IS THERE A URODYNAMIC PARAMETER ASSOCIATED WITH DETRUSOR AFTER-CONTRACTION? NEW INSIGHT.

Hypothesis / aims of study

Detrusor after-contractions (DAC) are non common in adults (1). Both definition (nothing in ICS reports) and significance (artefact, link with detrusor overactivity (DO) or bladder outlet obstruction (BOO)) remain discussed. DAC are detected by all kind of pressure transducers. Recently it has been proposed the following definition: "a detrusor pressure increase after flow ceases at the end of micturition" (2) but no new explanation. Our purpose was to make a VBN analysis of the voiding phase preceding DAC and to make simulations of pathophysiological conditions able to explain both voiding phase and DAC.

Study design, materials and methods

Our database consisted of 44 DAC observed during UDS in 44 patients (40 females and 4 males) who presented with voiding dysfunction. Cystometry was performed with a 7F triple lumen catheter allowing the recording of urethral pressure.

Criteria for DAC were: post void residual <30 mL and increase of detrusor pressure (p_{det})>10cm H₂O.

We used the VBN mathematical micturition model (3) for analysis of recordings and simulations.

Results

<u>1-UDS</u>

Onset of DAC (ODAC) occurred during the end of voiding: Q_{ODAC} = 8.8±6.0 mL/s (while Q_{max} =18.9±8.2 mL/s; p< .0001). There was a brisk increase of p_{det} at the onset of DAC in 20 files (Fig A). The bladder volume at ODAC was 17.9±13.4mL (i.e. bladder collapse condition).

The pressure amplitude of DAC (p_{DAC}) was significantly higher than $p_{det.Qmax}$: 82.3±57.4 vs. 33.1±16.6 cm H₂O (p<.0001).

The ratio voided volume at the onset of DAC vs. voided volume was .901 \pm .110.

DO was found in 16/44 (36%) patients and BOO in 7/44 (16%). Urgency was the complaint for 29/44 (66%) patients.

An increase of rectal pressure was observed at the onset of DAC in 8 files $(20.1\pm10.6 \text{ cm H}_2\text{O})$ but with concomitant increase of urethral pressure in only 1 file. No change of activity of both urethra and rectum was observed during DAC.

2-VBN

The voiding phase until the onset of DAC was always restored by VBN analysis (Fig A-D). As in voidings without DAC, the fitting of recorded and computed curves in their terminal part was sometimes obtained with the addition of an effective abdominal pressure (which only acts on the bladder) in 22 /44 (50%) files (Fig C-D). That additional pressure clearly began before the onset of DAC (Fig C-D).

After the onset, the flow curve was restored using the previous VBN parameters but the increase of p_{det} could not be restored. Neither hypothesis of a great (abnormal) detrusor force or an abdominal straining only effective after voiding nor that of abnormal nervous control before the end of voiding allowed restoring the recorded curves (pressure, flow rate and DAC).

Interpretation of results

Complaint of urgency and urodynamic diagnosis of detrusor overactivity are the more frequent scenarios associated with DAC. Ninety percent of both p_{det} and Q are restored by the standard VBN analysis, thus we can say that the VBN analysis is reliable. At the opposite of results given in a previous study (1), DAC does not correlate with a sphincter contraction.

The effective abdominal pressure allowing restoring the end of the flow curve in 50% files does not induce the DAC.

Urgency or detrusor overactivity are frequently observed in DAC patients. As all studied DAC occur in condition of bladder collapse, our hypothesis is that a local concentration of stresses occurs at the contact between the transducer and the bladder wall. Why the DAC stops remains understood.

Concluding message

The onset of DAC is the more significant phenomenon. DAC is not associated with BOO but more probably with bladder overactivity. That latest phenomenon occurring with concomitant bladder wall collapse could lead to a concentration of stresses around the transducer similar to that observed at the tip of a nail. DAC appears as the result of local conditions in an almost empty bladder and thus of weak clinical significance.



References

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Disclosures

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