream, feeling of incomplete emptying, retention
Sexual Symptoms - Pain	Pain during or after ejaculation at any of the above sites, pain with sexual thoughts or arousal
Sexual Symptoms - Function	Reduced sensual pleasure, erectile dysfunction, reduced volume and force of ejaculation, multiple unwanted daily erections (persistent arousal)
Rectal Symptoms	Proctitis fugax, pain prior to and/or during defecation, pain relieved by defecation, inability to defecate, infrequent stools, constipation, excessive numbers of stools, fecal/gas incontinence, loss of feeling of fullness or urge to defecate, pain as ampulla fills
Central Sensitization	Pain in feet/toes/calves, nausea, weight loss, diaphoresis, flushing, tachycardia

Table 3. *Neurophysiologic testing for diagnosis of pudendal neuropathy.*

Author	**Publication**	**No. of Patients**	**Gender**	**Neurophysiologic Tests Used**
Amarenco et al (12)	Rev Neurol 1997; 153:331-334. (in French)	170	M 53 F 117	EMG; SEP; SL; PNTMLT
Amarenco et al (13)	Muscle Nerve 2001; 24:116-119.	6	M	EMG, SEP, bulbocavernosus latency, PNTMLT, SCVDNP
Antolak & Antolak (4)	J Pelvic Med Surg 2006; 12:35-40.	8	F	WDT; PNTMLT
Bautrant et al (14)	J Gynecol Obstet Reprod Biol 2003; 32:705-712.	200	F	PNTMLT, EMG; SEP; sacral reflex latency
Beco (15)	BMC Surg 2004; 4:4-15.	74	F	EMG, PNTMLT
Benson et al (16)	Am J Obstet Gyn 2005; 192:1663-1668.	64	F	PNTMLT, EMG, bulbocavernosus reflex
Klausner & Batra (17)	J Urol 1996; 156:1425-1427.	55	M 40 F 15	SEP
Popeney et al (18)	Neurourol Urodynam 2007; 26:820-827.	58	M 32 F 26	PNTMLT & EMG
Robert et al (10)	Surg Radiol Anat 1998;20:93-98.	150	M1/3 F 2/3	PNTMLT
Shafik (19)	Pain Digest 1993; 3:252-256.	4	M	PNTMLT & EMG
Shafik (28)	Eur J Obstet Gynecol Reprod Biol 1998; 80:215-220.	12	M	PNTMLT; anal and levator EMG

EMG = electromyogram (anal sphincter, levator ani, pelvic floor); PNTMLT = pudendal nerve terminal motor latency test; SEP = somatic evoked potentials; SSCVDNP = sensory conduction velocity of dorsal nerve of penis; SL = sacral latencies; WDT = warm detection threshold; M = male; F = female

Methods

Twenty-five consecutive, unrecruited men scheduled appointments requesting evaluation at a pelvic pain clinic for their symptoms of CPP between January 5, 2010 and July 25, 2010. The patients were self-referred following review of symptoms on the internet or were referred by physicians alert to the possibility of neuropathic pelvic pain. No exclusions were made despite previous interventions, medical diagnoses, or therapies. The patients' ages ranged from 24 to 82 years (median 48). Each patient completed a CPP symptom survey

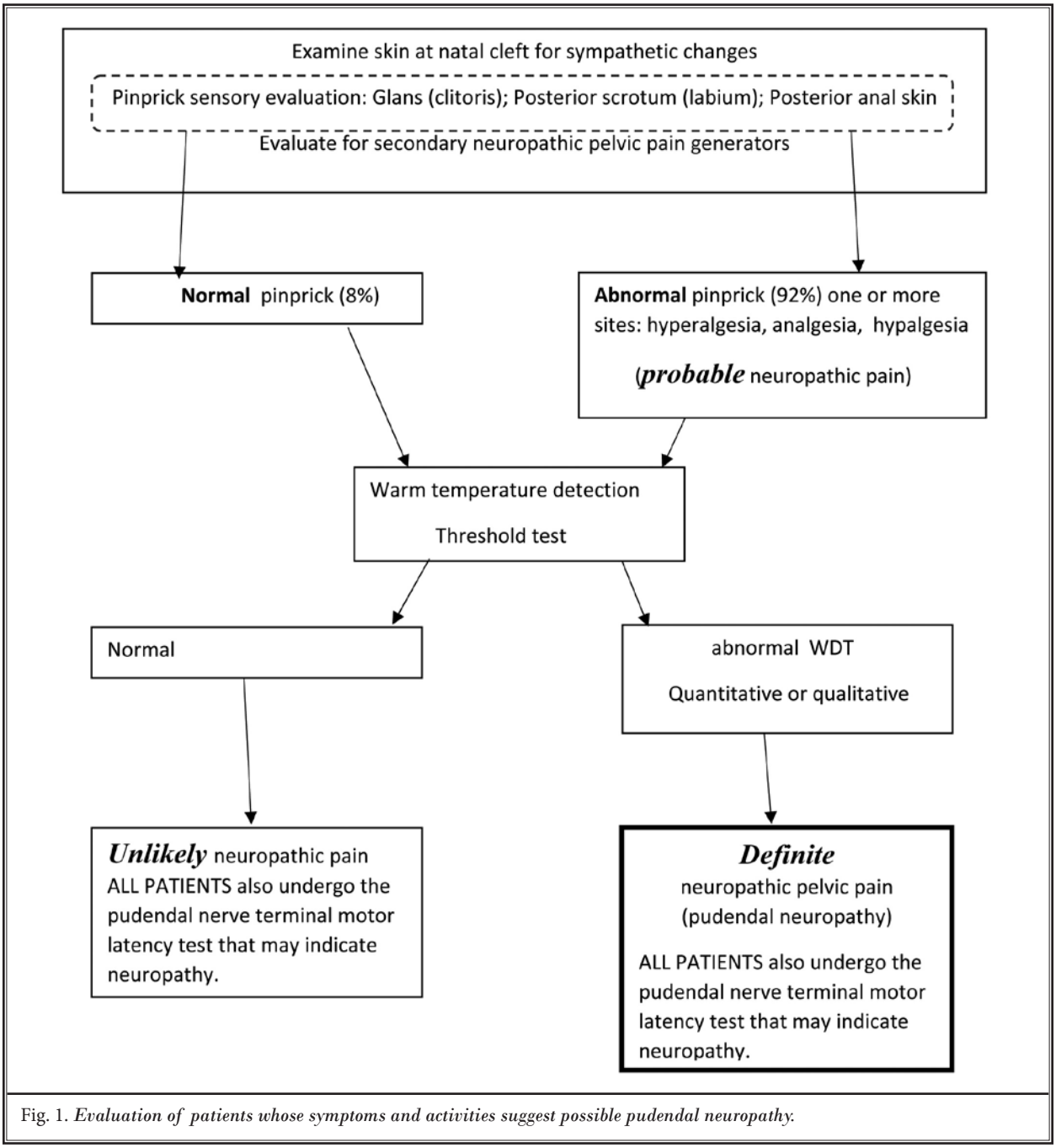

Fig. 1. *Evaluation of patients whose symptoms and activities suggest possible pudendal neuropathy.*

that focused on neuropathic pelvic symptoms, including aggravating and relieving factors. Pain levels were established and monitored using the National Institutes of Health Chronic Prostatitis Symptom Index (NIH-CPSI) (23). It includes 4 domains: pain, urinary symptoms, impact of symptoms, and quality of life.

The physical examination and evaluation focused on potential neuropathic sources of CPP, but it also included evaluation for prostate inflammation (Fig. 1). Pinprick sensory testing was performed at the lateral glans penis, the perineum anterior to the anus, and the posterior perianal skin. The anterior thigh (lumbar in-

nervation) was used as a normal site for comparison. The skin was observed for possible sympathetic changes at the coccyx, sacrum, and gluteal crease (S2-3-4 dermatomes). Additional neuropathic pelvic pain generators were sought including abdominal cutaneous neuropathy, ilioinguinal and iliohypogastric neuropathies, T-12 posterior ramus and T-12 posterior cutaneous perforating neuropathies, middle cluneal neuropathies, and neuropathies of the perineal branch of the posterior femoral cutaneous nerves. These are diagnosed by firm digital pressure over the nerve pathway. Prostate massage was performed to evaluate expressed prostate secretions for inflammatory prostatitis (24).

Pinprick testing was used in an attempt to include or exclude the clinical impression of possible pudendal neuropathy. The sites tested were within the distribution of each of the 3 pudendal nerve branches bilaterally – glans penis, posterior scrotum (perineum), and posterior perianal skin. The normal comparison site for the patient was the anterior thigh (lumbar territory). Light pressure on the skin assured a sharp sensation but avoided pain (3).

Definite evidence of neuropathy was sought using WDT. This employed the NTE-2C Thermoprobe and Controller (Physitemp Instruments Inc., Clifton, New Jersey), which has a small thermal probe that measures only 0.44 cm^2; a stepping algorithm was used (25). Prior to testing, a thorough explanation was given regarding the process and the patients' expected responses. They were asked to comment about any perceptions occurring in any part of their body during the testing. The small thermode was placed without pressure on the skin, with its temperature at 31.5°C (neutral in most patients). Contamination of the probe is prevented using a vinyl sheath made from an examination glove; this did not affect the results. Quantitative testing uses a stepping algorithm with temperature increments of 4°, 2°, and 1°C. The first sensation of warmth, the threshold, was reported by the patient. The maximum temperature used was 43.5°C to prevent denaturing of protein (at 45°C, erythema). Temperature increments of 4°C may stimulate severe pudendal pains due to wind up. Since 2005, patients are tested only with 2°C and 1°C increments. Three pudendal branches were tested bilaterally: glans penis laterally, the posterior scrotum at its perineal reflection, and the posterior perianal skin at the anal verge. Test sites are > 1 cm from the sagittal midline and, at the anal sites, > 1 cm posterior to the coronal midline. The locations assure testing within the receptive field of each individual pudendal nerve branch without overlap from contralateral pudendal nerves or adjacent nerve territories (26) (Fig. 2).

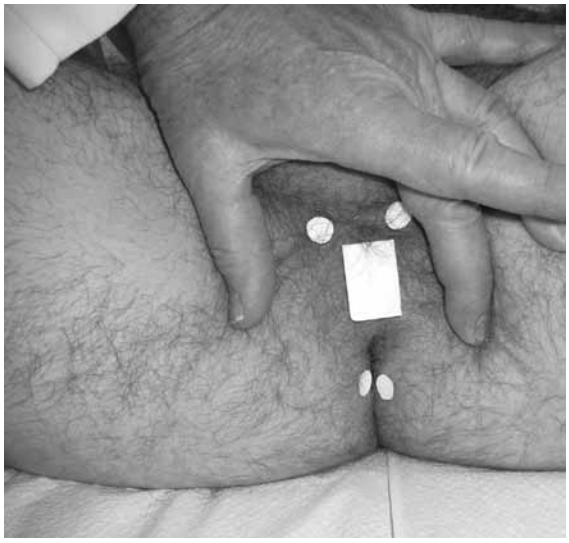

Fig. 2. *WDT testing showing placement sites for the 1 cm diameter NTE-2A Thermosensory Tester thermal probe (round circle) at the perineal nerve and inferior rectal nerve sites. Dorsal nerve sites are laterally near corona of glans (not shown). The 2x3 cm patch represents position of a different commercial thermode used in articles discussed in this paper. The large size overlaps into bilateral receptive fields.*

Normal penile WDT is 36°C +/- 1.89°C (22). To be conservative, we chose an upper range of normal as < 39.5°C. This is 2 standard deviation from Bleustein et al's (22) (vide supra) published normal values using the same NTE-2C Thermoprobe and Controller. An introductory test was performed on the anterior thigh.

Qualitative (subjective) abnormalities of the WDT, indicating central sensitization, were recorded. These include painful sensations (warm allodynia) or dysesthesias (tingling, cold, etc). After sensations persisting after removal of the thermode and/or dislocation of symptoms to distant sites were recorded.

Evaluation for inflammatory prostatitis used digital prostate massage to obtain expressed prostate secretions for microscopic examination. The expressage was immediately collected on a microscope slide, covered with a cover slip, and examined (400x power). The average number or range of white blood cells (wbc) and macrophages in each of multiple fields were counted per high power field (hpf) and recorded. Abnormal is considered > 10 wbc/hpf (24).

The study is considered exempt from the institutional review board under paragraph 45 CFR 46.101 (b)-4.

Results

25 men with CPP, ages 25 to 84, were evaluated because of symptoms suggesting pudendal neuropathy. Perineal pain occurred in only 13 of 25 (52%). However, sitting aggravated the chief complaint in 15 men (60%). Bilateral pudendal neuropathies predominated, being identified in 24 of 25 men (96%). Pinprick and WDT responses were incongruous at test sites (Table 4).

Table 4. *Responses to WDT (quantitative and qualitative), pinprick examination, and microscopy of prostate secretions.*

Patient; Number/Age (yrs)	Side	Glans	Scrotum	Anal	EPS	Skin Change	*Qualitative Responses to WDT Test
1 / 59	R L	33.5 *(+) 37.2*(+)	36.5*(=) >43.5*(+)	>43.5 (+) 35.6(+)	NO	Pdo Ca	Pulsating and tingling, throbbing, sharp pain L scrotum felt in L Leg
2 / 48	R L	>43.5 (o) 39.1 (+)	>43.5 (o) 39.1 (o)	>43.5 (+) 36.5 (+)	NO	NR	None
3 / 52	R L	39.9*(+) 35.5*(+)	41.5 (+) 37.5*(+)	>43.5 (=) 40.5 (+)	RP	O	Pinprick sensation, R glans also felt in R great toe, pinprick sensation left scrotum
4 / 50	R L	>43.5*(o) 37.2*(+)	33.6(o) 35.1 (+)	41.8*(o) 35.4(+)	NO	Pdo	Tingling, cold at glans, indescribable sensation anus
5 /25	R L	41.5 (+) 35.4 (+)	41.5* (+) 39.0 *(+)	39.6 (+) 43.5 (-)	Borderline	Ca	Felt dysesthesias only at 2°C increment
6/ 53	R L	36.0 (+) >43.5 (+)	35.8 (+) >43.5 (+)	36.6 (+) 35.5 (+)	Rare	NR	None
7 / 54	R L	38.3 (o) 40.1 (-)	38.3 (o) 40.1 (-)	35.4 (o) 36.1 (o)	0–8 wbc	Pdo	None
8 / 45	R L	>43.5 (+) >43.5 (+)	>43.5 (+) 42.5 (+)	43.5 (o) 35.5 (o)	0–40 wbc Macro	NR	None
9 / 45	R L	>43.5 (o) >43.5 (+)	36.8 (o) 38.9 (+)	40.8 (-) 41.2 (+)	NR	pdo	None
10 / 82	R L	>43.5 (=) >43.5 (-)	>43.5 (+) >43.5 (-)	>43.5 (=) >43.5 (=)	30–80 & Macro	NR	None
11 / 30	R L	37.5 (-) 34.5 (+)	35.5 (+) 37.5*(+)	35.5 (+) 37.5 (+)	Neg	O	Felt indescribable change at 2°C increment L glans, felt indescribable change at L scrotum
12 / 69	R L	34.9* (+) 34.7 * (=)	35.8*(+) 35.1*(+)	36.3 (+) 36.0*(-)	RP	Trophic Changes	Glans and anal sites-- tingling, L scr felt at L thigh, R scr felt at glans
13 / 63	R L	>43.5 (=) 36.%*(=)	37.5 (=) >43.5 (=)	42.5 (=) >43.5*(=)	NO	O	Indescribable sensation
14 / 24	R L	37.5 (=) 38.5 (+)	>43.5 (=) >43.5 (=)	37.5 (=) 39.5 (+)	NO	NR	None
15 / 48	R L	43.5 (=) 36.3* (+)	>43.5 (+) >43.5 (+)	>43.5 (=) >43.5 (+)	WNL	Ca	Glans felt cool at 2°C increment
6 / 26	R L	40.3 (=) >43.5 (-)	35.1 (=) 39.0 (=)	36.7 (-) 36.1 (-)	0–5 wbc	O	None
17 / 30	R L	37.0 (=) 43.5 (=)	43.5 (-) 34.9 (=)	36.0 (-) 40.0 (-)	No wbc	Ca	None
18 / 38	R L	>43.5 (=) 40.8* (=)	37.9 (+) >43.5 (+)	37.6 (=) 34.9 (=)	Neg	Ca Extensive	L glans felt warm tingling at 2°C increment
19 / 55	R L	35.4 (+) 33.8 (+)	35.1 (+) 39.8 (+)	38.7 (=) 43.5 (=)	NO	Pdo	None
20 / 47	R L	37.7 (-) 37.3*(-)	43.5*(+) 42.6*(+)	38.8 (-) 40.&*(-)	NO	O	Tingling at 5 of 6 branches

Table 4 (cont.). *Responses to WDT (quantitative and qualitative), pinprick examination, and microscopy of prostate secretions.*

Patient; Number/Age (yrs)	Side	Glans	Scrotum	Anal	EPS	Skin Change	*Qualitative Responses to WDT Test
21 / 29	R L	35.3 (+) 35.1*(+)	37.6*(=) 37.7*(+)	35.5 (-) 35.4*(+)	WNL	NR	Cold at glans and scrotal site, indescribable sensation at anal site
22 / 51	R L	39.7 (=) 39.3*(=)	>43.5 (=) >43.5 (=)	36.7 (=) 38.7 (=)	0–30 wbc clumps	CR sacrum	Indescribable sensation at glans
23 / 36	R L	37.8 (o) >43.5 (o)	37.0 (-) >43.5 (-)	>43.5 (=) 37.4 (-)	WNL	NR	None
24 / 43	R L	43.4 (-) 36.0*(-)	33.9 (+) 36.4 (+)	37.4 (=) 35.9 (=)	WNL	O	Indescribable sensation at glans
25 / 53	R L	>43.5 (-) >43.5 (+)	>43.5 (-) >43.5 (=)	38.5 (=) >43.5 (=)	NO	NR	None

R = right; L = left; (+) = hyperalgesia; (-) = hypalgesia; (=) = normal; Sc = scrotum; = vermification– a disorganized, repetitive convolutional movement of the dartos muscle; EPS = expressed prostate secretions per high powered field; NE = not expressed; NO = not obtained; WNL = within normal limits (< 10 WBC/HPF); WBC = white blood cells per high power microscopic field; Macro = macrophages; Ca = cutis anserina; CR = cutis reticularis; Pdo = peau d'orange; NR = not recorded; O = no changes observed

All 25 men had pudendal neuropathy diagnosed (100%) either by abnormal pinprick (92%) and/or abnormal WDT (100%) (Table 3). The 2 men with normal pinprick sensation, cases 13 and 22, had abnormal WDT studies. Pinprick testing showed no correlation with WDT results. For example, 4 patients could not feel 43.5°C at some sites, but pinprick at the same site varied from analgesia to hypalgesia to normal and hyperalgesia.

Abnormal *quantitative* warm detection threshold testing occurred in 22 of 25 men (88%). The 3 men with normal quantitative WDT (cases 11, 12, and 21) had *qualitative* impairment of warm perception, resulting in dysesthesias and dislocations, meaning the warm temperature signals were perceived at sites distal to the thermoprobe. Each also had bilateral abnormal pinprick sensory testing. Thirteen men noted allodynia or dysesthesia at normal or abnormal temperatures. The sensations included throbbing, tingling, pain, hot, cold, and a vague "indescribable sensation" or "change." In patient 3, the right glanular paresthesias were also felt in the right great toe.

Skin manifestations of sympathetic stimulation in the posterior sacral territory were found in 11 of 17 men where recorded or 64.6%. These included cutis anserina (5), peau d'orange (5), cutis reticularis (1), and trophic changes (1).

In 4 men, the dartos muscle responded to normal warmth by contractions of a twisting, rolling, convoluted nature that we call vermification. This appears to represent disorganized autonomic signaling and was stimulated at both normal and elevated temperatures.

Expressed prostate secretions showed inflammation in 4 of 15 (26.7%) men from whom expressage could be obtained. Two men with "prostatitis-like" complaints had previous radical prostatectomy for carcinoma of the prostate.

The NIH-CPSI scores ranged from 10 to 38, with a median of 25.

Additional neuropathic pelvic pain generators were noted in 64% of men with pudendal neuropathy. These included: thoracolumbar junction syndrome (Maigne syndrome) (2), abdominal cutaneous neuropathies (5), ilioinguinal and/or iliohypogastric neuropathies (12), and middle cluneal neuropathies (6).

Discussion

Among patients referred to our practices, past diagnostic errors in men with CPP are quite common. Misdiagnosis occurs because the polysymptomatic pudendal syndrome may not only include pain at multiple sites (scrotum, penis, urethra, perineum, prostatitis, etc.), but also causes functional abnormalities. Those affecting the bladder may be misdiagnosed as interstitial cystitis or "non-neurogenic neurogenic bladder" (27). The multiple issues within the context of sexual dysfunction (erectile dysfunction, anemission, painful ejaculation, persistent arousal syndrome) are typically not considered to be caused by pudendal neuropathy (28). Manifold interventions are associated with these symptoms, and they are often ineffective.

Pudendal neuropathy can be suspected at initial consultation based on symptoms. Key symptoms that are indicators of a "possible" neuropathic basis are as

follows: pain aggravated by sitting and driving that is relieved sitting on a toilet seat, allodynia to underclothing, and a history of youthful athletics.

An important caveat about a "possible" symptomatic diagnosis is that perineal pain is considered by some authors to be the sine qua non for diagnosing pudendal neuropathy (Nantes criteria) (29). However, in the present cohort, perineal pain was a symptom in only 52% of the men. Multiple additional "chief complaints" included penile pain (n = 7), scrotal (testicular) pain (n = 5), and coccydynia, proctalgia, abdominal pain, and erectile dysfunction. The Nantes criteria would exclude 48% of this cohort from the possibility of having pudendal neuropathy. This conflict arises because the major diagnostic criterion of the Nantes group is relief of perineal pain during sitting following a lidocaine pudendal nerve block. Such blocks are generally less than 80% effective (30). Moreover, up to 10% of patients with an International Association for the Study of Pain "definite" diagnosis of pudendal neuropathy do not require therapeutic perineural blockade for treatment. They respond simply to lifestyle changes (a self-care, nerve protection program) and medications (30). Perineal pain may also be caused by neuropathy of the perineal branch of the posterior femoral cutaneous nerve.

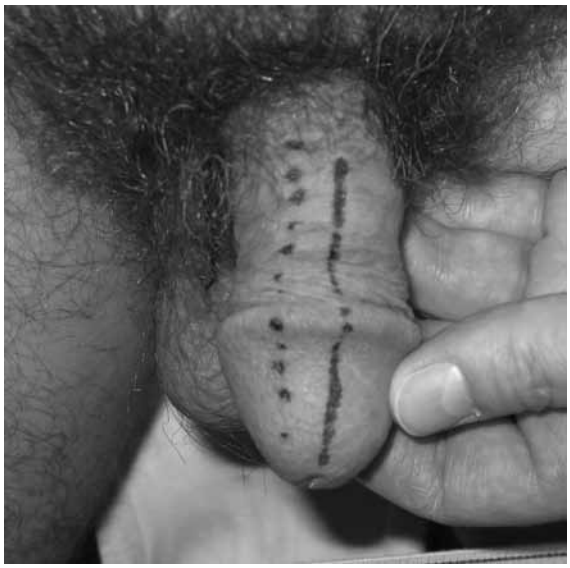

Fig. 3. *Unilateral penile anesthesia following a successful left unilateral perineural pudendal nerve injection. Anesthesia to pinprick crosses the midline (solid line) to the contralateral right side of the dorsum of the penis (dotted line). The right pudendal nerve had not been injected.*

QST is utilized in pain neurology for support of the neurological diagnostic process, e.g., when neuropathy is probable because of pinprick sensory changes (31). WDT is more reproducible than other QSTs (2). The WDT test can also be used in children (32). We have successfully tested children as young as age 13 for pudendal neuropathy.

In men with CPP, small nerve fiber neuropathy was postulated because application of topical capsaicin to the perineum caused excessive response compared to normal males (33). The WDT measures the integrity of small, unmyelinated C-fibers. Our experience with thousands of WDT tests indicates that damage to the small fibers that carry neuropathic pain is the crux of diagnosing pudendal neuralgia. Abnormal WDT also supports the diagnosis of complex regional pain syndrome II.

We chose to not record heat pain and cold pain because these measure large, myelinated A-beta and A-delta fibers. These large fibers may also be damaged in pudendal neuropathy as noted by abnormal sensory evoked potentials in a man with penile hypesthesia (34).

The size of the thermal probe affects test results (35). A small thermode, as used in the present study (0.44 cm^2), permits measurements within the receptive field of each individual pudendal nerve branch. An oversized thermode can overlap into receptive fields of adjacent cutaneous sensory nerves. Placement of even a small thermode must avoid the anatomical midline because of overlapping contralateral sensory nerves (26). This overlap causes midline penile testing results to differ when compared to lateral measurements in the same men (Antolak 2005 poster presentation, unpublished). An overlap of approximately 0.5 cm can be demonstrated by pinprick examination following a unilateral pudendal nerve anesthetic block. (Fig. 3)

Testing across the midline results in erroneous conclusions. One article concluded that CPP was not a peripheral neuropathy because midline perineal testing showed similar results in both normal men and men with CPP (36). This spurious opinion occurred because an extremely large thermode (6 cm2) was placed in the midline of the perineum (see Fig. 1). The normal values determined by Lefaucher are not useful because a 2.56 cm3 thermode was used at the penile midline (37). In both articles, the lack of awareness of anatomical nerve overlap caused specious conclusions. This can also be stated about the sensitivity and specificity of Bleustein and colleague's (22) results. One can only speculate about their damaging effect on research efforts.

Any practitioner or clinic can perform the tests described in our methods section. The WDT equipment in simple to use but requires an assistant. In Europe, the Physitemp Thermosensory Tester is not available. Beco et al (38) use a more sophisticated MSA Thermal Stimulator (Somedic AB, Horby, Sweden) to support the definite diagnosis of pudendal neuropathy.

Central sensitization and/or windup are evident when WDT evokes qualitative responses, i.e., paresthesias, dysesthesias, and warm allodynia. Dramatic somato-somatic reflexes via the sacral cord include paresthesias in the great toe (patient 1), leg (patient 3), or thigh (patient 12) during testing in the genitalia. Patients' sensations may occur on the side contralateral to the test sites. Somatovisceral reflexes include bladder or rectal urge at normal or abnormal temperatures during the WDT. Another indicator of neural plasticity is what we call "vermification" of the scrotum that occurs in some men during WDT testing (patients 5, 11, 20, and 24). This is a disorganized, rolling, convoluted movement of the dartos that appears to represent disorganized autonomic signaling. It may occur during testing at any of the pudendal branches. Central neuropathic symptoms can be treated initially with medications such as gabapentin, amitriptyline, baclofen, and clonidine.

Inflammatory prostatitis in 4 men in this cohort is considered a coincidental finding. They received 2 months of clindamycin 100 mg 3 times a day and follow-up testing of the prostate expressage. When antibiotic therapy eliminates the infection but pelvic pains persist, remission of pains after pudendal nerve blocks is also indicative of a neuropathic cause of their CPP. Leucocyte counts in expressed prostatic secretions do not correlate with symptoms of CPP (39).

Larger cohort studies are required and should include both neurophysiological testing and evaluation for inflammatory prostatitis in men with CPP. Results in the present study suggest that symptoms of "prostatitis-like" pains more likely represent pudendal neuropathy than inflammatory prostatitis, especially in the 2 men who had previous radical prostatectomy for cancer.

Monitoring treatment responses is important. We began the use of the NIH-CPSI in 1999, but it is not a diagnostic tool for CPP. It can demonstrate changes over short term (weeks, months) and long term (up to 12 years). Despite development of a newer version, we continue to use the NIH-CPSI for longitudinal comparisons with past evaluations. The total score at consultation reflects the severity of the CPP, e.g., the patient with a consultation score of 38 has a suicidal level of pain whereas the man with the consultation score of 10 had sexual dysfunction (not measured by NIH-CPSI), minimal pain, and his poor quality of life predominates. The average NIH-CPSI scores in our patients are consistently worse than those measured in primary care clinics and a university urology clinic (40).

It is essential to separate neurogenic from non-neurogenic CPP. Incorrect treatment of CPP as an inflammatory or morphological process is fraught with failures. Interventions have included many months or years of antibiotic therapy, multiple varicocele operations, inguinal hernia repairs, epididymectomy, unilateral or bilateral orchiectomy, prostate needle biopsies, and even radical prostatectomy for pain. The rapid and simple pinprick sensory test could shift the practitioner away from morphological causes of CPP to a neurological basis.

Treatment of a "definite" pudendal neuropathy can be successful using sequential processes (10,14,19,41). These begin with nerve protection and medications, progressing as necessary to pudendal nerve perineural injections of steroids and bupivacaine. Approximately 30% of patients require nerve decompression surgery. Failures of pain control often relate to presence of central sensitization and/or the "additional" neuropathic pelvic pain generators outlined in the Methods section. These must be treated concurrently.

The strengths of this report are: (1) it is evidence-based using pinprick testing and the WDT to provide a definite diagnosis of neuropathy; (2) it offers both clinicians and researchers a uniform, simple, practical means of diagnosing definite neurogenic pain and separating it from non-neurogenic pain in patients with CPP or suspected prostatitis; (3) the findings refute misleading statements found in the "Nantes criteria;" (4) the results suggest that adoption of a focused pudendal neurologic examination coupled with simple neurophysiologic tests might introduce a new era of evidence based research and change the paradigm of CPP resulting in "precision medicine" therapies.

The chief weaknesses of this report include: (1) it represents a private clinical practice to which all patients were referred (self or physician) because of possible pudendal neuropathy; (2) the cohort is not representative of a pain clinic or a standard urologic practice. This bias cannot be changed as we are committed to evaluate and treat "all-comers." Broad, population-based evaluations are needed to determine the incidence and prevalence of neuropathic pelvic pain; (3) another problem is that normative value testing of WDT was selected

from the medical literature although the same device was used. Our normal studies were limited to testing of normal spouses (Antolak, 2003–2004, unpublished). The numbers were too small for statistical analysis; (4) the conservative temperature used by fiat for the upper limits of normal excluded patients between 37°C and 39.5°C, who would be considered abnormal by data published by Bleustein (23). Finally, the NIH-CPSI is limited because it does not include questions regarding sexual function or bowel function.

This observational study and our 2006 publication underscore the importance of specialty research groups (pain, gynecology, colorectal surgery, urology, neuromodulation, National Institutes of Health) to develop uniform protocols for sensory and WDT testing in all institutional review board studies of CPP.

Conclusions

Pudendal neuropathy is typically a bilateral process in men with CPP. A definite diagnosis is possible using pinprick sensory testing in the pudendal territory and a QST called the WDT. Pinprick testing identified neuropathy in 92% of an unrecruited, consecutive patient cohort. The WDT test was abnormal in 100% of the men presenting with symptoms of pudendal neuropathy. Inflammatory prostatitis was not sought in 2 men with previous radical prostatectomy and was present in only 4 of the 15 men with pudendal neuropathy from whom secretions were obtained. Perineal pain caused by sitting was found in only 52% of the men and should not be considered a pathognomonic symptom of pudendal neuropathy, as suggested by the Nantes criteria.

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