511

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AN EXPERIMENTAL APPROACH ON THE PATHOPHYSIOLOGY OF DETRUSOR SPHINCTER DYSSYNERGIA

Hypothesis / aims of study

During normal micturition the relaxation of external urethral sphincter (EUS) is thought to be related to pontine micturition center (PMC) projecting to sacral inhibitory interneurons in the dorsal grey commissure that in turn modulate the EUS motor neurones of the Onuf's nucleus (ONu)[1]. Despite this longer motor pathway due to sacral interneurons EUS relax before the onset of bladder contraction. That is partly explained by higher nerve conduction velocity in efferent somatic fibres than in efferent parasympathetic fibres.

Animal studies suggest that recovery of bladder function following spinal injury depends upon the reorganisation of reflex pathways both in peripheral and central nervous system and leads to overactive bladder and detrusor sphincter dyssynergia (DSD).

It is assumed that the normal spino-bulbo-spinal micturition reflex is replaced by a spinal detrusor to detrusor reflex. However, very few is known about how this new reflex loop interacts with EUS motor neurones.

Animal observations in spinal transected cat have shown that the synaptic input to sphincter motor neurons is altered and in SCI patient with DSD EUS demonstrates hyperexcitability.

One hypothesis of mechanism of DSD could be that afferent input from the bladder, conveyed by C fibres to the intact sacral cord, stimulates parasympathetic motor neurons and concomitantly stimulates, instead of inhibits motor neurons of ONu, via new interneurons and/or new synaptic connections and/or changes in neurotransmitters. By higher nerve conduction velocity in efferent somatic fibres than in efferent parasympathetic fibres EUS should theoretically start being activated before bladder contraction even having taken into account increased intraspinal nerve conduction time.

The aim of our study was to evaluate, using urodynamics (UdS), if EUS contraction indeed precedes bladder contraction in DSD and by how many times.

Population and study design

We retrospectively reviewed the UdS of 25 consecutive complete SCI patients (January 2002, December 2003), and selected untreated patients that demonstrated neurogenic overactive bladder and DSD. **Delay A** was defined as the time period between the onset of EUS pressure increase and the onset of bladder pressure increase, at the first uninhibited detrusor contraction, **Delay B** as the time period between the onset of urethral sphincter pressure increase and the moment in which bladder pressure increased 10 cmH2O above the baseline value. We measured same delays at the second and third UIDC in the same UdS, and if available in a second UdS.

Results

25 patients demonstrated overactive bladder and DSD, 5 were excluded because of artefact during UdS.

Delay A was positive (>0sec.) in 16/20 patients (80%).

Mean delay A was 2.2sec. (sd:3.3, median:2.1, IQR:0.8-3.5).

10/20 patients had a second UIDC during the initial UdS and 9 a third one. Delay A was positive at the first UIDC in 7/10, at the second UIDC in 9/10 and at the third UIDC in 7/9. 10/19 patients have had a second UdS that matched selection criteria (mean time between UdS: 22 months).Delay A was positive at the first UdS in 8/10 patients and at the second UdS in 9/10 patients. Mean Delay A at first UdS, in these 10 patients was 1.5 s. (sd: 1.8, median: 1.2, IQR: 0.8-2.2) and 2.0 s. (sd: 3.1, median: 2.5, IQR: 1.1-4.4) at second UdS. There was no significant difference between Delay A at first and second UdS when compared by wilcoxon signed ranks test.

Delay B was positive (>0sec.) in 20/20 patients (100%). Mean delay B was 7.6sec. (sd:5.9, median:5.5, IQR: 3.8-8.4). Delay B was positive at all the UIDC in all patients and at the two different UdS.

Interpretation of results

Our results on retrospective data support the hypothesis that EUS contracts before bladder in DSD and the delay is approximately two seconds.

By common afferent pathways EUS contraction preceding bladder contraction can only be explained by difference in latencies in efferent motor pathways. The time between spinal activation of parasympathetic sacral centre and bladder activation is approximates according to data from Brindley anterior root stimulation around 1 to 3 seconds. On the other hand latency between spinal activation of ONu to EUS activation has been evaluated after lumbar magnetic stimulation and varies from 8 to 20 milliseconds (needle EMG or urethral pressure recording)[2]. This difference in estimated latencies has to be explained by the difference of myelinisation on autonomic and somatic fibres and is coherent with our findings. We need to confirm our retrospective findings in a prospective study recording both EUS pressure and EMG in parallel to bladder pressure.

Concluding message

In complete spinal cord injured patients with DSD, EUS activity seems start before the onset of bladder pressure increase and always before a bladder pressure increase of 10 cmH2O more than baseline. Thus sphincter activity could reliably predict the onset of uninhibited bladder contraction.

References

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