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1) Article from a Journal (Index Medicus):

a) Standard:

MacRae HM, McLeod RS. Comparison of haemorrhoid treatment modalities: a metanalysis. Dis Colon Rectum 1995; 38: 687-94.

Court FG, Whiston RJ, Wemyss-Holden SA, Dennison AR, Maddern GJ. Bioartificial liver support devices: historical perspectives. ANZ J Surg 2003; 73: 793-501.

or:

Court FG, Whiston RJ, Wemyss-Holden SA, et al. Bioartificial liver support devices: historical perspectives. ANZ J Surg 2003; 73: 793-501.

b) AA. VV.

The Standard Task Force, American Society of Colon and Rectal Surgeons: Practice parameters for the treatment of haemorrhoids. Dis Colon Rectum 1993; 36: 1118-20.

c) Cited paper:

Treitz W. Ueber einem neuen Muskel am Duodenum des Menschen, uber elastiche Sehnen, und einige andere anatomische Verhaltnisse. Viertel Jarhrsxhrift Prar. Heilkunde (Prager) 1853; 1: 113-114 (cited by Thomson WH. The nature of haemorrhoids. Br J Surg 1975; 62: 542-52. and by: Loder PB, Kamm MA, Nicholls RJ, et al. Haemorrhoids: pathology, pathophysiology and aetiology. Br J Surg 1994; 81: 946-54).

2) Paper from a Journal:

Bianco A. II medico tra "morale" e "moralismo". Questione morale: le ragioni della nostra professione. La Professione 2003: 3/4: 11.

3) Chapter from a book:

Milson JW. Haemorrhoidal disease. In: Beck DE, Wexner S, eds. Fundamentals of Anorectal Surgery. 1st ed. New York: McGraw-Hill 1992; 192-214.

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Legends must be typed on a separate page. Each table and illustration must be cited in the text in consecutive order. Electronic submission of images must include identification of each image by number (e.g., 1.jpg, 2.jpg) in order of citation. The appropriate position in the text should be indicated in the margin of the manuscript.

Editorial

Welcome to the first edition of Pelviperineology for 2009

March 2009 marks an important step in the development of this journal. We are pleased to announce a significant expansion of our editorial board with new contributors from the various specialties, reflecting our multidisciplinary nature. With our new editorial board the journal will have the resources to have regular contributions in the fields of Imaging, Anatomy, the Integral Theory as well as existing features of the European Perineology Group and the Pelvic Floor Digest.

The Journal now begins the next phase of our development striving to achieve Medline listing and start to develop an impact factor. Our new board is committed to developing the Journal as the preeminent multidisciplinary journal in the world. We are looking for people who wish to make a commitment to this process and willing to help us by submitting quality articles for publication, helping to review articles and generally make their colleagues aware of what the journal has to offer.

This year the AAVIS International Pelviperineology Congress will be held in Noosa on the Sunshine Coast of Queensland in conjunction with the International Pelvic Floor Dysfunction Society and the International Collaboration of the Pelvic Floor. This year the Urological Society and the Colorectal Society of Australasia have also been invited to participate. We are anticipating a good representation of all three pelvic floor specialties. There will be a special emphasis on colorectal dysfunction and treatment of posterior compartment defects. The multidisciplinary faculty and preliminary program can be reviewed by visiting the AAVIS Website at ***www.aavis.org***

THE EDITORS

Can pelvic floor ultrasonography "imagine" the future?

Pelvic floor disorders (PFD) include urinary and fecal incontinence, overactive bladder, constipation, pelvic pain and pelvic organ prolapse. These conditions are often assumed to be attributable to the effects of pregnancy and childbirth. It is still questionable whether pregnancy itself is a risk factor for PFD in later life or if it is the vaginal delivery that is the main risk factor. Pelvic floor laxity as a consequence of childbirth may result from weakening and stretching of the muscles and connective tissue during delivery or it may occur as result of spontaneous lacerations and episiotomies during delivery. Both can lead to impairment of the position and support of the pelvic organs. Damage to the levator muscle complex, that provides support to the lower urinary tract, reproductive tract and lower gastrointestinal tract, will result in impaired function of any, or all, of the structures that the muscles support. In addition, lack of integrity of connective tissue (as endopelvic fascia, uterosacral, coronal and pubourethral ligaments) that supports the three organ systems will lead to herniation of one organ system into another. Frequently damage occurs after the first delivery, however it may remain occult and patients may not become symptomatic until later in life or following subsequent vaginal deliveries.

There is a great need to increase our understanding of the natural history of pelvic floor dysfunction. An important issue is: can we detect occult pelvic floor damage and predict which damage can predispose and progress to PFD in certain women?

During the past decade, pelvic floor imaging techniques have become increasingly popular as diagnostic tools in the management of patients with PFD. Magnetic resonance imaging offers clear images of the entire pelvis, however MRI is expensive and with current technology is time-consuming. Ultrasonography also provides detail of the pelvic floor region. This modality is rapid, less costly, portable, and accurate. Recently introduced high frequency ultrasound transducers with a built-in 3D automated acquisition system can be successfully applied to improve our knowledge of the pelvic floor. Three-dimensional endovaginal, endoanal and translabial ultrasound provides a good visualization of anatomy and morphology of the pelvic organs, muscles with their attachments, soft tissues and vessels in several planes never obtained before. In addition dynamic ultrasonography provides information on the function of the three compartments. Levator ani or endopelvic fascia damages, which occur during the vaginal birth process, can be determined with the use of pelvic floor ultrasonography.

The goal is to use pelvic floor ultrasonography to identify the causative mechanism of PFD and its risk factors, and finally propose treatment. Using US assessment it may be possible "to imagine" the future and predict outcomes. Thus pelvic floor US can be performed after vaginal delivery to determine pelvic floor damages that are at risk for development of PFD in order to provide patients information regarding potential consequences of PFD and allowing patients to make a decision whether they wish undergo an elective caesarean delivery.

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Pelvic Floor Digest

*This section presents a small sample of the Pelvic Floor Digest, an online publication (**www.pelvicfloordigest.org**) that reproduces titles and abstracts from over 200 journals. The goal is to increase interest in all the compartments of the pelvic floor and to develop an interdisciplinary culture in the reader.*

FORUM

Stem cell differentiation by functionalized micro- and nanostructured surfaces. *Martínez E, Lagunas A, Mills CA et al. Nanomedicine 2009;4:65.* New nanotechnologies have provided with enormous possibilities when designing customized supports and scaffolds with controlled nanoscale topography and chemistry. This article reviews the main strategies followed to achieve solutions to the challenge for furthering fundamental biology studies.

Beyond the impact factor. *Watts G. BMJ. 2009;338:b553.* The article stresses the importance of measuring the social impact of research, and discusses about RAE (research assessment exercise) and its replacement, the REF (research excellence framework).

1 – THE PELVIC FLOOR

Effect of surgically induced weight loss on pelvic floor disorders in morbidly obese women. *Wasserberg N, Petrone P, Haney M et al. Ann Surg. 2009;249:72.* Bariatric surgery has a beneficial effect on symptoms of pelvic floor disorders in morbidly obese women with a significant reduction in total mean distress scores after surgery attributed mainly to the significant decrease in urinary symptoms but also for improvement in the pelvic organ prolapse domain. Age, parity, history of complicated delivery, percent excess body weight loss, BMI, type of weight loss procedure and presence of diabetes mellitus and hypertension have no predictive value for postoperative outcomes.

Surgical outcomes of VRAM versus thigh flaps for immediate reconstruction of pelvic and perineal cancer resection defects. *Nelson RA, Butler CE. Plast Reconstr Surg. 2009;123:175.* The surgical outcomes and complications in 133 cancer patients who underwent immediate reconstruction of defects following abdominoperineal resection or pelvic exenteration with vertical rectus abdominis myocutaneous (VRAM) versus thigh flaps are compared. Immediate VRAM flaps result in fewer major complications without increased early abdominal wall morbidity.

Combined surgery in pelvic organ prolapse is safe and effective. *Riansuwan W, Hull TL, Bast J, Hammel JP. Colorectal Dis. 2009 Jan 17 Epub.* Combined surgery for pelvic organ prolapse is safe and effective when considering outcomes of rectal prolapse surgery (93 operations in this study). Surgeons should not hesitate to address all pelvic floor issues during the same operation by working in partnership with the anterior pelvic floor colleagues.

2 – FUNCTIONAL ANATOMY

Circadian variation of rectal sensitivity and gastrointestinal peptides in healthy volunteers. *Enck P, Kaiser C, Felber M et al. Neurogastroenterol Motil. 2009;21:52.* There are significant differences in the perception of rectal distension stimuli for urge and pain depending on daytime, but the release of gastrointestinal peptides seems not to be involved. This circadian variation needs to be taken into account in patients and volunteer studies.

Pubo-urethral ligament injury causes long-term stress urinary incontinence in female rats: an animal model of the integral theory. *Kefer JC, Liu G, Daneshgari FJ. Urol. 2009;181:397.* A novel rat model (pubo-urethral ligament transaction that was compared to bilateral pudendal nerve transaction) can be used to investigate mechanisms of SUI in females, including the role of urethral hypermobility and potential therapeutic interventions.

Modulation of opioid actions by nitric oxide signaling. *Toda N, Kishioka S, Hatano Y, Toda H. Anesthesiology. 2009;110:166.* Endogenous oxide (nitric endothelial, neurogenic and inducible) plays pivotal roles in controlling physiological functions, participates in pathophysiological intervention, and is involved in mechanisms of therapeutic agents. This paper deals with modulation of morphine actions by nitric oxide as being useful in establishing new strategies for efficient antinociceptive treatment.

3 – DIAGNOSTICS

Factors influencing patient satisfaction when undergoing endoscopic procedures. *Ko HH, Zhang H, Telford JJ, Enns R. Gastrointest Endosc. 2009;69:883.* To identify factors related to patient satisfaction with endoscopy (EGD, colonoscopy) 261 patients were studied and 86.6% were very satisfied with doctor's personal manner and technical skills, nurse's personal manner, physical environment, and more time with doctor discussing the procedure. Initial satisfaction may depend on residual sedation, but it tends to decrease over time possibly because of recall bias.

Reliability of physical examination for diagnosis of myofascial trigger points: a systematic review of the literature. *Lucas N, Macaskill P, Irwig L et al. Clin J Pain. 2009;25:80.* Trigger points are promoted as an important cause of musculoskeletal pain, however there is no accepted reference standard for their diagnosis, and data on the reliability of physical examination for trigger points are conflicting and the matter needs to be further investigated with studies of high quality.

Do patients undergo prostate examination while having a colonoscopy? *Hammett T, Hookey LC, Kawakami J. Can J Gastroenterol. 2009;23:37.* Colonoscopy is an ideal opportunity for physicians to use a digital rectal examination to assess for prostate cancer. Physicians performing colonoscopies in men 50 to 70 years of age should pay special attention to the prostate while doing a digital rectal examination before colonoscopy. In a study on 846 colonoscopies performed by 17 physicians only in 15.0% of cases a comment regarding the prostate was made. This novel concept may help maximize resources for cancer screening and could increase the detection rate of clinically palpable prostate cancer.

CT Colonography: techniques and applications. *Yee J. Radiol Clin North Am. 2009;47:133.* CTC, also termed virtual colonoscopy, is increasingly accepted at sites throughout the world as a new effective tool for the diagnosis and screening of colorectal carcinoma. This article presents information of related issues of bowel cleansing, stool and fluid tagging, bowel distention, multidetector CT scanning parameters, appropriate applications, and potential complications.

Importance of gender, socioeconomic status, and history of abuse on patient preference for endoscopist. *Schneider A, Kanagarajan N, Anjelly D et al. Am J Gastroenterol. 2009;104:340.* Both men and women with a history of abuse are significantly more likely to prefer a woman endoscopist. Physicians should be aware of these high preference rates to increase compliance and optimize patient care.

4 – PROLAPSES

Robotic-assisted sacrocolpopexy: technique and learning curve. *Akl MN, Long JB, Giles DL, Cornella JL et al. Surg Endosc. 2009 Jan 27. Epub.* Laparoscopic sacrocolpopexy (LSCP) offers a minimally invasive approach for treating vaginal vault prolapse. The Da Vinci robotic

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surgical system may further decrease the difficulty of the procedure with acceptable complication rates and short learning curve: 2/80 patients had injury to the bladder, one a small bowel injury, and one patient had a ureteric injury, 5 developed vaginal mesh erosion, one a pelvic abscess, and one had postoperative ileus, 4 were converted to laparotomy.

Clinical pathway for tension-free vaginal mesh procedure: evaluation in 300 patients with pelvic organ prolapse. *Kato K, Suzuki S, Yamamoto S et al. Int J Urol. 2009;16:314.* Excluding five patients with concomitant hysterectomy, 305 consecutive women with POP-Q stage 3 or 4 between 2006 and 2007 were planned a TVM procedure with removal of the indwelling urethral catheter on the next morning, discharge on postoperative day 3. Perioperative complications were: bladder injury (11 cases), vaginal wall hematoma (2), rectal injury (1) and temporary hydronephrosis (1). The catheters were removed on the next morning in 95.6% of the cases, none required intermittent catheterization at home. Postoperative hospitalization was as planned in 93.3% of the cases. Two patients were re-hospitalized within one month due to vaginal bleeding or gluteal pain. Patients generally accepted the early discharge in spite of the Japanese culture preferring a longer hospital stay.

Trocar-guided transvaginal mesh repair of pelvic organ prolapse. *Elmér C, Altman D, Engh M et al. Obstet Gynecol. 2009;113:127.* A prospective multicenter cohort study performed throughout 26 clinics on 261 patients evaluated at 2 and 12 months with POP-Q, Incontinence Impact Questionnaire (IIQ-7), Urogenital Distress Inventory (UDI-6). Anatomic cure (POP-Q stage 0-I) was observed in 79% after anterior repair with polypropylene mesh, 82% after posterior repair. For anterior and posterior repair cure was 81 and 86% respectively for the anterior and posterior compartment. Bladder and rectal perforations occurred in 3.4%, vaginal erosions in 11%). Surgical intervention due to mesh exposure occurred in 2.8%. There were significant quality-of-life improvements in all domains of the IIQ-7. Despite significant improvements in UDI-6 scores, symptoms specific for SUI were not ameliorated.

Sexual dysfunction after trocar-guided transvaginal mesh repair of pelvic organ prolapse. *Altman D, Elmér C, Kiilholma P et al. Obstet Gynecol. 2009;113:127.* To estimate sexual dysfunction before and after trocar-guided transvaginal mesh surgery for pelvic organ prolapse in 105 sexually active women participating in a prospective multicenter study using the short form of the Pelvic Organ Prolapse/Urinary Incontinence Sexual Questionnaire (PISQ-12). Overall scores worsened 1 year after surgery due to a worsening of all symptoms in the behavioral-emotive and partner-related items, whereas improvements were observed in physical function. Rates and severity of dyspareunia neither improved nor worsened.

Nerve injury during uterosacral ligament fixation: a cadaver study. *Collins SA, Downie SA, Olson TR, Mikhail MS. Int Urogynecol J Pelvic Floor Dysfunct. 2009 Jan 27. Epub.* The inferior hypogastric plexus is vulnerable during uterosacral ligament fixation. Entrapment of S2 and S3 fibers could cause pain in their respective dermatomes and could be responsible for the postoperative pain described in the literature.

5 – RETENTIONS

Proposal for a urodynamic redefinition of detrusor underactivity. *Cucchi A, Quaglini S, Rovereto B. J Urol. 2009;181:225.* In male patients with nonneurological conditions, no obstruction, mainly voiding lower urinary tract symptoms and detrusor underactivity, intrinsic detrusor speed is more compromised than intrinsic strength. The definition of idiopathic detrusor underactivity of a slower and/or weaker bladder with or without poorly sustained micturition contractions is more effective than a definition of decreased detrusor contraction strength and/or poorly sustained micturition contractions. This may reflect the evolution from an initial stage to obviously impaired voiding function.

Constipation does not develop following elective hysterectomy: a prospective, controlled study. *Sperber AD, Morris CB, Greemberg L et al. Neurogastroenterol Motil. 2009;21:18.* There have been retrospective or uncontrolled reports that women develop constipation following hysterectomy. This study challenges existing data: in 132 elective surgery patients with hysterectomy compared to 123 controls there was no difference between the groups at any follow-up point in functional constipation, frequency of stools, stool consistency, straining, feeling of obstruction or need to manually evacuate stool, though many developed abdominal pain.

Reinterventions after complicated or failed STARR procedure. *Pescatori M, Zbar AP. Int J Colorectal Dis. 2009;24:87.* The stapled transanal rectal resection procedure has been suggested as a surgical option for patients presenting with evacuatory difficulty in the clinical presence of a rectocele. In 20 patients referred with 13 cases operated upon, reinterventions had to be performed for three complications and ten failures including recurrent OD, severe proctalgia, and fecal incontinence. Overall, 11 patients underwent biofeedback and psychotherapy. Only 5 patients with no psychological overlay became asymptomatic or improved. The STARR procedure requires careful patient selection to determine the associated pelvic floor pathology and pre-existent psychopathology.

Pilot study on the effect of linaclotide in patients with chronic constipation. *Johnston JM, Kurtz CB, Drossman DA et al. Am J Gastroenterol 2009; 104:125.* Linaclotide, a novel peptide agonist of guanylate cyclase-C receptors, has been shown in animal studies to stimulate intestinal fluid secretion and transit. In 42 patients with chronic constipation it improved bowel habits and symptoms, Further randomized controlled trials are warranted as this common gastrointestinal disorder has limited treatment options.

6 – INCONTINENCES

Simultaneous laceration of external iliac artery and vein complicating anterior vaginal wall sling operation for stress urinary incontinence. *Gul U, Turunc T, Yaycioglu O. Int Urogynecol J Pelvic Floor Dysfunct. 2009 Jan 27. Epub.* The needle carrier was inserted from the suprapubic area down to the vaginal lumen. The measures to be taken to avoid this life threatening complication are discussed.

Physiological, psychological, and behavioural characteristics of men and women with faecal incontinence. *Maeda Y, Vaizey CJ, Hollington P, Stern J, Kamm MA. Colorectal Dis. 2008 Oct 21. Epub.* The factors leading to faecal incontinence in males are less well understood than those in females. In this prospective study physiological, anatomical, psychological, and behavioural characteristics of male (34) and female (75) patients were compared. Nearly 40% of men with faecal incontinence report it in the absence of a definable functional or structural sphincter abnormality. Differences were observed in physiological characteristics and coping behaviours of men and women.

Post radical hysterectomy urinary incontinence: a prospective study of transurethral bulking agents injection. *Plotti F, Zullo MA, Sansone M et al. Gynecol Oncol. 2009;112:90.* Macroplastique transurethral injection can be a valid option having no surgical complications (24 patients). This therapeutic strategy is able to treat SUI and improve postoperative well being.

Radiofrequency energy delivery to the anal canal: is it a promising new approach to the treatment of fecal incontinence? *Kim DW, Yoon HM, Park JS et al. Am J Surg. 2009;197:14.* The SECCA procedure was used in 8 patients with fecal incontinence. The Fecal Incontinence Severity Index score and the Fecal Incontinence-related Quality of Life scale scale were not improved significantly and considerable complications (anal bleeding, pain, and mucosal discharge) were associated.

Effect of mode of delivery on the incidence of urinary incontinence in primiparous women. *Boyles S, Li H, Mori T et al. Obstet Gynecol. 2009;113:134.* Urinary incontinence is common in the immediate postpartum period after a woman's first pregnancy. Vaginal delivery increases the risk of urinary incontinence, but labor and pushing alone followed by cesarean delivery do not appear to increase this risk significantly.

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Classification System

Complications of vaginal supportive implants for prolapse surgery. New complications, new symptomatology, prevention and treatment

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Abstract: We describe a simple description of complications related to synthetic implants used in the treatment of stress incontinence or prolapse by the vaginal route. We describe their symptoms and propose a strategy for prevention and treatment of these complications. We will describe successively: *Type 1: defect of vaginal healing* - 1A: exposure of vaginal implant. 1B: abnormal healing: polyps, synechiae. *Type 2: infection of the implant* - 2A: persistent vaginal exposure with apparent local infection; 2B: infection along the implant; 2C: skin erosion near issue of the prosthesis, contiguous infection and fistulae along the supportive implant; 2D: contact abscess; 2E: distant abscess; 2F: fistulae; 2G: acute infection: pelvic cellulitis. *Type 3: contraction of implant* - Grade 1: palpation of supportive implant is painless, retraction moderate and asymptomatic, arm or body of the prosthesis is palpable but not thickened. Grade 2: retraction is moderate (less than about 30%) and\or without many symptoms, palpation may be sensitive, prosthesis globally moderately thickened without nodulae. Grade 3: important contraction (more than 50%) and\or painful palpation with localized thickening of the implant. 3A - important contraction, moderate symptomatology. 3B - important and symptomatic contraction. Grade 4: simple contact of implant is painful ++ even if contraction is not always palpable. *Type 4: erosions due to implant* a) erosions of vaginal fornix; b) uretral erosion; c) bladder erosion; d) rectal erosions; e) other distant erosions. This classification can only be temporary but distinguishes different types of complications too often mixed up in publications.

INTRODUCTION

Vaginal placement of synthetic meshes has become more and more popular for the treatment of genital prolapse. Recently many companies have commercialized some specific devices for this particular route. The term of meshes is probably inadequate as many devices are actually biological or both synthetic and biological. We will use here the term of vaginal supportive implants to address these material in their specific use for prolapse and incontinence surgery by thee vaginal route.

These meshes have been originally designed to be used in hernia abdominal surgery, and recently the success and good tolerance of sub urethral slings have encouraged their use by the vaginal route. Recently two companies have commercialized some specific devices for this particular route including for example The Prolift© (Ethicon Womens device as well as Apogee© and Perigee©). This specific vaginal implantation is responsible of specific complications that are not well described.

We will try here to summarize and classify these complications for future use in scientific complications.

After briefly reminding the materials and their classification we will describe complications after use of meshes, their symptoms, prevention and treatment.

We will not describe complications due to the technique itself such as dysuria or bladder instability after excessive tension of sub-urethral slings, or per operative complications as bladder perforation or major bleedings. These complications are not specific of the implant itself and should be described independently for each surgical technique.

I: CLASSIFICATION OF COMPLICATIONS due to supportive vaginal implants

Type 1: defect of vaginal healing (probably of vascular origin without infection).

1A: exposure of vaginal implant. This term should be preferred to vaginal granuloma or vaginal erosion both suggestive of infection for many surgeons.

1B: abnormal healing: polyps, synechiae.

– fortuitous, after lateral unnoticed perforation

– linked to defect of healing, median, under an incision.

Close to these, post surgical synechia or scar polyps are innocuous complications usually treated during consultation.

Type 2: infection of the implant

2A: persistent vaginal exposition with apparent local infection;

2B: infection along the implant;

2C: skin erosion near issue of the prosthesis, contiguous infection and fistulae along the supportive implant;

2D: contact abscess;

2E: distant abscess;

2F: fistulae;

2G: acute infection: pelvic cellulitis.

Type 3: contraction of implant

Grade 1: palpation of supportive implant is painless, retraction moderate and asymptomatic, arm or body of the prosthesis is palpable but not thickened.

Grade 2: retraction is moderate (less than about 30%) and\or without many symptoms, palpation may be sensitive, prosthesis globally moderately thickened without nodulae.

Grade 3: important contraction (more than 50%) and\or painful palpation with localized thickening of the implant.

3A - important contraction, moderate symptomatology.

3B - important and symptomatic contraction

Grade 4: simple contact of implant is painful ++ even if contraction is not always palpable.

Type 4: erosions due to implant

"true" erosions are distinct from "vaginal erosions" due to vaginal exposition of the implant. They occur after healing due to local friction or compression by thee implant. Erosions are exceptionally vaginal though prosthesis may be exposed or vaginal fornix wounded during intervention. Close organs can show erosions mostly due to excessive tension, sometimes with contractions. This is exceptional in our experience. But it sometimes happened years or decades after cure of herniae or promonto-fixation

a) erosions of vaginal fornix;

b) uretral erosion:

c) bladder erosion;

d) rectal erosions;

e) other distant erosions.

This classification can only be temporary but distinguishes different types of complications too often mixed up in reviews.

It will grow richer with new headings. It already helps us giving new indications of genital prolepses cure by vaginal route.

II : SYMPTOMATOLOGY OF COMPLICATIONS

Type 1A and\or 1B

can be asymptomatic, or give vaginal discharges, leucorrhoea with sometimes slight spontaneous bleeding. But in our experience, an asymptomatic vaginal erosion has a major risk of becoming symptomatic.

Type 2

Complications usually give very important leucorrhoea, yellow or brownish, with spontaneous or provoked metrorrhagias seldom painful at early stages. Fistula will always be detected between prosthesis and vagina or skin with an aperture allowing emission of pus or at least dirty looking leucorrhoea. Sometimes difficult to find (it can be millimetric), always found by careful examination eventually under anesthesia. Infected prosthesis is often encapsulated in inflammatory tissue and a painful induration may often be found during vaginal exam.

Type 3

Contractions usually asymptomatic (grade 1) may give spontaneous or stress incontinence, or even pelvic pains and, above all dyspareunias (grade 3 or 4). Prosthetic retraction may be asymptomatic but local palpation is often a little painful Palpation feels the prosthesis, hardened and crumpled under vaginal wall. At the utmost, retracted tissue around the prosthesis will feel like endometriosis nodula with contraction of vagina and near organs (grade 3b)...

Type 4

Implants can be painful at palpation even if contraction is not prominent but can still be palpable. Retropubic tracts may generate pain all along their way, often triggered by palpation of the prosthesis. Some trans obturator tracts are not really retracted but painful with dyspareunia and stress pains. Should this symptomatology go on after medical treatment, and last for more than a few months, when pain is felt touching the prosthesis at bone contact, secondary resection may become necessary.

Type 5

Symptoms are directly dependent of the affected organ Isolated vaginal erosions often but not always give vaginal discharges and metrorragia.

Urethral erosions will often give bladder urgencies, mictional sensation of burning, urethrorragia, repeated urinary infections. Urethroscopy and\or cystoscopy should be systematic.

Bladder erosions have more or less the same signs.

Rectal erosions will give rectorragia, violent colics, tenesmus. Diagnosis can be made by rectal palpation or rectoscopy.

In all these cases, complications and infection may appear if diagnosis is late. Pelvic sonography will show better than IRM or scanner the retracted implant, will measure the retraction, show the prosthetic recesses, the situation of close organs to overlook eventual per-or post -surgery complications. On the whole, any long lasting symptoms after implant of prosthesis, especially with bleeding or infection at distance of surgery must conjure up all these diagnoses and call for complete exploration under general anaesthesia if needed.

III: PREVENTION OF COMPLICATIONS DUE TO SUPPORTIVE VAGINAL IMPLANTS

Prevention of complications type 1

They did not seem to us directly infectious but linked to problem in early cicatrisation of the vaginal scar. We must try at the utmost to have neat and clean scars, respect vascularisation with a dissection not too near vaginal mucosa and a neat resection of wound banks before vaginal suture.

– Vaginal prosthesis especially synthetic one should not stay in contact with vaginal scar. The vaginal fascia can be interposed or the prosthesis buried as far away as possible from vaginal scar.

– Last, it is important not to have pointless large scars, intersecting scars, T incisions giving a high number of vaginal scars.

– vascularisation of vaginal scars may also be altered by hysterectomy with ligation of cervico-vaginal vessels.

Prevention of complications type 2

We do not know well the mechanism of these infections, their exact origin, the bacteriological type of germs Bacteriological study of infected implants may find no germs or many but with no prevailing one. Antibiotherapy cannot be an efficient prevention. Real infections are scarce with monofilamentous knitted polypropylene. We only saw one early (TVM group) cellulitis at day 7, or pelvic abscesses after surinfection of a true haematoma. We will describe later its treatment. Infection of vagina, urinary infections must be treated before any surgery. Infection is more frequent with certain materials:

– polyesters;

– silicone coated prostheses;

– micro porous synthetic mesh as Goretex.

Risks being higher with vaginal route a priori contraindicates these materials by this route (without proof of good animal and human tolerance).

Vaginal insertion of material always needs:

– good asepsy;

– strict respect of hospital hygiene rules;

– pre surgical diagnosis of local or general infection will post pone intervention;

– manipulation of implants implies changing gloves;

– insertion of implant during surgery must be as late as possible so it has shortest possible contact with surgical field;

– implants must be taken out of its wrapping as late as possible;

– implants will not be placed if there is a per surgical rectal wound.

Prevention of complications type 3

Retraction of prosthesis has no sure physiopathology, so prevention remains empiric. Symptomatology is more important when suspension of prosthesis is tight, more frequent when prosthesis is exposed. So we try:

– not to fix a prosthesis with suture thread and

– to treat quickly any exposition of prosthesis large or resisting medical treatment

– pre surgical vaginal atrophy may be treated by local oestrogens to have more supple tissues during and after surgery.

In all cases, prosthesis should not be too superficial so as not to exacerbate signs when placing it, dissection must be deep so prosthesis is not directly under vaginal mucous membrane.

Prevention of complication type 4

– any compression of adjacent organs must be avoided with

– no fixation of prosthesis by stitches

– no prosthesis placed next urethral, bladder or rectal wound.

IV: TREATMENT OF COMPLICATIONS DUE TO IMPLANTS

Type 1A

– Medical treatment

Should first be tried especially with old patients cured of their prolepsis and asymptomatic. But, in our experience, we have a high risk of secondary signs except with monofilamentous polypropylene. These complications are not surely infectious so we try and propose a simple and local disinfection and hope to enhance cicatrisation.

Treatment varies with the importance of vaginal defect. <1 cm^2: medical treatment has large chances of success. >2 or 3 cm^2 they are minimal and surgical resection must often be chosen. When there is association of vaginal infection, local antibiotics may be tried, then antiseptics or antibiotics.

– If cicatrisation of a large area is difficult, surgical resection must usually be done in operating theatre with usual asepsis measures. Technical gestures are simple but must be very cautious to avoid recurrence of deficient cicatrisation and secondary complications such as wound or secondary fistula of close organs.

Technical description: partial resection of a supportive vaginal implant

Presurgery check up

When there is big defect of vaginal healing under bladder, or the smallest doubt about bladder integrity, cystoscopy must be done. When implant is prerectal, rectal palpation must confirm rectal integrity, or even rectoscopy.

These explorations will be repeated under anaesthesia, implants seized with clamp, put under traction. Vagina is dissected around the implant and freed on half a centimeter about around the cicatrisation defect. Implant thus exposed is resected with cold scissors and surgical lancet. After incision of half the circumference about, free border of resected piece is seized with clip, traction separates prosthesis from underlying organs. Bipolar scissors may then help to dissect and coagulate without changing tool. Dissection and resection of exposed prosthesis is progressive, underlying organs are once more explored with a methylene blue test of bladder or rectum. When the wall of bladder or rectum is thin or wounded, it is classically stitched up, imperviousness then controlled. Vagina is shut " tension free " without resection.

Type 1B

Vaginal synechiae, vaginal polyps if symptomatic, may be split up during consultation.

Treatment of infectious complication type II

– Emergency withdrawal when there is infection or defect of cicatrisation of an implant "at risk";

– Medical treatment: defect of vaginal cicatrisation with monofilamentous knitted polypropylene implant;

– Partial resection of implant when defect of vaginal cicatrisation resists medical treatment or first intention defect is > 4 cm^2

Technical description: withdrawal of infected implant

Withdrawal is always easier when implant is infected, infection important and spread to the whole implant.

Presurgical check up

– When sub vesical vaginal healing is largely faulty or rectracted or when there is any doubt about vesical integrity, cystoscopy must be done.

– When supportive implant is prerectal, rectal exam must assess integrity of rectum.

Surgery begins by exploring exposition, repeating exams under anaesthesia, looking for aperture of vaginal fistula through which prosthesis might be palpable. More often, prosthesis is not palpable above all after sub urethral sling. A fine forceps will try to seize the prosthesis through aperture of fistula.

Implant is then gently and slowly tracted along its initial axis. It is easier when prosthesis is infected or encapsulated in an infectious shell. Implant will slowly and totally appear through the fistula if infection is diffuse all along its length, when there is a near abscess, a cutaneo vaginal fistula or cellullitis.

When infection is only partial with trans ligament, trans obturator or transperineal passage, infection often does not diffuse beyond the ligament. Retroligamental, transperineal or transobturator part of the implant remains firmly fixed. Progressive traction will break prosthetic arm near this zone. Ablation must be as complete as possible to avoid relapse. Dissection, as large as possible, tries to seize the implant next to obturator membrane or next to ligament of insertion. If prosthesis comes off at once exam must control its whole length is present. If not, dissection must go on until complete resection of the prosthesis sometimes changing route of access. If implant breaks and part of it is still palpable in dissection space it must be seized with the same forceps to realize total ablation of the material.

– Total resection: when intervention is finished, it is not necessary to close vagina, post surgical drainage will be better.

– Subtotal resection usually when implant is not totally infected. Resection of infected zone up to obturator membrane is usually enough for secondary cicatrisation. But the risk, at least theoretical, of distant relapse needing emergency consultation must be known by the patient.

– When supportive implant is synthetic, it is large and resection difficult. Intervention will use about the same technique as large resection for retraction we are going to describe.

Treatment of complications type III: retractions, pains

Prosthetic retractions need surgery when they are a discomfort for patients. They are often difficult to treat. There is no relapse of prolepsis, other organs are near. Partial ablation is usually enough, resection of lateral prosthetic arms hazardous by routine route . Large resection of median part of the prosthesis, as near the prosthetic arms as possible may reduce pain.

Technical description: complete resection of a supportive retracted implant

First surgical time is infiltration of retracted zone after palpation, deep between implant and nearest organ that is rectum. Rectal exam rules out rectal erosion of implant. Medial incision of vagina follows the first incision done when fixing the prosthesis. Between implant and vagina, dissection is easier after infiltration and usually easy at contact of implant seized with serrated dissecting forceps. Same is done between implant and rectum if there is ablation of anterior implant. After dissection, arms of the implant may be seen, only remaining suspension points which must be cut as far as possible.

Resection of sub urethral portion of a sub urethral implant

If it is done for pain, sling must be resected in the zone painful at palpation. Vaginal route may be complemented by laparotomy or laparoscopy to resect retro pubic portion of sling, or by trans obturator route if the sling goes through obturator. Resection may be done for a complication due to excessive tension; dysuria, bladder instability or such as dyspareunia. Resection technique is the same but, when tension is excessive, the sling stuck under urethra, access may be difficult and lateral approach better with less risk of urethral wound. Transverse incision will be done facing the implant. The implant is located by vaginal palpation or a transurethral sound giving a "striction" sign. Regular palpation of the sling guides dissection until contact. It is then seized by a forceps, tightened and dissected at vaginal contact, going towards urethra but cautiously not to injure it. Dissection then goes round the other side for complete resection of the sling. Before closing vagina, integrity of urethra is controlled by urethral sound or urethroscopy. Any injury needs strict suture in two plans of wounded vagina and its fascia for a lesser risk of secondary fistula.

Treatment of complications type IV: erosions of near organs

These complications remain exceptional if none of these organs has been wounded during surgery. Any pre surgical suspected lesion must be confirmed: Cystoscopy for bladder lesion, urethroscopy for urethral lesion, rectal palpation and rectoscopy for rectal lesion. Pelvic and perineal sonography may be very useful. Treatment is large resection of prosthesis as described above.

CONCLUSION

Supportive vaginal implants, for cure of urinary incontinence or genital prolapse, have specific complications that could be lessened by prevention. Any surgeon using these products should know early treatment and specific strategies.

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Anal sphincter structure and function relationships in aging and fecal incontinence. *Lewicky-Gaupp C, Hamilton Q, Ashton-Miller J et al. Am J Obstet Gynecol. 2009 Jan 9 Epub.* Thickening of the internal anal sphincter occurs with aging. Thinning of the external sphincter and a corresponding drop in squeeze pressure correlated with fecal incontinence but not aging. Rectal hypersensitivity is associated with fecal incontinence rather than aging and may play a role in the mechanism of fecal incontinence.

A longitudinal population-based survey of urinary incontinence, overactive bladder, and other lower urinary tract symptoms in women. *Wennberg AL, Molander U, Fall M et al. Eur Urol. 2009 Jan 13 Epub.* Female urinary incontinence, overactive bladder, and other LUTS are highly prevalent conditions with a few studies describing progression as well as remission in the short term, and no long-term longitudinal studies. UI and other LUTS constitute dynamic conditions, a marked overall increase in the prevalence of UI, OAB, and nocturia in the same women from 1991 to 2007 was observed. Both incidence and remission of most symptoms were anyway considerable.

7 – PAIN

Depressive symptoms, anxiety, and quality of life in women with pelvic endometriosis. *Sepulcri Rde P, Amaral VF. Eur J Obstet Gynecol Reprod Biol. 2009;142:53.* Of 104 women diagnosed with pelvic endometriosis 86.5% presented depressive symptoms and 87.5% with a positive correlation with current pain intensity. A rational approach to endometriosis should include an evaluation of the emotional profile and quality of life reducing the functional damage caused by the disease.

Treatment of fibromyalgia syndrome with antidepressants: a meta-analysis. *Häuser W, Bernardy K, Uçeyler N, Sommer C. JAMA. 2009;301:198.* Fibromyalgia syndrome is a chronic pain disorder associated with multiple debilitating symptoms and high disease-related costs. Antidepressant medications are associated with improvements in pain, depression, fatigue, sleep disturbances, and health-related QoL.

Study on personality and psychiatric disorder in fibromyalgia. *Rose S, Cottencin O, Chouraki V et al. Presse Med. 2009 Jan 22 Epub.* The importance of psychiatric comorbidities, with mental and personality disorders in patients with fibromyalgia has to be taken into account in the treatment by their impact on doctor-patient relation.

Macrophages and nerve fibres in peritoneal endometriosis. *Tran LV, Tokushige N, Berbic M et al. Hum Reprod. 2009;24:835.* Endometriosis is considered to be an inflammatory disease, and macrophages are the most numerous immune cells in endometriotic lesions possibly playing a role in the growth and development of the lesions and in the generation of pain through interaction with nerve fibres.

Results of lateral internal sphincterotomy with open technique for chronic anal fissure: evaluation of complications, symptom relief, and incontinence with long-term follow-up. *Kiyak G, Korukluoglu B, Kusdemir A et al. Dig Dis Sci. 2009 Jan 1 Epub.* In this clinical prospective study on 129 patients the complication rate was 41.8%, urine retention being seen frequently in males. Postoperative incontinence was only to flatus and two-thirds of these resolved in 6 weeks. No patient showed improvement of incontinence after 6 weeks.

A randomized clinical trial for women with vulvodynia: Cognitive-behavioral therapy vs. supportive psychotherapy. *Masheb RM, Kerns RD, Lozano C et al. Pain. 2009;141:31.* Psychosocial treatments for vulvodynia are effective. Cognitive-behavioral therapy, a directed treatment approach that involves learning and practice of specific pain-relevant coping and self-management skills, yielded better outcomes and greater patient satisfaction than a less directive approach like supportive psychotherapy.

Pain sensitivity and analgesic effects of mindful states in Zen meditators: a cross-sectional study. *Grant JA, Rainville P. Psychosom Med. 2009;71:106.* Zen meditators have lower pain sensitivity and experience analgesic effects during mindful states. This may reflect cognitive/self-regulatory skills related to the concept of mindfulness and/or altered respiratory patterns. Prospective studies investigating the effects of meditative training and respiration on pain regulation are warranted.

Antecedent nonbladder syndromes in case-control study of interstitial cystitis/painful bladder syndrome. *Warren JW, Howard FM, Cross RK et al. Urology. 2009;73:52.* Probing for clues to the pathogenesis of interstitial cystitis/painful bladder syndrome (IC/PBS) antecedent nonbladder syndromes were found: fibromyalgia-chronic widespread pain, chronic fatigue syndrome, sicca syndrome, irritable bowel syndrome, migraine, chronic pelvic pain, depression, allergy. Most syndromes appear in clusters. Among the hypotheses generated is that some patients with IC/PBS have a systemic syndrome and not one confined to the bladder.

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Case report

Intestinal and pelvic endometriosis: psychological and surgical considerations

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Abstract: Bowel endometriosis is an uncommon disease that can provoke severe symptoms including intestinal obstruction. The disease generally affects young women, and, often has psychological implications since it is associated with severe pain and infertility. Our patient, a 40-year-old woman, suffered from rectal bleeding, dysmennorrhea, and episodes of intestinal obstruction, as well as anxiety and depression documented by various psychological tests. Surgery revealed a tumor-like mass below the rectosigmoid junction and endometriotic nodules on the right ovary and ileum. The rectosigmoid colon was resected and the nodules removed. The patient, who had an uneventful postoperative course, is currently in good physical condition but is still depressed and receiving hormonal therapy and psychotherapy. A combined surgical-psychological-hormonal approach may be the most effective way to treat intestinal and pelvic endometriosis.

Key words: Endometriosis; Rectosigmoid colon; Intestinal resection; Ovarian cyst; Psychological evaluation; Trait and state anxiety; Drawing tests.

INTRODUCTION

Endometriosis occurs in women of reproductive age and is most commonly found on the peritoneal surface of the reproductive organs. The prevalence of the disease in the general population ranges between 1 and 8% according to different series.[1] The intestine is affected in less than 20% of patients suffering from gynaecological endometriosis. In the rectosigmoid colon, endometriosis may mimic either a neoplasm or inflammatory bowel disease and may even cause intestinal obstruction.

Nearly half of the patients with endometriosis suffer from infertility due to hormonal defects. The hormonal treatment of the disease prevents pregnancy. This problem, together with symptoms, such as severe pelvic pain, may affect the patient's quality of life and cause mental illness.

The aim of this paper is to report a case of large bowel endometriosis unusually affecting the entire circumference of the rectum and causing anxiety and depression. Major surgery and psychotherapy were needed to successfully treat the patient.

CASE REPORT

Clinical History: A 40 year-old woman was referred to our Unit after several episodes of intestinal obstruction. She had a long standing history of severe pelvic pain during her menstrual periods and was diagnosed with pelvic endometriosis at age 25. After she married she attempted to become pregnant without success.

She had undergone two operations for ovarian endometriosis cysts in the past 7 years and complained of rectal bleeding and constipation during her menstrual period. A colonoscopy showed a tumour-like mass below the rectosigmoid junction, 9 cm above the anal verge. Anorectal and trans-vaginal ultrasound were performed by means of a B & K machine (Bruel & Kiaer, Aarhus, Denmark) using a 10 mHz probe and did not show any localization of endometriosis within the anal canal and lower to middle rectum; the recto-vaginal septum was free of disease; the anal sphincters were intact.

Psychological History: The patient had been adopted when she was 1 week old. She never knew her biological parents and her adoptive father died 3 years before she was admitted to our Unit. She expressed the strong desire to know her natural parents and was taking oral antidepressant drugs (Duloxetine, 30 mg twice a day).

She had a psychological consultation at our Unit and underwent STAI X1 and STAI X2 tests (C.B.A.) aimed at evaluating her state and trait anxiety levels.[2] A significant trait anxiety level of 79.8 (normal value below 50) was found. State anxiety level was 47.9 (normal value below 50). These findings suggested that anxiety was a stable trait of her personality. The Depression scale consisting of 24 items was administered,[3] which showed a significant depression reaching a score of 87.6 (normal value below 50).

Others tests were administered, such as the draw-the-family-test,[4] the rain-test, the tree-test,[5] and the draw-a-person-test [6] aimed at evaluating socio-emotional adaptation (Figs. 1, 2). These tests showed that the patient had difficulties with interpersonal relationships, a high insecurity and immaturity levels, and poor emotional defences.

Surgical Intervention: Once the peritoneum was opened, an obstructive hard whitish mass was found below the rectosigmoid junction, without any significant dilatation of the proximal sigmoid. An endometriosis cyst of the right ovary, 2 cm in diameter, was coagulated with diathermy. A rectosigmoid resection was carried out with preservation of the superior rectal artery and a latero-terminal anastomosis was performed at 8 cm above the anal verge using a 29 mm circular stapler (Ethicon Endosurgery, Cincinnati, Ohio, USA) (Fig. 4). The specimen was sent to the pathologist and a typical histology of rectal endometriosis was found. Two smooth and soft chocolate-like nodules, 1 cm in diameter, were found and excised by diathermy at the level of the terminal ileum, and then sent to the pathologist, who diagnosed them as endometriosis cysts.

The postoperative course was uneventful. At 4-month follow-up the patient is in good physical health, her bowel motions are normal and she has no anal incontinence and no constipation, but still has marked symptoms of depression. She is receiving psychological counselling and has begun hormonal therapy aimed at preventing disease recurrence.

DISCUSSION

Psychological Considerations: Endometriosis is a disabling illness that affects about 8 million women worldwide.[7] Endometriosis compromises a woman's quality of life. Maintaining a regular job, or getting pregnant can be

Fig. 1. – The rain-drawing test shows that the patient is lacking of defence, as she did not drew any umbrella to protect her body under the rain. The attempt of the patient to stop the rain with her arms makes the drawing rather dramatic.

difficult. Dyspareunia during sexual intercourse is common. Chronic pelvic pain causes emotional and behavioural changes. Symptoms may be both vegetative and cognitive-affective; so because of pain caused by endometriosis the role of the woman changes both within her family and within society.[8]

Women who suffer from chronic pelvic pain frequently have abnormal psychological profiles, that can include a history of depression and/or a difficult family life. Moreover, the degree of pain reported by women with endometriosis is frequently not related to disease severity.[9] Therefore the success or failure of treatment for pain due to endometriosis depends on a multidisciplinary approach.

Chronic pain does not resolve completely with pharmacological treatment and causes psychological disorders, mostly depression. Patients are likely to complain that nobody

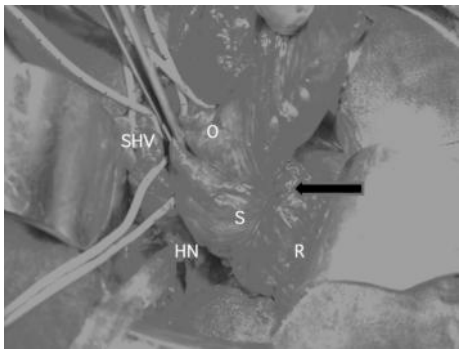

Fig. 3. – Intra operative field showing stenosis tumor-like endometriosis just below the rectosigmoid junction the surgical dissection has been commenced and some structures has been mounted on rubber band loop i.e. Ovarian vessels , SHV superior haemorroidal vessels which has been separated to ensure a better vascular supply, HN Hypogastric Nerve, S sigmoid, R rectum.

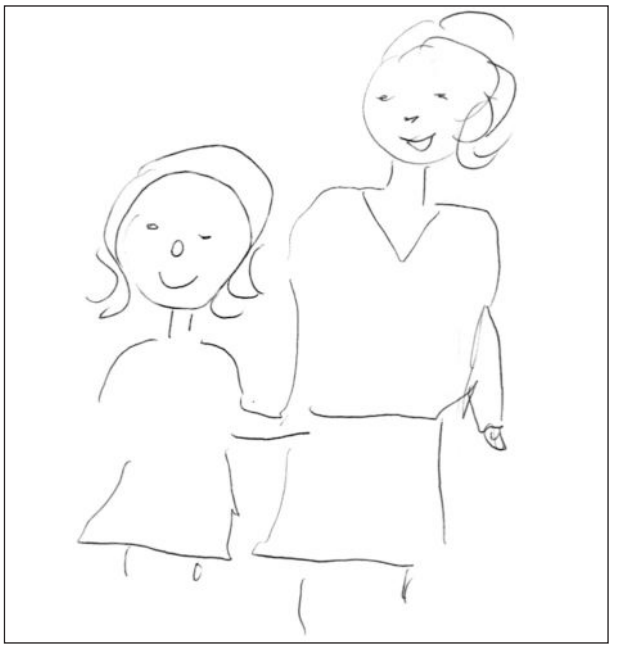

Fig. 2. – The draw-the-family test shows several abnormalities, such as wavering lines and body deformities (i.e. lack of legs and arms). Moreover the patient, despite being 40 years old, represented herself as a child which means she felt frail and insecure.

can understand their painful and troublesome condition. Endometriosis may frustrate a patient's expectations, cause loss of self-esteem, alteration of body image and, ultimately, social isolation. The patient may at first feel anger, which if not treated is likely to develop into aggressive behaviour, and increase the patient's social isolation.

Our patient presented many findings of the psychological pattern that has been associated with endometriosis i.e depression and difficulty in coping with her disease. She not only believed her problem was undervalued, but had an egodystonic body image, and trouble maintaining relationships.

Surgical considerations: Endometriosis, originally described by Rokitansky in 1860, is found in 1-8% of the general population and up to 35% of patients suffering from infertility. The first widely accepted theory regarding the pathophysiology of endometriosis was proposed by Samp-

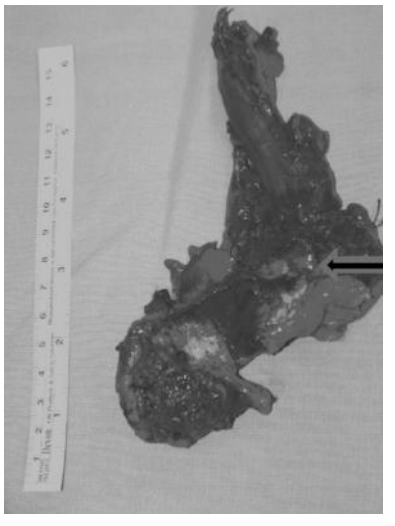

Fig. 4. – The specimen shows a tumor like-endometrioma in the upper rectum (arrow) with dark and whitish areas.

son, in 1922.[10] The prevalence of intestinal endometriosis is 5.4% to 25.4%. The most common location is the rectosigmoid colon; over 65% [11, 12] of these cases are located in sigmoid colon, followed by the rectum, ileum, appendix and cecum. The typical symptoms of rectal endometriosis are dysmenorrhoea, dyspareunia, cyclical rectal bleeding,[13] and intestinal obstruction due to the circumferential involvement of the rectum.[14]

Preoperative imaging of the pelvis in general and the rectum in particular is difficult but important for planning surgery. There are multiple techniques for this purpose. Magnetic resonance imaging (MRI) has a sensitivity of 80% and specificity of 90% for the evaluating rectal endometriosis.[15] The sensitivity of transvaginal ultrasound in identifying endometriosis in the muscular layer of the rectum is 100%, specificity is 85.7%, positive predictive value is 91%, and negative predictive value is 100%. Endorectal ultrasound has sensitivity of 97%, and specificity of 97%.[16,17] The endorectal ultrasound evaluation of our patient was normal. This suggests that although it has an elevated sensitivity and specificity, this diagnostic tool also produces false negatives probably related to the depth of the lesion. Colonoscopy is positive just in 10%-12% of cases because of the histological characteristics of endometriosis.

As in other painful conditions that can cause psychological distress, e.g. diverticular disease,[18] the treatment of large bowel endometriosis should be multidisciplinary. In addition to a skilled gynaecologist and colorectal surgeon, an expert psychologist is needed, because many of these patients have some degree of psychological distress due primarily to infertility.

In cases with rectal involvement there is evidence that endometriosis lesions are not just confined to the mucosa; in 36% of rectal specimens the lesions also involves the sub mucosal plane and in less than 12% of cases also is located at the mucosal plane.[19] This has some important implications regarding the removal of affected tissue. In cases where the disease involves small bowel and large bowel, as well as the rectum all affected tissue is, unfortunately, not always removed because many of patients are only evaluated and treated by gynaecologists, most of whom do not feel comfortable managing the dissection, resection and anastomosis of the colon and rectum.

A recent study by Brouwer et al.,[20] the largest series on treatment of rectal endometriosis, describes a 10-year experience with a total of 203 patients with rectal endometriosis who were treated surgically. One hundred seventy-three patients required segmental resection of the rectum, but on analysis there were no preoperative factors predicting the need of segmental resection of the rectum in the absence of symptomatic rectal obstruction. This illustrates the importance of participation of a colorectal surgeon in the management of the case when the suspicion of rectal involvement is high. Those patients who were candidates for segmental resection had on overall morbidity of 11%, the same morbidity on follow-up, and 9% reported gastrointestinal symptoms, most commonly frequency and urgency.

In conclusion we believe that prompt, radical surgical intervention, aimed at preventing rectal stricture and intestinal obstruction, with a multidisciplinary approach including psychotherapy, is the basis of a correct management of patients suffering from intestinal endometriosis.

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Original article

The use of flow equation in functional coloproctology: a new theory in anorectal physiology

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Abstract: The flow equation and hybrid law in coloproctology can be used to understand normal anorectal physiology and accordingly explains the controversies experienced due conflicting research data. It can be used equally for incontinence and constipation. According to the flow equation there are four primary mechanical factors maintaining continence and achieving unobstructed defecation, namely intra-rectal pressure (IRP), dynamic viscosity of the bowel contents, anal canal length and diameter. The last 3 factors are responsible for the anal canal resistance (ACR). All other factors are secondary, and working through one or more of the four primary mechanical factors each can be numerically calculated separately. The sensory and reflex components work through the IRP and ACR respectively, and can also be numerically calculated in each individual. This data can be used to plan treatment and predict outcomes. Calculation of the ACR before and after treatment is helpful when making an objective evaluation of different treatment modalities. If different treatments are given to the same patient, e.g. combining constipating agents with sensory biofeedback for the rectum and anal sphincter repair, each modality may affect a different component in the flow equation, such as the dynamic viscosity, IRP, anal canal length or diameter. Calculation of the ACR also gives new insight into the results of modifying existing treatment modalities or creating new ones. Norm-grams and the automated flow calculator were designed to illustrate and avoid tedious calculations and they suggest a final diagnosis for each patient.

Key words: Flow equation; Hybrid law; Intra-rectal pressure; Anal canal resistance; Incontinence; Constipation.

INTRODUCTION

The anorectum is a physiologically highly integrated segment of the bowel. The mechanical factors modified by sensory and reflex components are integrated instantaneously in order to initiate normal defecation within a few seconds and to maintain continence within a fraction of a second. This highly integrated nature may be responsible for the lack of answers to the enigmatic question of how the anal sphincter works. This enigma exists despite the availability of an enormous pool of research data where many different factors have been considered in an unintegrated approach. The use of the flow equation for mathematic integration of anorectal physiology first appeared in the international literature in 1998 when Farag attempted to answer the above-mentioned question and standardize our approach to functional colorectal disorders.[1]

THE RESISTANCE AND FLOW EQUATION IN FUNCTIONAL COLOPROCTOLOGY

The flow equation called the Hagen-Poiseuille law was originally designed to study the flow of newtonian fluids, such as water, in rigid tubes. Poiseuille had used the equation successfully in the study of blood flow (a non-newtonian solution).[2, 3] Newtonian fluids had been defined as those fluids that have a constant dynamic viscosity at different rates of flow.[3] The flow equation for the newtonian fluids can be used for non-newtonian fluids, e.g. stools, if their shear stress equals zero.[2, 3] According to Douglas et al, the Hagen-Poiseuille law can be used for gases and solids, which behave like a very low viscosity, and a very high viscosity fluids respectively.[4] The resistance of the anal canal to flow had been suggested by many authors to be a more important factor in maintaining continence than the ability of the muscles to squeeze around the anal canal. However trials to measure the anal canal resistance mechanically using probes, catheters, small balloons and obturators were unsuccessful.[5, 6, 7, 8] Recently the resistance and flow equations had been applied to the field of functional coloproctology for a mathematically integrated approach of anorectal manometry and defecography.[1] According to the flow equation, constipation can be defined as a low flow state during defecation while anal incontinence (AI) can be defined as abnormal flow of bowel contents through the anal canal during rest or squeeze where (Flow = Pressure/Resistance). Accordingly the recto-anal interaction is a pressure/resistance interaction rather than pressure/pressure interaction. The anal canal resistance is directly proportionate to dynamic viscosity (DV) or consistency of stools and anal canal length (ACL), and inversely proportionate to anal canal resistance.[4]

Conforming to the flow equation, four primary mechanical factors affect the anal continence and defecation in health and disease, namely:

1. intra-rectal pressure (IRP).
2. dynamic viscosity of the stools (DV)
3. anal canal length (ACL).
4. anal canal diameter (ACD).

$$\text{AC Resistance} = \frac{128 \times \text{Dynamic Viscosity} \times \text{ACL}}{3.14 \times (\text{ACD})4}$$

The flow equation will be finally as follows:

$$\text{Flow} = \text{IRP} \times \frac{3.14 \times (\text{ACD})^4}{128 \times \text{DV} \times \text{ACL}}$$

Other mechanical factors are secondary factors operating through one or more of the above mentioned primary factors. Type of food intake, amount of fluids ingested, rate of gastric emptying, small and large bowel absorption and motility, work through the dynamic viscosity factor. Rate of rectal filling, rectal capacity and rectal compliance work through the factor of IRP, while the pelvic floor muscles, anal sphincters and pelvic supporting connective tissue and fascia work through the factors of ACL and ACD. Sensory and reflex factors are known to intimately interact with the mechanical factors in order to maintain normal continence. Both work through the flow equation by determining which IRP interacts with which anal canal dimensions (length and diameter), at any given time sensory factors principally affects IRP due to delayed sensations (Fig. 1). Reflex factors work mainly by determining which anal canal dimensions (and hence resistance) are challenged by intra-rectal pressure during rest or squeeze for incontinent or during defecation for constipated patients. Each of the sensory or reflex factors can be

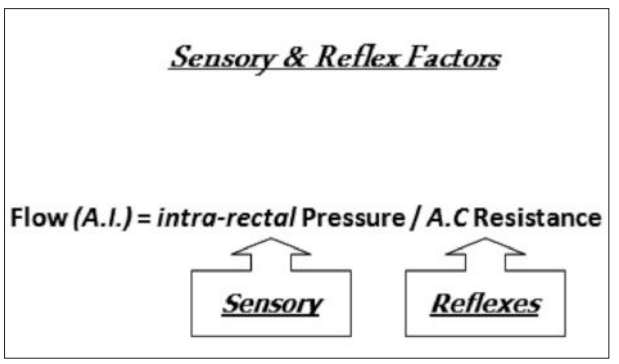

Fig. 1. – The interaction between the Mechanical, Sensory and Reflex Factors in the Anorectal Segment.

numerically quantified individually or in conjunction with other factors.

According to the resistance equation the anal canal resistance increases as the DV (consistency) of the stools increases, and this explains why the anal canal resistance to gas is lower than its resistance to fluid stools which is in turn is lower than its resistance to soft well formed stools.

If we use the DV to air, water and soft well formed barium sulphate paste at normal body temperature as representative to the DV to bowel gas, watery stools and normal well formed stools respectively, the anal canal resistance at any given situation is 1:38:69 respectively for gas, fluid stools and formed stools respectively. This numerically explains why the AI to gases is the easiest to occur and the last to be regained in those patients. It also explains the beneficial effect of constipating agents in patients with AI by increasing the DV (consistency) of the stools which proportionately increase the anal canal resistance.

The above mentioned fact also explains why hard stools are difficult to evacuate even in normally functioning anal canals and can explain the beneficial effect of laxatives in obstructed defecation in any particular patient by decreasing the DV of stools which proportionately decrease the anal canal resistance during defecation.

This beneficial effect of laxatives and constipating agents can also be quantitated numerically using the rotational viscometer and the flow equation in any given patient (Vide infra). Similarly according to the resistance equation the anal canal resistance is directly proportionate to ACL and inversely proportionate to the ACD. The later is the most important determinant of anal canal resistance than ACL and DV of stools being raised to the power four.

This confirms our general knowledge concerning the need of the anal canal to dilate and shorten in order to achieve normal unobstructed defecation. It also explains the findings of other authors that the postoperative ACL was the only statistically significant manometric parameter in predicting the functional outcome of anal sphincters repair for patients suffering from AI using logistic regression analysis.[9]

Measuring the anal canal resistance in an average control subject using lateral defecographic views on regular abdominal films during rest, squeeze, and defecation (Fig. 2), revealed an 8 fold increase in anal canal resistance during squeeze in order to overcome the tendency to increase the flow through the anal canal due to an increase in the intra-rectal pressure during urgency or coughing or Valsalva's maneuver. While during defecation this resistance is voluntarily decreased by 88 times as compared to AC resistance during rest in order to allow for normal unobstructed defecation.

In this control subject the anal canal resistance can be altered over a range of 704 folds (i.e. 8×88), from full contraction during maximum squeeze to full relaxation during defecation.

This interesting versatility of the anal canal resistance can only be achieved by muscular tissue which has a resting tone and can actively contract and actively relax maintaining continence during rest and during squeeze while allowing for normal unobstructed rectal evacuation during defecation. This function cannot be achieved by any other natural or synthetic tissue as effectively as the muscular tissue. It can also explain the role played by the anal sphincters being made of a combination of involuntary muscles [longitudinal muscle layer and internal anal sphincter (IAS)] which maintain continence during rest by the basal tone of the IAS, and the voluntary muscles: the external anal sphincter (EAS) maintains the anal canal diameter (primarily) and anal canal length (secondarily) by its basal tone during rest and by its contraction during squeeze, and the puborectalis muscles which work by upwards and forward pull of the ano-rectal junction maintaining anal canal length (primarily) and anal canal diameter (secondarily), by stretching the anal canal during its contraction during squeeze aided by the visco-elastic properties of the anal canal (Tab. 1). A marked decrease in the visco-elastic properties of the anal canal e.g. due to irradiation may attenuate the puborectalis ability to increase the ACL and decrease ACD in addition to the damage to the anal sphincter and the rectum.

The role of the longitudinal muscle can only be appreciated by looking at its unique attachment like strands of a tent passing through the lower part of both IAS and EAS to the perianal skin and to the anoderm anchoring both muscles to the skin and anoderm. If absent, upward recoil of both sphincters can lead to anal canal shortening-widen-

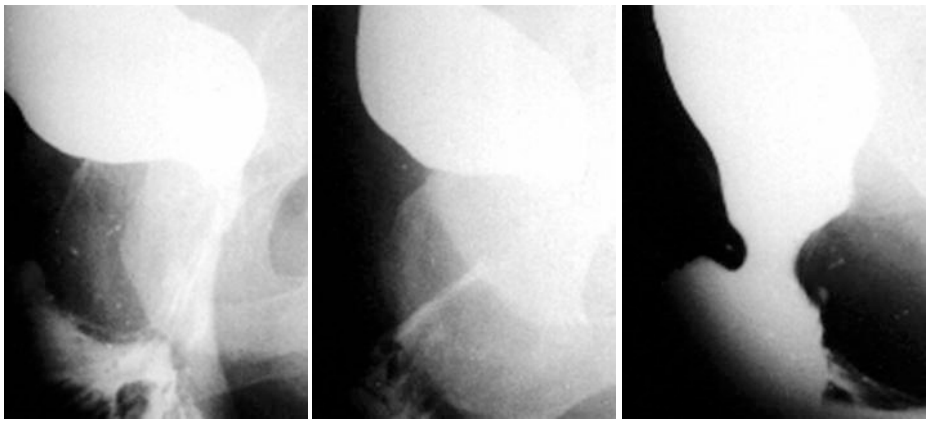

Fig. 2. – The Anal Canal Resistance as they appear on defecography during Rest, Squeeze and Defecation.

TABLE 1. – *Anatomic-function relation-ship according to the flow and resistance equations where the ACL and ACD represent the final common pathway for action of the anal sphincters.*

	Longitudinal muscle	***Internal analsphincter***	***External anal sphincter***	***Puborectalis muscle***
A.C. Length	It forms a supportive framework for the IAS and EAS during rest and squeeze. It decreases the A.C length during defecation	Maintains A.C.L. during rest (+)	Maintains ACL during rest (+) and during Squeeze (+)	Maintains ACL during rest (++) and during squeeze (++) and relaxes to increase ACL during defecation (++)
A.C. Diameter	Forms a supportive framework for the IAS and EAS during rest and squeeze. Opens the A.C. during defecation [10]	Maintains A.C.D. during rest (++) and relaxes to increase ACD during defecation (++)	Maintains ACD during rest (+), decreases ACD during squeeze (++) and relaxes to increase ACD during defecation (++)	Maintains ACD during rest (+) and during squeeze (+) and relaxes to increase ACD during defecation (+)

ing (incontinence) which will not be helped much by contraction of the puborectalis pulling the anorectal junction upwards and forwards, and will lose some of its effect on anal canal dimensions.

This effect theoretically may be responsible for AI after operations for anal fissures or low fistulae if they cut the longitudinal muscle attachments at certain critical points in otherwise previously normal anal canal muscles.

The resting tone of the EAS may be due to its vertical stretch between the pulling puborectalis while its lower part being fixed to the perianal skin by the longitudinal muscle unique attachment. This continuous mechanical stimulation is akin to the theory of dynamic graciloplasty where continuous electric stimulation of any skeletal muscle will increase the percentage of the slow twitch fibers in the muscle from 10-45% which lead to its fatigue resistant properties of the muscle and its resting tone contrary to all other skeletal muscles. In fact the puborectalis also is always mechanically stimulated by the weight of the rectum and its fixation to the presacral fascia in the traditional anatomic theory or by the levator plate and the uterosacral ligament in the musculo-elastic theory.[10] In fact division or loss of function of the puborectalis may also lead to a decrease in the EAS function through the loss of its physiological vertical stretch and hence the loss of some of its resting tone. This may lead to shortening and widening of the EAS and decompensation of the voluntary continence mechanism. On the other-hand division of the EAS will not lead to puborectalis dysfunction. This suggests that the puborectalis is the main muscle of continence while the EAS provides a functional reserve. The role of pelvic fascia for the proper function of the pelvic floor muscles and anal sphincter as was suggested by Petros and Swash,[10] sounds physiologically convincing because muscles has to gain fixed attachment to bones or strong fascia in order to maintain its physiological optimum length for optimum function (Starling's law). Loss of fixed attachment by laxity or injury to the ligaments will reduce the functionality of the muscles.

The upwards and forward pull of the anorectal junction by the puborectalis muscle will keep the longitudinal muscle ani (LMA) over-stretched as it is tethered to the perianal skin by its filamentous attachments, so that it stays in a state of isometric contraction during rest and squeeze. Embryologically the LMA and the IAS are a continuation of the bowel wall telescoped inside the puborectalis and EAS. The action of the LMA and the IAS occurs in two stages:

1. Stage of receptive relaxation: as the stools reaches the lower rectum both muscles relax aided by the relaxation of the puborectalis muscle which allows the relaxed EAS to flip outwards and shorten to facilitate unobstructed defecation;

2. Stage of isotonic contraction: this occurs during the stage of actual flow when the anal canal shortens vertically and becomes everted over the passing stools. According to the musclo-elastic theory [10] the LMA contracts to pull the recto-vaginal septum and perineal body in order to keep the anal canal open during defecation. This action most probably takes place at this stage of actual flow.

The flow equation can suggest answers to many controversial issues in functional coloproctology where the anorectal angle does not show itself as a primary factor in maintaining anal canal resistance. It is most probably an indication of a properly functioning puborectalis muscle which works by increasing the anal canal length and decreasing anal canal diameter through elongation and stretching the anal canal by the upwards and forward pull of the muscle aided by the visco-elastic properties of the anal canal and its surrounding muscles. The reverse action occurs during defecation through relaxation of the puborectalis muscle.

Similarly, perineal descent does not affect the equation directly. However it may represent pelvic floor weakness which yields excessively under straining. In the same way weak abdominal wall muscles form a big belly as a result of increasing intra-abdominal pressure.

THE HYBRID LAW IN COLOPROCTOLOGY

Since two thirds of continent individuals still have normal anal pressures as seen on manometry, and two thirds of patients with AI have low anal pressures, can the anal canal pressure show itself in the flow equation?

As was suggested by other authors, the anal canal pressure measured is the resistance of the anal canal to distension by the measuring probes and is proportionate to the probe diameter.[11]

According to the law of Laplace:

$$\text{Wall Tension (T)} = \frac{\text{Distending Pressure (p)} \times \text{Radius (R)}}{\text{Anal Canal Wall Thickness } (\delta)}$$

$R\ (\text{Inside}) = T\delta/P$ Since the $ACD = 2R$

Compensating for the ACD in the Flow equation with the Laplace's Law:

$$\text{Flow} = IRP \times \frac{3.15\ (T\delta)^4}{8 \times DV\ \ (ACP)^4}$$

This equation suggested by Farag [1] in 1998 was named the Hybrid Law in coloproctology, where: from the Hybrid law, the anal canal pressure is inversely proportionate to flow as is known in the literature and the anal canal wall tension increases as the flow increases i.e. during defecation, where

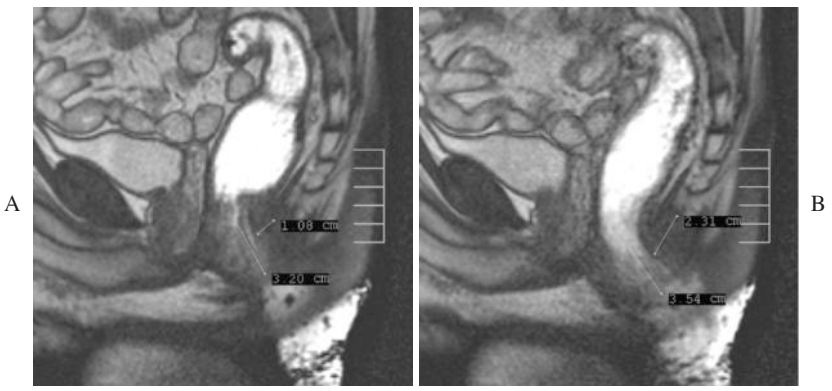

Fig. 3. – The measurement of the Anal Canal Sphincter Thickness during the beginning = 1.08 cm (A), and at the Zenith of Defecation = 2.31 cm (B).

anal fissure can happen and anal suppurations can start at the anal crypts. According to the equation, contrary to our believes, the anal canal wall thickness has to increase being proportionate to flow in order to protect the anal canal against the increasing wall tension, a physiologic necessity and priority in all the GIT to avoid bowel wall rupture. Our unpublished data in well selected controls proves this (Fig. 3). Failure of the anal sphincters to increase in thickness due to either a congenitally thin sphincter or due to opening of the anal canal by effacement-thinning of the relaxed EAS, (like the cervix uteri during delivery), rather than upwards and outwards recoil of the EAS by puborectalis relaxation and its active relaxation, may be responsible for the development of hemorrhoids in the submucosa in those predisposed to this disease in order to increase anal canal wall thickness in an attempt to minimize the wall tension. Those predisposed to anal fissure will develop cracks while the rest may develop anal suppuration. Anismus may also develop in a desperate attempt to increase the anal sphincter thickness by spasm.

Calculation of resistance and flow in health and disease

$$\text{AC Resistance} = \frac{128 \times DV \times ACL}{3.14 \times (ACD)^4}$$

$$\text{Flow} = IRP \times \frac{3.14 \times (ACD)^4}{128 \times DV \times ACL}$$

The Intra-rectal pressures are measured manometrically in K Pascal where: 100 mmHg = 13.3 K Pascal. ACL and ACD are measured from the lateral defecographic views in meters. Dynamic viscosity of bowel gas, liquid stools and soft well formed stools were approximated to that of:

air = 0.00001905 Kg m^{-1} s^{-1}, water = 0.000723 Kg m^{-1} s^{-1}

Barium sulphate paste = 0.0013092 Kg m^{-1} s^{-1}, flow index = liter/s. (×1000 = cc/s.) Recently those calculations can be done using an automated calculator available on the following address: http://www.integratedcoloproctology.com/cald.htm

FLOW INDEX IN CONSTIPATION

Using a fixed dynamic viscosity for air, water and barium sulphate, only 3 measurements are done in calculation of flow index in constipated patients:

1. mean IRP during defecation as measured using computerized anorectal manometry (mmHg)
2. ACL (cm)
3. ACD (cm).

Both 2 and 3 are measured from the lateral defecographic views on a standard abdominal film. A flow index of 1cc/sec was taken as well as a cutoff point between obstructed defecation (flow < 1 cc/sec) and unobstructed flow in the absence of anatomical obstruction or excessively hard stools during defecation. The mathematically calculated flow was taken as a flow index rather than an accurate measurement of flow in order to avoid minor corrections on the native equation. Norm grams representing the flow equation during defecation and continence had been plotted in order to facilitate understanding the flow equation and allocation of individual patients as a rough though rapid substitute for suggested mathematical calculations.

Defecation Norm Gram (Fig. 4)

The four primary factors involved in the flow equation could be successfully plotted in order to facilitate understanding the mechanism of normal defecation and continence and to allocate the majority of the patients into normal or abnormal physiology by simply plotting the line connecting the ACL and diameter during attempted defecation against the line connecting the mean IRP during attempted defecation and point M (see the norm gram) where the meeting of the 2 lines allocate the patient in its corresponding functional status. The above mentioned measurements are applied directly to the norm gram as mmHg and cm without conversion to the SI units.

Example:

IRP = 52 mmHg, ACD = 2 cm, ACD = 2,3 cm. Calculated flow index = 1,8 cc. barium sulphate/sec (i.e. zone I).

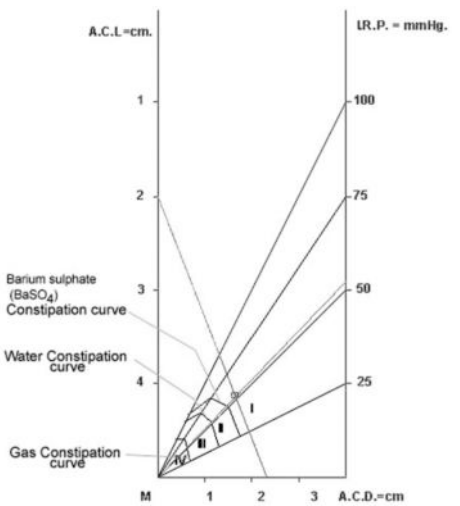

Fig. 4. – Defecation Norm Gram.

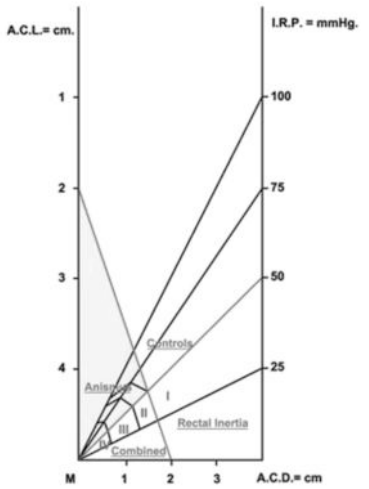

Fig. 5. – Types of patients and controls on Defecation Norm Gram.

Zone I includes: 1. normal unobstructed defecation; 2. patients that have constipation due to hard stools, colonic inertia or mechanical factor such as rectocele or intussusception that is expected to regain normal defecation after correction of the cause.

Zones II, *III* and *IV* include patients with underlying anismus and/or rectal inertia (RI). *Zone II*: obstructed defecation for soft well formed stools. *Zone III*: obstructed defecation for watery stools. *Zone IV*: obstructed defecation for gas.

The minimum normal anal canal resistance was represented by the line connecting ACL = 2 cm and ACD = 2 cm. The minimum normal intra-rectal pressure was represented by ... line connecting IRP = 50 mmHg and point M.

RI can be sub-classified into the following categories according to the IRPs measured during attempted defecation: *mild* from 40 to 50 mmHg; *moderate* from 30 to < 40 mmHg; *severe* from 20 to < 30 mmHg; *rectal atony* < 20 mmHg. The defecation norm gram can define anismus, rectal inertia, combined cases and normal controls (Fig. 5). Some of anismus patients lie in zone I of normal defecation, being compensated for by increased IRP during defecation which had led to the controversies about the role of anismus in constipation. Those cases are compensated anismus patients that should not be recruited as normal controls. In fact normal controls recruited only from the control area above and in front of both lines.

THE FLOW EQUATION IN ANAL INCONTINENCE

According to the flow equation, three main types of AI can be recognized:

– *passive AI* (i.e. during rest), where flow is calculated using maximum intra-rectal pressure during rest and ACL and ACD during rest;

– *stress AI* (i.e. during reflex squeeze e.g. on coughing). Where flow is measured using maximum IRP during cough or Valsalva's maneuver, and ACL and ACD during squeeze;

– *urgency AI* (during voluntary squeeze). Where flow is measured using maximum IRP during sense of urgency and ACL and ACD during squeeze.

Correction for sensory and reflex components

Passive AI: for *delayed first sensation* the flow index is measured using maximum IRP just before the first sensation and AC length and diameter during rest. For *reflex deficit:* if profound RAIR precedes the first sensation causing unconscious profound inhibition of the anal sphincter (*over-flow incontinence*) e.g. neurogenic AI, the flow is measured using maximum IRP during rest and ACL and ACD during full relaxation (defecation position).

Stress AI: correction for *defective reflex contraction* of the sphincters where the maximum IRP during coughing challenges the anal sphincter in its resting state instead of its contraction state. Flow is measured using maximum IRP during cough and ACL and ACD during rest.

Urgency AI: when profound RAIR precedes the sense of urgency where flow is measured using maximum IRP during urgency and ACL and ACD during full relaxation (defecation). Quantification of sensory and or reflex deficit can be made easily by the equation: flow index after correction-flow index before correction.

A flow index of 0.1cc/sec was taken as a cutoff point between normal continence and fecal soiling (flow <0.1 cc/sec). A flow index of 1cc/sec was taken as a cutoff point between true incontinence (flow ≥ 1 cc/sec) and fecal soiling.

Those corrections can be made much easy by the modern anorectal machines with simultaneous anorectal manometry during contrast defecography. The moments of leak of the contrast can be marked and later on freezed and the measurements done. However knowledge of the above corrections is essential for planning treatment as will be discussed later.

Continence Norm Gram (Fig. 6)

Five curves were calculated and subsequently plotted on the norm gram:

1. barium sulphate continence curve (upper solid curve);
2. water continence curve (middle solid curve);
3. barium sulphate soiling curve (upper dashed curve);
4. water soiling curve (lower dashed curve);
5. air continence curve (lower solid curve).

The above mentioned curves divide the norm gram into six zones forming the basis of a new functional classification of the degree of severity of incontinence.

Grade I: continent, anal staining due to minor anal problem (e.g. hemorrhoids), and incontinence on top of normal anal sphincter (e.g. fecal impaction); *grade II:* gas incontinence; *grade III:* fluid soiling; *grade IV:* solid soiling; *grade V:* fluid incontinence; *grade VI:* solid incontinence. Each grade from II to VI is assigned with letters *P*, *S* or *U* (as an indication for *passive*, *stress* or *urgency* AI) in order to signify which measurements are to be used in calculation of flow index or in

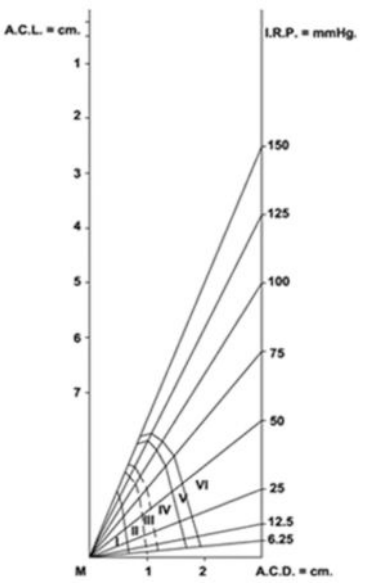

Fig. 6. – Continence Norm Gram.

the norm grams. Allocating each patient on the norm gram is done in the same way as in defecation. An important although difficult clinical tool to discriminate between anal staining due to minor anal problems (grade I) which requires no specific treatment or investigations and fluid or soft stool incontinence (grade III and IV) is by a simple look at the continence norm gram where the later is associated with gas incontinence (grade II). Discrepancies between *mathematical* anal incontinence despite absence of clinical incontinence is considered as sub-clinical sphincter weakness which may be unmasked later. E.g. a patient with sub-clinical gas incontinence may be masked by minimal amount of bowel gas produced in his bowel. Similarly a patient with mathematical stress fluid incontinence my be apparently normal because he will only manifest clinical incontinence when he has watery diarrhea simultaneously with a chest infection.

Frequency of anal incontinence may not correlate with quality of life scores because of different social status and intellectual abilities. The frequency of incontinence does not signify which mechanism is deranged and what can be improved by non specific treatments. As an example a high social rank person with gas incontinence may have a poor quality of life score when compared to a less intellectual person. Similarly a patient with stress gas incontinence may have increased frequency of his AI if he has an attack of bronchitis which can be successfully treated using antibiotics, mucolytics and expectorants.

Individual scores such as such as the flow (physiologic) score are less helpful in planning treatment. Due to the importance of frequency and QoL scores a composite score is suggested as "physiologic/frequency/QoL (PFQ) score" similar to the TNM score local/regional/systemic status of the tumors.

Planning for treatment and predicting outcome in patients with anal incontinence

The calculation of the flow equation and observing its elemental components will help to plan treatment in patients with functional anorectal disorders. Anal sphincter repairs should not be offered to the patients with normal anal canal resistance where the minimum AC dimensions during rest were set as ACL = 3.0 cm and ACD = 0.8 (AC resistance = 5639.6 and tolerating IRP up to 42 mmHg) and the minimum AC resistance during squeeze was taken as ACL = 3.5 cm and ACD = 0.6 cm (AC resistance = 20794.7, tolerating IRP up to 155 cm) as calculated from the flow calculator. Different treatment modalities can be assessed by studying the flow equation in patients suffering from multifactorial causes of AI. The effect of different biofeedback or operations can be followed up by its ability to increase anal canal resistance and decrease the Fl as they appear on the flow equation. Assessment of the anal sphincter function before closure of colostomy can be done preoperatively since the calculations can be done using defecography and anorectal manometry of the defunctionalized anorectal segment as accurately as in normal individuals.

THE USE OF THE FLOW EQUATION IN PLANNING TREATMENT: PREDICTION AND EVALUATION OF OUTCOME IN CONSTIPATED PATIENTS

Patients should be divided into two main groups after exclusion of dietary, hormonal and drug induced causes for constipation.

1. Patients with an apparent cause for constipation such as small bowel inertia, colonic inertia, hard stools, organic stricture, large rectocele or intussusceptions;

2. patients with no apparent cause for constipation.

The patients in the first group are further divided by the result of the flow calculator and the defecation norm gram into:

– patients suffering from apparent cause for constipation and lie in the zone I (normal unobstructed defecation) can be treated medically or surgically with expected normalized defecation after treatment since they have a normal underlying anorectal segment;

– patients with apparent cause of constipation and lying in constipation zones II-IV are expected to have residual obstructed defecation after treatment of the apparent cause of constipation. In fact correction of the underlying rectal inertia and/or anismus is recommended in those patients before correction of their apparent cause for constipation;

– patients suffering from no apparent cause of constipation and who lie in obstructed defecation zones II-IV should be treated for rectal inertia, anismus or both.

Some of the patients who do not have an anatomical abnormality, have a normal flow on the flow calculator and fall in zone I on the norm gram still complain of excessive straining during defecation. These patients have a hidden rectal inertia and have to strain vigorously in order to raise the intra-abdominal pressure for evacuation. These patients can be detected by simultaneously measuring the intravesical pressure or by palpating their abdomen and observing their face during attempted defecation. Repeating the test while asking them to strain gently will unmask their rectal inertia which should be treated by prokinetics.

Treatment of patients who have a normal flow but have a compensated rectal inertia or compensated anismus (Fig. 5), sounds logical if they present for evaluation for other reasons. This group may develop subsequent problems which appear to be due to prolonged periods of compensation by excessive conscious straining.

Normal reflex straining is a natural event during initiation of defecation but it should be reflex, non-laborious and is usually unnoticed by normal individuals. The resistance and flow equation can help in operator independent evaluation of different modalities of treatment for constipation by calculating anal canal resistance and flow equation pre- and postoperatively. A study on the flow equation could predict

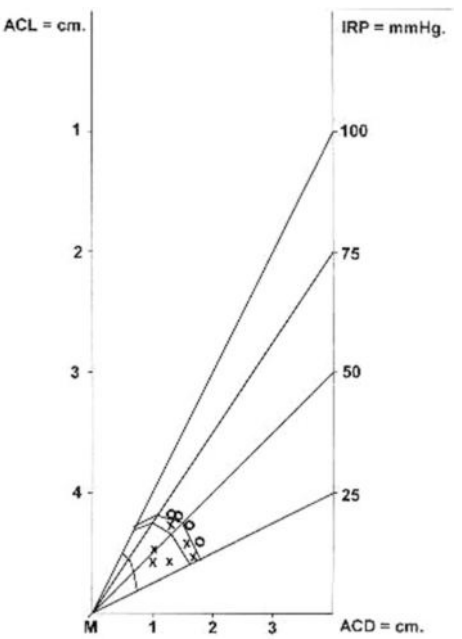

Fig. 7. – The use of Flow equation for predicting outcome in Rectocele Patients (Farag [12]).

the outcome of surgery in 10 female patients suffering from anterior rectocele and obstructed defecation (Farag [12]). The equation was not used to select patients for surgery. The four patients that had successful surgery had a preoperative flow index ≥ 1 cc of barium sulphate/ sec (i.e. abnormal underlying anorectal segment), while the six failures had a preoperative flow index < 1cc barium sulphate/ sec i.e. an abnormal underlying anorectal segment (Fig. 7).

THE FLOW EQUATION AND EXPLANATION OF THE LITERATURE

In a multicenter retrospective analysis of the outcome of artificial anal sphincter implantation for severe fecal incontinence published in the Br J Surg in 2001, Altomare and co-workers concluded that artificial anal sphincter with a diameter 2.9 cm was associated with a high incidence of postoperative obstructed defecation as compared to the cuffs with a 2 cm diameter. Numerical explanation of the authors findings can be given by the flow equation as follows. From the perspective of the flow equation the artificial anal sphincter works by maintaining adequate ACL determined by the diameter of the cuff and minimizes the ACD by the inflation of the balloon. The presence of a pliable anal canal is essential for the action of the artificial anal sphincter in order to reduce the ACD. The procedure should be accompanied by lysis of any fibrosis, which may cause tethering of the AC to the peri-anal structures. The artificial sphincter tries to mimic the normal anal sphincter by its ability to relax in order to achieve normal unobstructed defecation on volition. Use of an artificial inflatable anal sphincter replacement was followed by anismus if the cuff diameter used is 2.9 cm as compared to cuff diameter of 2 cm in this study from Italy. This finding can be explained by the flow equation simply by the fact that the cuff diameter represents the minimal ACL which can be achieved during defecation. For example at any given ACD (e.g. 2 cm), using 2.9 cm cuff would lead to a 45% increase in AC resistance during defecation which needs a proportionate increase in IRP during defecation in order to maintain the same Flow Index achieved with 2 cm diameter cuff during defecation (more straining for the increasing resistance). The use of cuffs with 2.9 cm diameter should be abandoned and the inner diameter of the artificial sphincters with 2 cm diameter cuffs should be tailored according to the mean intra-rectal pressures during defecation measured preoperatively and accordingly the expected flow index postoperatively.

IMPROVING THE PRESENT TECHNIQUES AND PLANNING FOR NEW TREATMENT MODALITIES

Failure of the muscle layers that wrap around the anal canal may result from perineal trauma. The rigidity of the anal canal may be fixed by dense perianal adhesions. The muscles are held by the adhesions and cannot contract to decrease the anal canal diameter. An adequate perianal adhesiolysis is an essential step to be added for the success of such operations. Similarly the use of behavioral treatment and biofeedback to teach the patients how to have an urgency defecation is added in my lab as an essential step for the conservative treatment of simple anorectal problems namely, hemorrhoids, anal fissure, anal fistulae, anismus, rectal intussusception, mucosal and complete rectal prolapse.

CONCLUSION

The use of flow equation suggests new definitions for anal incontinence and constipation from the flow point of view, determines the intra-rectal pressure, dynamic viscosity and the anal canal length and diameter as the primary mechanical factors maintaining continence. It also suggests how the sensory and reflex factors interact with the mechanical factors in order to maintain continence in a fraction of a second and initiate normal unobstructed defecation in few seconds. All these factors can be measured numerically in health and disease. This knowledge helps in planning treatment modalities for each individual patient, predicting outcome, and objectively evaluating the outcome postoperatively or after treatment. In cases of combined treatment modalities, the effect of each modality can be evaluated separately. The use of the flow equation can help to anticipate and avoid postoperative constipation after reconstruction of the anal sphincter for the treatment of anal incontinence. It also can be used for improving the present treatment modalities and planning for new treatment options. The hybrid law in coloproctology gives an insight on how the anal sphincters behave during defecation and a new insight on the etiology of haemorrhoids, anal fissure, anal fistulae, anismus, rectal intussusception, mucosal and complete rectal prolapse.

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Original article

Differential staged sacral reflexes allow a localization of pudendal neuralgia

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Abstract: The objective of this work is to localize pudendal nerve compression by measuring sacral reflexes and to explain differences in latencies by an anatomical investigation. Electrophysiological data was obtained from 59 patients by measuring the healthy side and the painfull side of 79 patients with uni- or bilateral pain. In addition, 7 formalin-fixed female cadavers were dissected to identify nerve trajectories. Dissections demonstrated that the levator ani nerve runs directly from the sacral plexus, to innervate the levator ani, while the pudendal nerve traverses the sacrospinal and sacrotuberal ligaments, entering the perineum with an inferior rectal nerve, already separated runs to the posterior or caudal part of the external anal sphincter muscle, while the anterior parts of the external anal sphincter muscle are innervated by perineal nerve branches coming from the pudendal canal. Significant differences in latencies suggest that main nerve compression occurs in the pudendal canal. In conclusion, it is possible to localize differences in pudendal nerve compression by separate electrophysiological examination of staged sacral reflexes and allows to narrow down the location of nerve compression.

Key words: Anatomy; Compressive neuropathy; Electrophysiology; External anal sphincter innervation; Pudendal nerve.

INTRODUCTION

Compressive pudendal neuropathy is a frequent condition that is often ignored.[1-3] Its incidence is approximately 1% in the general population and the condition probably affects women more often than men. Because healthcare professionals lack an adequate method to diagnose accurately pudendal neuropathy, the affected individuals often embark on an endless quest for effective relief with serious physical and psychological consequences.

There are several possibilities of a treatment to diminish pain. The first line is conservatory followed by infiltrations [4, 5] and, ultimately by surgical procedures.[6-9] It is therefore important to identify precisely the site where the nerve is compressed. The classical approach consists of measuring pudendal nerve terminal motor latencies after electrophysiological stimulation. However, the results obtained with this technique lack of reproducibility and sensitivity.[10-14] The endo-vaginal or endo-rectal stimulation at the level of the ischial spine have been criticized because of poor precision in the localization of the stimulation point, the distortion of the stimulation potential by the different intervening tissues and due to the fact, that albeit an increased latency did demonstrate myelinopathy, it could not indicate its location. Several conditions may interfere with conduction times, including vascular factors, presence of distal synaptic ends, vegetative reactivity and time-dependent variability.[15-17] Moreover, a compression at the level of the ischial spine or just beside it cannot be detected by pudendal nerve terminal motor latencies. Thus, the sacral reflex measurements were thought to be a good indicator for the extent of the affected terminal nerve area. However, it is usually recorded via the bulbocavernosus muscle; yet it reveals to be unsatisfactory.[18] Therefore, the aim of our study was the development of a more precise diagnostic test to localize pudendal neuropathies. Preliminary electrophysiological tests of the anterior and posterior innervations in the anal sphincter area (Eric de Bisschop, unpublished results), revealed marked latency differences. Therefore, we decided to compare sacral reflex transmission latencies through the pubococcygeus loop and the levator ani and the anterior and posterior anal sphincter parts, calling them *"staged sacral" reflexes*. Differences in latencies due to electrophysiological measurements were validated and so it can be explained by differential innervation of the anal sphincters by rectal inferior and perineal nerve branches.

SACRAL REFLEXES

When a nerve is compressed, its vascularization is compromised and consequently the nerve suffers. Nerve suffering has several consequences, including a decrease in nerve conduction velocity that affects both the motor fraction of the sacral reflex and the sensory transmission owing to the evoked somesthesic potentials. This decrease can be recorded as a prolonged latency period between the stimulus and the record points. In a pudendal nerve terminal motor latency measurement, the stimulation electrode is placed over the ischial spine usually by endo-rectal insertion. An impulse is delivered and a recording is made using a surface electrode placed on the perineal muscle, usually at the level of the anal sphincter (Fig. 1A). The stimulus may also be delivered through an endo-vaginal insertion. In both cases, the examination consists of measuring the latency, i.e. the time elapsed between the moment of impulse delivery and the record of the electric potential in the target region. By comparing the recorded latencies to normal values, it is possible to estimate the influx transmission capacity of the nerve. Normal latencies, reported in the literature, vary from 2 to 5.35 msec.[14, 19] The measurement of the sacral reflex is carried out in an analogous manner: a stimulus is delivered at one point and the potential is recorded at a second point. The stimulus is delivered in this case at the level of the clitoris. The impulse travels through the sensory pudendal nerves towards the sacral spine (Onuf's nucleus) and returns back through motor efferent nerves. The potential is recorded in one of the zones of the pudendal nerve that is innervated by motor fibers. In case of nerve compression, latency is increased due to the impairment of nerve conduction (Fig. 1B).

CONVENTIONAL ANATOMY

In a conventional anatomical description, the pudendal nerve originates as a fusion of the S2, S3 and S4 roots, with an occasional involvement of the S1 and L5 roots, while the levator ani muscle is innervated by a nerve that originates

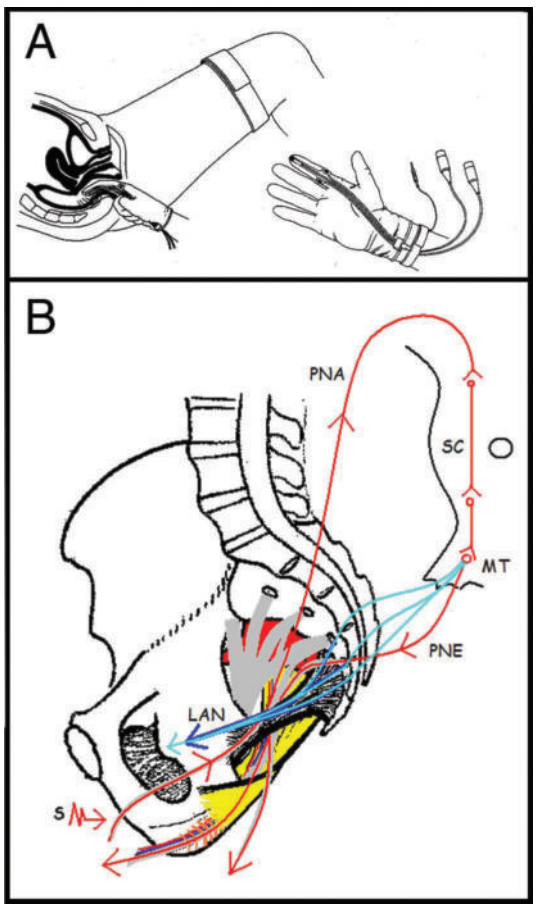

Fig. 1. – (A) The technique of pudendal nerve terminal motor latency measurements. (B) Electrophysiological Anatomy. S: indicates the stimulus at the level of the clitoris. Records are made at the level of the anterior and posterior anal sphincter quadrants. LAN: levator ani nerve. PNA: pudendal nerve afferents. PNE: pudendal nerve efferents. MT: medullar transfer. SC: spinal cord.

in the sacral plexus and runs above the levator ani muscle (on top of the pelvic floor) on the pelvic side and then innervates the iliococcygeus and pubococcygeus muscles as well as the upper part of the puborectalis.[18-20] The pudendal nerve circumvents or perforates the sacrospinous ligament (formerly called small sacro-sciatic ligament) and enters between sacrospinous and sacrotuberous ligaments (formerly also called great sacro-sciatic ligament) into the perineum, also called "ligament clamp" as it constitutes a site for potential pudendal nerve compression. Nerve and blood vessels run within the ischiorectal fossa towards the anterior perineum and vasculo-nervous elements enter the pudendal canal (also termed Alcock's canal), that is constituted by duplication of the fascia of the internal obturator muscle, thus may also cause a nerve compression. The inferior rectal nerve classically innervates all parts of the anal sphincter, and separates from the pudendal nerve at the beginning of the pudendal canal and runs across the ischiorectal fossa towards the anal sphincter. In the middle of the pudendal canal, the pudendal nerve divides into two branches: the dorsal nerve of the clitoris and the perineal nerve, with both nerves traversing the pudendal canal in its entirety. The perineal nerves give off the sensory branches to the perineum as well as the motor branches for the perineal muscles and for the external anal sphincter. The dorsal nerve of the clitoris is a terminal sensory branch of the pudendal nerve.

Here, we confirm that the anterior and posterior quadrants of the anal sphincter are innervated by different branches and by different trajectories and provide several possible entrapments of parts of the nerves and consequently pudendal neuropathy. Because the afferent path of the dorsal nerve of the clitoris is always the same, it is theoretically possible to locate the site of compression with some precision by examining the different efferent paths. To confirm the validity of this assertion, we carried out an anatomical study aimed at determining:

i) the systematic presence of a nerve in the levator ani (pubococcygeus) muscles;

ii) the location of the dorsal nerve of the clitoris starting point relative to the main trunk;

iii) the presence of a starting point of the inferior rectal nerve that would be more proximal than what is described in the literature;

iv) to identify differences in the innervation of the anterior and posterior anal sphincter quadrants.

MATERIALS AND METHODS

We relied on 27 consecutive male and 49 female patients suffering from unilateral or bi-lateral pudendal neuropathy. The diagnosis was ascertained because symptoms disappeared after conservative treatment or surgery. We hypothesize that the absence of symptoms on the controlateral part was the guaranty of an intact pudendal anatomy. Also for ethical reasons we decided to use each patient as his own comparative and the healthy side as control value. Each patient had 3 measurements on each side meaning 6 values at all. The first record was the reference for the pubo-rectal muscle, the second was taken at the superior quadrant of the external anal sphincter and the third at the inferior quadrant of the external anal sphincter. The obtained values show that nerves may be affected differentially and exhibiting significantly higher electrophysiological values on the affected side than on the normal side. To ascertain that our hypothesis was correct, we decided to confirm the anatomical situation on cadavers. The seven corpes used in this study were obtained by the donation program to the Department of Cellular Biology and Morphology. All donors gave previously a written consent. The cadavers were perfused through the femoral artery with a mixture of 0.9 L of formaldehyde (38%), 0.5 L of phenol (85%), 1.0 L of glycerol (85%), 4.0 L of ethanol (94%) and 10.6 L of water. The cadavers, were stored at 8 °C until dissected by second year medical students as part of their training. Half pelvises that had not been dissected in the course were used to study the trajectories of the anal, pudendal and perineal nerves described in this work.

RESULTS

The nerve fibers for levator ani (pubococcygeus) muscle run directly from the sacral plexus, above the pelvic floor on the side of the levator ani and follow a different direction from the pudendal nerve (Figs. 2A and 2B). The pudendal nerve traverses the ligament clamp located between the sacrospinal and the sacrotuberal ligament in the lateral space of the ischiorectal fossa where nerves and vessels are surrounded by the fascia of the internal obturator muscle and form the pudendal canal. In one of the seven dissections performed, the pudendal nerve actually perforates the sacrospinal ligament. In five cases, the pudendal nerve divides into the dorsal nerve of the clitoris and perineal nerves after passing underneath the sacrospinal ligament but before entering the pudendal canal (Fig. 2C). The same anatomical section also shows that the dorsal nerve of the clitoris runs parallel to the pudendal canal. The neurovascular bundle within the pudendal canal gives rise to multiple perineal branches that branch off either at the point of entry into the ischiorectal

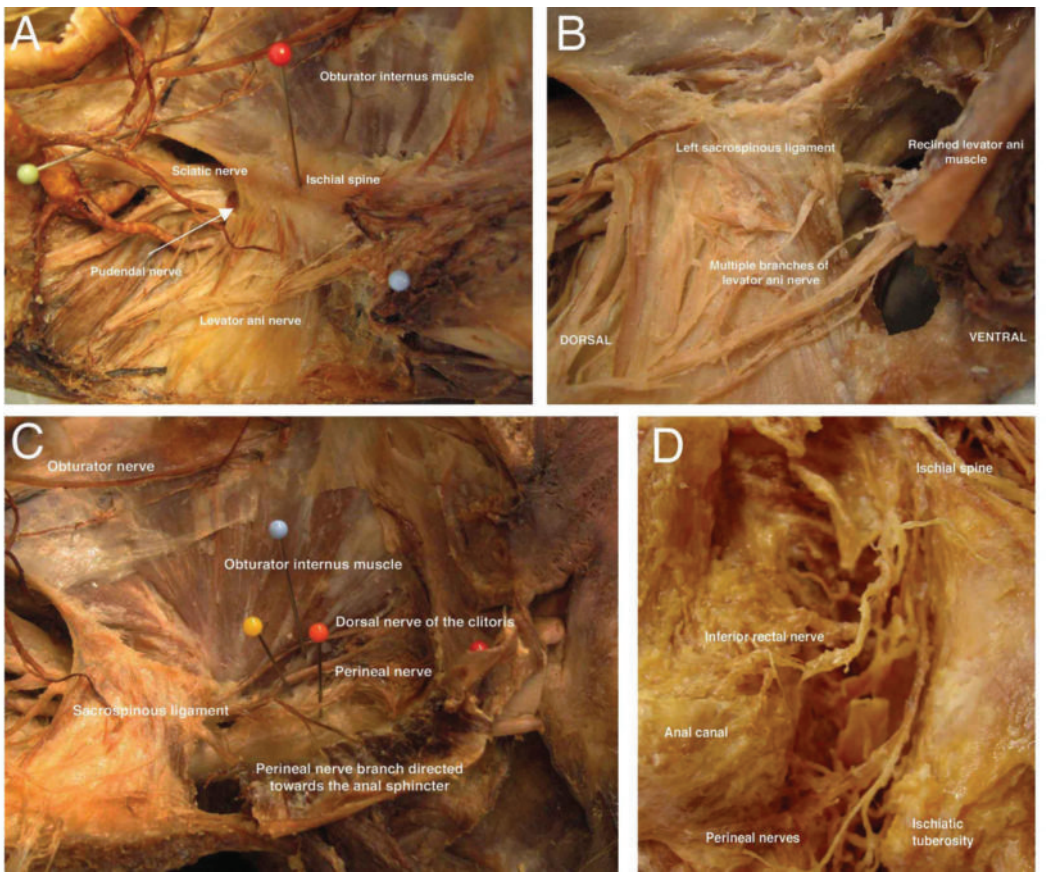

Fig. 2. – (A) The internal face of the left half pelvis. The red needle indicates the position of the ischial spine: the levator ani nerve passage through the sacrospinal ligament is clearly visible. (B) The passage of the levator ani nerve above the sacrospinal ligament is easily distinguishable. (C) The pudendal canal has been opened and the internal obturator fascia removed. The nerve that passes above the blue needle is the dorsal nerve of the clitoris, the one above the yellow needle is the main branch of the pudendal nerve, i.e. the perineal nerve. The ramifications of the inferior rectal nerve are seen underneath the red needle. (D) Ischiorectal fossa in the right half pelvis. The inferior rectal nerve that innervates the posterior anal hemisphere originates in a much higher zone and does not traverse the pudendal canal as classically described in the literature. In addition to other functions, perineal nerves act as motor nerves for the anterior anal hemisphere.

fossa, inside the pudendal canal in the direction of the anal canal (sphincter) or the anterior perineum. In all of the dissections, the inferior rectal nerve originates prior to the entry into the ischiorectal fossa and runs straight towards the back of the anal canal, through the ligament clamp but not through the pudendal canal (Figure 2D).

The electrophysiological measurements of the staged sacral reflexes show a significant higher latency time of the ventral quadrant of external anal sphincter innervation, while the posterior or caudal quadrant was just slightly prolonged and levator ani innervation was unaffected (Fig. 3A-C).

DISCUSSION

Historically, the pudendal nerve has been investigated using terminal motor latency,[14-21] a technique which is not very reliable.[22] Indeed, because of the distance between the stimulation point and the nerve (approximately 1 cm),[23] it is possible that the electrical impulse delivered at the point of contact with the ischial spine does not merely travel along the nerve but diffuses in the entire perineal region. Several investigators have made different contributions to the available tests.[24, 25] The levator ani nerve appears to be recognized as a distinct entity by all authors. Our work has shown that the levator ani is innervated by a nerve that originates in the sacral plexus. This nerve runs above the sacrospinal ligament and terminates in the pubococcygeus, the iliococcygeus, and the puborectalis muscles. This finding is supported by investigations carried out by Hallner[26] who failed to find any pudendal nerve innervation of these muscles in 200 dissections. At the same time, it should be noted that a few authors have nevertheless proposed possible pudendal nerve innervation of the levator ani muscle. In most cases, these opinions are based on experiments in which this nerve was anesthetized at the level of the ischial spine, resulting in the paralysis of the levator ani muscle.[27] The methodology of such experiments is however questionable: the anesthetic could have easily diffused towards the levator ani nerve, located less than 1 cm away from the anesthetic injection point. The dorsal nerve of the clitoris is classically described as a terminal branch of the pudendal nerve.[26] However, in our dissections it often appears as a branch that is parallel to the pudendal nerve and that does not run through the pudendal canal. This observation, which contradicts the classical description in literature, has also been confirmed by other authors.[7] If this observation is correct, then the dorsal nerve of the clitoris can hardly be compressed, which explains the interest in using this nerve as an afferent branch in sacral reflex studies. An even bigger controversy surrounds the inferior rectal nerve.[28-32] Certain authors believe that it originates at the beginning point of the pudendal canal,[33] with some variations.[32] Thus, its origin may be independent with or without an anastomotic branch from the perineal nerve. Moreover, the inferior rectal nerve may perforate the sacrospinal ligament in its middle portion at an approximate distance of 1 cm from the ischial spine, extending to a distance of up to 1.5 cm. Other investigators[33] estimate that 60% of

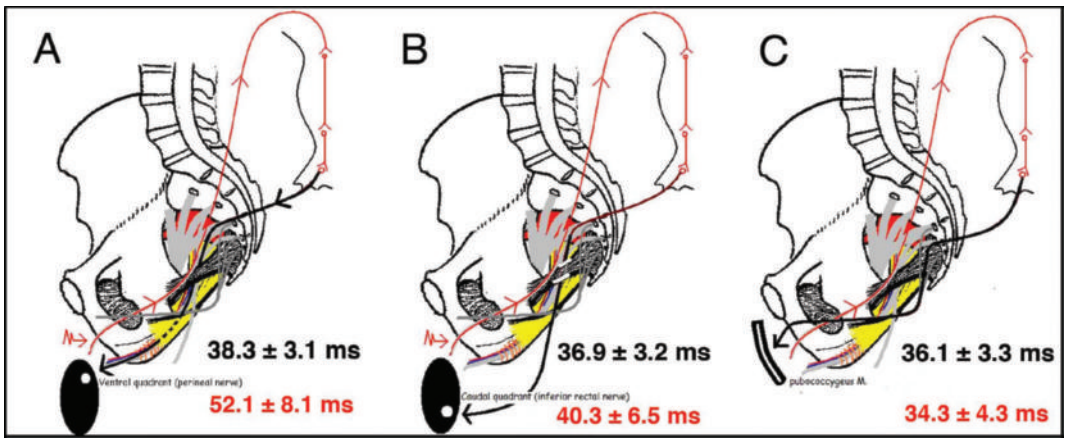

Fig. 3. – The concept of staged scared reflexes and its electrical impulse circuits. Image (A) represents the recording made from the anterior or ventral quadrant of the external anal sphincter. Image (B) represents the recording made from the posterior or caudal quadrant; and image (C) represents the recording from the levator ani (pubococcygeus) muscle. To establish normal latency of staged reflexes, the healthy side of patients with uni-lateral neuralgia were measured (n = 59), black numbers. Staged reflexes were measured in the painful side of 76 patients with uni- or bi-lateral neuralgia (numbers in red). Note the highly significant difference (t-test $p < 0.1$) of latencies of the anterior quadrant when compared with the healthy side, suggesting a compression of the pudendal nerve in the pudendal canal.

the lower rectal nerves originate from the pudendal canal. This view is supported Shafik and Doss that also describe an emergence from the pudendal canal. [31] In fact, it seems that any structure that terminates at the anal sphincter is referred to as an inferior rectal nerve. However, according to our findings, the anal sphincter shows at least two distinct innervations that may explain the differences with the classical descriptions in literature. The first innervation network originates from the inferior rectal nerve itself, while the second network is generated by the pudendal nerve via perineal branches. We believe that the multiple origins of innervation are important from both the clinical and electromyographical points of view (Fig. 3A-C). Indeed, one branch is the inferior rectal nerve that branches off very early (proximally) and runs parallel to the nerve, but is not located in the pudendal canal and no longer at risk for compression. This nerve terminates in the posterior hemisphincter. A second branch that innervates the anterior hemisphere appears to originate from a more distal region, i.e. the perineal branch of the pudendal nerve. It is thus subject to compression at the level of the ligament clamp and the pudendal canal or the falciform process. In other words, we believe that it is anatomically correct to postulate that the anterior and posterior anal sphincter quadrants as well as the pubococcygeus muscle all have separate and distinct innervations. As a consequence, a staged sacral reflex analysis of the reflex loops specific for these three muscles should make it possible to better locate the site of compression with greater precision compared to the current techniques. The anatomical analysis conducted in this work argues in favor of the proposed concept, and demonstrates that part of the external anal sphincter innervation comes from pudendal nerves and that the pudendal nerve compression in the pudendal canal may be the cause of pudendal neuralgia.

CONCLUSION

Our anatomical study confirms the existence of separate innervations of the anterior and posterior parts of the external anal sphincter muscle as well as for the pubococcygeus muscle. It may be possible to better identify the site of compression of the pudendal nerve by separate electrophysiological examinations of the three zones. The concept of staged sacral reflexes is introduced and should lead to apply more precisely infiltration treatments or to start surgical interventions.

ABBREVIATIONS

LAN: levator ani nerve; PNA: pudendal nerve afferents; PNE: pudendal nerve efferents, MT: medullair transfer; SC: spinal cord.

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8 – FISTULAE

Hidradenitis suppurativa presenting as a polypoidal lesion at the anal verge. *Seneviratne SA, Samarasekera DN. Colorectal Dis. 2009;11:97.* Hidradenitis suppurativa is a chronic disease characterized by painful recurrent abscesses, fistulas and scarring lesions in axilla, groin, perineum and rarely mass lesions at the affected site. A case of hidradenitis suppurativa with a polypoidal growth at the anal verge is presented.

Modified York-Mason technique for repair of iatrogenic rectourinary fistula: the Montsouris experience. *Kasraeian A, Rozet F, Cathelineau X et al. J Urol. 2009;181:1178.* Rectourinary fistula is a devastating complication of rectal and genitourinary surgery. Failure in conservative management calls for surgical intervention. A series of 12 patients (after radical prostatectomy and following high intensity focused ultrasound, 6 with fecal diversion) treated by a modified York-Mason technique is presented. The urethra is not closed after fistula excision, only a multilayer, nonoverlapping closure of the anterior rectal wall being performed. Three patients required multiple York-Mason procedures. All patients reported intact fecal continence. Median hospital stay was 4 days.

Repair of giant vesicovaginal fistulas. *Ezzat M, Ezzat MM, Tran VQ, Aboseif SR. J Urol. 2009;181:1184.* To repair giant vesicovaginal fistula (35 patients, 7 with a complete loss of the urethral floor), the abdominovaginal approach using a rotational bladder flap was evaluated. patients had. Fistula etiology was secondary to obstructed labor in 25 patients, the result of iatrogenic surgical injuries in 5, sling erosion in 3 and pelvic irradiation in 2. The bladder was bisected sagittally, and a bladder flap was rotated downward and medially to fill the extensive fistula defect. An additional vascularized flap was interposed in 23 patients including gracilis muscle, omental, peritoneal or Martius flap. Fistulas were successfully repaired in 31 of 35 patients (88%). The remaining 4 patients underwent surgical correction with a second, more limited repair.

Long-term success rate after surgical treatment of anorectal and rectovaginal fistulas in Crohn's disease. *Löffler T, Welsch T, Mühl S et al. Int J Colorectal Dis. 2009;24:521.* Among 777 patients with Crohn's disease undergoing surgery (1991-2001) 147 had anorectal or rectovaginal fistula requiring 292 operations, 98% with Crohn's disease in the colon or rectum. Over long-term follow-up, 29 patients required proctectomy. Submucosal fistulas needed major surgery in only 14% of cases compared to 56% of cases with rectovaginal fistulas. After 5 years complex fistulas showed a strong trend towards a higher recurrence rate after surgery than simple submucosal fistulas. Whereas recurrences occurred over the whole observation period in the group of patients with complex fistulas, there was no further recurrence in patients with submucosal fistulas 13 months after surgery.

The PFD continues on page 32

Author's Reply

Invited comment: A new theory of anorectal function
(D. Chatoor, A. Emmanuel - issue 4, 2008)

We have been interested to study Drs Chatoor and Emanuel's analysis and comments on our work. We note that they do not comment overall on the musculo-elastic theory itself, or on the concepts underlying our series of publications. As we explained in the preamble we sought to test the musculo-elastic theory of pelvic function and continence by challenging its predictions, a method proposed as the basis of the scientific method by the late Sir Karl Popper, and regarded as the most rigorous test procedure. Thus the studies we reported were designed to test the musculo-elastic theory by seeking direct tests that would refute the theory.

For example, in Study No 1, if we failed to demonstrate the three predicted directional movements, then the Theory would be invalidated. Since the theory states that faecal incontinence (FI) is caused by lax suspensory ligaments, in this case, the pubourethral and uterosacral, and we found a >80% cure of FI on repair of those ligaments, then the theory remains valid, although not necessarily proven. Popper held that nothing in science is ever finally proven, but that any theory remains valid until it is refuted. The classic example of this principle in Physics is Newton's Laws of Gravity, which required modification in relation to Einstein's Theory of Relativity, yet still remain accurate enough that they are used to calculate satellite orbits.

We define idiopathic FI as a disorder of faecal continence despite a normal external and internal anal sphincter, and in the absence of any causative central neurological or other disorder.

Chatoor and Emanuel state that "the aetiopathogenesis of urinary and faecal incontinence is rarely a singular process. Insults to the pelvic floor are usually multiple (including chronic straining, increased intra abdominal pressure effects, parturition and the menopause)". This statement reflects generally held opinion but is essentially phenomenological rather than mechanistic. It does not lead to understanding of the abnormal functional forces acting on the pelvic floor leading to incontinence. Our musculo-elastic theory, on the other hand leads to predictions of abnormalities in pelvic floor function, and that their correction should mitigate FI when present. It also offers functional explanation of symptoms such as straining and abdominal pressure effects as secondary manifestations of connective tissue damage, especially to the suspensory ligaments, because this abnormality will cause unloading of muscle forces, and weakening of the vector acting across the pelvic floor during its normal function.

The comments made by Chatoor and Emanuel regarding collagen abnormalities as risk factors for the development of FI, and probably also applicable to stress urinary incontinence, regarding collagen abnormaities are entirely in accord with our perspective of these problems, and are especially valuable since they should be amenable to experimental testing.

We note that Chatoor and Emanuel have misinterpreted some of our ideas, and comment as follows:

Experimental study No 1.

We did not set out to alter the puborectal angle, which we agree is not in itself crucial to faecal continence. We set out to restore the functional integrity of certain intrapelvic ligaments, in order to restore muscle forces. Any resultant change in the puborectal angle would therefore be secondary to restoration of ligamentous function and muscle force vectors acting across the pelvic floor.

We regard the outer longitudinal muscle of the rectum (LMA) as contracting against, and angulating, the levator plate, which is attached to the ligamentous structure via its fascial coverings. We do not suggest the LMA merges with the uterosacral ligaments.

We have not claimed to quantify muscle forces, but rather to confirm the direction of these muscle forces. We agree that the position of the ligaments is necessarily only an estimate, but it is evident by studying the radiographic illustration of the normal subject with the pelvic floor in the "resting position", that there is a definite bend at midurethra, which is consistent with the anchoring point of the three muscle forces we have described. With regard to the uterosacral ligaments, we compared the straining to the resting data. The downward angulation of the tip of the levator plate is the directly in line with the position of the cervix, the insertion point of the uterosacral ligaments.

It is not possible to assess laxity in the living subject, even with MRI.

Experimental study No 2.

We have indeed suggested a possible mechanism; that is, restoration of the pubourethral anchoring point for levator plate contraction and we provide a diagram to illustrate this concept.

Experimental study No 3.

Chatoor and Emanuel offer an explanation of the phenomenon described on the basis of increased sensory input caused by digitation of the vagina, leading to an enhanced reflex response. We prefer the notion, based on clinical examination that this change in function was simply due to a mechanical change associated with restitution of abnormalities in function associated with lax connective tissue structures. This concept is testable by more detailed experimental work in affected patients.

Experimental study No 4.

The bladder was full of urine when the testing was done. Although we accept that there is discussion regarding the best techniques for measurement of abdominal pressure changes, any criticisms regarding the methods we used apply equally to both the squeezing and straining maneouvres, and so would apply to both.

Experimental study No 5.

Chatoor and Emanuel make a definitive statement "One of the frequent causes of faecal incontinence in the elderly is internal sphincter atrophy." This may or may not be so.

We must emphasize that we are suggesting a different approach to understanding the functional basis of faecal incontinence and stress urinary incontinence; i.e., that it may be due primarily to ligamentous laxity. This concept does not exclude a role for other factors, especially anal sphincter tears, and even internal anal sphincter dysfunction, but we do not think the latter is a likely cause of faecal incontinence as a unique and solitary abnormality. We agree entirely that we have taken an arbitrary 2 mm definition of internal anal sphincter thinning on the basis of advice from our Radiologist. What we did demonstrate was:

a) Only a minority of patients with FI had IAS thinning.

b) IAS thinning had no impact on whether such patients were cured following the tape insertion procedure.

One has to conclude therefore that IAS defect was not a major cause of FI in our patients.

As regards the ultrasound probe, a 7 Mhz probe may be less sensitive but its use was consistent, and this does not therefore alter our conclusions.

Once again we point out that our observations and interpretations are open to further experimental study, which will verify or refute them.

Experimental study No 6.

We note the comments. However, the presenting symptoms were relieved by the procedure, and we have presented our observations and interpretation of the mechanism of benefit.

Experimental study No 7.

We agree that the puborectalis is an important part of the anorectal closure mechanism; indeed, this has been known for more than 30 years. We regard the main function of the puborectalis muscle as anchoring the anorectum, so that closure of the anus can be effected by stretching the rectum backwards and downwards. A relevant analogy might be maintenance of urinary continence after excision of he distal part of the urethra. In that case, we consider it is the backward/downward stretching which narrows the urethral tube, exponentially raising its resistance according to the 4^{th} power law of Poiseuille.

Experimental study No 8.

The histological findings described were features that do not occur in normal subjects, as compared with historical data on the anatomy and histology of these perineal muscles. Biopsies from control subjects are clearly unethical, not least since normal subjects are not subject to surgical procedures. We have made the point that histological features in muscles in which muscle fibres could be detected were consistent, in part, with changes found in limb muscles after tenotomy. The essential feature of this abnormality is that it is due to unloading of muscle by the tenotomy, or by ligamentous laxity in the case of our pelvic floor biopsies, and that these changes are reversible.

Even a weak muscle, caused by direct injury, denervation or myopathy can function better if its ligamentous attachments are reinforced. People with severe muscle atrophy, as in inherited neuropathies, poliomyelitis or myopathy can remain mobile as long as joints and ligaments are intact. It is well-recognized that when there is stretching of ligaments, mobility may be lost.

We agree however, that other than stating that the tape provides a strong insertion point, we did not demonstrate how the contractility improved.

Experimental study No 9.

A strength of this report is that it represents the experience of a generalist gynaecologist who based his report on a preoperative questionnaire. Symptoms are important. Gradation of the severity of incontinence would not have contributed to the aim of testing the hypothesis although we agree that will be necessary in attempting to better define the indications for the procedure. The important point to note is that patients were cured of their FI symptoms. This report reflects the remarkably successful results of one practitioner.

Experimental study No 10.

Chatoor and Emanuel take issue with our algorithm. This is intended to reflect our experience. We agree that it differs in some respects from other algorithms of pelvic floor dysfunction and, of course, like all algorithms it certainly oversimplifies the issues. We draw attention to the various algorithms in the book "The Pelvic Floor", (Pemberton J, Swash M, Henry MM, WB Saunders 2002) which also represent summaries of ideas and practice. The unexpected placement of certain symptoms, such as nocturia, unexpectedly as a posterior defect represents our experience of its resolution following a posteriorly-directed, reconstructive tape-insertion procedure.

In reply to Chatoor and Emanuel we again point to the results described – a large percentage of these patients were cured or improved of their FI symptoms following operations that only repaired suspensory ligaments. The symptoms and their relation to the algorithm have been validated in two ways.

1. Use of 'simulated operations'; i.e., anchoring specific ligaments and observing the effect on symptoms such as urge, SI and pelvic pain.

2. Tracking pre-operative symptom fate after site-specific repair to the three zones in large numbers of patients. The surgical techniques are fully described elsewhere in the literature.

We agree with Drs Chatoor and Emanuel, that the algorithm may not have described the functional defects sufficiently for optimal cure of all patients in this group. For example, some patients required a second procedure directed to another pelvic floor ligament. However, because we wished to assess whether laxity in either the anterior or posterior suspensory ligaments (or both) caused FI, we followed the protocol described. The operations are minor in-patient procedures, so that an argument can be made for repairing both the anterior and posterior in all patients with FI.

We agree that it would have been helpful to repeat the tests in failed cases. However, this re-examination was dependent on patient consent.

Mean anal pressure and functional anal length were determined by standard methods, using balloon manometry, in the Dept of Colorectal Surgery at the Royal Perth Hospital.

Our results regarding the difficulty in securing reliability in pudendal nerve terminal motor latencies speak for themselves. The neurological co-author (M Swash), who introduced this technique as an experimental method in the 1980s never intended that it should be used in clinical practice in individual patients, for the very reason that the length of the terminal segment of the pudendal nerve over which the measurement was made could not be verified and might vary from test to test. At the time this method provided useful verification of damage to the pudendal nerve, when treated as a change in group data, but the standard deviation of the grouped results was always too great for application in individuals. Our results reported here reflected these comments.

With regard to "validated scoring sheets' we believe that nothing could be more validating than a patient stating she does not soil anymore. This is a yes/no response that answers the question absolutely.

Poisseuille's Power Law may be confusing. However, we set out to explain why some patients with no obvious anatomical defects were incontinent, or had emptying problems. By regarding the anorectum as a tube which is opened or closed by muscle forces, this explanation becomes rationalized in terms of physics. If we then understand that these same muscle forces effectively contract against suspensory ligaments, as do all muscles in the body, then the argument becomes rationalized also in biomechanical terms.

Experimental study No 11.

Once again, Chatoor and Emanuel have ignored our results – a large percentage of these patients were cured

or improved of their FI symptoms with simple out-patient, daycase, operations which repaired suspensory ligaments.

Cystocoeles were present in a number of these patients and required repair. This necessity afforded us the opportunity to test whether this repair also improved the FI cure rate; it did not.

We agree with Chatoor and Emanuel that it would have been helpful to add subgroup data in the paper. However, the numbers of patients involved were too small to allow this. We agree that the question, which ligament causes which symptom is of critical importance. Our approach as stated, was to restore all the anatomical defects, based on the principle of "restore the anatomy, and you will restore the function". Further work may yet address this question.

Experimental study No 12.

Chatoor and Emanuel make no comment on the major observation made in this report by Abendstein; that is, a large percentage of these patients were cured or improved of their rectal intussusception and obstructive symptoms by repairing the posterior suspensory ligaments, thus avoiding a major invasive abdominal procedure. From our perspective, this observation validates another of the musculo-elastic theory's predictions. All these patients were symptomatic prior to surgery.

Again, we do agree that subgroup data in this study would have been beneficial to our aims.

We believe overwhelmingly that psychological problems in this disorder are secondary to the incontinence. It is remarkable how many such problems disappear overnight after successful surgery.

Final comments

In their criticisms Chatoor and Emanuel have unmasked what is perhaps a major imperative for future research, close collaboration with colleagues whose knowledge and advice will provide more information, and better test the musculo-elastic theory.

We have presented information that supports a concept that emphasizes the role of ligaments applied to the muscle forces activating anorectal closure (continence) and evacuation. Our aim has been to indicate a new direction for treatment and research. We hope that the many doubts and questions raised by Chatoor and Emanuel will be taken up and used to further test the musculo-elastic theory. What we have done can only be considered a small beginning which, we trust, will unfold into a rich new era of research in anorectal function and dysfunction.

March 2009

PETER PETROS
MICHAEL SWASH

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9 – BEHAVIOUR, PSYCHOLOGY, SEXOLOGY

Single institution 2-year patient reported validated sexual function outcomes after nerve sparing robot assisted radical prostatectomy. *Rodriguez E, Finley DS, Skarecky D, Ahlering TE. J Urol. 2009;181:259.* Overall 90% of men reported return of potency at 24 months and 46% returned to baseline with normal 5-item International Index of Erectile Function (IIEF) scores and 100% firmness. There was no difference in 5-item IIEF scores or fullness between unilateral and bilateral nerve sparing.

Chronic interpersonal stress predicts activation of pro- and anti-inflammatory signaling pathways 6 months later. *Miller GE, Rohleder N, Cole SW. Psychosom Med. 2009;71:57.* Chronic interpersonal difficulties (a study on 103 healthy young women) accentuate expression of pro- and anti-inflammatory signaling molecules. Although this process does not result in systemic inflammation under quiescent conditions, it does accentuate leukocytes' inflammatory response to microbial challenge. These dynamics may underlie the excess morbidity associated with social stress, particularly in inflammation-sensitive diseases like depression and atherosclerosis.

Effect of vaginal polypropylene mesh implants on sexual function. *Gauruder-Burmester A, Koutouzidou P, Tunn R. Eur J Obstet Gynecol Reprod Biol. 2009;142:76.* Since a very high rate of dyspareunia and impairment of sexual function is reported in women after vaginal mesh repair, a validated questionnaire was administered to 120 women to explore sex life before and after polypropylene mesh insertion (Apogee(R) / Perigee(R)) 1 year after surgery, and gynecologic examinations were performed preoperatively and postoperatively to assess urogenital anatomy and function. No woman complained of dyspareunia at 1-year follow-up. In 40 patients (33.3%), analysis of the validated questionnaires revealed more deeply rooted sexual disorders based on partnership problems and unrelated to surgery. The Authors conclude that sexual dysfunction is only rarely associated with urogynecologic surgery.

10 – MISCELLANEOUS

Female cosmetic genital surgery. *Goodman MP. Obstet Gynecol. 2009;113:154.* Genital plastic surgery for women has come under scrutiny and has been the topic of discussion in the news media, online, and in medical editorials. In the absence of measurable standards of care, lack of evidence-based outcome norms, and little standardization either in nomenclature or training requirements, concern has been raised by both ethicists and specialty organizations. Some women request alteration of their vulvas and vaginas for reasons of cosmesis, increasing self-esteem and improving sexual function.

Budesonide induction and maintenance therapy for Crohn's disease during pregnancy. *Beaulieu DB, Ananthakrishnan AN, Issa M et al. Inflamm Bowel Dis. 2009;15:25.* Budesonide (Entocort EC, AstraZeneca) is an enteric coated locally acting glucocorticoid preparation whose pH- and time-dependent coating enables its release into the ileum and ascending colon for the treatment of mild to moderate Crohn's disease. Budesonide was used during pregnancy at the 6 mg/day dose in 6 patients and 9 mg/day dose in 2 patients. There were no cases of maternal adrenal suppression, glucose intolerance, ocular side effects, hypertension or fetal congenital abnormalities.

Prevalence of anal squamous intra-epithelial lesion in women presenting genital squamous intra-epithelial lesion. *Giraldo P, Jacyntho C, Costa C et al. Eur J Obstet Gynecol Reprod Biol. 2009;142:73.* To determine the frequency of anal squamous intra-epithelial lesions (ASIL) in women with genital squamous intra-epithelial lesions (GSIL), 184 patients with histopathological diagnosis of GSIL and 76 controls without GSIL, were submitted to anoscopy in order to determine the presence of ASIL. All the women were HIV-negative. The frequency of ASIL was 17.4% in the GSIL group and only 2.6% in the control group. All the high grade ASIL diagnoses were found in women with cervical SIL.