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**PELVIC FLOOR DYSFUNCTION: ITS ASSESSMENT BY MRI AND
STRENGTH DURATION TEST AND ITS RESPONSE TO BIOFEEDBACK**

by

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A thesis presented to The University of Dublin, Trinity College, Ireland,
for the degree of Doctor of Medicine (M.D.)

in

The Faculty of Health Sciences, University of Dublin

Trinity College

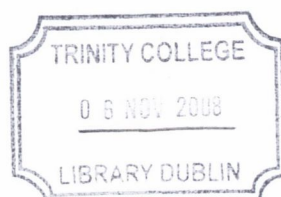
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E.I. Eguare, MB. BS, FRCSI

DEDICATION

I would like to dedicate this thesis to my wife Sandra for her unfailing love and ever present support.

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This work would not have been without the help and encouragement of Professor Frank B.V. Keane, who took me on board, tutored me and broadened my horizon. He painstakingly saw through every step of this project and his thought-provoking questions always brought me back to the drawing board to search for answers and to improve. He also kindly provided the photographs of the pelvic floor anatomy used in chapter one.

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SUMMARY

The component features of pelvic floor dysfunction (PFD) are interrelated but the development of a unified strategy to investigate and treat these problems has been slow; this has invariably affected the outcome of available treatment options. This lack of an integrated approach to the concept of PFD, stems from our inadequate understanding of its pathophysiology, and the traditional compartmentalisation of the pelvic floor.

To improve our understanding of pelvic floor function, various investigative modalities have been employed but none of them has been able to offer sufficient explanation in this regard. There also exist difficulties with the establishment of objective parameters, which can be used in evaluating patients' response to therapy for PFD. The present situation where a response to treatment is based principally on the patients' perceived improvement, needs to be reviewed.

This thesis aims to prospectively evaluate the use of phased array, body coil dynamic pelvic floor MRI in the global assessment and identification of pelvic floor abnormalities in patients with PFD. It also aims to evaluate techniques for assessing and objectively monitoring functional improvements in anorectal function following therapy.

To achieve these aims, multi-modal cohort studies were carried out. These included the use of phased array, body coil dynamic pelvic floor MRI to prospectively assess subgroups of patients with idiopathic combined faecal and urinary incontinence, obstructive defecation and asymptomatic controls. This study has enabled us to demonstrate distinct anatomical abnormalities in the levator plate complex in patients with pelvic floor dysfunction. Furthermore the combination of our findings on MRI and evacuation proctography has also

offered us the ability to better classify abnormalities in the obstructive defecation subgroup of patients, thus enhancing the tailoring of therapy to patients' needs.

This thesis also evaluated the use of strength duration test (SDT) as an alternative method of assessing pelvic floor innervation and anal sphincter function. We now know that pudendal nerve terminal motor latency test is not a very accurate test as the presence of few surviving fast conducting nerve fibres will provide a normal latency test. Analysis of SDT parameters shows that it correlates well with the pressure and sensory components of anorectal manometry. It also showed a high level of repeatability and good correlation with the quality of life scores in patients with faecal incontinence. The ease of learning and performing this test; and its minimal invasiveness, makes it a potential adjunct to the armamentarium for assessing the pelvic floor.

Another section of this work looked at the role of electromyographic (EMG) biofeedback in the treatment of faecal incontinence and the determination of objective parameters for evaluating patients' response to it. This study concludes that using validated incontinence scoring systems and manometric parameters, recordable improvements were achieved with EMG biofeedback therapy.

The use of anorectal ultrasonography (performed by colorectal surgeons or senior trainees under supervision), as a complimentary investigative tool for assessing PFD was also evaluated. Comparing our findings with phased array pelvic MRI, and using pathological staging as the "gold standard", tend to suggest that anorectal ultrasonography was more accurate and more sensitive. The need for colorectal surgeons and trainees to acquire skills in the performance of anorectal ultrasonography is highlighted and should be developed.

***“Man should strive to have his intestines relaxed
all the days of his life”***

Moses Maimonides, AD 1135-1204

***“A good set of bowels is worth more than any
quantity of brains”***

Josh Billings (Henry Wheeler Shaw), AD 1818-1885

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LIST OF ABBREVIATIONS

ANOVA	Analysis of variance
ARM	Anorectal manometry
CP	Circularly polarized
CT	Computerized axial tomography
DRE	Digital rectal examination
EAS	External anal sphincter
EAUS	Endoanal ultrasonography
ELUS	Endoluminal ultrasonography
EMG	Electromyography
ENS	Enteric nervous system
ERUS	Endorectal ultrasonography
FD	Fibre density
FDV	First desire to void
FI	Faecal incontinence
FIQOL	Faecal incontinence quality of life
HPZ	High pressure zone
IAS	Internal anal sphincter

IG	Intersphincteric groove
IL	Inner layer of the external anal sphincter
LAM	Levator ani muscle
LP	Levator plate
MRI	Magnetic resonance imaging
OD	Obstructive defecation
PET	Positron emission tomography
PFD	Pelvic floor dysfunction
PNE	Percutaneous nerve evaluation
PNS	Peripheral nerve stimulation
PNTMLT	Pudendal nerve terminal motor latency test
PR	Puborectalis
PW	Pulse width
QOL	Quality of life
RAIR	Rectoanal inhibitory reflex
SD	Standard deviation
SDC	Strength duration curve
SDT	Strength duration test
SDV	Strong desire to void
SNS	Sacral nerve stimulation
STC	Slow transit constipation
TEMS	Trans anal endoscopic microsurgery
TNS	Temporary nerve stimulation

CHAPTER 1

INTRODUCTION

1.1 Overview of pelvic floor dysfunction

Functional disorders of the pelvic floor have been a common clinical problem over the years; and though a significant proportion of the population suffer from it, very little attention has been paid to it. The reason for this is attributable to social embarrassment, which prevented the sufferers from speaking about it. For instance faecal incontinence affects approximately 2% of the general adult population (Nelson, Norton et al. 1995) and the prevalence of pelvic prolapse is up to 16% in the population (Hagstad, Janson et al. 1985; Hording, Pedersen et al. 1986; Cruikshank 1987; Yang, Mostwin et al. 1991). The incidence of urinary incontinence in primiparous women six months after child birth is about 26% and at nine months postpartum 26% of women still had some degree of anal incontinence. Constipation affects between 3 and 15 per cent of the general population at any one time (Thompson and Heaton 1980; Sandler and Drossman 1987) and more than 4 million people in the U.S. (Sonnenberg and Koch 1989; Zetterstrom, Lopez et al. 1999; Farrell, Allen et al. 2001).

Furthermore, the historical turf battle of compartmentalisation amongst the Urologists, Gynaecologist and Coloproctologists hampered concerted efforts to find solution to the problems of pelvic floor disorders. Pelvic floor disorders present with signs and symptoms, which include pain, faecal, urinary, or combined faecal and urinary incontinence, obstructive defecation, organ descensus, organ prolapse, urethral obstruction and possible rectal ulceration (Zacharin 1985; Healy, Halligan et al. 1997; Bertschinger, Hetzer et al. 2002). These signs and symptoms occur in varying combinations and severity, but the concept of treating these disorders according to compartments has made their investigation and management difficult.

In recent years there has been an increasing interest in anorectal functional abnormalities and greater awareness of the available treatment modalities has increased demand for specialized imaging and investigative tools (Osterberg, Graf et al. 2000; Stoker, Halligan et al. 2001). Investigations for pelvic floor assessment fall broadly into two categories: physiological and radiological investigations. Laboratory neurophysiological tests include anorectal manometry, balloon expulsion test, pudendal nerve terminal motor latency test, electromyography and urodynamic test; while the radiological tests include evacuation proctography, which involves static and dynamic fluoroscopy; video cystourethrography, endoanal-ultrasonography, CT-pelvimetry and dynamic magnetic resonance imaging of the pelvis.

The availability of so many investigations looking at a particular aspect of body function suggest that either none of these tests is completely satisfactory or the complex nature of the pelvic floor requires various tests that will assess its different aspects. Many of these investigations have played pivotal roles in enhancing our understanding of pelvic floor function, but inherent deficiencies still exists. The results of many of these tests are not reproducible and therefore not of absolute diagnostic value. In addition, many of these investigations are invasive, intrusive and uncomfortable, thereby compromising patients' compliance. Some of them also have the added disadvantage of imparting high-dose irradiation, which makes them contraindicated in many circumstances and renders them ethically unacceptable as research tools.

1.2 Thesis aims and objectives

The aims and objectives of this research thesis are to explore and evaluate pelvic floor investigative procedures that are:

- 1) Minimally invasive
- 2) Less intrusive on patients' privacy
- 3) Devoid of high-dose radiation
- 4) Biologically acceptable to patients of every age and sex
- 5) Capable of assessing the pelvic floor globally with detailed depiction of pelvic floor structures
- 6) Have reproducible, measurable parameters that can be used as prognostic and predictive indices for pelvic floor assessment.

I shall critically evaluate Magnetic Resonance Imaging (MRI) as an investigative tool for the assessment of pelvic floor dysfunction and to determine the usefulness of Strength Duration Test (SDT) as a useful ancillary neurophysiological investigation for pelvic floor assessment. In this regard, I will attempt to correlate morphological findings of pelvic floor MRI with clinical signs and symptoms in patients with pelvic floor dysfunction.

1.3 Subtypes of pelvic floor dysfunction

1.3.1 Faecal incontinence

Faecal incontinence is defined as recurrent uncontrolled passage of faecal material in an individual with a developmental age of at least four years (Whitehead, Wald et al. 2000). The incidence of faecal incontinence is about 7-9.5% in adults over 65 years old and it has a significant impact on the quality of life of these patients (Kinnunen 1991; Drossman, Li et al. 1993). Thomas et al reported the prevalence of faecal incontinence in the general population as 4.2/1000 men and 1.7/1000 women in the 15-64 years age range and 10.9/1000 men and 13.3/1000 women in the over 65 years age range (Thomas, Egan et al. 1984).

Faecal incontinence encompasses a spectrum of diverse aetiologies that include anal sphincter complex injuries, pudendal nerve neuropathy, and abnormalities in intestinal motility, leading to diarrhoea or constipation with overflow; impaired rectal sensation, rectal reservoir dysfunction, or weak pelvic floor musculature. In

the absence of an identifiable underlying cause for a patients' incontinence then a clinical diagnosis of idiopathic faecal incontinence is considered.

1.3.2 Combined faecal and urinary incontinence

Combined faecal and urinary incontinence occurs more frequently than is generally assumed with prevalence, estimated to be about 5% in males and 10% in females (Roberts, Jacobsen et al. 1998) . In the hospital setting, the prevalence of faecal incontinence in female patients attending urology and uro-gynaecology clinics varies from 15-30% (Jackson, Weber et al. 1997) . Stress urinary incontinence is a symptom that results from damage to the muscles, nerves, and connective tissue of the pelvic floor leading to disruption of urethral support and vesical neck function. Similarly, symptoms of faecal incontinence arise when there is damage to the muscles and endopelvic fascial support system of the anorectal complex. When these defects coexist or overlap, it results in the manifestation of symptoms of combined faecal and urinary incontinence (DeLancey 1997; Steinke, Hetzer et al. 2002).

1.3.3 Constipation

Constipation is said to occur when at least two of the following feature are present: (1) straining during more than 25% of bowel movements. (2) Sensation of incomplete evacuation with at least 25% of bowel movements. (3) Hard or pellet-like stools at more than 25% of bowel movements and (4) Bowel movement frequency of less than 2 per week with or without symptoms of constipation (Whitehead, Chaussade et al. 1991; Rotholtz and Wexner 2001).

The problem of constipation has a significant impact on the health care system with as many as 4,000,000 people affected in the United States; and constipation accounting for 0.9% and 1.2% of physician visits yearly in the UK and USA respectively (Sonnenberg and Koch 1989; Sonnenberg and Koch 1989; Johanson and Sonnenberg 1990). About \$800 million is spent annually in the USA for the procurement of prescribed laxatives (Anderson 1985). The symptoms of obstructive defecation are reported in about 40-74% of patients referred to specialist centres with complaints of constipation, and the intractability of this problem significantly impairs the quality of life of the patients (Kuijpers 1990; Lemieus and Kamm 1994).

Chronic constipation comprises of functional slow transit constipation (colonic inertia) and difficult (Obstructive) defecation, which is characterised by symptoms of straining, feeling of incomplete evacuation or the need to facilitate defecation digitally either by manual evacuation or application of trans-vaginal digital pressure or perineal support manoeuvre (Rotholtz and Wexner 2001; Wiesel, Dorta et al. 2001).

1.3.4 Solitary rectal ulcer syndrome

Solitary rectal ulcer syndrome (SRUS) is a rare clinical condition, which is often under-diagnosed (Haray, Morris-Stiff et al. 1997). It is characterized by manifestations such as rectal bleeding, mucous discharge, tenesmus, prolonged straining, difficult evacuation, and localized perineal pain (Halligan, Nicholls et al.

1995; Ertem, Acar et al. 2002). Little was known about SRUS as a clinical entity before the classic description of its clinical and pathologic features in 1969 (Madigan and Morson 1969). Since then investigations such as barium enema, evacuation proctography, endorectal ultrasonography, and anorectal manometry have been used to diagnose it and detect the causative disorder (Simsek, Yagci et al. 2004).

Due to the poor understanding of the pathogenesis of SRUS, many of the cases (25%) are still being misdiagnosed, and the correct diagnosis usually delayed for about 5 to 7 years (Sondheimer, Slagle et al. 1985; Figueroa-Colon, Younoszai et al. 1989; Tjandra, Fazio et al. 1992). The most widely accepted postulation of causal mechanism is that associated with rectal prolapse, which is brought about by excessive straining during defecation. The concomitant increased intra-abdominal pressure generated causes the anterior rectal wall mucosa to prolapse through the contracting puborectalis muscle and the anal canal. This mucosal intussusception through the high-pressure zone of the non-relaxing puborectalis and anal canal results in mucosal strangulation, congestion, oedema and ulceration (Figueroa-Colon, Younoszai et al. 1989; Eigenmann, Le Coultre et al. 1992).

Making a diagnosis of SRUS poses significant clinical problem because non of the features associated with the syndrome are not exclusive to it nor a prerequisite for its diagnosis (Halligan, Nicholls et al. 1995). However the presence of a combination of features and having a high index of suspicion is

essential to make a correct diagnosis. Features associated with SRUS are the presence of a single ulcer on the anterior rectal wall; strict adherence to this feature or the nomenclature of “solitary” can be misleading because ulcers can vary in position, be multiple or absent (Tjandra, Fazio et al. 1992). The characteristic histological feature of fibromuscular proliferation and obliteration of the lamina propria with muscle fiber disorientation is attributed to chronic mechanical and ischemic trauma, inflammation by hard stools, and intussusception of the rectal mucosa (Sondheimer, Slagle et al. 1985; Figueroa-Colon, Younoszai et al. 1989; Tjandra, Fazio et al. 1993). However, this feature can be seen at any site with prolapsing large bowel mucosa (du Boulay, Fairbrother et al. 1983). Extensive radiological study of this syndrome has also been carried out. Halligan et al. concludes that evacuation proctography features (usually seen in combination) associated with SRUS are prolonged or incomplete rectal evacuation, extended descent of the pelvic floor, and an increased frequency of internal or external rectal prolapse (Halligan, Nicholls et al. 1995).

The treatment of SRUS is difficult and traditional therapy mainly focuses on behavioural approach (Malouf, Vaizey et al. 2001). Recent studies have shown that biofeedback therapy is associated with successful outcome as demonstrated by increased rectal mucosal blood flow (Jarrett, Emmanuel et al. 2004). In properly selected patients and in patients with significant rectal prolapse as demonstrated by clinical examination or defecography good result is achieved from surgical repair of the prolapse (Choi, Shin et al. 2005).

1.3.5 Rectal prolapse

Rectal prolapse is defined as partial or full thickness protrusion of the rectal wall through the anal canal. It is a disabling condition, which typically occurs in elderly females but may occur in both sexes and at any age. Historically rectal prolapse has been reported since the Egyptian and Greek civilisation, but its aetiology remains poorly understood (Boutsis and Ellis 1974; Heitland 2004). The pathophysiology is thought to arise from pelvic floor weakness or atrophy, (present in the elderly, multi-parous women and spinal cord disease) and mid rectal intussusception, which occurs about 8-10cm from the anal verge. The commonest features associated with aetiology are the antecedent history of chronic straining and slow-transit constipation. However, in most cases, no single cause can be identified.

Investigations comprise of initial history and clinical examination. The technique of examining the patient in the squatting position, while straining, is useful in demonstrating the prolapse. Other investigations are video defecography, anorectal manometry and more recently, dynamic magnetic resonance imaging.

Rectal prolapse is classified as:

- ***Concealed prolapse***, in which there is internal intussusception of the upper rectum into the lower rectum, without the emergence of the prolapse through the annual verge.

- ***Incomplete or mucosal prolapse*** is limited to mucosal prolapse and can occur at any age. It is often associated with straining, constipation and haemorrhoids.
- ***Complete prolapse (proctidentia)*** is the full thickness prolapse of the rectum through the anal verge. It contains two layers of the rectal wall with an intervening peritoneal sac.

Most of the patients affected by this disorder are generally poor in health with numerous co-morbidities (Kimmins, Evetts et al. 2001). This has necessitated the ongoing search for an ideal surgical repair that can offer functional and morphological correction of pelvic floor dysfunction, be minimally invasive and cost-effective, and result in minimal morbidity and recurrence (Kimmins, Evetts et al. 2001; Heitland 2004; Sobrado, Kiss et al. 2004). This ongoing controversy of the ideal surgical procedure has led to the development of more than one hundred operations. Broadly, the surgical approaches for repair are classed into abdominal, perineal, and more recently the laparoscopic approach (Kim, Tsang et al. 1999; Kimmins, Evetts et al. 2001; Madbouly, Senagore et al. 2003; Chun, Pikarsky et al. 2004; Schiedeck, Schwandner et al. 2005). The choice of operation is influenced by the age and general health of the patient, and whatever the choice the principal goal should be to prevent recurrence of prolapse and improve continence and bowel function.

1.3.6 Descending perineum syndrome

Descending perineum syndrome (DPS) is a symptom complex, which shows considerable variation, ranging from obstructed defecation to combined faecal and urinary incontinence and including different types of prolapse (Schwandner, Poschenrieder et al. 2004). It is the increased mobility and the ballooning down of the perineum, usually because of chronic straining, multiple vaginal deliveries and previous perineal surgeries. Though, the concept of DPS was first described by Alan Parks in 1970, the syndrome remains difficult to interpret clinically and pathophysiologically (Hardcastle and Parks 1970; Villet, Ayoub et al. 2005). Some reasons adduced for this difficulty are that symptoms observed are secondary to associated lesions, and radiological signs of DPS are not always associated with clinical symptoms (Bartolo, Read et al. 1983; Villet, Ayoub et al. 2005).

To elucidate the complexity of this syndrome, detailed history, clinical examination with proctoscopy as well as investigations such as anorectal manometry, endoanal ultrasound, defecating proctogram and dynamic MRI of the pelvic floor are undertaken (Schwandner, Poschenrieder et al. 2004). Studies have shown that dynamic MRI is superior to fluoroscopic methods in evaluating DPS. A study carried out by Harewood et al. showed that the most prevalent abnormality on clinical testing is perineal descent >4cm; they also showed that balloon expulsion test was an insensitive screening test for this disorder (Harewood, Coulie et al. 1999).

Because of the multiple aetiologies of DPS, a multi-disciplinary approach to treatment is recommended. Pelvic floor retraining by biofeed-back can be employed but its success is dependent on the severity of perineal descent (Harewood, Coulie et al. 1999; Schwandner, Poschenrieder et al. 2004). D'Amico and Angriman conclude that most treatment of DPS is aimed at the predominant symptoms since the correction of the primary problem of excessive perineal descent is impossible (D'Amico and Angriman 2000). No consensus has been reached concerning the surgical management of DPS, however where there are obvious associated pathologies that are amenable to surgery then they should be treated surgically (Villet, Ayoub et al. 2005).

1.3.7 Dysfunction due to neoplasm

Anorectal neoplasm can produce symptoms, which can be mistaken for pelvic floor dysfunction. The presence of large villous adenoma in the rectum can produce copious amounts of mucus, which can result in sensation of urge incontinence. Presence of rectal lesions can also present with sensation of incomplete emptying or obstructive defecation. Occasionally, locally advanced anorectal tumours can damage the sphincter mechanism or even infiltrate the pelvic nerves leading to pelvic dysfunction, in form of pain or incontinence.

1.4 Anatomy of the pelvic floor

1.4.1 Pelvic Floor

The pelvic floor consists of three layers: endopelvic fascia, levator ani muscle and perineal membrane (Figure 1.1 and 1.2). The endopelvic fascia drapes the superior surface of the pelvic viscera and is condensed in a number of areas to form distinct ligaments. These include the pubourethral, urethropelvic, pubocervical and cardinal ligaments. The endopelvic fascia is a fibromuscular tissue composed of collagen, elastin and smooth muscle (DeLancey 1992). The histological structure of the endopelvic fascia varies in different areas of the pelvis.

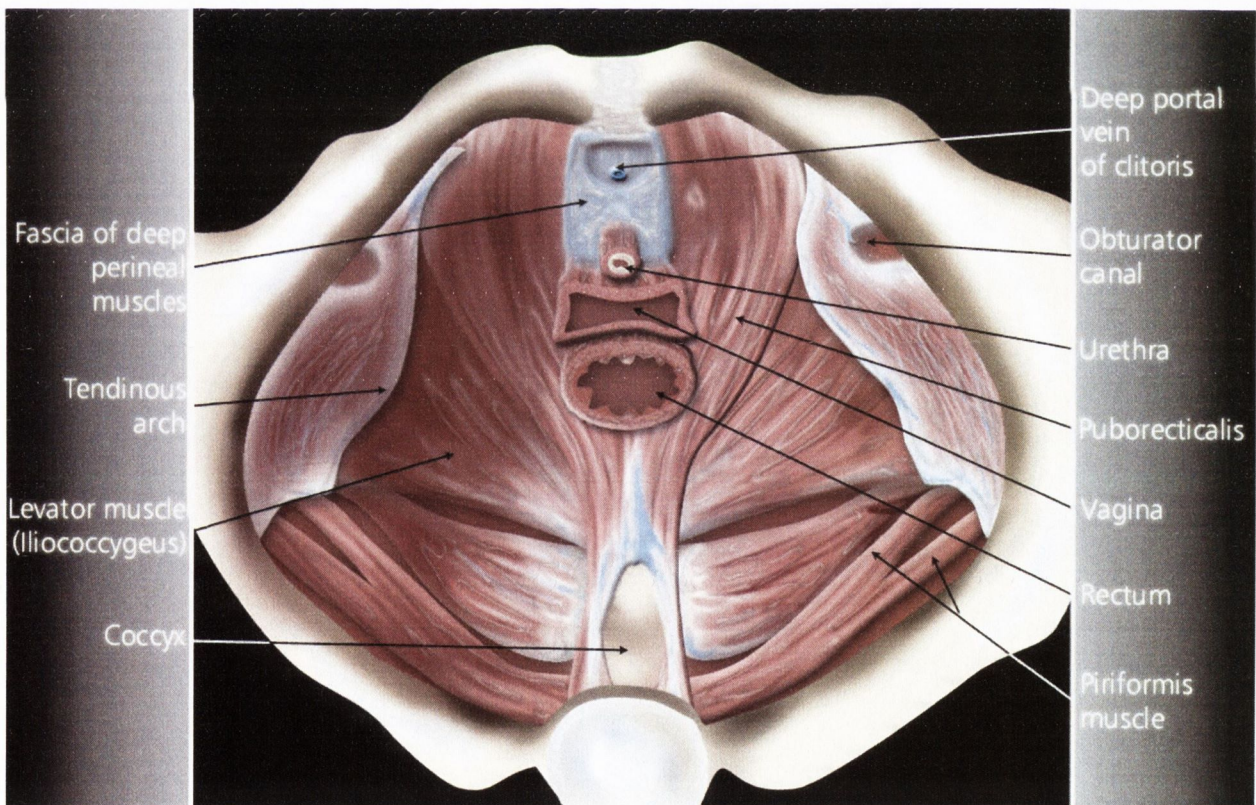


Figure 1.1: Axial view of the pelvic floor (Photograph kindly provided by Professor FBV Keane)

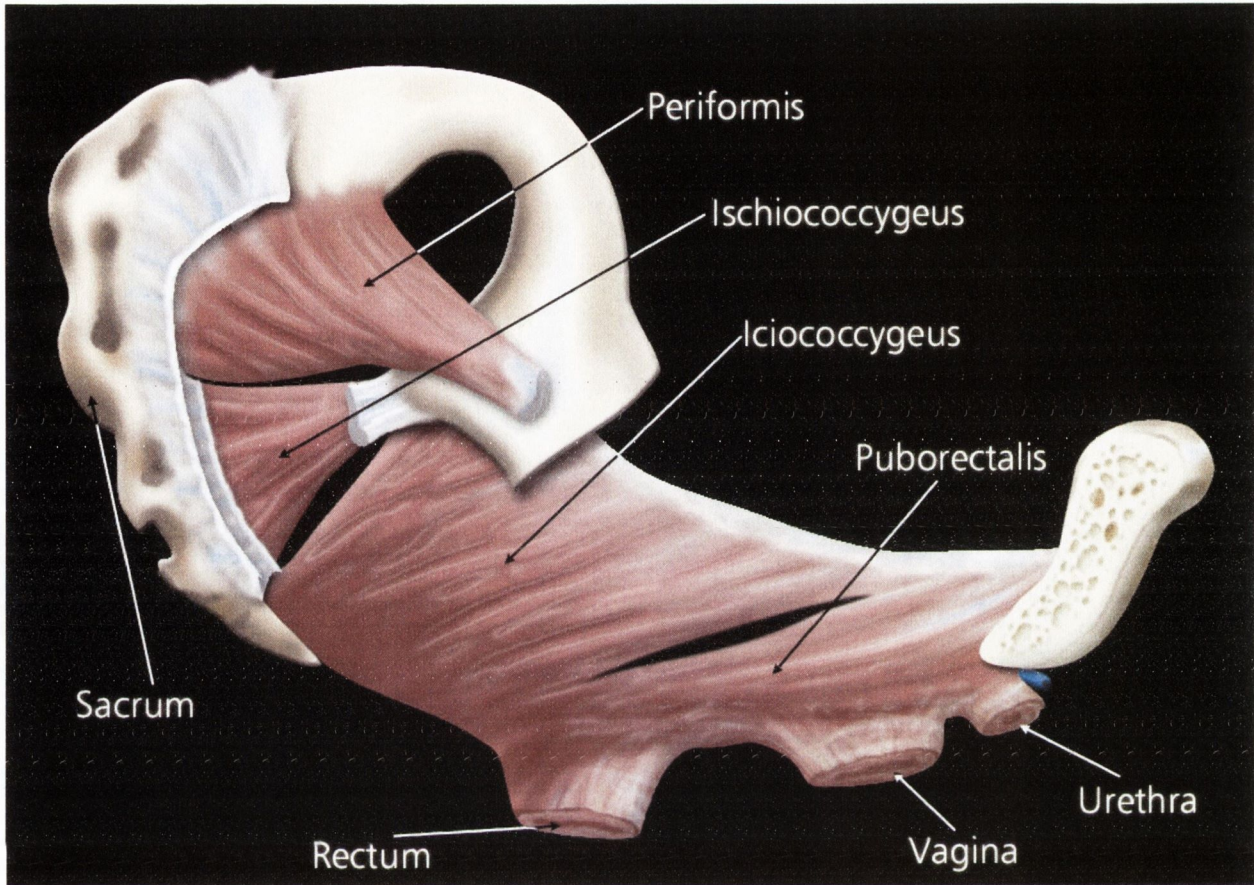


Figure 1.2: Sagittal view of the pelvic floor (*Photograph kindly provided by Professor FBV Keane*)

The autonomic nerve supply to the pelvic viscera runs in this layer (Figure 1.3). The muscle layer is formed by the “so called” gutter-shaped sheet of muscle, the pelvic diaphragm, slung around the mid line body effluents (urethra, anal canal and in the female, the vagina) that transverse it in close proximity (Lawson 1974; Last 1994) . This sheet of muscle, which is predominantly composed of striated muscle, is made up of the coccygeus and levator ani. The coccygeus is further divided into, ischiococcygeus, iliococcygeus and pubococcygeus. Though separate parts of the muscle are given different names, they are phylogenetically a unit and a morphological entity. The iliococcygeus, which arises from the posterior part of the arcus tendineus and the pelvic surface of the

ischial spine, is inserted into the side of the coccyx and the anococcygeal ligament and raphe. This muscle extends from the tip of the coccyx to the anorectal junction and is particularly prone to passive stretching during straining at defecation or in the second stage of labour.

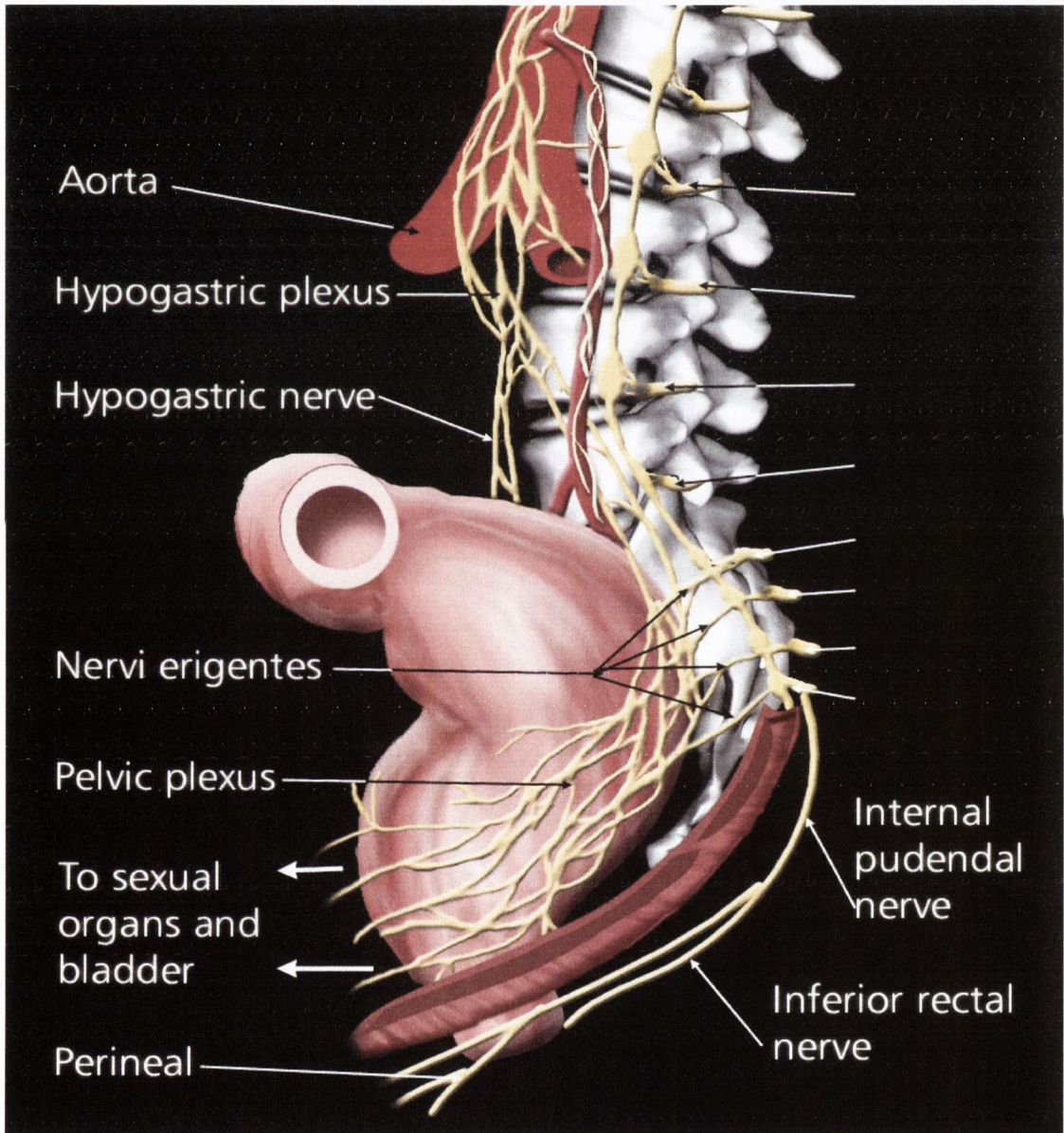


Figure 1.3: Innervation of the pelvic floor (*Photograph kindly provided by Professor FBV Keane*)

The pubococcygeus arises from the anterior half of the arcus tendineus and the posterior surface of the body of pubis, its more anterior fibres arising from the periosteum of the body of the pubis swings inferiomedially around the anorectal junction and unites with equivalent fibres from the opposite side to form the U-shaped hammock-like sling called the puborectalis. The puborectalis holds the anorectal junction angled forward and it plays a major role in the mechanism of defecation and maintenance of continence. More medial fibres of the pubococcygeus form similar slings around the prostate, vagina and urethra and are named, puboprostatae, pubovaginalis and pubourethrae respectively.

The opening in the levator ani muscle through which the effluent structures exit the pelvic floor is called the levator (urogenital) hiatus. The levator muscle is composed of both type 1 (slow twitch) and type 2 (fast twitch) muscle fibres. The type 1 fibres predominate reflecting its main role in maintaining a constant resting tone. Different parts of the levator ani have different relative proportions of type 1 and 2 fibres (Critchley 1980). The normal baseline activity of the levator ani muscle keeps the urogenital hiatus closed. It squeezes the urethra, vagina and rectum closed by compressing them against the pubic bone and lifts the floor and organs in a cephalic direction. It maintains a constant resting tone and this constant action eliminates any opening within the pelvic floor through which prolapse could occur and forms a relatively horizontal shelf on which the pelvic organs are supported (Berglas and Rubin 1953; Parks, Porter et al. 1962; Nichols, Milley et al. 1970).

1.4.2 Perineal membrane

The perineal membrane, which is a dense, triangularly shaped membrane lies in the anterior pelvis, below the pelvic diaphragm. It spans the inferior surface of the levator ani muscles and provides attachment for the urethra, vagina, and perineal body to the ischiopubic rami. It supports the perineal body and is believed to be important in preventing the downward descent of the perineal body and lateral vaginal walls beyond their elastic limit, during relaxation of the levator ani muscle. Support of the pelvic floor outlet is maintained by the perineal membrane, its attachments and the strong upward pull exerted by the levator ani (Koelbl, Strassegger et al. 1989) . There is controversy about the importance of the connective tissue component of the pelvic floor. However, it has been suggested that deficient or abnormal collagen may cause or contribute to pelvic floor dysfunction.

1.4.3 Rectum

The rectum commences at the rectosigmoid junction, which is usually at the level of the sacral promontory (Figure 1.4). It measures about 15-20cm in length and it follows the anterior concavity of the sacrum. It transverses the levator hiatus and ends at the top of the anal canal. The rectum has three intraluminal valves (valves of Houston), which partially encircles the rectum. The middle rectal valve (Kohlrausch's) is most consistent in its position and corresponds to the anterior peritoneal reflection. The rectum is surrounded by areolar and mesorectal tissue, which contains terminal branches of the inferior mesenteric artery and lymphatics. The upper third of the rectum is covered by peritoneum on its

anterior and lateral sides, the middle third has anterior peritoneal covering, and the lower third is completely without peritoneal covering. The anorectal junction is situated 2-3cm in front of and below the tip of the coccyx. Distal condensations of the visceral pelvic fascia form the lateral ligaments of the rectum, which may contain branches of the middle haemorrhoidal vessels. These ligaments attach the rectum to the lateral pelvic wall. The presacral fascia (fascia of Waldeyer) covers the sacrum, coccyx and pelvic nerves. Anteriorly, a thick fascial septum (fascia of Denonvilliers) separates the rectum from the prostate and seminal vesicles in the male, and the vagina in the female.

1.4.4 Anal Canal

The anal canal is the last 4cm of the alimentary tract and is developed from the anorectal canal and the proctoderm. Anatomically, the anal canal extends from the anal verge to the dentate line (Figure 1.4). Above the dentate line, the mucosa is arranged in a number of vertical folds called the columns of Morgagni, and below the dentate line, the canal is lined by modified squamous epithelium that is smooth, pale and devoid of hair or glands. Anteriorly the anal canal has a crucial relationship with the perineal body and the posterior wall of the vagina in the female, as they lie in close proximity. The anal canal is composed of two muscles: the internal and external anal sphincter muscles. The internal anal sphincter is an involuntary smooth muscle. It is a direct continuation of the circular muscle layer of the rectum and is under autonomic control. It is 2-3mm thick and 2.5-4.0cm in length. The internal anal sphincter derives its enervation from sympathetic, (L₅) and parasympathetic (S_{2, 3, 4}) nerve

fibres.

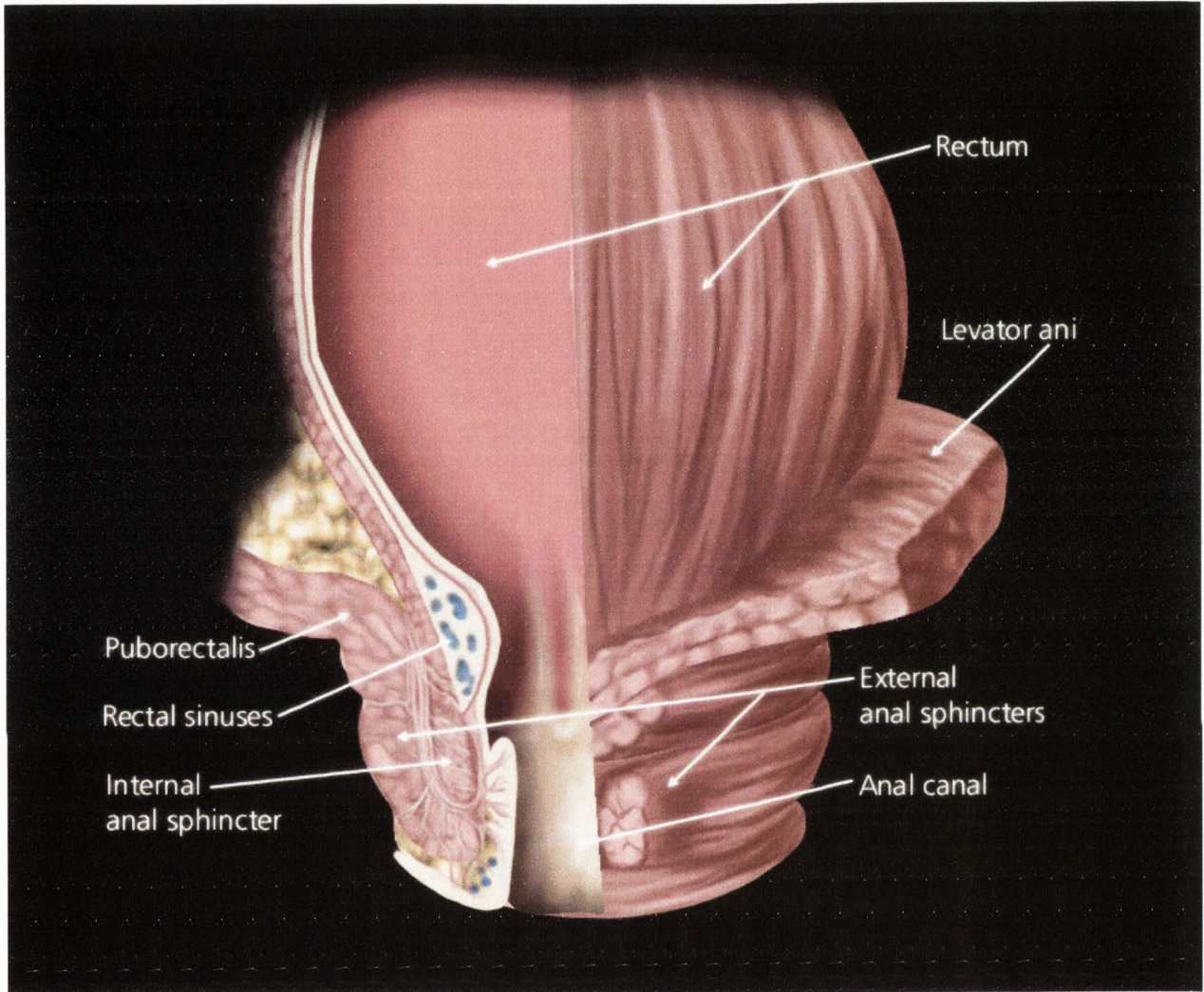


Figure 1.4: Anatomy of the Anorectum (*Photograph kindly provided by Professor FBV Keane*)

It is composed of slow twitch fibres. This muscle accounts for 50-75% of anal resting pressure (Frenckner and Euler 1975; Wunderlich and Parks 1982; Lestar, Penninckx et al. 1989). External anal sphincter is a voluntary skeletal muscle, which is composed of fast twitch muscle fibres. It is the outermost muscle of the anal sphincters and anatomically, has previously been described as consisting of

three individual components. However functionally it acts as a single unit. It extends below the level of the internal sphincter. The external anal sphincter muscle is responsible for 10-15% of resting pressure and the majority of anal sphincters squeeze pressure. The external anal sphincter increases anal canal pressure under conditions of increased intra-abdominal pressure and is supplied by inferior rectal branches of the pudendal nerve.

1.4.5 Innervation of the anorectum

The colon, rectum and anal canal have both autonomic and motor innervation. The sympathetic innervation is derived from the T₁₁₋₁₂ and the lumbar segments, and the parasympathetic supply is derived from the vagus and S₁₋₃ sacral segments. Parasympathetic supply of the left colon, rectum and anus comes from the second, third and fourth sacral nerves, the pelvic splanchnic nerves or *nervi erigentes* (Figure 1.3). While the sympathetic component of the autonomic innervation causes inhibition of colonic peristalsis and secretion, the parasympathetic stimulation increases them. Motor fibres to the external anal sphincter are carried in the pudendal nerve (S₂ and S₃) and, if present, the perineal branch of S₄.

Apart from the autonomic and motor nerve supply, the gut also has an intramural, intrinsic nervous system termed the enteric nervous system (ENS), which primarily controls gastrointestinal motility (Langely 1921). The two major components of ENS, interlinked by connecting ganglia are the myenteric plexus of Auerbach and the submucosal plexus of Meissner. The myenteric plexus is

situated between the circular and longitudinal layers of the muscularis propria, while the Meissner's plexus is located in the submucosal plane. Postganglionic sympathetic fibres from thoracolumbar outflow, and parasympathetic innervation via the vagus and splanchnic nerves to the postganglionic cell bodies within the bowel walls innervate the myenteric plexus. This system contains an entire reflex pathway, which permit peristaltic contraction independent of extrinsic enervation. This system also contain neurons that are able to monitor factors such as tension in the bowel wall or the chemical nature of luminal contents, and motor neurons which alter the mechanical activity of the intestine (Cook and Mortensen 2002).

1.5 Functional anatomy of the lower urinary tract

The urinary tract comprises of the bladder, urethra and a functionally distinct transition zone between these two called the vesical neck. The mechanism of continence of urine is maintained by the integrated function of the bladder, vesical neck, and urinary sphincters, supporting ligaments, nerves and muscles of the pelvic floor (Figure 1.5).

1.5.1 Bladder

The bladder consists of the detrusor muscle, covered by an adventitia and serosa over its dome. The bladder is composed of a dome and a trigone. The lumen is lined with transitional cell epithelium, and the submucosa is made of loose areolar tissue, which allows for the distension of the bladder.

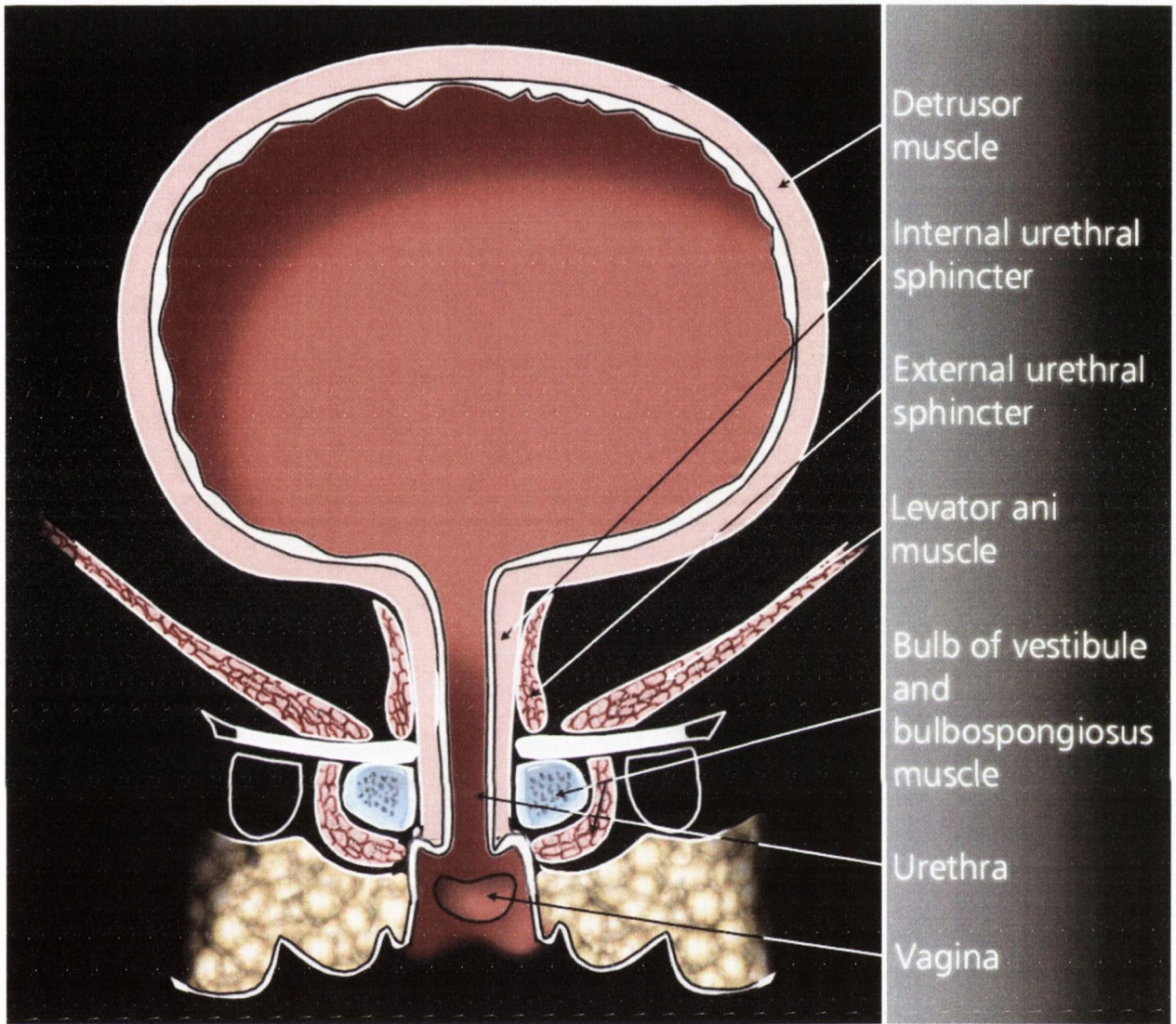


Figure 1.5: Urinary bladder with its musculofascial support structures
(Photograph kindly provided by Professor FBV Keane)

The detrusor muscle was originally described as consisting of three layers, namely outer longitudinal, middle circular-oblique and inner longitudinal. This description has been found to be inaccurate as these anatomical layers are not discrete. Functionally the detrusor muscle acts as a unit and is adapted for mass contraction, rather than peristalsis. With the exception of the

bladder trigone, the histological and histochemical characteristics of the detrusor muscle are uniform. They contain significant amounts of acetylcholinesterase, consistent with a rich parasympathetic nerve supply.

1.5.2 Trigone

This is a visible triangular elevation at the base of the bladder known as the vesical trigone. The trigone lies between the internal urethral orifice (centrally and below) and the two ureteral orifices (above and laterally). The trigone is composed of two anatomically distinct layers; the outer layer, containing deep trigonal muscle, which is similar to bladder detrusor muscle and the inner layer composed of a thin layer of superficial muscle bundles. The trigonal elevation is caused by the deposition of specialized smooth muscle, which are distributed circumferentially at the vesical neck, and are suggested to play some role in the closure of the vesical neck and the maintenance of continence. It has also been postulated that contraction of these fibres may be important in aiding the opening of the bladder neck, at the initiation of micturition. Individual muscle cells in this layer are devoid of acetylcholinesterase and the mucosa overlying the trigone frequently undergoes squamous metaplasia.

1.5.3 The vesical neck

This is located at the base of the bladder where the urethra traverses the musculature of the bladder wall. It is in this area that the detrusor musculature surrounds the trigone and the urethral meatus. It is now regarded as a distinct anatomic entity because of the uniqueness of its functional characteristics.

Autonomic neuropathy, which involves sympathetic denervation can result in the inability of this functional sphincter to close at rest and when this occurs in combination with stress urinary incontinence, attempts at surgical correction of the problem is frequently ineffective (McGuire 1981; McGuire 1986). It is known that only about 75% of continent nulliparous women have identifiable closed bladder neck; the remaining 25% with open bladder necks were not incontinent (Chapple, Helm et al. 1989). It is also known that about 50% of perimenopausal continent women have open bladder necks (Versi, Cardozo et al. 1990). This would tend to support the findings of Rud et al that the voluntary muscle of the urethra contributes largely to the mechanism of continence (Rud, Andersson et al. 1980). Fluoroscopic studies have shown also that contraction of the levator ani elevates the vesical neck and relaxation obliterates the posterior urethrovesical angle.

1.5.4 Urethra

The urethra is the conduit of urinary effluent from the bladder and is therefore an important determinant of urinary continence. In the female, it is about 4cm long and with the exception of its uppermost segment; the urethra is embedded within the vaginal wall. This makes the urethra particularly prone to stretching during pregnancy and parturition. The walls of the bladder neck and proximal urethra contain a high percentage of elastic fibre. It is hypothesized that they provide a passive occlusive resistance to bladder neck opening. This passive resistance in tandem with transmitted intra-abdominal pressure is thought to be important in

maintaining bladder neck closure. The urethra is composed predominantly of slow-twitch muscle fibres, which are well adapted to maintaining the constant tone exhibited by this muscle (Gosling, Dixon et al. 1981). This passive resistance is augmented by the activation of voluntary muscles during urethral contraction to ensure continence when increased closure pressure is needed. The voluntary muscle contributes approximately one third of resting urethral closure pressure (Rud, Andersson et al. 1980). Two arches of muscle, the compressor urethrae and urethral vaginal sphincter, are in close proximity to the distal urethra and act as a backup mechanism which can maintain urethral closure in spite of vesical neck incompetence or under circumstances where the urge to micturate must be deferred.

1.5.5 Innervation of the urinary tract

The nerve supply of the bladder derives from three sources: somatic, sympathetic and parasympathetic (Figure 1.3). The efferents from S₂₋₄ supply somatic innervation, particularly motor inputs to the urethral sphincter and the pelvic floor. Some of the fibres use the pudendal nerve as a conduit to supply the urethra and others travel directly to the superior surface of the levator ani muscle to supply it. Somatic afferents, which mediate sensation of bladder filling, and sensation from the urethra and pelvic floor, also travel in the pudendal nerve to the dorsal horn of S₂₋₄. Sympathetic efferents originate from T_{11-L2} segments of the spinal cord and they travel in the superior and inferior hypogastric plexuses to provide excitatory inputs to the smooth muscle of the bladder neck, urethra and seminal vesicles. However, these excitatory inputs stimulate the inhibitory

intramural ganglia in the bladder wall. Parasympathetic efferent fibres originate from the intermedio-lateral horns of S₂₋₄ segments of the spinal cord and their postganglionic axons provide cholinergic excitatory inputs to the bladder (Arnold 2002).

1.6 Pudendal nerve anatomy

The pudendal nerve arises from the anterior surfaces of S₂₋₄ nerves. The three branches fuse to form the nerve, which passes back between piriformis and coccygeus, medial to the pudendal vessels. It appears in the buttock and curls around the sacrospinous ligament to run forward into the ischioanal fossa. The nerve gives off the inferior rectal branch at the posterior end of the pudendal canal of Alcock then runs in the lower lateral wall of the fossa and medial to the ischial tuberosity, via the lesser sciatic notch to the deep perineal pouch situated above the perineal membrane and containing the urogenital diaphragm. The nerve divides in the canal into terminal branches (dorsal nerve of the penis and the perineal nerve). The perineal nerve is the larger terminal branches and it supplies the perineal muscles, external urethral sphincter and the mucous membrane of the urethra.

1.7 Physiology of the pelvic floor

The maintenance of normal continence in the three pelvic compartments of an individual is so important that Moses Maimonides (AD 1135-1204) was quoted

thus, *“Man should strive to have his intestines relaxed all the days of his life”*. Josh Billings (Henry Wheeler Shaw, AD 1818-1885) opined, *“A good set of bowels is worth more than any quantity of brains”*. While incontinence due to loss of integrity of the pelvic floor musculature and its fascial support system can be debilitating, the inability to effectively evacuate body effluents without undue strain is equally distressing. This results in significant disruption to lifestyle, frequently reported by patients presenting with these problems. Incontinence can be either in the form of faecal or urinary incontinence and in some cases, combined faecal and urinary incontinence. The maintenance of faecal or urinary continence has traditionally been considered separately. The extent to which abnormalities of one system affect the control of the other is largely unknown and has not been extensively investigated. On the other hand inability to evacuate, is frequently due to functional or structural obstructive causes, and no existing investigation is capable of fully elucidating these factors.

1.7.1 Mechanism of normal faecal continence

The mechanism of normal continence involves an integration of somatic and visceral functions with sensory information under local, spinal and central nervous system control. The sensory input involves the processing of information such as the stool consistency, gut transit time, rectal compliance, anal canal sensation, neural integrity and intact sphincter musculature (Bielefeldt, Enck et al. 1990). The complexity of the physiology of continence is reflected in the various factors that are involved, such as: high-pressure zone formed by

functioning anal sphincters, anorectal sensation, the anal sampling reflex, and rectal compliance (Bartolo and Macdonald 2002).

The pelvic components that play key roles in this area are the pelvic muscles, the anorectum and their innervation. The muscular wall of the anal canal is bounded superiorly by the puborectalis, where it fuses with the internal and external anal sphincter muscles. The internal anal sphincter is an involuntary smooth muscle under autonomic control and is composed of slow twitch fibres, which contracts tonically to ensure continence to liquid and flatus. The external anal sphincter is the voluntary component of the sphincter complex, which is composed predominantly of fast twitch muscle fibres. The external anal sphincter also contains some slow twitch fibres and it has been suggested that these are the fibres, whose function is optimised with biofeedback therapy and sacral nerve stimulation. There has been a reappraisal of the earlier theories of continence by Phillips et al. and Parks et al. (Phillips and Edwards 1965; Parks 1975). The current concept is that the mechanism of continence is predominantly sphincteric and dependent on the external anal sphincter and puborectalis muscle (Bartolo, Roe et al. 1986; Bannister, Gibbons et al. 1987; Ferrara, Pemberton et al. 1992; Ferrara, Pemberton et al. 1993).

1.7.2 Phases of the defecation process

1.7.2.1 Colonic peristalsis and mass movement

The propulsion of colonic contents into the rectosigmoid by peristalsis and mass movement causes the rectosigmoid to distend. This is the only form of sensation

recognisable by the rectum (Duthie and Bennett 1963). The degree of perception of rectal distension depends largely on the stool consistency, the rate of rectal filling and the volume of stool in the rectum. Stool entering the rectum causes excitation of the rectal wall stretch receptors, resulting in a burst of electromyographic activities; this is perceived by the higher centres as an urge to defecate. Initially these urges are intermittent but as the rectum fills and pressure rises. The frequency and duration of each urge increases and a point is reached where the sensation to defecate is consciously perceived.

1.7.2.2 Rectoanal inhibitory reflex (RAIR)

The initiation of rectal distension causes reflex relaxation of the internal anal sphincter (IAS) known as the recto-anal inhibitory reflex (Miller, Lewis et al. 1988). This reflex (RAIR) serves two purposes. The relaxation of the IAS exposes the mucosa of the upper anal canal to rectal contents, thus facilitating the anal sampling reflex, used for distinguishing flatus from liquid or formed stool (Duthie and Bennett 1963; Miller, Bartolo et al. 1988; Bielefeldt, Enck et al. 1990). The RAIR also helps to preserve continence by the contraction of the external anal sphincter (EAS). The anal canal is equipped with an abundance of free nerve endings and organized sensory cells such as Meissner's corpuscles, Golgi-Mazzoni bodies, Krause end bulbs and genital corpuscles which responds to touch, pressure, temperature and friction respectively (Duthie and Gairns 1960). Once the IAS is inhibited and opens to inflow of rectal contents, the various sensory apparatus are triggered. If the circumstances are not convenient, voluntary contraction of the EAS, assisted by contraction of the

pelvic floor muscles and puborectalis, can postpone defecation by retrograde propulsion of stool back into the rectum and the urge to defecate is aborted. This is called the recto-anal excitatory reflex and is thought to be a spinal reflex, which can be modulated by conscious perception and is important in preventing soiling during anorectal sampling (Whitehead, Orr et al. 1982; Sangwan, Collier et al. 1996; Zbar, Aslam et al. 1998).

1.7.2.3 Defecation

For the process of defecation to be complete, the intra-rectal pressure has to exceed the anal canal pressure. This pressure gradient is created by the concomitant relaxation of the IAS, EAS, pelvic floor and puborectalis muscles, widening of the anorectal angle and increased intra-abdominal pressure by the performance of the valsalva manoeuvre. At the completion of defecation, the closing reflex is initiated. This causes contraction of the EAS, which helps in milking residual stool in the anal canal back into the rectum and aiding the IAS to regain its normal resting tone. The anorectal angle is also restored consequently.

1.7.3 Mechanism of normal urinary continence

For normal urinary continence to occur the lower urinary tract has to be capable of accommodating urine that is continuously being produced by the kidneys, and be able to void completely through the natural urethral orifice as and when circumstances are appropriate. Similar to faecal continence, maintenance of urinary continence is dependent on normal anatomy and integrity of the pelvic

floor musculature, and on adequate support structures of the bladder neck and urethral sphincters (Figure 1.4).

The anatomy of the urinary bladder has been discussed previously. However there are special characteristics to note with regard to the physiology of micturition. The bladder is a hollow viscus composed of smooth muscle with no distinct layering, and with the capacity for extensive passive or active distension. The bladder is supported on the pelvic floor and surrounding endo-pelvic fascia, which is specially adapted to accommodate the volumetric changes of the bladder. The bladder smooth muscle forms a meshwork with the cells sliding over each other as the bladder distends.

1.7.3.1 Phases of micturition

The normal functioning of the lower urinary tract requires inputs from both autonomic and somatic neuronal systems. These two systems function in concert to ensure an effective and coordinated voiding mechanism. In normal individuals, the ureters convey about 2-5mls of urine per minute to the urinary bladder.

During the stages of normal micturition, the bladder initially fills without producing a significant increase in the bladder pressure (detrusor pressure), or in some individuals, the pressure may rise gradually as the maximal functional capacity (350-500 mls) is attained. This phenomenon is known as receptive relaxation

and is expressed as the volume during filling per unit pressure rise (mL/cm pressure rise) (Arnold 2002). Receptive relaxation depends largely on the compliance of the individual's bladder wall, and this is influenced by the morphologic makeup of the bladder wall, e.g. the preponderance of non-contractile elements of elastin, collagen and other inter-cellular substances.

As the threshold volume is reached stretch receptors in the bladder wall are triggered, sending impulses to the brainstem via the pelvic nerves and through the ascending spinal cord pathways (de Groat 1993), causing the individual to become aware of the bladder filling. Subsequently efferent supraspinal inputs via the thoracolumbar and sacral spinal cord to the sympathetic hypogastric plexus causes detrusor relaxation and increased urethral sphincteric tone. This increased urethral activity is initiated by the autonomic nervous system, and it is an involuntary phenomenon referred to as the guarding reflex. With progression in filling, the guarding reflex becomes voluntarily augmented through pudendal nerve-mediated contraction of the striated urethral sphincter. People with normal bladder can often distinguish three types of sensation: an initial sensation of filling, a first desire to void (FDV), and a strong desire to void (SDV). First sensation occurs at about 40% of SDV volume and first desire to void at 60% of SDV (Wyndaele 1998). If the circumstances are inappropriate, inhibitory impulses from the cerebral cortex will dampen the afferent inputs and consequently defer micturition.

In the initiation of micturition by the cerebral cortex, urethral sphincter relaxation is followed by detrusor contraction with opening of the bladder neck and relaxation of the pelvic floor (Clemens and McGuire 2002). As urine enters the proximal urethra, positive feedback impulses strengthen the detrusor contractions to ensure complete emptying of the bladder. At the end of micturition, the urethra and pelvic floor contract, the bladder detrusor relaxes and a small amount of urine is milked back into the bladder in a retrograde fashion (Mullner 1951).

1.7.4 Pathophysiology of incontinence

Due to the intricate nature of the relationship of pelvic floor organs and structures, a dysfunction in one compartment is frequently reflected in the others. Pelvic floor dysfunction may manifest as faecal or urinary incontinence, organ prolapse, obstructive defecation or a combination of these manifestations in varying degrees and severity. The various aetiological factors involved in the global concept of pelvic floor dysfunction will now be discussed and the anatomical defects associated with them will be highlighted.

1.7.5 Faecal incontinence

Just as the aetiology of faecal incontinence has not been fully elucidated, its definition still lacks universal consensus. Henry et al. defined major faecal incontinence as the frequent and inadvertent voiding per anum of formed stool,

while Oliveira et al. defined it as the loss of anal sphincter control resulting in unwanted release of gas, liquid or solid stool (Henry, Swash et al. 1992; Oliveira and Wexner 1998).

There also exist significant disparity in the reporting of symptoms from various reports; this is due to the different faecal incontinence scoring systems used by various reports. While the linear unweighted scoring systems of Wexner and Vaizey (Jorge and Wexner 1993; Vaizey, Carapeti et al. 1999) are easy to use, they are weak in the sense that they may over-score symptoms in patients with frequent but mild symptoms; and conversely may be under-scored in those with severe and embarrassing symptoms but with rarer episodes of incontinence events. The validated disease specific quality of life questionnaire developed by Rockwood et al. (Rockwood, Church et al. 1999), and the non-linear weighted scoring system of Mellgren et al. (Mellgren, Jensen et al. 1999) have attempted to address some of these weaknesses. However, they also suffer from the limitations of using the same domains of lifestyle disturbance and urgency for all social classes. Thus while those with mild symptoms (e.g. a business executive who has to spend long hours attending meetings or a taxi driver who frequently gets stuck in traffic), may over-report symptoms and over-score impact on lifestyle, individuals with ready access to a toilet may under-report symptoms and under-score impact on lifestyle.

Faecal incontinence may result from a single abnormality however; its aetiology in many cases is multifactorial. These can be classified as local (concerned with

the anorectum), regional (related to the pelvic floor and the supporting structures of the anal sphincters), or general medical factors (Table 1). Some authors have also classified faecal incontinence as passive or urge incontinence because it is thought that they reflect different pathophysiological processes; passive incontinence relates to internal sphincter defects and urge incontinence to external sphincter defects (Engel, Kamm et al. 1995). However, in practice this would appear to be an oversimplification as there is no distinct line of demarcation between the two and most patients would have a combination of the two howbeit in varying severity. Because of the coexistence of aetiological factors and synchrony of symptoms, the pathology of incontinence will be discussed in relation to anatomical structures and physiological functions that are impaired resulting in incontinence.

1.7.6 Anal sphincter injury

Anal sphincter injuries are the commonest cause of faecal incontinence and in women, obstetric related sphincter injuries account for most of the cases of incontinence (Cook and Mortensen 1998). Injuries arising from vaginal delivery can be because of direct trauma to the anal sphincters or traction injury to the innervation of the anal canal and the pelvic floor. Direct trauma could be as a result of direct tear from the presenting foetal head or surgically induced (episiotomy). Traction neuropathy tend to be common in women with history of prolonged second stage of labour and the use of forceps appears to increase this risk; however whether the neuropathy is due to the forceps or primarily as a result of the difficult second stage is difficult to determine (Groutz, Fait et al.

1999). It has been reported that about 6-30% of primiparous women who had vaginal delivery had endosonographic evidence of occult sphincter defect, which persisted after six months. However it was shown that less than 1% of all deliveries present with overt external sphincter defect (Sultan, Kamm et al. 1993).

1.7.6.1 External sphincter

Direct trauma can result in tears that can traverse the transverse perineal muscle and even extend to the sphincter muscles in severe cases. The severity of symptoms will depend on the extent of involvement of the sphincter. In cases where only the superficial fibres of the sphincter are involved in the damage, it leads to partial interruption of the sphincter mechanism with a segment of non-contractile scar tissue. In such cases, the anal resting pressure is largely unaltered but other functions of the external anal sphincter such as the anal sampling pressure may be reduced and less well maintained. A more severe or complete anal sphincter disruption will result in frequent symptoms incontinence to flatus, liquid and solids. A weakening of the closing reflex in this situation may result in post defecation soiling (Eccersley and Williams 2002).

1.7.6.2 Internal anal sphincter

Internal sphincter injuries have been found to be more common than once thought. With the introduction of endoanal ultrasonography, some forms of incontinence once classified as idiopathic are now known to be due to internal anal sphincter injury and this has been shown to occur in as much as 35% of

women during vaginal delivery. Apart from ultrasonographic features of internal sphincter disruption, anorectal manometric data may also show low resting pressure, especially in patients with sphincter injury confined to the internal sphincter alone. The mechanism of this injury is attributed to the shearing force exerted on the sphincters during vaginal delivery.

1.7.7 Neuropathic incontinence

Pudendal neuropathy is the other aspect of the pathophysiology of incontinence associated with childbirth trauma. About 42% of women who had vaginal delivery showed evidence of pudendal nerve damage as demonstrated by delayed terminal latency time, but this was reversible within six months in about 60% of patients (Snooks, Setchell et al. 1984; Snooks, Swash et al. 1990; Sultan, Kamm et al. 1993).

The advent of pudendal nerve terminal motor latency testing has reduced the number of patients that were hitherto classified as idiopathic faecal incontinence. Previous studies have demonstrated denervation injury to the anorectum and pelvic floor in about 75% of patients with idiopathic faecal incontinence (Snooks, Setchell et al. 1984). Pudendal neuropathy can also occur secondary to obstructive defecation leading to repetitive straining, which may in turn have been caused by structural damage in the pelvis during vaginal delivery. It is however important to note that pudendal neuropathy alone does not necessarily result in incontinence; there are a number of reports of advanced pudendal neuropathy identified in patients with normal incontinence (Wakeman and Allen-

Mersh 1989). In addition to injuries inflicted on the somatic innervation of the pelvic floor, the autonomic innervation also suffers varying degree of neuropraxia even in uncomplicated vaginal delivery.

The resulting autonomic neuropathy affects the internal anal sphincter mainly, leading to low resting pressure, hypotonia and hyporeflexia of the internal anal sphincter. In conditions where these neuropathic processes are prevailing, the degree of rectal perception of distension is reduced, the RAIR will be absent and the IAS opening, which triggers the cascade of defecation, will not occur. Furthermore, if anal sensation is not intact, appropriate EAS contraction (recto-anal excitatory reflex) and the closing reflex will be impaired.

1.7.8 Iatrogenic injury

Surgically induced trauma to the anal sphincters can predispose the patient to immediate or delayed faecal incontinence. Surgical procedures such as lateral internal sphincterotomy, fistulae surgery, surgery for rectal prolapse, haemorrhoidectomy, trans-anal resection of low rectal tumours have been documented to cause faecal incontinence. Digital anal stretch has also been known to cause widespread sphincter defects, due to the uncontrolled force applied in the procedure (Nielsen, Rasmussen et al. 1993). Trans-anal endoscopic microsurgery (TEMS) can cause a transient decrease in anorectal function which normally improves after 6 months (Banerjee, Jehle et al. 1996; Hemingway, Flett et al. 1996; Kreis, Jehle et al. 1996).

1.7.9 Anorectal function and the aging process

Anal canal pressures are lower in the elderly and the reduction in pressure is greater in women than men and greater for squeeze rather than resting pressure (Bannister, Abouzekry et al. 1987; Barrett, Brocklehurst et al. 1989; Laurberg and Swash 1989). This is thought to be due to the age related reduction in muscle bulk and to neurological damage to the sphincters. Oestrogen withdrawal associated with the onset of menopause women has also been identified as a significant predisposing factor to the development of faecal incontinence in women (Haadem, Ling et al. 1991).

1.7.10 General medical conditions

Incontinence in diabetic patients is usually thought to be of mixed autonomic and somatic neuropathy of the pelvic floor and sphincters. Patients with multiple sclerosis suffer from faecal incontinence due to pelvic floor denervation (Jameson, Rogers et al. 1994), and faecal impaction with spurious diarrhoea underlies the aetiology of incontinence in some elderly, immobile, and institutionalised patients.

1.8 Types of urinary incontinence

Urinary incontinence can be classified as passive, stress or urge urinary incontinence.

1.8.1 Passive urinary incontinence

This occurs in insensate patients, who have lost voluntary sphincter control. This commonly results from spinal cord lesions such as cord transection, chorda equina syndrome and spinal stenosis.

1.8.2 Stress urinary incontinence

Is caused mainly by factors, which results either in incompetence of the sphincter muscles or weakness of the pelvic floor support, particularly the pubourethralis (Gosling, Dixon et al. 1981; DeLancey 1988). The pelvic floor weakness allows the bladder neck and the intrabdominal segment to descend below the hammock supporting it, resulting in hypermobility of the bladder neck and failure of effective urethral closure during periods of increased abdominal pressure. Obstetric stress and injuries are thought to be major contributory factors, but the role that other factors such as prior pelvic surgery, overweight and menopause have to play, is unclear. A variant of genuine stress urinary incontinence can occur in some patients with normal pelvic floor support, but with a defect in the urethral sphincters. This type is known as type III genuine stress incontinence (McGuire, Lytton et al. 1976). It is characterised by severe stress incontinence in the context of a well-supported bladder neck. Most of the affected patients frequently have the antecedent history of previous pelvic floor surgery (often multiple operations) for incontinence, resulting in a scarred proximal urethra.

1.8.3 Urge urinary incontinence

Exist in a situation where a patient loses the ability to defer micturition for a meaningful period of time, which usually results in partial or complete loss of bladder content. This is very embarrassing to the patient and has significant impact on the patient's quality of life. It is usually caused by detrusor instability, which has both a primary and a secondary component. Primary detrusor instability is caused by an intrinsic abnormality in bladder smooth muscle or in the autonomic nerve supply to the detrusor muscle, which results in spontaneous and spasmodic contraction of the bladder muscle (Whorwell, Lupton et al. 1986). Secondary detrusor instability may result from irritation of the bladder by intraluminal, intramural or extraluminal lesions such as polyps, tumours, interstitial cystitis, or recurrent urinary tract infections. In the male, instability may also be provoked by bladder outflow obstruction secondary to urethral stricture or obstructive prostatic disease. A link between detrusor instability and irritable bowel syndrome has been described, showing that about 37.5% of patients with irritable bowel syndrome had urodynamic findings of detrusor instability in comparison to 6.2% of control patients. This would suggest that some patients with detrusor instability have a functional abnormality of their lower gastrointestinal tract related to a general smooth muscle or autonomic dysfunction (Whorwell, Lupton et al. 1986; Monga, Marrero et al. 1997).

1.8.4 Mixed urinary incontinence

This occurs in the presence of combination of features of urge and stress incontinence. This may occur when detrusor instability coexists with other

anatomical defects such as pelvic floor weakness, urethral hypermobility or urethral sphincter defects. The patients apart from having the paroxysmal incontinence of the urge type also tend to leak urine whenever they perform any activity or manoeuvre that causes raised intra-abdominal pressure.

1.8.5 Combined faecal and urinary incontinence

A significant proportion of patients presenting with complaint of either faecal or urinary incontinence are found to have combined symptoms. While a few of them present *de novo* with combined symptoms, others are discovered after obtaining a thorough history. The pathophysiology of combined faecal and urinary incontinence is poorly understood; however, it is thought to be related to global weakness of the pelvic floor musculofascial support system. It is a problem predominantly affecting the female population, and more so parous women. In the absence of local causes for incontinence, pelvic floor denervation is thought to be the common aetiological factor in patients with combined incontinence. Symptoms of stress urinary incontinence and faecal incontinence may be directly associated with each other as patients with stress urinary incontinence have demonstrated evidence of subclinical anorectal dysfunction as measured by anorectal manometry (Pannek, Haupt et al. 1996). Links have also been established between the incidence of detrusor instability and irritable bowel syndrome.

1.9 Prevention of pelvic floor disorders

For a prevention strategy of any problem to be formulated, there must be sufficient information and adequate understanding of the problem. There have been so many conjectures and speculations about the aetiology of pelvic floor disorders but most of these are inadequately researched. It will require enormous political and economic willpower of governments and practitioners for a perceptible impact to be made with regard to this problem with far-reaching health care implications. Prevention can be categorised into three stages.

The **primary prevention** aims to identify causative or predisposing factors, so they can be rectified before they cause symptoms. In the current context, natural factors such as pregnancy and vaginal delivery are inevitable so the aim will be directed at the secondary prevention.

Secondary prevention is aimed at ameliorating the effects of causative factors and preventing symptoms from progressing. Again, the problem here is that most of the patients presenting with pelvic floor disorders probably sustained the insult(s) culminating into these symptoms many years previously.

Tertiary prevention which is aimed at stopping symptoms from getting worse, reducing complications and preventing chronic disabilities, will appear to be the only way of salvaging some of the patients (Norton 2002).

From the above discussion, it becomes clear that trying to prevent a problem that is mainly amenable to tertiary prevention will be difficult, expensive and associated with high degree of failure. In a bid to prevent the problem of pelvic

floor disorders, some risk factors have been identified and they include: forceps delivery, episiotomy, prolonged second stage of labour, a large baby (over 4kg birth weight), and an occipital posterior position (Handa, Harris et al. 1996). Higher maternal age and first delivery are also risk factors (Sultan and Kamm 1997; Zetterstrom, Lopez et al. 1999). Midline episiotomy as is commonly practised in the USA has been shown to have subsequent risk of faecal incontinence 3 times that of patients with spontaneous perineal tears and 5 times that of patients with intact perineum at 3months post partum (Signorello, Harlow et al. 2000). The role of mediolateral episiotomy in the protection against incontinence has not been fully clarified however its risk is not as high as the midline episiotomy (Klein, Gauthier et al. 1994). Third degree perineal tear leads to subsequent bowel problems in about 80% of women and immediate post-delivery overlapping repair by colorectal surgeons yield better functional results than the end-to-end repair (Sultan and Kamm 1997; Sultan, Monga et al. 1999). Managing subsequent deliveries of patient with back ground history of sphincter or perineal damage is still a grey area but opinion seem to be in the favour of elective caesarean sections for subsequent deliveries. Pelvic floor exercises (PFE) yields good results in the immediate post delivery period, but reassessment at 12 months post-partum does not confer a protective advantage on those who do it, except they have continued to perform PFE (Morkved and Bo 1997; Sampsel, Miller et al. 1998). The applications of other factors such as dietary manipulation; use of pharmacological agents and the employment of neuromodulation such as biofeedback and sacral nerve stimulation-even before the onset of symptoms has been advocated.

1.10 Treatment of pelvic floor disorders

The treatment of pelvic floor disorders, apart from being wide and varied, has undergone a major development over the last three decades. In the early period of the development, the surgical approach to the management of this problem was at the forefront. Numerous repair and suspension procedures were described by the different specialties with interest in pelvic floor disorders. The Urologists described procedures such as Burch colposuspension, Marshall-Marchetti-Krantz procedure, Sling procedures (Stamey procedure and Dacron sling procedure), artificial urinary sphincters, etc. Gynaecologists described procedures such as anterior colporrhaphy (anterior repair), posterior colporrhaphy (posterior repair), and abdominal and vaginal hysterectomy. The Coloproctologists repaired rectal prolapse using perineal procedures such as Thiersch procedure, Delorme's procedure, perineal rectosigmoidectomy; and abdominal procedures such as rectopexy, resection rectopexy and anterior resection. Levatorplasty was performed either singly or in conjunction with any of the previously mentioned procedures.

None of these procedures offered the perfect solution to the problem and in many cases the repair of one problem led to the unmasking of another, causing it to become symptomatic, or even possibly create new problems by distorting anatomy or disrupting the blood supply (Norton 2002). The reason for this was the failure of specialists to appreciate the fact that normal continence depended upon multiple components of the pelvic floor and their appropriate interaction.

The isolated treatment of a disorder based on clinical examination alone frequently yielded poor results. For example the repair of a vaginal prolapse could result in the subsequent development of urinary and or faecal incontinence because the prolapse was actually buttressing a cystocele, bladder neck or indeed a rectocele; conversely a suspension procedure performed for a cystocele, without paying due attention to adjacent structures may unmask the symptoms of a rectocele or faecal incontinence. Wiskind et al found that colposuspension led to or unmasked symptoms of prolapse in a proportion of patients (Wiskind, Creighton et al. 1992).

The aim of urinary continence surgery is to elevate the bladder neck and restore it to its normal intra-abdominal location. This will help in restoring a positive pressure gradient in situations of increased intra-abdominal pressure. If however the resting urethral pressure is so low that it is less than the resting intravesical pressure, no amount of bladder neck elevation will restore the pressure gradient (Chamberlain 1995). It is also known that some patients presenting with significant symptoms of obstructive defecation were found to have large cystocele compressing and indenting the anterior wall of the rectum, thus giving a misleading picture of a rectocele on defecating proctogram. This is where further preoperative imaging of the pelvic floor has a role.

Some studies have examined the success rates of pelvic floor surgery. A retrospective study of a population of patients who had hysterectomy showed that about a third of them developed symptoms of alteration in bowel habit and

late onset urinary incontinence (Taylor, Smith et al. 1989; Bump and Norton 1998). While in the short term most of these procedure post results of 60-80% success rates, in the long term, less than 2% of these patients remain fully continent. The development of new investigative modalities such as anorectal manometry, urodynamics, nerve studies, and dynamic magnetic resonance imaging has helped in improving our conceptualisation of the morphological, physiological and neurological relationships of pelvic floor structures. This newly acquired knowledge of pelvic floor dysfunction is now helping to shape and direct the way forward in terms of new treatment modalities.

1.10.1 Biofeedback

The developments in the diagnosis and management of pelvic floor dysfunction (PFD) in the form of faecal and or urinary incontinence and obstructive defecation has led to the recognition of non-surgical management of PFD as an effective alternative to the various forms of surgical management. Though external anal sphincter damage can be repaired surgically with reasonably good short-term results, some of the patients' symptoms persist despite apparently good anatomical results from surgery (Engel, Kamm et al. 1994; Norton and Kamm 1999). For this group of patients and for those with mild to moderate symptoms without an underlying, surgically correctable abnormality, conservative management such as biofeedback has emerged as the first line treatment of choice (Enck 1993; Whitehead and Drossman 1996; Pager, Solomon et al. 2002).

Biofeedback is a technique intended to teach patients self-regulation of certain physiologic processes not normally considered to be under voluntary control. It is a behavioural technique using external equipment to demonstrate and alter physiological events using auditory or visual feedback (Fynes, O'Herlihy et al. 2002). The technique involves the feedback of information not normally available to the patient, followed by a concerted effort on the part of the patient to use this feedback to alter the physiological process in some specific way. As a therapy for incontinence, it helps the patient to learn to control and coordinate the contraction of voluntary sphincter muscles (Norton and Kamm 1999; Laycock and Jerwood 2001; Pager, Solomon et al. 2002). In obstructive defecation, the treatment is aimed at retraining the muscles of the pelvic floor to relax appropriately during defecation (Wiesel, Dorta et al. 2001). For patients with slow-transit constipation, biofeedback has been used for treatment but the mechanism of action has not been fully elucidated (Chiotakakou-Faliakou, Kamm et al. 1998; Brown, Donati et al. 2001; Emmanuel and Kamm 2001). It has been shown that biofeedback improves urinary and faecal incontinence symptoms subjectively, but so far no objective changes have been demonstrable in anal canal pressure as detected by manometry (Ryhammer, Bek et al. 1995; Fynes, Marshall et al. 1999).

1.10.1.1 History of biofeedback

Biofeedback has been in use for many years now and has been reported as an effective treatment modality for faecal incontinence. The goal of biofeedback training for faecal incontinence is to improve the patient's ability to voluntarily

contract the external anal sphincter and puborectalis muscles in response to rectal filling (Heymen, Jones et al. 2001). This is accomplished by (1) improving the strength of contraction of the pelvic floor muscles (2) improving the patient's rectal sensation and consequently the ability to perceive distension of the rectum (3) improving the endurance of the muscles and the patient's ability to defer defecation and (4) by a combination of these factors. Various studies have been carried out looking at the efficacy of biofeedback treatments. Rao et al reviewed 14 studies carried out between 1988 and 1997, and reported an improvement rate of 40% to 100% (Rao, Enck et al. 1997). In a review of 35 adult and paediatric studies carried out between 1974 and 1999, Heymen et al reported improvement rates of between 19% and 100%, however the individual studies employed different biofeedback treatment protocols (Heymen, Jones et al. 2001). Pagar et al (Pagar, Solomon et al. 2002) looked at the long-term outcomes of pelvic floor exercises and biofeedback treatment in a group of 120 patients. He assessed outcomes with four types of incontinence scores (Pescatori[0-6], St. Marks[0-13], Self-rating[0-10] and Quality of life score[10-0]), and achieved improvement rates of 72.6%, 78.1%, 75.3%, 83.6% respectively. Norton et al reviewed the outcomes of 100 patients who underwent biofeedback treatment and the result showed that 43% were reported as cured, 24% improved and 33% reported their symptoms as unchanged (Norton and Kamm 1999). Enck in a summary of published studies concluded that there was an overall cure and improvement rate of 80% (Enck 1993).

While these results have been encouraging, there has been a major difficulty in their interpretation. This is because of the poor categorization of patients, and lack of well-controlled research methodology. There was also a lack of standardization of treatment protocols e.g. the number of treatment sessions used by therapist varied widely. Gilliland et al found that the number of sessions (five or more) was the only identifiable predictor of success for patients with faecal incontinence (Gilliland, Heymen et al. 1997).

1.10.1.2 Types of Biofeedback therapy

The treatment protocols that are widely used are the sensory, strength, and coordination, biofeedback. The **sensory** biofeedback utilizes intrarectal pressure-balloon feedback device and it trains the patient to improve rectal sensation to diminishing rectal distension. The **strength training** biofeedback uses either the anal canal pressure or intra-anal electromyographic (EMG) feedback of pelvic floor muscle (Engel, Nikoomanesh et al. 1974; Heymen, Jones et al. 2001). In this technique, a surface EMG sensor is placed in the anal canal or adjacent to the anus to provide EMG activities of the pelvic floor muscles (Rieger, Wattchow et al. 1997). A further innovation of this technique is its combination with active stimulation of the anal sphincters and it has been shown to augment the effectiveness of strength training biofeedback (Fynes, Marshall et al. 1999). The **coordination** biofeedback training is the most performed type. It utilizes an intrarectal balloon that generates pressure feedback from the balloon distension and pelvic floor muscle coordination. The theoretical advantage of the coordination biofeedback is that it combines the

attributes of the sensory and strength biofeedback protocols, resulting in improved endurance (deferral time) and contractile strength of the pelvic floor muscles. However, a meta-analysis weighted by the number of subjects, and comparison of treatment outcome (defined by the percentage of subjects with a reduction in incontinent episodes), of coordination training versus strength training did not show any significant advantage of one treatment protocol over the other (Engel, Nikoomanesh et al. 1974; Cerulli, Nikoomanesh et al. 1979; Goldenberg, Hodges et al. 1980; Wald 1981; Wald 1981; Whitehead, Parker et al. 1981; Wald and Tunuguntla 1984; Whitehead, Burgio et al. 1985; Buser and Miner 1986; Berti Riboli, Frascio et al. 1988; Enck, Kranzle et al. 1988; Loening-Baucke, Desch et al. 1988; Chiarioni, Scattolini et al. 1993; Arhan, Faverdin et al. 1994; Faure, Ferriere et al. 1995; Glia, Gylin et al. 1998). The strength training using EMG biofeedback was reported to be superior to pressure feedback strength training (74% Vs. 64% respectively) (Olness, McParland et al. 1980; MacLeod 1983; McIntosh, Frahm et al. 1993; Keck, Staniunas et al. 1994; Sangwan, Coller et al. 1995; van Tets, Kuijpers et al. 1996; Ho and Tan 1997; Iwai, Iwata et al. 1997; Ko, Tong et al. 1997; Patankar, Ferrara et al. 1997; Rieger, Wattchow et al. 1997).

The improvements demonstrated in faecal incontinence using biofeedback techniques have only been seen with subjective assessment parameters. There has also been great controversy over outcome measurements because of the variety of outcome measurements that have been used in the treatment of faecal incontinence (Rao, Welcher et al. 1996; Heymen, Wexner et al. 1999). The most

commonly used measurement is by subjective report in a diary format and others have proposed incontinence severity scores, which vary significantly in their constituent measurable parameters (Keighley and Fielding 1983; Miller, Bartolo et al. 1988; Pescatori, Anastasio et al. 1992; Rockwood, Church et al. 1999; Reilly, Talley et al. 2000).

While surgical repair remains the primary mode of managing parous women with extensive anal sphincter and perineal injuries, augmented biofeedback may be used as an adjunct treatment to optimise successful surgical outcome. In less severe injuries, augmented biofeedback may improve faecal continence and co-ordination of pelvic floor muscle activity, to make surgical intervention unnecessary.

1.10.2 Sacral nerve stimulation

The concept of electro-stimulation for the control of urinary dysfunction was first introduced in the 1950s (Ganio, Luc et al. 2001). Initial attempts were focused on the direct stimulation of the spinal cord (Held, Agrawal et al. 1966; Bradley, Timm et al. 1971), the detrusor muscle and the striated sphincter (Ingersoll, Jones et al. 1957; Held, Agrawal et al. 1966; Holmquist 1968; Bradley, Timm et al. 1971; Jonas, Jones et al. 1975). Most of the initial tests were performed on dogs and the results were not very satisfactory in terms of initiating bladder voiding; subsequently attempts to directly stimulate the sacral nerve roots for the treatment of bladder voiding dysfunction showed some promising results. This principle was used for the treatment of patients with voiding difficulties by

Tanagho and Schmidt of the University of California, San Francisco (Tanagho and Schmidt 1982).

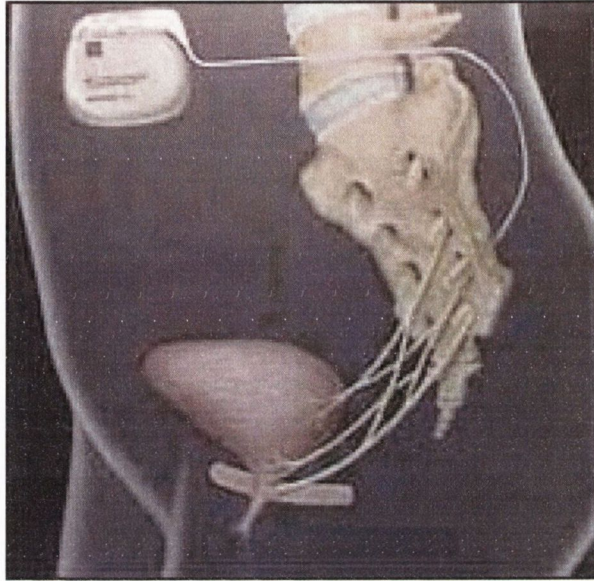


Figure 1.6: Schematic representation of an implanted sacral nerve stimulator and lead

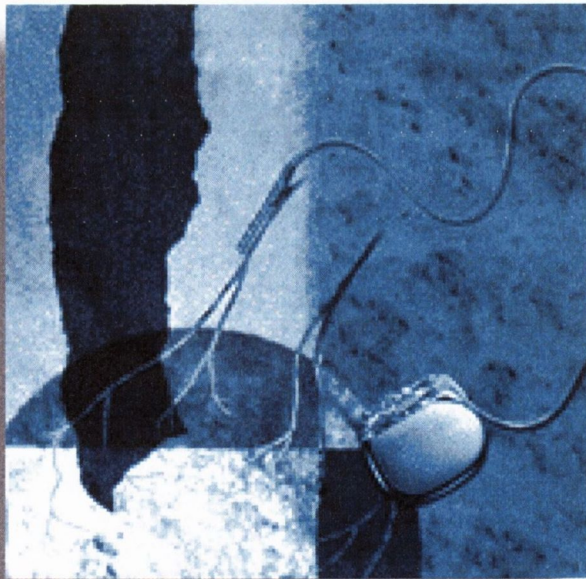


Figure 1.7: Fluoroscopic imaging of an implanted sacral nerve stimulator and lead

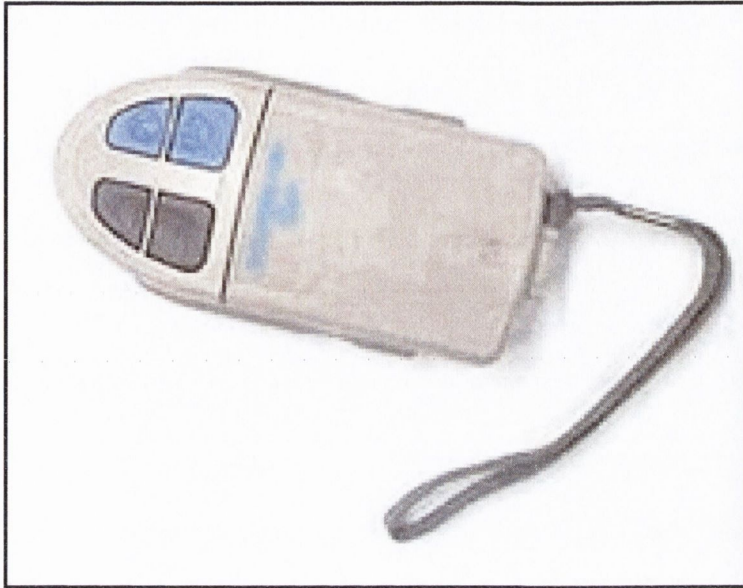


Figure 1.8: Portable remote control for the stimulator

The positive effects of sacral nerve neuromodulation in incontinence work in a multi-modal fashion, via the direct effect of stimulation of the efferent pathway on the pelvic floor, and the striated sphincter muscle. It also acts through the activation of autonomous neural pathway, inhibition of the detrusor muscle, activation of internal anal sphincter, and modulation of sacral reflexes that regulate rectal sensitivity and motility (Ganio, Luc et al. 2001; Ruud Bosch and Groen 2001). Stimulation of the efferent motor nerves to the anal sphincters increases the pressure in the anal canal by contraction of the pelvic floor and the anal sphincter muscles. It has also been hypothesised that this improvement is aided by the conversion of fast twitch fibres of the anal sphincters into the slow twitch type, and by increasing the threshold of pelvic floor muscle closing capacity (Tanagho and Schmidt 1982; Matzel, Stadelmaier et al. 1995).

The body of evidence favouring this mode of treatment as the future of treatment of pelvic floor dysfunction is rapidly increasing-both in Europe and North America. In 1981 the first sacral nerve stimulation (SNS) implant was successfully carried out. The evaluation of its effectiveness for the management of pelvic floor disorders in the short and medium term is still ongoing. Outcome measures used for evaluation are patients' symptomatology, urodynamic and anorectal manometry (Matzel, Stadelmaier et al. 1995; Vaizey, Kamm et al. 1999; Malouf, Vaizey et al. 2000; Ganio, Luc et al. 2001; Leroi, Michot et al. 2001; Rosen, Urbarz et al. 2001; Kenefick, Nicholls et al. 2002; Kenefick, Vaizey et al. 2002). The primary objective of SNS is to reduce the symptoms of faecal and urinary incontinence and a reduction of symptoms by at least 50% will imply a successful outcome.

Another advantage of SNS is that with minimal access surgery, it provides the most peripheral location for bilateral control of the nerves supplying the pelvic floor. This has a direct effect on the voluntary contraction of the sphincters, and inhibition of activities mediated by the parasympathetic outflow through the pontine centre or the sacral interneuron arch. SNS can also exert its effect by activating the internal sphincter. This is mediated by sympathetic and parasympathetic fibres, causing a stabilization of sphincteric pressure; and activation of the afferents in the local reflex arcs to maintain the tone of the striated sphincter (Ganio, Luc et al. 2001).

1.10.2.1 Exclusion criteria of SNS

Analysis of complications of the treatment and the predictive factors of success or failure, has guided the definition of exclusion criteria, which include: patients with rectal and/or uterine prolapse, cauda equina lesions, sacral agenesis, inflammatory bowel disease, previous proctectomy or pelvic irradiation or any other endoluminal pathology as determined by endoscopy or barium enema. Patients with diabetes mellitus, psychiatric disorders, cardiac diseases, age over 75 years and pregnant women or women of childbearing age who still have the intention of bearing children were also excluded (Ganio, Luc et al. 2001; Leroi, Michot et al. 2001).

1.10.2.2 The techniques of Sacral nerve stimulation

[As adapted from (Ganio, Luc et al. 2001)]

Sacral nerve stimulation consists of two stages

- 1) The diagnostic stage (Percutaneous nerve evaluation-PNE)
- 2) The therapeutic stage (Permanent implant)

The diagnostic stage: The percutaneous evaluation of the sacral nerve roots (S2, S3 and S4) is divided into two phases: the *acute phase* test, used to assess the integrity of each sacral spinal nerve to striated anal sphincter and the *chronic phase* used to assess the therapeutic potential of sacral nerve stimulation in individual patients.

The **acute phase** is a minimally invasive procedure performed with the patient in the prone position. After identifying the S2, S3 and S4 sacral foramina and under local anaesthetic infiltration a 20-gauge insulated spinal needle is inserted perpendicular to the sacrum, with an angle of inclination to the skin of 60-80 degrees. After the needle is inserted into the chosen foramen, it is connected to the external neurostimulator.

The stimulation parameters used for the acute phase are:

- 1) Pulse width (PW) of 210 μ sec
- 2) Frequency of 25 Hz
- 3) Adequate amplitude-usually of the range of 1-6 volts.

The **chronic phase** (Temporary sacral nerve stimulation-TNS) is commenced once an adequate muscle response is obtained, by inserting a temporary stimulator lead through the needle positioned in the chosen sacral foramen. The needle is then removed after ensuring that the lead is in place. The lead is connected to an external stimulator to allow evaluation of the sphincter response to the test. Subjective assessment is done with incontinence score and objective assessment using anorectal manometry. Seven days of stimulation is the minimum period accepted as a test period and for patients who respond well and maintain satisfactory lead position, the test stimulation period can be extended to 15 days. To standardize post-lead insertion environment of patients and to supervise the positioning and management of the lead, patients are admitted into hospital for the duration of the TNS. The stimulation parameters for this phase are:

- 1) Unipolar monophasic impulse, rectangular, PW of 210 μ sec
- 2) Frequency of 25 Hz
- 3) Amplitude range of 2-8 volts (according to the maximum comfortable amplitude for the patient).

The therapeutic stage: This involves positioning of the patient in the prone or Jack-knife position and after confirming the position of the sacral foramen with a needle inserted percutaneously, an incision is made to expose the foramen and the definitive electrode is inserted and anchored to the periosteum. The choice of the side and sacral nerve root to be used will be determined by the best muscle response at the time of PNE.

The lead is then tunnelled underneath the skin with the aid of a tunnelling instrument, the patient is repositioned and a small subcutaneous pocket is created in the lower quadrant of the abdomen. However, because of the prolongation of operating time due to the time required in turning the patient and the common occurrences of periostitis of the iliac crest related to the connecting wire, a new approach of performing a one-stage procedure in which the neurostimulator was implanted in the ipsilateral buttock rather than the anterior abdominal wall. These patients will subsequently be followed up by subjective and objective assessments at regular intervals.

1.11 Equipments and procedures for pelvic floor assessment

1.11.1 Anorectal manometry

1.11.1.1 History

This is one of the earliest physiological tests that, was devised to assess pelvic floor disorders. In its crude form an endotracheal tube (Mallinckrodt Critical Care, Athlone, Ireland; 29F) was used. "The cuff was filled with 2.5 ml of water at body temperature. A thin-walled, high-compliance, polyethylene disposable bag with a maximum volume of 500 ml was used as a rectal balloon. It was connected to a plastic tube (Ch 18), which was led through the endotracheal tube, thereby allowing simultaneous measurement of rectal volumes and anal pressures. Isobaric rectal distension was generated by creating a difference in height between two water reservoirs" (Osterberg, Graf et al. 2000). However, this apparatus has undergone a lot of refinement and there are now various types of balloon-pressure sensors that can be used for this test. This test is performed to assess anorectal function by measuring anorectal sensation and pressures, volume compliance, and the rectoanal inhibitory reflex. The straining pressure and the vector volume compliance are optional tests, which can give additional information.

This procedure does not require any special preparations and no sedation is needed. While this test is informative, it has no absolute diagnostic value and so

can only be used as a complimentary investigation. In a previous study (Holmberg, Graf et al. 1995), some of the parameters showed good reproducibility when the test was repeated after five minutes ($p < 0.001$) however the day-to-day variation was larger ($p > 0.05$). Low resting and squeeze pressures may indicate internal and external sphincter disorders respectively; and poorly sustained squeeze pressures might suggest easy fatigability, but they do not provide any information about the morphology of the pelvic floor musculofascial system. Furthermore, normal ARM in patients with pelvic floor dysfunction does not preclude structural or neuromuscular defects of the pelvic floor, which occurs commonly in "supra-levator syndrome". In such cases, the ARM will correctly evaluate the anal sphincters and puborectalis but not the levator ani muscle. Apprehension of some patients especially those who have suffered sexual abuse in the past may also lead to erroneously high or low parameters.

1.11.1.2 Equipments

Anorectal manometry equipment consists of a computer with installed, Polygram software (Medtronic Synetics Anorectal Manometry Analysis Module Version 2.0, Synetics Medical, Stockholm, Sweden). This computer receives and transcribes information from a transducer connected to the 4-channel water perfusion catheter with a distal balloon (MUI Scientific 4 channel catheter, Code 9012P2301). The catheter has imbedded pressure sensors positioned tangentially from each other and one centimetre apart, thus facilitating concentric pressure measurements of the anal canal. The anorectal manometry catheters

were reusable and sterilized according to the hospital infectious control committee, approved protocol.

1.11.1.3 Procedure

In performing this procedure, informed consent was obtained and the patient placed in the left lateral position. The manometry catheter was introduced into the rectum to a distance of about 6cm from the anal verge. The stationary pull through technique in, which the catheter was withdrawn at 1cm intervals, was employed. At each level, a stable resting, squeeze, straining, and coughing pressures were obtained with the aid of the computer software. The rectoanal inhibitory reflex was measured by inflating the distal balloon at increments of 10mls of air and this procedure is performed to assess the integrity of the autonomic enervation and the intrinsic, myenteric plexuses of the rectal wall. Rectal sensation was evaluated using the balloon insufflation technique. Rectal volumes at, which the patient experienced the first sensation, urgency to defecate, and maximum tolerable volume were recorded.



Anorectal Manometry

Patient Name:

Patient ID #:

Referring Physician: Keane, Prof F

Date of Test: 19/11/02

Physician:

Assistant:

Dr. Eguare Emmanuel.

Bmc Govern, Specialist Nurse

Interpretation and Comments

Feeling of Genital and Rectal prolapse x 2-3 yrs. Worsening now. Faecal urge Incontinence. Triggered by Food and Beer. Occasional Passive soiling with Urinary Urgency. Bad tear on second child.

MUI tube 138

Findings: Adequate Resting Pressures.

Adequate Squeeze Presures but poorly sustained.

First Desire @ 90mls, Urgency @ 150mls, Max tolerated volume 160mls.

Radial Pressure Analysis

Units: Pressure = mmHg Duration = sec. Rate = mmHg/sec.

Resting pressures

Depth (cm)	Anter.	Left	Post.	Right	Max.	Min.	Median	Mean	HPZ
6	7				7	7	7.0	7.0	
5	4	11			11	4	7.4	7.4	
4	18	33	9		33	9	18.3	20.0	
3	37	34	36	49	49	34	36.7	39.3	
2	62	39	71	82	82	39	66.7	63.6	X
1	54	29	64	39	64	29	46.5	46.4	X

Pressure increases during squeeze

Depth (cm)	Anter.	Left	Post.	Right	Max.	Min.	Median	Mean
6	0							
5	11	34			34	11	22.5	22.5
4	0	20	32		32	20	26.0	26.0
3	0	25	21	18	25	18	21.0	21.2
2	17	34	42	20	42	17	27.4	28.4
1	57	41	55	49	57	41	52.4	50.7

	Resting	Normals	Squeezing	Normals
HPZ length (cm)	2	2.5 to 3.5		
Mean pressure over HPZ	55	40 to 70	40	
Maximum pressure over HPZ	82		57	30 to 110
Verge to maximum (cm)	2		1	

RectoAnal Inhibitory Reflex (R.A.I.R)

Present for 10 ml at 1.0 cm from anal verge without sensation

Maximum Sustained Squeeze

		Normals
Peak amplitude	42	30 to 110
Regression amplitude	27	
Duration	16.6	
Duration at 50%	3.2	
Fatigue rate	1.1	

Jorge JMN, Wexner S, "Anorectal Manometry: Techniques and Clinical Applications"; Southern Medical Journal, Vol.86, No.8; August 1993, Pages 924-931.

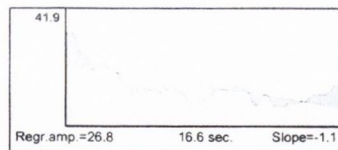


Figure 1.9: Anorectal manometry report sheet

1.11.2 The pudendal nerve terminal motor latency test

1.11.2.1 Concept

The functional integrity of the pudendal nerve can be determined by transrectal electrical stimulation of the motor nerve and measuring the time taken for a digitally delivered stimulus to elicit the first measurable contraction of the external anal sphincter. This procedure is performed using a flexible, disposable device that contains stimulating and recording (bipolar) electrodes and other ancillary materials. The pudendal nerve terminal motor latency (PNTMLT) curve is displayed on the LCD panel monitor of the keypoint portable computer. The latency is measured from the stimulation of the pudendal nerve to the start of the muscle motor potential (Laurberg and Swash 1989; Osterberg, Graf et al. 2000). The initial response to pudendal nerve studies was wide spread acceptance as a diagnostic test for pelvic floor dysfunction, however further evaluation has shown that the clinical relevance of prolonged PNTML and its routine use as a sole test for investigating patients with pelvic floor dysfunction is unclear (Osterberg, Graf et al. 2000; Stoker, Halligan et al. 2001). There were early indications of a strong correlation between perineal position and PNTML and that the degree of perineal descent could be predicted by the severity of pudendal neuropathy and vice-versa but subsequent studies did not show this correlation (Jorge, Wexner et al. 1993; Ryhammer, Laurberg et al. 1998). There were also attempts to establish a link between the so-called “neuropathic sphincter degeneration” and pudendal neuropathy but a recent study did not show any association between delay in PNTML and external sphincter atrophy as measured using the endoanal ultrasound (Voyvodic, Schloithe et al. 2000). One aspect of pudendal nerve

studies that has yielded consistent findings is the relationship of neuropathy with age. Increasing age has shown a significant correlation with higher incidence of pudendal neuropathy (Pfeifer, Salanga et al. 1997; Lefaucheur, Yiu et al. 2001).

Another study looking at various disease patterns and their PNTML did not show any significant correlation among patients with faecal incontinence, chronic constipation or chronic idiopathic rectal pain, in fact some of these patients had normal PNTML (Pfeifer, Salanga et al. 1997). The correlation between electromyography (EMG) and PNTML is also weak, and an explanation that was proffered for this is that while PNTML was mainly a reflection of conduction velocity, which is dependent on myelin function, the fibre density (FD), which is measured by EMG, is a reflection of axonal function (Osterberg, Graf et al. 2000). This is where the **strength duration test** might have a role because it combines the properties of muscle and nerve response to stimulation.

Considering the foregoing the pudendal nerve studies cannot be regarded as a satisfactory test to be used for absolute diagnosis in pelvic floor dysfunction, at best it may serve as a complimentary test in pelvic floor assessment. However, it has an important role as a prognostic indicator of success in the surgical treatment of faecal incontinence, as it has been found that both pudendal nerves must be intact to achieve normal continence after sphincter repair. Patients with unilateral pudendal neuropathy are less likely to have satisfactory postoperative sphincter function compared to those without neuropathy. The functional results of delayed anal sphincter repair after obstetric injuries was partly dependent

upon whether the nerve supply was intact (Laurberg, Swash et al. 1988; Wexner, Marchetti et al. 1991; Sangwan, Collier et al. 1996). Pudendal nerve studies did not show a correlation with the results of sacral nerve stimulation, however pudendal nerve integrity was an important factor in that patients with complete lesions of the pudendal nerve (lack of distal conduction) did not respond to sacral nerve stimulation.

1.11.2.2 Procedure

This test is performed using the St. Mark's flexible finger electrode (Ref 9013L4401, 13640 Dantec Electronic, Skovlunde, Denmark). This electrode, which has a stimulating electrode at the tip and recording electrodes situated around the base of the finger, is attached to a gloved finger and is connected to portable computer with software able to analyse and interpret the results. The procedure is performed with the patient in the left lateral position. A grounding electrode is strapped to the patient's right thigh. The course of the terminal segment of the pudendal nerve is determined by identifying the ischial spine with the tip of the finger. Rectangular pulse waves are applied to stimulate the nerve and the position of the examining finger is adjusted until a perceptible sphincteric contraction is obtained and a motor unit action potential is displayed on the computer monitor.

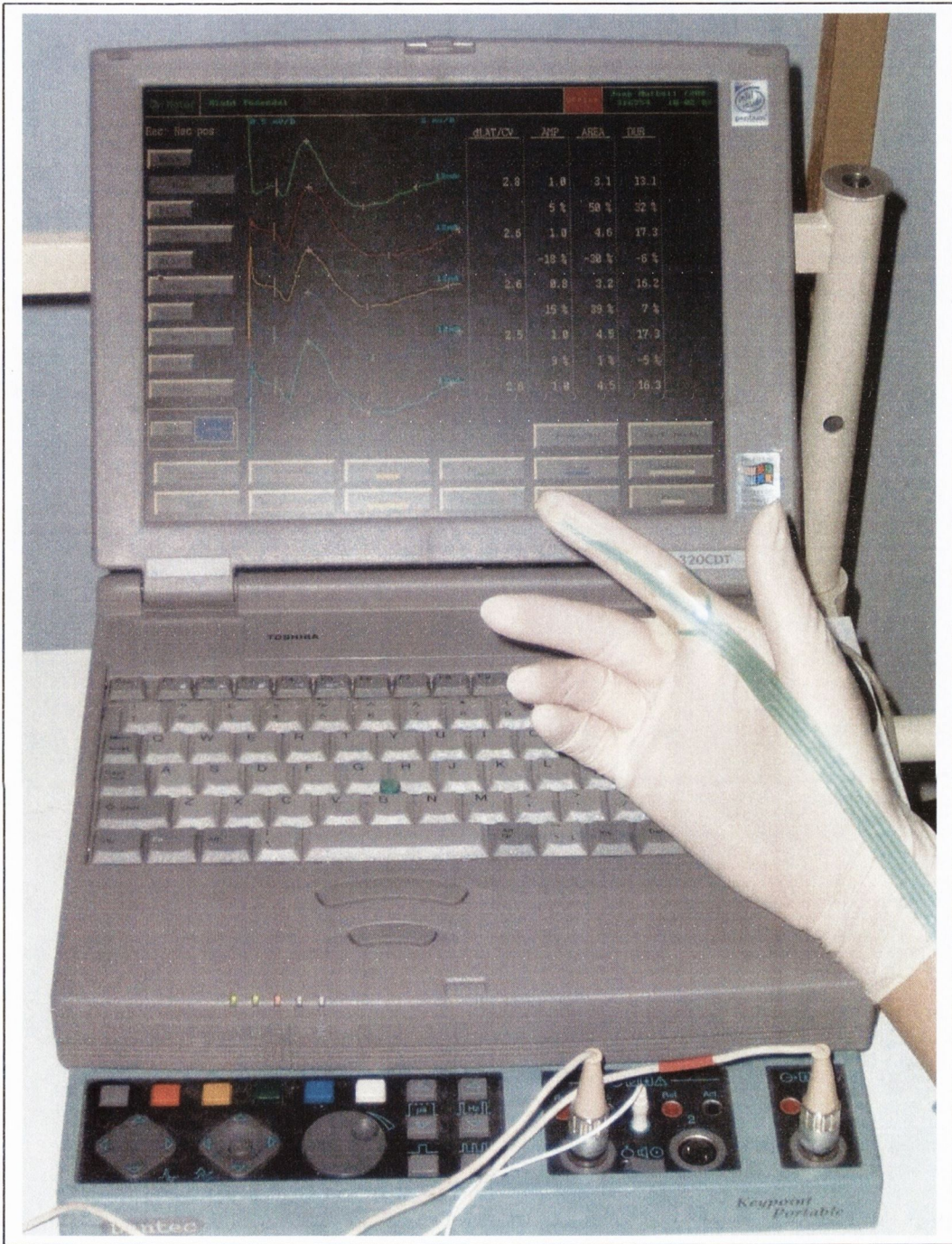


Figure 1.10: St. Marks Pudendal nerve electrode attached to a gloved finger and the integrated Dantec computer.

These action potential curves are demonstrated at five different positions and the stimulation may have to be repeated at the same location to ensure that the

latency time obtained is the shortest time possible for that position. The gloved finger is then rotated to identify the ischial spine on the opposite side and the procedure is repeated. The normal pudendal latency time is $2 \pm 0.2\text{msec}$.

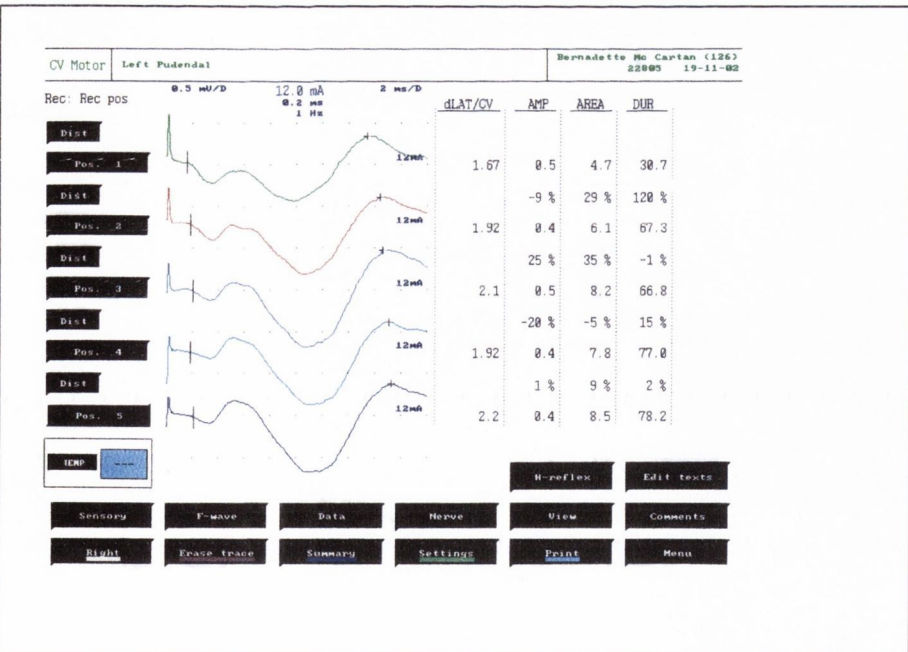
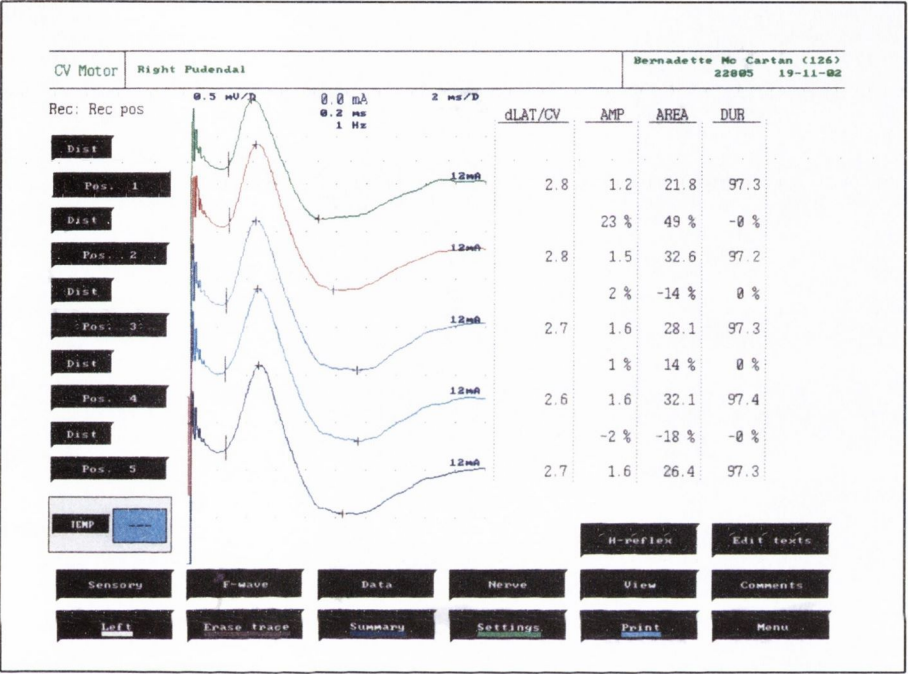


Figure 1.11: Pudendal nerve report sheets

1.11.3 Anorectal ultrasonography

Endoluminal ultrasonography (ELUS) has become a useful, minimally invasive technique for the assessment of anal sphincters and in determining the stage of anorectal tumours in terms of depth of invasion and nodal involvement. Historically the diagnosis of rectal tumour was based on clinical history, digital rectal examination (DRE) and sigmoidoscopy. DRE has a reported accuracy of 40%-80% in predicting the extent of tumour invasion of the rectal wall but these investigations, though useful, have limitations and are no longer adequate to precisely predict the extent of tumour invasion and identify peri-rectal lymph nodes (Akbari and Wong 2003).

Since endorectal ultrasonography (ERUS) was first described in 1985 (Hildebrandt and Feifel 1985), its role in the preoperative staging and planning of treatment strategy for rectal cancer has become increasingly apparent. Other helpful imaging modalities used for preoperative assessment of rectal cancer include computerized axial tomography (CT) and magnetic resonance imaging (MRI). CT has accuracy ranging from 53%-94% in the staging of primary rectal or rectosigmoid tumour depth and 56%-72% accuracy in the assessment of nodal status (Thoeni, Moss et al. 1981; Akbari and Wong 2003). However CT is not sensitive in determining the extent of local tumour invasion through the various layers of the rectal wall (Saitoh, Okui et al. 1986); and its accuracy in staging small lesions is poor (Hunerbein 2003). Meta-analysis of 78 CT studies

showed overall accuracy for depth of rectal wall invasion and nodal involvement of 73% and 66% respectively (Kwok, Bissett et al. 2000).

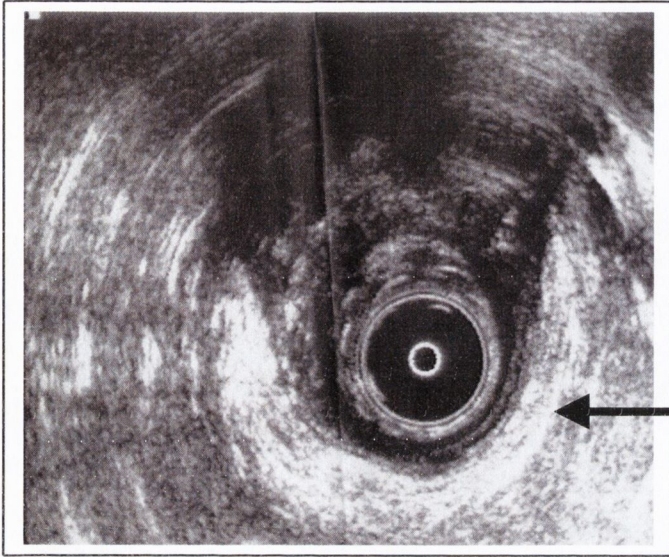
Magnetic resonance imaging and more recently MRI with endorectal coil, has the added advantage of multiplanar capability with an accuracy of about 66%, but lacks the ability to distinguish between T1 and T2 carcinomas and tend to underestimate tumour growth, (Starck, Bohe et al. 1995). Meta-analysis of MRI studies has shown overall accuracy for tumour invasion and nodal status of 82% and 74% respectively (Kwok, Bissett et al. 2000). Initial results of endorectal MRI have shown results comparable to ERUS, but its availability, cost and complexity of use is likely to impede its wide spread acceptance and use.

For tumours localized in the distal rectum, identification of patients with early stage carcinoma amenable to local endoscopic excision represents the only alternative to abdominoperineal resection (Thaler, Watzka et al. 1994). Ultrasonography seem to be the most accurate staging technique, with a reported overall accuracy of 75%-94% and 71% for tumour invasion and nodal status respectively (Hildebrandt and Feifel 1985; Dershaw, Enker et al. 1990; Sentovich, Blatchford et al. 1993; Starck, Bohe et al. 1995; Lindmark, Kraaz et al. 1997). The use of ultrasonography is limited by the presence of rectal strictures or stenosis, which prevents the advancement of the probe and thereby precludes complete assessment and staging; about 17% of rectal cancers cannot be fully staged as a result (Hawes 1993). Secondly, limitation in the visualisation of deeper structures with ERUS also affects accurate staging of

locally advanced tumours, which may be invading surrounding structures (Starck, Bohe et al. 1995).

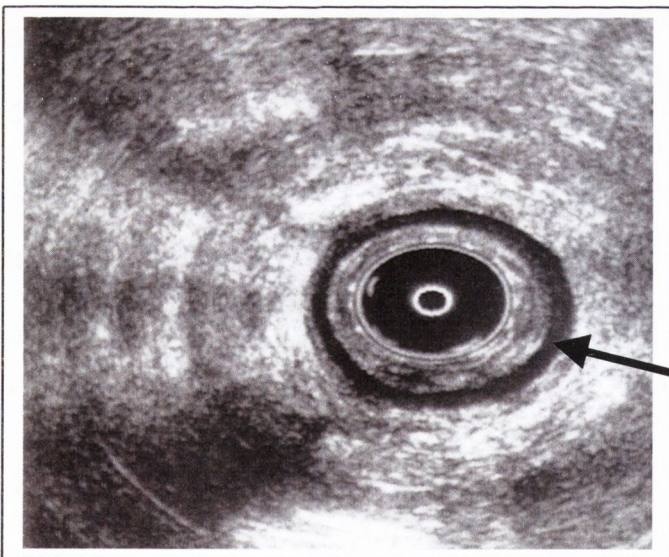
1.11.3.1 Evaluating faecal incontinence with endoanal ultrasound

Endoanal ultrasonography (EAUS) is useful for the imaging and mapping of anal sphincter musculature. It can be performed in an ambulatory setting with minimal discomfort to the patient and is therefore preferred by patients over more invasive and painful procedures such as the single fibre and concentric needle electromyography (EMG). EAUS also has the capability of depicting the anatomy of the anal sphincter clearly. The fixed structural landmarks of the anal canal makes it possible to adequately assess the sphincter morphology. The proximal border (upper anal canal) of the anal canal is bounded by the hammock-like puborectalis, which slings around the rectum and extends outward in a “U or V” shaped fashion to their insertion on the pubic rami (Figure 1.12). The section where the external anal sphincter (EAS) begins to form, and the IAS is at its maximum thickness, represents the middle anal canal (Figure 1.13). The distal anal canal is the segment where the EAS is fully formed and IAS is not visible because its distal margin terminates above this area (Figure 1.14). The puborectalis and the EAS are demonstrated as mixed echogenic structures, and the EAS forms a circular band outside and around the IAS. The internal anal sphincter (IAS) is particularly well demonstrated because of its contrasting hypoechoic features with its surroundings.



Puborectalis

Figure 1.12: EAUS of Upper anal canal



Internal anal sphincter

Figure 1.13: EAUS of Middle anal canal

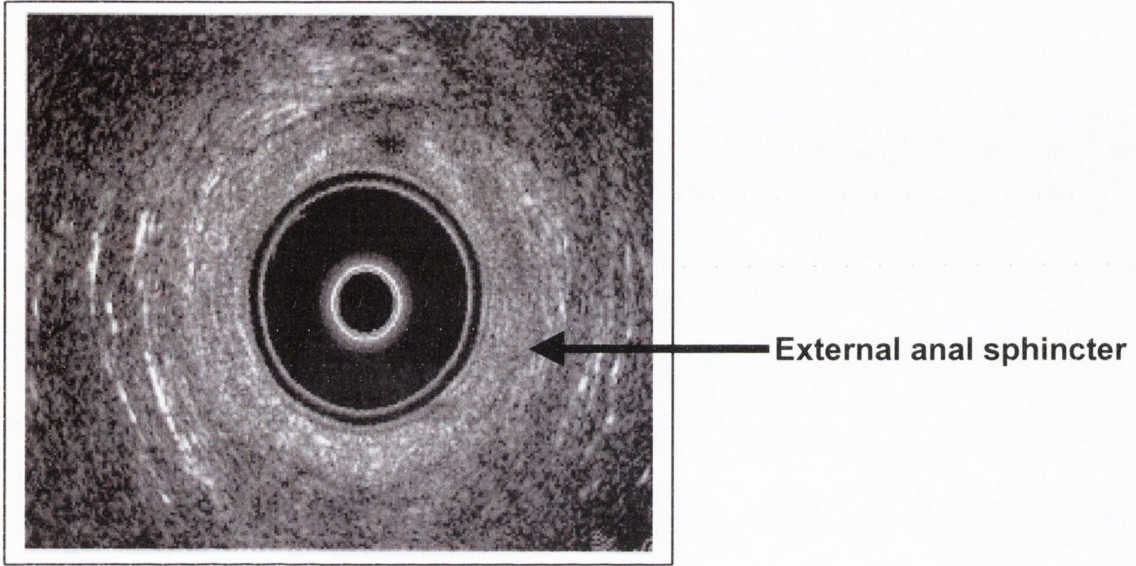


Figure 1.14: EAUS of Distal anal canal

The normal range of thickness of the IAS is 1.5 - 4mm and 2 - 4mm for males and females respectively, and variation in weight, height or body-mass index does not seem to affect it. Defects of both the EAS and IAS are identified as gaps or scars in the corresponding rings, often of contrasting echogenicity. Inserting a finger into the vagina and gently indenting the posterior vaginal wall provides a landmark for measuring the perineal body. An ultrasonographic reflection of the tip of the finger is seen on the monitor and measuring the distance between it and the anal mucosa gives the perineal body thickness.

The comparison of the findings of EAUS with other physiological parameters have confirmed it as reliable investigative tool for the evaluation of pelvic floor dysfunction. It has been shown that IAS disruption correlated with significant

reduction in the mean maximum resting pressures, and EAS disruption was inversely proportional to the mean maximum resting pressures (Felt-Bersma, Cuesta et al. 1992; Falk, Blatchford et al. 1994). It has also been shown that areas of scarring or sphincter atrophy determined by EAUS demonstrated low EMG activity (Swash and Hooks 1985); and decreased electrical activity is associated with faecal incontinence due to sphincter defects (Law, Kamm et al. 1990; Burnett, Speakman et al. 1991; Felt-Bersma, Cuesta et al. 1992; Deen, Kumar et al. 1993).

Good concordance has been demonstrated comparing sphincter evaluation with intra-operative findings. In a study of 44 patients, all sonographically detected EAS disruption and 21 of 22 IAS disruptions were confirmed at surgery; this translates to a sensitivity of 100%, and specificity of 100% and 95.5% for EAS and IAS respectively (Deen, Kumar et al. 1993). Another study by Sentovich et al, looking at 22 women with faecal incontinence due to sphincter injury yielded 100% accuracy (Sentovich, Wong et al. 1998). By imaging the sphincter musculature, previously unrecognised sphincter injuries have been identified in up to 40% of patients with idiopathic incontinence (Law, Kamm et al. 1991; Felt-Bersma, Cuesta et al. 1992; Eckardt, Jung et al. 1994). However the pitfall in endoanal ultrasonography is in the inter observer variability and the relatively high incidence of false positives (5%-25%) in nulliparous women (Sentovich, Wong et al. 1998). The various investigations used in the evaluation of pelvic floor dysfunction offer useful information about respective components of the pelvic floor and EAUS serves as a useful complimentary investigative tool.

However, for patients' convenience, cost-containment, and higher diagnostic yield, it has been advocated that combining EAUS and pudendal nerve studies be recognized as the procedures of choice for the evaluation of incontinence (Burnett, Speakman et al. 1991; Emblem, Dhaenens et al. 1994; Sentovich, Wong et al. 1998). EAUS is also useful as a surveillance tool for monitoring patients, post-sphincteroplasty. The overlapping (6-sign) or end-to-end signs can be seen on ultrasonography (Figure 1.15). Correlations between functional improvement and the identification of these signs have been described (Nielsen, Dammegaard et al. 1994; Savoye-Collet, Savoye et al. 1999).

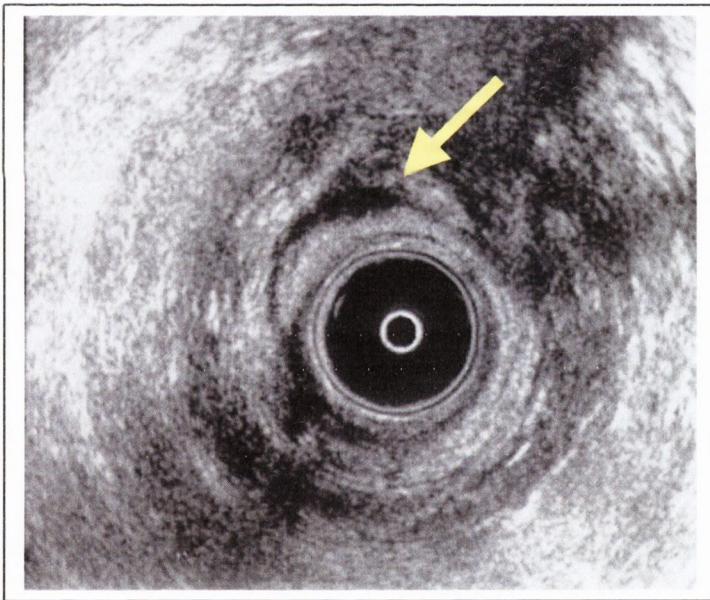


Figure 1.15: Post sphincteroplasty Endoanal ultrasound showing the overlapping 'six' sign (arrowed)

1.11.4 Urodynamic Test

The urodynamic equipment used in our unit is the "Dantec Menuet Compact plus" manufactured by Dantec Electronics LTD in Bristol. The bladder catheter

used with this system is a 4.5F double lumen cystometry catheter (Mediplus 5702). A 4.5F non-latex balloon catheter (Mediplus 5415) was used for measuring baseline rectal pressure. All the forms of catheters used were disposable.

This test is performed to assess bladder and urethral sphincter function in the filling and voiding phases of the continence cycle. It was performed after obtaining informed consent and in accordance with standard protocol (McGuire 1995). The flow rate was calculated by asking the patient to void urine into a flowmeter. A double lumen cystometry catheter (Mediplus 5702, 4.5fr) is then introduced into the bladder to measure the residual volume; the residual volume can also be obtained with the aid of vesical ultrasonography. Subsequently a catheter (non latex balloon catheter Mediplus 5415, 4.5fr) is placed in the rectum to measure the intra-abdominal pressure. After calibrating the equipments, the bladder is filled with isotonic saline at a rate of 30ml/min and the volume of bladder filling sufficient to produce a sensation of bladder filling, urgency and the maximum bladder capacity were recorded.

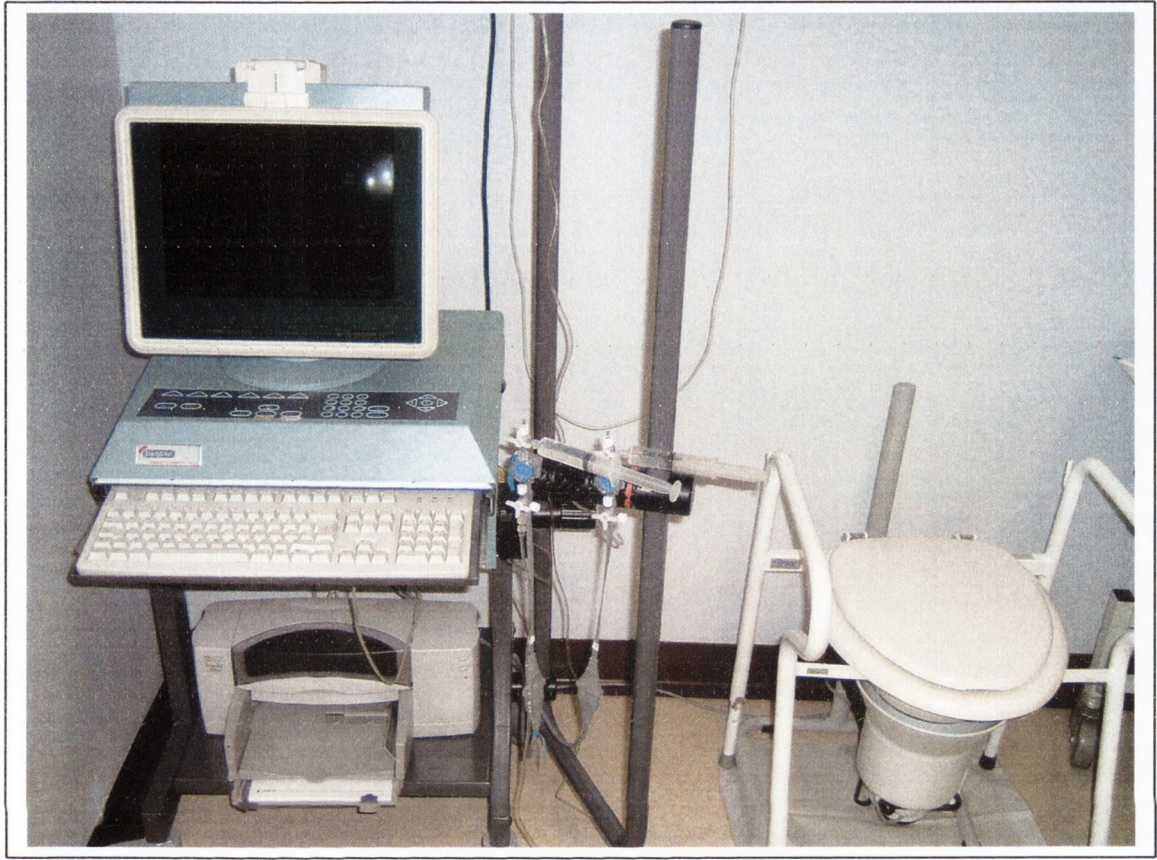


Figure 1.16: Urodynamics machine

1.11.5 Sterilization technique of anorectal catheters

In our institution, we developed a technique of automated sterilization of anorectal catheters by adapting the sterilizing machine used for flexible cystoscopes (Medivator DSD 91, Golytely Ltd). To enable effective irrigation of the anorectal catheter channels, they were connected to the main port of the sterilizing machine with the aid of four flexiflo connectors (H 157.61, Adaptac). This allowed automated sterilization of all catheters in a 20-minute cycle and with full immersion in Perasafe solution. The advantage of this innovation was that it significantly reduced the risk of staff exposure to toxic chemicals such as is obtained in the open system of washing with cidex.

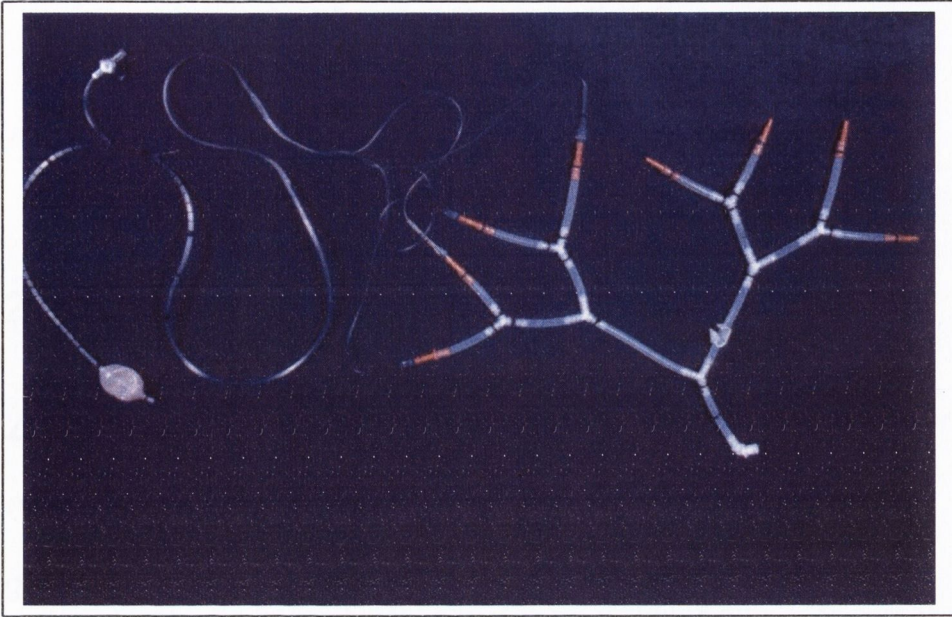


Figure 1.17: Flexiflo connectors for sterilizing catheters

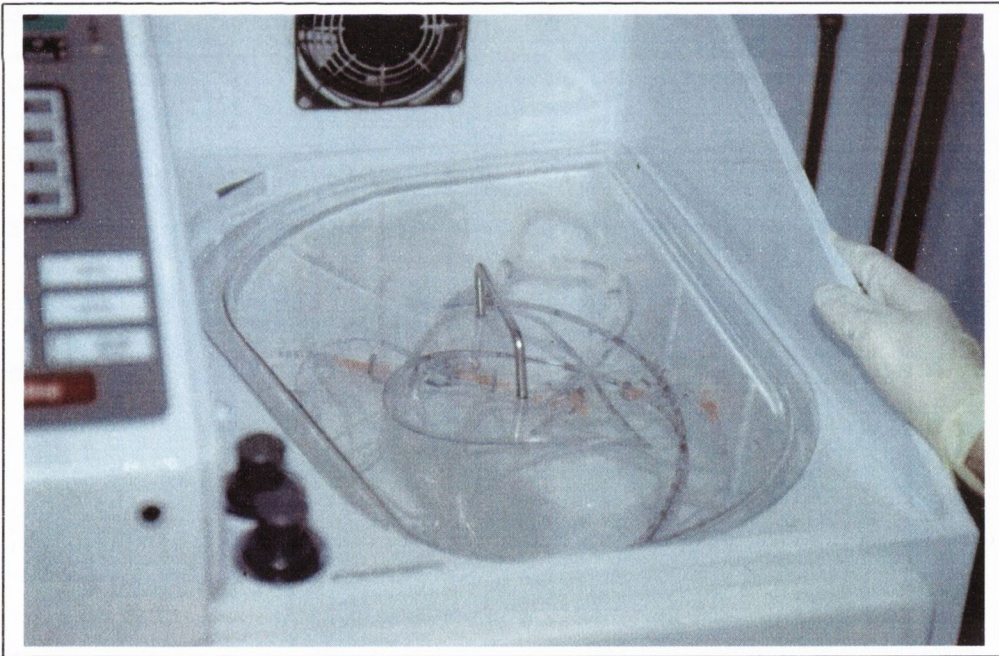


Figure 1.18: Closed sterilizing unit for catheters

CHAPTER 2

ASSESSING COMBINED INCONTINENCE WITH DYNAMIC PHASED ARRAY MAGNETIC RESONANCE IMAGING

2.1 Idiopathic combined faecal and urinary incontinence

2.1.1 Introduction

Combined faecal and urinary incontinence occurs more frequently than is generally assumed with prevalence, estimated to be about 5% in males and 10% in females (Roberts, Jacobsen et al. 1998) . In the hospital setting, the prevalence of faecal incontinence in female patients attending urology and urogynaecology clinics varies from 15-30% (Jackson, Weber et al. 1997) . Stress urinary incontinence is a symptom that results from damage to the muscles, nerves, and connective tissue of the pelvic floor leading to disruption of urethral support and vesical neck function. Similarly, symptoms of faecal incontinence arise when there is damage to the muscles and endopelvic fascial support system of the anorectal complex. A combination or overlap of these defects, results in the manifestation of combined symptoms of faecal and urinary incontinence (DeLancey 1997; Steinke, Hetzer et al. 2002).

Most patients referred for assessment in the pelvic physiology laboratory, from either a colorectal, gastrointestinal, gynaecological or urologic clinic, are referred with solitary symptoms of faecal or urinary incontinence, or with symptoms arising from organ prolapse (rectocele, enterocele, cystocele or uterine prolapse). Detailed assessment of these patients, show that a large proportion of

them have synchronous symptoms in adjacent compartments that were overlooked or unreported. The failure to identify these associated symptoms may in part relate to specialist focus but may also be attributed to the lack of access to an investigative modality that is capable of assessing the pelvic floor globally. This in turn may lead to inappropriate patients selection for therapeutic procedures and less than optimal results in the amelioration of all pelvic symptoms.

To understand the morphology and function of the pelvic floor, routine investigations include endoscopic procedures such as colonoscopy, cystoscopy, hysteroscopy, physiological tests of urodynamics, anorectal manometry, electromyography, pudendal nerve function, and imaging which includes endoanal ultrasonography and radiological contrast studies such as evacuation proctography, cystography and cystocolpoproctography. While all these investigations are useful in detecting specific abnormalities in particular compartments, they offer little or no information about the surrounding support structures and are often intrusive and interfere with normal physiology and function (Enck, Heyer et al. 1997; Gold, Halligan et al. 1999; Beets-Tan, Morren et al. 2001).

Magnetic Resonance Imaging (MRI) avoids these disadvantages and can provide a high image resolution global assessment of the pelvis, its constituent organs and the musculofascial support structures. Endoanal MRI using an EndoCoil (C.R. Bard, Inc., Murray Hill, NJ) is also used to evaluate the pelvis, but

it has similar disadvantages to endoanal ultrasound in that it props up the anal canal, causes anatomical distortion and interferes with normal physiological function.

The objective of our study was to evaluate dynamic, phased array, pelvic MRI in the assessment of patients with idiopathic combined faecal and urinary incontinence, using the widely available closed magnet system.

2.1.2 Materials and methods

All patients requiring assessment of disorders of faecal or urinary incontinence at the Adelaide and Meath Hospital, Dublin, are referred to the Pelvic Physiology Laboratory, which has the facility to assess both urodynamics and anorectal physiology. Our study population consisted of a group of patients that had a confirmed diagnosis of idiopathic combined faecal and urinary incontinence, and these were compared to a group of healthy asymptomatic age-matched controls.

To exclude known causes of faecal and urinary incontinence, after a full history and clinical examination, all the combined incontinence patients underwent colonoscopy, cystoscopy, endoanal ultrasound, anorectal manometry, urodynamic studies and pudendal nerve terminal motor latency testing. The severity of patient's faecal and urinary incontinence was evaluated using the Vaizey, Wexner and urinary incontinence scoring systems (Jorge and Wexner 1993; Vaizey, Carapeti et al. 1999). We defined idiopathic combined faecal and urinary incontinence as incontinence in which no physiological or anatomical cause could be identified using the standard tests mentioned above.

Anorectal manometry was performed on all patients using MUI scientific, eight-channel, water-perfusion, catheter (Medtronic Synaptic®). The station pull-through technique as previously described was adopted in this study (Stendal 1997). The parameters measured were mean maximal resting pressures; mean incremental squeeze pressures, rectal volume compliance, rectal sensation and rectoanal inhibitory reflex. Pudendal nerve terminal motor latency test (PNTMLT) was performed in the standard fashion to assess the integrity of the pudendal nerves (St. Marks pudendal stimulating electrode; Dantec Medical, Denmark). The normal reference value used was 2.0 ± 0.2 ms and latency periods greater than 2.5 ms were regarded as abnormal (Stendal 1997)¹⁰. An experienced colorectal surgeon (FBVK) performed the endoanal ultrasound in the standard fashion (Bruel and Kjaer Medical 2002 Panther®) to out-rule sphincter defects in the symptomatic patients. Urodynamic study was performed in the standard fashion (Lewis and Abrams 2000).

Phased array dynamic MRI was performed in all the patients and healthy volunteers after obtaining detailed informed consent (1.5-T Magnetom Symphony®, Siemens Corp., Erlangen, Germany). The phased-array scan utilizes a combination of CP-spine array coil which comprise a 12 coil design with 6 CP pairs of preamplifiers integrated in the patient table and CP body array which comprise of 4 coil design with 2 CP pairs of integrated preamplifiers which connects and operates in an integrated fashion with the CP-spine array.

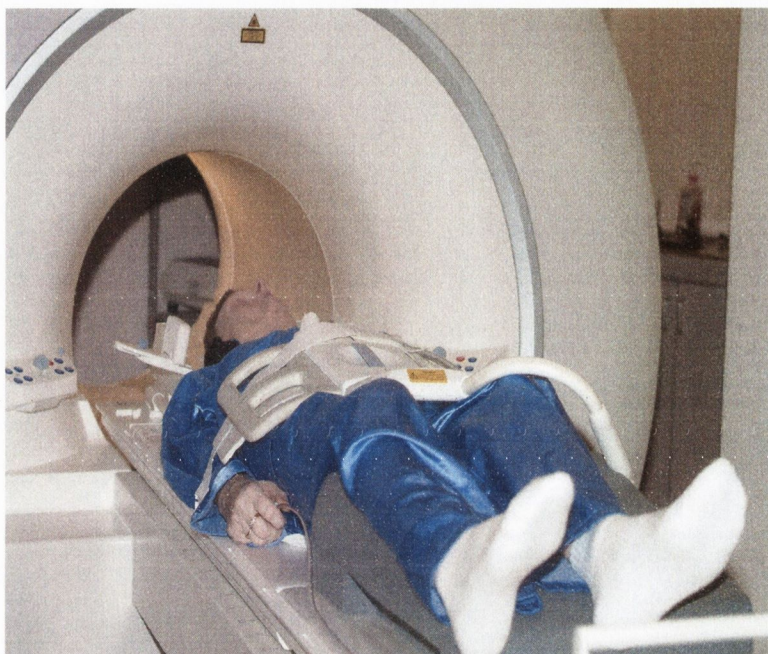
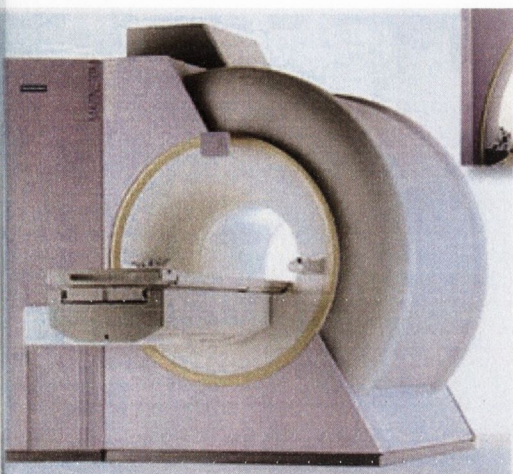


Figure 2.1: Patient lying supine on closed MRI scanner

Initial localizer images were obtained, in the coronal, sagittal and axial planes using T2-weighted turbo spin echo sequences. Because, we wanted to simulate natural physiological circumstances, no special bowel preparation or rectal contrast was administered. The examination was performed with the patient lying supine-head first on the scanner and legs kept slightly apart (Figure 2.1). The dynamics of the pelvic floor and its constituent structures were assessed with axial T1-weighted spin echo, coronal and sagittal T1 and T2-weighted turbo spin-echo sequences (with repetition time of 5130ms, echo time of 109ms and 2-3 mm slice thickness). Images were obtained in the coronal, sagittal and axial planes; and in the static and straining phases (Figures 2.2 and 2.3). Because of the problem of artefacts created by movement and the inability of patients to sustain prolonged breath holding during the straining mode, the straining phase

was performed with the fast gradient Echo sequence. With the patient in the supine position, images were obtained in the resting phase and the patient is then asked to strain, with breath holding for the acquisition of the straining phase images. The image acquisition time was approximately 25 minutes. Twenty-five individual parameters were measured using built-in software (callipers, Compass and densitometer) of the workstation. The parameters measured were validated by comparison to previously published data Beets-tan RGH et al (Beets-Tan, Morren et al. 2001), Morren GL et al (Morren, Beets-Tan et al. 2001), Kruyt RH et al (Kruijt, Delemarre et al. 1991), Comiter CV et al (Comiter, Vasavada et al. 1999), Fielding JR et al (Fielding, Dumanli et al. 2000), Yang A et al (Yang, Mostwin et al. 1991), Healy JC et al (Healy, Halligan et al. 1997), Rociu E et al (Rociu, Stoker et al. 2000). Levator ani muscle (LAM) length was measured by drawing the straightest line through the long axis of the muscle (from its pelvic ring insertion—arcus tendineus levator ani—to the commencement of the puborectalis) on both sides in the mid coronal image (Morren, Beets-Tan et al. 2001; Steinke, Hetzer et al. 2002).

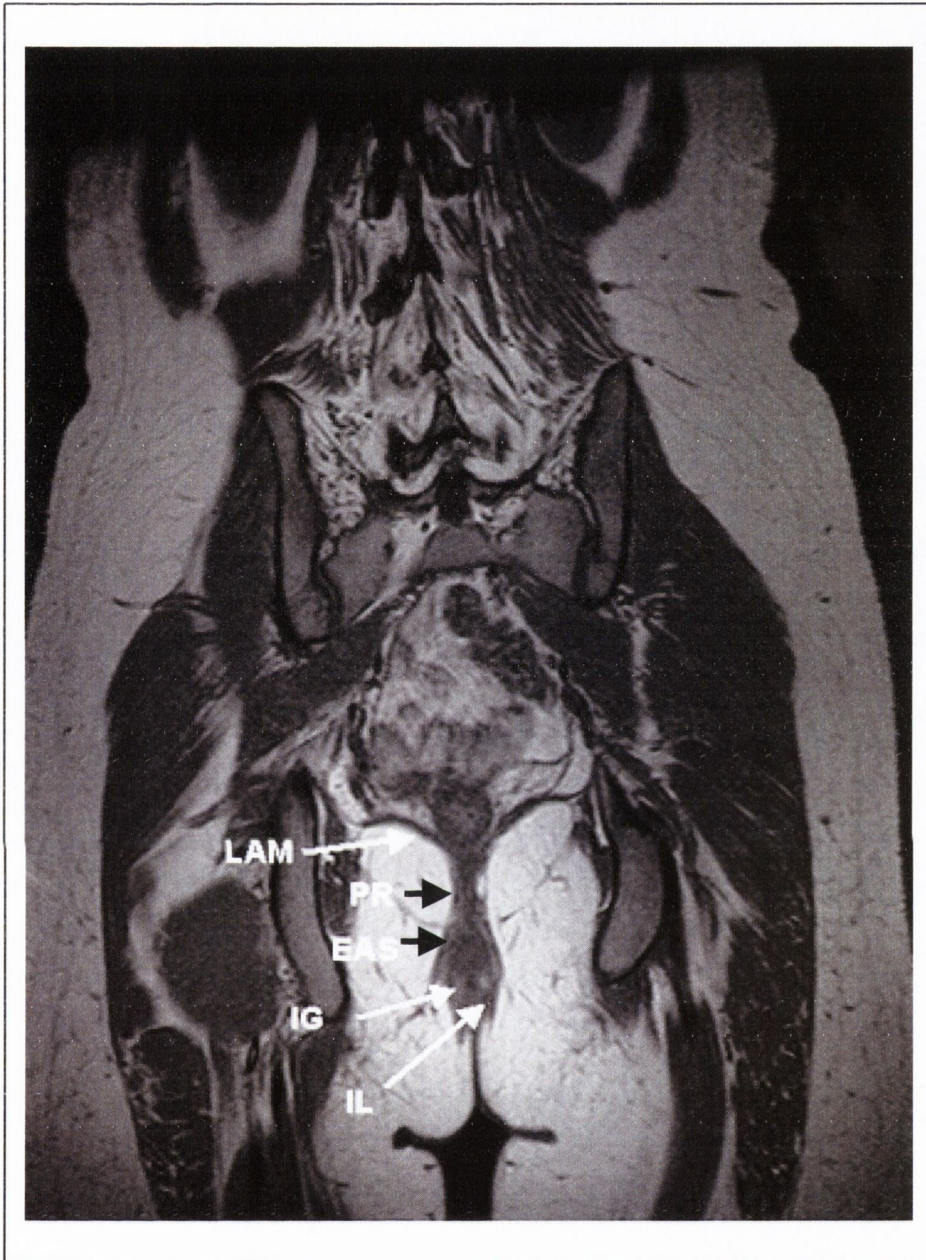


Figure 2.2: Mid coronal phased-array magnetic resonance image of the pelvis showing levator ani “levator dome” (LAM), puborectalis (PR), external anal sphincter (EAS), inner layer of external anal sphincter (IL), intersphincteric groove (IG)

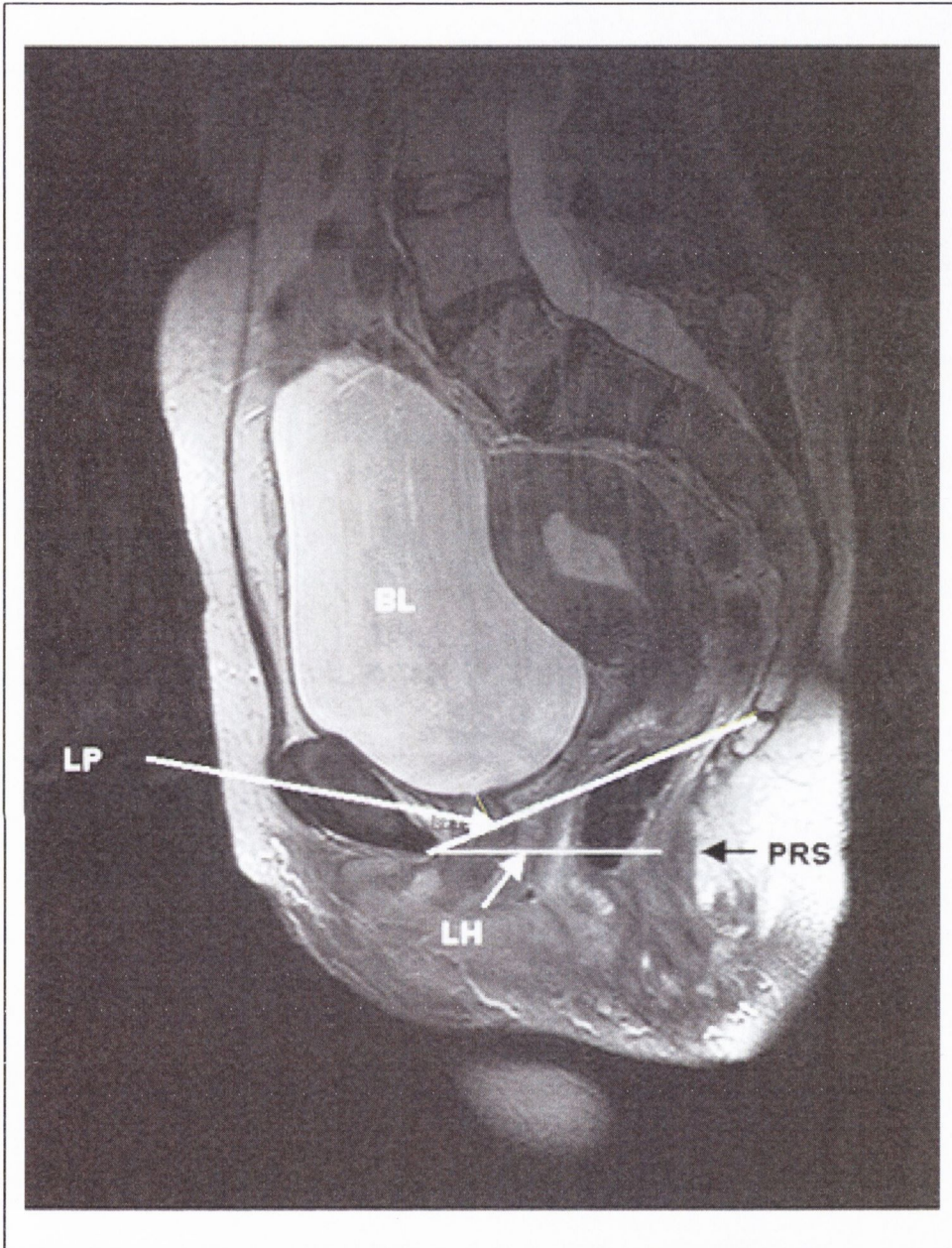


Figure 2.3: Mid sagittal phased array magnetic resonance image of the pelvis showing components of the levator and anal sphincter complex: Levator plate “pubococcygeal line” (LP), levator hiatus (LH), puborectalis sling (PR)

The LAM thickness was measured at the region of maximum thickness of the muscle in the mid coronal plane. The levator ani angle was measured (in the

coronal plane) by joining a straight line connecting both ends of the LAM with another line from the apex of the levator arch, the angle of intersection of the two lines forms the levator angle. The LAM area and tissue density were measured with the specialised image analysis software of the workstation. The mid coronal image was selected in each case and the pixel values for pure black and white colours were standardised. The LAM area on both sides was calculated by tracing the perimeter of the levator ani muscle on both sides in the mid coronal section and the mean cross-sectional LAM area was derived. The minimum, maximum and mean pixel values (LAM tissue density) were automatically calculated (Figure 2.4). Great care was taken to trace the muscle perimeter. The levator tissue density was measured using a built-in tissue densitometer in the workstation. To eliminate the differences likely to arise from equipment set-up, penetration of images and variation in body mass, each patient was used as her own control by measuring the tissue density of an equivalent area of the ipsilateral gluteus maximus. This was done on both sides (using the mid coronal segment image) and the difference of the levator-gluteal mean density was calculated.

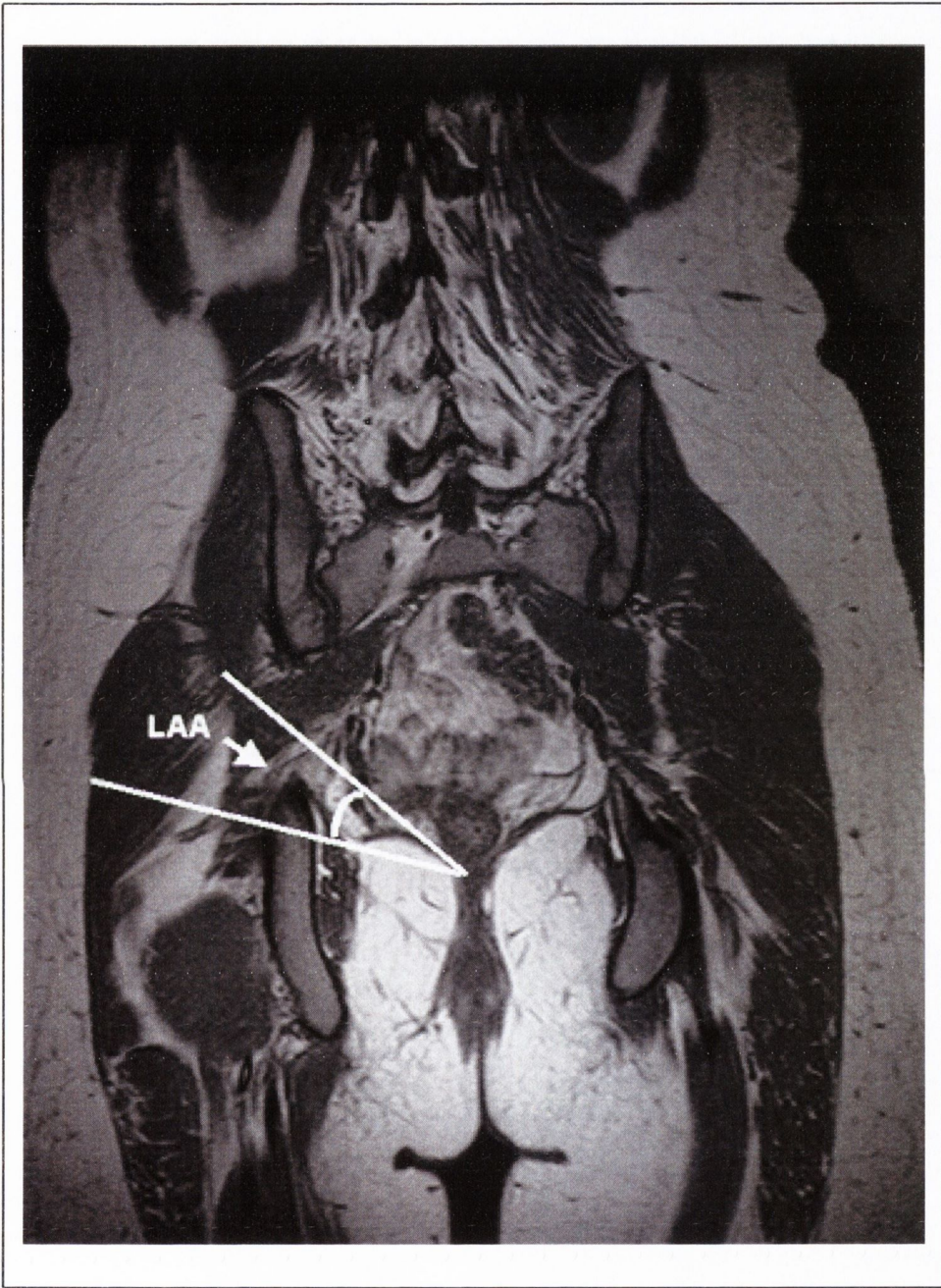


Figure 2.4: Mid coronal phased-array magnetic resonance image of the pelvis showing levator ani angle and the levator ani area

The tissue density was measured in pixels. The higher the pixel values, the lower the tissue density. The percentage fat content for each LAM complex was calculated using the formula described by Williams AB et al (Williams, Malouf et al. 2001).

$$\text{Percentage fat in LAM} = \frac{(X - X_m) \times 100}{(X_f - X_m)}$$

Where X is the mean pixel density for LAM, X_f is the mean pixel density for fat, and X_m is the mean pixel density for muscle.

The pubococcygeal line, levator hiatus and anorectal angle descent, were measured as described by Comiter CV et al and Fielding JR et al (Comiter, Vasavada et al. 1999; Fielding, Dumanli et al. 2000). The anorectal angle was measured in the standard way, as described by Matsuoka H et al. and Healy JC et al (Healy, Halligan et al. 1997; Matsuoka, Wexner et al. 2001). The anal sphincter complex was analysed as described by Beets-Tan RGH et al and Morren GL et al (DeLancey 1997; Fielding, Dumanli et al. 2000). The investigator performing the MR image analysis was blinded to both the clinical history and findings of the physiological studies of the patients. Approval for the study was obtained from the hospital's research and ethics committee.

2.1.3 Statistical analysis

Statistical analysis was performed using the software package (SPSS 10.0 for windows®, SPSS Inc. Chicago, Illinois). All results are presented as means \pm

Standard Deviation (SD) unless otherwise stated. The independent T-test for equality of means was applied. A P-value of less than 0.05 was considered to indicate a statistically significant difference.

2.1.4 Results

Of the 1501 patients referred to the pelvic physiology laboratory over a two-year period with complaints of pelvic floor dysfunction, 85 presented with combined faecal and urinary incontinence. A diagnosis of idiopathic combined faecal and urinary incontinence was made in 18 (21.2%) female patients after fulfilling the established criteria (12 urge incontinence and 6 mixed stress and urge incontinence). A control group of 14 asymptomatic females, with no history of faecal or urinary incontinence, constipation, gastrointestinal disease, or anorectal or urinary surgery were also recruited. Both groups were matched for age and parity; with mean age of 46.2 ± 14.4 years and 37.1 ± 12.9 years, $p = 0.07$, for the symptomatic and control groups respectively. The parity was 2.6 ± 1.7 and 1.7 ± 1.2 , $p = 0.8$, for the symptomatic and control groups respectively. The mean Vaizey score for the incontinence patient group was 11.6 ± 3 . The mean Wexner score for faecal incontinence was 9.7 ± 3 . Colonoscopy and cystoscopy were normal in all incontinent patients.

All symptomatic patients had a physiological evaluation of the pelvic floor. The means of the anal high-pressure zone length (HPZ), resting, squeeze pressures

and maximum tolerated volume were 2.25 ± 1 cm; 63 ± 32 mmHg, 45 ± 20 mmHg and 57 ± 69 mls respectively. The mean PNTMLT for the combined faecal and urinary incontinence group was within normal limits (1.96ms and 2.2ms on the right and left pudendal nerves respectively). In this study, a morphologically intact anal sphincter was determined in each patient with endoanal ultrasound and this was confirmed with axial MRI of the anal sphincter. The body-mass index did not show a correlation with age, parity or parameters for thickness or length of muscles of the pelvic floor on MRI imaging. The mean of the urinary incontinence quality of life score was $75.2/110 \pm 20$. Urodynamic studies showed that these patients had a mean flow rate of 8 ± 5 ml/sec (normal value = 10-18ml/sec) and a mean voiding volume of 147 ± 100 ml (normal values = 300-450ml). All the 32 participants tolerated the phased array dynamic MRI procedure satisfactorily.

2.1.4.1 Levator Plate Complex

Analysis of the levator plate complex in the mid coronal image (Figures 2.5a and 2.5b) demonstrated significant decrease in angle of the arch of the levator ani muscle in the combined incontinence group compared to the controls ($3.0^{\circ} \pm 5$ vs. $14^{\circ} \pm 10$; $P=0.004$).

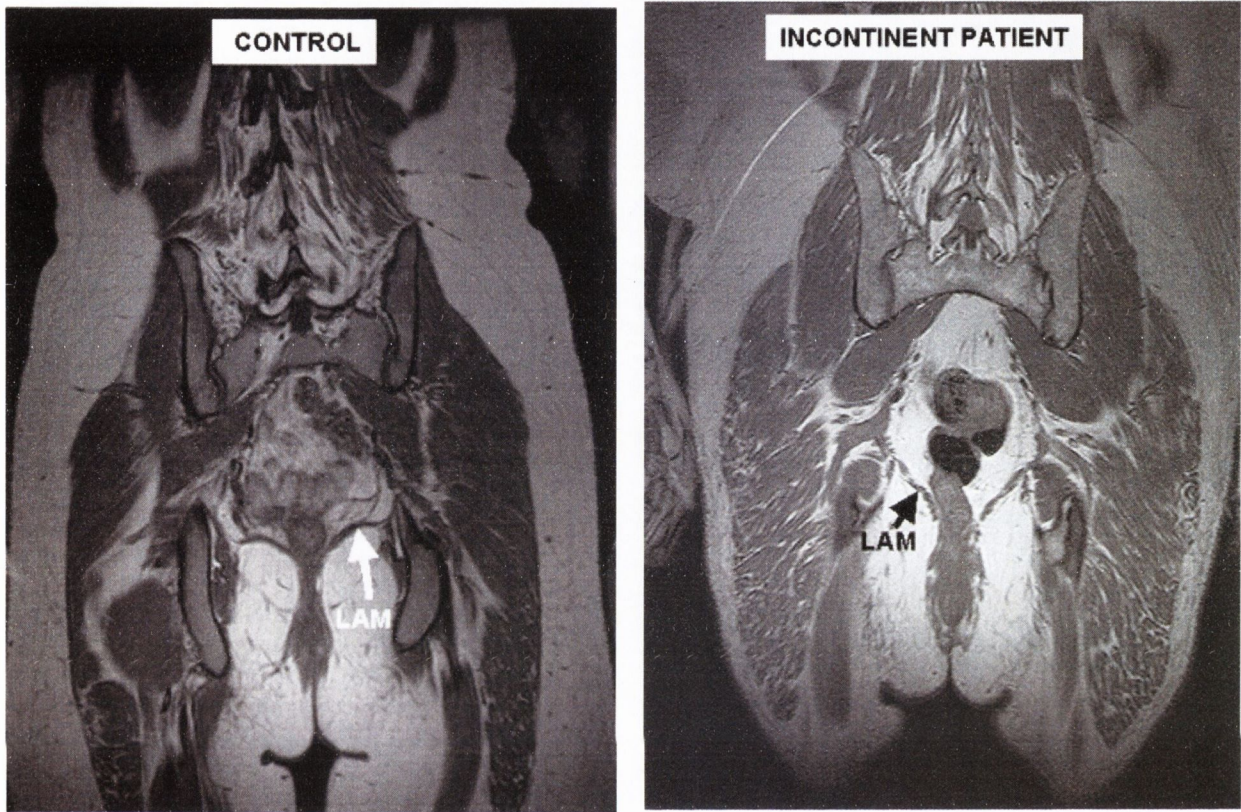


Figure 2.5 (A and B): Comparative mid coronal phased-array magnetic resonance images of a symptomatic patient and a healthy control

The levator ani angle was completely lost in 12(67%) and significantly decreased in 6(33%) of the incontinence group. The width of the levator hiatus showed a significant increase in the symptomatic group compared to controls (58.3 ± 8 mm vs. 46.5 ± 8 mm; $P=0.001$). Although the levator-ani muscle length and thickness did not differ significantly in the two groups (45 ± 6 mm vs. 44 ± 5 mm, $p=0.6$ and 4.5 ± 1 mm vs. 5.0 ± 1 mm, $P=0.2$ respectively), there was a significant reduction in the area of the levator ani muscle of the symptomatic group compared to the

controls ($19.5 \pm 1 \text{ mm}^2$ vs. $26.9 \pm 1 \text{ mm}^2$, $p=0.001$). There was also a significant reduction in the tissue density of the levator ani muscle of the symptomatic group versus the controls (157.3 ± 47 pixels vs. 126.1 ± 23 pixels, $p=0.025$) (Table 2.1).

Table 2.1: Parameters of the levator plate complex

Parameters	Study group (N=18)	Controls (N=14)	P-values
Urethrovesical Angle (degrees)	91 ± 23	73.5 ± 10	0.004
Levator Hiatus (mm)	58.3 ± 8	46.5 ± 8	0.001
Levator Angle (degrees)	3.0 ± 5	14 ± 10	0.004
Levator Density (pixels)	157.3 ± 47	126.1 ± 23	0.025
Levator Area (mm^2)	19.5 ± 1	26.9 ± 1	0.001
Puborectalis thickness (mm)	9.5 ± 3	12.5 ± 3	0.016

(Significance: $P < 0.05$)

2.1.4.2 Anal sphincter complex

There were significant differences in measurement of the external anal sphincter (EAS) length between symptomatic and controls ($20.0 \text{ mm} \pm 5$ vs. $26.6 \text{ mm} \pm 13$; $p=0.03$). The length of the inner layer of EAS (Stoker, Rociu et al. 1999) was also significantly decreased ($3.1 \text{ mm} \pm 3$ vs. $7.0 \text{ mm} \pm 2$; $p=0.001$). Similarly, there was a significant decrease in the measurement of the intersphincteric groove

(2.4mm±2 vs. 3.9mm±1; $p=0.003$), which was measured using a reported protocol (Jorge and Wexner 1993). There was however, no statistically significant difference in the thickness of the EAS muscle (3.7mm±1 vs. 7.7mm±11; $p=0.3$) (Table 2.2). There was complete obliteration of the inner layer of the external anal sphincter in 8 patients.

Table 2.2: Parameters of the anal sphincter complex

Parameters (mm)	Study group (N=18)	Controls (N=14)	P-values
External Anal Sphincter (EAS) length	20.0±5	26.6±13	0.03
Inner Layer of EAS length	3.1±3	7.±2	0.001
Intersphincteric groove	2.4±2	3.9±1	0.003
Superficial Transverse Perineal thickness	5.3±2	7.5±2	0.009
Perineal Descent	8.7±6	3.8±3	0.005
Puborectalis thickness	9.5±3	12.5±3	0.016

(Significance: $P < 0.05$)

2.1.4.3 Urethrovesical complex and perineal descent

The anterior urethrovesical angle was significantly wider in the incontinence group compared to the controls $91^{\circ} \pm 23$ vs. $73.5^{\circ} \pm 10$ (mean \pm SD) respectively, $p = 0.004$. The degree of perineal descent (Figure 2.6A and 2.6B)

was also significantly more in the symptomatic group compared to the control group $8.7\text{mm} \pm 6$ vs. $3.8\text{mm} \pm 3$ (mean \pm SD), $p = 0.005$.

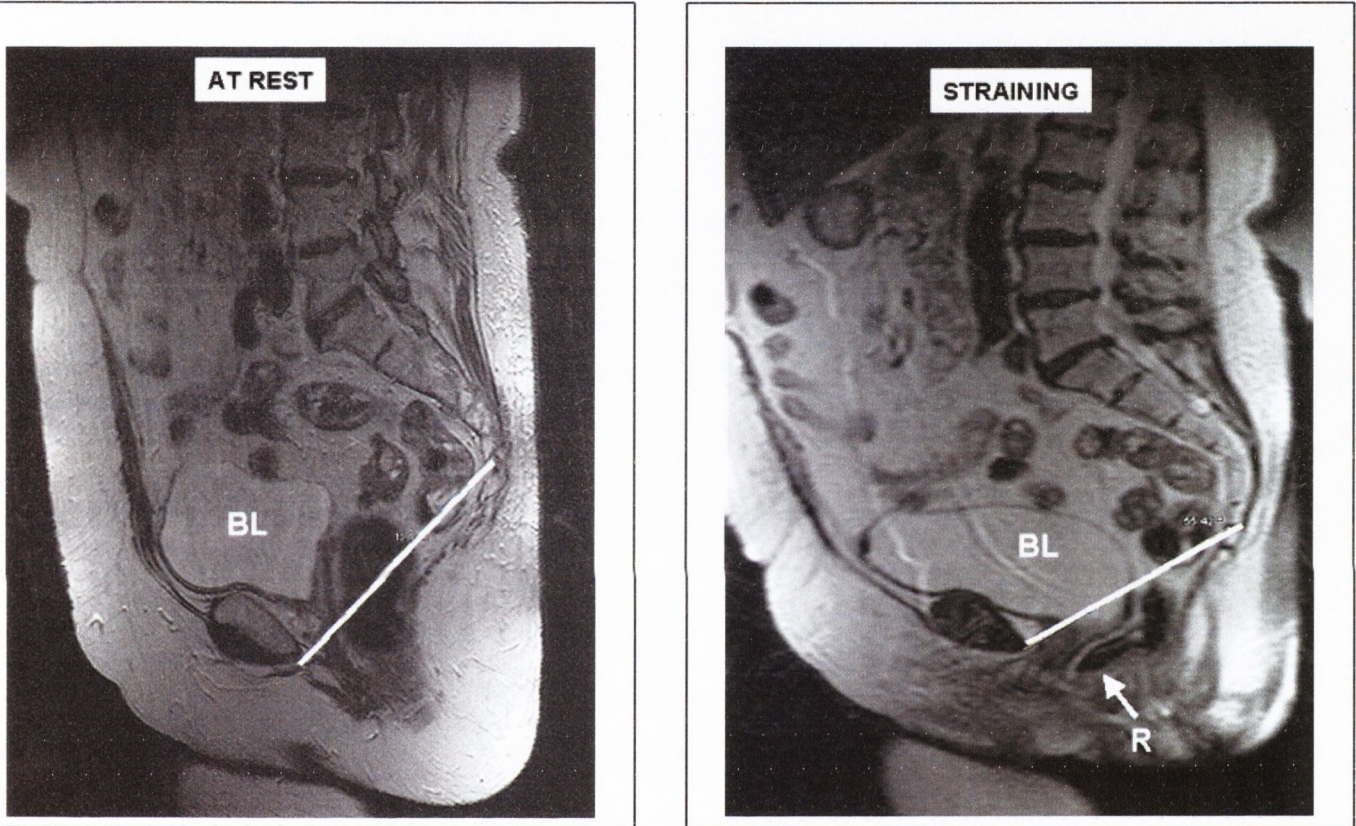


Figure 2.6 (A and B): Sagittal images of a patient illustrating perineal descent and organ prolapse

2.1.5 Discussion

Since the introduction of MRI as an imaging modality, there has been a growing interest in its use as an investigative tool in the evaluation of patients with pelvic floor dysfunction (PFD). This interest is reinforced by the fact that MRI, apart from offering the capability of global visualisation, with enhanced tissue resolution of the pelvic organs and their musculofascial support structures, is also free of ionising irradiation. The invasiveness of fluoroscopic procedures and the high doses of ionising irradiation they deliver render many of them unacceptable for many patients. For instance, with evacuation proctography, the somatic dose of ionising irradiation ranges from 100-200Gy (in men and women) and gonadal dose ranges from 40Gy in men to 90Gy in women. Defecating proctography (synonyms: defecography, evacuation proctography) has been used in the assessment of defecation disorders for nearly 50 years but its clinical value remains controversial. Although some investigators have defined a role for it in the assessment of faecal incontinence, its main indication is in the evaluation of constipation due to obstructive defecation (Kelvin, Maglinte et al. 1994; Stoker, Halligan et al. 2001). Rentsch et al commented that although defecating proctography has increased our knowledge of evacuation disorders, the causes of combined pelvic floor disorders in females and complex disorders of the posterior compartment in males remains unclear in some patients (Rentsch, Paetzel et al. 2001). Frequently, multiple opacification techniques of the different compartments and additional neurological and physiologic measurements are necessary to identify the predominant disorder (Karasick, Karasick et al. 1993;

Sentovich, Rivela et al. 1995). The result of the advent of MRI is the broadening of the spectrum of patients that can be investigated for PFD.

Employing previously validated techniques used in the measurement of pelvic floor parameters, we have demonstrated new and significant findings in the structure and quality of the levator ani muscle (LAM) and anal sphincter muscle complexes (Jorge and Wexner 1993; Stoker, Fa et al. 1998; Briel, Stoker et al. 1999; Choi, Wexner et al. 2000; Rentsch, Paetzel et al. 2001). The length and thickness of LAM were not significantly different when comparing control and incontinence subjects, but the LAM area was significantly reduced in the combined incontinence group. Thus, measurement of the muscle area gave a better representation of the muscle volume than the length or thickness, which were measured at points of maximum values. This was in agreement with the findings of Williams et al in which, the measurements of the length and width of the anal sphincter at specified levels did not correlate with the cross-sectional area due to the vast heterogeneity of sphincter morphology (Williams, Malouf et al. 2001).

The LAM tissue density was significantly diminished in the combined incontinence group compared to the control group. Neither the mean tissue density nor the percentage fat content correlated with age. This suggests that age is not a causal factor in the alteration of LAM tissue morphology. Previous studies looking at the anal sphincter have identified changes in the anal sphincter, which correlated with increasing age (Haas and Fox 1980;

Klosterhalfen, Offner et al. 1990; Speakman, Hoyle et al. 1995), although this correlation was not confirmed by Williams et al (Williams, Malouf et al. 2001).

These findings suggest that apart from anal sphincter problems, patients with idiopathic faecal and urinary incontinence also have structural defects (above the puborectalis) in the levator ani muscle; this may explain why some of these patients may have relatively normal anorectal neurophysiological and urodynamic studies and still suffer from significant symptoms of faecal and urinary incontinence. The levator ani angle, which is an index of the degree of arching of LAM, was significantly reduced in the symptomatic group. This, we feel, would alter the dynamics of the pelvic floor especially in the upright position or during phases of increased intra-abdominal pressure, when considerable stress is brought to bear predominantly on the levator plate.

The most striking finding of our study was the identification of the arch of the levator ani (“levator dome”) and this is the first report of this distinct anatomical entity as an MRI finding. Our contention is that in normal subjects with the arch of LAM intact, pressure from above, while trying to depress the arch, is deflected to closing the levator hiatus thus reinforcing continence. Conversely, in patients with idiopathic faecal incontinence the arch of LAM is either lost completely or significantly diminished, causing the muscle to assume a “funnel” or “bowl” configuration. The result of this is that when pressure is applied on the levator plate from above the levator hiatus opens and causes incontinence particularly if this is coupled with an already compromised anal sphincter. This hypothesis has

been corroborated by a study conducted by Shafik et al on the intrinsic myoelectric activity of the LAM (Shafik, Doss et al. 2003). In this study, it was determined that the LAM had resting myoelectric activity and this could be attributed to histological evidence of the presence of smooth muscle bundles interlaced with the striated muscle bundles. These smooth muscle fibres appear to generate an increased tone, reflected in an elevation of electrical activity of LAM, in response to an increase in intra-abdominal pressure and visceral weight thus reinforcing continence. Shafik further suggested that increased LAM myoelectric activity and contraction appears to protect the infralevator pelvic structures that compromise the rectal neck (anal canal), urethra, and vagina from the effect of increased intra abdominal pressure thus preventing leakage. In conditions such as chronic straining at defecation or in acute straining during labour, the brunt of the increased intra abdominal pressure falls on the LAM, which may ultimately subluxate or sags, directly exposing the infralevator structures and leading to the levator dysfunction syndrome (Shafik 1983; Busacchi, De Giorgio et al. 1999).

The parameters (such as perineal descent, organ descent, cystocele and rectocele) measured on phased array MRI performed with the patient in supine position were probably lower than those obtained in the upright position with the patient sitting in an open MRI unit. This was previously confirmed by Kelvin et al, who showed that the supine values were underestimated by about 10%-15% compared with the upright position, and we know that gravitational influence is known to exacerbate pelvic floor weakness (Kelvin, Maglinte et al. 2000).

Identifying structural alterations in the levator plate complex as part of a pre-operative evaluation in the selection of patients for incontinence surgery may be very important. While in the short term, the success rate of surgical intervention for faecal incontinence is as high as 70-80%, in the medium and long term the outcome is disappointing, with less than 2% remaining fully continent (Matzel, Stadelmaier et al. 1995; Vaizey, Kamm et al. 1999; Malouf, Vaizey et al. 2000; Rosen, Urbarz et al. 2001). Surgical procedures such as anterior levatoplasty or post-anal repairs do not restore or sustain the LAM and its intrinsic myoelectric properties. The loss of the LAM arch and its function may better respond to activation by physiotherapy, biofeedback or electrical stimulation (Busacchi, De Giorgio et al. 1999). Neuromodulation by sacral nerve stimulation (SNS) might be the way forward in the management of these patients (Matzel, Stadelmaier et al. 1995; Malouf, Vaizey et al. 2000; Ganio, Luc et al. 2001; Leroi, Michot et al. 2001; Rosen, Urbarz et al. 2001; Kenefick, Nicholls et al. 2002; Kenefick, Vaizey et al. 2002). Sacral nerve stimulation improves anal resting and squeeze pressures, rectoanal inhibitory reflex and volumetric compliance, as well as significantly increasing defecation deferral time and anal canal length. Rosen et al confirmed the positive stimulation response during acute-phase testing, by the typical contraction features of the pelvic floor and the anal sphincters; they were also able to demonstrate a significant increase in sphincter pressures and anal canal length (Rosen, Urbarz et al. 2001). Shafik et al have also shown that skeletal muscle may have some adaptive response to increased use of chronic electric stimulation and that the presence of smooth muscle fibres represents a

process of structural-functional adaptation in the LAM (Salmos and Hendriksson 1981; Pette and Vrbova 1992; Shafik, Doss et al. 2003). The initial results of SNS are very encouraging with recovery from incontinence of up to 70-80% in treated cases (Pette and Vrbova 1992). Future prospective studies are planned to rescan patients after neuromodulation therapy, to determine what structural changes are effected and how these might correlate with symptomatic improvement.

2.1.6 Conclusion

We have demonstrated for the first time distinct anatomical abnormalities in the levator plate complex in patients with idiopathic combined faecal and urinary incontinence. Our findings suggest that pelvic floor MRI may play an important role in selecting patients that will benefit from these new, minimally invasive treatment modalities. Findings on MRI may also suggest novel surgical remedies as well as provide better selection criteria for established surgical procedures in order to achieve better outcomes. Further studies to standardise reference values are therefore necessary to more, clearly define the clinical role of MRI.

CHAPTER 3

ASSESSMENT OF PATIENTS WITH OBSTRUCTIVE DEFECATION

3.1 Introduction

Constipation is said to occur when at least two of the following feature are present: (1) straining during more than 25% of bowel movements. (2) Sensation of incomplete evacuation with at least 25% of bowel movements. (3) Hard or pellet-like stools at more than 25% of bowel movements and (4) Bowel movement frequency of less than 2 per week with or without symptoms of constipation (Whitehead, Chaussade et al. 1991; Rotholtz and Wexner 2001). Chronic constipation consists of functional slow transit constipation (colonic inertia) and difficult (Obstructive) defecation. Obstructive defecation is characterised by symptoms of straining, feeling of incomplete evacuation or the need to facilitate defecation digitally either by manual evacuation, application of digital trans-vaginal pressure or perineal support manoeuvre (Rotholtz and Wexner 2001; Wiesel, Dorta et al. 2001).

Disorders of constipation has significant impact on the health care delivery with as many as four million people affected in the United States; and constipation accounting for 0.9% and 1.2% of physician visits yearly in the UK and USA respectively (Sonnenberg and Koch 1989; Sonnenberg and Koch 1989; Johanson and Sonnenberg 1990). About \$800 million is spent annually in the USA for the procurement of prescribed laxatives and the intractability of this problem significantly impairs the patients' quality of life (Anderson 1985).

3.2 Aetiology

The symptoms of obstructive defecation are reported in about 40-74% of patients referred to specialist centres with complains of constipation (Kuijpers 1990; Lemieus and Kamm 1994). Because many of these patients present with a combination of symptoms, it is important to assess these patients thoroughly to clearly differentiate the causes of the three variants of constipation, which are: slow transit constipation (STC), obstructive defecation and a combination of both types. Isolated slow-transit constipation occurs as a result of disturbance of motility manifest by prolonged transit times (Brown, Donati et al. 2001). This is caused by disorders such as:

- (1) Generalized gastrointestinal dysmotility (Spiller 1999).
- (2) Endocrine disorders such as hypothyroidism.
- (3) Autonomic neuropathy, which occurs in diabetes mellitus.
- (4) Iatrogenic causes of pelvic autonomic dysfunction, which follows surgical procedures such as hysterectomy, tubovarian surgery and childbirth (Roe, Bartolo et al. 1998; Giaroni, De Ponti et al. 1999; Scott, Knowles et al. 2001).

In contrast, obstructive defecation is caused by structural and functional disorders such as:

1. **Rectocele:** defined as a bulge of more than 2cm between the extended line of the anterior border of the anal canal and the tip of the rectal descensus (Figure 3.1) (Yoshioka, Matsui et al. 1991; Lienemann, Anthuber et al. 1996; Karlbom, Nilsson et al. 1999). It is recognised as the

commonest cause (39%-72%) of obstructive defecation, and it develops due to a defect or attenuation of the posterior rectovaginal septum in the direction of the vagina, its cause can be congenital, ageing, chronic straining in constipation, pregnancies and child birth trauma. (Khun and Hollyvok 1982; Marti and Deleaval 1999; Stojkovic, Balfour et al. 2003)

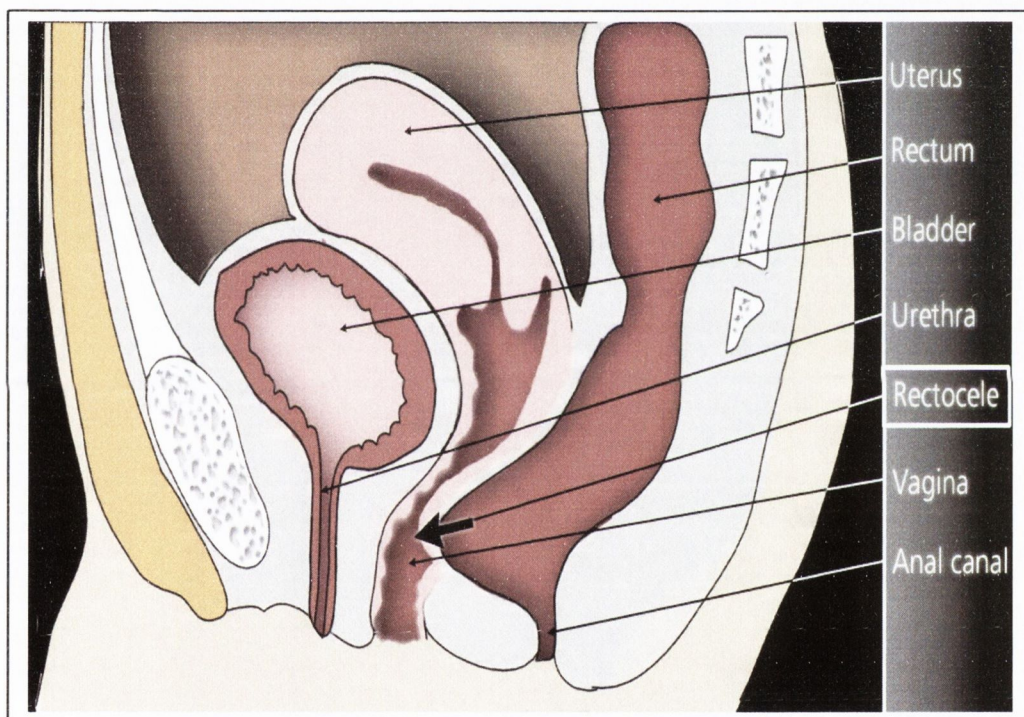


Figure 3.1: Diagram of the pelvis showing a rectocele (*Photograph kindly provided by Professor FBV Keane*)

2. **Sigmoidoceles** and **enteroceles** are commonly preceded by history of hysterectomy (6%-25% of cases) and frequently associated with rectal intussusception in about 55% of cases (Hawksworth and Roux 1958; Mellgren, Anzen et al. 1995) (Figure 3.2).

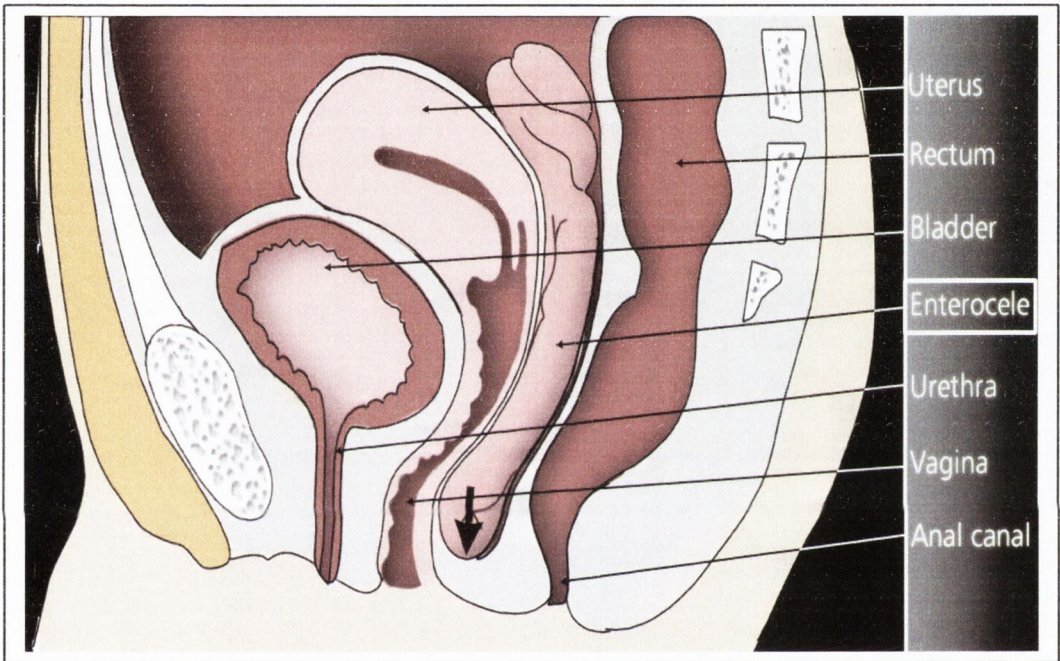


Figure 3.2: Diagram of the pelvis showing an enterocele (*Photograph kindly provided by Professor FBV Keane*)

3. **Cystocele** is defined as descent of the bladder base below the pubococcygeal line (Comiter, Vasavada et al. 1999). It can often cause extrinsic compression of the rectum leading to symptoms of obstructive defecation (Figure 3.3).

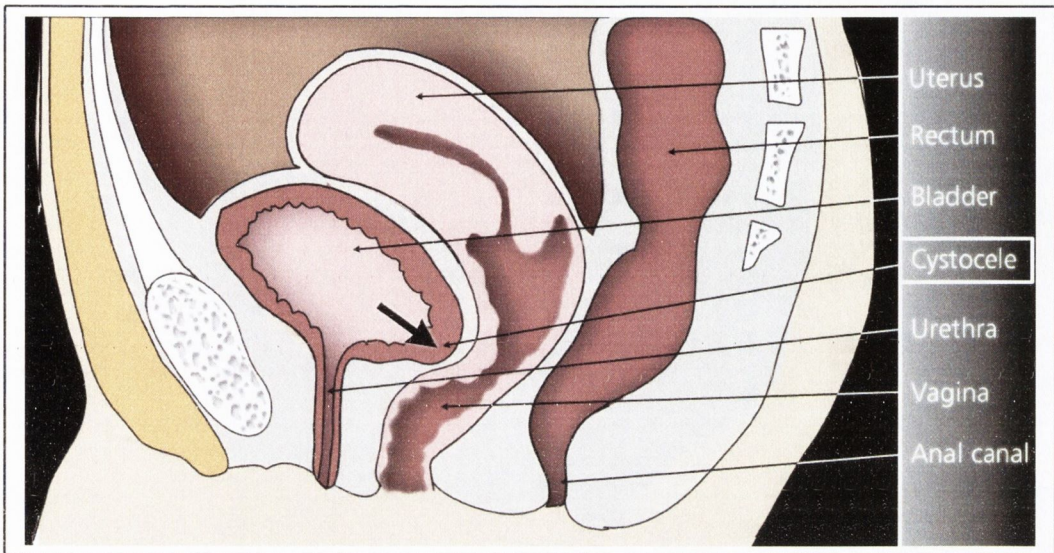


Figure 3.3: Diagram of the pelvis showing a cystocele (*Photograph kindly provided by Professor FBV Keane*)

4. Paradoxical puborectalis contraction (puborectalis dysynergia) or anismus is the disturbance and failure of relaxation of the striated pelvic floor and anal sphincter musculature, leading to functional obstruction of defecation at the pelvic outlet. A common association between this problem and history of sexual abuse in women has been described (Leroi, Berkelmans et al. 1995; D'Hoore and Penninckx 2003).

Classification of rectocele and sigmoidocele:

Rotholtz et al classified rectocele (according to their location on defecating proctogram), as: high, mid and low level rectoceles. The **high** rectocele occurs due to stretching or disruption of the upper third of the vaginal wall and uterosacral ligaments. The **mid-level** rectocele is the commonest type and is due to weakness of the pelvic floor support, caused by obstetric trauma. The

low-level rectocele occurs as a result of defects in the perineal body due to stretching or distension at childbirth (Rotholtz and Wexner 2001). Marti et al described another method of classification-based on fluoroscopic appearance, and there are three types. **Type I** is the digitiform rectocele or single hernia through the rectovaginal septum. **Type II** is a complex consisting of large sacculations, lax rectovaginal septum, anterior rectal mucosal prolapse, and deep pouch of Douglas; and is frequently associated with an enterocele. **Type III** is rectocele associated with intussusception and or prolapse of the rectum (Marti and Deleaval 1999).

Sigmoidoceles are classified as first, second and third degrees-based on their level of occurrence. The **first-degree** sigmoidoceles occurs cephalad to the pubococcygeal line; a **second-degree** sigmoidocele lies below the pubococcygeal line but above the ischiococcygeal line; and a **third-degree** sigmoidocele descends below the ischiococcygeal line (Jorge, Yang et al. 1994). These classifications have so far been based on radiologic and fluoroscopic findings, which entail the use of various contrast agents and high doses of irradiation.

3.3 Patient selection for Surgery

Because of the heterogeneity of the aetiology of constipation, its surgical management is often associated with poor outcome and significant morbidity (Wexner and Bartolo 1995; Pfeifer, Agachan et al. 1996; Alabaz, Nessim et al. 1999; El-Salhy 2003). It is therefore necessary to investigate these patients

comprehensively in order to properly select and tailor surgical management in accordance with the extent of the lesion and functional disturbances. It is instructive to note that diagnosis based solely on physical examination tends to underestimate the degree of prolapse or may indeed miss it; also, physical examination is not as accurate as MRI (Comiter, Vasavada et al. 1999; Goh, Halligan et al. 2000; Dohke, Mitchell et al. 2001). In a review by Marti et al it was shown that with proper patient selection, many centres had good results (79.5%-95% success rate, with a follow-up period of between 1year to 3years) with the endoanal approach for managing rectoceles; he also described procedures of choice for correcting the various types of rectocele (Marti and Deleaval 1999). Furthermore, the dominating abnormality can obscure other concomitant pelvic floor abnormalities and failure to identify all of the involved sites may result in incomplete surgical repair with subsequent persistence or recurrence of the prolapse (Wiskind, Creighton et al. 1992; Lienemann, Anthuber et al. 1997).

3.4 Investigations

Investigations include: screening blood tests to exclude metabolic, endocrine or systemic diseases. Physiologic evaluation includes tests such as anorectal manometry, anal sphincter electromyography, pudendal nerve study, and colonic transit study.

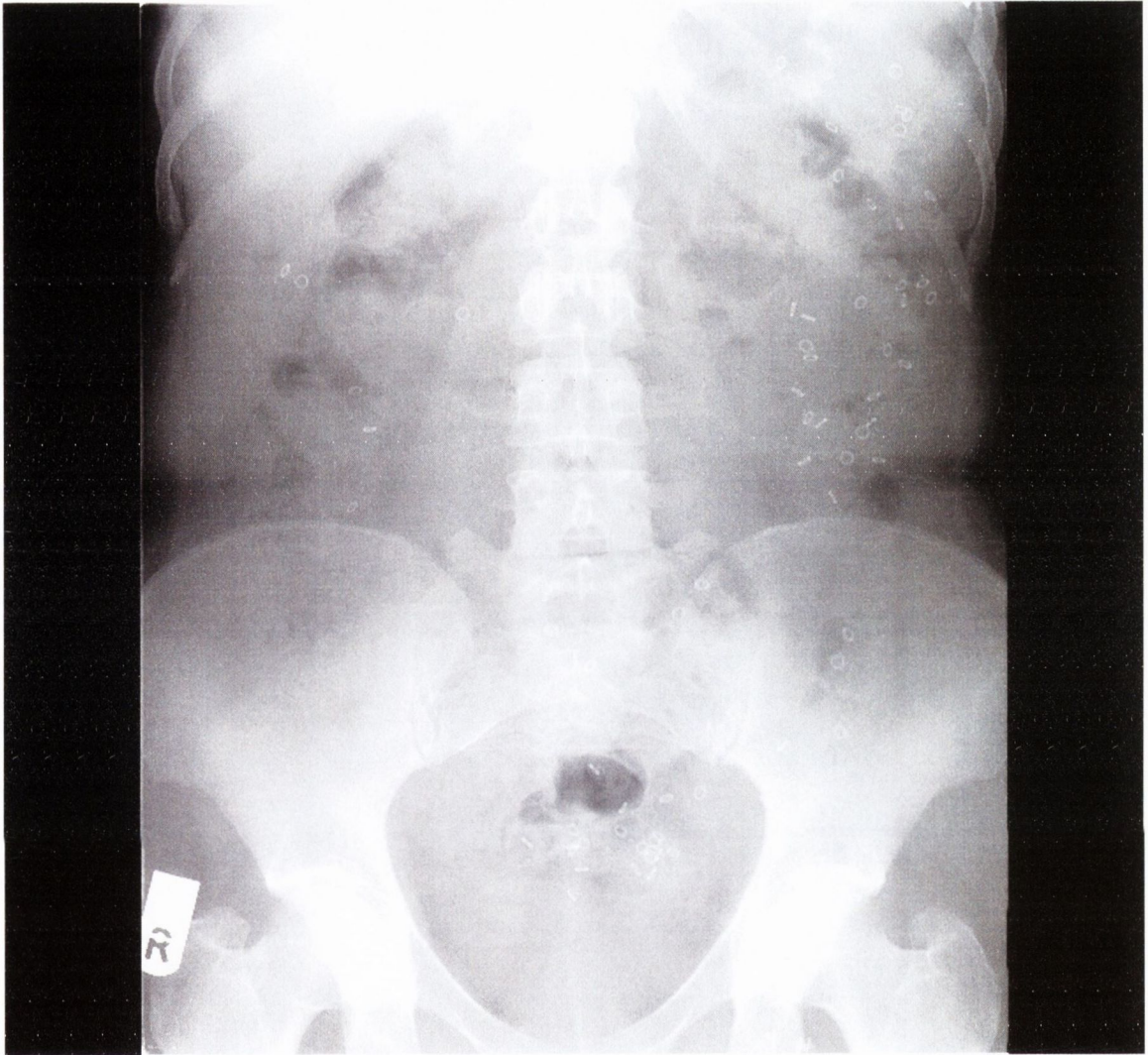


Figure 3.4A: Initial colonic transit image (taken on day 4) of a patient with obstructive defecation

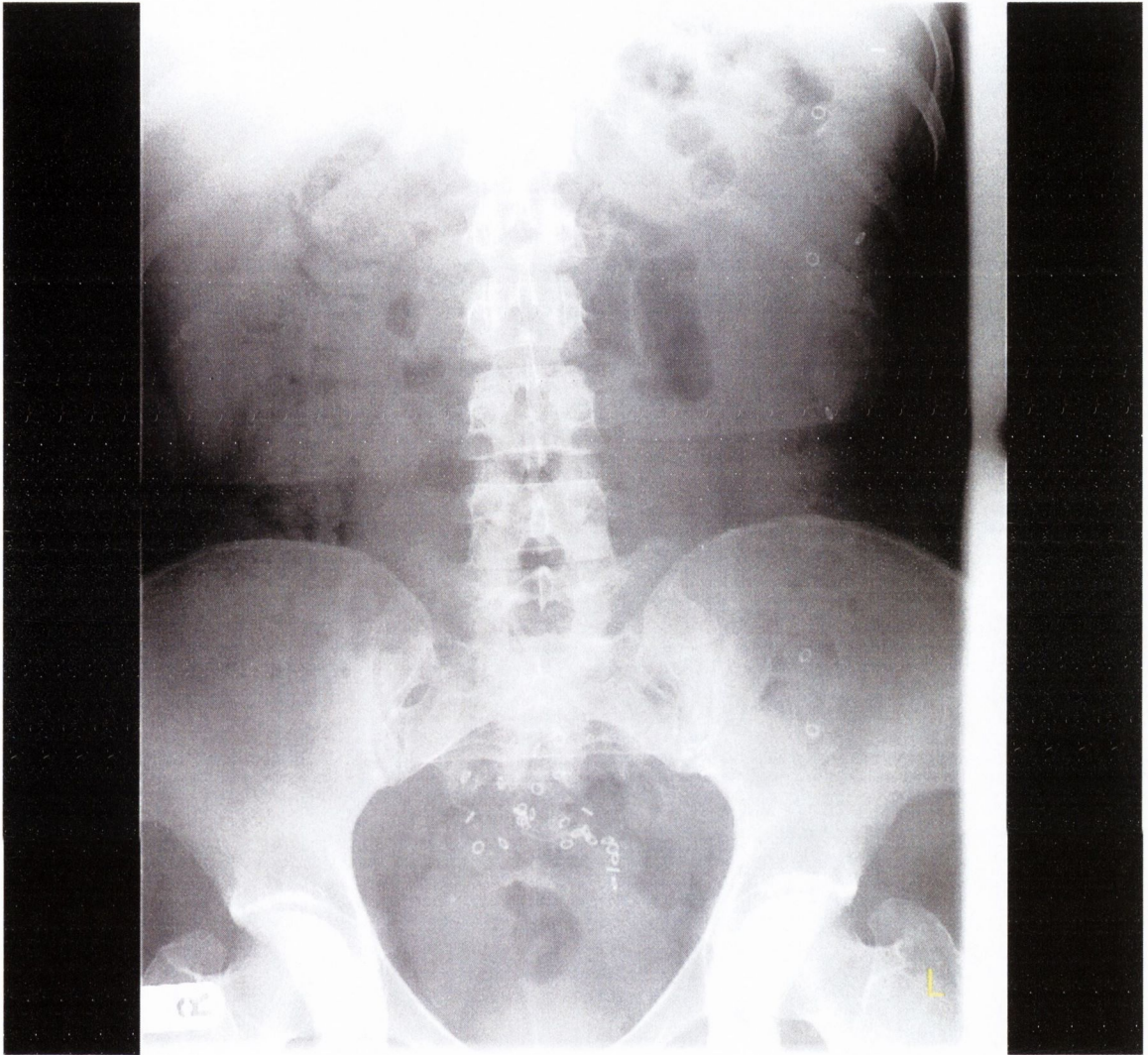


Figure 3.4B: Colonic transit image of same patient with obstructive defecation (taken on day 7) showing retention of markers in the rectosigmoid

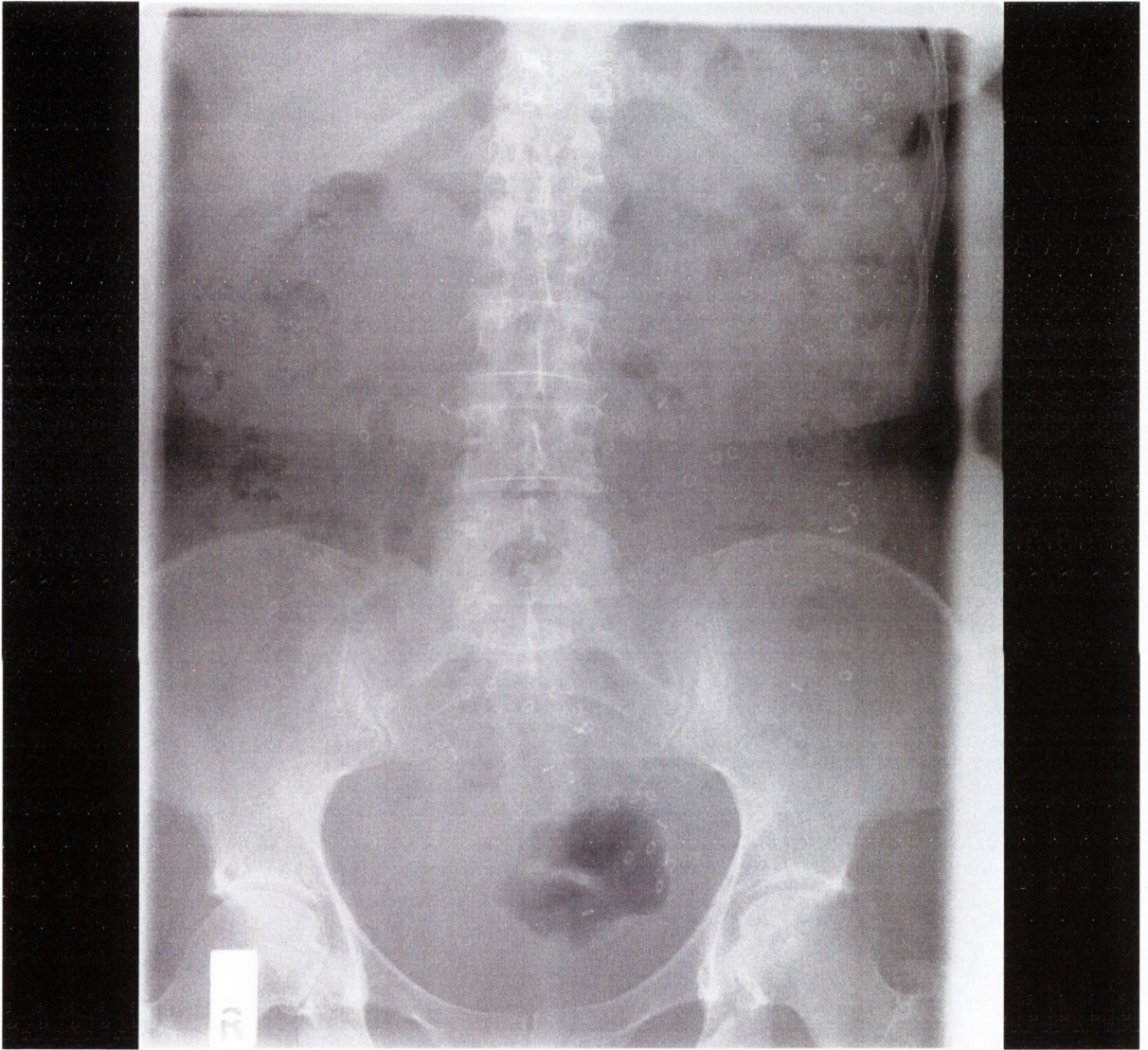


Figure 3.5A: Initial colonic transit image (taken on day 4) of a patient presenting with constipation

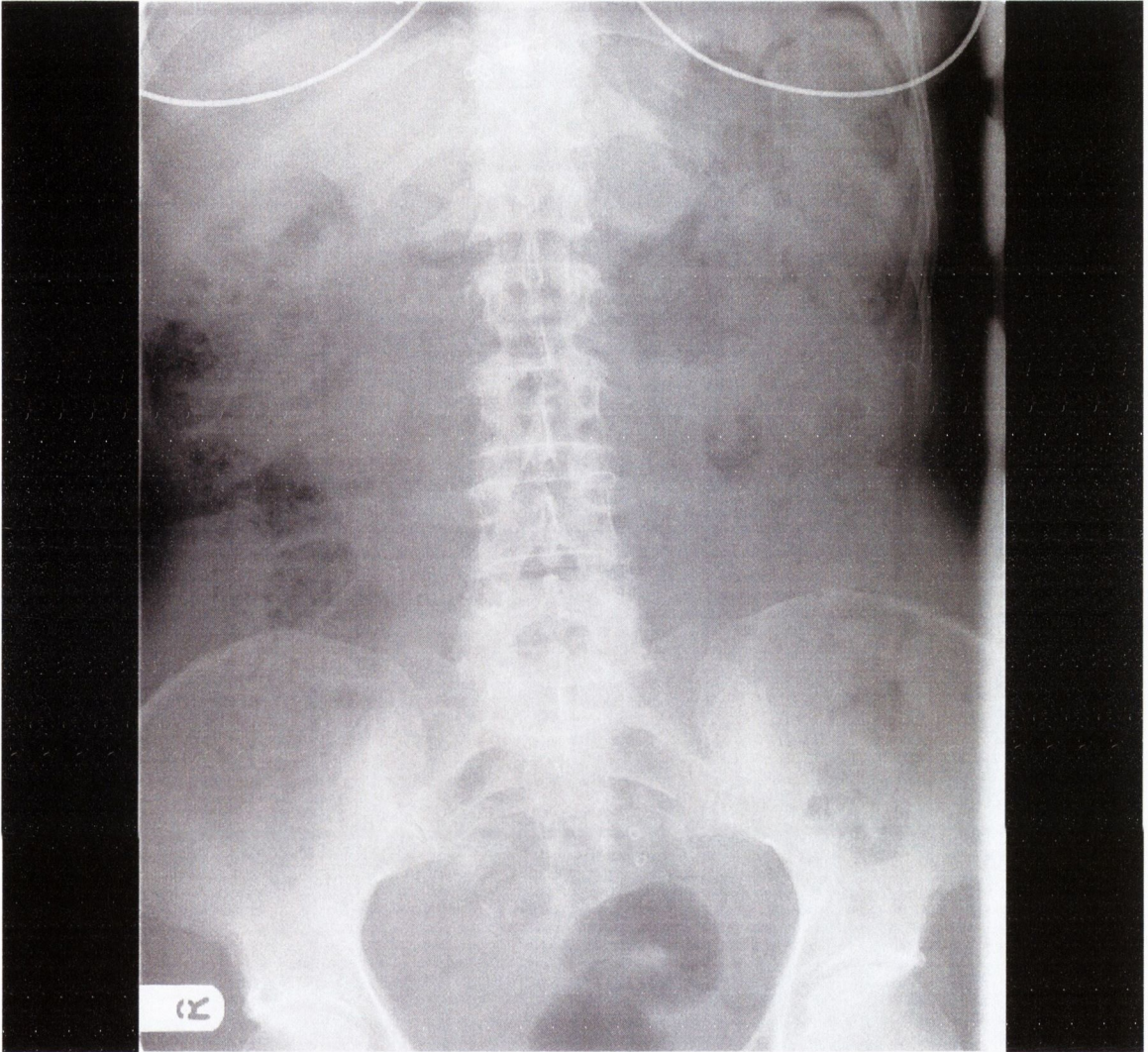


Figure 3.5B: Colonic transit image of same patient (taken on day 7) post ingestion of markers showing effective clearance of markers

Imaging techniques traditionally used for the evaluation of constipation include barium enema, evacuation proctography and cinedefecography. While these investigations are useful in assessing the colon and the anorectum, they offer little information about adjacent structures, which may impart on the anorectum. Defecating proctography (synonyms: defecography, evacuation proctography)

has been used in the assessment of defecation disorders for nearly 50 years but its clinical value remains controversial. Its main indication is in the evaluation of constipation due to obstructive defecation (Kelvin, Maglante et al. 1994; Stoker, Halligan et al. 2001). Rentsch et al commented that although defecating proctography has increased our knowledge of evacuation disorders, the causes of combined pelvic floor disorders in females and complex disorders of the posterior compartment in males remains unclear in some patients (Rentsch, Paetzel et al. 2001).

Frequently, a thorough, often time consuming multiple opacification techniques of the different compartments (colpocystoproctography), neurological and physiologic measurements are necessary to identify the predominant disorder. This represents a relatively invasive method for achieving this goal (Kelvin, Maglante et al. 1992; Hock, Lombard et al. 1993; Karasick, Karasick et al. 1993; Kelvin, Maglante et al. 1994; Halligan and Bartram 1995; Sentovich, Rivela et al. 1995). The invasiveness of fluoroscopic procedures and the high doses of ionising irradiation they deliver, render them unsuitable for many patients. For instance, with evacuation proctography, the somatic dose of ionising irradiation ranges from 100-200Gy (in men and women) and gonadal dose ranges from 40Gy in men to 90Gy in women.

The introduction of Magnetic Resonance Imaging (MRI) as an investigative tool in this area is overcoming this limitation and is capable of providing high-resolution global assessment of the pelvis, its constituent organs and the

musculofascial support structures. MRI has the unique ability of depicting actual anatomic structures, such as the anorectum and levator ani muscles, which are important for pelvic support (Strohbehn, Ellis et al. 1996). Apart from shedding light on the structural abnormalities that may cause loss of normal levator function, MRI is beginning to provide information that may influence the clinical management of patients with obstructive defecation or pelvic floor prolapse. Healy et al commented that while excessive pelvic floor descent in constipated patients had previously been inferred at evacuation proctography, MRI has clearly demonstrated multiple organ prolapse along with ballooning of the puborectalis muscle and marked depression of the levator ani muscle. They also opined that evacuation proctography resulted in underestimation of the frequency of enterocele and rectogenital hernias, even when the small bowel and vagina are opacified, because it may not depict hernias filled with mesenteric fat; while in contrast MRI enabled accurate delineation of small bowel and mesenteric fat in rectogenital hernias (Healy, Halligan et al. 1997).

The rapid development in the MRI technology has now made its use very adaptable for the evaluation of pelvic floor disorders. Some of the limitations of MRI are the problem of motion artefacts and its inability to adequately recognise functional anorectal disorders such as anismus and rectal intussusception (Kelvin, Maglinte et al. 2000). The development of stronger gradient sequences with rapid image acquisition has made dynamic pelvic assessment, in the straining and breath holding phases possible, with minimal problems of motion artefacts (Matsuoka, Wexner et al. 2001). The complete absence of ionising

irradiation, and non-contrast techniques has also made MRI an attractive investigative tool as it can be employed in patients of all ages especially women of child bearing age. The objective of our study was to evaluate the use of dynamic, phased array, pelvic MRI in the assessment of patients with obstructive defecation, using the widely available closed magnet system.

3.5 Materials and Methods

Our patient population comprised of all patients who were referred to the Pelvic Physiology Laboratory, at the Adelaide and Meath Hospital, Dublin, requiring assessment for obstructive defecation. This group of patients were compared to a group of healthy asymptomatic age and parity-matched controls.

After a full history and clinical examination, all the patients underwent colonoscopy, anorectal manometry, and pudendal nerve terminal motor latency testing. Anorectal manometry was performed on all patients with obstructive defecation, using MUI scientific, eight-channel, water-perfusion, catheter (Medtronic Synaptic®). The station pull-through technique as previously described was adopted in this study (Stendal 1997). The parameters measured were mean maximal resting pressures; mean incremental squeeze pressures, rectal volume compliance, rectal sensation and rectoanal inhibitory reflex. Pudendal nerve terminal motor latency test (PNTMLT) was performed in the standard fashion to assess the integrity of the pudendal nerves (St. Marks pudendal stimulating electrode; Dantec Medical, Denmark). The normal reference value used was 2.0 ± 0.2 ms and latency periods greater than 2.2 ms

were regarded as abnormal (Stendal 1997). All these patients also had colonic transit studies and evacuation proctography (defecography). The classification used for describing defecographic findings in our study was that described by Marti et al. (Figure 3.6).

Phased array, non-contrast, dynamic MRI was performed (as previously described) in all the patients and healthy volunteers after obtaining detailed informed consent (1.5-T Magnetom Symphony®, Siemens Corp., Erlangen, Germany). The investigator performing the MR image analysis was blinded to both the clinical history and findings of the physiological studies of the patients. Approval for the study was obtained from the hospital's research and ethics committee.

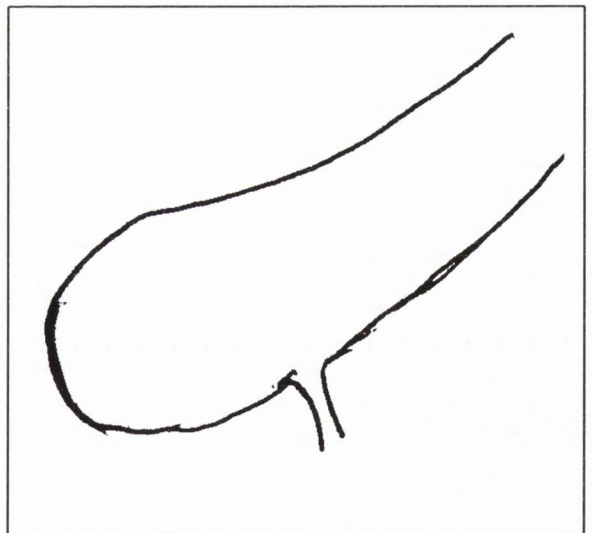


Figure 3.6 A: Type 1 Digitiform rectocele or single hernia through the rectovaginal septum

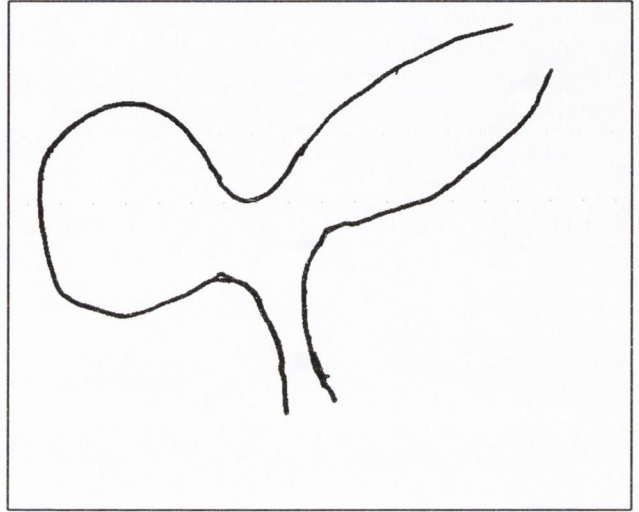
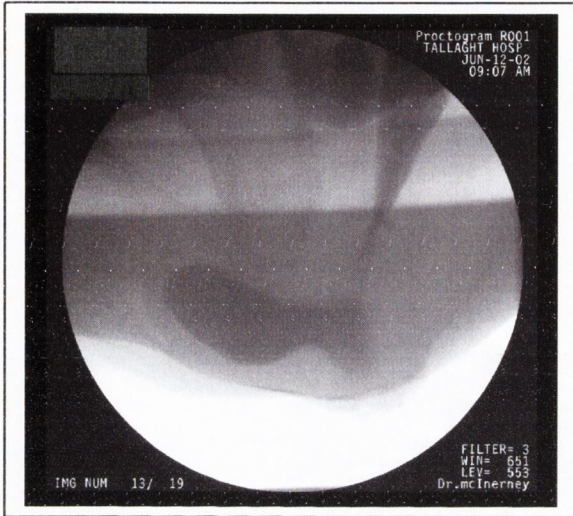


Figure 3.6 B: Type II Rectocele showing large sacculation and deep pouch of Douglas frequently associated with an enterocele

3.6 Statistical analysis

Statistical analysis was performed using the software package (SPSS 10.0 for windows®, SPSS Inc. Chicago, Illinois). All results are presented as means \pm Standard Deviation (SD) unless otherwise stated. The independent T-test for equality of means was applied. A P-value of less than 0.05 was considered to indicate a statistically significant difference.

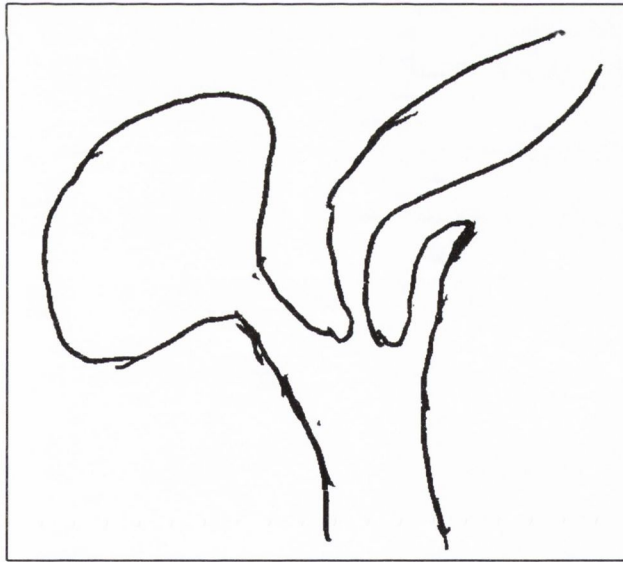
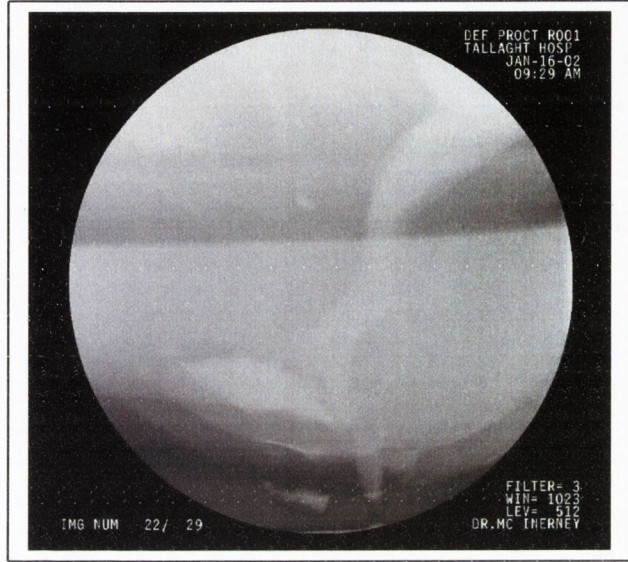
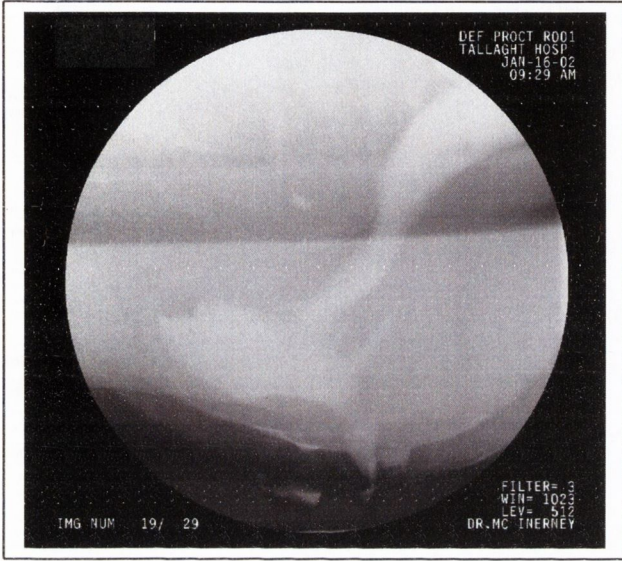


Figure 3.6C: Type III Rectocele showing intussusception and prolapse of the rectum

3.7 Results

Eighteen consecutive female patients referred to the pelvic physiology laboratory with complain of obstructive defecation (OD) were assessed. A control group of 15 asymptomatic females, with no history or symptoms of gastrointestinal or urinary disorders were also recruited. Both groups were matched for age and parity; with mean age of 46.6 ± 13.15 years and 40.47 ± 12.08 years, ($p = 0.2$), for the OD and asymptomatic control groups respectively. The mean parity was 2.08 ± 1.8 and 2.40 ± 1.7 , ($p = 0.6$), for the symptomatic and control groups respectively. The OD patients had anorectal manometric evaluation, which showed the mean of the anal canal high-pressure zone length (HPZ) to be 1.7 ± 1 cm; mean resting pressure of 88.76 ± 24 mmHg; mean squeeze pressure of 87.31 ± 37.49 mmHg, and mean maximum tolerated volume of 221 ± 112.66 mls. The control subjects did not have manometry. The colonic transit studies showed significant delay in the rectosigmoid, with a mean rectosigmoid segmental transit time of 31.2 hours (normal = 12.4 hours).

3.7.1 Levator Plate Complex

Analysis of the levator plate complex in the mid coronal image demonstrated significant decrease in angle of the arch of the levator ani muscle in the OD group compared to the controls ($6.4^0 \pm 5$ vs. $10^0 \pm 10$; $P=0.04$). There was no statistically significant difference in the width of the levator hiatus in the symptomatic group compared to controls (53.9 ± 10 mm vs. 47.7 ± 7 mm; $P=0.7$).

Although the levator-ani muscle length and thickness did not differ significantly in the two groups ($45\pm 6\text{mm}$ vs. $44\pm 5\text{mm}$, $p=0.6$ and $4.7\pm 1\text{mm}$ vs. $4.4\pm 1\text{mm}$, $P=0.5$ respectively), there was a significant reduction in the area of the levator ani muscle of the symptomatic group compared to the controls ($19.2\pm 1\text{ mm}^2$ vs. $35.7\pm 1\text{ mm}^2$, $p=0.001$). There was no difference in the tissue density of the levator ani muscle of the symptomatic group versus the controls (127.3 ± 25 pixels vs. 126.2 ± 24 pixels, $p=0.3$). Table 3.1 shows the comparative data of the two subgroups of symptomatic patients compared to the controls

3.7.2 Anal sphincter complex

There was no difference in measurement of the external anal sphincter (EAS) length between symptomatic and controls ($23.7\text{mm}\pm 6$ vs. $25.6\text{mm}\pm 11$; $p=0.6$). However, the length of the inner layer of EAS was significantly decreased ($4.0\text{mm}\pm 3$ vs. $7.5\text{mm}\pm 2$; $p=0.02$). Similarly, there was a significant decrease in the measurement of the intersphincteric groove ($2.4\text{mm}\pm 2$ vs. $4.0\text{mm}\pm 1$; $p=0.005$). There was however, no statistically significant difference in the thickness of the EAS muscle ($4.6\text{mm}\pm 1$ vs. $7.0\text{mm}\pm 9$; $p=0.4$).

3.7.3 Perineal descent and organ prolapse

There was no significant difference in the anterior urethrovesical angle of the OD group compared to the controls $80^0 \pm 21$ vs. $76.2^0 \pm 15$ respectively, $p = 0.6$. Though the anorectal angle was lesser in the constipation than in the control group, this difference was not statistically significant (101.6 ± 12 vs. 106.3 ± 12 , $p=$

0.3). The degree of perineal descent was also significantly more in the symptomatic group compared to the control group $15.74\text{mm} \pm 13$ vs. $6.1\text{mm} \pm 6$ (mean \pm SD), $p = 0.02$.

Table 3.1: Parameters of the combined incontinence and the obstructive defecation subgroups compared to the controls

Parameters	Controls	Combined incontinence (<i>P</i>)	Obstructive defecation (<i>P</i>)
Levator Hiatus	48 ± 6	58.3 ± 8 (0.001)	53.9 ± 10 (0.7)
Levator Angle	14 ± 10	3.0 ± 5 (0.004)	6.4 ± 5 (0.04)
Levator Area	26.9 ± 1	19.5 ± 1 (0.001)	19.2 ± 1 (0.001)
Levator Density	126.1 ± 23	157.3 ± 47 (0.025)	127 ± 25 (0.3)
EAS Length	6.6 ± 13	20.0 ± 5 (0.03)	23.7 ± 6 (0.6)
Inner Layer of EAS	7.0 ± 2	3.1 ± 3 (0.001)	4.0 ± 3 (0.02)
Urethrovesical Angle	73.5 ± 10	91 ± 23 (0.004)	80 ± 21 (0.5)
Anorectal Angle	106.3 ± 12	118.5 ± 15 (0.1)	101.6 ± 12 (0.3)

3.7.4 Comparing evacuation proctography and straining dynamic MRI

Evaluation of rectocele and cystocele (Figures 3.6, 3.7 and 3.8) showed that of the 18 patients, minimal, moderate and severe rectocele occurred in 3, 6, and 5 patients respectively; four patients did not have demonstrable rectocele. Minimal, moderate and severe cystoceles were demonstrated in 5, 1, and 4 patients respectively; cystocele was not demonstrated in eight patients. Using the

classification of Marti et al, the findings of evacuation proctography and dynamic MRI are summarised in Table 3.2. There was concordance between proctography and MRI in determining rectocele type in 10/18 (56%); and 12/18 (67%) concordance in determining the degree of rectocele.

Table 3.2: Classification of rectocele using proctography and MR

Class	Evacuation Proctography		Magnetic resonance imaging	
	Type	Degree	Type	Degree
I	3	5	8	3
II	5	6	5	7
III	4	1	1	4

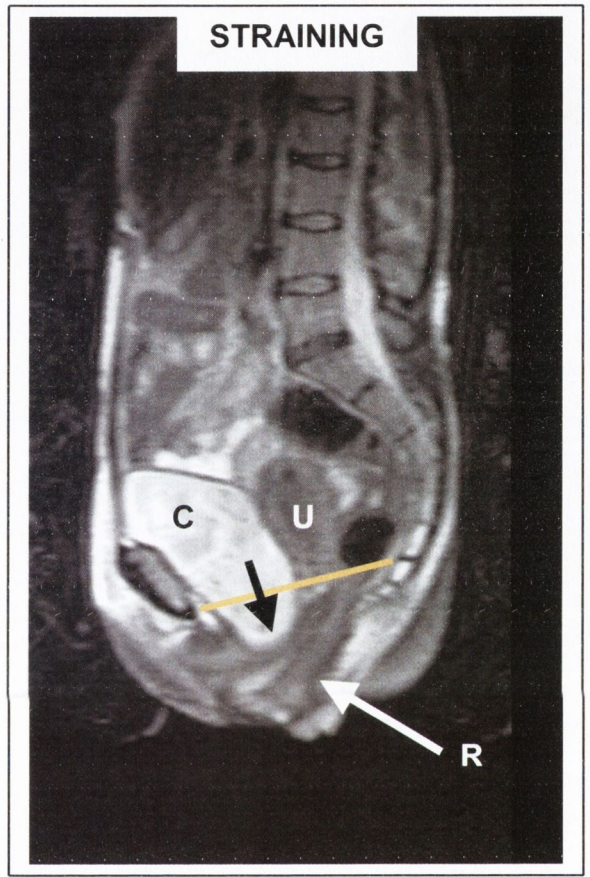


Figure 3.6: Images of the same patient taken at rest and during straining. Straining image shows multiple organ prolapse, cystocele (C), rectocele (R), and uterine prolapse (U)

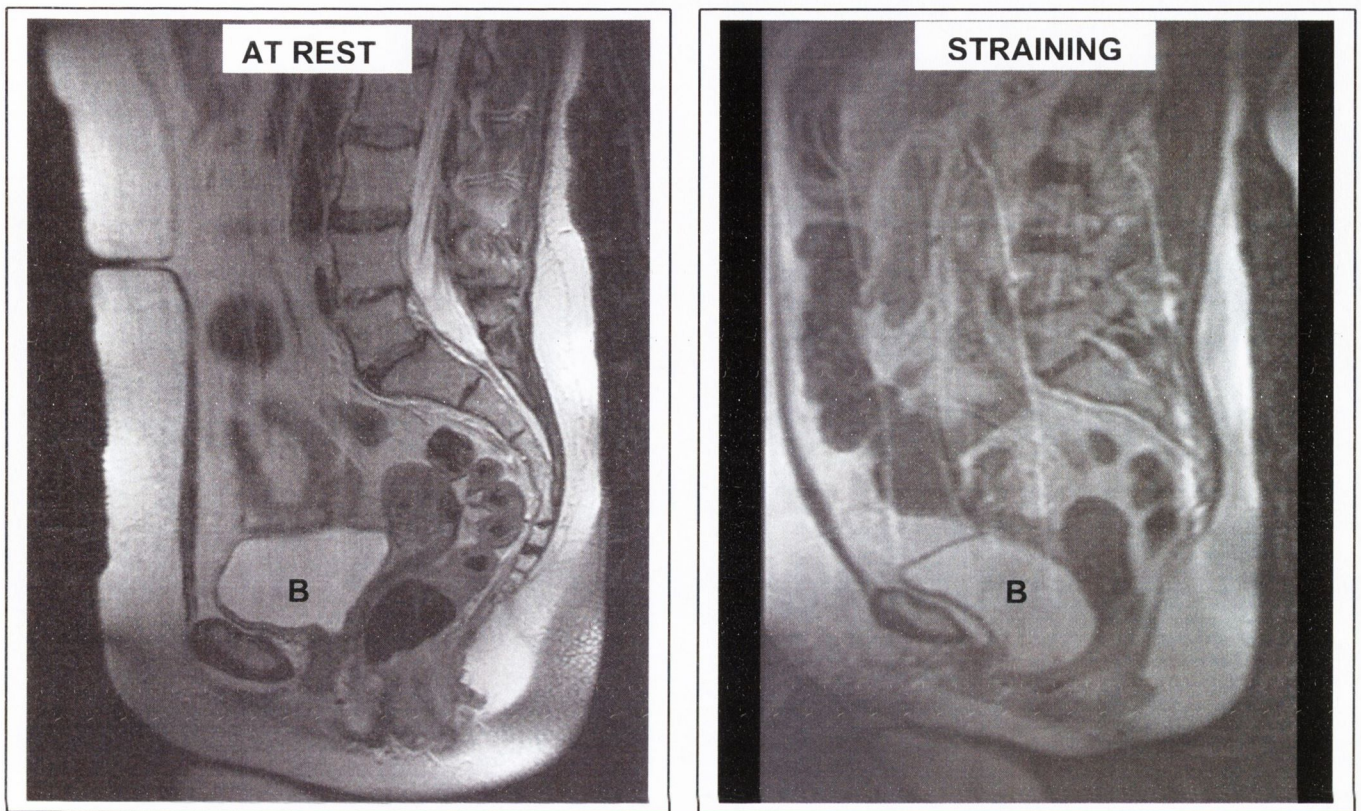


Figure 3.7: Images of the same patient taken at rest and during straining. Straining image shows significant bladder descent and retro-pulsion (B), causing extrinsic compression of the vagina and rectum

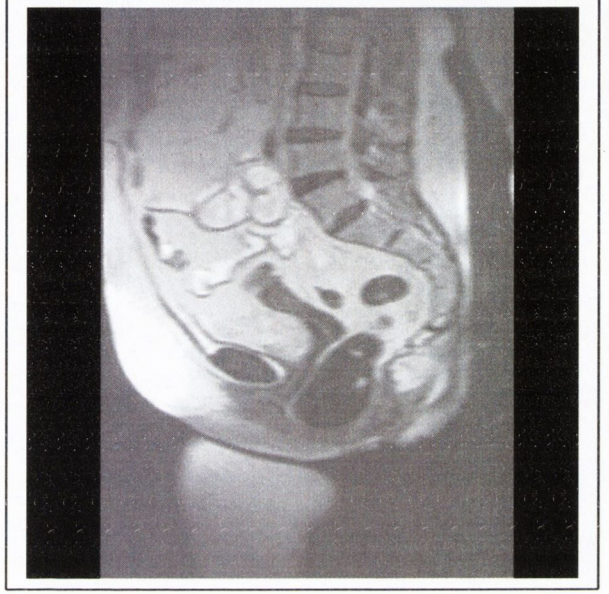
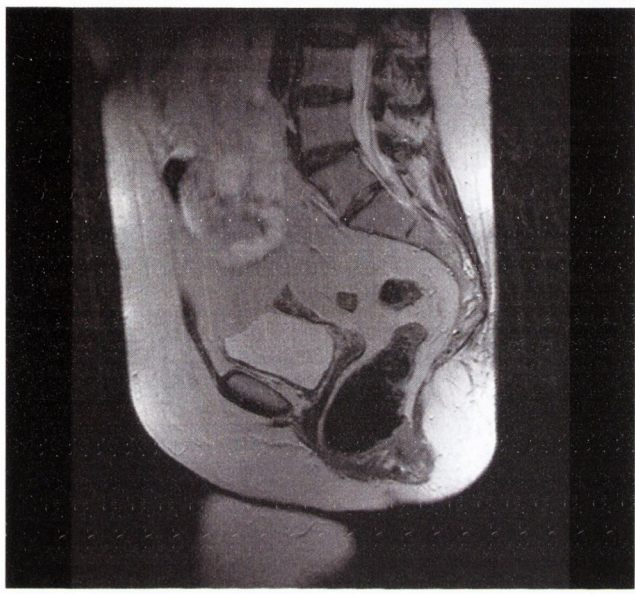


Figure 3.8: Images of the same patient taken at rest and during straining, showing significant rectocele

3.8 Discussion

Because of the multiple aetiologies and complex nature of combined pelvic floor disorders, a thorough evaluation of the pelvis is crucial for any patient presenting with complains of constipation and obstructive defecation. These disorders frequently involve both anatomic and functional disturbances of different intrapelvic structures and compartments (Figure 3.7), and we know that physical

examination alone is often insufficient for defining the nature and degree of visceral prolapse and pelvic floor relaxation (Comiter, Vasavada et al. 1999; Rentsch, Paetzel et al. 2001). Up until the advent of MRI careful medical history and clinical examination combined with investigations such as defecating proctogram and anorectal physiology were used in the assessment of various pelvic floor disorders, but these often led to incorrect or incomplete surgical management with unsatisfactory outcomes (Wiskind, Creighton et al. 1992; Rentsch, Paetzel et al. 2001). The use of pelvic floor MRI as a complimentary investigative tool is progressively overcoming these limitations.

Though the levator ani muscle (LAM) length, thickness and area were similar to those of faecal incontinence when compared to the controls, the LAM tissue density did not differ significantly from that of the control group. This finding was in contrast to the significantly reduced tissue density that was demonstrated in the incontinence group. The main causes of reduction in tissue density are fibrosis and fatty infiltration of muscle tissue (Williams, Malouf et al. 2001), which might be the sequelae of pelvic floor ischaemia suffered during childbirth. However, the effect of denervation in incontinence or the role of chronic denervation in obstructive defecation, with regard to tissue density alteration remains unclear.

The anal canal length of the symptomatic group did not differ significantly from the controls; this again contrast to the findings of the incontinence group where the EAS length was significantly reduced compared to the assyptomatic

controls. The anorectal angle was lesser in the OD group compared to the control this also contrasted the results of the combined incontinence group where the anorectal angle was wider than the control group. The puborectalis sling largely, maintains the anorectal angle and in OD patients, puborectalis dyssynergia may be a cause of the symptoms. To fully identify the aetiology of patients symptoms, combining the findings dynamic pelvic MRI, proctography and colonic transit study is needed. Of note, the anterior Urethrovesical angle did not differ significantly between the OD and the control groups, while there was a significant widening of the angle in the incontinence group compared to the controls.

Apart from the direct anorectal causes of OD, this study also identified extra colonic factors that often caused or contributed largely to the symptoms of obstructive defecation. A significant number of our patients demonstrated rectocele and cystocele of sizes, significant enough to cause symptoms (Figure 3.7). Some of our patients had large cystocoeles or enterocele, which descended into the pelvic cavity on straining, causing significant compression of the rectum. Interestingly the indentation caused by this extraluminal compression was reported as a rectocele on proctography, because they are often associated with retention of contrast (Figure 3.9). It is in these types of cases that dynamic pelvic floor MRI has proven to be a very useful clinical tool. It is important to note that though there was moderate level of concordance between the findings of evacuation proctography and dynamic MRI, the former is essentially a test of evacuation in a distended rectum while the later is a straining test. The MRI was

more accurate in determining the degree of rectocele because it clearly visualized the surrounding pelvic structures in relation to the rectocele, however proctography gave a better picture of the type of rectocele as shown earlier. It is the combination of this morphological information that makes both tests effective complimentary investigations.

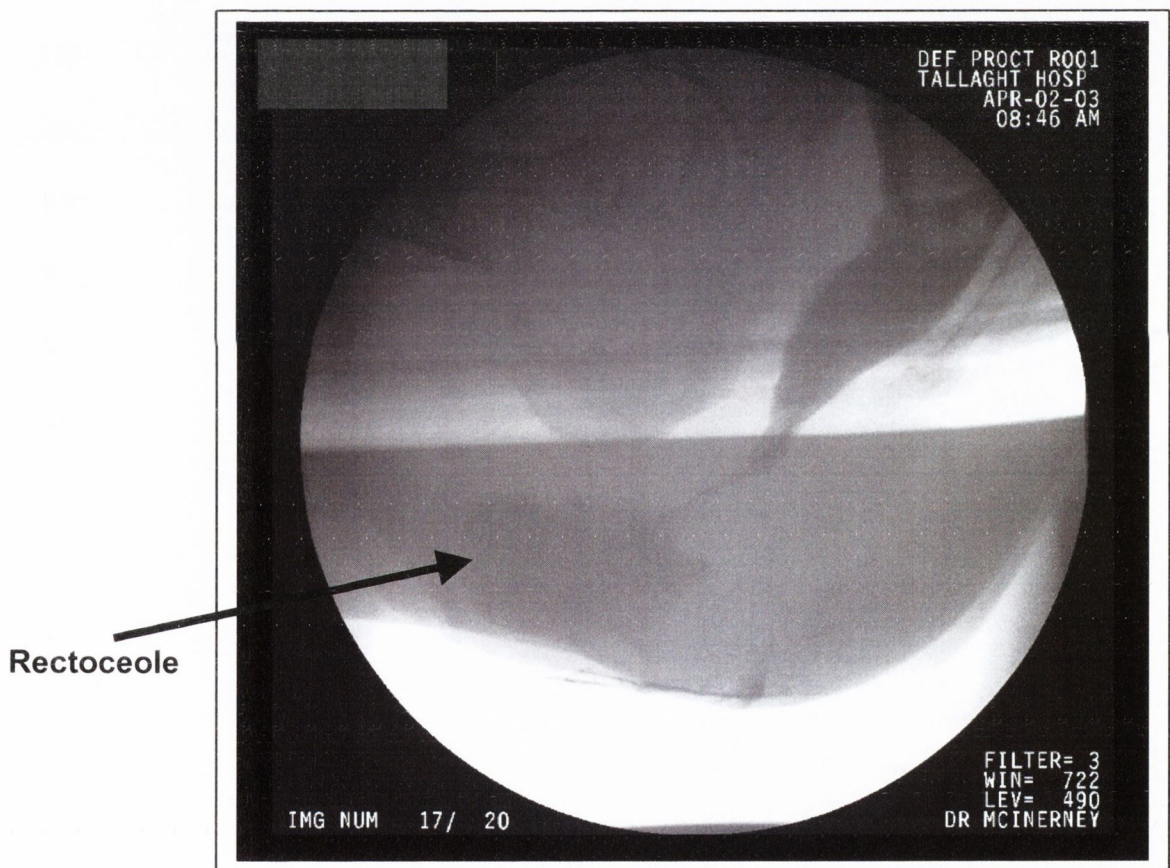


Figure 3.9: Defecating proctogram demonstrating an anterior rectocele

Enterocele was not demonstrated in many of our patients; an explanation for this could be that our MRI scans were performed in the supine position. It has

been advocated that the use of the open MRI scanner, that will enable images to be taken in the sitting position. The additional factor of gravity in the upright position will accentuate the features identified on the MRI images. However, these open scanners are presently not readily available in many institutions.

3.9 Conclusion

This study has again highlighted the important role that dynamic pelvic floor MRI can play in the identification of the various defects, which may result in the symptoms of obstructive defecation. When combined with the findings of other investigative tools, it will help in better patient selection and in the choice of the surgical remedy that is likely to yield optimum results for the patients. A larger study population will be needed to standardize the parameters used in this assessment.

CHAPTER 4

THE ROLE OF STRENGTH DURATION TEST IN ASSESSING ANAL SPHINCTER DYSFUNCTION: AN OLD TOOL WITH NEW USEFULNESS

4.1 Introduction

The act of normal defecation and continence requires an intact neurophysiological and musculofascial support system. An aberration in these systems results in the phenomenon of pelvic floor dysfunction (PFD). PFD is a common problem with huge health care implications, manifesting either as faecal or urinary incontinence, constipation and/or obstructive defecation; and pelvic organ prolapse. Faecal incontinence has a prevalence of about 1% of community-dwelling adults and 7.8% to 18.4% of the general population (Jorge and Wexner 1993; Nelson, Norton et al. 1995; Vaizey, Kamm et al. 1998). Urinary incontinence affects 4% of nulliparous women under 35 years, 9% of women up to the age of 65 years and 12% of women greater than 65 years. Various aetiological factors have been implicated in PFD and these, broadly classified are: anatomical, neurological and functional. Numerous clinical and imaging tests developed for investigating these problems include colonoscopy, cystoscopy, endoanal ultrasound, anorectal manometry, urodynamic studies, pudendal nerve terminal motor latency test (PNTMLT), electromyography (EMG), defecating proctogram and magnetic resonance imaging. However, none of

these has been adequate in offering sufficient understanding of the Pathophysiology of PFD.

The pudendal nerve studies though useful when it detects neuropathy, normal latency is of little importance, as latency measurement is inherently an insensitive indicator of nerve damage because, even when the nerve has undergone considerable trauma, the survival of a few fast conducting neurones can preserve normal latencies. The latency measurement only includes the terminal pathway along the pudendal nerve (from the ischial spine) to the muscle, thus nerve damage occurring proximal to this segment will not usually be reflected in the latency (Mills, Hosker et al. 2002) . It has also been shown that between 49-75 per cent of patients with faecal incontinence will have prolonged latency (Deen, Kumar et al. 1993; Roig, Villoslada et al. 1995). Rectoanal inhibitory reflex and rectal sensation, which are indicative of the integrity of pelvic autonomic innervation, are vital parameters but there is considerable overlap of the profile of faecal incontinence patients and normal controls (Felt-Bersma, Klinkenberg-Knol et al. 1990). Concentric-needle EMG is also a useful investigative tool for neurological evaluation. It can detect muscle action potentials in up to 50 motor units in response to active contraction. However, it is not readily available, requires great deal of skill and time to perform. In addition, it is an invasive and painful procedure, and requires an intact pathway from cerebral hemisphere to motor endplate (Monk, Mills et al. 1998).

The inability of PNTMLT and EMG to fully elucidate pelvic neuropathy has led to the re-evaluation of the strength-duration test (SDT) as a complimentary investigation for PFD. Keith Lucas (1907) first mooted the concept of SDT, experimenting on the excitabilities of muscle and nerve. Adrian (1917) described strength-duration curve by applying the findings of Lucas to investigations on human nerve injuries. Since then, the equipment for this investigation has undergone series of refinement through the era of Bauwens and Ritchie (1944), Pollock, Wayne Parry, MacKenzie and a host of others (Waldsworth and Chanmugam 1988). SDT is a measure of the excitability of muscle or nerve tissue and has been suggested as a means of assessing external anal sphincter function (Trojaborg 1962; Mills, Hosker et al. 2002).

Mills et al and Monk et al respectively, conducted previous studies using this technique to assess anal sphincter function and faecal incontinence. Monk et al found that when combined with manometric data, SDT could discriminate between normal and faecal incontinence patients with a sensitivity and specificity of 95% and 100% respectively. Mills et al concluded that SDT significantly correlated with established measures of external anal sphincter function and its innervation. (Monk, Mills et al. 1998; Mills, Hosker et al. 2002). The aim of our study was to further refine the technique of this procedure and to determine its relationship with other established measures of anorectal function.

4.2 Physiology of strength-duration test

When nerve and muscle tissues are stimulated with electrical impulses, they respond in a characteristic form that reflects the strength and duration of the exciting stimulus. A curve obtained by plotting points representing the minimum current required to elicit detectable muscle contraction (along the ordinate), for series of square wave pulses of graded durations of stimulus (displayed along the abscissa), is called the strength-duration curve (Figure 4.1). Lapicque, in 1909 first described the hyperbolic nature of this relationship, which provides valuable information on the state of excitability of nerves and muscles (Lapicque 1909).

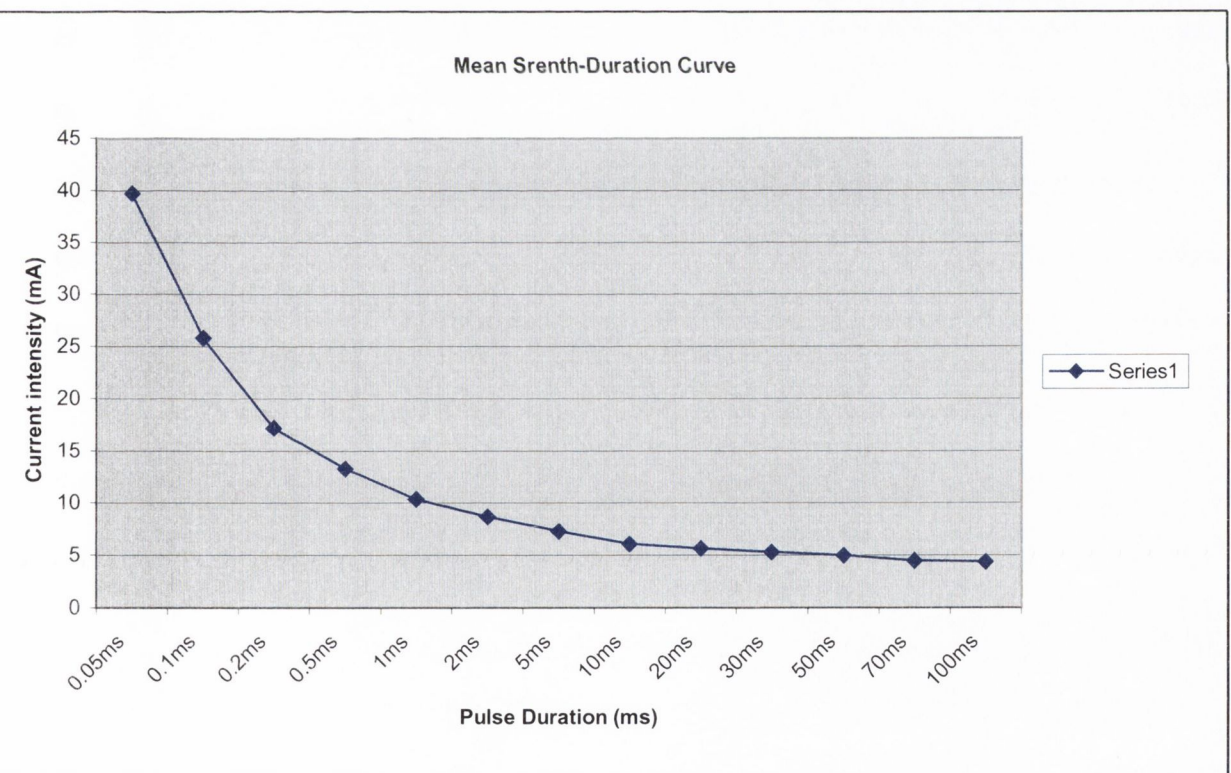


Figure 4.1: Strength Duration Curve

The current intensity needed to produce a minimal perceptible contraction varies and has an inverse relationship with current pulse duration. The normal muscle comprises of intramuscular nerve fibres that respond to currents of shorter duration and muscle fibres, which respond better to electrical pulses of longer duration (Kimura 1989). Consequently, the typical curve of a normal muscle reflects the short pulse constant characteristic of nerve fibres and a shift of the hyperbola upwards and to the right will indicate neuropathy or denervation.

4.3 Patients and Methods

All patients requiring assessment for disorders of faecal or urinary incontinence and obstructive defecation at the Adelaide and Meath Hospital, Dublin, are referred to the Pelvic Physiology Laboratory, which has the facilities for urodynamics and anorectal physiological assessment. Our study population consisted of a consecutive group of 50 female patients who volunteered to participate in this study. To exclude known causes of incontinence, after a full history and clinical examination, all the patients underwent relevant investigations, which included colonoscopy, cystoscopy, endoanal ultrasound, anorectal manometry, urodynamic studies and pudendal nerve terminal motor latency test (PNTMLT). In addition, these patients had strength-duration test after obtaining informed consent. Approval for this study was obtained from the hospital's research and ethics committee.

Anorectal manometry was performed on all patients using MUI scientific, eight-channel, water-perfusion catheter (Mui Scientific, Mississauga, ON, Canada). The station pull-through technique as previously described was adopted in this study (Neill, Parks et al. 1981; Stendal 1997). The parameters measured were mean maximal resting pressures; mean maximal incremental squeeze pressures, rectal volume compliance, rectal sensation and recto-anal inhibitory reflex. The peak and regression amplitude; and duration of the maximum sustained squeeze pressure were measured manometrically. The severity of patient's faecal incontinence was evaluated using the Wexner, Vaizey and faecal incontinence scoring systems (Jorge and Wexner 1993; Rockwood, Church et al. 1999; Vaizey, Carapeti et al. 1999). Pudendal nerve terminal motor latency test (PNTMLT) was performed in the standard fashion to assess the integrity of the pudendal nerves (St. Marks pudendal stimulating electrode; Dantec Medical, Denmark). The normal reference value used was 2.0 ± 0.2 ms and latency periods greater than 2.2 ms were regarded as abnormal (Neill, Parks et al. 1981; Kiff and Swash 1984). An experienced colorectal surgeon or senior trainees under his supervision performed the endoanal ultrasound in the standard fashion to out rule sphincter defects in symptomatic patients (2002 Panther®; Bruel and Kjaer Medical Systems, Inc. Mileparken 34, DK-2730 Herlev. Denmark). Urodynamic study was also performed in the standard fashion (Lewis and Abrams 2000).

Strength-duration test was performed with some modifications to the technique employed previously by Monk et al. and Mills et al. These modifications were

adopted after we identified steps that were likely to generate errors and inconsistent results.

1. With the original technique, the patient is positioned in the left-lateral position and the operator retracts the upper (right buttock) with the left hand while holding the stimulating electrode with the right hand. From our experience, this was cumbersome and more so in overweight or obese patients. It was also impossible to maintain a consistent force in retracting the buttock for the entire duration of the procedure. Consequently, a point is usually reached when the hand used for retraction becomes fatigued; this leads to tremor, which could mask the fine muscle contraction being sought. To overcome this problem, we devised a means of retracting the right and left buttocks with strips of adhesive tape (Fixomull® stretch, Beiersdorf AG, Hamburg, Germany), which are then fixed to the side railings of the trolley (Figure 4.2). This frees up the operator who can then assume the best position for sighting the sphincter contractions.

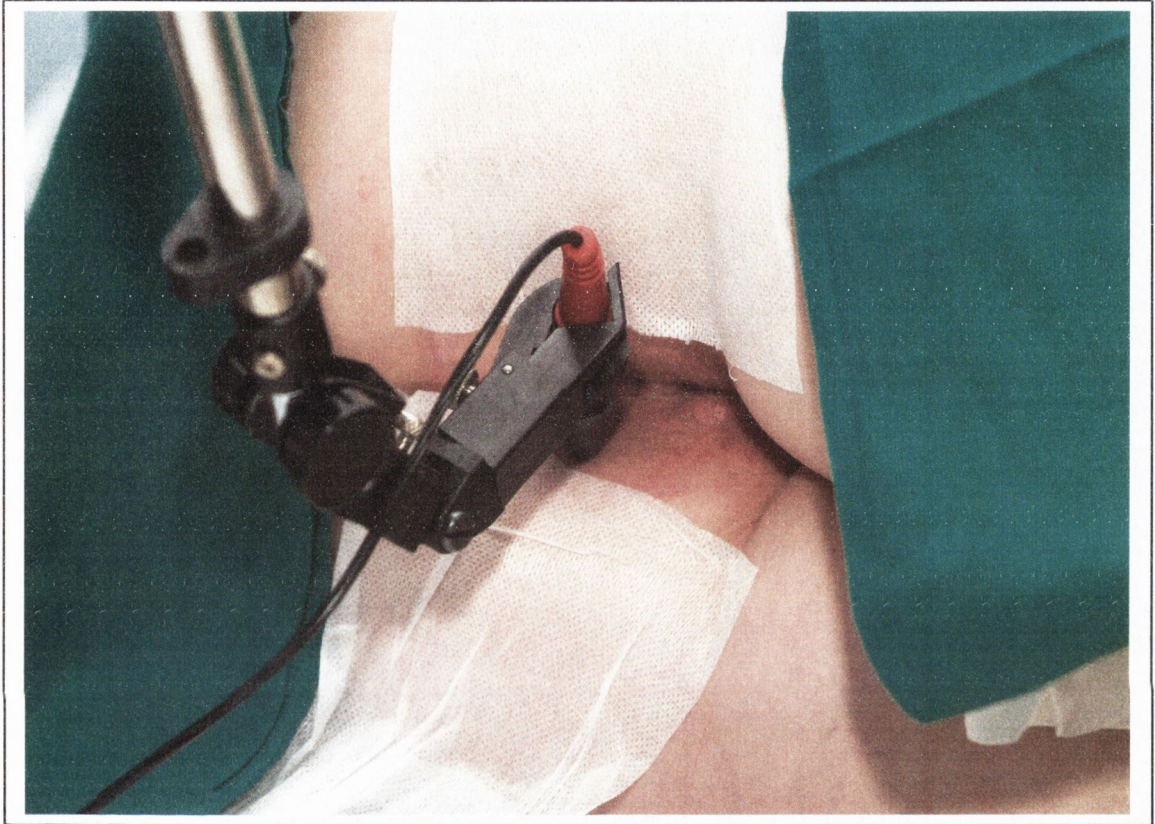


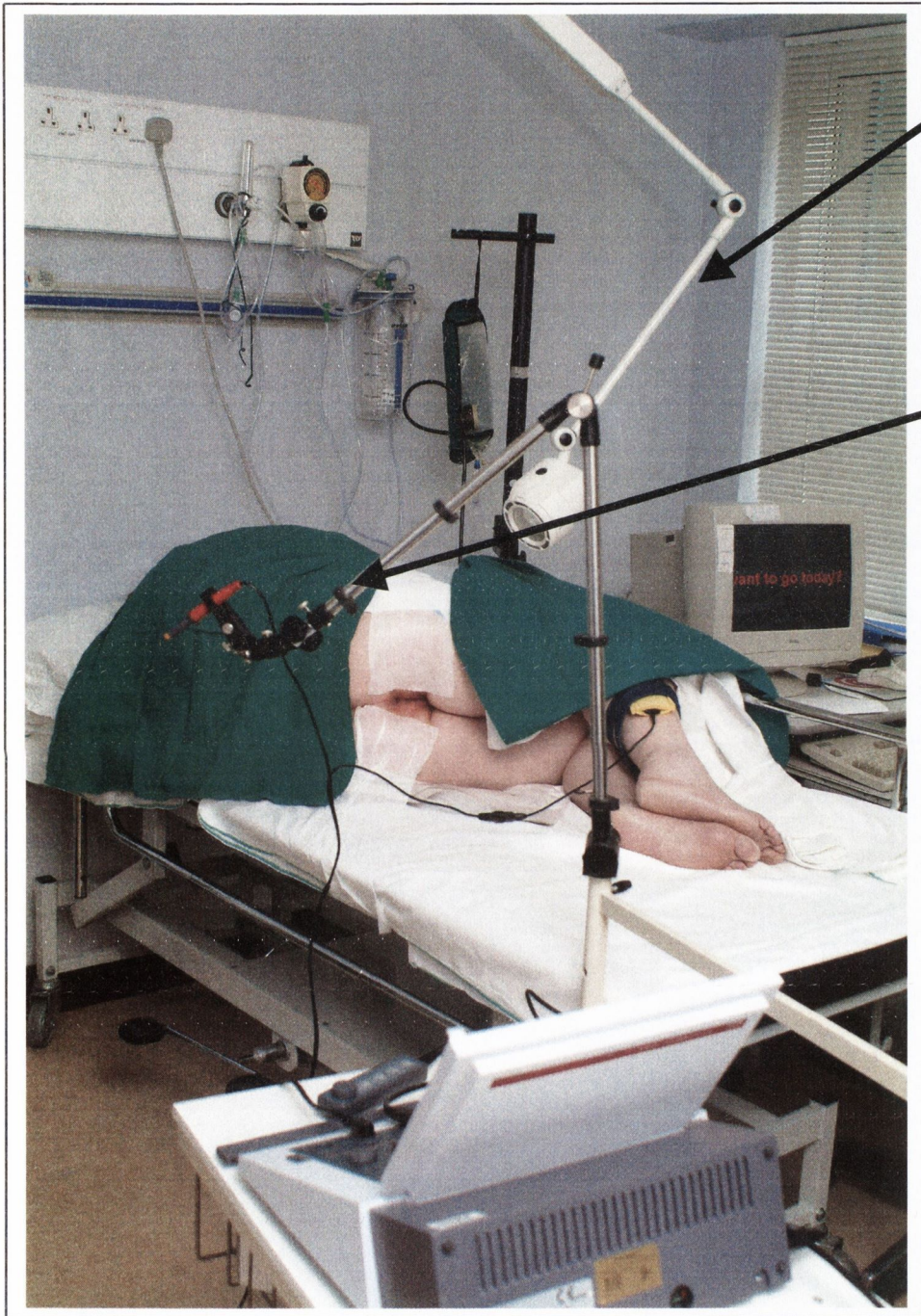
Figure 4.2: Retraction of the buttocks for SDT

2. We also discovered that with the hand-held electrode, it was impossible to maintain a constant pressure at the point of contact between the electrode and the perianal skin. It has previously been determined that pressure variations during the procedure was the commonest source of faulty results (Waldsworth and Chanmugam 1988; Monk, Mills et al. 1998). To rectify this problem, we designed a 'versatile electrode holder' (Figure 4.3). This holder has a wide range and angle of movement, and is attached to a motorised trolley, which houses the monitor and current

pulse generator (Myomed 932, Enraf Nionus, Rontgenweg, The Netherlands®). The main advantage of this compact unit is that once the stimulating electrode point position is set, it is maintained throughout the test and this eliminates the errors that are introduced into the procedure by the hand tremors and variation in pressures. It also has the added advantage of ease of transportation to bedsides outside the pelvic floor physiology laboratory.

3. The availability of a good light source that can be moved around for optimal visibility is also essential for observing the fine muscle contraction (Figure 4.3).

The procedures of PNTMLT and SDT were explained to the patient. We made the patient aware that one of them was still at an experimental stage but was blinded to which of them was the experimental test. At the end of the procedures, the patient was made to score both test using a visual analogue scale of 0-10 (0 being the most uncomfortable and 10, the most comfortable). They were also asked to comment on both test and to give reasons for their scores.



Light source

Electrode holder

Figure 4.3: Versatile electrode holder on a motorised trolley and light source

After positioning the patient in the left-lateral position, the buttocks were retracted with adhesive tape to expose the perianal region. A rubber electrode (6x8cm/4mm female, Enraf Nionus, Rontgenweg, The Netherlands®) encased in a moist pad, is strapped to the patient's right leg with an elasticised Velcro strap. The perianal skin is wiped dry and the stimulating electrode is positioned on the perianal skin at 3 o'clock with the aid of the versatile electrode holder (Figure 4.4).



Figure 4.4: Positioning of electrode

Patients were excluded from the test if there was evidence of perianal skin cracks or excoriation. An electrical impulse starting with pulse duration of 100 milliseconds (ms) is applied and the current intensity needed to elicit the minimal perceptible contraction, is noted and expressed in milliamperes (mA).

Two observers and the operator observed for the anal sphincter twitch and they were blinded to the current intensity needed to evoke the stimulus. The muscle twitch is accepted as a response if there is agreement between the operator and the observers. The operator and two observers were the same for all the procedures.



Figure 4.5: Myomed 932 Enraf Nionus

The procedure is repeated with progressively shortening pulse durations of 70, 50, 30, 20, 10, 5, 2, 1, 0.5, 0.2, 0.1 and 0.05 ms; and at the completion of the test, Myomed 932, Enraf Nionus (Figure 4.5) automatically generates the strength-duration curve (SDC). From the SDC generated, the following can be derived (Figure 4.6):

1. Rheobase: The minimum current intensity required to elicit a response in the form of anal sphincter contraction.
2. Chronaxie: The time required to obtain a sphincter response with current intensity of twice rheobase on the curve.
3. Utilisation time: The time at which the curve begins to plateau.

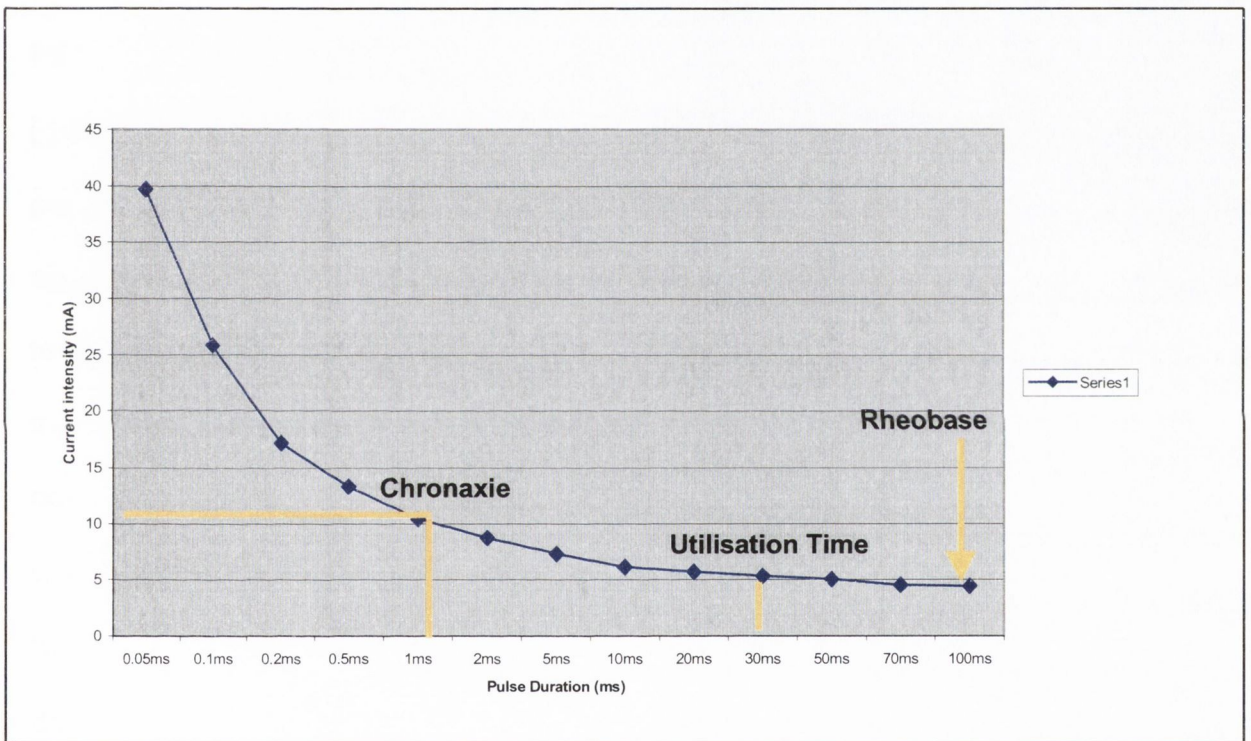


Figure 4.6: Strength Duration Curve Showing Rheobase, Chronaxie and Utilisation Time

4.4 Statistical Analysis

Statistical analysis was performed using the software package (SPSS 10.0 for windows®, SPSS Inc. Chicago, Illinois, USA). All results are expressed as means \pm Standard Deviation (SD) unless otherwise stated. Where appropriate, parametric statistical methodology was employed. One-way Analysis of Variance (ANOVA) was used to compare multiple group means. Pearson correlation and

Kendall's tau correlation tests were applied to test for correlation in parametric and non-parametric data respectively. Where applicable the Student t-test was used to compare means. A P-value of less than 0.05 was considered to indicate statistical significance.

4.5 Results

Fifty patients volunteered to have SDT and the test was not completed in 7 (14%) patients (5 patients could not tolerate the SDT, 1 patient did not show any perceptible contractile response even at very high currents, and 1 procedure was abandoned because the patient had generalised body tremors that rendered the test unreliable). Forty-three patients were therefore included in this study and of these, 23 presented with faecal incontinence, and the other 20 patients were continent patients presenting with constipation. The mean age of the study group was 53.3 ± 14.6 years, and mean parity of 3 ± 3 . Forty (93%), of the patients have had previous vaginal delivery and none of them had evidence of sphincter disruption on endoanal ultrasonography.

All the patients had pudendal nerve studies of which, 15 (35%) had normal pudendal nerve latency test, 7 (16%) had bilateral neuropathy, 3 (6.5%) had right-sided mononeuropathy, and 18 (42%) had left sided mononeuropathy. The mean PNTMLT for the faecal incontinence group was 2.42 ± 0.2 (2.1 ± 0.3 , and 2.7 ± 0.8 , for the right and left side respectively); and the mean PNTMLT for the constipation group was 2.1 ± 0.2 (1.9 ± 0.4 and 2.3 ± 0.3 , for the right and left side respectively).

The findings of anorectal manometric studies performed in incontinence and constipation subjects are as shown in Table 4.1. Using the visual analogue scoring scale, the mean score for SDT was 7.4 (median = 8, range = 4-10) and PNTMLT was 3.6 (median = 3, range = 0-7). Their difference in satisfaction rating was statistically significant (T-test, $P=0.0001$), and showed SDT to be a more tolerable test than PNTMLT. The patients' comments at the end of the test described PNTMLT, as cruel, undignified, dehumanising, degrading and invasive; while SDT was described as time consuming, more dignified, less uncomfortable and less invasive. The average time for equipment set-up for the two procedures was similar, but SDT and PNS took about 20 minutes and 8 minutes respectively to perform. To test for reproducibility of the SDT technique, the test was repeated in 35 patients and their means were compared using the student t-test. There was no significant difference in any of the pulse durations (Table 4.2).

Table 4.1: Manometric data and incontinence scores of the faecal incontinence and constipation subgroups (Mean \pm SD)

Parameters	Faecal incontinence (N = 23)	Constipation (N = 20)
Maximum resting pressure (in mmHg)	46.0 \pm 24.1	58.1 \pm 19.9
Maximum squeeze pressure (in mmHg)	52.6 \pm 26.3	62.6 \pm 26.3
Anal canal length (in cm)	1.9 \pm 1.3	1.9 \pm 0.9
First sensation (in ml)	80.2 \pm 42	68.9 \pm 29.5
Call to stool (in ml)	178.8 \pm 54.6	191.1 \pm 68
Urgency of defecation (in ml)	197.1 \pm 57.6	202.2 \pm 69.4
Peak Amplitude of maximum sustained squeeze	53 \pm 28	
Regression Amplitude of maximum sustained squeeze	44.7 \pm 27.4	
Duration of maximum squeeze amplitude	13.6 \pm 4.4 secs	
Vaisey Incontinence score (Max.=24)	10.6 \pm 3.2	
Wexner Incontinence score (Max.=20)	9.74 \pm 3.8	
Faecal Incontinence Quality Of Life score (Maximum = 119)	64.06 \pm 20	

Table 4.2: Comparison of the First and Second Strength Duration Tests
(Repeatability Test)

<u>Pulse</u> Duration (ms)	First SDT Mean Intensity mA (SD)	Second SDT Mean Intensity mA (SD)	N	P (Student t-test)
0.05	39.54 (18.10)	39.29 (18.14)	32	0.96
0.1	26.05(11.50)	25.05 (11.53)	30	0.74
0.2	17.2 (8.29)	15.90 (8.9)	29	0.57
0.5	13.35 (5.62)	13.16 (5.39)	33	0.99
1	10.38 (3.63)	10.23 (3.16)	34	0.86
2	8.66 (3.22)	8.60 (2.89)	30	0.94
5	7.06 (2.64)	6.70 (2.44)	35	0.56
10	5.96 (2.34)	5.68 (2.03)	35	0.59
20	5.75 (2.19)	5.34 (1.91)	29	0.45
30	5.27 (2.00)	4.61 (1.62)	35	0.13
50	5.04 (2.04)	4.55 (1.57)	34	0.27
70	4.50 (1.8)	4.0 (1.38)	31	0.23
100	4.48 (2.09)	4.13 (1.75)	35	0.44

4.5.1 Relationship of pudendal nerve studies and strength duration test

Applying Pearson's correlation test, a strong correlation was found between right pudendal nerve latency test and SDT at pulse durations of 0.05ms, 0.1ms,

0.2ms, 0.5ms and 2ms (*P-values* = 0.02, 0.01, 0.00, 0.02, 0.001 respectively)

(Appendix 3).

4.5.2 Relationship of anorectal manometry and strength duration test

To assess this relationship, the anal sphincter pressure component (Maximum squeeze pressure, Maximum resting pressure, the peak and regression amplitude of maximum squeeze pressure and duration of maximum squeeze) and the rectal sensory component (first rectal sensation to distension, urgency of defecation and maximum tolerated volume) were evaluated. Significant correlations were demonstrated between these parameters and SDT, at varying pulse durations (Table 4.1).

a) Anal sphincter pressure component

At pulse duration of 20ms, 30ms, 50ms, 70ms and 100ms, SDT correlated with:

- Maximum squeeze pressure ($p=0.02, 0.01, 0.06, 0.04$ and 0.02 respectively).

At pulse duration of 70ms and 100ms, SDT correlated with:

- Maximum resting pressure, $P = 0.05$ and 0.03 respectively

Utilisation time correlated (Kendall's tau correlation test) with:

- Peak amplitude of maximum squeeze pressure ($p=0.02$)
- Regression amplitude of maximum squeeze pressure ($p=0.02$)
- Duration of maximum squeeze ($p=0.04$)

- Pearson's correlation test showed significant correlations between utilisation time versus right and left pudendal nerve latencies ($P = 0.048$ and 0.039 respectively) (Appendix 4).

b) Anorectal sensory component

At pulse duration of 0.05ms, 0.1ms, 0.5ms and 1ms, SDT correlated with:

- First rectal sensation to distension, $p=0.04$ (at 0.5ms)
- Sensation of urgency of defecation, $p=0.05, 0.04, 0.01$ and 0.03
- Maximum tolerated volume, $p=0.04, 0.03, 0.01$ and 0.03

4.5.3 Relationship between strength duration test and faecal incontinence quality of life score

Using the Rockwood disease specific faecal incontinence quality of life scoring system (FIQOL), correlation was demonstrated between:

FIQOL and Utilisation time ($p=0.04$)

FIQOL and SDT at pulse durations of 50ms, 2ms, 1ms, 0.5ms, 0.2ms, 0.1ms and 0.05ms, ($P=0.03, 0.05, 0.02, 0.03, 0.01, 0.01, 0.006$ respectively).

To check for presence of cross innervation of the pelvic floor, the strength duration curves (SDC) on the right and left sides were compared in 12 individual patients. The tests on both sides were performed concurrently, allowing just enough time intervals for the repositioning of the peri-anal electrode. The curves obtained by plotting the current intensity against the pulse duration in individual patients were analysed using the analysis of variance (ANOVA). Statistically

significant difference between the right and left SDCs was found in 50% of the patients (Appendix 5). The severity of pudendal neuropathy did not influence the SDC obtained for each patient. Comparison of the SDCs of the subgroups of patients (faecal incontinence and constipation, Figure 6) did not show statistically significant difference ($P = 0.493$).

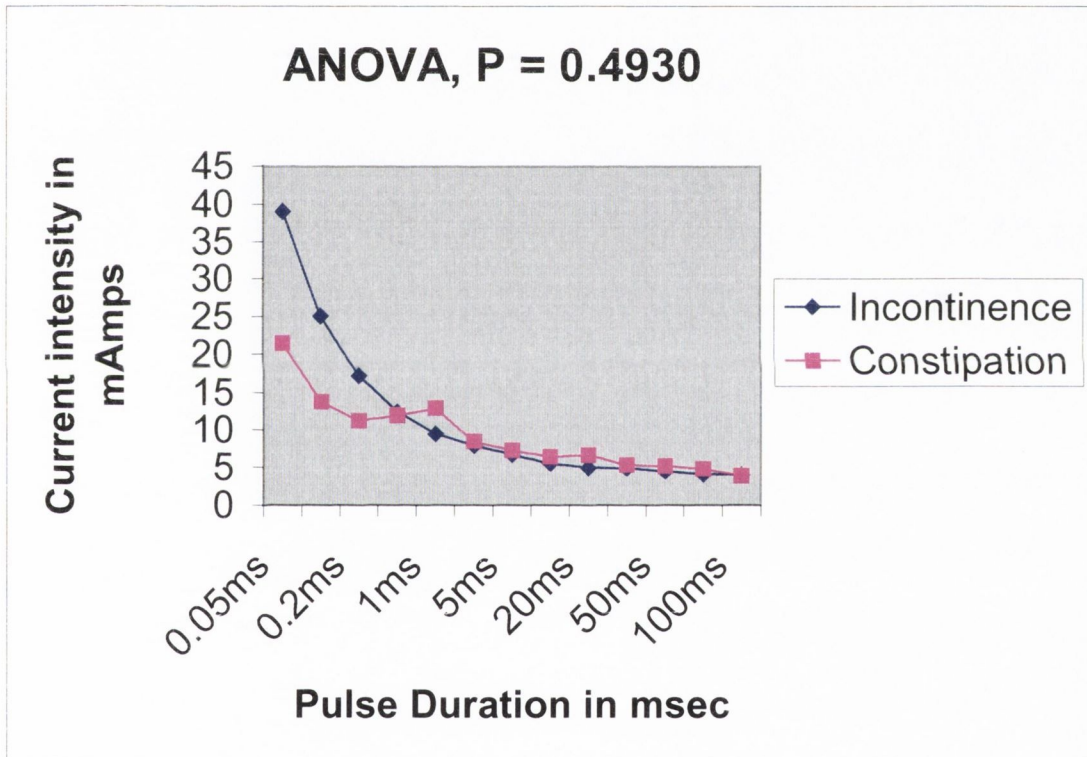


Figure 4.7: Comparative strength duration curves of the various sub groups using the ANOVA

Using the model of Monk et al, logistic regression was performed for the faecal incontinence and constipation subgroups (Monk, Mills et al. 1998). Parameters used for the calculation were the resting and squeeze pressures, and the current intensity at 1ms (I):

$$[\text{logit}(P) = 4.1605 - (0.0559 \times \text{squeeze pressure}) - (0.1755 \times \text{resting pressure}) + 0.8622 I_{\text{tms}}]$$

Where I_{tms} is the current required to produce a contraction at 1ms.

The $\text{logit}(P)$ for the faecal incontinence and constipation subgroups was +0.344 and -0.581 respectively. A negative score indicates normality and a positive score denotes faecal incontinence.

Analysis of the characteristics of patients' contractile response to stimulation showed that 15 patients had normal contractions, 9 patients responded with brisk contractions, which were poorly sustained, 9 had sluggish contractions, and 4 had hyperactive contractions that involved the contraction of the gluteus muscle on the ipsilateral side. Six patients experienced pain on stimulation but the pain was not severe enough to cause discontinuation of the test.

4.6 Discussion

This study has shown that with modification and standardisation of the methodology of SDT, significant correlation can be demonstrated between it and other physiological measurements of pelvic floor function. Correlations relating to the neural components of the anal sphincter (e.g. pudendal nerve latency and

rectal sensation) were observed with shorter pulse durations, while those relating to muscle function (e.g. resting and squeeze pressures), were found with pulses of longer durations. This is expected because nerves respond to currents of short durations while the muscle component respond to longer duration electrical impulses. Applying this knowledge, it may be possible to determine if the anal sphincter dysfunction is neural, muscular or a combination of both in origin.

The aim of using the visual analogue scale to compare SDT with a validated test such as PNTMLT was to determine its tolerability. A significant correlation was demonstrated between pudendal nerve latency test and the various points on the hyperbola of the SDC. This would suggest that worsening pudendal neuropathy would result in the requirement of increased current intensity to elicit a response in the SDT.

While the maximum squeeze and resting pressures correlated with pulse duration values of SDT, the utilisation time, which is derived from the strength duration curve also correlated with parameters such as the peak amplitude, regression amplitude and the duration of maximum squeeze. In a previous study by Sangwan et al (Sangwan, Collier et al. 1995), it was concluded that manometric parameters fail to indicate response to biofeedback therapy. However, in our chapter on biofeedback, we have demonstrated measurable manometric parameters, which can be used to objectively assess response to biofeedback therapy and this includes the defecation deferral time. Manometric parameters such as the peak and regression amplitude and the duration of

sustenance of maximum squeeze have a direct relationship to defecation deferral time. It is hoped that targeting an improvement in the utilisation time of the anal sphincter muscle either by way of biofeedback or sacral nerve stimulation may help in the monitoring and tailoring these treatment modalities to individual patients. Since the utilisation time also has a direct relationship with the disease specific, faecal incontinence quality of life score (Rockwood, Church et al. 2000), an improvement in utilisation time should serve as an objective parameter in assessing symptomatic improvement.

Using the logistic regression model as described by Monk et al it was possible to discriminate between continent and incontinent patients. It must be stated here that our control continent group comprised of patients with constipation. This group of patients were used because of the difficulty in recruiting normal asymptomatic females for this study; and we know that external anal sphincter abnormalities are pertinent to incontinence only. Monk et al also suggested that since they detected significant correlations at specific pulse durations, the whole SDC might not be necessary to discriminate between the controls and faecal incontinence patients. However, in our study, we found significant correlations across the spectrum of pulse durations and the study of the complete curve might offer further understanding of the sphincter characteristics and also offer further information such as the chronaxie, rheobase and utilisation time.

The patient who did not show any perceptible response to electrical stimulation was observed to have normal pudendal nerve terminal motor latency test.

However, her manometric parameters (resting and squeeze pressures) were poor and this combined with the SDT indicates poor prognosis. This case highlights the problem that may be encountered occasionally, when using PNS as the only test for neurological evaluation. In our study, 5(10%) patients could not tolerate SDT because of the discomfort. This number was much higher than previously reported (Monk, Mills et al. 1998; Mills, Hosker et al. 2002); and most of the patients that could not tolerate the test also had delayed pudendal latency tests. Thus while this test is generally more acceptable than the PNS as shown by the visual analogue scores, the expression of pain or discomfort in itself may be an indication of the pathology.

The outcome of SDT performed bilaterally did not prove or disprove conclusively the concept of cross enervation as can be seen in appendix 2. Our hypothesis was that if cross innervation was present, there should be no significant difference between the SDC on both sides of each patient, but the results for and against the hypothesis were evenly distributed. This finding is also similar to what obtains in pudendal nerve mononeuropathy where a proportion of the patients remain continent despite severe neuropathy on one side and vice versa. Further studies in this regard and with a larger population, might clarify this issue.

Our present study has demonstrated more correlations than the two previously published studies (Monk, Mills et al. 1998; Mills, Hosker et al. 2002). This could be attributed to the modifications we introduced in our technique of SDT. It is

known that one of the commonest sources of error in this test is the pressure variation caused by the hand held stimulating electrode. With the use of the 'versatile electrode holder' and the strapping of the buttocks, we have eliminated manual contact with the patient. As has been suggested by Mills et al, there is a need to develop an equipment, which is capable of detecting the muscle twitch response, rather than relying on human observation (Mills, Hosker et al. 2002). In our study, we have attempted to reduce the error arising from this by ensuring that there was agreement between the operator and the two observers before a muscle twitch can be accepted as the response.

Strength duration test offers us the ability to observe the characteristics of the anal sphincter muscle in response to stimulation. Some patients respond briskly while others respond with sluggish muscle contraction, which ceases after a few contractions; this may indicate easy fatigability and patients that are unlikely to respond to therapy. The clinical significance of rheobase, chronaxie and utilisation time derived from the strength duration curve is not clear at present, but it is hoped that further studies in this area might enable us to apply this information to individualise and tailor treatment regimen such as biofeedback and electro-stimulation therapy according to the severity of patients' symptoms. The character of the anal sphincter contraction may also be useful in determining the area of therapy that should be focused on e.g. those with brisk contraction will need more endurance therapy; and the rheobase and utilisation time may help in setting the current intensity at which therapy should commence and for

how long each treatment session should be. This we hope will optimise patients' response to treatment.

4.7 Conclusion

The use of strength duration test to assess anal sphincter function is still at an experimental phase. This test however has the potential of assessing the neuromuscular complex of the anal sphincter with tolerability levels that compares favourably with other established tests such as pudendal nerve terminal motor latency test and needle electromyography. The test is easy to perform and with our modification of the procedure, the attainment of the learning curve is easily achievable. Accrual of further data and long-term follow up of the patients will help to better clarify its role as an investigative tool.

CHAPTER 5

OBJECTIVE OUTCOME MEASURES IN ELECTROMYOGRAPHIC BIOFEEDBACK IN THE TREATMENT OF FAECAL INCONTINENCE

5.1 Introduction

Faecal incontinence (FI) is estimated to affect between 7% and 18% of the general population (Jorge and Wexner 1993; Vaizey, Kamm et al. 1998). It is a debilitating and often underreported condition (Johanson and Lafferty 1996). The therapeutic options for restoration of continence in these patients are largely dependent upon their underlying aetiology. Patients with external anal sphincter disruption secondary to trauma, whether obstetric related or otherwise, do benefit from early surgical intervention and delayed or repeated sphincter repair has been shown to have reasonable outcome. However, in a significant proportion of patients whose anal sphincter muscles are determined to be intact, therapeutic options are limited and surgical management is not successful in treating the underlying aetiology. For almost 30 years biofeedback has been used in the treatment of incontinence, which is not amenable to surgical intervention (Heymen, Jones et al. 2001). Previously published data report a subjective improvement in patient's overall symptoms with a reported efficacy ranging between 40% and 100% (Rao, Enck et al. 1997).

Biofeedback aims to improve the ability of the patient to voluntarily contract both the external anal sphincter and puborectalis muscles in response to rectal filling.

This is achieved by improving the contraction strength of the pelvic floor musculature, increasing patients' perception of rectal sensation or by a combination of these parameters (Heymen, Jones et al. 2001). Two commonly used techniques are routinely employed. In the first, a balloon is positioned in the rectum and the patient is taught to contract the external anal sphincter in response to sensation of rectal distension.

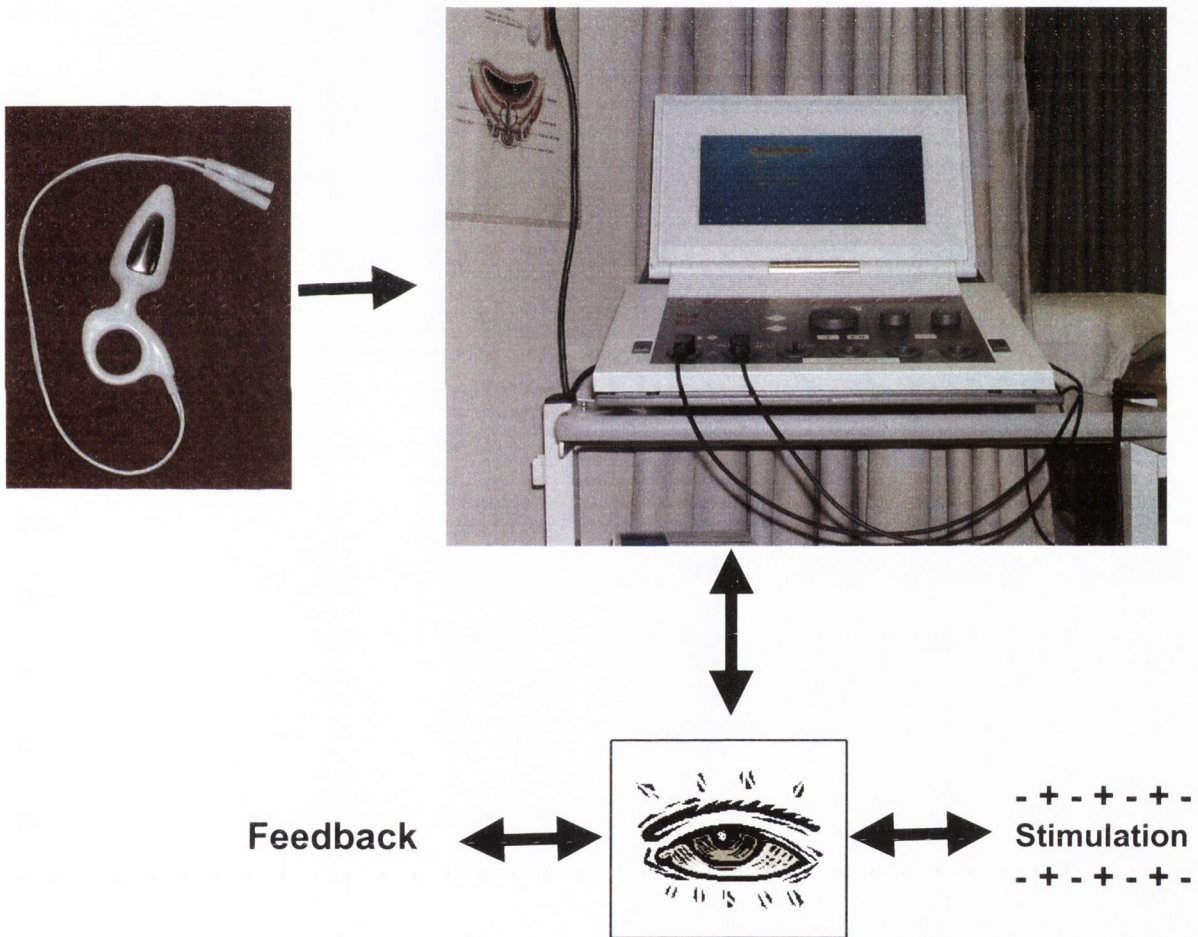


Figure 5.1: Biofeedback improves strength and coordination of anal sphincters

This volume is progressively reduced by decrement of 10ml, and it is aimed at reducing the threshold of sensibility of the rectum. (Berti Riboli, Frascio et al.

1988; Glia, Gylin et al. 1998). The other technique is that of Electromyographic (EMG) biofeedback in which a surface EMG sensor (Figure 5.1) is placed in the anal canal or adjacent to the anus (transvaginally) to provide readings of EMG activity of the pelvic floor muscles (Rieger, Wattchow et al. 1997).

In a further modification of this technique, the use of standard EMG biofeedback has been combined with active stimulation of the anal sphincters. This technique has been shown to augment the effectiveness of biofeedback (Fynes, Marshall et al. 1999).

The employment of biofeedback therapy has not been widely reported in the treatment of patients with faecal incontinence of diverse aetiologies. In addition, most of the improvements demonstrated in faecal incontinence using biofeedback techniques have been seen with subjective assessment parameters. In this study, we have retrospectively assessed the efficacy of electromyographic biofeedback in a population of patients with FI due to different aetiologies. Their response to treatment was documented, using both subjective and objective validated scoring systems and anorectal manometry.

5.2 Materials and Methods

5.2.1 Patient cohort

Our study population comprised of patients assessed in our colorectal clinic with a presumptive diagnosis of FI between June 1999 and June 2002. Patients were selected for this study on the basis that they had faecal incontinence for greater

than 6 months and were not amenable to surgical intervention. They were highly motivated and had normal pudendal nerve terminal motor latency test at least on one side. We excluded patients that had bilateral pudendal nerve neuropathy and those considered unable to undergo training due to limited comprehension or intellectual capacity. In total twenty-eight consecutive patients were included in the study. Patients underwent a full medical and clinical assessment to determine the underlying aetiology for their incontinence. Patients were evaluated using both subjective and objective scoring systems. As part of their diagnostic work-up for faecal incontinence, they underwent colonoscopy, anorectal manometry, endoanal ultrasonography and pudendal nerve terminal motor latency testing. Patients were then referred for biofeedback therapy.

5.2.2 Electromyographic Biofeedback therapy

A single specialist pelvic floor physiotherapist administered the biofeedback program. She obtained an initial history and performed an examination to determine the pre-therapy pelvic floor muscle strength, using the modified Oxford Scale. The modified Oxford scale measures the muscle strength by bimanual palpation during the maximal voluntary contraction of the pelvic floor muscles. The score is from 0 to 5. A score of 0 indicates no discernable muscle contraction. A score of 5 indicates strong resistance against the examining finger. Perineometry is considered a highly effective measurement of pelvic floor contraction strength, Isherwood et al showed good correlation between the Oxford score and perineometry in the assessment of pelvic floor strength. The Oxford score measures muscle strength by bimanual palpation during maximal

voluntary contraction of the pelvic floor (Isherwood and Rane 2000; Laycock and Jerwood 2001). All patients were given an explanation of the relevant anatomy and muscle function of the pelvic floor with emphasis on the external anal sphincter complex. They were all instructed in selective pelvic floor muscle recruitment and exercise with particular emphasis on the anal muscles, using visual aids and verbal explanation. Patients with an Oxford score of \geq Grade 3 commenced a muscle strengthening exercise program using EMG biofeedback.

The EMG biofeedback technique involves placement of an intra-anal surface EMG sensor, which detects the activity of the anal sphincter muscles. This electrical activity is converted to an audible signal, which the patient can also view on a monitor. The biofeedback unit used was the Myomed 398, (Enraf Nonius, Netherlands) and the intra anal device (Anuform™), which consist of a surface electromyographic sensor and stimulating electrodes (NEEN Healthcare, Norfolk, England). For patients that could not tolerate the intra anal probe because of intense sensation, an alternative intra vaginal probe (Periform™) was used (NEEN Healthcare, Norfolk, England). Patients were treated in supine, sitting and standing position as their anal muscle strength improved. The patients were reviewed at 1 to 4 weekly intervals for monitoring of treatment and to assess progress. If the modified Oxford score was poor (less than Grade 3), then augmentation muscle stimulation was also included in their program. Muscle stimulation at 35Hz with pulse duration of 250 μ s, as recommended by Laycock, was applied using the intra-anal stimulating electrode. This protocol includes a repetition cycle of 2 seconds ramp up time, which is sustained for 8

seconds and then followed by a ramp down time lasting for 1 second. A rest interval of 10 seconds is allowed between each cycle.

Patients that had a modified Oxford scale score of grade 0-1 used a home muscle stimulator on a daily basis for 4–6 weeks. All patients started with 5 minutes duration of treatment and progressed up to 15 minutes. Patients who scored a grade 2 were treated once weekly for 3-4 weeks. For patients who improved to a score of grade 3 they were commenced on biofeedback therapy. The remaining patients with a grade 2 modified Oxford score were also commenced on biofeedback therapy to optimise their limited anal sphincter function. Patients were instructed to perform a customized pelvic floor exercise program three times a day at home for the duration of therapy.

5.2.3 Quality of Life assessment

To assess the impact of faecal incontinence on patients' life-style, each patient completed a standard quality-of-life (QOL) scoring card (Carol Andrejasich, Ely Lilly). This subjective scoring system rates from 0 to 120, the higher the score the better the quality-of-life. Patients were required to respond to this QOL questionnaire before and after the biofeedback program. The formal scoring of our patients QOL was introduced as part of our standard evaluation protocols shortly after the initial patients were treated. As a result, not all the patients in our study had pre and post biofeedback QOL scores available for analysis.

5.2.4 Incontinence assessment

Patients were assessed with the validated Vaizey and Wexner faecal incontinence scoring systems. The Vaizey incontinence score is a twenty-four-point measure of faecal incontinence. Patients with a score of zero are considered to have normal continence (Vaizey, Carapeti et al. 1999). Fifteen minute deferral of evacuation after initial sensation forms part of the Vaizey score and is considered to be a significant index of a patient's continence. The Wexner score is a twenty point assessment of faecal incontinence (Jorge and Wexner 1993). Patients with a score of zero are considered to have normal continence.

5.2.5 Anorectal Manometry

Anorectal manometry was performed on all patients who presented with FI, using a four-channel water perfusion catheter (MUI Scientific 4 channel catheter-Code 9012P2301; Medtronic Synetics Anorectal Manometry Analysis Module Version 2.0 using Polygram for windows function testing software, Synetics Medical, Stockholm, Sweden). The procedure was performed in a standard fashion, using the stationary pull through technique (Stendal 1997).

5.2.6 Endoanal Ultrasound

Endoanal ultrasound was performed using the Bruel and Kjaer (B&K) Medical 2002 Panther© ultrasound machine with a radial endoscopic probe and a 10MHz (Code number 6004) transducer. The internal and external anal sphincters were assessed in a standard fashion.

5.2.7 Pudendal Nerve Terminal Motor Latency

Pudendal nerve terminal motor latency test (PNTMLT) was performed in the standard fashion to assess the integrity of the pudendal nerves (St. Marks pudendal stimulating electrode; Dantec Medical, Denmark). The normal reference value used was 2.0 ± 0.2 ms and latency periods greater than 2.2 ms were regarded as abnormal (Stendal 1997).

5.3 Follow up

All patients were reassessed with the subjective and objective scoring systems; and anorectal manometry at 6 to 18 months post EMG biofeedback therapy.

5.4 Statistical analysis

All data were stored on an IBM-compatible Microsoft Excel spreadsheet. Statistical analysis was performed using the software package (SPSS 10.0 for windows®, SPSS Inc. Chicago, Illinois, USA). Data is generally expressed as means (range) unless otherwise stated. Differences in nonparametric data sets were examined using Mann-Whitney-U test. A P-value of less than 0.05 was considered to indicate statistical significance.

5.5 Results

5.5.1 Patients

Twenty-eight females were referred for EMG Biofeedback therapy. The mean age of the study group was 49.2 (range, 31-70) years. The mean duration of symptoms before therapy was 43 (range, 7-120) months. Thirteen patients were post-obstetric injury (4 patients with parity greater than 5; 5 patients had multiple forceps delivery; and 4 patients sustained 3rd degree perineal tear), 11 had idiopathic faecal incontinence and 4 were post-surgical trauma (3, anterior resection; 1 repair rectovaginal fistula). There was no significant difference in the duration of the symptoms between these patients however; the mean age of those with idiopathic faecal incontinence was significantly higher (Table 5.1). The four patients lost to follow up were excluded from the analysis. In total twenty-four patients completed the therapy. The mean number of sessions was 6.4 and the median follow up is 18 (range, 5-30) months. In total 21 patients are currently more than one-year post biofeedback therapies.

Table 5.1: Patient Cohort

	No. of patients	Age (years) (range)	Mean Duration of symptoms (months) (range)
Postobstetric Injury	13	46.6 (range 31-68)	44.4 (8-120)
Idiopathic	11	43.6 (range 39-58)	43.2 (10-96)
Postsurgical intervention	4	59 (range 54-70)*	39.6 (12-96)

*p value considered significant

5.5.2 Quality of Life Data

The Quality of life was assessed in 13 of the 24 patients. The mean quality of life score pre-biofeedback was 62 (range 38-100) and post-biofeedback therapy was 77 (range 53-106). This represented a significant improvement in the patient's quality of life ($p < 0.01$; Mann-Whitney).

5.5.3 Vaizey and Wexner scores

The Vaizey and Wexner scores were assessed in 24 patients. The mean Vaizey score pre-biofeedback therapy was 13.7 (range 9-18) and post-biofeedback was 9.5 (range 3-16). This represented a significant improvement in patient continence ($p < 0.001$), Mann-Whitney (Figure 5.2). In total, 80% of patients had significant improvement in their Vaizey score. With regard to defecation deferral

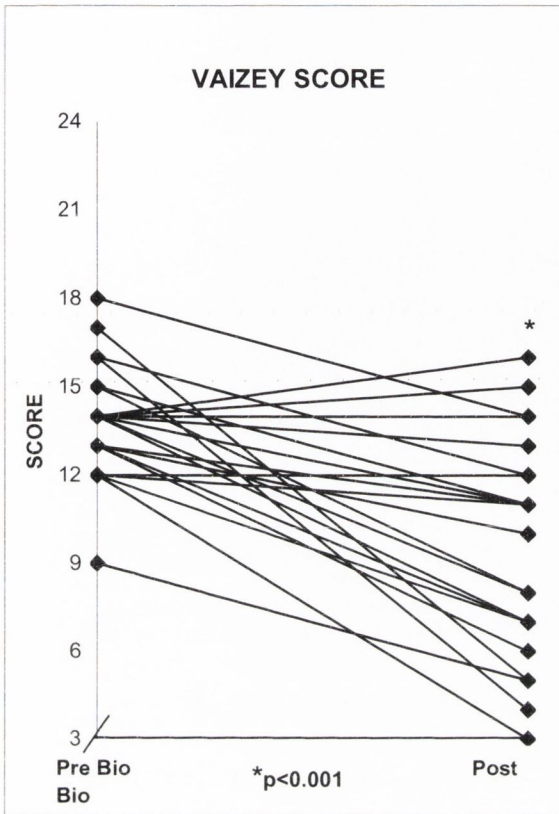


Figure 5.2: Vaizey score before and after EMG biofeedback.

time, the number of patients who could defer defecation by fifteen minutes increased from two pre-biofeedback to fourteen patients post-biofeedback therapy (Figure 5.3). This improvement was statistically significant ($p<0.001$) (Table 5.2). The mean Wexner score pre-biofeedback was 12.3 (range 7-16) and post-biofeedback was 8.7 (range 2-17). This again represented a significant improvement in the patient's continence levels ($p<0.001$), Mann-Whitney (Figure 5.4).

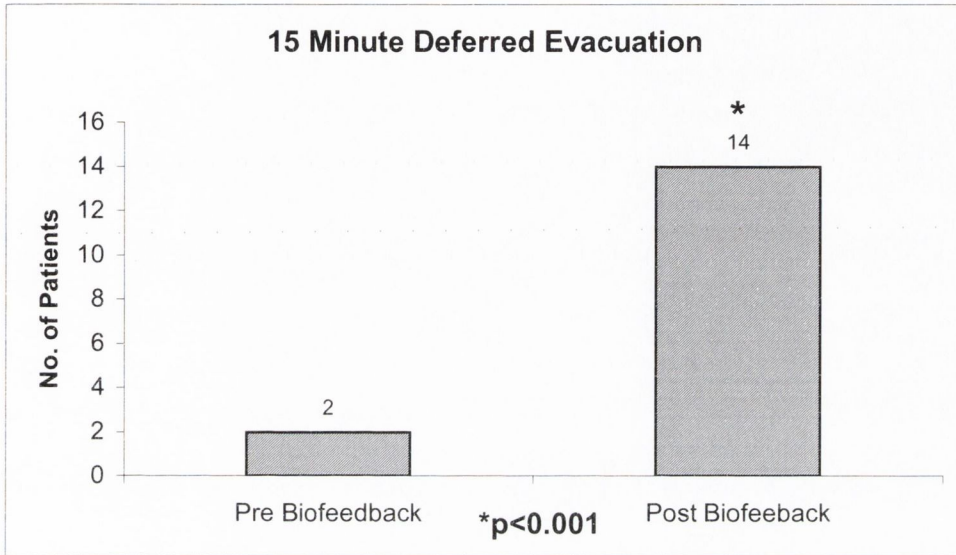


Figure 5.3: Fifteen-minute deferred evacuation before and after EMG biofeedback.

5.5.4 Anorectal Manometry

All the twenty-four patients in the study underwent anorectal manometry prior to EMG biofeedback therapy. Subsequently, 18 patients had a follow-up anorectal manometry at the completion of EMG biofeedback therapy. Of the six patients that were not re-assessed; four-refused reassessment and two declined on medical grounds. There was a significant improvement in the resting pressure, duration of the squeeze and amplitude of the squeeze post EMG biofeedback therapy (Table 5.2). There was however no statistically significant improvement in the squeeze pressure post EMG biofeedback.

Table 5.2: Results before and after EMG biofeedback

	No. of patients	Before Biofeedback (range)	After biofeedback (range)	P value
Scoring System				
Vaizey Score	24	13.7 (9–18)	9.5 (3-16)	< 0.01*
Wexner Score	24	12.3 (7–16)	8.8 (2–17)	< 0.01*
Quality of life	13	61.6 (38–100)	78.9 (55-106)	< 0.01*
15 min deferred evacuation	24	8.3%	54.1%	< 0.01*
Manometry				
Mean Max RP (mmHg)	18	20.3 (10.2-30.5)	26.65 (10-3.6)	< 0.01*
Mean Max SP (mmHg)	18	32.95 (14.7-88.3)	34.1(14.5-79.1)	0.3
Duration of Squeeze (sec)	18	13.31 (4.8-19.9)	16.16 (9.9-9.9)	< 0.01*
Peak Amplitude (mm)	18	50.44 (15-124)	62.88 (30-106)	0.05*

Mean Max RP – Mean Max Resting Pressure; Mean Max SP – Mean Max Squeeze Pressure; Duration of Sq – Duration of Squeeze; Peak Amp – Peak Amplitude of Squeeze.

***p value considered significant**

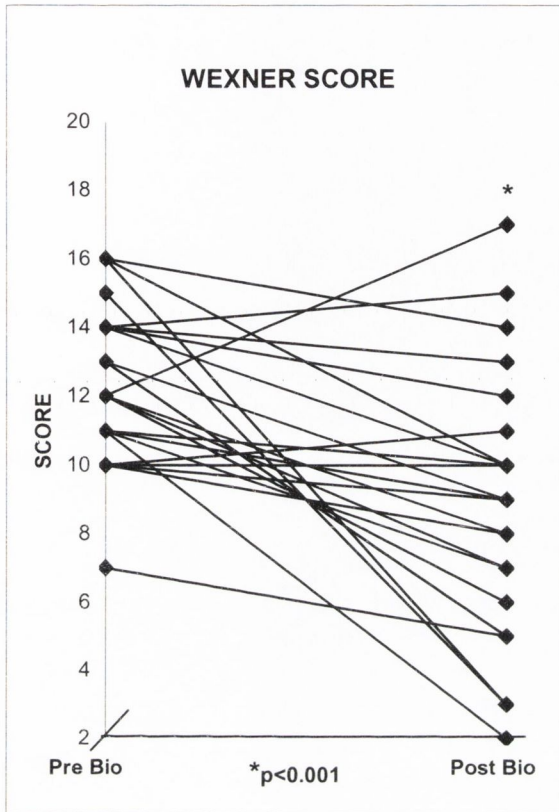


Figure 5.4: Wexner score before and after EMG biofeedback.

5.6 Discussion

Faecal incontinence is often a socially debilitating condition for which there are a number of therapeutic options. These range from simple dietary manipulation and use of pharmacological agents to that of surgical reconstruction and external sphincter stimulation. Surgery remains the optimum treatment in those cases with documented sphincter defects if deemed suitable. However, for patients in whom surgery will not be an effective therapeutic option, the consideration of other treatment modalities is indicated and biofeedback therapy in selected patients has been shown to have results comparable to that of surgery (Keighley, Makuria et al. 1980).

The mechanism of action of biofeedback therapy, though not fully understood, is thought to combine a reduction in the threshold of perceived rectal sensation to distension with augmentation of the contractile strength of pelvic floor muscle. The training protocols employed may be categorised into coordination, sensory and muscle strengthening training components. The EMG biofeedback technique that we employed in our study is designed to improve strength and coordination of pelvic musculature. Our understanding of the exact role, technique and benefit of biofeedback is yet not completely clear. Several units have published data, employing various forms of biofeedback treatment modalities. Improvements in rectal sensory perception and patient perceived continence using a variety of incontinence scoring systems have been reported (Guillemot, Bouche et al. 1995; Rieger, Wattchow et al. 1997; Glia, Gylin et al. 1998; Fynes, Marshall et al. 1999). Critics of the treatment argue that the improvement in continence is predominantly psychological due to the supportive interaction of the biofeedback therapist with the patient. These authors identify decreased anxiety, increased confidence and lack of published long-term follow-up data showing any benefit in these patients as support for their criticism. Furthermore biofeedback therapy has not, until now, been shown to impact on any objective anorectal manometric measurements (Heymen, Jones et al. 2001).

In this initial pilot study, we have assessed our own unit's experience with this technique. The study was not restricted to patients with obstetric injuries alone and our population was representative of mixed aetiologies. Patients were

assessed using the validated Vaizey and Wexner incontinence scoring systems along with a QOL questionnaire and anorectal manometry. Our results have confirmed the subjective improvements seen in other studies using this technique (Heymen, Jones et al. 2001). Our patients recorded an increase in their QOL score, which represented a significant improvement in their overall QOL status. Similar subjective results have been reported in the literature using a variety of QOL scores (Norton and Kamm 1999; Ryn, Morren et al. 2000). We also showed significant objective improvements in the level of continence following biofeedback therapy using the Vaizey and Wexner scoring systems. We further showed objective improvements after biofeedback in the anorectal manometry data. Patients significantly increased their maximum resting pressure, along with an increase in the duration of sustenance of the maximum squeeze. While the peak amplitude of squeeze pressure increased significantly, there was no significant improvement in the maximum squeeze pressure, a finding that is consistent with previously published literature (Guillemot, Bouche et al. 1995; Glin, Gylin et al. 1998).

There were difficulties with initial attempts to demonstrate objective manometric changes following biofeedback therapy. This study, to the best of our knowledge, represents the first objective documentation of manometric improvement after biofeedback in a population of faecal incontinence patients with diverse aetiologies. Factors that tend to negatively impact on the efficacy of biofeedback therapy include evidence of any underlying neurological impairment and a wide spectrum of patient selection (Cerulli, Nikoomanesh et al. 1979; van Tets,

Kuijpers et al. 1996). Our patients, despite having diverse aetiologies, were well motivated and had at least unilateral preservation of pudendal nerve function. The recent study by Fynes et al, using augmented (stimulated) EMG biofeedback focussed upon patients with faecal incontinence after obstetric trauma. They have demonstrated significant improvements in maximum resting pressure, maximum squeeze pressure and incremental squeeze pressure (Fynes, Marshall et al. 1999). In our study, the selection of well-motivated and neurologically intact patients may partially account for some of the subjective improvements seen; and we believe that patient's motivation is essential for compliance and success of this treatment modality. This however should not diminish the significance of the objective manometric findings demonstrated in our patients following treatment. We believe that the standardisation of our treatment protocols, having a dedicated biofeedback physiotherapist; and the initial augmented stimulation of those patients with an Oxford scale score less than grade 3 prior to commencement EMG biofeedback may account for our encouraging results

The objective assessment of biofeedback has suffered from a lack of consensus on treatment protocols and variability in the recording of outcome parameters. There have been many different scoring systems for assessing faecal incontinence and biofeedback therapy many of, which were designed by individual units. The use of the Likert-scale, the Rockwood system, the Pescatori and American Medical System scales have similarly been employed over the past number of years (Browning and Parks 1983; Miller, Bartolo et al. 1988;

Pescatori, Anastasio et al. 1992; Jorge and Wexner 1993; Rockwood, Church et al. 1999; Vaizey, Carapeti et al. 1999). These differences create difficulties in achieving a meaningful comparison between different studies. We are the first group to use both the Vaizey and Wexner incontinence scoring systems to assess our results. These well-validated scoring systems are now widely used in clinical practice, and are specific for the assessment of faecal incontinence. They are specific indicators of the severity of faecal incontinence. We did not highlight the specific number of incontinence episodes as they are built into the scores. Our results have confirmed an improvement in these incontinence scores that is achievable in the short term after biofeedback therapy. This is further emphasized by the significant improvement of the '15 minutes deferred evacuation time' recorded in our patients and this measurement alone must translate into a particular benefit for this group of patients. We would advocate the combined use of these scoring systems when reporting results of biofeedback therapy in the future. We found it difficult to show correlation between our quality of life indices and our objective scoring systems as this subjective element was only built into our incontinence protocol after it was initiated. However as part of the Vaizey and Wexner score there is one question that deals with quality of life and there was a significant improvement in these scores.

In our treatment protocol, we used the modified Oxford score to assess muscle strength thus stratifying the patients into different therapeutic approaches. We did not use this score to assess the response to therapy because of its

subjective component. The gold standard test for sphincter strength is anorectal manometry. It would be interesting in future studies to compare the modified Oxford score with anorectal manometry to determine if any correlations exist. There was good correlation of improvements in anorectal manometry and Vaizey and Wexner scores.

The short term efficacy of biofeedback therapy has been shown to be comparable to that of surgery (Sangwan, Coller et al. 1995). Initial improvements in faecal incontinence following biofeedback therapy are reported as approaching 70% of patients treated (Rieger, Wattchow et al. 1997). This figure is largely applicable to those assessed 12 to 24 months after treatment (Glia, Gylin et al. 1998). There is no doubt that biofeedback therapy is an extremely effective treatment for selected patients in the short term. The permanency of these results however has not been satisfactorily addressed in the literature. Enck et al assessed patients after a five year follow-up period and found no significant decrease in the functional improvements gained (Enck, Daublin et al. 1994). Ryn et al had a median follow-up period of 44 months in their 37 patients. They reported a decrease in the success rate from 60% to 41% over this time period (Ryn, Morren et al. 2000). A similar finding was reported by Glia et al with an initial success rate of 54% decreasing to 41% with time (Glia, Gylin et al. 1998). Other authors have however failed to confirm the benefit of these improvements over time (Whitehead, Burgio et al. 1985; Guillemot, Bouche et al. 1995). In our study, the median follow-up period was 18 months and the results must therefore only be interpreted as further evidence of

the initial successful impact of this promising treatment modality. Further long term prospective studies are therefore required to conclusively determine whether there is indeed a lasting benefit with biofeedback therapy.

The developments of innovative approaches in the treatment of patients with faecal incontinence have indeed progressed apace in recent years. These include stimulated graciloplasty, artificial anal sphincters and sacral nerve stimulation. Graciloplasty was first used in the treatment of end stage faecal incontinence in 1946; additional electric stimulation was introduced almost 15 years ago. Rongen et al reported good long term results with success in over 70% of patients treated however there were frequent complications, dynamic graciloplasty is mostly indicated in patients with end stage faecal incontinence (Wexner, Baeten et al. 2002; Rongen, Uludag et al. 2003). Artificial anal sphincters are a relatively new therapy. The initial reports revealed a high failure rates of over 50% (Christiansen, Rasmussen et al. 1999). With further refinement of techniques, recent reports have had better success. Michot et al reported success in nearly 80% of patient's in their latter series (Michot, Costaglioli et al. 2003). This result is similar to the findings of our study in, which 80% of patients had improvement in their Vaizey score. Both of the above surgical techniques are highly invasive procedures and few patients in our study would meet their inclusion criteria. A promising new technique for the treatment of faecal incontinence is sacral nerve stimulation. It is considered very useful in the treatment of incontinence where there is an internal anal sphincter problem (Malouf, Vaizey et al. 2000).

The benefits of EMG biofeedback therapy are clear in the short term however; we believe that EMG biofeedback is still evolving as a definitive treatment option. The improvements in rectal sensation(Glia, Gylin et al. 1998), rectal squeeze and resting pressures documented by our group and others has demonstrated the clear benefit of EMG biofeedback therapy in selected patients. Its' application as an adjunctive therapy to these exciting innovative therapies however has yet to be addressed but may be the future direction of faecal incontinence amelioration.

5.7 Conclusion

Despite the retrospective nature of this study, when critically assessed and using the validated scoring systems, our data supports the efficacy of EMG biofeedback in the treatment of faecal incontinence. This study also lends itself to the increasing body of evidence showing manometric improvements in anal sphincter muscle strength in patients following biofeedback therapy. Our successful results are indeed reflective of the short-term follow-up period studied, however they do indicate that this treatment may be applicable to a wider spectrum of patients than hitherto recorded. We would therefore suggest that EMG biofeedback should be considered a valuable part of the integrated treatment package for patients with documented faecal incontinence that are not amenable to surgical correction.

CHAPTER 6

GENERAL DISCUSSION AND FUTURE AREAS OF INTEREST

6.1 Discussion

The specialty of pelvic floor dysfunction is fast evolving and there still remain many questions to be answered. For instance, the use of pudendal nerve terminal motor latency testing in diagnosing pelvic floor neuropathy initially was thought to be the panacea but recent evidence is beginning to discount this. Thus while delayed latency may signify neuropathy, a normal latency time does not entirely exclude neuropathy as it is now known that the survival of few fast conducting fibres may reflect normal latency time. This problem highlights the need for a complimentary, if not an alternative neurological test. The use of strength duration test is still in its infancy but it has inherent potentials in this regard and attempts should be made to fully elucidate its physiological importance.

6.2 MRI in monitoring levator ani muscle morphology post therapy

Currently there is no reliable means of assessing the quality of pelvic floor musculature pre and post therapy. We know that exercise or increased stimulation of any muscle will result in hypertrophy and this can easily be assessed in the limb muscles by anthropometrical measurements, however this is not feasible with pelvic floor muscles. The most reliable way of doing this

would be by direct biopsy and histochemical study of the muscle, but there are many ethical issues militating against this. Consequently, there is the need to explore an acceptable imaging modality that is minimally invasive and MRI appears to meet this requirement, with its ability to assess soft tissue density and percentage fat content of muscles. However, the problem we are presently facing is the inability to perform MRI in patients with implanted electrical pulse generator used for sacral nerve stimulation. Having assessed these patients pre-therapy, MRI would have provided an objective qualitative method of monitoring the patients' response to treatment. Perhaps the development of an MRI compatible electrical pulse generator in the future will provide a solution to this problem.

6.3 Effect of therapy on strength duration test

From our study of strength duration test, we have been able to show a relationship between utilisation time and manometric measures of anorectal endurance such as regression amplitude and duration of maximum squeeze. The next phase of this study will be the evaluation of these parameters, pre and post-therapy either by biofeedback or sacral nerve stimulation. We hope that the identification of a consistent pattern in this relationship will define a role for strength duration test.

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APPENDIX

Appendix 1: Proforma of MRI parameters measured

THE PARAMETERS MEASURED IN THE PELVIC FLOOR MRI.

Name:

Chart Number:

Tel:

Age:

Sex: F/M

Date

of

Procedure

1. Urethrovesical angle =

2. Urethral length (Sagittal) =

3. Distance from symphysis pubis to:
 - a. Proximal vagina =
 - b. Distal vagina =

4. Vaginal length
Lateral (Sagittal) =

5. Thickness of EAS
 - a. Anterior
 - a. Posterior =

6. Anorectal angle =

7. Descent of Perineal Body (measured from the pubococcygeal line):

a. Level at straining =

b. Level at rest =

Descent = (a-b)

8. Length of Levator plate (Pubococcygeal distance) =

9. Levator Hiatus (**H-line**)

10. Length of bladder base from PBC line

Resting =

Straining =

Descent =

11. Length of anorectal junction from PBC line

Resting =

Straining =

Descent =

12. Descent of Puborectalis (**M-Line**)

- At Rest =

- Straining =

- Descent =

13. Length of Uterocervical junction from PBC line

Resting =

Straining =

Descent =

14. Position of Uterus: Anteverted Retroverted Neutral

15. Uterine size in cm (long x Transv) =

16. Vaginal vault Descent

17. Length of EAS

b. Lateral =

i. Right =

ii. Left =

18. Length of inner layer of EAS =

a. Right =

b. Left =

19. Angles of the inner layer of EAS =

RT =

LT =

20. Levator-Gluteal Densitometry Ratio

a. Levator:

- Min/Max =
- Mean =
- SD =

b. Gluteaux maximus =

- Min/max =
- Mean =
- SD =

21. Length of inter-sphincteric groove =

a. Right =

b. Left =

22. Length of anal canal (coronal) =

23. Identification of

- Rectocele: Min Mod Severe
- Cystocele Min Mod Severe

24. Levator ani------(RT) (LT)

a. Length =====

b. Thickness =====

25. Levator angles at Rest: RT = LT =

26. Puborectalis

a. Lateral = [RT =]; [LT=]

b. Posterior =

27. Superficial transverse perineal muscle (coronal) =

(RT) (LT)

Length =

Thickness =

28. Thickness of EAS (Axial) =

29. Presence of sphincter defects?

YES.

NO

Appendix 2: Strength Duration Test Report Sheet

PELVIC FLOOR PHYSIOLOGY LABORATORY

ADELAIDE AND MEATH HOSPITAL

TALLAGHT, DUBLIN

STRENGTH DURATION TEST PROFORMA

NAME: -----

D.O.B: -----

HOSPITAL No:-----

SEX: -----

PRESENTING COMPLAINT(S):

RESULTS:

Post Biofeed back assessment. Feels no improvement
SDT = No discomfort 9/10
PNS = Ucomfortable 1/10, sluggish response

Pulse Duration (in Milliseconds-ms)	Current Intensity [LT] (im Milliampere-mA)	Current Intensity [RT] (im Milliampere-mA)
100	5.6	5.2
70	5.4	5.0
50	5.6	4.8
30	5.6	4.6
20	5.7	4.9
10	5.7	5.4
5	5.6	7.8
2	6.2	8.3
1	8.0	9.7
0.5	9.3	12.0
0.2	10.2	19.0
0.1	13.4	27.0
0.05	22.5	48

Appendix 3: Report Sheet for Colonic Transit studies

DEPARTMENT OF SURGERY

ADELAIDE AND MEATH HOSPITAL TALLAGHT, DUBLIN 24

REPORTING SHEET FOR COLONIC TRANSIT STUDIES

Name: -----

MRN: -----

Date of Birth: -----

Sex: -----

	Right Colon	Left Colon	Rectosigmoid	Total Colon
4th Day	8	22	10	40
7 th Day	1	1	3	5
Transit Time	9 hours	23 hours	13 hours	45 hours

NORMAL VALUES

Right Colon = 11.3hrs

Left Colon = 11.3hrs

Rectosigmoid = 12.4hrs

Total Transit Time = 35hrs

NOTE: Delay in left colonic transit leading to marginal delay in total transit time

Appendix 4: Pearson Correlation Coefficients and (P Values) for the Current Intensity at each pulse duration against pudendal nerve and anorectal manometric parameters

Pulse Duration		PNTML Right	PNTML Left	PNTML Mean	Anal Canal Length	Maximum Resting Pressure	Maximum Squeeze Pressure	First Rectal Sensation	Urgency of Defecation	Maximum Tolerated Volume
(0.05ms)	Pearson Correlation	-.472*	.227	.051	-.086	-.135	.056	-.348	-.433*	-.460*
	Sig. (2-tailed)	.023	.297	.817	.697	.539	.801	.122	.050	.036
(0.1ms)	Pearson Correlation	-.535*	.316	.111	-.055	-.136	.016	-.349	-.445*	-.485*
	Sig. (2-tailed)	.009	.142	.615	.803	.537	.943	.120	.043	.026
(0.2ms)	Pearson Correlation	-.737*	.156	-.083	-.168	-.074	.020	-.383	-.391	-.430*
	Sig. (2-tailed)	.000	.499	.722	.466	.751	.931	.106	.097	.066
(0.5ms)	Pearson Correlation	-.492*	.489*	.307	.109	-.035	.174	-.460*	-.549*	-.556*
	Sig. (2-tailed)	.017	.018	.155	.622	.873	.427	.036	.010	.009
(1ms)	Pearson Correlation	-.397*	.402*	.253	.073	.014	.186	-.439*	-.464*	-.460*
	Sig. (2-tailed)	.061	.058	.244	.741	.949	.395	.046	.034	.036
(2ms)	Pearson Correlation	-.698*	.325	.119	-.020	-.029	.142	-.382*	-.369	-.389
	Sig. (2-tailed)	.001	.162	.618	.933	.904	.549	.118	.131	.110
(5ms)	Pearson Correlation	-.199	.130	.053	-.014	.174	.254	-.292	-.286	-.265

	Correlation									
	Sig. (2-tailed)	.362	.554	.809	.950	.429	.243	.199	.210	.245
(10ms)	Pearson	-.167	.011	-.053	-.051	.192	.368*	-.271	-.215	-.172
	Correlation									
	Sig. (2-tailed)	.446	.960	.811	.817	.379	.084	.235	.349	.455
(20ms)	Pearson	-.229	-.046	-.126	.055	.274	.511*	-.285	-.124	-.081
	Correlation									
	Sig. (2-tailed)	.331	.847	.598	.817	.243	.021	.251	.624	.749
(30ms)	Pearson	-.041	-.011	-.025	.140	.299	.522*	-.227	-.093	-.032
	Correlation									
	Sig. (2-tailed)	.852	.959	.909	.524	.165	.011	.322	.689	.890
(50ms)	Pearson	-.035	-.119	-.132	-.029	.332	.400*	-.180	-.049	-.009
	Correlation									
	Sig. (2-tailed)	.876	.598	.558	.899	.131	.065	.447	.836	.971
(70ms)	Pearson	.073	-.014	.014	.012	.427	.455*	-.190	-.080	-.025
	Correlation									
	Sig. (2-tailed)	.753	.952	.954	.959	.053	.038	.436	.745	.919
(100ms)	Pearson	-.107	.031	-.002	.051	.444	.469*	-.179	-.170	-.136
	Correlation									
	Sig. (2-tailed)	.628	.887	.992	.816	.034	.024	.436	.462	.558

*Correlations at or close to significance

Appendix 3: Comparison of Adolescent and Adult Learning Differences

Age Group	Attention Span	Memory Retention	Learning Style	Emotional Stability	Abstract Thinking
Adolescents	Shorter attention span, easily distracted	Lower retention of information over time	Concrete, experiential learning	Higher emotional volatility	Developing abstract reasoning
Adults	Longer attention span, focused	Higher retention of information over time	Abstract, conceptual learning	Higher emotional stability	Advanced abstract reasoning

Appendix 5: Correlation of Rheobase, Chronaxie, Utilization Time

		PNSRT	PNSLT	RHEOBASE	CHRONAXIE	UTILITIME
PNSRT	Pearson Correlation	1.000	.902	-.001	.040	.264
	Sig. (1-tailed)	.	.000**	.497	.403	.048*
	Covariance	1.259	2.168	-2.673E-03	.426	5.728
	N	41	41	41	41	41
PNSLT	Pearson Correlation	.902	1.000	.065	.083	.279
	Sig. (1-tailed)	.000**	.	.343	.302	.039*
	Covariance	2.168	4.585	.243	1.704	11.582
	N	41	41	41	41	41
RHEOBASE	Pearson Correlation	-.001	.065	1.000	-.368	.049
	Sig. (1-tailed)	.497	.343	.	.008**	.378
	Covariance	-2.673E-03	.243	2.957	-5.933	1.618
	N	41	41	43	43	43
CHRONAXIE	Pearson Correlation	.040	.083	-.368	1.000	.393
	Sig. (1-tailed)	.403	.302	.008**	.	.005**
	Covariance	.426	1.704	-5.933	88.052	71.210
	N	41	41	43	43	43
UTILISATION TIME	Pearson Correlation	.264	.279	.049	.393	1.000
	Sig. (1-tailed)	.048*	.039*	.378	.005*	.
	Covariance	5.728	11.582	1.618	71.210	372.988
	N	41	41	43	43	43

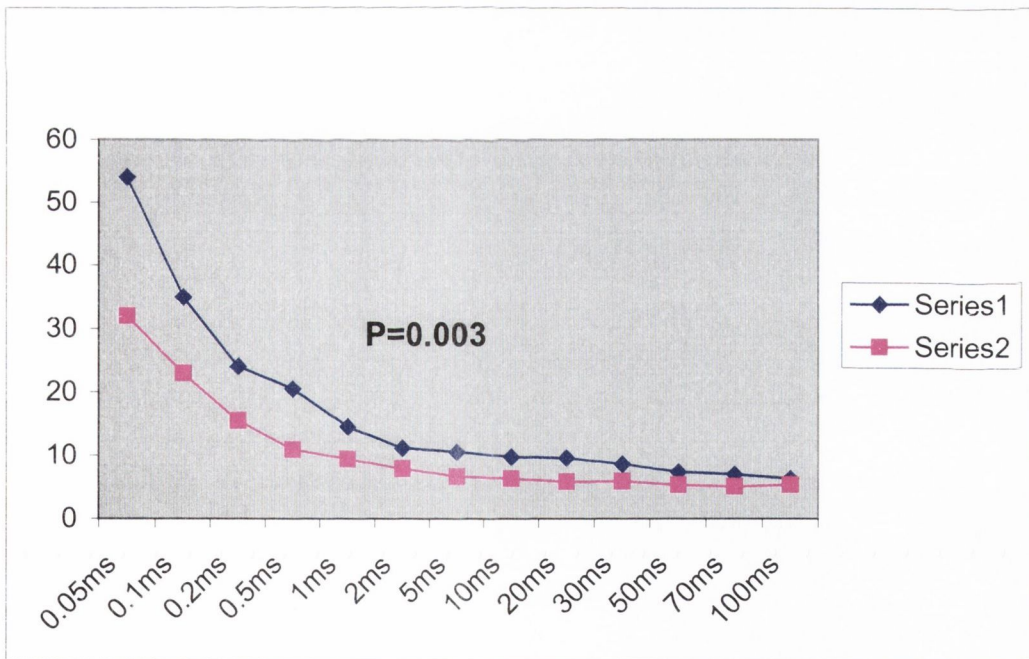
** Correlation is significant at the 0.01 level (1-tailed).

* Correlation is significant at the 0.05 level (1-tailed).

Appendix 6: Comparing Strength duration curves in individual Patients (ANOVA)

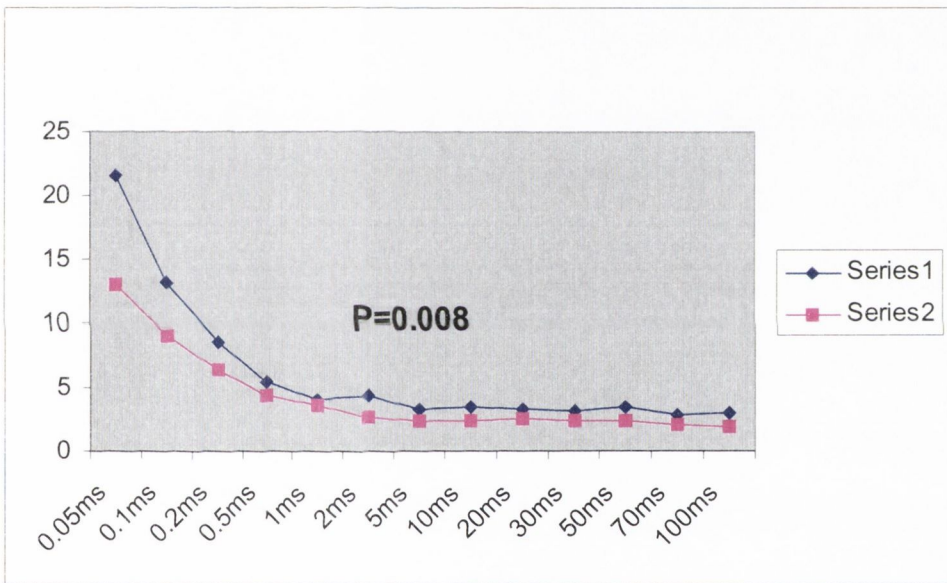
Patient 1:

0.05ms	0.1ms	0.2ms	0.5ms	1ms	2ms	5ms	10ms	20ms	30ms	50ms	70ms	100ms
54	35	24	20.5	14.5	11.1	10.5	9.7	9.5	8.6	7.3	7	6.



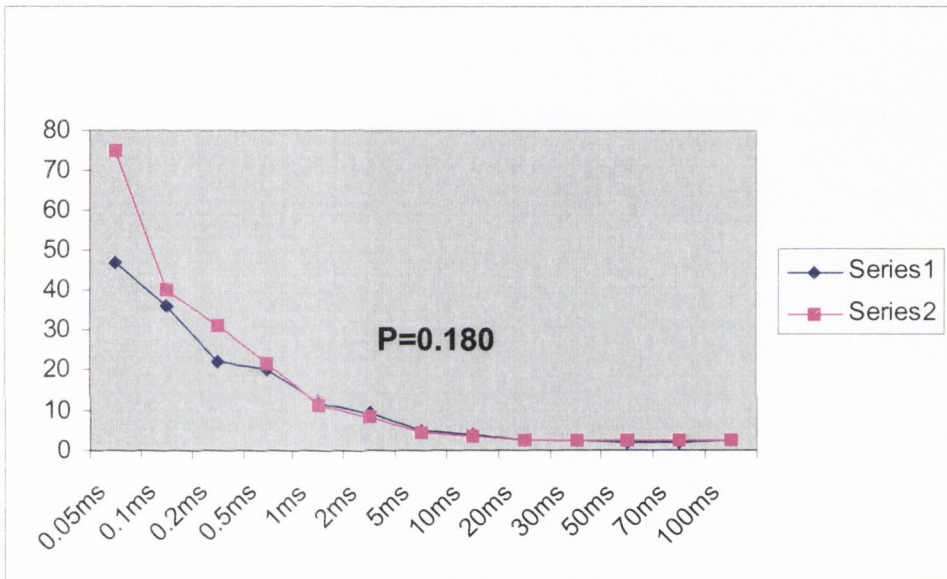
Patient 2:

0.05ms	0.1ms	0.2ms	0.5ms	1ms	2ms	5ms	10ms	20ms	30ms	50ms	70ms	100ms
21.5	13.2	8.6	5.5	4.1	4.4	3.3	3.4	3.3	3.2	3.4	2.8	3



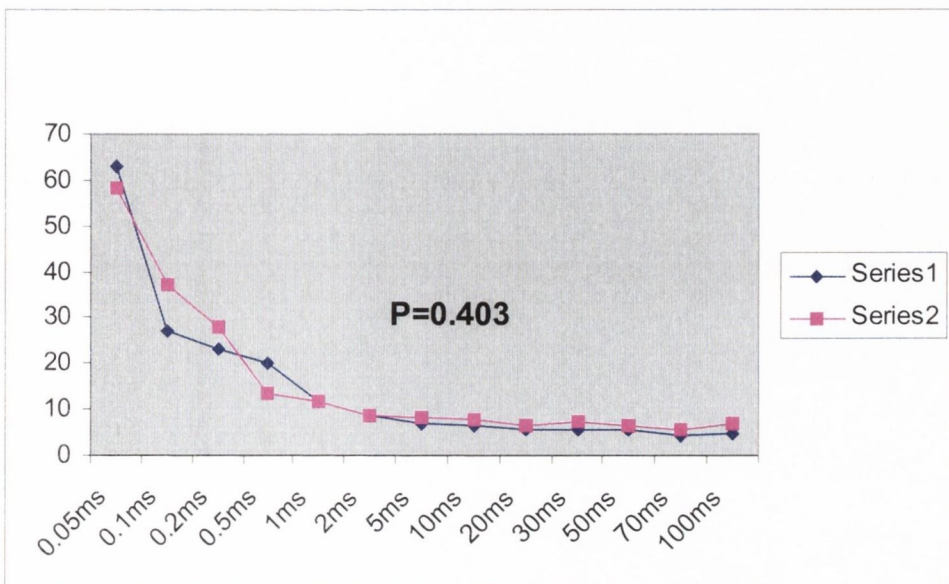
Patient 3:

0.05ms	0.1ms	0.2ms	0.5ms	1ms	2ms	5ms	10ms	20ms	30ms	50ms	70ms	100ms
47	36	22	20	12.2	9.4	5.2	3.8	2.6	2.3	2.1	2.1	2.3



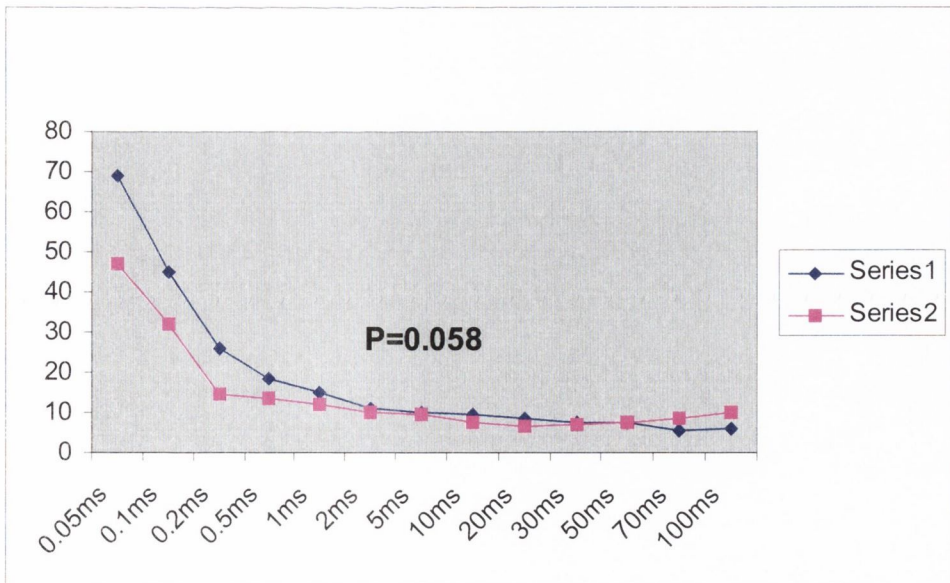
Patient 4:

0.05ms	0.1ms	0.2ms	0.5ms	1ms	2ms	5ms	10ms	20ms	30ms	50ms	70ms	100ms
63	27	23	20	11.8	8.7	7.1	6.4	5.8	5.8	5.8	4.3	5



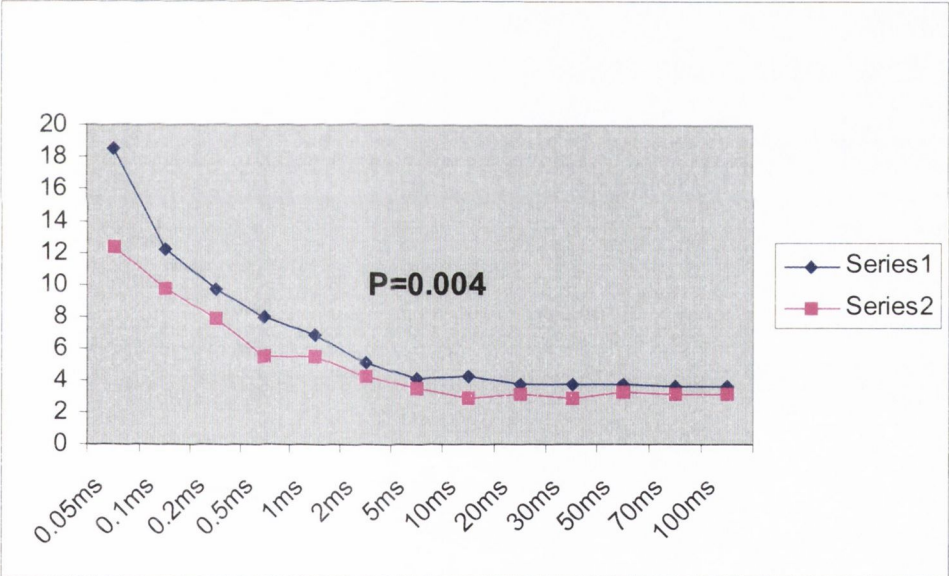
Patient 5:

0.05ms	0.1ms	0.2ms	0.5ms	1ms	2ms	5ms	10ms	20ms	30ms	50ms	70ms	100ms
69	45	26	18.5	14.9	11.2	10.1	9.7	8.5	7.3	7.4	5.7	6.2



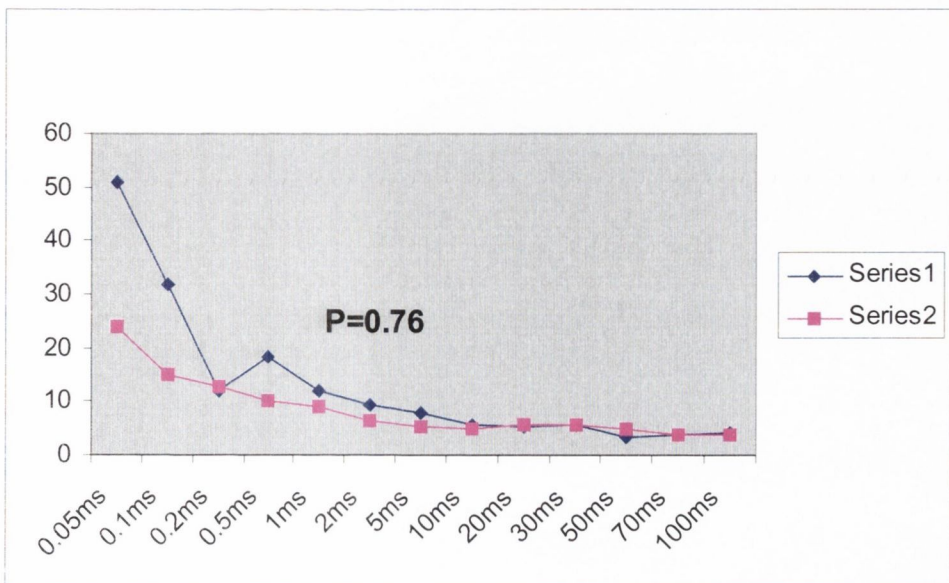
Patient 6:

0.05ms	0.1ms	0.2ms	0.5ms	1ms	2ms	5ms	10ms	20ms	30ms	50ms	70ms	100ms
18.5	12.2	9.7	8	6.9	5.1	4.1	4.3	3.8	3.7	3.7	3.6	3.6



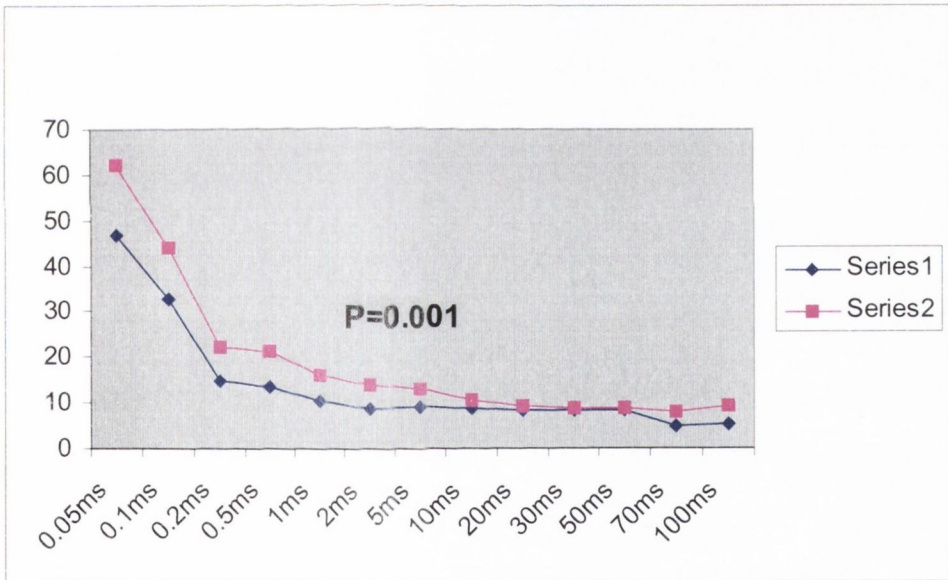
Patient 7:

0.05ms	0.1ms	0.2ms	0.5ms	1ms	2ms	5ms	10ms	20ms	30ms	50ms	70ms	100ms
51	32	12	18.5	12	9.3	7.8	5.7	5.3	5.7	3.5	3.7	4



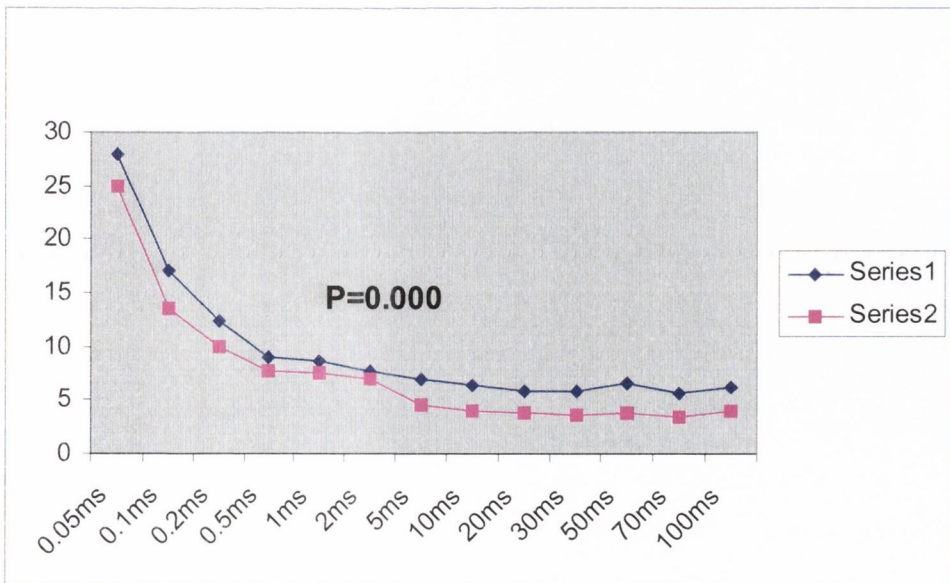
Patient 8:

0.05ms	0.1ms	0.2ms	0.5ms	1ms	2ms	5ms	10ms	20ms	30ms	50ms	70ms	100ms
47	33	15	13.5	10.6	8.8	9.1	8.9	8.4	8.3	8.1	4.9	5.1



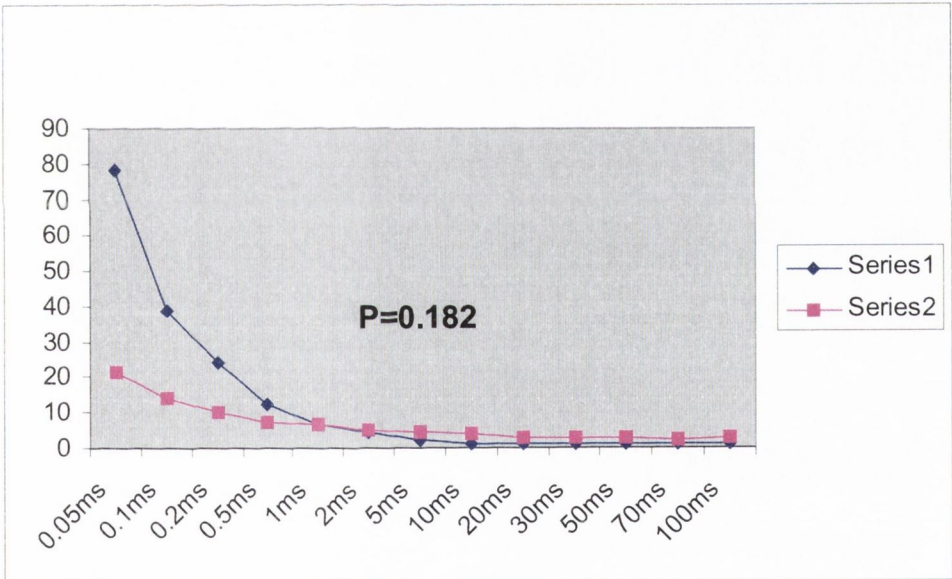
Patient 9:

0.05ms	0.1ms	0.2ms	0.5ms	1ms	2ms	5ms	10ms	20ms	30ms	50ms	70ms	100ms
28	17	12.3	9	8.6	7.6	6.9	6.3	5.8	5.8	6.5	5.7	6.2



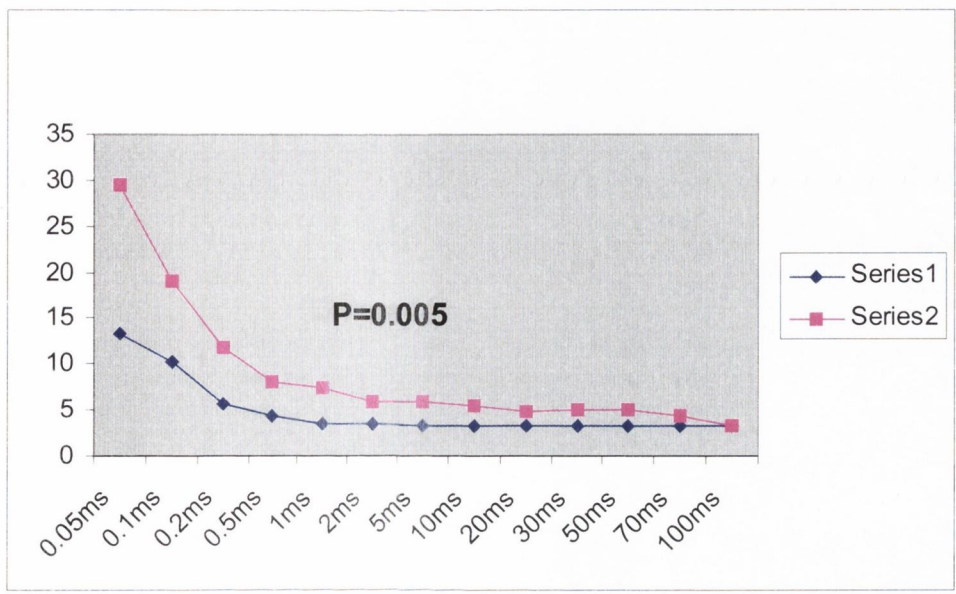
Patient 10:

0.05ms	0.1ms	0.2ms	0.5ms	1ms	2ms	5ms	10ms	20ms	30ms	50ms	70ms	100ms
78	39	24	12.2	6.6	4.6	2	1.2	1.2	1.1	1.3	1.2	1.1



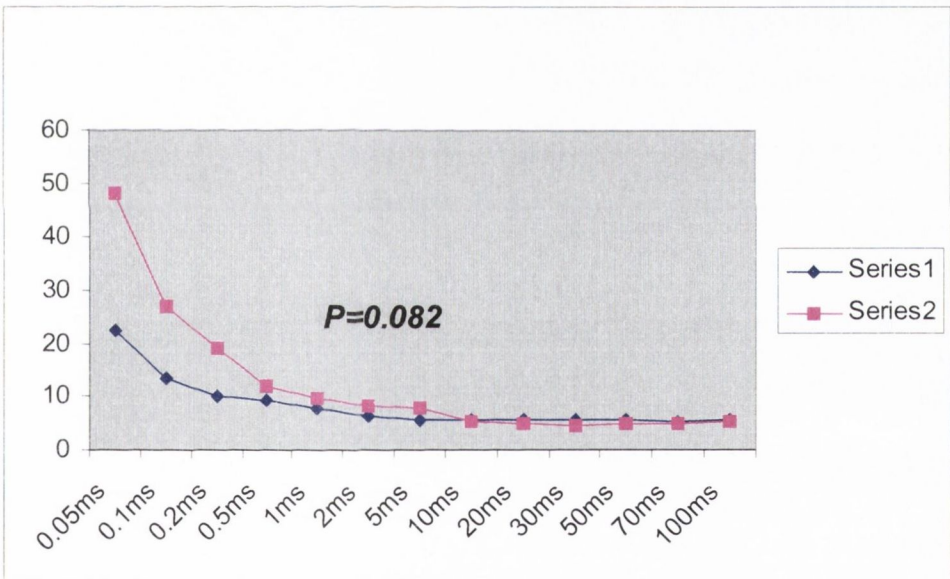
Patient 11:

0.05ms	0.1ms	0.2ms	0.5ms	1ms	2ms	5ms	10ms	20ms	30ms	50ms	70ms	100ms
13.3	10.2	5.6	4.4	3.6	3.4	3.3	3.2	3.2	3.2	3.2	3.2	3.2



Patient 12:

0.05ms	0.1ms	0.2ms	0.5ms	1ms	2ms	5ms	10ms	20ms	30ms	50ms	70ms	100ms
22.5	13.4	10.2	9.3	8	6.2	5.6	5.7	5.7	5.6	5.6	5.4	5.6



Appendix 7: Faecal and Urinary incontinence scoring sheet

Assessment date: 13/05/04

Medical Record Number

Name

Date of Birth

Sex

Address

Phone Home
 Work
 Mobile
 GP

History Summary

Faecal Incontinence *None*

Grade
 Solid
 Liquid
 Gas

Associated Urinary Incontinence
 None
 Stress
 Urge
 Mixed

Associated Urinary Incontinence Other:

Vaisey Score Card

Solid Stool	<input type="checkbox"/>
Liquid Stool	<input type="checkbox"/>
Gas	<input type="checkbox"/>
Lifestyle Alteration	<input type="checkbox"/>
Wears Pad or Plug (y/n)	<input type="checkbox"/>
Constipating Medicines (y/n)	<input type="checkbox"/>
Lack of ability to defer defaecation for 15 mins (y/n)	<input type="checkbox"/>
Yes = 2, No = 0	
Vaisey Score	<input type="text" value="/22"/>

Wexner Score Card

Solid	<input type="checkbox"/>
Liquid	<input type="checkbox"/>
Gas	<input type="checkbox"/>
Wears Pad	<input type="checkbox"/>
Lifestyle Alteration	<input type="checkbox"/>
Wexner Incontinence Score	<input type="text" value="/20"/>

Systems Review

In the last month have you had any of the following symptoms?

- Constipation
- Diarrhoea
- Bloating
- Abdominal Cramps
- Blood in Bowel Motion
- Mucus in Bowel Motion
- Pain on Defaecation
- Sensation of Incomplete emptying

Vaisey Score Key

0 never	no episodes in the last 4 weeks
1 rarely	1 episode in the past 4 weeks
2 sometimes	> 1 episode in the last 4 weeks but < 1 per week
3 weekly	1 or more episodes per week but < 1 per day
4 daily	1 or more per day

Wexner Score Key

0 never	never
1 rarely	less than once per month
2 sometimes	more than 1 / month and less than 1/week
3 usually	more than 1 / week and less than 1 / day
4 always	more than once per day

Medication

Anti-Diarrheol Agents Mobility Index

Calcium Channel Blockers Mental Status

Other Medication

Patient Report

MRN:

Name:

Phone No.:

Address:

Date of Birth:

GP:

Consultant:

History:

Gynaecological History

Surgical History

Medical History

<input type="checkbox"/> Pre-Menopausal	<input type="checkbox"/> Haemorrhoidectomy	<input type="checkbox"/> Diabetes Mellitus
<input checked="" type="checkbox"/> Post Menopausal	<input type="checkbox"/> Anal Fissure Repair	<input type="checkbox"/> Neurological Disease
<input type="checkbox"/> HRT	<input type="checkbox"/> Rectal Prolapse Repair	<input type="checkbox"/> Ulcerative Colitis
<input checked="" type="checkbox"/> Genital Prolapse	<input type="checkbox"/> Bowel Resection	<input type="checkbox"/> Crohns Disease
<input type="checkbox"/> Prior Surgery	<input type="checkbox"/> Urological Operation	<input type="checkbox"/> Gastrointestinal Malignanc
<input checked="" type="checkbox"/> Hysterectomy - Vaginal	Other: <input type="text" value="Uterine prolapse repair"/>	<input type="checkbox"/> Irritable Bowel Syndrome
<input type="checkbox"/> Hysterectomy - Open		<input type="checkbox"/> Constipation
<input type="checkbox"/> Bladder Repair		Other: <input type="text" value="Hypertension"/>

Obstetric History

Parity: 3 of 1

Pregnancy	1	2	3	4	5	6	7	8	9	10	11	12
Normal Delivery	<input checked="" type="checkbox"/>	<input checked="" type="checkbox"/>	<input checked="" type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Assisted - Forceps	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
- Suction	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
- Episiotomy	<input checked="" type="checkbox"/>	<input checked="" type="checkbox"/>	<input checked="" type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Prolonged 2nd Stage	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Malpresentation	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Large	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Elective Caesarian	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Emergency Caesarian	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Epidural	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

Faecal Incontinence Quality of Life Instrument

Total: /110

<input type="checkbox"/>	I worry about not being able to get to the toilet on time	<input type="checkbox"/>	I worry about my incontinence getting worse as I get older
<input type="checkbox"/>	I worry about coughing and sneezing	<input type="checkbox"/>	I have a hard time getting a good night's sleep
<input type="checkbox"/>	I have to be careful about standing up and sitting down	<input type="checkbox"/>	I worry about being embarrassed or humiliated because of my incontinence
<input type="checkbox"/>	I worry where the toilets are in new places	<input type="checkbox"/>	My incontinence makes me feel like I'm not a healthy person
<input type="checkbox"/>	I feel depressed	<input type="checkbox"/>	My faecal incontinence makes me feel helpless
<input type="checkbox"/>	I don't feel free to leave my home for long periods of time	<input type="checkbox"/>	I get less enjoyment out of life because of my faecal incontinence
<input type="checkbox"/>	I feel frustrated because my faecal incontinence prevents me from doing what I want	<input type="checkbox"/>	I worry about soiling myself
<input type="checkbox"/>	I worry about others smelling Faeces on me	<input type="checkbox"/>	I feel like I have no control over my bowel motion
<input type="checkbox"/>	Incontinence is always on my mind	<input type="checkbox"/>	I have to watch what I eat/drink
<input type="checkbox"/>	It's important for me to make frequent trips to the toilet	<input type="checkbox"/>	My faecal incontinence limits my choice of clothing
<input type="checkbox"/>	Because of my incontinence it is important to plan every detail in advance	<input type="checkbox"/>	I worry about having sex

Urinary Incontinence Quality of Life Instrument

Total: 79 /110

<input type="checkbox"/>	3 I worry about not being able to get to the toilet on time	<input type="checkbox"/>	4 I worry about my incontinence getting worse as I get older
<input type="checkbox"/>	5 I worry about coughing and sneezing	<input type="checkbox"/>	5 I have a hard time getting a good night's sleep
<input type="checkbox"/>	5 I have to be careful about standing up and sitting down	<input type="checkbox"/>	2 I worry about being embarrassed or humiliated because of my incontinence
<input type="checkbox"/>	2 I worry where the toilets are in new places	<input type="checkbox"/>	5 My incontinence makes me feel like I'm not a healthy person
<input type="checkbox"/>	5 I feel depressed	<input type="checkbox"/>	5 My faecal incontinence makes me feel helpless
<input type="checkbox"/>	3 I don't feel free to leave my home for long periods of time	<input type="checkbox"/>	5 I get less enjoyment out of life because of my faecal incontinence
<input type="checkbox"/>	3 I feel frustrated because my faecal incontinence prevents me from doing what I want	<input type="checkbox"/>	2 I worry about soiling myself
<input type="checkbox"/>	1 I worry about others smelling Faeces on me	<input type="checkbox"/>	3 I feel like I have no control over my bowel motion
<input type="checkbox"/>	2 Incontinence is always on my mind	<input type="checkbox"/>	5 I have to watch what I eat/drink
<input type="checkbox"/>	2 It's important for me to make frequent trips to the toilet	<input type="checkbox"/>	2 My faecal incontinence limits my choice of clothing
<input type="checkbox"/>	5 Because of my incontinence it is important to plan every detail in advance	<input type="checkbox"/>	5 I worry about having sex

Key

- 1 = Extremely
- 2 = Quite a bit
- 3 = Moderately
- 4 = A little
- 5 = Not at all

Appendix

301690
 Mrs. 01/09/1951 (F)
 Dolores McLaughlin
 25 St. Brigid's Park
 Foxrock
 Dublin 15



Q 1: In general, would you say your health is:

- 1 Excellent
- 2 Very Good
- 3 Good
- 4 Fair
- 5 Poor

Q 2: For each of the items, please indicate how much of the time the issue is a concern for you due to accidental bowel leakage. (If it is a concern for you for reasons other than accidental bowel leakage then check the box under Not Apply, (N/A).)

Q2. Due to accidental bowel leakage:	Most of the Time	Some of The Time	A Little of the Time	None of the Time	N/A
a. I am afraid to go out	1	2	3	4	<input type="checkbox"/>
b. I avoid visiting friends	1	2	3	4	<input type="checkbox"/>
c. I avoid staying overnight away from home	1	2	3	4	<input type="checkbox"/>
d. It is difficult for me to get out and do things like going to a movie or to church	1	2	3	4	<input type="checkbox"/>
e. I cut down on how much I eat before I go out	1	2	3	4	<input type="checkbox"/>
f. Whenever I am away from home, I try to stay near a restroom as much as possible	1	2	3	4	<input type="checkbox"/>
g. It is important to plan my schedule (daily activities) around my bowel pattern	1	2	3	4	<input type="checkbox"/>
h. I avoid traveling	1	2	3	4	<input type="checkbox"/>
i. I worry about not being able to get to the toilet in time	1	2	3	4	<input type="checkbox"/>
j. I feel I have no control over my bowels	1	2	3	4	<input type="checkbox"/>
k. I can't hold my bowel movement long enough to get to the bathroom	1	2	3	4	<input type="checkbox"/>
l. I leak stool without even knowing it	1	2	3	4	<input type="checkbox"/>
m. I try to prevent bowel accidents by staying very near a bathroom	1	2	3	4	<input type="checkbox"/>

Q 3: Due to accidental bowel leakage, indicate the extent to which you AGREE or DISAGREE with each of the following items. (If it is a concern for you for reasons other than accidental bowel leakage then check the box under Not Apply, N/A).

Q3. Due to accidental bowel leakage:	Strongly Agree	Somewhat Agree	Somewhat Disagree	Strongly Disagree	N/A
a. I feel ashamed	1	2	3	4	<input type="checkbox"/>
b. I can not do many of things I want to do	1	2	3	4	<input type="checkbox"/>
c. I worry about bowel accidents	1	2	3	4	<input type="checkbox"/>
d. I feel depressed	1	2	3	4	<input type="checkbox"/>
e. I worry about others smelling stool on me	1	2	3	4	<input type="checkbox"/>
f. I feel like I am not a healthy person	1	2	3	4	<input type="checkbox"/>
g. I enjoy life less	1	2	3	4	<input type="checkbox"/>
h. I have sex less often than I would like to	1	2	3	4	<input type="checkbox"/>
i. I feel different from other people	1	2	3	4	<input type="checkbox"/>
j. The possibility of bowel accidents is always on my mind	1	2	3	4	<input type="checkbox"/>
k. I am afraid to have sex	1	2	3	4	<input type="checkbox"/>
l. I avoid traveling by plane or train	1	2	3	4	<input type="checkbox"/>
m. I avoid going out to eat	1	2	3	4	<input type="checkbox"/>
n. Whenever I go someplace new, I specifically locate where the bathrooms are	1	2	3	4	<input type="checkbox"/>

Q 4: During the past month, have you felt so sad, discouraged, hopeless, or had so many problems that you wondered if anything was worthwhile?

- 1 Extremely So - To the point that I have just about given up
- 2 Very Much So
- 3 Quite a Bit
- 4 Some - Enough to bother me
- 5 A Little Bit
- 6 Not At All

Mrs. [redacted] 01/09/1951 (F)
 Dolores Mc Loughlin
 25 St Brigids Park
 Foxrock
 Dublin 18



Anorectal Manometry

Patient Name: **Mc Loughlin, Dolores**
 Patient ID #: **301690**
 Referring Physician: **, Prof. Prenderville**
 Date of Test: **14/05/02**

Physician: **Dr. Eguare Emmanuel.**
 Assistant: **Bmc Govern, Specialist Nurse**

Interpretation and Comments

50 yr old married lady with combined Faecal and Urinary Incontinence. Rectocele on examination. Previous Vaginal Hysterectomy and Repair 1998.
 MUI tube 138.
 Findings: Good Resting Pressures
 Adequate Squeeze Pressures though poorly sustained.
 First Desire @ 60mls , Urgency @ 220mls, Max tolerated volume 240mls.

Radial Pressure Analysis

Units: Pressure = mmHg Duration = sec. Rate = mmHg/sec.

Resting pressures

depth (cm)	Anter.	Left	Post.	Right	Max.	Min.	Median	Mean	HPZ
6	15				15	15	14.5	14.5	
5	8	77			77	8	42.5	42.5	
4	24	41	90		90	24	41.3	51.7	X
3	43	54	67	119	119	43	60.5	70.9	X
2	63	74	77	85	85	63	75.7	74.9	X
1	38	68	67	67	68	38	67.0	59.8	X

Pressure increases during squeeze

depth (cm)	Anter.	Left	Post.	Right	Max.	Min.	Median	Mean
6	0							
5	0	0						
4	44	20	22		44	20	22.4	28.9
3	40	31	21	51	51	21	35.6	35.9
2	57	50	52	44	57	44	50.8	50.8
1	105	113	63	60	113	60	84.2	85.4

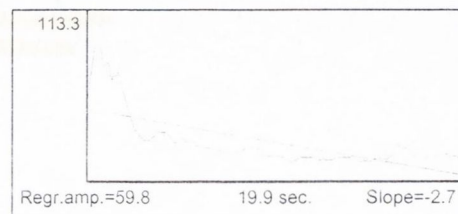
	<u>Resting</u>	<u>Normals</u>	<u>Squeezing</u>	<u>Normals</u>
HPZ length (cm)	4	2.5 to 3.5		
Mean pressure over HPZ	65	40 to 70	52	
Maximum pressure over HPZ	119		113	30 to 110
Verge to maximum (cm)	2		1	

RectoAnal Inhibitory Reflex (R.A.I.R)

Present for 20 ml at 1.0 cm from anal verge with sensation

Maximum Sustained Squeeze

		<u>Normals</u>
Peak amplitude	113	30 to 110
Regression amplitude	60	
Duration	19.9	
Duration at 50%	2.3	
Fatigue rate	2.7	



Appendix 8: Summary of MRI data

Total number of patients	24
T2	10
T3	14
Correctly staged	7/16(44%)
Understaged	6/16(38%)
Overstaged	3/16(18%)
Post Neoadjuvant Staging	
Correctly Staged	4/6(67%)
Understaged	2/6(33%)
Without Adjuvant therapy	
Correctly staged	1/3(33%)
Understaged	2/3(67%)