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ALPHA-BLOCKERS INCREASE VESICAL AND PROSTATIC BLOOD FLOW AND BLADDER CAPACITY

Aims of Study
Alpha blockers have ben shown to relieve symptoms suggestive of bladder outlet obstruction in patients presenting with lower urinary tract symptoms but do not treat the obstruction itself. Recent findings indicate that the site of action of these drugs may not be exclusively on the smooth muscle cells in the prostatic urethra or in the bladder. In the present study the effect of alpha-blockers on vesical and prostatic blood flow as well as urodynamic parameters was investigated.

Methods
7 patients suffering from lower urinary tract symptoms (LUTS) were treated with 0.4 mg tamsulosin for 5 weeks. In all patients colour Doppler ultrasound of the urinary bladder and the prostate as well as comparative cystometry were performed before and after treatment. Normal saline (NaCl) was used for the first run, followed by a second run with 0.2 M potassium chloride (KCl). Additional to standard urodynamic parameters recorded in each run peak systolic blood flow velocity (PSBFV) and end diastolic blood flow velocity (EDBFV) were measured in several intramural arteries in the urinary bladdder and the prostate at filling volumes of 0ml, 100 ml and maximum cystometric capacity (Cmax). For these measurements a color Doppler unit (Acuson, USA) fitted with an endorectal probe was used. The resistance index (RI) was defined as (PSBFV-EDBFV)/PSBFV.

Results
Contrary to healthy men all patients showed a strong pathologic response to intravesical KCl (mean Cmax 431 ml with NaCl and 313 ml with KCl) before treatment with tamsulosin. In the presence of NaCl, mean PSBFV increased with distension from 9 cm/s to 17 cm/s. Mean PSBFV in the urinary bladder and the prostate did not increase significantly during filling with KCl (mean PSBFV rose only from 10 cm/s to 13 cm/s), which provided further evidence that the normal autoregulation of vesical and prostatic blood flow was pathologic. After therapy maximum urinary flow and bladder contractility did not change significantly. Cmax during filling with NaCl remained unchanged while mean Cmax during filling with KCl was significantly increased after therapy (409 ml). Furthermore, after therapy PSBFV was siginificantly increased in the prostate as well as in the urinary bladder in the presence of KCl (12 cm/s in the empty bladder and 21 ms/s at Cmax with KCl). RI values remained unchanged before and after therapy, indicating that the increase of PSBFV after therapy was associated with a concomitant increase in blood flow. After treatment with tamsulosin the values of Cmax and PSBFV during filling with KCl were comparable to normal values measured in healthy volunteers in earlier studies.

Conclusions
Our data strongly suggest that benign prostatic hyperplasia is associated with a dysregulation of normal blood flow in the urinary bladder and prostate. Furthermore, alpha blockers increase blood flow in the lower urinary tract and maximum bladder capacity. These results may explain the therapeutic effects of alpha-blockers on lower urinary tract symptoms, while standard urodynamic parameters are not influenced.