Original Article

ISSN (0): 2347-3398; ISSN (P): 2277-7253

Detrusor afterContraction - Is it Pathological

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Abstract

Background: Aim: Detrusor after-contraction (DAC) is seen in many patients undergoing urodynamic studies. There are no established criteria to define DAC. There is very little data available to suggest whether it is a normal physiological or a pathological finding. The aim of our study was to find the practical significance of DAC. **Subjects and Methods:** Retrospectively study by collecting data of UDS from period of October 2008 to September 2013. Criteria for DAC were increase of detrusor pressure more than 10 cm H2O after the cessation of urine flow. **Results:** 540 patients underwent urodynamic study from October 2008 to September 2013. Out of 368 were males and 172 were females. Overall 28 patients of 540 (5.2%) showed DAC during urodynamic assessment. DAC was more frequent in women (16) than men (12). Mean age of women with DAC was 48 ±7.8 years (Range 17-59 years). Mean age of men with DAC was 51±13.3 years (22-71 years). Of the 16 women, 8 had detrusor overactivity, 4 had bladder outlet obstruction (BOO), 2 had detrusor sphincter dyssynergia (DSD) and 2 had other normal bladder parameters. Of the 12 men, 7 had bladder outlet obstruction, 3 had detrusor overactivity and 2 had small capacity and poorly compliant bladder. The pressure amplitude of DAC (pDAC) was significantly higher than pdetQmax: 79.5±41.1 vs. 31.8±13.0 cm of H2O (p<0.0002). Voided volume was 280±105mL and PVR 19±6.3mL. Volume at onset of DAC (VODAC) was 20 ml ± 4.5ml. The ratio of voided volume at the onset of DAC and total voided volume was 0.987 ± 0.37. The flow rate at onset of DAC was 6.9 ± 4.3 ml/sec while Qmax was 18.7± 6.8 ml/sec (p=0.0002). **Conclusion:** There is variability in prevalence of DAC and no association or correlation of DAC with various bladder pathologies with statistical significance. It is not possible to give an explanation to this urodynamic finding. Further prospective studies with adequate sample sizes are needed to give the exact meaning and clinical significance of this urodynamic finding.

Keywords: Detrusor, Urinary Bladder.

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Received: January 2018
Accepted: February 2018

Introduction

A detrusor after contraction (DAC) (urodynamic phenomena) - Is it an artefact or clinically significant entity? Although first description of DAC dates to 1987, very few investigators have studies this phenomenon. Prevalence of DAC varies in the literature. One of the large observational study done recently has reported it to be 13.9%.[1] It is frequently observed in children but also seen in adults. Detrusor after contraction is defined as increase in Pdet after cessation of voiding detrusor contraction in the absence of urinary flow when bladder is nearly empty[Figure 1].[2] However, magnitude of Pdet increase is not standardized and varies in different studies ranging from 5 cm of water to 20 cm of water. Indeed, ICS does not mention DAC in its recent standardization of terminology in urodynamic report.[3,4]

Various speculations have been made in the past regarding it's pathophysiology. It has been found to be associated with detrusor overactivity, Bladder outlet obstruction, detrusor sphincter dyssynergia and, otherwise urodynamically normal bladder.[4,5] Few reports have described DAC as artefact occurring solely as a consequence of urodynamic procedure and without any diagnostic value.[6-9] Most of the studies have used conventional urodynamics to investigate DAC whereas few have used ambulatory urodynamics to mimic and reproduce physiological conditions. Therefore, in the absence of standardize definition, poor understanding of pathophysiology and unclear clinical relevance, we think that DAC remains an enigmatic entity in neurophysiology of lower urinary tract which require further research.

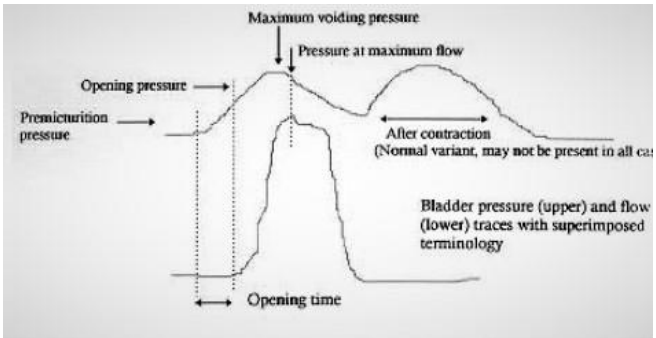

Figure 1: Detrusor after contraction (Arrow)

The aim of our study is to find the clinical association of DAC with various bladder pathologies and to establish its practical importance.

Subjects and Methods

After approval from institutional research review board we retrospectively reviewed data and urodynamic traces of patient who underwent UDS at Department of Urology, Yenepoya Medical College and Hospital and Institute of Nephro Urology, Bangalore, irrespective of the cause for which UDS was done during the time period from October 2008 to September 2013.

Criteria of DAC: In the absence of standardize criteria, following criteria selected, as this is the most widely used definition in published reports10: PVR should be <30mL to avoid a possible resumption of the voiding process and increase of detrusor pressure (pdet)>10cmH2O to avoid error of reading. To use mathematical modelling, additional conditions were necessary for voiding phase before DAC: Qmax>2mL/s, voided volume ≥100mL, continuous flow curves without predominant abdominal straining.

Study protocol: Before urodynamic study, detailed history of the patients with special attention to lower urinary tract symptoms noted. All patients are asked to complete 3 days of voiding diary. Urodynamic study was carried out using a standardized technique in line with the ICS standardization report on urodynamics.[11] Patient is asked to void and uroflowmetry recordings noted. Two 6F infant feeding tubes inserted per urethra and externally connected to filling channel and pressure transducer. Intra-rectal balloon catheter filled with 2 ml saline inserted for measurement of abdominal pressure according to good practice urodynamic guidelines.12 EMG leads attached to perineum and thigh region. Pressure transducers were zeroed to atmospheric pressure at the upper edge of the symphysis pubis. Cystometry was performed with the patient in the seated position with urethral catheter perfused with saline at room temperature, using a filling rate of 50mL/ min. Volume and pressure changes during filling phase recorded. After completion of filling phase patients were asked to void and flow rate, pressure & volume changes recorded. Post void residual volumes (PVR) were measured by ultrasound (US) using a Bladder-Scan.

Urodynamic data from 540 patients collected. DAC was found in the 28 patients.

MMS software was used to compute pressure and volume recording during urodynamic study. The urodynamic records were analyzed according to definitions and units as per ICS standards.[11] This study was conducted in accordance with the Declaration of Helsinki

Statistical Analysis

Data are presented as mean ± 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 SPSS, version 22.0

Results

540 patients underwent urodynamic study from October 2008 to September 2013. Out of 368 were males and 172 were females. Overall 28 patients of 540 (5.2%) showed DAC during urodynamic assessment. DAC was more frequent in women (16) than men (12). Mean age of women with DAC was 48 ±7.8 years (Range 17-59 years). Mean age of men with DAC was 51±13.3 years (22-71 years) [Table1]. All patients were symptomatic for frequency, urgency, incontinence or combination of above-mentioned complaints [Table2].

Of the 16 women, 8 had detrusor overactivity, 4 had bladder outlet obstruction (BOO), 2 had detrusor sphincter dyssynergia (DSD) and 2 had other normal bladder parameters. Of the 12 men, 7 had bladder outlet obstruction, 3 had detrusor overactivity and 2 had small capacity and poorly compliant bladder. Out of 28 patients with DAC, 2 males showed small functional capacity on pre procedure bladder diary recording and 1 patient had history of prior surgery for Benign prostatic hyperplasia [Table3].

Table 1: Demographics of study population

Detrusor after contraction- Total patients- 28/540 (5.1%)		
	Male	**Female**
Number	12(42%)	16 (58%)
Prevalence	3.2%	9.3%
Age	51±13.3 years (22-71 years)	48 ±7.8 years (Range 17-59 years)
Comorbidities		
Diabetes	6	3
Hypertension	2	1
Neurologic disorder	1	-

Table 2: Presenting symptoms.

Detrusor after contraction- Total patients- 28/540 (5.1%)		
Presenting Symptoms		
Frequency	6	12
Nocturia	2	1
Urgency	4	7
Urge incontinence	2	3

Table 3: Urodynamic findings associated with detrusor after contraction

Urodynamic findings	**Detrusor after contraction- Total patients- 28/540 (5.1%)**	
	Female	**Male**
	16 (58%)	**12(42%)**
Detrusor overactivity	8 (66%)	3 (18.75%)
Bladder outlet obstruction	4(34%)	7(43.75%)
Detrusor sphincter dyssynergia	2(16.6%)	-
Small capacity and poorly compliant bladder	2(16.6%)	-
Normal parameter	-	2(12.5%)

BOO defined according with Abrams-Griffiths number (A-G>40) (13) for man and cut-off values Qmax< 12mL/s and PdetQmax>25cmH2O for woman. The pressure amplitude

of DAC (pDAC) was significantly higher than pdetQmax: 79.5±41.1 vs. 31.8±13.0 cm of H2O (p<0.0002). Voided volume was 280±105mL and PVR 19±6.3mL. No change of activity of urethra was observed in 12/28 (42.85%) patients. An increase of rectal pressure was observed at ODAC in 4/28 (14.3%) patients (18.1±9.6cmH2O) indicating some straining component associated with DAC (Table 4). Both patients with DSD showed rise in rectal pressure. DAC is characterized by an increase of detrusor pressure starting during the phase "return to continence" of micturition, detrusor pressure reached its maximum after flow ceased in all cases.

Onset of DAC did not occur after bladder is empty rather it started in later part of the voiding cycle when bladder was near empty. Volume at onset of DAC (VODAC) was 20 ml ± 4.5ml. The ratio of voided volume at the onset of DAC and total voided volume was 0.987 ± 0.37. The flow rate at onset of DAC was 6.9 ± 4.3 ml/sec while Qmax was 18.7± 6.8 ml/sec (p=0.0002) [Table4]

DAC is characterized by an increase of detrusor pressure starting during the phase "return to continence" of micturition, detrusor pressure reached its maximum after flow ceased in all cases. The pressure amplitude of DAC (pDAC) was significantly higher than PdetQmax in 26 cases. Except for two cases having small bladder capacity with low compliance all other case had voided volume greater than 280 ml. An increase in rectal pressure was seen in 4 cases indicating some straining component associated with DAC and this was seen in cases with DSD.

Patterns of DAC were assessed according with the description of Ruarteet al.16 were bell shaped curves, 5 curves were with sharp peak, 1curve showed fluctuations in the falling phase. Increase in Pdet was not associated with change in flow rate irrespective of pattern of curve

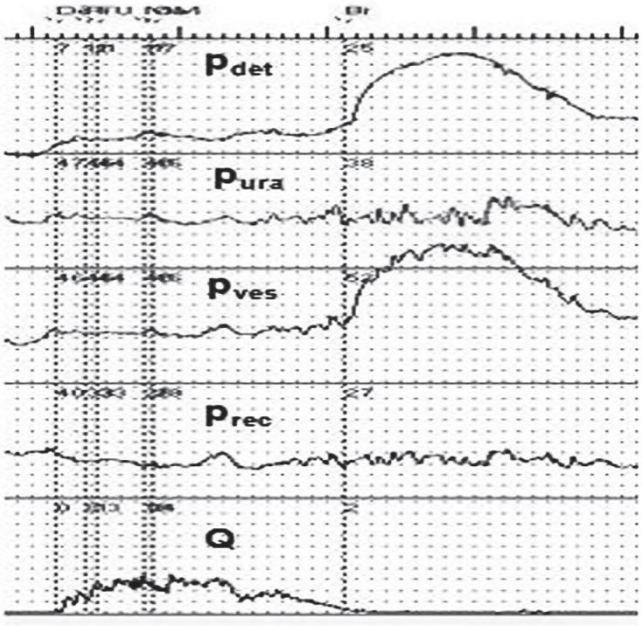

Figure 2: Recording of Detrusor after-contraction (DAC) observed during intubated flow of a male patient. From top to bottom: detrusor pressure (pdet), urethral pressure (pura), vesical pressure (pves), rectal pressure (prec) and flow rate (Q). Note the high amplitude of DAC compared with pdet during voiding

Table 4: Urodynamic parameters in patients with DAC

pDAC	79.5±41.1 cm of H2O	P<0.0002
PdetQmax	31.8±13.0 cm of H2O	
Voided volume	280±105mL	
PVR	19±6.3ml	
VODAC	20 ml ± 4.5ml	
voided volume at the onset of DAC /total voided volume	0.987 ± 0.37	
QODAC	6.9 ± 4.3 ml/sec	p=0.0002
Qmax	18.7± 6.8 ml/sec	

pDAC- Pressure amplitude of detrusor after contraction. PdetQmax- Maximum detrusor pressure during voiding. PVR post void residue, VODAC- Volume at the onset of detrusor after contraction, QODAC- Flow rate at the onset of detrusor after contraction (DAC)

Discussion

Although there have been many definitions for DAC, it is most commonly reported as any increase in Pdet after cessation of urinary flow. While some reports have reported any magnitude of Pdet rise (typically 5–20 cmH2O), others have insisted on an increase of Pdet of at least twice the level of maximum Pdet during the voiding phase.[3,5–7] Variation in reported prevalence can be attributed to the different definitions used by investigators. For example, the last of these definitions would have required a mean aftercontraction of 60 cmH2O simply to be classified as DAC in our study.

In the studied adult population, prevalence of DAC was 5.1%. Various other studies by Webster et al (2.0%),[2] Cho et al (8.1%),[3]& Françoise A et al (5.7%),[4] have also shown similar results. In males, prevalence was 3.2% whereas in females it was 9.1%. Recently, the largest observational study of symptomatic women reporting 13.9% prevalence which is similar to finding in our study.[3-8,13]

Table 5: Conditions associated with DAC

Study	Condition associated with DAC	Comment
Hoebeke et al ,[5]	Detrusor instability	Children with non-neurogenic incontinence
D'Amico et al ,[14]	Detrusor overactivity (DO) and overactive bladder (OAB)	Adult population
Cho et al ,[3]	Bladder outlet obstruction (BOO)	Adult population, significant correlation
Françoise A. Valentini,[15]	Urgency frequency syndrome detrusor over activity, BOO	Adult population but weak correlation
Our study	Detrusor overactivity, BOO, Detrusor sphincter dyssynergia, normal urinary bladder	Adult population

Clinical Significance of DAC is still unclear. In Previous

studies, DAC is found to be associated with non-neurogenic incontinence problem, detrusor instability, detrusor overactivity, urgency frequency syndrome and Bladder outlet obstruction.[3,5,14,15] In Our study, most common urodynamic diagnosis was Detrusor overactivity (66%) in females and bladder outlet obstruction (43.75%) in males. Other associated conditions were Detrusor sphincter dyssynergia. In 12.5 % of males it was also seen with normal urodynamic results. [Table5]

For all the studied urodynamic recordings, flow and pressure curves before ODAC are successfully analysed using the MMS software. In our study the first part of voiding does not possess any characteristic that could predict the occurrence of DAC.

When urinary bladder is near empty it is collapse and become wrinkled while it's shape is close to spherical when filled. In our study ODAC, the flow rate is still 0.3 times Qmax, the bladder volume is close to the condition of bladder collapse (VODAC=20 ml ± 4.5ml). We conclude that DAC is related in some way with bladder decompression with voiding. Françoise A. Valentini also observed similar findings in their study and speculated that occurrence of DAC may be related to the bladder collapse.[15] However, exact mechanism is yet to be elucidated.

The flow rate before and after ODAC did not show any significant change in our study but values of detrusor pressure did show changes. Two possible explanation for this could be either the recorded pressure is not the real detrusor pressure but is an artefact or the voiding phase during DAC results from a brisk change of the voiding parameters. RL Vereecken who performed needle EMG and concluded that DAC "is only the expression of a sphincter contraction interrupting an incomplete detrusor contraction",[4] i.e. "a late dyssynergic urethral sphincter contraction".

Previous studied have shown that significant PVR or/and subnormal maximum flow rate during intubated flow without DAC can be the consequence of a compression-like effect possibly due to a urethral reflex induced by the catheter in situ.[17,18] Françoise A. Valentini et al tested RL Vereecken hypothesis using VBN model and found that when strong sphincter excitation is introduced at the onset of DAC, an insufficient increase of detrusor pressure and arrest of the flow was observed. However, in our study we did not observed any changes in urethral pressure or EMG recording and corresponding changes in detrusor pressure and flow rate to support Vereecken hypothesis. Hence, we think that artefact hypothesis should be reconsidered, and it needs further investigation. 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.[8,18] In our study we observed that DAC differed in all characteristics when observed during two successive intubated flow. Hence, we agree with last assumption.

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 by altering local environment. This may produce brisk change in detrusor pressure as evident by change in the slope of Pdet curve. However, this change in local pressure may not be enough to produce change in the flow rate. 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 thus preventing further propagation of stresses in rest of the bladder which may have resulted into the changes in flow rate.

The limitation of our study includes the fact that it was a retrospective analysis with comparatively small sample size. We did not use video urodynamics could have provided better understanding of the changes in various parameters at the time of DAC. We believe that ADC needs further investigation to determine its significance.

Conclusion

Detrusor after contraction is seen in urodynamics as an isolated phenomenon at the end of voiding phase. There is variability in prevalence of DAC and no association or correlation of DAC with various bladder pathologies with statistical significance. It is not possible to give an explanation to this urodynamic finding. Further prospective studies with adequate sample sizes are needed to give the exact meaning and clinical significance of this urodynamic finding.

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
pdet, pdet.Qmax = Detrusor pressure, detrusor pressure at maximum flow rate
pDAC = Pressure amplitude of detrusor after-contraction
PVR = Post void residual volume
Q,Qmax = Flow rate, maximum flow rate

References

1. Rodrigues P, Hering F, Campagnari JC. (2014) Urodynamic after contraction waves: a large observational study in an adult female population and correlation with bladder and ureter emptying functions in women. Urol. Int. 2014; 93: 431–6.
2. Pesce F, Rubilotta E. Detrusor after-contraction: is this important? Curr. Urol. Rep. 2004; 5: 353–8.
3. Cho SY, Yi JS, Park JH, Jeong MS, Oh SJ. Detrusor after-contraction is associated with bladder outlet obstruction. Neurourol. Urodyn.

2011; 30: 1361–5.
4. Vereecken RL. The after-contraction: a true detrusor contraction or a late dyssynergic urethral sphincter contraction? BJU Int. 2000; 85: 246–8.
5. Hoebeke PB, Van Gool JD, Van Laecke E, VandeWalle J, Plancke H. The after-contraction in paediatricurodynamics. Br. J. Urol. 1996; 78: 780–2.
6. Webster GD, Koefoot RB, Zakrzewski CJ, Todd S. The after-contraction in urodynamic micturition studies. Neurourol. Urodyn. 1983; 2: 213–8
7. Wadie BS, Elsaadany MM. Detrusor after contractions in men with lower urinary tract symptoms: myth or reality? Arab J. Urol. 2013; 11: 336–9
8. Mutchnik SE, Sukin S, Boone TB. Case studies in voiding dysfunction. Contemp. Urol. Arch. 1999; 11: 110–3
9. McLellan A, Cardozo L. Urodynamic techniques. Int. Urogynecol. J. 2011; 12: 266-70
10. Abrams P, Blaivas JG, Stanton SL, Andersen JT. The standardisation of terminology of lower urinary tract function. The International Continence Society Committee on Standardisation of Terminology. Scand J UrolNephrol Suppl. 1988; 114:5-19
11. Van Waalwijk van Doorn E, Anders K, Khullar V et al. Standardisation of ambulatory urodynamic monitoring: report of the Standardisation Sub-Committee of the International Continence Society for Ambulatory Urodynamic Studies. Neurourol. Urodyn. 2000; 19: 113–25
12. Schäfer W, Abrams P, Liao L, Mattiasson A, Pesce F, Spangberg A, et al. Good urodynamic practices: uroflowmetry, filling cystometry, and pressure-flow studies. NeurourolUrodyn. 2002;21:261-74
13. Rodrigues P, Hering F, Campagnari JC. (2014) Urodynamic after contraction waves: a large observational study in an adult female population and correlation with bladder and ureter emptying functions in women. Urol. Int. 2014; 93: 431–6
14. D'Amico A, Pesce F, Rubilotta E, Sarti A, Curti P, Artibani W. Correlation between detrusor after-contraction and overactive bladder syndrome, detrusor overactivity and post micturition dribble. NeurourolUrodyn 2003; 22:457-8.
15. Detrusor after-contraction: a new insight Françoise A. Valentini 1,2, Brigitte G. Marti 3, GilberteRobain 1,2, Pierre P. Nelson 1 IBJU May - June, 2015Vol. 41 (3): 527-534, doi: 10.1590/S1677-5538.
16. Ruarte AC, Podestá ML, Medel R. Detrusor aftercontractions in children with normal urinary tracts. BJU Int. 2002;90:286-93.
17. Valentini F, Marti B, Robain G, Nelson P. Differences between the data from free flow and intubated flow in women with urinary incontinence. What do they mean? NeurourolUrodyn. 2008;27:297-300
18. Valentini FA, Robain G, Hennebelle DS, Nelson PP. Decreased maximum flow rate during intubated flow is not only due to urethral catheter in situ. IntUrogynecol J. 2013;24:461-7.

How to cite this article: Shivalingaiah M, Souza ND, Tamboli N. Detrusor after Contraction - Is it Pathological. Asian J. Med. Res. 2018;7(1):MC06-MC10.
DOI: dx.doi.org/10.21276/ajmr.2018.7.1.MC2

Source of Support: Nil, **Conflict of Interest:** None declared.