In recent years, there has been a rise in interest in detrusor underactivity (DU) [1–3], a bladder dysfunction that affects both sexes and causes bothersome symptoms. DU is defined by the International Continence Society (ICS) 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” [4].

As much as 48% of older men and 45% of older women undergoing evaluation for lower urinary tract symptoms (LUTS) show evidence of DU [5,6]. These patients may be affected by symptoms or require catheterisation for bladder drainage. Despite this apparent frequency, DU is largely underresearched in comparison to other lower urinary tract dysfunctions, such as detrusor overactivity (DO) or bladder outlet obstruction (BOO). Moreover, there is no simple, effective treatment.

At present, it is widely thought that the LUTS experienced by patients with DU overlap significantly with the LUTS associated with BOO and that it is not possible to reliably differentiate the two without an invasive urodynamic study. This has hampered the acquisition of epidemiological data and, in turn, has led to a lack of comprehensive evaluation of...
the true scale of the problem, its natural history, and its
effects in terms of symptoms, symptom bother, and
complications (eg, urinary retention, impairment of renal
function).

Clinical experience and evidence from available urody-
amic case series suggest that DU occurs in diverse patient
groups, pointing towards the existence of multiple aetio-
logical factors. These factors are likely to manifest in DU by
disrupting the processes involved in the generation of an
effective coordinated voiding contraction [2,7]. Interruption
to efferent neural pathways secondary to traumatic injury
or disease and intrinsic myogenic dysfunction due to
fibrosis are well-recognised mechanisms. More recently,
the potential importance of the urethral system and the afferent
system has been suggested [8,9].

There is currently a remarkable lack of consensus on
many aspects pertaining to DU as a diagnosis. A plethora of
terms are used to refer to DU and/or its associated symptoms,
despite the ICS terminology having been published more than a decade ago. Moreover, no accepted diagnostic criteria exist. Furthermore, the ICS report falls short in specifying parameters for reduced contraction strength, prolonged bladder emptying, or normal time span. Most current criteria focus on strength, either applying specific cut-offs for maximum flow rate (Q\text{max}) and maximum detrusor pressure Q\text{max} or using indices and calculations such as the bladder contractility index [10] or the Watt factor, which estimate isovolumetric contraction strength [11]. The application of these criteria to DU is limited for several reasons:

- The criteria do not consider definitional aspects, such as contraction speed or how effectively the bladder empties, mostly related to the duration of the contraction.
- Assumptions regarding bladder volumes and energetics are contained within these calculations, which likely are not applicable to some or all instances of DU.
- The rise in detrusor contraction strength with increasing BOO grade in elderly men suggests that it is difficult or impossible to define single threshold values for DU [12].
- Normative data in highly affected populations (eg, the aged) are not available.

There is a need for further research on all aspects of DU. In contrast, DO is well researched, and it is worth revisiting the development of the OAB symptom complex as a concept. This was based on recognition that patients present with symptoms that may not always correlate with an underlying urodynamic abnormality (ie, DO). This has proved to be an effective means of categorising patients in clinical practice to guide the instigation of therapy, particularly at the primary-care level. Consequently, an expansion of research followed that has contributed to our understanding of bladder storage function and pathophysiology and that allowed the development of novel therapies.

In terms of DU, a definition currently exists but is fairly
nonspecific due to the extremely limited evidence base
from which it was derived. Nevertheless, the urodynamic
abnormality is clearly related to a group of recognised
symptoms (eg, slow flow, hesitancy). In addition, there
are some associated, poorly defined, clinical presentations
(eg, impaired or absent bladder sensation) and sequelae
(eg, raised postvoid residual and urinary retention). A
variety of patient groups are affected, both with and
without neurologic disease or injury. In this context, it is
easy to recognise some parallels to the example of DO
and OAB. Categorisation of the symptoms and/or signs of
dU seems like a logical initial step to facilitate standardisa-
tion and further research in this area.

A consensus group met at the International Consultation
on Incontinence–Research Society and ICS annual meetings
in September and October 2014 to review the available
evidence base and consider the feasibility of developing a
working definition of a symptom complex for underactive
bladder (UAB). It was agreed that although patients with DU
can present with a variety of storage, voiding, and
postmicturition LUTS, the voiding symptoms often predominate. These symptoms appear to be variably associated
with the symptoms and signs of incomplete bladder
emptying and impaired bladder sensation.

It was clearly recognised that the clinical features of DU
may show significant overlap with those of BOO. Despite
this, it was felt that a definition of a symptom complex for
UAB would be of potential clinical value and could form the
basis of a definition on which further qualitative and
quantitative epidemiological studies could be conducted.

We propose the following working definition: The
underactive bladder is 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.

Associated factors that need to be considered include
sex, age, and any known neurologic pathology. It should be
pointed out that the underactive bladder symptom complex
is not synonymous with DU, which can be confirmed only
by urodynamic testing. The definition and the role of
impaired detrusor contractility in DU and UAB also remain
to be elucidated.

It must be emphasised that the proposed definition has
been developed on the basis of expert opinion and
discussion rather than the results of prospective studies.
Such studies are now in progress, as are efforts to obtain
qualitative data from focus groups. These efforts should
help refine this working definition further. Nevertheless, we
feel that the development of the definition presented in this
paper represents a significant step in the right direction and
will help raise the profile of this much-neglected problem
and facilitate further research.

In summary, DU is a common but poorly understood lower
urinary tract dysfunction that occurs in a heterogeneous
group of men and women and that arises due to multifacto-
rial aetiologies. Currently, it can be confirmed only after
urodynamic testing. We propose a working definition for a
complex of symptoms that we suggest are known as
underactive bladder and associated with DU. We feel UAB
could prove useful as a means of identifying affected patients,
rather analogous to the relationship between DO and OAB,
and could provide a basis for further definitive qualitative and quantitative research on the subject.

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References


