PAIN OF UROGENITAL ORIGIN (PUGO)  
NEWSLETTER of the IASP Special Interest Group on Pain of Urogenital Origin

Executive Committee
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Florella Magora, MD,  
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Richard Berger, MD, Coordinator  

Web Page Task Force
Tony Buffington, MD, Coordinator  

Mailing Address for the SIG:  
SIG on PUGO  
Dr. A. Baranowski, PUGO Chair,  
University College London  
Pain Management Centre  
National Hospital for Neurology and Neurosurgery, Queen Square,  
London WC1N 3BG, United Kingdom  
Email: aparan@btinternet.com

Message from the Chairman

Medicine is exciting because it is always changing. This change is stimulation for further exploration and learning providing us with opportunities for improved patient care. The first major meeting that I organized focusing on urogenital pain was in 1997. To the meeting I was able to invite a number of friends and colleagues with a special interest in urogenital pain, it was a small group but did attract over a hundred participants. At the end of the day, the meeting produced more questions than answers. We were also aware that the world of urogenital pain was rapidly changing. In 1998 PUGO was officially recognized as a special interest group within the IASP and since then we have held three Clinical Scientific Meetings. The first was in Eilat, 1998 our inaugural meeting, we held our third Clinical Scientific Meeting this September just gone (the second being Hamburg, 2000). This third Clinical Scientific Meeting covered considerably more ground, but what was particularly thrilling was the significant increase in both basic science and evidenced-based medicine that was presented. Summaries of some of those presentations appear within this newsletter. There is still a long way to go and a lot of work to be done!

Recently there have been changes within your executive committee of PUGO. Sadly Dr. Richard Schmidt and Dr. Ragi Doggweiler have resigned from the committee. As a result and in accordance with SIG bylaws I have arrived as chairman one year early and Richard Berger has been co-opted as Newsletter editor. It is our intention to have elections for officers prior to our next annual general meeting (AGM), which will occur during the IASP World Congress in San Diego. Officers to be elected are the Chairman-Elect and the Secretary/Treasurer, and a "Call for Nominations" for this purpose is included with this Newsletter. These officers plus the Chairman comprise the Executive Committee, and the Executive Committee appoints the chairs of standing committees and other working bodies within the SIG. These appointments include a Research Coordinator, a Web page Coordinator, and a Newsletter Editor. Everyone has a job and responsibility. SIG members who are interested in serving on Committees are asked to submit their names on the enclosed nominations form.

How do I see the next four years progressing for PUGO? The titles of the standing committees have been chosen carefully with it being envisaged that each member will have important responsibilities. These should not be free ride committees. Over the next year I look forward to the members of the standing committees drawing up guidelines as to exactly what they feel are the core responsibilities of those positions and as to how they will be achieved. I am sure that we will all have opinions and perhaps that task may be easier for some positions than others. If you have strong views please feel free to contact us. We shall use the List Server to air our views.

Over the next year I would like to see the Web page being frequently updated. We all have a responsibility towards that, so please send your ideas to Tony Buffington (email buffington.1@osu.edu) who has the onerous task of coordinating the Web page. We will aim to produce two newsletters a year, one based on our Clinical Scientific Meeting and one based upon recent advances. Richard Berger (email: rberger@u.washing-ton.edu) has been co-opted to coordinate these. Again, please help him with your ideas.

I envisage that the Chairmen Elect will have many responsibilities preparing for their three years in office. The main challenge for them will be organizing the Clinical Scientific Meetings that will occur during their term. As Chairman I have been very lucky because Allan Gordon has volunteered to organize a meeting in Toronto in 2003 and Maurice Bensignor has agreed to organize the Clinical Scientific Meeting in France, in 2004. Both Allan and Maurice have been co-opted onto the Executive Committee.

Timely topics in pain research and treatment have been selected for publication, but the information provided and opinions expressed have not involved any verification of the findings, conclusions and opinions by the IASP or the SIG on Pain of Urogenital Origin. Thus the opinions expressed in this Newsletter do not necessarily reflect those of IASP, the SIG, or of the Officers and Councilors of either IASP or the SIG on Pain of Urogenital Origin. No responsibility is assumed by IASP or the SIG for any in-jury, and/or damage to persons or property as a matter of products liability, negligence or otherwise, or from any use or operation of any methods, products, instruction or ideas contained in the material herein. Because of the rapid advances in the medical sciences, the publisher recommends that independent verification of diagnoses and drug dosages should be made.

December 2001

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Committee.

Of all the responsibilities I see that of Research Coordinator as being the most difficult. The coordination of research is something that we will have to work on. I think that we ought to start with simple tasks, such as looking at data collection – but not collection for collections sake! Ursula Wesselmann (email pain@jhmi.edu) has a great brain but will need a lot of input into designing her job and in making plans for future collaborative research.

There is no doubt that in medicine collaboration is important. At our last Clinical Scientific Meeting we had representation from the International Pelvic Pain Society, the Sex and Gender SIG of IASP and the Vulval Pain Society. I would like to further these links and in the first instance would like to set up a registry of all those societies that have an interest in pain that affects the urogenital system. With this in mind I would be grateful for any web sites or e-mail address that you can give me. Please e-mail me at ap-baran@btinternet.com.

One of our great successes over the past year has been the List Server which Tony Buffington set up. I highly recommend that you sign up to it. You can do this by contacting Tony. He will check that you are a fully paid up member of PUGO and then place you on the list. It’s as easy as that and whenever you have a question you need an answer to you send it by e-mail to the server. Any of the members of the server who has an answer can get back to you via the list server and we are all educated by seeing the discussion as a series of e-mails!

We have a busy year ahead of us preparing for the future. Hopefully by this time next year we will see our efforts of the past two years consolidated with PUGO being firmly established and with well laid plans for how the society should develop and what the role of the executive committee members and standing committees should be in the future.

Andrew Baranowski, MB BS, FRCA, MD
Chair, SIG on Pain of Urogenital Origin

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Abstracts from the 3rd Annual Scientific Meeting on Urogenital Pain, September 2001

Sacral Neuromodulation
D. Andrich, National Hospital for Neurology and Neurosurgery, UCLH, London, United Kingdom

Pain in the genitourinary tract is poorly understood and remains a challenging urological problem. In the past, the cause for pelvic pain was thought to be infective in origin, but recent trends support the hypothesis, that pelvic pain represents a neural breakdown in the regulation of pelvic floor function [1].

As a result of this new approach to pelvic floor dysfunction, new therapies have emerged which are aimed at modulating the pelvic floor, such as biofeedback and sacral anterior root stimulation. Neuromodulation of sacral nerve roots is an established alternative approach for the treatment of various urinary storage and voiding dysfunctions [2] and [3]. The application of this treatment modality in pelvic pain syndromes is new, but a few studies show promising results [4], [5], [6] and [7]. This presentation will outline the operative technique of sacral neuromodulation and will highlight the advantages and common complications of this treatment option.

References

Development of a Program for Sequential Therapy of Urogenital Pain Attributed to Pudendal Neuropathy
Antolak SJ, Hough D and Pawlina W. Departments of Urology, Radiology and Anatomy, Mayo Clinic, Rochester, MN, USA

Development of an evaluation and treatment program for chronic pelvic pain has been an evolutionary process and remains a “work in progress”. Cooperation with the Departments of Anatomy and Radiology has been of paramount importance.

The Problem. Chronic pelvic pain has the health impact of acute myocardial infarction, unstable angina or acute ulcerative colitis. Over 7.7 million patient visits for “chronic prostatitis” occurred in the USA in 1996. It is estimated that 90% DID NOT have infection or inflammation. Most urologists consider pain in the urogenital/rectal area “chronic prostatitis”. The United States National Institute of Health established the International Prostatitis Collaborative Network (IPCN) which defines the "prostatitis-like" symptoms in the absence of infection or inflammation as Category IIIB chronic prostatitis/chronic pelvic pain syndrome. Robert et al and others define the same symptom complex as pudendal neuralgia, pudendal nerve entrapment or pudendal canal syn-
drome. We consider the symptom complex to be a compressive neuropathy which meets the criteria of chronic regional pain syndrome. There is pain, motor impairment (urethral sphincter, ischiocavernosus muscle) and autonomic dysfunction (irritable bladder, penile/scrotal retraction, abnormal sweating, inability to achieve erection) and skin changes manifested by peau d’ orange over the sacrum or buttocks in some patients.

_Evaluation._ After urological evaluation excludes reproductive tract infection, the physical examination highlights the sensory examination of the pudendal nerve distribution. An attempt is made to reproduce the subjective pain by compression of Alcock’s canal and the sacrospinous ligaments medial to the ischial tuberosity. These sites are the anatomical pathway of the pudendal nerve. Gluteal skin is examined for peau d’ orange. Neurophysiological testing is difficult but may include pudendal nerve terminal motor latency testing and EMG of the ischiocavernosus muscles. These tests are not commonly performed at our institution.

_Diagnosis._ Pudendal nerve entrapment is a clinical diagnosis: urogenital/rectal pain aggravated by sitting, reduced when standing, not present when recumbent and relieved by sitting on a toilet seat. The IPCN defines Category IIIB chronic prostatitis as lower genitourinary symptoms, particularly pain in the perineum or genitalia, voiding symptoms such as dysuria or frequency, and sexual dysfunction in the absence of uropathogens in the urine or prostatic secretions.

_Etiology._ The etiology of pudendal nerve entrapment appears to be compression and/or stretching of the pudendal nerve at the interligamentous space between the sacrospinous and sacrotuberous ligaments and in the pudendal (Alcock’s) canal. The symptoms in males and females are remarkably similar depending on which branch(es) of the nerve is involved and the degree and duration of the injury. The position of the ischial spine is frequently abnormal (surgical observation and/or Judet views of pelvis) and may be the common anatomical denominator for this syndrome. The variety of symptoms is explicable by the marked variation in the composition of the pudendal nerve during segmentation and segregation of the spinal cord and the peripheral nerves also the variable sites of nerve impingement and the variable microtrauma.

_Treatment._ Treatment is based upon the concept of relief of a compression neuropathy, analogous to carpal tunnel syndrome. Treatment is monitored with the NIH-CPSI.

1. Perineal hyperprotection or self-care.
   This consists of avoidance of hip flexion exercises, standing as much as possible, avoidance of cycling and using a sitting pad cut specially to "suspend" the perineum when sitting-a "perineal suspension pad". In the absence of previous medication therapy the patients are given ketorolac 10 mgm q6h for five days and amitriptyline increasing from 10mgm @hs to 50 mgm @hs at 10 days intervals.

2. Perineal corticosteroids injections.
   Three injections are given at two week intervals using a mixture of bupivacaine and dexamethasone. Effectiveness is evaluated by sensory examination of the pudendal distribution 1 to 2 hours after the injection. Cadaver studies including section of unembalmed hemipelvis after CT guided injection of dye and radiographic contrast into the interligamentous space and into Alcock’s canal confirm the accuracy of this procedure.

_Treatment._ When conservative treatment is not totally effective, neurolysis with sectioning of the offending ligaments and fasciotomy of the obturator fascia forming Alcock’s canal is performed by a neurosurgeon.

_Results._ Response to treatment varies from 42% to 72% depending upon treatment modality used and technique used to analyze the response. The use of the NIH-CPSI is valuable for monitoring patient response. Case reports illustrating positive response to each modality are presented.

_Pudendal Nerves and Chronic Pain in the Perineal, Rectal and Urogenital Areas._

Maurice Bensignor¹, Jean-Jacques Labat², Roger Robert³.
¹Anesthesiology, Clinique Viaud, 40 rue Fontaine de Barbin, 44000 Nantes, France; ²Neurology. Clinique urologique, Hotel Dieu, 44093 Nantes Cedex 1, France; ³Neurosurgery. Hotel Dieu, 44093 Nantes Cedex 1, France

Pain syndromes of the perineal, urogenital, and rectal areas are often poorly understood and recognised when no specific lesion can be identified. A psychological distress is often present but may rather be a consequence of chronic pain than a symptom of a characterised psychiatric disease. Besides the visceral, musculoskeletal and emotional components of these complex pain syndromes, an activation of autonomic neurons may result in an inappropriate peripheral adrenergic outflow and in a sensitisation of nociceptors and mecanoreceptors. A burning perineal pain extending to the vulva, vagina, testis, urethra, or to the anorectal region specially when unilateral, worsened in sitting position, except on the toilets and relieved standing up or lying down, thus not affecting sleep, evoke an involvement of pudendal nerves. The pain later tends to extend with time as a consequence of the associated muscle spasm, sensitisation and plastic changes in the central nervous system. Pudendal neuropathy is more frequent in women. It can be clinically suspected: neurological examination of the perineum is normal. Gentle pressure on the ischial spine, sacrospinous ligament or the lateral wall of the ischiorectal fossa reveals tenderness. Diagnostic anaesthetic blocks may give more informations. Electrophysiology may bring further data: electromyography of bulbospongious muscle and sphincter ani may show a spontaneous denervation activity. Pudendal nerve distal motor latency is obtained by intra rectal stimulation applied on the ischial spine and recording through a needle in the bulbospongious muscle. Anatomical and surgical studies suggested that many patients may present with pudendal nerve entrapment. Improvement may be obtained from therapeutic nerve blocks in the sacrospinous ligament or in the pudendal (Alcock’s) canal. In an open study on 178 patient who underwent 1 to 4 steroid blocks (mean 2.1) at six weeks intervals with 6 months follow up after the last block, 68% had good to excellent relief. Among 36 patients transiently improved by local anesthetic blocks who had no benefit from
steroid blocks, 11 have been referred for surgery, all of them were found to have severe nerve ligament entrapment. A few carefully selected patients may benefit from surgery. More than 300 patients had pudendal neurolysis and were assessed one year or more after surgery: 44% considered being completely or almost completely relieved, 23% considered they were substantially improved but still felt pain particularly in prolonged sitting position. 33% had no benefit from surgery. None was aggravated. A prospective randomised controlled study will end in October 2001.

These difficult problems have a better chance to be solved when a comprehensive treatment plan is worked out through a close multidisciplinary collaboration. Better understanding of physiopathological mechanisms should lead to improve specific treatment strategies.

Development of CPPS(Chronic Pelvic Pain Syndrome) Tender Point Score.
Richard E. Berger, Jay C Lee, Marcia Ciol, Ivan Rothman. Department of Urology and Rehabilitation Medicine, University of Washington, Seattle, WA, USA

Introduction: The American College of Rheumatology has described The Manual Tender Point Survey (MTPS) as a tool to diagnose and evaluate fibromyalgia. We have used and expanded this tool for use in CPPS.

Methods: We examined 59 men with CPPS and 80 control men in an IRB approved protocol. CPPS men had pelvic pain and no pathogens on 4-glass localization tests. Examiners standardized exams by learning to press 4kg pressure in a standardized fashion using a dolorimeter and periodically recalibrated their finger during the study. Each man was told to rate pain caused by pressure at each point for 1-10. Scores of pain were developed for control. FM points, external pelvic points, internal pelvic points, prostate points and scrotal points.

Results: Men with CPS had higher pain scores on FM (p<.01), external pelvic (p<.001), prostatic (p<.001) and scrotal pain scores (p<.001) than men without CPPS. There was no difference in control point pain scores or number of EPS leukocytes. CPPS men and controls had no difference in estimated prostate size or “bogginess.” EPS leukocytes count correlated inversely with amount of prostate tenderness in CPPS men but not in controls. NIH symptoms scores and EPS leukocyte counts were higher in CPPS men with higher tenderness scores.

Conclusions: Men with CPPS have more tenderness in non-pelvic areas than men without CPPS. Men with CPPS have more tenderness in external pelvic, internal pelvic, prostatic, and scrotal sites than men without CPPS. Tenderness in men with CPPS is not limited to the prostate but is present throughout the pelvic and in extra-pelvic sites raising the possibility that CPPS may be related to FM. Pain and tenderness in CPPS may be related to prostatic fluid inflammation.

Is Interstitial Cystitis in Cats a Naturally Occurring Model of Visceral Pain?
Tony Buffington, DVM, PhD, DACVN (buffington1@osu.edu) Professor of Veterinary Clinical Sciences, The Ohio State, University Veterinary Hospital, Columbus, OH, USA

Interstitial cystitis (IC) is a chronic pelvic/perineal pain syndrome of unknown etiology. The clinical features of IC include chronic, recurrent urinary frequency, urgency, and pain referable to the lower urinary tract. These symptoms often appear acutely, and generally follow a waxing and waning course. Epidemiological studies reveal that more than half of IC patients report daily or constant pain, which is exacerbated by stressful circumstances. At least two variants IC have been described. In the more common form, only glomerulations (submucosal petechial hemorrhages) are seen at cystoscopy. In the less common ulcer form, fissures and scars that crack and bleed when the bladder is distended are present.

Histopathology may be used to rule out other disorders, but findings in IC are inconsistent. In non-ulcer IC, scattered glomerulations, small mucosal tears, and submucosal hemorrhages with or no a mild inflammatory infiltrate can be seen, although abnormalities usually are limited to vasodilatation, and submucosal edema. In the classic form, chronic inflammation, fibrosis, dilatation of vessels with hemorrhage, neural proliferations and perineuritis may be identified in the suburothelium; these abnormalities occurred in only 3.9% of the 209 biopsy samples from the IC database study. Unfortunately, these histopathologic lesions are not unique to IC, and lesion severity does not appear to correspond well with the severity of clinical or cystoscopic findings. Moreover, the clinical symptoms of IC and glomerulations are not restricted to patients with IC.

Understanding of IC is further complicated by the observations that symptoms may remain even after removal of the bladder, and that bladder lesions can be present in patients reporting significant improvement in clinical signs. IC patients also have a variety of other problems. Epidemiological studies have reported that headaches, fibromyalgia, irritable bowel syndrome, cough, and tingling in fingers/toes can be more prevalent in IC patients than in age-matched controls, suggesting that the causes of IC may extend beyond the bladder.

These complexities, the absence of identified causes, and the lack of generally effective treatments make IC a particularly challenging disease to model. Investigators have approached this problem by attempting to re-create bladder-related features of IC in healthy animals using noxious intravesical, systemic, and environmental stimuli. These studies have provided a valuable description of the complexity of responses of normal bladder to a variety of insults. Their relevance to the etiology of IC, however, is less clear. The identified responses usually are not specific to the bladder, and when the stimulus is removed, healthy animals appear to return to normal.

A spontaneously occurring disease analogous to IC also occurs in domestic cats, referred to as Feline Interstitial Cystitis (FIC) to distinguish it from IC in humans. FIC primarily
resembles the non-ulcerative form of IC. The waxing and waning course of clinical symptoms and aggravation by environmental stressors, and the many abnormalities of local bladder factors and sensory, central, and sympathetic function that have been identified in humans also occur in cats with FIC.

Like the induced models, FIC also has limitations as a model of IC. One is the seemingly different gender distribution between affected males and females of the two species. In cats, both genders are affected roughly equally, whereas in humans, 90% of patients are women. One reason for the gender discrepancy may be related to differences in diagnoses rather than diseases. Men with IC symptoms are more likely to be diagnosed with non-bacterial prostatitis rather than with IC; if only half of the cases of non-bacterial prostatitis are the same disease as IC, the difference in gender distribution between humans and cats would dissolve.

Another significant limitation to using cats with FI to investigate etiologic mechanisms of IC is that affected animals are not easy to acquire without veterinarian and owner cooperation. This is particularly frustrating because some 4 million cats are destroyed annually in the United States for “elimination problems”, the majority of which are related to the urinary tract. Additionally, cats are more expensive to maintain in laboratory animal facilities than are rodents.

References:

Purinergic Signalling in Visceral Pain.
Geoffrey Burnstock. Autonomic Neuroscience Institute, Royal Free and University College Medical School, Rowland Hill Street, London NW3 2PF, United Kingdom

The rapidly expanding field of purinergic signalling will be reviewed, including a summary of the current classification, distribution and properties of the cloned receptor subtypes for purines and pyrimidines and examples of their physiological and pathophysiological roles described (see Burnstock, 1997).

A novel hypothesis will be explored for purinergic mechanosensory transduction where ATP released from epithelial cells lining tubes (including ureter, uterus and gut) and sacs (such as urinary bladder) during distension acts on P2X3 purinoceptors on subepithelial sensory nerve plexuses; this initiates impulses that are relayed via sensory ganglia and the spinal cord to pain centres in the brain (see Burnstock, 2001). Supporting evidence will be presented, including: release of ATP during distension of ureter and bladder; immunohistochemical localization of P2X3 receptors on subepithelial sensory nerve plexuses in ureter, bladder and intestine; impulse generation in sensory nerves during application of ATP and its significant reduction in P2X3 knockout mice. The possible mechanisms involved in cellular release of ATP in response to distension and the changes occurring in inflammatory conditions will be discussed.

References:

Chastetree Fruit for the Treatment of Premenstrual Complaints. Sigrun Chrubasik, Institut für Rechtsmedizin, Albertstr. 9, 79104 Freiburg, Germany

Medicinal use of chastetree fruit dates back to the Greek antique. The drug consists of fresh or dried fruits of Vitex agnus castus (VAC) containing not less than 0.1% casticin. For the treatment of premenstrual complaints, the Commission E monograph recommends aqueous-alcoholic extracts (50-70%) corresponding to 30-40 mg of the drug1 (with at least 30-40 μg casticin). Casticin may serve as a leading compound for the lipophilic active principle that includes labdane and clerodane type diterpenes. Presently it is still unknown if hydrophilic compounds (e.g. agnusid, aucubin) contribute to the therapeutic activity. Results of clinical studies with particular preparations cannot be transferred to other preparations. This is because the total amount of effective compounds is dependent on the origin of the plant, extraction and manufacturing procedures, and on the plant individual quantity and spectrum of effective compounds.

PMS is characterized by psychic and somatic symptoms (e.g. depressive mood, nervousness, irritability, anxiety, mastalgia, abdominal bloating, fatigue, headache) during the last 8 to 10 days prior to onset of menstrual bleeding. Clinical studies give evidence to the clinical effectiveness of VAC preparations (dose equivalent up to 240 mg crude drug) in the treatment of PMS2. The rationale of the effectiveness of VAC is its dopaminergic, prolactin-inhibiting mode of action3,4. In vitro studies using pituitary cell cultures and studies in rodents confirm the chastetree fruit effect on prolactin release. However, non-dopaminergic effects5 (mainly interaction with opioid receptor sites) may also be involved in the VAC effect mechanism and may correspond to the clinical efficacy, especially alleviation of psychic alterations and pain. The overall mechanism of the VAC action is, however, not yet fully elucidated. Serious adverse events have not occurred during treatment over several months2. In rare cases gastrointestinal disturbances (nausea), allergic skin reactions, headache, fatigue, and cycle disorders were observed.

There is general agreement that VAC preparations (as prolactin inhibitors) should not be used during pregnancy and lactation (due to inhibition of prolactin release). Concomitant use of dopamine receptor antagonists may decrease the effectiveness of VAC (likewise, a potentiation of co-administered dopaminergics may occur). In case of feeling of tension and swelling of breasts and at disturbances of menstruation, a physician should be consulted for diagnosis. Preclinical safety data have proven a very low toxic risk in
rodents during acute, subacute and chronic tests, and on gene toxicity, but full safety data still need to be obtained.

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6. Unpublished, property of Bionorica AG [BNO]

Sexual Abuse, Rape And Chronic Uro-Genital Pain.
Beverly J Collett, Pain Management Ctr, Leicester Royal Infirmary, Leicester, United Kingdom

Chronic urogenital pain in both men and women is a poorly understood condition. An increasing body of research has highlighted the increased incidence of childhood and adult sexual and physical abuse in women with chronic pelvic pain (1,2). In contrast, little research has been done on childhood sexual and physical abuse in men. A Medline search revealed no papers specifically looking at this area of research. It is therefore unresolved at present as to whether men with urogenital pain have a higher incidence of childhood sexual or physical abuse. The medical conditions that may arise in men who have been sexually abused as children are unknown.

The question arises as to whether it is abuse per se or whether children raised in households where either physical or sexual abuse can take place have been subjected to other influences which may predispose to chronic urogenital pain (3). Not all abuse victims go on to develop pelvic pain. What is the mechanism which leads to the development of medical problems, including pelvic pain? Can profiles of children be developed which will identify children who are at risk of developing pelvic pain.

Once sexual abuse has been identified, does it matter? What type of help is needed for these women and more pertinently does it help?

References

How to Work Up a Patient from the Urogenital Perspective.
Clare J Fowler, National Hospital for Neurology and Neurosurgery, UCLH, London, United Kingdom

A patient with uro-genital pain often poses a difficult clinical problem. First it is important for the patient to have been thoroughly examined by a urologist or gynaecologist and local pelvic pathology to have been excluded. Once a structural cause has been eliminated a neurologist opinion is often sought and again the prime aim of the neurologist is to exclude any form of conus or sacral root pathology. Magnetic resonance imaging is the investigation of choice to examine both the neural tissue and surrounding structures.

Clinical neurological examination of the perineum is of limited value and neurophysiological investigations are rarely contributory either. Although it has been claimed that measurement of the pudendal terminal motor latency is valuable in identifying patients with pudendal nerve entrapment [1] this is an abnormality that is an exceptional finding in practice. Other pelvic floor clinical neurophysiological investigations are more helpful in identifying changes of denervation and reinnervation than investigating pain syndromes. A major defect of currently available clinical neurophysiological investigations is that they examine mostly large myelinated nerve fibre function rather than the unmyelinated and small myelinated fibres that subserve autonomic innervation, pelvic organ sensation and pain.

Unfortunately there are many patients with perineal or pelvic organ pain in whom investigations cannot demonstrate an abnormality. The rich sensory nerve supply of the region and its complex interaction with the autonomic innervation may well lead to the development of neurogenic pain syndromes which have yet to be defined.

Reference:

Is Clitoral Pain a Model of Neuropathic Pain?
Allan S Gordon MD, FRCP(C), Neurologist and Director, Wasser Pain Management Ctr and Head of the Chronic Pelvic Region Pain Unit, Mount Sinai Hospital, Toronto ON, Canada

Genital pain syndromes such as vulvar vestibulitis (VVS) or painful urethral syndromes are increasingly recognized as the cause of significant pain and disability in women. Most of these women do not have clitoral pain as a prime feature.

Indeed clitoral pain should be uncommon in most cases of VVS, as VVS usually involves the inferior part of the introitus although one might expect it with vulvodynia. Clitoral pain is not a thing a woman would easily talk about. Most physicians, even urologists and gynecologists, would not ask about it in routine or even specific questioning. Little data exists about the prevalence of clitoral pain.

Yet persisting, severe, and often unexplained clitoral pain does occur. It is briefly mentioned in Wall and Melzack's
Textbook of Pain (1) where it states that very few reports have been published. A PubMed search showed few entries and also was not that helpful in discovering articles about constant clitoral pain. It is not mentioned in Chronic Pelvic Pain, An Integrated Approach (2). At the 1999 Santa Fe Meeting of the ISSVD (International Society for the Study of Vulvovaginal Disorders), President Marilyne McKay, made a platform plea for more study of clitoral pain.

The author, a pain neurologist with an evolving interest in Chronic Pelvic Region Pain, recently presented a series of more than 10 women with what he called the PNPC syndrome (Painful Nipples Painful Clitoris) at the Pain Of Urogenital Origin (PUGO) meeting in Hamburg in September 2000 (3). More than half of the women were volunteer/collaborators derived from various online patient lists of which the author has or had been a member. Some of the patients reported upon here were presented in poster form at the Female Sexual Function Forum in Boston in October 2000 (4) and will be published in the proceedings of that symposium. Paul Perry has written a description of Clitoral Pain which appears on the International Pelvic Pain Society website (http://www.pelvicpain.org) under "resources".

This paper reports 15 patients from the author's practice and personally evaluated and more than 20 women who contacted the author through the Internet. Most were seen to have or reported to the author that they had clitoral pain at rest clitoral pain both at rest and with touch or pressure. The pain was usually quite severe. Some looked upon it as part of an overall genital pain syndrome. But most were very specific that it was the clitoris that hurt and everything else was secondary.

When the author's own patients were examined very few had clitoral pain only. Often there was other genital sensitivity just as perirectal or vulvar sensitivity and allodynia. Cases were associated with pudendal neuralgia, multiple sclerosis, Guillain Barre syndrome, direct and indirect trauma, and vulvar vestibulitis or vulvodynia, and conditions such as fibromyalgia. Therapy so far has been relatively unsuccessful but 3/15 improved on their own.

The online volunteers suffered from a number of conditions including Lichen Sclerosis (5), direct trauma, vestibulitis or vulvodynia. In several the cause was not known. Several reported improvement but most reported that treatments were unhelpful.

Most reported frustration with lack of diagnosis and/or indifferent care and were so troubled by their condition that they were anxious to find treatments and would be willing to participate in research programs.

Severe pain at rest, allodynia and hyperalgesia, lack of obvious local pathology (other than LS), would all suggest that clitoral pain is primarily neuropathic and should be approached that way. Cases of a slipped vibrator, an errant glass bottle, vigorous masturbation, pelvic surgery, and the presence of neurological disease would all support that contention as would associated vestibulitis considered by some to be neuropathic.

These patients, although there are relatively few of them would be a good 'model' for testing hypotheses, agents or treatments for effectiveness in neuropathic pain. Once the paradigm shift is made by practitioners who normally do not think that they are treating neuropathic pain, this opens up new approaches to management.

Thus well-documented clitoral pain should be the subject of organized clinical trials in neuropathic pain, both pharmacological and non pharmaco logical trials. There is a need to look both at the pain issues and the serious quality of life issues associated. Management needs to be both multidisciplinary and multidimensional. A database of cases should be established.

The author will present some of the cases and a more detailed evaluation of the characteristics of the groups. This is a descriptive paper but it is hoped that it will serve as an impetus for the development of a multicenter research protocol.

References:

Chronic Pelvic Pain in Women and Rehabilitation
Claudine Hattab-Blum, Physiotherapist, M.Ph. Specialist in Uro-Gynecological Rehabilitation 6 Hagdud Haivri St., 92345 Jerusalem, Israel. jocelyn@vms.huji.ac.il

Chronic Pelvic Pain (CPP) is a common syndrome affecting many women of all ages, races, and backgrounds. Its study is often disconcerting, owing to: the great variety of symptoms described by the patients; the difficulty in classifying those symptoms; the depressive state of many patients (due to their pain and personality)

CPP frequent manifestations are urological, anorectal and sexual dysfunctions. CPP is often characterised by a burning sensation and vulvar irritation which cause local muscular contractions. This in turn provoke a dorsal neural root stimulation thus inducing a vicious circle of primary and secondary pain, as well as antalgic compression focused on trigger-points. This pain is nociceptive and results in a negative sexual circle manifested by contraction of the pelvic musculature, anatomical constriction of vaginal introitus, inflammation, vaginitis, anxiety, corporal memory of the locus of irritation and dyspareunia.

In our uro-genital rehabilitation clinic many patients are referred with this type of nociceptive pain, and a diagnosis of vulvodynia /vestibulodynia. New bio-techinics emerged recently for treating this highly incapacitating disease through a holistic approach. More than any other region of the body, the pelvic area is delicate borderline between the psyche and the soma, and it is difficult to specify exactly where each begins and ends.

The first step is having a physician treat the known etiologies or symptoms (infections, inflammations, and mechanical irritations) Decision is made regarding referral either to techniques of general physical therapy, or focusing on
the reeducation of the sphincters and perineal musculature. The latter method involves a wide assortment of techniques such as computerised bio-feedback using surface EMG, perineal exercises, massage, stimulation, relaxation, behavioral modifications and other procedures.

After holistic treatment, during the last three years, of over a hundred women suffering from vulvo/vestibulodynia, our results show increase in muscle tone in 95% of the patients, increased muscular relaxation in 68%, and balance in muscular stability in 62% of the patients. But, the most important result is that 845 of the patients regained a normal life, including satisfactory sexual experience, with 16% feeling only some improvement. The proposed treatment is a multi-faceted approach, conducted by a multidisciplinary team, and takes into account the bio-psycho-social aspects of sexual pathologies.

**Gender Differences in Pain.**

Anita Holdcroft, MB ChB, Reader in Anesthesia, Magill Dept. of Anaesthesia, Chelsea and Westminster Hospital, 369, Fulham Road, London SW10 9NH, United Kingdom. Email: aholdcro@ic.ac.uk

Sex differences in responses to pain result from a variety of factors such as genetic, physiological, anatomical, neural, hormonal, lifestyle and cultural. Biological effects can be explored in animals but clinically it is recognised that while women are more vulnerable to pain than men, a broader-based pain management program can be activated for them. Recognition of gender differences in pain may improve diagnosis as well as outcome following analgesic drugs, physical and behavioral therapies.

What is the clinical evidence for gender differences? Epidemiological studies, often based on health insurance assessments, report that women experience pains of higher severity, more frequently, in more sites and of longer duration than men [1]. The prevalence of painful diseases is higher in women [2] and age is a covariant [3]. More importantly the clinical presentation of visceral pain, such as coronary artery ischaemia, differs between the sexes. Even in healthy individuals experimentally induced pain demonstrates gender differences [4] and the configuration of the sexual organs and their hormonal milieu [5] are also factors influencing pain sensitivity.

What should be the general approach to sex differences in patients presenting with pain and how do these relate to urogenital disorders?

The wide variety in acute pain responses to a clearly defined noxious stimulus can partly be explained by gender differences at a number of levels. The maintenance of a chronic pain state should similarly be considered in terms of gender effects. For example, women vary in their responses to a predetermined experimental pain during their childbearing years depending on the phase of their menstrual cycle whereas men do not vary. In the long term pain may be modulated by hormonal manipulations such as oral contraceptives to treat dysmenorrhea. When analgesics are used to manage pain any gender differences in pharmacokinetics or pharmacodynamics should be identified both for pain relief and also side effects.

In the application of gender differences to pain of urogenital origin the general effects of sex differences may apply to the attitude of the clinician, the clinical setting and the willingness of the patient to use supportive situational or physical therapies. Adverse events to procedures are more common in women and possibly the choice of pain therapies should reflect this. Within the process of pain assessment changes in hormone concentrations, either as a result of disease or therapies, and their effects on pain symptoms should be elicited. Urogenital disorders may be sex-specific but basic principles of pain management may be defined based on gender differences.

**References:**

**References:**

**Barriers and attitudes of doctors managing urogenital pain.**

R William Stones MD FRCOG. Senior Lecturer in Obstetrics and Gynaecology, Univ. of Southampton. rws1@soton.ac.uk

Women report negative experiences of consulting doctors for the complaint of chronic pelvic pain. Key questions addressed in this paper are: What do doctors think about women with pelvic pain? Can “medical attitudes” to women with pelvic pain be characterized? Does the medical consultation influence outcome?

**Implications for clinical practice:** Clinicians need to be aware of the importance of the initial medical consultation as a factor influencing patients’ outcome from investigation and treatment. While consulting styles reflect the individual personality of the doctor, we need to be aware of our own underlying attitudes and how these might enter into the dynamics of the consultation. Women presenting with pelvic pain in whom no clear diagnosis is present, or where diagnoses overlap, need to be given clear explanations which do not undermine the legitimacy of their experience of pain or convey a message of dismissal. The setting in which consultations for pelvic pain take place need adequately to reflect the referral pattern: patients with long standing or disabling symptoms require extended consultation time and access to other advice and treatment resources, as in a multidisciplinary model.

**Implications for research:** Further studies are required of the relationship between consulting style and patient outcomes, perhaps using observational techniques such as video recording of consultations. Research is needed to clarify the importance of elements in the patient’s experience such as continuity of care and the contribution of different members of a multidisciplinary team.

**References:**
Chronic pelvic pain: A difficult clinical problem, the development of an animal model.

Ursula Wesselmann MD, Associate Professor of Neurology and Biomedical Engineering, Johns Hopkins Hospital, Department of Neurology, 720 Rutland Avenue, Trayor Bld 604, Baltimore MD 21205, USA. Tel: (410) 614-8840; Fax: (410) 955-9826; e-mail: pain@jhmi.edu

Chronic pelvic pain in women presents a very difficult clinical problem because existing treatment strategies are often unsuccessful. Estimated medical costs for outpatient visits for chronic pelvic pain in the United States are $881.5 million/year. Overall, a women has about a 5 percent risk of having chronic pelvic pain in her lifetime. In patients with a previous diagnosis of pelvic inflammatory disease this risk is increased fourfold.

Pelvic pain belongs to the category of visceral pain. Although persistent pain of visceral origin is a much greater clinical problem than that from skin, the overwhelming focus of experimental work on pain mechanisms relates to cutaneous sensations. The neurophysiological mechanisms underlying visceral pain are poorly understood. In the past 15 years several different animal models have been developed to study the behavioral manifestations, the neurophysiology and neuropharmacology of somatic pain. The greatest contribution of these animal models may lie in their use to study the effects of traditional analgesic therapies and to develop new analgesic therapies, rationally targeted upon the pathophysiological mechanism. In contrast, very few animal models have been developed to study visceral pain. The reason for this is that, similar to the clinical situation, the manifestations of visceral pain in animal models are more difficult to describe and to quantify than somatic pain. Until relatively recently, it was often assumed that concepts derived from cutaneous studies could be transferred to the visceral domain. However, there is experimental evidence demonstrating that the neural mechanisms involved in pain and hyperalgesia of the skin are different from the mechanisms involved in painful sensations from the viscera.

To gain further insight into the mechanisms and pain pathways mediating chronic pelvic pain in women, we have recently developed a new model in the rat that resembles closely a state of inflammatory uterine pain (Wesselmann et al., Neurosci. Lett., 246, 1998). Rats with uterine inflammation show characteristic movements indicative of visceral pain: hunching, hump-backed position, licking of the lower abdomen, repeated waves of contraction of the ipsilateral oblique musculature /inward turning of the hindlimb, stretching of the body, squashing of the lower abdomen. For quantitative analysis of pain behavior a scoring system of abnormal movements has been designed. The majority of visceral pain episodes occur on days 2 to 4 post inflammation, then the rats return to normal behavior. In addition to spontaneous pain behavior, we have observed muscle hypersensitivity over the flanks and lower back, indicating referred visceral pain. Ovariectomy results in a significant reduction of abnormal behavioral characteristics on days 2-4 post inflammation, suggesting that the development of uterine pain in this rat model is influenced by the gonadal hormonal milieu. This is in agreement with the clinical observation that acute pelvic inflammatory disease and chronic non-malignant pelvic pain typically occur in women during their reproductive ages.

In conclusion, we have designed a model of visceral pain in the rat which resembles closely a state of inflammatory uterine pain. This model will allow to gain further insight into the neural processes, which contribute to pelvic pain syndromes, and will allow to study the effects of interventions for the treatment of pelvic pain.

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The role of biofeedback in urogenital pain.

Kimberly Martin, Clinical Research Fellow, Pain Management Centre, National Hospital for Neurology and Neurosurgery, Queen Square, London, United Kingdom.

Some pelvic pain conditions produce similar symptoms to chronic abacterial prostatitis, or prostatodynia. These observations led to the recent reclassification of prostatodynia as Chronic Pelvic Pain Syndrome. Biofeedback is frequently suggested as a therapeutic option for prostatodynia patients for whom other methods fail, although there as yet no randomised controlled trials to support this. Recently, the National Institutes of Health identified biofeedback as a high priority for prospective evaluation. Within this context, two distinct approaches have been described: urodynamic biofeedback, which is a behavior modification strategy designed to re-educate patients with voiding dysfunctions; and biofeedback-directed pelvic floor muscle re-education. It is the latter of these two which forms the focus for this discussion.

The anatomy of the pelvic floor musculature will be described. From the sixteenth century to the present day, opinion has been divided over the existence of a levator prostate. Recent evidence suggests that different regions of the pelvic floor muscle display differences in the relative sizes and distribution of fibre types and in the proportions of muscle spindles.

The scientific rationale behind the use of pelvic floor re-education is discussed in some detail. The putative link between prostatodynia and its female counterpart, vulvodynia, is traced. There is electromyographic evidence that vulvodynia may be secondary to a hyperactive pelvic floor and that fast twitch (type II) fibres may be responsible. It has been proposed that biofeedback stabilises these fibres. With the combined support of the Prostate Research Campaign and the St Peter’s Trust, the University College London Hospitals Pain Management Centre is currently under-
taking a pilot study to develop protocols for the use of biofeedback-assisted pelvic floor muscle training and electrical muscle stimulation in patients diagnosed with Chronic Pelvic Pain Syndrome. The methods and results of this study are presented. After ten weeks, five out of eight patients (63%) showed greater than 25 percent reduction in total NIH Chronic Prostatitis Symptom Index. Improvements were also seen in short McGill Pain and VAS scores. Concurrent changes in surface EMG allude to the possibility that these methods induce physiological alterations in the pelvic floor muscle.

The methods developed in this study will hopefully for use in a randomised controlled trial in order to establish a formal evidence base for this type of treatment. Although this is a limited cohort, these data are sufficient to support the conduct of such a trial.

Meetings Calendar

August 17-22, 2002, San Diego, California, USA: 10th World Congress on Pain
International Association for the Study of Pain (IASP). Info: IASP, 909 NE 43rd St, Suite 306, Seattle, WA 98105, USA, Tel 206-547-6409; Fax 206-547-1703; Email iaspdesk@juno.com; Web: www.iasp-pain.org

Tuesday, August 20, 2002, 16:30-17:30: SIG business meeting at the 10th World Congress on Pain, San Diego, USA.

May 2003 Toronto, Ontario, Canada: PUGO Scientific Meeting.
Contact: Allan Gordon. Email: allan.gordon@toronto.ca

Contact: Maurice Bensignor. Fax: 33-2-4037-2616

Membership in IASP SIGs is open only to members of IASP. The cost of joining a SIG is US$20.00. Members wishing to join a SIG should indicate their preference on the annual membership renewal form or contact the IASP main office: IASP Secretariat, 909 NE 43rd St., Suite 306, Seattle, WA., 98105-6020 USA. Fax 206-547-1703. Email: iaspdesk@juno.com

The SIG on Pain of Urogenital Origin (PUGO) currently has 111 members in 23 countries representing 19 specialties

See Enclosed "Call for Nominations"

Please return this form by March 1, 2002 to:
Ms. Kathy Havers
IASP Secretariat
909 NE 43rd Ave, Suite 306
Seattle, WA 98105 USA

Fax: 206-547-1703
email: iaspadmin@juno.com

Correspondence to the Newsletter Editor
R. Berger. Newsletter Editor.
University of Washington Medical Center
Department of Urology, Box 356510
1959 NE Pacific Street, Seattle, WA 98195, USA
Tel: 206-543-3270; Fax: 206-543-3272
email: rberger@u.washington.edu
Call for Nominations

Members of PUGO are invited to submit nominations for the next SIG election which will elect a Chairman-Elect and a Secretary/Treasurer. **Please use this form to submit your nominations for these offices.** A list of all current SIG members is printed on the reverse side of this nominations form.

The election will be completed prior to the 10th World Congress on Pain (August 17-22, 2002, San Diego, CA, USA) via a mail ballot to all current members of the SIG, and results announced at the SIG business meeting that will be held on Tuesday, August 20, 2002. The current Chairman-Elect, Dr. Andrew Baranowski, who is currently serving as Acting Chairman, will take office at that time.

*Any member of the SIG may stand for election for these offices*

**CHAIRMAN-ELECT:**

________________________________________

**SECRETARY/TREASURER:**

________________________________________

The Chairman, Chairman-Elect, and Secretary/Treasurer comprise the Executive Committee which also appoints Committees and working parties within the SIG.

If you are interested in serving on any SIG Committees or are willing to chair a committee, please volunteer your name in any of the spaces below:

**Web Page Coordinator:**

________________________________________

**Committee on Research:**

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**Committee on Meetings:**

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**Committee on Finance:**

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**Committee on Newsletter:**

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IASP Secretariat
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