THE CHANGES OF EXPRESSION OF ALPHA- AND BETA-ADRENOCEPTOR RELATED TO URODYNAMIC CHANGES IN RAT BLADDER OUTLET OBSTRUCTION

Hypothesis / aims of study
To explore possible changes in expression and/or function of a1- and beta-adrenoceptor subtypes as a cause for bladder dysfunction and to assessed the possibility of the usage of beta-adrenoceptor in a rat model of bladder outlet obstruction (BOO) with storage symptoms.

Study design, materials and methods
Rats were randomized into 2 groups; sham operation (control group) and BOO group. Non-anesthetic urodynamic study (UDS) was performed with bladders from BOO group and sham-operated (control) rats 4 weeks after BOO induction. Of these BOO group, bladder only with increased bladder weight and intravesical pressure and decreased micturition interval in UDS was choiced and mRNA expression of a1- and b-adrenoceptor subtypes was assessed by quantitative real-time PCR.

Results
The mRNA expression of alpha1A-, alpha1D-, beta2- and beta3-adrenoceptors were increased in BOO group (respectively, 2.04, 1.68, 1.28 and 1.46 times than control group).

Interpretation of results
The mRNA expression of alpha1A-, alpha1D-, beta2- and beta3-adrenoceptors were increased in BOO group than control group.

Concluding message
In a rat BOO model with storage symptoms, most of adrenoceptors were upregulated. These suggest that beta agonist might ameliorate the storage symptoms in the management of male overactive bladder.

Figure. Relative expression of adrenoceptor subtype mRNA in bladder of partial obstruction as compared to sham operated rats. Data are expressed as folds of corresponding expression in sham-operated rats.