Diagnosing the Prostatitis Patient:
The Dilemma Continues
Jeannette M. Potts, MD

Introduction
It has been estimated that greater than 50% of men will experience an episode of prostatitis during their lifetime [1]. The vast majority of these men are diagnosed with abacterial prostatitis or prostatodynia, also referred to as the National Institutes of Health (NIH) Category III prostatitis/chronic pelvic pain syndrome (CP/CPPS). Community-based surveys have demonstrated a prevalence of this disorder in 8% [2], and as high as 11.5%, of men younger than 50 years of age [3]. The prevalence of self-reported history of prostatitis among health care professionals was 16% [4].

Quality of life is significantly diminished for patients with prostatitis who complain of frustration and disability as a result of this disease. It has been demonstrated (as measured by symptom-severity scores) that the impact of prostatitis on a patient’s life is comparable with that experienced by a person suffering from acute myocardial infarction or Crohn’s disease [5•].

Despite its prevalence and significant impact on the life of the patient, medical research is lacking an established cause for abacterial prostatitis. Unfortunately, the paucity of evidence-based medical research exists for all areas of prostatitis, but this is especially true with respect to NIH CP/CPPS [6].

The literature that does exist has been thoroughly reviewed by Collins et al. [7••], who concluded that no gold standard currently exists for the diagnosis of prostatitis. The reviewers also observed weak methodology in the application of diagnostic testing.

The Dilemma: What Is Abacterial Prostatitis?
Is it an Abnormality of the Prostate Gland?
In 1995, the NIH/National Institute of Diabetes and Digestive and Kidney Diseases consensus established the prostatitis classification system, defining NIH Category III chronic abacterial prostatitis as chronic pelvic pain syndrome and thereby acknowledging that the most common form of prostatitis “may in fact be caused by a disorder unrelated to the prostate gland alone” [8]. Unfortunately, traditional prejudice and industry support from pharmaceutical and surgical companies have perpetuated a “prostatocentric” approach to patient care. Furthermore, examiners are too eager to diagnose prostatitis in any young man with pelvic/genital pain who then experiences discomfort during digital rectal examination, citing this symptomatic examination as a corroborative finding! The end result of this is the promotion of a fictitious diagnosis, and what I call antibiotic codependency.

The diagnostic criteria for this syndrome as a prostatic disease is loaded with contradiction. The patient describes his pain as coming from the prostate gland only because someone in the past explained the location of the gland and/or the patient read about this on the Internet or in other self-help literature. Many patients who initially present to urologists with genital discomfort or rectal complaints are told that they have prostatitis and subsequently receive a relatively long course of antibiotics. Few, if any patients are evaluated for other sources of referred pain, or occupational or psychologic stressors that may be contributing to their symptoms. Fortunately, patients can be assessed more accurately for genital/pelvic pain and associated symptoms through the NIH Chronic Prostatitis Symptom Index (CPSI). The establishment of the CPSI is a welcome standardized tool that has promising value in research for both diagnostic and outcome measurement [9••].

Microbiologic evaluation for CPPS lacks reliability, as demonstrated by conflicting clinical findings and treatment outcomes. Although the quantification of leukocytes in expressed prostatic secretion (EPS) allows researchers and
practitioners to further subclassify CP/CPPS into Category IIIa (inflammatory) and Category IIIb (noninflammatory, historically prostatodynia) disease, this distinction has little clinical significance. It is well known that leukocyte count rarely correlates with clinical presentation and findings; that is, leukocytes may vary regardless of patients’ symptomatology or efficacy of therapy for both chronic bacterial and abacterial forms of prostatitis [10]. Furthermore, findings in EPS, postprostatic massage urine, and ejaculated specimens vary greatly. When comparing EPS, postprostatic massage urine, and seminal fluid from patients with prostatitis, Krieger et al. [11] found that inflammation was detected in only 28% of patients when EPS was evaluated alone. However, according to the new NIH consensus classification, which also considers inspection of the postprostatic massage urine and seminal fluid, twice as many patients were found to have evidence of inflammation [11]. When combining diagnostic tests such as measurement of leukocytes in EPS, four-glass culture, and transrectal ultrasonography, differentiation between categories of prostatitis for purpose of prescribing therapy was found to be difficult [12].

Leukocytospermia is observed with frequency among asymptomatic men in infertility clinics [13]. The role of infection in this condition remains unclear. Among asymptomatic men with elevated serum prostate-specific antigen, 42% had positive EPS with greater than 20 leukocytes per high powered field in the absence of urinary tract infection. Histologic evidence of prostatic inflammation was demonstrated in 52% of patients who underwent transrectal prostate gland biopsy [14]. Surprisingly, however, when patients with symptomatic prostatitis were evaluated by transrectal biopsy only 5% of collected specimens exhibited significant evidence of inflammation [15].

An infectious cause of CP/CPPS has been thoroughly investigated without producing compelling evidence for a culpable organism [6]. The urologic community is in agreement that only 5% to 7% of patients with chronic prostatitis will have positive cultures when tested. Ironically, surveyed urologists reported they rarely (47%) or never (33%) perform the four-glass test for localization cultures, yet always (40%) or frequently (42%) prescribe antibiotics to their patients [16]. Physicians who routinely performed the four-glass test were more likely to prescribe treatments other than antibiotics. Further complicating this issue are the observations of Nickel et al. [17], who noted similar proportions of patients diagnosed with NIH Category II or NIH Category III prostatitis responding positively to antibiotics. The presence of bacteria or inflammatory cells was not a predictor of patient response to treatment. Randomized-controlled trials are necessary to assess the potential placebo effect of antibiotics in all categories of prostatitis, especially the nonbacterial types.

Other research has targeted the prostate gland as the source of malady. Kirby et al. [18] and later Persson and Ronquist [19] found evidence of intraprostatic urinary reflux. This finding supports a nonbacterial cause for prostatic inflammation; however, it does not provide an explanation for similar symptoms in patients diagnosed with noninflammatory disease or prostatodynia. Persson and Ronquist [19] also noted increased levels of urate and xanthine in the EPS of men with prostatitis, a finding that led to one clinical trial using allopurinol [20]. When compared with control subjects, patients receiving medication reported decreased symptoms, had lower levels of serum and urine urate, and decreased levels of xanthine and urate in their EPS. However, no significant differences were seen in EPS leukocyte counts with this therapy [20]. In a related study, Mehik et al. [21] noted significantly higher intraprostatic tissue pressure in all patients with CP/CPPS when compared with control subjects. Pressures were also noted to be higher among patients with NIH Category IIIa disease when compared with patients diagnosed with NIH Category IIIb prostatitis [21].

Given these results, it is possible that intraprostatic urinary reflux and high intraprostatic tissue pressure may be nothing more than a secondary manifestation of other urologic or pelvic pathology, such as dysfunctional voiding and/or pelvic floor tension.

Is Prostatitis a Misdiagnosed Voiding Dysfunction?

In 1994, Kaplan et al. [22] observed a high incidence of vesical neck obstruction in men misdiagnosed with chronic nonbacterial prostatitis. In a subsequent study, men younger than 50 years of age diagnosed with chronic nonbacterial prostatitis were urodynamically evaluated. Fifty-four percent of patients had primary vesicular neck obstruction, 24% had pseudodyssynergia (contraction of the external sphincter during micturition), 17% had impaired bladder contractility, and 5% had acontractile bladders [23]. Patients with bladder neck obstruction, sometimes associated with perineal pain, responded favorably to bladder neck incision, while 83% of those with pseudodyssynergia were successfully treated using biofeedback [24].

Acquired voiding dysfunction in association with prostatitis and/or prostatodynia was observed by Meares [25] and may share features with the non-neurogenic/neurogenic bladder described by Hinman [26]. The first patient described by Hinman [26] was successfully treated with hypnosis.

Other researchers have observed urodynamic evidence of pseudodyssynergia in patients with prostatitis but have not considered this to be a different diagnosis, rather a corroborative component of a CPPS perpetuated by pelvic floor tension and autonomic dysregulation. These features are consistent with myofascial pain syndromes.

Is Prostatitis a Myofascial Pain Syndrome?

Myofascial pain is defined as a focus of hyperirritability in a muscle or its fascia that causes significant discomfort in symptomatic patients. The pain may be referred in a pattern