SPS NEWS

The Official Publication of The Southern Pain Society

Fall 2002

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Editor's Desk

Maureen J. Simmonds, PT, PhD Editor

Conferences and Cruises

I've just returned from San Diego where I - and more than 5,000 others - attended the Triennial Congress of the International Association for the Study of Pain. The meeting was well organized, well attended and the content was superb. As with any conference, it was wonderful to renew personal and professional friendships and meet many new, interesting and interested people from around the world. I enjoy international conferences because I lived for many years in three different countries, and I've visited many others – including third world and emerging countries (I was born and trained as a physiotherapist in England, worked as a clinician and did my graduate work (PhD Rehabilitation Science) in Canada, and am now on Faculty at a university in the USA). In that sense, I feel I can relate to some of the issues and understand some of the concerns associated with studying and managing pain problems in some different countries. Although the details may differ, many of the major issues and trends in the study and management of pain are similar across countries. However, as a physical therapist with a special research interest in pain and movement I was most pleased to see that research and clinical management approaches are integrating sensory *and* motor neural networks in pain states. Movement is essential to life, and for people with pain, movement is compromised. Whether movement compromise is a result of, or part of, the pain experience remains to be determined. Perhaps we will be closer to addressing such questions at the next pain conference. And of course the next major conference is the SPS Conference, which promises to be an enriching experience. The theme of the SPS conference is "What we have learned in the past ten years about pain." I look forward to cruising the SPS annual conference with you.

Newsletter Submissions

All submissions to SPS News should be typewritten and double spaced with title and name of author(s). The article should be copy-ready. Please include biographical information.

Submission Deadlines

Winter edition-November 1; Spring edition-February 1; Summer edition-May 1; Fall edition-August 1.

President's Column

Peter Staats, MD

This is my last President's address for the Southern Pain Society. When I became President two years ago, I had big shoes to fill taking over from Bert Ray. I am extremely fortunate that Bert has remained a great friend and colleague and has continued to provide the Society with his guidance and help. When I agreed to accept the President's position, I had several goals for our Society. I wanted us to expand our member base, provide education on all levels--from practitioners to consumers and lobbyists, and either improve our administrative structure or make the decision to hire an independent Executive Director, all while maintaining our financial viability.

I am pleased to report that this year we have achieved most, if not all, of our goals. We have expanded our financial base, and during the past two years we have managed to keep our expenses down. Our annual meetings have been tremendously successful. We are now in the best financial position ever enjoyed by the Southern Pain Society. We have educated hundreds of primary care physicians, pain specialists, and allied health providers at our past two meetings, while generating a profit for the society.

During the past several years, we have seen a growth in the awareness of pain medicine as a specialty, and our advice has been sought by Congress and by health leaders from around the world. This growth has not been without challenges, however. While we have been busy trying to educate consumers about the appropriate use of opiates, for example, the media has been full of stories of criminals posing as patients to illegally obtain drugs. This media attention could have backfired on the specialty of pain medicine; however, we faced these problems proactively. When the problem of OxyContin diversion arose, we raised a collective voice urging that our elected officials adopt a balanced and well-considered response. We have also worked Drug Enforcement Agency to help solve the problem of under-treatment of pain and addiction.

This year, we started a new program offering seven small educational conferences around the country on a variety of topics relevant for pain. Working collaboratively with Mike Ainsworth of Synergy and under the direction of Shashidhar Kori, we began this new program in various locations in the Southeast. These conferences were supported by an unrestricted educational grant from Elan Pharmaceuticals. They succeeded in providing in-depth information about pain topics ranging from fibromyalgia to high-end interventional options to hundreds of primary care specialists. This year's Southern Pain Society Annual Meeting on the cruise ship Sovereign of the Seas, also chaired by Dr. Shashidhar Kori, promises to be one of our most successful meetings ever.

We have expanded our member base and even added a new Puerto Rican chapter of the Southern Pain Society. Under the direction of Drs. Albert Ray and Victor Mojica the 2003 meeting will be held in Puerto Rico.

We have also made an important change by moving our administrative structure to the direction of Lori Postal, our diligent and hard-working, new Executive Director.

Finally, I am extremely pleased that Jeannie Koestler will be assuming the Presidency of the Southern Pain Society. Jeannie has long been a tremendous advocate of the Society, and she has a most able leader of the Mississippi Chapter (our largest). The next two years will undoubtedly bring her challenges that none of us can foresee, and she will need our help to continue the educational mission of our Society. The Southern Pain Society will undoubtedly benefit from Jeannie's experience, knowledge, and leadership ability, and I am confident that we are in the best hands possible.

Thank you all for your confidence in me and your support. It has been a great two years.

Science

Daniel M. Doleys, PhD   Assistant Editor

Current Concepts of Chronic Pelvic Pain in Women

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Editor's Note: This submission is Part 2 of a 2 part scientific article. Part 1 appeared in the Summer issue.

Urinary System Causes of Chronic Pelvic Pain

The urinary tract is a common site for inflammatory pathology, stones and other conditions, which can activate visceral nerves and cause CPP. Since visceral pain is diffuse and may be poorly localized, a history of voiding abnormalities will offer the best sign of origin. The most common triggers for CPP originating from the urinary system are: 1) hypersensitivity due to recurrent, chronic urinary tract infections, 2) interstitial cystitis and 3) urolithiasis.

Recurrent infection with hypersensitivity

The urothelium is ordinarily bathed in a constant flow of sterile urine. Uropathogens such as bacteria, fungus and viruses are capable of infecting this tissue and causing inflammation and pain. If the tissue injury is repetitive or chronically present, the dorsal horn neurons of the spinal cord become sensitized. They then can become activated by even normal stimuli (e.g., bladder filling). Visceral nerve hypersensitivity produces pelvic muscle spasm, soft tissue trigger points and skin hyperesthesia through the convergence-projection phenomenon. These changes may persist long after the initial insult[11].

Although initiated by infection, there may be no persistent infection after the cascade of events is set in motion. Neuropathic pain and myofascial pain are common. Women describe a "constant burning" in their urethra or bladder. Some patients complain of intermittent "shock-like" pains. Pelvic floor spasm may produce postcoital aching or a sense of vaginal fullness. Stranguria is experienced by patients with vesical-sphincter dyssynergia. Dehydration, anxiety, stress, depression and diet have all been implicated in symptom exacerbation.

The diagnosis of hypersensitive lower urinary tract depends on the history of voiding dysfunction, absence of a positive culture and absence of cystoscopic evidence of interstitial cystitis. The physical exam consists of a one-finger palpation of the lower urinary tract starting at the distal urethra proceeding proximally. The trigone and each ureteral insertion should be evaluated separately. Urodynamics, uroflowmetry and pelvic floor electromyography can help define difficult voiding dysfunction such as vesicle-sphincter dyssynergia.

Prevention of recurrent urinary tract infections is the best way to interdict hypersensitivity of the lower urinary tract and CPP. Asymptomatic bacteriuria and cystitis should be promptly treated with appropriate antimicrobials. Relief of dysuria with phenazopyridine hydrochloride is recommended. Frequency and nocturia can be helped by Imipramine pamoate 100mg at bedtime. Tolterodine 2mg twice a day will usually be helpful. When bladder capacity is reduced by chronic low volume voiding, bladder diaries and retraining is beneficial. Stranguria is treated by diazepam 2.5 to 10mg every 6 hours. In difficult cases of vesicle-sphincter dyssynergia, Terazosin may be started at 1mg at bedtime and slowly increasing the dose while avoiding hypotension.

Interstitial Cystitis

Some consider interstitial cystitis (IC) the end stage of lower urinary tract syndrome. Others feel the clinical findings are distinct enough to qualify this condition as a separate disease. No infectious agents are found associated with IC. In fact, cultures are consistently negative. Urinalysis may show a few red blood cells. Bladder capacities are progressively reduced. The urethra becomes extremely tender and catheterizations are very painful.

Cystoscopic examinations usually require general anesthesia due to the patient's pain. Throughout the bladder, surface blood vessels are attenuated. After hydrodistension, multiple bleeding points are noted. Occasionally, an ulceration (Hunner's ulcer) will be present.

The pathogenesis of IC has long been believed to be an autoimmune phenomenon. Increased mast cells in the bladder mucosa in many of these patients may be evidence of this or mast cells may be a protective mechanism of the host. It is clear, for whatever reason, the urothelium of the bladder is "leaky". Urea and other noxious agents in the urine gain access to the nociceptors of the submucosa to produce pain and spasm. The preponderance of evidence now implicates a neuro-inflammatory process resulting from direct bladder insult or antidromic visceral pain[12].

The typical patient with IC has frequency, urgency, nocturia, constant suprapubic pelvic pain and dyspareunia. The dyspareunia may be the result of bladder tenderness, pelvic floor myalgia or vestibulitis. Some patients have all three components. Normal sexual activity is almost impossible. Nocturia is sometimes severe, often occurring more than 3 times each night. Ninety per cent of the patients will have a bladder capacity of less than 350mL and a bladder capacity during general anesthesia of less than 850mL.

Treatment of the CPP from IC involves both empirical treatments such as hydrodistensions and diet therapy. Other therapy includes bladder instillations and oral therapy. Dimethylsulfoxide (DMSO) or sulfate polysaccharide heparin placed and held in the bladder will offer some patients substantial relief. Pentosan polysulfate sodium given orally is effective to decrease symptoms in about one third of patients.

Surgical approaches to the patient failing to respond to conservative therapy include: 1) cystoplasty, bladder augmentation, 2) bladder denervation procedures and 3) cystectomy. None of these offers women assurance that their CPP will resolve.

Urolithiasis

The predominant quality of pain from urolithiasis is colic. This severe visceral pain usually leads to immediate medical intervention for analgesia and relief of obstruction. Recurrent ureteral colic can produce prolonged hyperalgesia and referred pain. Depending on the intensity and frequency of ureteral colic, musculoskeletal and dermatome-specific referred pain can present as undiagnosed CPP.

The patients with a history of recurrent lumbar pain, flank pain and shooting pain into the genitofemoral distribution may have this type of neuropathic CPP. The prevention of this type CPP depends on early analgesia for ureteral colic. The dietary therapy for preventing the specific type stones formed should be instituted. Integral to urolithiasis prevention is increased water intake. Treatment consists of those medications previously discussed to be efficacious for neuropathic pain.

Bowel as an Etiology of Chronic Pelvic Pain

Chronic pelvic pain may be produced by a number of conditions affecting the bowel. Pathology can be divided into extrinsic and intrinsic diseases. Extrinsic conditions include intermittent bowel obstruction and endometriosis. Intrinsic conditions include dysmotility disorders and inflammatory changes.

Extrinsic Disorders

Intermittent, partial bowel obstruction can be a cause of CPP. The pain can be diffuse and perceived to originate low in the pelvis. Adhesive bands from previous surgery are the most common etiology. Pelvic tumors can also produce this luminal compromise. Ovarian cancer may present in this fashion. Endometriosis will involve the bowel in up to 37% of cases. The terminal ileum seems to be a common site of this condition with resultant menstrual exacerbations of symptoms. Cramping, sharp pain often accompanied by nausea and vomiting is the most common presentation. The abdomen may be slightly distended during episodes of partial obstruction. High-pitched bowel sounds may be heard during episodes of pain. The diagnosis can be confirmed with a flat and upright abdominal X-ray. The treatment is surgical relief of the luminal compromise by adhesiolysis or partial bowel resection[13].

Intrinsic Disorders

Symptoms of intermittent bowel obstruction can originate with disorders of bowel motility (dysmotility). The most common type is irritable bowel syndrome. Although it is thought to be a disturbance of the large bowel, the small bowel function may also be abnormal This condition affects women of all ages, but is especially common in the younger women experiencing school or career stresses. It may often be a component of complex pelvic pain syndromes such as endometriosis, vestibulitis and pelvic floor myalgia. There are two distinct patterns: Constipation dominant and diarrhea dominant. The small bowel dysmotility is most often seen in the patient complaining of multiple loose stools per day (diarrhea dominant). These patients may have sharp abdominal and pelvic pain with bloating. The abdominal exam will usually reveal normal bowel sounds and no point tenderness. The constipation dominant dysmotility is manifested by infrequent, hard stool. These patients complain of intermittent, sharp lower abdominal-pelvic pain. There may be some discomfort to pressure over the sigmoid colon of cecum. Hard stool is often palpable on abdominal exam. Endoscopy and imaging studies will be nonspecific.

Inflammatory conditions of the bowel are among the most frequently seen causes of CPP from the bowel. Early Chron's disease and ulcerative colitis will often present as pelvic pain of undetermined etiology. These conditions may be perplexing before the onset of bloody diarrhea. Endoscopic and radiographic studies will usually yield the correct diagnosis.

The presence of CPP in the older female may be from diverticular disease. It is thought to occur in one third of the population over 45 and two thirds of the population over 85. Since the colon and sigmorectum share the same innervation as the cervix, uterus and adnexa, it may be difficult to determine if lower abdominal-pelvic pain is gynecologic or enterocolic in origin. Diagnosis may be even more difficult with the presence of a pelvic mass called a phlegmon. This can be made up of colon, diverticular abcess, bladder wall and pelvic sidewall. The patient usually complains of cramping, lower abdominal-pelvic pain. Fever, nausea and dyschezia may be present. Endoscopy and barium enemas should not be done in the actively inflamed patient for fear of perforation. CT scan is usually diagnostic. Oral antibiotics may be sufficient conservative therapy, but for recurrent bouts in the younger patients, surgical resection may become necessary[14].

Musculoskeletal Causes of Chronic Pelvic Pain

The pelvic musculoskeletal frame is a common source of CPP, but often goes unrecognized and untreated. This can be a primary pathology source or can be a secondary response to true visceral pain. Most commonly, patients with CPP have a combination of both. Usually, the visceral pain initiates the muscular spasm, which in turn causes joint dysfunction and skeletal changes. The mechanism for this sequence

of events resides in the inter-neuronal communications of the spinal cord. The visceral nociceptors cause reflex activation of the motoneurons, which produces muscle contraction and activation of muscle nociceptors[2]. Muscles sharing dermatome innervation with the pelvic viscera are thus susceptible to this spasm-pain reaction. These patients complain of activity specific pelvic pain. For example, patients with piriformis muscle spasms will have exacerbations when climbing stairs and patients with pubococcygeus spasms will experience dyspareunia or pain when sitting. The diagnosis of these conditions is made by careful palpation of the muscles looking for spasm and tenderness. Physical therapy is essential if progress is to be made in pain reduction. Trigger point injections may be necessary. Only temporary improvement will be realized if the site of visceral nociception is not remedied (e.g., endometriosis).

Uterine Fibroids

Uterine leiomyoma are the most common tumors of the female pelvis occurring in one in every four of five women. The size of myomas depends on difference in vascular supply, proximity to adjacent tumors, degenerative changes and hormonal growth factors. These tumors may be submucous, intramural, subserosal or pedunculated. While most fibroids are asymptomatic, 10-40% may produce menorrhagia, infertility or pain.

Chronic pelvic pain has been attributed to uterine fibroids more frequently than justified. Special conditions such as acute red degeneration during pregnancy and transcervical prolapse of a submucous fibroid ("aborting fibroid") are notable exceptions. There is evidence that CPP is more likely produced by associated pathology (endometriosis, adhesions, etc.) than the uterine fibroid. Impingement of leiomyomata on surrounding structures can cause a variety of symptoms. Collision dyspareunia, rectal pressure and pelvic discomfort are likely with a fixed retroverted fibroid[15].

Hernias

Pelvic and abdominal hernias are an often overlooked as primary generators of chronic pelvic pain. The most common hernia in the female is the indirect inguinal hernia. They may produce the same symptom complex as a direct or femoral hernia. These patients often complain of pain in the lower pelvis and groin with some radiation into the anterior thigh or labia majora. The next most common hernias are ventral hernias, which are usually produced by previous surgery and will cause pain in the area of the anterior fascial defects.

More uncommon hernias include: sciatic (pain in the buttocks and back of the leg), obturator (pain in the inner aspect of the thigh and behind the knee or hip) and Spigelian (pain just lateral to the rectus abdominis). These defects are not easily diagnosed in women on physical exam. Laparoscopy is a great aid in their detection and most can be repaired via minimally invasive surgery at the time of diagnosis[16].

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Commentary on "Current Concepts in Pelvic Pain in Women by C. Paul Perry, M.D."

by Daniel M. Doleys, Ph.D.

Dr. Perry's article has provided us with a valuable insight into the multiple causes of chronic pelvic pain (CPP). Indeed, the complexity of the anatomy encourages pain syndromes of mixed types, i.e. neuropathic, somatic and visceral. The contributions of endometriosis, ovarian cysts, adhesions, bladder, and bowel can be difficult to differentiate because of convergence, projections and referral patterns. Although only briefly alluded to, the need for expert consultation from a pain oriented gynecologist seems self evident.

The impossible part of writing a brief synopsis of pathophysiological considerations is being able to convey the degree of suffering experienced by these, all too frequently, young women. Many of them have had pain as a significant part of their lives since the onset of puberty. This omnipresent pain can rob these young teenagers of normal psychosexual and developmentally critical experiences. It takes very little imagination to appreciate the emotional impact on the patient and family. The all too often cavalier approach and/or condescending attitude such as dismissing the problem as a "pelvic migraine" and relating it to some assumed history of sexual abuse or fear of sexual intimacy, is unwarranted and demeaning.

One recent study by Pukall, et al (2002) focused on women with vulvar vestibulitis syndrome. Although the authors did identify significantly more catastrophizing thoughts related to intercourse pain as compared to control subjects, no difference was found regarding non-intercourse related pain. In addition physiological studies revealed systemic hypersensitivity to tactile and pain stimuli. This finding led the authors to discuss the possibility of a "generalized disorder of sensory modulation". Studies of this type put and help to keep both psychological and physiological factors in perspective.

Caution must be exercised about viewing patients with chronic "pelvic" pain as potentially qualitatively and/or quantitatively different from patients experiencing pain in some other part of their body. Woolf, et al (1998) have suggested a "mechanism-based" approach to pain classification. This approach has more recently been echoed by Mantyh (2002) as regards cancer pain. Under this system assessment and treatment strategies would be based upon identified pain mechanisms such as primary afferent versus CNS mediated pain as opposed to relying on anatomical descriptions. This approach would not necessarily de-emphasize the relevant psycho/social or behavioral issues, but would help to insure appropriate attention to the relevant pathophysiology.

The contribution of past childhood abuse and victimization continues to be of interest and is receiving considerable scrutiny. Whether or not such histories are more prevalent in CPP patients versus irritable bowel syndrome or low back pain is unclear. The hypothesized mechanism(s) by which the history manifests itself have included changes in hippocampal volume, cortical sensitization, neural hormonal changes and/or the development of psychiatric comorbidities. Additionally, the role of such histories as mediators, modulators, or maintainers of CPP also remains undetermined.

We must take pause and learn from our colleagues involved in the treatment of spine pain. The over emphasis on spinal structures as "pain" versus "nociceptive" generators has contributed to increased frequency of spinal surgeries and invasive procedures frequently of limited benefit to the patient. To be sure, there are lesions which require surgical correction. However, surgery, in this context, should not be confused with, though can oftentimes be part of, "pain therapy".

Finally, though not as frequent a topic as pelvic pain in women, males can suffer pain in the region of the pelvis as well. However, it seems as though we are more inclined to consider the offending structures, i.e. bowel, penis, abdomen, bladder rather than the general location, i.e. pelvis. Clearly, men are not immune to childhood abuses. It would be of interest to examine possible differences in type, that is sexual versus physical abuse, between men and women. Schofferman, et al (1992) has suggested that the number of different types of trauma, which he refers to as risk factors, to be important in predicting changes in pain in response to spinal surgery. The role of perception may also be relevant. That is, males may be more inclined to discount certain types of abuse, such as physical, as being "just part of growing up" or a regrettable but acceptable feature of a parents personality.

Dr. Perry's article should be a stimulus toward greater understanding and exploration into chronic pelvic pain. Like many other pain types such as low back, headache, fibromyalgia it appears multifactorial and best addressed within a multidisciplinary model.

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Announcements

Change of Address

If you change your mailing or email address, please notify SPS by contacting Lori H. Postal, RNC, MS, Executive Director at the following SPS address:

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Web Site Update

Our web site is currently in revision and is off line. We will have a new and exciting look within the next month.

District News

Angela J. Koestler, PhD

SPS Districts

Greater Atlanta
Charles MacNeill, M.D.
(770)419-9902, ext. 219

Dade County
Mary Alice Yoham, MSN, ARNP
(305)324-4455, ext. 4276

The Miami-Dade County Pain Society wants you for a member, Board Memeber and active participant in Educational Progarams. Please contact President MaryAlice Yoham, MSN ARNP (myoham@med.Miami,edu) or Secretary Treasurer Albert Zbik, PsyD. (Miami Pain and Integrative Medicine Center, 305.595-4681). Watch for announcements for upcoming meetings via flyers. All Welcome.

Mississippi
Eric Pearson, M.D.
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Cruise the Bahamas with the Southern Pain Society and participate in the Annual Scientific Meeting aboard the Sovereign of the Seas!

Southern Pain Society
Annual Scientific Meeting
Agenda
October 17-20, 2002

Thursday October 17th	**Sail 5PM**	
1:00 – 1:15 pm	Welcome and Introductions	Shashidhar Kori, MD
1:15 – 2:00 pm	New Advances in Acute Pain Management	B. Todd Sitzman, MD
2:00 – 2:45 pm	Psychological Assessment of Pain	Angela J. Koestler, PhD
2:45 – 3:00 pm	Break	
3:00 – 3:45 pm	Fibromyalgia and Myofascial Syndromes	Albert Ray, MD
3:45 – 4:30 pm	Economics of Chronic Pain Interventions	Benjamin W. Johnson, Jr. MD
4:30 – 5:15 pm	Recent Advances in the Pathophysiology and Management of Headaches	Maria Carmen Wilson, MD
7:30 – 8:15 pm	Cocktail Reception	
Friday October 18th	**Arrive Nassau 11 am**	
8:00 – 8:45 am	Issues in the Use of Opioids with Chronic Pain Patients	Daniel M. Doleys, PhD
8:45 – 9:30 am	An Update on Complex Regional Pain Syndrome and Other Neuropathic Pains	Stephen Bruehl, PhD
9:30 – 9:45 am	Break	
9:45 – 10:30 am	Rational Polypharmacy with Adjuvant Analgesics	Shashidhar Kori, MD
10:30 – 11:15 am	No Mystery: If It's Physical, It's Therapy	Maureen Simmonds, PT, PhD
	Sail at midnight	
Saturday October 19th	**Arrive Coco Bay 8 am**	
8:00 – 8:45 am	Will Opioids Drive You Crazy? The Effect on Taking Them on Cognition And Driving Skill	Stanley L. Chapman, PhD
8:45 – 9:30 am	Epidural and Other Steroid Injections	John Satterthwaite, MD
9:30 – 9:45 am	Break	
9:45 – 10:30 am	High End Interventional Therapies	B. Todd Sitzman, MD
10:30 – 11:15 am	Alternative Medicine Therapies… No Longer Alternative	Mary Alice Yoham, MSN, ARNP
11:15 - 11:30 am	Course Summary and Closing Comments	Shashidhar Kori, MD
	Sail 5 PM	
Sunday October 20th	**Dock at 8 am**	

It's not too late to register! Call 1-800-422-0711 and join us for a great meeting and lots of fun!

Southern Pain Society

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Cary, North Carolina 27512

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