



## **Physiology :**

The possible causes of an overactive bladder are still not completely understood. A common subdivision differentiates overactive bladder syndrome and urge incontinence into neurogenic and non-neurogenic[3] . \_\_\_ In terms of differential diagnosis, undiscovered infections that cause no characteristic symptoms (chlamydia, mycoplasma, trichomonas, etc.), central and peripheral nervous disorders [4] , changes in bladder outlet and psychosomatic causes should be discussed. Basically, the detrusor undergoes a normal aging process, which can also lead to overactive bladder syndrome.

For example, in the bladder of the elderly, there is complete penetration of the submucosa and the surrounding neurovascular and muscle bundles by collagen fibers, leading to a loss of elasticity of the detrusor [5] , [ 6 ] , [7] , [8] . Activation of stretch receptors in the urothelium and adjacent connective tissue results in increased afferent signals in the CNS, which leads to an efferent response in the form of urinary tract sensations. Under physiological conditions, afferent signals from the bladder are normally inhibited in the thalamus and prevent conscious perception in the CNS. When these central inhibitions are removed, the heightened stress signals enter consciousness and are perceived as a draining stimulus.

## **Detrusor Overactivity and Detrusor Neurogen Overactivity**

When classified by urodynamic features, detrusor overactivity (idiopathic) with overriding symptoms is prominent. Obviously, the center of sacral urination constantly generates sensory stimuli which reach the functional center of the hypothalamus by afferents. The imbalance between the strength of the afferent impulses and the central inhibition of the voiding reflex causes detrusor hyperactivity[9] . Neurogenic detrusor overactivity results from reduced central nerve inhibition of the voiding reflex, which may occur for example in MS, M. Parkinson or after stroke, and not respond to treatment with rMSP in anticipation of the following chapters.

Detrusor overactivity is often also symptomatic, namely, as already described, inflammation, tumors, anatomical features (Descensusvaginae) or estrogen deficiency.

## **OAB**

Overactive bladder is generally considered a precursor to dranine incontinence and is characterized by pollakiuria, nocturia, and imperative urinary urgency. There doesn't always have to be detrusor overactivity.

## **Integral theory**

The emergence of a female UI can be explained very well in the integral theory according to Petros [10] , [11] . Thus, the vagina in the overall structure of the female pelvic floor is considered a kind of trampoline, which is always in a state of elastic tension due to various ligaments and muscle trains located in the ring of the bony pelvis. If the bottom of the bladder now sinks by loosening the trampoline membrane (“pelvic floor insufficiency”), the bottom of the bladder can stretch independently of the filling of the bladder. That

increases the likelihood of irritation for dilator receptors concentrated at the bottom of the bladder, which transmit signals to the CNS via afferent pathways [12]. Normally, the body can counter the increased and unregulated signals via the central inhibitory centers, so that the trampoline runs stable again due to the contraction of the pelvic floor. But if the ligaments are too tight or loose, it is no longer possible for the muscles to contract the vaginal wall sufficiently. Thus, the expansion receptors located at the bottom of the bladder "pull" even at low filling volume. It should also be mentioned here that bladder discharge can also lead to urinary retention [13]. , [14].

### **Gate control theory**

Another explanatory model and perhaps a therapeutic point of attack arises from the "gate control theory" first described in 1965 for pain control [15]. . Thus, it is known that afferent bladder signaling can be inhibited by sacral medulla interneurons [16]. . Thus, the activity of the myelinated thick fibers blocks or closes the "gateway", thus inhibiting the passage of afferents from the peripheral bladder which circulate in the thin A-delta/C fibers. The thin fibers, on the other hand, open the gate, so that stimuli from the urinary tract are transmitted to the CNS [17]. The gate is inhibited or closed by the somatosensory fibers of the pudendal nerve, which contain both somatosensory and somatomotor nerve fibers.

### **Pelvic organ prolapse (POP) / uterine descent**

Vaginal prolapse is the sinking of the pelvic organs, which can affect both the vagina and the uterus or cervix (cervix) [18]. Affected individuals typically present to their gynecologist with symptoms of lower urinary tract (LURT), which may consist of stress urinary incontinence, but also urgency, pollakisuria, and urge incontinence [19].

It is significant that an overactive bladder (OAB) is more common in POPs (pelvic organ prolapse) and therefore has a causal relationship [20]. According to one study, 56-88% of patients with vaginal prolapse had symptoms of urgency, but they also had to offer 20-64% of patients without prolapse [21]. However, the relationship between prolapse and LUTS is rarely mentioned in the literature [22]. . This is also confirmed by the ICS (International Continence Society), according to which "LUTS are a major problem for women, but the cause of the symptom is unknown" [23]. After all, according to one study, lowering of the anterior vaginal wall or prolapse should have links to overactive bladder and correlate directly with the severity of overactive bladder [24].

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### **PelviCenter Rpms QRS effect :**

Early encouraging attempts at painless, non-invasive magnetic stimulation of the sacral nerves in the 1990s suggested that perineal magnetic stimulation or rMSP should also be used in urge incontinence. This is also related to the extended Integral Theory model, which allows pelvic floor training to restore the anterior architecture of the pelvic floor and lost trampoline function of the vagina, thereby reducing bladder filling pressure and the risk stimulation of

extensor receptors. However, this only applies to overactive bladder syndrome or UI where the cause is pelvic floor weakness.

Based on the tuning parameters or stimulus pattern of an rPMS, the frequency to be applied would be identical to the therapy of an SUI. However, if bladder irritation or impulsive impulsive conduction in the CNS is prominent in OAB/UI, rPMS training designed for pelvic floor support could not address the actual cause. Here, gate control theory should be included in the processing procedure. After an rPMS activates only the thick myelinated fibers of N. pudendus and thus closes the gate ("transmission in the sacral reflex arc"), so that afferents from the peripheral bladder are not transmitted to the thin fibers A-delta/C do not respond to rPMS, the transmission of the urinary urgency stimulus into the CNS is prevented.

It recalls the experience of each of us that, by "pinching" the buttocks (or the pelvic floor), a strong urge to urinate can disappear for a brief moment. The derived strategy is therefore to activate the branches of N. pudendus by an rPMS and thus inhibit or superimpose pathologically increased bladder irritation (the need-reducing effect of a strong pelvic floor via a negative feedback mechanism is in the literature). known since the 1970s).

Everything therefore depends on a frequency of stimulation which interrupts an independent signaling not directed towards the central nervous system. Here are the results of useful peripheral electrical stimulation, after a frequency of 5 Hz allows maximum bladder inhibition on sympathetic fibers. Also, frequencies between 5 and 10 Hz should lead to central inhibition of efferents (motor signaling) in the bladder or detrusor overactivity[25] , [26] , [27] , [28] . \_\_\_\_\_ . However, this does not include neurogenic bladder dysfunction such as paraplegia [29] . \_\_\_\_\_

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### **Treatment scope and treatment period :**

If OAB is accompanied by pelvic floor insufficiency (mixed incontinence), a treatment frequency of 2-3 times per week over a period of 6-8 weeks (16-20 applications) is recommended. Since the underlying cause of the disorder is usually unclear, treatment frequencies should be divided. For isolated overactive bladder, an increased frequency of treatment may be considered.

Choosing the right frequency setting and timing them is explained in the QRS Pelvicenter manual. These you receive as operator of a QRV Pelvicenter included. Likewise, the instructor will explain this topic in detail when setting up the device.

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### **Expectation of success :**

After 16-20 treatment sessions, 50% of OAB patients show a marked improvement in their symptoms and 10-15% are expected to recover. Therapeutic success occurs with increasing duration of treatment. In the majority of cases, patients first reported an improvement in sleep, followed by a gradual reduction in imperative urgency. Long-lasting therapeutic success can be expected for up to 6 months. It is therefore advisable to carry out a follow-up treatment (8 to 10 treatment sessions) a few months after the initial treatment.

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### **Place of study :**

For rPMS treatment of OAB or UI syndrome (OAB is the ending of CSI, urge incontinence is a symptom of OAB) so far there are 39 studies.

Of these, 11 studies have been presented at urology meetings, but the abstracts or posters have not been published. Additionally, it should be noted that many of the studies cited below relate to a mixed population of patients with stress, urgency, and mixed incontinence.

**Study 1** : prospective randomized double-blind multicenter study [30] \_\_\_\_

In this multicenter (randomized, double-blind) study, 151 women with II randomized 2:1 were randomized to stimulation and a sham group treated twice weekly for 6 weeks with treatment parameters: maximum flux density of 560 mT , frequency 10 Hz (5 s on / 5 s off) for 25 minutes each The shaver only works with a maximum flux density of 20.4% of the active device.

### **Result:**

Compared to baseline, the number of urge incontinence episodes per week in the active group decreased by  $-13.08 \pm 11.00$ ; placebo of  $-8.68 \pm 13.49$  ( $p = 0.038$ ). Exasperation periods decreased by  $-2.65 \pm 2.52$  ( $p = 0.011$ ) compared to placebo ( $-1.53 \pm 2.39$ ). Urine loss in the verum group decreased by  $14.03 \pm 34.53$  ml compared to placebo - by  $4.15 \pm 40.60$  ml ( $p = 0.0056$ ).

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**Study 2**: randomized double-blind multicenter study [31] \_\_\_\_

The patient population consisted of SUI, UI and mixed patients treated 3 times per week for 6 weeks (10 minutes 5 Hz / 3 minutes off / 10 minutes 50 Hz). For the placebo treatment, the intensity was reduced to zero. Patients were told that a scanner's silence procedure would work accordingly.

## **Result :**

Of the initial 49 patients, 33 patients (14 patients (58%) in the active group and 19 (76%) in the control group) completed the study. After patients drank 500 ml of fluid and waited 30 minutes of sitting, there was no urine loss (21% from baseline) in post-treatment patients in the verum group, while no change had been recorded in the shampoo group. The tampon weight was 2.59 g in the verum group and 14.6 g in the placebo group ( $p = 0.079$ ). Mean compress consumption decreased from 3.33 to 2.0 ( $p=0.02$ ) in the active group and remained unchanged in the placebo group. The quality of life score fell from 72.86 (initial value) to 84.69 ( $p=0.04$ ) in the active group and remained unchanged in the placebo group.

*Comment: The methodology and the presentation of the results, however, need to be discussed.*

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## **Study 3:** 37 patients with urgency or stress incontinence [32] \_\_\_\_\_

20 emergency patients (3 men, 17 women,  $\bar{X} 68.5 \pm 14.2$  years) and 17 patients with stress urinary incontinence. Detrusor contractions were observed in 8 patients at baseline. rPMS treatment took place twice a week for 8 weeks (10 min at 10 Hz - 2 min break - 10 min at 50 Hz).

## **Result (user interface only)**

In 25% of urge incontinence cases, healing was achieved. There was an improvement in 60% and no treatment in 15% ( $p<0.003$ ). In 7 of the original 8 patients, urodynamic detrusor contractions persisted. After 2 weeks, the amount of unwanted daily urine in the verum group was reduced from 5.6 to 3.6 per day. At 24 weeks, two patients were fully continental (no models) and four patients averaged less than one model per day.

In the vermic group, the functional capacity of the bladder (filling by deflation) increased from  $141 \pm 50.6$  ml before treatment to  $188 \pm 77.8$  ml after treatment ( $p < 0.018$ ). Mean I-QOL before treatment was 62.7 and increased to 77.8 at 4 weeks ( $p<0.004$ ). The VAS score also improved from 7.82 to 5.45 at 2 weeks ( $p<0.04$ ). 24 weeks after the last treatment, the result persisted in 9 out of 17 patients (52.7%). In 8 patients (47.1%), the previous symptoms reappeared. 3 of these 8 patients wanted more applications of rPMS.

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## **Study 4:** 48 patients with overactive bladder [33] \_\_\_\_\_

rPMS treatment was performed at a frequency of 10 Hz, twice a week for 20 minutes each over an 8-week period.

### **Result after 2 weeks:**

A total of 27 patients (56.3%) experienced a marked decrease in symptoms, with 33 of 48 (68.8%) experiencing urgency symptoms, 27 of 48 (56.3%) and 8 of 16 patients. (42.8%) improvement in urge incontinence. The average frequency of daily toileting decreased by 42.8% ( $p < 0.001$ ). The total urine volume remained the same, which increased the respective single urine volume accordingly.

### **Result after 24 weeks:**

Almost all 27 patients (96.3%) showed improvement in symptoms even after 24 weeks.

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### **Study 5:** 26 patients with overactive bladder syndrome [34] \_\_\_\_\_

Twenty-six patients (2 men, 24 women/39.5 years) with overactive bladder syndrome were studied. rPMS treatment was carried out twice a week at a frequency of 10 Hz over a period of 7 to 8 weeks.

### **Result after 8 weeks and 3 months:**

In 23 of the 26 patients, there was a significant decrease in urgency symptoms (frequency of toilet visits). Only in 2 patients showed no improvement. The average number of daily urinations decreased by 38.1% ( $p < 0.001$ ). Prior to treatment, 8 OAB patients (31%) reported one or more episodes of incontinence. After treatment, this was only the case in 4 patients. There was no significant change in bladder capacity from baseline.

### **Result after 6 months:**

After 6 months, the previously obtained result was maintained in 14 patients (93%) of the 15 evaluable patients. The number of daily urinations decreased from  $15.8 \pm 5.3$  to  $9.9 \pm$  ( $p < 0.001$ ).

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### **Study 6:** mixed study group [35]\_\_\_\_\_

The study population was 66 patients (23 men, 43 women) with mixed and compelling symptoms. The average number of male applicants was only  $7.7 \pm 3.8$  and  $10.4 \pm$  for females. The treatment itself was carried out for 20 minutes at lower and higher frequencies.

**Result :**

57.1% of men showed significant improvement and a further 9.5% slight improvement, while in 29% of cases the treatment had no effect. Among the women, only 35.1% showed a significant improvement and 37.8% a slight improvement, while in 24.3% of the cases the treatment had no effect. Before treatment, 66.7% of women complained of compelling symptoms, after treatment only 7.4%.

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**Study 7:** Prospective study [\[36\]](#)

Mixed incontinence group with 24 participants (12 cravings and 12 mixed). Patients with urge incontinence were treated at 10 Hz for 20 minutes with a 2 minute break, mixed patients at 10 and 50 Hz (twice a week for 8 weeks).

**Result :**

Unwanted urine loss improved in 58% of patients. A significant number of patients were satisfied with the treatment ( $p < 0.001$ ). 3 of 24 were completely continental after therapy. Of 12 patients with urge incontinence, the treatment was effective in 6 patients (50%) ( $p < 0.005$ ). In the mixed group, this was the case in 8 patients (67%) ( $p < 0.01$ ). Subjectively, 70% of all patients experienced improvement ( $p < 0.01$ ) – while 30% reported either no change or worsening of symptoms.

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**Study 8:** Mixed SUI/OAB Incontinence Group [\[37\]](#)

Mixed incontinence group consisting of 49 patients with SUI and 44 patients with overactive bladder. Only the OAB group is referenced here. 34 people completely completed the treatment. The therapy took place twice a week for 9 weeks. Treatment parameters were 50 Hz (3 seconds) pause (6 seconds), 10 Hz (3 seconds) 6 seconds pause.

**Result :**

The response rate ("marked improvement in overactive bladder symptoms") was 61.7% (21 of 34 people). If you include defaulters (intention-to-treat analysis), it's 47.7% (21 out of 44 people). According to the UDI-6 (Urogenital Distress Inventory), validated for older women and men [\[38\]](#) and questioning the symptoms and their degree of severity, and differentiating between obstructive and irritative symptoms, the mean score decreased from  $9.7 \pm 4.2$  (baseline) after 9 weeks to  $4.0 \pm 1.7$  ( $p < 0.01$ ). In the IIQ-7, the short form of the Impact Incontinence Questionnaire, which examined the negative impact of incontinence on daily activities, the score went from  $10.8 \pm$  (baseline) to  $3.5 \pm 4.4$ .

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## **Conclusion :**

OCC or urge incontinence is often associated with detrusor overactivity, which can result from inflammation, age-related bladder remodeling, pelvic floor sagging and tumors, or an imbalance between the influx. With mixed incontinence, this is the predominant age of incontinence, affecting 10.8% (men) and 12.8% (women), with a high incidence in the elderly .

If you include only those who are severely affected, that's 28% of all female patients and 45% of all patients over 60. Mixed incontinence accounts for 38% of all forms of incontinence.

The high rate of side effects and discontinuation of first-line propagated anticholinergic drugs, intravesical injection of botulinum toxin with a rapidly diminishing long-term effect despite repeated use or even surgical use of a neurostimulator of the sacral roots, represent a very important non-invasive and above all effective form of therapy such as rPMS.

With a treatment duration of only 16-20 applications (6-8 weeks), with significant improvement in symptoms or healing in 50-65% of all patients, rPMS should be the treatment of choice as therapy of OAB with more targeted side effects.

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