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).

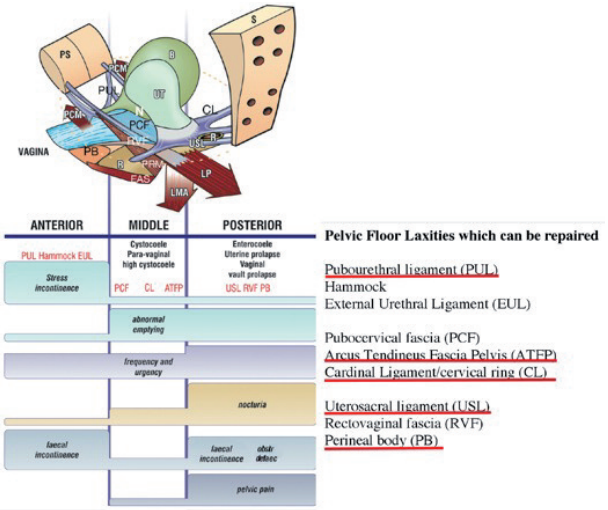

Figure 1. – Diagnostic algorithm. The posterior vector forces levator plate "LP" and conjoint longitudinal muscle of the anus "LMA" (arrows) selectively contract against the cardinal "CL" and uterosacral ligament "USL" during urethral and anorectal closure to stretch PCF to support the bladder base stretch 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.

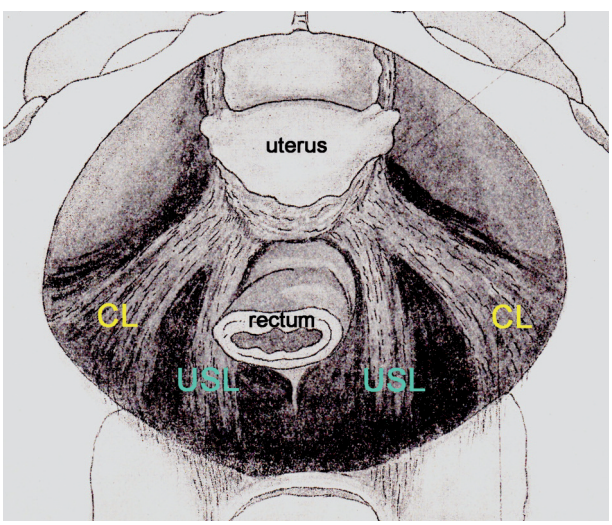

Figure 2. – Parametrium according to Martius (2). The Figure demonstrates that the parametric tissue fibres are mainly running backwards to the the iliosacral region connecting and suspending the cervix to the posterior pelvis. USL = uterosacral ligaments, CL = cardinal ligament.

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]

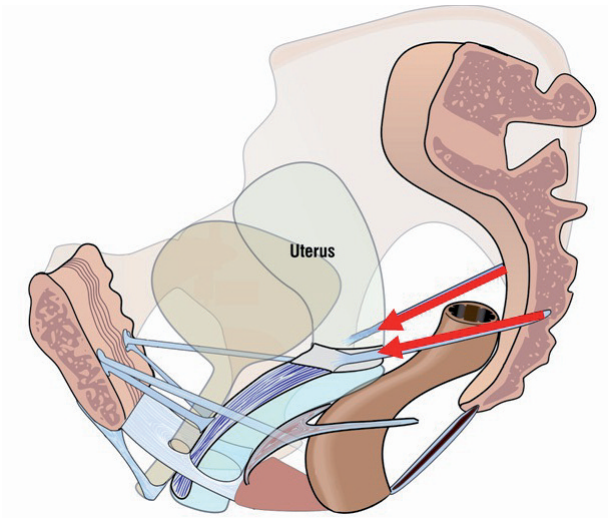

Figure 3. – The USL (red arrows) arises from the sacral vertebrate S2-4 and attaches to the cervical ring posteriorly.

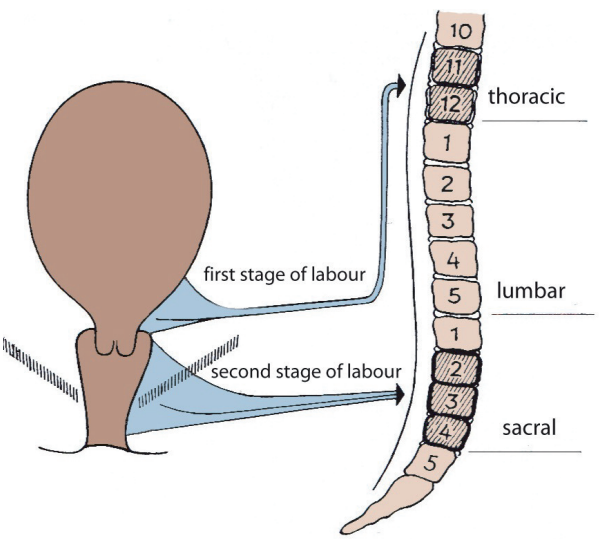

Figure 4. – Schematic diagram of visceral uterine innervation.

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]

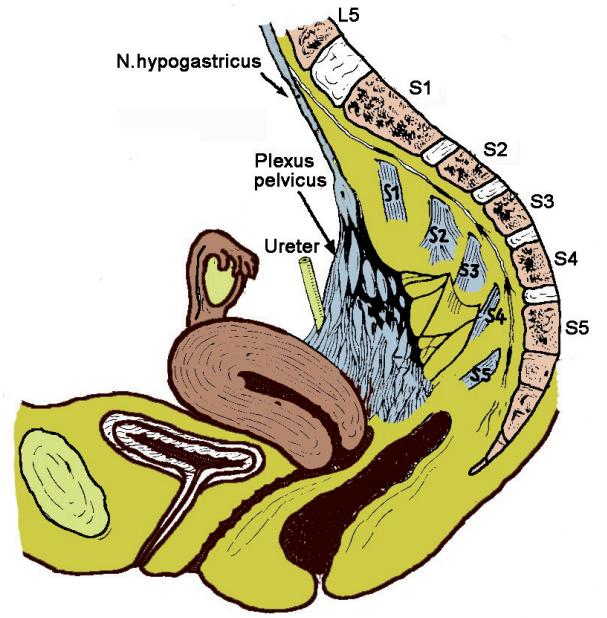

Figure 5. – Schematic diagram of pain transmission during birth. In the first stage of labour pain conduction is transmitted mainly to TH 11 and 12, in the second stage to the plexus pelvicus and sacralis.

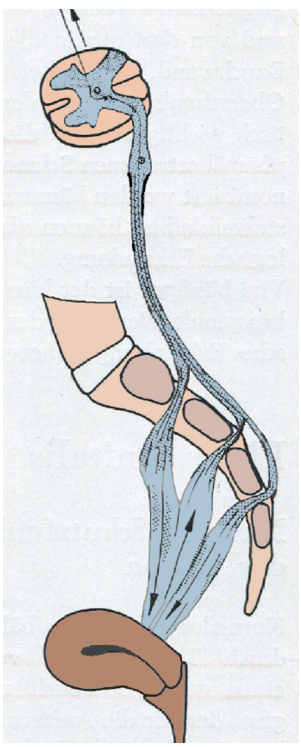

Figure 6. – Origin of mechanical, cerebrospinal transmitted gynaecological back pains.

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:

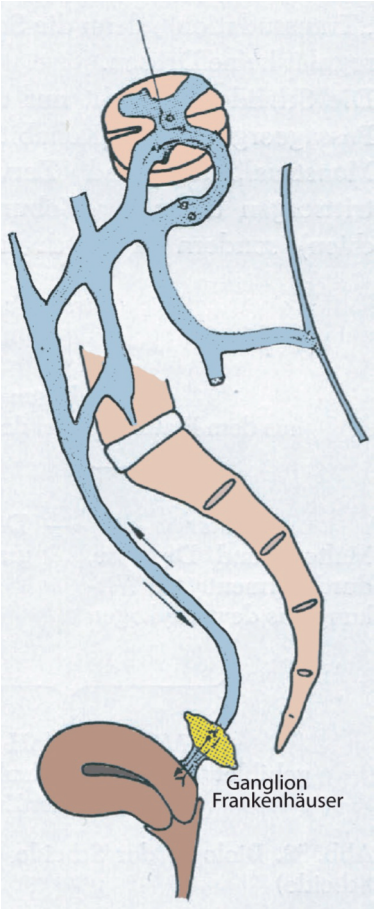

Figure 7. – Origin of visceroperipheral transmitted gynaecological back pains.

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.

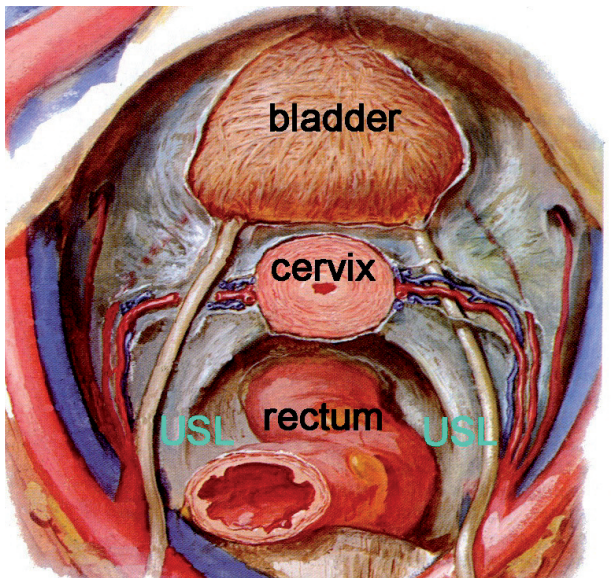

Figure 8. – Schematic diagram of the female pelvis mainly extending backwards. USL = uterosacral ligaments.

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.

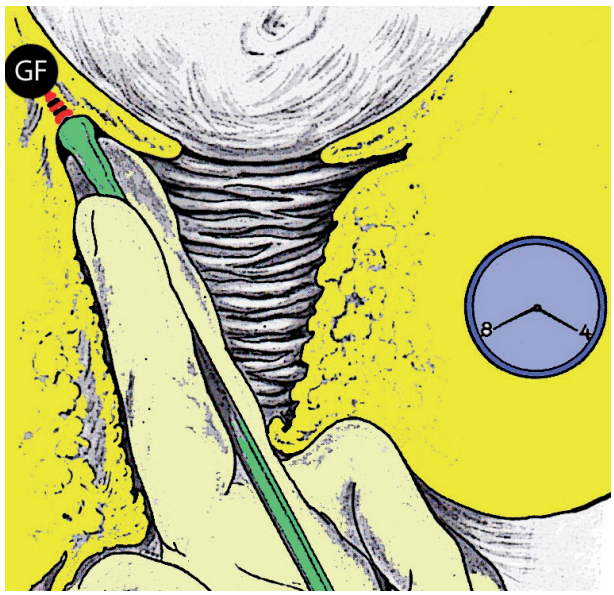

Figure 9. – Schematic picture of the paracervical block. Injection of local anaesthetics into the Ganglion Frankenhäuser area (GF) at 8 ó clock.

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.

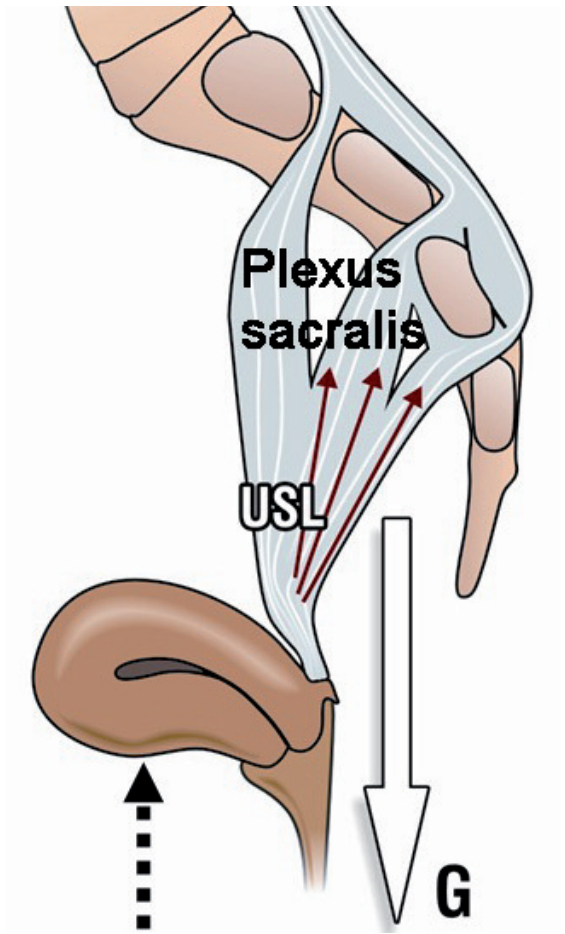

Figure 10. – In case of loose uterosacral ligaments (USL) or insufficient support from pelvic floor (black dotted arrow) gravity (G) pulls the uterus downwards producing pressure against the plexus sacralis.

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.

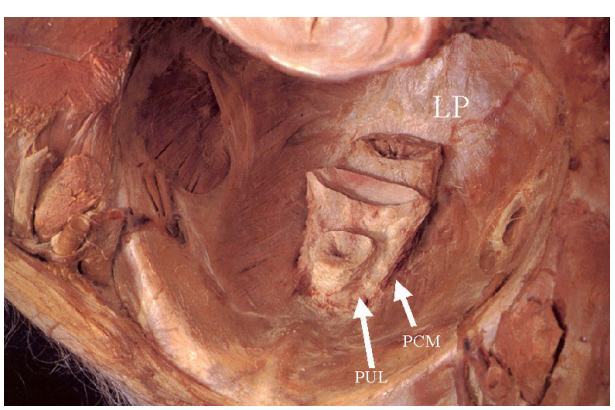

Figure 11. – Interconnectedness of organs and levator crus by connective tissue. Cadaveric specimen –view from above - front to back, urethra, vagina and rectum. PUL=pubourethral ligament; PCM=pubococcygeal muscle; LP=levator plate.

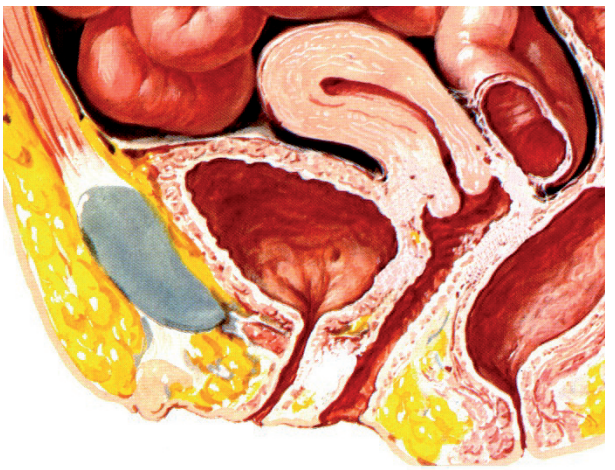

Figure 12. – Normal anatomy: A normal position of the uterus requires a stable support of pelvic floor.

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]

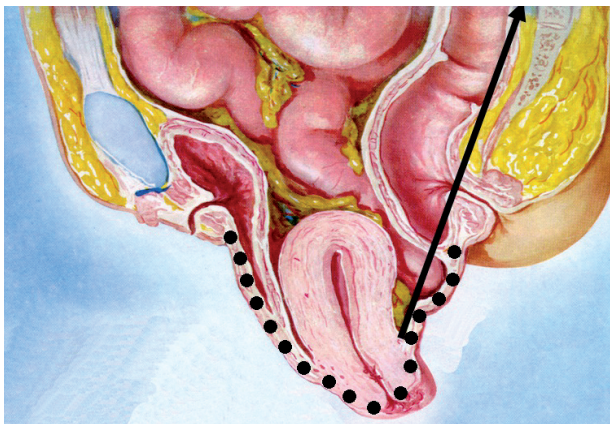

Figure 13. – Intestine prolapse: A damaged pelvic floor base (dotted black line) cannot support the pelvic organs causing prolapse, tremendous stretching of suspending ligaments (black arrow) and pain.

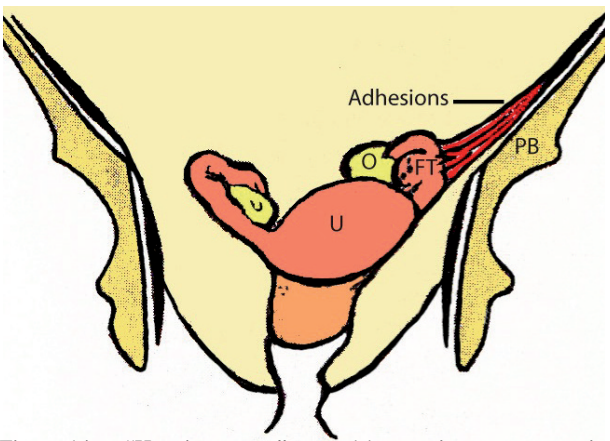

Figure 14. – "Hanging agony" caused by traction on parametric scar tissue. U = uterus, O = ovary, FT = fallopian tube, PB = pubic bone.

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.

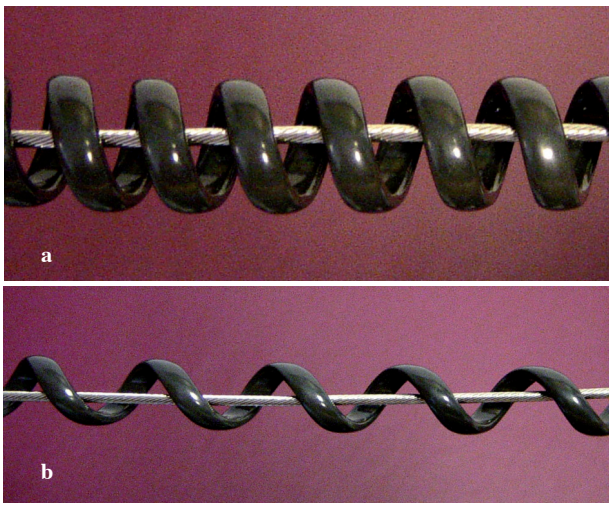

Figure 15a. and 15b. – Analogy of telephone cable and USL: a) USL with firm elastic connective tissue (black spiral cable outside) prevent stretching of nerve fibres inside b) Lax USL are not able to inhibit strain on nerve fibres inside

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

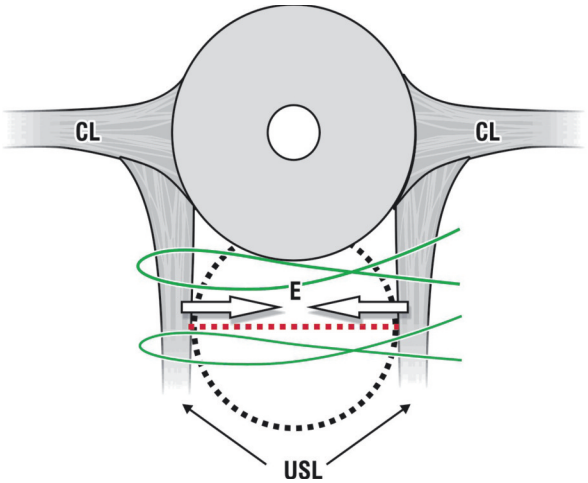

Figure 16. – Simple posterior fornix repair. A transverse incision is made in the posterior fornix 3-4cm below the cervix, A large No1 needle is inserted widely laterally below the vaginal skin and the loose uterosacral ligaments (USL) are approximated (arrows) with a strong Vicryl or polypropylene sutures.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

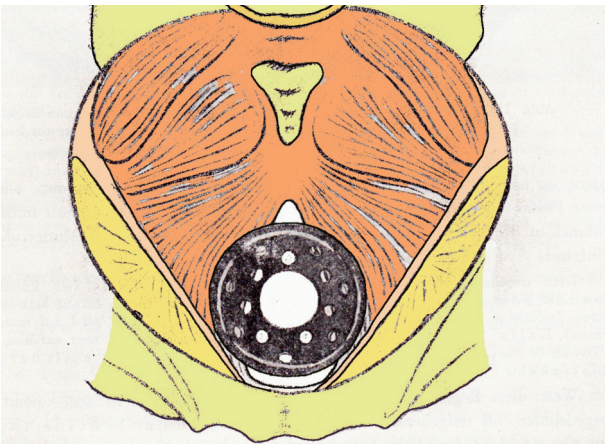

Figure 17a. – Schatz pessary in adequate position.

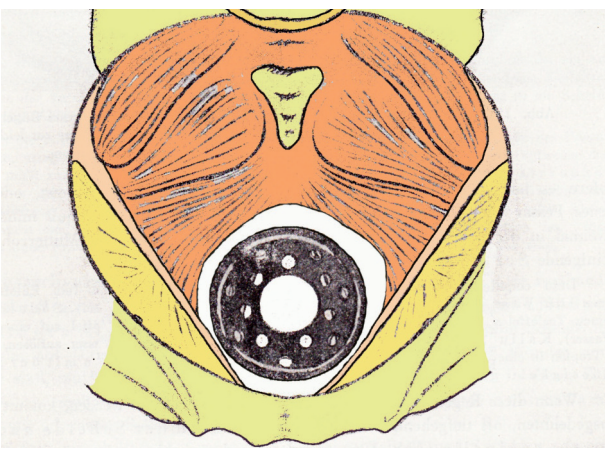

Figure 17b. – Not working Schatz pessary due to large levator muscle gap.

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

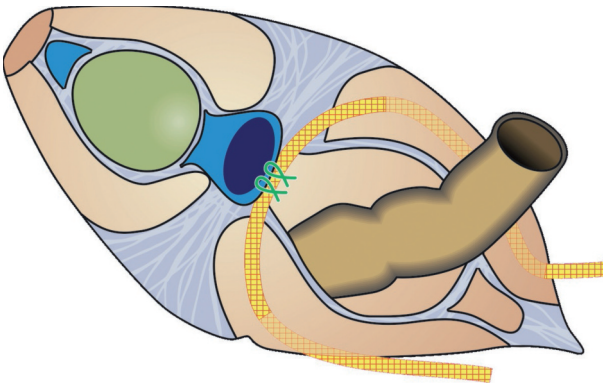

Figure 18. – Posterior intravaginal slingplasty (pIVS): The tape is placed along the exact position of the uterosacral ligament (USL).

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

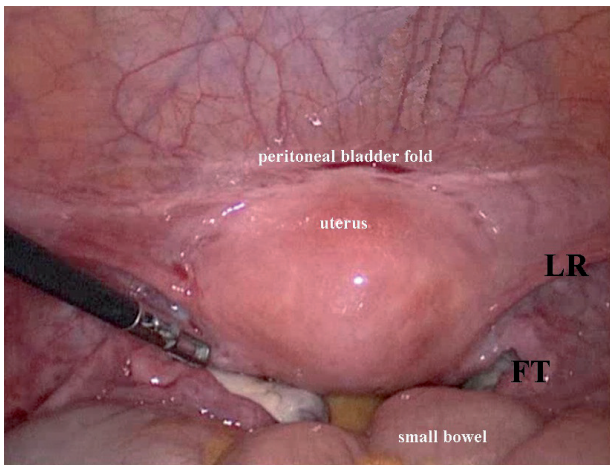

Figure 19a. – Uterus in normal position after vaginal insertion and sacrospinous fixation of a USL- and CL- neoligament. FT and LR, with normal shape, are far away from Douglas cavity.

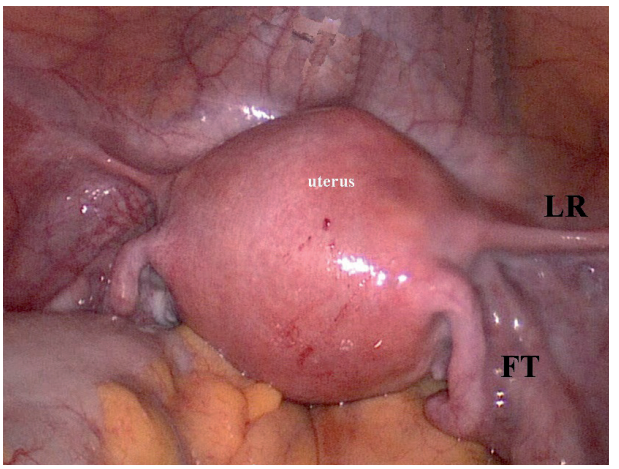

Figure 19c. – Uterus in descendent position. Ligamenta rotunda are lax (LR), fallopian tube (FT) deeply in Douglas cavity.

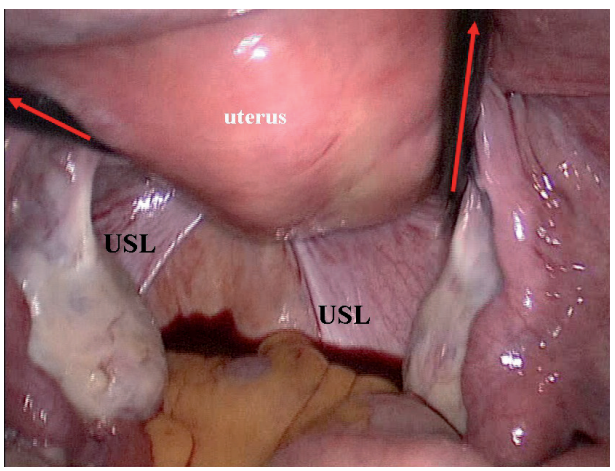

Figure 19b. – : Anteversion and elevation of uterus (red arrows) generate tension on the thin USL.

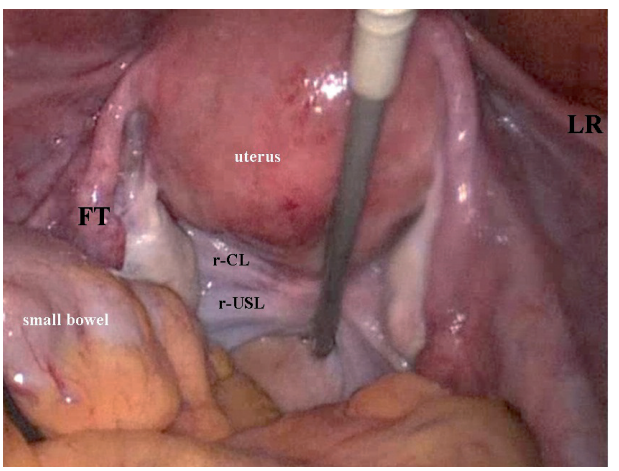

Figure 19d. – Reconstructed USL (r-USL) and CL (r-CL) in physiological position on both sides prevent a descent of the uterus.

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]

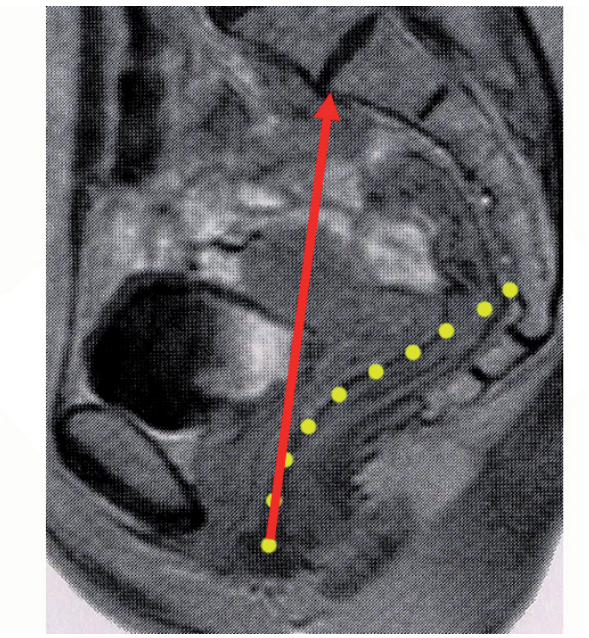

Figure 20. – MRI – Picture of the female pelvis in upright position. Sagital view of pelvic organs.Dotted yellow line = normal axis of vagina in a banana shaped backwards curveRed arrow = unphysiological vertical unbowed axis of vagina after laparoscopic/laparotomic sacrocolpopexy.

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.

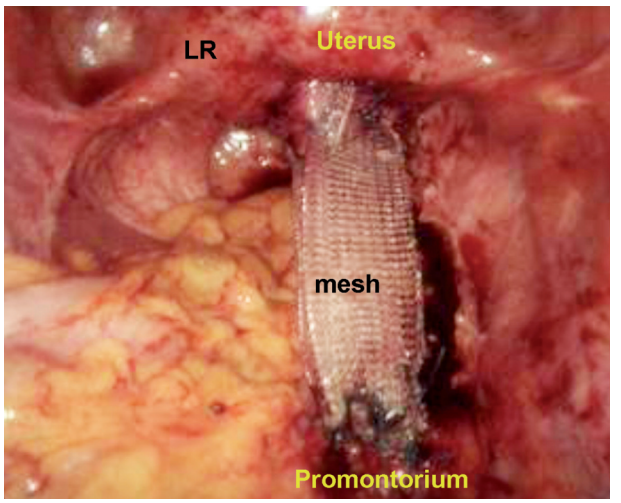

Figure 22. – Laparoscopic fixation of the uterus to the promontorium with a mesh-graft. In contrast to vaginal repair illustrated in Fig. 18 this procedure reconstructs neither vaginal axis nor USL and CL along the physiological course.

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

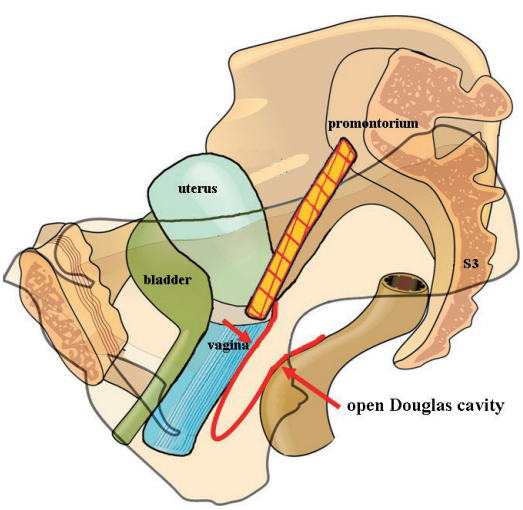

Figure 21. – Schematic picture of the female pelvis in upright position after abdominal colposacropexy. Sagital view. Fixation of vaginal apex and cervix to the promontorium with mesh pulls the uterus forwards and opens the Douglas cavity.

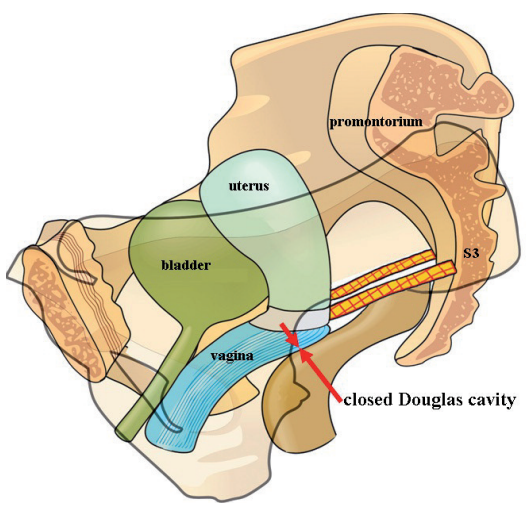

Figure 23. – Schematic picture of the female pelvis in upright position after vaginal colposacropexy. Sagital view. Fixation of vaginal apex and cervix to the vertebrate S3 with mesh creates a normal vaginal axis keeping the Douglas Cavity close.

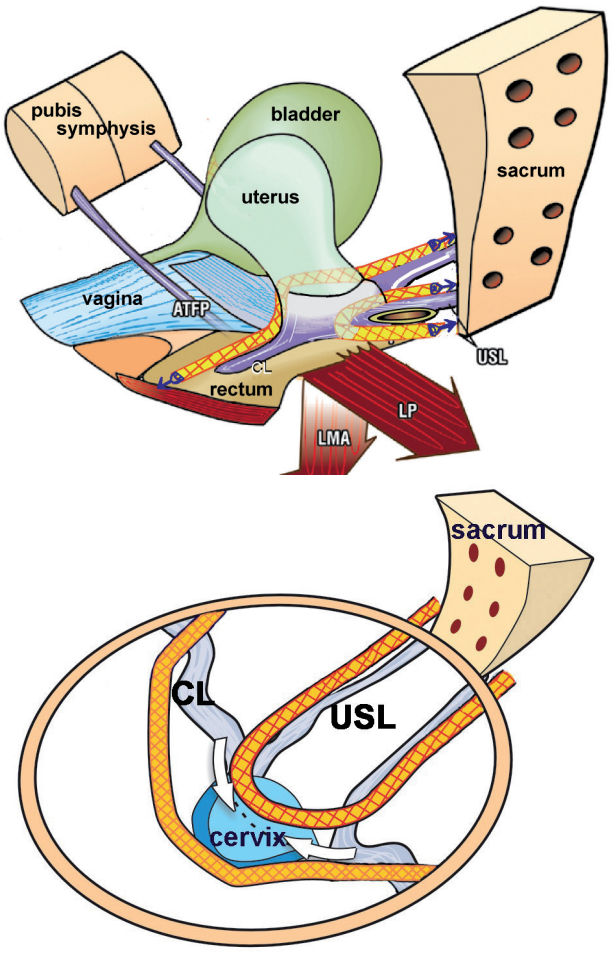

Figure 24a. and 24b. – Physiological reconstructed vaginal axis and shape after vaginal insertion of artificial ligaments along the exact course of uterosacral (USL) and cardinal (CL) ligament. ATFP = arcus tendineus fascia pelvis, LP = levator plate, LMA = longitudinal muscle of the anus.

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

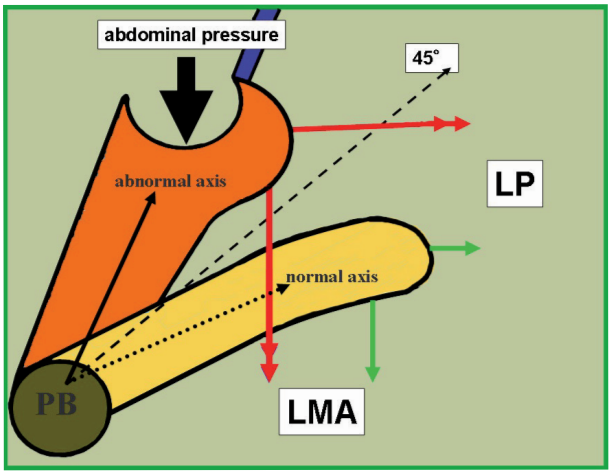

Figure 25. – Abnormal vertical axis due to promontorium fixation (blue).In case of vertically inclined vagina with an axis to the horizontal >45 degrees abdominal pressure, backward force of levator plate (LP), downward force of longitudinal muscle of anus (LMA) accelerate prolapse/entero/rectocele formation.PB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

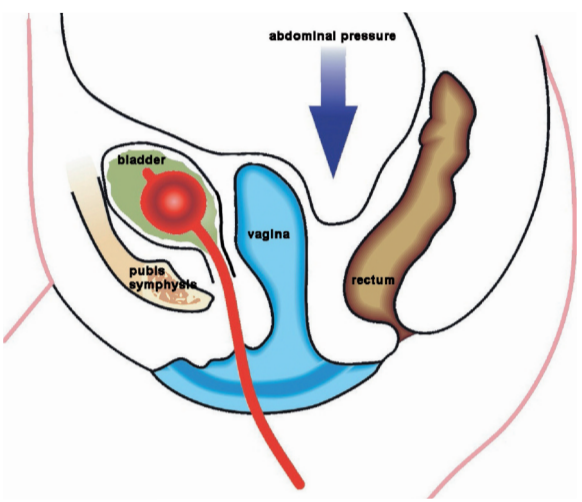

Figure 26. – Vagina in unphysiological vertical position after abdominal repair. The unsupported open posterior space allows the intraabdominal pressure and gravity force (dark blue arrow) to push the Douglas Cavity downwards creating vault prolapse, entero/rectocele and rectal intussusception.

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-

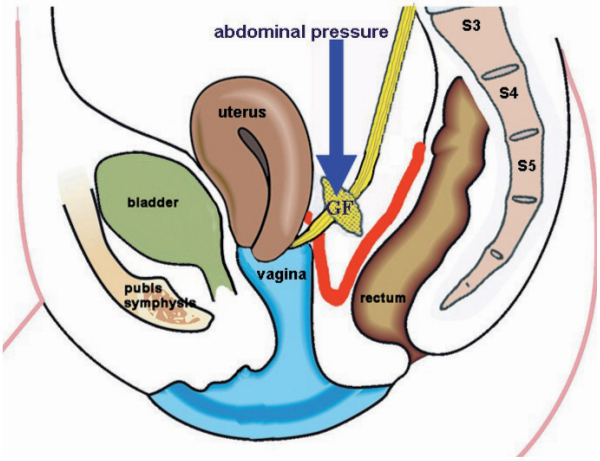

Figure 27. – Vagina in unphysiological vertical position after abdominal repair. Intraabdominal pressure (dark blue arrow) and gravity force squeeze the unsupported Ganglion Frankenhäuser (GF, yellow) downwards generating pai.

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).

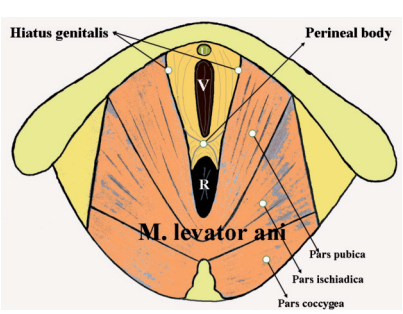

Figure 28. – Stable and firm genital hiatus protected by a strong pubic branche of the levator ani muscle.

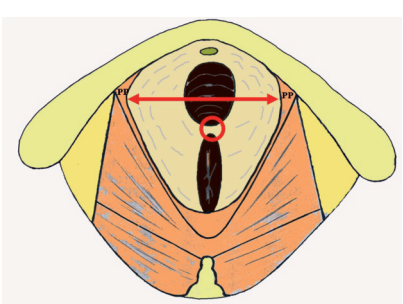

Figure 29. – Extremely wide genital hiatus (red arrow) caused by damage of levator ani muscle and endopelvic fascia. The levator crura are thin and far lateral, the perineal body (red circle) deficient.

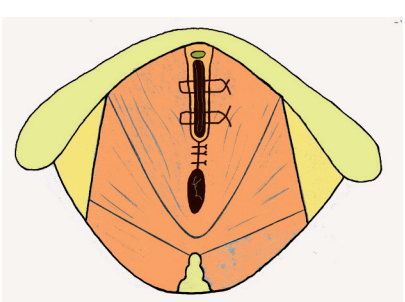

Figure 30. – Reconstructed genital hiatus after repair of perineal body and levator crura.

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.

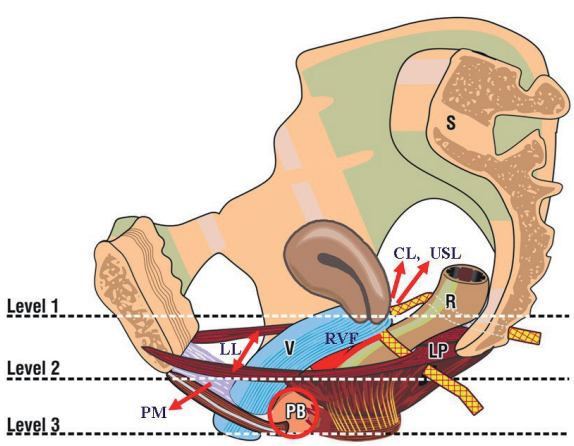

Figure 31. – Fig. 31 Three level repair:
Level 1 cardinal ligament (CL) and uterosacral ligament (USL)
Level 2 rectovaginal fascia (RVF) and levator cruses (LL)
Level 3 perineal body (PB) and perineal membrane (PM)

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.

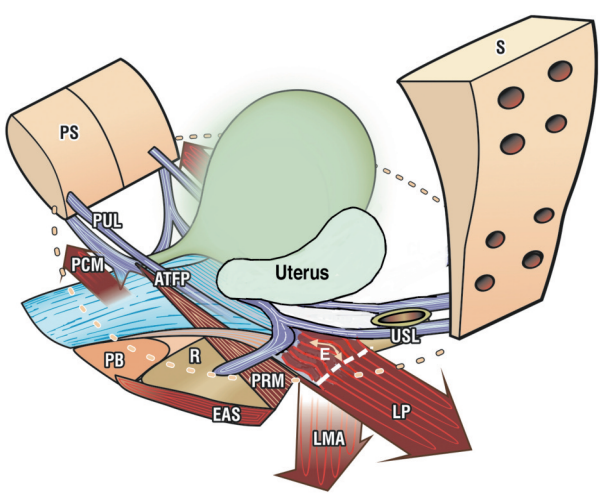

Figure 1. – How lax uterosacral ligaments (USL) may inactivate the muscle vectors according to Gordon's Law. The wavy lines 'E' above the horizontal broken lines along LP (levator plate) indicate how prolapse can lengthen the LP muscle. According to Gordon's Law, muscle lengthening will inactivate the 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
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* * *

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.

REFERENCE

Gineco.ro, 2010, year VI, volume VI, nr.20.2/2010, pg. 118 - 122 Index and abstracted in Thomson Reuters Science Citation Index Expanded Journal Citation Reports/ Science Edition Elsevier Bibliographic Data base: SCOPUS (ISI).

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%)

ANZJOG

REFERENCE

Petros PEP, Inoue *H Letter - Pelvic pain may be caused by laxity in the uterosacral ligaments as part of the "Posterior Fornix Syndrome"*. ANZJOG 2013; 53(3): 325-6. DOI:10.1111.

Dr Hiromi Inoue MD
Kamakura, Japan

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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

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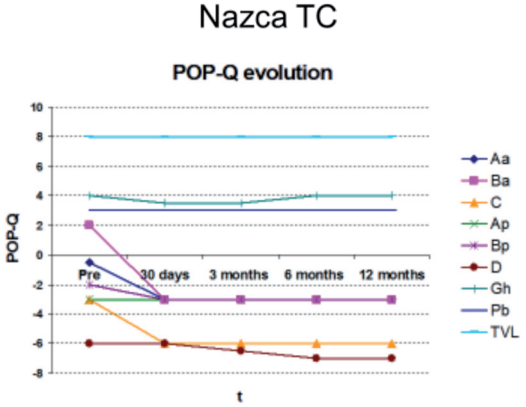
In my view Goeschen's paper is an important one that encourages the topographic diagnosis of chronic pelvic pain, instead of sindromic diagonosis and this is relevant.

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

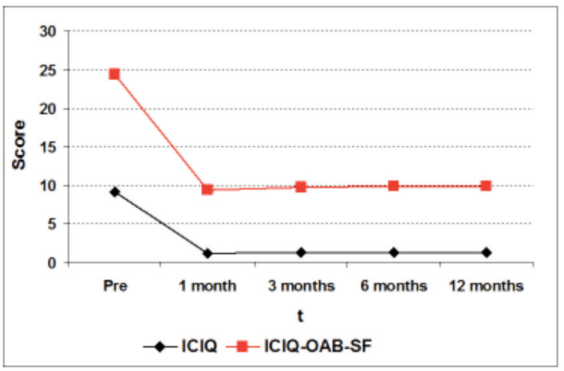
Demographics

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

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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

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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

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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
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