

Recto-enterocele repair: past problems and new horizons

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Abstract: Standard operations for treatment of recto-enterocele are based on incorrect anatomical assumptions from the past. The anatomy of prolapse and the principles of hernia repair are reviewed. A new concept of the dynamic bridging graft is proposed.

Key words: Recto-enterocele; Hernia principles; Site specific defects; Mesh repair; Dynamic bridging grafts.

Present methods for treating recto-enterocele are based on assumptions of 100 years ago. These assumptions are now known to be flawed. However, new anatomic and surgical insights into the pathogenesis of recto-enterocele have not flowed on to improved therapy. Standard operations for recto-enterocele, whether done as a posterior colporrhaphy or a Delorme's procedure, are still rooted in concepts of 20 years ago.

ERRONEOUS ASSUMPTIONS FROM THE PAST

The true basis of pelvic organ support was a complete mystery to 19th Century surgeons. The ambient belief in Victorian times was that pelvic organ support derived mainly from the stiffness of the vaginal walls, which were in turn thought to be held up by their insertion into the levator ani muscles and perineal body. Little distinction was made between prolapse of uterus, bladder or rectum. Constricting the genital hiatus and creating an obstructive shelf in the lower third of vagina was seen as a way to strengthen upper tract support (Fig. 1a & b).

However, the impression of improved uterine support after such surgery was completely erroneous. It arose because the still unsupported cervix and uterus often remained hidden within a voluminous pocket above the rigid perineal shelf, created by levatorplasty. Nonetheless, this entirely non-anatomic operation held sway until well after World War II.¹

Beginning in the 1960's, this very deforming operation of high transverse levatorplasty began to fall into disfavour, because of its high incidence of dyspareunia² and because it contributed little to enterocele repair. In a search for a less morbid technique, focus shifted to Denonvillier's fascia. This structure was first described in males, but was later recognized as having significant supportive value in women.³ Based on the assumption that rectocele arose because of fascial attenuation, surgeons began plicating the central portion of the rectovaginal septum, as a potentially less morbid strategy. This surgery has been done by both the transvaginal⁴ and transanal routes.⁵ Direct comparison of the trans-vaginal and trans-anal approaches through the medical literature is impossible, because gynaecologists operate primarily for bulge control and colorectal surgeons operate primarily for obstructive defaecation. We know from clinical experience that both methods of rectocele repair deliver reasonable symptom control, at least for a while. However, normal anatomy cannot be restored by either transvaginal or transanal plication.⁶ Both techniques have also been largely ineffective at restoring normal defaecation mechanics,⁷ and both still carry a risk of postoperative dyspareunia.⁶

Gynaecologists next turned to the concept of locating

Conflict of Interest Declaration: The author is supervising investigator for a global multicentre randomized clinical trial comparing 'suture-only' and SurgiSIS®-augmented vaginal paravaginal repair. Beyond this research funding from Cook Incorporated, I have no commercial or employment ties to any company.

and specifically correcting any specific tears in the rectovaginal septum, as had been pre-empted by Richardson.⁸ Several reports of so called "defect-specific" rectocele repairs appeared in the American literature, citing good bulge control, better functional outcomes and much reduced dyspareunia rates.^{7,9,10} However, the repair of these "defects" amount to nothing more than placing a finger of the non-dominant hand in the rectum at the time of surgery, and re-enforcing any area of perceived fascial weakness with isolated sutures. As explained below, this approach has failed to grasp the true nature of the "site-specific" defects that lead to recto-enterocele formation. Not surprisingly, later reports have shown these mechanically misguided attempts at "defect-specific" rectocele repair to be quite inefficient.¹¹

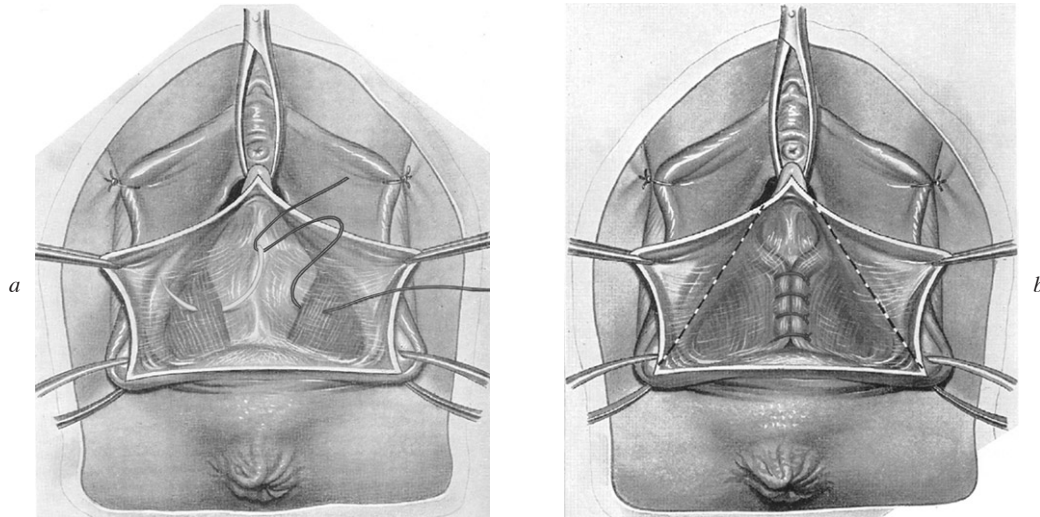
If we are to achieve optimal anatomic and functional results from rectocele repair, our surgical techniques must satisfy the principles of biomechanics. First and foremost, we must set aside the erroneous belief that rectocele arises because of fascial attenuation. In reality, endopelvic fascia is like canvas – it does not easily stretch, but will tear along lines of stress.^{12,13} Second, surgeons must understand the true nature of the fascial defects that cause recto-enterocele. The connective tissues of the postero-apical compartment form a thick and highly collagenized leash, running from sacrum to perineum (Fig. 2a).

This fascial aggregation plays an important support role, and has been designated the 'vaginal suspensory axis'.¹² Obstetric damage to the vaginal suspensory axis almost always occurs in the mid-pelvis, because expulsive forces increase exponentially as the presenting part tries to negotiate the "plane of least dimensions". If the endopelvic fascia is torn as the head tries to enter the mid-pelvis ("engagement"), the upper margin of the pericervical ring is likely to separate from the uterosacral ligaments – setting the stage for a 'cervix-first' prolapse. Conversely, if damage occurs as the presenting part is exiting the 'plane of least dimensions' ("rotation" and "extension"), the rectovaginal septum is likely to be shorn away from the inferior border of the pericervical ring (Fig. 2b).

The fetal head then pushes the detached rectovaginal septum downwards and outwards, much like a snow plough (Fig 3).

This injury creates a low pressure zone in the upper vagina, into which the pelvic contents can herniate, driven by sustained intra-abdominal pressure:

- Loss of the stiffening effect of an intact rectovaginal septum allows the rectal wall to bulge forwards. Anatomically, this creates the bulge of a rectocele (Fig. 4a).
- Functionally, stool wedges in the resulting "bowel pocket", thus disrupting the mechanics of defaecation (Fig. 4b).
- With careful dissection, the apical edge of this detachment can usually be seen in the lower vagina (Fig. 4c).
- Pre-peritoneal fat is invariably found above the line of septal avulsion – a sure sign of accompanying enterocele or sigmoidocele (Fig. 4d).



Figs. 1a & 1b. – Depict the reparative concepts of pre WWII surgeons, based on the belief that the uterus was basically “propped up” by the walls of a stiff vaginal tube. As shown in this diagram, modified from Wilfred Shaw’s textbook of 1935, prolapse repair in this era concentrated on constricting the genital hiatus and creating a rigid perineal shelf. The belief that this anatomically inappropriate technique was an effective means of supporting upper tract was entirely illusory. In reality, the prolapsing uterus and/or enterocele simply dangled ‘out of sight’, in an artificial pocket that formed above the perineal shelf. What was not illusory, however, is the severe dyspareunia that high transverse levator-plasty caused.

WHY HAS PROLAPSE REPAIR BEEN SO INHERENTLY DIFFICULT?

The key reason is that *pelvic connective tissues are NOT structurally suited to chronic load bearing*. Hence, Nature relies upon a complex inter-relationship between the muscles and the connective tissues (Fig. 5).

- Pelvic floor muscles act as a dynamic backstop, which absorbs most of the load.
- Endopelvic fascia is also important, but in a less direct way.

The role of the pelvic floor muscles

Skeletal muscle brings two unique advantages to the bio-mechanics of pelvic organ support: *durability* and *contractility*.

i) *Durability*: All biological fibres are susceptible to strain-

induced fatigue fracture, unless continuously remodelled in response to every day forces. Hence, even strong connective tissue would have difficulty in passively suspending the pelvic viscera in a species of bipeds that lives for 85+ yrs. These inherent bio-mechanical difficulties are brought to the fore by the combined insult of aging and prior childbirth injury. Both events disrupt the vital process of collagen homeostasis, and hence amplify the tendency of fascia (especially very weak fascia) to fail over time.

Stress-strain forces also create fibre fracture in skeletal muscle. However, in contrast to fascia, microtears in the

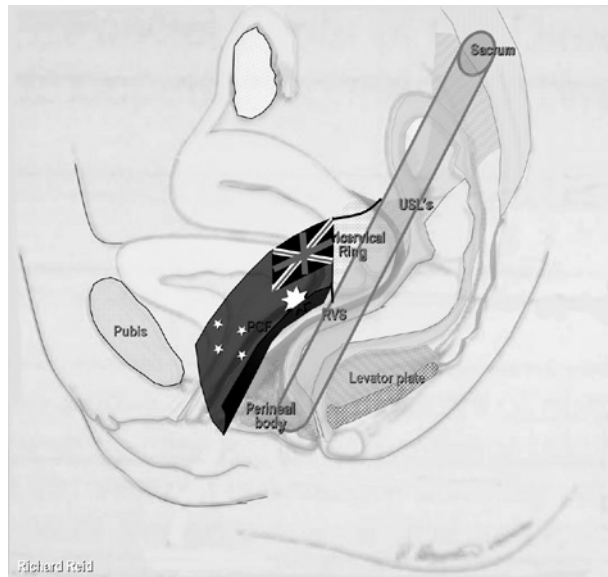


Fig. 2a. – A sagittal section of female pelvis, showing how the vaginal suspensory axis and the anterior vaginal hammock intersect like a flag, at half mast on a flagpole. Obstetric forces tear the fascia in the mid-pelvis (ie, where the “flag” joins the “flagpole”).

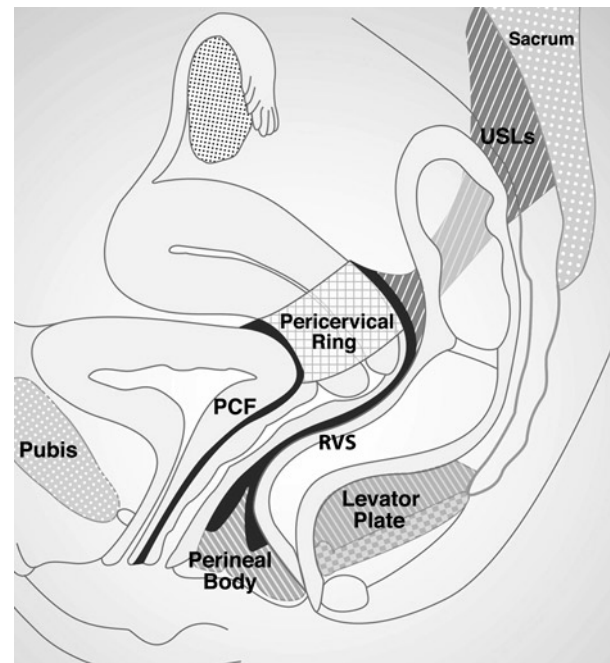


Fig. 2b. – The connective tissues of the postero-superior axis (“flag-pole”) form a continuous strong band that runs from the sacral periosteum, through the uterosacral ligaments, into the pericervical ring, and down through the rectovaginal septum, to insert into the perineal body. When this is intact, bowel motions are guided smoothly through the pelvis and easily out the anus. However, when it is torn, pelvic dragging discomfort and obstructive defecation become a problem.

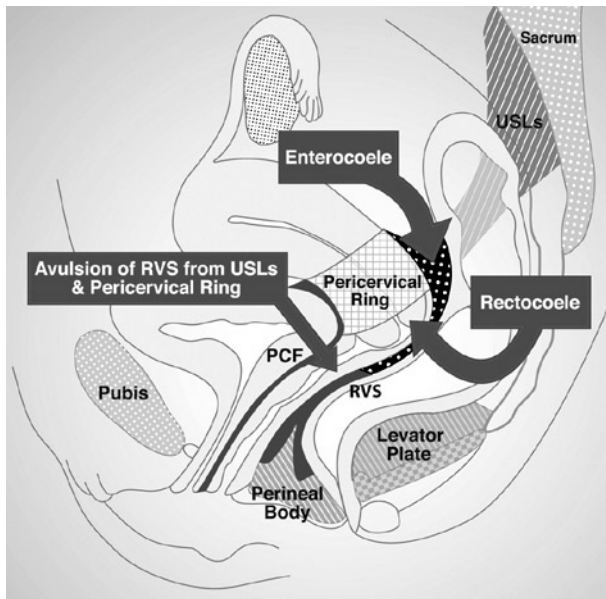


Fig. 2c. – This diagram shows the anatomic consequences of damage to the vaginal suspensory axis. Laceration of the uterosacral ligaments above the pericervical ring creates uterine descensus, while avulsion of the rectovaginal septum below the pericervical ring leads to herniation of ileum, sigmoid or rectum into the vaginal lumen.

muscle bundles induce compensatory hypertrophy, making the injured muscle stronger. However, the capacity for muscle hypertrophy is also diminished in older women by catabolism (age or illness related breakdown of the body's proteins)^{14, 15} and sarcopaenia (age-related acceleration of myocytes loss via apoptosis).¹⁶ Even so, muscle is a far more robust resource than fascia.

ii) *Contractility*: Being contractile, the pelvic floor muscles actively oppose intra-abdominal pressure, in two different but crucial ways.

– The *slow twitch fibres* maintain constant postural tone, thus narrowing the urogenital hiatus and elevating the levator plate into a convex, dome-like configuration. The former action directly opposes any tendency for the pelvic viscera to exteriorize, and the latter action creates a dynamic back-stop that acts as a flap valve to neutralize passive intra-abdominal forces.

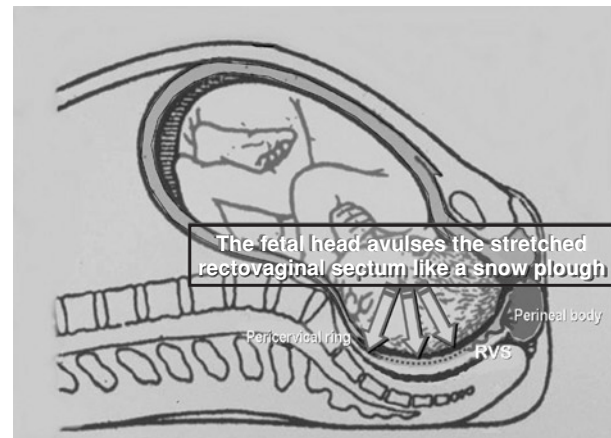


Fig. 3. – Is a diagram from William's Textbook on Obstetrics, showing how the primary fascial injury occurs as the fetal head passes through the mid pelvis. In other words, endopelvic fascia is like canvas - it does not stretch, but will tear at points of extreme stress.

– The *fast twitch fibres* provide rapid reflex contractions which equalise the sudden violent pressure waves generated by coughing or straining, thus preventing Valsalva forces from overwhelming urethral and anal closure pressures.¹⁷⁻²⁰

In other words, the physiologic role of the pelvic floor muscles in pelvic organ support is important and irreplaceable.

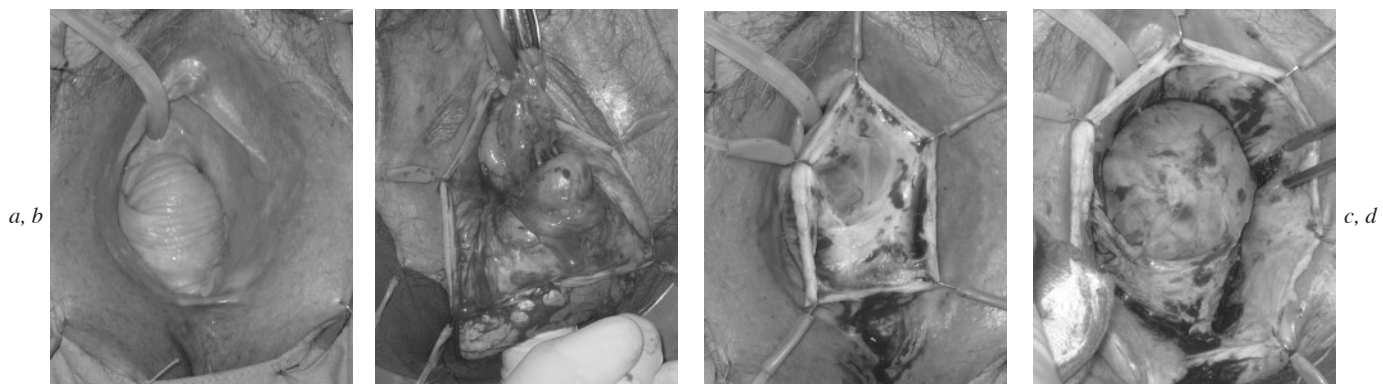
The role of the endopelvic fascia

The endopelvic fascia functions more as an investing mesentery, than as a direct visceral suspensory system (such as depicted by Fig. 5d).

In this role, it attaches the pelvic organs to the axial skeleton, and thus stabilizes them over the centre of the levator plate. The endopelvic fascia has considerable mechanical strength, and can resist short term expulsive forces. However, any fascial suspension is prone to fail under sustained load, especially if ravaged by age and childbirth damage.

The pathogenesis of prolapse

Pregnancy itself softens the pelvis connective tissue, thus potentially weakening apical supports. However, the key event in recto-enterocele formation is vaginal delivery, which can cause several complementary patterns of support failure.



Figs. 4. – Shows the clinical effects of a recto-enterocele avulsing the rectovaginal septum below the pericervical ring. 4a: The typical posterior bulge, caused by "partition failure" in the postero-apical compartment. Whether the rectocoele or the enterocele component assumes greater relative prominence is largely a matter of random variation. 4b: An assistant's finger in the rectum, highlighting the rectocoele component of the bulge. A "bowel pocket" (causing symptoms of obstructive defaecation) is well demonstrated. 4c: A careful dissection on the cranial side of the rectovaginal septum, using Lone Star retractor hooks to aid the dissection. A line of avulsion can be seen between the dense white fascia of the rectovaginal septum (below) and the yellow pre-peritoneal fat of the cul de sac (upper areas). 4d: On completing the dissection shown in the previous photo, the cul de sac can be seen to be projecting downwards and forwards as a large enterocele. The futility of "repairing" this bulge by plicating this pre-peritoneal fat is obvious.

Manoeuvres with a surgeon's glove

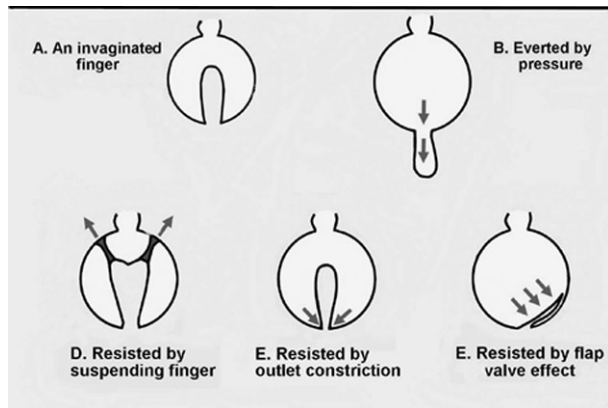


Fig. 5. – The pelvic organ support mechanisms used by Nature are similar to the maneuvers that one would use to prevent an invaginated finger of a surgical glove (A) from being everted under pressure (B). To prevent such eversion, the finger apex could be suspended to the top of the glove by connective tissue (C), the base of the finger could be constricted by postural tone from the pelvic floor muscles (D) or Valsalva forces could be dissipated against the ‘flap valve’ mechanism of an intact levator plate (E).

– Perhaps the prime insult is a direct avulsion of the pelvic diaphragm from its origin on the levator tendon. Such injuries are seen in about 20% of parous women, and are powerful risk factor for the subsequent of prolapse and incontinence.²¹⁻²³

– Elongation of the nerve to levator ani during descent of the fetal head can also cause a stretch neuropathy and eventual muscle fibre atrophy.²⁴⁻²⁷

– Finally, the endopelvic connective tissue is generally torn by the same obstetric events, creating a group of “site-specific” fascial defects.^{12, 28-30}

Avulsive and stretch neuropathy injuries to the pelvic diaphragm result in a sagging concave levator plate and a widened urogenital hiatus (Fig. 6a & b).

Valsalva pressures are now deflected downwards and outwards, creating a sliding stress on the pelvic viscera. If the fascial mesentery is also torn, the pelvic viscera align over this widened genital hiatus, and are thus susceptible to descensus (Fig. 6c).

Childbirth is “an essential but not sufficient factor” in the pathogenesis of prolapse. However, these myofascial injuries are generally compensated for years by the strong connective tissues of young women. Whether or not this series of adverse mechanical events ever results in overt prolapse

depends upon the operation of secondary factors, such as nutritional deficiency, repeated abdominal straining, central obesity and the acquired collagen weakness that inevitably develops in a torn anterior or posterior suspensory hammock.

THE HERNIA ANALOGY

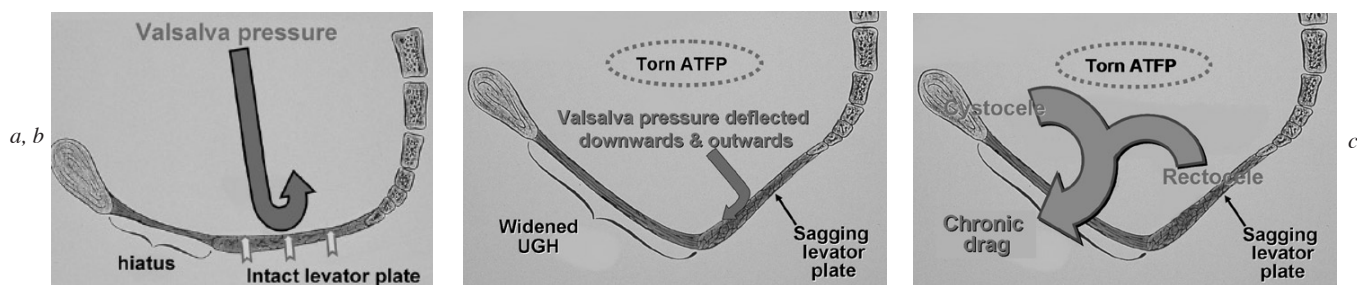
Weakened connective tissue adjacent to the ‘site-specific’ tears has been identified as an important failure mechanism by hernia surgeons. This same phenomenon is probably just as relevant to prolapse repair.

Gynaecologists are beginning to articulate that prolapse is a form of hernia. Let us explore the implications of that assertion in a little more detail.

Hernia is the protrusion of an internal organ (usually small bowel) through the muscular wall of the body cavity, generally occurring at a site of congenital weakness. The pathogenesis of hernia has two components.

– A *mechanical event*: namely, a ‘site-specific’ tear in the transversalis fascia at either the groin (inguinal hernia) or the anterior abdominal wall (incisional hernia).

– A *metabolic event*: namely, secondary (acquired) degenerative weakness in the connective tissue of the anterior abdominal wall adjacent to the initial tear.³¹⁻³² This phenomenon is particularly evident in treatment failure patterns for incisional hernia. Firstly, the unsatisfactory results of Mayo duplicative suture repair for incisional hernia have been repeatedly documented, as in a National survey of German hernia surgeons.³³ Analysis has not identified any consistent technique factors that predispose to failure. Secondly, a retrospective, population-based cohort study from a Washington State hospital discharge database (1987-99) demonstrated that the 5-year re-operative rate was 23.8% after the first re-operation, 35.3% after the second, and 38.7% after the third failure. In response to these tissue weakness factors, the use of synthetic mesh in incisional hernia repairs increased from 34.2% in 1987 to 65.5% in 1999. Controlling for age, sex, co-morbidity index, year of the initial procedure, and hospital descriptors, the principal hazard in this population-based cohort study proved to be the use or non-use of a tissue augmentation material (recurrence being 24.1% higher in the ‘suture-only’ repairs).³⁴ Thirdly, a multicenter RCT comparing suture versus mesh hernioplasty in 200 patients showed the three-year cumulative recurrence rates to be 80% higher if mesh was not used (43% vs 24%; $p = 0.02$). Risk factors for recurrence were suture repair, infection, prostatism (in men), and previous surgery for abdominal aortic aneurysm (another disorder known to reflect collagen weakness).³⁵ Similar reductions



Figs. 6. – A diagram showing the dynamic interaction between the pelvic floor muscles and the endopelvic fascia. 6a: The slow twitch fibres maintain constant postural tone, thus narrowing the urogenital hiatus and elevating the levator plate into a convex, dome-like configuration. The former action directly opposes any tendency for the pelvic viscera to exteriorize, and the latter action creates a dynamic backstop that acts as a flap valve to neutralize passive intra-abdominal forces. 6b: Loss of postural tone in the pubococcygeus and puborectalis section of levator ani allows the introitus to gape, while loss of postural tone in the iliococcygeus portion would allow the levator plate to sag (thus destroying the flap valve effect). 6c: When levator ani weakness is combined with fascial defects, the pelvic organs tend to move forwards and sit over a widened genital hiatus. As such, the combination of muscular and fascial injury has a synergistic effect in predisposing to pelvic organ descensus.

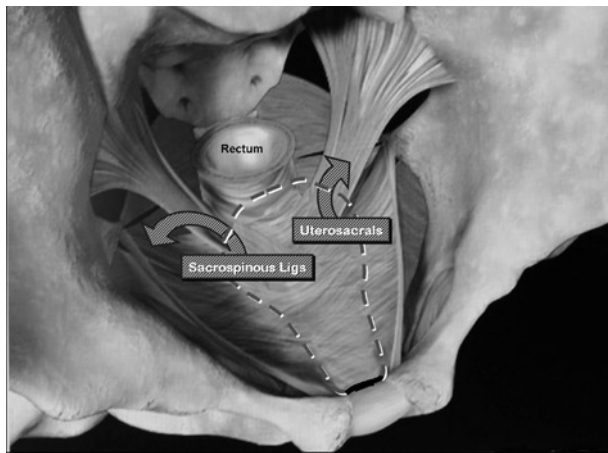


Fig. 7. – Fascia of postero-apical plane, seen in the coronal plane. It takes origin from uterosacral ligaments (centrally) and lower edge of sacrospinous ligaments (laterally), then running down the arcus tendineus fascia pelvis (upper half of vagina), before dipping backwards on the arcus tendineus fascia rectovaginalis, to insert into perineal body.

in inguinal hernias have also been documented in a prospective Denmark-wide study.³⁶ Finally, the late appearance of incisional hernias several years after laparotomy and the high recurrence rates after ‘suture-only’ repair (irrespective of surgeon or technique) point to the importance of disordered collagen metabolism in the pathogenesis of both primary and recurrent incisional hernias.³⁷ This view is supported by the demonstration of a reduced proportion of high tensile strength (type I) collagen and an increased amount of immature (type III) collagen in hernial sacs.³⁸ Acquired degeneration in collagen quality probably occurs because the ‘site-specific’ defects in the investing fascia disrupt continuous tissue remodelling, a process that is driven by the transmission of everyday mechanical stress. Such collagen homeostasis is affected by the balance between growth factors and tissue collagenase levels (mainly matrix metalloproteinases-1 and -13).^{39,40} There is a suggestion of disordered MMP-1 and MMP-13 activity in both skin and scars from hernia patients, but evidence to this point has been inconsistent.⁴¹

Likewise, prolapse is the protrusion of an organ (uterus, bladder or bowel) through the vaginal fibromuscularis, usually at a site of childbirth injury. It also has mechanical and metabolic components.

– The mechanical event is a group of ‘site-specific’ tears in the endopelvic fascia, as discussed above.

The high prolapse incidence and treatment failure rates in patients with inherited collagen disorders like Ehlers Danlos or benign joint hypermobility syndromes is well known.^{42,43} However, it is also likely that biochemically normal prolapse patients acquire a metabolic collagen weakness in the endopelvic fascia when daily transmission of mechanical forces to the torn suspensory hammocks is disrupted. The argument that tissue weakness is also an important factor in the aetiology of prolapse mirrors that of herniologists. The risk of operative failure rises with each successive reparative attempt, even though subsequent procedures are usually done at tertiary referral centers.⁴⁴ Moreover, in a cohort of women with pelvic floor disorders who were followed prospectively for 5 years, a history of prior pelvic prolapse and urinary incontinence surgery was actually a marker for a 42% increase in the likelihood of that patient coming to re-operation.⁴⁵ Such failures do not reflect tissue thinning in prolapse women – in fact, the vaginal muscularis layer in enterocele has been shown to be thicker than normal.⁴⁶

Given that the mechanical of the vaginal wall is likely to reflect composition, thickness and tissue architecture, it is noteworthy that Boreham et al have shown a reduced proportion of physiological smooth muscle and an increased proportion of disorganized smooth muscle bundles with decreased α -actin staining.^{47,48} Prolapse tissue biopsies have been shown to have a decreased collagen concentration,⁴⁹ lower collagen I: III ratios, and up to four times higher levels of lytic protease enzymes (as indicated by MMP activity).⁵⁰⁻⁵²

General surgeons have been able to reduce the failure rate for inguinal hernia from about 35% to $\leq 2\%$. The main vehicle of this success has been adherence to a group of rules called the ‘Hernia Principles’. Logic would suggest that the same approach may help gynaecologists to improve their prolapse repair outcomes.

THE HERNIA PRINCIPLES

Over the course of a couple of centuries, surgeons developed a group of cardinal operative rules to reduce hernia recurrence.⁵³

These principles are: 1) Avoid wound infection, 2) Protect repair from intra-abdominal pressure, 3) Repair tears in investing fascia, 4) Re-anchor the torn investing fascia back onto skeleton.

The aim is to repair all ‘site-specific’ fascial defects, using permanent suture, and with no tension *in any direction*. However, mobilising the retracted vaginal hammocks back to the mid-pelvis, so many years after childbirth, does inevitably produce a degree of suture line tension.

To these traditional rules, modern surgeons have added the proviso that the most effective way to avoid tension in hernia repair is through the use of mesh. There are two main rules for the prudent use of mesh in hernia surgery:

- The type of mesh (eg weight, stiffness, Amid class⁵⁴) must be appropriate to the intended implantation site
- Surgeons must distinguish between suspensory and bridging functions.

These theoretic principles also fit recto-enterocele repair – but we cannot directly extrapolate the choice of materials, from hernia to prolapse. The vagina is not the abdomen. Specifically:

– *In the groin*, mesh is implanted through a sterile environment, between two tough & highly collagenized aponeurotic layers, where it lies 5-10 cm deep to body surface. There is minimal tissue-on-tissue movement, and the mesh is well separated from intra-abdominal hollow viscera.

– *In the vagina*, mesh is implanted through a contaminated environment, between a basement membrane and a fragile layer of smooth muscle, just 1/2 cm deep to vaginal mucosa. This is an area of maximal tissue-on-tissue movement. Finally, the implantation site is immediately adjacent to the bladder, ileum and rectum.

Rules have been devised for the tension free placement of mesh:

- 1) Mesh must suit surgical site, 2) Isolate mesh from contact with a hollow viscus, 3) Limit bacterial colonisation of the mesh, 4) Choice of mesh must suit surgical objectives, 5) Mesh implant must overlap the defect on all sides, 6) Stabilize against doubling, wrinkling & undue shrinkage, 7) Mesh must be placed in a tension-free manner.

ADAPTING HERNIA RULES TO RECTO-ENTEROCOELE REPAIR

Rectocele, enterocele and vault inversion share a common origin - namely, childbirth damage to the endopelvic fascia. Such injuries often occur concomitantly.^{11,12} Gynecologists

have traditionally regarded these three conditions as discrete entities. However, support failure within the anterior and postero-apical compartments are highly correlated.^{55,56} Typically, a patient will present with overt support failure in one segment and incipient weakness in adjacent sites. Paradoxically, despite marked differences in their clinical prominence, both dominant and incipient support defects are of almost equal importance to the reconstructive gynaecologist. That is to say, the fascial supports at the secondary sites may well be strong enough to maintain the *status quo*, but they are often too damaged to resist the new force vectors created when an adjacent vaginal segment is re-suspended. Leaving an area of incipient weakness unrepaired in such circumstances sews the seeds of early failure – often within 6 months or so. In the words of Wayne Baden,³⁰ the prudent surgeon will always “leave the entire tract intact”, or face an unacceptable risk of early postoperative bladder, vault or rectal prolapse.

From a pragmatic perspective, pelvic visceral mesenteries resolve into two semi-independent systems – the anterior and postero-apical compartments. These two systems intersect like a flag and flagpole (Fig. 2a). The *anterior hammock* is vital to urinary continence, but has no major supportive role for the vagina as a whole. Conversely, the *vaginal suspensory axis* both suspends the vaginal apex and partitions the vagina from the cul de sac and rectum. When intact, this vaginal suspensory axis forms a membrane that guides faeces efficiently through the pelvis and out the anus. The proximate cause of recto-enterocele is a ‘site specific tear’ in the vaginal suspensory axis – creating *suspensory failure* if the injury occurs above the pericervical ring and *partition failure* if damage occurs more distally (Fig. 2b).

Effective repair of postero-apical compartment prolapse requires that fascial integrity be restored in two different planes.

– In the *sagittal plane*, fascial continuity must be restored from the sacral periosteum, through the uterosacral ligaments, into the pericervical ring, down the rectovaginal septum and into the perineal body. Historically, this has been most effectively done by threading a narrow ribbon of polypropylene from the sacral promontory to the rectovaginal space (abdominal sacrocolpopexy). However, transvaginal placement of a remodeling biomesh has the potential to deliver even better performance than abdominal sacrocolpopexy, by a cheaper and less invasive technique.

– In the *coronal plane*, restoration of normal anatomy requires that fascial continuity be established from the ischial spines and lower margin of sacrospinous ligament, down the two fascial white lines,⁵⁷ to the distally retracted edge of the rectovaginal septum (Fig. 7). Such a repair in the coronal plane cannot be done from above, but is readily accomplished from below.⁵⁸

Such a repair can be done by re-suturing native tissues. However, given that damaged endopelvic connective tissues undergo a slow but relentless deterioration in collagen quality, use of an appropriate tissue augmentation material is more in accordance with modern hernia principles. From a biomechanical perspective, mesh re-enforcement must satisfy two goals:

– *Re-attachment of the vagina onto the uterosacral ligaments (and hence the axial skeleton)*: Mesh used for this task must act as a ‘*suspensory strut*’, for which tensile strength is the dominant consideration. Polypropylene is the strongest available material, but it creates a foreign body reaction and dense avascular scarring.⁵⁹ Such inflammatory, non-lubricated fibrosis can be morbid. However, suspensory struts are usually located in static sites (where there is little movement of one tissue on another). Hence, polypropylene

has generally been well tolerated, when used as a mid-urethral sling or for sacrocolpopexy. Whether SIS offers any advantage over polypropylene for these operations is presently being debated. It is likely that the wisest choice depends on other patient factors.

– *Closure of any low pressure zone within the postero-apical compartment*: This needs a bridging graft, not a strut. The graft material must be strong but not excessively so. Mesh used for this task must act as a ‘*bridging graft*’, for which tissue flexibility and a low risk of erosion or pain is more important than extreme tensile strength. In my judgement, polypropylene is a poor choice in this situation. Conversely, SIS performs very well as a bridging graft in almost all patients.^{60,61}

A SMALL CLINICAL SERIES

Effective mesh correction of the posterior defect in two planes requires a roughly diamond-shaped graft. Key points in ensuring a *safe and effective operative technique* were:

– Routine use of the Lone Star Retractor[®], to optimize exposure and to create traction / counter-traction throughout the wound. Effective sharp dissection depends heavily on the use of this invaluable surgical tool.

– Pararectal spaces were entered via an essentially bloodless embryologic cleavage plane between the endopelvic and parietal fasciae, allowing easy passage to each sacrospinous ligament.

– A combined bridging and suspensory graft of SurgiSIS[®] biomesh was secured to the extraperitoneal portions of the uterosacral ligaments (antero-medially) and to the sacrospinous ligaments (postero-laterally). This implant was shaped, somewhat like a “gingerbread-man” cookie (but with very long “arms” and a short “body”) – thus suspending the vagina within the mid-pelvic axis.

In electing to use primarily biological implants, there is one important point must be made about the choice of materials. In the early 1990’s, manufacturers “leatherized” various cadaveric and animal grafts, in the hope getting an equally permanent but “more natural” scaffold. Outcome proved to be paradoxical. Although first generation biomesh is strong *in vitro*, reports soon surfaced of an unduly high repair failure rate when Pelvicol[®], etc was used *in vivo*.⁶² Re-operation often showed no residual graft material. With the wisdom of hindsight, the reason for this phenomenon is obvious. *In vivo*, any denatured collagen – whether of endogenous or exogenous origin – is seen by the host’s immune system as “dead tissue”, and hence subjected to an intense biodegradation reaction (ie, encapsulation and enzymatic autolysis). In addition, Pelvicol[®] provokes a strong foreign body reaction, meaning that the resulting wound can be just as hard and just as stiff as with synthetic mesh. Thus, first generation biologicals with cross-linked collagen are poorly suited for use as a bridging graft, as illustrated a recent rectocele repair series showing a 41% failure rate at 3 years.⁶³

My experience in a pilot study using SurgiSIS[®] as a bridging graft has been most encouraging.⁶⁴⁻⁶⁶ At one year follow-up, 46 of 49 patients had outright or qualified anatomic success. There were statistically significant reductions in all pre-operative symptoms, including bulge, drag and defaecatory difficulties. Intraoperative complications were minimal, and no graft-related morbidity or dyspareunia has been seen.⁶⁶

IN SUMMARY

Prolapse repair is associated with stretch dilatation of the anterior rectal wall. However, this is a *secondary* event. The *primary* cause is a combination of pelvic muscle avulsive

and denervation injury, together with various 'site-specific' lacerations of the suspensory hammocks. To be curative, any operation for recto-enterocele must repair the sites of fascial tearing (rather than just plicating the non-specific dilatation of the rectal muscularis).

Surgical options for prolapse repair place more reliance on the endopelvic fascia than occurs in Nature. Attaining a durable repair under these circumstances is a biomechanically difficult task – even in young women with strong, anabolic tissues. However, this inherently difficult task is much harder by secondary collagen degeneration within adjacent connective tissues. Hernia surgeons have faced and overcome similar obstacles, and it is likely that some of these surgical principles are relevant to gynaecology.

In using tissue augmentation materials, surgeons need to distinguish static struts (where tensile strength is the dominant issue) from *dynamic bridging grafts* (where tissue flexibility and low morbidity are the main considerations). SurgiSIS® is an ideal bridging graft, under almost all circumstances. If properly sized and shaped, it can also perform well as a suspensory strut. However, there are clinical circumstances where the addition of polypropylene is prudent.

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