QRS 1010 Pelvicenter

Repetitive peripheral magnetic stimulation to correct functional pelvic floor disorders

Scientific documentation and medical information

Women with urge incontinence/OAB

Publisher:

Industriering 3 FL-9491 Ruggell Tel.: +423 798 80 88 / +31630916144 (WhatsApp)

E-Mail: emiel.spiessens@qrs-international.com / emiel.spiessens@gmail.com

YouTube: QRS GERMANY

QRS MedWiss Service

definition

The overactive bladder syndrome (OAB) - consisting of the imperative urge to urinate, pollakiuria, nocturia with and without urge to urinate (urge or urge incontinence) - is characterized by a sudden urge to urinate [1] , which forces you to go to the toilet even with small amounts of fluid. Consequently, the urge to urinate is paired with frequent voiding (pollakiuria) and this of course also at night (nocturia). The causes are uninhibited contractions of the detrusor (M. detrusor vesicae) and/or excessive bladder sensitivity [2] . The disease is usually diagnosed by excluding other disorders, such as urinary tract infections or other organic causes.

Epidemiology and Prevalence

The prevalence data differ considerably between the individual authors. We refrain from listing the various evaluations and the various reasons for the deviations. In the following information, we are concentrating on prevelance data from the so-called EPINCONT study, which is listed in the brochure of the Robert Koch Institute "Federal Health Reporting" [2] . This study usefully differentiates between different forms of urinary incontinence and also highlights the prevalence of "seriously affected".

Occurrence of urinary incontinence (all forms) in women overall:

under 30 years	12% (light 57%, moderate 31%, heavy 12%)
between 50 to 54 years	30% (light 46%, medium 33%, heavy 21%)
over 90 years	40% (light 24%, medium 31%, heavy 44%)

Differentiation between the forms of urinary incontinence:	
stress incontinence	50%
Urge incontinence/OAB	**11%**
mixed incontinence	36%
Other	3%

The prevalence data of the highest severity by age subdivision:	
stress incontinence	17% (25-44 y. 10% - 45-59 y. 15%, 60+ 33%)
Urge incontinence/OAB	**28% (25-44 8%, 45-59 18%, 60+ 45%)**
mixed incontinence	38% (25-44 y. 19%, 45-59 y. 33%, 60+ 53%)

If only the data on severely affected are taken into account, this is 28% of all female OAB patients or 45% of all OABs in women over 60 years of age. Mixed incontinence accounts for 38% of all forms of incontinence.

---

physiology

The possible causes of an overactive bladder have not yet been fully clarified. A common subdivision differentiates the OAB syndrome and urge incontinence into neurogenic and non-neurogenic [3] . In the differential diagnosis, undiscovered infections that do not cause any characteristic symptoms (chlamydia, mycoplasma, trichomonads, etc.), in addition to central nervous and peripheral disorders [4] , changes in the bladder outlet and psychosomatic causes must be discussed. Basically, the detrusor muscle is subject to a normal aging process, which can also lead to OAB syndrome.

In the urinary bladder of old people, for example, collagen fibers have completely penetrated the submucosa and the area surrounding the neurovascular bundles and muscle cells, which causes the loss of elasticity of the detrusor [5] , [6] , [7] , [8] . The activation of stretch receptors in the urothelium and the adjacent connective tissue leads to increased afferent signals into the CNS, which lead to an efferent response in the form of urinary urgency sensations. Under physiological conditions, afferent signals from the bladder are normally blocked in the thalamus and prevent conscious perception in the CNS. If these central inhibitions are absent, the increased stretching signals reach consciousness and are perceived as an emptying stimulus.

Detrusor hyperactivity and neurogenic detrusor hyperactivity

If classified according to urodynamic characteristics, (idiopathic) detrusor overactivity with urge symptoms is in the foreground. Apparently, sensory stimuli are constantly being generated via the sacral voiding center, which reach the functional center in the hypothalamus via afferents. The imbalance between the strength of the afferent impulses and the central inhibition of the micturition reflex causes detrusor overactivity [9] . Neurogenic detrusor hyperactivity is caused by reduced central nervous inhibition of the micturition reflex, as can occur, for example, in MS, Parkinson's disease or after a stroke and, anticipating the following chapters, may not respond to treatment with rPMS. Detrusor hyperactivity is often caused by symptoms, ie - as already described - it is caused by inflammation, tumours, anatomical peculiarities (descensus vaginae) or an estrogen deficiency.

OAB

OAB is usually seen as a precursor to drain incontinence and is characterized by pollakiuria, nocturia and an imperative urge to urinate. Detrusor hyperactivity does not always have to exist here.

integral theory

The emergence of a female UI can be explained in some cases with the integral theory according to Petros [10] , [11] . In the overall construction of the female pelvic floor, the vagina can be seen as a kind of "trampoline" that is always in an elastic state of tension due to the various ligaments and muscle trains in the ring of the bony pelvis. If the bladder floor now sinks due to loosening of the trampoline structure ("insufficiency of the pelvic floor"), the bladder floor can stretch independently of the bladder filling. This increases the probability of stimulus for the stretch receptors concentrated on the bladder floor, which transmit the signals via afferent pathways

into the CNS [12] . Normally, the body can counteract increased and unregulated signals via central inhibition centers, so that the trampoline "tightens" again by contracting the pelvic floor. However, when the ligaments are overstretched or loose, it is no longer possible for the muscles to contract the vaginal wall sufficiently. The stretch receptors located at the bottom of the bladder thus "fire" signals into the CNS even when the filling volume is low. It should also be mentioned here that bladder emptying disorders can also result in urinary retention [13] , [14] .

gate control theory

Another explanatory model and therapeutic point of attack results from the “gate control theory” for influencing pain, which was first described in 1965 [15] . It is known that afferent signaling from the bladder can be inhibited by interneurons in the sacral cord [16] . Thus, the activity of thick, myelinated fibers blocks or closes the "gate" and thus inhibits the conduction of the peripheral bladder afferents, which run in thin A-Delta/C fibers. The thin fibers, on the other hand, open the gate so that the urge to urinate is passed on to the CNS [17] . The gate is blocked or closed by the somatosensory fibers of the pudendal nerve, since this contains both generally somatosensory and somatomotor nerve fibers.

Pelvic organ prolapse (POP) / descensus uteri

Vaginal prolapse is understood to mean the sinking of the pelvic organs, which can affect both the vagina and the uterus or cervix (earth uterus) [18] . Those affected usually present to their gynecologist with “lower urinary tract symptoms” (LUTS), which can consist of stress urinary incontinence, but also urge symptoms, pollakiuria and urge incontinence [19] . It is significant that overactive bladder (OAB) occurs more frequently in POP (Pelvic Organ Prolapse) and therefore a causal relationship is suggested [20] . According to a study, 56 to 88% of patients with a vaginal prolapse had urge symptoms - which 20 to 64% of patients without a prolapse also had [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 is a major problem for women, but the cause of the symptoms is unknown" [23] . According to one study, anterior vaginal wall sagging or prolapse is thought to have links to overactive bladder and directly correlate to OAB severity [24] .

QRS Pelvicenter rPMS effect

The first encouraging attempts at painless and non-invasive magnetic stimulation of the sacral nerves suggested as early as the 1990s that perineal magnetic stimulation or rPMS should also be used in urge incontinence. This is also related to the extended model of the integral theory, according to which the previous architecture of the pelvic floor and the lost trampoline function of the vagina can be restored with pelvic floor training, thus reducing the filling pressure on the bladder floor and the risk of stimulation of the stretch receptors. However, this only applies to an OAB syndrome or an UI, the cause of which is to be found in a weakness of the pelvic floor.

Based on the setting parameters or stimulus configuration of an rPMS, the frequency to be applied would be identical to the therapy of a SUI. However, if the irritation of the bladder or a disordered afferent transmission of stimuli to the CNS are the focus

of OAB / UI, rPMS training designed to strengthen the pelvic floor would not be able to take the actual cause into account. Here, the gate control theory must be included in the treatment procedure. After rPMS only activates the thick myelinated fibers of the pudendal nerve and thus closes the gate (“transmission in the sacral reflex arc”), so that the peripheral bladder afferents are not transmitted to the thin A-Delta / C fibers and such thin fibers as well do not react to the rPMS, the transmission of the urge to urinate to the CNS is prevented.

This is reminiscent of everyone's experience that by "pinching" the buttocks (or the pelvic floor) a strong urge to urinate can disappear for a brief moment. The strategy derived from this consists of activating the branches of the N. pudendus by rPMS and thus inhibiting or superimposing a pathologically increased bladder stimulus (the urge-reducing effect of a strong pelvic floor via a negative feedback mechanism is in the literature known since the 1970s).

So everything depends on a pacing rate that interrupts autonomous and undirected signaling to the CNS. Findings from peripheral electrical stimulation are helpful here, according to which a frequency of 5 Hz enables maximum inhibition of the bladder via sympathetic fibers. Frequencies between 5 and 10 Hz should also produce a central inhibition of the efferents (i.e. motor signaling) to the bladder or detrusor hyperactivity [25] , [26] , [27] , [28] . An exception to this, however, are neurogenic bladder dysfunctions such as paraplegia [29] .

Scope of treatment and duration of therapy

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

The selection of the correct frequency settings and their chronological sequence is explained in more detail in the QRS Pelvicenter manual. You will receive this as the operator of a QRS Pelvicenter. The instructor will also explain this topic in detail when setting up the device.

expectation of success

After 16 to 20 therapy sessions, a significant improvement in symptoms can be expected in about 50% of OAB patients and healing in 10 to 15%. The success of the therapy occurs with increasing treatment duration. In the majority of cases, patients first report an improvement in their night's sleep, followed by a gradual reduction in the need to urinate. A therapeutic success lasting up to 6 months can be expected. It is therefore advisable to carry out a follow-up treatment (8 to 10 therapy sessions) a few months after the initial treatment.

study situation

To date, there have been 39 studies on the rPMS treatment of OAB syndrome or UI (OAB is the term for ICS, urge incontinence is a symptom of OAB). Of these, 11 studies were presented at urological congresses, but no abstracts or posters were published. In addition, it must be taken into account that many of the studies cited below refer to a mixed group of patients with stress, urge and mixed incontinence.

Study 1 : randomized, prospective, double-blind, multicenter study [30]

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

Result:

Compared to baseline, the number of urge urinary incontinence episodes per week in the active group increased by -13.08 +/- 11.00; for placebo by -8.68 +/- 13.49 (p=0.038). Urge periods decreased by -2.65 +/- 2.52 (p=0.011) compared to placebo (-1.53 +/- 2.39). The urine loss was reduced in the verum group by 14.03 +/- 34.53 ml versus placebo with - 4.15 +/- 40.60 ml (p = 0.0056).

Study 2: randomized, double-blind, multicenter study [31]

The group of patients consisted of SUI, UI and mixed patients who were treated 3 times a week for 6 weeks (10 minutes 5 Hz / 3 minutes break / 10 minutes 50 Hz). For the placebo treatment, the intensity was adjusted down to zero. The patients were informed that the procedure would work according to the noiselessness of a CT.

Result:

Of the original 49 patients, 33 patients (14 patients (58%) in the active group and 19 (76%) in the sham group) completed the study. After the patients had drunk 500 ml of liquid and had to wait 30 minutes in a sitting position, none of the patients in the verum group experienced urine leakage (21% baseline), while no change was registered in the sham group. The average pad weight was 2.59 g in the verum group and 14.6 g in the placebo group (p = 0.079). Average pad usage decreased from 3.33 to 2.0 in the active group (p=0.02) and remained unchanged in the placebo group. The QOL score improved from 72.86 (baseline) to 84.69 (p=0.04) in the active group and remained unchanged in the placebo group.

Study 3: 37 patients with urge or stress incontinence [32]

20 patients with urge (3 men, 17 women, Ø 68.5 +/- 14.2 years) and 17 patients with stress incontinence. Detrusor contractions were evident in 8 patients at baseline. The rPMS treatment took place twice a week for 8 weeks (10 min at 10 Hz - 2 min break - 10 min at 50 Hz).

Result (UI only)

Cure was achieved in 25% of urge incontinence patients. 60% improved and 15% had no effect (p<0.003). In 7 of the original 8 patients, urodynamic detrusor contractions persisted. After 2 weeks, the daily, unwanted urine leakage in the verum group was reduced from 5.6 to 3.6 per day. At 24 weeks, two patients were fully continent (no pads) and four patients were using less than one pad per day on average.

In the verum group, the functional bladder capacity (volume per emptying) increased from 141 +/- 50.6 ml before treatment to 188 +/- 77.8 ml after treatment (p < 0.018). Mean I-QOL was 62.7 before treatment and increased to 77.8 at 4 weeks (p<0.004). The VAS score also improved from 7.82 to 5.45 after 2 weeks (p<0.04). 24 weeks after the last treatment, the result was maintained in 9 out of 17 patients (52.7%). The previous symptoms recurred in 8 patients (47.1%). 3 of these 8 patients wanted further rPMS applications.

Study 4: 48 female patients with OAB [33]

The rPMS treatment was carried out at a frequency of 10 Hz, twice a week for 20 minutes each over a period of 8 weeks.

Result after 2 weeks:

A total of 27 patients (56.3%) achieved a significant reduction in symptoms, with 33 of 48 patients (68.8%) having urge symptoms, 27 of 48 (56.3%) the frequency and 8 of 16 patients (42.8%) improved urge incontinence. The mean frequency of daily toilet visits decreased by 42.8% ($p < 0.001$). The total volume of urine remained the same, which means that the individual urine volume increased accordingly.

Result after 24 weeks:

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

Study 5: 26 patients with an OAB syndrome [34]

26 patients (2 male, 24 female / Ø 39.5 years) with an OAB syndrome were examined. The rPMS treatment took place 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 urge symptoms (frequency of going to the toilet). Only 2 patients showed no improvement. The mean number of daily voids decreased by 38.1% ($p<0.001$). Before treatment, 8 OAB patients (31%) reported one or more episodes of incontinence. After the treatment, this was only the case in 4 patients. However, there was no significant change in bladder capacity from baseline.

Result after 6 months

At 6 months, of the 15 evaluable patients, the previously achieved result was maintained in 14 patients (93%). The number of daily voids had decreased from 15.8 +/- 5.3 to 9.9 +/- ($p < 0.001$).

Study 6: mixed study collective [35]

The study group consisted of 66 patients (23 male, 43 female) with mixed and urge symptoms. The average number of applications was just 7.7 +/- 3.8 for men and 10.4 +/- for women. The treatment itself was carried out over 20 minutes with low and high frequencies.

result

57.1% of the men showed a significant improvement and an additional 9.5% showed slight improvements - while 29% had no effect on the treatment. Among the women, only 35.1% experienced a significant improvement and 37.8% a slight improvement,

while 24.3% had no effect from the treatment. Before the treatment, 66.7% of the women complained about urge symptoms, after the treatment only 7.4%.

Study 7: Prospective study [36]

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

Result:

Unintentional urine leakage improved in 58% of patients. A significant number of patients were satisfied with the treatment ($p<0.001$). 3 of 24 were fully continent after therapy. Of 12 urge incontinence patients, treatment was successful 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 felt an improvement ($p < 0.01$) - while 30% reported either no change or worsening of the symptoms.

Study 8: Mixed incontinence group SUI/OAB [37]

Mixed incontinence group with 49 SUI and 44 OAB patients. Only the OAB group is referred to here. 34 people completed the treatment in full. The therapy took place twice a week for 9 weeks. Treatment parameters were 50 Hz (3 s), pause (6 s), 10 Hz (3 s), pause (6 s).

Result:

The responder rate (“significant improvement in OAB symptoms”) was 61.7% (21 of 34 people). If the dropouts are included (intention-to-treat analysis), it is 47.7% (21 out of 44 people). According to the UDI-6 (Urogential Distress Inventory), which has been validated for older women and men [38] and asks about symptoms and their degrees of severity and differentiates between obstructive and irritant symptoms, the average score fell from 9.7 +/- 4.2 (baseline) at 9 weeks to 4.0 +/- 1.7 ($p<0.01$). In the IIQ-7, the short form of the Impact Incontinence Questionnaire, which examines the negative impact of incontinence on activities of daily living, the score fell from 10.8 +/- (baseline) to 3.5 +/- 4.4.

---

summary

The high rate of side effects and discontinuation of the anticholinergics propagated as first-line therapy, the intravesical botolinum toxin injection with rapidly diminishing long-term effects despite repeated use, or ultimately, as a last resort, the surgical use of a neuro-stimulator of the sacral roots, represent the great importance of a simpler, painless, non-invasive and, above all, effective form of therapy such as rPMS.

rPMS is proving to be an easy-to-use, target-oriented and (almost) side-effect-free urinary incontinence therapy (SUI/UI/OAB/mix) and consequently claims to establish itself as one of the first-line therapy options over the next few years . This claim is justified because with a manageable duration of therapy of 16 to 20 applications, each lasting 15 to 20 minutes, according to evident studies, there is a clear improvement in symptoms or healing in 50 to 8 weeks over a period of 6 to 8 weeks 65% of all patients can be assumed. In addition, patients who deny an invasive procedure, or who are foreseeably unsuitable for conventional BeBo training, or who are ashamed of electrostimulation, can be offered an effective therapy solution.

Sources

[1] Abrams P et al. The standardization of terminology of lower urinary tract function: report from the Standardization Sub-committee of the International Continence Society. neurourol urodyne . 2002;21(2):167-178
[2] Federal Health Reporting . Booklet 39. Urinary incontinence . Ed . Robert Koch Institute September 2007
[3] Yokoyama O. Pathophysiology and treatment of the overactive bladder. Hinyokika Kiyo. 2005; 51(9):599-601
[4] Reynolds WS et al. Does central sensitization help explain idiopathic overactive bladder ? Nat Rev Urol. 2016; 13(8): 481-91
[5] Goepel M, Schwenzer T, May P et al. Urinary incontinence in the elderly. German Ärztebl 2002; 99(40): A2614-A2624 / B-2230 / C-2093
[6] Elbadawi A, Yalla SV, Resnick NM. Structural basis of geriatric voiding dysfunction. II. J Urol 1993, 150: 1650-1656
[7] Elbadawi A, Yalla SV, Resnick NM. Structural basis of geriatric voiding dysfunction. III. J Urol 1993; 150: 1668-1680
[8] Elbadawi A, Yalla SV, Resnick NM. Structural basis of geriatric voiding dysfunction. IV. J Urol 1993; 150: 1681-1695
[9] Hennecke S. Studies on the validity of the Gaudenz questionnaire in the diagnosis of female urinary incontinence. Dissertation. Charité – University Medicine Berlin, 2011
[10] Petros PE. The Female Pelvic Floor. Function, Dysfunction and Management According to the Integral Theory. Springer Medicine Verlag, Heidelberg. 2007
[11] Petros PE. The Female Pelvic Floor. Function, Dysfunction and Management According to the Integral Theory. Springer Medicine Verlag, Heidelberg. 2007
[12] Liedl B. Urinary incontinence in women and men also from a pelvic surgical perspective. Urologist 2010. 49: 289-301
[13] DasGupta R, Fowler CJ. The management of female voiding dysfunction: Fowler's syndrome - a contemporary update. Curr Opin Urol. 2003; 13:293-299
[14] Dutta I, Dutta DK. Voiding disorders - Review of current advances. J Evol Med Dent Sci. 2013; 2(37): 7197-7201
[15] Melzack R, Wall P. Pain mechanism: a new theory. Science 1965; 150(3699):971-979
[16] Craggs M, McFarlane J. Neuromodulation of the lower urinary tract. ExpPhys 1999; 84: 149-150
[17] Kronenberg RM. Permanent cessation of severe symptoms of "Chronic Pelvic Pain Syndrome". Dissertation. University of Bern.
[18] Jelovsek JE, Maher C, Barber MD. Pelvic organ prolapse. Lancet.2007; 369:1027-38.
[19] Cetinkaya SE, Dokmeci F, Dai O. Correlation of pelvic organ prolapse staging with lower urinary tract symptoms, sexual dysfunction, and quality of life. Int Urogynecol J 2013; 24:1645-1650
[20] De Boer TA et al. Pelvic Organ Prolapse and Overactive Bladder. Neurourol Urodyn. 2010; 29(1):30-39
[21] De Boer TA et al. Pelvic Organ Prolapse and Overactive Bladder. Neurourol Urodyn. 2010; 29:30-39
[22] Tunn R et al. Urogynecology in practice and clinic. Walter de Gruyter GmbH & Co. KG, Berlin. 2010
[23] Abrams P et al. The standardization of terminology of lower urinary tract function: report from the standardization sub-committee of the ICS. neurourol urodyne . 2002; 21:167-178
[24] Yuan ZY, Shen H. Pelvic organ prolapse quantification in women referred with overactive bladder. Int Urogynecol J 2010; 21:1365-1369
[25] Reitz A, Schurch B. Neuromodulation of the lower urinary tract by peripheral electrical stimulation of somatic afferents. current Urol 2001; 32(5): 245-251
[26] Case M et al. Effects of electrical intravaginal stimulation on bladder volume: an experimental and clinical study. Urol Int 1978; 33:440-442
[27] Lindstrom S et al. The neurophysiological basis of bladder inhibition in response to intravaginal electrical stimulation. J Urol 1983; 129: 405-410
[28] Lindstrom S et al. Rhythmic activity in pelvic efferents to the bladder: an experimental study in the cat with reference to the clinical condition "unstable bladder". Urol Int 1984; 39:272-279
[29] Vereecken RL, Das J, Grisar P. Electrical spinkter stimulation in the treatment of detrusor hyperreflexia in paraplegics. neurourol Urodyne 1984; 3: 145-154
[30] Yamanishi T, Homma Y, Nishizawa O et al. Multicenter, randomized, sham-controlled study on the efficacy of magnetic stimulation for women with urgency urinary incontinence. Int J Urol 2014; 21(4): 395-400
[31] Cardozo L, Miska K, Schuessler B et al. Extracorporeal innervation (EXMI ™) for treatment of urinary incontinence in a population of European patients.
[32] Yokoyama T, Fujita O, Nishiguchi J et al. Extracorporeal magnetic innervation treatment for urinary incontinence. Int J Urol 2004; 11: 602-606
[33] Choe JJ Choo MS, Lee KS. Symptom change in women with overactive bladder after extracorporeal magnetic stimulation: a prospective trial. Int Urogynecol J Pelvic Floor Dysfunction. 2007; 18(8): 875-880
[34] Kim ST, Han DH, Choe JH, Lee K. Extracorporeal magnetic stimulation for the treatment of overactive bladder. ICS 2003, #470, Florence, Italy
[35] Perianan M, Huat C, Peter L. Efficiency of extracorporeal magnetic innervation (EXMI) in urinary incontinence: a symptomatic assessment. Presented ICS Congress 2002, Heidelberg Germany Abstract # 435

[36] Chandi DD, Groenendijk PM, Venema PL. Functional extra corporal magnetic stimulation as a treatment of female urinary incontinence: the chair. BJU Int. 2004; 93(4): 539-542
[37] Lo TS, Tseng LH, Lin YH et al. Effect of extracorporeal magnetic energy stimulation on bothersome lower urinary tract symptoms and quality of life in female patients with stress urinary incontinence and overactive bladder. J Obstet Gynecol Res. 2013; 39(11): 1526-1532
[38] Donovan JL: Symptom and Quality of Life Assessment. In: Abrams P: Incontinence, Plymouth: Health Publications Ltd, 2001, 267-316