Results: 56 women were recruited in total (23 in the prolapse group and 33 in the control group). The mean age in the prolapse group was 40 (range 28-50) and in the control group 40 (range 27-51). The mean parity in each group was 2.9 and 2.2 respectively. The glycation end products are expressed as mole of collagen per mole pentosidine where a reduction in levels indicates an increase in pentosidine present.

	Control (n = 33) Median (range)	Prolapse (n = 23) Median (range)	Significance
Total collagen (%dry wt)	50.6 (23.0 – 79.2)	27.7 (16.7 – 37.3)	< 0.001
Protein Content (%dry wt)	89.2 (58.3 – 99.6)	85.3 (41.1 – 99.2)	0.48
DSC (Pk Ht °C)	67.7 (65.9 – 69.8)	66.5 (65.1 – 68.5)	0.06
Proteoglycan (µg/g wet wt)	5944 (3103 – 9594)	3711 (2020 – 14865)	0.04
Metalloproteinase (/µg coll) MMP 2 expression	3.27 (0.20 – 8.01)	15.42 (3.35 – 42.14)	< 0 001
Immature cross-links (/mole coll)			
HLNL	0.11 (0.00 – 0.21)	0.18 (0.04 – 0.72)	< 0.001
DHLNL	0.05 (0.00 – 0.20)	0.06 (0.01 – 0.40)	0.7
Mature cross links (/mole coll)			
Pyridinoline	0.14 (0.04 – 0.45)	0.42 (0.04 – 1.26)	< 0.001
HHL	0.16 (0.02 0.34)	0.49 (0.08 – 2.07)	< 0.001
Glycation end products (mole/mole) Pentosidine	333 (90 – 1221)	113 (51 – 268)	< 0.001

Conclusions: Women with genitourinary prolapse appear to have an alteration in collagen metabolism causing a decrease in total collagen therefore reducing its ability to provide structural support. The increase in intermediate cross linking indicates de novo synthesis is occurring but not at a level to restore the overall collagen content. The extracellular components (proteoglycans and protein) decrease making it unlikely that dilution of the tissue leads to the reduction in collagen. Gelatinase activity (MMP2) indicates turnover of the collagen is occurring but it is the new collagen rather than the old being degraded which is indicated by the increase in advanced glycation end products. The peak temperature at denaturation is reduced in the prolapse group although not significantly and as there is no reduction in cross links this would suggest that the prolapse collagen is damaged. Enthalpy values measuring the energy required to denature collagen should corroborate this and is part of our ongoing work.

1. *The Lancet* 1996; 347:1658-1661.

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QUANTITATIVE ELECTROMYOGRAPHIC ANALYSIS OF LEVATOR ANI AND EXTERNAL ANAL SPHINCTER MUSCLES OF NULLIPAROUS WOMEN

Aims of study: To introduce the use of digital quantitative electromyography (QEMG) of the levator ani and external anal sphincter, and establish reference values for these muscles. QEMG is both reproducible and independent of the operator, and is a technique routinely used in the analysis of striated muscles outside the pelvis. We present a study using QEMG to evaluate pelvic muscles in nulliparous subjects.

Methods: Fifteen nulliparous women without symptoms of pelvic floor dysfunction underwent EMG examination of the levator ani (LA) and the external anal sphincter (EAS) using a concentric needle electrode with a recording area 0.07mm² and diameter of 0.45mm (Medelec NDMC 37). The levators were sampled via a transvaginal approach at two sites on each side: on the levator near the insertion of the levator arcus into the ischial spine, and 1 cm medial to the first site. Muscle bellies palpated at these sites were presumed to be either iliococcygeus or puborectalis. Two sites on the left and right of the posterior external anal sphincter were sampled just inside the mucocutaneous junction. The resulting signal was filtered (range 5Hz-10KHz) and amplified and recordings made at three levels of voluntary activation at each site: at rest, at partial activation, and at maximal activation. Epochs of up to 20sec of each site and activation level were recorded on digital audio tape (Sony DTC-A8). Analysis of taped signals was performed on a Medelec Synergy electromyograph capable of analyzing motor unit action potentials (MUAPs) and the interference pattern (IP). Measurements were made of MUAP amplitude, area, amplitude/area ratio, duration, number of phases and turns, and of the activity, envelope amplitude (env amp), and number of small segments of the interference pattern for both muscles. Normal ranges were generated and compared to those established for other striated muscles.

Results: The mean age of the group was 28.7±7.5 years (range 20-49, median 25). A median of 24 (range 12-44) MUAPs were recorded in each LA, and median of 6 (range 2-13) MUAPs in each EAS. IP was measured in 12 epochs in the LA and 6 epochs in the EAS. Mean and standard deviations for parameters are presented below:

	MUAP Amplitude (μV)	MUAP Duration (msec)	Turns/sec	Amplitude/Area Ratio
LA	480 ± 200	10.5 ± 2.2	2.8 ± 0.8	1.44 ± 0.3
EAS	370 ± 130	8.3 ± 2.5	2.3 ± 0.4	0.91 ± 0.4
Biceps	364 ± 296	10.58 ± 5.1	2.14 ± 0.99	1.75 ± 0.7

	IP Amplitude (μV)	IP Activity	IP Env Amp (μV)	Turns/sec	# Small Segments
LA	308.6 ± 58.2	98.7 ± 50.5	879.4 ± 219.7	248 ± 82.8	109.4 ± 62.9
EAS	221.6 ± 42.4	60.8 ± 39.9	560 ± 192.9	181.9 ± 91.8	77.5 ± 52.7

Published values for MUAP of the biceps brachii muscle as a representative limb muscle are shown for comparison(Musc & Nerve,18:1155,1995). LA,EAS,and biceps parameters are similar. Conclusions: EMG of the LA and EAS is feasible and well tolerated. We have established normal ranges for important QEMG parameters for both the LA and EAS in a nulliparous group of subjects. Ongoing study involves comparison of these data to that of older normal subjects and that of patients with pelvic floor dysfunction.

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VARIATION IN QUANTITY AND DISTRIBUTION OF NEURAL TISSUE IN THE STRIATED UROGENITAL SPHINCTER MUSCLE

Aims of Study: A neurogenic hypothesis proposing that pudendal nerve injury causes stress urinary incontinence (SUI) is based on prolonged conduction times. These conduction delays, however, are not sufficient to plausibly affect continence. Nerve damage can cause muscle loss. If loss of neural tissue caused loss of sphincter muscle and sphincteric weakness, this could affect continence.

Previous work has shown decreased muscle cells in the striated urogenital sphincter of women (1). The present study was undertaken to analyze 1) the quantity and 2) the distribution of neural tissue within the striated urogenital aphincter muscle (SUGS) and to relate changes in neural tissue and number of muscle cells present.

Methods: Thirteen cadaveric urethras (mean age 47 years, range 15-78,) from our earlier work had sufficient tissue remaining for further analysis and were selected for study. Five of these samples were from nulliparous women, seven from parous women and in one cadaver, parity was unknown. None of the patients had neuro-muscular disease that would affect the pelvic floor.

A sagittal histologic section was made from the paraffin blocks. An S100 stain was used to identify neural tissue. The upper end of the SUGS region began at the caudal end of the detrusor loop and the lower end, at the caudal extent of the striated muscle. This region was then divided into ten equal segments called "deciles". The number of times that a nerve section was seen within the SUGS ("*nerve number*") was counted under 400X magnification of a microscope (1). The axons within each nerve fascicle were similarly counted. The total number of nerves and axons seen in the section within the SUGS was obtained by adding the number of nerves and axons obtained at every 10% of urethral length. Regression analysis between nerve density and muscle cell number was performed using a statistical program (Statview®, Abacus Concepts, Berkeley, California). Comparative data was available for 11 urethras. Regression analysis between cadaver age and the nerve density was also carried out.

Results: Remarkable variation was seen in the quantity of neural tissue in the sampled region of the SUGS. There was a 7-fold variation in number of nerves present. Nerve counts ranged from 72 to 543 (mean 247.1 SD 123.24), and the range of number of axons was 431 to 3523 (mean 2201 SD 1152.6).