209

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MODELIZED ANALYSIS OF UROFLOWS: A WAY TO UNDERSTAND THE MOTOR NEURONS ACTIVITY DURING DETRUSOR EXCITATION.

Aims of Study

The nervous efferent pathways to the detrusor during micturition are well known. Some models assume an all-or-none behavior of the M-region [1-2]. Unfortunately, it is difficult to apply these models in order to explain the evolution of the flow rate and of the detrusor pressure in a standard voiding and obviously almost impossible in case of lower urinary tract dysfunction.

Our goal was to use recorded pressure-flow studies (PFs) as experimental results (and with some assumptions free flow (FF) recordings) in order to allow a quantitative study of the detrusor excitation and to propose a description of the firing of the motor neurons.

Methods

The isovolumetric detrusor force versus time during voiding can be directly evaluate at each time from the simultaneous measurement of the intravesical pressure, the flow rate and the bladder volume. We call "excitation-force" E_F the ratio between this force and the maximum force. From the well known studies of the calcium turnover in the muscular cell, we can link E_F to the concentration of the free Ca^{2+} ions in the cytosol. We call "excitation-calcium" E_{Ca} this concentration expressed in an appropriate unit. Then, we establish a relation (which will be the same for all patients) between the number of firing motor neurons at a given time and E_{Ca}. We call "recruitment ratio" rr the ratio of firing motor neurons (or, more precisely the ratio of the mean firing frequency to the maximum frequency).

Using a computer analysis of urodynamic tracings (VBN method [3]), the evolution of E_F, E_{Ca} and rr versus time was evaluated from each of the PFs of 107 men with lower urinary tract symptoms (LUTS) due to benign prostatic enlargement (BPE) and of 52 women with stress urinary incontinence (SUI). All patients were neurologically normal individuals. Excitations were also evaluated from FF of healthy volunteers of both sexes, assuming a normal detrusor function.

Results

1) For all the voids of healthy volunteers, 53% of these of men with BPE and 25% of women with SUI, we find an all-or-none behavior for the recruitment ratio: rr = 0 during the continence phase and rr = 1 till the end of voiding. Then E_F and E_{Ca} are slowly increasing curves which explains the slow increasing part of the bell shaped flow rate curve (while the decreasing part can be related to the decrease of the bladder volume).

2) For all the other cases, the recruitment ratio exhibits a two steps behavior: rr = 0 for t = 0, rr = 1 (as in the standard case) at the beginning of the flow. Then, at a critical time t_c, rr takes a value R ($0<R<1$) which remains constant till the end of the flow. Looking at E_F and E_{Ca}, this phenomenon appears as a fading of the detrusor excitation. The consequence is a sudden change of the slope of the flow rate curve at the time t_c. The mean value of R is 0.53 ± 0.21 for men and 0.41 ± 0.18 for women; these mean values do not differ significantly despite the difference in the origin of LUTS. On the opposite, the critical time differs significantly: 9.7±2.5s for men and 4.0 ± 2.7s for women.

Conclusions

An accurate analysis of the PFs recording (VBN method) allows to observe and quantify the variation of the nervous excitation of the detrusor during micturition. At our knowledge, no other experimental way to obtain such information exists. By applying this method to a large number of patients we bring to the fore one systematic pattern of the nervous control of the detrusor excitation: 1) in the standard case the course of excitation is the same for man and woman, 2) in many cases of patients without nervous desease, a fading of the excitation occurs, later for men than for women, which could be related to a feedback starting at the urethral level, 3) observed smoothly increasing or decreasing detrusor excitations can be related to sudden changes in the number of firing motor neurons.

References

[1] Neurourol Urodyn 1990. 9: 601-18; [2] Neurourol Urodyn 1998. 17: 175-96; [3] Neurourol Urodyn 2000. 19: 153-76.