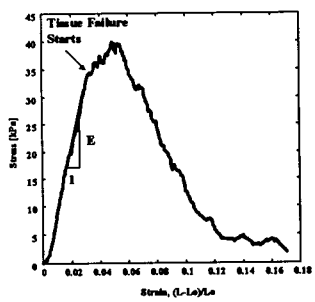
Nonparametric analysis of variance shows a significantly greater ultimate stress (2.5 kPa vs. 1.1 kPa, p=0.006) and stiffness (8.1 kPa vs. 3.5 kPa, p=0.013) of the posterior vaginal wall/adventitia in the non-prolapse group. This indicates greater tensile strength in the non-prolapse group in the tissues of the posterior compartment. There is no difference between the mechanical properties of the uterosacral ligaments in these two groups.

Comparing anterior and posterior vaginal wall/adventitia within the prolapse group, there are no significant differences in mechanical properties, indicating these are similar tissues. Comparing these tissues with the uterosacral ligaments in the prolapse group, the ligaments have a significantly greater ultimate stress and stiffness. This implies that these tissues are different. The uterosacral ligaments are composed of endopelvic fascia, while the adventitia of the vesicovaginal wall is loose areolar tissue [1]. In the no – prolapse group, there is no significant difference between the anterior and posterior vaginal wall, but this tissue had a significantly greater ultimate stress and stiffness than the uterosacral ligaments.

CONCLUSIONS: This research shows the anterior and posterior vaginal walls/adventitia have different mechanical properties than the uterosacral ligaments, providing evidence that these tissues are not the same in composition. Therefore, insufficient mechanical strength of the posterior vaginal compartment may be one of the primary causes of pelvic organ prolapse. Once prolapse occurs in the posterior compartment, the force vectors in the pelvic organ system changes, resulting in an increased load placement on the uterosacral ligaments. We hypothesize that since there is no enhancement in the inherent tissue strength after prolapse repair with native tissue, and the same causative intra-abdominal forces are present, recurrence of the prolapse may occur.

Figure 1. Typical Stress-Strain Curve showing ultimate tensile stress and strain at tissue failure. Stiffness (Young's modulus) is described as the slope of the linear part of the curve obtained by linear regression.



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J. Manning A. Korda C. Benness

Urogynaecology Unit, Royal Prince Alfred Hospital

Sydney AUSTRALIA

DOES UTEROVAGINAL PROLAPSE CAUSE VOIDING DYSFUNCTION?

Aims of Study

Uterovaginal prolapse and voiding dysfunction often coexist. Large cystoceles are considered to cause voiding dysfunction (1,2). However, the relationship between voiding difficulties and other forms of uterovaginal prolapse is unclear. The aims of this study were to further assess the role of large cystoceles in causing voiding difficulties and to determine whether other forms of uterovaginal prolapse , particularly rectocoeles, cause voiding dysfunction.

Methods

1200 consecutive women referred for urodynamic evaluation were assessed. Details of symptoms, urodynamic diagnosis, pelvic examination findings (3) and relevant medical, surgical, obstetric and demographic information were recorded. Investigations included uroflowmetry, subtracted cystometry, radiological imaging, urethral pressure profilometry and pelvic examination. A diagnosis of voiding dysfunction was made following assessment of symptoms, peak flow rate and voiding pattern and urinary residual. The prevalence of voiding dysfunction was compared between groups of women with significant uterovaginal prolapse (3) and age matched controls with no prolapse. Each group was compared with a minimum of 600 controls.

Results

196 women had severe uterovaginal prolapse (at least 3rd degree). The prolapse groupings are indicated in table 1 together with the associated prevalence of voiding dysfunction. Nearly 1 in 4 women with a large cystocele alone had a diagnosis of voiding dysfunction and this was significantly greater than among controls (Table 1).

TABLE 1 : VOIDING DYSFUNCTION In CASES vs CONTROLS

Prolapse group	age	N=cases	Cases %	Controls %
3rd ° rectocele +/- cystocele	56	113	10.6	9
3rd ° rectocele alone	57	56	12.5	8.1
3rd ° rectocele + 3rd °cystocele	54	57	8.8	8
3rd ° cystocele alone	58	30	23.3*	8.1
>=2nd °uterovaginal or vault prolapse (UV/V)	56	42	24*	8.5
>=2nd °UV /V prolapse , no 3rd cystocele	62	18	11.1	10.2

* = P< 0.05

Interestingly, when large cystoceles and rectoceles co-existed, the prevalence of voiding dysfuction was not increased. The group with 3rd degree uterine or vault descent also had a high prevalence of voiding difficulties. However, this prevalence was no different from controls when those with large cystoceles were excluded. There appeared to be a trend toward more voiding dysfuction in those with 3rd degree rectocele and no concurrent cystocele (12.5% vs 8.1%) Table 2 compares the prevalence of previous anterior vaginal wall surgery between age matched cases and controls.

TABLE 2 : PREVALENCE (%) ANTERIOR VAGINAL WALL SURGERY IN PROLAPSE GROUPS VS CONTROL

Prolapse group	Case- %	Control -%
3rd °rectocele + 3rd °cystocele	17	20
3rd °rectocele alone	38*	21
3rd °cystocele alone	22	21
>= 2nd ° uterine / vaginal prolapse (U/V)	26	21
>= 2nd °U / V prolapse + no 3rd °cystocele	56*	29

* = P< 0. 05

The prevalence of such vaginal wall surgery was significantly higher for those with 3rd degree rectocele alone. This may account for the apparent higher prevalence of voiding dysfunction in these women. This impression is supported by a comparison of 169 women with previous anterior colporrhaphy with 335 age matched controls with no prolapse and no past history of vaginal surgery (Table 3).

TABLE 3 : % VOIDING DYSFUNCTION IN WOMEN WITH PREVIOUS ANTERIOR COLPORRHAPHY vs CONTROLS

Surgery group	n	age	Case %	Control %
no current 3rd ° cystocoele	149	62	10.7*	6.3
recurrent 3rd ° cystocoele	20	66	45**	7.4

**=P=0.00005, *= P=0<0.05

The prevalence of voiding dysfunction among women with a severe cystocele that recurs after anterior colporraphy is 45%, significantly higher than in the control group (7.4%) and significantly higher than among women with a history of successful anterior colporrhaphy (10.7%). Multivariate analysis indicates that voiding dysfunction is significantly associated with increasing age (P=0.0001), severe cystocele alone (P=0.0011) and with a history of anterior vaginal wall surgery (P=0.0088), particularly anterior colporrhaphy (P=0.01). 3rd degree rectocele, 3rd degree uterine or vaginal prolapse, neurologic disease, diabetes and obstetric factors were not significantly related.

Conclusion

This study confirms a strong relationship between large cystoceles and urodynamically proven voiding dysfunction. The highest prevalence is in those with recurrent cystoceles. However we found no evidence that other forms of uterovaginal prolapse cause voiding dysfunction. Coexistent rectoceles may be protective of the voiding dysfunction caused by cystoceles. Anterior vaginal wall surgery is independently associated with voiding dysfunction.

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