

DOES URODYNAMICS CAN EXPLAIN WHICH OCCURS DURING DETRUSOR HYPERACTIVITY WITH IMPAIRED CONTRACTILITY (DHIC)?

Hypothesis / aims of study

Detrusor hyperactivity with impaired contractility (DHIC) is a complex voiding dysfunction [1]. If detrusor overactivity (DO) is clearly defined, impaired contractile function can be due to several causes. Our purpose was to investigate whether urodynamics could provide information on the mechanism of DHIC.

Study design, materials and methods

Voiding is a voluntary process needing sustained detrusor contraction and sphincter relaxation.

For normal subjects, there is no detrusor excitation during bladder filling while sphincter excitation is maximum. For a given volume, bladder wall sensors trigger the voiding process: detrusor excitation increases toward its asymptotic value while sphincter excitation decreases toward zero. For bladder volume close to 20 mL, bladder sensors stop the voiding process and sphincter excitation increases. In DHIC, the bladder is overactive but empties ineffectively. Our purpose was, in patients with DHIC, to search for urodynamic relationship with cortical deficit that are believed to cause urge incontinence.

Retrospectively, pressure-flow studies (PFs) of 145 non neurological women with urodynamic diagnosis of DO and without symptom suggestive of obstruction, or diabetes, or grade >2 prolapse, or previous pelvic floor surgery were analyzed.

All PFs were performed using a triple lumen urethral catheter 7F, allowing recording of urethral pressure p_{ura} , therefore to analyze the coordination between the behaviour of detrusor and sphincter during voiding.

To define detrusor underactivity (DU) reduced detrusor strength, increased voiding time (t_{mic}) and large post void residual (PVR) are considered [2]. To assess DHIC, $t_{mic} > 20\%$ of the mean t_{mic} and $PVR > 20\%$ of the bladder filling were considered in first line. Detrusor contractility was evaluated by the VBN parameter k [3].

Results

Mean age was 63.3 ± 16.1 y. DO was phasic (DOP) in 74 files and terminal (DOT) in 71. DOT was observed in older women (66.4 ± 13.5 vs. 59.5 ± 17.5 y; $p = .0096$).

Association of chosen criteria for DU were founded in 21 (14%) files. Among them 14 (66.7%) had urge incontinence.

Women with DHIC were younger, values of $p_{det.Qmax}$ and k were not consistent with a reduced detrusor strength (Table)

	age (y)	t_{mic} (s)	PVR (mL)	Q_{max} (mL/s)	$p_{det.Qmax}$ cm H ₂ O	k
N = 21 (12 DOP; 9 DOT)	60.2 ± 16.0	79.1 ± 21.7	166.4 ± 90.4	6.6 ± 3.5	41.8 ± 19.3	$.61 \pm .35$
N = 124 (62 DOP; 62 DOT)	63.9 ± 16.4	43.3 ± 21.3	52.7 ± 76.5	11.3 ± 5.3	29.4 ± 15.1	$.48 \pm .33$

Looking at the pressures during the voiding phase of the 21 files with DO and DU,

1- urethral pressure: the end of flow resulted from the increase of urethral pressure becoming equal to the detrusor pressure though there was bladder is far from empty.

2- detrusor pressure: there was a tendency to an early decrease of p_{det} .

Interpretation of results

In that population of non-neurological women with urodynamic diagnosis of DO, combination of DO and DU is observed in not frequent. The type of DO is not characteristic while one could suppose that terminal DO (single involuntary detrusor contraction resulting in bladder emptying) may not be present in DHIC.

Two results are very intriguing in that population with idiopathic DO and DU:

1- The DHIC sub-group is younger. In all reports DHIC is more prevalent in elderly.

2- In the DHIC sub-group, $p_{det.Qmax}$ is higher as the value of parameter of detrusor contractility.

To explain the high $p_{det.Qmax}$ concomitant with a large PVR one has to assume a wrong coordination between the centers governing respectively the excitation of the detrusor and of the sphincter.

Concluding message

This study underlines abnormal conditions: relative young age, high detrusor pressure and high detrusor contractility in a population of non-neurological women, with idiopathic DO and DU, which association consists in DHIC. Recording of urethral pressure during PFs bring to the fore the wrong coordination between detrusor and sphincter during DHIC.

References

1. Yalla SV, Sullivan MP, Resnick NM. Update on detrusor hyperactivity with impaired contractility. *Current Bladder Dysfunction Reports*. 2007; 2: 191-6
2. van Koeveeringe GA, Vahabi B, Andersson KE, Kirschner-Herrmans R, Oelke M. Detrusor underactivity: a plea for new approaches to a common bladder dysfunction. *NAU* 2011; 30: 723-8
3. Valentini FA, Besson GR, Nelson PP, Zimmern PE. Clinically relevant modelling of urodynamics function: The VBN model. *NAU* 2014; 33(3): 361-66. doi 10.1002/nau.22409

Disclosures

Funding: none **Clinical Trial:** No **Subjects:** HUMAN **Ethics not Req'd:** It involved retrospective analysis of urodynamic studies from a database. **Helsinki:** Yes **Informed Consent:** No