



Urinary Incontinence in Women-Large Steps to Cure (“Feel Free!”)

Jager W* and Ludwig S

Department of Urogynecology, University of Cologne, Germany

Symptoms are Signs of Changes from Normal

Voiding is an intended procedure which is self-induced by the women. The term “Urinary Incontinence” (UI) covers a spectrum of symptoms that completely describe the fact that voiding was unintended (“loss of urine”).

UI usually starts with the uncontrolled loss of urine during coughing or sneezing. The situation continues with the loss of urine when the woman rises from a chair to visit the bathroom. The time interval between the need to void (“urge”) and the actual spontaneous loss of urine will diminish consequently. To avoid the loss of urine she starts to void prophylactically i.e. increases voiding frequency. The final stage is when the woman has lost all control of voiding and loses urine all the time (total incontinence).

It was assumed that these different symptoms were signs of different incontinence entities and were named accordingly as Stress Urinary Incontinence (SUI) and Urgency Urinary Incontinence (UUI). The sum of all symptoms is named Mixed Urinary Incontinence (MUI). Furthermore, incontinence is a problem during the day but rarely during the night. Patients go to the bathroom 15 times per day but only 2 times per night. In most patients’ UI starts at menopause or short before. It is an interesting observation that further 20% of patients develop the incontinence at elder ages after stopping Hormone Replacement Therapy (HRT). Urinary incontinence is only observed in humans. It is never seen in animals.

The Search for the Causes of that Change will Lead to Treatment

The always identical sequence of symptoms indicates an inherent common pathology which has an identical pathophysiology. Therefore, the different classifications as SUI, UUI, MUI or total incontinence are misleading since we do not have to deal with 4 different disorders but with one disorder (defect) which increases with time (pathophysiological continuum). Since quadruplets never develop incontinence we hypothesized that incontinence is based on the fact that the humans raised the body. Thereby, the bladder lies above the urethra in contrast to animals in which the bladder is always below the urethra. That indicates the UI is caused by the upright body position. This assumption is supported by the observation that incontinence is seldom a problem when the woman is in a horizontal position (sleeping). This dependence of body position and the day/night difference led us to assume that UI is not a neurological disorder (“nerves don’t sleep at night!”) but a problem of a changing anatomy of the bladder and its anatomical surroundings. We hypothesized that the critical part of the problem is the bladder outlet to the urethra. Since we do not know the exact localization of the problem we decided to define that area as the Urethra-Vesical Junction (UVJ). (Incidentally, the same pathophysiology could be applied to UI in men after total resection of the prostate) (It is tempting to speculate that decreasing levels of the ovarian hormones play a critical role in the pathogenesis and pathophysiology of UI.)

Treatment Must Result in Repair of the Causes

If UI is a pathophysiological continuum of one defect than the cure rates of SUI by Trans-Vaginal-Tapes (TVT) or Trans-Obturator Tapes (TOT) demonstrate that surgery, i.e. the anatomical repair in the anterior part of the bladder base, is the right way to go. This effect is explained by a passive closing of the urethra when the pressure on the bladder base (and the UVJ) is increased and the urethra is compressed between the bladder and the tape. Therefore, these tapes are a correction of the UVJ anterior part. In patients with the more advanced disorder (UUI) an additional part of the UVJ must be affected. We assumed that this could be the posterior part of the UVJ. The area of this defect the Urethral-Vesical Junction (UVJ) - is located on the upper/frontal part of the vagina. The length of the (unoperated, not stretched) vagina usually varies between 8.5 cm and 9.5 cm. The

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*Correspondence:

Wolfram Jager, Department of
Urogynecology, University of Cologne,
Germany,

E-mail: wolframjaeger@gmx.de

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length of the urethra before it reaches the UVJ is between 2 cm and 5 cm. When we consider the distance between the hymenal rings to the external opening of the urethra and add 1 cm than the UVJ must be in the range between 3 cm and 6 cm from the remnants of the hymenal ring in the anterior vagina. The UVJ is tightly fixed at the anterior vaginal wall and when the bladder needs to be elevated from the anterior vaginal wall e.g. for anterior colporrhaphy, this fixation side has to be dissected with scissors. Considering the length of the vagina, the critical areas are between the first-third and the second-third of the length of the vagina. In the upright body position that part of the vagina is pulled downwards by the vertical pressure of the bladder. If that is accompanied by a partial prolapse of the uterus, a tension downwards will accompany the traction on the closing mechanism of the UVJ. This will lead to further opening of the UVJ. That can easily be found during a vaginal examination where the patient standing position is with a “not empty” bladder.

Correct Treatment will Lead to the Disappearance of Symptoms and Return to Normal

The respective treatment for these different phases of urinary incontinence would be the surgical repair of the defect. The TVT/TOT was the first steps on the way to a reproducible success (“cure”). In order to take that tension from the posterior part of the UVJ, we decided to elevate the bladder by elevating the upper part (half) of the vagina. We replaced the uterosacral ligaments by PVDF ligaments with the physiological length. We realized that the length of the uterosacral ligaments was important for the success of that treatment. When standardized, the CESA (cervico-sacropexy) or the VASA (vagina-sacropexy) in patients with urinary incontinence, we observed immediate restoration of continence in a third of all the patients. When we then added a TOT 75% of patients became continent again [www.cesa-vasa.com]. Clinically, these patients presented with the typical symptoms of Urgency Urinary Incontinence (UUI) and MUI

as mentioned above. However, when we started to treat these MUI patients with a TOT alone, no continence was obtained. One has to speculate that the effects on the anterior urethra/bladder by the sub-urethral tape cannot lead to continence when the posterior part of the UVJ/vagina is not strengthened by a mechanical apical support. That support must exert the effect on the upper part of the defect. So, the remaining incontinent patients must have a defect between the anterior and posterior part of the UVJ. When we started to focus our surgical attempts on the UVJ we immediately recognized that the missing gap was closed with that step.

That Led to the Disappearance of all Symptoms

It is now only a matter of standardization to cure urinary incontinence in women. Our hypothesis is a leverage of the “hammock-hypothesis” and the “Integral Theory”. Smaller defects can be closed by a TOT. When the underlying pathogenesis is based on decreasing hormonal levels (loss of tension of pelvic muscles) then the development cannot be stopped and the next defect will appear. Associated with a (even minor) prolapse of the anterior vagina, the patients have more problems to avoid involuntary loss of urine and develop UUI. When the upper part of the urethral outlet is elevated by elevating the prolapse by CESA or VASA continence is established. But if the defect even touches the outlet directly further support of this area leads to continence. We have already done large steps to cure incontinence in women. We assume that the final steps will be done soon and patients then will “feel free!” to do what they want whenever they want and whatever they want without losing urine anymore.

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