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ALPHA BLOCKER TEST: AN ALTERNATIVE TO PRESSURE-FLOW STUDY FOR THE ASSESSMENT OF BLADDER OUTLET OBSTRUCTION AND DETRUSOR CONTRACTILITY IN PATIENTS WITHOUT PROSTATIC ENLARGEMENT

Aims of Study
Bladder outlet obstruction (BOO) in men with a non-enlarged prostate might be caused by dynamic obstruction mediated by alpha-1 adrenoreceptors rather than by volume-dependent static obstruction. Alpha-1 blockers are effective in these cases, if the cause of voiding dysfunction depends on BOO. In other words, it could be speculated that men might have BOO when an alpha-1 blocker is effective for them. Moreover, since the outcome of therapy, which aims to remove BOO, is influenced by detrusor contractility, the status of detrusor contractility could be supposed by means of analysis of treatment efficacy. In the present study. Transrectal ultrasound and Pressure flow study (PFS) were done in men with a prostate volume less than 20 ml, and it was investigated whether the differences in anatomical changes of the prostate, urethral resistance, and detrusor contractility lead to the differences in the degree of improvement in subjective and objective symptoms after administration of an alpha-1 blocker. In addition, it was analysed whether a cause of urinary disturbance could be determined by analysing the treatment efficacy with an alpha-1 blocker.

Methods
Thirty-two men over 50 years old (mean: 66.3) with an International Prostate Symptom Score (I-PSS) over 8 points (mean: 17.6), a Quality of Life (QOL) index over 2 points (mean: 4.6), a maximum flow rate (Qmax) below 15 ml/s (mean: 8.5) and a prostate volume less than 20 ml (mean: 16.4) were enrolled. Patients with neurogenic bladder, prostatic cancer, urethral stricture, or active urinary tract infection, as well as those who were taking medications that could influence urination were excluded.

An alpha-1 blocker, 0.2 mg/day of tamsulosin, was administered for 4 weeks. I-PSS, QOL index, free flowmetry and PFS were performed before and after tamsulosin administration. Bladder Outlet Obstruction Index (BOOI) and Bladder Contractility Index (BCI) were obtained from the measurements. Prostate Specific Antigen (PSA) was measured before treatment using a Tandem-R RIA kit. A prostate volume, transition zone volume and residual urine volume were obtained by ultrasound. The data were analysed to determine the degree of BOO and bladder contractility in these men.

Results
All subjects completed the 4-week treatment with tamsulosin without an evidence of adverse reactions. Tamsulosin administration led to an improvement in I-PSS, QOL index, Qmax and BOOI, but no change in voided volume, residual urine volume and BCI. The treatment efficacy was predicted by the degree of BOO and detrusor contractility only. There was no correlation between other factors (ex. age, prostate volume, transition zone volume, PSA and PSA / prostate volume) and the treatment efficacy. In the improved factors, the measurement that showed the highest correlation with the pre-treatment BOOI and BCI was the degree of improvement in the Qmax. (BOOI: p < 0.002, r = 0.60, BCI: p < 0.002, r = 0.59) When Qmax was improved by more than 3.5 ml/sec, the positive predictive value for both scores of indexes of more than 40 in BOOI and more than 100 in BCI was 100%.

Conclusions
In patients with non-enlarged prostate, the effectiveness of tamsulosin depended upon the degree of BOO and detrusor contractility, but not upon anatomical factors. In addition, the degree of BOO and detrusor contractility could be indicated by means of the improvement rate of Qmax after treatment with tamsulosin. The test administration of an alpha-1 blocker could be an alternative to PFS for the assessment of the degree of BOO and detrusor contractility.