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Author(s): G. Hohlbrugger, *F. Frauscher, T. Stötzer, H. Strasser, G. Bartsch

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Institution City Country: Department of Urology and *Radiology II, Innsbruck, Austria

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Title (type in CAPITAL LETTERS): **COMPARATIVE (NACL VS =0.2 M KCL) CSYTOMETRY OF NEUROGENIC BLADDER: INTRAVESICAL POTASSIUM ALTERS BLOOD FLOW OF BLADDER WALL BUT NOT INTRAVESICAL PRESSURE**

Aims of study: In the normal bladder with its 'tight' urothelium, stretching and in particular, the presence of intravesical KCl (0.2 M) enhance bladder wall circulation. However, in a majority of patients with urgency/frequency syndrome, urge incontinence or interstitial cystitis, KCl reduces not only the maximal cystometric capacity (Cmax) but also circulation. The former is considered to result from an increased urothelial permeability due to a urothelial GAG layer deficit (a lack of mucous production by urothelial cells). In view of their markedly increased susceptibility to infection, neurogenic bladders might also exhibit such a phenomenon. For this reason, we expected to have analogous results.

Methods: Comparative cystometry (NaCl vs 0.2 M KCl; 50 ml/min) was perfomed in 27 patients (6 with complete upper motor lesions and 21 with complete lower motor lesions; UMNL and LMNL). Simultaneously, vesical blood flow was measured at several visible arteries of the accessible wall segment at 50 ml volume, until unstable detrusor contraction set in (UMNL) or until a maximal filling volume of 500 ml was reached (LMNL). Measurement was carried out using an endorectal ultrasound probe and a colour Doppler (Acuson 128 XP).

Results: Not a single urodynamic parameter was altered by KCl in neurogenic bladders. Similarly to normal bladder, at first the mean peak blood flow velocity (PBFV) increased by stretching caused by NaCl alone and in UMNL, in the presence of KCl, it increased from 0.07 (50 ml) to 0.18 m/sec. With the onset of unstable detrusor contraction, in neither cases of filling volume employed could perfused arteries be detected. In patients with LMNL, blood flow remained constant (0.12 m/sec.) even during stretching caused by NaCl. In contrast, the PBFV dropped in the presence of KCl from 0.08 (50 ml) to 0.04 m/sec. (500 ml).

Conclusions: In neurogenic bladders, urodynamic diagnosis through comparative cystometry yields no additional information. In contrast and despite a suspected GAG layer deficit, it suggests that the urothelial barrier function is maintained. Because denervation affects also other urothelial functions such as active transurothelial NaCl-transport (1), retention of epidermal growth factor receptor protein (2), arrest of cell proliferation (2) as well as release of NO (3), it can be assumed that as far as barrier function is concerned, GAG layer deficit is compensated by a closing of urothelial ion channels. The obviously reflex-guided reduced blood flow probably makes an essential contribution to the morphologically detectable degenerative changes in the neurogenic bladder.

References:

1. Proc. Int. Cont. Soc. 11, p. 122, 1981
2. J. Urol. 153, Suppl., Abstr. 420. 1995
3. J. Urol. 159, Suppl., Abstr. 76, 1998