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# A NEW METHOD OF INDUCING DETRUSOR OVERACTIVITY IN CONSCIOUS RATS 3 DAYS AFTER RETINYL ACETATE INSTILLATION.

Hypothesis / aims of study
A credible animal overactive bladder model used in basic research is an indispensable harbinger of safe and ethical clinical trials on human subjects [1]. Our objective was to develop a new animal model of a hyperactive bladder that will be void of inflammatory urothelium lesions and display significant sensitivity to muscarinic receptor antagonists.

Study design, materials and methods
To examine the influence of 0.75 % retinyl acetate solution on cystometric parameters, it was infused into the bladder for 5 min [2]. Cystometric studies with physiological saline were performed in conscious unrestrained rats 3 days later. To examine the influence of retinyl acetate, acetic acid or cyclophosphamide on morphology of urinary bladders, the bladders were subjected to histopathological examination.

Results
We demonstrated that in rats subject to previous 5-minute bladder instillations with retinyl acetate, an increase of basal pressure, threshold pressure, micturition voiding pressure, bladder contraction duration, relaxation time, detrusor overactivity index, nonvoiding contractions frequency and amplitude occurs. On the other hand, a decrease in voided volume, post-void residual, volume threshold, voiding efficiency, intercontraction interval, bladder compliance and volume threshold to elicit nonvoiding contractions was observed. Administration of oxybutynin chloride (0.5 mg/kg, i.v.) reversed changes of cystometric parameters evoked by retinyl acetate. Contrary to acetic acid and cyclophosphamidete, bladders subjected to retinyl acetate infusion had no signs of bladder inflammation.

Interpretation of results
The results obtained indicate that transient infusion of 0.75% retinyl acetate can induce detrusor overactivity, which is often observed in patients with overactive bladder syndrome (OAB). In addition, it was demonstrated that stimulating afferent C-fibres using retinyl acetate did not induce evident histopathological inflammatory lesions in the urinary bladder wall.

Concluding message
It appears that in the future this model can prove useful in gaining more knowledge on the pathophysiology of OAB, and contribute to the preparation of new, more effective options of OAB pharmacotherapy.

References
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2. 2. Andaloussi-Lilja, J., Lundqvist, J., & Forsby, A. (2009). TRPV1 expression and activity during retinoic acid-induced neuronal differentiation. Neurochemistry International, 55(8), 768–774.

Disclosures
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