



REVIEW

The underactive bladder: detection and diagnosis [version 1; referees: 2 approved]

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Abstract

The inability to generate a voiding contraction sufficient to allow efficient bladder emptying within a reasonable time frame is a common problem seen in urological practice. Typically, the symptoms that arise are voiding symptoms, such as weak and slow urinary flow. These symptoms can cause considerable bother to patients and impact upon quality of life. The urodynamic finding of inadequate detrusor contraction has been termed detrusor underactivity (DUA). Although a definition is available for this entity, there are no widely accepted diagnostic criteria. Drawing parallels to detrusor overactivity and the overactive bladder, the symptoms arising from DUA have been referred to as the “underactive bladder” (UAB), while attempts to crystallize the definition of UAB are now ongoing. In this article, we review the contemporary literature pertaining to the epidemiology and etiopathogenesis of DUA as well as discuss the definitional aspects that are currently under consideration.



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Introduction

Detrusor underactivity (DUA) is a common bladder dysfunction observed in both men and women undergoing urodynamic studies for the assessment of lower urinary tract symptoms (LUTS). It is typically associated with voiding LUTS such as slow urinary flow and intermittency but may also result in storage and post-micturition LUTS. Despite being commonly encountered in routine clinical practice, DUA has been given relatively little attention in clinical and scientific research. Not only can DUA arise in bothersome symptoms but its occurrence may impact upon the outcome of treatments for other dysfunctions, such as bladder outlet obstruction and stress urinary incontinence. Recently, there has been renewed interest in DUA, the associated symptoms, and potential treatments. This article aims to review the recent literature regarding the epidemiology, etiopathogenesis, and diagnosis of DUA and its associated symptoms.

Definition

A plethora of terms have been used to describe a bladder that does not contract efficiently. Terms such as detrusor areflexia, hypotonic bladder, atonic bladder, detrusor failure, impaired detrusor contractility, and chronic retention all reflect the lack of consensus and uncertainty as to the pathophysiological mechanisms at play. In an effort to standardize terminology, the International Continence Society report in 2002 used the term DUA to describe a urodynamic abnormality. This was defined 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”¹. This definition is necessarily vague on specifying the parameters for length and strength of contraction and prolonged voiding, as what is normal has not yet been established.

Signs and symptoms

There is a lack of prospective studies correlating DUA and its associated clinical signs and symptoms. In clinical practice, the typical presentation is of voiding LUTS such as reduced flow, prolonged flow, hesitancy, and intermittency. After voiding, some patients may report a sensation of incomplete bladder emptying, which may be related to a raised post-voiding residual (PVR).

One could speculate that the presence of storage symptoms is related to the bladder sensory function in the individual patient, such that patients with poor or absent bladder sensation will have a reduced urge to pass urine, which will result in infrequent voiding. Anecdotally, some patients may report they lack any perception of their bladder emptying during the voiding process. Should these patients retain a large enough PVR, they may also experience incontinence, particularly during the night. This clinical picture is commonly termed chronic urinary retention (CUR). Patients with retained bladder sensation may void with a normal frequency pattern or have increased frequency, nocturia, and incontinence should the PVR be high enough.

There has been some debate about whether a symptom complex is associated with DUA, referred to by some as the underactive bladder (UAB), which is rather analogous to detrusor overactivity (DO) and the overactive bladder (OAB) symptom complex. This is certainly an attractive concept, as it could, in theory, allow patients to

be diagnosed and treated on the basis of symptomatology. Nevertheless, there are clear difficulties in defining a specific set of symptoms that do not overlap significantly with LUTS due to benign prostatic enlargement (BPE). Clearly in women this is less likely to be problematic given the relatively rare occurrence of obstructive causes. A working definition for UAB was recently proposed by an expert consensus group: “...a symptom complex suggestive of detrusor underactivity and is usually characterised by prolonged urination time with or without a sensation of incomplete bladder emptying, usually with hesitancy, reduced sensation on filling, and a slow stream”².

Epidemiology

Given the inability to diagnose DUA without an invasive urodynamic study, it is unsurprising that there is little information on its prevalence on a population basis. There is no single non-invasive proxy measure (e.g. raised PVR, reduced urinary flow rate) that could reliably identify patients with the condition. Clinical series of patients with LUTS undergoing urodynamic studies offer the best available evidence as to the prevalence of DUA. In younger men (<50 years), prevalence is 9–28%, which rises to 48% in elderly men (>70 years)³. In elderly women, prevalence rates are between 12 and 45% and peak in the institutionalized elderly. Older frail patients often have co-existent DO, and this entity was originally described by Resnick and co-workers in 1987 as detrusor hyperactive impaired contractility (DHIC)⁴.

Etiopathogenesis

Given the diverse patient groups who can be affected by DUA, it is clear that the etiopathogenesis is likely to be multi-factorial. There seems to be an assumption in the literature that aging leads to a decline in detrusor contractile function. While plausible, it must be emphasized that there is no conclusive evidence that this is the case. The available data from animal and human studies is often quite contradictory.

The etiological factors resulting in DUA can be broadly classified as neurogenic or myogenic.

Myogenic factors

Any pathological process that changes the normal structure and/or functioning of the extracellular matrix or the myocytes of the detrusor may cause a loss in the generation or transmission of a contraction. Factors that may affect the contractile activity of the myocytes include disruption of important cellular mechanisms (e.g. ion storage/exchange, excitation-contraction coupling, calcium storage, and energy generation). The implication of this is that even if efferent neuronal activity is normal, an impaired contraction will result⁵.

Supporting the idea that myogenic factors may be important, morphological changes have been observed within both the normally aged and the diseased detrusor. Much of this work was conducted by Elbadawi, who suggested that specific ultrastructural features detected by electron microscopy were associated with aging and different bladder dysfunctions including DUA^{6–8}. The specific pattern associated with DUA was described as the “degeneration” pattern, characterized by widespread disrupted myocytes with axonal

degeneration⁶. What is not clear is whether detrusor myocyte disruption is in itself the cause of DUA or the result of an underlying pathological process.

Neurogenic factors

Intact bladder sensation is central to the voiding reflex. The afferent nerves monitor bladder volume during the storage phase and also during the voiding phase. Afferent nerves arising at the level of the urethra may also have a contributory role in perceiving flow through the urethra^{9,10}. If afferent functioning is reduced, this may lead to a reduction in the strength or premature termination of the voiding reflex¹¹.

Bladder outlet obstruction

It has long been held that DUA occurs as a secondary effect of prolonged bladder outlet obstruction (BOO). This assumption has been largely derived from work in animals, particularly the rodent, where partial BOO is induced by a constricting metal ring or ligature. The process by which DUA occurs has been widely described and includes three phases^{12,13}:

1. Following obstruction, there is increased bladder outlet resistance leading to bladder wall distension
2. Compensatory hypertrophy and hyperplasia of the detrusor then occurs associated with a rise in tissue blood supply
3. Initially, contractile function is maintained; however, after a variable period of time, contractility is dissipated with a reduction in bladder emptying ability (decompensation)

Relating the insights from this work to humans is problematic, as the animals studied are usually young and female with an acute obstruction induced using a constricting ring. This is unlikely to be a good representation of the common clinical scenario of an elderly man with compressive benign prostatic obstruction. Interestingly, the largest longitudinal study available has shown that in man, prolonged BOO does not usually result in decompensation and DUA. In a group of 170 men with BOO demonstrated on pressure-flow studies, no significant deterioration in urodynamic parameters was observed at a mean follow up of 13.9 years (no change in $p_{det}@Q_{max}$ [detrusor pressure at maximum flow] and a reduction in Q_{max} [maximum flow rate] of only 1 ml/s)¹⁴.

Diabetes mellitus

Diabetic bladder dysfunction (DBD) (or diabetic cystopathy) is a well-recognized cause of DUA. Diabetes mellitus probably affects function through both a myogenic and a neurogenic mechanism leading to a decline in bladder emptying ability throughout the course of the disease^{15,16}. It is traditionally thought that this predominantly occurs through hyperglycemia-induced axonal degeneration and segmental demyelination leading to impaired afferent and efferent bladder function (autonomic neuropathy)¹⁷. Myogenic mechanisms are less clearly elucidated, though it is likely that changes in intercellular connections and excitability, intracellular signaling, and receptor density and distribution are implicated¹⁸.

Diagnostic aspects

Currently, the only accepted modality for diagnosing DUA is an invasive pressure-flow test (or urodynamic study). However, there are no widely accepted criteria for diagnosing DUA. Most proposed

methods relate to the measurement of the strength of the detrusor contraction.

Detrusor contraction strength

A detrusor contraction generates pressure and flow¹⁹; therefore, many authors have used two measurements to approximate contraction strength and thereby diagnose DUA: the maximal flow (Q_{max}) and the detrusor pressure at maximal flow ($P_{det}@Q_{max}$). Most often a reduction in either is considered to be lower than the “normal” range. Historically, for men the normal ranges are derived from a series of men undergoing de-obstructive surgery^{20,21}. In other groups, such as healthy men and women, in particular, the ranges are less well established^{22–24}. Although relatively simple to apply, this method has two flaws. Firstly, it is likely to be an inaccurate method of determining the maximal pressure generated by the bladder due to the bladder outlet relation (BOR), the normal inverse correlation between detrusor pressure and urine flow during voiding²⁵. Essentially, the BOR can be summed up as during voiding the pressure is highest when flow is lowest and vice versa, hence the $p_{det}@Q_{max}$ represents the point of lowest bladder pressure during voiding. Secondly, the fact that the flow rate is also impacted upon by the degree of bladder outlet resistance is not considered. This is important because when detrusor pressure is low, the cause of low flow may also have its basis in obstruction (e.g. due to BPE) or alternatively a normal Q_{max} can result from reduced outlet resistance even if detrusor pressure is low (e.g. post-prostatectomy incontinence).

In order to gain a more accurate assessment of contraction strength, different methods of measuring isovolumetric bladder pressure have been introduced:

1. Watts factor

This is an estimate of the power per unit area of the bladder surface that is generated by the detrusor. The formula for calculation is $WF = [(p_{det} + a)(v_{det} + b) - ab]/2\pi$ where v_{det} is detrusor shortening velocity and a and b are fixed constants ($a=25$ cmH₂O, $b=6$ mm/s) derived from experimental and clinical data²⁶. The main pros of the WF are that it is not dependent on bladder volume²⁶ and is not influenced by increased outlet resistance²⁷. There are, however, no validated values for what is considered normal. Currently, the WF is little used in practice due to its complexity as well as the fact that it assesses only strength of contraction, rather than duration.

2. Projected isovolumetric pressure (PIP)

The PIP was proposed by Schäfer, who used a straight line to represent the BOR and then “projected” back to the y-axis (p_{det}) from the point representing $p_{det}@Q_{max}$ to obtain the isovolumetric pressure. This projection is calculated by the formula $PIP = P_{det}@Q_{max} + KQ_{max}$ where K is a fixed constant representing the steepness of the angle of the BOR to x-axis²⁸. K is dependent on the specific population studied and differs between men and women²⁹; in men it is usually taken as 5. The suggested groupings for PIP are as follows:

- >150 – strong contractility
- 100 to 150 – normal contractility
- 50 to 100 – weak contractility
- <50 – very weak contractility

There are two other reported variants of the PIP: the detrusor coefficient (DECO), which is the PIP divided by 100, and the bladder contractility index (BCI), which is essentially the same as PIP but with different groupings. The main advantage with these formulae is that they are easy and quick to use; however, they may overestimate PIP and have less test-retest reliability than measuring isovolumetric pressures directly³⁰.

3. Mechanical occlusion of flow

This entails the direct measurement of isovolumetric pressure by the mechanical obstruction of urine flow³¹. There are two methods for this measurement: (1) a stop test, the interruption of flow once it has already started, or (2) continuous occlusion test, where urine flow is impeded before and throughout the duration of the detrusor contraction. Stop tests can be voluntarily induced by the patient by contraction of the rhabdosphincter or alternatively the urodynamicist can impede flow through methods such as manual occlusion of the urethra. The voluntary stop test tends to underestimate pressure by about 20 cmH₂O, which is likely to be attributable to a reflex detrusor inhibition brought on by sphincter contraction^{30,32}. The voluntary test may not be possible in patients with sphincter weakness and in the elderly, whereas continuous occlusion can be painful and does not provide the opportunity for synchronous flow measurement.

Detrusor contraction speed

If a bladder is slow to contract, then this could also potentially result in DUA. Cucchi and co-workers proposed the parameter of detrusor shortening velocity, as calculated by the formula $v_{det} = Q/2[3/(V + V_t)/4\pi]^{0.66}$ where Q represents the flow rate (ml/s), V represents bladder volume (ml), and V_t represents the volume of

non-contracting bladder wall tissue. They found that a reduction in shortening velocity was evident before a decline in Watt factor in both men^{33,34} and women³⁵.

Ambulatory urodynamics

It is not uncommon for a patient to be unable to void during a urodynamic study due to anxiety; this has been termed “bashful bladder”, which has been attributed to inadequate relaxation of the rhabdosphincter combined with reflex detrusor inhibition. This situation is usually identified by taking a good clinical history of previous voiding symptoms; however, if doubt persists, then an ambulatory urodynamic study may be helpful^{36,37}.

Conclusions

DUA remains a poorly characterized and poorly understood bladder dysfunction. Its clinical correlate, UAB, has recently been introduced into clinical usage, yet it remains unclearly defined. To progress the current situation, there is a need to investigate the common mechanisms by which patients develop DUA as well as to collect prospective data that can be used to correlate the symptoms and signs with an underlying urodynamic abnormality.

Competing interests

The authors declare that they have no competing interests.

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The short commentary 'The underactive bladder: detection and diagnosis' discusses the topic from the perspective of symptoms, epidemiology, pathogenesis, and diagnostic aspects. The paragraphs on diagnostic aspects are a summary of urodynamic testing, more specifically: pressure-flow analysis. I assume that the words '...are typically associated with...' in the introduction and the words '...typical presentation...' in the signs and symptoms section, refer to the historical beliefs of professionals. The last paragraph of the signs and symptoms section mentions that there is a debate on whether there is a symptom complex associated with detrusor underactivity (the underactive bladder syndrome?). I agree with this statement and careful reading of the working definition cited from reference 2 in the commentary makes it obvious that the working definition of (the) bladder outflow obstruction (syndrome) would be exactly identical, if it was introduced nowadays. The elements of the working definition for UAB (as a signs and symptoms syndrome) are equal to those considered, both currently and traditionally, very relevant in men with benign prostatic enlargement¹. I conclude that the underactive bladder syndrome as it is suggested here is not specific, like all LUTD-syndromes, and that, indeed, underactive detrusor (contraction during voiding) is a condition only assessable with a pressure-flow study.

There is some observational clinical data on how the detrusor compensates for the growing prostate and the—by consequence—increase in bladder outflow obstruction, in addition to the animal studies referred to in the commentary, to explain the pathophysiology². With the perspective of DU, this clinical study may allow two interpretations; first: the detrusor of aging men increases (maximum) power to compensate for the growing prostate and second: some detrusors are better able to compensate than others. The validity of the clinical observation² was subsequently confirmed in a canine experiment³. If relevant neurogenic abnormalities are excluded on the basis of history and clinical examination, myogenic factors remain as the cause of detrusor underactivity, and based on biologic variance, some detrusors will be stronger than others and some will be better able to conquer challenges. The search for relevant amino-acids or other molecules will be important here if we want to discover ways to improve the symptomatically weak detrusors.

Urodynamic diagnosis of detrusor contraction work or power during voiding is not yet standardized, and the commentary gives a neat overview of parameters that may prove to be useful. Especially objective parameters that will demonstrate predictive value and responsiveness to management will become relevant in the future.

Cooperation with the patient is necessary to obtain a representative pressure-flow test, and the autonomic

voiding reflex requires parasympathetic dominance in a—for the patient involved—stressful situation. This is even more important when the patient has a ‘bashful bladder’. I consider it valuable that this is introduced here in this context. Based on the relevance of this dysfunction, that will exist in a continuum from ‘low grade’ to ‘very troublesome’, the new ICS Good Urodynamic Practices and Terms (that will be available in 2016) will introduce the terms (or diagnoses) ‘situational inability to void’ and ‘situational inability to void as usual’. Whether ambulatory urodynamics provides a reliable method to obtain a pressure-flow test remains speculative however, because of the lack of evidence.

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I have read this submission. I believe that I have an appropriate level of expertise to confirm that it is of an acceptable scientific standard.

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