the identification of connexin 26 and 43 at an optimal dilution of 1:1,000 and a hydrogen peroxidase labeled antibody to vinculim (dilution of 1:1,750). Infant mouse heart was used as the connexin control and rate small bowel granulation tissue as the vinculin control.

Results: Immunohistochemistry- No cell membrane staining for connexin 26 or 43 was seen in the bladder biopsies of the 5 cases and 4 controls. Entire cell border staining with labeled antibody to vinculin was present in all cases and controls.
Electron Microscopy- No gap junction was identified in any of the cases or controls. Adherens (intermediate)junctions in classic and rudimentary forms were present in all cases and controls. Dense plaques ('hemijunctions') were present on the membranes of all patients and usually a 'complementary' dense plaque could be identified on an adjacent cell membrane. Adherens junctions and dense plaques occupied most of the cell border in all patients. Membrane caevolae occupied the spaces between these adherens junctions and dense plaques.

Conclusion: Immunohistochemistry and electron microscopy of the detrusor did not identify gap junctions. In organs such as the human uterus and mouse mammary glands, gap junctions have a rapid turnover with formation and involution occurring within several hours (2, 3). Many connexin subtypes have been identified. It could be argued that we failed to identify gap junctions in the overactive bladder because we happened to biopsy the bladders at a time when the junctions had undergone involution or the junctions were composed of connexin other than 26 and 43. In this study, the entire cell border stained positive to vinculin confirming that adherens junctions are the predominant and probably only junction present on detrusor smooth muscle cell membranes. Vinculin is a protein associated with adherens junctions and is likely to play an important role in the linkage of actin to the cell membrane. The function of adherens junctions is to mediate mechanical coupling between adjacent cell. Electron microscopy demonstrated the entire cell border to be occupied by adherens junctions, dense plaques (adherens 'hemijunctions') and caveolae leaving no space for gap junctions. We interpreted 'protrusion junctions' to be rudimentary adherens junctions rather than possible gap junctions as reported by Elbadawi et al (2). Connexin 43 was chosen because it has been found in the human uterus and was considered the most likely connexin to be identified if gap junctions were present in the human bladder. Connexin 26 has been identified in human cardiac muscle. The cause of increased membrane excitability in detrusor muscle in patients with bladder instability remains unanswered. This study failed to demonstrate that detrusor instability is caused by the presence of gap junctions.

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H. C. Klingler, A. Pycha, J Schmidbauer, M. Marberger
Department of Urology, University of Vienna, Austria

THE USE OF PERIPHERAL NEUROMODULATION FOR TREATMENT OF DETRUSOR OVERACTIVITY: AN URODYNAMIC BASED STUDY

Aim of Study: An urgency and frequency syndrome due to an overactive bladder can successfully be treated by afferent nerve stimulation of the S3 spinal regions. Aim of this study was to determine the efficacy of peripheral neuromodulation of the S3 region in patients with urgency-frequency syndrome due to an overactive bladder.
Patients and Methods: 15 patients (11 women and 4 men), suffering from urgency-frequency syndrome, as documented by a voiding chart were diagnosed with overactive bladder. Pelvic pain was assessed by an visual analogue scale (VAS). Full urodynamic work-up was performed before and after 12 peripheral stimulations with a 9V monopolar generator, the so-called Stoller Afferent Nerve Stimulator (SANS™) utilising a minimal invasive transcutaneous access to the posterior tibial nerves. Patients follow-up was for a mean (SD) 10.9 (4–15) months post treatment.
Results: No complications were observed. Reduction in pain was achieved in all patients, with a decrease in VAS from a mean (SD) of 7.6 (5–10) to 3.1 (1–7) (p=0.00049). Seven patients (46.7%) had a complete response and were considered

cured; three (20.0%) showed significant improvement; and five (33.3%) were classified as non-responders. Urodynamic evidence of bladder instability, evident in all patients prior to treatment, was eliminated in 76.9% of patients. In all patients, mean (SD) total bladder capacity increased significantly from a 197 (35–349) to 252 (78–384) ml (p=0.00795), mean (SD) volume at first bladder sensation from 95 (16–174) ml to 133 (32–214) ml (p=0.00166) and mean (SD) bladder volume at normal desire to void from 133 (27–217) ml to 188 (47–296) ml (p=0.00232). In the responding group, the mean (SD) total numbers of voids was reduced from 16.1 (9–24) times during the day and 4.4 (2–6) times during the night to 8.3 (6–10) and 1.4 (1–2) times (p=0.002539), respectively. In addition we observed a decrease in the number of protective pads from 4.9 to 1.6 per day.

Conclusions: Peripheral neuromodulation of the S3 region can successfully treat patients suffering from urgency-frequency syndrome due to an overactive bladder. Morbidity of this treatment is negligible. Clinical results may be improved by proper patients selection since patients with severe anatomical deficiency, interstitial cystitis and neurological disorders seem to be unsuitable candidates for this technique.

Aknowledgment: The SANS device was provided by UroSurge Inc. Iowa, USA and the study was approved by the local ethics committe (No. EK 327/98).

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Author(s): J. Groen, J.L.H.R. Bosch

Institution, city, country: University Hospital Rotterdam, Rotterdam, The Netherlands

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RELATIONSHIP BETWEEN DETRUSOR INSTABILITY AND URETHRAL RESISTANCE IN PATIENTS TREATED WITH SACRAL NEUROMODULATION

AIMS. Sacral neuromodulation is a treatment option for patients with detrusor instability [1]. This method has also been shown to decrease urethral resistance during voiding [2]. We previously found that urethral resistance was higher in a group of females with detrusor instability than in females with mixed incontinence or stress incontinence or females without a demonstrable cause of the incontinence and postulated that functional obstruction could be a potential cause of instability [3]. In the present study, we examined the relationship between the decrease of urethral resistance and that of the grade of instability in patients with detrusor instability treated with neuromodulation. In addition, we examined if the symptomatic changes as derived from voiding / incontinence diaries depended on urethral resistance. Such a study might contribute to the understanding of the role of voiding characteristics in the signs and symptoms of detrusor instability.

METHODS. Neuromodulation is applied at our department since 1990. Voiding / incontinence diaries and cystometric studies with subsequent pressure / flow studies before and 6 months after the operation are part of the evaluation. Cystometry is done in the supine and standing position and, after implantation, with the stimulator on and off. The pressure / flow studies are done in the standing position. Only patients with symptoms of urge incontinence and urodynamically demonstrated detrusor instability who had passed the 6 month evaluation period were included in the present study. The maximum detrusor pressure Pmax during the filling phase was used as the parameter characterising the grade of instability. Urethral resistance was characterised by URA. In addition, the maximum flow rate Qmax and the associated detrusor pressure pQmax were determined. The measurements in the standing position and, after implantation, with the neurostimulator on, were used. Three categories of symptomatic success were defined: a more than 90% decrease in the number of pads used per day or the number of incontinence episodes