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for the pain, 31 had hysterectomy for pain, and 61 had laparoscopy with the same pain returning after therapy (unpublished observations).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

REFERENCES

1. Bonica JJ. Neurophysiological and pathological aspects of acute and chronic pain. Arch Surg 1977;112:750.
2. Melzack R. Myofascial triggerpoint relation to acupuncture and mechanisms of pain. Arch Phys Med Rehabil 1982; 62:114.
3. Travell J, Bigelow NH. Role of somatic trigger areas with patterns of hysteria. Psychosom Med 1947;9:353.
4. Renaer M, Vertommen H, Nijs P, Wagemaus L, Van Hemelrijck T. Psychological aspects of chronic pelvic pain. AM J OBSTET GYNECOL 1979;134:75.
5. Yaksh TC, Hammond DL. Peripheral and central substrates involved in the rostrad transmission of nociceptive information. Pain 1982;13:1.
6. Owman C, Alm P, Sjoberg N. Pelvic autonomic ganglia: structure, transmitters, function, and steroid influence. Elfvin LG, ed. Autonomic ganglia. New York: Wiley, 1983:126.
7. Irwin J, Morse E, Riddick D. Dysmenorrhea induced by autologous transfusion. Obstet Gynecol 1981;58:286.

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]

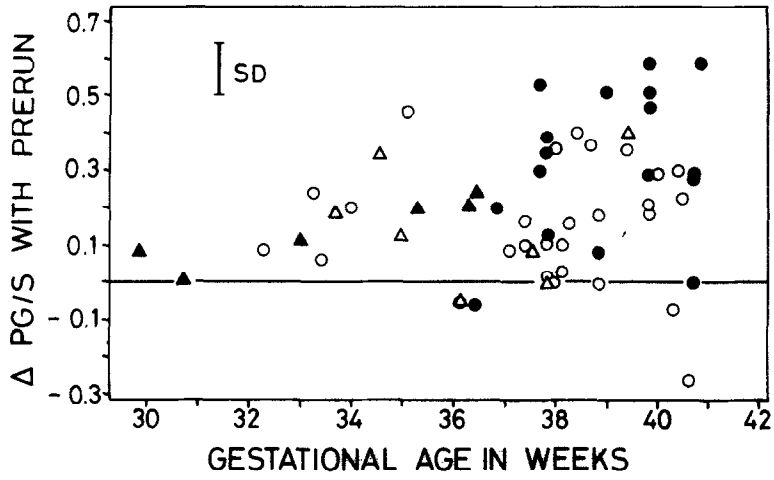

Fig. 1. Amount of pseudo-phosphatidylglycerol found in amniotic fluid, expressed as a ratio to sphingomyelin (*PG/S* ratio). The values are coded as to outcome as discussed in Reference 3, with *closed triangles* representing cases of respiratory distress syndrome; *open triangles*, mild transient difficulty; and *closed circles*, absence of any respiratory difficulty (delivery within 48 hours).

In addition, we have recognized and recently reported the presence of a substance found in most amniotic fluids which imitates phosphatidylglycerol with this system.[3] The substance ("pseudo phosphatidylglycerol"), of which Dr. Barnes and associates may have been unaware, is present in many cases in amounts greater than the genuine phosphatidylglycerol. In a sample of 75 fluids that had appeared phosphatidylglycerol-positive with the original procedure, we found 20 to be phosphatidylglycerol-negative after the removal of the contaminating substance.

The accompanying Fig. 1 shows the amount of pseudo phosphatidylglycerol we found, expressed as a ratio to sphingomyelin. Notably, the cases of respiratory distress syndrome in this population had pseudo phosphatidylglycerol as great as a ratio of 0.2, but of these cases, four were shown to actually be phosphatidylglycerol-negative after removal of the pseudo phosphatidylglycerol.

We believe that the confusion of pseudo-phosphatidylglycerol for phosphatidylglycerol with this system may be an important factor in the results that Barnes et al. report. The authors may wish to reanalyze the specimens, using the modification we have developed to eliminate the interference of "pseudo phosphatidylglycerol" in the Helena method.

David B. Cotton, M.D.
Department of Obstetrics and Gynecology
Baylor College of Medicine
One Baylor Plaza
Houston, Texas 77030

Thomas Spillman, Ph.D.
Department of Pathology and Laboratory Medicine
University of Texas Medical School
6431 Fannin
Houston, Texas 77030

REFERENCES

1. Touchstone JC, Chen JC, Beaver KM. Improved separation of phospholipids in thin layer chromatography. Lipids 1980;15:61.
2. Cotton DB, Spillman T, Bretaudiere JP. Effect of blood contamination on lecithin to sphingomyelin ratios in amniotic fluid by different detection methods. Clin Chim Acta 1984;17:299.
3. Spillman T, Cotton DB, Lynn S, Bretaudiere JP. Removal of a component interfering with phosphatidylglycerol estimation in the Helena system for amniotic fluid phospholipids. Clin Chem 1984;30:737.

Reply to Cotton and Spillman

To the Editors:

The concept of "pseudo-phosphatidylglycerol" mentioned by Drs. Cotton and Spillman is intriguing (see Reference 3 in their letter). It remains an unidentified substance, a co-migrator with phosphatidylglycerol found resistant to digestion by *Bacillus cereus* phospholipase C. It is suggested that a prechromatography step of developing the sample on the plates for 70 minutes, equilibrating with the solvent for 10 minutes, and air-drying for 10 minutes removes pseudo-phosphatidylglycerol.

Cotton and Spillman describe a sample of 75 amniotic fluids reported initially as phosphatidylglycerol positive; however, 20 fluids (27%) were subsequently found to be, in fact, PG negative with use of the prechromatography step. We find this figure high and would expect a greater incidence of the respiratory distress syndrome with positive phosphatidylglycerol by use of our current one-dimensional thin-layer chromatography method with the Helena Tek 200 system with that percentage of error from contamination. Our experience has been that respiratory distress syndrome is rare in cases reported as phosphatidylglycerol positive. Whit-