Short Communication Open Access

There is a Chance to Cure Urinary Incontinence in all Women!

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Short Communication

Urinary incontinence (UI) is a widespread disorder especially of the elderly women. Considering only women who asked for medical treatment it was calculated that every third woman would suffer from UI during her lifetime [1].

The symptoms, which make UI a bothering disorder, can be summarized as involuntary urinary loss.

The etiology of UI is unknown.

UI usually starts with the loss of some drops of urine during coughing or sneezing, i.e. when the intra-abdominal pressure increases. It is a characteristic finding that this urinary "stress urinary incontinence" usually presents as the first symptom of UI. In most patients that starts in their forties [2]. After an undefined time interval of few years most patients have increasing problems to reach the toilet without loosing urine. In order to prevent this involuntary loss of urine these patients increase their voiding frequency and get up from the bed at night.

Because that "loss of control" over the bladder could not be attributed to a specific cause the reason for this kind of UI remained a matter of speculation. Most people speculated the responsible actor either in the brain or the nerves or the bladder muscle itself. They usually favor one of the two main hypothesis:

Either it's a neuronal regulation dysfunction between bladder and brain or the bladder muscle is without any control and contracts on its own

Several treatments were developed according to these hypotheses [3]. They focus on the nervous system or the nerves which influence bladder function. However; after treatment of thousands of women the results were summarized by the Cochrane Collaboration as "only slightly better than placebo" [4].

Only the local injection of Botox in the bladder muscle of the patient can cure the UI. This treatment leads to a paralysis of the bladder muscle so that the patient must catheterize herself. The bladder is paralyzed of all its physiological actions – it becomes a kind of "medical neo-bladder". One may argue if that is a treatment or an action of despair.

The final step is the life with a permanent catheter.

Since the end of the last Century the understanding of the pathophysiology of UI changed. As usual in the beginning only few people started the intellectual change with new assumptions.

DeLancey, Ulmsten and Petros hypothesized that incontinence is not a neuronal disorder or chronic infection of the bladder but they interpreted the UI as a consequence of destroyed pelvic anatomy [5,6]. They changed the scientific focus from the neuronal pathways to changes of the "genital holding apparatus".

As usual – if one cannot convince the auditorium immediately the chance for latter success of the hypothesis is poor ("If the soup is not delicious the guests will be skeptical for the rest of the menu - but usually they remain seated and wait for the next course!")! In the surgical treatment of the pelvic floor in order to cure UI it turned out in another way: the first steps were a great success but the guests "left the restaurant" thereafter.

For practical purposes the urogenital system was divided in three levels or compartments. Based on old experiences one started with the repair of the anterior compartment (Level 1) which is the part of the urethra. It was assumed that in this Level the pubourethral ligaments were defective and different kinds of slings were placed in order to support the defective ligaments.

The success of this surgical approach to repair/replace anatomical structures was dramatic with nearly 80% of patients cured of their stress urinary incontinence.

The same effects were observed with an elevation of the vagina in the region of the urethral-vesical junction ("BURCH operation"). Even the repair of the endopelvic fascia as part of the treatment of a "cystocele" – a typical middle zone (Level 2) defect - led to a cure of UI in a substantial percentage of women.

These treatments were effective for the "mildest" forms of UI but anecdotally it was also noted that more severe forms of UI ("total loss of urine before reaching the toilet") were also cured in some of these patients. However, the scientific community did not pay further attention to these observations.

We focused our interest on the apical fixation of the vagina (Level 3).

We realized that the apical part of the vagina – even still with the uterus – or after hysterectomy – started to "descend" along the vaginal axis predominately after menopause. That is the time of life when most patients become incontinent.

The estrogens seem to be able to keep the "holding system" under tension. That assumption was further strengthened by the observation that patients with hormone replacement therapy developed the descensus after finishing this replacement in their later life. Usually they started to complain about UI within few years thereafter.

So we hypothesized that the drop of the local estrogens levels may play a critical role in the development of UI; however, as a first step these patients needed help to become continent again.

We developed a surgical procedure to fix the vaginal apex in its original position.

We assumed that a repair or replacement of the uterosacral ligaments (USL) would be the essential step. We therefore searched for ways to repair the USL. However, we had to realize that in these patients the histologically composition of the tissue changed dramatically. In histological studies the connective tissue had diminished and could not be repaired. Therefore, we realized that we had to look for a replacement of the USL. Furthermore, the results of sacrocolpopexy clearly indicated that a central single replacement would lead to incontinence. We therefore decided to follow the original anatomy and replace both USL [7].

According to MRT measurements the female bony pelvis has nearly identical dimensions [8]. Therefore, the USL must be nearly identical in all women [9]. Therefore, we standardized this bilateral replacement as vagina-sacropexy (VASA) or cervico-sacropexy (CESA) (www.cesa-vasa.com) using tapes of identical lengths for all patients (Dynamesh, FEG, Aachen, Germany) [10,11].

We immediately realized that just by the bilateral repair of these ligaments between 33% and 62% of patients were cured of their uncontrolled urine loss. In the remaining patients, which were still incontinent after CESA or VASA the repair of the anterior compartment by a TOT more than two-thirds of these women were cured, leading to an overall cure rate between 71% and 77% [12].

These results led us to speculate that the still incontinent patients need an additional repair of the middle zone. This is our research aim for the future.

The experience of the last 20 years has helped us to make a giant step towards cure of incontinence. We learned that urinary incontinence is an anatomical disorder which appears with increasing lifetime. It becomes a widespread disorder in the elderly affecting probably every third woman older than 60 years of age.

So far, there was no cure for them. Now we can cure that disorder by CESA or VASA either alone or in combination with a suburethral sling. The overall cure rate of 77% is not helpful for the patient because either she is cured after surgery or not. But we are very optimistic that the near future will close the remaining gap.

While incontinence leads to social isolation continence restoration brings them back into the center of the society.

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