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### Editorial Comment on: Urinary Incontinence at Orgasm: Relation to Detrusor Overactivity and Treatment Efficacy

Hann-Chorng Kuo

Department of Urology, Buddhist Tzu Chi General Hospital and Tzu Chi University, Hualien, Taiwan  
hck@tzuchi.com.tw

The pathophysiology of overactive bladder (OAB) is complex. Disturbances in nerves, smooth muscle, and urothelium can cause urgency sensation and detrusor overactivity (DO) [1]. Treatment targeting at muscarinic receptors, presynaptic cholinergic fibers as well as suburothelial sensory receptors have been shown effective on OAB and DO [2,3]. This paper [4] is interesting in finding a high incidence of DO in patients with coital urinary incontinence (UI) at orgasm compared to that occurring during penetration (65.4% vs. 15.7%). However, treatment with antimuscarinics by tolterodine ER failed in 41.2% of patients with coital incontinence at orgasm and urodynamically

proven DO compared to 17% in a group of controls. On the other hand, women with coital incontinence during penetration are more likely to have urodynamic stress incontinence (48.2% pure stress UI and 13.2% mixed UI) and five women (45%) with DO did not respond to antimuscarinic treatment.

From the pathophysiologic viewpoint, orgasm might be a trigger of DO and penetration might potentiate the occurrence of UI in women with intrinsic sphincter deficiency or cystocele. The results of this study seem to provide evidence for this hypothesis. Interestingly, women with DO and coital incontinence either at orgasm or during penetration have a high failure rate of antimuscarinic treatment. This result highlights that a different pathophysiology may exist in women with coital incontinence and DO. Bladder base and trigone locate in the anterior aspect of vagina and are rich in sensory nerve supply. Previous study has shown an increase in vanilloid receptors at the trigone in patients with sensory urgency [5]. Injection of botulinum toxin A into

the bladder base and trigonal has been effective in treating patients with refractory DO [6], suggesting that dysfunction in the bladder base might mediate at least a part of the patient's DO. During intercourse, the bladder base is easily affected either during penetration (external compression) or at orgasm (contraction of pelvic floor muscles). DO mediated by neurologic dysfunction might occur without a distended bladder. Since this triggered DO is mediated by other than muscarinic receptors, antimuscarinic treatment will not be effective.

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## Editorial Comment on: Urinary Incontinence at Orgasm: Relation to Detrusor Overactivity and Treatment Efficacy

Thierry Roumeguère

University Clinics of Brussels, Department of Urology, Erasme Hospital, Brussels, Belgium  
[thierry.roumeguere@ulb.ac.be](mailto:thierry.roumeguere@ulb.ac.be)

Overactive bladder (OAB) is a highly prevalent condition with a significant impact on sexual function [1,2]. Coital urinary incontinence is a more common symptom reducing sexual desire and ability to achieve orgasm than currently suspected and affects the quality of life of sexually active women [3,4]. Only little information is available in the current literature on the relationship to intercourse regarding penetration or orgasm. Serati et al [5] should be commended for their study. They deal with the underlying pathophysiologic features of incontinence during sexual intercourse and provide interesting data on detrusor overactivity (DO) and orgasm-associated incontinence. For the first time, the authors evaluate pharmacologic treatment efficacy (with tolterodine ER) in patients with this specific form of incontinence. Nearly 20% of women suffer from intercourse urinary incontinence and at least one third during the orgasm phase with a high

prevalence of DO. Treatment efficacy is significantly lower in patients presenting urinary incontinence at orgasm compared to controls. Orgasm has been considered as a trigger for involuntary detrusor contractions [5], but we do not have any rationale to support the hypothesis of incontinence at orgasm to be considered as a more severe form of DO and that orgasm incontinence is a negative predictive factor for antimuscarinic treatment of OAB.

This paper reflects, among other things, the importance of urodynamic assessment to improve the treatment cure rate. Comparison of urodynamic parameters between responders and non-responders would have been helpful as would an evaluation of the urethral function lacking in this study. Further research is still needed especially on the potential role of urethral incompetence. It is difficult to choose the most appropriate treatment because the clinical entity remains incompletely defined. It is also matter of course to remind us to assess the impact of OAB on sexual function before and after treatment via validated questionnaires.

Clinicians should routinely assess OAB-related anxiety about sexual health and the impact of this concern on sexual intimacy when caring for patients with OAB because women do not necessarily nor easily initiate discussion of sexual issues with their physicians.