

## ACONTRACTILE DETRUSOR IN STRESS INCONTINENCE: AN OVERLOOKED PROBLEM ?

### Aims of Study

Stress urinary incontinence (SUI) is defined by the International Continence Society as involuntary leakage from the urethra, synchronous with exertion/effort, or sneezing or coughing (1). An acontractile detrusor is one that cannot be demonstrated to contract during urodynamic studies (1).

SUI is due to a decreased urethral resistance. The perimictional detrusor pressure appears to be impaired in SUI patients (2,3). This reduced detrusor pressure can be explained as no higher detrusor strength is needed to overcome the lower outflow resistance.

Clinical experience has shown that many SUI patients void without rise in detrusor pressure with or without abdominal pressure rise.

We compared the voiding in three different groups of women.

### Methods

We included 126 SUI patients, 15 healthy women without urinary complaints, surgical history in the pelvic region or neurological disease, and 51 patients with bladder overactivity (BOA). All underwent a standardized 6-channel cystometry and pressure flow measurement. A pressure rise of the detrusor of more than 15 cm H<sub>2</sub>O during voiding is accepted as minimal sign of active detrusor involvement in voiding (4). Only women who were able to void 100 ml were included in the data analysis. We rearranged each group according the detrusor function and straining behaviour (conform to ICS terminology) : 1. no straining combined with normal detrusor function, 2. perimictional straining and normal detrusor function, 3. perimictional straining and acontractile detrusor.

### Results

The mean age was 56,5 years (30- 83) in the SUI group, 50 years in the volunteers ( 40- 60) and 56,6 years in the OAB group (25- 82). The groups are of matching age ( $p < 0.05$ ).

14 women were not able to void on command, 15 women voided less than 100 ml. These women were equally divided over the three groups ( $p < 0.05$ ). Finally 12 volunteers are included, 103 SUI patients and 48 BOA patients.

The voiding behaviour is described in table 1.

Table 1: the voiding behaviour in women

	<u>control</u>		<u>SUI</u>		<u>BOA</u>	
	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%
<b><i>No perimictional straining and normal detrusor function</i></b>	3	25.0	35	34	23	47.9
<b><i>Perimictional straining and normal detrusor function</i></b>	7	58.3	36	35	22	45.8
<b><i>Perimictional straining and acontractile detrusor</i></b>	2	16.7	32	31	3	6.3

### Conclusions

In SUI patients more women strain to void without development of detrusor contraction. Previously it was shown that straining is hardly present in young women without symptoms. Is straining the consequence of ever diminishing detrusor contraction because of the low urethral resistance? Or is straining a possible cause of the SUI? As treatment of voiding dysfunction and restoring of detrusor contraction often results in the disappearance of the SUI

(5) we have tendency to believe that straining can be the cause of SUI in a large group of women with this symptom. A larger study is ungoing.

### **References**

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