

DOES THE NEOBLADDER FILLING ACTIVATE VISCERO-SOMATIC REFLEXES? A NEUROPHYSIOLOGICAL AND URODYNAMIC INVESTIGATION

Hypothesis / aims of study:

Unlike preoperative condition, patients with neobladder urinate when they feel an increased pressure in the pelvis; micturition is performed by straining the abdomen and relaxing the perineum. This type of voiding is due to the loss of physiological micturition reflex which is activated by afferent unmyelinated C fibers and finely myelinated A delta fibers connected to the slow adapting mechano-receptors of the bladder wall in healthy subjects. Previous studies showed that bladder filling induces changes in the excitability of somatic spinal motoneurons as tested with the soleus H reflex study and suggested that this modulation takes place at spinal level possibly through propriospinal pathways. Overall the above studies also suggested that spinal motoneurons inhibition arises from bladder afferent input during bladder filling. Whether spinal motoneurons modulation during bladder filling at least partly depends also on an increase of abdominal pressure is unknown. Basing on these assumptions, the aim of this study was to evaluate whether ileal neobladder distension also affects the excitability of spinal motoneurons.

Study design, materials and methods

We performed soleus muscle H reflex study during ileal neobladder distension in 8 male patients (age range 64-72 years, mean 67,5) with ileal orthotopic neobladder. Patients underwent the H-reflex study during urodynamic assessment. Because evidence in humans suggests that hip angle and forearm positions are critical for soleus and flexor carpi radialis (FCR) H-reflex modulation, for H reflex recordings and urodynamic assessment subjects laid supine in a gynaecological position with the forearm in pronation. Participants were instructed to relax the forearm and keep the head in a neutral position during recordings. Median nerve stimulation was performed with an electromyographic (EMG) device by placing an Ag/AgCl surface stimulating bar electrode with coupling gel at the distal third of the upper arm over and in line with the median nerve, proximal to cubital fossa. The active electrode was positioned proximal to the reference electrode to avoid anodal block. The stimulating electrode delivered percutaneous electrical stimuli of 1.0-millisecond square-wave pulses. The stimulation intensity was adjusted to evoke a low-amplitude M-wave and a 50% maximum H-reflex. EMG signals were recorded from the FCR muscle (bandwidth 20 Hz–10 kHz) using Ag/AgCl surface recording bar electrode with coupling gel which was placed over the belly of the FCR to record the H-reflex amplitude and M-wave of the muscle, with the active electrode approximately over the FCR motor point and the reference electrode 2 cm lateral to it. One 2-cm (diameter) round metal ground electrode was placed on the lateral aspect of the cubital fossa between the stimulating and recording electrode sites. Ten trials were collected and then averaged for each condition. Each trial was repeated at 10 seconds interval. H-reflex and M-wave size were measured peak-to-peak.

Soleus H reflex

Electrical stimuli were delivered to the right tibial nerve (1.0 ms duration square-wave pulses) with an electromyographic (EMG) device through monopolar needle electrodes placed in the popliteal fossa (soleus H reflex). For the H-reflex study, stimulus intensity was adjusted to evoke a low-amplitude M-wave and a 50% maximum H-reflex. To check the efficacy and stability of nerve stimulation, the M-wave size was measured when H-reflex recording began and monitored throughout testing. EMG signals were recorded from the soleus-muscle using Ag–AgCl surface electrodes (bandwidth 20 Hz–10 kHz). Ten trials were collected and then averaged for each condition. Each trial was repeated at 10 seconds intervals. H-reflex and M-wave size were measured peak-to-peak.

Urodynamic investigation

A double lumen 6 Fr catheter was inserted in the neobladder; abdominal pressure was derived from a rectal balloon inserted in the rectal ampulla. Sterile saline solution at room temperature was used to fill the neobladder through a cystometric speed infusion of 30 mls/min.

Experimental paradigm

The H-reflex was tested under two bladder filling conditions: empty neobladder (control value) and maximum neobladder capacity (when subjects felt they can no longer delay neobladder voiding or reported pain).

Statistical analysis

Statistical analysis was based on Wilcoxon matched paired test; the R² value was calculated to analyze the correlation between the neobladder filling and changes in the FCR and soleus muscle H-reflex amplitude.

Results

Clinical and Urodynamic outcomes

Date from surgery ranged from 8 months to 7 years. Mean neobladder capacity resulted 235.5±18 mls and mean neobladder pressure 29±7 cmH₂O. Two subjects presented residual peristaltic activity (maximum amplitude, respectively, of 23 and 45 cmH₂O) without urinary incontinence episodes. Post void residual volume was not significant in all the individuals (mean value 10.75±8.8 ml).

Neurophysiological evaluation

Median nerve electrical stimulation elicited an FCR H reflex at empty bladder with a mean amplitude±SD 0.63±0.17mV. Tibial nerve electrical stimulation elicited a soleus H reflex at empty bladder with a mean±SD amplitude 2.90±1.09mV. Paired sample T test showed that the amplitude of FCR H reflex and soleus H reflex did not statistically differ when tested at empty neobladder and at maximum neobladder capacity (FCR H reflex p=0.88; Soleus H reflex p=0.83).

Interpretation of results

No studies have yet investigated viscerosomatic reflexes of the small intestine in humans. Our findings showed that neobladder filling left the H-reflex size unchanged suggesting that bladder filling-induced modulation on H reflex size, previously reported, was actually due to bladder afferents activity and did not depend on an aspecific increase in pelvic and abdominal pressure during micturition. Furthermore the results suggest that small intestine afferents do not modulate spinal motoneurons excitability, as shown by the lack of changes in the H reflex size from the FCR and soleus muscles. Whether small intestine afferent-induced modulation on spinal motoneurons is weaker than bladder filling-induced modulation making it undetectable with our techniques remains unknown. The lack of neobladder filling induced modulation on somatic spinal motoneurons might be partly be responsible for the absence of a real desire to void in patients with neobladder and might suggest that increase of pressure in the pelvis, probably transmitted by mechanoreceptors of abdominal wall, makes them deciding to void. For this reason, even if they may maintain corporeal image, patients with bladder reconstruction need to continuously used timed-voiding to avoid excessive distension of the neobladder and, for the micturition, are prompted to strain with the abdomen previously relaxing perineum in the way to completely empty the neobladder limiting the risk of residual urine.

Concluding message

This experimental investigation showed that in humans the filling of ileal orthotopic neobladder does not determine any modification of H-reflex amplitude, therefore suggesting that in humans ileal afferent pathways do not modulate spinal motoneurone's activity.

References

1. . Alrowayeh HN, Sabbahi MA, Etnyre B: Flexor carpi radialis H-reflex modulation during spinal loading and unloading with varied forearm postures. J Clin Neurophysiol 2010;27(2):116-9.

Disclosures

Funding: None **Clinical Trial:** No **Subjects:** HUMAN **Ethics Committee:** ASL Latina **Helsinki:** Yes **Informed Consent:** Yes