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Detrusor after-contraction: a new insight

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ABSTRACT

Aims: Detrusor after-contractions (DAC) are non-common in adults. Both definition (nothing in ICS reports) and significance (artefact, link with detrusor overactivity (DO) or bladder outlet obstruction (BOO)) remain discussed. Our purpose was to carry out an analysis of the urodynamic parameters during voidings with DAC and, using the VBN model, to simulate pathophysiological conditions able to explain both voiding phase and DAC.

Materials and Methods: From large urodynamic database of patients referred for evaluation of lower urinary tract dysfunction, DAC were observed in 60 patients (5.7%). Criteria for DAC were post-void residual <30mL and increase of detrusor pressure >10cmH₂O. VBN model was used for analysis of both pressure and flow curves, and simulations of pathophysiological conditions.

Results: Onset of DAC (ODAC) occurred when $Q=7.3\pm 5.7$ mL/s and bladder volume= 17.9 ± 15.4 mL. Urgency-frequency syndrome and urodynamic diagnosis of DO were the more frequent scenarios associated with DAC. ODAC was associated to an inversion of the slope of detrusor pressure curve without any perturbation in flow curve. Among tested pathophysiological hypothesis (great, abnormal, detrusor force, sphincter contraction), none allowed restoring all recorded curves (flow rate, voiding pressure and DAC).

Conclusion: No urodynamic characteristic of the first part of voiding is an index of occurrence of DAC. ODAC is a significant phenomenon linked with the bladder collapse. DAC is not associated with BOO but more probably with DO and appears as the result of local conditions in an almost empty bladder (concentration of stresses around a transducer); thus DAC seems of weak clinical significance.

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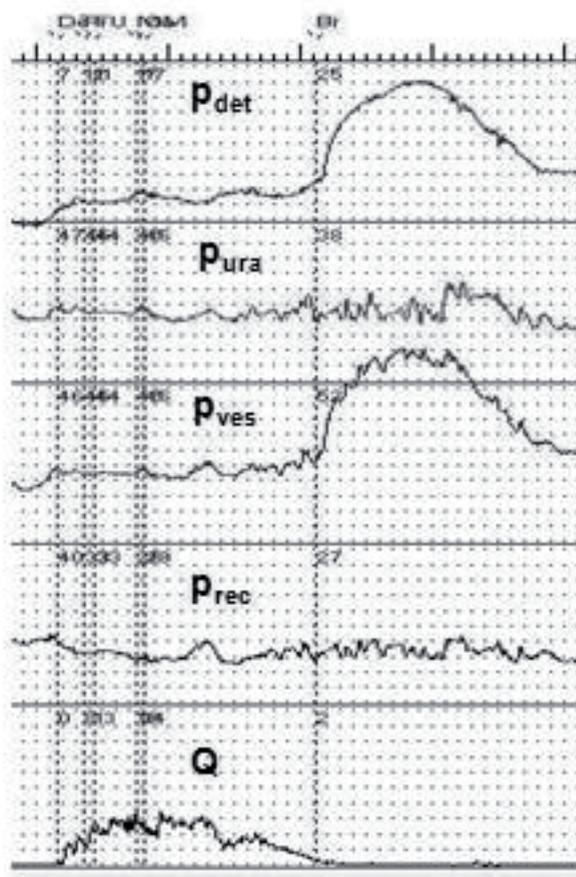
INTRODUCTION

Detrusor after-contraction (DAC) is an urodynamic phenomenon (Figure-1) which definition (nothing in ICS reports) (1, 2) is not clear despite a recent proposal as “a detrusor pressure increase after flow ceases at the end of micturition” (3) although DAC is frequently observed when the bladder is close to empty (4). The first observation by Rehflish dates from 1897, and few studies have focused on DAC; strict definition, causes and significance of that phenomenon remain far from elucidation.

Although DAC is frequently observed in children (4), it is also observed in adults. DAC is detected by all kind of pressure transducers (5). Several hypothesis about its mechanism had been suggested (6, 7): DAC is an artifact or can be due to some dysfunction of the bladder or/and the urethra and thus be eventually correlated with detrusor overactivity (DO) (6) or bladder outlet obstruction (BOO) (7).

Our purpose was to propose strict criteria to define DAC, then to analyze the urodynamic characteristics and to use mathematical modeling

Figure 1 - Recording of a typical example of detrusor after-contraction (DAC) observed during intubated flow of a female patient. From top to bottom: detrusor pressure (p_{det}), urethral pressure (p_{ura}), vesical pressure (p_{ves}), rectal pressure (p_{rec}) and flow rate (Q). Note the high amplitude of DAC compared with p_{det} during voiding.



to test earlier hypotheses proposed as causes of DAC in order to answer to the question: artifact or clinical significance.

MATERIALS AND METHODS

Urodynamic recordings of patients referred to our urodynamic laboratory for evaluation of lower urinary tract dysfunction over the period January 2006 to December 2009 were retrospectively analyzed.

To state unambiguously the occurrence of DAC, two criteria were chosen: PVR should be $<30\text{mL}$ to avoid a possible resumption of the

voiding process and increase of detrusor pressure (p_{det}) $>10\text{cmH}_2\text{O}$ to avoid error of reading.

To use mathematical modeling, additional conditions were necessary for voiding phase before DAC: $Q_{max} > 2\text{mL/s}$, voided volume $\geq 100\text{mL}$, continuous flow curves without predominant abdominal straining.

Cystometry was performed with the patient in the seated position with a 7-F triple-lumen urethral catheter perfused with saline at room temperature, using a filling rate of 50mL/min . The type of catheter enabled the recording of urethral pressure during cystometry and pressure-flow study; the distal eye-hole, kept in the lateral position, was located at the site of maximal urethral closure pressure. Pressure transducers were zeroed to atmospheric pressure at the upper edge of the symphysis pubis. Rectal pressure was recorded using a punctured intra-rectal balloon catheter filled with 2mL of saline according to the report of Good Urodynamic Practice guidelines (8). Post void residual volumes (PVR) were measured by ultrasound (US) using a Bladder-Scan®.

Among 1823 patients who underwent filling cystometry, only 1047 succeeded (714 women and 333 men) in a pressure-flow study which fulfilled the required criteria for mathematical modeling. Among these 1047 patients and according with the proposed criteria, DAC was found in 60 patients (53 women and 7 men). Three women performed two cystometries with intubated flow in the same session, and DAC was observed in both.

Method

In a first step, careful analysis of urodynamic recordings allowed to study the urodynamic events occurring during the onset of DAC (ODAC). There was no use of mathematical modeling.

In a second step, the VBN mathematical micturition model (9, 10) was used to analyze the main part of the voiding phase and to test pathophysiological hypotheses able to explain the occurrence of DAC.

We recall that the VBN software, based on a global model of micturition, allows computing voiding curves: flow rate and detrusor pressure vs.

time for any set of characteristic parameters, such as detrusor contractility, urethral parameter (compressive or constrictive), effective abdominal pressure and degree of nervous excitation of both detrusor and urethral sphincter. The model and the software are fully described in references 9 and 10. When a good fitting between computed and recorded curves is obtained (quadratic error <1%), one can say that the evaluation of characteristic parameters is good. Gender, initial bladder volume and catheter geometrical obstruction are included in the model.

This study was conducted in accordance with the Declaration of Helsinki. According to the local practice of our Ethics Committee, there is no formal Institutional Review Board approval required for retrospective studies.

Statistical analysis

Data are presented as mean \pm SD and range. t test, analysis of variance (ANOVA) and the chi-square test were used as appropriate. All statistical results were considered significant at $p < 0.05$. Statistical analyses were performed using SAS, version 5.0 (SAS Institute, Inc., Cary, NC).

RESULTS

Population

DAC occurred in 5.7% of the studied population (60/1047) and was more frequent in women (53/714=7.4% vs. 7/333=2.1%) than in men.

For the group with DAC, mean age was 48.4 ± 16.4 y [21-84 years] for women and 53.0 ± 22.3 years [26-79 years] for men. Nine women (16.9%) and 2 men (28.6%) had a history of neurological disease.

Urgency-frequency syndrome was reported by 43/60 (71.6%) patients. Urodynamic DO was found for 32/60 (53.3%) patients (phasic DO for 19 patients, terminal DO for 13). Other urodynamic diagnoses were intrinsic sphincter deficiency for 13 women (11) and hypertonic sphincter for 8 women (12). BOO defined according with Abrams-Griffiths number (A-G>40) (13) for man and cut-off values $Q_{max} < 12$ mL/s and $p_{det.Qmax} > 25$ cmH₂O for woman (14) was found in 10/60 (16.6%) patients, respectively 1 (14.3%) man and 9 (16.9%) women.

Characteristics of urodynamic recordings

1- Two significant events were observed:

a- Onset of DAC (ODAC) did not occur for an empty bladder but during the latest part of flow and for a small bladder volume: $V_{ODAC} = 17.9 \pm 15.4$ mL (i.e. bladder collapse condition). The ratio voided volume at the onset of DAC vs. voided volume was 0.932 ± 0.060 . The flow rate at ODAC was $Q_{ODAC} = 7.3 \pm 5.7$ mL/s (while $Q_{max} = 17.4 \pm 7.7$ mL/s; $p < 0.0001$). Thus, DAC is characterized by an increase of detrusor pressure starting during the phase "return to continence" (15) of micturition; detrusor pressure reached its maximum after flow ceased in 45/60 (75.0%) of the studied DAC.

b- In 47 (78.3%) files, ODAC was an angular point on the pressure curve (i.e. a discontinuity of its slope with a change from negative to positive value) without disruption of the flow curve at this time (Figure-2A). Remark: the behavior of the flow curve was the same (no disruption) in absence of angular point.

2- Other urodynamics characteristics of voidings with DAC:

The pressure amplitude of DAC (p_{DAC}) was significantly higher than $p_{det.Qmax}$: 75.5 ± 51.7 vs. 34.8 ± 18.0 cmH₂O ($p < 0.0001$). Voided volume was 316 ± 145 mL and PVR 1.8 ± 5.4 mL.

Urethral behavior could not be assessed in 13 files because the transducer moved (leveling of both urethral and vesical pressures). No change of activity of urethra was observed in 21/47 (44.7%) files. An increase of rectal pressure was observed at ODAC in 8/60 (13.3%) files (20.1 ± 10.6 cmH₂O) but with concomitant increase of urethral pressure in only 1 file. No change of rectal activity has been observed in the 52 remaining files.

No change of activity of both urethra and rectum in 17/47 (36.2%) was observed during DAC.

3- Patterns of DAC

Assessment of patterns of DAC was made according with the description of Ruarte et al. (4). Among the more simple patterns, 30 bell shaped curves, 5 with small waves during the rising phase and 4 of high amplitude and tower shape were

found. Other patterns were more complicated: 11 irregular and prolonged, 6 with sharp peak in the rising phase, 4 two headed and 2 with fluctuations in the falling phase. Whatever the pattern of DAC (4), the increase in p_{det} was not associated to any change in flow rate.

Looking at the three files with 2 DAC, the 2 DAC exhibited a difference in shape, pressure and duration.

VBN analysis of voidings with DAC and simulation of pathophysiological conditions

For each patient the voiding phase until the onset of DAC was restored by VBN analysis (Figures 2 A-D). As in voiding without DAC, the fitting of recorded and computed curves in the terminal part was sometimes obtained with the addition of an effective abdominal pressure (which only acts on the bladder) in 30/60 (50%) files (Figures 2 C-D). That additional pressure clearly began before the onset of DAC (Figures 2 C-D).

After the onset of DAC, the flow curve was restored using the same VBN mechanical parameters (detrusor force and urethral parameter) as in the initial phase of voiding. But we did not succeed to restore the slope of the increase of p_{det} and its maximum value with these parameters.

Tested hypothesis to restore DAC were to introduce in the computation a strong sphincter excitation or a great (abnormal) detrusor force starting during that last phase of micturition. None of these hypotheses enabled us to restore the whole recorded curves (flow rate, detrusor pressure including DAC). More, the VBN model or any global model of micturition could not explain the brisk change in the slope of the detrusor pressure curve without any flow perturbation (Figure 2-A).

DISCUSSION

From analysis of urodynamic recordings of micturition with DAC, we propose the following definition of DAC: a detrusor pressure increase (higher than $10\text{cmH}_2\text{O}$ to avoid artifact of interpretation) during the phase "return to continence" of micturition (15) with PVR less than 30mL (to avoid confusion with reinforcement of detrusor pressure to empty the bladder).

If reference to a limit in detrusor pressure is usually mentioned in the studies on DAC (16) there is a lack of information about the PVR (4, 7).

In the studied adult population, prevalence of DAC is 5.7% which is in the range of the results of Webster et al. (17) and Cho et al. (7) who respectively found a prevalence of 2.0% and 8.1%.

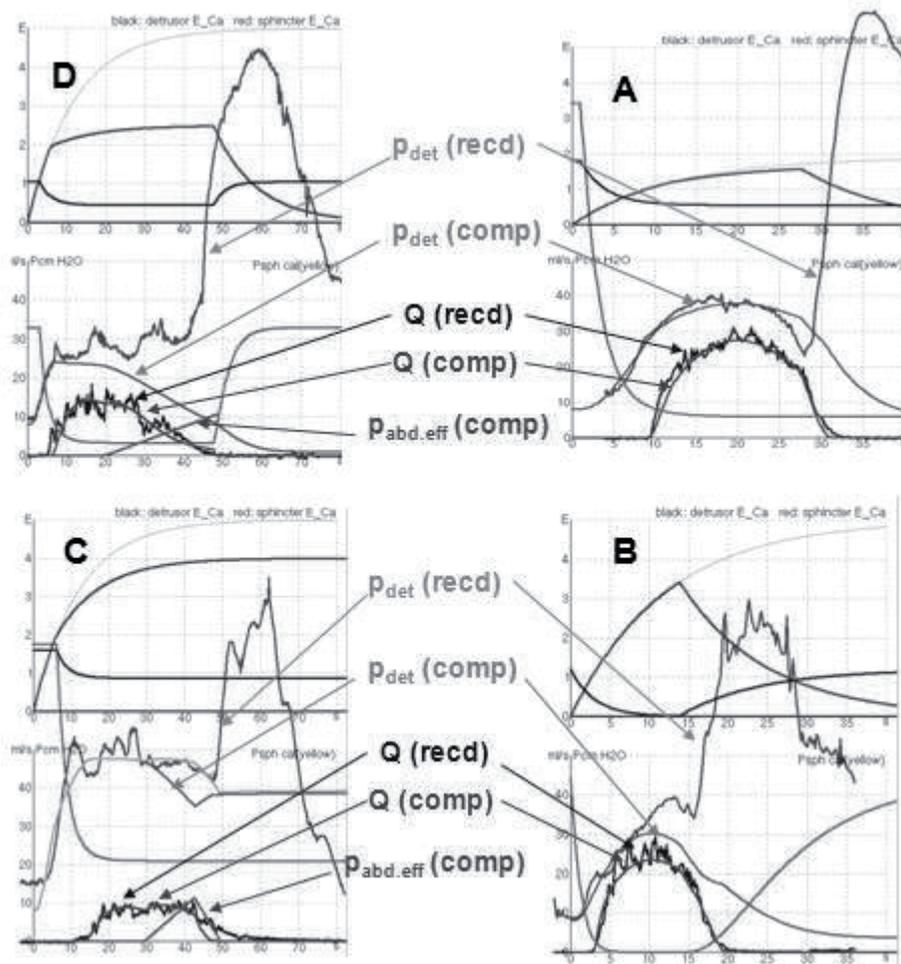
Significance of DAC is often discussed. In their study in children with non-neurogenic incontinence problems, Hoebeke et al. (18) concluded that DAC were indicators of detrusor instability. In adults, D'Amico et al. (6) found a statistical correlation among DAC and detrusor overactivity (DO) and overactive bladder (OAB), Cho et al. (7) concluded that DAC was significantly correlated to the presence of BOO. In our study (only adult individuals), urgency-frequency syndrome and urodynamic diagnosis of DO are the more frequent scenarios associated with DAC, BOO is less frequent than DO. But the liability of the correlations is weak.

For all the studied urodynamic recordings, flow and pressure curves before ODAC are successfully analyzed using the standard VBN model: the first part of voiding does not possess any characteristic that could predict the occurrence of DAC.

Close to emptying ($V < 40\text{mL}$) the bladder collapses and becomes flat or wrinkled while its shape is close to be spherical when filled. While at ODAC, the flow rate is still $0.42 Q_{max}$, the bladder volume is close to the condition of bladder collapse ($V_{ODAC} = 17.9 \pm 15.4\text{mL}$). We conclude that DAC is related in some way with collapse.

The flow rate before and after ODAC obeys the standard VBN model without any change in the parameters value; this is not the case of the detrusor pressure. Two kinds of explanations can be considered. Either the recorded pressure is not the real detrusor pressure, but is an artifact, or the voiding phase during DAC results from a brisk change of the voiding parameters. That second mechanism has been proposed by RL Vereecken (19) who performed needle EMG and concluded that DAC "is only the expression of a sphincter contraction interrupting an incomplete detrusor contraction", i.e. "a late dyssynergic urethral sphincter contraction".

Figure 2 - Four examples of VBN analysis of voidings followed by detrusor after-contraction (DAC) in female patients. The description only concerns the lower register (flow and pressures): Q (recd) (red)= recorded Q; Q (comp) (blue)= computed Q; p_{det} (recd) (fuchsia)= recorded p_{det} ; p_{det} (comp) (grey)=computed p_{det} ; $p_{abd.eff}$ (comp) (green) = computed $p_{abd.eff}$.



In the lower register, the yellow curve (A-D) without label is the computed sphincter pressure, and the cyan curve (C) the sum computed p_{det} plus computed $p_{abd.eff}$

A) A very impressive brisk increase of p_{det} at the onset of DAC.

C, D: additional effective abdominal pressure ($p_{abd.eff}$) which only acts on the bladder to achieve emptying. That additional pressure clearly begins before the onset of DAC.

In the upper register of each analysis: computed detrusor and sphincter excitations (for details about the VBN model, see references 9, 10).

It has been shown that large PVR or/and decreased maximum flow rate during intubated flow without DAC can be the consequence of a compression-like effect possibly due to an urethral reflex induced by the catheter in situ (20, 21).

So, the Vereecken hypothesis is realistic and we have tested it. Starting from the VBN analysis of the first part of a voiding with DAC,

a strong sphincter excitation is introduced in the computation at the time of ODAC. The result is an insufficient increase of detrusor pressure and arrest of the flow.

More generally, whatever the change in mechanical parameter tested (sphincter contraction or/and increased detrusor force, effective abdominal pressure only acting on the bladder) we

never succeeded to reproduce simultaneously the flow curve, both slope and amplitude of the recorded pressure and still less the discontinuity of the slope at ODAC. Thus the artifact hypothesis must be reconsidered.

Some authors asserted that DAC is a possible artifact related to recording during the final phase of micturition (22). In this study, despite a small number of patients, when DAC is observed during two successive intubated flows, DACs differ in all characteristics. Two papers advocated the hypothesis that the sensible membrane of a microtip transducer could be pressed against the wall of the empty bladder and falsely record an increase in detrusor pressure (22, 23). From our analysis we agree with these last assumptions.

A plausible explanation is that in most cases, at the end of voiding, the transducer shifts on a smooth bladder wall which does not create any pressure but that in some cases, the transducer is trapped in a bladder fold which leads to a local concentration of stresses and induces the observed increase in pressure. That last condition can explain the brisk change in the slope of p_{det} curve without any change in the flow curve. Why the DAC stops remains not understood. A hypothesis is that the stresses exerted by the bladder wall on the tip of the catheter are sufficient to expel the catheter from the bladder fold.

Limitations of our study include the fact that it is retrospective, and that EMG and video-urodynamics are not used. We don't routinely use EMG because needle EMG which gives the best information increases the invasiveness. Video-urodynamics would be informative if the location of the transducer could be clearly identified on the pictures; as it is not the case, mainly when the bladder is close to collapse condition, its usefulness remains discussed.

CONCLUSIONS

No urodynamic characteristic of the first part of voiding is an index of occurrence of DAC. The onset of DAC is a significant phenomenon which is linked with the bladder collapse. During a voiding with DAC, the flow rate obeys a global model of micturition while the detrusor pressure

obeys the model only until the onset of DAC; DAC pressure cannot be restored using this model. Thus, DAC appears to be more probably an artifact. The interaction of the transducer with the bladder wall leads 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.

ABBREVIATIONS

A-G = Abrams-Griffiths number

BOO = Bladder outlet obstruction

DAC = Detrusor after-contraction

DO = Detrusor overactivity

EMG = Electromyography

ICS = International Continence Society

ODAC = Onset of detrusor after-contraction

p_{det} , $p_{det.Qmax}$ = Detrusor pressure, detrusor pressure at maximum flow rate

p_{DAC} = Pressure amplitude of detrusor after-contraction

PVR = Post void residual volume

Q , Q_{max} = Flow rate, maximum flow rate

US = Ultra sound

VBN = Valentini-Besson-Nelson (authors name)

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CONFLICT OF INTEREST

None declared.

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EDITORIAL COMMENT

DAC is not a common finding in urodynamics evaluation, and is a phenomenon with no clear definition even in the ICS.

In ancient times it was a common belief that urodynamics must reproduce the patient's complaint to be of any value. Following this statement, we hardly try to find involuntary detrusor contractions in patients with reliable reports of urge incontinence. We did not had the clear knowledge that, for instance, more than half patients with OAB would not show involuntary bladder contraction during cystometry.

By that time, DAC worth as something to report in a patient with urgency and a normal cystometry, and this finding was also associated with BOO.

In this paper, the authors analyzed 60 urodynamic evaluations with DAC, and although DO were associated with this finding, they did not find a strong clinical significance for this finding.

Is DAC a true detrusor contraction or a late dyssynergic urethral sphincter contraction or an artifact? It cans be everything, but I do agree that this phenomena in more an artifact. The proposition for further studies with EMG and video urodynamics to clarify our doubts is worthy, but its clinical usefulness perhaps will be little.

From this paper, we can give to DAC its true value: weak clinical significance.

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