

## URETHRAL SPHINCTER INSTABILITY – AN IMPORTANT CONTRIBUTING FACTOR IN OAB PATIENTS?

### Hypothesis / aims of study

Overactive bladder syndrome (OAB) is according to the International Urogynecological Association (IUGA) and the International Continence Society (ICS) defined as a symptom combination of urgency, accompanied by frequency and/or nocturia, with or without urge urinary incontinence (UUI). OAB is a symptomatic diagnosis, while detrusor overactivity (DO) is a urodynamic observation defined by involuntary detrusor contractions during the filling phase, which may be spontaneous or provoked. The aim of this study is to investigate if urethral sphincter instability is a contributing factor in OAB patients ignored not only in diagnostics but also in therapeutical concepts.

### Study design, materials and methods

We did continuous urethral pressure profilometry (cUPP) during CMG (cystometrogram) in 38 female patients. 13 were investigated because of OAB, not responding to first line therapy with antimuscarinics and conservative treatment, in 25 female patients with stress incontinence were tested prior to surgery, including 8 patients presenting themselves with mixed incontinence. All patients were recruited within our routine investigation in our continence clinic – ethical approval and patient consent was obtained. Patients with genital prolapse >grade I were excluded, as well as patients with impaired cognitive function or neurogenic disorder, which prevented them from lying still on the table. Bacteriuria at the time of investigation was excluded by urine analysis. All patients underwent conventional filling cystometry including pressure/flow studies followed by a conventional urethral pressure profile measurement with and without cough test at 2ml filling and a continuous filling cUPP with the catheter positioned at the site of the maximum urethral closure pressure determined by urethral pressure profile under rest. The bladder was filled with medium filling speed of 30ml/min. The cUPP was done in the supine position to reduce moving artefact and the patient was asked to lie relaxed with as little movement as possible. Throughout the investigation pelvic floor electromyography (EMG) was registered by surface electrodes. Urethral pressure change was calculated as the difference of the highest closing pressure minus the lowest closing pressure during the filling period ( $\Delta$ UP- urethral pressure). A urethral pressure drop with urgency to void at maximum bladder capacity before micturition was neglected.

### Results

The profilometry study in the genuine stress incontinent showed significantly ( $p < 0.05$ ) less pressure change during constant urethral pressure measurement compared to patients with OAB. (Table 1).

Table 1. Comparison of  $\Delta$ UP in patients with OAB and with stress incontinence component

	OAB	Mixed and pure stress incontinence	p-value
$\Delta$ UP	46.0 $\pm$ SD 44.11	11.95 $\pm$ SD 14.08	<0.05 s

Measurement of UPP and cUPP in women with stress incontinence revealed median 53 and 43 cm H<sub>2</sub>O, respectively. This was significantly lower ( $p < 0.01$ ) than in patients presenting with an instable detrusor during the filling phase with a median 75 and 65 cm H<sub>2</sub>O. The presence of urethral instability seems is not associated with age and maximal urethral closure pressure (Table 2).

Table 2. Correlation of  $\Delta$ UP with age and maximum urethral closure pressure (MUCP)

	OAB		Mixed and pure stress incontinence	
	r -value	p-value	r -value	p-value
$\Delta$ UP Age	-0.711	<0.01 HS	-0.105	> 0.05 NS
$\Delta$ UP MUCP	-0.555	<0.05 S	0.173	> 0.05 NS

### Interpretation of results

This study confirms that urethral pressures obtained during the filling phase of the CMG are far more stable in stress incontinent women in contrast to what is seen in patients with OAB. Moreover urethral instability is quite independent from often precipitating factors as age and external sphincter sufficiency, expressed here as a MUCP. All patients with urodynamically proven DO had pressure changes during cUPP of more than 10cmH<sub>2</sub>O. None of the patients with OAB and pressure change less than 10cmH<sub>2</sub>O had DO during the urodynamic investigation. The aetiology of urethral instability is still unknown. Is it primarily a defect of the urethra or it's secondary to a change in bladder pressure during the filling phase?

### Concluding message

Up to now we do not really understand bladder sensation and specifically the symptom of urgency. Up The term „overactive bladder” may distract from the important role of urethral sphincter.

Knowing which patients with OAB have additional urethral sphincter instability might shed light in the multifactorial aetiology of OAB. With a wide armamentarium of new anticholinergic drugs with combined activity as on muscarinic receptors and on calcium channel blockers of smooth urethral muscles on the market and in the pipeline this knowledge could influence future diagnostic approach of OAB and eventually even on a choice of medication.

Disclosures

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