Original article

Interventional manometry: transvaginal support of pelvic floor ligaments raises endoanal pressure

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Abstract: *Background:* The interaction between pelvic floor ligaments and muscles and anorectal pressure is not well characterized. Pelvic floor muscle vectors act against pelvic floor suspensory ligaments, including pubourethral (PUL) uterosacral (USL) and, inferiorly, perineal body (PB). Laxity of the pelvic floor leads to reduced endoanal pressures at rest and during a voluntary anal squeeze. *Aim:* We tested pelvic floor function using a new technique; interventional anal manometry. *Methods:* In a heterogeneous group of 14 women, with pelvic floor dysfunction of various causes, anorectal pressure measurements were obtained at rest and during maximal voluntary anal squeeze, before and during the following per-vaginal interventions, a] digital support at midurethra to support the pubourethral ligament (PUL); b] after a 3x6 cm tampon was inserted into the posterior fornix to support the uterosacral ligaments (USL); c] with combined PUL and USL support; d] with PUL, USL and perineal body (PB) support. *Results:* Resting and squeeze anorectal pressures increased during the support manoeuvres described, especially during experiment [d]. *Conclusions:* Creation of firm insertion points at PUL, USL, PB enabled muscle vectors to act more efficiently, leading to increased endoanal pressure. These interventions do not increase pressure generation by internal (IAS) and external sphincters (EAS) themselves, but result from changes in anorectal cross-sectional area. "Interventional manometry" offers a method for better understanding functional abnormality in the pelvic floor in women with clinical problems due to pelvic floor weakness.

Key words: Anal manometry; Pubourethral ligament; Uterosacral ligament; Perineal body; Pelvic floor laxity.

INTRODUCTION

Anorectal manometry correlates poorly with diagnosis of pelvic floor dysfunction and prediction for cure after an intervention. Nonetheless resting anal pressure measurements in individuals are reproducible.[1] Although anorectal resting pressure is reduced in subjects with faecal incontinence, there is insufficient sensitivity and specificity to consider anorectal pressure measurement as a specific diagnostic test.[2,3]

We have previously reported that upward pressure applied at midurethra by digital support of the pubourethral ligament caused a mean increase in endoanal pressure of 47cm water in the control group, and a mean 30 cm water pressure increase in a group of women with faecal incontinence ($p = 0.034$), suggesting less tight anal closure by directional muscle forces in the group with faecal incontinence.[4] This increase in endoanal pressure was considered to be due to improved efficiency of the muscle closure mechanism caused by supporting the ligament at the muscle insertion point.[5]

Therefore, as a test of principle, we have studied the effect of pelvic muscle contraction against pelvic floor suspensory ligaments and the perineal body (Figure 1) by measuring endoanal pressure before and during transvaginal digital support to these ligamentous structures in a heterogeneous group of women with pelvic floor disorders.

PATIENTS AND METHODS

Fourteen patients were studied. Eight presented with anorectal pain, five presented with constipation and one had double urinary and faecal incontinence. Mean age was 49.9 years (range 36-64). The principles of the Helsinki Declaration of 2008 were followed.

The interventions

Digital support of the pubourethral ligament (PUL)

The examiner's index finger was inserted into the vagina immediately behind the pubic bone at the level of midurethra and gently pressed upwards.

Tampon support of the uterosacral ligaments (USL).

A 3x6 cm tampon was inserted into the posterior fornix to support the uterosacral ligaments (USL).

Digital support of the perineal body (PB).

Two fingers were inserted into the vagina and separated laterally to support the right and left perineal bodies, taking care not to compress the anus.

Endoanal pressure measurements

Pressure measurements were performed using a ManoScan 360 High-Resolution catheter-based Anorectal Manometry System (Given Image Company, Israel).

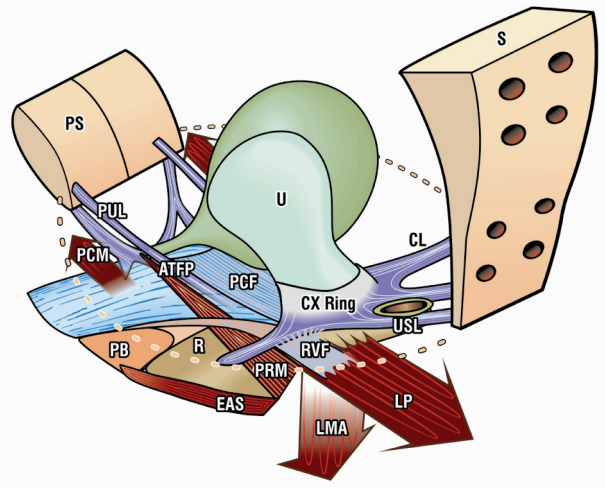

Figure 1. - Muscle vectors (arrows) showing planes of contraction forces against suspensory ligaments and perineal body. Patient in sitting position. According to this model, the action of anal canal "squeezing" is activated by forward contraction of m.puborectalis (PRM) which inserts directly into the posterior surface of the pubic symphysis (PS). M.pubococcygeus (PCM) contracts against the pubourethral ligament (PUL) anteriorly. M. levator plate (LP) contracts against the pubourethral ligament (PUL) anteriorly and against the perineal body (PB) inferiorly via its rectovaginal fascial attachment (RVF). The longitudinal muscle of the anus (LMA) contracts against the cardinal/uterosacral (CL/USL) complex. EAS=external anal sphincter.

Anorectal pressure measurements were made at rest and on maximal voluntary anal squeezing when supporting PUL only, USL only, both PUL and USL, and a final reading when simultaneously supporting all points, i.e., PUL, USL and perineal body (PB).

Statistical analysis:

We used Student's two-tailed t test to test for significance in the paired groups of data. Calculations were performed with Graphpad software.

RESULTS

The mean resting unsupported endoanal pressure before intervention was 70 mmHg (range 49.5-90.3mmHg). The mean unsupported maximum anal squeeze pressure was 125.3mmHg (range 86.6-184.4mmHg). With digital support as described above there was an increase in resting and maximal voluntary squeeze pressures, especially marked with combined support of PUL, USL and PB. Resting pressure increased by 28% and squeeze pressure by 22%. There was relatively little benefit when PUL or USL were supported alone. The full results are given in Table 1. Since the small number of patients studied were heterogeneous in terms of their clinical diagnosis no attempt was made to differentiate the results according to different syndromes.

TABLE 1. – Mean endoanal pressure measurements at rest and during voluntary anal squeeze with the pubourethral (PUL), uterosacral (USL) pelvic floor ligaments and perineal body (PB) unsupported and digitally supported per vaginam. Differences between the mean values at rest (columns 1 and 2) and between mean values during voluntary anal squeeze contraction (columns 3 and 4) were tested using Student's two-tailed t test.

	Resting pressure (range, mm Hg + SD)	**p vs baseline**	**Squeeze pressure (range, mm Hg + SD)**	**p vs baseline**
Baseline	70.1+14.5		125.3+30.5	
	(49.5-90.3)		(86.6-184.4)	
PUL support	81.2+18.5	0.01	130.8+29.9	NS
	(47.2-104.2)		(83.6-175)	
USL support	77.8+16.8	NS	140.5+31.9	0.01
PUL+USL	88.0+21.6	0.008	148.7+27.4	0.008
	(47.8-115.3)		(115.9-210)	
PUL+USL+	90.5+31.1	0.001	153+37.8	0.01
PB support	(56.5-166)		(101.5-242.2)	

DISCUSSION

The pubococcygeal (PCM), levator plate (LP) and conjoint longitudinal muscle of the anus (LMA) components of the pelvic floor musculature insert into pelvic floor connective tissue in relation to the anal canal and, in women, the vagina.[6, 7] These muscles contain a predominance of slow twitch fibres, and are thus adapted to tonic contractile force.[8] Contraction of these muscles stretches and narrows the anorectum causing an increase in intra-anal pressure. In patients with pelvic floor dysfunction, for example, prolapse and faecal or urinary incontinence, pelvic floor muscles are weak and there is associated with laxity and lack of elasticity of pelvic floor ligaments, so that muscle contraction is less effective. Anal squeezing is an important protective mechanism in the maintenance of faecal continence. Our understanding of the dynamics of pelvic floor function (Figure 1) implies that supporting the pubourethral ligament would cause little if any increase in anorectal pressure. We confirmed this in this study (Table 1).

The uterosacral ligament (USL) is attached to the lateral side of the rectum by fascia. Mechanical support of the uterosacral ligament alone creates a firm anchoring point superiorly, leading to an enhanced forward vector during contraction of the puborectalis muscle (PR) which we consider is the main muscle activated during a voluntary anal squeeze. This leads to increased anorectal pressure on squeezing, associated with narrowing of the intra-rectal luminal area. It can be seen from Fig 1 that digital support of the rectovaginal fascia (RVF) and perineal body (PB) should improve levator plate (LP) contraction. This contractile force stiffens the anterior wall of the rectum, permitting a stronger force vector to act during anorectal closure by the puborectalis muscle.

However, viewed from a basic physics perspective, it is not the intra-anal pressure itself which influences continence or defecation, but the frictional resistance to the flow of faeces within the anorectum. In a mathematical model, Bush (9) showed that this resistance followed an exponential relationship with the radius of the anorectum, being inversely proportional to the 3rd power of the radius. However, anorectal diameter is not uniform and this relationship is therefore complex. In addition, local factors such as lubrication of the anorectal wall by mucus, and stool consistency, will both play a role in anorectal frictional resistance to the passage of faeces. Anorectal pressure (Pressure=Force/Area) is itself derived inversely from the 2nd power of the anorectal radius since area can be simplified as $\pi r2$. These concepts indicate that resistance to faecal flow, and anorectal pressure, are related phenomena, so that the increased pressure which we measured on anchoring pelvic floor ligaments would also increase the intra-anal resistance. Thus, changes in anorectal pressure induced by appropriate correction of pelvic floor ligamentous laxity will improve symptomatic pelvic floor dysfunction.

The question arises, "Does interventional manometry have any practical value?" and "Can it serve as a predictive test?" There is evidence that reinforcing the PUL and/or USL suspensory ligaments may improve pelvic floor support and therefore anal closure (10,11) or obstructive defecation (11-13). We consider it likely that pressure measurements made before and after digital support of specific pelvic floor ligaments may become a useful predictive test in planning surgical repair procedures based on ligament reconstruction (10-13). However, this suggestion requires that appropriate measurements should be made before and after surgical techniques designed to restore normal continence by repair of suspensory ligaments and the perineal body in specific pelvic floor syndromes. Such studies remain to be carried out.

CONCLUSION

We have presented a new concept in clinical investigation of the pelvic floor, which we have termed "Interventional manometry". This technique requires digital reinforcement of the ligamentous insertion points of the pelvic muscles while measuring intra-anal pressure. We interpret the results of these interventions as improving muscle vectors during pelvic floor muscular contraction, causing an increased intra-anal resistance by narrowing the anal canal. This technique enables the precise anatomic-physiological deficit due to pelvic floor ligamentous laxity to be characterized and surgical correction to be planned accordingly.

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