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Increased intraprostatic pressure in patients with chronic prostatitis

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Abstract The purpose of this prospective study was to develop a method for measuring intraprostatic pressure. Intraprostatic, extraprostatic and perineal subcutaneous pressures were measured in 43 patients. Twenty-four patients had chronic nonbacterial prostatitis (CNP) and prostatic hyperplasia (group A), 10 patients had benign prostatic hyperplasia (BPH) (group B) and 9 patients served as controls (group C). The pressure measurements were performed with a Stryker pressure monitor under transrectal ultrasonographic control at three different points: perineal subcutaneous tissue, paraprostatic tissue and the apex of the prostate beneath the capsule. Significantly higher intraprostatic pressure values ($P < 0.001$) were recorded in the patients with CNP compared with the BPH patients or the controls. We conclude that this novel method of measuring intraprostatic pressure, which has not been reported earlier, could be a new tool in the diagnosis of CNP and in the evaluation of the therapeutic effects of the different treatment modalities used in CNP.

Key words Tissue pressure · Prostate gland · Chronic prostatitis · Pressure measurement

Introduction

Chronic prostatitis is a common problem in men around the world [1, 5, 9, 14, 15]. One of two men suffer from symptoms of prostatitis during their life. The etiology of chronic nonbacterial prostatitis is unclear. There are no specific diagnostic signs or symptoms associated with

this disease. The chronic syndrome is usually characterized by persistent perineal pain and functional and somatic urological problems, such as frequency, urgency, dysuria and decreased urine flow [1, 8, 10, 13–15]. The aim of our study was, first to develop a method for measuring intraprostatic tissue pressure and, second, to assess whether this pressure is increased in patients with chronic nonbacterial prostatitis with pain.

Patients and methods

Three groups of patients were included in this study:

Group A: 24 patients with painful nonbacterial prostatitis and symptoms of obstruction referred for transurethral resection of the prostate (TURP), mean age 65.5 years, range 42–81 years. The diagnosis of chronic nonbacterial prostatitis (CNP) was made primarily on the basis of a typical history, a physical examination, laboratory tests [white blood cells in urine, serum creatinine, prostatic-specific antigen (PSA), urine culture and expressed prostatic secretion (EPS) with bacteriology] and transrectal ultrasonography (TRUS). The Meares-Stamey procedure was not performed [14]. Instead, we used a simplified approach with a cell count and urine culture from EPS before and after massage of the prostate as proposed by Nickel [11]. The urine cultures in all the three groups of patients were negative, and the EPS in group A showed more leukocytes than 10 per power field, but no bacteria. The histology of TURP chips in group A showed, first of all, signs of chronic inflammation with edema and benign prostatic hyperplasia.

Group B: 10 patients with benign prostatic hyperplasia (BPH) referred for TURP without prostatitis symptoms, mean age 71.5 years, range 63–88 years. The histology of TURP chips showed benign prostatic hyperplasia.

Group C: 9 patients with no prostatic disease, mean age 45.5 years, range 35–55 years. All of these patients had been admitted for haemorrhoid surgery.

The mean prostatic volumes measured by transrectal ultrasonography (Aloka SSD, 1700, Japan) in the different groups were: group A 42.4 ml, range 34.0–50.9 ml, group B 49.4 ml, range 33.2–65.6 ml, group C 27.9 ml, range 23.4–32.4 ml. The PSA values were as follows: group A mean 3.6, range 1.4–6.0, group B mean 4.4, range 0.4–9.4, group C mean 2.7, range 1.8–3.2 (PSA, prostatic specific antigen enzyme immunoassay by Hybritech, Orion Diagnostica, Finland).

Prostatic pressure was measured with the Stryker intracompartmental pressure monitor system (295-1 pressure monitor, 295-2 Quick Pressure Monitor Set; Bio Tec Instruments, Model DPM-1,

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Winooski, V.). This tissue pressure monitor is widely used in orthopedics to measure compartmental pressure in patients with tibial bone fracture [17, 18]. The side-holed needle for the measurement of pressure was 18 G in size and 2.5 inches (6.0 cm) long. The monitor was calibrated before the pressure measurement. We used the standard package available from Stryker (Fig. 1).

The following method of measuring prostatic tissue pressure was used. The patient was in a lithotomy position. Spinal anesthesia was used in all cases. The perineal area was washed and prepared. The puncture needle was guided by TRUS. One milliliter of sterile physiological saline was injected into the tissue. The pressure reading was recorded after 10 seconds. The pressures of randomized sides of the apex of the prostate (left vs. right), randomized paraprostatic sides (left vs. right) and perineal subcutaneous tissue were measured. All patients received prophylactic antibiotic medication (ciprofloxacin 500 mg). No urosepsis or other acute infections were seen. All patients were discharged after the procedure in a good condition.

Statistics

Statistical analyses included the use of variance test and Student's *t*-test with Bonferroni's corrections.

Results

The pressures in subcutaneous perineal tissue, intraprostatic tissue and paraprostatic tissue in each study

Fig. 1 Stryker intracompartmental pressure device

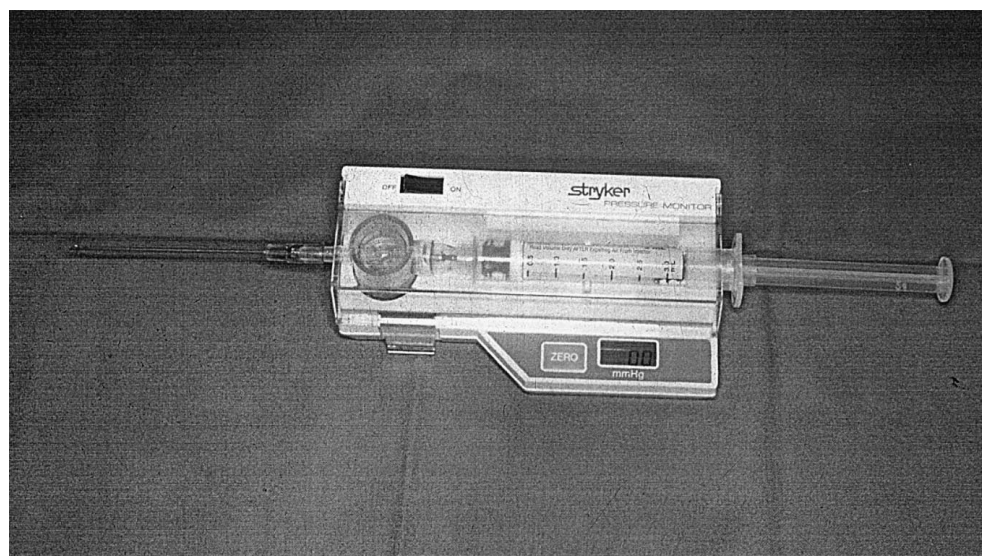


Table 1 The pressure measurement readings (mmHg) obtained with the Stryker intracompartmental device in subcutaneous perineal tissue, intraprostatic tissue and paraprostatic tissue. Group A, patients with chronic nonbacterial prostatitis (CNP); group B, patients with benign hyperplasia of prostate (BPH); group C, control patients. *N* number of patients, *SD* standard deviation, *CI* confidence interval

	N	Mean	SD	95% CI	Min	Max
<i>Pressures in subcutaneous perineal area (mmHg)</i>						
Group A	24	17.38	7.71	14.12–20.63	6	38
Group B	9	11.22	3.38	8.62–13.82	5	16
Group C	10	14.50	3.78	11.80–17.20	9	22
<i>Pressures in apical intraprostatic area (mmHg, measured randomly)</i>						
Group A	24	85.46 ^a	24.88	74.95–95.96	36	132
Group B	10	45.60	14.67	35.11–56.09	20	69
Group C	9	53.33	15.79	41.20–65.47	40	78
<i>Pressures in paraprostatic area (mmHg, measured randomly)</i>						
Group A	24	29.58	19.73	21.25–37.91	7	82
Group B	10	21.70	7.32	16.46–26.94	8	32
Group C	9	30.56	14.06	19.75–41.37	13	58

^a Between A and B-C, *F* = 15.6, *P* < 0.001

group are presented in Table 1. The only significant difference between the groups was seen in the values of intraprostatic tissue pressure, which were significantly (*P* < 0.001) higher in the patients with chronic non-bacterial prostatitis than in the patients with BPH or the control patients.

Discussion

Chronic prostatitis is a diffuse and obscure term commonly used to describe a collection of symptoms characterized by the presence of pain or discomfort in the perigenital area often radiating towards the lower back or the inside of the thigh. Its etiology is still unknown. The location of discomfort may vary from individual to individual and differ in the same individual on different occasions. A variety of micturitional symptoms in the lower urinary tract may accompany the pain or dominate the picture. Even ejaculation is sometimes painful or followed by crampy discomfort. The prostatitis syndrome has many different etiologies leading to identical or similar symptoms through a variety of pathogenetic mechanisms [1, 2, 5, 10, 12–15].

We have found no earlier reports on the measurement of intraprostatic pressure in chronic prostatitis. We found intraprostatic pressure to be high in our patients with typical symptoms of chronic nonbacterial prostatitis. Our study also showed that it is possible to measure intraprostatic pressure with the Stryker device without complications. We postulate that the increased pressure causes the pain experienced by patients with chronic nonbacterial postatitis. We know that the level of pain in these patients fluctuates. This phenomenon might be due to local edema in the prostate. Similar pathophysiological criteria and histopathological findings are also available for other parts and conditions of the human body, such as the anterior tibial syndrome, as also are findings on pancreatic tissue [2–4, 6, 7, 15, 17, 18].

There was a significant difference in tissue pressure between groups B and C on the one hand and group A, with the chronic nonbacterial prostatic syndrome and BPH, on the other. The compartmental syndrome represents a well-documented medical phenomenon. One factor must be present in the compartmental syndrome, namely increased tissue pressure [6, 7, 17, 18]. The measurement of tissue pressure in cases of compartmental syndrome in traumatology is widely used in daily practice [17, 18]. There have also been attempts to clarify the mechanism of chronic pain in patients with chronic pancreatitis [6, 7].

The reason why we chose the pressure measurement of prostatic tissue was the close functional and anatomical similarity between the tibial muscles, the pancreas and the prostate. The prostate is surrounded by Denonvillier's fascia, the endopelvic fascia and the anterior surface of the surgical capsule with puboprostatic ligaments. The stroma of the prostate is rich in blood vessels, smooth muscle and sympathetic nerve fibers connected to baro- and pain receptors [2–4, 8, 13, 16]. These three different physiological entities are comparable: traumatically injured tibia, chronically inflamed pancreas with congestion and inflamed prostate. The main symptom in all cases is pain through local tissue damage despite the different etiological backgrounds. The inflammation and/or congestion due to the tissue edema may lead to increased intraprostatic pressure and pain [2, 3, 6, 7, 16–18].

Increased sympathetic activity might be one reason for inappropriate spasm of the distal urethra, leading to increased pressure in the prostatic urethra and consequent urine reflux into the prostatic ducts. The presence of urine (sterile or infected) could induce ductal and periductal inflammation with edema of prostatic tissue and pain. This could further aggravate the spasm of the involved pelvic musculature, exacerbating the pain and the voiding dysfunction [8, 10, 13].

The paraprostatic area was not so much affected by the rising tissue pressure in the patients with CNP. The different anatomical structure and the loose tissue separated from the prostate gland by fascia might explain this finding [2, 3, 16].

On the basis of this preliminary study, it is apparent that pressure in the intraprostatic and extraprostatic regions can be measured. It also seems possible that the typical symptoms in patients with chronic nonbacterial prostatitis correlate with the increased pressure in the apical region of the prostate gland. We will therefore continue both to develop a better pressure monitoring system and to measure intraprostatic pressure in a larger group of patients with only chronic prostatitis without hyperplasia of the prostate gland.

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