Neuromodulation of the superior hypogastric plexus: a new option to treat bladder atonia secondary to radical pelvic surgery?

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Introduction

It is well documented that radical pelvic surgery can lead to postoperative dysfunction of the lower urinary and intestinal tracts, such as loss of bladder and rectum sensation and impairment in the command to spontaneously void the bladder or rectum (1,2). For this reason „nerve-sparing“ techniques have been developed. They mainly consist of identifying and sparing the different bands of neural tissue involved in micturition and defecation (3,4,5,6,7). Even in cases where a radical resection of the parametria is required, it is possible to selectively spare the parasympathetic pelvic nerves (8). Therefore postoperative functional morbidity should not be accepted as a routine side-effect of radical pelvic surgery. Despite this, the incidence of bladder atonia secondary to the destruction of the pelvic autonomous motoric nerves during pelvic surgery remains high. Management of bladder atonia is based on the need to empty the bladder before overflow, leakage or stretching occurs and the majority of the patients still use self catheterization on a regular basis. Several therapies for recovery bladder voiding function in patients with bladder atonia have been evaluated in the past, but none permit effective recovery of bladder function when atonia is secondary to radical pelvic surgery. In this present study, we report on one technique never reported before, the neuromodulation of the superior hypogastric plexus.

Methods

We report on four patients suffering from bladder atonia secondary to pelvic surgery; Diagnosis was secured by previous neurourologic reports and urodynamic testing and all four patients managed their bladder atonia by intermittent selfcatheterisation.

The first patient, a 30 –year-old woman, suffering from chronic pelvic pain and bladder atonia following pelveoperitonitis secondary to a postpartal hysterectomy 2005 performed following a uterine rupture. The patient
underwent three further laparoscopies with adhesiolysis due to pelvic pain, unfortunately without any improvement in pain. The patient was referred to a multidisciplinary pain center and still rated her pain 8/9/10 despite multiple analgesics (paroxetine 40mg/d, mirtazapine 20mg/d, tramadol 300mg/d, pregabalin 450 mg/d, fentanyl 50µg/3days). As we have demonstrated that laparoscopy allows exposure (9,10) and the placement of electrodes on all pelvic nerves (11,12), the objective of our proposal was to control the pelvic pain using the neuromodulation of the superior hypogastric plexus as it is well known that it plays an important role in the transmission of visceral pelvic pain (13). We performed a laparoscopic exposure of the entire superior hypogastric plexus ventral of the inframesenteric portion of the aorta and placed an „On-Point PNS Electrode – Model 3987” (Medtronic GmbH Germany) covering the entire plexus and fixed with a non-resorbable suture while the cable was passed dorsal of the colon descendens avoiding direct contact with the left ureter.

The second patient, a 61-year-old women developed bladder atonia following an abdominal radical hysterectomy with pelvic lymphadenectomy and postoperative pelvic radiotherapy 1998 for endometrial cancer. The third patient, a 28 year-old women suffered from bladder atonia and severe constipation after laparotomic deep anterior colorectal resection/anastomosis for deep infiltrating endometriosis of the rectum in 2000. The last patient, a 58-years old man developed bladder atonia after radical prostatectomy for cancer and postoperative pelvic abscess formation which required surgical revision.

The second and the third patients previously underwent a transforaminal sacral nerve test-stimulation without any positive effect on bladder function.

In all three patients, the LION procedure on the superior hypogastric plexus was performed in the same way as with the first patient. While in the first patient recovery of micturition occurred after implantation of the permanent electrostimulator and after the patient was discharged, in the last three reported patients, the effect of the neuromodulation to the superior hypogastric plexus on the bladder function was assessed during a test phase of external neuromodulation before the decision was made to implant the permanent stimulator (Interstim Modell 3023 Neurostimulationssystem - Medtronic GmbH Germany): On the first postoperative day urodynamic testing was performed under a stimulation 250 µs/ 15Hz with increasing intensity starting at 0,5 Volt until electrical induced discomfort or pain sensation. With every modification of the parameters of the neuromodulation, the patients were encouraged to attempt micturition.

Results

In the first patient, we had planned a neuromodulation of the superior hypogastric plexus in an attempt to control her pelvic visceral pain. The LION procedure on the superior hypogastric plexus was uneventful and took 55 minutes.
On the third postoperative day, the patient defaecated normally and all analgesics were stopped. The test phase to prove efficacy of the neuromodulation was then started. The external stimulation was alternately switched on and off for a period of 5 hours. The patient estimated her pain to be 7-8/10 without stimulation while during stimulation (15Hz/250µs/0,6V) she reported a direct decrease of the pain to a VAS score of 2/10. It was therefore decided to implant a permanent neuromodulator (Itrel 3 – Medtronic GmbH Germany) and the patient was discharged from our institution with continuing self-catheterisation. Four weeks later, the patient informed us by phone about recovering spontaneous micturition. We referred the patient directly to a neuourologist in her home country for urodynamic testing: It revealed during the filling phase a maximal intravesical pressure of 15cm H2O with apparition of a first sensation of fullness at 460ml. During provocation testing, no incontinence occured. Micturition was obtained with an intravesical pressure of 36 cm H2O, an abdominal pressure of 3cm H2O and a middle urethral flow of 8ml/sec (Max: 16ml/sec) while no pelvic floor activity or postmictional resturine could be observed. Unfortunately, the patient would not undergo urodynamic testing as she was afraid of the possible recurrence of the pain. 21 months later, the patient is still not taking any medication and estimates her pain score to be 1-2/10; she is still voiding her bladder under normal micturition conditions without requiring catheterisation and also reported on the recovery of a vaginal sensation of pleasure during sexual activities with an increase in her libido. In the second patient, full recovery of bladder function could be obtained and was urodynamically documented as with the first patient. The third patient also reported on recovery of spontaneous bladder voiding but urodynamic testing documented a postmictional resturine varying between 80ml and 150 ml urine. In the last patient in our series no effect of the neuromodulation of the superior hypogastric plexus could be observed neither clinically nor during urodynamic testing so that the electrode was removed by simple traction of the electrode cable. In all three patients who underwent implantation of the permanent neurostimulator, position of the electrode was controlled before by X-ray (figure 2).

Discussion

The more extensive a surgical procedure in the pelvis is, the higher the risk of damage to the lower urinary tract will be. The most common postoperative complication is the an- or hypocontractility of the detrusor secondary to injury of the vesical motoric innervation. To avoid overstretched of the bladder and damage to the upper urinary tract, bladder atonia is mostly managed by intermittent self catheterisation. Medical treatment with alpha-blockers can reduce hypertonia of the dorsal urethra (14) but can only support recovery of spontaneous micturition when the measured bladder pressure and residual urine volume are within acceptable limits. Surgical procedures to reduce the
voiding resistance of the bladder always carries the risk of postoperative incontinence (15) and current surgical treatments such as enteroplasty with intermittent catheterisation or intravesical electrostimulation are far from ideal solutions (16). A further option for the treatment of urine retention mentioned in literature is sacral neuromodulation but this method does not work in patients with bladder atonia secondary to surgical destruction of the pelvic splanchnic nerves. A partial voiding of the bladder can be obtained by simple Valsalva or a Créde manoeuvre, but a complete emptying is normally not obtained (17).

In lesions of the lower motor neurons, a paresis of the pelvic floor muscles with loss of sphincter tone and the absence of induced reflexes exists. However the external sphincter is not an obstructive element for bladder voiding, but the internal sphincter which present a paradoxal hypertonie induce by the intact sympathetic innervation from the lower thoracic segment which is sustained and even strengthened. In addition intermittent self-catheterisation can lead over the time to structural changes of the urethra increasing the voiding disturbances to the bladder outlet. This paradoxal urethral hypertonus also explains why patients with a cauda equina cannot obtain passive micturition although the destruction of the lower-motor-neurons to the detrusor is combined with a destruction of the pudendal motor-neurone and logically incontinence should more likely be expected. This paradoxal hypertonia of the internal sphincter which is also accompanied by a further relaxation of the detrusor is induced by a “sympathetic arc reflex”: The sympathetic afferent fibers of the internal urethral sphincter run via the inferior and superior hypogastric plexi to the intermediolateral nuclei placed in the medulla at T11-L3 where efferent motoric fibers are stimulated (18). This information is then conducted back to the bladder through the same hypogastric plexi and produces a “paradoxal hypertonus” of the internal urethral sphincter and an additional relaxation of the detrusor (19). Thus in view of the results of our small series, it appears that the neuromodulation to the superior hypogastric plexus is able to modify or interrupt this “sympathetic arc-reflexe” which results in a reduction in the tone of the internal urethral sphincter. That a block-effect is obtained on the afferent sympathetic fibres contained in the superior hypogastric plexus is proven by an improvement in pain in the first patient of our series. The urodynamic testing also demonstrated that micturition occurred not just due to a blockade of the paradoxal urethral hypertonia but also to an effective contraction of the detrusor. Thus it appears that the effect of the neuromodulation on the sympathetic fibres of the superior hypogastric plexus involved in this arc reflex, permits the rest of the vesical parasymathetic efferent fibres of the bladder to contract the detrusor. It is unknown if this principle of neuromodulation to the superior hypogastric plexus could also permit passive micturition in patients with a cauda equina, but the LION procedure is such a minimal invasive surgical procedure that it could also be tested in this indication. A further new field of the neuromodulation of the superior hypogastric plexus could also be the management of autonomic dysreflexia in patients with spinal cord injury above mid-thoracic level...
Conclusion

We report in our small series on the neuromodulation of the superior hypogastric plexus as a therapy option to control visceral pelvic pain and to recover micturition with bladder atonia secondary to pelvic surgery, it is clear that these results need to be confirmed on a larger number of patients and with a longer follow-up. The technique of neuromodulation of the superior hypogastric plexus is a completely new surgical procedure that has never been performed or reported on before.

References


