The frustrations of chronic pelvic pain

To the Editors:

I would like to address a number of issues raised by Dr. Slocumb in his paper on chronic pelvic pain (Slocumb JC. Neurological factors in chronic pelvic pain: trigger points and the abdominal pelvic pain syndrome. AM J OBSTET GYNECOL 1984;149:536). The subject is most important because there is a large number of women whose extensive complaints cannot be diagnosed precisely and whose treatment remains unsatisfactory.

I agree with Dr. Slocumb that conditions such as functional ovarian cysts, pelvic adhesions and congestion, the "universal joint syndrome" are, as causes of chronic pelvic pain, often merely figments of our frustration. I believe that the complaints may be psychosomatic, but this hypothesis is difficult to prove.[1] Certainly our patients generally detest the suggestion that their suffering is due to anything other than physical disease. Apart from reassurance and support, mainstream gynecology has little to offer in the care of these patients.

If Dr. Slocumb's success can be duplicated, patients and gynecologists will be indebted to him. However, success cannot be evaluated from a mixture of telephone calls, letters, and office visits. A great many treatments of this refractory condition have not stood the test of time. There may be a very substantial placebo effect from the care and treatments provided by Dr. Slocumb. Above all, Dr. Slocumb used trigger-point injections as well as supportive therapy and medications for insomnia, depression, and/or anxiety in his management. Which of these helped the most? Outcome should be asesssed from carefully designed protocols and instruments, not from a mixture of treatments applied in undescribed proportions.

Questions should be asked about the pathologic conditions identified in the dorsal horns. A physical disease is postulated in an effort to explain the pain, but emotional factors are remarked upon, and thus psychosomatics are again invoked. Is there solid evidence of dorsal horn malfunction apart from subjective phenomena such as pain and hypersensitivity? Is it possible that higher centers produce whatever disease is identified in the dorsal horns? Has Dr. Slocumb given his patients another face-saving but scientifically suspect diagnosis?

A number of gynecologists have noted that chronic pelvic pain patients frequently present with multiple nongynecological symptoms such as headaches, lethargy, irritability, and depressive symptoms.[1-4] While these observations have been uncontrolled, more recent controlled studies have shown psychopathologic conditions in patients with chronic pelvic pain but no demonstrable disease.[5,6]

Much work remains to be done. Meanwhile, I hope that gynecologists will continue to perform laparoscopies instead of producing diagnoses unsupported by inspection of the internal genitalia. Dr. Slocumb's success with chronic pelvic pain patients appears to be impressive. If his methods can be duplicated, we should proceed to investigate the mechanisms involved.

Ivan K. Strausz, M.D., F.A.C.O.G.
St. Luke's/Roosevelt Hospital Center
Amsterdam Avenue at 114th Street
New York, New York 10025

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Reply to Strausz

To the Editors:

I would like to thank Dr. Strausz for his questions and comments concerning the paper on abdominal-pelvic pain. As pointed out by Dr. Strausz, current therapy for pelvic pain is unsatisfactory and the wide range of theories and the degree of operative intervention with little improvement is of concern. It was for these reasons that I felt it necessary to reassess my gynecologic skills and began incorporating neurological concepts in the evaluation of abdominal and pelvic pain.

The consistent finding of locally tender tissues in the abdominal wall, para cervix, dorsal sacrum, and levator muscles all reproducing the same chronic pain sensations strongly implicates abnormal neurological thresholds in both visceral somatic and peripheral sensory pain fibers of the T12 and S2-4 dermatomes. The association of onset with physical trauma such as that after an operation, rape, delivery, and intrauterine contraceptive device infections, etc., is commonly followed with persistence of the same pain sensations for years after resolution of the traumatic process.

The techniques of reproducing pain with (1) focal pressure (single finger, cotton tip, and/or needle tip), (2) examination of the abdominal wall by tensing the rectus muscles, and (3) blocking of tender tissues with a local anesthetic to document extended pain relief be-

yond the duration of action of the local anesthetic are all necessary in assessing neurological pelvic pain syndromes.

In any valuable therapy based on clinical criteria, the first step in molding the clinical approach is to gain experience under many conditions and to describe the associations of possible causes, factors, therapies, and observed outcomes in a large population.

The purpose of this paper on chronic pelvic pain thus is the description of techniques in diagnoses of what appears to be a neurologically mediated pelvic pain and the response of local infiltration. In the growing field of pain therapy these observations are consistent with those observed in other chronic pain conditions such as phantom limb, myofascitis, deafferentation syndromes, and other posttrauma chronic pain conditions.[1, 2]

Dr. Strausz questions whether the placebo effect can explain the reported successful response and to what extent "supportive therapy and medication" affected the outcome. Most of the patients seen in the pain clinic had received therapy elsewhere for the pain, 31 had hysterectomy for pain, and 61 had laparoscopy with the same pain returning after therapy (unpublished observations). In the study population all received local infiltration, 10% hysterectomy, 14% narcotics primarily for postinjection or rebound pain, 6% antidepressants, all for <1 month duration, etc. Supportive therapy then was infrequently used and only for short intervals in patients with complex clinical findings.

In a pilot study for our present double-blind saline/lidocaine/bupivacaine study, needling and operating room saline injection had no effect on pain response in six patients.

The association of psychological findings of depression, insomnia, lethargy, hypochondriases, and hysterical traits with chronic pelvic pain could as easily be a result of chronic pain as it could be a cause of chronic pain.[3] These findings are not seen in all pain patients, and when treated, the condition involves more the reaction to pain than the pain itself. These same psychological functions have been observed in other painful disease states including pelvic pain caused by pathologic diseases (endometriosis).[4]

The absence of macroscopic disease in dysmenorrhea does not exclude what we now understand is a complex neurotransmitter process of end-organ ischemia, altered thresholds with prostaglandin, vasopressin, and substance p release.[5, 6] The ability of autologous transfusions to reproduce symptoms of severe dysmenorrhea even after hysterectomy should raise serious questions about unproved but much quoted theories that primary dysmenorrhea is a result of the patient's "rejection of her role as a woman."[7]

The association of stress and emotional factors in patients with pelvic pain should not be ignored, however, because the descending control (endorphins) of the dorsal horn function in modulating threshold levels is well documented. Depression and anxiety lower the threshold, and anxiety and depression can be a result of neurochemical alterations in central nervous system function resulting from chronic pain. Psychological events resulting in stress would have the same effect of lowering thresholds but would not have to be the cause of pain in a patient with a presensitized pelvic threshold dysfunction. A Minnesota Multiphasic Personality Inventory evaluation of pain patients and control subjects with long-term follow-up is in process.

Trigger-point infiltration is not a cure but an observed clinical response to alterations in pain threshold. Much needs to be done to understand the role of pelvic pain and alterations in peripheral thresholds, including a controlled double-blind study. Let us begin by questioning these theories that have not explained the condition and by expanding our tools of evaluation.

I welcome your questions because each of us assesses pain through eyes biased by our past and honed by our experiences. Would your questions, however, be the same if you modified your examination techniques?

John C. Slocomb, M.D., S.M.H.
Department of Obstetrics and Gynecology
The University of New Mexico School of Medicine
2211 Lomas Boulevard, Northeast
Albuquerque, New Mexico 87106

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False phosphatidylglycerol with Helena Fetal Tek 200 thin-layer chromatography method for fetal lung maturity

To the Editors:

We read with interest the communication by Barnes et al. (AM J OBSTET GYNECOL 1984;148:347) reporting occurrences of respiratory distress of the newborn in cases where phosphatidylglycerol had been detected in the amniotic fluids. The method of analysis was the Helena Fetal Tek 200 system, which was developed by Touchstone et al.[1] We also have utilized this procedure in routine analysis and quantified the effects of blood contamination with it.[2]