VOIDING DYSFUNCTION/FEMALE UROLOGY
MINI-REVIEW

The challenges in the diagnosis of detrusor underactivity in clinical practice: A mini-review

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ABBRVIATIONS
BCI, bladder contractility index;
CUR, chronic urinary retention;
DHIC, detrusor hyperactivity with impaired contractility;
DO, detrusor overactivity

Abstract
Objective: To review the current definitions, terminology, epidemiology and aetiology of detrusor underactivity (DU), with specific attention to the diagnostic criteria in use. In addition, we address the relation and the overlap between DU and bladder outlet obstruction (BOO). In this mini-review, we hope to help identify DU patients and facilitate structured clinical evaluation and research.

Methods: We searched the English literature using ScienceDirect and PubMed for relevant articles. We used the following terms: ‘detrusor underactivity’, ‘underactive bladder’, ‘post voiding residual’, ‘post micturition residual’, ‘acontractile bladder’, ‘detrusor failure’, and ‘detrusor areflexia’.

Result: DU is one of the most common conditions causing lower urinary tract symptoms (LUTS). Unfortunately, it is also the most poorly understood bladder dysfunction with scant research. To our knowledge there is no clear definition and no non-invasive method to characterise this important clinical condition. DU may result from the normal ageing process; however, it has multiple aetiologies including neurogenic and myogenic dysfunction. In many cases the symptoms of DU are similar to those of BOO and it usually requires invasive urodynamic study (UDS) for diagnosis to differentiate the two diagnoses. A number of diagnostic tests may be used including: UDS testing, the Schafer pressure/flow nomogram, linear passive urethral resistance relation, Watts factor, and the bladder contractility index. Of these, UDS testing is the most practical as it determines both the maximum urinary
Introduction

Detrusor underactivity (DU) is one of the most common conditions causing LUTS, yet it is poorly understood and therefore remains a topic of ongoing research [1,2]. Various terminologies have previously been used to describe DU, such as underactive bladder [3], impaired detrusor contractility [4], bladder failure, bladder decompensation, hypotonic bladder [5], detrusor areflexia, and detrusor failure [6]. The variety of terminology and various implied definitions reflect a lack of consensus. In 2002, the International Continence Society (ICS) defined DU as ‘a contraction of reduced strength and/or duration, resulting in prolonged bladder emptying and/or failure to achieve complete bladder emptying within a normal time span’ [2]. This definition is based mainly on urodynamic study (UDS) findings, not on symptoms. Whilst a definition based on the clinical syndrome of the condition may aid in the understanding of DU, the symptoms of DU are similar to those of BOO, such as weak or interrupted stream, and significant post-void residual urine (PVR) volume, which cannot be differentiated except by UDS.

Methods

We searched the literature for relevant articles from January 1972 to January 2016 using the electronic English databases ScienceDirect and PubMed. We used the terms: ‘detrusor underactivity’, ‘underactive bladder’, ‘post voiding residual’, ‘post micturition residual’, ‘acontractile bladder’, ‘detrusor failure’, and ‘detrusor areflexia’. Studies that were not in English, case reports, or those not including any of the following: definition, terminology, epidemiology, aetiology of the DU, and the overlap between DU and BOO were excluded.

Results

The primary search identified 258 articles. After applying the above exclusion criteria, we included 33 articles. We found that the ICS definition does not include symptomatology. DU may be defined as ‘decrease in sensation of the micturition desire that may be accompanied by nocturia and frequency with decrease in voiding volume associated by incomplete bladder empting and incontinence that may increase at night’ [7]. Including the symptom complex, as in definition of overactive bladder (OAB), may improve the diagnosis and treatment of DU. However, unfortunately, symptoms alone cannot be used for diagnosis in clinical practice due to the overlap between the symptoms of DU and BOO. The only practical method of differentiating these two conditions is UDS, an invasive technique. To our knowledge there is no clear definition or non-invasive method to resolve this important clinical condition [8]. Clinicians require multiple data for diagnosis such as the strength of detrusor contraction, whether detrusor contraction is sustained, and the presence or absence of incomplete bladder emptying. In contrast, clinicians may begin first-line management in the presence of OAB symptoms without confirming the diagnosis [1].

Epidemiology

Many of the clinical studies for patients with non-neurogenic LUTS showed that DU was present in 9–
28% of men aged < 50 years increasing to 48% in men aged > 70 years. In elderly women, prevalence ranges from 12% to 45%. However, estimating the epidemiology of DU is difficult, as it is largely based on clinical picture rather than UDS findings [1,9].

Aetiology

DU cannot only be explained as an ageing process, but also occurs in response to multiple aetiologies [10], including both myogenic and neurogenic factors. Myogenic causes include changes in the ultrastructure of myocytes and gap junctions that inhibit detrusor contraction and deposition of collagen between muscle bundles, similar to changes mostly occurring in BOO where detrusor decompensation occurs [11,12]. Neurological factors also play an important role in the pathogenesis of DU, as any interruption in afferent or efferent supply from and to the detrusor muscle leads to major DU [13–15]. Many neurological diseases can lead to lower motor neurone symptoms resulting in DU such as diabetic cystopathy, Parkinsonism, multisystem atrophy, multiple sclerosis, Guillain–Barre syndrome, spinal lumbar disc hernia, and spinal cord injury. Further, it may occur as a result of iatrogenic neurological injury such as in pelvic surgery, radical prostatectomy, abdominoperineal resection, and radical hysterectomy [2].

Clinical picture

Clinically patients with DU present complaining of LUTS, hesitancy, difficulty, interrupted stream, weak stream, post-micturition dribbling, and sensation of incomplete voiding with chronic urinary retention (CUR) and an elevated PVR, generally <300 mL [2,16]. The ICS definition does not specify PVR volumes in CUR. Patients may present in a late stage with complications of CUR such as nocturnal enuresis, overflow incontinence, recurrent UTI, stone formation, and reflux with azotaemia [2]. As stated previously, presenting symptoms are similar to the symptoms of BOO and it may be difficult to differentiate the two conditions without invasive UDS testing.

Notably, during UDS, DU should be differentiated from Alzheimer dementia. In Alzheimer dementia, the patient may be unable to follow the command to contract the detrusor muscle, although proper function is not compromised. This is a common problem in elderly patients [17].

DU may also be associated with detrusor overactivity (DO), known as detrusor hyperactivity with impaired contractility (DHIC), a condition that mostly occurs in elderly patients where the patient complains of both storage and voiding symptoms. In this condition, on UDS, detrusor contractions occur during filling and there is also failure of effective detrusor voiding contrac-

tions [18]. Mahdy and Ghoniem [19] proposed a change to the old terminology (DHIC) to DO with DU (DODU), to better describe and diagnose this condition.

Classification

According to UDS testing, DU can be classified in men into three categories: mild, moderate, and severe. This is based mainly on detrusor pressure at the maximum urinary flow rate ($P_{\text{det}}@Q_{\text{max}}$) and degree of emptying values of $>40 \text{ cmH}_2\text{O}$ are normal, 30–40 cmH$_2$O are mild DU, 20–30 cmH$_2$O are moderate DU, and $<20 \text{ cmH}_2\text{O}$ are severe DU. Classification is highly relevant for choosing the method of treatment and to evaluate the follow-up of the patients [8]. In women, an exact definition or classification is even less clear and mainly relates to the degree of bladder emptying, i.e., residual urine volume. The values of DU equal those of the lower limit of the normal range, which have been estimated by studies done on adult males, who underwent bladder outlet operations [20]. Other studies have also been done on female patients [21] and healthy young adults as well [22].

Diagnosis

UDS testing measures the relationship between $P_{\text{det}}$ and the expulsion of urine through the urethra [23]. When the flow increases the urethral pressure decreases, reaching its lower limit at maximal flow (i.e., lowest outlet resistance). Bladder strength can be estimated by measuring the isovolumetric $P_{\text{det}}$, which is the highest pressure value reached when outflow is stopped during voiding (i.e., highest outlet resistance) [1]. UDS testing is the only practical method to diagnose DU [8], as it estimates the sustainability of contraction and strength of detrusor muscle by measuring $Q_{\text{max}}$ and $P_{\text{det}}$ relative to $Q_{\text{max}}$. A $Q_{\text{max}}$ of $<15 \text{ mL/s}$ and $P_{\text{det}}@Q_{\text{max}}$ of $<40 \text{ cmH}_2\text{O}$ may indicate DU; however, ‘normal’ ranges are not widely accepted. UDS testing is advantageous because it is simple to use, but it may still underestimate detrusor strength.

Other methods may be used to diagnose DU by measuring the isovolumetric detrusor contraction. The practical UDS methods in evaluating detrusor function are: the Schaefer pressure/flow nomogram or linear passive urethral resistance relation (LinPURR) to evaluate and grade BOO, Watts factor, and the bladder contractility index (BCI) for detrusor work.

The Schaefer LinPURR curve was developed to evaluate detrusor function relative to BOO. The urethral resistance relation is a straight line that characterises the relationship between $P_{\text{det}}$ and flow. It consists of seven zones and indicates increasing grades of obstruction on a scale of 0–6. Grades 0 and 1 indicate no obstruction; grade 2, equivocal or mild obstruction;
grades 3–6, increasing severity of obstruction [24]. Each of these data points indicates the degree of obstruction and the detrusor strength; it is plotted according to the $Q_{\text{max}}$, $P_{\text{det}}$, and detrusor strength on the LinPURR diagram. This may be particularly useful in differentiating patients with obstructive patterns from those with true DU [24].

The Watts factor is a mathematical analysis of UDS data, measuring the detrusor power per bladder surface unit. It measures the isovolumetric $P_{\text{det}}$ independent of BOO [25], but not the sustainability of contraction or BOO [26]. The equation for the Watts Factor is: $\text{Watts factor} = \frac{(P_{\text{det}} + a)(V_{\text{det}} + b) - ab}{2\pi}$, where $V_{\text{det}}$ is the detrusor shortening velocity and $a$ and $b$ are constants ($a = 25 \text{ cmH}_2\text{O}$, $b = 6 \text{ mm/s}$). As $V_{\text{det}}$ and $P_{\text{det}}$ vary throughout the voiding cycle, the Watts factor changes as well. There are no widely accepted ‘normal’ values; however, a Watts factor of $7 \text{ W/m}^2$ may indicate DU [1].

BCI can be calculated by the following formula: $\text{BCI} = P_{\text{det}}@Q_{\text{max}} + 5Q_{\text{max}}$. A BCI of $>150$ is strong, BCI 100–150 is normal, and BCI $<100$ is weak, indicated on a bladder contractility nomogram [24]. The BCI is fast and easy to use, and therefore amenable to clinical settings. However, the index does not account for BOO and therefore cannot assist in identifying between these two independent or concomitant conditions.

The three measurements are highly correlated. Still, in our opinion, multichannel UDS testing remains the most practical to diagnose DU.

Finally, evaluation of sensation is very important, as it reflects the intact afferent supply. Sensation is estimated by asking the patient to inform about the first sensation, first desire and strong desire during the filling phase in UDS [28]. It is usually correlated with DU; however, because of its subjectivity there has not been much research to characterise impaired bladder sensation.

**Points of interest**

Although DU can result from advanced BOO, the relationship between the two conditions is not usually straightforward. However, not all patients with BOO develop DU and not all patients with BOO have DU [29]. The outcome of prostate surgical procedures in those patients with DU is still controversial. Some studies suggest that there are no favourable outcomes for those with DU [30], whilst other studies suggest that patients with combined DU and BOO will benefit from prostate surgical procedures [31]. Other studies go to the extremes, where they address that patients with DU will benefit from prostate surgical procedures even when they are not associated with BOO [32]. Finally, in a retrospective analysis of 4272 UDS, it was concluded that underactive bladder is not a symptom complex and DU should be based on UDS diagnosis [33].

**Conclusion**

DU is a frequent finding and commonly contributes to LUTS. There are many challenges that make it difficult to accurately diagnose based on the clinical presentation alone, as it has the same clinical appearance as advanced BOO. Thus, it is not easy to screen for DU based on symptoms alone. UDS testing is the cornerstone of DU diagnosis. Moreover, it can aid in the classification and follow-up of DU. For many years there was no consensus on the definition of DU. In 2002, the ICS defined DU according UDS findings. Although UDS testing is invasive, it is simple, reliable, and practical. There has been recent renewed interest in DU with the necessary basic research to shed light on the pathophysiology of this common condition. We hope more research is done to find other less invasive and more practical methods for diagnosis. This will help to advance the understanding of DU and hopefully treatment of this common urological condition.

**Conflict of interest**

None declared.

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None.

**References**

[1] Osman NI, Chapple CR, Abrams P, Dmochowski R, Haab F, Nitti V, et al. Detrusor underactivity and the underactive bladder: a new clinical entity? a review of current terminology, definitions, epidemiology, aetiology, and diagnosis. *Eur Urol* 2014;65:389–98.

[2] Abrams P, Cardozo L, Fall M, Griffiths D, Rosier P, Ulmsten U, et al. The standardisation of terminology of lower urinary tract function: report from the standardisation sub-committee of the International Continence Society. *Am J Obstet Gynecol* 2002;187:116–26.

[3] Anderson JB, Grant JB. Postoperative retention of urine: a prospective urodynamic study. *BMJ* 1991;302:894–6.

[4] Madjar S, Appell RA. Impaired detrusor contractility: anything new? *Curr Urol Rep* 2002;3:373–7.

[5] Alexander S, Rowan D. Treatment of patients with hypotonic bladder by radio-implant. *Br J Surg* 1972;59:302.

[6] Kirby RS, Fowler C, Gilpin SA, Holly E, Milroy EJ, Gosling JA, et al. Non-obstructive detrusor failure. A urodynamic, electromyo graphic, neurohistochemical and autonomic study. *Br J Urol* 1983;55:652–9.

[7] Chancellor MB, Kaufman J. Case for pharmacotherapy development for underactive bladder. *Urology* 2008;72:966–7.

[8] Yu Z, Li J, Li Z, Hou R. Photoselective vaporization of the prostate and simultaneous suprapubic cystostomy for the treatment of benign prostatic hyperplasia in patients with mild to severe detrusor underactivity. *Urol Int* 2015;95:269–75.
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[9] Smith PP, Hurtado EA, Appel RA. Post hoc interpretation of urodynamic evaluation is qualitatively different than interpretation at the time of urodynamic study. Neurourol Urodyn 2009;28:998–1002.

[10] Suskind AM, Smith PP. A new look at detrusor underactivity: impaired contractility versus afferent dysfunction. Curr Urol Rep 2009;10:347–51.

[11] Brierly RD, Hindley RG, Mclarty E, Harding DM, Thomas PJ. A prospective controlled quantitative study of ultrastructural changes in the underactive detrusor. J Urol 2003;169:1374–8.

[12] Levin RM, Longhurst PA, Barasha B, Mcguire EJ, Elbadawi AJ, Wein AJ. Studies on experimental bladder outlet obstruction in the cat: long-term functional effects. J Urol 1992;148:939–43.

[13] Gosling JA, Gilpin SA, Dixon JS, Gilpin CJ. Decrease in the autonomic innervation of human detrusor muscle in outflow obstruction. J Urol 1986;136:501–4.

[14] Febe JL, van Asselt E, van Mastigt R. Neurophysiological modeling of voiding in rats: urethral nerve response to urethral pressure and flow. Am J Physiol 1998;274:R1473–81.

[15] Bump RC. The urethrodetrusor facilitative reflex in women: results of urethral perfusion studies. Am J Obstet Gynecol 2000;182:794–804.

[16] Chancellor MB, Blaivas JG, Kaplan SA, Axelrod S. Bladder outlet obstruction versus impaired detrusor contractility: the role of outflow. J Urol 1991;145:810–2.

[17] Valenti FA, Robain G, Marti BG. Urodynamics in women from menopause to oldest age: what motive? What diagnosis? Int Braz J Urol 2011;37:100–7.

[18] Griffiths DJ, Mccracken PN, Harrison GM, Gormley EA, Moore KN. Urge incontinence and impaired detrusor contractility in the elderly. Neurourol Urodyn 2002;21:126–31.

[19] Mahdy A, Ghoniem G. Role of urodynamics in the evaluation of elderly voiding dysfunction. Curr Bladder Dysfunct Rep 2014;9:350–62.

[20] Abrams PH, Griffiths DJ. The assessment of prostatic obstruction from urodynamic measurements and from residual urine. Br J Urol 1979;51:129–34.

[21] Pflisterer MH, Griffiths DJ, Schaefer W, Resnick NM. The effect of age on lower urinary tract function: a study in women. J Am Geriatr Soc 2006;54:405–12.

[22] Schmidt F, Shin P, Jorgensen TM, Djurhuus JC, Constantinou CE. Urodynamic patterns of normal male micturition: influence of water consumption on urine production and detrusor function. J Urol 2002;168:1458–63.

[23] Griffiths DJ. Editorial: bladder failure—a condition to reckon with. J Urol 2003;169:1011–2.

[24] Nitti VW. Pressure flow urodynamic studies: the gold standard for diagnosing bladder outlet obstruction. Rev Urol 2005;7(Suppl. 6): S14–21.

[25] Lecamwasam HS, Yalla SV, Cravalho EG, Sullivan MP. The maximum watts factor as a measure of detrusor contractility independent of outlet resistance. Neurourol Urodyn 1998;17:621–35.

[26] Van Koeveringe GA, Vahabi B, Andersson KE, Kirschner-Herrmans R, Oelke M. Detrusor underactivity: a plea for new approaches to a common bladder dysfunction. Neurourol Urodyn 2011;30:723–8.

[27] Wyndaele JJ. The normal pattern of perception of bladder filling during cystometry studied in 38 young healthy volunteers. J Urol 1998;160:479–81.

[28] Thomas AW, Cannon A, Bartlett E, Ellis-Jones J, Abrams P. The natural history of lower urinary tract dysfunction in men: minimum 10-year urodynamic follow-up of untreated bladder outlet obstruction. BJU Int 2005;96:1301–6.

[29] Seki N, Kai N, Seguchi H, Takei M, Yamaguchi A, Naito S. Predictives regarding outcome after transurethral resection for prostatic adenoma associated with detrusor underactivity. Urology 2006;67:306–10.

[30] Lomas D, Krambeck A. MP42-02 Long-term efficacy of holmium laser enucleation of the prostate in patients with detrusor underactivity or acomtractility. J Urol 2016;195:e569.

[31] Potts B, Belsante M, Peterson A, Le NB. MP74-15 bladder outlet procedures are an effective treatment option for patients with urodynamically-confirmed detrusor underactivity without bladder outlet obstruction. J Urol 2016;195:e975.

[32] Laudano M, Benedon M, Stern M, Blaivas J. MP89-08 Underactive bladder is not a symptom complex. J Urol 2015;193:e1102–3.