



Do the definitions of the underactive bladder and detrusor underactivity help in managing patients: International Consultation on Incontinence Research Society (ICI-RS) Think Tank 2017?

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Aims: The Think Tank aimed to discuss the pitfalls and advantages of current definitions in terms of research and management of underactive bladder (UAB). UAB broadly defines a symptom complex of bladder emptying problems and does not indicate a specific pathology. Detrusor underactivity (DU) is a urodynamic diagnosis from pressure-flow studies. The correlation of UAB with DU remains to be precisely determined.

Methods: The presentations and subsequent discussion, leading to research recommendations during the Think Tank of the International Consultation on Incontinence Research Society in Bristol, 2017, are summarized.

Results: To develop more specific individualized management strategies, the Think Tank panel proposed (i) that, since defining a single type of index patient to represent all UAB will not fulfill all clinical research needs, several index patients should be defined by phenotyping of patients with UAB, including, children, young men and women, elderly male and female patients with co-existing DU and detrusor overactivity, and neurological patients with UAB; (ii) prospective longitudinal studies to assess the natural history of UAB, in the different target populations, based on different UAB phenotypes, should be initiated; (iii) DU should be precisely defined by urodynamic parameters; and (iv) work to develop validated specific questionnaires combined with non-invasive tests for screening, diagnosis and follow up, needs to be continued.

Conclusions: The precise relationship of UAB to DU remains to be defined. Phenotyping patients with UAB/DU, performing prospective trials of natural history, and developing symptom questionnaires and diagnostic investigations will improve our ability to identify and treat UAB/DU.

KEYWORDS

detrusor underactivity, dysfunctional voiding, Fowler's syndrome, terminology, underactive bladder, urodynamics

1 | TERMINOLOGICAL HIERARCHY OF BLADDER EMPTYING PROBLEMS: SYMPTOMS VERSUS FUNCTION VERSUS CAUSE

Detrusor underactivity (DU) has been defined by the International Continence Society (ICS) from pressure-flow studies (PFS), as a contraction of reduced strength and/or duration, resulting in prolonged bladder emptying and/or a failure to achieve complete bladder emptying within a normal time span.¹ DU does not indicate a specific symptomatology, etiology, or need for treatment and does not fulfil the needs for a clinical definition. Furthermore, parts of the urodynamic definition, such as reduced strength or prolonged duration or normal time span have not been defined and may be age and/or gender dependent.

The term underactive bladder (UAB) has been proposed as the clinical picture associated with DU. UAB is defined as “slow urinary stream, hesitancy and straining to void, with or without a feeling of incomplete bladder emptying and dribbling, often with storage symptoms,” and again does not imply a specific pathology.² Management of UAB greatly varies depending on the bother of the patient. UAB may be suggestive of DU, but PFS may not always confirm DU and the degree of bother is often variable.^{3,4}

The underlying cause may be impaired detrusor contractility that indicates diminished detrusor contractile function assuming that there is maximal stimulation with no shortage of energy substrate.⁵ Contractility is a measure of muscle response, downstream of ample neurotransmitter stimulation in the presence of ample energy substrate.⁵ A neurogenic disease may play a role in the etiology.⁵ The previous ICI-RS report agreed on these three definitions namely, UAB, DU, and impaired contractility as the symptomatic, functional and etiologic correlates of bladder emptying problems, respectively (Figure 1).⁵

2 | DO WE NEED A BROAD DEFINITION FOR THE BLADDER EMPTYING SYMPTOM COMPLEX SUCH AS IN OAB?

The Think Tank focused on the research needed in view of the opportunities and the pitfalls of a broad definition of a symptom complex. A comparison was made between UAB and the other broad-spectrum definition, OAB that has a similar hierarchy of symptoms, dysfunction, and cause (Table 1).

The presenting symptoms and the urodynamic findings do not always correlate in both conditions. For example, OAB is not necessarily associated with DU and UAB is not always associated with DU. OAB and UAB mostly present as quality

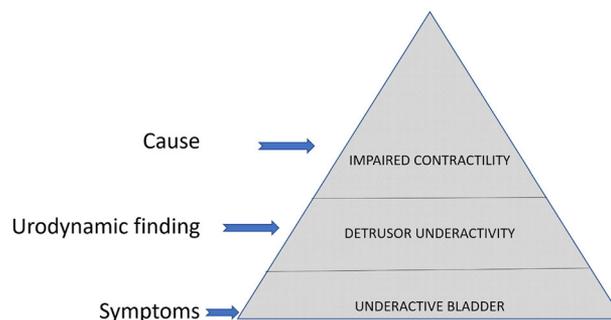


FIGURE 1 The hierarchy of symptoms, urodynamic findings, and cause in bladder emptying problems

of life issues, where secondary upper urinary tract (UUT) changes are unlikely, unless there is co-existing bladder outlet obstruction (BOO). Both conditions may be associated with benign prostatic obstruction (BPO) and negatively affect the outcome of prostatectomy.⁴

Obviously, the lack of a successful medical treatment for UAB/DU has discouraged research in this field resulting in significantly less epidemiological data, compounded by the absence of validated symptom scores or scales to be used for screening studies. Exact epidemiological data may be difficult to acquire, as at least some DU patients do not experience “symptoms.” On the other hand, OAB can usually be screened for, even by a telephone interview or can be studied more intensively by a bladder diary.

For conditions that affect quality of life, treatment is often based on symptoms without a definitive diagnosis, assuming that the treatment is neither dangerous nor prohibitively expensive. Some false positive and false negative diagnoses can be tolerated if there are no serious consequences of inappropriate treatment or delayed diagnosis. The necessity to perform diagnostic testing which is uncomfortable, expensive or has potential complications is likely to thwart the use of medications for a condition largely affecting quality of life, such as UAB. Hence, if scientifically justified, patient care is facilitated by as broad a definition as possible, assuming that the available effective treatments can improve or reverse the cause of the condition. However, if the diagnostic process is lengthy and expensive it may be difficult to identify enough sufferers of the condition to economically justify work to discover and develop new medications or surgical procedures. Therefore, broad definitions of a symptom complex may be helpful to generate funds for research and to make an adequate business case for research into new pharmacological compounds. It would be helpful to find investigational methods that confirmed DU in patients with one or more UAB symptoms if and when treatments that resolve one or more of the symptoms become available. This would certainly draw doctors’ attention to the subject if the patients they see with the condition, could more easily be diagnosed. Furthermore, a

TABLE 1 A tentative comparison of OAB and UAB

	Overactive bladder	Underactive bladder
Etiology	Poorly understood, assumed to be multifactorial	
Epidemiology		
Prevalence	Well-studied	Not known
Age and gender distribution	Affects children and adults and both genders	
Symptoms		
Index patient	Defined	Not defined
Bladder diary for the diagnosis	Useful	Not useful
Symptom awareness	Generally high	Low, at least in some patients
Screening by telephone interview	Commonly utilized	Not applicable
Validated questionnaires for diagnosis and screening	Exist	Do not exist
Cardinal symptom	Urgency	Not defined, yet
Other measurable symptoms useful as end points in studies	Urgency incontinence, daytime frequency, nocturia	Not defined, yet
Urodynamic findings and definitions		
Main Urodynamic finding	DO, usually accompanied by urgency or DO incontinence	DU, usually with loss of bladder sensation
DO vs DU	DO is a finding of filling CMG and it is well defined	DU is finding of pressure-flow study and its parameters are not clearly defined
Treatment		
Effective medical treatment	Exists	Does not exist
Effective surgical treatment	Exists	Does not exist
Prognosis		
Risk for UUT damage	Very low in both, unless it is associated with BOO	

large number of patients would be able to recognize themselves if there were a broad definition.

However, one pitfall of a broad definition is when several conditions share the same symptoms. In the case of UAB we are facing a major challenge to differentiate DU from BOO based on symptoms, bearing in mind that DO and DU may co-exist. In addition, should we treat UAB to relieve symptoms or treat the cause of the DU in an attempt to correct DU and thereby resolve its symptoms? A symptomatic cure might be achieved by improving the compensatory mechanisms, but may not be warranted since its long term effects are not known. Obviously, UAB has to be treated based on the underlying condition, not based on the symptoms. To overcome this pitfall, we need to develop algorithms to detect underlying conditions and initiate treatment, tools to evaluate treatments based on evaluation of the condition and symptoms, and to develop person-specific treatments including prevention of disease progress. Understanding the etiological factors leading to UAB and its natural history is the key to fulfill the aforementioned requirements.

3 | CHALLENGES IN DISTINGUISHING DU BASED ON SYMPTOMS AND DEVELOPING SPECIFIC QUESTIONNAIRES

A perfect correlation between the symptoms of UAB and underlying DU has not been proven in any prospective study. Rademakers et al reported an increased prevalence of incomplete bladder emptying, hesitancy, and a weak stream in women with DU compared with women with normal PFS.⁶ They found a correlation between PVR (>100 mL) and almost all of the voiding questions that were addressed, except for “applying abdominal pressure during voiding?” In addition, a low bladder contractility index (BCI) showed a significant association with the questions related to “a feeling of incomplete emptying,” “hesitancy” and “presence of a weak stream.”

Likewise, Gammie et al have evaluated a database of 28 282 adult PFS records of both genders.⁴ Men who had both a low BCI and a BOO index in the obstructed range,

suggesting simultaneous DU and BOO, and women with clinical obstruction were excluded from the analysis. After exclusion, they classified 1788 patients (men: 507; women: 1281) into: (i) those with DU without BOO; (ii) those with BOO without DU; and (iii) those with normal PFS. They found that patients with DU reported a statistically significantly higher occurrence of decreased and/or interrupted urinary stream, hesitancy, feeling of incomplete bladder emptying, palpable bladder, and absent and/or decreased sensation compared with patients with normal PFS.⁴ Despite these encouraging results, the study population was not a general urology or screening population but it was a referral patient group with bothersome LUTS. The symptom questions in the database were not validated and the study naturally carried the disadvantages of a retrospective clinical evaluation. Considering the strict patient inclusion criteria, one can also argue that the definitions in the latter study were designed to prevent any doubt that these patients really had DU, and therefore may have been too strict.

In contrary to the latter two studies, two other studies correlating symptoms with urodynamic findings in young men with LUTS revealed that clinical symptoms could not predict urodynamic evidence of DU or its etiology.^{7,8} Obviously, the aforementioned four studies contain significant dissimilarities in methodology that may account for their conflicting findings.

Apparently, the question as to whether all UAB/DU patients are “in need of treatment” needs to be explored. Some of the hidden cases may include patients who cope with their symptoms and do not seek medical help, whereas another group may consist of symptomatic but under-evaluated cases. For example, in a screening population of ambulatory elderly women, Huang et al have shown that 27% of women with PVR >200 mL reported no significant symptoms at the baseline evaluation.⁹ However, being asymptomatic in spite of high PVR does not necessarily mean that these patients are safe in the long term. For example, Wu et al have shown that incidentally detected elevated PVR is associated with increased risk of urinary tract infection (UTI) in both genders.¹⁰ Likewise, Hijazi et al have diagnosed DU as the sole underlying cause in 22% of women with recurrent UTI who were otherwise asymptomatic.¹¹ The latter two studies have revealed the need for diagnosing asymptomatic cases since these can lead to significant morbidity, especially in the elderly.

Thus, there is a strong need for prospectively designed studies to investigate the correlation between symptoms and urodynamic findings in better defined patient groups in order to develop a specific symptom questionnaire. This questionnaire may be combined with noninvasive tests such as measurement of bladder wall thickness, uroflow and PVR measurement.^{4,12} Therefore, it may be used in assessment for detection, diagnosis, and evaluation of treatment outcomes of

UAB. Very strict symptomatic definitions may have a high specificity for DU, but their sensitivity may be low and therefore may not be as clinically useful as hoped, such issues may help to explain conflicting findings in the literature.

4 | PHENOTYPING PATIENTS WITH UAB/DU FOR RESEARCH, DIAGNOSTIC, AND THERAPEUTIC PURPOSES

Our Think Tank suggested phenotyping patients with bladder emptying problems according to following index types:

- 1) Children with UAB/DU
- 2) UAB/DU in younger patients
 - a) Young men with idiopathic UAB/DU
 - b) Young women with idiopathic UAB/DU with or without dysfunctional voiding (DV) or with Fowler's syndrome.
- 3) DU in the elderly male and female with or without DO
- 4) Neurological patients, (eg, spina bifida and sacral agenesis).

1) Children with UAB/DU

Both OAB and UAB are seen in childhood and their possible role in the occurrence of adult UAB is intriguing. The main question is whether some people are born with weak bladders, others with stronger bladders or whether UAB can develop in every child due to bad bladder habits. *One hypothesis* proposes that, in children, UAB evolves from a cascade of conditions, starting with OAB, continuing through voiding postponement and dysfunctional voiding (DV), before UAB develops. Urgency incontinence is the most common type of daytime urinary incontinence (UI) in children.¹³ It can be considered as a developmental disorder with a persistence of infantile voiding patterns and a genetic disposition.^{14,15} Contractions of the detrusor are inadequately inhibited by the central nervous system (CNS), manifesting as urgency which, in turn, is counteracted by holding manoeuvres such as squatting, sitting on one's heel, pressing legs together, etc.¹⁶ By thus overexerting pelvic floor muscles, frequency decreases. By continuing to defer micturition with the aid of holding manoeuvres, voiding postponement develops.

Voiding postponement leads to the second most common type of daytime UI characterized by a habitual deferral of micturition in certain situations such as during school, while playing video games, etc.¹³ Incontinence occurs when the bladder is distended and despite the use of holding manoeuvres the detrusor cannot be inhibited. Low

micturition frequency is common. In some children, it is a habit which is maintained despite negative consequences. In others, it is associated with oppositional defiant disorder (ODD) and family dysfunction.¹⁷ In the further course, DV can develop, characterized by a habitual contraction of the urethral sphincter or pelvic floor during voiding, with “staccato” or interrupted urine flow pattern and increased EMG-activity measured during pelvic floor contractions. Large post-void residuals, constipation, UTI's, and even UUT damage can evolve. The last of the cascade would then be UAB, as described above.

This progression or sequence theory is advocated by Franco, who states that: “OAB triggers ‘bladder overactivity’ usually in the early filling phase, causing the pelvic floor to respond by voluntary contraction,” which leads to holding maneuvers and temporary reduction of “bladder contractions.”¹⁷ According to this theory, the starting point of the cascade described above would be OAB. However, only a longitudinal study can prove or refute this concept: as far as we know such a study is not in progress.

The second model proposes that different types of lower urinary tract disorders (LUTD) can be primary conditions—without the necessity of preceding disorders. From a large, cross-sectional series of 1000 children, Hoebeke et al argue that the age distribution of different conditions does not reflect a typical sequence, as DV often occurs before OAB.¹⁸ Children with functional, non-organic LUTD “have different primary diseases, but all have a common risk of incontinence, UTI's, reflux and constipation”; and “the traditional concept of a DV sequence may be true in some children, but primary forms of LUTD may be more common.”

According to the third model, voiding postponement is the starting point of progression.¹⁹ The “volitional delaying of voiding” induces detrusor muscle hypertrophy and fibrosis, leading to decreasing compliance and “uninhibited bladder contractions,” often associated with constipation. This, in turn, leads to detrusor hypertrophy and tonic pelvic floor contractions, so that DV can follow.

All three models have their validity since different etiologies could be possible in individual children.¹⁸ In some children, OAB could trigger the progressive sequence of disorders; in others, they could be primary conditions; and finally, in some, the deferral of micturition could lead to OAB. All three models focus on bladder function which is mediated by holding maneuvers and voiding behaviors characterized by deferral, retention, and postponement. They do not adequately incorporate the inhibitory functions of the CNS. Prospective studies with carefully defined populations with documented bladder and CNS function are needed to understand how and why UAB and DU develop.

2) DU in young adults

In young adults, a major differentiation for phenotyping is based on gender, because of anatomical and functional differences.

a) Young men with idiopathic DU

The exact prevalence and natural history of DU in young men is unknown. The causative factors may be congenital or acquired from childhood. Many young men with UAB/DU are mistakenly labeled as having chronic prostatitis and treated empirically by antibiotics, alpha blockers or anti-inflammatory agents. For example, Kaplan et al retrospectively studied 137 men with chronic LUTS, all previously misdiagnosed with chronic prostatitis and treated unsuccessfully.²⁰ Videourodynamic studies revealed primary vesical neck obstruction, DV, impaired bladder contractility, and acontractile bladders in 54%, 24%, 17%, and 5% of these cases, respectively. In another study, Karami et al retrospectively analyzed young men with chronic idiopathic LUTS.²¹ They found acontractile detrusor in 10.5%, underactive detrusor in 2.4% patients, and DO plus acontractile detrusor in 1.3%.²¹ In a similar patient group, Joeng et al have found DU or acontractile detrusor in 13%.²² These three studies indicated that few clinical symptoms were useful for the diagnosis in young men with chronic LUTS. Therefore, videourodynamic studies were necessary for the correct diagnosis. DU was the underlying urodynamic pathology in up to 22% of young men with idiopathic LUTS. Further research in this patient population is badly needed to define more accurate clinical tools in the differential diagnosis, and to discover the cause or causes of UAB/DU in young men.

b) Young women with idiopathic UAB/DU, with or without DV or with Fowler's syndrome

The diagnostic dilemma is most challenging in the younger female population because small changes in detrusor pressure may define BOO or DU, making it very difficult to develop reliable diagnostic urodynamic descriptions. Additionally, women may not be able to void during PFS due to the obstructive effect of the catheter and/or the unnatural environment and artificial retrograde filling used in conventional urodynamics. As a consequence, DU may be overlooked in some women with very low urethral resistance, particularly those with stress incontinence, and detrusor function can only be assessed by obstructing their micturition mid-void. In contrary, there are other groups of women with BOO due to either sphincter overactivity, such as in Fowler's syndrome, or pelvic floor overactivity, as in DV. This is in contradistinction to women with idiopathic DU who have normal outlet resistance.²³ Therefore, phenotyping may be necessary, even among women, to

TABLE 2 Comparison of dysfunctional voiding with Fowler's Syndrome as two main phenotypes of young women with UAB/DU and increased pelvic floor/sphincter activity

	Dysfunctional voiding	Fowler's syndrome
Assumed etiology	Learned misbehavior	Primary relaxation disorder of the EUS
History of childhood bladder dysfunction	Present	Not present
Trigger event	Not present	Usually present
Co-morbidities (gynecological, neurological and functional)	Not present	Commonly present
Specific EMG finding	Not defined	Defined
Sphincter volume	Normal	Increased
MUCP	Less than 100 cmH2O	Usually above 100 cmH2O
Urgency	Exist	Does not exist
Urethral catheterization	Easy	Painful with gripping sensation
Bladder sensation	Normal	Absent
Bladder capacity	Usually normal	Usually over 1000 mL
Treatment	Benefits from sphincter relaxation exercises and SNM	Benefits almost only from SNM

EUS, external urethral sphincter; EMG, electromyography; MUCP, maximum urethral closing pressure; PFMT, pelvic floor muscle training; SNM, sacral neuromodulation.

differentiate those that have idiopathic DU and those that have DU which may be secondary to, or associated with, BOO, whether from sphincter overactivity or pelvic floor overactivity.

Recently, Brown et al investigated 200 women with DU stratified into four cohorts: cardiovascular disease manifestations, cardiac risk factors, neurologic, or idiopathic.²⁴ They found no differences between cohorts for presenting symptomatology or urodynamic parameters. However, patients were retrospectively recruited using ICD-9 codes where the diagnostic accuracy of these symptom codes for UAB/DU remains unclarified. UDS were also retrospectively reviewed and interpretation was certainly open to error and bias. Furthermore, there was no phenotyping of DU in terms of the co-existence, or not of increased outlet resistance. The Think Tank agreed that phenotyping of female patients with UAB symptoms, in future studies, should define two groups, DU alone and DU with co-existing BOO: the question as to whether DU is secondary to urethral obstruction remains unclarified. The group of women with DU and increased sphincteric or pelvic floor activity include women with Fowler's syndrome and patients who develop DU secondary to DV, respectively, however, the distinction between the two subgroups is debatable. Fowler's syndrome is postulated to be the isolated idiopathic retention of young women due to a hormonally-determined primary relaxation disorder of external urethral sphincter associated with a specific EMG abnormality.²⁵ However, a recent study revealed that

50% had unexplained chronic pain syndromes, and 9% of these were taking opiates.²⁶ Furthermore, abdominopelvic surgery with general anesthesia was the leading trigger in 35%, whereas 24% had "functional" neurological symptoms such as non-epileptic attacks or leg weakness and 30% had psychological symptoms.²⁶ These findings further raise the issue as to whether Fowler's syndrome is really due to primary spasticity of external urethral sphincter or whether it is a marginal phenotype of DV. A transition between DV and Fowler syndrome is difficult to postulate because both conditions have unique, even paradoxical features.²⁷ Our Think Tank has further elaborated the clinical, urodynamic, and therapeutic differences between the two conditions (Table 2).

3) UAB/DU in the elderly male and female with or without DO

The Think Tank has further classified the UAB/DU in the elderly in three phenotypes as (i) elderly symptomatic men with UAB/DU after failed prostatectomy and/or high PVR and/or UI; (ii) elderly women with UTIs and/or high PVR due to UAB/DU and; (iii) elderly women with UTIs and/or high PVR and/or urgency UI due to UAB/DU with co-existing DO. However, there is very little data in the literature to precisely delineate the clinical features of these subgroups.

In the elderly, DU often co-exists with DO so that patients have both OAB and UAB. Resnick and Yalla referred to this group as having DHIC (detrusor hyperactivity, impaired contractility) in men. Later they

described the same phenomenon in women.^{28,29} “DHIC” or according to contemporary terminology “co-existing DO and DU” was clinically defined as an overactive detrusor during filling that empties ineffectively due to diminished detrusor contractile function. Other associated features were bladder trabeculation, a slow velocity of bladder contraction, little detrusor reserve power, and a significant amount of residual urine. The outcome of prostatic surgery is usually poor in patients with “DHIC.”²⁸ Women with “DHIC” may also present with stress UI. However, any surgery to prevent SUI, may increase outlet resistance and worsen the clinical outcome as women who pre-operatively void with a weak detrusor contraction are most likely to have urinary retention postoperatively.³⁰

The co-existence of DO with DU has led to an intriguing theory, as yet unproven, of a possible common mechanism for the pathophysiology of UAB. Chancellor recently hypothesized that chronic untreated or treatment refractory OAB may progress to DHIC and ultimately to UAB.³¹ He suggested that UAB and OAB may not be an entirely separate disease entity. Instead, chronic untreated or treatment refractory OAB-due to diabetes, BOO or aging may progress to DHIC and, finally, UAB. He further recommended that early education, behavioral modification and medical treatment may alter and/or prevent progression to UAB. However, this theory lacks the essential natural historical data for confirmation.

If this hypothesis is true, DO-DU is not a unique disease state but it is a step in the pathophysiological progress of DO to DU. Unfortunately, there is a big gap of knowledge regarding the natural history of OAB/DO and UAB/DU. In one of the few studies, Thomas et al have followed neurologically intact men aged >18 years diagnosed with DU and who initially opted for no specific treatment with a mean follow-up of 13.6 years.³² There were few symptomatic and urodynamic changes over a long period of time. In another study, Heidler et al studied the natural history of OAB on women with median follow-up of 6.5 years and failed to show a progressive character for OAB.³³

However, Garnett et al have shown evidence of deterioration of OAB and DO in a period of more than 10 years.³⁴ They retrospectively analyzed 174 women with OAB and DO and found that 88% of women with OAB/DO continued to have their symptoms after 10 years. They also demonstrated statistically significant increases in detrusor pressure at the time of DO incontinence, more involuntary detrusor contractions, and reduced bladder capacity. Although clinically insignificant, there were more women who developed DO incontinence than had a resolution of it. Only one in eight women did not reveal persistent symptomatic DO at repeat UDS after 10 years.³⁴ They did not show progression to UAB/DU.

Our Think Tank has proposed that pelvic ischemia is a promising area of research to explain DO-DU, as chronic moderate bladder ischemia in the rabbit is associated with DO while chronic severe bladder ischemia causes impaired detrusor contraction.³⁵ It has been postulated that even moderate ischemic states of the bladder that are associated with DO, could in the long-term lead to DU.^{35,36} Atherosclerosis-induced arterial insufficiency or BOO alone may produce bladder ischemia, leading to functional and structural changes in the bladder, and cause LUTS. The coexistence of arterial insufficiency with BOO may expose the bladder to a more severe ischemia and worsen the functional and structural alterations in the bladder secondary to BOO. An ischemia-induced bladder fibrosis can further compromise the bladder blood flow through non-compliant high-pressure filling.³⁶

Although “decompensation” of the detrusor, due to long term BOO and/or chronic ischemia, has been a commonly cited hypothesis no study has yet shown that, in humans, long standing BOO can lead to decompensation resulting in an initially normal bladder developing DU or UAB/DU, in the long term.³⁶ The decompensation concept is supported only in short-term acute animal experiments in maturing animals over a few months.³⁷ These animal models may not be a good model for the many years of changing pathophysiology in patients, and that the concept of “decompensation” remains unproven.

5 | CONCLUSIONS

The precise relationship of UAB to the urodynamic definition of DU remains to be clarified. Our Think Tank agrees on following recommendations for research priorities on UAB/DU:

1. Use the current working hypothesis of UAB and definition DU until data supports a change in these definitions.
2. The existing urodynamic definition of DU lacks precision. There is a need for gender- and age-adjusted quantitative definitions for DU.
3. There needs to be phenotyping of UAB/DU patients in the populations suffering from LUTS suggestive of DU, as suggested by this Think Tank.
4. Better animal models need to be developed to test new medical and other treatment options. Animal models for DU should follow the phenotyping of UAB, as much as possible. Acute models may not mimic the human disease at all.
5. A widely accepted and fully validated questionnaire to enable screening for UAB, that can be further combined with non-invasive urodynamic tests, to enable the diagnosis of UAB/DU is needed.

6. Prospective longitudinal studies to assess the natural history of UAB are needed. Target populations should include the phenotypes of UAB as indicated by this Think Tank. The transition of childhood disorders into adulthood should also be assessed by longitudinal studies.
7. Exploration of the possible drug mechanisms of increasing detrusor contractile function, reducing urethral resistance and “modulating” the nervous system on different phenotypes, will hopefully lead to effective drug treatment of UAB/DU.

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