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Mini Review: A ligamentous explanation for overactive bladder symptoms as defined by International Continence Society in the female

[Autor's unedited version]

Peter Petros

University of Western Australia, Perth, Australia
Professorial Department of Surgery University of NSW St. Vincent's Hospital Sydney

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Corresponding author

Prof. Peter Petros, M.D.
pp@kvinno.com

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The International Continence Society (ICS) committee has defined Overactive bladder (OAB) as "a syndrome characterized by symptoms of urgency, with or without urgency incontinence, usually with increased daytime frequency and nocturia (increased night time urination). The term OAB can only be used if there is no proven infection or other obvious pathology". Though the ICS gives no anatomical basis for OAB, it is suggested that the cause is from the detrusor itself. In this review, urodynamic evidence is presented that OAB in the female may be a prematurely activated but normal micturition reflex, as predicted by the Integral Theory. Anatomically, a trampoline analogy explains how loose ligaments prevent the vaginal stretching so important for support of the bladder base stretch receptors which control the reflex. Surgical cure of OAB by repair of loose cardinal/uterosacral ligaments are an important proof that the origins of OAB are outside the bladder, laxity of the suspensory ligaments which in turn, inactivate the striated muscle vector forces which contract against them. This concept is not in conflict with the definitions of the ICS. Rather it provides a causative anatomical background to the definitions.

INTRODUCTION

Standardization of terminology (definitions) of symptoms and conditions of the pelvic floor has been a raison d'etre of the International Continence Society (ICS) committees for more than 40 years [1, 2]. OAB has been defined as follows [2].

Overactive bladder (OAB) is a syndrome characterized by symptoms of urgency, with

or without urgency incontinence, usually with increased daytime frequency and nocturia (increased night time urination). The term OAB can only be used if there is no proven infection or other obvious pathology.

Though etiology is sometimes suggested by the ICS [1, 2], generally, no specific anatomical basis is given for the majority of these definitions. The vast majority of bladder, bowel and pain conditions are considered as being incurable.

Messelink [3] commented on the importance of definitions and guidelines as follows:

There can be no guidelines without definitions, and no definitions without understanding the item that is defined. The definitions and terminology we use reflect our present knowledge. Definitions and terms are of utmost importance because they are the basis for the language we use.

"Understanding the item" [3], clearly involves an understanding of normal anatomy and physiology of the pelvic floor, and how it can go wrong.

METHODS

This review is confined to 'Overactive bladder' (OAB) as defined by the ICS. The aim of this work is to review existing studies of anatomy, physiology and surgery relative to an anatomical basis for OAB and to discuss a way forward for cure. The 2013 ICS fact sheets [2] and 2002 publication [1] and recent CEJU publication by Liedl et al. [4] were chosen as reference points.

Evidence

ICS definition OAB ('overactive bladder) symptoms consist of frequency, urgency/ urge incontinence, nocturia [2]. According to [2], "the symptoms of OAB are suggestive of detrusor (bladder muscle) overactivity."

Analysis If what [2] states is correct, it would not be possible to cure OAB symptoms surgically. Yet cure rates of up to 90% for frequency and urgency have been published in patients following repair of cardinal/uterosacral ligaments [4–11].

Anatomical and physiological explanations for an anatomical origin of OAB

As part of the 1990 Integral Theory, frequency, urgency and nocturia were defined as being manifestations of a prematurely activated, but normal micturition reflex [12]. It was hypothesized that loose suspensory ligaments, pubourethral anteriorly and uterosacral posteriorly, weakened the musculoelastic stretching mechanism of the vagina which supported the bladder base stretch receptors 'N', Figure 1. In 1993, it was demonstrated that the events occurring in patients with DI (urodynamic 'detrusor instability'), now 'DO', were identical with those occurring during micturition, first sensory urgency, then urethral relaxation, then detrusor contraction, then urine loss [13]. In a further study [14], in a small group of patients, it was possible to control urodynamic "DI" by digital support of the bladder base. This would not be possible if the problem was in the detrusor itself [2]. This experiment also confirmed that the activator of the urodynamic DO were peripherally located stretch receptors, 'N', Figure 1.

Surgical evidence of the anatomical origin of OAB

A recent study of 611 patients who had a TFS posterior sling repair for uterine/apical prolapse found a high co-occurrence of chronic pelvic pain, bowel symptoms and OAB symptoms [4].

The results following posterior TFS sling were 90% prolapse cure in 611 patients. Symptom incidence in these 611 patients (% Cure at 12 months in brackets) was: urge incontinence: n = 310 (85%); frequency: n = 317 (83%); nocturia: n = 254 (68%); chronic pelvic pain (CPP): n = 194 (77%); fecal incontinence: n = 93 (65%).

Though co-occurrence of CPP, bladder & bowel dysfunction was convincingly demonstrated by [4], it has been demonstrated that patients tend to complain of mainly one of these symptoms [15]. This phenomenon has been called the 'Pescatori Iceberg' after Pescatori who first reported it. For example, in 198 patients who presented with CPP [15], 127 had frequency, 55 urge incontinence, 63 nocturia, 59 obstructive defecation, 66 USI, 44 residual urine, 68 bladder emptying symptoms, 56 fecal incontinence. It follows from this that any patient who has any type of uterine/apical prolapse is likely to have OAB symptoms and that these are potentially curable surgically.

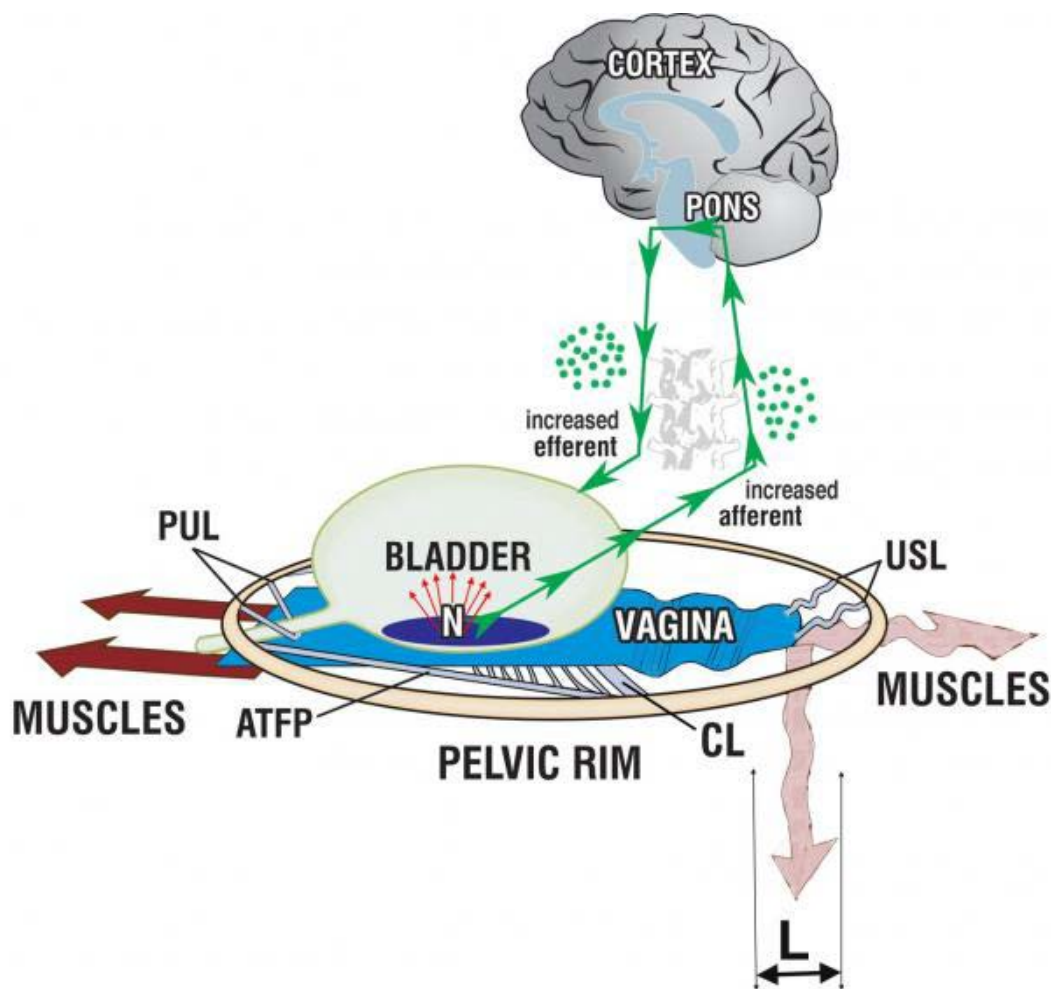
The importance of competent posterior ligaments

Urge incontinence concerns the symptom 'inability to hold on' [1, 2]. This begs the questions, 'hold on to what'? Clearly inappropriate activation of the micturition reflex, which is the Integral Theory's definition of OAB, fig1. The role of uterosacral ligaments in OAB and fecal incontinence (FI) cure was discussed more comprehensively recently [16]; the posterior vectors (arrows), Figure 1, act to close and open both the urethra and anus [17] and both act against the uterosacral ligaments (USL) [18]. Therefore weakness in USL explains the co-occurrence of OAB and FI symptoms [4] and how both are potentially curable by shortening and reinforcing USLs, as demonstrated [4]. As regards nocturia, a similar mechanism to fig1 holds. To visualize this, turn Figure 1 90 degrees. The patient is supine and the pelvic muscles are relaxed. As the bladder fills, it stretches downward by the force of gravity. Normally intact USLs support the volume of urine. If USLs are weak, as the volume of urine increases, the bladder base continues to descend. This stimulates the stretch receptors 'N' which in turn activate the micturition reflex. The patient is woken by an urge to pass urine. Conclusions The evidence for an external mechanism for causation of OAB and OAB as a prematurely activated micturition, but normal micturition reflex as described in this work is strong if not overwhelming. This concept is not in conflict with the definitions of the ICS. Rather it provides a causative anatomical background to the definitions.

Conflicts of interest

The authors declare no conflicts of interest.

Figure 1. If the uterosacral ligaments (USL) lengthen say by 'L', USLs are unable to suspend the vagina adequately. The downward/backward muscle forces (arrows) weaken as a muscle requires a firm anchoring point to contract efficiently. The muscle forces (arrows) cannot stretch the vagina sufficiently to support the stretch receptors 'N'. 'N' fire off increased afferent impulses at a low bladder volume and this is perceived by the cortex as urgency. If the number of afferent signals is sufficient to activate the micturition reflex, the efferents are activated; the forward muscles relax; the backward muscles open out urethra; bladder contracts; the patient may uncontrollably lose urine ('urge incontinence').



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