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VISION

THE INTERNATIONAL PELVIC PAIN SOCIETY

Professionals engaged in pain management for women.

GYNECOLOGICAL PAIN IN THE CLINIC:

Is There a Link with the Basic Research?

By Andrea J. Rapkin, M.D.

The basic research of visceral pain mechanisms does not explain the chronic pelvic pain phenomena a gynecologist confronts in the clinic. There is, however, a link between the basic research and the acute gynecologic pain processes observed in the emergency room. Acute pain refers to pain that is brief, spanning hours to days, whereas chronic pelvic pain has been defined as pain of greater than six month's duration.

Acute Pain

Early studies performed under local anesthesia have shown that crushing, or burning the bowel, for example, evokes no pain, whereas distention of hollow viscera, sudden stretching of the capsule of solid organs, hypoxia or necrosis of viscera, production of algesic (pain-producing) substances, rapid compression of ligaments or vessels, and inflammation may cause severe pain. All of the acute gynecologic pain processes are actually manifestations of these noxious stimuli: labor and dysmenorrhea are associated with abnormal distention or contraction of muscular walls of hollow viscera. Hemorrhagic corpus luteum cysts can result in rapid stretch of the capsule of a solid organ, in this case the ovary. Degeneration of leiomyomata (smooth muscle uterine tumors) leads to hypoxia of visceral muscle. Intraperitoneal hemorrhage or leakage of an irritating fluid from a cyst (such as a ruptured teratoma) or ruptured tubal pregnancy are examples of situations in which there is an accumulation of pain-producing substances in the peritoneal cavity. Endometritis and pelvic inflammatory disease are acute inflammatory states initiated by infection. Torsion of an adnexa causes compression of ligaments and vessels, and hypoxia of involved tissues. Pathology associated with acute pelvic pain involves mediators of inflammation present in high concentration and possibly entailing spatial summation.

Variability of Pain Sensitivity

Numerous pathologic conditions result in severe anatomic distortion but are not associated with symptoms of pelvic pain. Carcinoma (ovarian, uterine) is rarely associated with pain. However, with nerve invasion, this pain is no longer "visceral", but neuropathic, and is painful.

The sensitivity to stimulation of the reproductive organs in the normal, as well as the acute and chronic pain states is of interest; however, there is no recent comparative information in humans. Theobald performed hysterectomies under local anesthesia in a group of asymptomatic women. The vagina and cervix were fairly insensitive to incision or pressure. In the abdominal cavity, the extraperitoneal tissues (somatic tissues) were exquisitely sensitive to incision. The parietal peritoneum, intestines, body of uterus, and ovaries were all fairly insensitive to manipulation including incision, pinching, and pressure. Injection of saline under the ovarian capsule caused a dull ache. The fallopian tubes, particularly the uterine end, were extremely sensitive. Without inflammation, infection, traction on, or lesion of neurovascular bundles, the pelvic organs with the exception of the fallopian tubes were all relatively insensitive. However, "rough handling" led to hypersensitivity of every structure and general anesthesia was necessary to proceed with the surgery. Sensitization was thus important for alteration of the threshold for response or development of mechanosensitivity.

Chronic Pelvic Pain

Pelvic pain research has focused on structures distal to the spinal cord with exhaustive search for somatic and visceral pathology. Up to 80% of patients with low back pain, myofascial pain, irritable bowel syndrome, or chronic pelvic pain lack physical signs or tissue distortion that correlate with pain. The high frequency of failure in attempts to treat peripheral causes also suggests that we must consider central mechanisms.

(continued next page)

The President's Perspective

Fred M. Howard, M.D., President

As our new organization moves into a time of transition, it is appropriate that we reflect on accomplishments and evaluate our goals. In a short year-and-a-half, the IPPS has moved from an idea to a reality, from a challenge to an achievement. The IPPS is now a strong and maturing multidisciplinary professional society dedicated to pain management for women. We should all thank one another for this accomplishment, but especially we should thank Dr. C. Paul Perry, who guided us through our challenging first one-and-one-half years. I am personally eternally grateful to Paul for his leadership, ethics, compassion, dedication, guidance, and friendship. Paul will remain in a leadership position in the IPPS as Chairman of the Board, ensuring continuity as well as his continued active participation in the Society.

Although we have accomplished much thus far, three projects initiated, but not completed, will be my goals for us during my tenure as president. First, we initiated, under Dr. Deborah Metzger's guidance, the development of standardized history and physical examination forms, for use in caring for women with chronic pelvic pain. We will endeavor to complete them this year and have them available by spring of 1999 for practitioner's use.

Directed by Dr. Michael Wenof, we have worked on educational information for our patients. Our goal should be to complete sufficient segments of this project to have information available by spring of 1999, also.

Finally, one of our dreams for the IPPS has been that it would lead to multidisciplinary research that might improve our knowledge and care for women with pain. I hope we will be able to initiate one or two studies within the next twelve months. Multidisciplinary research has the potential to be the IPPS's most significant contribution.

I am really looking forward to my chance to serve as President of the IPPS — please do not hesitate to let me know how I can serve you and the IPPS best. See you in San Diego!

Fred M. Howard, M.D.
President

Patients and Families

The IPPS needs the support of patients and families. If you would like to support further research into chronic pelvic pain, and receive our newsletters regarding recent advances and treatment, please consider joining! Dues are $35.00 annually.

Gynecological Pain (cont. from front)

The Search for an Underlying Pathology

Adhesions

Laparoscopic studies of women with chronic pelvic pain have revealed adhesions and endometriosis to be the main visible pathologies. Sixteen to 48% of women undergoing laparoscopy for pain have adhesions. However, women with tubal infertility often have a high incidence of pelvic adhesions. When a group of asymptomatic infertility patients with adhesions was compared with a group of women with pelvic pain and adhesions, the incidence, location, and density of adhesions did not differ between groups.

If adhesions were truly a frequent cause of pain, lysis of adhesions would be expected to relieve pain. The only published randomized prospective study of adhesiolyis by Peters et al. Involved 48 women, 24 of whom has lysis of extensive adhesions. At the time of evaluation 12 months later, there were no differences in pain scores. A subgroup of patients with dense vascular adhesions involving bowel did have a reduction in pain. A prospective noncontrolled study was performed by Steege in which all patients has lysis of adhesions. The postoperative pain ratings did not differ significantly from the preoperative ratings. However, in women without psychosocial factors, there was significant improvement in postoperative rating. This study highlights the importance of psychological or other central factors in maintaining pain.

Endometriosis

Endometriosis can be demonstrated in 15–40% of patients undergoing laparoscopy for chronic pelvic pain. Endometriosis produces a low-grade inflammatory reaction. However, there is no correlation between the location of disease and pain symptoms and no relationship between the incidence or severity of pain and the site or stage of endometriotic

Gynecological Pain (cont. from opposite)

lesion. Prostaglandin (PG) production from explants of petechial lesions was significantly greater than from the explants of powder burn or black lesions, which are more common in patients with higher-stage endometriosis. PG production may account for severe pain in some patients with mild disease.

Minimal Association Between Organic Disease and Pain

The degree of organic disease is thus minimally related to the level of pain and functional impairment. A substantial proportion of patients with pain may have sources of pain outside the reproductive organs. Abdominal wall and pelvic floor muscle trigger points, nerve entrapment in surgical scars, irritable bowel syndrome, and interstitial cystitis represent the most common sources of nonreproductive system chronic pain. Myofascial pain, irritable bowel syndrome, urgency frequency syndrome, and interstitial cystitis probably all entail alterations of central processing.

Pelvic Congestion

The syndrome of pelvic congestion was delineated in the 1940's (Taylor, 1949). Women with pelvic congestion were of reproductive age and complained of lower abdominal pain that was often lateralizing, a sharp pain with sudden movement, secondary dysmenorrhea, dyspareunia, postcoital ache, urgency and frequency of urination, nausea, diarrhea, constipation, bloating, premenstrual exacerbation of all symptoms, and increased pain with fatigue, stress, and activity. A large percentage of these women had undergone prior operations. Taylor hypothesized that the pelvic pain in these women was related to a disorder of the autonomic nervous system involving both afferent and efferent pathways. The problem affected all end organs of the visceral autonomics and occurred in patients with a predisposition to psychiatric symptoms. Congestion of the veins draining the ovaries and uterus in the pelvic congestion syndrome was supported by Beard's transuterine venogram studies.

Central Modulation of Pelvic Pain

In 1984, Slocumb noticed that patients suffering from pelvic pain with symptoms identical to those described by Taylor had on examination, the same pain produced by pressure over localized points in different tissues (trigger points). Tissues innervated by the same thoracic segment exhibited increased sensitivity (dermatomic area hypersensitivity). Injection of these trigger points with 2–3 cc of 0.25% bupivacaine relieved pain. These findings, though obtained in an uncontrolled study, raise the consideration of central modulation of pelvic pain.

Primary dysmenorrhea may be a better model in which to explore the role of central pain modulation, although dysmenorrhea is not truly a chronic, persistent pain state. Painful contractions in women with dysmenorrhea are associated with intense contractile myometrial activities secondary to excess prostaglandins that give rise to hypoxic conditions. Either the mechanical pressure itself or the chemical sequelae of hypoxia could activate uterine afferent fibers that would then convey information to be processed in the central nervous system (CNS) as pain. Various lines of evidence point to central modulation in addition to peripheral factors in the pain of primary dysmenorrhea: (1) Travell has stated that chronic dysmenorrhea can produce abdominal wall trigger points; (2) Electrical stimulation of the abdominal wall in the region of the T12 and L1 dermatome in women who have had dysmenorrhea produces pain akin to dysmenorrhea; (3) prolonged relief can often be obtained by injection of local anesthetics into hyperalgesic tissues; (4) accompanying the pelvic pain, there may be an alteration in reflex responses leading to associated bloating, diarrhea, constipation, and urgency and frequency of urination; (5) exacerbation of pain associated with stress, anxiety, and depression is more likely to develop in patients with previous adverse experiences such as physical or sexual abuse.

The cause of the observed dermatomic hypersensitivity of diverse tissues and altered reflex responses in many women with chronic pelvic pain is uncertain. The hyperalgesia and persistent changes in autonomic reflex activity seen in many chronic pelvic pain patients could be related to alterations in central excitatory or inhibitory phenomena.

Giamberardino's studies of patients with ureteral lithiasis suggest that at least some of the plastic changes occurring after acute inflammation may persist. Years after passage of a kidney stone, residual alteration in the electrical response properties of the overlying ipsilateral subcutaneous tissue and muscle was commonly observed. Gebhart and Mayer have independently shown that patients with IBS have lower colonic pain threshold and enlarged, atypical areas of pain referral with colonic balloon distension. The cause of this hypersensitivity is unknown.

Directions for Future Research

Unanswered questions regarding chronic pelvic pain include the following: What is the stimulus for alteration in central processing in women with chronic pelvic pain? Can long-lasting alterations in descending inhibitory or excitatory systems lead to sensitization? What is the effect of estrogen or cyclic hormonal changes on the development or maintenance of the chronic pain state? ▲

IPPS Calendar

Spring Meeting

April 30–May 1, 1999
Simsbury Inn
Hartford, Connecticutt
Details coming soon!

The International Pelvic Pain Society

Name: _____ Title: _____ Specialty: _____

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Annual Dues for Health Care Providers (based on income) and Patients

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Phone your request to (205) 877-2950, or Fax it to (205) 877-2973
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