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ROLE OF α-BLOCKERS IN THE TREATMENT OF CHRONIC PROSTATITIS

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ABSTRACT

Treatment of nonbacterial chronic prostatitis/chronic pelvic pain syndrome continues to be a challenge for the treating physician. However, results from studies on the use of α-blockers seem to show some promise. Further studies on this class of drugs in the treatment of this condition are recommended. UROLOGY **60:** 27–28, 2002. © 2002, Elsevier Science Inc.

Treatment of chronic prostatitis is challenging because the causative factors underlying this condition are not fully understood. However, there is information in the literature on its pathology that may assist in planning a treatment with some sense of rationality. Observations have been made about the urodynamic changes in the lower urinary tract that possibly have an impact on the pathogenesis of chronic prostatitis.[1–3]

Anatomic studies by Donker *et al.*[4] show that urethral sphincteric muscles are under α-adrenergic control. In video pressure flow studies and synchronous recording of the electromyographic activity of the external urethral sphincter, Barbalias *et al.*[5] observed increased maximum urethral closure pressure caused by hypertonia of the prostatic urethra from adrenergic overactivity in chronic prostatitis. What causes this adrenergic overactivity is a matter of speculation. The increased intraurethral pressure possibly results in a retrograde urethroprostatic ascent of urethral contents into the prostate, resulting in an inflammatory response that leads to prostatitis. Decreasing the adrenergic overactivity by the use of α-blockade appears to be a logical approach to reverse this process, with a possible beneficial effect on prostatitis.

Osborn *et al.*[6] documented markedly elevated urethral pressure profiles in patients with nonbacterial prostatitis. They also noted incomplete funneling of the bladder neck and narrowing of urethral sphincter on urodynamic evaluation of these patients.

Hellstrom *et al.*[3] described reflux of urine into the intraprostatic ducts in association with urethral spasm, as well as urethral stricture. It is suggested that this event of intraprostatic reflux of urine may initiate an intraprostatic inflammatory process resulting in nonbacterial prostatitis. If the refluxing urine is infected, it may result in bacterial prostatitis.

Using phenoxybenzamine, Osborn *et al.*[6] observed a symptomatic response in 13 of 27 patients (48%). Neal and Moon[7] showed a response rate of 76% (19 of 25 patients) after 1 month of therapy with 1- to 10-mg doses. Altogether, 11 responders (58%) remained asymptomatic 3 months later. Barbalias *et al.*[5] conducted a prospective study with the use of α-blockers and ciprofloxacin in nonbacterial prostatitis.[5] They used terazosin 1 to 2 mg a day or alfuzosin 2.5 mg once or twice a day for a period of >8 months. Mean follow-up time was 22 months (range, 6 months to 3 years). They observed that α-blockade significantly reduced recurrence rate in both bacterial and nonbacterial prostatitis. They concluded that use of α-blockade in the treatment of chronic prostatitis is justified.

It is evident from a careful review of these studies that they were not properly designed. However, their observations suggest that α-blockade deserves further evaluation by well-designed studies involving a large number of patients. The Chronic Prostatitis Research Network is currently conducting a study using tamsulosin and ciprofloxacin.

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DISCUSSION FOLLOWING DR. DATTA'S PRESENTATION

J. Curtis Nickel, MD (Kingston, Ontario, Canada): There is an abstract that was just published in the *Journal of Urology* looking at tamsulosin versus placebo.[1] It is a company-sponsored trial, which was underpowered. However, the investigators did use appropriate inclusion criteria and validated outcomes. It seemed to show some benefit for tamsulosin versus placebo.

Scott I. Zeitlin, MD (Los Angeles, California): You said that reflux through the acini to the peripheral zone can lead to chronic prostatitis. Are you implying that is where chronic prostatitis exists?

Nand S. Datta, MD (Los Angeles, California): When they did microdissection studies, in most patients, they found lesions in the peripheral zone.

Dr. Zeitlin: You mean on transrectal biopsy, correct?

Dr. Datta: I am talking about postmortem studies.

Dr. Zeitlin: However, do we know where chronic prostatitis exists in those patients who had been diagnosed with chronic prostatitis?

Dr. Nickel: Those studies were done in Britain, first of all by Blacklock[2] and then by Kirby when he was a fellow.[3] Basically, they put India ink or fine charcoal particles in the bladder before a transurethral resection of the prostate (TURP), and the patient voided it out. Then, when they did the TURP, they tried to determine where the particles had collected. Their conclusion was that the particles were mainly in the periphery. When we actually go back and look at it, it was just a simple comment that there appeared to be more inflammation as the duct went out to the periphery.

John N. Krieger, MD (Seattle, Washington): If you do step sections of prostates removed for various reasons, whether for cancer or benign prostate hyperplasia, you will find inflammation in the great majority of cases. In patients with no history of pelvic or prostatic pain, I have had the experience of sending pathology specimens that exhibited pathologic inflammation in the prostate.

Anthony J. Schaeffer, MD (Chicago, Illinois): So it is fair to say we do not know whether there is a role in inflammation in this condition.

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