Asian Journal of Urology (2019) 6, 208–209

Available online at www.sciencedirect.com

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Commentary

Referring to the article published on pp. 169–173 of this issue

Moving beyond BPH — A contemporary update on male LUTS

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Received 19 October 2017; accepted 21 October 2017

This insightful state-of-the-art article reviews lower urinary tract symptoms (LUTS) and their interface with "so-called" benign prostatic hyperplasia (BPH), in particular, emphasising that LUTS are not just related to the prostate. Highlighting the importance of looking beyond the symptoms, beyond the prostate at not just the potential for a treatment, but rather at the underlying functional basis for the symptomatology. It is essential to take into account not only the patients' LUTS, but their general medical history, drug history and comorbidities and to carefully evaluate patients with a voiding diary, particularly where nocturia is a significant symptom. It is now well-established that the term LUTS includes storage, voiding and post-micturition symptoms. In particular it is the storage symptoms that lead to the majority of patients with "BPH" presenting to see us as urologists. It has been recognised for over 40 years that the bladder is an unreliable witness and that LUTS are not disease specific. Nevertheless, in 2017 there is still a tendency for the term "BPH" to be used as though it is a clinical condition, as is also the case for the storage symptoms designated by the term "overactive bladder symptom complex (OAB)", when in fact, the terms descried a collection of relatively non disease specific symptoms.

LUTS undoubtedly produce significant interference with quality of life and activities of daily living relating to both work and social activities. An increased prevalence of LUTS is associated with sexual dysfunction and the metabolic syndrome [1]. Indeed, it is the underlying detrusor overactivity which is seen in two thirds of patients with benign prostatic obstruction, which has been the subject of considerable scientific research in the past. The principal emphasis has been on potential detrusor muscle dysfunction. There has been comprehensive investigation of detrusor muscle morphology on microscopy, but it is difficult to separate out changes relating to aging from those responsible for altered detrusor muscle function [2]. Brading [3] used an animal model comprising adolescent female pigs with an obstructing ring placed around the urethra to evaluate his hypothesis that there is neuromuscular dysfunction resulting in denervation hypersensitivity. A plethora of other animal models using a similar design followed over the years. A limitation of any such model is that the innervation of the lower urinary tract in many animals such as rodents is very different from that seen in a primate. Furthermore, how representative is an immature animal (often female) of the situation seen in an aging male and what is the nature of the underlying neuropathophysiological aetiology? In this context it is important to recall that detrusor overactivity is in fact uncommon in male patients presenting with a urethral strictures. With these reservations in mind, it is not surprising to recall that none of the animal models have ever resulted directly in the development of any new pharmaco-therapeutic agents.

Contemporary opinion supports the view that storage LUTS (OAB) are likely to be the consequences of altered afferent nerve stimulation consequent upon a combination of mechanical obstruction to the bladder, ageing and incidental neurological dysfunction [4]. This afferent hypothesis is supported by an old observations that if

Peer review under responsibility of Second Military Medical University.

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https://doi.org/10.1016/j.ajur.2017.12.003

lidocaine is injected into the prostate in a patient undergoing urodynamics then bladder overactivity is ameliorated [5]. It can be further hypothesised that in fact prostatic therapies such as transurethral resection of the prostate (TURP) and laser therapy that either ablate or resect the prostatic urothelium result in a deafferentation of the prostatic urethra which ameliorates the storage symptoms, which are indeed known to improve following such therapy.

There are significant ultrastructural changes in the obstructed bladder and it has been suggested that lower urinary tract obstruction in the male is the principal cause of bladder hypertrophy [6]. Conversely it must be remembered that in the female patient bladder overactivity and not obstruction is the principal cause of hypertrophy of the bladder [7]. Whilst some data published to date do support the hypothesis that obstruction is the primary cause of detrusor hypertrophy it is also very important to consider that detrusor overactivity is present in the majority of these patients. Furthermore, voiding only occupies a fraction of the bladder's functional time, and I would speculate that in the male, just as in the female, it is overactivity which is the principal cause of the bladder wall thickening which is also very evident in patients with neuropathic bladder dysfunction.

Conversely, it is important to consider the potential for detrusor underactivity being present. In this context the concept of "voiding efficiency" is of great importance (the ratio of post voiding residual to functional capacity [residual + average voided volume]); a voiding efficiency of >40% should be considered as the threshold at which consideration should be given to a pressure flow urodynamic assessment to exclude detrusor underactivity.

Patients should be evaluated holistically and urologists and other clinicians need to adopt a tailored approach to the management of LUTS. It is only by using this approach to the management of patients that we can move beyond the term BPH and the mechanistic concept of reaching for the prescription pad or surgical instruments to improve these patients' quality of life.

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