Nonselective Blocking of the Sympathetic Nervous System Decreases Detrusor Overactivity in Spontaneously Hypertensive Rats

Introduction and Objective: The detrusor overactivity (DO) in OAB represents overactive parasympathetic activity, because the parasympathetic nervous system is responsible for the detrusor contraction. However, it remains unknown whether sympathetic activity, the opposing partner in bladder function, is increased or decreased during DO in patients with OAB. The present study was undertaken to compare bladder function and some characteristics of DO during the filling phase before and after the administration of the nonselective sympathetic blocking agent labetalol in spontaneously hypertensive rats (SHRs).
Materials and Methods: We used SHRs, which are a model system for studying DO and altered sympathetic activity. The involuntary dual control systems of the autonomic nervous system (ANS) in the bladder of awake SHRs were investigated through simultaneous registrations of intravesical and intraabdominal pressures to observe detrusor overactivity (DO) objectively. We checked on the cystometric detrusor overactivity-related parameters such as DO occurrence rate, DO frequency and DO pressure during filling phase in SHRs after intraarterial labetalol injection.
Results: SHRs showed the features of overactive bladder syndrome during urodynamic study, especially DO during the filling phase. After injection of the nonselective sympathetic blocking agent labetalol, DO disappeared in 3 of 6 SHRs (50%). DO frequency decreased from 0.98 ± 0.22 min^{-1} to 0.28 ± 0.19 min^{-1} ($p < 0.01$), and DO pressure decreased from 3.82 ± 0.57 cmH_2O to 1.90 ± 0.86 cmH_2O ($p < 0.05$). This suggests that the DO originating from the overactive parasympathetic nervous system is attenuated by the nonselective blocking of the sympathetic nervous system.
Conclusions: We demonstrated herein that intraarterial labetalol, a nonselective sympathetic blocking agent, suppresses the occurrence, frequency, and pressure of DO in SHRs. This suggests that the DO from the overactive parasympathetic nervous system requires compensatory overactive sympathetic nervous system activity in a kind of balance between these two systems.