Original article

The rhabdosphincter has a role in pressure generation but not continence

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Abstract: Background: The rhabdosphincter is a thin horseshoe shaped layer of striated muscle which sits outside the smooth muscle layer of the mid urethral zone. Though evidence has been presented for the role of a musculoelastic mechanism involving forward contraction of the suburethral vaginal hammock, this tiny structure , the "rhabdosphincter" is still considered to be the main continence mechanism by many authors. *Aim:* To assess the contribution of the rhabdosphincter to urethral pressure and continence. *Patients and methods:* Four women, mean age 61 years (range 51-71 yrs), parity 0-4 (mean 2.6) with urodynamically demonstrated genuine stress incontinence and no detrusor overactivity (DO). The pressure exerte d by the "rhabdosphincter" was measured during coughing by single Gaeltec microtip transducers before and after surgical dislocation of the suburethral vaginal hammock during a "tension free" midurethral sling operation performed under local anesthesia. The transducer catheter was oriented ventrally at 12 o'clock to maximize the pressure recorded during rhabdosphincter contraction. Five pressure readings were taken from each patient during coughing, taking care to ensure a valid reading at the high pressure zone.
Results: Intraoperatively, urine loss occurred for the first time, or greatly worsened when the suburethral vagina was disconnected from the urethra. Maximum intraurethral pressure increase recorded on four patients at midurethra was 78, 94,112, and 170% of the preoperative reading. *Conclusions:* The pressure "P" readings are potentially misleading in that they measure only the formula, P=force/area, in this case force from the rhabdosphincter, a weak muscle less important in the closures mechanism of the urethral tube. We hypothesize the rhabdosphincter acts as an adjunctive sealing mechanism.

Key words: Rhabdosphincter; Urethral pressure; Continence mechanisms; Stress incontinence; Urethral closure.

INTRODUCTION

The rhabdosphincter (Figure 1), was described by Gosling[1] as composed of striated muscle shaped in a horse-shoe and located outside the smooth muscle layer of the urethra. It is most prominent in the midurethral area. The posterior portion is relatively thin. The muscle cells are all of the slow twitch variety. These cells were unusually small with diameters of only 15 to 20 μm. DeLancey,[2] studied the anatomical relationships of the urethra to its surrounding structures in the series of dissections and microscopic examinations. He summarized these in terms of their occurrence as a percentage of total urethral length. Zero was taken at the bladder neck. He defined the *intramural urethra* as that length of the urethra traversing the bladder wall and it occupied zero to twentieth percentile. The *midurethra* was that length occupying the twentieth to sixtieth percentile. It was occupied mainly by the striated urethral sphincter muscle. In an extensive anatomical study, Huisman[3] considered that these structures were far too small to constitute an effective sphincter. He demonstrated that in older women, the striated muscle fibres degenerated with time, sometimes consisting only as a few atrophied fibres infiltrated with fatty tissue. Perrucini et al.[4] also demonstrated muscle atrophy with time. Yet, in spite of ultrasound evidence of urethral closure from behind during coughing or straining[5, 6] this tiny structure is frequently considered as constituting the main continence mechanism.

The aim of this study was to assess the contribution of the rhabdosphincter to urethral pressure and continence under stress (coughing) by isolating it from the musculoelastic closure mechanism.

PATIENTS, MATERIALS AND METHODS

Ethics

The operation was approved by local IRB and all patients signed informed consent.

Pre-operative investigations

Initially five patients were tested. All patients attended for testing with a comfortably full bladder, and were urodynamically tested on an Ormed 5000 system using the methods described by Asmussen and Ulmsten.[7] Instability (including a handwashing test), urethral pressure profile, cough, strain, and "cut-off"(squeeze) pressure transmission ratios, peak urine flow rate, emptying time, and residual urine were routinely assessed. Cough, strain, and "cut-off" pressure transmisssion ratios were repeated a few seconds later in all patients.

Operation

A "tension-free" midurethral sling operation was performed under local anaesthesia in 5 patients, using bilateral paraurethral incisions to dislocate the suburethral vaginal hammock from both pubococcygeus muscles as per the classic Goebell –Stoeckl-Frangenheim operation. The patient was sedated with an opiate analgesic pre-operatively, but received no muscle relaxant. An intravenous line was inserted, and the patient kept sedated, but co-operative with appropriate doses of midazolam (dosage 5-12mgs), administered by an anaesthesist. Prilocaine 1% diluted 1 in 3 (80-100 mls) was used to infiltrate the suprapubic skin, rectus muscles, periurethral and subpubic tissues, bilaterally. In the first part of this operation, bilateral incisions were made in the lateral sulci. A specially designed rigid tunneller with an external cross-sectional diameter of 6mm was inserted through the incisions and entered the Cave of Retzius, before emerging directly above the pubic bone through a prior 1.5 cm horizontal incision. Two plastic inserts were left in situ. After the measurements were completed, a tape was inserted by means of the tunneller in order to create an artificial pubourethral ligament.

Intra-operative pressure transmission testing

This was performed during the operation with a) intact vagina, b) after the two full depth lateral incisions had been made and opened out, with insertion of two plastic inserts

of the IVS tunneller, but prior to completion of the operation. Prior to testing each patient, the bladder was filled with 240 ml of saline. A single Gaeltec transducer was calibrated for accuracy using a water column between 10 and 100 cm H2O high before each operation. The transducer was introduced with the pressure sensor ventrally oriented at 12 o'clock to maximize the pressure recorded during rhabdosphincter contraction. The purpose of orienting the transducer ventrally at 12 o'clock was to render the measurement of the closure force from the rhabdosphincter as relevant as possible.

Pressure recording began within the bladder at a starting distance of between 7cm and 9cm from the external meatus with the patient coughing at each stage. Because the functional urethral length varied considerably between patients, in all patients care was taken to acquire readings in the zone of maximal urethral pressure. A minimum of 5 pressure readings were taken from each patient during coughing, taking care to ensure a valid reading at the high pressure zone.

RESULTS

The mean age was 61 years (range 51-71 yrs), parity 0-4 (mean 2.6). All 5 patients had urodynamic genuine stress incontinence (GSI), with no detrusor overactivity (DO) on urodynamic testing.

Vagina intact

Taking maximum readings only within each patient, mean pressure measured during coughing at the high pressure zone in the 5 patients was 41 cm H2O (range 20-73 cm H2O).

After bilateral incisions in the sulcus, plastic inserts in situ

In two patients the maximum cough pressure within the urethral pressure zone was below 100% of that measured with intact vagina (78% and 94%); in two patients results were in excess of 100% (170% and 112%). No recording was obtained in the 5th patient because of an unforeseen anaesthetic problem at that point of the procedure. Therefore this patient was excluded from the study.

Urine leakage

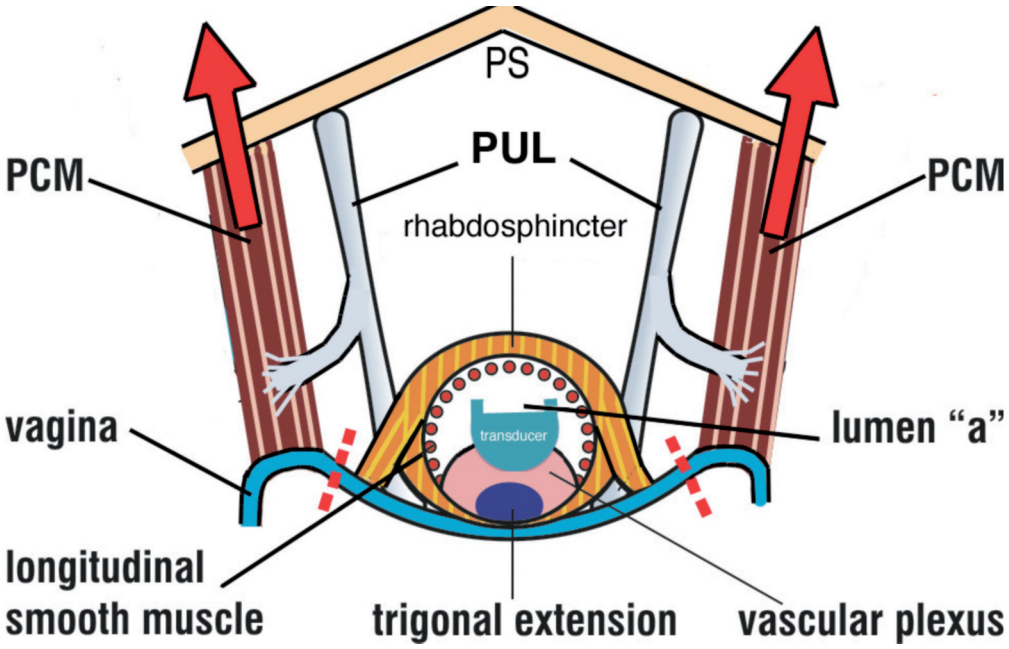
With the vagina intact, slight urine leakage was noted on stress (straining and coughing) in two patients after the bladder had been filled to 240 ml, but no urine leakage in the other two. After the bilateral incisions were made in the sulci, plastic inserts in situ, all four patients lost profuse amounts of urine on coughing. On completion of the operation, urine leakage ceased immediately in all 4 patients.

DISCUSSION

This study raises three important questions:

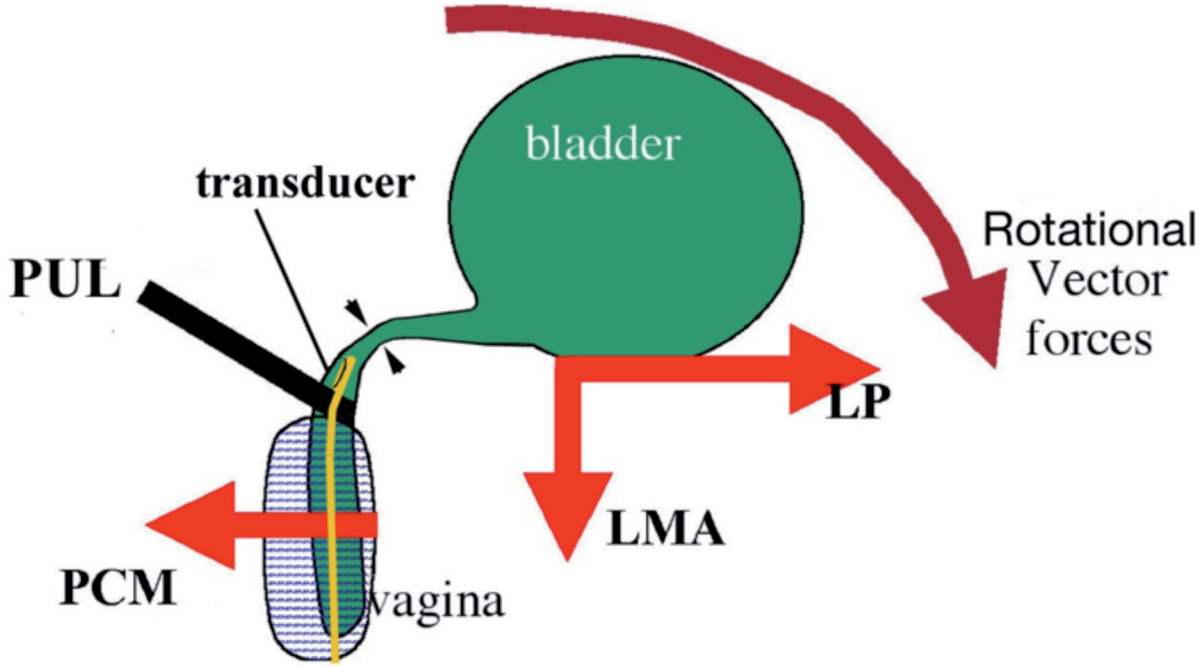
1. *What generates the pressure measured during MUP and during coughing?*
2. *What is the role of intraurethral pressure in continence?*
3. *What is the role of the rhabdosphincter in urethral closure and continence?*

With reference to question 1, "what generates intraurethral urethral pressure", in this study, all pressure measurements were carried out with a vagina dislocated from its lateral attachments to the closure muscle m.pubococcygeus, so that the transducer had to be measuring only the pressure exerted in the space 'a' directly above the transducer (Figure 1), a consequence of rhabdosphincter contraction and urethral stretching by the posterior vectors LP/LMA (Figure 2) which would narrow space "a" (Figure 1), small arrows (Figure 2). As pressure is Force/Area, pressure measured in the four patients was force rhabdosphincter /area 'a'.



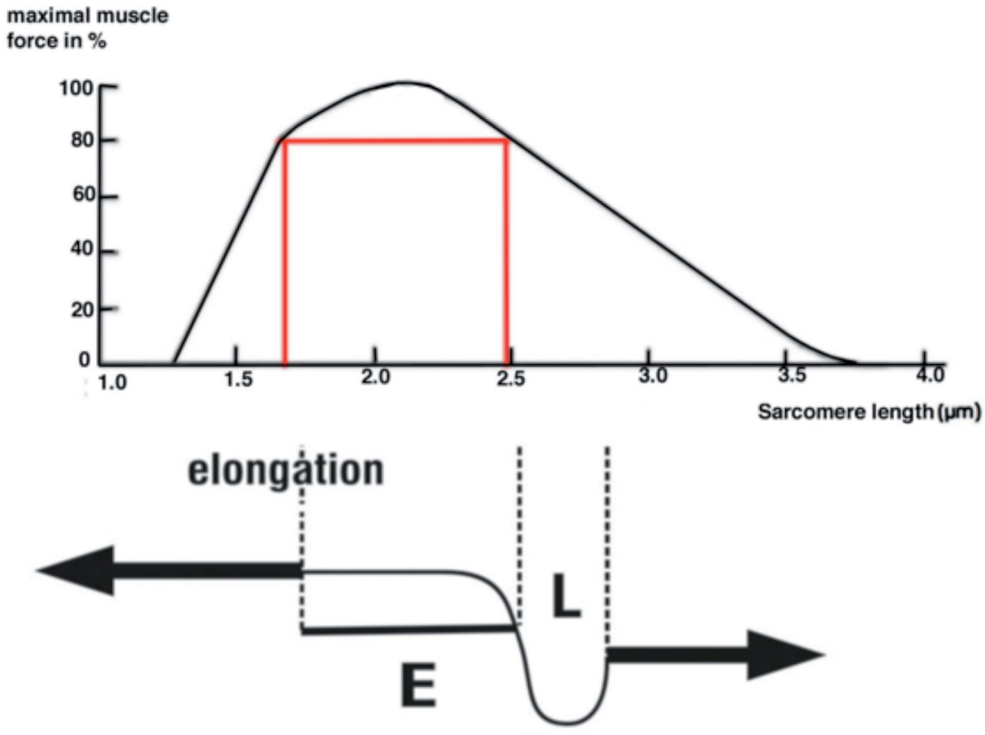
Hypothesized components of urethral closure. Perspective: coronal section of vagina at midurethra. The trigonal extension and vascular plexus are known as the "Cresta Urethralis"; arrows = vector closure forces (fast and slow twitch) from contraction of the anterior pubococcygeus muscle (PCM) which stretches the vagina upwards to close the urethral lumen'a'. Contraction of the rhabdosphincter over the "Cresta Urethralis" provides a water-tight mucosal seal, and is recorded as a pressure rise on effort. The vagina was dislocated from PCM surgically by a full thickness incision between vagina and PCM (broken lines). Note narrowing of space "a" by the transducer.

Figure 1. – Rhabdosphincter ("horse-shoe" striated muscle).



M. pubococcygeus (PCM) pulls the vagina forwards against the pubourethral ligament, "PUL" to close the distal urethra; LP/LMA stretch the proximal urethra against PUL to narrow it (small arrows; LP=levator plate; LMA=conjoint longitudinal muscle of the anus. This action occurs around the tip of the transducer which is effectively pushed forwards to further narrow the space "a" in Figure 1.

Figure 2. – Distal and proximal closure mechanisms of the urethra.[5]



Any laxity "L" in the muscle insertion points, whether rhabdosphincter or the three vectors in Figure 2, will effectively lengthen the striated muscles and result in a lower contractile force.

Figure 3. – Gordon's Law, a striated muscle contracts only over a fixed distance "E".

With reference to question 2, "role of intraurethral pressure in continence". With age especially, the collagenous attachments of vagina to PCM, PUL to urethra, rhabdosphincter to urethra and trigone, even the collagenous components of the vascular plexus,

Figure 1, all weaken; the lumen "a" expands and pressure falls, as Pressure=Force/Area. The rhabdosphincter also atrophies with age[3, 4] so that Force applied to "a" also weakens, causing a further pressure drop when measured. This hypothesis explains the onset of ISD, especially in the older age group. Though this hypothesis explains fall in urethral pressure with age, fall in urethral pressure is not per se causative of USI, as demonstrated by Kapoor et al.[8] It has been demonstrated that a high rate of cure can be achieved in patients with ISD with a midurethral sling.[9, 10] In two studies,[11, 12] a subgroup of patients with ISD (MUPs <20cm H2O) became 100% continent, *yet their post-operative MUPs remained <20cm H2O*. These data further support Kapoor et al.'s findings[11] that pressure per se is not an indicator of continence. Pressure, whether low, as in intrinsic sphincter deficiency (ISD), or high, as in our study, influences neither leakage nor continence. In contrast, it has been demonstrated by upward pressure at midurethra with a hemostat under transperineal ultrasound control that *an intact pubourethral ligament* (PUL) is the key factor for control of urinary stress incontinence and restoration of urethrovesical geometry.[6] (See video at www.integraltheory.org). Further validation comes from > 1,500,000 midurethral sling operations since 1995[13] for cure of USI. A lax PUL will result in effective lengthening of PCM (Figure 1). According to Gordon's Law,[14] this will weaken all three muscle closure forces (arrows Figure 3).

With reference to question 3, "role of the rhabdosphincter in closure and continence", it has been discussed in the answer to the question 2, that pressure per se is not an indicator of continence. Pressure merely measures force relative to the area over which it is measured. The role of the rhabdosphincter in continence control was examined thoroughly by Huisman[3] from a biomechanical/anatomical perspective. Huisman described how the rhabdosphincter (Figure 1) inserted inferiorly into the prolongation of the superficial trigone. He also described a cavernous type plexus submucosally in the cresta urethralis which was analogous to that in the corpora cavernosa of the male. In a series of histological studies extending from the new-born to old age, he was able to demonstrate that severe atrophy occurred in this muscle with time. He concluded that this was a weak muscle, and was not a major factor in continence control.

How to explain urine leakage with cough pressure increases of 112% and 170%?. Clearly, these measurements are misleading, as it is known that in urinary stress incontinence (USI), intraurethral pressure falls in relative terms to zero.

It has been demonstrated[5, 6] that on effort, backward/downward vectors stretch and rotate the proximal urethra around PUL (Figure 2). This action occurs around the tip of the transducer which in certain circumstances may "splint" the midurethra, effectively pushing the anteriorly oriented tip closely towards the anterior urethral wall to create a false reading, measuring the force transmitted by the rhabdosphincter over a greatly constricted area. The dislocated hammock could not stretch the vagina upwards to close the space lateral and posterior to the transducer. The urine ran out from around the three sides of the transducer.

We conclude:

1. The pressure readings in this and other studies can be potentially misleading in that they only measure the formula, Pressure=Force/Area.

2. The muscle force, in this instance, can only derive from the rhabdosphincter, which we have shown is a weak muscle incapable of closing the urethral tube.

3. Closure (continence) is different from pressure recorded, as demonstrated recently by Kapoor et al.[8]

4. We hypothesize the rhabdosphincter acts principally as an adjunctive sealing mechanism.

5. The musculofascial mechanism is the most important mechanism for urethral closure.

Limitations of the study. Only 4 patients were tested. However, according to Popper[15], this was sufficient to invalidate the "pressure hypothesis", one validated exception being sufficient to invalidate a hypothesis.

Note: Despite pressure rises >100% when the suburethral vagina was detached during midurethral sling surgery, 2/4 patients lost large amounts of urine. This indicates that musculoelastic closure is a most important mechanism for continence.

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Multidisciplinary Uro-Gyne-Procto Editorial Comment

To improve the integration among the three segments of the pelvic floor, some of the articles published in **Pelviperineology** are commented on by **Urologists**, **Gynecologists** and **Proctologists/Colo Rectal Surgeons** with their critical opinion and a teaching purpose. Differences, similarities and possible relationships between the data presented and what is known in the three fields of competence are stressed, or the absence of any analogy is indicated. The discussion is not a peer review, it concerns concepts, ideas, theories, not the methodology of the presentation.

Uro… In our continued quest for understanding the pathopysiology of female stress incontinence and how our operations may work, these authors, using the Integral theory, try to explain the relative contribution of the two most important factors in the female continence mechanism, namely the rhabdosphincter and anterior vaginal support. This paper in a way challenges the traditional belief of the understanding of ISD (intrinsic Sphincter Deficiency) which is defined by a low leak point of <60 cm H2O or MUCP of <20cmH20. They have shown, although in only a small patient cohort, that by restoring pubourethral ligament (PUL) function via a mid urethral sling (MUS), urethral closure and hence continence, can be obtained regardless of the leak point pressure or MUCP at which leakage takes place. This is reflected, and may explain, in clinical practice why the MUS can be so effective in restoring continence in most patients with either hypermobility-, or ISD-predominate incontinence. So far, literature is scarce on the anatomical explanations of these observations, other than that recent data do show the MUCP and LPP can be useful in selection of the surgical approach as the retropubic route maybe more effective than transobturator for lower LPP or MUCP.

This paper may also explain in those pts where the MUS fails (ie. after effective restoration of the PUL), that possibly the rhabdosphincter was more important IN THAT patient than the PUL, and hence a compressive procedure like bulking agent, pubovaginal sling, or artifical urinary sphincter would be more effective than a mid urethral sling.

Although the subject number is small and the results yet to be validated by others, this paper is an interesting read and certainly offers some food for thought with respect to the pathophysiology of female stress incontinence. It is by constant careful observation and critical thinking "outside the box" that science continue to advance.

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Gyne… Urinary continence is just one aspect within the vast complexity of human being functions. Nevertheless, even considering only female urinary continence, a full understanding is lacking. The article from Wagenlehner et al. clearly highlights the uncertainty of our knowledge. Quite surprisingly the smaller the field of observation, the greater the complexity we discover. In this light has the urinary continence mechanism to be considered, as a complex phenomena where many different factors interact. Urologist and colorectal surgeons have real sphinteric structures to take care of, gynaecologists may hardly consider the bulbocavernous (otherwise *constrictor vaginae*), or at a higher level the pubococcigeal muscles as sphincteric structures; but this is not the case, at least from a functional point of view, for both structures and for different reasons.

Therefore strictly from the gynecological corner of a multidisciplinary perspective I don't have much to comment on in the present paper. My only concern is on the static of the distal anterior vagina after "bilateral paraurethral incisions to dislocate the suburethral vaginal hammock from both pubococcygeus muscles". Further numbers and a long follow-up would be necessary to address this topic.

Incidentally I notice that the terminology adopted throughout the paper sometimes is adherent to the most recent ICS standardization (*Detrusor Overactivity*), sometimes not (*Genuine Stress Incontinence, Instability*); in a multidisciplinary perspective adherence to a standardized language is a major concern. That's why one can only partially agree with statement 5 in the conclusions: structural integrity of the musculofascial mechanism is just one mechanism of urethral closure, not necessarily the most important one. Throughout the paper, as well as commonly in debates around this subject, various biological components are considered: muscles, connective tissue, bones, vascularisation. No words about neurological components, but neuromuscular junction integrity plays for sure a role in urinary continence mechanism. Surprisingly this aspect is completely ignored in the clinical assessment of urinary stress incontinence. On the contrary anorectal function testing also include the assessment of *rectoanal inhibitory reflex* and *cough reflex*. Should one learn from the other? Anyway the paper mainly addresses the concept of urethral pressure measurements. The Authors criticize its role, and this is absolutely acceptable. But it has to be emphasized that "*measurements*" are the problem, not the pressure per se: at present methods of pressure measurements are misleading. The pressure generated within the urethra conceptually remains the synthesis of all the forces cooperating in continence. At present a non interference instrument to measure it is not available, but further research in this direction is welcome.

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Procto… This interesting article by Wagenlehner et al along with other published evidence lends further weight to the proposed Integral Theory by Petros, that tension in the pubourethral ligaments and their close relationship to the pubococcygeus muscle is the main mechanism by which urinary continence is achieved, and that the rhabdosphincter plays only a supportive role. Is it possible to take this further and make further hypotheses as to how faecal continence is maintained.

There is good evidence from manometric studies that continence is not directly related to external sphincter pressures. Perhaps the external sphincter is the rhabdosphincter of the anus, augmenting continence but not primarily responsible. The interrelationship of the puborectalis/pubococcygeus, levator plate and longitudinal muscle of the anus are a much more likely explanation. Simultaneous contraction of the levator plate pulling backwards; the puborectalis/pubococcygeus pulling forwards; and longitudinal muscle of the anus pulling downwards, acts to increase the anorectal angle and close the anorectal opening, deferring defecation and at the same time, as suggested in this paper inhibiting micturition. Anyone who has ever suppressed their flow of urine mid-micturition will be well aware of the simultaneous sensation of contraction and closure of their anus. The converse is also true. Perhaps time has come for colorectal surgeons to stop focussing on the external sphincter as the cause of incontinence and play closer attention to the integrated actions of the other muscles as outlined above. We should try to understand their anatomy, the possible role of loss of tension, and the various mechanisms that may contribute to such a loss. We might as a consequence, investigate the effect on faecal incontinence of repairing their respective ligamentous supports.

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