median pad test loss was 2.3 grams (IQR 1.18-8.20), digital grade 2 (IQR 2-3) and perineometry measurement 10cmH_20 (IQR 8-15).

Voluntary pelvic muscle floor contraction resulted in a highly significant reduction in the length and surface area of the urogenital hiatus, and in the transverse measurement of the hiatus in the region of the urethra on ultrasound (Table 1). Values are given as mean (SD). The Wilcoxson Signed Ranks Test was used to compare rest and squeeze measurements.

MEASUREMENT	REST	CONTRACTION	SIGNIFICANCE
Surface area (SA) (cm^2)	17.5 (2.8)	16.1 (3.1)	P<0.001
Length (L) (mm)	56.8 (7.4)	53.4 (6.8)	P<0.001
S1 (mm)	34.8 (4.1)	32.7 (4.7)	P=0.001

The change in length of the urogenital hiatus on ultrasound between rest and a maximal contraction correlated with pelvic floor function using perineometry (r=0.437, P=0.029) but not with digital assessment. However, there was no correlation between transvaginal ultrasound, digital grading or perineometry and the degree of leakage as assessed by either cystometry or pad testing suggesting that pelvic floor function is not the major factor in determining the severity of genuine stress incontinence.

CONCLUSIONS

Voluntary contraction of the pubococcygeous results in changes in the dimensions of the urogenital hiatus which can be assessed using transvaginal ultrasound. Maximal pelvic floor contraction produces a reduction in the length of the hiatus and narrowing of its transurethral diameter. The change in length of the hiatus correlates with measurement of pelvic floor strength using perineometry. However, none of the measurements of pelvic floor function correlate with the severity of genuine stress incontinence and we therefore question if their continuing use in the assessment of this group of patients can be justified.

67

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PELVIC FLOOR CONNECTIVE TISSUE MECHANICAL PROPERTIES: IS THERE AN ASSOCIATION WITH PELVIC ORGAN PROLAPSE?

AIMS OF STUDY: The purpose of this research was to measure the mechanical properties of the pelvic connective tissues thought to be associated with prolapse The null hypothesis is that the mechanical properties of these pelvic tissues in women would be the same regardless of prolapse.

METHODS: A prospective observational study enrolled thirty-four women undergoing benign hysterectomy, vaginal or abdominal. Five abdominal and 29 vaginal hysterectomies were performed. The Pelvic Organ Prolapse Quantification system was used on 27 patients, and the Baden half-way system on seven others, extrapolated to POPQ staging. Patients with POPQ stage 0 – 1 were designated no prolapse, stage 2 – 4 was designated as prolapse. Exclusion criteria included prior hysterectomy, bladder surgery, cone or LEEP of the crevix, personal history of connective tissue disease or chronic steroid use. At surgery for prolapse, tissues obtained were: vaginal wall anteriorly and posteriorly with underlying connective tissue; vesicovaginal adventitia, rectovaginal wall with connective tissue, and uterosacral ligaments bilaterally. Anterior and posterior vaginal wall and uterosacral ligaments were obtained from women without prolapse. Mechanical properties measurements were done on a servohydraulic-testing machine, after 10 pre-conditioning cycles. Cross-sectional area and initial gauge length were measured with a digital caliper. The tissue was loaded in tension until failure. A typical stress – strain curve is shown in Figure 1. Ultimate tensile stress, strain and stiffness were calculated. T-tests for independent means, paired t-tests for within group differences, non-parametric tests for comparison of means and regression analysis for outcome variables were used where appropriate.

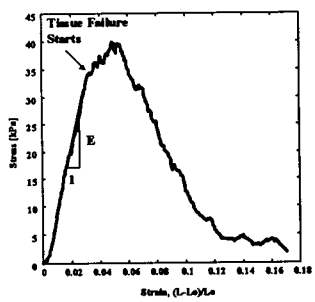
RESULTS: Regression analysis shows no correlation between age, weight or parity, and stress, strain and stiffness. Mechanical properties are not significantly different when the effect of hormones on prolapse is analyzed in the groups of premenopausal (endogenous) and postmenopausal (exogenous HRT) patients vs. postmenopausal patients without hormones. No significant difference is found as a function of hormone replacement in postmenopausal women with prolapse.

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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68

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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.