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## Letter to the Editor

**Reply to Bernhard Liedl, Klaus Goeschen, and Florian Wagenlehner's Letter to the Editor re: Benoit Peyronnet, Emma Mironska, Christopher Chapple, et al. A Comprehensive Review of Overactive Bladder Pathophysiology: On the Way to Tailored Treatment. Eur Urol 2019, 75:988–1000**

We welcome the important comments raised by Liedl and colleagues and would like to thank them for their interest in our recently published manuscript [1]. This comprehensive review of overactive bladder (OAB) pathophysiology was meant to stimulate such discussions and to promote research in the field. We fully concur that the causative link between pelvic organ prolapse (POP) and OAB symptoms has been largely demonstrated [2]. From a broader perspective, we believe that OAB is a nonspecific symptom complex to which anything affecting the bladder can contribute to the pathogenesis, including lower urinary tract prolapse.

The relationship between POP and OAB has mostly been attributed to the prolapse-induced bladder outlet obstruction (BOO) that may trigger histological changes in the bladder wall resulting in myogenic detrusor overactivity. The other mechanism that has been postulated to underpin this relationship is that the prolapse would open the bladder neck/proximal urethra and stretch the bladder trigone, with urine stimulating urethral afferents and triggering uninhibited detrusor contraction through the urethrovesical reflex [2]. These two mechanisms nicely fit in the myogenic and urethrogenic hypothesis that we set out in our review [1].

While we fully agree on the inclusion of POP as a possible cause of OAB, we would strongly advocate against considering that this relationship is exclusive. As we strived to demonstrate, OAB pathophysiology is highly complex and is multifactorial in nature [1]. The view that OAB symptoms in all female patients with POP should be attributed to the prolapse would be looking through the wrong end of the telescope.

Likewise, we disagree that OAB symptoms "can be predictably cured" by POP repair. Evidence on the outcomes for OAB symptoms after POP repair remains scarce, and mostly comprises small retrospective series lacking use of validated questionnaires [2]. Much lower rates of OAB symptom resolution after POP surgery than the 60–80% that Liedl and colleagues referred to have been reported in the literature (as low as 10% [3]), underscoring that POP repair cannot be regarded as the appropriate treatment for OAB for all these patients. In their review, de Boer and Vierhout [4] concluded that it is still impossible to reliably predict the presence of OAB after POP surgery. Future research assessing preoperative predictors of post-POP OAB improvement might help in further deciphering this relationship and improve the selection of optimal candidates for surgery.

In summary, we clearly recognize the validity of the point Liedl et al raised on the plausible implication of POP in OAB pathogenesis and on the possibility that OAB symptoms resolve after POP repair. However, we would temper their enthusiasm regarding the proportion of patients who could be "cured" of their OAB symptoms by POP surgical repair owing to the heterogeneity of the literature on that topic. Most importantly, we would encourage providers to avoid tunnel vision regarding OAB pathophysiology and therapeutic management, as there is compelling evidence of its multifactorial nature and the need for an individualized treatment approach.

***Conflicts of interest:*** Benoit Peyronnet is a consultant for Astellas, Allergan, Medtronic, and Boston Scientific. Christopher Chapple is a consultant, researcher, and speaker for Astellas, Allergan, Pfizer, and Medtronic; has received personal fees and nonfinancial support from Allergan and Pfizer, and grants, personal fees, and nonfinancial support from Astellas. Jean-Nicolas Cornu is a consultant/speaker for Astellas, Pfizer, Boston Scientific, Medtronic, and Bouchara Recordati.

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Benoit Peyronnet[a,*]
Christopher Chapple[b]
Jean-Nicolas Cornu[c]

[a]*Department of Urology, University Hospital of Rennes, Rennes, France*
[b]*Department of Urology, Sheffield Teaching Hospitals, Sheffield, UK*
[c]*Department of Urology, University Hospital of Rouen, Rouen, France*

*Corresponding author. Service d'Urologie, Hopital Pontchaillou, 2 rue Henri Le Guilloux, 35000 Rennes, France.
E-mail address: peyronnet@chu-rennes.fr (B. Peyronnet).