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# THE SURGICAL TREATMENT OF URGE- AND MIXED URINARY INCONTINENCE – IS THE OUTCOME DEPENDANT ON THE POP-Q STAGE?

### Hypothesis / aims of study

DeLancey, Ulmstein, and other researchers have stated that urinary incontinence (UI) in women is not a neurological or inflammatory disease. According to their hypotheses, UI is caused by a defect in the holding apparatus of the bladder (i.e., the endopelvic fascia). This theory is supported by the finding that UI symptoms only occur in the upright body position.

When intact, the endopelvic fascia supports the urinary bladder in maintaining its position. The endopelvic fascia extends from the cervix to the os pubis and is fixated at its apical, basal, and lateral ends. If the fascia loses its tension, UI symptoms occur. A defect in any part of the fascia can lead to incontinence.

Pelvic organ prolapse (POP) is caused by a defect in apical fixation. The uterosacral ligaments (USLs) prevent POP and therefore incontinence. If these ligaments weaken, POP and UI symptoms occur.

Cervico–sacropexy (CESA) and vagino–sacropexy (VASA) are established surgical methods for physiological and bilateral USL replacement through the elevation of the anterior vaginal wall. Therefore, these methods can be used to treat an apical defect in the endopelvic fascia.

Decreased tension in the basal parts of the endopelvic fascia also leads to UI; this condition can be treated using transobturator tape (TOT).

The lateral suspension of the fascia can be another cause for the loss of tension. Furthermore, structural deficits in the fascia may be caused by connective tissue or hormonal deficits, which consequently might cause a loss of tension in the fascia. Therefore, no diagnostic or treatment method has yet been established. We focused on the apical and basal fixation of the fascia in this study. Women with POP and UI were treated through CESA or VASA. If the patients remained incontinent after the first procedure, additional TOT placement was proposed to them.

We estimated that women with advanced POP [POP-quantification system (POP-Q) stages II–III] would present more favorable outcomes for continence than would those with less advanced POP (POP-Q stage I). This study compared these two groups in terms of the continence rates after CESA or VASA, followed by TOT application (if necessary).

#### Study design, materials and methods

The incontinence symptoms of the patients were evaluated by conducting interviews by using established questionnaires. Woman with urge UI (UUI) or mixed UI (MUI) were included if they had previously received conservative treatments for their UI symptoms but without success. Patients with only stress UI, POP-Q stage 0, or body mass index > 35 kg/m² were excluded.

The patients were divided into two groups depending on their POP-Q stage. Group A consisted of patients with POP-Q stage I, and group B included patients with POP-Q stages II and III.

CESA or VASA was performed after obtaining informed consent from the patients; the patients were reevaluated at 4 months postoperatively. If they still presented with UI symptoms, TOT placement was proposed. If they accepted, the TOT was implanted, and they were reevaluated 4 months postoperatively.

Groups A and B were compared in terms of the continence rates postoperatively.

#### Results

A total of 211 patients were included. Group A comprised 165 patients (UUI: n = 40 and MUI: n = 125), and group B included 46 patients (UUI: n = 16 and MUI: n = 30).

Through CESA or VASA, continence was reestablished in 19 of 40 (48%) and 11 of 16 (69%) patients with UUI in groups A and B, respectively (p > 0.05).

#### Interpretation of results

Reestablishing the apical fixation of the endopelvic fascia led to continence in 26%–43% and 48%–69%patients with MUI and UUI, respectively. Through additional TOT placement as treatment for the basal fixation of the endopelvic fascia, overall continence rates of 63%–65% and 85%–92% were achieved for patients with MUI and UUI, respectively. Thus, the severity of POP had no significant influence on the outcome.

#### Concluding message

UI is not a neurological or an inflammatory disease. According to the hypotheses of DeLancey, Ulmstein, and other researchers, UI is caused by a defect in the holding apparatus of the bladder. The endopelvic fascia supports and maintains the bladder in a stable position, and a loss of tension in this fascia leads to UI.

In women with POP and UI, the apical fixation of the fascia is no longer intact. Standardized bilateral and physiologic USL replacement through CESA or VASA is an effective treatment for POP and has resulted in reestablished continence in 26%–69% of patients.

After the application of additional TOT as treatment for the basal defects in the endopelvic fascia, 63%–92% of patients regained continence.

POP severity had no significant influence on continence outcomes.

In patients who remained incontinent after CESA or VASA + TOT, a defect in the lateral fixation of the endopelvic fascia or structural deficits in the fascia may be present.

Group A			UUI n=40			MUI n=125				
POP-Q I	cont	inent	incontinent		mv	continent		incontinent		mv
	n	%	n	%		n	%	n	%	
CESA/VASA	19	(48)	21	(52)		32	(26)	93	(74)	
TOT	4*	(50)	4*	(50)	3	34	(47)	39	(53)	
overall continence rate	23*	(85)	4*	(15)	4	66	(63)	39	(37)	
	*10 patients recieved no TOT					*20 patients recieved no TOT				

Group R			UUI n=16			MUI n=30				
Group B POP-Q II - III				ncontinent mv		continent		incontinent		mv
	n	%	n	%		n	%	n	%	
CESA/VASA	11	(69)	21	(31)		13	(43)	17	(57)	
TOT	1	(50)	4	(50)		2	(20)	8	(80)	
overall continence rate	12	(92)	4	(8)		15	(65)	8	(35)	
	*3 patients recieved no TOT					*7 patients recieved no TOT				

Additional TOT was placed to achieve continence in 4 of 8 (50%) and 1 of 2 (50%) patients with UUI in groups A and B, respectively (p > 0.05).

Äfter CESA or VASA, 32 of 125 (26%) and 13 of 30 (43%) patients with MUI in groups A and B, respectively, achieved continence (p > 0.05).

Additional TOT restored continence in 34 of 73 (47%) and 2 of 10 (20%) patients with MUI in groups A and B, respectively (p > 0.05).

Thus, after excluding the patients who rejected a necessary TOT, overall continence rates of 63%-85% in group A and 65%-92% in group B were achieved.

No statistically significant differences were noted between the two groups (p < 0.05).

## References

- 1. DeLancey JO: Structural support of the urethra as it relates to stress urinary incontinence: the hammock hypothesis. American journal of obstetrics and gynecology 1994; 170: 1713-20; discussion 20-3.
- 2. Petros PE, Ulmsten UI: An integral theory and its method for the diagnosis and management of female urinary incontinence. Scandinavian journal of urology and nephrology Supplementum 1993; 153: 1-93.
- 3. DeLancey JO: The anatomy of the pelvic floor. Current opinion in obstetrics & gynecology 1994; 6: 313-6

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