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Monographic issue: Chronic Pelvic Pain

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A multidisciplinary pelvic floor journal

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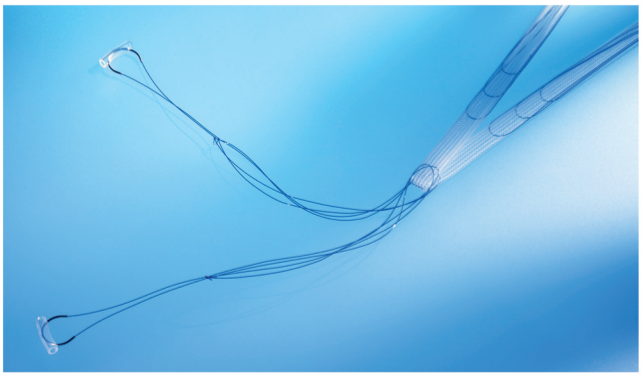

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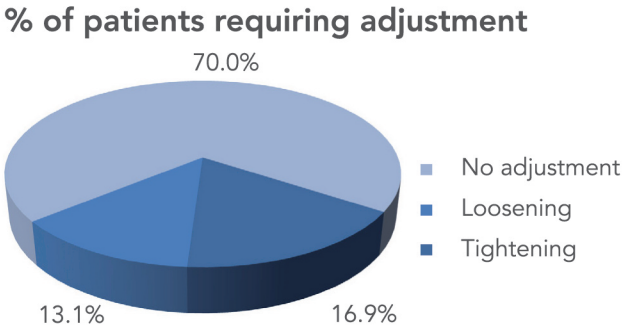

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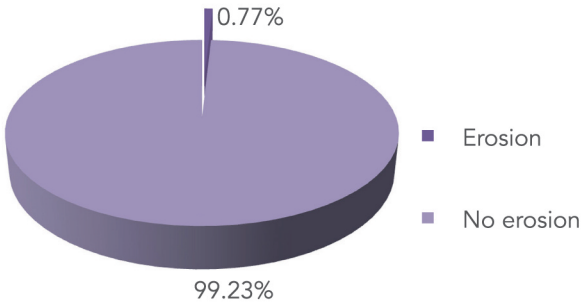

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Original article

Role of uterosacral ligaments in the causation and cure of chronic pelvic pain syndrome

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Abstract: Aim: To critically analyse the role of apical support and suspension in order to understand the genesis of chronic pelvic pain syndrome (CPPS).
Method: Based on historical and recent scientific publications 5 possible reasons for lumbosacral pains are extracted. All directly or indirectly relate to uterosacral ligament (USL) support: 1) spastic, colicky uterosacral contractions, 2) irritation of Ganglion Frankenhäuser, 3) unphysiological tension on the plexus sacralis due to deficient suspending ligaments or support from pelvic floor, 4) overstretching of uterosacral ligaments (USL), 5) disturbance of blood circulation. Two neurological pathways of pain transmission are examined to explain cure of CPPS by reinforcing USL support. An anatomically based analysis was made of existing operations which restore apical anatomy.
Results: It was concluded: 1. That damaged USLs are mainly responsible for prolapse induced pain. 2. CPPS is almost variously associated with urgency, nocturia and "obstructive: urination and defecation, an important factor in the differential diagnosis from causes such as endometriosis. 3. Because of the complex interaction of muscles, ligaments and extremely sensitive nerves, any reconstruction should aim as much as possible to mimic the natural anatomy if good symptomatic cure rates are to be achieved. 4. Comparative analysis as follows.
* *Simple approximation of USLs* initially cures CPPS but does not last in the longer term. * *Abdominal sacrocolpopexy* does not mimic normal anatomy. Promontorial fixation creates an unphysiological vertical vaginal axis which may result in high recurrence of prolapse and pain. * *Traditional level 2 vaginal operations* are also insufficient, because it is difficult for re-sutured weakened tissues to achieve the tensile strength of vaginal wall needed for symptom cure. * *Posterior sling operations* provide lasting success using artificial tapes which create strong artifical neoligaments, exactly as per the TVT tension free vaginal tape.
Conclusion: Symptoms, especially pain, are the sentinels of pelvic floor dysfunction and they are invariably associated with uterine prolapse, often minimal. Symptoms cannot validly be ignored by any expert body issuing recommendations on prolapse management and results. Failure to cure pre-existing symptoms equals failure of that type of surgery. The presence of urgency, nocturia, "obstructive" micturition or defecation strongly indicates that the cause of the CPPS is loose uterosacral ligaments. For good longer term results, a polypropylene tape precisely inserted to support USL is required.

Keywords: Chronic pelvic pain syndrome; Uterosacral ligaments; Cardinal ligaments; Integral theory; Pelvic congestion; Pelvic organ support.

INTRODUCTION

In the female organism, the pelvis is an especially vulnerable site for major, often disabling pathology, in particular, pain, bladder and bowel disorders. Dysmenorrhea, uterine fibroids, cycle disorders, immovable retroflexed uterus, endometriosis, inflammation of ovaries or fallopian tubes, ovarian tumour, vaginal or uterine prolapse, are all implicated in the causation of chronic back pain. These pains are characterized as low dragging abdominal pain or low sacral backache.

However, most chronic pelvic pain syndrome (CPPS) conditions are deemed to be of unknown origin, classified as a "neurological" or in the German literature as "Pelvipathia vegetativa", "Parametropathia spastica", "Spasmophilia genitalis", "Plexalgia hypogastrica", "pelvic neuralgia" or cervical syndrome".[1]

Pelvic pain can be caused by disturbance of blood flow in the small pelvis, for example in form of functional hyperemia during menstruation or due to inflammation. In the literature, venous congestion caused by varicosis of pelvic veins, "Pelvic congestion syndrome", has been well known for many years.

Since some decades, Heinrich Martius published in the German literature that in about 30% of cases, backaches are provoked by damaged suspending or supporting ligaments of the pelvic organs.[2] The paired "Ligamenta recto-uterina", which are connected via paraproctium to the bony sacrum und therefore in general are termed "plica or ligamenta sacro-uterina" or "uterosacral ligaments" (USL), are placed in the centre of numerous pathophysiological considerations. Unfortunately, Martius's concepts have remained largely unknown in the English literature. In 1993, Petros and Ulmsten independently described CPPS as being caused by lax uterosacral ligaments as part of the "Posterior Fornix Syndrome",[3] along with other pelvic symptoms, nocturia, urgency, abnormal emptying. They reported a significant cure rate of CPPS and other posterior fornix symptoms following repair of the uterosacral ligaments.[3] Petros wrote a classic description of this pain in 1996.[4]

"In its acute state of manifestation, the pain was invariably severe, frequently one-sided, situated low in the right or left iliac fossa, usually relieved on lying down, frequently relieved by insertion of a ring pessary, reproducible on palpating the cervix and displacing it posteriorly, patient in supine position. Although the pain was chronic in nature, itvaried considerably from time to time as concerns intensity. There was a history of deep dyspareunia which only occurred on deep penetration, or in specific positions. Frequently the patient complained of a constant lower abdominal pain the day after intercourse. Half the patients complained of low sacral backache which was also cured by the surgery. Six patients, 2 of whom were nulliparous, entered the study through Emergency".

In 2008, Abendstein et al. expanded the Posterior Fornix Syndrome with their report of cure of Obstructive Defecation Syndrome (ODS), severe sacral and abdominal CPPS and non-sphincteric fecal incontinence with a posterior sling.[5] These works led to a diagnostic algorithm which immediately separates uterosacral induced CPPS from other types such as endometriosis, the key differential being that invariably other posterior zone symptoms accompany the CPPS symptoms (Figure 1).

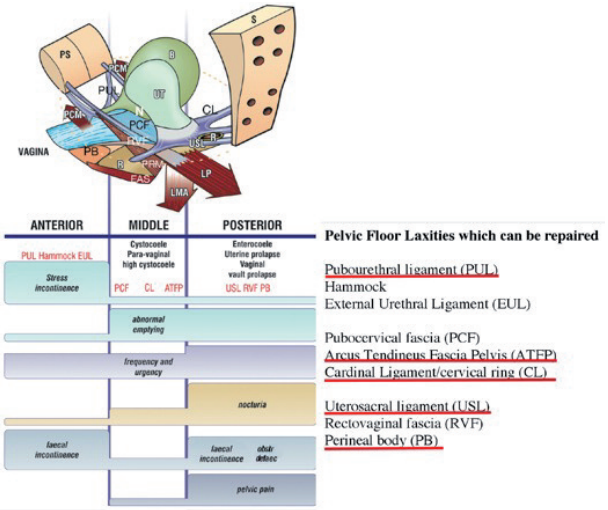

Figure 1. – Diagnostic algorithm. The posterior vector forces levator plate "LP" and conjoint longitudinal muscle of the anus "LMA" (arrows) selectively contract against the cardinal "CL" and uterosacral ligament "USL" during urethral and anorectal closure to stretch PCF to support the bladder base stretch receptors "N and anorectal receptors (not shown), thereby controlling urge symptom afferents. Loose ligaments will weaken the muscle forces giving rise to the symptoms and prolapses indicated. The height of the bar indicates probability of occurrence of a particular symptom and therefore its relationship to a specific ligament*. The underlined structures indicate the ligaments which can be surgically reinforced using polypropylene tapes. Diagnosis of which ligament to repair is indicated by the algorithm's symptoms and confirmed by systematically assessing the damage of 3 structures in each zone of vagina (Chapter 3 ref. 19).
* for example stress incontinence is caused by a damaged PUL, nocturia and pain by USL etc.

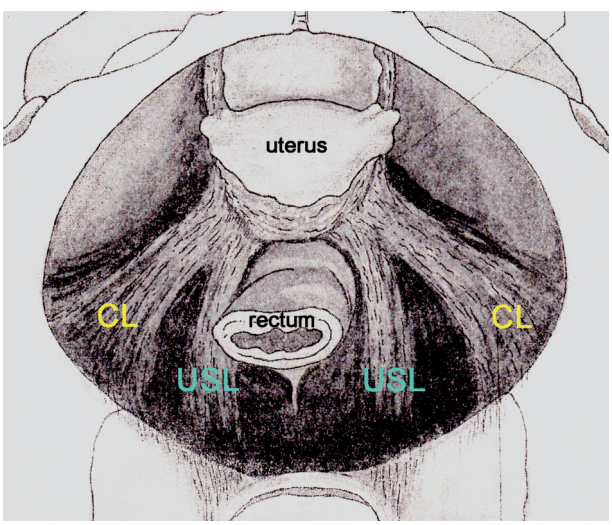

Figure 2. – Parametrium according to Martius (2). The Figure demonstrates that the parametric tissue fibres are mainly running backwards to the the iliosacral region connecting and suspending the cervix to the posterior pelvis. USL = uterosacral ligaments, CL = cardinal ligament.

Up to now the discussion about the significance of the supporting and suspending system of the pelvic organs is still largely unknown in the English literature. Expert committees such from the International Continence Society (ICS) and the European Urology Association do not refer to USL laxity as a major cause of CPPS.[6,7] Neither does a recently published review article on CPPS mention deficient posterior pelvic organ ligaments as a cause.[8] Important publications in 1993, 1996, 2001, 2010 and 2012 which concentrate intensively on this focus continue to be ignored.[3,4,9-11]

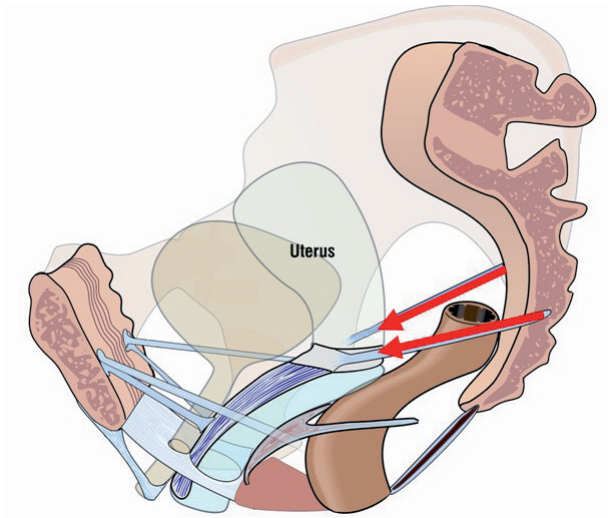

Figure 3. – The USL (red arrows) arises from the sacral vertebrate S2-4 and attaches to the cervical ring posteriorly.

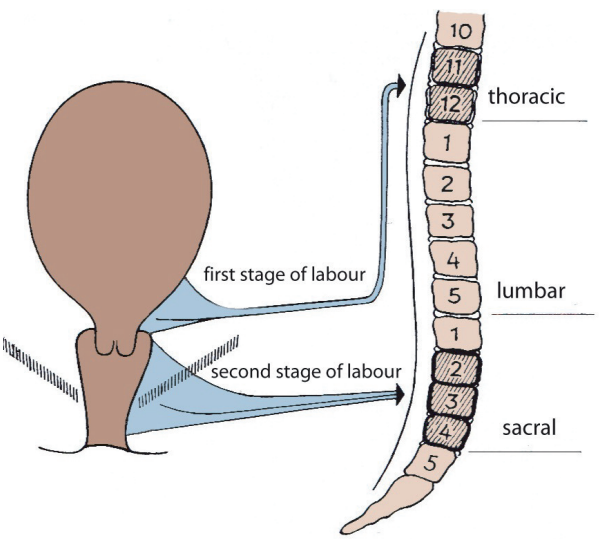

Figure 4. – Schematic diagram of visceral uterine innervation.

The aim of the present review is to asses the evidence for:
1. The significance of the posterior pelvic supporting and suspending system for chronic pelvic pain induction.
2. The therapeutical possibilities to cure these symptoms.

Anatomy of posterior suspension system

One of the first reports about the uterusacral ligament is from Philip Verheyn in the year 1708.[12] Under the title "uteri connexio", Verheyn pointed out that the neck of the uterus is connected at the bottom to the vagina, posteriorly to the rectum and anteriorly to the bladder.

In 1862 the anatomist Hyrtl from Vienna specifically emphasized in his textbook "Anatomie" that apart from the ligamenta rotunda there are restiform peritoneal plicates reaching from the bladder to the uterus, called "Ligamenta vesico-uterina" as well as from the rectum to the uterus, called "Ligamenta recto-uterina". These ligaments contain connective tissue fibres of considerable strength and are therefore able to lock the uterus in place.[13] Long-standing works explain the importance of USL for anorectal support and explain the cure of Obstructive Defecation Syndrome (ODS), severe sacral pain and fecal incontinence reported by treatments based on the Integral Theory.[2,5,9,10,20,40]

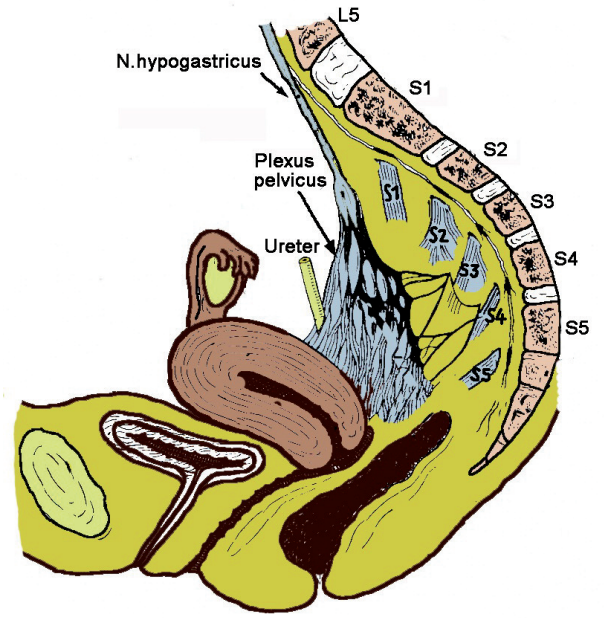

Figure 5. – Schematic diagram of pain transmission during birth. In the first stage of labour pain conduction is transmitted mainly to TH 11 and 12, in the second stage to the plexus pelvicus and sacralis.

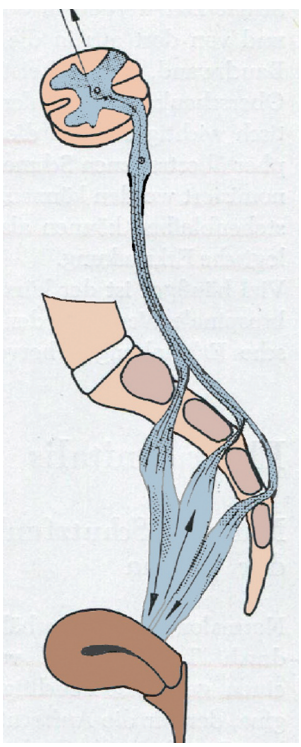

Figure 6. – Origin of mechanical, cerebrospinal transmitted gynaecological back pains.

In 1914 Symington[14] wrote in his textbook "Quain's Elements of Anatomy" for the first time that in the "utero-sacral ligaments" muscle tissue is present with close topographically connection to the Plexus pelvicus (Figure 4).

In 1917 Blaisdell confirmed in a comprehensive publication concerning the macroscopic and microscopic construction of "Plicae sacro-uterinae" that smooth muscle cells forms an integral part of these structures.[15]

In the year 1950 Campbell detected fibres of smooth muscle only in the anterior and middle part of the "Ligamenta sacro-uterina" whereas posteriorly just loose connective tissue, vessels and nerves could be found.[16]

1938 Martius published, that uterosacral ligaments (Figure 2) are mainly made of smooth muscles contracting spastically and painful in case of irritation.[17]

In order to interpret this problem and to find strategies of treatment, Petros performed a prospective study in 1996[4] including patients with pelvic pain of otherwise unknown origin and those with laxity in the posterior vaginal fornix. Histological examinations of uterosacral ligaments were part of this study and typically demonstrated the presence of smooth muscle, collagen, elastin, and nerve endings, both myelinated and unmyelinated in all specimens examined. The nerve fibres in the uterosacral ligaments were classified as parasympathetic visceral fibres. In his opinion, the visceral innervation incorporating fibres from T12-L1 provides an adequate explanation for pain distribution in the lower abdomen, specifically in the area of the ilioinguinal nerve (Figure 5). He hypothesized that stretching of weakened and loose uterosacral ligaments by gravity may stimulate the nerve endings within these tissues to cause pain.

Up to now there are still different views about the importance of the posterior suspension for pelvic organs. For example, in his textbook from 2008 Fritsch mentioned only a ligamentum recto-uterinum as a plication extending from rectum to the uterus. This ligament entirely forms the cranial boundary of "Douglas Cavity". For him there is no evidence of a structure coming from the os sacrum to the rectum or uterus.[13]

Petros,[19] Goeschen and Petros 2009[20] pointed out in their textbooks that the uterosacral ligaments (USL) arise from the sacral vertebrate S2-4 (Figure 3) and attach to the cervical ring posteriorly. The USL retains the fornix in place. Age or birth related loss of collagen/elasticity can lead to a uterine prolapse and USL-stretching. The blood support of the proximal USL is provided by the ramus descendens of the uterine artery, so that hysterectomy may cause further atrophy and weakening of USL by removing its main blood supply. The nerves contained within USL are sensitive to tension. This is easily demonstrated in such patients using the lower blade of a bivalve speculum. Gentle support generally relieves the pain. Excessive stretching will exacerbate it.[21]

In 2012 Forgács et al.[11] were able to localize the Ligamentum recto-uterinum macroscopically in all examined female cadaveric dissections. They detected a conjunction between the lateral part of the rectum and the cervical ring** and additionally along the connective tissue of the paraproctium to the surface area of the sacrum. Taken this in account they conclude that the term "Ligamentum sacro-uterinum" is absolutely correct. They furthermore removed two 1cm long pieces of tissue from the anterior and posterior part of the ligament for histological examination. Both specimens obtained firm fibrous connective tissue, what is typical for a ligament, vessels and lengthwise running smooth muscle fibres.

**This finding provides an anatomical basis for Abendstein's pioneering work on cure of "obstructive defecation" , CPPS and fecal incontinence using a posterior sling.

Pathway of pain transmission

Time limited pain caused by tension, compression, contraction or spasm is a well known physiological phenomenon in women during childbirth. These pains mainly have a mechanical origin and are therefore comparable to situations, which outside pregnancy, induces similar pressure to the pelvic floor. In this context the following points are of particular interest:

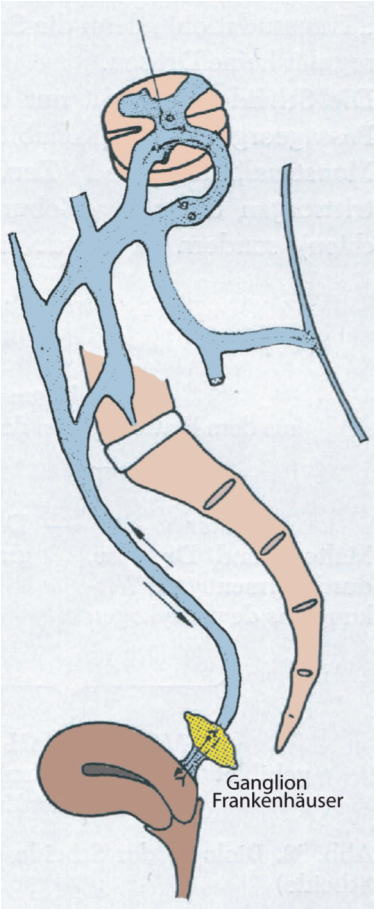

Figure 7. – Origin of visceroperipheral transmitted gynaecological back pains.

1) How does birth pain actually emerge?
2) Through which nerve tracts are these pains transmitted?
3) Do have similar pains in non-pregnant woman a comparable origin and identical transmission path?

Pain caused by deliveries consist of
- labour ache due to uterine contraction or spasm,
- pain induced by continuous cervix dilation and
- pain provoked by pressuring and stretching the pelvic floor as a consequence of fetal descending

In the first stage of labour aches related to uterine contraction are transmitted via the caudal placed Ganglion Frankenhäuser (Figure 7), the Plexus pelvicus, the sympathetic fibres of the N. hypogastricus to the dorsal roots of spinal cord at TH 11 until TH12 (Figures 4 and 5).

Cervix dilatation pains are transferred predominantly through the parasympathetic fibres of the plexus pelvicus to the sacral roots at S2 - S4.

Pain in the second stage of labour provoked by the descending fetus reaches via the pudendal nerve the plexus sacralis in the same region at S2 - S4 (Figure 4).

In the last 2 decades the epidural anaesthesia has become the most frequent procedure to interrupt labour pain conduction, whereas in former years the Ganglion Frankenhäuser was the preferable point to block the paracervical transmission (PCB see below Figure 9). In the hands of experts both methods are very effective for analgesia.[22]

For non pregnant women Martius[2] mentioned two pathways of lumbosacral pain-transmission already in the year 1946.

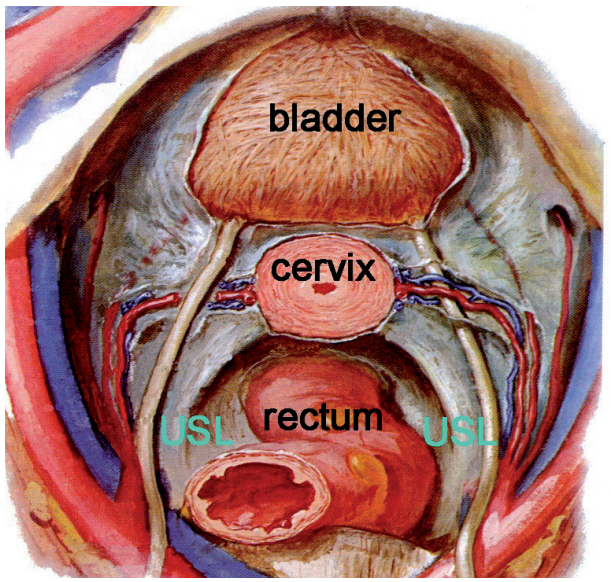

Figure 8. – Schematic diagram of the female pelvis mainly extending backwards. USL = uterosacral ligaments.

1) The first pathway goes directly to the brain via the fibres of the cerebrospinal nerve system induced by mechanical irritation of the sensitive receptors (Figure 6).

2) In case of pathological increased visceral irritation primary visceroperipheral pain conduction in the spine can be skipped to sensitive fibres of the peripheral cerebrospinal nerve system causing pain in those body segments that belongs to the related part of the spinal cord. These pains radiate mainly to the lumbosacral region, the anterior and lateral abdominal wall, the inguinal region and the thighs (Figure 7).

More frequent and important is the shorter, direct cerebrospinal way. The upright posture of humans imposes serious tension on the suspension and support system of the uterus, especially due to the fact that the female pelvis mainly extends backwards (Figures 2,8). On the wall of the pelvis there are numerous sensitive receptors from the cerebrospinal nerve system located, which can cause lumbosacral pain by pulling against the suspension apparatus. These pelvic pains are characterized by low dragging abdominal pain or deep sacral backache.

It is not surprising, that both concepts, pregnancy or not, are compatible. Regardless of pregnancy it is very likely that pelvic pain caused by tension, compression, contraction or spasm of pelvic organs emerge in the same way, because nerve transmission pathways will not change after delivery.

5 Possible reasons for organic lumbosacal pains

This paper especially concerns the significance of the posterior suspension and supporting system for vagina, uterus, bladder and rectum. The aim of this article is not to list all the numerous possibilities causing pelvic pain. That would lead to a monotone enumeration of nearly all gynaeco-pathological entities such as inflammation of genital organs, tumors, endometriosis, diseases of bladder, intestine, spine and so on. The discussion is limited to the structures supporting the uterus and posterior vaginal wall as detailed in figure 1, cardinal and uterosacral ligaments, perineal body and rectovaginal fascia.

Due to the fact that the uterosacral ligaments incorporate connective tissue, collagen, elastin and furthermore nerves, smooth muscles and vessels the question arises which mechanical or pathophysiological alterations can create chronic pelvic pain.

1) Pain as a result of spastic, colicky uterosacral contractions

It has long been assumed that smooth muscle fibres inside the uterosacral ligaments can cause painful spastic contractions. These colicky pains are especially localized on the left side of the pelvis combined with spastic constipation. For this condition Martius created the term "Parametropathia spastica sinistra cum obstipationem" in 1938 and indicated the connection between spasm and pain in the area of the posterior suspension system.[17]

A typical finding for theses cases is a radiation of severe pain into the sacrum whilst touching the posterior vaginal fornix during a gynaecological examination. Particularly painful is the area of the uterosacral ligament insertion points. The intensity of pain increases tremendously if the cervix is moved laterally or anteriorly. Identical back aches can emerge during deep sexual penetration.

This situation was and is still falsely confused with adnexitis, parametritis or proctitis. A proctitis is often taken into account, because pain accelerates during defecation. However this is not the result of inflammation. This exclusively is caused by condensed faeces passing and stretching the uterosacral ligaments.

2) Pain induced by irritation of Ganglion Frankenhäuser

The Ganglion Frankenhäuser,[23] cervical ganglion or so called Pelvic brain[24] is located bilaterally to the cervix uteri and vaginal fornix (Figure 7). It is situated in the connective tissue of the parametrium, on a level with the middle of the cervix uteri and about 2,5 cm lateralwards from the cervix. The pelvic brain is located in the base of the ligamentum latum at the distal end of the plexus hypogastricus. It is lodged practically at the junction of the cervix uteri with the vaginal fomix and has profound and extensive connection with the uterus, vagina, and rectum, ureter and bladder.

Distension and contraction of pelvic organs, with consequent change of visceral location, alters to a relative degree the syntopic relations of the pelvic brain.

During parturition, labour is initiated by the distalward movement of the child and the consequent mechanical irritation, pressure, excitement on the pelvic brain. The greater the distalward movement of the child in the pelvis the more mechanical irritation from the fetal head occurs on the pelvic brain, and consequently the greater number of nerve elements are excited. This is accompanied with increasing pain during labour due to pressure and tension to the pelvic brain.

Older obstetricians mainly know the Ganglion Frankenhäuser from the paracervical block (PCB). Injection of local anaesthesia in the paracervical area blocks the nerve connection to the N. praesacralis and Plexus sacralis. Immediately after injection patients are free of pain.

Pain caused by dilatation of the lower uterine segment as well as pain generated by the tremendous stretching of the uterine support system during birth is not longer present whilst the duration of anaesthesia. The injection area is located in the level of the uterosacral insertion points at 4 and 8 o'clock position. Too deep penetration of the needle can be prevented by the use of a guide sleeve (Figure 9).

Outside pregnancy the following considerations regarding pain generation can be derived from this item:

1. The Ganglion Frankenhäuser, located in the parametrium, undergoes permanent stimulation, if uterus or vagina descent. This can cause identical serious pains as during birth.
2. Mechanical support of uterus and vagina by restoration of the supporting and suspending structures should be able to stop the permanent stimulation of the paired ganglia. These patients should be free of pain lifelong, unless the supporting system gives way again.

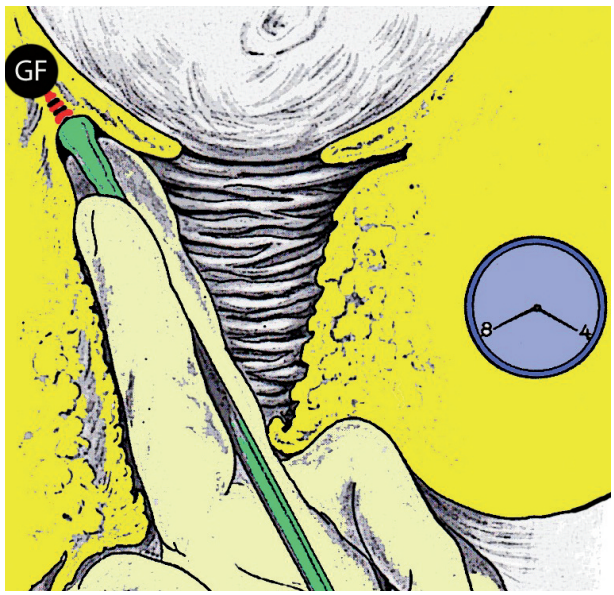

Figure 9. – Schematic picture of the paracervical block. Injection of local anaesthetics into the Ganglion Frankenhäuser area (GF) at 8 ó clock.

3) Pain generated by unphysiological tension on the plexus sacralis due to

- a) deficient suspending ligaments or
- b) deficient support from pelvic floor

a) deficient suspending ligaments

The female lumbosacral area provides an extensive widespread support and suspension apparatus for the pelvic organs, intensively connected with the periosteum of the posterior bony pelvis, the skeleton muscles and the sensitive receptors of the somatic nerve system. It is logical that a deficient support of the genital organs can lead to serious tension against the plexus sacralis with the result of severe back pain in this area (Figure 10).

There are two simple mechanical reasons regarding the fact that pelvic organs leave their normal position causing tension on the supporting and suspending system:

1) Due to the upright posture of humans the pelvic organs are exposed to the effects of gravity. Therefore the pelvic organs are predestinated to fall down.

2) The fixation of the genital organs has to be so flexible that the tremendous change of uterine position during pregnancy is possible.

Therefore since several years it is well known that pelvic pain is mainly related to uterine and/or vaginal prolapse provoked by loose suspending or supporting structures. As a result of this even women with minor prolapse can suffer from major symptoms, because these pains are generated by the downward force pulling against the plexus pelvicus (Figure 10).

1946 Martius[2] already published this issue related knowledge in the following sense:

In case of vaginal or uterine prolapse, severe, even "torturing" pain can occur in the lower abdomen or the low posterior pelvis, induced by a marked strain of prolapsed organs on the plexus sacralis. Symptoms are not only correlated to the stage of prolapse, but much more to the sensibility of the affected patients.

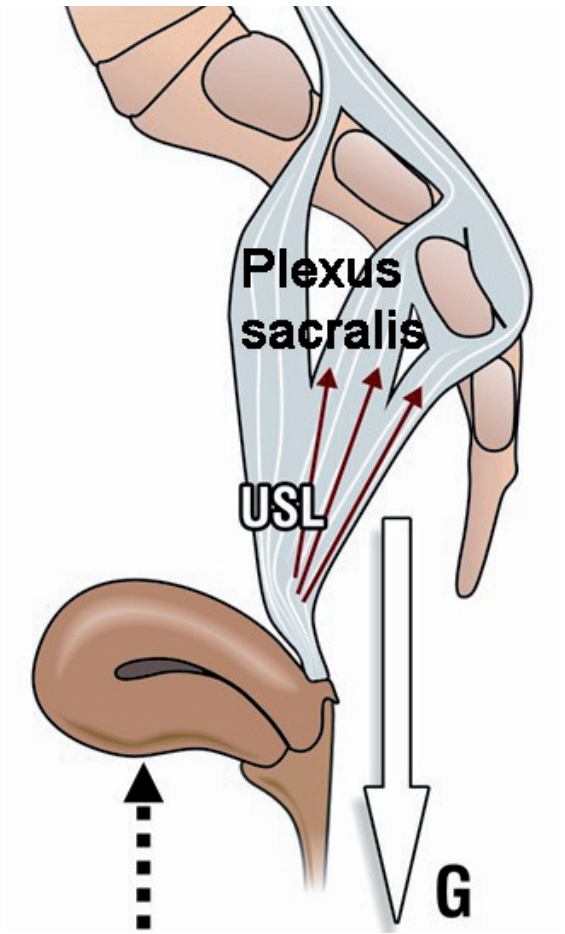

Figure 10. – In case of loose uterosacral ligaments (USL) or insufficient support from pelvic floor (black dotted arrow) gravity (G) pulls the uterus downwards producing pressure against the plexus sacralis.

After birth nearly every woman has at least a slight descent of her pelvic organs. Some have major prolapse of their organs. Not all patients experience pain and there is no relationship between the quantum of prolapse and the experiencing of pain. Because the pain is neurological in origin, major symptoms may occur with even minimal prolapse.

Patients complain that their intestines push downwards, mention a feeling of losing something and relate their present back pain to the prolapse on their own initiative.

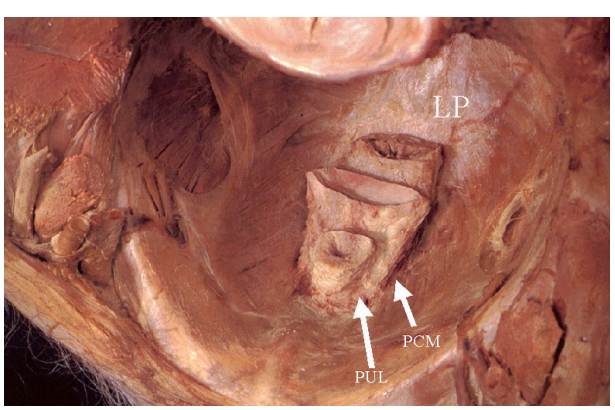

Figure 11. – Interconnectedness of organs and levator crus by connective tissue. Cadaveric specimen –view from above - front to back, urethra, vagina and rectum. PUL=pubourethral ligament; PCM=pubococcygeal muscle; LP=levator plate.

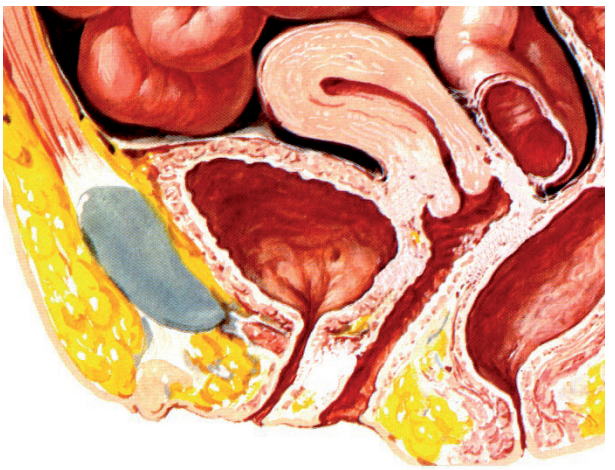

Figure 12. – Normal anatomy: A normal position of the uterus requires a stable support of pelvic floor.

b) deficient support from pelvic floor

As already mentioned, the content of the small pelvis is not only suspended, but also supported from the base. The pelvic floor contains three muscle layers located roof tile shaped one above the other. Striated muscles are not able to guarantee the necessary permanent tonus for the support of these organs by themselves. It is evident from figure 1 that the 3 directional muscle forces contraction (arrows, Figure 1) which stretch and support the organs contract against the suspensory ligaments. A weakened ligament will cause weakened muscle. Therefore the whole system is interrelated and ultimately reliant on firm suspensory ligaments. For this purpose the elastic system of visceral innervated muscle-connective-tissue-plate is required, the so-called lissomuscularfibrous system or endopelvic fascia. This unit is closely connected with skeleton muscle fibres, sealing interstices and acting as a rubber mat (Figure 11).

The pelvic floor has two functions: Firstly to obturate the abdominal cavity downwards and secondly to assure an exit for the intraabdominal organs (Figure 12). The fact that the outlet tract from bladder, rectum and uterus corresponds to the direction of gravity due to posture requires a particular well coordination of the lissomuscularfibrous system, especially as this system still has to work after deliveries.

A decline of pelvic floor followed by a descent of pelvic organs unavoidably must cause tension against the suspending ligaments (Figure 13). This can generate pain, primarily initiated by the deficient pelvic floor. However the suspending ligaments are stretched as well, but only secondarily, nevertheless still with the consequence of pain induction in the lumbosacral area.

Predominantly these pains can be addressed by a repair of the damaged pelvic floor which returns the organs to their normal position by restoration of loose ligaments. As the ligaments are weakened, simple plication will not work. Reinforcing the ligament by surgeries which incorporate the neoligament principle[25] are required.[5,9,10,20]

Severe sacral dragging pain can be the result of previous alterations in the parametric region. Martius[2] stated this situation as follows:

In case of a deficient pelvic floor or a damaged uterine suspension, intensive back pain can arise if the uterus is stiffly fixed to the pelvic wall by old parametric scar tissue (Figure 14). Insufficient support from below leads to heavy traction against the suspension area even in patients with minor prolapse. According to Sellheim this situation is called “hanging agony” in the old literature.[26]

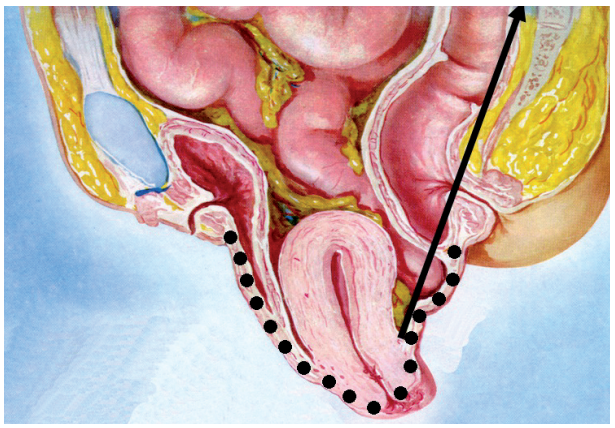

Figure 13. – Intestine prolapse: A damaged pelvic floor base (dotted black line) cannot support the pelvic organs causing prolapse, tremendous stretching of suspending ligaments (black arrow) and pain.

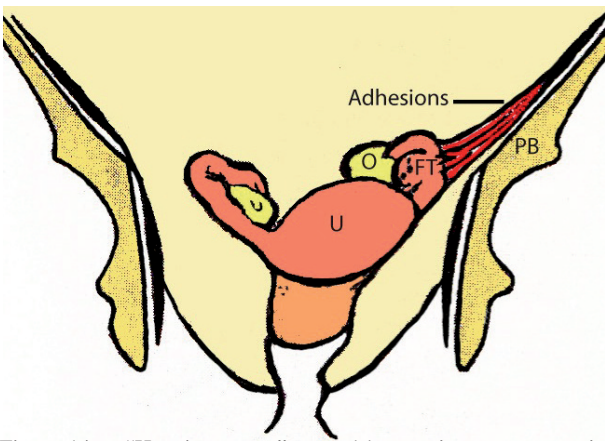

Figure 14. – "Hanging agony" caused by traction on parametric scar tissue. U = uterus, O = ovary, FT = fallopian tube, PB = pubic bone.

4) Pain induced by overstretching the uterosacral ligaments (USL)

Concerning the USL, Petros published the following hypothesis for the pathogenesis of chronic back pain:[4] Pelvic pain related to laxity of the uterosacral ligaments (USL) is characterized by low dragging abdominal pain, (often one-sided), deep dyspareunia, and often, low sacral backache. It can vary in intensity. Sometimes it can be sufficiently severe for the patient to present as an emergency.

The nerve fibres in the uterosacral ligaments are parasympathic visceral fibres. A visceral innervation incorporating fibres from T12-L1 adequately explains pain distribution to the lower abdomen, specifically, distribution in the area of the ilioinguinal nerve. It is hypothesized that stretching of lax ligaments by gravity may stimulate the nerve fibres within these tissues, and cause pain (Figure 15).

This pain is often relieved on lying down, and usually exists as part of the "posterior fornix syndrome"[3] which may include urge, frequency, nocturia and more recently, fecal incontinence, abnormal emptying of bladder and rectum.[5] CPPS pain may occur with only minor degrees of prolapse. This pain can be reproduced ('simulated') by digital palpation of the USL. It is hypothesized that the pain relief obtained after posterior IVS surgery is related to the physical support given to the S2-4 unmyelinated nerve fibres carried along the uterosacral ligaments.

A ring pessary may work in the same way by providing temporary mechanical support for the ligaments, and therefore, the nerve endings contained within.

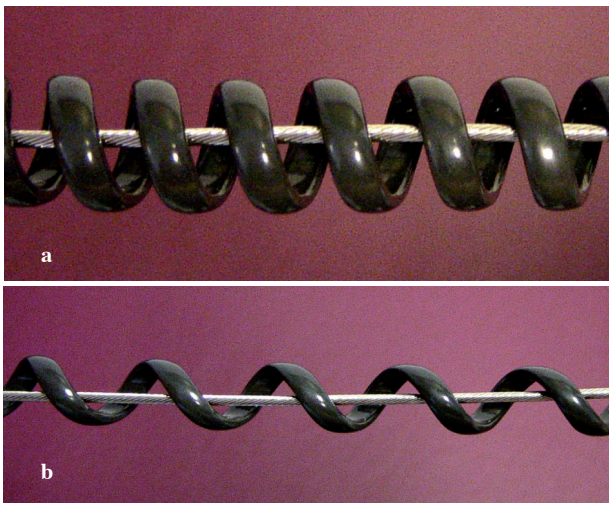

Figure 15a. and 15b. – Analogy of telephone cable and USL: a) USL with firm elastic connective tissue (black spiral cable outside) prevent stretching of nerve fibres inside b) Lax USL are not able to inhibit strain on nerve fibres inside

5) Pain caused by disturbance of blood circulation

The "Pelvic Venous Congestion Syndrome" (PVCS) is a chronic pain condition affecting 13-40% of women caused by varicose veins in the lower pelvis. PVCS generates chronic pain as well as dragging sensations in the lower abdomen and/or in the pelvis.

Varicosis commonly occurs in legs, when the valves in the veins stop working or there is an obstruction in circulation. In these cases the venous blood flows backwards and not to the heart causing pain and varicosis. This identically happens with pelvic veins in case of PVCS.

PVCS most commonly occurs in younger women, who have born children. During pregnancy the pelvic veins can be compressed by the fetus. This is thought to affect the valves in the veins causing them to stop working.

In many women after birth the connective tissue is lax and the suspension system overstretched. This leads to a descent of pelvic organs with the result of obstructed circulation and enlarged, bulging and knotty veins. Pain worsens on standing, lifting, during or after sexual intercourse and is usually improved by lying down.

Vaginal ultrasound examination is very helpful to detect abnormal dilated veins. But in lying position it can be difficult to demonstrate dilated pelvic veins because in supine position the pressure on the vessels is reduced, and the calibre of lumen appears physiological. Taken this in account the examination should be performed in standing position.

From Petros point of view[4] pelvic congestion is secondary to ligamentous laxity and can even arise in nulliparae or independent of pregnancy under the following conditions:

The uterus is normally supported by the cardinal and uterosacral ligaments, possibly assisted by contraction of the pelvic floor muscles. It is hypothesized that where the supporting ligaments are lax, the force of gravity acting on the uterus could cause congestion by "kinking" of the pelvic veins within these tissues, preventing outflow, thereby generating congestion.

Pain Symptomatic:

Women with deficient suspension or support of their pelvic organs have already gone through a long history of suffering, often accompanied by failed therapeutic attempts. The pain is sometimes so severe that these patients can become mentally deranged***. Some express suicidal

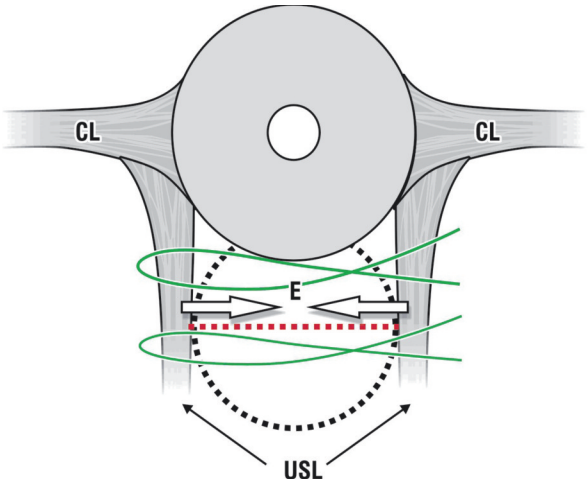

Figure 16. – Simple posterior fornix repair. A transverse incision is made in the posterior fornix 3-4cm below the cervix, A large No1 needle is inserted widely laterally below the vaginal skin and the loose uterosacral ligaments (USL) are approximated (arrows) with a strong Vicryl or polypropylene sutures. CX= cervix; CL=cardinal ligament; E=enterocele.

thoughts. Invariably laparoscopic findings are negative and this leads many of their treating physicians to refer them to psychiatrists.[4] This is a major tragedy as these pains can be vastly improved or cured. Even a simple USL plication as described (Figure 16)[4] can be effective, especially in the short term.

*** The consequences of this and failure of the medical establishment to recognize USL causation of CPPS is constitutes the theme "Conspiracy of Silence" in the patient book "The Bliss of Continence Restored", authors Peter Petros, Joan McCredie, Patricia Skilling, Amazon.com. Many case report examples amplify the devastating psychological effects as part of the Conspiracy theme.

Their pains are typically characterized as:
- Low abdominal dragging pain, often unilateral
- Low sacral pain
- Deep dyspareunia and postcoital ache
- Tiredness
- Irritability

Pain worsens during the day and is relieved by lying down. Pain can be reproduced by palpation of cervix and posterior fornix. Chronic pain may cause tiredness and irritability,[4] decreasing libido and creating marital stress and depression, all of which, in another context, could be interpreted as psychological associations, or even causes.

According to Martius[2] this pain can be provoked by moving the cervix laterally or anteriorly during gynaecological examination (see above).

In this context Petros[4] pointed out that insufficient connective tissue support for the non-myelinated nerve endings which course along the uterosacral ligaments, may cause referred low abdominal pain or sacral backache. Deep dyspareunia may induce pain by pressure on these nerves.

The lower abdominal pain and sacral backache may be reproduced by gently touching the posterior fornix digitally, or with a ring forceps. This is described as "cervical" or "vaginal" "excitation pain".

As already mentioned above pelvic pain is often part of the 'posterior fornix syndrome', which include urge, frequency, nocturia, abnormal emptying of bladder and rectum. The pain may occur with only minor degrees of prolapse.

Possible Therapy Options:

In pre-antibiotic times mercury or iodine was instilled intravaginally, leeches or "cantharidenpflaster" were applied vaginally as well as hot and cold compresses or baths were administered. With discovery of local anaesthetics these drugs have been injected into the posterior vaginal fornix. Furthermore since 1932 the osteopathic therapy is established for lumbosacral back aches.[27]

On the other hand patients with chronic abdominal or sacral pain have been treated in special sanatoria for women such as Bad Kissingen and Bad Pyrmont in Germany or Harkány and Hévíz in Hungary for along time.

Traditional health cures lasted at least four weeks. During this period the patients could recover from normal everyday life. In most cases this lead to an alleviation of symptoms as a proof for many doctors that these symptoms were mainly of psychosomatic nature. In order to put more emphasis on the psychosomatic component and the resulting psychotherapeutic treatment many new terms were created. Most of them were predominantly related to symptoms of vegetative dystonia [17,27-35] such as "Pelvipathia vegetativa, Spasmophilia genitalis, cervical syndrome, Plexalgia hypogastrica, congestion-fibrosis-syndrome, pelvis neuralgia".

On the other hand, in 1938 Martius[17] considered, that these symptoms were more likely related to somatic problems caused by local, mechanical stimuli amplifying the tension in the parametric tissue. In a high percentage of women, this local, mechanical irritation was originated by a deficient supporting or suspending system of the pelvic organs.

In his opinion, the pessary therapeutic option for pelvic organ support is not a good idea because a device acts only palliatively without any cure effect. On contrary, a pessary treatment can even worsen the situation by generating ulcerations and/or inflammation. The device will only stay in position, if the levator muscle gap is smaller than the circumference of the ring. If the gap is too large or the supporting area of the levator is weak, pelvic organs and the pessary will prolapse (Figure 17). Therefore insertion of a pessary has to be only a preliminary, time limited makeshift since it can cause a lot of troubles for many patients.

Taken this in account Martius in his surgical textbook 1936[36] stated *"This "problem can only be solved by a sufficient operation that is able to restore the natural anatomy"*. He furthermore pointed out *that it is not enough to narrow the vagina by the so-called "colporrhaphia anterior and posterior", because the holding ability of the vagina is inadequate*. Unfortunately this widespread so-called prolapse operation is not effective for an anatomical repair, but encourages surgeons to use bad operation techniques because the name of the operation sounds convincing.

In 1993 Petros in cooperation with Ulmsten[3] described pelvic pain as being a part of the "Posterior Fornix Syndrome" with symptoms comprising pelvic pain, nocturia, urgency, frequency and abnormal emptying. In 1996, still ignorant of Martius's work in the German literature, Petros[4] substantiated Martius´s statements by scientific research. He published his results about the relationship between pelvic pain of otherwise unknown origin and laxity in the posterior vaginal fornix in a prospective study. Twenty-eight patients with negative laparoscopy findings, lower abdominal pain and laxity in the posterior ligamentous supports of the uterus underwent surgical approximation of their uterosacral ligaments.

At 3-month review, 85% of patients were cured, and at 12 months, 70%. Petros's conclusion was that nonorganic pelvic pain has frequently been attributed to psychological factors. He suggested that this may be a T12-Ll parasympa-

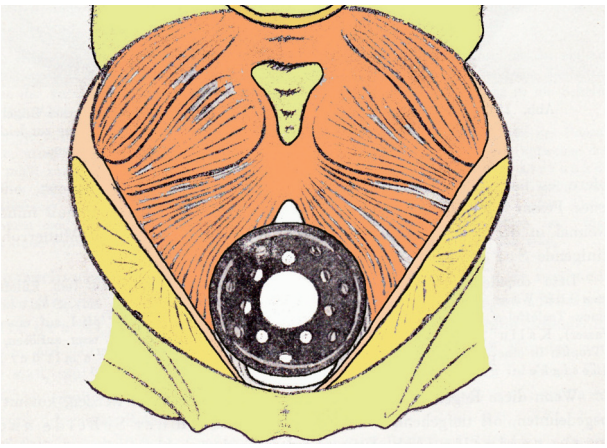

Figure 17a. – Schatz pessary in adequate position.

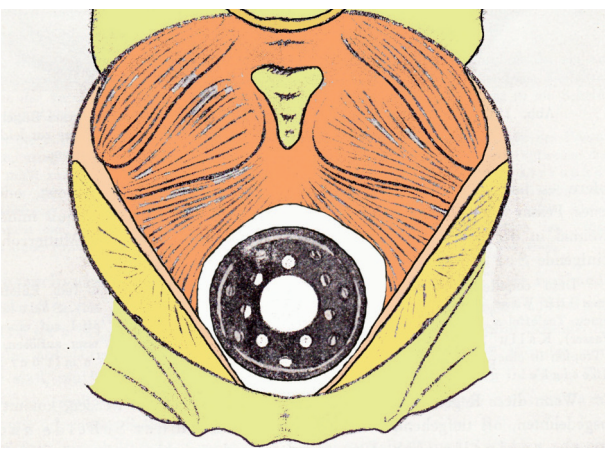

Figure 17b. – Not working Schatz pessary due to large levator muscle gap.

thetic pain referred to the lower abdomen, perhaps due to the force of gravity stimulating pain nerves unable to be supported by the lax uterosacral ligaments in which they are contained. It was concluded that laxity in the posterior ligaments of the vagina should first be excluded before referring patients with pelvic floor discomfort or pain for psychiatric care.

A ring pessary before operation may be useful as a diagnostic tool to relieve pain ("simulated operation") by providing mechanical support for the ligaments, and therefore, the nerve endings contained within. Another more recent diagnostic test is to gently insert the bottom blade of a bivalve speculum[21]. Yet another test method is to gently insert a large tampon into the posterior fornix (Gunnemann A. personal communication).

Because of deterioration in cure rate over time following the simple plication operation,[4] Petros developed a posterior sling operation (Figure 18) to reinforce the damaged uterosacral[38, 39] and nowadays the cardinal ligaments[10] as well.

Figure 19 demonstrates the physiological reconstruction of anatomy before and directly after vaginal reinforcement of the uterosacral und cardinal ligaments according to Petros. The laparoscopic taken pictures show a sufficient support of the uterus from the renewed ligaments. Keeping the uterus in natural position prevents traction against the lumbosacral plexus and pain.

In the meantime, the convincing data from Petros[4] are validated by numerous surgeons.

In 2002 Farnsworth[9] already published his data for pelvic pain cure after repair of posthysterectomy vaginal vault

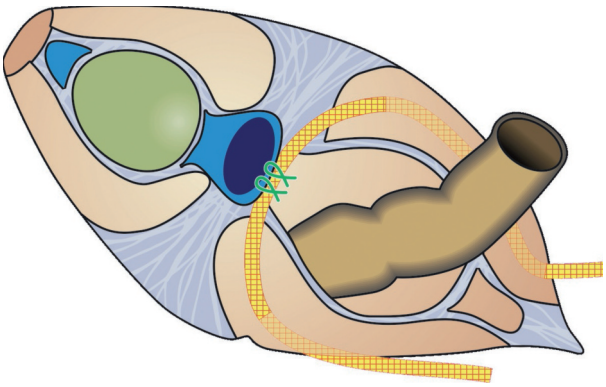

Figure 18. – Posterior intravaginal slingplasty (pIVS): The tape is placed along the exact position of the uterosacral ligament (USL).

prolapse. He performed the posterior intravaginal slingplasty (Figure 17), first reported by Petros 1997,[39] in 93 patients with posterior fornix syndrome. 21 from 27 patients (78%) with chronic pelvic pain were cured by the operation.

2004 Goeschen et al[40] analysed 59 patients with chronic pelvic pain before and after posterior intravaginal slingplasty. Follow up 1 year after the operation showed 42 patients (71)% having no pain any longer,[11] (19%) with improvement of more than 50% and only 6 (10%) without any effect.

Since that time numerous studies confirm these results.[10, 41-44] The cure rates for chronic pain are in a range between 62 and 83%.

Further treatment options for pelvic pain in literature consist of surgery to interrupt nerve pathways such as laparoscopic uterine nerve ablation and presacral neurectomy, hysterectomy with or without removal of the ovaries[45] or neuromodulation, where patients reported 40% improvement in their pain symptoms at 15 months mean follow-up.[46] But according to Daniels[47] laparoscopic destruction of nervous tissue is not more effective than a simple diagnostic pelviscopy.

In patients with Pelvic Venous Congestion Syndrome (PVCS) pelvic vein embolisation has been shown to be a safe procedure with relief of the symptoms of pain, and improvement in the appearances of the varicose veins. Up to 80% of women obtain relief using this method within 2 weeks of the procedure.[48,49]

However, this treatment is not able to eliminate the cause of venous dilatation. Congestion problem and pain will recur, if the following hypothesis is true: the uterus is normally supported by the cardinal and uterosacral ligaments, assisted by contraction of the pelvic floor muscles. It is hypothesized by Petros[4] that where the supporting ligaments are lax, the force of gravity acting on the uterus can cause congestion by "kinking" of the pelvic veins within these tissues, preventing outflow, thereby causing congestion.

This same laxity may also be an important cause of haemorrhoids.[50] The inward collapse of the anterior rectal wall may inhibit the venous return, distending the veins and creating backward pressure which may cause pain and bleeding.

This theory gives an explanation for the fact that PVCS and haemorrhoids emerge not only in women, who have born children, but also in nulliparae. Therefore PVCS and haemorrhoids cannot not only occur due to birth damage but to congenitally tissue laxity as well.

It has often been observed that pain, PVCS and haemorrhoids disappear after a three level posterior sling repair.[50]

Based on 1200 examined patients Forgács[11] recently assumed that muscle fibres located in the Plica recto-uterina can contract as in a colic and by this cause visceral pain in

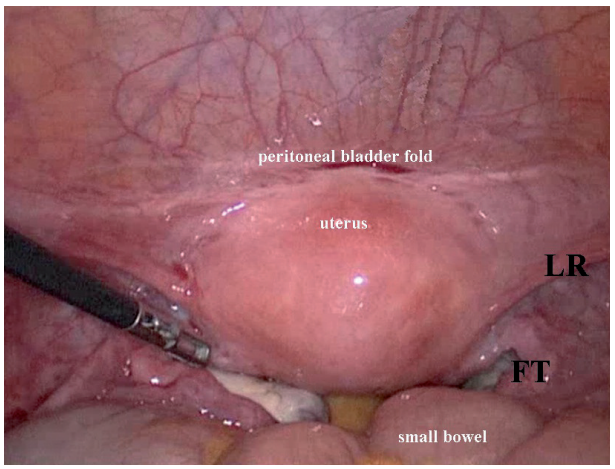

Figure 19a. – Uterus in normal position after vaginal insertion and sacrospinous fixation of a USL- and CL- neoligament. FT and LR, with normal shape, are far away from Douglas cavity.

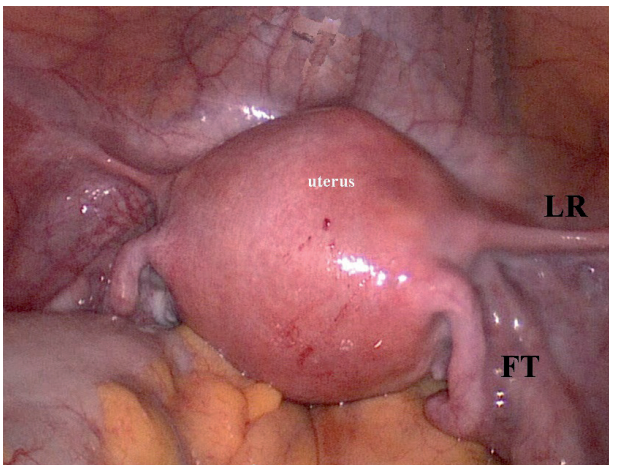

Figure 19c. – Uterus in descendent position. Ligamenta rotunda are lax (LR), fallopian tube (FT) deeply in Douglas cavity.

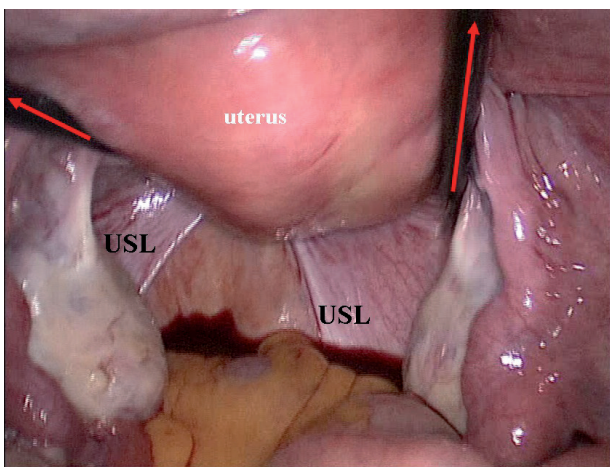

Figure 19b. – : Anteversion and elevation of uterus (red arrows) generate tension on the thin USL.

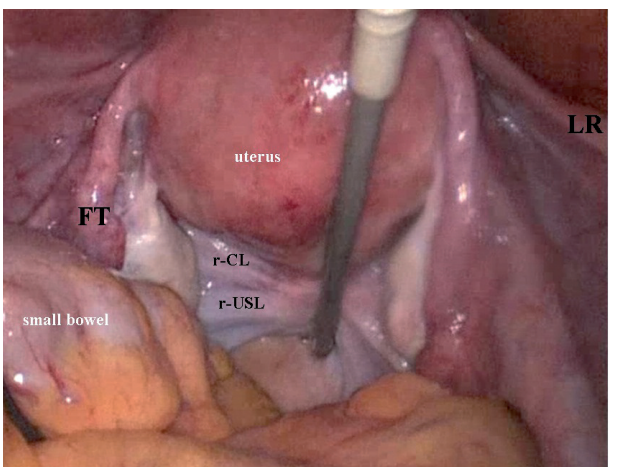

Figure 19d. – Reconstructed USL (r-USL) and CL (r-CL) in physiological position on both sides prevent a descent of the uterus.

the low pelvis. By triggering special points at skin and vagina with laser he was able to stop or reduce pain in about 80% of patients. However if a damaged anatomy is responsible for pain the effect of this method cannot remain for a longer period.

DISCUSSION

As stated earlier, the aim of this article is not to list all the numerous possibilities causing pelvic pain. Therefore the following discussion exclusively concentrates on the significance of the posterior suspension and supporting system for vagina, uterus, bladder and rectum, as summarized by Figure 1.

Chronic pelvic pain syndrome (CPPS) is a major health problem not only for the individual, but for society also.[6,7] Investigation by laparoscopy frequently reveals no obvious cause for the pain,[4] leading to ascribe causation to psychological reasons.

Chronic pain of moderate to severe intensity occurs in 19% of adult Europeans, seriously affecting the quality of their social and working lives.[51]

Though it is well known since years that CPPS in about 30% is provoked by damaged suspensory ligaments of the pelvic organs,[2] experts and expert committees state that the pathogenesis of chronic pelvic pain is poorly understood.[6-8] In a 1996 study, the estimated direct medical costs for CPPS outpatient visits alone for this group in the U.S. was $881.5 million per year. In addition, 15% reported time lost from paid work and 45% reported reduced work productivity.[52] Taking this in account is it very important to cure these patients.

Observational studies indicate that the prevalence of menopausal patients with pelvic organ prolapse is between 31-41.1%.[53,54] A multicenter study presents the following distribution of prolapse patients: 24% with stage 0 prolapse, 38% with stage I, 35% with stage II, 2% with stage III, and 0% with stage IV.[55] Nygaard et al[56] found 2.3% with stage 0 prolapse, 33% with stage I, 62.9% with stage II, 1.9% with stage III, and 0% with stage IV. The lifetime risk of undergoing an operation for pelvic organ prolapse is reported to be 11-19%.[57,58]. However, none of these studies address the fact that CPPS can occur in patients with quite minimal prolapse.[10,19,20]

Regarding anatomical changes in the suspension or support system of pelvic organs, there are 5 different reasons that can cause pelvic dragging pain in the back (see above). All 5 possibilities lastly lead to the fact that either the nerve endings or muscle fibres contained within the uterosacral ligaments (USL) are stretched, leading to traction against the plexus sacralis or the Ganglion Frankenhäuser or dilatation of pelvic veins generates pressure to the surrounding area. However, as stated previously, the pelvic congestion may be secondary to the ligament looseness.[4]

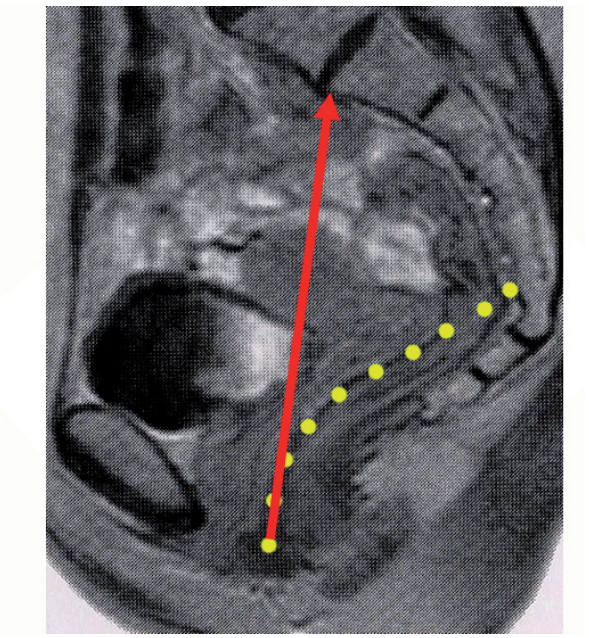

Figure 20. – MRI – Picture of the female pelvis in upright position. Sagital view of pelvic organs.Dotted yellow line = normal axis of vagina in a banana shaped backwards curveRed arrow = unphysiological vertical unbowed axis of vagina after laparoscopic/laparotomic sacrocolpopexy.

From the therapeutic point of view, these patients can be pooled in three groups:

Group 1) Patients with intact pelvic floor, but damaged ligamental suspension. In these cases uterus, vagina, rectum and bladder can leave their normal position causing a tension to the nerve fibres inside the USL or to the lumbosacral plexus.

Group 2) Patients with damaged pelvic floor but sufficient ligaments. This situation leads to insufficient support of the intestinal package followed by a descent of these organs generating painful traction against the plexus sacrospinalis. These pains arise even if the suspension system is sufficient, because gravity pulls the deficient supported pelvic organs downward creating tension via the USL to the lumbosacral plexus.

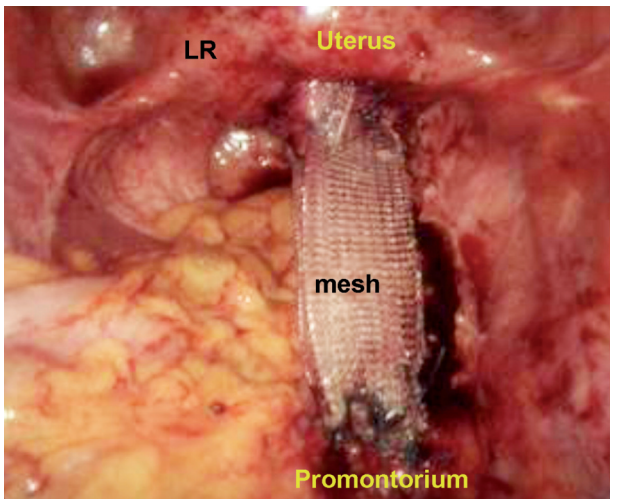

Figure 22. – Laparoscopic fixation of the uterus to the promontorium with a mesh-graft. In contrast to vaginal repair illustrated in Fig. 18 this procedure reconstructs neither vaginal axis nor USL and CL along the physiological course.

Group 3) Patients with a combination of both.

This differentiation is not only of scientific interest, but has even important therapeutic consequences and allows explanations for the different cure rates after vaginal or abdominal surgery in literature.

Numerous surgeons favour the abdominal way to restore the anatomy or to cure anatomical lumbosacral pains either by laparoscopy[59-64] or by laparotomy.[65-68] The success rate, when defined as lack of apical prolapse postoperatively, ranged from 78-100% and when defined as no postoperative prolapse, from 58-100%.[69]

Taken this in account Cochrane analyses[68] and a recent review article[69] come to the conclusion:

"Abdominal sacrocolpopexy (ASCP) is the **SCP** (sacrocolpopexy) **standard** for vaginal vault prolapse and is superior to vaginal sacrocolpopexy, with fewer recurrent prolapses and less dyspareunia. Laparoscopic sacrocolpopexy **upholds** the outcomes of the gold standard abdominal sacrocolpopexy with minimal morbidity".

As there is only a weak correlation between the extend of prolapse before and after sacrocolpopexy and pelvic symptoms.[2,4,70,71] Bojahr et al[59] performed a retrospective cohort

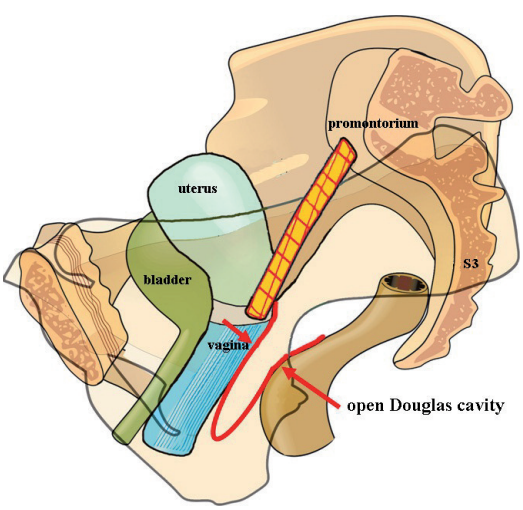

Figure 21. – Schematic picture of the female pelvis in upright position after abdominal colposacropexy. Sagital view. Fixation of vaginal apex and cervix to the promontorium with mesh pulls the uterus forwards and opens the Douglas cavity.

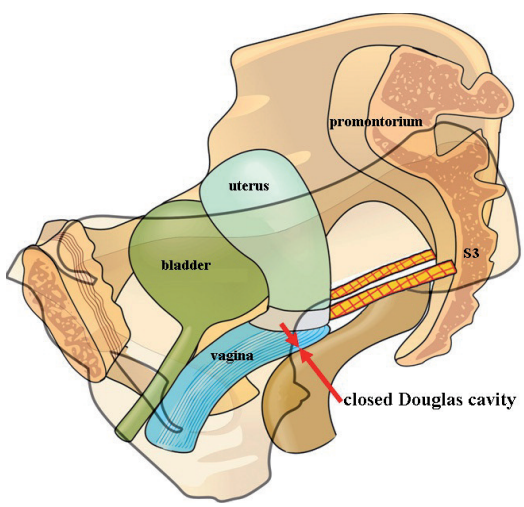

Figure 23. – Schematic picture of the female pelvis in upright position after vaginal colposacropexy. Sagital view. Fixation of vaginal apex and cervix to the vertebrate S3 with mesh creates a normal vaginal axis keeping the Douglas Cavity close.

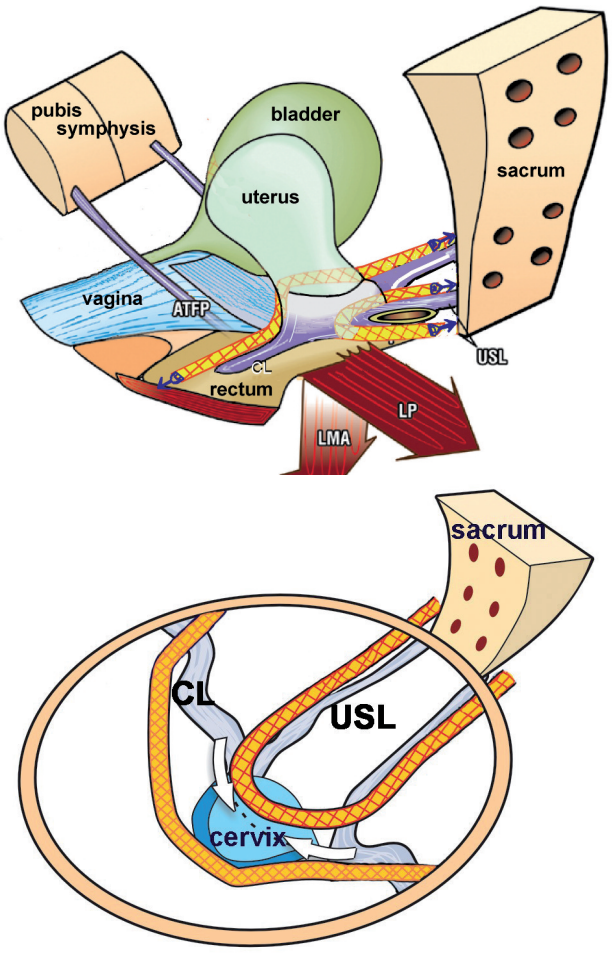

Figure 24a. and 24b. – Physiological reconstructed vaginal axis and shape after vaginal insertion of artificial ligaments along the exact course of uterosacral (USL) and cardinal (CL) ligament. ATFP = arcus tendineus fascia pelvis, LP = levator plate, LMA = longitudinal muscle of the anus.

study among 310 women with the aim to assess the subjective outcome following laparoscopic sacropexy. Subjective success of prolapse surgery was determined by the absence of symptoms.

The study shows "a significant postoperative reduction of nearly all assessed symptoms". However, the persistence of back pain was 82.9%. That means the cure rate was only 17%. Additionally 40% of the women wearing pessaries preoperatively still needed a pessary therapy after surgery and 22.4% required a further prolapse surgery during the mean follow up of 24.5 months.

In contrast to abdominal surgery, numerous more recent studies present much better results after vaginal sacrocolpopexy regarding back aches and other symptoms. The cure rates for lumbosacral pain after posterior sling operations[37,38] range between 62 and 83%.[9,10,38,40-44] Data from numerous studies[9,10, 40-44] demonstrating a high cure rate for CPPS leads to the conclusion that abdominal surgery obviously cannot no longer be accepted as Gold Standard if we include symptoms in the assessment criteria.

Is there an explanation for this contradiction between adequate anatomical restoration of the apex and failure to cure accompanying symptoms?

From the view of an engineer, an architect or a surgeon optimal results regarding symptoms and anatomy can only be achieved by an accurate reconstruction of the anatomy

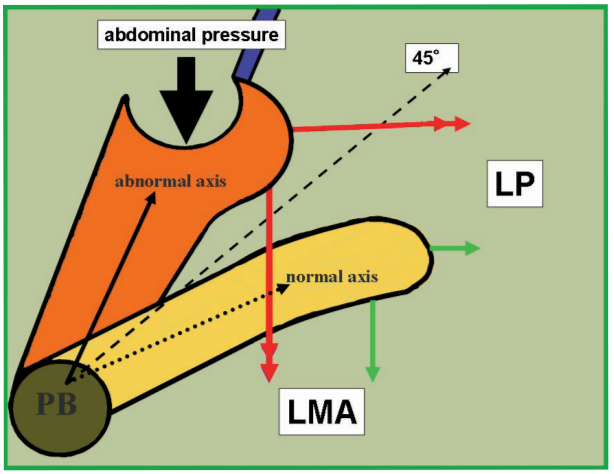

Figure 25. – Abnormal vertical axis due to promontorium fixation (blue).In case of vertically inclined vagina with an axis to the horizontal >45 degrees abdominal pressure, backward force of levator plate (LP), downward force of longitudinal muscle of anus (LMA) accelerate prolapse/entero/rectocele formation.PB = Perineal Body.

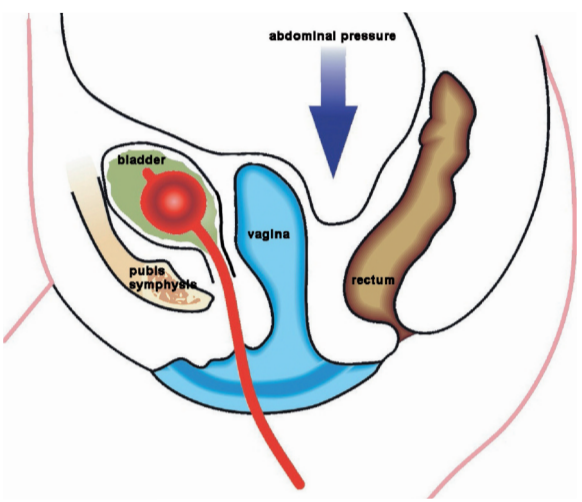

Figure 26. – Vagina in unphysiological vertical position after abdominal repair. The unsupported open posterior space allows the intraabdominal pressure and gravity force (dark blue arrow) to push the Douglas Cavity downwards creating vault prolapse, entero/rectocele and rectal intussusception.

as it occurs in Nature. "*150 years after Christ Galen already stated that normal function of organs follows reconstruction of form and structure*".

That means:

- If entirely the suspension system is deficient it might be enough to repair only the loose ligaments.
- In case of pelvic floor damage this problem must be solved by restoration of the base in order to support the pelvic organs and the intestine.
- If ligaments and pelvic floor are lax, both structures have to be renewed.

Taken this in account the following question arises: Which surgical way, abdominal or vaginal, provides the best results concerning the 3 different situations.

Group 1) If exclusively damaged uterosacral and/or cardinal ligaments are responsible for pelvic pain, this problem can be solved abdominally as well as vaginally. However abdominal surgery as ***it is performed today*** does not recreate the natural axis of the vagina because the at-

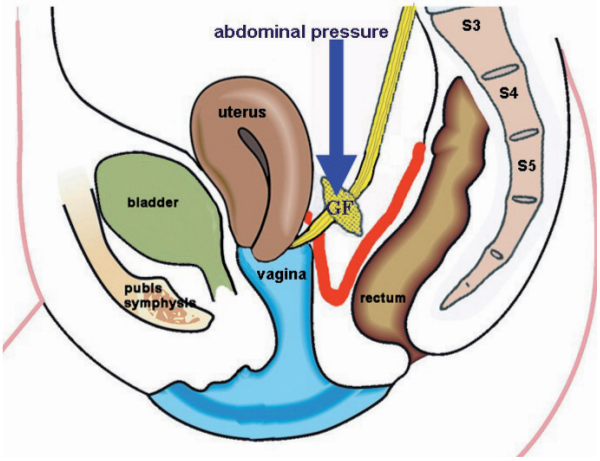

Figure 27. – Vagina in unphysiological vertical position after abdominal repair. Intraabdominal pressure (dark blue arrow) and gravity force squeeze the unsupported Ganglion Frankenhäuser (GF, yellow) downwards generating pai.

tachment area of mesh is the promontorium (Figures 20, 21, 22). Therefore this procedure creates an abnormal vertical inclined vaginal axis.

Physiologically the uterosacral ligaments (USL) arise at sacral vertebrate S2-4 (Figure 3) a significant distance from the promontorium. In contrast, new vaginal procedures such as "posterior-intravaginal-sling" (pIVS) or "tissue-fixation-system-" (TFS) operations[10] are able to place the neoligament along the exact position of the uterosacral or cardinal ligaments (USL) (Figures 18, 19, 23, 24).

A vertical inclined vagina after abdominal SCP surgery is unphysiological and can therefore generate three problems:

The more the vagina and the pelvic organs are in vertical position

1) the more they are exposed to the effect of gravity and predestinated to fall down (Figure 25a).

2) the greater the posterior space is opened for causing enterocele (Figures 21, 26).

3) the less the vagina can be compressed downwards by the muscle forces of the levator plate (LP) and the longitudinal muscle of the anus (LMA) that opens and close bladder and rectum (Figure 26).

As the axis of vagina after laparoscopy/laparotomy is more vertical, a recurrence or new formation of prolapse (Figures 20, 21, 26) can be expected more often compared to the vaginal approach.

Furthermore, the unsupported open posterior space allows the intraabdominal pressure and gravity force to push the Ganglion Frankenhäuser (GF) downwards (Figure 27). Mechanical irritation on GF consequently excite a great number of nerve fibres generating pain due to pressure and tension.

That means: If GF is not supported from below, abdominal pressure can generate pain by pushing GF downwards, even though there is no traction against the plexus sacralis. That explains why, in case of new formed enterocele due to vertical vaginal axis, sufficient reconstruction of uterine suspension is not always effective against pain.

Group 2) Pain patients with damaged pelvic floor but sufficient ligament suspension (Figure 12) primarily need a reconstruction of the base. In healthy women the pelvic floor physiologically is stable and firm guarantied by the three muscle layers closely connected with the endopelvic fascia (Figure 10). The strongest muscle layer is formed by the M. levator ani and M. coccygeus. The M. levator ani is composed of two components: 1) pars pubica and 2) pars ischiadica (Figure 28).

Regarding prolapse patients, the pars pubica is the most interesting part of the levator ani. The "levator crus", the inner parts of the levator ani muscle plate are located near to the body midline and girdle the genital hiatus, the passage for urethra, vagina and rectum. The genital hiatus has a triangular shape with the largest extension at the pubis symphysis. A stable and narrow hiatus genitalis is necessary to prevent a descent of genital organs.

In case of genital prolapse the hiatus genitalis is dilated. This may be due to damaged levator insertions and/or damaged perineal body (Figure 29). In many cases, the levator crura have moved laterally, vastly opening the hiatus during straining. This problem can only be solved by a sufficient operation enabling the restoration of natural anatomy (Figure 30).

It is logical and has been recognized for decades that abdominal operations provide no access to the important hiatus area.[36] However, even vaginal procedures can only be successful, if surgery reconstructs both, the dilated hiatus genitalis and perineal body as well.

Narrowing the vagina by the so called "colporrhaphia anterior and posterior" is not effective, because the holding ability of the vagina is inadequate. Therefore Martius[36] already mentioned the importance of levator and perineal body sutures to constrict the hiatus genitalis (Figure 30). However, suturing the hiatus will inevitable create pain and tension and the sutures may tear out, as is well known with native tissue perineal body repair. The TFS method uses an adjustable sling to narrow the hiatus by joining the divaricated muscle bellies. The pain is minimal, as the tissues are simply restored to their original position, and are joined by a TFS tape which in time is infiltrated with fibrous tissue to form a new central tendon (Figure 24).

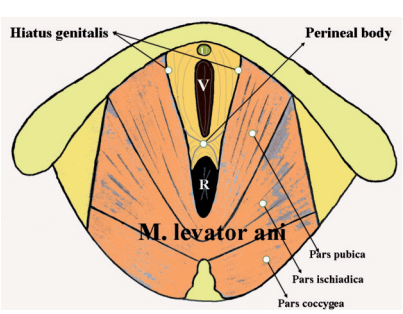

Figure 28. – Stable and firm genital hiatus protected by a strong pubic branche of the levator ani muscle.

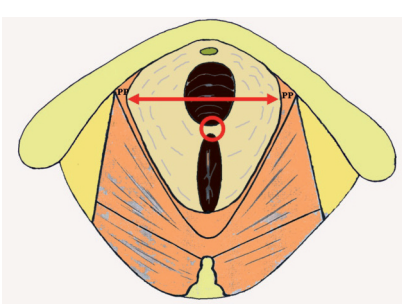

Figure 29. – Extremely wide genital hiatus (red arrow) caused by damage of levator ani muscle and endopelvic fascia. The levator crura are thin and far lateral, the perineal body (red circle) deficient.

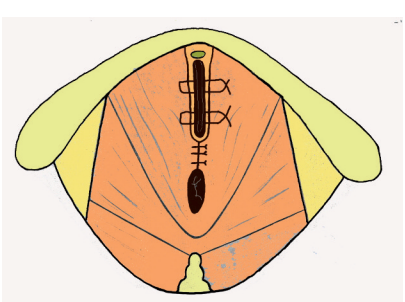

Figure 30. – Reconstructed genital hiatus after repair of perineal body and levator crura.

Group 3) Patients with a combination of both

Due to the fact that deficient connective tissue is mainly responsible for prolapse and pelvic floor dysfunction, an isolated damage of ligaments represents an exception. In the majority of cases, a descent of pelvic organs is the consequence of both, insufficient support and suspension.

A new dimension of understanding arose in 1992 when De Lancey,[72] based on cadaveric dissections, demonstrated the significance of connective tissue structures for organ suspension by specifying three levels of vaginal support (Figure 31):

Level 1: superior attachment (cardinal/uterosacral ligament complex)

Level 2: lateral attachment (superolateral insertion points of anterior vaginal wall, rectovaginal fascia)

Level 3: distal attachments, i.e. perineal body, perineal membrane

Petros created a new vaginal strategy of pelvic floor surgery based on the Integral Theory[73] which regards symptoms and organ prolapse as being both caused by lax suspensory ligaments (pubourethral, cardinal, ATFP, uterosacral and perineal body). Application of the neoligament principle used in the TVT,[73] cure rates have been reported that have not been achieved before.[38,40-44] He accurately reconstructed the three levels (Figure 31) by

1) insertion of a tension free tape to create an artificial pubourethral, uterosacral and cardinal neoligament (Level 1 repair)

2) reinforcement of rectovaginal fascia and narrowing the genital hiatus (Level 2 repair) and

3) repair of perineal body and membrane (Level 3 repair) (Figure 31).

Following the Integral Theory[73] abnormal symptoms due to prolapse are mainly caused by connective tissue laxity in the pelvis. Therefore an isolated damage of suspending ligaments is an exception. In the majority of cases the supporting system will be deficient as well. Thus, in most patients with pelvic floor problems, a 3 level repair is necessary to reconstruct the natural anatomy and to cure the symptoms.

Keeping all these considerations in mind, there is only a small gate for abdominal procedures. Laparotomy or laparoscopy **as it exists today**, enables only the elevation of the descended level 1 structures such as vaginal apex or uterus and can suture a displaced anterior vaginal wall to the arcus tendineus fascia pelvis (ATFP). However, even these procedures reconstruct the anatomy not physiologically.

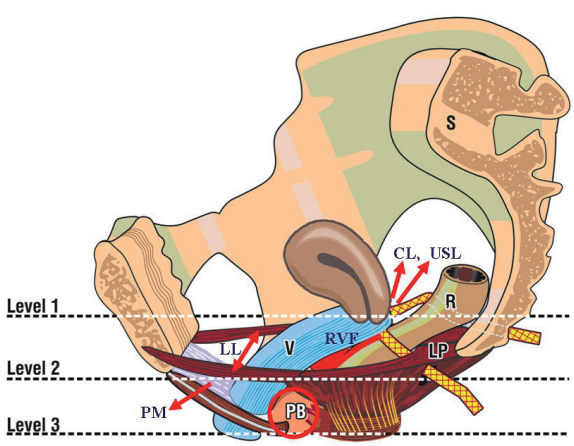

Figure 31. – Fig. 31 Three level repair:
Level 1 cardinal ligament (CL) and uterosacral ligament (USL)
Level 2 rectovaginal fascia (RVF) and levator cruses (LL)
Level 3 perineal body (PB) and perineal membrane (PM)

Therefore it is not surprising that abdominal surgery **as it exists today** provides worse cure rates for symptoms than vaginal surgery.[59] That said, it is envisaged that application of the TFS laparoscopically may be able to re-suspend the vaginal apex as accurately as the vaginal operation (Petros personal communication).

Patients with persistent pelvic pain after sufficient pelvic floor restoration should be examined whether spastic parametropathy or pelvic congestion due to varicosis is responsible. Forgács et al[11] published cure rates of about 80% in patients with spastic parametropathy by triggering special points at skin and vagina with a laser.

According to Ignacio et al[48] and Ganesh et al[49] up to 80% of women with pelvic pain caused by varicosis obtain relief within 2 weeks after pelvic vein embolisation.

CONCLUSIONS

Chronic pelvic pain syndrome (CPPS) is a major problem seriously affecting the quality of life in up to 20% of women. Differentiation from other causes of pelvic can be made by using the pictorial algorithm to identify one or more co-existing posterior fornix symptoms which almost invariably co-occur. The diagnosis can be checked by the use of "simulated operations" to provoke or alleviate the pain during office examination. Any operation which supports the apex can theoretically cure CPPS. A low transverse incision 3-4 cm below the cervix with plication of the loose USLs will produce a high initial cure rate for CPPS. On this basis alone, it is worth considering, especially by surgeons untrained or unwilling to use as mesh tapes. Because this method has an increasing failure rate with time, it has been found that most effective longer-term surgical technique for CPPS is to reinforce the USLs with a polypropylene tape precisely inserted into the position of the USLs. Abdominal SCP operations, though effective for prolapse, are far too imprecise to restore symptoms effectively.

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INVITED COMMENTS

In this issue, Goeschen K. has presented a review on the Chronic Pelvic Pain Syndrome (CPPS) in women and its relation to pelvic floor dysfunction. Several pelvic floor surgeries, such as that by Heinrich Martius1, the Integral Theory2 or experiences of improvement of pain by means of reinforcing lax uterosacral ligaments3 are cited. He details how this method also may cure other symptoms, described in the 1993 publication of the Integral Theory as the "Posterior Fornix Syndrome"4: urgency, frequency, nocturia, abnormal bladder and bowel evacuation and non-sphincteric fecal incontinence.

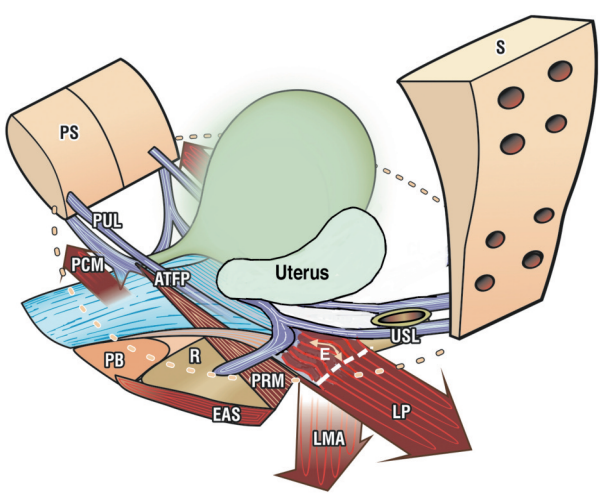

Figure 1. – How lax uterosacral ligaments (USL) may inactivate the muscle vectors according to Gordon's Law. The wavy lines 'E' above the horizontal broken lines along LP (levator plate) indicate how prolapse can lengthen the LP muscle. According to Gordon's Law, muscle lengthening will inactivate the muscle forces which act on the both LP and LMA (longitudinal muscle of the anus). As LP and LMA are key vectors in urethral and anorectal closure and opening, this may result in obstructive and incontinence symptoms for both organs. It is also clear that a firmly contracted LP muscle will support the apex and uterosacral ligaments (USL) and the nerve endings contained within it.

It is important to understand the pathogenesis of pelvic pain: Peripheral hypersensitisation and central (systemic) hypersensitisation both contribute to the full extent of the chronic pelvic pain syndrome. It seems that the peripheral hypersensitisation is reversible to some extent, while the central hypersensitisation is more difficult to treat. Therefore it is important to diagnose and treat all phenotypes possibly causative for pain arousal and chronification. It has been shown in several occasions, that uterosacral ligament laxity can produce pain symptoms, even with minor prolapse. If this is the case a phenotype directed approach should include pelvic floor reconstruction in the pain management. In this regard an important aspect is to define the exact anatomical site of the injury, which usually includes the uterosacral and cardinal ligaments, which then should lead to site specific reconstruction of the damaged anatomical sites.

The complex anatomical associations have been discussed in detail by this review of Goeschen (Figure 1), also in a historical context.

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* * *

Professor Goeschen has provided an excellent historical survey of the Chronic Pelvic Pain Syndrome (CPPS), from Heinrich Martius to the Integral Theory. I have been applying the Integral Theory paradigm since 2006, when I began using the Tissue Fixation System (TFS). In an experience spanning several hundred cases, I have found a high cure rate for posterior fornix syndrome symptoms, urgency, frequency, nocturia, abnormal bladder and bowel evacuation following anatomical correction of apical prolapse and also for CPPS. I have also found that even minor apical prolapse can produce severe symptoms.

Of course, the pictorial algorithm is very important to differentiate between other types of CPPS and that caused by looseness in the uterosacral ligament.

I have found that a Pro Dry pessary inserted into the posterior fornix mechanically supports the apex and reduces or eliminates the various loci of CPPS. In my experience, these are lower abdominal or groin pain, low sacral backache, vulvodynia. It also often reduces urgency. If left in overnight, it can significantly lessen the episodes of nocturia. As the support is mechanical and depending on the anatomical condition, a large menstrual tampon soaked with estrogen cream could achieve the same result.

Dr Alfons Gunnemann MD PhD
Chefarzt Dept of Urology, Klinikum Lippe Detmold, Germany

* * *

I rarely had the chance to read such an article. This is about hard work and a lot of wisdom.

Concerning the key aspects I think:

1. I absolutely agree the existence of posterior fornix syndrom. My experience began in 2007 and till now I operated 178 women with complaints of frequency, urge, nicturia and emptying troubles. Only 21% had chronic pelvic pain as associated symptom. In 80% of cases all complaints disappeared in next 24 hours and results were stable at 1 year in 90% of cases

My initial experience was with TFS (50 cases) with good results. Because of availability and cost, I now do Mc Call associated with a procedure which anchors the anterior aspect of cervical ring transobturatorly with a small piece of mesh. I named this procedure "spatial stabilization of cervical ring".

2. I published in 2014 a paper "Laparoscopically assisted vaginal hysterocolposacropexy" where the concept of posterior fornix syndrome is emphasized.

3. The concept of "force equilibrium in pelvic reconstructive surgery" that means that any procedure in one pelvic compartment must be accompanied by another procedure (curative or prophilactic in the opposite compartment). This concept I considered helpful especially in early degree of prolapse.

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Professor Petre Bratila MD PhD,
Bucharest Rumania

* * *

I am very pleased to confirm Professor Goeschen's comments on a loose uterosacral ligament being responsible for the causation of a wide rang of bladder, bowel and chronic pelvic pain symptoms (of posterior fornix syndrome).

There is no question about the existence of the posterior fornix syndrome.

My experience began in 2009 and it concerns the posterior IVS (pIVS). Up to now I have operated approximately 400 women with complaints of frequency, urge, nocturia and chronic pelvic pain (CPPS), who had have a terrible social life. Because they couldn't sleep through, they had have many problem in their marriage and daily life. In almost 90% of cases all complaints disappeared within the next 24 hours after pIVS and results were stable at follow up 1 year post op. The effect on quality of life following cure of CPPS and nocturia is remarkable.

Since 2009 all my prolapse patients were treated with posterior IVS in addition to other surgical steps. At 12 months, the anatomical recurrence rate is less than 2%, the symptomatic cure rate more than 80%.

Most of the patients were hopeless due to numerous frustrating drug treatment and operations. Fortunately, this was the past. Nowadays we can help these patients with a reconstruction of the posterior suspension system. As a gynecologist, I know how is this success precious by urogynecological patients.

I work in Denizli Turkey, where we do not have the facilities of large hospitals. The pIVS which I use is a very simple operation with few problems and it is therefore very suitable for cure of these problems".

Dr Alpaslan Caliskan MD
Denizli, Turkey

* * *

I write to congratulate Professor Goeschen on a major contribution to pelvic floor science and to support his conclusions that chronic pelvic pain syndrome (CPPS) is curable by surgically supporting the posterior vaginal fornix.

Our group in Kamakura Japan has been using the TFS system since 2006. Based on our experience over many hundreds of patients where TFS was used to correct apical prolapse, we can confirm that at least in our practice, mainly patients with a mean age of 70 years, chronic pelvic pain as described by Petros in 1996 is a common condition and it is associated with symptoms of urgency, frequency, nocturia, obstructive defecation and fecal incontinence ("Posterior Fornix Syndrome"). Our group has achieved high cure rates for apical prolapse at 12 months (>90%) but also, associated symptoms, CPPS and other Posterior Fornix Syndrome symptoms, Table 1. The tape rejection rate for apical prolapse has been <1%.

Posterior TFS sling

Table 1 Symptom Outcome – 403 patients.[4]

Symptom change with surgery			% cure in brackets		
Faecal incontinence	Frequency > 10/Day	Nocturia > 2/night	Urge incontinence > 2/day	Abnormal emptying	Pelvic pain
Australia[4] 67 patients					
$n = 23$ (87%) $P \leq 0.005$	$n = 27$ (63%) $P \leq 0.005$	$n = 47$ (83%) $P \leq 0.005$	$n = 36$ (78%) $P \leq 0.005$	$n = 53$ (73%) $P \leq 0.005$	$n = 46$ (86%) $P \leq 0.005$
Japan[5] 336 patients					
$n = 52$ (82.7%)	$n = 179$ (84.9%)	$n = 129$ (60.5%)	$n = 171$ (91.2%)	NA NA	$n = 76$ (71.1%)

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Dr Hiromi Inoue MD
Kamakura, Japan

* * *

I found Klaus Goeschen's review paper on CPPS very enlightening

The symptoms of CPP and posterior fornix syndromes are common affecting approx 20% of Caucasian women of all ages with or without significant urogenital prolapse.

The review paper explores a historical synopsis of knowledge about the anatomy of the female pelvis and its ligaments in the archives of gynaecology and obstetrics in the German literature. In 1938 Martius postulated that CPPS albeit PVFS was due to the pathophysiology of ligamentus supporting structures in the pelvis- the earliest records date back to Verheyn in 1708 and in 1862 Hytel described the antro-lateral, posterior lateral and posterior supporting ligaments supporting the uterus, bladder and rectum.

In 1993 (60 years later) Petros independently and intuitively postulated the same paradigm but went on to scientifically prove this theory and named it the Integral Theory as the causation of CPPS PVFS. The pathophysiology of fibro muscular vascular and neurogenic nature of the USL and CL which histologically and applied anatomy and physiology of these structures can explain visceral dysfunction and pain. These lax and damaged ligaments cause:

- overstretching of the fibro muscular components histologically being more than 50% of the structure of these ligaments, causing fasciculation, as described by Gordon in the 1960's. The "colic" which then causes tension on visceral muscle fibres, mylenated and non- mylenated nerves involving Frankenhauser

plexus associated with hyper stimulation of pelvic floor resulting in somatic nerve pain.
- Striated muscle N receptors in the levator plate the vascular component is also important and resulting in associated pelvic congestion syndrome of varicosities in pelvic ligaments - sacral vein plexus as well as broad ligament & infundibulo-pelvic ligaments

Goeschen's review paper very eruditely covers the history of the intuitive and scientific discovery of the Integral Theory of Peter Petros. This is presented with clinically astute algorithms of the symptoms correlating this with pelvic floor dysfunction and pain expounded by Peter Petros' application of the Integral Theory.

In my experience our initial patient cohort of 44 patient with multiple comorbidities which basically had precluded them from being offered any active management in our public hospital setting were offered TFS neoligament surgeries for their pelvic floor distressing symptoms.

- 20% of this cohort had CPPS & PVFS (in conjunction with other associated pelvic compartment symptoms
- At 12 months data the cure rate of PVFS & CPPS was 80-85% and 3-4 year data cure rate of 72-75%
- Our experience now has been over 900 TFS neoligament prosthetic tape insertions – all done transvaginally, experitoneal and through keyhole transvaginal incisions of 4cms or less for treatment of pelvic floor reconstruction ie urogenital prolapse and visceral dysfunction with or without CPPS and PVFS

These outcomes are very gratifying for patients with very low morbidity and excellent rapid recovery.

The paradigm shift is the concept that pathophysiological (PVFS) neurogenic dysfunction (CPPS) is inherent in the ligament support of the female pelvis. If these ligaments are supported with the TFS technology, accurately placed and tensioned, that visceral function be well restored and neurogenic pain syndrome improved, often with complete resolution of the 20% or more of patients presenting to our clinics with chronic pain syndrome.

Dr. Max Haverfield, MB BS FRCOG FRANZCOG
Melbourne, Australia

* * *

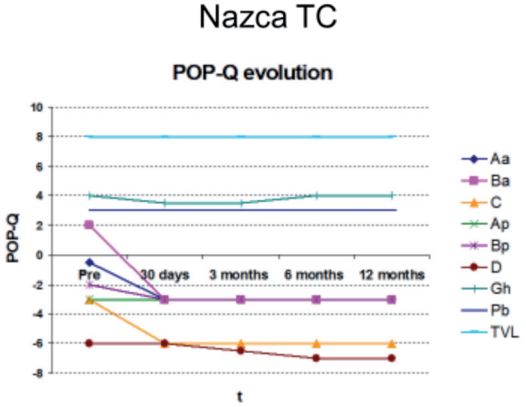
In my view Goeschen's paper is an important one that encourages the topographic diagnosis of chronic pelvic pain, instead of sindromic diagonosis and this is relevant.

The author should be congratulated for the extensive review of the literature including paper in German.

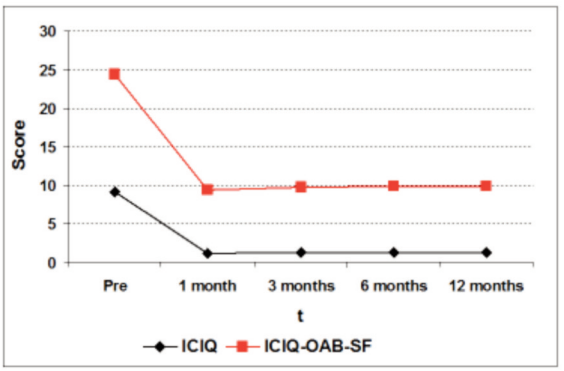
Demographics

n	mean	SD
104	61,1	11,0
102	4,3	2,8
102	3,5	2,4
102	0,4	0,9
82	35,1	35,9
87	22,8	9,8

Nazca TC



Nazca TC



The nerve fibres in the uterosacral ligaments were classified as parasympathetic visceral fibres. In his opinion, the visceral innervation incorporating fibres from T12-L1 provides an adequate explanation for pain distribution in the lower abdomen, specifically in the area of the ilioinguinal nerve (Figure 5). He hypothesized that stretching of weakened and loose uterosacral ligaments by gravity may stimulate the nerve endings within these tissues to cause pain.

The fiber T12-L1 are sympathetic and not parasympathetic.

The ilioinguinal nerve is somatic.

As Petros states in his book, all is endopelvic fascia and all the structures are interconnected. I think this comment should be added.

Let me add some of our results with the Nazca POP Repair System. This is a POP (pelvic organ prolapse) repair system, consist of a polypropylene type 1 mesh implant and a kit of needles, for anatomic and functional repair of the pelvic floor.

Professor Paolo Palma MD PhD, Titular Professor
Dept. of Urology, University of Campinas, Brasil

* * *

The article of Prof. Goeschen is analytical brilliant in bringing at least two breaking fundamental differences to all article written until now:

1. It shows that some physicians knew BEFORE 1960 much more about pelvic pain than our contemporary colleagues!

2. It shakes the fundament of the surgical treatment of the pelvic floor on abdominal/laparoscopic way

3. The way of pain transmission through the Frankenhaeuser plexus is the only way to explain why patients have pains BEFORE and AFTER a laparoscopic promontofixation: this technique only anchors the upper vagina/uterus, but doesn´t repair the enterocoele, so that the pain remains caused by pressure on Douglas and weak sacrouterin ligaments.

I can fully agree all of the points of view in this article.

The posterior fornix syndrome (PFS) is the main target of my activity since 2008 as pelvic floor surgeon and sustainer of the Integral Theory. In Sept 2014 I have communicated my experience about 492 cases of PFS in Muenich at the International Congress of the ISPP and defended this study a month later at the University for Medicine of Bucarest/Romania and became my PhD Graduation with „Magna cum laudae" about that (Title : „The posterior fornix syndrome: a new urogynecological entity. Physiopatology and suggestion for surgical cure")

The surgical cure of the posterior compartment and the level I brings evidently a dramatic improvement of the symptoms of the PFS up to 98%!

Improvement of:
defecation 97,84%
bladder emptying 94,24%
nocturia 88,06%
frequency/urgency 73,58%
fecal leakage/non-sphincterian incontinence 65,45%
pelvic pain and improvement of the intercourse 62,56%
are exemplary mentioned in my study.

Complications are rare. Intraoperative: hematoma 3,32% rectal lesion 0,88% and short post-op abscess 0,22%

Post-operative after 3 months: granulation polyps 3,54% erosions 1,55% wound dehiscence 1,55% recto-vaginal fistula 0,22% (only one in 452 cases.

My conclusions about the experience got in the treatment of 492 patients with PFS are:
-appears between 40-80 years, BMI non-specific
-usually after 1-3 deliveries
-50% post-hysterectomy, 50% with intact uterus
-minor prolapse may cause major symptoms!
-vaginal hysterectomy seems to favorize PFS (versus abdominal/laparoscopic hysterectomy)
-supracervical hysterectomy gives no protection against PFS
These conclusions induce some important aspects:
The clinical/ ultrasonographic examination must detect EVEN THE MINIMAL ANATOMICAL FAILURE in order to give a realistic chance to cure the symptoms.
The transvaginal surgery is more challenging versus abdominal/laparoscopic/robotic surgery, because operating in virtual spaces and need of experience, but is the only way of THE ANATOMIC RECONSTRUCTION. It maintains the elasticity and axis of the vagina and through that, the normal function of the pelvic organs.

Dr Andri Muller-Funogea MD PhD
Dusseldorf, Germany

* * *

I congratulate Professor Goeschen on a comprehensive work. I can confirm his views in a very practical way. I have performed TFS surgery on approximately 200 patients, inserting more than 400 tapes mainly in patients with POP. I have found that the Integral Theory Questionnaire and the pictorial algorithm are essential practical aids to decide which ligaments need repair.
The cure rate for CPP and other posterior fornix syndrome symptoms such as nocturia in our clinic has been well above 80% and the cure of POP>90%.
Out of the 400 tapes, there was only one tape erosion, in the anterior compartment. It resolved with E2 cream. No anchors migrated and none required removal. Some patients had post-operative pain such as buttock pain for short periods, but none had ongoing pain beyond a few weeks.

Dr Peter Ashton, MB BS FRCOG FRANZCOG
Melbourne, Australia

* * *

In my 2 years data (N=66) of TFS for POP, there were 30 patients who complained of low abodominal discomfort preoperatively.
And their points of P-QOL Q18 (vaginal discomfort and back pain) improved statistical signficantly from 1.82±1.04 to 1.04±0.39 following the TFS uterosacral (USL) and cardinal operations.($p<0.01$)
Therefore we can treat CPPS surgically by TFS for POP, especially USL repair.
It is important to emphasize that CPPS can also be treated non-surgically.
We are doing pelvic floor rehabilitation (PFR) according to Integral Theory as described in Chapter 5 of the textbook "The Female Pelvic Floor", 3rd edition, Springer 2010. The patients who complained of pelvic pain and coital pain are initially treated by Integral Theory based PFR exercises which emphasize strengthening of the posterior muscles and ligaments of the pelvic floor.
Our PFR data included seventeen patients who complained pelvic pain and coital pain.
Their Visual analogue scale and sexual pain point of FSFI (female sexual function index) were improved statistically significantly from 93mm to 41mm and from 12 to 35 .
($p<0.05$).

Professor Yuki Sekiguchi MD PhD
Yokohama, Japan

Original article

Mapping chronic urogenital pain in women: review and rationale for a muscle assessment protocol - Part 1

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INTRODUCTION

Chronic urogenital pain (CUP) is a prevalent, yet complex pain phenomenon, affecting women of all ages. CUP is difficult to understand because it exists where there is no visible pathology, continues long after tissue irritation or damage, and persists well beyond the expected time of healing. In simple terms, CUP occurs where there is little, if any, obvious reason for it to exist. CUP syndromes encompass a wide range of pain sites and symptoms, which affect the functioning of the urinary and reproductive systems. Women diagnosed with CUP report symptoms of urge, frequency, suprapubic pressure, nocturia, dyspareunia, abdominal and lower back pain and a range of other comorbidities. The pervasive nature of CUP can be debilitating, interfering with daily activities, relationships, vocational capacity and reproductive potential. Given the high prevalence of these pain disorders,[1,2] it is surprising that even what is considered as a state-of-the-art response in relation to CUP management, is deemed unsatisfactory by clinicians and sufferers.[3]

Women diagnosed with CUP, invariably consult multiple specialists, present with lists of inconclusive investigations, and often have a long history of unsuccessful attempts at treatment. This reflects a notorious lack of response to antibiotics, antifungals, corticosteroids, hormone therapies, antidepressants, and less frequently surgeries. These ineffective management strategies tend to protract suffering and are disparaging to both patient and clinician, not to mention costly to the individual and the health care system. In the last 30 years, vast resources have been expended on the study of CUP and the growing body of scientific literature attests to the complexity and impact these disorders have on women. Yet, one of the key, but elusive tasks, is the need to establish the cause and source of pain.

Reviewing the current management guidelines formulated by various peak associations,[4-6] pain of pelvic muscle origin, though mentioned in literature, has been long overlooked as a potential cause of CUP symptoms.[7] Over the last thirty years evidence linking dysfunctional pelvic muscles with CUP syndromes has increased significantly.[8] Each of the peak associations endorse pelvic muscle assessment as part of diagnostic screening, and recommend muscle related therapies as frontline interventions in the management of CUP.

The authors of this two part series contend that there is an evident need for developing a standardized assessment protocol based on the link between CUP and pain of pelvic muscle origin. The first article introduces the *integrated mapping and assessment protocol* (IMAP) and establishes the rationale for pain mapping in the context of two CUP syndromes, vulvodynia and bladder pain syndrome/interstitial cystitis (BPS/IC). The second article introduces the clinical use of the IMAP in CUP, illustrating its benefits. Together these two articles provide new insights into the origins and mechanisms of pain, thereby expanding the evidence base for improved management of CUP.

Pain Mapping and CUP Syndromes

Pain mapping, sometimes referred to as the cartography or iconography of pain, is an assessment method for identifying the origins of pain. The history of pain mapping is not extensive but its evolving use is encouraging and relevant to CUP.[9]

In recent times Travell and Simons enriched the understanding of pain through their extensive compilation of pain maps linking the origins of pain with body regions to which pain was referred.[10] The mechanisms of pain, proposed by Travel and Simons, focused on changes in myofascial structures that gave rise to trigger points (TrPs) within muscles, ligaments and fascia. TrPs were identified as tender and irritable points within muscle spindles responsible for tension regulation. TrPs have been shown to be sympathetically innervated and the mechanisms by which emotions affect muscle tension were subsequently identified.[11,12] TrPs can be either active, producing constant pain, or latent, leading to referred pain only when provoked by pressure or the activation of muscles. Because TrPs are often latent, pain of myofascial origin can remain unidentified, unless muscles are examined and palpated. It is important to remember that TrPs, whether active or latent, not only refer pain but also compromise the function of muscles and cause autonomic reactions within proximal regions.[10]

The potential contribution of pain mapping to the understanding of CUP arises from its systematic approach to localising the source of pain and ascertaining its quantitative and qualitative characteristics. Theoretically, certain fundamental features of chronic pain can compound any attempts to localise it. Given that pain is a sensory and emotional experience, it can be elusive, and pain pathways difficult to identify. The modulating impact of mental and emotional states affects any attempts to objectively assess pain severity, and the impact of chronic pain syndromes on sufferers.[13] Currently there are no qualitative or quantitative measures available to logically establish the location, intensity and impact of CUP.[14]

Most CUP disorders trace their onset to noxious triggers (acute tissue trauma, infections and inflammatory states), which are known to initiate a cascade of nerve and immune reactions, as well as reflex muscle responses. When these reactions continue beyond the acute phase, they give rise to what ultimately characterises chronic pain syndromes; decreased pain thresholds, increased field of nociceptor reception, increased nociceptor responsiveness (allodynia), increased intensity of response (hyperalgesia), prolonged post stimulus sensations (hyperpathia) and the occurrence of unexplained spontaneous pain, affecting the overall functioning and wellbeing of the individual. Given the

known involvement of pelvic muscles in CUP syndromes, if pelvic muscles are the primary generators of persistent pain, they are easily accessible for assessment and pain mapping. Various functional assessments of pelvic muscles have been published,[15,16] but what is evidently lacking is a standardised pain assessment protocol that would assist with localising pain in the urogenital area. The IMAP has been developed to meet this specific clinical need, and its use and efficacy are illustrated in Part 2 of this series.

The benefits of pain mapping are manifold. In the context of CUP, pain mapping assists with:

- Localising the generators of pain;
- Linking the origins of pain with patient's symptoms;
- Quantifying the severity of pain;
- Ascertaining the sensory qualities of pain (i.e. burning, stabbing pain);
- Authenticating the patient's experience of pain by reproducing what the patient considers to be "their pain";
- Identifying the muscular origins of pain;
- Highlighting the role of peripheral mechanisms of pain;
- Guiding therapy based on the results of each individuals pain profile;
- Creating a practical and tangible pathway for resolution of pain; and
- Providing an objective means for evaluating the effectiveness of therapeutic interventions.

With these benefits, pain mapping facilitates a more pragmatic approach to the assessment and management of CUP. It goes beyond the common but basal question of 'where is the pain?' and addresses the more complex issue of 'where is the pain coming from?' By doing so, it directs the inquiry to the source of pain, and in particular focuses on its muscle origin. Unlike pain diaries and body forms, which are a standard feature of pain questionnaires,[17] pain mapping goes beyond phenotyping,[18] and operationalizes the task of chronic pain management.

Rationale for Muscle Pain Mapping in CUP Disorders

The management of chronic pain is most effective when the pathogenesis and mechanisms are clear and reasonably understood. Two study methodologies are often utilised in investigating pathophysiology; the first looks at the anatomical and functional changes associated with a disease, and the second examines treatments that are effective in halting the disease, enabling the identification of mechanisms by which interventions work. The outcomes of both methodologies are relevant to the study of CUP.

Traditionally, the end-organ specialist has been entrusted with excluding all medical causes of urogenital pain before making a diagnosis, explaining the patient's symptoms and offering treatment and a prognosis.[19] However, given that CUP symptoms are poorly localised and not only affect the bladder, urethra and vulvovaginal area, but also the colorectal, perineal, groin, thigh, suprapubic, abdominal and lower back areas, the absence of pathology led to certain assumptions which gave rise to the development of nomenclature and classification systems that relied almost exclusively on organ-based, symptomatic criteria. As a result bladder and vulvar symptoms were thought to originate from an undiagnosed disease or neurological disorder affecting the organs named in the pain syndrome (*bladder* pain syndrome, *urethral* syndrome, *vulvar vestibulitis* syndrome, etc.). This organ-based classification system, arising from a questionable assumption, gave rise to a further dilemma. Because pain was "…thought to stem from the kidney, bladder… pelvic organs or vulva," treatment was organ directed, until it became very evident that such postulations were erroneous and that "pain felt in these regions may not originate in the organs themselves…".[20] The misleading hypothesis that pain stemmed from the organ at the centre of the syndrome gave rise to a high frequency of failure in organ directed treatments, and confirmed the organ-based approach to be wrong, suggesting that the mechanisms being considered were also wrong.[20]

The organ based labelling of syndromes created the false impression that something was known about the underlying pathology. The terminology itself, gave rise to erroneous thinking and misguided, and at times dangerous therapies.[21] It was assumed that if "the disease" in the organ could be cured, or more radically the "diseased organ" removed, the pain would no longer exist. This proved not to be the case.[22,23] The removal of the presumptively "diseased" end-organs, by means of cystectomies, clitoridectomies, colectomies and hysterectomies, did more to "ingrain and accelerate these pain conditions than to relieve them".[24]

Many women currently attending pain clinics present with a history of antecedent pelvic surgery, having undergone major invasive procedures, including hysterectomies, sometimes at an early age, even in their early 20's. The present-day practice of multiple laparoscopies, ultrasounds, MRI's and CT scans produces little evidence of disease or anatomical abnormalities that explain CUP.[25]

Another extreme and equally erroneous assumption gave rise to the belief that the absence of pathology was indicative of underlying psychopathology. The reality of patients pain came into question.[26] Some medical texts suggested that CUP syndromes represented the end-stage of repressed emotional disturbances. However, even allowing for the questionable possibility of a psychiatric aetiology, surgical treatments were advocated for chronic pain syndromes "most of which worsened the illness".[24]

With the undue focus on a range of erroneous assumptions about the causes of chronic pain, the functional role of the musculoskeletal system in CUP was consistently overlooked. How this may have come about is not very clear. However, given that the three major systems of the human body that converge in the pelvic cavity (the reproductive, urinary and digestive systems), are traditionally managed by end-organ specialists (the gynaecologist, urologist and gastroenterologist), in ruling out potential causes of pain, they do not routinely evaluate muscles and muscle dysfunction as a possible source of CUP.[27] Despite evidence that links dysfunctional pelvic muscles with CUP, and the established benefits of muscle oriented therapies,[8, 28, 29] the muscular system as a cause of pain was, and continues to be, systematically overlooked.[7] Yet, dysfunctional pelvic muscles can be both a cause of pain and potentially impact all of the organ systems that converge in the pelvis, providing a common base for a range of comorbidities seen in CUP.

Muscles, both external and internal, together with the supporting soft tissue are integral to the proper function of the pelvis and organ related systems.[30] The bladder and the urethra are themselves muscular structures that are closely integrated with pelvic muscles and fascia, which anchor them within bony pelvis. The urethra is fused to the anterior vagina, and the endopelvic fascia attaches the bladder, urethra and vagina to the pelvic bones through ligaments and muscles that support them in a hammock like fashion.[31,32] During contraction of the levator ani muscle the pelvic organs lift and move anteriorly, and during relaxation they descend into a resting position. Failure to tighten muscles can result in loss of continence and sexual response, while failure to relax can lead to pain. When the postural and pelvic muscles are stressed, traumatized, weak or imbalanced they become a potential source of pain.[33] As a result, the major association guidelines, including those

of the European Association of Urology (EAU), American Urology Association (AUA), the International Continence Society (ICS), and the International Society for the Study of Vulvovaginal Diseases (ISSVD), advocate muscle assessments and the use of myofascial therapies as frontline interventions.[4,5,34,35] Given the need for evidence-based and mechanisms-oriented approaches to the classification and management of chronic pain,[36] pain mapping accords with the current paradigm shift.

From the spectrum of CUP conditions, the two most frequently discussed are vulvodynia and BPS. A brief review of current literature on vulvodynia and BPS is provided, and the role of myofascial variables highlighted.

Vulvodynia. The term *vulvodynia* is a descriptive term for unexplained pain in the vulvar area. Since vulvodynia is considered to be one of the chronic urogenital pain syndromes, the differential diagnoses of urethral syndrome and BPS should be considered given that the pathophysiology and prevalence is similar.[34] Anatomically the vulvar area includes the external portion of the female reproductive organs; the vestibule, hymen, urethral opening, ducts of the of the minor and major vestibular glands, labia minora and majora, the clitoris, mons pubis and the perineum.[37] The ISSVD defines vulvodynia as "vulvar discomfort, most often described as burning pain, occurring in the absence of relevant visible findings or specific, clinically identifiable, neurologic disorder".[38] The definition identifies the location of the pain, its sensory qualities and its unknown, but potentially multi-factorial nature. The quality of pain is most often reported as burning, rawness, itching, or stabbing, and the intensity of pain is rated by the majority of patients as severe.[39]

The reported prevalence of vulvodynia has varied between 4 and 16 per cent. Even though the ISSVD has provided a clear definition of vulvodynia, different methodologies of data collecting, and underreporting of the condition make its true prevalence difficult to establish.[2,34, 40-42] A large study of a cohort of vulvodynia patients showed that the prevalence peaks at age 24, and even though it affects women of all ages, it is a disorder primarily impacting young women, most of whom are in their prime reproductive years and are seeking to enter long-term relationships.[2]

The aetiology, diagnosis and management of vulvodynia have not been clearly delineated.[34] It is suggested that multiple pathologies may be involved including immune system up-regulation (increased mast cells), proliferation of local pain fibers (increased peripheral sensitivity) and contraction of the levator ani muscle in response to pain (increased pelvic muscle tone). Assessment predominantly consists of self-report questionnaires and quality of life measures. Clinical evaluations range between medical, psychological and sexual assessments. The treatment of vulvodynia is predominantly focused on prescribed medications in the absence of defined pathology. As a result, management strategies are usually ineffective.[34,43]

The most common diagnostic test is the cotton swab test, sometimes referred to as the Kaufman Q-tip touch test.[44] This swab test is used to localise the pain within the vulva area.[45, 46] The test starts in the thighs and progresses medially to the vestibule. In the vestibule testing follows the perineal clock starting at 2, going onto 4, 6, 8 and 10 o'clock positions and, if pain is present, the patient is asked to quantify the pain as mild, moderate, or severe. The Q-tip test was originally used by Friedrich (1988), who, in a landmark paper, proposed three diagnostic criteria for vestibulodynia, a localised form of vulvodynia (referred to in his paper as vulvar vestibulitis syndrome).[47] The criteria included (i) severe pain on vestibular touch or attempted entry, (ii) tenderness to Q-tip pressure localised within the vulvar vestibule, and (iii) physical findings confined to vestibular erythema of various degrees. The third criterion proved to be unreliable, but patient's self-reporting of pressure-related-pain, and pain with Q-tip test, continue to be widely used in the diagnosis of vulvodynia. From the perspective of pain mapping, two pertinent points arise in relation to vulvodynia; the diagnostic reliability of sensitivity to pressure, and variation in tenderness, as demonstrated by Friedrich's initial pain mapping of the vestibular area.

The consensus statement on definition, diagnosis and management of vulvodynia[34] recommended that in addition to medical tests, a careful urogenital examination should be performed which includes pain mapping and a functional assessment of pelvic muscles. It specifically stated that a thorough pain map should include the vestibule, perineal area and thighs. Furthermore, the pelvic muscles were to be routinely examined with bilateral palpation of the levator ani to assess the potential contribution of referred myofascial pain. Then, the lower third of the anterior vaginal wall was to be examined as part of the assessment to establish any associations with bladder-related comorbidities, which were thought to affect a third of vulvodynia patients.[34] Given the extensive literature linking dysfunctional pelvic muscles with CUP, very few clinicians have followed these recommendation and little has been mentioned about pain mapping with vulvodynia patients. The IMAP addresses each of these points in the mapping of CUP.

Several studies have compared general pain sensitivity between vulvodynia sufferers and asymptomatic controls. A study on quantitative sensory testing used pressure ranging from 0g to 1500g while assessing pain thresholds of the vulva in 23 defined locations.[48] Pain thresholds were significantly lower for vulvodynia cases than controls at all 23 sites tested, with no significant differences between pressure sensitivity at analogous right and left locations. In a study utilising EMG biofeedback and manual therapy, assessment went beyond the Q-tip test of the vestibule, and included appraisal of the role of pelvic muscle function in the aetiology and maintenance of vestibulodynia symptoms.[49] In the vestibulodynia group, muscles were found to be hypertonic but weak and inelastic, thus restricting the vaginal opening. Muscle tension was attributed to pain related guarding, with the suggestion that tension holding created increased pressure at the level of the vestibule and was responsible for perpetuating and exacerbating pain in vestibulodynia. A significant finding of the study was that 90% of women reporting pain with intercourse demonstrated pelvic floor muscle dysfunction. The researchers concluded that regardless of what the primary or adjunct therapy may involve, pelvic floor pathology must be assessed, as it can exacerbate and maintain the pain.

In studies examining the inter-relationship between vulvodynia and BPS/IC, pelvic muscle dysfunction was identified in 87% of BPS/IC cases, 60% of whom also presented with vulvodynia.[50] In a further analysis comparing BPS/IC with vulvodynia, and BPS/IC without vulvodynia, levator ani pain levels were significantly higher in the group with vulvodynia[51] Ratings of levator ani pain were derived from internal palpation of the muscle, and scored on a 10-point Visual Analogue Scale (VAS). Vulvar pain was assessed by palpating the vestibule at the 1, 3, 5, 6, 7, 9, 11 and 12 o'-clock positions. From this comparative analysis, it was evident that the two CUP syndromes, BPS/IC and vulvodynia, appear to be very closely related, and muscle tenderness was a significant feature in both. The connection between these two syndromes is further highlighted by findings that the majority of vulvodynia patients have a positive potassi-

um sensitivity test (a standard test for BPS/IC) and that vulvodynia pain may be referred from the urinary bladder.

A pilot study examining patients diagnosed with clitorodynia (a localized form of vulvodynia) found that palpation of muscles along the paraurethral area reproduced clitoral pain. It was also noted that in some patients, points in the paraurethral area reproduced symptoms of arousal, and in some instances clitoral tumescence, in a subgroup of patients suffering from persistent genital arousal disorder.[53] This study identified the pelvic muscles and paraurethral soft tissue as involved in generating symptoms and pain.

Another study looked at tactile pain sensations using functional magnetic resonance imaging (fMRI). It found augmented genital sensory processing in patients with vestibulodynia.[54] Another found increased sensitivity to touch and pain in the vestibule as a result of physiological arousal.[55] Overall, the evidence points to a generalized sensory abnormality, compounded by increased catastrophizing, hypervigilance, and fear of intercourse and non-intercourse pain.[48, 56, 57]

There are very few studies evaluating the effectiveness of therapy in the management of vulvodynia. A number of electromyographic (EMG) studies confirmed abnormal muscle tone in vulvodynia patients, marked by elevated resting baselines, poor muscle strength, and poor recruitment and recovery.[16] The dysfunctional state of the muscles was significant enough to be of diagnostic value, differentiating between controls, vulvodynia patients and women suffering from incontinence.[58] In studies specifically looking at management of vulvodynia using EMG biofeedback, therapy focused on down-training of hypertonic muscles which resulted in an 83% reduction in symptoms.[58] Very few studies exist reporting benefits of manual therapies in the management of vulvodynia. In studies where dysfunctional pelvic muscles, characterised by high muscle tone, instability, poor contraction and recovery, and tenderness to pressure were noted, soft tissue mobilization and myofascial release techniques produced complete resolution of symptoms or significant improvement.[41, 59]

In summary there is significant evidence linking increased PFM hypertonicity with hyperalgesia and chronic pain. Dysfunctional and tender PFM may be the source and cause of pain, and the vulvar and bladder areas may simply be "innocent bystanders".[60] The extent to which pain of PFM origin gives rise to central sensitization needs to be examined and studied further. It follows that if peripheral mechanisms can give rise to peripheral and central sensitization, reversing the process through the effective rehabilitation of pelvic muscles may be key to reversing the sensitization process and to the management of CUP.

Current literature discusses assessment techniques for evaluating functional pelvic muscle tone but provides no tools for the assessment of pain of pelvic muscle origin.[15] This is a significant oversight which compromises potential outcomes.[61]

Bladder Pain Syndrome. Concepts of bladder pain have varied over time, with different criteria and terminology used to describe pain and associated voiding symptoms.[62] The term Painful Bladder Syndrome/Interstitial Cystitis (PBS/IC) was introduced in 2004 at the inaugural meeting of the Multinational Interstitial Cystitis Association meeting in Rome. Within two years, at the biannual IC conference, PBS was redesignated as BPS to highlight the involvement of the end organ and the neuro-visceral (myopathic) mechanisms.[62] IC came to be considered a subgroup of the syndrome, presenting with a identifiable pathology. The term IC is being phased out, leaving BPS as the label describing chronic pain thought to arise from the bladder.

BPS is defined as "an unpleasant sensation (pain, pressure, discomfort) perceived to be related to the urinary bladder, associated with lower urinary tract symptoms of more than six weeks duration, in the absence of infection or other identifiable causes".[63] The reported prevalence of BPS ranges from 5-16%.[1] Its true prevalence is difficult to establish due to ongoing disparity of definitions, classification, methods of data collection and underreporting.[1,64] Patients present with symptoms of urge, frequency, nocturia, suprapubic pressure, and pelvic, abdominal and back pain, as well as other common comorbidities.[3,64] Though the pain involves the bladder and multiple other body sites in the same patient, it is attributable to a single disorder.[65] The severity of symptoms can vary between patients and can fluctuate widely in the same patient but the quality of life is significantly compromised among all sufferers.[66]

Patients presenting with BPS are often diagnosed on the basis of clinical symptoms, diagnostic tests, such as the potassium sensitivity test, cystoscopy with hydrodistention, and validated questionnaires. Theoretical models hypothesising the pathophysiology of BPS abound, although some clarity is emerging in relation to four potential primary processes. These have been identified as (i) GAG/proteoglycan layer disruption, (ii) immune & inflammatory system up-regulation, (iii) neurological sensitization (central and peripheral), and (iv) pelvic floor dysfunction (PFD).[67]

The link between bladder pain and pelvic muscle dysfunction has been well established.[8,23,64] One of the early studies of BPS reported pain upon digital examination of the levator ani, the sacrum, and anal region. Subsequently, different methods of treatment were compared among participants, including bladder distension, total cystectomy and ultrasound treatment of the perineal area. Of 4 cases treated with total cystectomy, pain continued to persist in 2 of the patients. The authors concluded that pain was partly bladder induced and partly caused by the painful levator muscle. Based on digital examination, the diagnosis of levator ani spasm syndrome was made.[23] However, in a recent pilot study which examined pelvic muscle tenderness and sensitivity in the paraurethral area, the symptoms of bladder pain and urge were fully reproduced in 100% of the cases upon palpation.[68] Urge to void and bladder pain were localized and shown to originate from to the paraurethral area.

Studies of urodynamics and neurostimulation also showed an association between pelvic muscles and symptoms of BPS.[64,69,70] The location of pain varied but included suprapubic, perineal, rectal, and genital pain, and in some cases was reported as radiating to thighs, ankles, and feet. The quality of pain was described as burning, pressure and stabbing, with patients reporting voiding dysfunction, including urgency, frequency, incontinence, straining-to-void, hesitancy, and urinary retention.[64] The functional inability of patients to exercise effective voluntary control over pelvic muscles was a key feature of long standing duration. The authors stated that, "pain and soreness, even inflammation, will develop if the muscle system is functionally abused," suggesting that changes within the bladder can be explained by changes in pelvic muscle function. It was noted that relaxation of pelvic muscles, whether through stimulation or biofeedback-assisted retraining, "can produce immediate relief in the muscle soreness.".[64] The authors recommend that "urologists should be encouraged to deal with pain on as conservative a level as possible" and that "if pelvic muscle dysfunction is not corrected, then the chances are against therapy being successful even with the help of medication".[64]

In a landmark study evaluating the impact of manual therapy on urgency-frequency syndrome, 42 patients who had previously undergone ineffective treatment were examined. Ineffective treatments included antibiotics (55%), urethral dilation (50%), anticholinergics (30%), diazepam (22%), tricyclic antidepressants (15%), α-blockers (12.5%), phenazopyridine hydrochloride (10%), acupuncture (10%) and surgery (5%). Each patient then participated in myofascial treatment and biofeedback-assisted retraining (8). Initial assessment consisted of intravaginal and intrarectal examination of the urinary and anal sphincter, and pelvic muscles in order to identify tightness, tenderness and pain that duplicated symptoms. Treatment consisted of compressing, stretching, strumming of internal muscles, and stretching of external muscles, with heat application to facilitate greater muscle relaxation. In women presenting with tenderness of the urinary sphincter, paraurethral tissue was repeatedly compressed against the pubic bone using increasing pressure. With repeated compressions a reduction in tenderness, softening and thinning of the contracted tissue and decreased sensitivity was noted. Trigger points that were resistant to therapy after 6-8 weeks of treatment underwent trigger point lidocaine injections. As a result of manual therapy, patients with urgency-frequency syndrome experienced an 83% improvement rated moderate to marked.

The study concluded that manual therapy, muscle relaxation techniques using biofeedback, bladder retraining and psychological therapy "arrests the neurogenic trigger leading to bladder changes, decreases central nervous system sensitivity and alleviates pain due to dysfunctional muscles".[8]

In a further study of 70 women with a diagnosis of bladder pain, on examination 87% had levator pain consistent with pelvic floor dysfunction.[50] Of these patients 71% reported dyspareunia, 50% irritable bowel syndrome (IBS) and 36% urge incontinence. Neurostimulation devices were present in 16% of the sample and this subgroup experienced significantly higher levels of pain in the levator ani muscle upon palpation. Given that treatments directed only at the bladder produced disappointing outcomes, the researchers assessed all of the pelvic floor muscles to identify levels of tenderness and pain. Pain from individual muscles was evaluated using the VAS, and pressure applied to pelvic floor muscles elicited pain in the supra-pubic area, perineum, rectum and labia.

Consistent with previous studies which identified 85% of patients as suffering from myofascial pain and hypertonic pelvic floor dysfunction,[8,64] the authors conclude that "the pelvic floor may be a significant source of pain in women with IC, making therapy directed only at the bladder less effective".[50] The authors recommended that muscle directed therapies might provide a new direction for management of BPS/IC in women.

Another study identified myofascial pain in 78.5% of patients with 67.9% having 6 or more separately identifiable trigger points.[71] The most common locations were the obturator internus, puborectalis, arcus tendinineus, and iliococcygeus muscle. The average pain scores for trigger points ranged from 5.2 - 6.8 on a ten-point scale. Suprapubic, back, anal, and vulvar pain were correlated with myofascial trigger points, with the strongest association noted with left sided myofascial trigger points. Urethral pain only correlated with left puborectalis trigger point scores. Patients with a longer duration of BPS had overall higher myofascial trigger point scores. The authors conclude that performing a musculoskeletal examination allows for proper identification of pain, which can be utilised to direct therapy with patients.

In a randomized multicentre clinical trial of physical therapy in women with BPS, those presenting with pelvic floor tenderness during vaginal examination were assigned to either a global therapeutic massage or pelvic floor myofascial physical therapy.[29] Of a total of 81 subjects, 59% reported moderate or marked improvement in the pelvic floor myofascial physical therapy group, while only 26% reported improvement in the global therapeutic massage group. It was noted that 43% in the global therapeutic massage group and 18% in the myofascial therapy group, reported no improvements as a result of treatment. Both treatment groups showed benefits in secondary outcomes of pain, urgency, frequency and quality of life. The authors conclude that myofascial therapy may be beneficial in the management of BPS and pelvic floor tenderness.

The studies on BPS focused primarily on outcomes of various manual, muscle oriented therapies. From the comparative outcomes, some conclusions have been drawn regarding potential mechanisms. Though no causal relationships have been established as yet, the pivotal role of dysfunctional muscles in conditions such as BPS has been recognised.[24]

CONCLUSION

CUP, considered by some as the "black box" of medicine,[33] affects a large number of women with a reported lifetime prevalence of 5-16%. The etiology of CUP is considered to be multifactorial, but often unknown. With the mechanisms of pain poorly understood, the outcomes of current interventions have been notoriously ineffective. Lacking an evidence base, the more invasive therapeutic options potentially cause more physical and emotional harm than good.[24] With growing evidence highlighting the inherent connection between dysfunctional muscles and CUP, greater emphasis needs to be placed on peripheral mechanisms, and less on the end-organ as a cause of symptoms. Though muscle pain is often generalised and poorly localised it can be generated by myofascial changes which not only mimic visceral pain but can induce the pain.[24] Developing a standardized assessment protocol focusing on pain of pelvic muscle origin has become a necessity. The IMAP has been developed to assist with identifying the sources of pain and in doing so providing much needed insight into the mechanisms of CUP.

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INVITED COMMENT: Pelvic Pain Trigger Points

These manuscripts explore an area in desperate need of scientific clarification; pelvic pain disorders. Jantos and colleagues have shown the usefulness of careful pain mapping of the pelvic cavity for myofascial trigger points(TPs). For years my colleagues and I have tried to establish a model for chronic muscular pain by studying muscle structures and their function in chronic pain conditions. Some years ago we published a series of studies showing that myofascial TPs produced high levels of electromyographic activity, even when the adjacent muscle was silent. We further showed that this activity was responsive to psychological stress, was not blocked by cholinergic blockade, but was blocked by alpha sympathetic blockade, establishing a close link between emotions and pain. Through the systematic identification of TP's in the pelvic cavity these two papers demonstrate that not all TP's are equal. The results show that some areas such as the paraurethral region were previously underestimated and drive most of the variance in pain reporting. This type of work, when extended, should help to greatly improve treatment strategies by integrating mind/body techniques and physical modalities.

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Original article

Mapping chronic urogenital pain in women: insights into mechanisms and management of pain based on the IMAP Part 2

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Abstract: Objective: To introduce the *integrated mapping and assessment protocol* (IMAP), designed to systematically localise the origins of pain in chronic urogenital pain (CUP) syndromes. *Method:* A retrospective analysis of IMAP data, from 82 participants, comparing pain scores of two CUP subgroups; vulvodynia and bladder pain syndrome (BPS), with asymptomatic controls. A further analysis was carried out to assess the use of the IMAP as a tool for evaluating the impact of interventions. *Results:* The IMAP scores showed significant differences between CUP syndromes and asymptomatic controls. Pain scores from the two CUP subgroups confirmed that pain of pelvic muscle origin is characteristic of chronic pain conditions, and does not represent the normal state. The paraurethral area was shown to be the source of the highest pain scores, when compared with other maps. Analysis of pre and post intervention pain scores indicated a statistically significant reduction of pain scores, demonstrating the IMAP's sensitivity to intervention-related change. *Conclusion:* The IMAP is a promising tool in localizing the origins of pain, and providing insight into the role of peripheral pain mechanisms in CUP syndromes. Mapping will assist in the development of evidence-based interventions, and provides an objective means of evaluating their impact.

Keywords: Chronic urogenital pain (CUP); Vulvodynia; Bladder pain syndrome (BPS); Integrated mapping and assessment protocol (IMAP); Ischemia.

INTRODUCTION

The *integrated mapping and assessment protocol* (IMAP) was specifically developed for the mapping of chronic urogenital pain (CUP). CUP syndromes represent a group of functional pain disorders, which, in contrast to structurally related pain problems, lack well-defined pathophysiology and consequently are poorly understood. Mapping, whether in the mathematical, medical or geographical sense, is the process of delineating, linking, matching and establishing relationships between different sets of points, spaces, regions, themes, functions or symptoms. Scientists have mapped the brain, chromosomes, and peripheral nervous, circulatory and lymphatic systems but little work has been done on the mapping of pain disorders. This pilot study aims to systematically map the origins of CUP.

The science of mapping is referred to as cartography, or in the context of pain, as cartography of pain, and is commonly known as pain mapping.[1] In keeping with the current paradigm shift in the study of CUP, where greater emphasis is being placed on the need to understand the mechanisms of pain, innovative methods are required to develop evidence-based management strategies for disorders such as vulvodynia and bladder pain syndrome (BPS).

The IMAP as a tool is designed to localize the origins of pain, assess its severity, characteristics and spatial distribution. As a process, it engages the patient in delineating pathways that link the source of pain with "their" symptoms, and empowers and reassures them that their pain is not linked to sinister causes. Likewise, it enables the clinician to be discerning in the choice of suitable interventions. Intuitively, pain mapping, using the IMAP, shifts the focus to peripheral mechanisms involving dysfunctional muscle states as discussed in Part 1 of this series.[2]

Structurally and functionally the pelvic floor muscles (PFM) provide support to the pelvic organs; maintain urinary and faecal continence; enable sexual intercourse; facilitate parturition; provide postural support and assist with movement. The PFM form a musculoskeletal complex that is one of the most intricate in the human body,[3] consisting of a large number of individual muscles, numerous ligaments and fascia that hold in place all of the pelvic viscera. The pelvic muscles are organised in several layers which span the pelvic cavity, and form a horizontally oriented platform.[4] These layers of soft tissue are penetrated anteriorly by the urethra, centrally by the vagina and posteriorly by the anorectum, in an area known as the urogenital hiatus or pelvic outlet. Because the pelvic muscles relate to more than one organ system, their dysfunction can impact multiple systems at the same time, and each system can be a potential source of pain and symptoms. For purposes of pain mapping the PFM are accessible to digital and Q-tip palpation.[5] This form of assessment is in keeping with the guidelines for the study of vulvodynia, BPS and pelvic pain disorders.[6-9]

The meaningful interpretation of pain maps requires comparison with normative data from asymptomatic women, who have no history of lower urinary tract dysfunction. A recent study assessed pelvic muscle tenderness in nulliparous, asymptomatic women, 18-30 years of age.[10] The level of pelvic muscle tenderness was measured using the visual analogue scale (VAS). Using a cut off pain rating score of 3, pain ≤ 3 was considered clinically not significant, whereas pain scores >3, were deemed to be significant. The study found that in all of the 17 participants, there was no tenderness. Furthermore, none of the subjects had high-tone PFM. The study concluded that pain on palpation could not be considered a variation of normal. By contrast, the incidence of pelvic muscle tenderness in bladder pain patients was reported to be as high as 94%, with the majority showing dysfunctional, hypertonic pelvic muscles.[11] These studies show that in asymptomatic, nulliparous women, without lower urogenital tract symptoms, the pelvic muscles should be painless and of normal tone, and that a pain score > 3 should be considered as an uncommon finding.[10]

There is general agreement that pain, commonly attributed to painful end-organs such as the bladder, bowel and external genitalia, arises from high-tone pelvic muscles.[2] Furthermore, that dysfunctional pelvic muscles account for

urinary urgency, constipation, dyspareunia, and neurogenic inflammation of the bladder.[12] These conclusions are consistent across studies that have integrated muscle assessment into their research designs.[2]

While the role of dysfunctional pelvic muscles in CUP symptoms is acknowledged and better understood, there are no detailed pain maps localizing the origins of symptoms and pain. Earlier reports demonstrated a high degree of specificity with which certain symptoms can be linked to focal areas of pelvic anatomy.[13, 14] This study, using the IMAP, undertook a comprehensive analysis of CUP mapping of the external urogenital area, internal pelvic muscles, and the paraurethral/bladder region with the aim of localizing the source of pain and symptoms.

METHOD

This study is based on a retrospective review of pain maps from 82 patients who were assessed by the authors. Patient data was obtained from two study centres, a CUP clinic in Adelaide, South Australia, and the Terpa Clinic in Lublin, Poland, in collaboration with the Medical University of Lublin.

The inclusion criteria for the CUP group consisted of consecutive patients diagnosed with CUP, aged 18-60, who attended urogenital pain consultations between January and December 2014. The exclusion criteria were: concurrent illness with overlapping symptoms; history of pelvic reconstructive surgery, and any pelvic surgical procedures, pregnancy, birth or lactation within the last 3 months. The control group consisted of 28 asymptomatic women who met the inclusion criteria, who were undergoing a regular gynaecological examination.

Of the 82 participants, only 70 were retained for the final analysis. A total of 9 CUP participants were excluded: 7 on account of coexisting infections and medical problems, and 2 due to prolapse. Of the remaining 45 CUP participants, 27 were diagnosed with vulvodynia and 18 with BPS. Of the 28 participants in the control group, 25 were retained for the final analysis. Two were excluded on account of lower urinary tract symptoms disclosed during assessment, and one on account of severe pelvic muscle tension and pain during internal mapping. This case was of interest as she presented as asymptomatic, but reported adhering to a rigorous, daily pelvic muscle exercise program, which resulted in hypertonic and painful pelvic muscles, which were identified during mapping.

Pain mapping was performed in the lithotomy position on an empty bladder. For each palpation point the patient was asked to provide three items of information; pain rating on a 0-10 numerical rating scale (NRS), pain characteristics, using up to three adjectives from a modified McGill Pain Questionnaire list;[15] and description of the spatial distribution of pain. Only pain scores were analyzed in this study; pain descriptors and spatial distribution will be reported in a separate study. A therapeutic intervention was performed on a subgroup of CUP participants (n=13) in order to evaluate the IMAP's ability to detect change in pain scores. Pain maps were completed prior to the commencement of therapy, and at its conclusion. The intervention consisted of electromyographic (EMG) assisted relaxation training, myofascial therapy, pelvic massage and desensitization of the paraurethral points.

The three pain maps were developed on the basis of clinical work, research studies and cadaver dissections. The cadaver studies specifically focused on the anatomical, physiological and functional relationships between the vulvar, urethra and bladder. While validating the three pain maps,

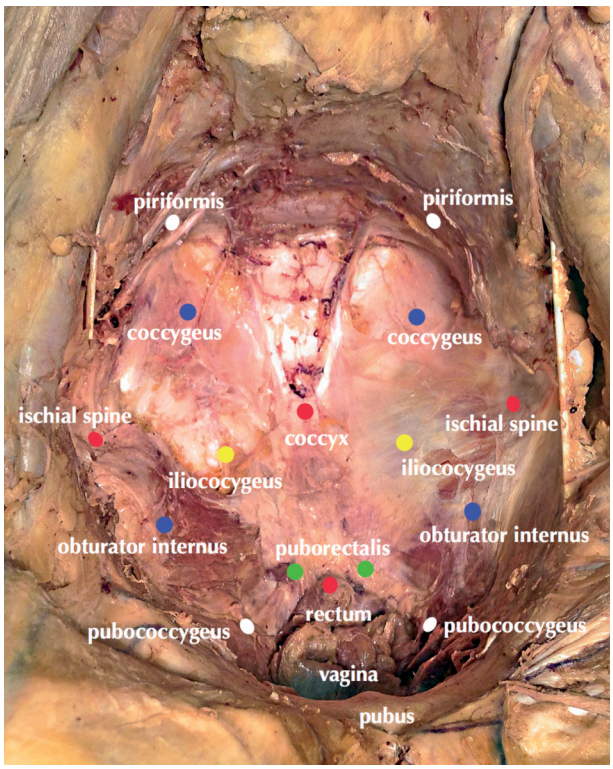

Figure 1a. – Cadaver image showing anatomical reference points (marked red), and internal pelvic muscle palpation sites (marked white).

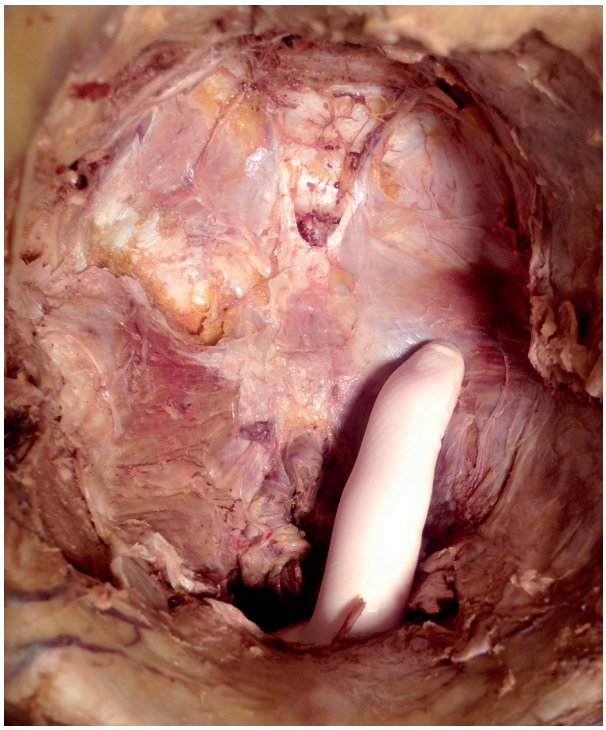

Figure 1b – Pelvic Floor Image of Cadaver - palpation.

reference and mapping points were identified for internal pelvic muscles, these are marked in Figure 1a, and palpation is demonstrated in Figure 1b.

Mapping the external urogenital area (Map 1). The first map focused on the external urogenital area as shown in Figure 2.

Muscles marked with a solid black dot were palpated digitally, while points marked with a white dot were mapped using a moistened Q-tip. The pressure used ranged

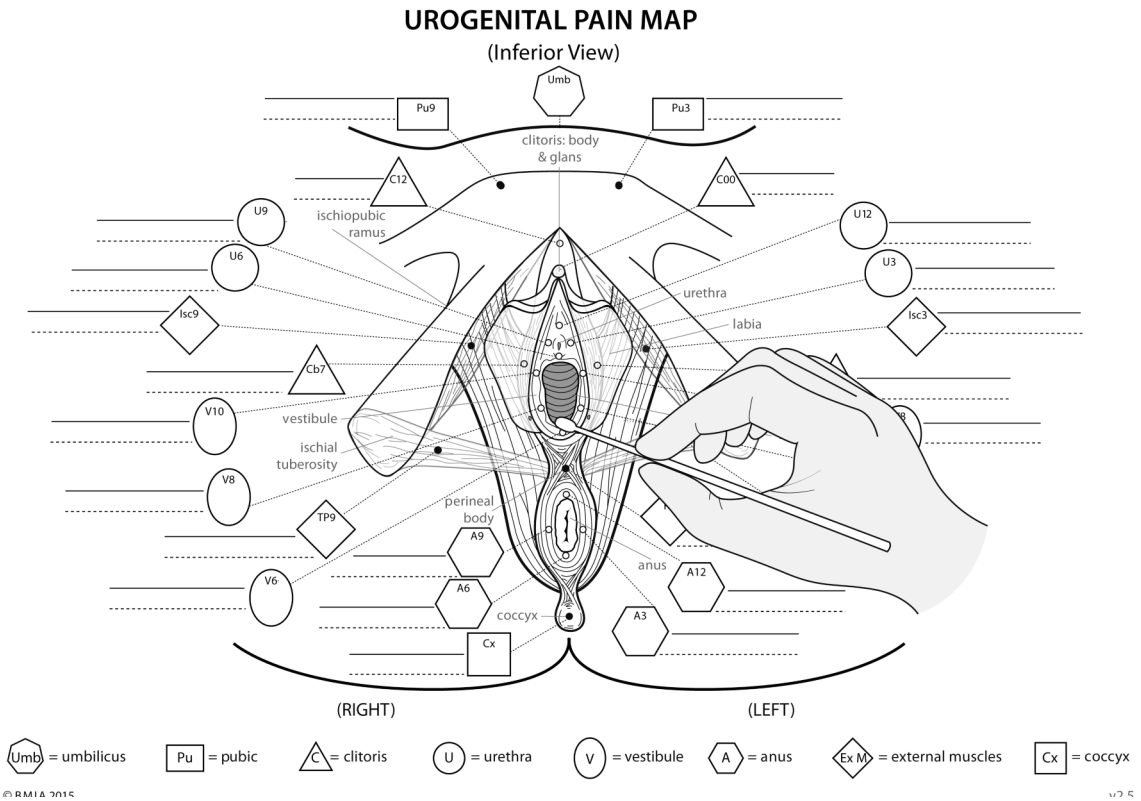

Figure 2. – Urogenital pain map (Map 1), identifying external points for pain mapping.

from 0.1 - 0.2 kg/cm² for Q-tip palpations, and 0.4 - 0.5 kg/cm² for digital palpation of superficial muscles as based on an earlier study.[16] Several devices have been tested by the authors, but were considered not conducive to the task of intravaginal pain mapping.[17,18] The clinician's skill and training in locating pelvic muscles and applying correct and consistent pressure are a requisite for quality mapping. The urogenital pain map labelled points around the clitoris, urethra, vestibule and anus on the basis of the perineal clock. However, the order of palpation did not follow the clock sequence in order to minimise the patient's anticipatory reactions.[19]

Mapping internal pelvic muscles (Map 2). The second map consisted of the palpation of internal pelvic muscles as shown in Figure 3.

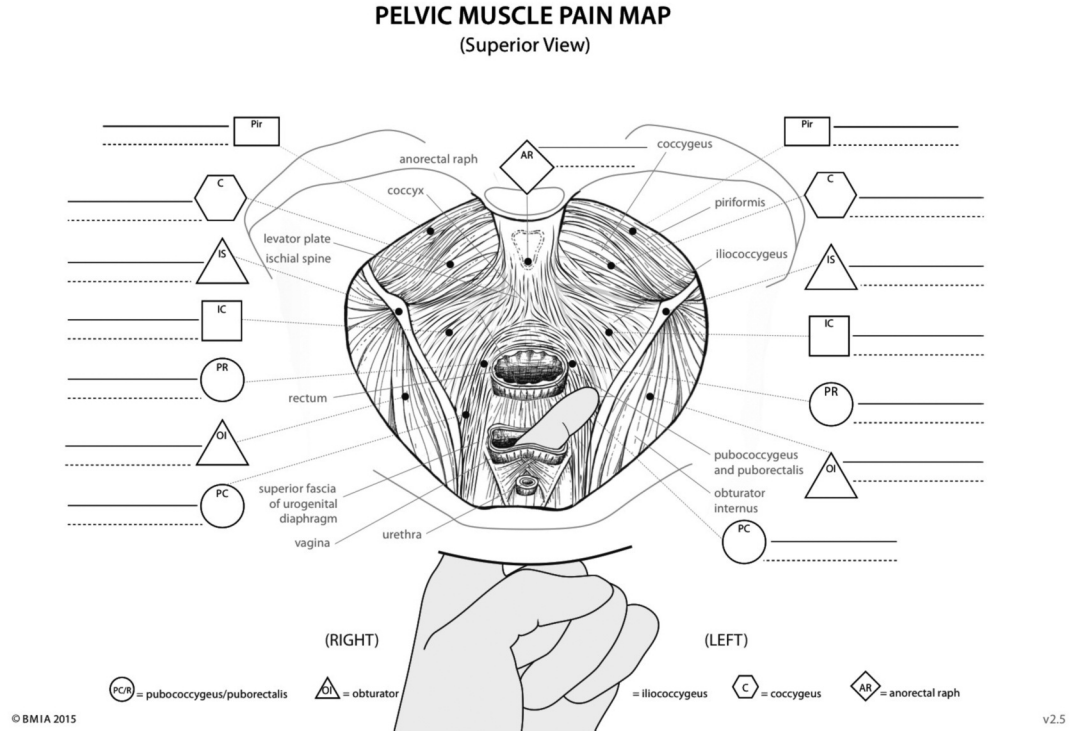

Figure 3. – Pelvic muscle map (Map 2), identifying internal points for pain mapping.

The pressure used during assessment of the internal muscles ranged from 0.4 to 0.5 kg/cm^2. A single digit was gently inserted into the vaginal canal to palpate the levator ani, and muscles lateral and posterior to the introitus. Each participant was then asked to voluntarily contract and relax the PFM, to assess general muscle strength using the six-point Oxford Scale.[20] In CUP cases the PFM are frequently in a non-relaxing state, revealing a functional contracture, which has also been shown by EMG assessments.[21] A healthy pelvic muscle should feel bulky, but well elasticized, maintaining appropriate pressure around the examiners finger, with no pain on contact.[22]

Mapping the para-urethral area (Map 3). When palpating the paraurethral area, a maximal pressure of 0.4 -– 0.5 kg/cm^2 was applied, always starting with very light pressure and progressively increasing to maximal pressure. The paraurethral points were palpated lateral to the urethra, as shown in Figure 4.

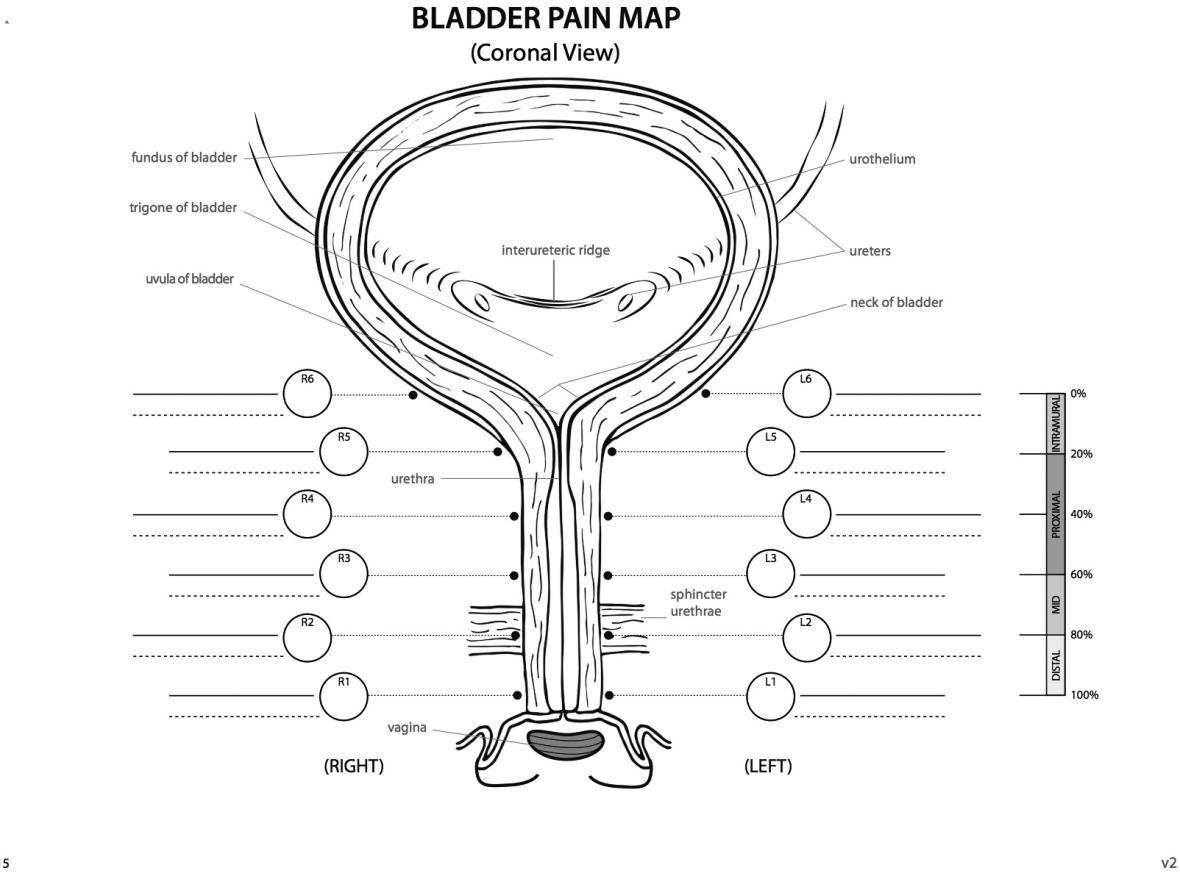

Figure 4. – Bladder pain map (Map 3), identifying paraurethral points for pain mapping.

Due to the fact that the paraurethral area is not normally examined as a source of pain, there are no protocols in literature for its assessment. The rationale for the protocol used in this study is based on the anatomical structure of the urethra. The urethra is a multilayered hollow tube of approximately 4 cm in length. It is surrounded by striated and smooth muscle layers organised in circular and longitudinal formations. For much of its length the urethra is fused with the anterior vaginal wall. At the level of the levator ani muscle the urethra is "incorporated into the pelvic floor rather than piercing a layer of muscle tissue".[4] Since the urethra, bladder and supporting structures of the pelvis are all part of the pelvic floor, the tone and sensitivity of the urethra are closely linked to pelvic muscle tone.

Anatomically the urethra can be divided longitudinally into percentiles, with the opening of the internal vesical neck representing point 0, and the external meatus representing the 100th percentile.[4, 23] Detrusor muscle fibres extend as far as the 15th percentile and the striated urogenital sphincter muscle begins where the detrusor muscles fibres end and extend to the 64th percentile.[4] For mapping purposes palpation points are separated in 20th percentile increments, beginning at the external meatus around the 100th percentile, then moving up to the 80th, 60th 40th, 20th, and 0 at the level of the vesical neck. An additional palpation point, estimated at a 20th percentile above the bladder neck, is recommended to assess pain originating from the trigone.

Analyses. Total pain scores were computed for each pain map for the three groups. The following analyses were undertaken; calculations of demographic data; means of pain scores across three maps and nine anatomical regions for the three groups; a comparison of pre and post treatment scores for CUP groups; and comparison of post treatment scores of the two diagnostic groups and the control group. The aim of the pre and post comparison was to show the sensitivity of the IMAP to changes in muscle pain scores.

As the study data consisted of a retrospective review of de-identified patient information, based on the written consent of each participant, no formal institutional approval was required.

RESULTS

A total of 82 participants were enrolled in the study, and data from 70 of the participants was used in the final statis-

tical analysis. Following 12 exclusions, 27 women remained in the vulvodynia group, 18 in the BPS group, and 25 in the control group. Statistical analysis was performed using SPSS v22.0.

There were no significant differences between the age means for vulvodynia, BPS and control group (Age Means = 34.4, 31.0, 33.9, respectively, $F(2,67) = 0.70$, $p = 0.50$). A significant positive correlation was noted between age and parity ($r = .53$, $p < .001$). A significant negative correlation was found between age and external urogenital pain scores (Map 1), for the vulvodynia and BPS groups, ($r = -.28$, $p = .02$). There was no significant negative correlation between age and internal pelvic muscle pain (Map 2), for either diagnostic group ($r=.18$, $p=.14$); but there was a significant negative correlation between age and paraurethral pain (Map 3) for the combined diagnostic groups ($r=-.26$, $p=.03$).

One-way ANOVAs were applied to test for differences between the vulvodynia, BPS, and control groups, in the average pain scores derived from the three pain maps. There were significant group differences for all three maps: external urogenital map (Map 1), $F(2, 35.55) = 28.1$, $p <.001$); internal pelvic muscle map (Map 2), $F(2, 35.01) = 88.3$, $p<.001$; paraurethral map (Map3), $F(2, 39.12) = 100.79$, $p<.001$) as shown in Figure 5.

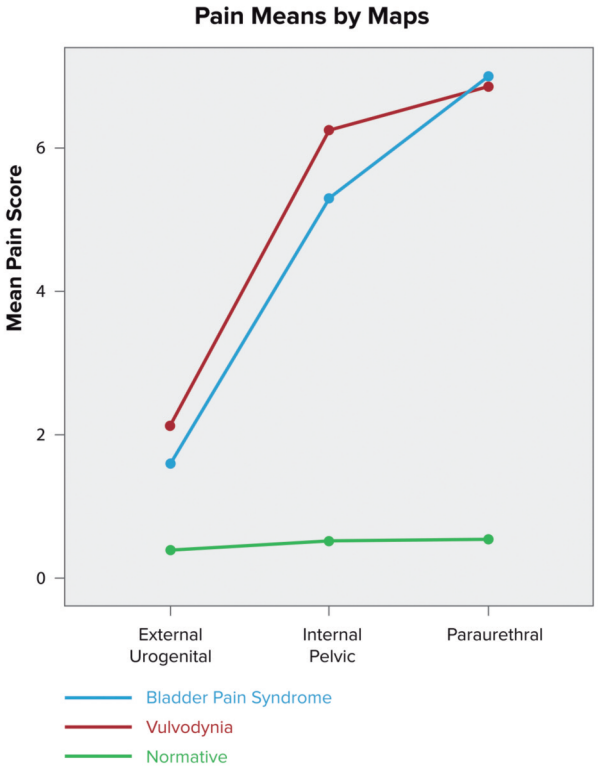

Figure 5. – Comparison of three pain map means for vulvodynia, BPS and asymptomatic controls.

Post-hoc pairwise t-tests revealed that there were significant differences between controls and vulvodynia groups, and control and BPS groups, for all three pain maps. There were no significant differences in pain scores on each of the pain maps between the vulvodynia and BPS groups. The vulvodynia and BPS group experienced the least pain with the external urogenital pain points (Map 1), significantly more pain with internal pelvic muscles (Map 2) and most pain with the paraurethral points (Map 3).

An analysis of pain by nine specific anatomical regions, showed the lowest pain scores to be from the suprapubic, clitoral, anal and superficial muscles (as per Map 1). Higher scores were provided for the urethral and vestibular regions (as per Map 1). A significant increase in pain scores for the two internal pelvic muscle regions - the levator muscles and obturator internus, and piriformis and coccyx (as per Map 2). Maximal pain scores were recorded in the paraurethral area (as per Map 3). The regional pain scores are shown in Figure 6.

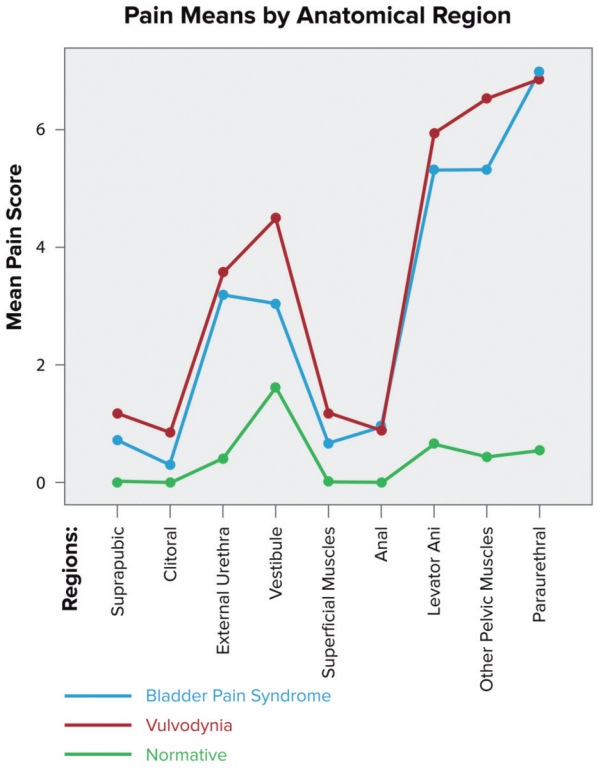

Figure 6. – Comparison of means of nine anatomical regions for vulvodynia, BPS and asymptomatic controls.

There were no significant differences in regional pain scores between the vulvodynia and BPS groups. Both groups showed the same pain score trends on all three maps.

A 3 x 2 x 2 mixed ANOVA was applied to investigate the relationship between the pain maps, treatment, and diagnosis. This three-way interaction was non-significant, $F(2, 11) = 1.96$, $p = .16$). The main effect of treatment was significant, $F(1, 11) = 166.05$, $p < .001$). Patient pain scores were significantly lower after treatment. However, the treatment by diagnosis interaction was non-significant, $F(1, 11) = 0.02$, $p = .90$). This suggests that the effect of the treatment was the same for both the vulvodynia and BPS groups, for each of the pain maps as shown in Figure 7.

A 3 x 2 x 2 mixed ANOVA was applied to investigate the relationship between the nine pain regions, treatment, and diagnosis. This three-way interaction was non-significant, $F(8, 11) = 1.23$, $p = .29$). The main effect of treatment was significant, $F(1, 11) = 96.14$, $p < .001$). Patient pain scores were significantly lower after treatment. However, the treatment by diagnosis interaction was non-significant, $F(1, 11) = 0.06$, $p = .81$). This suggests that the effect of the treatment was the same for both the vulvodynia and BPS groups, across all the pain regions as shown in Figure 8.

In the final analysis, the post treatment pain scores for the vulvodynia and bladder pain group showed no significant differences to the asymptomatic, control group pain scores for all three pain maps (external urogenital map, $F(2, 35) = 0.25$, $p = .78$); internal pelvic, $F(2, 35) = 0.77$, $p = .77$; and paraurethral, $F(2, 35) = 0.79$, $p = .79$).

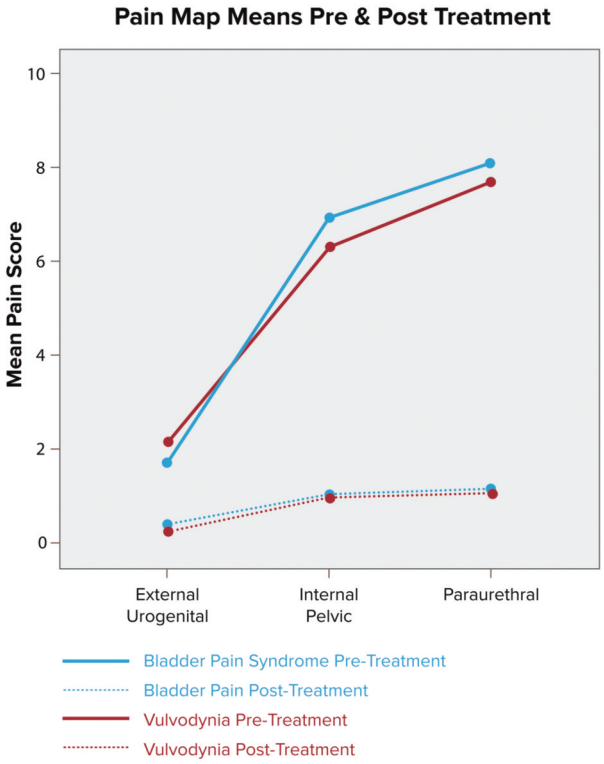

Figure 7. – Comparison of pain map means for pre and post treatment scores (three maps) for Vulvodynia and BPS.

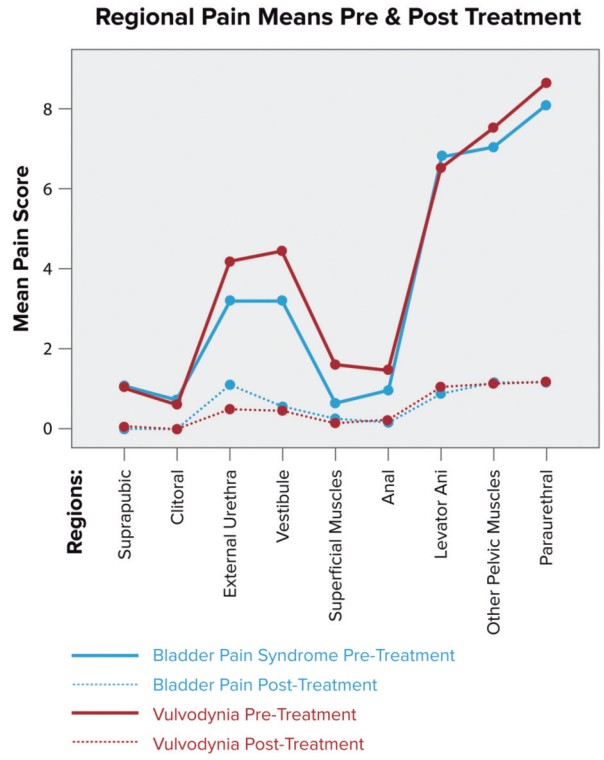

Figure 8. – Comparison of regional means for pre and post treatment scores (nine anatomical regions) for vulvodynia and BPS..

In the final analysis, the post treatment pain scores for the vulvodynia and bladder pain group showed no significant differences to the asymptomatic, control group pain scores for all three pain maps (external urogenital map, $F(2, 35) = 0.25$, $p = .78$); internal pelvic, $F(2, 35) = 0.77$, $p = .77$; and paraurethral, $F(2, 35) = 0.79$, $p = .79$).

DISCUSSION

This is the first study to provide a full urogenital pain map for the two CUP syndromes of vulvodynia and BPS. The results show that pain of pelvic muscle origin is characteristic of chronic pain syndromes. It is not a feature of asymptomatic controls, and does not constitute a normal state. This finding is consistent with a recent study assessing tenderness of pelvic muscles in asymptomatic nulliparous women.[10]

The results showed a significant negative correlation between age and pain on the urogenital and bladder pain map (Maps 1 & 3), indicating that pain was not linked to ageing. Quite to the contrary, it is the younger women who more frequently present with CUP symptoms. This is consistent with earlier findings showing that the prevalence of CUP disorders peaks at an early age.[24, 25]

When the pain scores for vulvodynia and BPS groups were compared on the three pain maps, and across the nine anatomical regions, there were no significant differences between the two groups. This raises the important question of whether these two pain syndromes constitute different pain disorders, or form the same syndrome with variations in symptom presentation.[26] The data supports the later view and is consistent with the conclusions of other studies.[27, 28]

The highest pain map scores were reported in the paraurethral area (Map 3). However, the areas traditionally mapped as part of diagnostic assessments for vulvodynia and BPS focus only on the external urogenital and perineal area. This study demonstrates that the paraurethral area may be of greater diagnostic and therapeutic relevance than the external urogenital region. This was consistent with the earlier findings of the authors.[13, 14] Only two other studies have made mention of bladder base tenderness, one in the context of dyspareunia, the other in the context of BPS.[29, 30] In this study, paraurethral pain provoked by palpation was commonly described by patients as sharp, piercing, stabbing and burning pain, reproducing sensations of urge, suprapubic pressure, low abdominal pain, groin discomfort and in some cases, sensations of burning in the soles of the feet, as shown in Figure 8. Vulvodynia and BPS patients commonly use these pain descriptors, and palpation of the paraurethral points completely and accurately reproduced each patients symptoms.

An earlier study reported that BPS patients, on average, identified 2.1 pain sites.[27] Suprapubic pain was most commonly reported (83%), followed by urethral (36%), non-genital (29%) and genital (23%) pain. Pain was described as throbbing, tender, piercing and aching. Since 84% to 90% reported worsening of pain with bladder filling, it was hypothesized that the bladder was the generator of pain.[27] While the results of pain sites and descriptors are consistent, the current study showed that it is the paraurethral area, not the bladder that is in fact the generator of pain and symptoms.

The proximal paraurethral points associated with urge and frequency may be linked to the descent of the detrusor muscle fibres that extend down as far as the 15th percentile (refer to Figure 4). The sensation of urge increased in intensity with proximity to the bladder neck and the trigone. However, there were exceptions where urge was also reproduced by mid urethral points. These observations tend to confirm earlier reports that myofascial trigger points (TrPs) in the paraurethral area are linked to symptoms of frequency and urge as well as antidromic inflammation of the interstitium, as seen with BPS/IC.[31]

Some participants sought to differentiate between discomfort associated with urge and the experience of pain. They reported lower pain scores, yet experienced the most

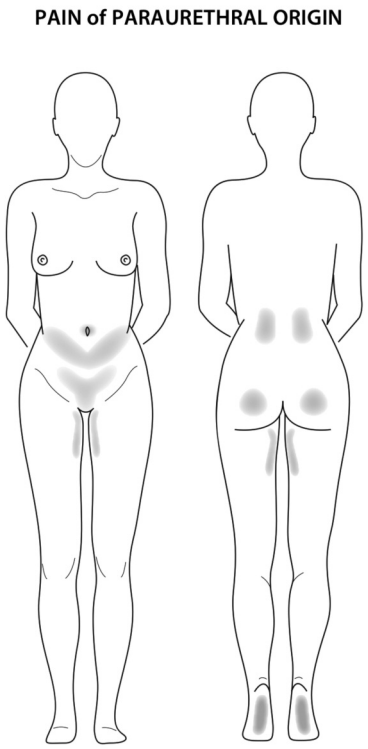

Figure 9. – Distribution of pain originating from the paraurethral area.

intense and distressing sense of urge when the proximal paraurethral points were palpated. Future studies need to differentiate between pain and the distress associated with an intense urge to void. During the course of therapy, as symptoms would abate, the rigidity of the urethra was reduced and the paraurethral area became more supple and markedly less sensitive. When the paraurethral area was successfully desensitized, urge triggered by palpation was eliminated and patients reported a reduction in voiding frequency. Even patients without voiding problems commented on reduced hesitancy and improved flow post desensitization.

Paraurethral hypersensitivity noted in this study is consistent with earlier studies by the authors which showed that palpation of paraurethral points reproduced bladder pain and voiding symptoms in 100% of BPS cases.[13] The authors also found that in a subgroup of patients with clitorodynia and persistent genital arousal disorder, the upper paraurethral points reproduced clitoral pain and symptoms of arousal. Upon desensitization these symptoms would also subside.[14]

The extreme sensitivity of the paraurethral area appears to be a trigger for the bracing and guarding response of the deeper pelvic muscles. Such muscle reactions could be misdiagnosed as defining the involuntary muscle spasm noted in vaginismus. Yet, the reaction appears to be a normal pain reflex. In time, such a pain reflex may develop into an anticipatory protective reaction and become associated with anxiety, fear and phobia.

Lack of clinician's awareness of the existence of paraurethral hypersensitivity gives rise to a range of costly and invasive tests in search of pathology that might explain the persistent pain. Despite repeated laparoscopies and ultrasounds being performed, results are usually negative or pathology is disproportionate to the severity of pain. During the course of mapping the majority of patients stated that their pain and symptoms were accurately reproduced. Identifying the source of their pain was reassuring and reduced the perceived threat of more ominous causes of pain.

Analysis of data from the deeper pelvic muscles map (Map 2) showed this to be the second most-tender area. These muscles have been the subject of various research reports in literature.[32, 33] The levator muscle, as it supports the pelvic organs and encloses the hiatus, is structurally one of the most susceptible muscles to functional change. The over-activation of the levator ani muscle leads to a functional contracture resulting in the narrowing of the introitus and ischaemic hypersensitivity.[21] During pain mapping, palpation of the vestibule reproduced the sharp burning pain that women reported experiencing when wearing tight clothing, sitting or attempting penetration. Vestibular hypersensitivity is typically assumed to arise from peripheral nerve damage, yet appears to be the result of the hypertonus state of the levator ani muscles.

During pain mapping, the puborectalis was found to refer pain into the anorectal area and cause a sense of bowel urge. The coccygeus and piriformis muscles referred pain into the lower buttocks, coccyx and lower back. The obturator internus muscle accounted for intense pain radiating into the hip, lower back, abdominal quadrants and thigh region.

Mapping scores from the urogenital area (Map 3) provided the lowest overall pain scores for the vulvodynia and BPS groups. The pubic area, perineal body and superficial muscles, including the bulbocavernosus, ischiocavernosus and the transverse muscles, were not found to be generators of pain. The pain scores from the external urethral area and the vestibule received the highest pain ratings on the urogenital pain map. Pain in the anal sphincter area was rated low and appears to occur in conjunction with fissures, haemorrhoids, constipation and coccygeal pain.

The analysis of the pre and post treatment scores demonstrated the value of the IMAP to assess the effectiveness of a therapeutic intervention. According to patient's subjective estimates, the conclusion of therapy was marked by an 80-95% reduction in pain and voiding symptoms. The post mapping pain scores verified the patient's estimates by showing a statistically significant reduction in pain. The post therapy scores for vulvodynia and BPS patients showed no significant difference with the scores the control group. Some of the patients expressed the view that the treatment had provided them with a complete cure. A patient who previously suffered severe pain and voided at 15-minute intervals, noticed that she was voiding at 2.5-hour intervals and experienced no pain at the conclusion of therapy. Patients who suffered constipation reported restored regularity. Most patients were able to comfortably resume daily activities. The IMAP validated the patient's subjective estimates of improvement, and provided an objective means for evaluating the impact of a therapeutic intervention.

Given that pelvic muscles relate to more than one organ system, normalising the function of PFM appears to affect multiple systems simultaneously. If dysfunctional pelvic muscles lead to inflammation and tenderness of the urogenital viscera, they may have a similar impact on the gastrointestinal system. With comorbidities such as irritable bowel syndrome being common in CUP patients, future research should examine common links and mechanisms. Specifically, studies should evaluate what impact the normalizing of pelvic muscle function may have on reversing comorbidities.

In terms of potential mechanisms, several changes within muscles arise in association with muscle over-activation, commonly referred to as "wind-up". These are discussed in more detail elsewhere.[34] However, there are two primary mechanisms recognised in literature. The first of these is ischemia, or reduced blood perfusion, with hypoxia, reduced

oxygen supply during increased demand. Ischemia is associated with muscle over-activation and leads to deep tissue pain of moderate to high intensity.[35-37] Ischemic pain is most often described as "stabbing", "burning", "heavy" and "exhausting" pain and leads to lower pain thresholds consistent with peripheral sensitization.[35, 37, 38]Given that peripheral sensitization can be reversed through muscle normalization, the mechanisms are clearly different to those of central sensitization which require different management strategies.[39, 40] Ischemic pain characteristics appear to be totally consistent with symptoms of vulvodynia and BPS, making ischemia one of the likely mechanisms.[21, 41] Impaired microcirculation due to vasoconstriction, tissue hypoxia and tissue acidosis have been previously suggested as a cause of chronic pelvic and urogenital pain.[42]

The second mechanism of pain that arises from muscle over-activation is mediated by TrPs which can give rise to myofascial pain syndromes.[43, 44] Some estimate that 85% of chronic pain conditions are muscle mediated, giving rise to regional pain.[45] Pain from pelvic muscle TrPs is well documented but can go unrecognized unless the clinician is prepared to actively look for, and identify the source of pain, by palpating muscles and soft tissue that harbour these points of tenderness.[31, 46]

In summary pain mapping provides a range of benefits, the most important being its systematic approach to localising and identifying the source of pain. The localising of pain within pelvic muscles and soft tissue of the urogenital area further disproves the hypothesis of the end-organ being the cause of CUP. Based on the outcomes of this study and the clinical experience of the authors, it is the dysfunctional pelvic muscles that act as the generators of pain, and the end-organs appear to be the "innocent bystanders".[47] The wrongful attribution of pain to organs arises on account of the similarities in myofascial and visceral pain characteristics.[12] It is only through a systematic assessment of the urogenital area, using a tool such as the IMAP, that the true origins of pain can be established.

CONCLUSION

Pain mapping removes some of the perplexity associated with CUP syndromes. The systematic approach of the IMAP enabled pain to be localised, and peripheral mechanisms to be identified. Pain mapping directly links dysfunctional pelvic muscles to CUP symptoms. The use of the IMAP provided pertinent information that may be central to the development of evidence-based interventions. With further validation, the IMAP can serve as an important tool in the assessment and management of CUP.

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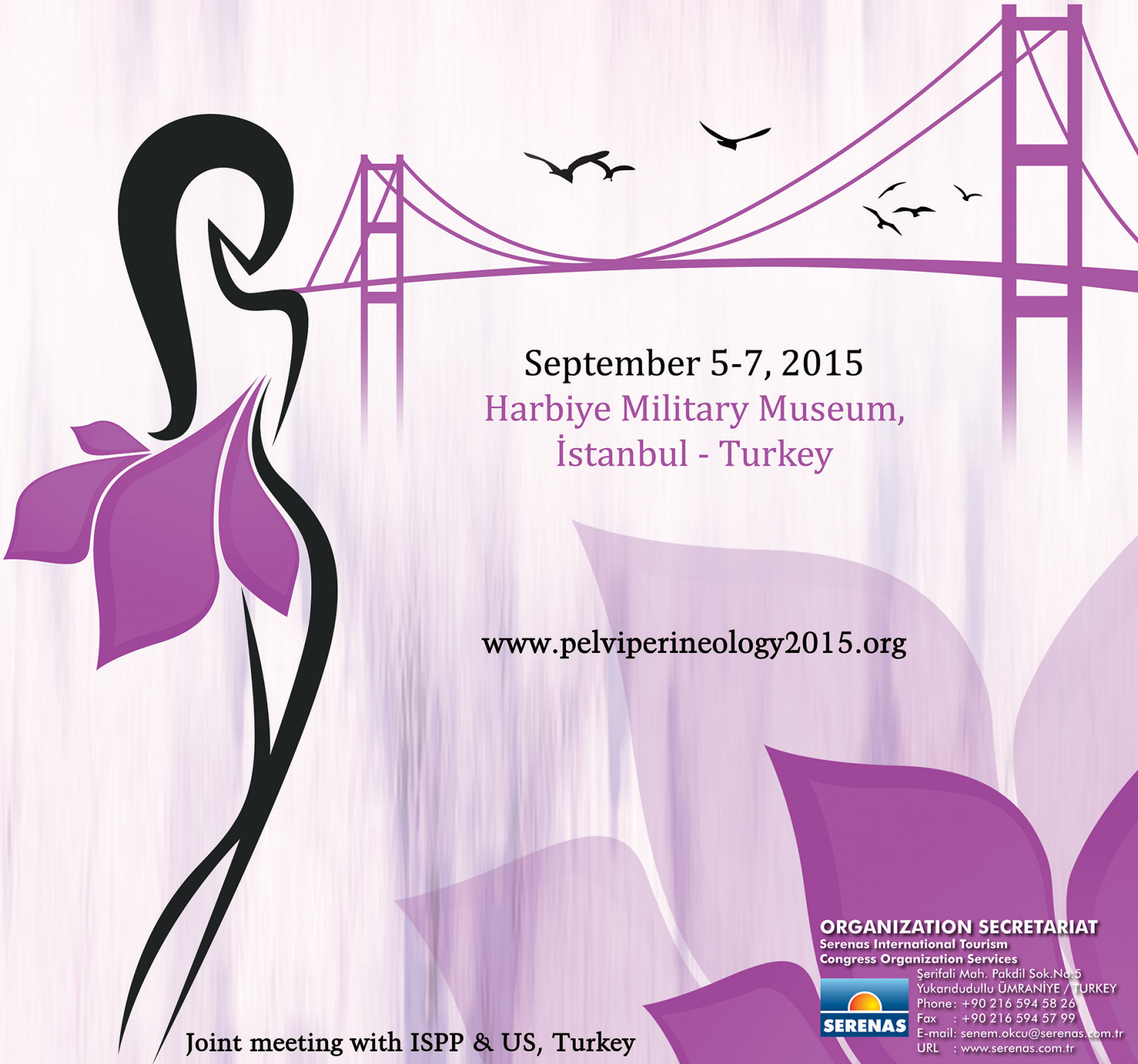

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