VOIDING DYSFUNCTION AND DETRUSOR INSTABILITY AFTER THE COLPO SUSPENSION OPERATION FOR GENUINE STRESS INCONTINENCE

by

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A thesis submitted to the University of Plymouth in partial fulfilment for the degree of

DOCTOR OF MEDICINE

June 1999
ABSTRACT

VOIDING DYSFUNCTION AND DETRUSOR INSTABILITY AFTER THE COLPOSUSPENSION OPERATION FOR GENUINE STRESS INCONTINENCE

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Colposuspension is an effective treatment for genuine stress incontinence. Continence is restored by positioning the bladder neck in a fixed and elevated retro-pubic position. Despite a high success rate of up to 90%, post-operative complications occur which may have an adverse effect on quality of life. Voiding difficulties develop in 0-43% of patients and detrusor instability in 2-25%. This considerable variability is due to differences in definition, the timing of assessment, patient selection, and probably also in surgical technique. The natural history of these complications is not clearly known due to the lack of prospective follow-up studies. There is also general uncertainty with regards to their causes. While retrospective studies have attempted to identify pre-operative risk factors, there are no prospective studies which attempt to correlate the anatomical and functional changes caused by surgery with the development of voiding dysfunction and detrusor instability.

This study has investigated prospectively 77 women undergoing the operation of colposuspension in relation to the incidence, natural history and causes of post-operative voiding dysfunction and detrusor instability. The complications were identified and followed-up objectively by means of serial urodynamic studies. Patients were also assessed clinically and using quality of life measures. The development of complications were correlated to a number of anatomical and functional changes caused by surgery. Anatomical changes were identified mainly by imaging the bladder neck with Magnetic Resonance Imaging (MRI). Functional changes were identified using urodynamic studies.
Voiding dysfunction after colposuspension was common, with 69% of women requiring a catheter for more than seven days, and 28% for longer than 14 days. Improvement occurred gradually in most cases, with only 7.7% and 2.5% of them needing catheterization at three months and one year respectively.

De novo detrusor instability occurred in 21% of women at three months follow-up, and was symptomatic in 66% of these cases. Objective and subjective resolution was seen in 50% of these at one year follow-up.

Quality of life after colposuspension improved in most cases despite the development of these complications, probably due to the resolution of their incontinence.

Voiding dysfunction and detrusor instability after colposuspension were found to be multifactorial, due to patient related factors (age and detrusor contractility for voiding dysfunction, and age and a past history of bladder neck surgery for detrusor instability), and to operative factors (amount of bladder neck elevation and urethral compression). These findings might lead to the development of preventative measures.
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GLOSSARY OF ABBREVIATIONS

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ACKNOWLEDGEMENTS

My grateful thanks are due and herein expressed to the many people without whose help this work would not have been possible.

Mr R.M. Freeman, Consultant Obstetrician and Gynaecologist, for supervising this work, for his teaching in research methodology and presentation techniques, for his involvement in the reproducibility studies of the methods used in this project, for his constant support and infectious enthusiasm, and for his training in Urogynaecology.

Prof. K. Rogers (Postgraduate Dean, University of Plymouth), for supervising this work, and for his advice and support.

Dr J. R. Sneyd (Consultant Anaesthetist, University Reader), for supervising this work, and for his expert advice.

Mrs A. Waterfield and Mrs R. Bishop, for their help during urodynamic tests and post-operative management of patients. Thanks to their care and enthusiasm, they have greatly contributed towards our patients satisfaction and compliance. Also to the other Nurses of the Gynaecology Ward of Derriford Hospital for their help.

Mr S. Shaw, Statistician (School of Mathematics and Statistics, University of Plymouth), for his advice and statistical expertise.

Mrs E. Watson, Senior Radiographer, for performing the MRI studies and for her involvement in the reproducibility studies of this technique. Also to the other Radiographers in the MRI Department for their kind assistance.

Dr M. Williams, Consultant Radiologist, for allowing us to perform MRI studies under his supervision and for checking the quality of the results.

Dr P. Dubbins, Consultant Radiologist, for his teaching and support in relation to the intra-operative ultrasound studies.
Mrs C. Arnold, Mrs M. Down and Mrs L. Durbin, Medical Secretaries, for helping to
arrange patient’s appointments.

Mr A. Barton (Research and Development Unit, Derriford Hospital), for his helpful
advice.

Mr A. Hassard, Clinical Psychologist, for his advice on psychological questionnaires and
pain scoring systems.

Mr C. Kelleher and Prof. L. Cardozo, for allowing me to use the King’s Health
Questionnaire.

Mrs D. Hill (Business Manager, Directorate of Obstetrics and Gynaecology, Derriford
Hospital), for supporting our project.

Mrs S. Waring (Medical Photography, Derriford Hospital), for her help with pictures and
illustrations.

Finally, this thesis is dedicated to my family, my Mother and Father, my wife Elizabeth
and my children Filippo and Francesca. Special thanks go to my wife Elizabeth, for her
endless support and dedication.
AUTHOR'S DECLARATION

During registration for the degree of Doctor of Medicine, the author has not been registered for any other University award.

This study was carried out in collaboration with the departments of Obstetrics & Gynaecology and Radiology, Derriford Hospital, Plymouth. Support was also provided by the department of Mathematics and Statistics of the University of Plymouth.

The work of which this thesis is a record was carried out by me while working as a Research Fellow in Urogynaecology at Derriford Hospital, Plymouth. During this time I obtained clinical training in Urogynaecology and also training in research methodology.

All references cited were personally consulted during the preparation of this thesis.

Relevant scientific seminars and conferences were regularly attended. Contacts with external institutions and clinical experts in the field of Urogynaecology were established.

The contents of the thesis have provided material for presentations to learned societies.

Presentations at the International Urogynecology Association (IUGA) Annual meetings:

- 'Why do women have voiding difficulty after colposuspension?', IUGA 1997, Amsterdam (oral presentation).
- 'Why do women have detrusor instability after colposuspension?', IUGA 1998, Buenos Aires (oral presentation).

Other presentations:
- I have been invited to speak on 'voiding dysfunction' at the 1997 Joint Ulster Obstetrical Society / South West Obstetricians and Gynaecologists Society meeting.
- I have been invited to speak on 'prevention of voiding dysfunction after colposuspension' at the Research Urogynaecology Society (RUGS) at the Royal College of Obstetricians & Gynaecologists (RCOG) in 1998.

The contents of the thesis will contribute to future publications.

Signed

Luigi Bombieri

Date

June 1999
CHAPTER 1

INTRODUCTION
Urinary incontinence, defined as ‘the involuntary loss of urine which is objectively demonstrable and a social or hygienic problem’ [1], is reported by 12-44% of women [2]. Up to 5.7% of women interviewed in a population based study reported urinary incontinence within the preceding week, thus providing a measure of the prevalence of severe incontinence [3].

Genuine stress incontinence (GSI) is defined as ‘the involuntary loss of urine occurring when, in the absence of a detrusor contraction, the intravesical pressure exceeds the maximum urethral pressure’ [1]. Leakage of urine typically occurs during a rise in intra-abdominal pressure. Loss of support of the urethra and bladder neck, and urethral sphincter weakness are considered the major causative factors [4]. The exact prevalence of genuine stress incontinence is not known, as there are no population based urodynamic studies. The subjective symptom of stress incontinence is reported by 49% of incontinent women in epidemiological studies, while 22% report urge incontinence and 29% have mixed symptoms [2]. However, symptoms are not a reliable guide to the cause of incontinence [5], and urodynamic studies are required for an accurate diagnosis.

In incontinent women presenting to a urodynamic clinic, GSI is reported to be the commonest cause of incontinence, occurring in 41-62% of cases [5,6]. As not all incontinent women present to a doctor and the criteria for referral for urodynamics may vary, this proportion may not be representative of the whole incontinent population.

Urinary incontinence is a symptom that may profoundly impair quality of life. A high proportion of incontinent women are affected by the need to wear pads, resort to restricting their fluid intake, avoid going away from home, restrict their physical activities and find that incontinence affects their work [7]. The social stigma associated with incontinence makes many women feel ‘odd, different and less attractive’, and makes them avoid sexual activity or seeing other people [7]. However, the psychosocial impact of incontinence on
each individual is different and is not directly proportional to the objective severity of symptoms [8].

In addition to stress incontinence, women with genuine stress incontinence have a variety of additional urinary symptoms. In a large series of women with this condition, 68% reported urgency, 59% urge incontinence, 47% frequency, 43% nocturia, 30% incomplete bladder emptying, 18% coital incontinence, and 3% nocturnal enuresis [9]. These associated symptoms may have a psychosocial impact on women which is as significant as the incontinence itself [8].

Although stress urinary incontinence can be effectively treated, it is estimated that only one-quarter to one-third of incontinent women in developed countries seek medical help [2]. This is because many women hope that their symptoms would get better on their own, while others think that incontinence is normal, and others are too embarrassed to complain [7].

Available therapies include pelvic floor exercises and surgery.

Treatment with pelvic floor exercises was first introduced by Kegel in 1948 [10] and has a variable success rate of 17-84% [11]. Cure and satisfaction rates may reach 60-70% in well motivated patients who are instructed and followed-up by enthusiastic professionals [11]. The beneficial effect of pelvic floor exercises can also be maintained in the long-term [11]. As a consequence, pelvic floor exercises are usually offered as a primary treatment to women with stress incontinence.

The ideal surgery for genuine stress incontinence should be effective in the long-term and should also have low morbidity. Since Baker-Brown performed a suprapubic cystostomy in 1864, more than 150 different operations have been described for the treatment of stress incontinence [12]. This reflects the uncertainty as to which procedure best achieves these
aims. The colposuspension operation was first described by Burch in 1961 [13], as a modification of the Marshall-Marchetti-Krantz procedure [14]. Continence is restored by positioning the bladder neck in a fixed and elevated retro-pubic position. There is evidence from retrospective [15-17] and prospective [18-20] studies, that the colposuspension operation has a high success rate of up to 85-90%, and that the beneficial effect tends to be long-lasting [17]. As a consequence, this operation is often taken as the ‘gold standard’ against which new procedures are measured. Despite the high success rate, complications occur which may adversely affect quality of life.

Post-operative voiding difficulties have been reported with a mean frequency of 12.5% [15], with a range of 0 to 43% [18,19,21-27]. This considerable variability is due to differences in the definition of “voiding difficulty”, in the timing of assessment, in patient selection, and probably also in surgical technique. The natural history of this complication is not clearly known and it has been suggested that patients with ‘late dysfunction’ constitute a group that is different from those with ‘early dysfunction’ [24]. There is general uncertainty with regards to the causes of dysfunction, with only age having been firmly identified as a risk factor [27].

Bladder neck overerelevation is often considered the most important cause [28], but there is no evidence to support this theory. Elevation in relation to post-operative voiding dysfunction has been assessed subjectively at the time of surgery [29], and no correlation has been found. This type of observation is likely to be inaccurate, as the bladder neck cannot be directly visualized. Similar conclusions were reached by Dundas et al [30], who measured elevation by means of videocystourethrography; although the mean bladder neck elevation in patients with voiding dysfunction was higher than in those who voided normally, the difference was not statistically significant. Their method may have been inaccurate, because they positioned the bladder neck (a medial structure) in relation to a
lateral pelvic landmark (the ischial tuberosity); as a consequence, even a minimal lateral pelvic tilt could result in considerable loss of accuracy.

**Urethral compression** could result in post-operative voiding dysfunction and Tanagho [31] suggested that this could be avoided by placing the sutures as far laterally as possible, away from the bladder neck, thus leaving enough space to accommodate two fingers between urethra and pubic bone after tying the sutures. Unfortunately, there is no available objective data on which to base such a policy.

**Poor bladder contractility** (assessed pre-operatively) has been considered a cause of post-operative voiding dysfunction [22,24,32], but others [27] could not find an association. None of the studies have assessed voiding function (contractility) during the post-operative period.

**Psychological factors** may contribute to post-operative voiding dysfunction [27], possibly by decreasing detrusor power and inducing pelvic floor spasm, but there is no objective data to support this theory. Post-operative **pain** may similarly contribute to voiding dysfunction, but this variable has not been assessed.

Post-operative **detrusor instability** has been reported with a mean frequency of 9.6% [15], with a range of 2 to 25% [18,24-26,33-35]. The natural history of this complication is unknown, as few studies have attempted to assess this over a period of time. Those which have been carried out are retrospective and include many patients lost to follow-up [34]. Although urodynamic studies have suggested that obstruction is unlikely to be the causative mechanism [33], subtle degrees of obstruction may not be easy to diagnose in women using this method [36]. Surgical variables such as bladder neck elevation and urethral compression, which may have an obstructive effect, have never been studied in relation to the development of this complication.
Other important complications after colposuspension may also occur, such as vaginal prolapse in 4-31% of cases [23,25,26,37-40], which is correctable and therefore less likely to have prolonged or permanent adverse effects. Other complications are uncommon.

A recent survey on the impact of incontinence surgery on morbidity found that only 68% were satisfied and would recommend surgery to a friend, while 26% reported worsening of mental health, and 11% reported feeling worse than before surgery [41]. These observations from patients questioned by an independent observer are in contrast to the higher success rates reported by surgeons. When assessing the effects of an intervention, it is necessary for outcome measures to include objective and subjective data on the effects on function, development of complications, socioeconomic data and effect on quality of life [42].

It has been pointed out that there are no valid and reliable data on the frequency of complications following incontinence surgery and that there is a lack of patient-perceived outcomes regarding quality of life [16]. The development of new problems after surgery or the worsening of pre-existing conditions, may lead to a deterioration in quality of life despite cure of the genuine stress incontinence.

There is a need to standardise and improve the assessment of surgical outcomes, so that the impact of complications is taken into account. While research continues into new methods to improve the success rate of incontinence surgery, there is arguably a more urgent need to reduce its morbidity.

This study aims to investigate prospectively morbidity after the operation of colposuspension, in particular voiding dysfunction and detrusor instability, which are the complications most likely to adversely affect quality of life in the long term. A better
understanding of the incidence, natural history and causes of these complications, may lead to the development of preventative measures.

AIMS OF THESIS

1. To identify the incidence of post-operative voiding difficulty, study its natural history and investigate its causes.

2. To identify the incidence of post-operative detrusor instability, study its natural history and investigate its causes.
CHAPTER 2

PELVIC FLOOR ANATOMY IN RELATION TO URINARY

STRESS INCONTINENCE
Urinary continence at the time of intra-abdominal pressure rises depends on the integrity of the urethral supportive mechanism and of the urethral sphincter. An extensive literature review of the anatomy of the lower urinary tract is beyond the scope of this thesis. Only aspects relevant to the maintenance of continence during stress will be discussed.

1- ANATOMICAL BASIS OF URETHRAL SUPPORT

Urethral support cannot be considered in isolation as it depends on the connection of the vagina and periurethral tissues to the muscle and fascia of the pelvic wall [43]. The pelvic organs (especially vagina and uterus) are normally held in place by two main supportive structures: the endopelvic fascia, which suspends the organs from the pelvic sidewalls, and the levator ani muscles, which maintain the urogenital hiatus closed and form a shelf on which the pelvic organs rest [44].

The endopelvic fascia is a fibromuscular tissue consisting of collagen, elastin and smooth muscle. DeLancey has performed anatomical studies on cadavers and described the role of the endopelvic fascia for vaginal support [45].

Where it attaches to the upper vagina, cervix and uterus it is referred to as cardinal and uterosacral ligaments. It originates over the region of the greater sciatic foramen and lateral sacrum and inserts into the side of the cervix and upper part of the vagina (level I of vaginal support).

The middle third of the vagina is attached more directly to the lateral pelvic sidewalls in correspondence to the arcus tendineous fasciae pelvis and fascia of the levator ani muscles by a downward continuation of the cardinal and uterosacral
ligaments which are called the pubocervical fascia anteriorly and the rectovaginal fascia posteriorly (level II of vaginal support). The endopelvic fascia in the lower regions of this level (in contradistinction to level I), extends over both the anterior and posterior surfaces of the vagina in the midline.

The arcus tendineous fasciae pelvis is a fibrous band that stretches from the lower part of the pubic bone close to the symphysis to the ischial spine. The margin of the endopelvic fascia close to its point of lateral attachment can be seen as a ‘white line’.

In the distal third (level III of vaginal support), the vaginal wall is directly attached to surrounding structures (urethra, levator ani muscles and perineal body) without any intervening fascia.

The **levator ani muscle** has two different parts. The first part, the **pubococcygeous muscle**, arises from the pubic bones, attaches to the lateral walls of the vagina (over its lower third) and rectum and inserts posteriorly into a midline raphe connected to the coccyx. The second part, the **iliococcygeus muscle**, arises from a fibrous band on the pelvic wall which stretches over the obturator internus muscle (the arcus tendineous levator ani) and joins in the midline raphe behind the rectum.

The levator ani muscles are innervated mainly by the pudendal nerve, with only its posterior component (puborectalis muscle) receiving branches from the pelvic nerves [46].

The muscle cells of the levator ani consist of a majority of type I (slow twitch) fibres and also type II (fast twitch) fibres [47]. Fast twitch fibres are probably recruited during events which raise intra-abdominal pressure (e.g. coughing, sneezing) [47]. Their contraction pulls the rectum, vagina and urethra anteriorly, thus compressing their lumen. The resting tone of the levator ani muscles is
considered as a separate function which is critical for pelvic support. Tone is maintained by constant activity of fatigue-resistant (slow twitch) striated fibres [47].

2-ANATOMY OF THE URETHRA

The urethra is a multilayered hollow tube, 3-4 cm long, composed of mucosa, submucosa with a well developed vascular plexus, longitudinal smooth muscle and circular smooth muscle [4]. It is surrounded for most of its length by the urethral sphincter which consists of a smooth muscle component, the proximal (internal) sphincter and a striated muscle component, the distal (external) sphincter (figure 1).

![Anatomy of the female lower urinary tract](image)

**Fig. 1.** Anatomy of the female lower urinary tract (coronal section). D = detrusor. T = trigone. SM = smooth muscle. ES = external (striated) sphincter. PS = periurethral striated muscle (levator ani)

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The proximal sphincter lies at the level of the bladder neck occupying the first 15% of the length of the urethra and includes loops of detrusor muscle [48]. Although its structure does not correspond to an anatomical sphincter, it maintains bladder neck closure.

Its autonomic innervation is indistinguishable from that of the bladder, and travels from the pelvic plexus to the retro-trigonal region along the vascular pedicles of the bladder [49].

The distal sphincter lies below the bladder neck and surrounds the urethra from 20 to 80% of its length [48]. In its upper part the muscular fibres lie in a circular orientation, whereas distally in the area of the urogenital diaphragm fibres leave the urethra and attach to the vaginal wall as the 'urethrovaginal sphincter' or to the perineal membrane near the pubic rami as the 'compressor urethrae' [50]. Most of the bulk of the distal sphincter is in the middle third of the urethra, and its posterior part is relatively thin [48,51]. It is composed largely of slow twitch fibres deputised to maintain tone [46] and is innervated mostly by the pudendal nerve [47]. An additional somatic contribution, which also originates from the sacral segments S2-S4 and travels separately from the pudendal nerve following an intrapelvic course, has been recently described [49].

The distal 20% of the urethra passes between the vestibular bulbs and the bulbocavernosus muscles and does not have a significant muscular component [48].

Closure of the urethra is thought to be due in equal proportion to smooth muscle, striated muscle and vascular plexuses, producing a hermetic seal [52].
3. THE CONTINENCE MECHANISMS:

There is no single anatomical lesion that has been consistently and scientifically related to stress incontinence. Stress incontinence must therefore be viewed as a multifactorial and dynamic event. Enhorning has shown, using simultaneous urethral and bladder pressure measurements, that stress incontinence occurs because the normal transmission of abdominal pressure to the urethra is lost [53]. The anatomical and physiological factors responsible for this loss of pressure transmission are poorly understood. Enhorning hypothesized that it is due to displacement of the proximal urethra from its normal intra-abdominal position [53]. However, there is no part of the urethra which is truly intra-abdominal [54], and there is little relationship between stress incontinence and urethral position (assessed with Q-tip or urethrocystography) [55,56]. Also, studies on normal continent women show that pressure transmission is greater in mid-urethra rather than in the proximal part [57].

While structures such as endopelvic fascia, levator ani muscles and urethral sphincter are considered important for bladder neck support and continence, their relative contribution is a matter of debate. It is generally accepted that while the urethral sphincter has an important role in maintaining continence during basal conditions, adequate bladder neck and urethral support are essential during increases in abdominal pressure [4]. As a consequence, the main cause of stress incontinence is thought to be loss of bladder neck and urethral support, with weakness of the sphincteric mechanism constituting a second less common cause [4].
A) URETHRAL SUPPORT

The anatomical relationship between urethra, vagina, endopelvic fascia and levator ani muscles is crucial for the understanding of continence mechanisms. DeLancey has performed anatomical studies on cadavers aiming to explore the relations between these structures [54]. He found the urethra to lie on a supportive layer composed of endopelvic fascia and anterior vaginal wall (figure 2). This layer gains structural stability through its lateral attachment to the pelvic sidewall at the arcus tendineous fasciae pelvis (figure 3). The endopelvic fascia in turn is strictly connected to the levator ani muscles. The arcus tendineous fascia pelvis in its posterior part merges with the arcus tendineous levator ani (which is more lateral in its foremost part). In addition, the endopelvic fascia surrounding the vagina attaches to the levator ani muscle on its medial margin. Urethral support is then provided by a hammock-like structural layer composed by endopelvic fascia, anterior vaginal wall, arcus tendineous fasciae pelvis and levator ani. The connection of the endopelvic fascia with the levator ani muscles explains why pelvic floor contraction can be seen to control bladder neck position and also why pelvic floor exercises may benefit women with stress incontinence.
Fig. 2. Urethral supports (USu). The urethra (U) lies on a supportive layer composed of endopelvic fascia and vaginal wall (V = vagina, VM = vaginal muscle). This layer is attached laterally (FA = fascial attachment) to the pelvic sidewall at the arcus tendineous fasciae pelvis (ATFP) and is also connected to the levator ani (LA) muscles (Mat = muscular attachment). Additional abbreviations: PVM = pubovesical muscles. SFLA = Superior fascia of levator ani. PVP = paraurethral vascular plexus. R = rectum. RP = rectal pillars. Reproduced from DeLancey, Neurol Urodyn 1989; 8: 53-61, with permission of Alan R. Liss Publishers.
Fig. 3. Lateral pelvic wall attachment of the urethra (U). The arcus tendineous fasciae pelvis (ATFP) stretches from the lower part of the pubic bone close to the symphysis (PS = pubic symphysis) to the ischial spine (IS). The margin of the endopelvic fascia close to its point of lateral attachment can be seen as a 'white line'. The arcus tendineous levator ani (ATLA) stretches over the obturator internus muscle and fascia (OIM&F), and provides attachment to the levator ani muscles (LA). The arcus tendineous fascia pelvis in its posterior part merges with the arcus tendineous levator ani. Additional abbreviations: B = bladder. PVP = paraurethral vascular plexus. PVM = pubovesical muscle. VN = vesical neck. Reproduced from DeLancey, Neurourol Urodyn 1989; 8: 53-61, with permission of Alan R. Liss Publishers.
While it has been known for a long time that structural support of the urethra is an important factor for continence, the anatomical basis of this support had never been extensively researched. DeLancey performed pioneering anatomical studies on cadavers and studied the effects of simulated increases in abdominal pressure on the pelvic floor and also the effects of selectively cutting supporting structures [54]. He hypothesized that urinary continence during stress relies on the strength and integrity of the hammock-like support (endopelvic fascia and anterior vaginal wall) below the urethra. If the underlying supportive tissues are firm, downward forces would compress the urethra against them and have the effect of closing its lumen. Conversely, if they are lax and mobile, compression would not be as effective and the transmission of abdominal pressure to the urethra is lessened.

There is some controversy over which structure provides the most support to the bladder neck.

Those who favour the endopelvic fascia argue that support defects represent breaks in the endopelvic fascia network [58]. Evidence from surgical observations [59,60] and MRI [61] suggests that such breaks do occur in women with stress incontinence. The defect in patients with stress incontinence has been located mainly where the endopelvic fascia attaches to the lateral pelvic wall in correspondence to the arcus tendineous fasciae pelvis. The repair of these defects has been reported to cure stress incontinence [60]. However, most observations of endopelvic fascia breaks are observational and uncontrolled and cannot be considered specific of stress incontinence.

The supportive role of the endopelvic fascia is highlighted by evidence that its main component, collagen, plays an important part in the pathogenesis of genuine stress incontinence. Nulliparous women with stress incontinence have been shown to have
less collagen in periurethral vaginal biopsies compared to continent controls [62]. They also have an increased proportion of weaker type III collagen (with a decrease of the stronger type I), and significantly reduced collagen cross-links [62]. It has been claimed that the collagen of periurethral vaginal biopsies is representative of collagen in the endopelvic fascia [62].

Other investigators consider the levator ani muscles as the main supporting structure and recognise breaks of the endopelvic fascia as a secondary phenomenon [44,63]. They argue that muscle tissue properties are better suited for long-term support and that stress urinary incontinence is due to neuromuscular damage to the pelvic floor. This has been shown to occur by neurophysiological studies. Pudendal nerve conduction studies have shown that vaginal delivery results in damage to the innervation of the pelvic floor muscles [64,65] and that this damage is increased by subsequent deliveries [64]. Electromyographic [66] and histopathological [67] studies have shown that women with stress incontinence and genitourinary prolapse have a significant increase in denervation of the pelvic floor. However, neurophysiological evidence of muscle denervation is not present in all individuals with pelvic floor weakness, thus suggesting that weakness of the endopelvic fascia may be a primary feature in at least some women [68].

The hypothesis that both endopelvic fascia and levator ani muscles play an important role in the maintenance of continence is supported by MRI studies on five women with stress incontinence who were compared to continent controls: marked thinning of the levator ani was seen in two of five incontinent women and paravaginal defects were seen in four of five incontinent women [69].
B) URETHRAL CLOSURE

Urethral function also plays an important role in the maintenance of continence. It has been shown that increases in urethral pressure precede the increase in abdominal pressure by 250 milliseconds, and the pressure rise in the urethra frequently exceeds the increase in abdominal pressure, thus implicating contraction of the urethral striated muscle [70]. The secondary but important role of the distal urethral sphincter is also suggested by nerve conduction studies of the terminal (perineal) branches of the pudendal nerve, showing evidence of denervation injury to the urethral sphincter striated muscle in women with stress incontinence [71,72], although this finding has not been confirmed by others [73].

The role of the bladder neck closure mechanism is not clear. Although there is an association between an open bladder neck at rest and urethral sphincter incompetence [74], up to 21-50% of continent women have been shown to have incompetence of the bladder neck, expressed as opening of the bladder neck on ultrasound [75] and radiological [76] studies.

While loss of bladder neck support and damage to the urethral sphincter are considered the cause of genuine stress incontinence, identification of these defects (e.g. by imaging) has so far proven difficult. Radiography, ultrasound and more recently magnetic resonance imaging (MRI) have been used to facilitate diagnosis and assess the results of treatment. Loss of the posterior urethrovesical angle and descent, funneling and increased mobility of the bladder neck, have all been associated with genuine stress incontinence. The evidence linking these imaging features to genuine stress incontinence will be reviewed in chapter 3.
CHAPTER 3

THE INVESTIGATION OF THE LOWER URINARY TRACT:

IMAGING
Radiography, ultrasound and MRI have been used to study the lower urinary tract in patients with stress incontinence. Most interest has been directed towards the anatomy of the bladder neck, the pathological changes of stress incontinence and the alterations induced by corrective surgery. These techniques and their impact on clinical practice will be described and discussed in this chapter. The anatomical changes revealed by these techniques in association with stress incontinence will be discussed separately.

A) IMAGING TECHNIQUES

1- RADIOGRAPHY

CYSTOURERETHROGRAPHY

This method of examination had already been used in the male for a number of years before it was first applied to study women with stress incontinence by Norris and Kimbrough in 1928 [77]. They injected 180-240 ml of a 10 % sodium iodide solution through a soft rubber tube into the bladder and made antero-posterior x-ray exposures with the patient standing and recumbent, at rest and while bearing down. They compared normal women to women with stress incontinence before surgery and after failed anterior colporrhaphy; a cone-shaped bladder neck was considered typical of stress incontinence. Several other studies followed, and the history of those pioneers has been reviewed by Roberts [78]. Images were obtained using antero-posterior and oblique exposures of the urethra and bladder. Anatomical changes considered typical of stress incontinence were
funnelling of the internal urethral meatus, undue descent of the bladder neck on straining, and sometimes dilatation of the urethra.

Roberts (1952) [78] described a technique for direct lateral cystourethrography which was based on the work of Mickulicz-Radecki (1931) [79]. The difficulties of obtaining a true lateral view of the pelvis and achieving adequate penetration of the soft tissues and bone were overcome, although patients had to be subjected to a considerable dose of x-rays. A special chair was designed and the sitting position was adopted for all lateral views; this prevented the patient from moving during straining or during micturition, and solved the problem of collecting the radio-opaque fluid. Jeffcoate and Roberts (1952) [80] used lateral cystourethrography in their classical studies which highlighted the importance of the loss of the posterior urethrovesical angle in relation to stress incontinence.

Although the first to introduce a metallic bead chain into the urethra to aid imaging of the bladder neck were Stevens and Smith (1931) [81], it was Hodgkinson (1953) [82] that popularised its use and gave a considerable boost to the use of lateral urethrocystography. The chain was 25 cm long, with metallic beads of 3 mm diameter separated by 1.5 mm long connecting rods. The bead chain was laid in the lumen of a bisected rubber catheter, which acted as an introducer; once into the bladder, the chain was disengaged from the catheter by holding taut the end of the chain and further introducing the split catheter; the catheter was then withdrawn leaving the chain in place. The technique was simple, avoided the distortion caused by rubber catheters and allowed the study of patients in the erect position.

Hodgkinson’s technique and Jeffcoate and Roberts observations on the importance of the posterior urethrovesical angle in relation to stress incontinence, led to the widespread use
of cystography for the investigation of incontinent women. The history of cystography over
the next two-three decades is inextricably linked to the importance attributed to this
radiological sign. The virtual disappearance of cystography from routine clinical practice
was due less to the intrinsic limits of the technique in terms of reproducibility, than to the
loss of credibility of the posterior angle as a useful marker of genuine stress incontinence
(see below).

**COLPO-CYSTOURETHROGRAPHY**

Although Ardran et al (1956) [83] were the first to outline the vagina (and rectum) in
addition to the bladder, simultaneous radiological imaging of bladder and vagina was first
extensively studied in relation to incontinence by Bethoux and Bory (1962) [84]. Their
technique involved the insertion of radio-opaque dye into the bladder, urethra, vagina, and
rectum; in addition a metal sound was introduced in the uterus as a marker. Dynamic
pictures were taken with patients standing, firstly while contracting the pelvic floor and
secondly during Valsalva. The ‘colpocystogram’ was then constructed by drawing on the
same transparent paper the bony landmarks and the visceral contours of both radiographs.
The technique was used to study the dynamics of the pelvic organs during prolapse. It was
applied also to the study of urinary incontinence. They recognised two different
radiographic types associated with stress incontinence. ‘Type A’ (anterior) was thought to
be due to weakness of the anterior structures of support, and was characterised by
funnelling of the bladder neck. ‘Type P’ (posterior) was considered due to weakness of the
posterior structures, and was characterised by descent of the bladder. In addition, a
combined ‘type P-A’ was noticed.

Lazarevski et al [85] modified the technique, mainly by introducing a third radiograph
into the ‘colpocystogram’. This image was produced while the prolapsed organs were
pushed up by means of a forceps with a cotton tampon, in order to reveal masked prolapse of a different structure or latent stress incontinence.

Colpocystography in relation to stress incontinence was popularised in Denmark by Olesen and Walter [86], while its application elsewhere for this indication remained limited. It was suggested that the test should be used for the recognition of the basic anatomic defect behind each case of stress incontinence, in order to plan the most appropriate operation [87]. It was thought to be theoretically sound to perform a colposuspension in patients with an anterior defect and a vaginal repair for posterior defects [88]. As a consequence, colpocystography became a routine investigation in Denmark for patients with stress incontinence. This despite the complexity of the test, the exposure to radiation, the lack of anatomical studies correlated to the radiographic findings and the lack of controlled studies to assess the type of operation required by each type of defect.

Various authors [89-92] subsequently reported on the poor reproducibility of colpocystography, the low sensitivity and specificity for the diagnosis of stress incontinence, and the lack of correlation with the results of surgery. It was concluded that the test should not be offered routinely to patients with stress incontinence.

VIDEO-CYSTOURETHROGRAPHY (VCU)

Enhorning et al (1964) [93] were the first to perform simultaneous cystometry and dynamic radiological screening of the bladder and urethra; both tests were recorded by separate television cameras and fused in one image. It thus became possible to correlate morphologic and pressure changes in the bladder and urethra. Bates et al (1970) [94] simplified the procedure for routine clinical use: the test took only 30 minutes to perform and exposure to radiation was limited (equivalent to an intravenous urogram dose). They
considered the test as particularly useful in cases where the distinction between stress incontinence and urge incontinence is difficult, as it was possible to visualise escape of contrast during incontinence while the detrusor pressure was simultaneously recorded.

Although VCU is considered by some [95] as the ‘gold standard’ for the investigation of female incontinence, there is general agreement that such an expensive and complex test is not required for the routine management of most patients with stress incontinence. This is highlighted by a report from a tertiary referral centre [96], where 25 women with stress incontinence were assessed by VCU: it was concluded that this test made a negligible contribution to the final clinical outcome and that simple cystometry would have been more appropriate.

However VCU remains useful as a research tool and in the evaluation of complex problems in tertiary referral centres [97]. It enables the correlation of anatomy with function in cases of incontinence of unclear etiology. It detects diverticula of the bladder and urethra, localises urethral strictures, reveals vesico-ureteral reflux and is useful to assess patients with complex neurological problems. In addition, VCU allows visualisation of the lower urinary tract during voiding, which can be difficult using other imaging techniques such as ultrasound.

2- ULTRASOUND

Transabdominal sonography using a linear array scanner was first used to assess the bladder neck by White et al (1980) [98]. They observed 30 women, both continent and incontinent, at rest and during Valsalva. Although they did not report on their observations, they stated that the technique could provide information similar to that obtained with radiographic studies while avoiding exposure to radiation.
Using the same technique, Bhatia et al [99], assessed bladder neck mobility before and after retropubic urethropexy and suggested that ultrasound could be used to assess the adequacy of the procedure (in terms of bladder neck fixation) in cases of persistent incontinence.

Both authors commented on the difficulty of visualising the bladder neck in some patients due to obesity or pubic symphysis shadowing despite the insertion of a urethral catheter. Except for bladder volume measurement, the transabdominal technique is no longer in use.

The technique of transrectal ultrasound was already in use by the urologists for the study of the prostate but it was first described in relation to the study of the bladder neck by Nishizawa et al (1982) [100]. They suggested that it could be used in conjunction with urodynamic studies in a manner similar to VCU, with the advantages of more user-friendly equipment and no exposure to radiation. Another advantage of rectal sonography over abdominal sonography is its ability to clearly demonstrate the bladder neck without interference from the symphysis or subcutaneous fat.

Potential pitfalls are prevention of bladder neck movement by the probe and movement of the probe itself during stress [101]. To control for this possibility, rectal ultrasound has been compared with bead chain urethrocystography [102]. Bladder neck mobility was defined using different criteria for each method, thus making a quantitative comparison difficult. However, the insertion of a rectal probe has not been found to alter bladder neck position and mobility as assessed by Q-tip angle change [103].

Concerns with regards to the possible effect of the rectal probe on urethral pressure profile parameters and on bladder function during filling due to direct pressure or to anal sphinter stimulation, have been shown to be without foundation [104]. However, voiding
function with a rectal probe in situ is difficult to assess [104], and despite the creation of a specially modified chair [105], the presence of the probe may inhibit micturition [106].

Patient acceptability in comparison to other techniques is lower [107] and has limited the application of rectal ultrasonography in clinical practice.

The technique of **transvaginal ultrasonography** was first described by Quinn et al (1988) [108]. They placed an endoprobe into the vagina with a comfortably full bladder and used a catheter to obtain accurate positioning of the bladder neck. Opening of the bladder neck with a cough was considered to provide direct evidence of stress incontinence. When compared to urodynamics, they showed that vaginal ultrasound had a sensitivity of 96% and a specificity of 82% for the diagnosis of stress incontinence [109].

Distortion of the anatomical features by the endoprobe, impairment of bladder neck mobility and difficulties in the assessment of patients with significant anterior vaginal wall prolapse, were thought to be potential problems [110]. The effect of the vaginal probe on lower urinary tract function was studied by urethral pressure profilometry (UPP) and its effect on bladder neck mobility by bead chain urethrocystography: UPP parameters were found to be altered by the probe and radiography showed that the probe resulted in elevation of the bladder neck at rest and restriction of descent during Valsalva [111]. However, others found bladder neck mobility to be unaffected by the vaginal probe and comparable to colpocystourethrography [112,113].

Despite evidence that, with experience and a small probe, the technique of vaginal ultrasonography is reproducible for the assessment of position and mobility of the bladder neck [107], the unresolved controversy over the extent of distortion caused by the vaginal probe is probably the reason why many clinicians have resorted to less invasive techniques.
Kohorn et al (1986) [114] first described perineal ultrasonography with a linear probe applied to the perineal region and compared it with VCU. The techniques were said to correlate well, although the parameters compared were not mentioned and no statistical analysis were performed. Unlike rectal and vaginal ultrasonography, perineal positioning of the ultrasound probe does not alter function and anatomy, and the position of the probe is not affected by straining.

Koebi and Bernaschek (1989) [115] first described introital ultrasonography, and suggested that the smaller probe applied below the urethra has the additional advantage of allowing the performance of simultaneous urodynamic studies.

Linear-curved-array and sector scanners with frequencies between 3.5 and 5.0 Mhz are recommended for perineal sonography, while small sector probes with frequencies between 5.0 and 7.5 Mhz are used for introital sonography [116]. Marking of the urethra with a catheter for clear visualisation of the bladder neck is now not required due to improved technology and resolution.

Introital and perineal ultrasound have been compared with lateral bead chain urethrocystography. For the assessment of bladder neck mobility sonography has been found to be comparable [117] or superior [118] to radiography. For the assessment of bladder neck position [118,119] and urethrovesical angles [120] the two techniques have been found to be comparable. Radiography is superior only for the demonstration of funnelling [118].

Perineal ultrasound has also been compared to videocystourethrography [121]: the two methods were found to be comparable for the assessment of bladder neck descent (although the average descent observed by ultrasound was greater); there were discrepancies in the assessment of opening of the bladder neck on Valsalva and observation of urine loss.
Although voiding studies have been performed by perineal sonography using a specially devised chair [122], it is doubtful whether this technique will supersede VCU for this particular application.

One particularly useful application of introital and perineal ultrasound is the diagnosis of urethral diverticula. It is a simpler alternative to VCU or double-balloon urethrography [123].

Perineal ultrasound can also be used to measure the degree of bladder neck elevation that can be achieved by pelvic floor contraction when assessing the efficacy of Kegel exercises or pelvic floor electrical stimulation [124].

Methods of sonographic assessment of bladder neck position and mobility

a) Position:

Various methods have been used to localise the bladder neck in relation to the pubic bone using two distances or one distance and one angle. The more commonly used methods are illustrated in figures 4 and 5.
Fig. 4. Methods of localisation of the bladder neck using ultrasound. Arrow = distance measured, A = angle measured, S = lower edge of symphysis pubis, B = bladder neck. The bladder neck is localised by measuring a distance and an angle (1), two distances in a system of co-ordinates (2), or a distance and an angle in a system of co-ordinates (3). In (1) the reference is the pubic axis. In (2) the co-ordinates are made from a line tangent to the anterior surface of the pubis and a perpendicular over the lower edge of the symphysis. In (3) the co-ordinates are made from a line (BP), which is parallel to the vaginal probe (V) over the bladder neck, and a line which is perpendicular to BP over the back of the symphysis (X).
Fig. 5. Methods of localisation of the bladder neck using ultrasound recommended by the German Association of Urogynaecology. For description see text (p. 52). Distances are illustrated by arrows. S = lower edge of symphysis pubis, B = bladder neck, X axis = line from superior to inferior edge of symphysis pubis, Y axis = line perpendicular to X over lower edge of symphysis, Dx = distance between bladder neck and Y axis, Dy = distance between the bladder neck and the X axis.
In 1996, the German Association of Urogynaecology made recommendations for ultrasound studies of the lower female urinary tract, as a basis for comparisons of different sonographic evaluations and to help ensure examination quality [129].

When performing introital, vaginal or rectal ultrasonography (when visualisation of the entire symphysis pubis is not usually possible), a method which uses the lower point of the symphysis pubis as a reference point is recommended (figure 5a). A horizontal line is drawn at the lower border of the symphysis, and the distance between the bladder neck and the horizontal line is measured. The posterior urethrovesical angle (angle beta) can also be measured.

When using perineal ultrasound, and the whole of the symphysis pubis is visible, the method proposed by Schaer et al [118] is recommended (figure 5b). A system of coordinates is used: the X axis is constructed by drawing a line between the superior and inferior borders of the symphysis pubis and the Y axis is constructed perpendicular to the X axis at the inferior symphysis border. Two distances are measured: Ox, which is the distance between the bladder neck and the Y axis, and Dy which is the distance between the bladder neck and the X axis. For precise localisation of the bladder neck, the upper and ventral point of the urethral wall at the immediate transition into the bladder is used. The reproducibility of this method for the determination of bladder neck position was assessed by two investigators on 40 patients and there was good inter-examiner agreement [118].

These recommendations have not yet been considered by the International Continence Society (ICS) Standardisation Committee or by the International Urogynecology Association (IUGA). They are however the most commonly used methods at present.
b) Mobility:

Bladder neck mobility has also been assessed by ultrasound in a variety of ways. While some methods measure bladder neck movement per se without indicating direction or position, others are able to measure movement in two directions (antero-posterior and supero-inferior) and can relate bladder neck movement to fixed structures such as the symphysis.

Bladder neck movement can be measured as a straight distance between the location of the bladder neck at rest and after Valsalva (figure 6a, oblique arrow) or the distance of vertical descent after Valsalva (figure 6a, vertical arrow). Alternatively, a 'rotation angle' can be measured before and after Valsalva. Angles that can be measured are the angle between pubic axis and the bladder neck to lower symphysis line (B-S line), or the angle between a horizontal line drawn from the lower edge of the symphysis and the B-S line, or simply the angle between the B-S line at rest and after Valsalva (figure 6b).

Movement can also been measured by localising the bladder neck at rest and after valsalva in a system of co-ordinates (figure 5b). This results in the measurements of two distances at rest and two distances after Valsalva. Bladder neck mobility can then be determined by calculating the length of the vector between the two positions using a mathematical formula (vector length = square root of (x1 - x2)^2 + (y1 - y2)^2). This method is favoured by the German Association of Urogynaecology [129], as it allows clear discrimination of direction of movement, accurate localisation in relation to bone landmarks and comparison between studies.

A crucial aspect that needs to be addressed in relation to the assessment of bladder neck movement is the quantification of the abdominal force during straining, without which comparisons are likely to be inaccurate.
Fig. 6. Methods of assessment of bladder neck mobility by ultrasound. For description see text (p. 53). B1 = position of bladder neck at rest, B2 = position of bladder neck after Valsalva, S = lower edge of symphysis pubis, B-S = line between bladder neck and lower edge of symphysis.
Influence of body position, bladder volume and urethral catheter on sonographic parameters

The patient’s position has an influence on some measurements. In the standing position the bladder neck lies lower [129,130] and the posterior urethrovvesical angle is larger [129] than in the supine position; in addition, funnelling is found more frequently with the patient standing [130]. Likewise, in the sitting position, the bladder neck has been found to be lower and less mobile than in the supine position [131].

Bladder volume has no influence on bladder neck position [129,131,132], but increasing volumes slightly widen the posterior urethrovvesical angle; this may be due to involuntary contraction of the pelvic floor. In addition, bladder neck funnelling is more often detected with a greater bladder volume [129,130].

The presence of urethral catheters have been shown to increase urethral width but do not affect bladder neck location and urethral mobility [133]. However, the presence of a catheter may reduce the distance between bladder neck and pubic bone in postmenopausal women not taking hormone replacement [132]; this may be due to mechanical distortion or involuntary contraction of pelvic floor muscles.

New developments in ultrasound

Three dimensional ultrasound has been used to measure urethral sphincter volume [134-136] and the surface area of the levator ani hiatus [136,137].

Intra-urethral ultrasound, with a 9 French polyurethane catheter containing a mechanically rotating 20 Mhz single element transducer, has been used to measure urethral sphincter volume [138]; patient acceptability of this relatively invasive technique was found to be good.

These techniques are to be considered at present as research tools.
3- MAGNETIC RESONANCE IMAGING (MRI)

MRI has been a recognised imaging technique since the early 1980s. It was first applied to the study of stress incontinence by Klutke et al (1990) [139].

MRI is based on the principle that protons have a positive electrical charge and perform a motion that resembles the wobbling of a spinning top; this motion is called precession and results in the creation of a magnetic field. The hydrogen proton has the strongest magnetic momentum and is the most abundant nuclear species in living tissue; because of its abundance, the vast majority of clinical magnetic resonance imaging today is of hydrogen.

The single steps of an MRI examination can be described quite simply: the patient is placed in a magnet, a radio wave is sent in, the radio wave is turned off, and the patient emits a signal which is received and used for reconstruction of the picture [140]. When an external magnetic field is applied, the precession frequency changes and is directly dependent on the strength of the magnetic field. When a patient is put in the MR magnet, the protons, being little magnets, align with the external magnetic field, some parallel and some anti-parallel to it. Parallel and anti-parallel forces will cancel each other, but as there are more protons in a parallel position, there is always a net magnetic field left. This cannot be measured directly because it is longitudinal to the external field of the MR magnet. The transmission of a radiofrequency pulse that has the same frequency of the precessing protons can cause a transfer of energy to the protons, a process called resonance; this results in more protons being anti-parallel and thus neutralising more protons in the opposite direction, with a decrease in longitudinal magnetisation. The radiofrequency pulse also causes the protons to precess in synchrony, which results in the creation of a new magnetic field in a different (transversal) direction. When the radiofrequency pulse is switched off, the longitudinal magnetisation increases again and the transversal
magnetisation decreases and disappears; the process is called relaxation and is picked up as a radiofrequency signal. Longitudinal and transversal relaxation occur in definite times which are called respectively T1 and T2 and are dependent on tissue composition and structure. Protons can be stimulated with different pulse sequences and the echo listened to several times, to gain information about tissue composition. In addition to their T1 and T2 relaxation times, tissues also differ in their concentration of loosely bound hydrogen: this parameter is called ‘spin density’, and also influences MRI images.

Initially only static images could be obtained, but the more recent advent of fast MRI has allowed to create dynamic images, which can be applied to the study of prolapse.

The advantages of MRI are its excellent tissue contrast, its non-invasive nature, its direct multi-planar imaging, its ability to provide flow information and the complete lack of ionising radiation. The high costs limit the clinical application of MRI. At present, the technique is used in incontinent patients as a research tool only.

Although no studies have formally compared MRI to radiography and ultrasonography in relation to imaging of the bladder neck, the high quality of image obtained suggests that MRI may be more reproducible and less operator dependent.
B) ANATOMICAL FEATURES ASSOCIATED WITH STRESS INCONTINENCE

Imaging techniques have been used in patients with stress incontinence with the aim of identifying the causative anatomical lesions and thus providing a rationale for corrective surgery. Attempts to identify the specific lesions of genuine stress incontinence have failed mainly because of its complex and multifactorial pathology, and the presence of confounding factors such as prolapse. Various theories have been proposed, but few (if any) have withstood the test of rigorous scrutiny. The anatomical features detected with various imaging techniques in association with stress incontinence will be described and discussed.

1- LOSS OF THE POSTERIOR URETHROVESICAL ANGLE

The history of cystography is in great part linked to the observation of this radiological sign. The first to highlight it were Jeffcoate and Roberts (1952) [80], who studied with lateral cysto-urethrography 67 women with stress incontinence and associated prolapse, 23 women with prolapse and without stress incontinence, and 24 normal controls. They found the normal bladder to have a posterior urethrovesical angle of about 100 degrees. In cases of stress incontinence, where great care was taken over the diagnosis (including cystometry), funnelling of the urethrovescical junction and in particular loss of the normal posterior urethrovesical angle were considered typical of stress incontinence. The effects of surgery on the posterior urethrovesical angle were studied and correlated to the clinical success rate. The study concluded that the most significant anatomical deformity associated with stress incontinence is loss of the posterior urethrovesical angle and that restoration of this angle is a feature of successful operations for this complaint. This despite the observation that 20% of patients with stress incontinence did not show this abnormality.
and that restoration of the angle was not observed in the majority of successful anterior colporrhaphies. Other shortcomings were the lack of statistical analysis of the data presented and the lack of reproducibility studies of the imaging method used.

Theoretical support for the association between loss of the posterior urethrovaginal angle and the development of stress incontinence came from earlier cystourethrographic studies on women in labour by Malpas et al (1949) [141]. They studied 32 women in the first and second stage of labour. Most of the information was obtained from lateral views taken with the patient lying down. The urethra was highlighted with a catheter. They found that displacement of the bladder neck depended on descent of the presenting part, which caused the bladder base to roll up from behind forwards until it came in line with the urethra; both bladder base and vesico-urethral junction then moved forwards towards the symphysis pubis. There was very little movement of the bladder neck upwards. The shape of the junction ‘came to resemble a pear and its stalk, in contrast to the normal appearance of an apple and its stalk’. The forward direction of movement of the bladder base and the alteration in the shape of the bladder neck with loss of the posterior urethrovesical angle led them to postulate that extreme stretching of the fascial investment of the posterior aspect of the bladder neck during vaginal delivery was the causative mechanism of stress incontinence.

A subsequent radiological study by Francis (1960) [142] on 83 pregnant women, who performed urodynamics on women complaining of incontinence in order to exclude detrusor instability, also supported the importance of an absent urethrovesical angle as a sign of stress incontinence. This angle was found to be absent in 90% of the women with stress incontinence on radiographs performed in pregnancy and during the puerperium, while it was absent in only 8% of women with no incontinence.
The bead chain technique introduced by Hodgkinson [82], combined with the findings of Jeffcoate and Roberts on the association between stress incontinence and loss of the posterior urethrovessical angle [80], were instrumental in the widespread implementation of urethrocystography in clinical practice. The main impetus for this practice came from the recognition that the standard operation performed at the time for stress incontinence (anterior colporrhaphy), was associated with a relatively high failure rate despite the successful correction of prolapse. Pre- and post-operative X-rays were then performed with the intent of correctly diagnosing stress incontinence by observing altered anatomy not otherwise visible, selecting a suitable surgical procedure for each individual case and assessing post operative results.

Bailey’s observations in three communications (1954, 1956 and 1963) [143-145] are typical of this approach and contributed strongly to its widespread application. In a follow-up of 206 cases treated by colporrhaphy, he was dismayed to discover that only 32 of 83 cases of stress incontinence (38%), were cured after two or more years. He also noted that the cure rate following a ‘modified colporrhaphy’ (in essence an anterior colporrhaphy combined with a suprapubic urethro-vesical suspension similar to the Marshall-Marchetti-Krantz procedure) was higher. On the basis of pre- and post-operative urethrocystograms he sought to determine the reason for this difference in results. Following observations on 350 patients that underwent surgery for prolapse and/or incontinence, he described the normal bladder and urethra as having a posterior urethrovessical angle of 95-100 degrees and a urethral angle of inclination to the vertical of less than 45 degrees. He found stress incontinence to be almost invariably associated with loss of the posterior urethrovessical angle. It is not stated whether the diagnosis of stress incontinence was made after urodynamics. Also it is not stated whether observations were done on normal controls. He attempted to classify the various urethrovessical relationships observed into 7 anatomical groups, in a progressively severe deviation from the ‘normal’ radiological appearance. It is
not stated whether there was a clear correlation between this classification and the severity of symptoms reported by patients. He found the urethral axis (or angle of inclination of the urethra to the vertical axis) to be relevant to the type of surgery required. An inclination of more than 45 degrees was more likely to require an additional suprapubic approach. However he admitted that the interpretation of the X-ray images was not easy and that the observer was 'constantly confronted with what might appear to be atypical cases'. As a result, 'clinical' cure (assessed subjectively) did not always correlate well with 'radiological' cure.

Despite the considerable shortcomings in Bailey's methodology, his conclusions were taken-up by Green (1962) [146], who later further developed them in his widely quoted review on stress incontinence [147], in which cystourethrography was promoted as central to its diagnosis and management. Green's appreciation of the importance of the posterior urethrovesical angle was based on the uncritical acceptance of previous studies and on his own observations on 152 patients that underwent urethrocystography before and after surgery. Despite the absence of normal controls, he concluded that the loss of this angle was a constant feature of stress incontinence. His analysis of Bailey's work led him to consider the angle of inclination of the urethral axis as a very important additional feature. He simplified Bailey's complex anatomical-radiological classification into two basic types: Type I, in which only loss of the normal posterior urethrovesical angle is present; and Type II, where in addition the angle of inclination of the urethral axis to the vertical is greater than 45 degrees. In his retrospective study of 90 patients treated for stress incontinence, he found that the success rate of vaginal surgery for patients with a pre-operative type I anatomic configuration was 81 %, while it was only 48 % for patients with a type II configuration. However, the success rate for patients with type II configuration who had undergone a suprapubic urethrovesical
suspension was high (93%). As a consequence, he promoted cystourethrography as essential to the diagnosis of stress incontinence and to the choice of the type of surgery. He also speculated that the loss of the posterior urethrovesical angle was not only an index of inadequate urethral support, but also the cause of incontinence. He argued that the loss of the posterior angle could render impossible the equal transmission of sudden elevations of intra-abdominal pressure to the proximal urethra, thereby resulting in incontinence.

These views gained widespread acceptance and as a consequence urethrocystography became a routine investigation in the management of incontinence in the 1970s and 1980s.

This purely mechanical concept of stress incontinence was first challenged by Crisp (1965) [148] in a facetious editorial entitled ‘What’s the angle?’, where he argued that to consider the loss of the posterior urethrovesical angle as the cause of incontinence ignores the basic physical laws on resistance (resistance to flow in a pipe is inversely proportional to its diameter and directly proportional to its length; angulation of the pipe does not significantly change this principle) and hydrodynamics (angulation at the bladder neck does not prevent transmission of sudden rises of intravesical pressure to the bladder neck as Pascal’s law dictates that pressure within a container of fluids is transmitted equally in all directions).

Already in 1956, a small but methodologically correct study by Ardran et al [83] had failed to identify any specific radiological features of stress incontinence. They performed serial rapid cystourethrography at rest, on straining and coughing, during voiding and while interrupting the stream, in 16 women with stress incontinence and in 11 normal controls (of which seven were multiparous). Apart from the expected observation of entry of contrast medium into the urethra during coughing or straining in the incontinent group, they found no other distinguishing features. In particular, stress incontinence could not be related to
any loss of the posterior urethrovesical angle or any change of urethral angulation. Their conclusions, which were dissimilar to the other contemporary studies, were largely ignored. Subsequent studies, which for the first time included large numbers of continent controls, have criticised the predictive value of observations made by cystourethrography [56,149-151] or ultrasound [127], when adopting Green’s criteria (i.e. loss of the posterior urethrovesical angle and abnormal urethral angulation). A considerable overlap between continent and incontinent women has been found. In addition, the degree of blinded inter-observer variability for cysto-urethrography was found to be unacceptably high, with a lack of agreement in interpretation between three different radiologists varying from 19 to 54 %, depending on the radiological sign examined [56].

It is now generally accepted that the loss of the posterior urethrovesical angle is related more to pelvic relaxation than to any form of incontinence and that there is no role for static cystourethrography in the routine investigation of female incontinence.

2- BLADDER NECK AS THE MOST DEPENDENT PART OF THE BLADDER

Hodgkinson [82] is the only investigator that has attached great importance to this finding in relation to stress incontinence. He studied by means of bead chain urethro-cystography a small number of women with and without stress incontinence. He demonstrated that both continent multiparae and patients with stress incontinence had deficient support to the bladder base, as shown by its pointed appearance at rest and its mobility during straining. The difference between the two groups was that in continent multiparae, straining caused a downward and backward rotation of the bladder in its relation with the urethra. This movement of the bladder did not occur in patients with stress
incontinence, and this resulted in the internal urinary meatus plunging to the most
dependent bladder level, where it was subject to maximum stress. He concluded that these
changes in urethra-vesical relationships were important factors in relation to continence.

His findings, unlike his technique, did not influence clinical practice at the time.
Other investigators found a large overlap between women with stress incontinence and
continent controls when observing this radiological sign [56, 150]. The sign itself was
found to be poorly reproducible when assessed by three different observers [56].

3- OPEN BLADDER NECK

This imaging feature has also been termed ‘funnelling’ of the bladder neck. Initially it
was thought to be a feature of stress incontinence when observed during straining. Later,
the observation of funnelling at rest was considered diagnostic of stress incontinence due to
sphincter damage as opposed to an abnormally mobile bladder neck.

The association of bladder neck funnelling with stress incontinence was made in the early
days of cystography [78]. Also Jeffcoate and Roberts [80] thought that it was a feature of
stress incontinence, but not as important as loss of the posterior urethrovesical angle; they
did not report its incidence in any of the groups studied, including the stress incontinent
group, and they did not specify whether funnelling was visible at rest or only during
straining. Bethoux and Bory [84] observed funnelling of the bladder neck with colpo-
cystourethrogramy in women with stress incontinence during pelvic floor contraction but
more during straining; they considered it a feature of anterior pelvic floor weakness. Their
work led to the widespread use of colpo-cystourethrogramy in Denmark in the 1980’s and
the detection of funnelling by these means became an indication for a colposuspension (as
opposed to a vaginal repair) in Danish gynaecological practice [87].
Lazarevski et al [85], using the same technique on 586 patients with prolapse and stress incontinence, also found that funnelling was frequently associated with stress incontinence (no incidence reported); however, he observed that funnelling could occur also in continent patients.

Hutch [152] suggested that an essential feature of continence was the presence of a flat bladder base. This observation could be considered as analogous to the absence of funnelling. He argued that the internal sphincter (which he called ‘basal plate’) could function only if maintained in a ‘non-funnelled’ configuration, with all the concentrically arranged muscle fibres working together. He based his observations on 500 lateral cystograms; however the features of these patients were not specified.

The lack of matched control groups and rigorous assessment of the techniques used in all the above studies, was noticed by Fantl et al [56], who found the technique to have an unacceptably high inter-observer variability when assessed by three independent radiologists. In all cases where the three radiologists were in agreement, the diagnosis was detrusor instability (on subsequent urodynamics). They concluded that funnelling of the bladder neck was not typical of stress incontinence. Similar observations on the poor reproducibility of this observation was also made by other authors [90,92].

It should also be noted that the observation of funnelling in the absence of simultaneous urodynamics is intrinsically flawed, as funnelling may occur as a consequence of a detrusor contraction; funnelling can therefore be reliably linked to stress incontinence only if imaging is combined to cystometry. This was performed by Versi et al [76], who assessed by VCU bladder neck opening in 98 continent women (with a negative pad test) presenting with menopausal symptoms: 50% of them had an open bladder neck while coughing. They therefore questioned the importance of the internal sphincter for the maintenance of continence and suggested that an open bladder neck during stress should not be considered diagnostic of stress incontinence.
Funnelling of the bladder neck at rest (as opposed to during straining) was studied by Blaivas and Olsson [153] by means of VCU, in order to distinguish incontinence due to hypermobility (they called this a 'hernia' of the bladder neck and defined it as a bladder neck descent of more than 2 cm) from the much less common form of incontinence due to urethral sphincter dysfunction. They found that 21% of 181 patients with stress incontinence that were assessed by VCU had a bladder neck and proximal urethra which were open at rest. They called this 'type III' stress incontinence and included it in a modified version of Green's original classification.

They argued that patients with this type of incontinence have a damaged urethral sphincter and have a high failure rate after suprapubic suspension procedures. The study was retrospective and did not include normal controls. In addition, patients with the various types of incontinence were not randomised to different treatments. Despite these shortcomings, 'type III' stress incontinence remains a term in current use (particularly in the USA) as a synonym of sphincter damage (intrinsic sphincter deficiency). However, it is not included in the standard ICS terminology.

The concept that the demonstration of an open bladder neck at rest can diagnose intrinsic sphincter deficiency (ISD) has been challenged by other studies. Versi [74] investigated by VCU at rest and in the erect position 147 symptomatic women presenting to a urodynamic clinic; the prevalence of an open bladder neck was 21%; although there was an association with stress incontinence, it could not be considered as a diagnostic sign. Similarly, Dupont et al [154] observed 55 patients with and without stress incontinence by means of VCU at rest and during straining. They found that in patients without stress incontinence, 31% had an open bladder neck at rest and 62% with strain, while in patients with stress incontinence, 75% had an open bladder neck at rest and with strain.
In conclusion, although funnelling of the bladder neck at rest or during straining seems to occur more frequently in patients with genuine stress incontinence, the evidence does not suggest that it should be considered a predictor of continence, incontinence, degree of severity of incontinence or type of incontinence.

Whether an open bladder neck in an asymptomatic patient should be considered a risk factor for the development of incontinence is not yet clear. Chapple et al [75] investigated 29 asymptomatic young nulliparous women by vaginal ultrasonography and found that 21% of them had an open bladder neck. They postulated that they may constitute a group at risk of future development of stress incontinence. Prospective longitudinal studies are needed to clarify this issue.

4- INCREASED BLADDER NECK MOBILITY

Hypermobility of the bladder neck has been associated with stress incontinence since the early times of cystography [78], although Jeffcoate and Roberts [80] thought that it was more representative of prolapse than incontinence.

McGuire et al (1976) [155] were the first to highlight the importance of hypermobility in relation to stress incontinence. They performed VCU on 125 women with stress incontinence, and found that the most common abnormality (present in 80% of patients) was hypermobility of the proximal urethra. This was defined as ‘descent of the bladder neck below the pelvic diaphragm on straining’ (not quantified).

Bergman et al [103] used rectal ultrasonography to study bladder neck mobility in 44 patients with stress incontinence, 23 patients with detrusor instability and 24 continent controls. Bladder neck descent on straining was measured directly on the screen; the investigator was blinded to the diagnosis. They found that 86% of stress incontinent
patients had bladder neck descent during stress of one centimetre or more. The specificity of this finding for a diagnosis of stress incontinence was 91%. As a consequence, a bladder neck drop of 1 cm or more is now often used as a measure of hypermobility.

Poor support of the proximal urethra and bladder neck is considered the most common cause of stress incontinence [4]. Support for this concept comes from the observation that corrective surgery leads to a cure by providing support to the bladder neck. The demonstration of a ‘hypermobile’ bladder neck should therefore constitute an important diagnostic aid in the evaluation of patients with stress incontinence. To date, it is not clear as to what constitutes a ‘hypermobile’ bladder neck, as no study on hypermobility has fulfilled all the scientific requirements, which should include measurement of abdominal pressure generated during Valsalva, assessment of reproducibility of the imaging method, presence of controls matched for age, parity and degree of prolapse and an investigator blinded to the diagnosis (table 1). In addition many studies have been conducted using an endoprobe which may have prevented mobility.

It is difficult therefore to draw any conclusions on whether increased bladder neck mobility, as assessed by present imaging techniques, is the ‘cause’ or at least a sign of stress incontinence and not just a measure of prolapse. The best conducted studies [107,161] suggest at most a weak association between increased bladder neck mobility and genuine stress incontinence. There is therefore no evidence to support the routine imaging assessment of bladder neck mobility in patients with stress incontinence. This notwithstanding, the assessment of bladder neck mobility might be useful with regards to the choice of treatment for the incontinent patient after failed surgery.
Table 1. Association of the sign of increased bladder neck mobility with genuine stress incontinence.

<table>
<thead>
<tr>
<th>Method</th>
<th>Degree of association</th>
<th>Controls</th>
<th>Valsalva measured</th>
<th>Observer blinded</th>
</tr>
</thead>
<tbody>
<tr>
<td>McGuire et al (1976) [155]</td>
<td>Strong</td>
<td>No controls</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>VCU (125)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bergman et al (1988) [103]</td>
<td>Strong</td>
<td>Unmatched Controls</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Rectal US (91)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Richmond &amp; Sutherst (1989) [156]</td>
<td>Weak</td>
<td>Controls matched for parity</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Rectal US (84)</td>
<td></td>
<td>Unmatched Controls</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Carey &amp; Dwyer (1991) [157]</td>
<td>Weak</td>
<td>Controls matched for parity</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Bead-chain cystography (36)</td>
<td></td>
<td>Unmatched Controls</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Johnson et al (1992) [158]</td>
<td>Strong</td>
<td>Unmatched Controls</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Vaginal US (343)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mouritsen &amp; Rasmussen (1993) [125]</td>
<td>Weak</td>
<td>Unmatched Controls</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Vaginal US (61)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weil et al (1993) [127]</td>
<td>Weak</td>
<td>Controls matched for parity</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Vaginal US (55)</td>
<td></td>
<td>Unmatched Controls</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kiiholma et al (1994) [159]</td>
<td>Weak</td>
<td>No controls</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Perineal US (38)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hol et al (1995) [107]</td>
<td>Weak</td>
<td>Controls matched for age &amp; parity</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Vaginal US (100)</td>
<td></td>
<td>Unmatched Controls</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Meyer et al (1996) [160]</td>
<td>Weak</td>
<td>Controls matched for parity</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Perineal US (214)</td>
<td></td>
<td>Unmatched Controls</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Athanasiou et al (1996) [161]</td>
<td>No association</td>
<td>Controls matched for age, parity &amp; vaginal descent</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Perineal US (87)</td>
<td></td>
<td></td>
<td></td>
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</table>

The significance of the direction of movement has been explored by Athanasiou et al [137]. Their comparison by perineal ultrasonography of continent and incontinent women has shown that there was no difference between the two groups with regards to bladder...
neck mobility, but that the direction of movement was more likely to be downwards in the incontinent group. Unfortunately, Valvalva was not standardised in this study.

Bladder neck hypermobility has been evaluated during pregnancy as a possible risk factor for the development of stress incontinence after delivery by King and Freeman [162]. They performed perineal ultrasound on 116 primigravidae and found that increased bladder neck mobility was associated with an increased risk of post partum stress incontinence. An angle of rotation with Valsalva greater than 10 degrees (seen in 31 women antenatally), was associated with a 50% risk of post partum stress incontinence. The diagnosis of stress incontinence was not made objectively. Also it is not known whether post-partum incontinence necessarily leads to genuine stress incontinence in later life. Before any conclusions can be drawn on this issue, these results need to be replicated by other studies and patients need to be followed up longitudinally.

5- REDUCED URINARY SPHINCTER DIMENSIONS

A mean reduction in urinary sphincter dimensions in women with genuine stress incontinence has been reported using intra-urethral ultrasound [138], perineal ultrasound [135], and three-dimensional vaginal ultrasound [137], as compared to continent controls and women with detrusor instability. There is however considerable overlap of values.

Intrinsic sphincter deficiency is considered a less common cause of stress incontinence [4]. The measurement of the urinary sphincter dimensions could constitute a useful diagnostic tool and could improve our understanding of the mechanisms of continence. Perineal ultrasound has been used to study the predictive value of urethral dimensions (length, width and cross-sectional area) for the detection of the low pressure urethra. Decreasing changes in urethral diameters or cross-sectional areas were a sensitive (95%) but non-specific (59%) test for the detection of the low pressure urethra [163].
While these findings do not appear to have an immediate clinical application and need further evaluation, nonetheless they highlight the importance of the external (distal) urethral sphincter in the maintenance of continence.

**6- SOFT TISSUE DAMAGE AS SEEN BY MRI**

Before the advent of MRI, all direct anatomic knowledge in relation to stress incontinence was derived only from cadaver dissection and intra-operative observations. MRI, with its excellent soft tissue differentiation and capacity for multiplanar imaging, has provided a new tool for characterising important anatomical features.

Various types of damage to the muscles and fascial attachments of the pelvis have been identified: thinned and distorted urethropelvic ligaments [139,164-167], increased separation of the urethra from the pubic bone [139,164-166], loss of angulation and thinning of the levator ani [69,164,165,168], lateralization of the pubococcigeus muscles [169], and paravaginal defects [61,69,166,167].

Most studies so far are small in numbers, and contain no matched controls. Investigators are not blinded to the diagnosis and there is no agreement as to what constitutes normal anatomy [168]. Moreover, as evidence of the multi-factorial origin of stress incontinence, no single soft tissue lesion has been constantly associated with stress incontinence.
CONCLUSIONS

Imaging for stress incontinence has been the subject of dramatic technical evolution and has greatly contributed to the understanding of the mechanisms of continence. Ultrasound is a practical alternative to radiography for most applications. MRI is an interesting new research tool.

The clinical application of imaging techniques in relation to stress incontinence is at present limited because no associated anatomical lesions have been conclusively identified. Further work in this area is required.
CHAPTER 4

THE INVESTIGATION OF THE LOWER URINARY TRACT:

URODYNAMICS
The term ‘urodynamics’ was first used by Davis and Zimskind in 1962 in relation to upper urinary tract function [170]. In modern medical practice, the term describes ‘the medical science concerned with the physiology and pathophysiology of urine transport from the kidneys to the bladder as well as its storage and evacuation’, as defined by Susset in 1985 [170]. In practical terms, ‘urodynamics’ generally refers to tests that provide objective information about lower urinary tract function [4]. These include simple demonstration of urinary leakage on physical examination and quantification of urinary loss using a pad, but also more complex urinary flow studies (uroflowmetry), urethral and bladder pressure studies (urethral pressure profilometry and cystometry), imaging studies and electrophysiological studies.

Cystometry, urethral pressure profilometry and uroflowmetry will be described and their clinical applications discussed. Imaging studies have been described in chapter 3. Electrophysiological studies are beyond the scope of this discussion.

1- CYSTOMETRY

Cystometry is the method by which the pressure/volume relationship of the bladder is measured [1]. Although the first report of intravesical pressure measurement was made by Dubois in 1876 [170], the first observations on lower urinary tract function using cystometry were first made by Mosso and Pellacani in 1882 [171]. The bladder pressure was measured by recording the movements of a suspended test-tube filled with fluid and whose contents were connected to the bladder via a fluid filled catheter. Bladder contractions caused small fluctuations of volume within the tube thus altering its weight. These pioneers were already aware of the limitations of simple cystometry, mainly the presence of artefacts produced by rises in intrabdominal pressure, which they called ‘passive’ bladder contractions. They distinguished them by observing that ‘true’ bladder
contractions were of slower onset and caused a more gradual rise in pressure. This technical problem was overcome by Denny-Brown and Robertson in 1933 [172]. In addition to measuring bladder pressure, they performed simultaneous recording of intrabdominal pressure using a rectal catheter connected to a manometer, thus leading to the later development of ‘subtracted’ cystometry, where artefacts can be eliminated and the ‘true’ detrusor pressure observed.

Present day cystometry does not differ in principle, although modern technology has perfected the ease and accuracy of measurement. Bladder and rectal pressures are measured using fluid filled catheters connected to external pressure transducers or directly with microtip transducers. The detrusor pressure is obtained electronically by subtracting the intrabdominal pressure from the bladder pressure. A graphic record of the measured pressures is obtained.

The practice of cystometry requires the patient to be awake, unanaesthetized and neither sedated nor taking drugs that affect bladder function [1]. After the patient has voided, a filling catheter and a pressure catheter are inserted into the bladder using sterile technique (usually transurethrally, but also percutaneously). The residual urine is measured. The abdominal pressure catheter is inserted in the rectum or in the vagina. The patient is placed in the desired position for cystometry (supine, sitting or standing). If fluid filled lines are used, the detrusor pressure is set to zero in correspondence to the reference point, which is at the level of the superior edge of the symphysis pubis. Correct functioning of the equipment is checked by asking the patient to cough and observing the effect on the pressures measured. The bladder is then filled with liquid (sterile water) or gas. The rate of filling can be slow (up to 10 ml/min), medium (10-100 ml/min) or rapid (over 100 ml/min). The patient’s bladder symptoms are progressively recorded. These include the first desire to void, the normal desire to void (comfortably full bladder), a strong desire to void and the
cystometric bladder capacity. Urgency (defined as a strong desire to void accompanied by fear of leakage) or pain are noted. The patient coughs at intervals to demonstrate stress incontinence. Provocative manoeuvres are performed to demonstrate unstable detrusor contractions if none are observed during bladder filling. These include coughing, listening to the sound of running water, washing hands in cold water and changing position (e.g. standing). Incontinence in relation to unstable detrusor contractions is noted. The patient is then asked to void in private. The filling catheter is removed beforehand.

As suggested by the ICS [1], a diagnosis of genuine stress incontinence is made if loss of urine is seen to occur during rises in intrabdominal pressure, in the absence of a detrusor contraction. Overactive detrusor function is diagnosed if involuntary detrusor contractions occur during the filling phase, either spontaneously or after provocation. Contractions are phasic in type. The size of the pressure rise required to obtain a diagnosis is not now specified by the ICS. In its original report (1976), a pressure rise exceeding 15 cmH2O was considered significant, but for lower pressure elevations clinical judgement should be exercised [173]. A gradual increase in detrusor pressure without subsequent decrease is considered a change in compliance.

It has been recognised that variations in the methods can lead to variability of results [173]. For instance, fast bladder filling or using liquids at room temperature are more likely to lead to unstable detrusor contractions than slow filling and liquids at body temperature [4]. The performance of cystometry in supine position is considered less likely to reveal unstable contractions, as opposed to the sitting and standing positions [4]. Also, fast filling may lead to artificially high bladder pressure at the end of filling, thus causing a false impression of 'low compliance' [4]. For this reason, the ICS advises that any variations that may affect the results of cystometry should be specified. These include the method of
access to the bladder, the fluid medium used and its temperature, the rate of filling, the position of the patient during the test, the type, number and size of catheters, and the type of measuring equipment.

Cystometry was born as a research tool and it was initially applied to the investigation of the physiology and pathophysiology of the lower urinary tract [171,172]. Its first routine clinical application (Rose, 1927) was the study of the neurologically abnormal bladder [170]. It is however only in recent times that cystometry has gained widespread acceptance. Bates et al, in 1970 [94], described a simplified technique of simultaneous cystography and cystometry. They also highlighted the potential of the test to investigate the pathophysiology of female stress and urge incontinence, distinguish these two conditions in selected patients, and assess objectively the results of treatment. Since then cystometry, with or without simultaneous imaging, has gradually become a routine test for the assessment of women with lower urinary tract symptoms. This process has been prompted also by the recognition that the bladder is an ‘unreliable witness’ (Bates, 1970) and that cystometry is required to obtain a diagnosis.

The value of a clinical test is based on its reproducibility, on its sensitivity and specificity to identify a pathological condition, and on the improvement in quality of care based on its results. Cystometry has never been subjected to rigorous scientific assessment. Booth et al [174] have retrospectively assessed the results of cystometry on 64 patients after repeated testing (mean interval 19 months), and found that in 95% of cases the diagnosis remained unchanged. They concluded that cystometry was highly reproducible with regards to qualitative information, although quantitative information could be variable. Lose and Thyssen [175] have studied the reproducibility of cystometry over two consecutive tests on 36 women. They found that first desire to void and maximum bladder
capacity increased significantly at the second filling. There was agreement between the two investigations with regards to detrusor overactivity in 89% of cases, with more cases detected after the second test (14 of 36) as compared to the first (11 of 36). No studies have assessed the reproducibility of cystometry in a blinded fashion.

Cystometry is often used routinely to investigate female urinary incontinence and other lower urinary tract symptoms. Various studies have shown that the use of cystometry results in a re-allocation of patients to a different diagnosis, as opposed to simple history and physical examination [5,6,9,176-179]. The clinical value of cystometry in this context has been assessed retrospectively by Shepherd et al [178], who showed that treatment was more likely to be successful if decisions were based on urodynamic tests.

The most common application of cystometry in urogynaecology is the investigation of urinary incontinence, with the view of excluding detrusor instability before incontinence surgery. Reliance on symptoms for this purpose is thought to be inaccurate. Sand et al [6] found that only 22% of incontinent women presenting for urodynamics complained of pure stress incontinence with no other associated symptoms. Of those, only 58% had a diagnosis of genuine stress incontinence after cystometry, with 18% having mixed incontinence, and 16% having isolated detrusor instability (in 7% no incontinence was demonstrated).

Some believe that only women who have a history of stress incontinence alone, with demonstrable stress incontinence on examination, do not require cystometry, as they are invariably found to have genuine stress incontinence and stable bladders [179].

The value of cystometry for the purpose of selecting patients for incontinence surgery has been recently questioned by Black et al [180], who found similar subjective outcomes after surgery regardless of whether or not women underwent urodynamic investigations beforehand. It is difficult to draw firm conclusions from this study, as various operations (with different success rates and morbidity) were considered together, and objective outcomes were not included. Until more accurate means of diagnosis become available, the
value of cystometry in this context can only be resolved by a randomized study which evaluates the results of treatment with and without prior cystometry.

Despite the invasive nature of cystometry, its morbidity in women has been shown to be low. Transient irritative symptoms occur in one third of patients. A small minority may develop bacteriuria as a result of the test [181].

Attempts have been made to overcome the ‘unphysiological’ nature of cystometry and improve its diagnostic accuracy. The advent of portable storage devices has led to the development of ambulatory monitoring. This technique allows the bladder to fill naturally with urine at body temperature and enables patients to be fully ambulant. The insertion of bladder and rectal pressure catheters is still required. Using the standard criteria of normality for static cystometry, ambulatory monitoring has been shown to detect a higher incidence of abnormalities [182]. However, considering the very high incidence of abnormalities in asymptomatic volunteers [182], the use of these criteria may be inappropriate when interpreting data from ambulatory monitoring. To date, the place of ambulatory monitoring in routine clinical practice has not yet been established.

2-UROFLOWMETRY

The first observation of urinary flow was made by Rehfisch in 1897, using an air displacement technique in order to time the onset and finish of micturition [183]. The first uroflowmeter was developed by Drake in 1948. Patients were made to void in a receptacle and its change in weight as urine flowed was recorded on a kymograph [170]. In 1956 von Garrelts used an electronic transducer to convert the changes in weight caused by the flow of urine onto a photokymograph [184].
While various other methods have been subsequently devised, modern flowmeters remain based on the use of weight transducers, although spinning discs are a valid alternative. This latter method relies on the recording of the change of power required to maintain a disc rotating at the same speed while its movement is hampered by the flow of urine.

Uroflowmetry provides a graphic record of urine flow, together with information on volume voided, time of void, and speed of flow (peak and average flow rates). The immediate reproducibility (test re-test variation) of uroflow data in women has been found to be good [175].

The study of urinary flow has been mainly applied to the male for the evaluation of outflow obstruction. Uroflowmetry is considered less useful in women, as voiding dysfunction is less common. This application will be discussed in chapter 6.

3- URETHRAL PRESSURE STUDIES

Considering the important role played by the urethra in the maintenance of continence and during micturition, the measurement of the pressure within the urethra should in theory have important applications. The difficulty lies in the fact that the urethra is not a fluid filled organ and the pressures measured within the urethra are very variable and often reflect the tension of surrounding structures.

Bonney is credited with the first attempt to measure intraurethral pressure in 1923 by measuring the pressure required to force liquid up the urethra [170]. Denny-Brown and Robertson in 1933 [172] first recorded simultaneously intraurethral and intravesical pressure using a catheter positioned within the urethra through which a ureteric catheter was passed into the bladder. Karlson in 1953 [185] first made accurate simultaneous
recordings of bladder and urethral pressures using pressure transducers which had originally been devised for intrauterine pressure measurements.

The methods used to record pressure include balloons, infusion methods and catheter tip transducers. Balloons are mounted concentrically on a catheter which is dragged along the urethra. They are considered inaccurate as they result in the averaging of pressure variations which should be recorded separately. Infusion methods measure urethral pressure indirectly by recording resistance to a constant flow of fluid infused from side holes in a catheter that moves along the urethra. The response of this system is too slow to allow an accurate measurement of pressure variations during sudden rises in intra-abdominal pressure. Catheter tip micro-transducers constitute the method in current use. They allow for recording of rapid pressure changes, but they measure the tension of tissues at right angle to its surface. As a consequence, the pressure is dependent on the orientation of the transducer and is not a measurement of pressure over 360 degrees. Most catheters in use have two sensors mounted six centimetres apart. This allows for simultaneous intravesical and intraurethral measurements. Artefacts are unfortunately common and difficult to avoid [186]. They are due to the difficulty in standardising variables such as the position of the transducer, the position of the patient, the rate of withdrawal of the catheter, the bladder volume and the cough strength.

Urethral pressure measurements can be obtained in isolation at rest (static profilometry) or simultaneously with bladder pressure during coughing (stress profilometry). Numerous parameters can thus be obtained. Those more commonly used are the maximum urethral pressure (the largest pressure measured in the urethra), the functional urethral length (the length of the urethra in which urethral pressure exceeds bladder pressure), the maximum
urethral closure pressure (the largest pressure difference between the urethral pressure and the bladder pressure) and the pressure transmission ratio (the percentage of increment in urethral pressure during a cough in relation to the simultaneous increment of bladder pressure).

The use of urethral pressure profilometry (UPP) has been extensive especially in the field of research and has advanced our understanding of lower urinary tract physiology and pathophysiology. However, its clinical use has been limited. Urethral diverticula, urethral strictures and urethral instability can be diagnosed using UPP, but they are uncommon. Women with genuine stress incontinence have been shown to have a highly statistical difference in UPP variables as compared to continent women, but unfortunately there is a great deal of overlap between the two groups, thus limiting the value of UPP for the study of incontinence. UPP is commonly used to diagnose urethral sphincter damage, commonly defined as a maximum urethral closure pressure lower than 20 cmH2O, with the aim of predicting the success of incontinence surgery and guiding the choice of surgery. This will be further discussed in chapter 5.
CHAPTER 5

THE OPERATION OF COLPOSUSPENSION
Since Baker-Brown described suprapubic cystostomy for the treatment of stress incontinence in 1864, over 150 different operations have been devised for the treatment of this condition [12]. For its high and long lasting success rate, the colposuspension operation is now considered the 'gold standard' against which other procedures are measured. Uncertainties still exist as to how the procedure works and which patients benefit the most.

In this chapter, the history of the operation of colposuspension will be reviewed and the technique and its variations will be described in detail. The mechanisms by which the procedure works and the risk factors for failure will be discussed. Methods of outcome assessment will also be discussed.

1- HISTORY AND TECHNIQUE

Retropubic suspension procedures were first performed by Hepburn (1927) [187] and later by Miller (1945) [188] for the treatment of mucosal eversion of the urethra. Miller observed that one of his four patients had improved urinary continence after the procedure and suggested that the operation could have a place in the treatment of urinary incontinence.

Marshall, Marchetti, and Krantz (1949) [14], a Urologist and two Gynaecologists, were the first to describe and popularise retropubic suspensions for the treatment of stress incontinence. They recognised that bladder neck elevation and fixation could be an important factor for continence control when they noticed that a male patient, totally incontinent after an abdomino-perineal removal of rectum and a transurethral resection of the prostate, could regain control by applying perineal pressure. The patient underwent successful retropubic suspension, and they subsequently performed the procedure on 49
more patients, 45 of which were female. The operation involved the placement of three
sutures of no. 1 chromic catgut along the urethra on either side; the urethral wall was
transfixed by the suture, without entering the lumen; an additional suture was placed on
either side of the bladder neck. The sutures were then anchored to the periosteum of the
pubis and to the cartilage of the symphysis posteriorly, avoiding undue tension. Additional
sutures were placed on the bladder wall and attached to the rectus muscles, in order to
obtain maximal bladder elevation. They reported a 64% success rate, and they attributed
their failures to poor patient selection, as only 38 of 49 patients were thought to have stress
incontinence. They subsequently modified their technique and advised against the
placement of sutures through the urethra in order to avoid trauma [189]. To this day, the
Marshall-Marchetti-Krantz procedure remains a commonly performed operation for the
treatment of stress incontinence. Recognised disadvantages are the difficult surgical
definition of the edges of the urethra, the poor hold of the posterior aspect of the pubis for
suture insertion, and the development of osteitis pubis in 0.9% to 10% of cases [28].

In order to overcome this problems, Burch in 1961 proposed some modifications [13].
He suggested that the vaginal fascia lateral to the urethra should be the lower point of
anchorage, and that its dissection from the bladder could be aided by the insertion of the
operator's finger into the vagina. With regards to the upper point of anchorage, he initially
tried the ‘white line’, where the levator muscle becomes attached to the lateral wall of the
pelvis; this however offered a poor hold. He then found Cooper's ligament to be a much
firmer structure, and used it on 45 women with stress incontinence, with a 100% success
rate. Three no. 2 chromic sutures were placed on each side.

He also noted that the attachment to Cooper's ligament provided a more lateral and higher
point of fixation, and used it for the correction of cystocele on eight patients with good
results. He called the procedure ‘Cooper's ligament urethrovaginal fixation’ and thought
that it should be considered for the primary treatment of stress incontinence rather than being reserved for failures. The term ‘colposuspension’ for this procedure appears to have been used for the first time by Turner-Warwick and Whiteside in 1970 [190].

In his description of the colposuspension operation, Burch did not specify operative details such as the exact location of the sutures in relation to the urethra and the bladder neck, the site of placement of the sutures on the pectineal ligaments (medial or lateral), the amount of bladder neck dissection needed, or the ideal amount of bladder neck elevation and suture tension. As a consequence, variations of the colposuspension operation were described by different surgeons. The most commonly performed techniques are linked to the names of Stanton, a gynaecologist from London [37], and Tanagho, a urologist from San Francisco [31], and were described in 1976.

Stanton et al [37] described placing three no.1 Dexon sutures on either side: the first suture was placed caudally to the bladder neck, the second at the bladder neck, and the third cephalad to the bladder neck. The sutures were tied so as to approximate the paravaginal fascia to the ipsilateral ileopectineal ligaments (figures 7 and 8). In addition, two no.1 Dexon sutures were placed transversely across the bladder neck, to plicate it. Bladder neck plication was subsequently discontinued [191]. In both reports, patients were extensively investigated before surgery and scrupulously followed-up afterwards. Success was assessed subjectively and objectively and complications were reported in detail. Stanton’s scientific approach contributed to the acceptance of the colposuspension operation in the United Kingdom, and his technique has gained wide acceptance.
Tanagho’s modifications aimed to prevent urethral compression and avoid surgical damage to the urethral sphincter [31]. To that end, he advised minimal bladder neck dissection, and the placement of sutures as far laterally as possible on the ilio-pectineal ligament. Two no.1 Dexon sutures were placed on either side, opposite the mid-urethra, and at the level of the bladder neck. Tension was avoided, and tissue approximation to the ilio-pectineal ligament was not considered essential. After tying the sutures, there had to be
enough room between the pubic bone and the urethra to accommodate two fingers.

Tanagho's recommendations were generally adopted by surgeons in America in the belief that they helped to prevent post-operative voiding dysfunction.

A formal comparison between Stanton's and Tanagho's techniques has never been performed. Objective intra-operative measures that would allow such a comparison have yet to be proposed.

Other technical details of the colposuspension have not been clarified. The number of sutures placed (between one and four each side) and the type of suture material used (absorbable, slowly absorbable or permanent) have never been subjected to randomized comparison. Although chromic catgut has been used with good results by Burch [13] and has the advantage that it will dissolve should inadvertent entry into the bladder occur, there are concerns in relation to the duration of tensile strength. Slowly absorbable sutures such as polyglycolic acid (Dexon) and polyglyconate monofilament (Maxon), or nonabsorbable sutures such as polybutylate-coated polyester (Ethibond) are generally preferred. A retrospective comparison has not shown differences in cure rate and complications between these types of suture material [192]. In a review of 17 studies which specified suture material, there was no difference in cure rate between studies that used absorbable sutures and those that used permanent sutures [193].

Vancaillie and Schuessler (1991) were the first to report on the colposuspension operation performed laparoscopically [194]. Since then, the procedure has been taken up by several enthusiasts. The anatomical result achieved by laparoscopic colposuspension is similar to the open procedure. It is however not yet clear whether the investment in technology and surgical skills required for laparoscopic surgery will result in substantial
benefits for the patients treated [195]. Large multi-centre trials comparing the open and laparoscopic methods are being carried out.

2- MECHANISM OF ACTION

Despite the lack of conclusive evidence from anatomical and imaging studies, it is generally accepted that genuine stress incontinence results from the anatomical displacement of the bladder neck and proximal urethra outside of its normal intrapelvic location above the urogenital diaphragm [4]. This is thought to be associated with increased bladder neck mobility. The changes in bladder neck position and mobility prevent efficient transmission of intra-abdominal pressure rises to the urethra, thus causing the bladder pressure to exceed the maximum urethral pressure with resulting urinary leakage [53].

As a consequence, surgical procedures for stress incontinence aim to reposition the urethra in its normal intrapelvic location. The operation of colposuspension has been shown to elevate the bladder neck and to fix it into a high retropubic position. Penttinen et al [196] performed cystourethrography on 29 patients before and after colposuspension. Surgery resulted in a mean bladder neck elevation of 16 mm, and in a reduction of bladder neck mobility during straining from a mean of 24 mm down to a mean of 5 mm. Carey and Dwyer [157] also performed cystourethrography before and after 16 colposuspensions. It can be extrapolated from their data that surgery resulted in mean bladder neck elevation of 12.2 mm (range 2-20 mm), and in a reduction of bladder neck mobility during straining from a mean of 13.5 mm down to a mean of 3.2 mm. Enzelberger et al [197] used introital ultrasonography to measure bladder
neck position and mobility before and after colposuspension in 36 patients. The bladder neck was elevated by a mean of 9 mm and mobility during straining was reduced from a mean of 15 mm down to 3 mm.

Exactly how this anatomical changes result in improved continence remains a matter of debate.

Pressure-flow studies have consistently shown that after the operation of colposuspension there is a significant increase in detrusor pressure and a reduction in peak flow rate [24,25,88,198-203]. This suggests the introduction of an element of obstruction. It is however unlikely that obstruction is required in order to achieve continence. Similar degrees of obstruction have been shown in patients whose operations were successful and unsuccessful [199,204]. Also, successful surgery is often seen in patients that do not show any evidence of obstruction after surgery [199,205], and failure can be seen in patients with urodynamic evidence of increased urethral resistance [198].

There is no clear evidence that continence is achieved by improving the sphincter mechanism, despite experimental evidence that stretching of the urethra causes an increase in urethral functional length and urethral closure pressure [206]. The effects of colposuspension on urethral sphincter function have been found to be inconsistent. In some studies, colposuspension was found to increase urethral functional length and maximum urethral closure pressure (MUCP) [205,207-209]. In one study, colposuspension was found to increase urethral functional length but not urethral closure pressure [210]. However, urethral pressure variables have been found to be unchanged in most studies [19,197,199,203,211-215], suggesting that it is unlikely that continence is obtained by improved urethral function. Moreover, in individual cases, there is a poor correlation between success and changes in static urethral pressure profilometry [205].
Long-term follow-up studies [25,26,216] have reported a significant reduction of MUCP at rest both after successful and unsuccessful operations. It is not clear whether this is an effect of surgery or whether it represents a physiological reduction associated with ageing [217].

Enhorning [53] suggested that continence in normal women is maintained by **effective transmission of intra-abdominal pressure to the proximal urethra** and that successful surgical treatment of stress incontinence is due to relocation of the proximal urethra into the intra-abdominal pressure zone. The operation of colposuspension has been shown to enhance pressure transmission ratio to the proximal urethra during stress urethral pressure profilometry [19,20,26,196,197,199,203,209,212-216,218]. This change has been reported to occur in successful cases but not in failures [19,26,199,209,214-216,218]. Improved transmission ratio may not however signify restoration of normal anatomy and continence mechanisms, as the colposuspension operation enhances pressure transmission mainly at the proximal urethra, while in normal continent women maximal pressure transmission occurs at the mid-urethra [57,199]. Moreover, pressure transmission ratios greater than 100% have been reported after the colposuspension operation [196,199,215], thus suggesting an improvement on simple passive transmission of intra-abdominal pressure.

It has been suggested that colposuspension works by creating a new continence mechanism (Hertogs and Stanton [219]). These authors studied the effects of a modified speculum placed in the vagina of 48 patients cured by colposuspension. When placed behind the urethra (high in the posterior fornix) in order to interrupt the posterior route of abdominal force transmission, stress incontinence was demonstrated in 90% of patients. Urethral compression against the hard surface of the pubis bone by the posterior structures during stress might explain why pressure transmission ratios greater than 100% have been
reported after colposuspension. This study also highlights the importance of the structures behind the urethra as a compressive force.

**Elevation of the bladder neck** with apposition behind the pubic bone is a recognised aim of the colposuspension operation. The position of the bladder neck in relation to the pubic bone after surgery has been studied in relation to success and failure.

Hertogs and Stanton [220] have compared by means of urethrocystography 17 successful cases with 10 failures after colposuspension. Despite considerable overlap with regards to bladder neck position, the bladder neck was significantly more anterior (closer to the postero-superior surface of the symphysis pubis) after successful colposuspension, than after failure. Successful cases were elevated by a mean of 17.3 mm above the lower border of the symphysis, while failures were elevated by a mean of 11.1 mm. The difference was not statistically significant. They concluded that bladder neck approximation to the pubic bone was more important than elevation with regards to outcome of surgery.

Mouritsen and Rasmussen [125] reached opposite conclusions after performing vaginal ultrasonography on 39 patients that underwent surgery for incontinence. They found more approximation between the bladder neck and the pubic bone in patients that failed to respond to surgery.

In contrast to both studies, Dellas et al [209] found a clear correlation between amount of bladder neck elevation achieved by surgery and likelihood of success. They performed cystourethrography on 88 women before and 4-6 months after colposuspension. The bladder neck was positioned in relation to a line running from the inferior border of the pubis to the sacroccocygeal joint. Continent women after surgery had a mean bladder neck elevation of 4.6 cm, while patients with failed surgery had a mean elevation of 2.8 cm. The probability of post-operative continence increased in relation to the amount of bladder neck elevation. It is however disconcerting that the mean bladder neck elevation achieved in the surgical failures in this study is considerably higher than the elevation achieved in
successful cases by other authors [157,196] (as mentioned previously) who also used similar measuring techniques.

Enzelsberger et al [197] used introital ultrasonography to measure bladder neck elevation and reported that in five patients with failed surgery elevation was less than 10 mm. The significance of this finding is uncertain as elevation in successful cases is not mentioned. Others [128,221,222], using ultrasonography, found that the absolute position of the bladder neck in relation to the pubic bone did not discriminate between successes and failures. Also the subjective assessment during surgery of paravaginal approximation to the ilio-pectineal ligament has not been found to be predictive of outcome [223].

The issue of whether bladder neck position is a determinant of success or failure remains unresolved. All studies have considerable methodological limitations. In some studies, the number of patients with ‘failed’ colposuspension is either small [222], or not specified [125], or includes also patients with conditions other than persistent stress incontinence (e.g. detrusor instability and voiding dysfunction) [221]. Various procedures for incontinence which may have different mechanisms of action (e.g. needle suspensions or anterior colporrhaphies) are pooled together with colposuspensions [125,128]. Studies which assess subjectively bladder neck elevation during surgery [223] are likely to be inaccurate as the bladder neck is not visible. No studies have assessed the reproducibility of the technique used and observers were never blinded to outcome of surgery.

**Fixation of the bladder neck** behind the pubic bone is also a recognised aim of the colposuspension operation. This concept is supported by DeLancey’s anatomical studies that suggest that continence depends on the integrity of the hammock-like layer that stabilises the urethra [54]. Bladder neck mobility after surgery has been studied in relation to success and failure.
Kil et al [128], Mouritsen and Rasmussen [125] and Barbic and Kralj [222] found using ultrasonography that successfully treated patients had reduced bladder neck mobility as compared to failures, thus suggesting that bladder neck stabilization is an important determinant of success. Kujansuu et al [224] reached the same conclusions using urethrocystography and measuring bladder neck mobility by observing the difference in the urethral inclination angle when straining. Also Bergman et al [19] found that successful Burch procedures resulted in a well supported urethrovesical junction at the one year post-surgical evaluation as judged by the Q-tip test, while four of 12 failures had mobile bladder necks.

These studies have considerable methodological limitations. In most studies, various procedures for incontinence that may have a different mechanism of action were pooled together [125,128,224]. In all studies observers were not blinded to outcome. In most studies the reproducibility of the imaging technique used was not assessed [125,128,222,224]. Valsalva was standardised in only one study [128]. The vaginal probe used for ultrasonography may have prevented mobility [125,128]. The number of patients with failed colposuspensions was either small [128,222] or not specified [125]. And finally, the assessment of bladder neck mobility by Q-tip test has been shown to have shortcomings [225].

On the available evidence, it can be concluded that the colposuspension operation seems to work by creating a new continence mechanism, rather than by restoring normal anatomy and physiology. Repositioning of the proximal urethra in a high retropubic position maximises the compressive effects of the postero-superior structures during stress and increases pressure transmission to the proximal urethra. Although there is some evidence that bladder neck fixation is an important element for the restoration of continence, the
relative importance of elevation and fixation is not clear, and the amount of elevation (if any) required to achieve effective and long lasting fixation is not known.

3- SUCCESS RATE

Recently, two large literature reviews on the success of surgery for genuine stress incontinence have been published. Jarvis [15] hand-searched 16 English-speaking scientific journals from 1970 and performed a meta-analysis of 213 studies where results of surgery had been assessed objectively. Black and Downs [16] searched extensively English and non-English language journals, and reviewed systematically the literature available. They found the methodological quality of the studies to be generally poor and the design of the studies variable and unsuitable for meta-analysis. Their assessment of the effectiveness of surgery was based on the results of 11 randomised controlled trials, 20 prospective non-randomised cohort studies and 45 retrospective cohort studies.

Jarvis [15] reported an overall subjective success rate of 89.6% and an objective success rate of 84.3% for the colposuspension operation. This was considerably better than the success achieved by needle suspension (77.6% and 70% respectively) and anterior colporrhaphy (80.9% and 72% respectively). Black and Downs [16] stated that about 85% of women can expect to be continent one year after colposuspension, compared with 50-70% after anterior colporrhaphy or needle suspension.

When considering only randomised studies, the colposuspension operation appears clearly superior to anterior colporrhaphy and needle suspension (table 2).
Table 2. Objective cure rates (%) after incontinence surgery at 6-12 months follow-up in randomised studies (n. of patients in brackets).

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<td></td>
<td>12m follow-up</td>
<td>6m follow-up</td>
<td>12m follow-up</td>
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<tr>
<td>Colposuspension</td>
<td>87% (101)</td>
<td>84% (25)</td>
<td>70% (26)</td>
</tr>
<tr>
<td>Needle suspension</td>
<td>70% (98)</td>
<td>40% (25)</td>
<td></td>
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<tr>
<td>Anterior colporrhaphy</td>
<td>69% (99)</td>
<td>36% (25)</td>
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In the long term, the success rate of the colposuspension operation is comparatively greater, with the benefit of colposuspension being maintained for at least five years, whereas the benefits of anterior colporrhaphy and needle suspension diminish quite rapidly [16]. The only randomised study with long-term follow-up [20] reports a success rate at 5 year follow-up of 82% for the colposuspension operation, of 43% for needle suspension, and of 37% for anterior colporrhaphy.

With regards to **sling procedures**, the evidence available on their value is scanty, with only 150 patients having been included in prospective studies [16]. Taking into account this limitation, Jarvis [15] and Black and Downs [16] conclude that slings appear to be as effective as the colposuspension operation. The only randomised comparison of the colposuspension operation with a sling procedure [197] showed similar success rates but lower morbidity for the colposuspension operation. Recently, a sling procedure has been developed, involving the use of a tension free vaginal tape (TVT) [227]. A success rate of 84% has been reported at two year follow-up, with a low complication rate [227]. Prospective studies comparing this procedure with colposuspension are in progress.
Although no comparative studies have been performed, para-urethral injectables clearly have a success rate which is lower than colposuspension [15], and it is unlikely that they will constitute first choice treatment, except perhaps in frail and elderly patients [228,229].

**Paravaginal defect repair** has been compared to colposuspension in a randomised study by Colombo et al [230]: subjective and objective cure rates were considerably higher after colposuspension.

**Different methods of colposuspension** include the Marshall-Marchetti-Krantz procedure, the Burch colposuspension and the laparoscopic colposuspension. Although the Burch colposuspension is generally considered as one procedure, two variations described by Stanton and Tanagho are commonly performed.

Comparisons between the Burch colposuspension and the **Marshall-Marchetti-Krantz procedure** [201,231-233], including one randomised study [201] have shown the Burch colposuspension to be more successful, but not significantly so. Morbidity was greater after the Marshall-Marchetti-Krantz procedure [201]. The colposuspension operation was considered technically easier to perform [233].

Comparisons between open and **laparoscopic colposuspension** are at present difficult. In the short term the laparoscopic procedure has been reported to be as effective as the open colposuspension [234]. In the longer term, Lobel and Davis [235] report a declining success rate for the laparoscopic technique from 86% at one year down to 68% at 30 months. The only randomised study published to date [236,237] reports a higher failure rate at one year for the laparoscopic procedure and a success rate of only 60% at three years (comparing to 93% for the open colposuspension).

No direct or indirect comparisons have been made to date between the two variations of the Burch colposuspension described by **Stanton and Tanagho** and it is therefore not
known whether subtle differences in operative technique could lead to differences in long-
term success rates. A review of the available reports reveals similar objective cure rates
after five years or more (table 3).

Table 3. Cure rates after colposuspension using Stanton’s and Tanagho’s methods.

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<th>STANTON</th>
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<th>TANAGHO</th>
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<tr>
<td></td>
<td>n. patients (years follow-up)</td>
<td>Cure (%)</td>
<td>n. patients (years follow-up)</td>
<td>Cure (%)</td>
</tr>
<tr>
<td>Wiskind et al (1992) [40]</td>
<td>131 (3-14y)</td>
<td>81.8%</td>
<td>van Geelen et al (1988) [214]</td>
<td>34 (5y)</td>
</tr>
<tr>
<td>Herbertson &amp; Iosif (1993) [238]</td>
<td>72 (8-12y)</td>
<td>90.3%</td>
<td>Eriksen et al (1990) [25]</td>
<td>86 (5y)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Bergman &amp; Elia (1995) [20]</td>
<td>33 (5y)</td>
</tr>
</tbody>
</table>

There are no randomised studies reporting on success rates after five years, and few
authors have reported objectively on the results of the colposuspension operation beyond
five years. Alcalay et al [17] reviewed 109 women after a mean of 13.8 years. Although
there was a decline with time, a plateau was reached after 10 years, with a subjective and
objective success rate of 69%. Others were able to report higher long-term success rates.
Herbetsson and Iosif [238] followed-up 72 women, and the objective cure rate was 97.3%
at one year, 91.7% at five years and 90.3% at nine years. Similarly, Feyereisl et al [26]
reported an 82% objective cure rate in 87 women at 5-10 year follow-up.
Despite a possible decline with time, the colposuspension operation appears to have a greater long term success rate than other procedures [16]. Pressure transmission ratios are increased to a greater extent by colposuspension, comparing to needle suspensions and anterior colporrhaphy [212-214]. Bladder neck mobility assessed by Q-tip is reduced to a greater extent by colposuspension than by needle suspension [213].

This may be due to the firmer point of anchorage offered by the ilio-pectineal ligaments, and also to the more extensive dissection required by the procedure which allows for a greater connective tissue reaction [20]. There is concern that the minimal dissection required for the laparoscopic colposuspension (with reduced fibrosis) and the early return to normal activities which follows this procedure, might result in reduced long-term success, comparing to the open technique. The results of on-going randomised trials are awaited.
4- ADVERSE FACTORS

Numerous factors that may adversely affect the results of surgery have been considered in the literature. Most of them are debatable or anecdotal. The evidence available is presented and discussed.

Hysterectomy

Whether prior or concomitant hysterectomy affects the chances of successful anti-incontinence surgery has been a matter of some debate.

With regards to a history of prior hysterectomy, Green [239] found a higher success rate of 90% in 41 women who underwent retropubic suspension after prior hysterectomy, comparing to a cure rate of 82% in 40 women who still had their uterus. In contrast, Sand et al [240] found a lower cure rate of 58% in 62 women with prior hysterectomy, comparing to a cure rate of 83% in women with a uterus still in situ. Most authors [191,210,223,241] have been unable to demonstrate any effect of past history of hysterectomy on surgical outcome.

In relation to the performance of a hysterectomy at the time of anti-incontinence surgery, Green [239] found a higher subjective cure rate of 97% in 149 women treated with retropubic suspension and concomitant hysterectomy, comparing to a cure rate of 82% in 40 women who underwent retropubic suspension alone. Most authors [191,192,214,223,232,241] have been unable to demonstrate any effect of concomitant hysterectomy on the likelihood of successful surgery. Langer et al [242] randomized 45 patients to colposuspension alone or colposuspension and concomitant hysterectomy, and found no difference in the cure rate at two years.

As concomitant hysterectomy may increase operative morbidity [243], there is no evidence at present to suggest that it should be routinely performed at the time of colposuspension.
Low pressure urethra

Intrinsic urethral sphincter deficiency (ISD) is considered the second most important cause of stress incontinence after bladder neck hypermobility [4]. Urethral pressure profilometry is commonly used to detect this defect, despite a great deal of overlap in results between continent and incontinent women [57]. Low urethral pressure, defined as a MUCP of less than 20 cmH2O [244], is present in 15-23% of women that undergo surgery for genuine stress incontinence [244,245]. When defined as a MUCP of less than 30 cmH2O, an incidence of 44% has been reported [246]. McGuire [244] reported that patients with failed incontinence surgery had lower post-operative urethral closure pressures than those whose surgery was successful. Such association cannot establish whether a low urethral pressure is a cause of failure or an effect of surgery.

Various authors [26,210,247,248] have found a lower mean pre-operative MUCP in surgical failures as compared to successes. Sand et al [249] compared the results of colposuspension in 41 patients with low pre-operative closure pressures (MUCP < 20 cmH2O) and 45 patients with normal pressures. The failure rate was 54% in the low-pressure group comparing to 18% in the normal-pressure group. The two groups were comparable except for a difference in age, with older patients in the low-pressure group. After correction for age, the low-pressure urethra was found to be an independent risk factor only for patients under the age of 50 years. Similarly, Baker and Drutz [250] compared 27 patients with low pre-operative closure pressure (MUCP < 20 cmH2O) with 63 patients with normal pressures. The failure rate in the low-pressure group was 18.5% comparing to 6.3% in the normal-pressure group. The two groups were however not comparable, as patients in the low-pressure group were significantly older. Koonings et al [245] were able to compare two groups of patients with low (less than 20 cmH2O) and normal MUCP that were matched for age. The failure rate at one year was 33% versus 12%
respectively, and the difference was statistically significant. Because the number of patients with low urethral pressure was low (only nine), it is difficult to draw firm conclusions.

Bowen et al [208] compared 21 patients that had unsuccessful colposuspensions with 21 successful cases that were matched for age, parity, number and type of previous anti-incontinence surgery, the presence of pulmonary disease and smoking, weight, menopausal status, and previous hysterectomy. The pre-operative MUCP was significantly lower in the failure group than in the success group, and 76% of patients in the failure group had a low-pressure urethra (less than 20 cmH2O) comparing to only 19% in the success group.

In contrast, Meschia et al [246] could not find a statistically significant difference in failure rate when comparing two groups of patients with normal and low MUCP. The two groups were comparable with regards to age and weight, but not with regards to a history of previous bladder neck surgery (more common in the low pressure group). They used a higher value of 30 cmH2O to define a low pressure urethra. Similarly, Monga and Stanton [248] did not find low MUCP a predictor of poor outcome after exclusion of cases with prior bladder neck surgery.

It has been proposed that patients with a low MUCP should undergo an alternative procedure. Horbach et al [251] suggested that these patients should have a sling procedure. They reported a success rate of 85% with slings on 13 patients with low-pressure urethras.

Bergman et al [252] suggested the introduction of a modification to the colposuspension operation, which included a plication of the paraurethral tissues over the proximal urethra aiming to increase urethral resistance (the 'Ball-Burch' procedure). They compared 48 women with low-pressure urethras undergoing the 'Ball-Burch' procedure with 18 historical controls with low-pressure urethras that had a Burch colposuspension alone. The controls were matched for age and number of previous anti-incontinence procedures. The one year objective failure rate of the Ball-Burch procedure was 10%, and that of the Burch procedure was 38%. The new technique increased urethral resting parameters of functional
length and closure pressure to a greater degree than did the simple Burch procedure in women with low urethral pressure.

In contrast, relatively high cure rates of 78% have been reported after colposuspension in 29 patients with low closure pressures [253].

There seems to be enough evidence to suggest that the presence of a low-pressure urethra is an independent risk factor for failure of anti-incontinence surgery, when using a lower defining value of 20 cmH2O. However, the extent of risk is controversial and there is no conclusive evidence that alternative surgery is required.

**Obesity**

It is unclear whether obesity constitutes a risk factor for failed incontinence surgery. Contrary to general opinion, weight or body mass index (BMI) at the time of incontinence surgery have not been found to affect outcome by most authors [26,204,210,223,248,254] even after long-term follow-up [26,210,254]. Studies however include only small numbers of obese patients [204] or do not specify their number [26,210,223,254]. Two studies without these shortcomings reach opposite conclusions. Alcalay et al [17] found at 10-20 year follow-up that 13 patients with weight greater than 80 Kg had a success rate of only 46% (comparing to 76% for patients of less than 80 Kg). In contrast, Kinn [247] found at five year follow-up that weight was not a risk factor for 22 of 153 patients weighing more than 80 kg.

Only one study has correlated weight at the time of outcome assessment to success or failure [255] and found that patients with failed surgery had a significantly higher BMI than patients successfully treated. However, the weight at the time of surgery was not recorded. It is therefore not known whether weight gain after surgery is an independent risk factor.
Previous incontinence surgery

Jarvis [15], following his meta-analysis, reports a slightly higher success rate for the colposuspension operation when performed as a primary procedure as compared to secondary surgery (89.8% versus 82.5%). However, when individual studies dealing with this issue are considered separately, methodological diversity and inconsistency of findings make it difficult to draw firm conclusions (table 4).

The inconsistency of results may be due to differences in length of follow-up and to the lack of separate analysis of results following different types of previous anti-incontinence procedures.

From the available reports, there is no consistent and conclusive evidence that a history of anterior colporrhaphy constitutes a risk factor, while previous failed retropubic suspension seems to be an adverse feature. Of reports that include a significant number of prior retropubic suspensions [210, 240, 250], only Sand et al [240] do not consider it as a risk factor: but while not reaching statistical significance, their reported success rate of patients with a prior history of retropubic suspension was only 45%. The mechanism of damage is unknown, but denervation and devascularisation are possible explanations. Paradoxically, vaginal dissection at anterior colporrhaphy has been shown to be more likely to produce neuropathy than retropubic dissection [259]. Multiple failed operations may result in a rigid, scarred, ‘drainpipe’ urethra, which is fixed and adherent to the back of the pubic bone [260].
<table>
<thead>
<tr>
<th>Type of previous bladder neck surgery (n. previous surgery / n. patients)</th>
<th>Follow-up</th>
<th>Prior bladder neck surgery is risk factor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stanton et al (1978) [204]</td>
<td>Not specified</td>
<td>1-3 years</td>
</tr>
<tr>
<td>Milani et al (1985) [232]</td>
<td>Ant. Repair (16/86)</td>
<td>1.8-6 years</td>
</tr>
<tr>
<td>Francis et al (1987) [210]</td>
<td>Retropubic (12/50)</td>
<td>3 months</td>
</tr>
<tr>
<td>Galloway et al (1987) [23]</td>
<td>Type not specified (19/50)</td>
<td>1-6 years</td>
</tr>
<tr>
<td>Freeman and Malvern (1987) [256]</td>
<td>Type not specified (117/278)</td>
<td>6 months</td>
</tr>
<tr>
<td>Rizvi et al (1988) [241]</td>
<td>Type not specified (20/62)</td>
<td>2-3 years</td>
</tr>
<tr>
<td>Baker and Drutz (1991) [250]</td>
<td>Ant. Repair (82/289)</td>
<td>0.13-12 years</td>
</tr>
<tr>
<td>Feyereisl et al (1994) [26]</td>
<td>Type not specified (20/72)</td>
<td>5-10 years</td>
</tr>
<tr>
<td>Kjolhede and Ryden (1994) [258]</td>
<td>Not specified</td>
<td>2-10 years</td>
</tr>
<tr>
<td>Monga and Stanton (1996) [223]</td>
<td>Type not specified (90/325)</td>
<td>1 year</td>
</tr>
<tr>
<td>Monga and Stanton (1997) [248]</td>
<td>Type not specified (44/76)</td>
<td>1 year</td>
</tr>
</tbody>
</table>
Age

Advancing age is often considered a risk factor for failure after incontinence surgery. However, there is no strong evidence to support this theory. Stanton et al [204] have shown age to be a risk factor, but only for patients with post-operative incontinence due to detrusor instability. Kjolhede and Ryden [258] and Kinn [247] have shown age to be a risk factor for failure, but their assessment of outcome was not objective.

In contrast, Gillon and Stanton [38] reported a high long term success rate of 89% after colposuspension in 35 elderly women (aged between 65 and 82 years), with no increase in morbidity. Baker and Drutz [261] compared the success rate after colposuspension of 50 women aged 65 years or more to 239 younger women. Despite a lower MUCP, age of more than 65 years was not a risk factor for failure and there was no increased morbidity; hospital stay was only two days longer. Monga and Stanton [223] did not find age to be a risk factor (at one year) in a study of 280 patients cured by colposuspension when compared to 45 women who failed. Even in the long term, age does not appear to be an adverse factor, as shown by Alcalay et al [17], who followed-up 20 patients older than 60 years for 10-20 years. This has been confirmed by other long-term follow-up studies [25,26].

Absence of bladder neck mobility

Bergman et al [262] found a high failure rate after colposuspension (55%) among nine women whose urethral mobility was less than 35 degrees as assessed by Q tip. There was no correlation between a negative Q-tip test and the presence of other risk factors such as low urethral closure pressure, age and a past history of bladder neck surgery. They suggested that incontinence in these patients was unlikely to be due to poor support. In contrast, Bowen et al [208] could not find a difference in pre-operative bladder neck mobility as assessed by Q-tip between successes and failures. Most of their patients
however had ‘hypermobile’ urethras (mean of 44.9 and 42.9 degrees of mobility respectively). Similarly, Monga and Stanton [248] studied bladder neck mobility with ultrasound and found that it did not affect outcome.

**Pre-operative vaginal prolapse**

It is not clear whether the presence or severity of pre-operative vaginal prolapse predisposes to failure of incontinence surgery. Milani et al [232] and Pigne et al [218] found pre-operative prolapse to be a risk factor. In contrast, Bowen et al [208] could not find an association between the severity of pre-operative cystocele and rectocele and the chances of successful surgery. In all studies prolapse was poorly defined and quantified, and there may have been differences in the incidence of severe prolapse.

Sacrocolpopexy performed at the time of colposuspension has been found by Pigne et al [218] to be an important adverse factor. They subsequently found that the risk of persistent incontinence could be reduced by avoiding anchoring the vault under tension. Excessive stretching of the anterior vaginal wall after vault suspension may limit vaginal mobility and prevent transmission of posterior forces during stress [263].

**Pre-existent detrusor instability**

In most reports, surgical failure is defined as continuing or worsening incontinence. This may however be due to causes other than stress incontinence, such as pre-existing or new onset detrusor instability [260]. Surgical failure should be urodynamically distinguished as persistent stress incontinence or persistent (or new onset) detrusor instability, as the two problems are likely to have different causes. Detrusor instability will be discussed separately as a ‘complication’ and **surgical failure** is here defined as **failure to cure genuine stress incontinence**.
While pre-operative detrusor instability is considered an adverse factor with regards to post-operative continence [204], there is no clear evidence to support the view that it constitutes a risk factor for failure to cure genuine stress incontinence. Colombo et al [264] reported a higher incidence of recurrent stress incontinence after colposuspension in women with mixed detrusor instability and genuine stress incontinence as compared to women with pure genuine stress incontinence. The difference was statistically significant, but the chances of success were high in both groups. Bowen et al [208] compared 21 failed colposuspensions with 21 successful cases and found the same incidence of pre-operative detrusor instability in both groups. They concluded that the presence of detrusor instability did not appear to affect the ability to cure genuine stress incontinence. Also Monga and Stanton [223] did not find detrusor instability a risk factor for the cure of stress incontinence when comparing retrospectively 280 cured patients (of which 40 had pre-operative detrusor instability) to 45 patients with failed surgery and persistent stress incontinence (of which five had pre-operative detrusor instability). Similarly, other authors could not find a significant difference in the cure rate of genuine stress incontinence between women who had mixed genuine stress incontinence and detrusor instability pre-operatively and women who had pure genuine stress incontinence [215,240,248].

Other factors

Patients are generally instructed to avoid heavy lifting after surgery, but although this seems reasonable advice, there is no strong evidence that this is actually detrimental. Maleika-Rabe [265] found that carrying weights over 5 Kg at work and middle-hard burden with housework were unfavourable features with regards to long term success after incontinence surgery. In contrast, Kjolhede and Lindehammar [255] could not find significant differences in the workload in employment or housework, assessed by means of
questionnaire, between women with successful or failed surgery. Objective data are needed to resolve this point (e.g. type of work, when recommenced and other risk factors).

**Chronic cough** and **chronic constipation** are considered risk factors for failure, but no data are available to support this theory. Monga and Stanton [223] did not find these factors to influence outcome.

It is controversial whether the presence of **pelvic floor neuropathy** is a risk factor for failure. Post-operative assessments with concentric needle electrode electromyography found a higher incidence of neuropathy in patients with recurrent stress incontinence after surgery [255,266]. Using the same method prior to surgery, Monga and Stanton [248] could not find a significant difference between women with failed or successful surgery.

It is not clear whether colposuspension causes neurological damage. Caputo and Benson [267] performed pudendal nerve terminal motor latencies on 20 women before and after colposuspension and found no significant differences, thus suggesting that the operation does not produce pudendal nerve neuropathy.

**Menopausal status** was found to be a risk factor by Meschia et al [246] in a study of 11 of 75 women with failed surgery, despite pre-treatment with topical estrogen. Eriksen et al [25] studied 86 women after colposuspension and found that 71% of premenopausal and 61% of postmenopausal women were cured. The difference was not statistically significant. Monga and Stanton [223] in a larger study including 280 cured women and 45 failures did not find menopausal status to be a determinant of outcome.
4- OUTCOME ASSESSMENT

To date, assessment of surgical outcome has been performed by asking patients whether they are still suffering from the condition treated (subjective outcome) and by performing a test to ascertain whether the condition is still present (objective outcome).

From the strictly clinical point of view, the patient’s opinion would appear the most important determinant of outcome. However, subjective cure rates have been repeatedly shown to be higher than objective cure rates, and patient’s evidence may not be reliable for a variety of reasons, including the desire to please the surgeon or the wish to avoid further treatment. The subjective report of urinary symptoms may be difficult to interpret, as the bladder remains an unreliable witness post-operatively as well as pre-operatively. Moreover, subjective data may be collected and interpreted in a variety of ways, thus making comparisons difficult.

For these reasons it has been suggested that an adequate scientific assessment of the results of surgery should additionally rely on objective tests [191], although no specific test has been recommended for this purpose by the ICS. A combined subjective/objective assessment has been the accepted form of outcome assessment to date. This may no longer be sufficient.

There is increasing evidence that the results of incontinence surgery are not as favourable as originally thought when taking into account quality of life issues [41]. This is obviously of concern, as incontinence surgery aims to improve quality of life. Simple subjective and objective outcome assessments do not take adequately into account the patient’s perception of a problem and the effect that this problem has on quality of life. This may occur because there is a poor correlation between the impact of urinary symptoms on the individual sufferer and the objective severity of the condition [8]. As a consequence, the development
of complications after surgery (e.g. voiding dysfunction or detrusor instability), which may not appear as a surgical ‘failure’, may have a worse impact on an individual patient’s quality of life than the problem originally treated. It is therefore now perceived as important that quality of life becomes an additional outcome measure for research in adult women with symptoms of lower urinary tract function [42].

‘Quality of life’ is an abstract and subjective concept which depends on many variables and is difficult to quantify. Structured questionnaires have been devised for this purpose. They are composed of several items or questions, which are combined to form ‘domains’ (or ‘dimensions’). The ‘domain’ is the specific area of experience that is being measured. Several domains can by combined into ‘instruments’, which are focused on a particular aspect of health. Questionnaires can be completed by the individual concerned or by another trained individual as part of an interview. Answers are graded and result in a score.

Two main types of health related quality of life questionnaires have been devised: generic and condition-specific. Generic questionnaires are designed for broad comparisons between groups and assess a wide spectrum of vital functions. They tend to have low sensitivity when measuring changes due to interventions in women with urinary incontinence, because the general health of these women is otherwise good. Condition-specific questionnaires are designed to measure the impact of a particular disease, and inquire about specific symptoms, their severity, and their effect on quality of life. They are therefore better suited to assess interventions on women with urinary symptoms. The ICS now recommends that intervention studies should include a condition-specific questionnaire [42].

The quality of condition-specific questionnaires depends on their capacity to measure what is intended (validity), their capacity to measure it in a consistent manner (reliability), and their capacity to be sensitive to clinically significant change (responsiveness). The validity can be measured against a ‘gold standard’ measure, such as an accepted clinical
instrument. The reliability can be assessed by its internal consistency (the degree of correlation between the items forming a scale) and by its test-retest correlation.

A number of disease-specific instruments have been recently devised and validated: the Incontinence Impact Questionnaire, the Urological Distress Inventory, the King's Health Questionnaire, the York Incontinence Perceptions Scale, the Bristol Female Lower Urinary Tract Symptoms [268]. The exact role of each one of these instruments has not yet been established, and clinicians should select the instrument better suited for a given purpose. However it should be remembered that while quality of life instruments are likely to constitute useful items for research, they should not be considered a substitute for objective testing and clinical judgement.

**CONCLUSIONS**

On the available evidence there is general agreement that the colposuspension operation constitutes the 'gold standard' for the treatment of genuine stress incontinence because of its high short and long-term success rate. It is not clear whether technical variations of the procedure are advantageous in terms of success rate and complications. Elevating and fixing the bladder neck results in improved pressure transmission to the proximal urethra. The relative importance of elevation and fixation is not clear, and the amount of elevation (if any) required to achieve effective and long-lasting fixation is not known. Despite the presence of risk factors that may decrease the success rate in some patients, most patients are likely to benefit from the procedure.

There is a need to assess the results of surgery in a more comprehensive way which includes quality of life measures.
CHAPTER 6

VOIDING DYSFUNCTION IN THE FEMALE AFTER SURGERY FOR STRESS INCONTINENCE
Voiding dysfunction is uncommon in women and occurs in only 13-16.5% of women presenting to Urogynaecology clinics [269,270].

Possible causes are neurological disease, psychological disturbances, drug therapy, pelvic masses, genital prolapse, genital atrophy, urethral pathology, painful perineal conditions, and pelvic surgery such as radical hysterectomy or rectal surgery. Often no cause is found, but some cases classified as idiopathic may be due to isolated impairment of relaxation of the striated muscle of the urethral sphincter [271].

Surgery for incontinence is thought to be an important cause of impaired bladder emptying in women, accounting for a quarter of cases [269].

In most cases, post-operative voiding problems are short-term, and constitute a source of frustration for patients and surgeons alike. Less commonly, post-operative voiding problems are long-term, and lead to profound alterations in quality of life.

In order to understand the pathophysiology of voiding dysfunction, voiding mechanisms in normal women and in women with stress incontinence will be described. The definition of voiding dysfunction, its incidence after surgery, its clinical effects and management will be described. Predictive factors and possible causes will be discussed.

1- NORMAL VOIDING IN WOMEN

Normal voiding occurs when tension receptors in the bladder sense fullness and stimulate a sacral reflex of somatically mediated relaxation of the pelvic floor and urethra and parasympathetically mediated detrusor contraction. Imaging studies on normal women have shown that the bladder base and the upper urethra move downwards (while the lower urethra remains fixed), the bladder as a whole becomes more ovoid in shape, the posterior urethrovesical angle becomes obliterated, funnelling occurs at the bladder neck, and the whole urethra dilates as the fluid passes [80]. In the adult, this simple reflex is under
voluntary control via a set of complex pathways which run from the cerebral cortex, down to the pontine micturition centre and the sacral spinal cord.

Studies on detrusor pressure during voiding in normal women suggest that most void with a detrusor contraction greater than 15 cm H2O, with mean values of 23-28 cmH2O of detrusor pressure at maximum flow, and 20-36 cmH2O of maximum detrusor pressure (table 5). Detrusor pressure during voiding in women is on average lower than in men [273]. A few normal women have been reported to void with a low pressure detrusor contraction of less than 15 cmH2O, but no normal women voided without any contraction, and it has been suggested that such an event should probably be considered abnormal and indicative of low urethral closure pressure [283].

Table 5. Detrusor pressure values in normal women*

<table>
<thead>
<tr>
<th>Authors</th>
<th>n. women</th>
<th>Age</th>
<th>pdet,Q max (cmH2O)</th>
<th>pdet,max (cmH2O)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Backman et al, 1966</td>
<td>15</td>
<td>5 of &lt; 25 y</td>
<td>mean 28</td>
<td>range 10-56</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5 of 25-50 y</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>5 of &gt; 50 y</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frimodt-Moller, 1974</td>
<td>11</td>
<td>Mean 26.9 y</td>
<td>mean 25</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Range 20-44 y</td>
<td>(+/- 11.9 SD)</td>
<td></td>
</tr>
<tr>
<td>Walter et al, 1979</td>
<td>11</td>
<td>Median 46 y</td>
<td>mean 23</td>
<td>mean 28</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Range 40-62 y</td>
<td>range 9-40</td>
<td>range 16-57</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Mean 37 y</td>
<td>mean 28.7</td>
<td>mean 29.9</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Range 20-63 y</td>
<td>(+/- 8.4)</td>
<td>(+/- 8.7)</td>
</tr>
<tr>
<td>Sjoberg and Nyman, 1981</td>
<td>19</td>
<td>Median 42 y</td>
<td>mean 36</td>
<td>range 15-72</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Range 29-77 y</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heslington and Hilton, 1996</td>
<td>22</td>
<td>Mean 33.8</td>
<td>mean 20.3</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>+/- 11.5 y</td>
<td>(+/- 14.2)</td>
<td></td>
</tr>
</tbody>
</table>

* Studies on normal women containing small numbers [278], inaccurate recording of maximum voiding pressure due to inclusion of the after contraction [279] or omission of intra-abdominal pressure measurements [183,280-282] have been excluded.

‡ Values of pressure given in mmHg in original paper, converted to cmH2O in table.
There are variations in the way women void. Some women, in addition to pelvic relaxation and detrusor contraction, strain by an unconscious but sustained Valsalva manoeuvre. Backman et al [272] have shown that 10 of 15 women had an intra-abdominal pressure rise of 15 cmH2O or more at bladder neck opening, indicating abdominal straining, and in eight this was noticed during maximum flow. Similarly, Karram et al [277] noted straining during voiding in 22 of 30 normal women. The contribution of abdominal pressure to the voiding process varies considerably between individuals and within the same individual during consecutive voids [273,274].

The 'normal' voiding curve in women has been described as having a single peak, with a fast crescendo and a relatively slow diminuendo, with minimal fluctuations [284]. However, an interrupted pattern is seen in 20% of normal women during a single void, and in 8% of normal women such a pattern occurs repeatedly [284]. Studies of flow rates in normal women suggest that most normal women void with a peak flow rate greater than 15 ml/s, with mean values of 23-27 ml/s (table 6). Peak flow rate values are on average higher in women than in men [285,287]. The main variable affecting flow rates in normal women is bladder volume, with greater flow rates seen with increasing volumes [281,284,287]. Haylen et al [287], after studying flow rates in 249 female volunteers (aged 16-63), have constructed nomogram charts, in centile form. These provide a reference range of normality for urinary flow rates in relation to volume voided. Parity, weight and height appear to have no influence on flow rates [284]. Age may cause a subtle deterioration in flow rates and voiding efficiency (see chapter 6, section 5e).
Table 6. Peak flow rates during voiding in normal women*.

<table>
<thead>
<tr>
<th>n. women (n. of flows)</th>
<th>Age</th>
<th>Qmax, ml/s (volume voided)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arbuckle and Paquin, 1963 [281]</td>
<td>27 (92, 10 excluded as volume &lt; 200 ml)</td>
<td>Range 19-26 y</td>
</tr>
<tr>
<td>Backman 1965 [285]</td>
<td>151 (at least 302)</td>
<td>All ages, but mean and range not specified</td>
</tr>
<tr>
<td>Smith 1968 [183]</td>
<td>50 (50)</td>
<td>Mean 32.4 y Range 6-68 y</td>
</tr>
<tr>
<td>Fantl et al 1983 [284]</td>
<td>60 (360)</td>
<td>Premenopausal 29.9 +/- 6.3 SD Postmenopausal 56.6 +/- 3.1 SD</td>
</tr>
<tr>
<td>Bottaccini and Gleason, 1980 [286]</td>
<td>36 (36)</td>
<td>age not specified</td>
</tr>
</tbody>
</table>

*Peak flow rates obtained with volumes of less than 150 ml, or using grossly unphysiological methods, e.g. in the presence of suprapubic catheters, are not included.
† Volume not clearly specified in text.

2- VOIDING IN WOMEN WITH STRESS INCONTINENCE

It is controversial whether women with stress incontinence have lower peak flow rates during voiding than normal women. Lower values have been reported in women with stress incontinence [286], but this has not been confirmed by others [284,288].

In contrast to normal women, who invariably void with a detrusor contraction (with or without abdominal straining), up to 11-15% of women with genuine stress incontinence have been shown to void without a contraction [277,289]. These women void by pelvic
relaxation, with or without abdominal straining. Women with stress incontinence also void with significantly lower detrusor pressures than normal continent women (mean of 12.3 cmH2O versus mean of 20.3 cmH2O respectively) [277]. A low voiding detrusor pressure of less than 15 cmH2O has been reported in up to 50% of patients with stress incontinence undergoing surgery [200,290]. It is not clear whether this is due to the lower mean urethral closure pressure of women with genuine stress incontinence, or whether there is a real impairment of detrusor muscle function in these women.

It has also been shown that women with stress incontinence are more likely to strain during voiding than normal women [288], and that they are more likely to initiate voiding with a Valsalva manoeuvre, as opposed to normal women who always initiate voiding by relaxing the pelvic floor [282]. It has been suggested that straining in women with stress incontinence may be a compensatory mechanism for their weaker detrusor contraction [288].

3- DEFINITION OF VOIDING DYSFUNCTION

A definition of female voiding dysfunction has not yet been included in the standardisation of terminology of lower urinary tract function published by the International Continence Society [1].

Symptoms of voiding difficulty (e.g. hesitancy, straining to void, feeling of incomplete bladder emptying, a poor and/or interrupted urinary stream) have been found to be an unreliable guide to diagnosis [269,270]. Commonly used definitions rely on objective assessment of voiding performance. As a consequence, chronic urinary retention has been variously defined as ‘painless failure of bladder emptying where catheterisation yields a volume equal to at least 50% of normal bladder capacity’ [291], as ‘painless retention of
urine, with a residual volume exceeding the normal capacity' [292], or as ‘the repeated presence of a residual urine volume of 150 ml or more’ [269].

Equally, there is no universally accepted definition of voiding dysfunction after incontinence surgery. When reporting the incidence of this complication after colposuspension, voiding performance is often measured in terms of ‘time required for resumption of voiding’, or ‘time of catheter removal’, or ‘number of patients requiring a catheter for more than seven days’ (as detailed in appendix 1, where all reports found in the literature on voiding function after colposuspension are listed). While these definitions may appear simple and practical, the lack of uniformity with regards to catheter management strips them of scientific precision.

Prolonged or ‘late’ voiding dysfunction after incontinence surgery (as opposed to ‘early’ dysfunction), may affect patients who did not have ‘early’ dysfunction [24], and may have different causes. It has been variously defined as ‘a peak flow rate of less than 15 ml/s’, ‘a residual of more than 100 ml’, or ‘the need for long term catheterisation’ (appendix 1). Unfortunately, the timescale of ‘early’ and ‘late’ dysfunction remains undefined and there is uncertainty as to what constitutes a ‘significant’ residual.

4- INCIDENCE

Post-operative voiding disorders (variously defined as above) have been reported to occur after most operations for stress incontinence. While bladder buttress procedures rarely result in prolonged voiding dysfunction, this complication occurs in 10.3% of patients after non-endoscopic bladder neck suspensions, in 5.8% of patients after endoscopic bladder neck suspensions and in 12.8% patients after bladder sling procedures [15]. Paravaginal repairs performed for stress incontinence have been reported to lead to voiding
dysfunction in only 0-5% of patients [60,230]. Prolonged voiding dysfunction is uncommon after para-urethral injectables, although an incidence of 5% (2/34) has been reported in a recent series [228]. The tension free vaginal tape is currently undergoing extensive studies, but their promoters claim that the procedure does not lead to prolonged voiding dysfunction [227].

The colposuspension operation leads to post-operative voiding dysfunction in a mean of 12.5% of patients [15]. In order to obtain reports on voiding dysfunction after colposuspension, a computer literature search was performed using Medline. Further reports were obtained from reference lists and abstracts from conference proceedings of the International Continence Society and the International Urogynecology Association annual meetings (1994-1998). These are listed in appendix 1. The reported range varies from none to 43%. Differences in definition, patient selection and surgical technique may account for such a wide range.

It is not clear whether differences exist in the incidence of voiding dysfunction after different methods of colposuspension.

The Marshall-Marchetti-Krantz (MMK) procedure is thought to have an incidence of post-operative voiding dysfunction similar to the Burch colposuspension [15]. However, the only randomised comparison of these two operations [201], found a statistically significant later resumption of spontaneous voiding after the MMK procedure as compared to the Burch colposuspension (13.8 vs 8.5 days).

Voiding dysfunction has been reported to occur rarely after laparoscopic colposuspensions [195,234,305]. The only randomised comparison to date does not report on this complication [236].
More subtle differences in technique between surgeons, which may be relevant to the development of voiding difficulties, are likely to occur and are difficult to measure objectively. The variations of Burch's colposuspension described by Stanton et al [37] and Tanagho [31] are the most widely performed (chapter 5), but while the modifications described by Tanagho aim to prevent voiding dysfunction, a randomised comparison of these two methods has never been performed.

Reports in the literature (see appendix 1) where the surgical technique is clearly described and is identifiable as following either Stanton's or Tanagho's method, and the definition of voiding dysfunction is comparable, are illustrated in tables 7 and 8.

Table 7. Days of catheter requirement after Stanton’s and Tanagho’s methods of colposuspension.

<table>
<thead>
<tr>
<th></th>
<th>STANTON</th>
<th>TANAGHO</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n. women</td>
<td>days (mean)</td>
</tr>
<tr>
<td>Stanton et al '76 [37]</td>
<td>40</td>
<td>14</td>
</tr>
<tr>
<td>Gillon&amp;Stanton’84 [38]</td>
<td>35</td>
<td>15</td>
</tr>
<tr>
<td>Milani et al’91 [289]</td>
<td>129</td>
<td>6.3</td>
</tr>
<tr>
<td>Colombo et al’94 [201]</td>
<td>40</td>
<td>8.5</td>
</tr>
</tbody>
</table>

Table 8. Percentage of patients requiring a catheter for more than seven days after Stanton’s and Tanagho’s methods of colposuspension.

<table>
<thead>
<tr>
<th></th>
<th>STANTON</th>
<th>TANAGHO</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n. women</td>
<td>%</td>
</tr>
<tr>
<td>Iosif’85 [296]</td>
<td>195</td>
<td>27.6%</td>
</tr>
<tr>
<td>Milani et al’91[289]</td>
<td>129</td>
<td>30%</td>
</tr>
<tr>
<td>Kremer&amp;Freeman’95 [27]</td>
<td>51</td>
<td>43%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
A retrospective comparison between the two techniques in relation to the development of post-operative voiding dysfunction is of limited value due to likely differences in catheter management between studies and the lack of objective measures that could distinguish one technique from the other. There is at present no convincing evidence that voiding dysfunction is less common when using Tanagho’s modification.

5- CAUSES

In the immediate post-operative period, many reversible factors are likely to contribute to early voiding dysfunction [306]. Atropine and other anticholinergic anaesthetic reversal agents (some with a half life of three to four days) may reduce detrusor power. Bladder overfilling may depress detrusor contractility, opiates may reduce bladder sensation, and pain may inhibit perineal relaxation. In addition, bruising and oedema may depress bladder contractility and cause temporary obstruction.

In addition to these temporary factors, more permanent causes of voiding dysfunction may be at work from the outset. Some factors may be related to the operative technique (e.g. bladder neck overelevation, urethral compression, or the performance of concomitant surgery). Other factors may be related to the patient (e.g. poor detrusor power, psychological factors, age, previous pelvic or bladder neck surgery).

The evidence in the literature on the relative importance of these factors is presented and discussed.
Overcorrection of the bladder neck and proximal urethra is commonly thought to be the main cause of chronic retention after bladder neck suspension procedures [28,36,269,307]. It has been suggested that overelevation causes voiding dysfunction due to obstruction [36] or interference with the normal voiding mechanisms that include bladder neck descent at the commencement of voiding [54]. However, there is no clear evidence to support the theory that overelevation causes voiding dysfunction.

The issue has been explored by various methods:

Elevation in theory could be assessed subjectively at the time of surgery, by observing the degree of approximation of paravaginal fascia to ilio-pectineal ligament. Colposuspension performed with minimal elevation has been claimed to have a lower incidence of voiding dysfunction [294]. However, using this method of assessment, Dwyer and Stanton [29] were unable to correlate the amount of elevation to the likelihood of developing post-operative voiding dysfunction. The subjective assessment of elevation during surgery is likely to be inaccurate as the bladder neck is not visible and no account is taken of bladder neck position prior to elevation.

Pelvic examination and cystoscopy have been used by Carr and Webster to assess elevation and diagnose ‘overcorrection’ following incontinence surgery causing obstruction [36]. However, they did not compare their findings with controls and their assessment was subjective and not quantifiable. Pelvic examination and cystoscopy do not seem to have the potential to provide an objective measure of bladder neck elevation.

It is generally thought that excessive suture tension may lead to overelevation and voiding dysfunction or to surgical failure due to tearing of the fascia. Kondo et al [308] have measured suture tension during needle suspensions using a spring scale and found no
correlation between increasing tension and post-operative voiding performance. Similarly, Kapoor et al [309] used a standardised measure of tension while performing needle suspensions, but this did not prevent the development of voiding dysfunction. Both authors concluded that overelevation was not likely to be a cause of voiding dysfunction. However, no studies have shown whether the measurement of tension during surgery is reproducible and whether tension correlates accurately with elevation.

Direct assessment of elevation using videocystourethrography has been performed by Stanton et al [21]: they found no ‘abnormal’ elevation of the bladder neck in patients with voiding dysfunction three months after surgery. However, objective measures of elevation were not obtained.

Dundas et al [30] using the same method have made the most serious attempt to date to correlate voiding dysfunction with bladder neck elevation. They performed VCU on 54 women before and after colposuspension. The amount of bladder neck elevation achieved by surgery was established by measuring the height of the bladder neck above the ischial tuberosity before and after surgery. Bladder neck mobility was also observed during voiding and on coughing using the same point of reference. Although the time of investigation in relation to surgery was not specified, the use of a urodynamic definition of voiding dysfunction (Qmax < 15 ml/s), rather than the time of catheter removal, suggests a late assessment. Mean elevation achieved by surgery was seven millimetres. Although the mean elevation achieved in 20 patients with post-operative voiding difficulty was higher than that achieved in patients with no voiding difficulty (9.3mm vs 4.8mm), the finding did not reach statistical significance. The decrease in descent on voiding achieved by surgery was significantly greater in the first group (i.e. poor voiders) as compared to the second (good voiders). They therefore concluded that decreased bladder neck mobility could be a precipitating factor in the causation of voiding difficulty. The finding of a mean elevation after colposuspension of only seven millimetres suggests that either the surgeons purposely
performed minimal bladder neck elevation (which was not compatible with the aims of the study), or that the method chosen was inaccurate. In fact, the reproducibility of the measurements was not assessed. Significant loss of accuracy in measuring bladder neck elevation may derive from the use of a laterally placed bone landmark (such as the ischial tuberosity) in relation to a medial structure (such as the bladder neck). Even a slight lateral tilt of the pelvis would result in a large variation of elevation measures.

To date, the issue of whether bladder neck overelevation causes post-operative voiding dysfunction remains unresolved, as no objective measurements using reproducible methods have been performed. Surgeons therefore have no guidelines on how much the bladder neck should be elevated, thus making incontinence surgery more of an art than a science.

b) URETHRAL COMPRESSION

Misplacement of sutures, causing kinking of the urethra, compression and peri-urethral scarring, is a possible cause of post-operative voiding dysfunction. Urethral distortion has been observed in women with long-term dysfunction after incontinence surgery. Lockhart et al [307], using VCU, found some degree of urethral deformity or distortion in five of 11 obstructed patients after incontinence surgery. Zimmern et al [310] found urethral deviation using urethroscopy and/or voiding urethrogram in five of 13 obstructed patients. Carr and Webster [36] reported mid-urethral kinking at urethroscopy in six of 51 women obstructed by incontinence surgery. Other authors reporting on obstructed patients did not quantify the incidence of urethral deformity [311], or did not report finding it [312,313]. Misplacement of sutures causing gross urethral distortion, while possible, does not appear to be a frequent cause of post-operative voiding dysfunction.
Subtle differences in technique of colposuspension may lead to different degrees of urethral compression. The Marshall-Marchetti-Krantz procedure, where stitches are placed in close proximity to the urethra, has been found to induce a later resumption of spontaneous voiding as compared to the Burch colposuspension [201]. Tanagho’s modification of the Burch colposuspension, where stitches are deliberately placed as far away from the urethra as possible, has been claimed to reduce the risk of voiding dysfunction, despite the lack of supporting evidence [31].

To date, no objective measures have been proposed to quantify subtle degrees of urethral compression.

Differences in urethral rigidity may predispose some women to urethral compression and obstruction. Lose et al [24] found that an increased pre-operative urethral resistance (defined as \( p\text{det},Q\max/Q\max^2 \)) was predictive of ‘late’ voiding dysfunction. They postulated that increased urethral resistance was due to increased urethral rigidity, as most patients with ‘late’ voiding dysfunction had undergone previous anterior colporrhaphy. Conversely, Milani et al [289], did not find an association between pre-operative urethral resistance and voiding function at six months follow-up. However, urethral resistance may not be an appropriate measure of urethral rigidity, as urethral distensibility is not included in the equation [314]. Attempts have been made to quantify the elastic properties of the urethra by using complex mathematical models based on pressure-flow curves [315], and by measuring urethral distensibility mechanically using balloons [316] or probes of progressively increasing diameter [317]. These methods have yet to find a useful clinical application and have never been used for the prediction of post-operative voiding dysfunction.
c) POOR DETRUSOR FUNCTION

The operation of colposuspension introduces an element of obstruction, as shown by the significant increase in detrusor pressure and decrease in peak flow rate which occurs post-operatively (chapter 5). While obstruction would seem to be the prime cause of voiding dysfunction after colposuspension, poor detrusor contractility may be an additional important factor. An intrinsically weak detrusor may be unable to cope with even the slightest increase in outflow resistance. In addition, temporary detrusor failure may occur in the post-operative period due to various surgical factors (e.g. anaesthesia, anticholinergics, analgesics, bladder overdistention).

Detrusor contractility has been assessed pre-operatively using a variety of methods:

Pressure/flow studies have been used as a measure of detrusor contractility for the prediction of voiding dysfunction after colposuspension (table 9).

Table 9. Urodynamic prediction of post-operative voiding dysfunction.

<table>
<thead>
<tr>
<th></th>
<th>low Qmax</th>
<th>low pdet,max</th>
<th>low pdet,Qmax</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stanton et al</td>
<td>Predictive</td>
<td>Not predictive</td>
<td></td>
</tr>
<tr>
<td>1978 [21]</td>
<td>Predictive</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dwyer and Stanton 1986 [29]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bathia and Bergman 1986 [290]</td>
<td>Not predictive</td>
<td>Predictive</td>
<td></td>
</tr>
<tr>
<td>Norton and Stanton 1988 [32]</td>
<td>Predictive</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bathia et al 1989 [200]</td>
<td>Predictive</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kremer and Freeman 1995 [27]</td>
<td>Not predictive</td>
<td>Not predictive</td>
<td>Not predictive</td>
</tr>
<tr>
<td>Heit et al 1997 [304]</td>
<td>Not predictive</td>
<td>Not predictive</td>
<td>Not predictive</td>
</tr>
</tbody>
</table>
The inconsistency of findings may be due to methodological differences. In most studies, patients are grouped as having 'low detrusor pressure' as opposed to 'normal detrusor pressure', on the basis of whether they void with pressures of less than 15 cmH2O [24,200,290], or less than 20 cmH2O [27,32]. Grouping of patients using arbitrary values may be inappropriate as normal values of voiding pressure have not been clearly defined. While studies of normal continent women suggest that voiding always occurs in the presence of a detrusor contraction greater than 15 cmH2O, it has been shown that many women with stress incontinence void without a detrusor contraction (chapter 6, section 2). Also rigid definitions of voiding dysfunction result in arbitrary grouping of patients. As a consequence, a patient that resumes spontaneous voiding eight days after surgery would be classed as normal in one study [21] and abnormal in another study [290]. The only study that assessed the predictive value of pre-operative urodynamic variables using the day of catheter removal as their endpoint for analysis (thus avoiding arbitrary definitions), concluded that pre-operative voiding studies were not predictive of duration of post-operative catheterisation [304].

The type of pre-operative voiding pattern has been correlated to post-operative voiding dysfunction, showing an equally inconsistent predictive value. Voiding with a detrusor contraction has been found to be protective against the development of post-operative voiding dysfunction, with one study showing that none of these patients required prolonged catheterization [22]. Similarly, voiding without a detrusor contraction has been shown to be a risk factor, with half of such patients developing voiding dysfunction [24]. Voiding without a detrusor contraction and with additional abdominal straining has also been found to be a risk factor for the development of post-operative voiding dysfunction [22,318]. However, Milani et al [289] reached different conclusions after their urodynamic assessment of 129 women prior to a colposuspension.
operation. They classified voiding function as: ‘detrusorial’, in the presence of detrusor pressure during voiding greater than 15 cmH2O (56.5% of women); ‘abdominal’, in the presence of abdominal straining with or without a detrusor contraction (32.5% of women); and ‘perineal’, in the absence of detrusor contraction and abdominal straining (11% of women). The mean time for resumption of spontaneous voiding after surgery was similar in the three groups.

The inconsistency of these findings may be partly explained by the lack of a clear definition of the concept of ‘voiding with abdominal straining’: most studies define abdominal straining simply as an abdominal pressure rise greater than 10 cmH2O. The number, size, and timing of these pressure rises may need to be defined in order to be meaningful, as a single pressure rise at the end of voiding may not have the same importance as repeated straining throughout voiding.

Detrusor contractility has been assessed by measuring the detrusor pressure that is generated when a voluntary contraction of the external (distal) sphincter interrupts the flow, the so called stop test [319]. However, clinical studies have not found the stop test predictive of post-operative voiding dysfunction [32]. Reflex detrusor inhibition induced by voluntary striated sphincter activation may underestimate the maximum detrusor pressure. In addition, patients are often unable to stop the flow of urine due to a weak pelvic floor or the presence of detrusor instability.

The presence of an ‘after contraction’ at the end of micturition may be considered a form of ‘stop test’. It should be noticed that ‘after contractions’ have been shown to occur in only 60% of normal women [272]. This urodynamic variable has not been assessed as a predictor of post-operative voiding dysfunction.
The **detrusor shortening velocity** [320], which reflects the speed of detrusor muscle shortening, aims to assess detrusor function independently of urethral resistance. It can be obtained using a mathematical formula based on urodynamic parameters which include the peak flow rate and the maximum detrusor pressure in the absence of flow (recorded prior to voiding or after the stop-flow test).

A low detrusor muscle shortening velocity has been found to be associated with post-operative voiding dysfunction [302]. This suggests that the capacity of individual bladders to overcome obstruction may differ amongst patients.

A **continuous occlusion test** has been proposed as a measure of potential detrusor strength [321]. An isovolumetric detrusor contraction is obtained by occluding the outlet with a balloon catheter positioned at the bladder neck before the initiation of a voiding contraction. Simultaneous electromyography has shown that detrusor inhibition due to reflex sphincter contraction does not occur. This test has not yet been applied to the prediction of post-operative voiding dysfunction.

While the overall evidence suggests that poor detrusor power may contribute to post-operative voiding dysfunction, its importance in relation to other possible factors has never been assessed. Moreover, the best method to assess detrusor power has not yet been established.

Whether temporary loss of detrusor contractility occurs in the early days after colposuspension is not known. No pressure-flow studies have been performed in the immediate post-operative period after colposuspension. However, such studies performed after endoscopic bladder neck suspensions have shown a rise in mean voiding pressure from 28 cmH2O before surgery to 61 cmH2O afterwards, and a rise in mean urethral
resistance from 0.025 to 0.23, more in keeping with an obstructive mechanism than loss of
detrusor contractility [322].

d) PSYCHOLOGICAL FACTORS

Psychosocial disturbances have been reported to cause or to contribute to voiding
dysfunction [269,292,323-325]. Urinary retention may develop secondary to centrally
mediated, unconscious inhibition of either detrusor contraction or pelvic floor relaxation or
both [323].

Psychological factors may contribute to cause post-operative voiding dysfunction.
Stanton et al [326], in a small preliminary study, found Diazepam to be beneficial in
preventing voiding delay after colposuspension, thus suggesting that anxiety could be an
important causative factor. Diazepam could however have acted as a muscle relaxant as
well as a sedative.

To date, no prospective studies using psychological measures have looked at
psychological factors in relation to post-operative voiding dysfunction. The association
remains unproven.

e) AGE

Despite the lack of large population based studies on normal women, and the absence of
longitudinal data, there is growing evidence that the incidence of voiding dysfunction in
women increases with age and that the process of ageing may decrease detrusor
contractility and increase urethral rigidity.
Few studies have investigated age related variations during voiding on normal women. Backman [285], in a study of 151 normal women of all ages, found that the peak flow rate decreased with advancing age. Backman et al [272] reported that older women were more likely to strain abdominally during voiding (4 of 5) than younger women (2 of 10), thus suggesting a possible impairment of voiding function. Sundblad [327] showed lower detrusor pressures during voiding, with an increasing contribution of abdominal straining, with advancing age. In contrast, other authors [179,183,277,284] found no deterioration of peak flow rate values with age, but few women older than 60 were included in these studies.

More data is available on the effects of age on voiding in women with urinary symptoms. Amongst women attending a urogynaecology clinic, women with urodynamically proven voiding dysfunction have been reported to be significantly older than women with normal micturition [269,270].

Urodynamic studies performed by Malone-Lee and Wahedna [328] on 1391 women of all ages with urinary symptoms but no obvious neurological disease, found that older patients had higher residual urine volumes, and that there was evidence of decreased detrusor contractility with advancing age (as reflected by lower values of detrusor shortening velocity, detrusor pressure at maximum flow and maximum detrusor pressure). In contrast, Karram et al [277] could not find an effect of age on voiding detrusor pressure in 70 women with stress incontinence. However, the mean age of their patients was relatively low (47.4 years +/- 10.7 SD).

An increasingly 'obstructive' pattern of void with advancing age has been reported by Malone-Lee [329] after studying the peak flow rate and detrusor pressure of 880 women of all ages.
Anatomical changes in the bladder wall that could lead to **reduced bladder contractility** in the elderly have been described. An increased collagen content in the detrusor muscle in association with increasing age has been found at autopsy [330-332] and in bladder biopsies [333]. A reduction in the number of cholinergic nerves in the normal human bladder with age was described by Gilpin et al [334], following histochemical and electron microscopy studies on 54 neurologically and urodynamically normal patients (of which 44 were women). Depletion of caveolae (surface vescicles) was described by Elbadawi [335] following electron microscopy studies of detrusor biopsies from 13 asymptomatic and urodynamically normal elderly women: the finding suggested de-differentiation of muscle cells with advancing age, which could result in some decrease in contractile activity.

Anatomical changes that could lead to **urethral atrophy and increased rigidity** have also been described.

Reduced striated muscle and vessels content and increased connective tissue in the urethra have been found at autopsy in relation with ageing [336,337].

A decrease in urethral closure pressure in normal women [217] and in women with stress incontinence [53,338], and an increase in urethral diameter [339] have been reported in relation to increasing age. These changes are thought to be related to increased urethral rigidity due to ageing.

Susset et al [340] assessed urethral compliance directly by measuring the pressure necessary to develop a cushion of water between a catheter of increasing size and the urethral mucosa. When using this method in 57 women of different ages they found age to be a significant factor for increased rigidity. This is however contradicted by Wagg et al [341], who measured urethral compliance by using a complex mathematical model based on pressure-flow curves, and found no evidence of decreased urethral
compliance with age. This discrepancy highlights the technical difficulty of measuring urethral rigidity and the lack of data on normal women.

Several reports have highlighted the increased risk of post-operative voiding dysfunction with increasing age after anti-incontinence surgery [27,303,304,309,342]. Only Norton and Stanton [32] did not reach the same conclusion; however, the mean age in their report was not specified and may have been lower.

While it is not clear whether voiding dysfunction is due to 'normal' ageing of the lower urinary tract or to the presence of disease processes, age is at present the only risk factor that has been convincingly associated with the development of post-operative voiding dysfunction after anti-incontinence surgery. Increased urethral rigidity may increase the likelihood of causing obstruction during anti-incontinence surgery, and reduced detrusor power may reduce the ability to cope with it.

**Menopause**

It is not clear whether the menopause has an effect on voiding which is distinct from age. There are no studies showing that the menopause per se has a deleterious effect on voiding function. However, the distal urethra is oestrogen dependent and therefore susceptible to undergo postmenopausal atrophic changes [343]. The reduction in urethral functional length seen after the menopause [217] is likely to be a manifestation of this process. There is evidence that these changes are more pronounced in a minority (18%) of postmenopausal women [344]. These women may have increased urethral rigidity and may be predisposed to the development of voiding dysfunction after surgery. Whether detrusor function is affected by the menopause is unclear. Karram et al [277] have found a lower mean detrusor
pressure during voiding in relation to the menopause in normal women and in women with stress incontinence. This finding is difficult to explain, although oestrogen receptors are known to exist in the bladder [345].

There are no reports suggesting that the menopause constitutes a risk factor for postoperative voiding dysfunction. Heit et al [304] found menopausal status to be predictive of voiding dysfunction, but more as a function of age than oestrogen status, as oestrogen replacement therapy did not reduce the risk of prolonged catheterisation. This is in accordance with observations made by others [27].

g) PREVIOUS SURGERY

**Radical Hysterectomy** is a well recognised cause of long term voiding dysfunction [269]. Anatomical studies by Mundy [346] have shown that the bulk of the plexus formed by terminal parasympathetic branches and pelvic nerves lies below the cardinal ligaments. Damage is likely to occur only if the cardinal ligaments or an unusually long cuff of vagina are removed. Scotti et al [347] evaluated urodynamically 12 patients before and after radical hysterectomy: three women developed long term voiding dysfunction, due to poor detrusor power or to inability to relax the sphincter.

There is controversy over whether simple **hysterectomy** affects voiding function. Smith et al [348] studied a series of 44 women with a previous history of hysterectomy and affected by the ‘urethral syndrome’, and found 28 cases of urethral obstruction. No pressure-flow studies were performed, and the diagnosis of obstruction was made on the basis of the presence of ‘trabeculation’ at cystoscopy, a sign which may be found also in patients with detrusor instability. Similarly, Parys et al [349] reported on four patients, from a group of 36 women, who developed urethral obstruction after simple hysterectomy;
they were shown to have abnormal pelvic nerve conduction studies, having had normal studies prior to the procedure. Sphincter spasticity and periurethral fibrosis were thought to be possible causes of obstruction. No definition was given of ‘obstruction’.

On the other hand, clinical and urodynamical studies have found no evidence of increased voiding dysfunction after vaginal hysterectomy (with anterior colporrhaphy performed at the same time) [350] and after abdominal hysterectomy [351].

Lalos and Bjerle [352] randomised 22 women to total or sub-total hysterectomy and reported that bladder function was not altered by either procedure, as shown by pre and post-operative urodynamic studies. Dwyer and Desmedt [269] could find no difference in the incidence of hysterectomy between patients with and without voiding dysfunction presenting to a Urogynecology clinic.

Finally, Kremer and Freeman [27] found no difference in the incidence of voiding dysfunction after colposuspension in patients with a past history of hysterectomy (17 of 51).

It can therefore be concluded that simple hysterectomy does not seem to cause voiding dysfunction, and that a history of hysterectomy does not constitute a risk factor for the development of post-operative voiding dysfunction after colposuspension.

It is also controversial whether a past history of bladder neck surgery predisposes to voiding dysfunction after colposuspension. While anterior colporrhaphy per se is unlikely to cause voiding dysfunction [269,270,350], it has been reported that patients with a past history of this type of surgery are at increased risk, possibly due to increased urethral rigidity [24,302,303]. However, other studies have not confirmed this finding [27,257].

Differences in the definition of voiding dysfunction and lack of detail with regards to the type of bladder neck surgery may explain these discrepancies. The matter remains unresolved.
h) CONCOMITANT SURGERY

Hysterectomy [27,318] and posterior repair [303,318], performed at the same time of colposuspension, have not been shown to increase the incidence of post-operative voiding dysfunction after colposuspension.

6- CLINICAL EFFECTS

Inability to void leads to prolonged use of urinary catheters and an increased incidence of urinary tract infection. The risk of acquiring catheter-associated bacteriuria relates to the duration of catheterisation and ranges from 4% to 7.5% per day over the first ten days of catheterisation [353]. Hospital stay is often prolonged and, when patients are discharged with a catheter, nursing care in the community is needed. Costs therefore escalate.

The specific symptoms of long term voiding dysfunction after colposuspension have not yet been reported in detail. Surgical reports on patients that underwent urethrolysis for presumed obstruction after incontinence surgery [36,311,354] suggest that typical symptoms of voiding difficulty are often combined with irritative symptoms (with and without coexistent detrusor instability). The majority of patients on long-term clean intermittent self catheterisation have bacteriuria and about one third of them require intermittent treatment with antibiotics [355]. In the long term, inability to void may lead to upper tract changes, and periodical checks by means of renal ultrasonography are normally performed. Inability to void may also cause profound alterations in quality of life and may have serious psychological effects. These adverse effects have yet to be quantified.
Unfortunately, the development of post-operative voiding dysfunction is not a sign of a better repair. Various authors have reported no correlation between the time required for spontaneous voiding and the likelihood of success in the long term [29,191,258,289,302].

7- INVESTIGATIONS

Uroflow features that are considered suggestive of voiding dysfunction in the female are changes in the normal bell-shaped curve, with intermittent or multiple peaked flow, suggestive of abdominal straining or unsustained bladder contractions, and/or low flow rates [356]. Although there are no clear-cut values, a peak flow rate of less than 15 ml/s on at least two occasions or residual urine volumes in excess of 100 ml are generally considered abnormal and suggestive of voiding dysfunction. Uroflowmetry however is considered only a screening test.

The assessment of voiding dysfunction in the female, like in the male, relies on pressure-flow studies. While in men low flow in the presence of high detrusor pressure usually signifies obstruction, and nomograms based on pressure-flow data are used to diagnose obstruction [357], in women a clear distinction between obstruction and poor detrusor function on the basis of pressure-flow studies is very difficult.

The presence of a detrusor contraction greater than 20 cmH2O [358], 35 cmH2O [359], or 60 cmH2O [360], in the presence of low or no flow, have all been considered suggestive of obstruction. Similarly, detrusor contractions of less than 15 cmH2O [24,200,289,290] or 20 cmH2O [27], have been arbitrarily considered suggestive of poor detrusor power (as opposed to obstruction) in women with voiding dysfunction. No data have been provided to justify these definitions.
In an attempt to obtain cut-off values for the definition of obstruction in women, urodynamic studies have been performed in obstructed patients and compared to controls. Zimmern et al [361] compared the pressure-flow values of 35 obstructed patients, to 120 non-obstructed controls. The presence of a peak flow rate of less than 10 ml/s with a detrusor pressure at maximum flow of more than 30 cmH2O, had a sensitivity of 83% and a specificity of 89% for the diagnosis of obstruction. Similarly, Massey and Abrams [362] studied 163 obstructed women and defined obstruction by the presence of two or more of the following parameters: (1) Flow rate (F) < 12 ml/s. (2) Detrusor pressure at maximum flow (P) > 50 cmH2O. (3) Urethral resistance (P/F^2) greater than 0.2. (4) ‘Significant’ (not quantified) residual urine in the presence of a raised detrusor pressure or urethral resistance. The practical value of these observations remains unclear, as in both studies the clinical and radiological criteria used to define obstruction were not specified, and the clinical features of the controls were not mentioned. In addition, no attempt was made to investigate poor detrusor function.

In contrast, other authors do not rely heavily on strict urodynamic criteria for the diagnosis of obstruction. Farrar et al [363] considered voiding essentially as a matter of balance between voiding forces on the one hand and outflow resistance on the other. They believed that ‘relative obstruction’ could exist in the presence of normal or low detrusor pressures, and defined obstruction as the presence of a peak flow rate of less than 15 ml/s in the presence of any amount of detrusor muscle activity. A similar view was taken by Carr and Webster [36], who diagnosed women as obstructed after incontinence surgery (and therefore suitable to undergo urethrolysis) purely on the basis of a clear-cut temporal relationship between surgery and the onset of persistent voiding problems and/or irritative symptoms. They supported their argument by performing successful urethrolysis even on women who had acontractile bladders (chapter 6, section 8).
The reported range of detrusor pressures in patients investigated for voiding dysfunction after incontinence surgery and considered ‘obstructed’ (after imaging studies and/or urethroscopy) is very wide (table 10).

Table 10. Detrusor pressure values in ‘obstructed’ women after incontinence surgery.

<table>
<thead>
<tr>
<th>Authors</th>
<th>n. patients</th>
<th>Detrusor pressure (pdet,max) (cmH2O)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Mean: 59</td>
</tr>
<tr>
<td>Webster and Kreder 1990 [313]</td>
<td>15</td>
<td>Range: 0-80</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Mean: 34</td>
</tr>
<tr>
<td>Foster and McGuire 1993 [364]</td>
<td>48</td>
<td>&lt; 30 cmH2O: 40%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Acontractile: 64%</td>
</tr>
<tr>
<td>Nitti and Raz 1994 [311]</td>
<td>41</td>
<td>&gt; 40 cmH2O: 16%</td>
</tr>
<tr>
<td>Austin et al 1996 [365]</td>
<td>18</td>
<td>0-40 cmH2O: 20%</td>
</tr>
<tr>
<td>Carr and Webster 1997 [36]</td>
<td>51</td>
<td>&lt; 30 cmH2O: 27%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Acontractile: 15%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>&gt; 40 cmH2O: 21%</td>
</tr>
</tbody>
</table>

Many ‘obstructed’ patients who were cured by a take-down procedure (chapter 6, section 8), had detrusor pressures of less than 30 cmH2O [36,311,313,364,365], or even had acontractile bladders [36,364], thus challenging the concept that obstruction exists only in the presence of high pressures. ‘Relative’ obstruction may therefore occur in the presence of low detrusor pressure, and a poorly contractile bladder may only become obvious after the relief of obstruction [312].

More detailed studies on patients with prolonged post-operative voiding dysfunction have been performed using video urodynamics [36,307,310-313,364-367]. Anatomical deformities, such as a narrowed or deviated urethra leading to obstruction, have been detected in a minority of patients [307,310,311] (chapter 6, section 5b).
Urethral profilometry is not considered useful for the investigation of voiding dysfunction in the female [312,313,362], while urethroscopy has been used to identify urethral deviation caused by a peri-urethral suture [310,313].

8- TREATMENT

In the short term, following bladder neck surgery, suprapubic catheters are preferred. They are more practical and allow patients to attempt urethral voiding without the need to be re-catheterised. Comparing to urethral catheters, they have a lower incidence of significant bacteriuria [243,368,369] and voiding occurs earlier [368,369]. In addition, patient acceptability appears to be higher [368].

In the medium to long term, clean intermittent self catheterization (CISC) as suggested by Lapides [370] is now considered the standard treatment. The advantages over an indwelling catheter are a lower incidence of bacteriuria and an ‘improved mental status’ [355]. Early CISC has been shown to have advantages in comparison to suprapubic bladder drainage in terms of hospital stay and infectious morbidity [371]. Also, CISC is easier to learn before the bladder neck is elevated. However, such a practice requires considerable resources, as all patients need to be instructed on CISC before surgery. Also, not all patients wish or are able to master the technique.

The effect of drugs for the treatment of post-operative voiding dysfunction after anti-incontinence surgery has been disappointing. Alpha-adrenergic antagonists to relax the urethra and bladder neck (e.g. Phenoxybenzamine), and detrusor stimulating drugs such as cholinergic agents (e.g. Bethanecol), anticholinesterase (e.g. Distigmine bromide) and
prostaglandins, have been used empirically with inconsistent results. The rationale for the
use of these drugs is not clear as women have few alpha receptors in the bladder neck and
urethra (unlike in the male), while there is no convincing evidence that post-operative
voiding dysfunction is due to poor detrusor function.

Most studies have investigated the potential benefits of drugs in relation to procedures
not likely to cause long-term voiding dysfunction (e.g. vaginal hysterectomy, anterior
colporrhaphy for prolapse), and there is little information on the efficacy of drugs after
anti-incontinence surgery.

Stanton et al [326] compared Diazepam, Phenoxybenzamine, intravesical Prostaglandin
E2 and Bethanecol chloride, in an uncontrolled non-randomised study of 40 women
undergoing the operation of colposuspension. Diazepam was found to be the most effective
drug.

Wagner et al [372] compared intravesical Prostaglandin E2 given on post-operative days
6-7 with placebo in a randomised study of 28 patients with urinary retention after anterior
colporrhaphy for stress incontinence (technique not specified). There was no clinical effect
of note.

Tammela et al [373] compared intravesical Prostaglandin F2 alpha with placebo in a
randomised study of 36 women with voiding dysfunction after various types of anti-
incontinence surgery. Prostaglandin F2 alpha was superior to placebo in the short term. The
long term benefit could not be assessed because no patients in either group experienced
prolonged voiding dysfunction.

Koonings et al [374] randomised 69 women after a Pereyra procedure (n=28) or Burch
colposuspension (n=41) to prophylactic intravesical PGF2 alpha, vaginal PGE2, or
placebo. Both PGE2 and PGF2 alpha significantly reduced the length of time required for
post-operative bladder drainage as compared to the control group. Also in this study there
were no cases of prolonged voiding dysfunction, and even in the control group the mean
number of days of catheter requirement was only four. It is therefore difficult to draw any conclusions as to the prophylactic effect of Prostaglandins on prolonged voiding dysfunction.

As mentioned previously, oestrogens do not appear to be of value either.

Due to their inconsistent effect and unproven value, drugs are not currently recommended for the prevention and treatment of post-operative voiding dysfunction.

Various *surgical procedures* have been performed on patients with post-operative voiding dysfunction with mixed results.

**Urethral dilatation and internal urethrotomy**

Urethral dilatation with Hegars up to 12-14 size and urethral incisions have been reported to be effective in patients with voiding difficulties due to 'urethral narrowing' following anti-incontinence procedures [375]. However, the benefit seems to be short lived as scarring is likely to recur and may even result in worsening of the problem. In addition, stress incontinence has been reported to recur [292,376].

Moolgaoker et al [377] reported that prophylactic urethrotomy and, to a lesser extent, urethral dilatation, performed at the time of surgery for stress incontinence, resulted in earlier resumption of voiding (comparing to patients that had neither procedure performed). There were no negative effects on the two year cure rate. Patients were not randomised, and of 128 patients, only five had sling procedures and all others had an anterior colporrhaphy with bladder neck buttressing, a procedure not likely to result in obstruction.

There is at present no rational basis for the performance of urethral dilatation or urethrotomy for the prophylaxis or treatment of post-operative voiding dysfunction and there is evidence that these procedures might be harmful.
Urethrolysis (with and without re-suspension)

Suprapubic and vaginal take-down procedures, involving urethrolysis, with or without additional re-suspension procedures, have been described by various Authors as an alternative to CISC for patients who were considered 'obstructed' after surgery for incontinence. Patients usually had a combination of irritative symptoms and symptoms of voiding dysfunction. Their urodynamic features are presented in table 10. Methods and outcome are presented in table 11. In all studies success is assessed subjectively and is usually defined as the complete resolution of symptoms.

Table 11. Methods and outcome of take-down procedures for voiding dysfunction after incontinence surgery.

<table>
<thead>
<tr>
<th>Route</th>
<th>n. women</th>
<th>n. women repeat suspension</th>
<th>Success %</th>
<th>n. recurrent GSI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zimmern et al (1987) [310]</td>
<td>Vaginal</td>
<td>13</td>
<td>92%</td>
<td>0</td>
</tr>
<tr>
<td>McGuire et al (1989) [312]</td>
<td>Vaginal</td>
<td>13</td>
<td>76%</td>
<td>0</td>
</tr>
<tr>
<td>Webster and Kreder (1990) [313]</td>
<td>Abdominal</td>
<td>15</td>
<td>93%</td>
<td>2</td>
</tr>
<tr>
<td>Foster and McGuire (1993) [364]</td>
<td>Vaginal</td>
<td>48</td>
<td>65%</td>
<td>0</td>
</tr>
<tr>
<td>Nitti and Raz (1994) [311]</td>
<td>Vaginal</td>
<td>41</td>
<td>71%</td>
<td>0</td>
</tr>
<tr>
<td>Austin et al (1996) [365]</td>
<td>Vaginal</td>
<td>16</td>
<td>50%</td>
<td>1</td>
</tr>
<tr>
<td>Carr and Webster (1997) [36]</td>
<td>Abdominal or vaginal</td>
<td>51</td>
<td>58%</td>
<td>7</td>
</tr>
</tbody>
</table>

Adverse factors for urethrolysis were the presence of detrusor instability [364] and high pre-operative post-void residual [311]. Absence of a detrusor contraction before urethrolysis has not been found to constitute an adverse factor [311,364].
Despite the lack of uniformity with regards to patient selection, time since original operation, definition of obstruction, surgical treatment, and outcome measures, it would appear that urethrolysis is a reasonable alternative to CISC, suitable perhaps for patients that are unwilling or unable to master the technique. The reported success rate is variable and unpredictable, and there is a risk of recurrent stress incontinence.

CONCLUSIONS

The reported frequency of voiding dysfunction after the operation of colposuspension varies considerably, suggesting that subtle differences in operative technique are likely to be important. Urethral compression caused by too medial placement of sutures may be a factor, but evidence is not conclusive. Bladder neck overerelevation is another possible factor, but it has never been assessed with strict methodology. Anatomical changes induced by surgery need to be measured objectively and accurately before they can be correlated to the development of voiding dysfunction. It may then be possible to advise clinicians on how to prevent this distressing complication.

Factors related to the patient, such as age and poor detrusor contractility, may also play a role. Their relative importance needs to be quantified, so that women at risk of prolonged voiding dysfunction can be detected prior to surgery.
CHAPTER 7

DETRUSOR INSTABILITY AFTER SURGERY FOR STRESS INCONTINENCE
Detrusor instability is a condition which causes symptoms of urinary frequency, nocturia, urgency and urge incontinence. It is a common cause of urinary incontinence and occurs in approximately one third of women undergoing urodynamic studies [9]. Detrusor instability has been shown to have a greater adverse effect on quality of life than genuine stress incontinence [378] and treatment in the medium to long term is largely disappointing [379]. It is therefore of great concern that surgical treatment of genuine stress incontinence can result in the development of this condition.

In this chapter, the incidence, natural history and management of detrusor instability occurring de novo after incontinence surgery will be presented, and possible causes will be discussed. The effects of incontinence surgery on pre-existent detrusor instability will also be discussed as the possibility exists that de novo detrusor instability constitutes an undiagnosed pre-operative problem.

1- DEFINITION

The unstable detrusor, as defined by the International Continence Society, is one that is shown objectively to contract, spontaneously or on provocation, during the filling phase of cystometry, while the patient is attempting to inhibit micturition [1]. The ‘normal’ bladder during the filling phase expands to accommodate the increasing volume without a significant rise in pressure. This observation had already been made by Mosso and Pellacani in 1882 [171], was later confirmed by Denny-Brown and Robertson in 1933 [172], and is described as a standard of normal bladder behaviour by the ICS [1]. While unstable detrusor contractions may be asymptomatic [1], they are generally considered abnormal and give rise to the sensation of urgency and may result in urge
incontinence. When they are documented by cystometry, in the presence of symptoms of frequency, urgency or urge incontinence, a diagnosis of detrusor instability can be made.

The correlation of symptoms with cystometric findings is complex, and the significance of unstable detrusor contractions is often unclear. Isolated symptoms of urgency and urge incontinence have been found to be associated with a urodynamic diagnosis of detrusor instability in only 51-77% of cases [5,6,9], thus suggesting a high false negative rate. The poor specificity of cystometry is highlighted by studies showing urodynamic evidence of detrusor instability in 35% of women with the isolated symptom of stress incontinence [6] and even in 18% of normal asymptomatic volunteers [276,279]. An even higher incidence of detrusor instability of 38-69% [276,278,279] has been found in asymptomatic volunteers after an extended period of observation by ambulatory monitoring, thus suggesting that unstable detrusor contractions may to a certain extent be physiological. In this respect, it is of interest that a study on asymptomatic female volunteers using suprapubic catheterisation showed that none had unstable detrusor contractions on static cystometry [274], thus suggesting that they may be an artefact produced by irritation from urethral catheters.

2- CAUSES

In clinical practice, detrusor instability is classified as neuropathic (when it is defined as 'detrusor hyperreflexia'), obstructive or idiopathic. More recently the term 'overactive bladder' has been introduced, which indicates both detrusor instability and detrusor hyperreflexia and is easier for patients to understand.

The passive function of accommodation of the bladder to increasing volumes is dependent on the quiescence of the parasympathetic reflex. Detrusor hyperreflexia can result from lesions of the cerebral cortex (e.g. Parkinson’s disease and cerebro-vascular accidents), the
spinal cord (e.g. spinal injury, multiple sclerosis, cord compression, spina bifida), and the pelvic nerves (e.g. diabetes, iatrogenic injury). These can disrupt the integrity of the micturition reflex and of the higher pathways which control it, thus leading to involuntary detrusor contractions.

**Bladder outflow obstruction** has been associated with detrusor instability in men. The occurrence of instability in men has been correlated with the degree of obstruction [380] and relief of obstruction has resulted in reversion from unstable to stable detrusor behaviour in up to two thirds of patients [381]. This association has not been clearly shown in women, among whom obstruction is rare.

**Idiopathic detrusor instability** occurs in patients that have neither evidence of neuropathy nor evidence of obstruction.

The similarity in clinical symptoms and cystometric appearances in these three groups has raised the possibility of a common pathophysiological process. There is increasing experimental evidence that the occurrence of unstable detrusor contractions may be due to the development of post-junctional supersensitivity of the detrusor muscle secondary to a disorder in neurotransmission [381,382]. Post-junctional supersensitivity is the result of a chronic decrease in the normal contact between a neurotransmitter and the smooth muscle cell, as may occur after partial denervation. As a consequence, a non-specific increase in smooth muscle sensitivity occurs, thus lowering the threshold for muscle contraction. Detrusor muscle hypertrophy, which has been shown to occur in women with idiopathic detrusor instability using ultrasound [383], may then simply be a consequence of detrusor supersensitivity.

The cause of de novo detrusor instability after surgery for genuine stress incontinence is unknown.
It has been suggested that detrusor instability that develops after surgery may be present pre-operatively and that low outflow resistance seen in GSI may make pre-operative diagnosis difficult. The presence of unstable detrusor contractions on ambulatory urodynamics in patients with a stable bladder on static cystometry has been shown to predict the development of post-operative symptoms of detrusor instability, and the method has been proposed as a mean to diagnose occult pre-operative detrusor instability [384,385]. However, the practical value of ambulatory urodynamics in this context is probably limited, as the test has been shown to detect detrusor contractions in up to 70% of asymptomatic women [276,279]. Increased bladder wall thickness measured on ultrasound has been shown in association with detrusor instability [383] and a pre-operative bladder wall thickness greater than five millimetres has been shown to predict the development of irrititative symptoms after colposuspension [386]. While this observation lends support to the concept that some patients may be predisposed to the development of post-operative detrusor instability, the exact clinical value of bladder wall thickness assessment in this context awaits confirmation.

Obstruction would seem to be a natural explanation as the colposuspension operation has been shown to be an obstructive procedure (chapter 5). Pressure/flow studies before and after surgery have not confirmed this hypothesis. Cardozo et al [33] have studied 75 patients with follow-up at three and 15 months. Patients were divided in ‘stable’ and unstable’ (18.5%) groups: there was no difference in pressure/flow variables between the two groups. They concluded that the development of post-operative detrusor instability was not due to excessive urethral compression. However it is of interest that the peak flow rate in the ‘unstable’ group was significantly reduced post-operatively (from a mean of 24 ml/s before surgery down to a mean of 15 ml/s after surgery), while this change did not occur in the ‘stable’ group. Although detrusor pressure during voiding did not rise significantly after
surgery in the 'unstable' group, this may still suggest a mild degree of obstruction, as obstructed women do not necessarily respond with a detrusor pressure rise (chapter 6, table 10). Pressure/flow studies therefore may not be sensitive enough to detect minor degrees of obstruction.

In favour of an obstructive hypothesis are the findings of Bump et al [387], who performed urethral pressure profilometry after incontinence surgery on patients with post-operative detrusor instability, patients without post-operative detrusor instability and patients with pure detrusor instability that did not undergo surgery: pressure transmission ratios were significantly higher and greater than 100% in patients with post-operative detrusor instability as compared to the other groups. They postulated that surgery had overcorrected the urethral position and caused relative obstruction and considered that post-operative detrusor instability is a condition that differs from idiopathic detrusor instability. In addition, clear evidence of obstruction (on VCU) has been reported in 29% of women with post-operative detrusor instability [307] and surgical treatment of this condition with take-down procedures (and relief of obstruction) has been shown to be effective (chapter 7, section 6).

It has been suggested that surgical dissection of the bladder base may damage the nerve supply to the bladder and cause detrusor instability [33,388]. The motor function of the detrusor bladder muscle is controlled by the parasympathetic nervous system via the pelvic nerves. These nerves originate from the sacral spinal cord in segments S2-S4 and run to Frankenhauser's plexus and the bladder following its vascular supply. Autonomic terminal nerves are then distributed to the bladder in the retro-trigonal region, where damage due to surgical dissection may occur [49]. However, anatomical and surgical considerations do not clearly support a neurogenic theory. The greater concentration of terminal nerves has been shown to be in the 4 o'clock
and 8 o’clock positions [49,389], and vaginal dissection has been shown to be more likely to cause nerve damage than abdominal dissection [390]. A higher incidence of *de novo* detrusor instability after vaginal surgery (such as anterior colporrhaphy) should then be expected, compared to retropubic procedures, but there is no evidence that this occurs [15]. In addition, procedures such as needle suspensions where minimal surgical dissection is performed (and where nerve damage should be minimal), should be associated with a very low incidence of *de novo* detrusor instability. In fact this complication has been reported as frequently after needle suspension as after colposuspension [18].

An increased likelihood of *de novo* detrusor instability after incontinence surgery in patients with a past history of bladder neck surgery would also support a neurogenic theory. Cardozo et al [33] had initially reported that women with post-operative *de novo* detrusor instability had a higher mean number of previous operations for incontinence as compared to women whose bladders remained stable after surgery. Although in a subsequent report the same group [34] stated that the difference was not statistically significant, there was a significant trend towards the development of *de novo* detrusor instability with an increasing number of previous bladder neck operations. Sand et al [35] did not find previous incontinence surgery to be a risk factor for the development of *de novo* detrusor instability. However, they analysed their data by individual type of previous incontinence procedure, which may have been inappropriate given the low number of women who developed *de novo* detrusor instability (n=5). Observation of their data shows that all women who developed this complication had actually had previous incontinence surgery.

Recently, Cardozo et al [391] have reported the results of colposuspension on 52 women with a past history of bladder neck surgery (including vaginal and abdominal procedures), and there were no cases of *de novo* detrusor instability. There is therefore no conclusive evidence that a past history of bladder neck surgery increases the risk of *de novo* detrusor instability.
It has also been proposed that post-operative detrusor instability may be due misplacement of sutures and creation of a supportive shelf under the trigone rather than under the bladder neck. This would result in irritation of a sensitive area and development of symptoms of urgency. Although this is an interesting hypothesis, it remains unsubstantiated as it results from observations made with vaginal ultrasound on only two patients with symptoms of urgency and no urodynamic studies [221].

3. INCIDENCE

Post-operative de novo detrusor instability has been reported in 8% of patients after anterior colporrhaphy, 11% after Marshall-Marchetti-Krantz procedure, 9.6% after colposuspension, 5.8% after needle suspension procedures, and 16.6% after sling procedures [15]. This complication has been reported to occur also after paravaginal repair performed for genuine stress incontinence [392]. Injectables are normally considered to be free from complications [15], although a surprisingly high incidence of de novo detrusor instability of 39% has been reported in a recent series [228]. The incidence of this complication with the tension free vaginal tape is not yet clear, although their promoters claim that urge incontinence does not occur [227]. Unfortunately, valid and reliable data on the frequency of complications following incontinence surgery are lacking [16] and it is difficult to draw conclusions as to the potential of each procedure to cause de novo detrusor instability.

Some large retrospective comparative studies suggest that no individual operation has a significantly higher rate of occurrence of de novo detrusor instability [393,394], but retropubic procedures overall have a higher rate than anterior colporrhaphy [393]. Little information with regards to de novo detrusor instability can be obtained from the few
prospective randomised comparative studies [18,19,197,226]. Mundy [18] reported an incidence of 20% after needle suspension compared to only 7.5% after colposuspension. Enzelsberger et al [197] reported an incidence of 16% after sling procedures comparing to only 8% after colposuspension. Numbers were however small and the differences were not statistically significant.

All reports on \textit{de novo} detrusor instability after the operation of colposuspension supported by pre and post-operative urodynamics on all patients are included in Table 12. The literature search was conducted using the same method described for voiding dysfunction (p. 121). Reports on more than one type of operation (where it is impossible to unravel the proportion of instability for each procedure), and reports with incomplete urodynamic assessment, are excluded. Long-term reports with urodynamics performed years after surgery are also excluded due to the difficulty in such cases to establish a direct link between cause and effect. It should be noted that the same patients may have been included in more than one report as some authors have reported on more than one occasion (e.g. Walter et al, 1978 and Walter et al, 1982, Lose et al, 1987 and Jorgensen et al, 1988, Colombo et al, 1994 and Colombo et al, 1996).
Table 12. Incidence of *de novo* detrusor instability (% *de novo* DI) after colposuspension.

<table>
<thead>
<tr>
<th>n. women</th>
<th>Follow-up</th>
<th>% de novo DI</th>
<th>Technique</th>
</tr>
</thead>
<tbody>
<tr>
<td>Walter et al 1978 [198]</td>
<td>37</td>
<td>1 year</td>
<td>5.4% (all symptomatic)</td>
</tr>
<tr>
<td>Cardozo et al 1979* [33]</td>
<td>92</td>
<td>3 months</td>
<td>18.5% (47% symptomatic)</td>
</tr>
<tr>
<td>Walter et al 1982 [88]</td>
<td>38</td>
<td>12-30 months</td>
<td>5.2% (all symptomatic)</td>
</tr>
<tr>
<td>Mundy 1983 [18]</td>
<td>26</td>
<td>1 year</td>
<td>7.5% (all symptomatic)</td>
</tr>
<tr>
<td>Lose et al 1987 [24]</td>
<td>51</td>
<td>4-72 months</td>
<td>20% (symptoms not specified)</td>
</tr>
<tr>
<td>Sand et al 1988 [35]</td>
<td>66</td>
<td>3-4 months</td>
<td>7.6% (60% symptomatic)</td>
</tr>
<tr>
<td>Langer et al 1988 [215]</td>
<td>62</td>
<td>3-6 months</td>
<td>27% (symptoms not specified)</td>
</tr>
<tr>
<td>Jorgensen et al 1988 [395]</td>
<td>56</td>
<td>Time not specified</td>
<td>20% (symptoms not specified)</td>
</tr>
<tr>
<td>Rizvi et al 1988 [241]</td>
<td>62</td>
<td>3-6 months</td>
<td>3.2% (symptoms not specified)</td>
</tr>
<tr>
<td>Milani et al 1991 [396]*</td>
<td>31</td>
<td>6 months</td>
<td>6.5% (symptoms not specified)</td>
</tr>
<tr>
<td>Colombo et al 1994 [201]</td>
<td>40</td>
<td>6 months</td>
<td>5% (50% symptomatic)</td>
</tr>
<tr>
<td>Colombo et al 1996 [264]*</td>
<td>44</td>
<td>2 years</td>
<td>11.3% (60% symptomatic)</td>
</tr>
<tr>
<td>Heslington and Hilton 1995 [397]</td>
<td>41</td>
<td>3 months</td>
<td>17% (symptoms not specified)</td>
</tr>
</tbody>
</table>

* Includes patients with 'low compliance' detrusor instability

The wide reported range of post-operative detrusor instability may be explained by differences in the time of assessment and differences in cystometric technique. Factors such as rate of filling, position and number and type of provocative manoeuvres are known to influence the results. However even after excluding studies where details of technique were not described [18,215] or were not provocative [88,198], there is no difference in the reported range of *de novo* detrusor instability.

It is not known whether the incidence of *de novo* detrusor instability differs when retropubic suspensions are performed using different methods. A randomised study of
Colposuspension versus Marshall-Marchetti-Krantz procedure suggested that the latter procedure causes a higher number of severe cases of instability [201]. Numbers were however too small to reach statistical significance. Comparisons between open and laparoscopic colposuspension are inconclusive due to lack of data. Evidence so far from a randomised study [236] and a non randomised comparative study [234] suggests that there is no difference between the two methods with regards to the development of post-operative detrusor instability. There also appears to be no difference in this respect between Stanton’s and Tanagho’s method of colposuspension (table 12).

4- CLINICAL EFFECTS

Post-operative detrusor instability is reported to be symptomatic in 47-100% of cases (table 12). Unfortunately reports do not specify the severity of symptoms and their impact on quality of life.

The natural history of post-operative detrusor instability is not clearly known, as only one cohort of patients has been followed-up in the long-term. Cardozo et al [33] performed repeat urodynamics at one year on 12 of 17 patients found to have post-operative detrusor instability three months after surgery: 11 of 12 remained unstable. Their symptoms were not specified. On subsequent follow-up [34] 3-5 years later, one of the original cohort of 17 women was excluded (because on review of her cystometry she was thought to have had detrusor instability pre-operatively), and nine patients were added. A total of 25 patients with post-operative de novo detrusor instability were therefore reviewed: of these 11 were asymptomatic and 14 were symptomatic. Only eight of 25 patients underwent urodynamics: six (symptomatic) women were unstable and two (asymptomatic) women were stable. The only conclusion that can be drawn from these observations is that more than half of patients
found to have *de novo* detrusor instability three months after the colposuspension operation are likely to be symptomatic in the long-term. As no patients who were stable at three months were followed-up in the long-term, it is not known whether post-operative detrusor instability can develop at a later date. It is however of interest that the incidence of detrusor instability 10-20 years after the operation of colposuspension has been reported by the same group of investigators to be 14.7% [17], which is similar to the incidence of 18.5% found in the immediate post-operative period [33].

5- INVESTIGATION

The diagnosis of detrusor instability is made by cystometry. Unfortunately the correlation between symptoms of urgency and urge incontinence and urodynamic evidence of detrusor instability may be even poorer in patients with post-operative detrusor instability. While 51-77% of women with symptoms of urgency and urge incontinence are found to have an unstable bladder on cystometry [5,6,9], unstable contractions are seen in only 0-50% of women with similar symptoms after surgery (table 13). There is therefore concern about the sensitivity of urodynamics for the diagnosis of post-operative detrusor instability. A possible explanation is that the symptoms of urgency and urge incontinence occurring after surgery are not due to unstable detrusor contractions but to another as yet undefined pathology.
Table 13. Incidence of detrusor instability in patients with post-operative symptoms of urgency and urge incontinence.

<table>
<thead>
<tr>
<th>Study</th>
<th>n. of women with urgency and urge incontinence</th>
<th>% with unstable contractions on cystometry</th>
</tr>
</thead>
<tbody>
<tr>
<td>Galloway et al, '87 [23]</td>
<td>7</td>
<td>0%</td>
</tr>
<tr>
<td>Webster and Kreder, '90 [313]</td>
<td>13</td>
<td>38%</td>
</tr>
<tr>
<td>Foster and McGuire, '93 [364]</td>
<td>34</td>
<td>14%</td>
</tr>
<tr>
<td>Nitti and Raz, '94 [311]</td>
<td>30</td>
<td>46%</td>
</tr>
<tr>
<td>Austin et al, '96 [365]</td>
<td>13</td>
<td>46%</td>
</tr>
<tr>
<td>Carr and Webster, '97 [36]</td>
<td>38</td>
<td>50%</td>
</tr>
</tbody>
</table>

Urodynamic studies are also performed in order to establish whether post-operative detrusor instability is due to obstruction. However pressure/flow studies may not be informative when obstruction occurs in women, as many ‘obstructed’ patients have been found to have relatively low detrusor pressures of less than 30 cmH2O or even acontractile bladders (chapter 6, table 10).

Radiographic imaging and cystoscopy have been performed in patients with a mixture of irritative and obstructive symptoms after incontinence surgery, looking for signs of obstruction [36,307,310,311,313,364,365]. In the only study where it is possible to extrapolate the data for detrusor instability, the reported incidence of obstruction (defined as radiographic evidence of urethral distortion) associated with detrusor instability was 29% [307].

6- TREATMENT

The management of detrusor instability relies essentially on behavioural modification and pharmacological intervention, with surgery reserved for extreme cases. There is no standard management of detrusor instability occurring after incontinence surgery. This is due to uncertainty as to the cause of the condition and as to the possible role of obstruction.
Few reports exist on the results of medical treatment in patients with post-operative detrusor instability. Steel et al [34] found that only four of 14 women (16%) with post-operative detrusor instability were helped by medical treatment. In contrast Korda et al [257] found that nine of 16 patients (56%) with post-operative detrusor instability were treated effectively with anticholinergics.

Surgical 'take-down' procedures have been performed in patients who developed severe symptoms of urgency and urge incontinence after incontinence surgery, with and without urodynamic evidence of unstable detrusor contractions. Urethrolysis has been performed abdominally or vaginally, with or without re-suspension. The results of urethrolysis for symptoms of urgency and urge incontinence after incontinence surgery are presented on table 14. Unfortunately in all studies the outcome was only assessed subjectively.

<table>
<thead>
<tr>
<th>n. women (n. with proven DI)</th>
<th>Subjective cure rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Webster and Kreder, 1990 [313] 13 (5 unstable)</td>
<td>100%</td>
</tr>
<tr>
<td>Foster and McGuire, 1993 [364] 5 (all unstable)</td>
<td>40%</td>
</tr>
<tr>
<td>Nitti and Raz, 1994 [311] 30 (14 unstable)</td>
<td>43% for urgency 50% for urge incontinence</td>
</tr>
<tr>
<td>Austin et al, 1996 [365] 13 (6 unstable)</td>
<td>53%</td>
</tr>
<tr>
<td>Cross et al, 1998 [354] 39 (7 of 23 tested)</td>
<td>85%</td>
</tr>
</tbody>
</table>

In view of the unpredictable and variable success rate of take-down procedures for the treatment of post-operative detrusor instability, it has been suggested that surgery should be undertaken only in the presence of convincing evidence of urethral obstruction [311,364]. Others however take the view that obstruction may not be diagnosed with current methods and urethrolysis should be offered if there is a clear temporal relationship between onset of
symptoms and incontinence surgery [36]. As the natural history of detrusor instability after incontinence surgery is unknown, there is no information on when urethrolysis should be performed, although a minimum waiting period of three months for spontaneous improvement of symptoms has been suggested [36,354].

7- MIXED STRESS AND URGE INCONTINENCE

The symptom of urge incontinence is reported by 59% of women with pure genuine stress incontinence [9] and does not necessarily imply the presence of coexistent detrusor instability. Symptoms of urgency and urge incontinence often improve after surgery for genuine stress incontinence, and it has been suggested that per se, should not be considered a contraindication to surgery [41]. However, as many as 23% of women undergoing urodynamics have mixed genuine stress incontinence and detrusor instability [9]. The management of these women is difficult and controversial. Conservative measures (pelvic floor exercises, bladder drill, drug therapy) are usually tried first as they have been shown to reduce the need for surgery [398]. Surgery is usually performed when conservative measures fail, and up to 23-32% of women undergoing colposuspension have been reported to have mixed incontinence [25,35,215,395]. The outcome of surgery in this group of patients is uncertain and higher rates of post-operative incontinence have been reported as compared to patients with pure genuine stress incontinence [204,399]. While pre-operative detrusor instability does not appear to compromise the cure of genuine stress incontinence (chapter 5), surgery has an unpredictable effect on detrusor instability and its reported cure is variable. All reports on the effects of colposuspension on detrusor instability in patients with mixed incontinence which are documented by pre and post-operative urodynamics are included in Table 15. Reports on more than one type of
operation, where it is impossible to unravel the proportion of cases of instability for each procedure, are excluded. Patients with low compliance bladders are also excluded and considered separately (see later).

Table 15. Outcome of surgery in patients with mixed incontinence.

<table>
<thead>
<tr>
<th>n. women</th>
<th>Follow-up</th>
<th>Objective cure (stable bladder)</th>
<th>Technique</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lose et al, 1987 [24]</td>
<td>19</td>
<td>4-72 months</td>
<td>26%</td>
</tr>
<tr>
<td>Sand et al, 1988 [35]</td>
<td>20</td>
<td>3-4 months</td>
<td>55%</td>
</tr>
<tr>
<td>Langer et al, 1988 [215]</td>
<td>30</td>
<td>3-6 months</td>
<td>40%</td>
</tr>
<tr>
<td>Jorgensen et al, 1988 [395]</td>
<td>19</td>
<td>time not specified</td>
<td>21%</td>
</tr>
<tr>
<td>Karram&amp;Bathia, 1989 [398]</td>
<td>27</td>
<td>3-12 months</td>
<td>59%</td>
</tr>
<tr>
<td>Milani et al, 1991 [396]</td>
<td>14</td>
<td>6 months</td>
<td>57%</td>
</tr>
<tr>
<td>Scotti et al, 1996 [400]</td>
<td>46</td>
<td>3-6 months</td>
<td>56.5%</td>
</tr>
<tr>
<td>Colombo et al, 1996 [264]</td>
<td>23</td>
<td>2 years</td>
<td>60%</td>
</tr>
</tbody>
</table>

Reports are generally accurate with regards to the objective effect of surgery on urodynamic evidence of detrusor instability. The reported cure ranges from 21% to 67%, and there is no obvious difference in this respect between Stanton's and Tanagho's methods of colposuspension.

Unfortunately there is little information on subjective results (i.e. symptoms) and impact of surgery on quality of life, particularly on subjects with persistent detrusor instability. This information would be helpful in relation to pre-operative counselling. Failure to cure detrusor instability may be acceptable, as long as treatment of genuine stress incontinence is successful and symptoms of detrusor instability (in particular urge incontinence) do not worsen.
It is not known whether 'low compliance' is an entity separate from detrusor instability. The ICS defines 'low compliance' as a true detrusor pressure rise (greater than 15 cm H2O) without subsequent decrease [1]. It is often regarded as a form of detrusor instability and reported as such [33]. Others consider it as either an artefact due to rapid bladder filling or a 'stiff' bladder wall as a result of neurologic abnormalities or radiation [4]. Treatment with drug therapy has been shown to be equally disappointing in patients with 'low compliance' as in patients with detrusor instability [379]. The prognostic significance of a 'low compliant bladder' in relation to incontinence surgery would also seem to be similar to detrusor instability. Colombo et al [264] have reported separately on the clinical and urodynamic effects of colposuspension in patients with low compliant bladders and detrusor instability (in addition to their GSI): the incidence of symptoms of urge incontinence was the same in both groups before and after surgery, and so was the cure rate for stress incontinence.

There are no consistent pre-operative methods to ascertain which patients will do well following surgery. Pre-operative factors such as age, parity, urinary symptoms, history of previous incontinence surgery and urodynamic variables have not been found to be predictive of outcome [35,398]. A variable that may be helpful to predict outcome is the temporal order of onset of symptoms of stress and urge incontinence. Scotti et al [400] found that a history of stress incontinence preceding the urge incontinence was predictive of a favourable outcome after colposuspension, as compared to patients that report urge incontinence as antecedent to stress incontinence (79% vs. 21% objective cure rate). The disadvantage of this variable is that it relies on patients' memory and ability to distinguish stress and urge symptoms.

Attempts have been made to quantify the severity of detrusor instability and to relate it to results of surgery. It has been suggested that low-pressure instability (detrusor contractions
of < 25 cmH2O) has a better prognosis than high-pressure instability (detrusor contractions > 25 cmH2O) [401,402]. Studies are inconclusive either because only few patients with high-pressure instability were included [401] or because outcome was not assessed objectively [402].

The observation of a poorly sustained detrusor contraction during voiding has been associated with detrusor instability and considered a better prognostic variable than contractions during filling with regards to the outcome of surgery [403]. This is however based on the observation of only five patients. More recent observations actually suggest that detrusor contractility during voiding is greater in women with mixed incontinence as compared to women with genuine stress incontinence [404].

Instability indices, which take into account the size of the pressure rise during unstable contractions and other variables (e.g. bladder volume at the first contraction), have been proposed as a better way to quantify detrusor instability and predict surgical outcome in women with mixed incontinence [405]. Unfortunately, larger studies have failed to confirm their predictive value [264].

A pathophysiological explanation of why some patients with detrusor instability respond to conventional incontinence surgery could be that genuine stress incontinence and detrusor instability may be manifestations of the same condition, at least in some patients.

Papa Petros and Ulmsten [406] have proposed a complex integral theory of female urinary incontinence, suggesting that both stress and urge symptoms derive from the same anatomical defect, a lax vagina. Loss of vaginal support would result in defective bladder neck closure mechanisms and in stimulation of hypothetical bladder base stretch receptors, thus causing stress and urge incontinence. This theory however cannot explain the occurrence of incontinence in women with no vaginal descent and the occurrence of surgical failures despite adequate support.
It has also been suggested that an open bladder neck would cause genuine stress incontinence, and as urine enters the proximal urethra, a reflex detrusor contraction may be elicited. This phenomenon can be clinically observed on cystometry, when urinary loss from stress is immediately followed by an unstable detrusor contraction with a new episode of incontinence [407]. In support of this theory there are experimental studies in animals showing that infusion of urine into the proximal urethra may induce a strong detrusor contraction [408,409]. The same phenomenon has been shown to occur also in humans in a study of normal female volunteers [185], but this finding has not been confirmed by observations on patients with urinary symptoms [410]. Unfortunately there are no specific observations in the literature on the infusion of fluid into the proximal urethra in women with mixed incontinence.

The correlation of reduced urethral closure pressure with the presence of detrusor instability in patients with mixed incontinence also suggests a possible link between urethral incompetence and the development of detrusor instability [411].

Present methods of investigation may not be sensitive enough to demonstrate the complex interplay between bladder neck and detrusor function. The observation of bladder neck funnelling has been proposed as a predictor of surgical outcome [412]. This is however a sign that has been shown to be present at rest in 21% of asymptomatic nulliparous young women [75]. More promising is the observation that good results after surgery have been obtained in patients with mixed incontinence when multichannel urethrocystometry has shown that bladder contraction is preceded by urethral relaxation [413]. Unfortunately, the test was not validated by observing the effects of surgery on patients whose bladder contractions were not preceded by urethral relaxation. The observation however suggests that bladder neck opening may be a separate, earlier event in the genesis of a detrusor contraction.
Bladder neck electrical conductivity (BNEC) is a technique that evaluates bladder neck opening via two gold plated electrodes placed at the bladder neck: if the bladder neck opens, the entrance of urine into the proximal urethra completes an electrical circuit (depending on the conductivity of the medium). This has been shown to correlate better than conventional cystometry with the symptom of urgency [414] thus suggesting that bladder neck opening causing urgency may not necessarily be followed by a detrusor contraction visible on cystometry [415]. BNEC may have a role to play as a predictor of surgical outcome in patients with mixed incontinence.

While the search for pre-operative values may contribute to the understanding of mixed incontinence, temporary bladder neck elevation would seem to be a logical and practical way to assess the potential effects of surgery on patients with mixed incontinence. Bladder neck support prosthesis have been used on patients with mixed incontinence with good symptomatic effect [416]. The challenge lies in devising a technique that has the capacity to reproduce surgery as closely as possible.
CONCLUSION

Detrusor instability occurring de novo after the colposuspension operation is a relatively frequent and unpredictable complication. Patients should be warned beforehand. Little research has been conducted on its natural history and impact on quality of life, but the prognosis appears uncertain and treatment may be unsatisfactory.

There is urgent need for research to investigate its causes so that preventative measures may be implemented.

The problem of the effects of surgery on patients with mixed genuine stress incontinence and detrusor instability is equally complex and under-researched. Worsening of pre-existent detrusor instability and onset of de novo detrusor instability may be due to the same factors.
CHAPTER 8

MATERIALS AND METHODS
The study population consisted of 77 women with genuine stress incontinence who underwent colposuspension. These women were prospectively investigated, selected for colposuspension and studied in relation to the development of post-operative voiding dysfunction and detrusor instability. The number of women studied was determined by the limited availability of the method used (MRI) and by time constraints.

All women approached agreed to participate in the study. They all gave informed consent (appendix 2) and were provided with an information leaflet (appendix 3). An information leaflet for General Practitioners (GP) was provided (appendix 4). The study obtained local Ethics Committee approval.

Methods, definitions and units conform to the standards recommended by the ICS [1], except where specifically noted.

There was no conflict of interest.

SETTING

The study was performed in Derriford Hospital, Plymouth. This is a busy District General Hospital, serving a population of half a million people. It has a Urogynaecology Unit, which sees more than 500 new referrals per year. Women are usually referred by GPs or other Consultants.

DIAGNOSTIC WORK-UP AND INCLUSION CRITERIA

In all patients presenting with urinary incontinence, the history was taken as detailed in appendix 5. Weight and height were measured. General, abdominal, vaginal and neurologic
examination (with testing of S234 segments) was performed as detailed in appendix 6. Bladder neck mobility was assessed clinically during vaginal (Sims) speculum examination. Vaginal examination included an assessment of prolapse using the ICS Pelvic Organ Prolapse Staging System, which quantifies the degree of prolapse of each vaginal compartment in centimetres, using the hymen as the fixed reference point [417]. The system aims to measure prolapse objectively and it has been shown to have good reproducibility [418]. The main points of this system are summarised in appendix 7.

Patients were asked to complete a frequency/volume chart for one week, also including fluid intake, number of leakage episodes and number of pads used. A mid-stream specimen of urine was obtained to exclude infection.

Initial conservative measures included referral to a Continence Advisor for pelvic floor exercises, behavioural therapy (bladder drill) for any irritative symptoms, fluid management and coping strategies (e.g. use of vaginal tampons, incontinence pads, etc). Interferential therapy was recommended to women unable to contract their pelvic floor.

Failure to improve with conservative measures (as reported by women) was an indication for urodynamic studies. These included uroflowmetry, distal urethral electrical conductance (if stress incontinence not demonstrable clinically), filling and voiding cystometry and urethral pressure profilometry (as detailed in chapter 4).

The equipment available consisted of an Albyn Medical / Griffon (with microtip pressure transducers), a Dantec Etude and then a Dantec Minuet (both with external pressure transducers / Novadome, connected to 4.5 French fluid filled bladder pressure catheters / Mediplus). The abdominal pressure was measured by inserting a fluid filled 8 French rectal catheter (Connecta) covered with a finger cot, which was then connected to external pressure transducers (as per ICS standards).

The bladder was filled with Saline at room temperature, using 8 French catheters (Vygon 08). Urethral catheterisation was performed after swabbing the meatus with diluted
Chlorexidine, and using Lignocaine Hydrochloride 2% (Instillagel) as a lubricant and local anaesthetic. The filling speed was 50-100 ml/min. The patient coughed at intervals (of every 100 ml filling volume) to demonstrate stress incontinence. Provocative manoeuvres were performed to demonstrate unstable detrusor contractions if none were observed during bladder filling. These included coughing, listening to the sound of running water, washing hands in cold water and changing position (e.g. standing). If necessary, patients were asked to cough (i.e. perform a 'stress test') while standing at capacity (after removing the filling catheter). The patient was then asked to void (in private).

As suggested by the ICS [1], a diagnosis of genuine stress incontinence was made if loss of urine was seen to occur during rises in intra-abdominal pressure, in the absence of a detrusor contraction. Overactive detrusor function was diagnosed if involuntary (phasic) detrusor contractions occurred during the filling phase, either spontaneously or after provocation. While the ICS includes asymptomatic contractions [1], only detrusor contractions accompanied by a sensation of urgency were considered diagnostic of detrusor instability. The size of the pressure rise was noted, but no lower limit was established for the diagnosis of detrusor instability (as none has been specified by the ICS).

Women with urodynamically proven genuine stress incontinence, which had an adverse effect on quality of life, and had failed to respond to conservative measures, were offered the operation of colposuspension, and were considered for inclusion in the study. They were fully counselled with regards to the development of post-operative complications, including voiding dysfunction, detrusor instability and prolapse.

The presence of intrinsic sphincter deficiency, defined by a maximum urethral closure pressure of less than 20 cmH2O [244], was not considered a contraindication to colposuspension, provided there was bladder neck mobility.

Concomitant detrusor instability was not a contraindication to surgery provided the condition and its symptoms were mild and responded well to bladder drill and
anticholinergic therapy. Any such patients were studied in relation to post-operative voiding dysfunction but excluded from the investigation of *de novo* detrusor instability.

**EXCLUSION CRITERIA**

1- Patients with neurological abnormalities likely to affect the bladder were excluded.

2- Patients requiring additional procedures, which may have an effect on bladder function, were excluded (e.g. women with prolapse of the mid-vaginal compartment likely to require vault-elevating procedures).

3- Patients with concomitant detrusor instability (in addition to GSI). They were excluded only from the investigation of *de novo* detrusor instability.

Mild to moderate rectocele was not considered an exclusion criteria, as only severe prolapse of the posterior vaginal compartment has the potential to affect bladder function [419]. While patients with rectoceles were included in the study, none underwent concomitant posterior repair, as there is controversy over the success rate of posterior colporrhaphy when performed at the same time of colposuspension [420].

Concomitant hysterectomy and past history of hysterectomy do not seem to influence voiding function after colposuspension (chapter 5, section 4). These were not considered an exclusion.

It is controversial whether previous bladder neck surgery predisposes to voiding difficulty after colposuspension (chapter 5, section 4). Therefore this was not considered an exclusion criteria but an additional factor worth investigating. In all cases (including those with retropubic surgery), the bladder neck was required to be mobile.
OPERATIVE TECHNIQUE AND POST-OPERATIVE CARE

The operation of colposuspension was performed as described by Burch, following the guidelines suggested by Stanton (chapter 5, section 1). The operations were performed by various surgeons including the author (LB), his supervisor (RMF) or doctors in training (under the supervision of LB or RMF). While gross consistency of technique was ensured by the presence of the author or RMF, subtle inter-operator variability was clearly possible. This is not in contrast with the aims of this thesis, as subtle intra- and inter-operator variations in technique may influence post-operative morbidity. Measurement of operative variables is discussed later (p. 179).

A low Pfannenstiel incision was made in all women (with the exception of one patient, who had a lower midline incision, as she required an incisional hernia repair). Following exposure of the cave of Retzius and dissection of the bladder base from the paravaginal fascia, two n.1 Ethibond sutures were placed on each side of the bladder neck, the first close to it and the second cephalad and lateral to the first. The paravaginal fascia was elevated as close as possible to the ilio-pectineal ligament (but avoiding excessive tension). The cave of Retzius was routinely drained with a Redivac suction drain.

Surgery was performed under general anaesthesia, but women were offered epidural anaesthesia for post-operative analgesia. This was inserted at the time of surgery and generally discontinued 24 hours later.

Antibiotic prophylaxis was given per-operatively to all women using Co-Amoxiclav (Augmentin) 1.2 g intravenously, or Cefotaxime (Claforam) 1 g intravenously and Metronidazole 1 g rectally to those allergic to Penicillin.

Thromboprophylaxis included all or some of the following: the use of pneumatic compression boots during surgery, TED stockings, Minipar 5000 units subcutaneously twice per day (started before surgery and stopped when patients were fully mobile).

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Patients on HRT were not routinely advised to stop it pre-operatively, but were covered with all the above measures.

In all patients the bladder was drained with a suprapubic Foley catheter (8 French gauge), which was kept on free drainage for 48 hours. The catheter was then clamped and patients attempted to void as instructed and following a standardised catheter regime (appendix 8). The catheter was removed when the residual urine volume after clamping was consistently less than 100 ml and the volume voided more than 200ml over at least 48 hours.

After surgery constipation was actively treated if patients had not defecated within three days. Initial treatment included Lactulose, Senna, and Glycerine suppositories. Enemas were reserved for resistant cases.

Patients were discharged home when fully mobile and free from complications. Inability to void did not prevent patients from going home, as long as they were able to cope with their catheter. In such circumstances a dedicated nurse was provided to visit the patient daily. They were also seen regularly in hospital until the catheter was removed. An ‘open door’ policy was adopted and patients were free to return for ‘check-ups’, advice and reassurance.

Post-operative voiding dysfunction was initially managed with suprapubic catheters. Voiding difficulty greater than one month was managed by clean intermittent self-catheterisation (CISC).

Post-operative detrusor instability and/or symptoms of urge incontinence were managed with advice on fluid intake (e.g. avoidance of caffeine, ‘fizzy’ drinks, and alcohol), bladder drill and anticholinergics (e.g. Oxybutinin). Anticholinergics were used only in patients who voided efficiently.

The data was recorded on specially designed forms and subsequently stored anonymously in a computer database (spreadsheets) following registration to the data protection act. Quality assurance was performed by checking the data prior to analysis.
Patients and methods in relation to the study of voiding dysfunction and *de novo* detrusor instability will be described separately.

**PATIENTS AND METHODS:**

1) **THE INVESTIGATION OF THE CAUSES OF VOIDING DYSFUNCTION**

A total of 77 women were prospectively studied in relation to the development of post-operative voiding difficulty.

Their details are summarised in table 16.

**Table 16. The study of voiding dysfunction. General characteristics of women recruited (n=77).**

<table>
<thead>
<tr>
<th></th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age</strong></td>
<td>mean 54y, median 55y, range 28-77y</td>
</tr>
<tr>
<td><strong>Parity</strong></td>
<td>mean 2.7, median 2, range 1-8</td>
</tr>
<tr>
<td><strong>Menopausal status</strong></td>
<td>pre-menopausal n=22 (28.5%)</td>
</tr>
<tr>
<td></td>
<td>post-menopausal n=55 (71.4%)</td>
</tr>
<tr>
<td></td>
<td>on HRT (topical or systemic) n= 42 (76.3% of post-menopausal)</td>
</tr>
<tr>
<td><strong>Past history of bladder neck surgery</strong></td>
<td>n=17 (22%): 14 anterior repairs, 1 two anterior repairs, 1 colposuspension, 1 anterior repair and colposuspension.</td>
</tr>
<tr>
<td><strong>Past history of hysterectomy</strong></td>
<td>n=38 (49.3%)</td>
</tr>
<tr>
<td><strong>Weight</strong></td>
<td>BMI: mean 26.4, range 18-35</td>
</tr>
<tr>
<td></td>
<td>Garrow scale: Ideal weight n=25 (32.4%)</td>
</tr>
<tr>
<td></td>
<td>Overweight n=26 (33.7%)</td>
</tr>
<tr>
<td></td>
<td>Obese n=26 (33.7%)</td>
</tr>
</tbody>
</table>

Neurological examination was normal in all patients, including three women with diabetes mellitus (one insulin-dependent), 11 women with chronic vertebro-skeletal problems (two of which had spinal surgery), and one woman with a history of a transient ischaemic attack.
**Prolapse**: Details are presented in table 17. In no patients did the leading edge of the cystocele or rectocele extend beyond the level of the hymen. No patients had significant descent of the mid vaginal compartment.

**Table 17.** The study of voiding dysfunction. Associated vaginal prolapse.

<table>
<thead>
<tr>
<th>Prolapse</th>
<th>n (%)</th>
<th>ICS Stage*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cystocele</td>
<td>39 (50.6%)</td>
<td>ICS Stage I: 28 (36%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>ICS Stage II: 11 (14%)</td>
</tr>
<tr>
<td>Rectocele</td>
<td>53 (68.8%)</td>
<td>ICS Stage I: 40 (52%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>ICS Stage II: 13 (17%)</td>
</tr>
</tbody>
</table>

*Stage 0: no prolapse.
Stage I: leading edge of prolapse is >1 cm above the level of the hymen.
Stage II: leading edge of prolapse is 1 cm or less proximal to or distal to the plane of the hymen.
Stage III: leading edge of prolapse is >1 cm below the plane of the hymen.
Stage IV: complete vaginal eversion.

**Additional procedures** were performed in nine women. These were abdominal hysterectomy (two), bilateral salpingo-oophorectomy (three), sterilisation (one), incisional hernia repair (one) and Fenton’s procedure (two).

At urodynamics, 57 women (74%) had genuine stress incontinence only, and 18 (23%) had mixed detrusor instability and genuine stress incontinence. Two women were considered to have 'potential' detrusor instability in addition to genuine stress incontinence, as urinary leakage occurred during cystometry which was associated with sensation of urgency but without an unstable bladder contraction.
The variables investigated were divided into pre-operative and operative / post-operative. Pre-operative variables, if present, might identify patients at risk of voiding difficulty. Operative and post-operative variables, if present, could potentially be modified to reduce the risk.

**PRE-OPERATIVE VARIABLES**

*Detrusor contractility* was assessed pre-operatively on voiding cystometry. Pre-operative urodynamic variables studied were: the peak flow rate (Q max), the detrusor pressure at maximum flow (p det,Q max), the maximum detrusor pressure (pdet, max), the urethral resistance (pdetQmax / Qmax^2), the presence of an after-contraction at the end of voiding (and its magnitude), the presence of abdominal straining during voiding, and the type of voiding pattern (see below).

Quantitative data was used for analysis of peak flow rate, detrusor pressure at maximum flow, maximum detrusor pressure, and urethral resistance. No attempt was made to classify patients into arbitrary categories for the purpose of statistical analysis. Care was taken to exclude the influence of the after-contraction when obtaining values for the maximum detrusor pressure during voiding.

The presence of an *after-contraction* was determined by a detrusor pressure rise after completion of voiding. The magnitude of the contraction was also noted.

*Abdominal straining during voiding* has not been defined by the ICS. In other studies an abdominal pressure rise of 10 cmH2O has been used [22,289,318]. In this study, abdominal straining during voiding was considered to be present only if an abdominal pressure rise of at least 10 cmH2O was present *throughout* the voiding process (either as a continuous pressure rise or as repeated peaks).
Women were divided in four groups according to their voiding pattern:

1. Women who voided with a detrusor contraction (greater than 15 cmH2O).
2. Women who voided with a detrusor contraction (greater than 15 cmH2O) and abdominal straining (as defined above).
3. Women who voided without a detrusor contraction and with abdominal straining.
4. Women who voided without a detrusor contraction and without abdominal straining (pelvic relaxation alone).

A lower limit of 15 cmH2O was chosen to define a detrusor contraction as studies on detrusor pressure during voiding in normal women suggest that most void with a detrusor contraction greater than 15 cm H2O (chapter 6, section 1).

**Anxiety and depression** were assessed using the Hospital Anxiety and Depression scale (HAD scale) [421] (appendix 9). This is a validated self-assessment scale used as a screening test for clinically significant anxiety and depression. For both anxiety and depression, scores of seven or less are normal, scores of eight to ten are considered borderline, and scores of eleven or more are clearly abnormal. Subscale scores can be used as measures of severity. Patients were asked to complete the form pre-operatively (before admission), in order to assess background psychological problems as a possible cause of voiding dysfunction. Analysis of data was performed using the subscale score rather than the group of allocation by score.

Other factors such as age, menopausal status (classified as pre-menopausal, post-menopausal without HRT and post-menopausal on HRT), weight (BMI), past history of hysterectomy, past history of bladder neck surgery, presence of concomitant detrusor instability, were also considered.
The effects of surgery on the bladder neck in terms of elevation and compression were studied using MRI. Intra-operative measures of elevation and compression were also assessed (these measures might be useful in prevention). Reproducibility studies were performed for all these variables (chapter 9, section 4). MRI measures of elevation and compression were correlated to intra-operative measures. Post-operative variables studied were detrusor contractility (on urodynamics), anxiety and depression (HAD scale) [421], pain (McGill’s questionnaire) [422], use of epidural anaesthesia, constipation and urinary infection.

a) Bladder neck elevation was assessed using two methods:

1) Using MRI before and after surgery (chapter 3).

Bladder neck position was determined using a system of co-ordinates as used on perineal ultrasound by Schaer et al [118] (figure 9). The pubic bone was used as a fixed reference point. The bladder neck was marked anteriorly, and when funnelling was present, this was done at the level of the posterior urethrovesical angle.
Fig. 9. MRI measures of bladder neck position.

BN = bladder neck
X = X axis
Y = Y axis

- BN – symphysis = distance between bladder neck and posterior surface of symphysis
- Dx = distance between bladder neck and Y axis (i.e. distance travelled along X axis)
- Dy = distance between bladder neck and X axis (i.e. distance travelled along Y axis)
Patients underwent MRI scanning the day before surgery and the position of the bladder neck was noted (figure 10).

Fig. 10. Bladder neck position before colposuspension on MRI.
This was then compared to the new bladder neck position on MRI performed six days after surgery (figure 11).

Fig. 11. Bladder neck position after colposuspension on MRI.
Comparison of the Dx measure before and after surgery determined the amount of bladder neck elevation achieved by surgery for each patient.

MRI measurements were made by the author, who was also aware of the patients post-operative course and voiding performance. While in theory an element of bias could have been introduced, this was thought to be unlikely, as the post-operative MRI was performed six days after surgery, the author was blinded to pre-operative measures and in most cases there was still uncertainty over the outcome. In addition, the accuracy and reproducibility of MRI (see chapter 9) reduced considerably the risk of obtaining unreliable measurements.

Patients underwent static MRI with a one Tesla superconductive magnet (Siemens Magnetron), with a body coil centred on the symphysis pubis. Sagittal planes were imaged in 3 mm thick sections with STIR and T1-weighted spin-echo sequences. For STIR images, the pulse sequences were 3500/30 (repetition time msec / echo time msec), the time of inversion was 130 msec, and the flip angle was 180 degrees. For T1-weighted images, the repetition time was 450 msec. The field of view was 450 mm for both sequences. A saturation band was positioned to reduce bowel motion artefact.

Patients were asked to have a comfortably full bladder. Bladder volume was not measured, as this variable has not been shown to affect bladder neck position (chapter 3, section A2). Thirteen STIR and 13 T1 frames were obtained before and after surgery thus providing a total of 52 frames for each patient. Measurements were performed on the frames that best demonstrated the bladder neck and urethra. In all women the quality of image was deemed sufficient for accurate measurements (as checked by Dr P Williams, Consultant Radiologist).
2) Bladder neck elevation was also assessed intra-operatively, by subjectively observing the amount of suture 'bow-stringing' (figure 12).

An attempt was made to quantify the extent of suture 'bow-stringing' by estimating it in millimetres to the nearest multiple of five (e.g. zero, five, ten, fifteen, etc.). 'Bow-stringing' was estimated on both sides, and a single measure was obtained by averaging the two values.

Fig. 12. Observation of suture 'bow-stringing' during surgery.
b) **Urethral compression** was assessed using three methods:

1) **Using MRI after surgery.**

The *distance between the bladder neck and the posterior surface of the symphysis pubis* was measured on the post-operative MRI images (figures 9 and 11). It is assumed that the longer the distance, the lower the chance of urethral compression.

2) **Intra-operatively, by measuring the distance between the medial paraurethral stitches with a ruler** (figure 13).

It is assumed that the wider the distance, the lower the chance of urethral compression. Measurements were obtained with a ruler by holding up the two medial sutures vertically. The distance between medial stitches at the *paravaginal site* and *pectineal site* were considered separately, as they do not always correspond.

---

**Fig. 13.** Measurement of the distance between the medial paraurethral stitches with a ruler.
3) Intra-operatively, by measuring the width of the urethropubic space after tying the sutures with paired ‘Hegar’ dilators (figure 14).

It is assumed that the greater the size of the ‘Hegar’ dilators, the lower the chance of urethral compression. This measure aimed to test objectively Tanagho’s assertion that voiding dysfunction is unlikely when by the end of the procedure it is possible to insert two fingers between bladder neck and pubic bone [31]. Initial attempts using only one Hegar were found to be poorly reproducible and these data were discarded.

Fig. 14. Measurement of the width of the urethropubic space (after tying the sutures) with paired ‘Hegar’ dilators.
c) Detrusor contractility was assessed post-operatively on day four by voiding cystometry. Post-operative day four was chosen because in a previous study in a similar population, half of patients had voided more than 50 ml by day four [27], thus allowing an adequate comparison between voiders and non voiders.

Filling cystometry was performed prior to voiding. The technique did not differ from that performed pre-operatively. The bladder was filled via an 8FG urethral catheter with Normal Saline at room temperature (rate 100ml/m) up to capacity. The filling line was routinely removed prior to voiding. The maximum detrusor pressure generated during a voiding attempt was recorded. Following a failed attempt, a second attempt at voiding was allowed with a smaller volume of fluid of 200-250 ml in the bladder (obtained by releasing the suprapubic catheter inserted at the time of surgery).

d) Anxiety and depression were assessed post-operatively in order to ascertain whether the stress of surgery and the hospital environment could influence voiding function. The HAD scale was again used [421] (appendix 9). Patients completed the questionnaire as a diary for four days starting on post-operative day two. The mean of the scores obtained was used for analysis, in order to minimise the effect of daily mood fluctuations. Data was not collected on the first post-operative day, as women had their bladders on free drainage and were not expected to void. After day five, anxiety and depression could have been a consequence rather than cause of voiding difficulty.

e) Post-operative pain was measured using the McGill’s pain questionnaire [422] (appendix 10). This provides quantitative measures of clinical pain that can be treated statistically and be used to compare pain intensity between patients. The ‘pain rating index’ (PRI) was used for analysis. This consists of the sum total of the values of the words chosen by patients to describe their pain. No pain has a value of zero, while maximum pain
has a value of 64. Patients were asked to complete the questionnaire as a diary for four days starting on post-operative days two. The mean of four days values was used for analysis.

f) The use of epidural anaesthesia for post-operative analgesia was assessed in relation to voiding function.

g) Post-operative bowel function, defined by the day when defecation first occurred, was studied in relation to voiding performance.

h) The presence of bacteriuria (quantitative culture greater than $10^5$ colony forming units per ml), on a urine specimen obtained from the catheter on post-operative day four, was studied in relation to voiding performance.
MEASURES OF OUTCOME (VOIDING PERFORMANCE)

All variables were correlated to:

a) First day of void (i.e. volume greater than 50 ml).

b) Day of catheter removal (i.e. residual urine volume of less than 100 ml over a 48 hour period, after voiding more than 200 ml).

Variables were correlated with both outcome measures using Pearson's correlation coefficient, t-test and stepwise multiple linear regression analysis.

Defining prolonged catheterisation is difficult and arbitrary and a definition of female voiding dysfunction has not yet been included in the standardisation of terminology of lower urinary tract function published by the International Continence Society [1]. This issue is also discussed in chapter 6. Previous studies have defined voiding dysfunction after colposuspension as 'prolonged catheterisation' longer than an arbitrary number of days (usually seven days) (appendix 1). Such analysis runs the risk of putting patients with no common pathophysiology in the same group (e.g. patients catheterised for eight days together with those unable to void for months). We have avoided this type of definition and used the day of catheter removal as the main end point for analysis.

The catheter was removed when residuals were consistently less than 100 ml, and this measure is often used in clinical practice and in investigative reports (chapter 6 and appendix 1). While this measure is also arbitrary, it has been adopted in this study to allow comparison with other studies. Also, for the purpose of this investigation, rather than the choice of what constitutes 'voiding dysfunction', what is important is the uniform application of the chosen measure so as to allow comparisons between patients.
A preliminary analysis on 42 patients suggested that overelevation (i.e. elevation > 26 mm) was an important cause of voiding dysfunction, with no patients with elevation of 26 mm or less requiring prolonged catheterisation [423] (appendix 11). As a consequence, additional investigations were performed aiming to:

1) Evaluate the relationship between amount of elevation and success or failure in the treatment of genuine stress incontinence.

Women were reviewed one year after surgery. The presence of stress incontinence was assessed subjectively, using the King’s Health Questionnaire [378] (see later). Patients were asked whether they had stress incontinence and how much they were affected by it (‘a little, moderately or a lot’). Objective testing was performed on urodynamics (as previously described). Patients were asked to cough at intervals during filling and also at capacity while standing (after removal of the filling line).

The relationship between outcome and elevation (as measured with MRI) was analysed using Student’s t-test and chi-squared test.

2) Investigate the reason(s) for differences in amount of bladder neck elevation between patients.

Major differences in surgical technique were considered unlikely (despite the number of operators involved), as the operations were performed under supervision and using an agreed standard technique. Elevation (as measured by MRI) was correlated with variables such as age, menopausal status, use of HRT, parity, presence of cystocele (measured
centimetres in relation to the hymen as per standard ICS method), previous hysterectomy, previous bladder neck surgery, and weight (BMI). Analysis was performed using Pearson’s correlation coefficient, Student’s t-test, analysis of variance and multiple regression analysis.
METHODS:

2) THE STUDY OF THE NATURAL HISTORY OF POST-OPERATIVE VOIDING DYSFUNCTION

All 77 recruited women were prospectively studied in relation to the development and natural history of post-operative voiding dysfunction. Women were reviewed three months and one year after colposuspension, and underwent clinical and urodynamic assessment.

Symptoms of voiding difficulty ('hesitancy', 'feeling of incomplete bladder emptying', 'need to adopt the standing position' and 'poor and/or interrupted stream') are known to be unreliable predictors of voiding dysfunction [270]. They were noted only if reported consistently and in combination (i.e. at least two of the above symptoms).

A symptom specific quality of life questionnaire (King's Health Questionnaire) [378] was given to patients before surgery, and post-operatively at three months and one year (appendix 12). This questionnaire provides information on the presence and severity of urinary symptoms such as frequency, nocturia, urgency, urge incontinence, stress incontinence, nocturnal enuresis, coital incontinence, frequent urinary tract infections and bladder pain. It also assesses the impact of these symptoms on quality of life. The questionnaire has been shown to be easy to use, valid (it measures what is intended) and reliable (it is consistent). The questionnaire was used in this study for the dual purpose of gathering information on symptoms and to assess the impact on quality of life caused by the development of voiding dysfunction. During the consultation the information recorded was individually checked.

The version number seven of the King's Health Questionnaire does not specifically ask about voiding function. As a consequence, specific enquiries were made at interview in
relation to symptoms of voiding dysfunction (as described above), and the amount of
'bother' caused by them (a little, moderately, a lot).

For the assessment of quality of life after surgery, each woman with post-operative
voiding dysfunction acted as her own control. The impact of her bladder symptoms on
quality of life at three months and one year was compared to that before surgery.

Voiding cystometry was performed using the technique already described. Urodynamic
variables such as volume voided, peak flow rate, detrusor pressure at maximum flow,
maximum detrusor pressure, urethral resistance, presence of abdominal straining, voiding
pattern and urine residual, were noted. When a residual of more than 100 ml was found,
women underwent repeat uroflow studies with measurement of residual volume using
ultrasound (Bard Portable Ultrasound BVI 2500).

Women were considered to have objective voiding dysfunction when found to have
residuals greater than 100 ml on more than one occasion. This objective finding was then
correlated to symptoms and quality of life outcome measures.

The presence of subnormal peak flow rates (as defined by values below the 5th centile on
the Liverpool nomograms [179], and also as a PFR of less than 15 ml/s) was noted.

The presence of urodynamic ‘obstruction’, as defined by Massey and Abrams [362], was
also noted. Using their criteria, women were considered to have ‘obstruction’ when two or
more of the following parameters were present: a peak flow rate of less than 12 ml/s, a
detrusor pressure at maximum flow greater than 50 cmH2O, a urethral resistance greater
than 0.2, and a ‘significant’ residual urine (greater than 100 ml for the purpose of this
study) (chapter 6, section 7). Lower urinary tract ‘obstruction’ in the female has not been
defined by the ICS and there are no universally accepted criteria (chapter 6, section 7). The
above definition is commonly used and was chosen to allow comparisons with other
studies.
METHODS:

3) THE INVESTIGATION OF THE CAUSES OF ‘DE NOVO’ DETRUSOR INSTABILITY

A total of 56 women with genuine stress incontinence and stable bladders were prospectively studied in relation to the development of post-operative de novo detrusor instability (72% of recruited women). Of the original cohort of 77 women, 21 women were excluded: 18 (23%) because they had mixed detrusor instability and genuine stress incontinence, two because they were considered to have ‘potential’ detrusor instability (urinary leakage occurred during cystometry which was not associated with either stress or an unstable bladder contraction), and one because she declined to have post-operative urodynamics.

The details of the women studied in relation to de novo detrusor instability are summarised in table 18.

Table 18. The study of de novo detrusor instability. General characteristics of women recruited (n=56).

<table>
<thead>
<tr>
<th>Age</th>
<th>mean 52.7y, median 54y, range 28-76y</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parity</td>
<td>mean 2.5, median 2, range 1-4</td>
</tr>
<tr>
<td>Menopausal status</td>
<td>pre-menopausal n=17 (30.3%)</td>
</tr>
<tr>
<td></td>
<td>post-menopausal n=39 (69.6%)</td>
</tr>
<tr>
<td></td>
<td>on HRT (topical or systemic) n=29 (74.3% of post-menopausal)</td>
</tr>
<tr>
<td>Past history of</td>
<td>n=11 (19.6%): 10 anterior repairs, 1 anterior repair and colposuspension.</td>
</tr>
<tr>
<td>bladder neck</td>
<td></td>
</tr>
<tr>
<td>surgery</td>
<td></td>
</tr>
<tr>
<td>Past history of</td>
<td>n=25 (48%)</td>
</tr>
<tr>
<td>hysterectomy</td>
<td></td>
</tr>
<tr>
<td>Weight</td>
<td>BMI: mean 26, range 18-34</td>
</tr>
<tr>
<td></td>
<td>Garrow scale: ideal weight n=20 (35.7%)</td>
</tr>
<tr>
<td></td>
<td>overweight n=20 (35.7%)</td>
</tr>
<tr>
<td></td>
<td>obese n=16 (28.5%)</td>
</tr>
</tbody>
</table>
Pre-operative symptoms of urge incontinence are detailed in table 19.

**Table 19.** The study of *de novo* detrusor instability. Pre-operative symptoms of urge incontinence in 56 women with genuine stress incontinence and stable bladders.

<table>
<thead>
<tr>
<th>Urge incontinence</th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No symptoms</td>
<td>19 (33.9%)</td>
</tr>
<tr>
<td>Symptoms</td>
<td>37 (66%)</td>
</tr>
<tr>
<td>Mild</td>
<td>14 (25%)</td>
</tr>
<tr>
<td>Moderate</td>
<td>13 (23.2%)</td>
</tr>
<tr>
<td>Severe</td>
<td>10 (17.8%)</td>
</tr>
</tbody>
</table>

Neurological examination was normal in all patients, including one woman with diabetes mellitus, and nine women with chronic vertebro-skeletal problems (one of which had spinal surgery).

**Prolapse:** Details are presented in table 20. In no patients the leading edge of the cystocele or rectocele extended beyond the level of the hymen. No patients had significant descent of the mid vaginal compartment.

**Table 20.** The study of *de novo* detrusor instability. Associated vaginal prolapse.

<table>
<thead>
<tr>
<th>Prolapse</th>
<th>n (%)</th>
<th>ICS Stage*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cystocele</td>
<td>26 (46.4%)</td>
<td>ICS Stage I: 18 (32.1%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>ICS Stage II: 8 (14.2%)</td>
</tr>
<tr>
<td>Rectocele</td>
<td>36 (64.2%)</td>
<td>ICS Stage I: 29 (51.7%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>ICS Stage II: 7 (12.5%)</td>
</tr>
</tbody>
</table>

*Stage 0: no prolapse.
Stage I: leading edge of prolapse is > 1cm above the level of the hymen.
Stage II: leading edge of prolapse is 1 cm or less proximal to or distal to the plane of the hymen.
Stage III: leading edge of prolapse is > 1 cm below the plane of the hymen.
Stage IV: complete vaginal eversion.
Additional procedures were performed in five women. These were abdominal hysterectomy (one), bilateral salpingo-oophorectomy (two), sterilisation (one), and Fenton’s procedure (one).

The variables investigated were divided into pre-operative and operative / post-operative. Pre-operative variables, if present, might identify patients at risk of voiding difficulty. Operative and post-operative variables, if present, could potentially be modified to reduce the risk.

**PRE-OPERATIVE VARIABLES**

The presence of **urge incontinence** before surgery (despite a stable bladder on cystometry) was considered in relation to the development of de novo detrusor instability. Symptoms were detailed by patients in the King’s Health Questionnaire [378]. Patients were asked to state whether they had urge incontinence and how much they were affected by it (‘a little, moderately or a lot’).

Pre-operative **urodynamic variables** studied were: peak flow rate, detrusor pressure at maximum flow, maximum detrusor pressure, urethral resistance, the presence of an after-contraction at the end of voiding (and its magnitude), the presence of abdominal straining during voiding, and the type of voiding pattern. Definitions and methods were the same used for the investigation of voiding dysfunction (chapter 8, section 1).

**Anxiety and depression** were assessed using the HAD scale [421], as detailed before (chapter 8, section 1).
Other factors such as age, menopausal status (classified as pre-menopausal, post-menopausal and post-menopausal on HRT), weight (BMI), past history of hysterectomy, past history of bladder neck surgery, were also considered.

OPERATIVE AND POST-OPERATIVE VARIABLES

All measures of bladder neck elevation and urethral compression, as detailed in relation to voiding dysfunction (chapter 8, section 1), were correlated to the development of de novo detrusor instability.

Pain was assessed post-operatively using the McGill questionnaire [422] as detailed before (chapter 8, section 1). Pain was considered in relation to the development of de novo detrusor instability in case it might be a marker of nerve damage.

Voiding performance in the post-operative period, as determined by the first day of voiding (greater than 50 ml) and by the day of catheter removal (residual urine less than 100 ml), was considered in relation to the development of de novo detrusor instability.

Urodynamics were performed three months after colposuspension to establish whether the development of de novo detrusor instability could be correlated to any evidence of obstruction caused by surgery. Three strategies were followed:

1) A definition of obstruction in the female was used as detailed before (Massey and Abrams) [362] (chapter 8, section 2). The incidence of de novo detrusor instability in 'obstructed' women was compared to that in 'non-obstructed' women.
2) Post-operative urodynamic variables of women with *de novo* detrusor instability were compared to those of women who did not develop this complication. Variables studied were peak flow rate, detrusor pressure at maximum flow, maximum detrusor pressure, urethral resistance, straining during voiding, and voiding pattern.

3) Post-operative urodynamic variables were compared to the pre-operative variables in all women, in order to ascertain whether women with *de novo* detrusor instability had incurred relative changes suggestive of obstruction as compared to women without this complication. Variables studied were peak flow rate, detrusor pressure at maximum flow, maximum detrusor pressure, urethral resistance, and presence or absence of abdominal straining (as defined in chapter 8, section 1).

MEASURES OF OUTCOME

Objective and subjective outcome measures were considered:

**Objective:** The development of *de novo* detrusor instability as detected by urodynamics three months after colposuspension was the main outcome measure. Evaluation was made at three months so as to establish a clear causal link with surgery while avoiding confounding factors operating in the immediate post-operative period (e.g. UTI, use of catheters, etc.).

Urodynamics were performed using the technique already described.

**Subjective:** The presence of the symptom of urge incontinence after colposuspension was taken as a sign of ‘potential detrusor instability’ and recorded. Symptoms were detailed by patients in the King’s Health Questionnaire [378]. Patients were asked to state
whether they had urge incontinence and how much it affected their quality of life ('a little, moderately or a lot'). 'Urge incontinence' was chosen for analysis (instead of other 'irritative' symptoms), because it is more consistently associated with detrusor instability [176] and arguably is the most distressing symptom of detrusor instability.

Statistics were performed using Analysis of Covariance (Ancova), Mann-Whitney test, Chi-squared tests, and Student's t-test.
METHODS:

4) THE STUDY OF THE NATURAL HISTORY OF 'DE NOVO' DETRUSOR INSTABILITY

All 56 women with genuine stress incontinence and stable bladders were prospectively studied in relation to the development and natural history of post-operative de novo detrusor instability.

The condition was diagnosed objectively by filling cystometry. The technique used and the criteria for diagnosis have already been described. The natural history of the symptom of urge incontinence in patients found to have stable bladders was also studied.

Follow-up was at three and twelve months after colposuspension.

A symptom specific quality of life questionnaire (King’s Health Questionnaire) [378] was given to patients before surgery, and post-operatively at three months and one year, as mentioned before (chapter 8, section 2). The questionnaire was used for the dual purpose of gathering information on symptoms and to assess the impact on quality of life caused by the development of de novo detrusor instability.

For the assessment of quality of life after surgery, each woman with de novo detrusor instability acted as her own control. The impact of her bladder symptoms on quality of life at three months and one year was compared to that before surgery.
CHAPTER 9

RESULTS:

THE CAUSES AND NATURAL HISTORY OF VOIDING DYSFUNCTION
I- PRE-OPERATIVE VARIABLES

Data from all 77 recruited women are included for analysis.

Details on age, menopausal status, weight (BMI), presence of a past history of hysterectomy and bladder neck surgery, presence of concomitant detrusor instability, are presented in chapter 8, section 1.

All 77 women had pre-operative urodynamics. Urodynamic variables are presented in table 21.

Table 21. The study of voiding dysfunction. Pre-operative urodynamic variables.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean (range)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Qmax</td>
<td>28.9 ml/s (7-56)</td>
<td>&lt; 12 ml/s: n=1 &lt; 15 ml/s: n=5</td>
</tr>
<tr>
<td>pdet,Qmax*</td>
<td>21.3 cmH2O (0-44)</td>
<td></td>
</tr>
<tr>
<td>pdet,max*</td>
<td>28.3 cmH2O (0-69)</td>
<td>&lt; 15 cmH2O: n=14 Acontractile (pdet = 0): n=1</td>
</tr>
<tr>
<td>Resistance*</td>
<td>0.034 (0.009-0.18)</td>
<td></td>
</tr>
<tr>
<td>MUCP†</td>
<td>40.5 cmH2O (7-92)</td>
<td>&lt; 20 cmH2O: n=8 (10%)</td>
</tr>
<tr>
<td>After contraction‡</td>
<td>Present: n=42 (54.5%) Mean 75 cmH2O (range: 24-230 cmH2O)</td>
<td></td>
</tr>
<tr>
<td>Abdominal straining</td>
<td>Present: n=32 (41.5%) Absent: n=45 (58.4%)</td>
<td></td>
</tr>
<tr>
<td>Voiding pattern§</td>
<td>1) 39 (50.6%) 2) 24 (31.1%) 3) 8 (10.3%) 4) 6 (7.7%)</td>
<td></td>
</tr>
</tbody>
</table>

* In 3 women values could not be obtained (pressure lines voided).
† In 2 women UPP was not performed.
‡ In 10 women variable could not be recorded (pressure line voided).
§ 1) detrusor contraction alone, 2) detrusor contraction and abdominal straining,
3) pelvic relaxation with additional abdominal straining, 4) pelvic relaxation alone.
The HAD questionnaire was completed pre-operatively by 76 women (98.7%). Details are presented in table 22.

Table 22. The study of voiding dysfunction. Pre-operative anxiety and depression.

<table>
<thead>
<tr>
<th></th>
<th>Depression n (%)</th>
<th>Anxiety n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non cases</td>
<td>59 (77.6%)</td>
<td>36 (47.3%)</td>
</tr>
<tr>
<td>Borderline</td>
<td>13 (17.1%)</td>
<td>19 (25%)</td>
</tr>
<tr>
<td>Cases</td>
<td>4 (5.2%)</td>
<td>21 (27.6%)</td>
</tr>
</tbody>
</table>

2- OPERATIVE AND POST-OPERATIVE VARIABLES

Measures of elevation:

Objective mean bladder neck elevation achieved by colposuspension, as measured by MRI, was 28.7 mm (range: 10-48 mm). Two women declined MRI due to claustrophobia and two could not have the test due to equipment failure.

Intra-operative ‘bow-stringing’ was seen in 25 cases (32.4%). This was considered of 10 mm or less in 17 cases and of more than 10 mm in eight cases.

Measures of urethral compression:

The mean distance between the bladder neck and the posterior surface of the pubic bone, as measured by MRI, was 5.7mm (range: 2-13).

The distance between the medial stitches as measured during surgery with a ruler was a mean of 42.2 mm at the paravaginal insertion (range: 22-65), and a mean of 50.5 mm at the ilio-pectineal insertion (range: 27-77).

The mean size of paired Hegars that could be inserted between the urethra and the pubic bone after tying the sutures was 11.7 (range 8-17). This variable was obtained for 61
patients only (79.2%), as initial attempts using only one Hegar were found to be poorly reproducible and data from these 16 women were discarded.

**Detrusor contractility:**

Urodynamic assessment of detrusor contractility on day four after colposuspension was performed on 73 patients (94.8%). Four patients declined the test. The urodynamic findings are summarised in table 23.

**Table 23.** Urodynamic findings on day four after colposuspension.

<table>
<thead>
<tr>
<th>Cystometric capacity</th>
<th>374 ml (218-771 ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Volume voided:</strong></td>
<td></td>
</tr>
<tr>
<td>&gt; 50 ml</td>
<td>n=31 (42.4%)</td>
</tr>
<tr>
<td>&gt; 200 ml</td>
<td>n=18 (24.6%)</td>
</tr>
<tr>
<td><strong>Residual &lt; 100 ml</strong></td>
<td>n=14 (19.1%)</td>
</tr>
<tr>
<td><strong>Maximum detrusor pressure</strong>*:</td>
<td></td>
</tr>
<tr>
<td>mean (range)</td>
<td>38.1 cmH2O (0-100 cmH2O)</td>
</tr>
<tr>
<td>0-14 cmH2O</td>
<td>n=9 (6 acontractile) (12.3%)</td>
</tr>
<tr>
<td>15-35 cmH2O</td>
<td>n=17 (23.2%)</td>
</tr>
<tr>
<td>&gt; 35 cmH2O</td>
<td>n=47 (includes outlier - see later) (64.3%)</td>
</tr>
</tbody>
</table>

* Detrusor pressure values were obtained irrespective of whether patients were or were not able to void.

Nine of 73 women had a detrusor pressure contraction on day four which was less than 15 cmH2O. These women were considered to have temporary loss of detrusor contractility. Details of their pre-operative detrusor pressure values and post-operative voiding performance are illustrated in table 24. The statistical association between detrusor pressure on day four and voiding performance is shown in section 7.
Table 24. Pre-operative detrusor pressure and post-operative voiding performance of patients found to have low detrusor pressure values of < 15 cmH2O in the immediate post-operative period.

<table>
<thead>
<tr>
<th>pdet,max (day 4)</th>
<th>pdet,max (before surgery)</th>
<th>Volume voided before test</th>
<th>Volume voided during cystometry</th>
<th>Day of void &gt; 50 ml</th>
<th>Day of catheter removal</th>
</tr>
</thead>
<tbody>
<tr>
<td>12</td>
<td>44</td>
<td>0</td>
<td>0</td>
<td>8</td>
<td>16</td>
</tr>
<tr>
<td>0</td>
<td>28</td>
<td>0</td>
<td>0</td>
<td>7</td>
<td>11</td>
</tr>
<tr>
<td>8</td>
<td>24</td>
<td>200</td>
<td>0</td>
<td>3</td>
<td>14</td>
</tr>
<tr>
<td>0</td>
<td>28</td>
<td>0</td>
<td>0</td>
<td>8</td>
<td>15</td>
</tr>
<tr>
<td>0</td>
<td>25</td>
<td>100</td>
<td>0</td>
<td>2</td>
<td>13</td>
</tr>
<tr>
<td>0</td>
<td>15</td>
<td>0</td>
<td>0</td>
<td>4</td>
<td>8</td>
</tr>
<tr>
<td>0</td>
<td>25</td>
<td>0</td>
<td>0</td>
<td>5</td>
<td>13</td>
</tr>
<tr>
<td>0</td>
<td>25</td>
<td>0</td>
<td>0</td>
<td>7</td>
<td>19</td>
</tr>
<tr>
<td>12</td>
<td>55</td>
<td>0</td>
<td>0</td>
<td>6</td>
<td>12</td>
</tr>
</tbody>
</table>

**Psychological factors:**

The HAD questionnaire was completed post-operatively on days 2-5 by 74 women (96.1%). The mean values of days 2-5 are illustrated in table 25.

Table 25. The study of voiding dysfunction. Post-operative anxiety and depression (mean of days 2-5).

<table>
<thead>
<tr>
<th></th>
<th>Depression n (%)</th>
<th>Anxiety n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non cases</td>
<td>48 (62.3%)</td>
<td>35 (45.4%)</td>
</tr>
<tr>
<td>Borderline</td>
<td>15 (19.4%)</td>
<td>19 (24.6%)</td>
</tr>
<tr>
<td>Cases</td>
<td>11 (14.2%)</td>
<td>20 (25.9%)</td>
</tr>
</tbody>
</table>

**Other factors:**

Fifty six women had **epidural anaesthesia** (72.7%), while 21 declined it (27.2%).

The McGill **pain** questionnaire was completed by 75 women (97.4%). The mean value for days 2-5 was 12.7 (range: 0-64).

The mean day of resumption of **bowel function** (defaecation) was post-operative day 3 (range: 1-7).

**Bacteriuria** was detected in 10 women in post-operative day 4 (12.9%).
3- OUTCOME MEASURES (VOIDING PERFORMANCE)

a) The mean day of void of more than 50 ml was 5.8 days. The range was 2 to 42 days. After exclusion of one outlier with extreme values (42 days), the mean was 5.38 days (range 2-27 days), and the median was 3 days (S.D. 4.79).

b) The mean day of catheter removal was 14.9 days. The range was 4 to 190 days. After exclusion of the same outlier (190 days before stopping CISC), the mean was 12.66 days (range 4-47 days), and the median was 11 days (S.D. 8.32).

Voiding performance after colposuspension is detailed in table 26.

Table 26. Voiding performance of 77 women after colposuspension.

<table>
<thead>
<tr>
<th>Voiding &gt; 50 ml</th>
<th>Days of catheterisation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>5.8 days</td>
</tr>
<tr>
<td>Range</td>
<td>2-42 days</td>
</tr>
<tr>
<td></td>
<td>14.9</td>
</tr>
<tr>
<td></td>
<td>4-190</td>
</tr>
</tbody>
</table>
4- REPRODUCIBILITY STUDIES

All reproducibility studies were performed using Pearson's correlation coefficient.

The reproducibility of measurements of elevation obtained with MRI was determined on 20 patients. Measurements were performed separately and blindly by two observers (L.B. the author and E.W. a Senior Radiographer) and repeated a second time by one of the observers (L.B.) three months later. Measures of elevation were obtained by observing and independently choosing from 52 different images for each patient.

The intra-observer and inter-observer correlations were very high and positive (0.92 and 0.90 respectively). Mean values are shown in table 27.

The reproducibility of measurements of bladder neck approximation to the pubic bone obtained with MRI was determined on 20 patients using the same method described above. The intra-observer and inter-observer correlations were also high (0.91 and 0.87 respectively). Mean values are shown in table 27.

Table 27. Reproducibility studies of MRI measures. Mean values of bladder neck elevation and approximation to the back of the pubic bone obtained by two different observers (1 and 2) and by the same observer on two separate occasions (a and b).

<table>
<thead>
<tr>
<th></th>
<th>Bladder neck elevation</th>
<th>Bladder neck approximation to pubic bone</th>
</tr>
</thead>
<tbody>
<tr>
<td>Observer 1 (mean mm)</td>
<td>29.3</td>
<td>7.2</td>
</tr>
<tr>
<td>Observer 2 a (mean mm)</td>
<td>29.1</td>
<td>6.6</td>
</tr>
<tr>
<td>Observer 2 b (mean mm)</td>
<td>29.5</td>
<td>5.7</td>
</tr>
<tr>
<td>Correlation 1 – 2</td>
<td>0.90 (correlation 1 vs 2a)</td>
<td>0.87 (correlation 1 vs mean of 2)</td>
</tr>
<tr>
<td>Correlation 2a – 2b</td>
<td>0.92</td>
<td>0.91</td>
</tr>
</tbody>
</table>
The reproducibility of measurements of urethral compression using Hegar dilators was determined on 14 patients. Two surgeons (L.B. and R.M.F.) performed the measurements separately and blindly. There was good agreement between observers (0.93). Mean values are shown in table 28.

The reproducibility of measurements of urethral compression using the distance between the medial stitches measured with a ruler (at the paravaginal and pectineal sites) was determined on 20 patients. Two surgeons (L.B. and R.M.F.) performed the measurements separately and blindly. The reproducibility was very good for both distances measured. The correlation between the two observers was 0.99 for both distances measured. Mean values are shown in table 28.

Table 28. Reproducibility studies of intra-operative measures. Urethral compression is assessed by two observers (1 and 2) using Hegar dilators and measuring (with a ruler) the distance between the medial stitches at the paravaginal and pectineal sites.

<table>
<thead>
<tr>
<th>Hegars</th>
<th>Distance between medial stitches (paravaginal site)</th>
<th>Distance between medial stitches (pectineal site)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Observer 1</td>
<td>13.2</td>
<td>39.7 mm</td>
</tr>
<tr>
<td>Observer 2</td>
<td>13.7</td>
<td>40 mm</td>
</tr>
<tr>
<td>Correlation 1 vs 2</td>
<td>0.93</td>
<td>0.99</td>
</tr>
</tbody>
</table>
Objective mean bladder neck elevation achieved by colposuspension, as measured by MRI, was 28.7 mm (range: 10-48 mm).

Intra-operative ‘bow-stringing’ was seen in 25 cases (32.4%). This was considered of 10 mm or less in 17 cases and of more than 10 mm in eight cases (5 mm = 3, 10 mm = 14, 15 mm = 1, 20 mm = 5, 25 mm = 1, 30 mm = 1).

When using Pearson’s correlation coefficient, no correlation was found between objective (MRI) and subjective (suture ‘bow-stringing’) measures of elevation (p > 0.05). Given that the observation of ‘bow-stringing’ is a measure of elevation that does not take into account the position of the bladder neck before elevation but only its final point of placement, the amount of suture ‘bow-stringing’ was also correlated to the final position (Dx) as measured with MRI. But also this correlation was not found to be significant.

Mean values of measures of urethral compression are shown in section 2.

Using Pearson’s correlation coefficient MRI measures of urethral compression were not found to be correlated to intra-operative measures using Hegars and the distance between the medial stitches (at paravaginal and pectineal level). However, there was evidence of a weak but significant (p < 0.05) positive correlation between Hegars and both distances between medial stitches (correlations of 0.35 and 0.38 for distance between pectineal stitches and for distance between paravaginal stitches respectively). This suggests that a more lateral placement of sutures should result in more space for larger Hegars between the urethra and the pubic bone.
7- FACTORS ASSOCIATED WITH VOIDING DYSFUNCTION

Data from 76 patients was used for statistical analysis. One outlier with extreme values (as detailed above) was excluded.

The *quantitative variables* which were significantly correlated \((p < 0.05)\) to the **day of void of more than 50 ml**, using *Pearson’s correlation coefficient*, are shown in table 29.

**Table 29.** Quantitative variables correlated to the day of void of more than 50 ml.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elevation using MRI</td>
<td>0.40</td>
</tr>
<tr>
<td>Urethral compression as shown by a decreasing Hegar size</td>
<td>-0.32</td>
</tr>
<tr>
<td>Age</td>
<td>0.31</td>
</tr>
<tr>
<td>Pre-operative detrusor contractility:</td>
<td></td>
</tr>
<tr>
<td>- increasing urethral resistance</td>
<td>0.27</td>
</tr>
<tr>
<td>- decreasing strength or absence of an after-contraction</td>
<td>-0.24</td>
</tr>
</tbody>
</table>

The *quantitative variables* which were significantly correlated \((p < 0.05)\) to the **day of catheter removal** are shown in table 30.

**Table 30.** Quantitative variables correlated to the day of catheter removal.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>0.45</td>
</tr>
<tr>
<td>Elevation using MRI</td>
<td>0.38</td>
</tr>
<tr>
<td>Urethral compression:</td>
<td></td>
</tr>
<tr>
<td>- decreasing distance between bladder neck and pubic bone</td>
<td>-0.29</td>
</tr>
<tr>
<td>- decreasing Hegar size</td>
<td>-0.27</td>
</tr>
<tr>
<td>Pre-operative detrusor contractility:</td>
<td></td>
</tr>
<tr>
<td>- decreasing strength or absence of an after-contraction</td>
<td>-0.32</td>
</tr>
<tr>
<td>Post-operative detrusor contractility:</td>
<td></td>
</tr>
<tr>
<td>- decreasing detrusor pressure on day 4</td>
<td>-0.24</td>
</tr>
</tbody>
</table>
The strong correlation between the most important outcome measure ('day of catheter removal') and age is illustrated in figure 15.

Fig. 15. Plot of 'day of catheter removal' against age.
Preliminary results based on 42 women (38 MRI measures of elevation) had suggested that there is a maximum level of elevation below which voiding performance is likely to be good and above which it might be poor [423] (appendix 11).

This finding persisted at completion of the study. Plots of both outcome measures, ‘day of void > 50 ml’ and ‘day of catheter removal’, against elevation, suggest a ‘threshold’ of around 26 mm, below which prolonged voiding dysfunction does not occur (figures 15 and 16).

Fig. 16. Plot of ‘day of voiding > 50 ml’ against elevation.
No women with bladder neck elevation < 26 mm (to the left of demarcating line) needed more than five days to commence voiding (> 50 ml).
No women with bladder neck elevation < 26 mm (to the left of demarcating line) needed a catheter for more than two weeks.

Accordingly, patients were divided into two groups, those with an elevation of 26 mm or less (group 1) and those with an elevation of more than 26 mm (group 2).

The voiding performance of the two groups was compared using a t-test (allowing for unequal variances). Very strong evidence of a poorer mean voiding performance for group 2 (elevation > 26 mm) was found for both measures of outcome (table 31).

The outlier with a very poor voiding performance excluded from analysis had a bladder neck elevation of 36 mm.

The amount of bladder neck elevation before and after interim studies did not differ (mean of 28.2 in initial 38 women vs mean of 29.3 in subsequent 35 women).
A stepwise multiple linear regression analysis was used to investigate whether voiding dysfunction can be predicted from pre-operative variables and to investigate the relationship between voiding dysfunction and operative variables.

Pre-operative variables and operative variables were analysed separately. Regression models with 'days to voiding more than 50 ml' and 'days to catheter removal' as the dependent variables produced models where the residuals were clearly non-normal. This problem was overcome by using taking logarithms of each of the above measures and using these as the dependent variables in the regression. Taking logs also tended to give better fitting models. When analysing operative variables it was also found that using 'elevation group' (as defined above) rather than the actual elevation produced better models.

The variables found to be associated with post-operative voiding performance are summarised in tables 32 and 33.

Table 32. Pre-operative variables predictive of voiding dysfunction after colposuspension (p-value in brackets).

<table>
<thead>
<tr>
<th>Days to voiding 50 ml</th>
<th>Days to catheter removal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increasing urethral resistance (p=0.001)</td>
<td>Increasing age (p&lt;0.001)</td>
</tr>
<tr>
<td>Increasing age (p=0.003)</td>
<td>Decreasing peak flow rate (p=0.004)</td>
</tr>
<tr>
<td>Straining during voiding (p=0.045)</td>
<td>Straining during voiding (p=0.005)</td>
</tr>
</tbody>
</table>
Table 33. Operative variables associated with voiding dysfunction after colposuspension (p-value in brackets).

<table>
<thead>
<tr>
<th>Days to void 50 ml</th>
<th>Days to catheter removal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elevation (using MRI) &gt; 26 mm (p&lt;0.001)</td>
<td>Elevation (using MRI) &gt; 26 mm (p=0.009)</td>
</tr>
<tr>
<td>Urethral compression (decreasing Hegars size) (p=0.021)</td>
<td>Urethral compression (decreasing distance bladder neck-pubis) (p=0.007)</td>
</tr>
<tr>
<td></td>
<td>Urethral compression (decreasing Hegars size) (p=0.022)</td>
</tr>
</tbody>
</table>

After regression analysis, pre-operative variables found to be not predictive of voiding dysfunction were detrusor pressure at maximum flow, maximum detrusor pressure, presence or absence of an after-contraction, presence or absence of coexistent detrusor instability, voiding pattern, history of hysterectomy, history of bladder neck surgery, weight (BMI), menopausal status, and psychological factors (anxiety and depression).

Operative and post-operative variables found to be not associated with voiding dysfunction were elevation as measured by observation of suture ‘bow-stringing’, urethral compression as measured by the distance between the medial stitches (paravaginal and pectineal), post-operative psychological factors (anxiety and depression), post-operative pain, post-operative detrusor contractility (maximum detrusor pressure on day four), constipation (first day of defecation), bacteriuria (on day four), and use of epidural for post-operative analgesia.

Considering that voiding performance had shown greater variability in the group of patients elevated by more than 26 mm, a stepwise multiple linear regression analysis was used to investigate whether voiding dysfunction could be predicted from pre-operative variables in this group at risk of voiding dysfunction. The most important outcome...
measure, 'days to catheter removal', was regressed against all possible pre-operative factors.

Variables found to be predictive of voiding performance in this group of patients are shown in table 34. These factors combined explain up to 43.3% of the variability in voiding performance for bladder neck elevation greater than 26 mm ($R^2 = 43.3\%$).

**Table 34.** Pre-operative variables associated with voiding performance (day of catheter removal) in the group of patients elevated by more than 26 mm.

<table>
<thead>
<tr>
<th>Variable</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>($p&lt;0.001$)</td>
</tr>
<tr>
<td>Urethral resistance</td>
<td>($p=0.005$)</td>
</tr>
<tr>
<td>Straining during voiding</td>
<td>($p=0.003$)</td>
</tr>
</tbody>
</table>

The value of pre-operative urodynamic variables for the prediction of post-operative voiding performance (after multiple regression analysis) is summarised in table 35.

**Table 35.** Predictive value of pre-operative urodynamic variables (p-value in brackets).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Day of void $&gt;50\text{ ml}$</th>
<th>Day of catheter removal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peak flow rate</td>
<td>not predictive</td>
<td>predictive</td>
</tr>
<tr>
<td></td>
<td>($p=0.004$)</td>
<td>($p=0.005$)</td>
</tr>
<tr>
<td>Detrusor pressure at maximum flow</td>
<td>not predictive</td>
<td>not predictive</td>
</tr>
<tr>
<td>Maximum detrusor pressure</td>
<td>not predictive</td>
<td>not predictive</td>
</tr>
<tr>
<td>Urethral Resistance</td>
<td>predictive</td>
<td>predictive*</td>
</tr>
<tr>
<td></td>
<td>($p=0.001$)</td>
<td>($p=0.005$)</td>
</tr>
<tr>
<td>After contraction (absence and decreasing strength)</td>
<td>not predictive</td>
<td>not predictive</td>
</tr>
<tr>
<td>Straining during voiding</td>
<td>predictive</td>
<td>predictive</td>
</tr>
<tr>
<td></td>
<td>($p=0.045$)</td>
<td>($p=0.005$)</td>
</tr>
<tr>
<td>Voiding pattern</td>
<td>not predictive</td>
<td>not predictive</td>
</tr>
</tbody>
</table>

* Only in women with bladder neck elevation greater than 26 mm.
Subjective assessment at one year was possible for all 77 women. All women completed the King’s Health Questionnaire before surgery, at three months and one year. While the study aimed to assess quality of life only in women with post-operative voiding dysfunction and detrusor instability (see later), quality of life data before and after colposuspension were available for all women and are shown in appendix 13. Objective assessment was performed in 76 women. One woman who lived far could not be available for testing and was interviewed over the phone. Cystometry was performed in 74 women. Two women who declined cystometry had a pad test.

A total of 16 women were found to have stress incontinent either subjectively or objectively or both. Nine women reported symptoms of stress incontinence, with a subjective failure rate of 11.6%. Symptoms were severe in one case, moderate in one case and mild in seven cases. Only one patient with subjective stress incontinence (described as severe) had demonstrable stress incontinence on cystometry. All other women with symptoms of stress incontinence were urodynamically normal (except one with detrusor instability). Eight women were seen to leak urine during stress on cystometry, with an objective failure rate of 10.5%. No women had stress incontinence while coughing in the sitting position, and leakage was seen only when standing and coughing with a full bladder. Only one patient found to have objective stress incontinence was also symptomatic. All other women with a urodynamic diagnosis of GSI were asymptomatic.
The relationship between outcome and elevation (as measured with MRI) was analysed using a Student's t-test. The mean elevation of patients with objective 'success' and those with objective 'failure' was not found to be significantly different (p=0.53). However, the mean elevation of patients with subjective 'success' was significantly lower than the mean elevation of patients with subjective 'failure' (P=0.039) (table 36).

Table 36. Association between amount of bladder neck elevation (MRI measures) and outcome of surgery.

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Success</th>
<th>Failure</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean elevation (n. patients)</td>
<td>Mean elevation (n. patients)</td>
<td></td>
</tr>
<tr>
<td>Subjective</td>
<td>28.12 (64)</td>
<td>33.22 (9)</td>
<td>0.039</td>
</tr>
<tr>
<td>Objective</td>
<td>28.77 (64)</td>
<td>30.75 (8)</td>
<td>0.53</td>
</tr>
</tbody>
</table>

Patients were divided into two groups based on elevation: equal or less than 26 mm (group 1) and more than 26 mm (group 2). The objective failure rate was 10.7% (3 of 28) in group 1 (26 mm or less), and 11.1% (5 of 45) in group 2 (> 26 mm). The subjective failure rate was 3.5% (1 of 28) in group 1 (26 mm or less), and 15.5% (7 of 45) in group 2 (> 26 mm). A Fisher's exact test was used to investigate whether there was an association between elevation group and outcome. No evidence of an association was found (table 37).

Table 37. Association between elevation group and outcome.

<table>
<thead>
<tr>
<th>Elevation</th>
<th>Objective failure n (%)</th>
<th>Subjective failure n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1: elevation of 26mm or less (n=28)</td>
<td>3 (10.7%)</td>
<td>1 (3.5%)</td>
</tr>
<tr>
<td>Group 2: elevation &gt; 26mm (n=45)</td>
<td>5 (11.1%)</td>
<td>7 (15.5%)</td>
</tr>
<tr>
<td>P-value</td>
<td>p = 1</td>
<td>p = 0.14</td>
</tr>
</tbody>
</table>
FACTORS ASSOCIATED WITH VARIATIONS IN AMOUNT OF BLADDER NECK ELEVATION (i.e. reasons for variability in amount of bladder neck elevation between patients despite the use of the same operative technique)

In relation to bladder neck elevation, the variables studied were: age, BMI and position of the bladder neck before surgery (Dx) (using Pearson's correlation coefficient); history of previous hysterectomy, history of previous bladder neck surgery and menopausal status (using t-tests); parity and amount of cystocele (using analysis of variance).

The position of the bladder neck before surgery (Dx, see fig. 9) was on the Y axis (zero value) in 6 women (8.2%), above the Y axis (positive value) in 47 women (64.3%), and below the Y axis (negative value) in 20 women (27.3%). The mean value of Dx was 3.7 mm (above the Y axis). Mean values of all other variables are shown in tables 16 and 17.

Variables found to be significantly associated with the amount of bladder neck elevation obtained at colposuspension are shown in table 38.

**Table 38.** Variables associated with greater bladder neck elevation after colposuspension.

<table>
<thead>
<tr>
<th>Variables</th>
<th>p-value (correlation)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lower position of the bladder neck before colposuspension</td>
<td>p &lt; 0.001 (correlation - 0.79)</td>
</tr>
<tr>
<td>Increasing age</td>
<td>p &lt; 0.01 (correlation 0.34)</td>
</tr>
<tr>
<td>Post-menopausal status</td>
<td>p = 0.0031</td>
</tr>
<tr>
<td>Increasing cystocele</td>
<td>p = 0.01</td>
</tr>
</tbody>
</table>
There was no association between amount of bladder neck elevation and BMI, history of hysterectomy, history of bladder neck surgery and parity. Using analysis of variance, the use of HRT in post-menopausal women was not associated with differences in bladder neck elevation. When using multiple regression analysis, the only independent variable which was significantly associated with the amount of elevation was the position of the bladder neck before surgery (p<0.001), which explained 63.7% of the variability in elevation. If this variable is left out of the model, then age and amount of cystocele become significant (p=0.001 and 0.007, respectively), but the resulting model would explain just 18.4% of the variability in elevation.
10- THE NATURAL HISTORY OF VOIDING DYSFUNCTION

The natural history of voiding dysfunction was studied in all 77 recruited women.

a) Voiding urodynamic variables before and after colposuspension

All women had pre-operative voiding cystometry. All women were seen at three months. They all had uroflowmetry and 76 had voiding cystometry. One patient declined to have cystometry due to discomfort. All women but one were seen at one year. The patient who did not attend lived far away and was interviewed by phone. All women who attended had uroflowmetry, but two patients declined cystometry.

Mean values of peak flow rate, detrusor pressure at maximum flow, maximum detrusor pressure and urethral resistance before colposuspension and at three and 12 months follow-up are presented in table 39. Values were compared using a paired Student’s t-test. All the variables were clearly significantly different at three months and one year from before surgery, with a reduction in mean peak flow rate and increases in the means of the other variables. Generally the differences were more pronounced at three months. The ‘fall back’ from three months to one year after surgery was significant for peak flow rate, detrusor pressure at maximum flow and maximum detrusor pressure, but not for resistance.

Table 39. Cystometric voiding variables before colposuspension and at three and 12 months follow-up. Mean values (SD) and p-values when comparing pre and post-operative variables and when comparing variables at 12 months to those at three months. The number of observations is detailed below (missing values were due to non-attendance or technical failure as the patients voided the pressure line).

<table>
<thead>
<tr>
<th></th>
<th>Before surgery mean (SD)</th>
<th>3 months mean (SD)</th>
<th>1 year mean (SD)</th>
<th>p-value 3 months</th>
<th>p-value 12 months</th>
<th>p-value ‘fall back’ 3-12 months</th>
</tr>
</thead>
<tbody>
<tr>
<td>Qmax (ml/s)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>n=77</td>
<td>28.9 (10.5) n=77</td>
<td>19.2 (8) n=77</td>
<td>22.4 (12) n=75</td>
<td>0.0001</td>
<td>0.0002</td>
<td>0.0007</td>
</tr>
<tr>
<td>pdet, Qmax (cmH2O)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>n=74</td>
<td>21.3 (11.1) n=74</td>
<td>32 (13.5) n=75</td>
<td>28.6 (15) n=72</td>
<td>0.0001</td>
<td>0.004</td>
<td>0.049</td>
</tr>
<tr>
<td>pdet, max (cmH2O)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>n=74</td>
<td>28.3 (14.7) n=74</td>
<td>47.5 (18.9) n=75</td>
<td>37.7 (19.4) n=70</td>
<td>0.0001</td>
<td>0.0026</td>
<td>0.0001</td>
</tr>
<tr>
<td>Urethral Resistance</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>n=74</td>
<td>0.034 (0.034) n=75</td>
<td>0.175 (0.442) n=72</td>
<td>0.100 (0.108) n=72</td>
<td>0.011</td>
<td>0.0001</td>
<td>0.11</td>
</tr>
</tbody>
</table>
b) Incidence of post-operative voiding difficulty

No women were considered to have pre-operative voiding dysfunction as judged by urodynamic studies (i.e. no residual greater than 100 ml). Post-operatively, the percentage of women with voiding dysfunction in relation to time is illustrated in table 40.

Table 40. Voiding performance of 77 women after colposuspension.

<table>
<thead>
<tr>
<th>Residual urine &gt; 100 ml</th>
<th>n,%</th>
</tr>
</thead>
<tbody>
<tr>
<td>For &gt; 7 days*</td>
<td>53 (68.8%)</td>
</tr>
<tr>
<td>For &gt; 14 days*</td>
<td>22 (28.5%)</td>
</tr>
<tr>
<td>For &gt; 30 days*</td>
<td>5 (6.4%)</td>
</tr>
<tr>
<td>For &gt; 3 months**</td>
<td>6 (7.7%)</td>
</tr>
<tr>
<td>For &gt; 1 year**</td>
<td>2 (2.5%)</td>
</tr>
</tbody>
</table>

* Residuals measured using the suprapubic catheter or CISC.
** Residuals measured at urodynamics.

At three months follow-up, six patients were found to have residual urine volumes greater than 100 ml on more than one occasion (7.7%). These six women constitute the main subject of the investigation on the natural history of voiding dysfunction and on the impact of this complication on quality of life.

The pre-operative and operative features (including early voiding function) of these six women are illustrated in tables 41 and 42.

Table 41. Pre-operative features of women with voiding dysfunction three months after colposuspension.

<table>
<thead>
<tr>
<th>No</th>
<th>Age</th>
<th>Qmax (ml/s)</th>
<th>Pdet.Qmax (cmH₂O)</th>
<th>pdet.max (cmH₂O)</th>
<th>Resistance (pdet.Qmax/Qmax²)</th>
<th>Straining during void</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>56</td>
<td>20</td>
<td>34</td>
<td>48</td>
<td>0.08</td>
<td>yes</td>
</tr>
<tr>
<td>2</td>
<td>61</td>
<td>43</td>
<td>17</td>
<td>17</td>
<td>0.009</td>
<td>yes</td>
</tr>
<tr>
<td>3</td>
<td>76</td>
<td>22</td>
<td>23</td>
<td>30</td>
<td>0.04</td>
<td>yes</td>
</tr>
<tr>
<td>4</td>
<td>52</td>
<td>20</td>
<td>29</td>
<td>44</td>
<td>0.07</td>
<td>yes</td>
</tr>
<tr>
<td>5</td>
<td>63</td>
<td>28</td>
<td>8</td>
<td>14</td>
<td>0.01</td>
<td>yes</td>
</tr>
<tr>
<td>6</td>
<td>70</td>
<td>15</td>
<td>10</td>
<td>10</td>
<td>0.04</td>
<td>yes</td>
</tr>
</tbody>
</table>
Table 42. Operative features and early voiding function of women with voiding dysfunction three months after colposuspension.

<table>
<thead>
<tr>
<th>No</th>
<th>Bladder neck elevation at MRI (mm)</th>
<th>Day of void &gt; 50 ml</th>
<th>Day of catheter removal</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>33</td>
<td>16</td>
<td>21</td>
</tr>
<tr>
<td>2</td>
<td>26</td>
<td>11</td>
<td>16</td>
</tr>
<tr>
<td>3</td>
<td>36</td>
<td>42</td>
<td>190*</td>
</tr>
<tr>
<td>4</td>
<td>31</td>
<td>8</td>
<td>16</td>
</tr>
<tr>
<td>5</td>
<td>48</td>
<td>15</td>
<td>27</td>
</tr>
<tr>
<td>6</td>
<td>32</td>
<td>3</td>
<td>47*</td>
</tr>
</tbody>
</table>

* Time of stopping CISC (i.e. residuals consistently < 100 ml).

As detailed in table 42, only two of six patients had experienced voiding difficulty since surgery. They required prolonged catheterisation followed by CISC. All other patients had their catheters removed 16-27 days after colposuspension (when their urinary residuals were less than 100 ml) and were later (at three months) found to have a residual urine volume greater than 100 ml.

In five patients bladder neck elevation was greater than 26 mm as measured with MRI, while in one case elevation was 26 mm (table 42).

c) History of patients with 'late' voiding dysfunction

The clinical, urodynamic and quality of life (King’s Health Questionnaire) data of these patients are shown in detail in tables 43 and 44. The impact of post-operative symptoms on quality of life in women with post-operative voiding dysfunction, as compared to pre-operative symptoms, is shown in table 45. Details of all quality of life domains (as reported in King’s Health Questionnaire) are shown in appendix 14. Urinary symptoms affecting quality of life, which may or may not have been a consequence of voiding dysfunction, are presented in table 44. The presence of coexistent detrusor instability and genuine stress incontinence is shown in table 44.

None of these patients had genital prolapse which could impair voiding.
The individual history of these six patients is summarised as follows:

**Patient No 1:**

When seen at three months, this patient was greatly distressed by symptoms of hesitancy, decreased and interrupted flow, feeling of incomplete emptying and need to stand to achieve voiding. She was able to void good volumes of urine (maximum 500 ml, average 150 ml), but had mean residuals of 200 ml. She was offered CISC but declined. She also refused re-insertion of a suprapubic catheter. She went on to develop recurrent urinary tract infections which greatly affected the quality of her life. She was treated with low dose prophylactic antibiotics and additional intermittent full dose courses. Gradually her voiding function improved subjectively and objectively. On review at one year her voiding was normal, but quality of life was still impaired by recurrent urinary tract infections.

**Patient No 2:**

When seen at three months, this patient had ‘irritative’ symptoms and complained of urge incontinence and stress incontinence. On cystometry her bladder was stable and stress incontinence could not be demonstrated. Her residual urine volumes were found to be high (200-350 ml). She was instructed on CISC and performed it once per day for three weeks, after which she discontinued the practice as her residual urine volumes were consistently less than 100 ml. On review at one year her symptoms persisted and quality of life was still impaired, although she was found to be urodynamically normal.

**Patient No 3:**

This patient started performing CISC five weeks after colposuspension as she was totally unable to void. Gradually she started voiding, and at the three months visit she was performing CISC only once per day (with residuals of 200-250 ml). Her voiding function
continued to improve and she was able to stop CISC (with residuals consistently < 100 ml) 190 days after colposuspension. On review at one year her voiding was normal. Quality of life had not been significantly impaired.

Patients No 4 and 5:

On review at three months, these two patients were virtually asymptomatic. Using timed voiding and double voiding they were able to reduce their residual urine volume as shown by ultrasound assessments. On review at one year, patient No 4 had residual urine volumes of less than 100 ml, but patient No 5 still had residual urine volumes of 160-170 ml. Again she was found to empty her bladder completely after a double void. She was only mildly bothered and declined treatment and follow-up.

Patient No 6:

This patient had to have her suprapubic catheter removed 11 days after surgery due to constant bypassing of urine around it. As she was still unable to void, a Foley catheter was inserted and left on free drainage for 10 days. After its removal her residuals were found to be greater than 100 ml and she learned CISC. At three months she was performing CISC only intermittently (when required by worsening symptoms of voiding difficulty). She had discontinued regular CISC 47 days after colposuspension (but continued to perform it sporadically). At one year she was still having residual urine volumes of 75-160 ml and was performing CISC once per day. Quality of life had not been significantly impaired.
Table 43. Urodynamic features of women with ‘late’ voiding dysfunction (i.e. with residual urine volumes > 100 ml three months after colposuspension) (n=6), at three months and one year follow-up.

<table>
<thead>
<tr>
<th>No</th>
<th>Residual &gt; 100ml</th>
<th>Qmax (ml/s)</th>
<th>Pdet.Qmax (cmH2O)</th>
<th>pdet,max (cmH2O)</th>
<th>Resistance</th>
<th>Obstructed*</th>
</tr>
</thead>
<tbody>
<tr>
<td>3m</td>
<td>12</td>
<td>3m</td>
<td>12m</td>
<td>3m</td>
<td>12m</td>
<td>3m</td>
</tr>
<tr>
<td>1</td>
<td>Yes</td>
<td>No</td>
<td>13</td>
<td>17</td>
<td>31</td>
<td>62</td>
</tr>
<tr>
<td>2</td>
<td>Yes</td>
<td>No</td>
<td>7</td>
<td>15</td>
<td>29</td>
<td>24</td>
</tr>
<tr>
<td>3</td>
<td>Yes</td>
<td>No</td>
<td>8</td>
<td>12</td>
<td>32</td>
<td>21</td>
</tr>
<tr>
<td>4</td>
<td>Yes</td>
<td>No</td>
<td>12</td>
<td>9</td>
<td>30</td>
<td>35</td>
</tr>
<tr>
<td>5</td>
<td>Yes</td>
<td>Yes</td>
<td>4</td>
<td>10</td>
<td>61</td>
<td>33</td>
</tr>
<tr>
<td>6</td>
<td>Yes</td>
<td>Yes</td>
<td>14</td>
<td>9</td>
<td>45</td>
<td>35</td>
</tr>
</tbody>
</table>

* As defined by Massey and Abrams [362] (chapter 8, section 2).

Table 44. Clinical and quality of life features of women with ‘late’ voiding dysfunction (i.e. with residual urine volumes > 100 ml three months after colposuspension) (n=6), at three months and one year follow-up.

<table>
<thead>
<tr>
<th>No</th>
<th>Symptoms of voiding Dysfunction*</th>
<th>Need for CISC</th>
<th>No of proven UTI Since surgery</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>3m</td>
<td>12m</td>
<td>3m</td>
</tr>
<tr>
<td>1</td>
<td>3</td>
<td>0</td>
<td>Yes</td>
</tr>
<tr>
<td>2</td>
<td>0</td>
<td>0</td>
<td>Yes</td>
</tr>
<tr>
<td>3</td>
<td>1</td>
<td>0</td>
<td>Yes</td>
</tr>
<tr>
<td>4</td>
<td>0</td>
<td>0</td>
<td>No</td>
</tr>
<tr>
<td>5</td>
<td>1</td>
<td>1</td>
<td>No</td>
</tr>
<tr>
<td>6</td>
<td>1</td>
<td>1</td>
<td>Yes</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>No</th>
<th>'Irritative symptoms'* (DI on cystometry)</th>
<th>Stress incontinence* (GSI on cystometry)</th>
<th>Overall impact of urinary symptoms on quality of life**</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>3m</td>
<td>12m</td>
<td>3m</td>
</tr>
<tr>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>3</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>4</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>5</td>
<td>2 (DI)</td>
<td>3 (DI)</td>
<td>0</td>
</tr>
<tr>
<td>6</td>
<td>1 (DI)</td>
<td>1 (DI)</td>
<td>0 (GSI)</td>
</tr>
</tbody>
</table>

* Symptoms are described by their degree of ‘bother’ (0 = absent, 1 = mild, 2 = moderate, 3 = severe).
** King’s Health Questionnaire (how much do you think your bladder problem affects your quality of life?).
Table 45. Impact of urinary symptoms on quality of life before colposuspension, and at three and twelve months, in women with post-operative voiding dysfunction (n=6).

<table>
<thead>
<tr>
<th>No</th>
<th>Symptoms impact before colposuspension</th>
<th>Symptoms impact at three months</th>
<th>Symptoms impact at twelve months</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Moderate</td>
<td>A lot</td>
<td>Moderate</td>
</tr>
<tr>
<td>2</td>
<td>A lot</td>
<td>A lot</td>
<td>A lot</td>
</tr>
<tr>
<td>3</td>
<td>Moderate</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>4</td>
<td>Moderate</td>
<td>No</td>
<td>A little</td>
</tr>
<tr>
<td>5</td>
<td>A lot</td>
<td>No</td>
<td>A little</td>
</tr>
<tr>
<td>6</td>
<td>A lot</td>
<td>A little</td>
<td>A little</td>
</tr>
</tbody>
</table>

Summary of outcome

Patients No 1 and 2 had a poor outcome from colposuspension in relation to quality of life issues, although improvement in specific quality of life domains had occurred and is documented in appendix 14. It should be noted that these patients were found to be urodynamically normal at one-year follow-up. ‘Bothersome’ urinary symptoms in one patient were mostly due to recurrent UTIs (which were not present before surgery). In the other patient quality of life was impaired by ‘irritative’ symptoms of frequency, urgency and urge incontinence (despite a stable bladder), which had been present before colposuspension.

Patients No 3, 4, 5 and 6 had considerable improvement in all quality of life domains (appendix 14) at three months and at one year, despite the development of voiding dysfunction (and the need to perform CISC for patients No 3 and 6).
d) Other markers of impaired voiding

Markers of suboptimal voiding in all 77 patients before colposuspension and at three and twelve months follow-up (as shown by a peak flow of less than the fifth centile, a peak flow rate of less than 15 ml/s, and the presence of urodynamic obstruction) are shown in table 46.

Table 46. Peak flow rate < 5th centile, peak flow rate < 15 ml/s, and presence of urodynamic obstruction, before colposuspension and at three and twelve months follow-up. Percentages refer to number of patients tested (before colposuspension: uroflowmetry and cystometry = 77, three months: uroflowmetry = 77, cystometry = 76, twelve months: uroflowmetry = 76, cystometry = 74).

<table>
<thead>
<tr>
<th></th>
<th>Before colposuspension</th>
<th>3m  n (%)</th>
<th>12m  n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Qmax &lt; 5th centile</td>
<td>22 (28.5%)</td>
<td>50 (64.9%)</td>
<td>39 (51.3%)</td>
</tr>
<tr>
<td>(Liverpool nomograms)*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Qmax &lt; 15 ml/s</td>
<td>5 (6.4%)</td>
<td>19 (24.6%)</td>
<td>15 (19.7%)</td>
</tr>
<tr>
<td>Obstruction</td>
<td>0</td>
<td>14 (18.4%)</td>
<td>9 (12.1%)</td>
</tr>
<tr>
<td>(Massey and Abrams)**</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Haylen et al, 1989 [287]
**Massey and Abrams, 1988 [362]

e) A special case of voiding dysfunction

This case highlights the difficulty in diagnosing voiding dysfunction using current investigations. This patient complained of symptoms of voiding dysfunction (hesitancy, decreased, interrupted and prolonged flow) which affected severely her quality of life, despite successful cure of her stress incontinence (unchanged impact of bladder symptoms, as reported in the King's Health Questionnaire). Her voiding had been normal before colposuspension (Qmax 25 ml/s, pdet,Qmax 42 cmH2O, resistance 0.06, no residual urine).
When reviewed at three months and one year Qmax was 18 and 29 ml/s respectively, pdet,Qmax was 60 and 26 cmH2O respectively, and resistance was 0.18 and 0.03 respectively. Residual urine volumes were low. There was no obvious voiding dysfunction. Her voiding pattern however was clearly abnormal, repeatedly showing prolonged and interrupted flow (figure 18).

**Fig. 18.** A special case of voiding dysfunction. Voiding cystometry at one year. Voided volume 671 ml, Qmax 29 ml/s, pdet,Qmax 26 cmH2O, resistance 0.03, residual 2 ml, voiding time 234 seconds, flow time 180 seconds.
CHAPTER 10

RESULTS:

THE CAUSES AND NATURAL HISTORY OF 'DE NOVO'

DETRUSOR INSTABILITY
I- PRE-OPERATIVE VARIABLES

Data from 56 women with genuine stress incontinence and stable bladders were included for analysis.

Details on age, menopausal status, weight (BMI), presence of symptoms of urge incontinence pre-operatively, and presence of a past history of hysterectomy and bladder neck surgery are presented in chapter 8, section 3.

The pre-operative urodynamic data are presented in table 47.

Table 47. The study of de novo detrusor instability. Pre-operative urodynamic data.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mean (Range)</th>
<th>&lt; 12 ml/s: n=1</th>
<th>&lt; 15 ml/s: n=5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Qmax</td>
<td>28.5 ml/s (7-56)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>pdet,Qmax*</td>
<td>20 cmH2O (0-43)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Resistance*</td>
<td>0.034 (0.009-0.18)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pdet,max*</td>
<td>27.3 cmH2O (0-69)</td>
<td>Acontractile (pdet=0): n=1</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>&lt; 15 cmH2O: n=11 (19.6%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>15-35 cmH2O: n=32 (57.1%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>36-50 cmH2O: n=9 (16%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>&gt; 50 cmH2O: n=4 (7.1%)</td>
<td></td>
</tr>
<tr>
<td>MUCP</td>
<td>41.2 cmH2O (7-92)</td>
<td>&lt; 20 cmH2O: n=6 (10.7%)</td>
<td></td>
</tr>
<tr>
<td>After contraction†</td>
<td>Present: n=30 (53.5%)</td>
<td>Mean 75 cmH2O</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Absent: n=20 (35.7%)</td>
<td>(range: 24-230 cmH2O)</td>
<td></td>
</tr>
<tr>
<td>Abdominal straining</td>
<td>Present: n=23 (41%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Absent: n=33 (58.9%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Voiding pattern‡</td>
<td>1) 28 (50%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>2) 17 (30.3%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>3) 6 (10.7%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>4) 5 (8.9%)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* No values in 2 women (pressure lines voided).
† No values in 6 women (pressure lines voided).
‡ 1) detrusor contraction alone, 2) detrusor contraction and abdominal straining,
3) pelvic relaxation with additional abdominal straining, 4) pelvic relaxation alone.
The HAD questionnaire was completed pre-operatively by 55 women (98.2%). Values are presented in table 48.

Table 48. The study of de novo detrusor instability. Pre-operative anxiety and depression.

<table>
<thead>
<tr>
<th>Depression</th>
<th>Anxiety</th>
</tr>
</thead>
<tbody>
<tr>
<td>n (%)</td>
<td>n (%)</td>
</tr>
<tr>
<td>Non cases</td>
<td>43 (76.7%)</td>
</tr>
<tr>
<td>Borderline</td>
<td>10 (17.8%)</td>
</tr>
<tr>
<td>Cases</td>
<td>2 (3.5%)</td>
</tr>
</tbody>
</table>

2- OPERATIVE VARIABLES

Measures of elevation:

Objective mean bladder neck elevation achieved by colposuspension, as measured by MRI, was 28.8 mm (range: 12-48). Two women declined MRI due to claustrophobia. Two women could not be tested due to equipment failure.

Intra-operative ‘bow-stringing’ was seen in 16 cases (28.5%). This was considered of 10 mm or less in 10 cases and of more than 10 mm in six cases.

Measures of urethral compression:

The mean distance between the bladder neck and the posterior surface of the pubic bone, as measured by MRI, was 5.8mm (range: 2-13).

The distance between the medial stitches as measured during surgery with a ruler was a mean of 41.8 mm at the paravaginal insertion (range: 22-65), and a mean of 49.6mm at the pectineal insertion (range: 27-66).
The mean size of paired Hegars that could be inserted between the urethra and the pubic bone after tying the sutures was 11.7 (range 8-17). This variable was obtained for 45 patients only (80.3%), as initial attempts using only one Hegar were found to be poorly reproducible and data from these 11 women were discarded.

3- POST-OPERATIVE VARIABLES

Post-operative voiding: Patients were able to void more than 50 ml after a mean of 5.4 days (range: 2-27). The catheter was removed after a mean of 12.5 days (range: 4-36). When including the outlier who voided 50 ml only after 42 days and required catheterisation for 190 days, the means were 6.1 and 15.7 days respectively. Forty women (71.4%) required a catheter for more than seven days.

The McGill pain questionnaire was completed by 54 women (96.4%). The mean value for days 2-5 was 14.1 (range: 0-64).

4- VOIDING CYSTOMETRY AT THREE MONTHS

The post-operative voiding cystometric data (at three months) are presented in table 49. All 56 women underwent filling and voiding cystometry, except one woman who could not void with the pressure lines in situ.
Table 49. The study of *de novo* detrusor instability. Post-operative voiding urodynamic data (at three months).

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>n</th>
</tr>
</thead>
<tbody>
<tr>
<td>Qmax mean (range)</td>
<td>19.4 ml/s (4-45)</td>
<td>&lt; 12 ml/s: n=11</td>
</tr>
<tr>
<td>pdet,Qmax mean (range)</td>
<td>29.9 cmH2O (9-71)</td>
<td>&lt; 15 ml/s: n=13</td>
</tr>
<tr>
<td>Resistance mean (range)</td>
<td>0.18 (0.09-3.8)</td>
<td></td>
</tr>
<tr>
<td>pdet,max mean (range)</td>
<td>45.6 cmH2O (10-114)</td>
<td>Acontractile (pdet=0): n=0</td>
</tr>
<tr>
<td>Residual urine &gt; 100 ml n (%)</td>
<td>5 (8.9%)</td>
<td></td>
</tr>
<tr>
<td>Abdominal Straining</td>
<td>Present: n=16 (28.5%)</td>
<td></td>
</tr>
<tr>
<td>Voiding pattern**</td>
<td>1) 36 (64.2%)</td>
<td>1) detrusor contraction alone, 2) detrusor contraction and abdominal straining,</td>
</tr>
<tr>
<td></td>
<td>2) 16 (28.5%)</td>
<td>3) pelvic relaxation with abdominal straining, 4) pelvic relaxation alone.</td>
</tr>
<tr>
<td></td>
<td>3) 1 (1.7%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>4) 2 (3.5%)</td>
<td></td>
</tr>
<tr>
<td>Obstructed (Massey and Abrams) n (%)</td>
<td>10 (17.8%)</td>
<td></td>
</tr>
</tbody>
</table>

* Variables not obtained in 1 woman (could not void with the catheters in place).

** 1) detrusor contraction alone, 2) detrusor contraction and abdominal straining, 3) pelvic relaxation with abdominal straining, 4) pelvic relaxation alone.

5- OUTCOME MEASURES

a) **Objective / Detrusor instability:**

At three months, 12 women (21.4%) experienced unstable detrusor contractions accompanied by urgency and were diagnosed as having detrusor instability. The mean height of unstable contraction was 21.5 cmH2O (range: 10-38 cmH2O). In two cases the rise in detrusor pressure was lower than 15 cmH2O. Unstable contractions occurred at a mean volume of 255 ml (range: 85-600). In two cases unstable contractions occurred only
after provocation (hand wash), while in all others they were spontaneous. In only two cases did unstable contractions cause leakage. Mean cystometric capacity was 549 ml (range: 335-808 ml).

Eight women with *de novo* detrusor instability reported urge incontinence (66.6%). This was mild in four cases and moderate in four cases. Details are shown in table 50.

**Table 50. De novo detrusor instability: cystometric and clinical details.**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Maximum detrusor pressure rise (cmH2O)</th>
<th>Volume at first detrusor contraction (ml)</th>
<th>Cystometric capacity (ml)</th>
<th>Leakage</th>
<th>Urge incontinence</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>23</td>
<td>100</td>
<td>785</td>
<td>no</td>
<td>no</td>
</tr>
<tr>
<td>2</td>
<td>18</td>
<td>259</td>
<td>692</td>
<td>no</td>
<td>mild</td>
</tr>
<tr>
<td>3</td>
<td>14</td>
<td>90</td>
<td>516</td>
<td>no</td>
<td>moderate</td>
</tr>
<tr>
<td>4</td>
<td>18</td>
<td>141</td>
<td>437</td>
<td>no</td>
<td>moderate</td>
</tr>
<tr>
<td>5</td>
<td>10</td>
<td>264</td>
<td>502</td>
<td>no</td>
<td>no</td>
</tr>
<tr>
<td>6</td>
<td>22</td>
<td>616</td>
<td>808</td>
<td>no</td>
<td>no</td>
</tr>
<tr>
<td>7</td>
<td>22</td>
<td>85</td>
<td>574</td>
<td>yes</td>
<td>no</td>
</tr>
<tr>
<td>8</td>
<td>38</td>
<td>251</td>
<td>438</td>
<td>no</td>
<td>moderate</td>
</tr>
<tr>
<td>9</td>
<td>20</td>
<td>600</td>
<td>680</td>
<td>yes</td>
<td>mild</td>
</tr>
<tr>
<td>10</td>
<td>19</td>
<td>143</td>
<td>335</td>
<td>no</td>
<td>mild</td>
</tr>
<tr>
<td>11</td>
<td>25</td>
<td>200</td>
<td>394</td>
<td>no</td>
<td>moderate</td>
</tr>
<tr>
<td>12</td>
<td>29</td>
<td>320</td>
<td>436</td>
<td>no</td>
<td>mild</td>
</tr>
</tbody>
</table>

b) **Subjective / Urge incontinence:**

Twenty five women reported urge incontinence post-operatively (44.6%). This was mild in 17 cases, moderate in five cases and severe in three cases.

Cystometry showed detrusor instability in only eight of these women (32%), as detailed above.
6- FACTORS ASSOCIATED WITH *DE NOVO* DETRUSOR INSTABILITY

**PRE-OPERATIVE VARIABLES**

An analysis of Covariance was used, with age and BMI as covariates, to investigate differences in peak flow rate, detrusor pressure at maximum flow, maximum detrusor pressure, resistance, anxiety and depression. A **Student's t-test** was used to investigate age. A **Mann-Whitney test** was used to investigate differences in relation to the presence or absence of an after-contraction, as this was not normally distributed. **Chi-squared tests** were used for the categorical variables (presence or absence of straining, type of voiding pattern, menopausal status, history of previous hysterectomy, history of previous bladder neck surgery, the presence of pre-operative symptoms of urgency).

Factors associated with the development of *de novo* detrusor instability (objective outcome measure) were increasing age (*P* = 0.05) and previous bladder neck surgery (*P* = 0.04) (table 51).

The only pre-operative factor associated with the development of symptoms of urge incontinence (subjective outcome measure) was the presence of urge incontinence pre-operatively (*P* = 0.01) (table 52).
OPERATIVE AND POST-OPERATIVE VARIABLES

A Student's t-test was used to compare means for elevation (MRI and suture 'bow-stringing'), urethral compression (as measured with Hegars and distance between medial stitches), post-operative pain and post-operative voiding performance (day of voiding > 50 ml and day of catheter removal). A Mann-Whitney test was used for the distance between the bladder neck and the pubic bone, as this variable was not normally distributed.

The following factors were associated with the development of de novo detrusor instability (objective outcome measure): increasing bladder neck elevation ($P = 0.02$) and increasing urethral compression (as shown by decreasing distance between bladder neck and pubis as seen with MRI) ($P = 0.01$) (table 51). There were no post-operative variables associated with this outcome.

As no cases of prolonged voiding dysfunction were seen when the bladder neck was elevated by 26 mm or less (chapter 9, section 7), the possibility that this amount of elevation was 'safe' also in relation to the development of de novo detrusor instability was explored.

Twenty women (35.7%) had bladder neck elevation of 26 mm or less, and two of them (10%) developed de novo detrusor instability. Thirty two women (57.1%) had bladder neck elevation greater than 26 mm, and seven of them (21.8%) developed de novo detrusor instability. Using Chi-squared tests, this difference was not statistically significant ($P=0.27$). It should however be noted that MRI measures of elevation were not available for three women with de novo detrusor instability (three of the four women who did not have MRI).
The only operative/post-operative factor found to be associated with the development of the symptom of urge incontinence (subjective outcome measure) was an increasing pain score in post-operative days 2-5 ($P = 0.001$) (table 52).

Table 51. Variables associated with de novo detrusor instability (objective outcome).

<table>
<thead>
<tr>
<th>Variables</th>
<th>Stable n = 44</th>
<th>De novo D.I. n = 12</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increasing age (mean)</td>
<td>51.2 y</td>
<td>58.8 y</td>
<td>$P = 0.05$</td>
</tr>
<tr>
<td>Previous bladder neck surgery (n)</td>
<td>6</td>
<td>5</td>
<td>$P = 0.04$</td>
</tr>
<tr>
<td>Increasing bladder neck elevation (mean elevation)</td>
<td>27.7 mm</td>
<td>34.8 mm</td>
<td>$P = 0.02$</td>
</tr>
<tr>
<td>Increasing urethral compression (median distance bladder neck to pubis)</td>
<td>6 mm</td>
<td>4 mm</td>
<td>$P = 0.01$</td>
</tr>
</tbody>
</table>

Table 52. Variables associated with post-operative urge incontinence (subjective outcome).

<table>
<thead>
<tr>
<th>Variables</th>
<th>No urge Incontinence n = 31</th>
<th>Urge Incontinence n = 25</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-operative urge incontinence (n)</td>
<td>16</td>
<td>21</td>
<td>0.01</td>
</tr>
<tr>
<td>Pain score (post-operative days 2-5) (mean)</td>
<td>9.13</td>
<td>20.5</td>
<td>0.001</td>
</tr>
</tbody>
</table>

URODYNAMIC VARIABLES OF OBSTRUCTION

1) When using the criteria for diagnosing obstruction suggested by Massey and Abrams [362] (chapter 8, section 2), 10 women were found to be obstructed on post-operative urodynamics at three months (table 49). Two of them had symptomatic de novo detrusor instability, four had symptoms of urge incontinence but stable bladders, and four had stable bladders and no symptoms of urge incontinence.
Two of 12 women with detrusor instability (16.6%) and five of 25 women with symptoms of urge incontinence (20%) were obstructed using these criteria.

Analysis of this variable in relation to the outcome measures was performed using a Chi-squared test. No association was found between a urodynamic diagnosis of obstruction and the development of de novo detrusor instability and the symptom of urge incontinence.

2) When comparing urodynamic variables at three months between patients with and without de novo detrusor instability and with and without symptoms of urge incontinence, a Student's t-test was used to compare means for peak flow rate, maximum detrusor pressure, and detrusor pressure at maximum flow. A Mann-Whitney test was used for resistance as this appeared to be distributed non-normally. Chi-squared tests were used for the categorical variables (straining during voiding and voiding pattern).

No significant differences were found for any of the outcome measures.

3) When comparing pre-operative to post-operative urodynamic variables in relation to the development of de novo detrusor instability or of symptoms of urge incontinence, a Student's t-test was used to compare means for the variables (difference in) peak flow rate, maximum detrusor pressure, and detrusor pressure at maximum flow. A Mann-Whitney test was used for resistance as this variable was not normally distributed. A Chi-squared tests were used for the categorical variable straining.

No significant differences were found for any of the outcome measures.

Overall, patients with de novo detrusor instability and patients with post-operative symptoms of urge incontinence were not found to be urodynamically obstructed, as compared to other patients.
The natural history of *de novo* detrusor instability was studied in 56 women with stable bladders pre-operatively. One woman who declined post-operative cystometry is included only for analysis of subjective data.

*Twelve women (21.4%) were found to have de novo detrusor instability at three months and constitute the subject of investigation on the natural history of this condition.*

The remaining 44 women with stable bladders at three months are reported on separately in relation to possible late development of detrusor instability.

Overall, when considering together patients with stable and unstable bladders, urge incontinence was reported by 25 women at three months (44.6%) and by 15 women at one year (26.7%).

The incidence of detrusor instability (irrespective of symptoms) was 21.4% at three months and 12.7% at one year.

The incidence of *symptomatic* detrusor instability was 14.2% at three months and 9% at one year.

While some women with post-operative symptoms of urge incontinence were prescribed anticholinergics, this is not thought to have altered findings, as care was taken to stop treatment prior to testing, and in no cases was treatment prolonged.
A) THE HISTORY OF DE NOVO DETRUSOR INSTABILITY

All 12 women with de novo detrusor instability underwent clinical and urodynamic assessment one year after surgery. Their symptoms (as reported in King’s Health Questionnaire) and urodynamic findings before colposuspension and three and twelve months post-operatively are detailed in table 53.

**Table 53. Symptoms and urodynamic findings before colposuspension and three and twelve months afterwards in 12 women with de novo detrusor instability*.**

<table>
<thead>
<tr>
<th></th>
<th>Before colposuspension</th>
<th>3 months</th>
<th>12 months</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>3 -</td>
<td>0 +</td>
<td>1 +</td>
</tr>
<tr>
<td>2</td>
<td>1 -</td>
<td>1 +</td>
<td>0 -</td>
</tr>
<tr>
<td>3</td>
<td>2 -</td>
<td>2 +</td>
<td>3 -</td>
</tr>
<tr>
<td>4</td>
<td>1 -</td>
<td>2 +</td>
<td>2 +</td>
</tr>
<tr>
<td>5</td>
<td>0 -</td>
<td>0 +</td>
<td>0 +</td>
</tr>
<tr>
<td>6</td>
<td>2 -</td>
<td>0 +</td>
<td>0 -</td>
</tr>
<tr>
<td>7</td>
<td>1 -</td>
<td>0 +</td>
<td>0 -</td>
</tr>
<tr>
<td>8</td>
<td>3 -</td>
<td>2 +</td>
<td>3 +</td>
</tr>
<tr>
<td>9</td>
<td>2 -</td>
<td>1 +</td>
<td>0 -</td>
</tr>
<tr>
<td>10</td>
<td>0 -</td>
<td>1 +</td>
<td>0 -</td>
</tr>
<tr>
<td>11</td>
<td>2 -</td>
<td>2 +</td>
<td>0 +</td>
</tr>
<tr>
<td>12</td>
<td>1 -</td>
<td>1 +</td>
<td>1 -</td>
</tr>
</tbody>
</table>

* 0 = Absence of symptoms of urge incontinence
  1 = Mild urge incontinence
  2 = Moderate urge incontinence
  3 = Severe urge incontinence

Plus and minus signs = Presence (+) or absence (-) of detrusor instability

At three months, eight of 12 women with de novo detrusor instability were symptomatic (66.6%). Symptoms were mild in four women and moderate in four.

At one year, **objective resolution** of de novo detrusor instability occurred in seven women (58.3%), and **subjective resolution** occurred in four of eight symptomatic women (50%). The other four were either the same or worse. Of four women with asymptomatic de novo detrusor instability at three months, one had become symptomatic at one year. Overall five
of 12 women (41.6%) with \textit{de novo} detrusor instability at three months had symptoms of urge incontinence at one year (three of them with persisting unstable bladders).

The impact of symptomatic \textit{de novo} detrusor instability on \textbf{quality of life} issues, as compared to symptoms before surgery, is detailed in table 54. Data on other quality of life domains (as reported in King’s Health Questionnaire) is shown in appendix 15.

\textbf{Table 54.} The impact of urinary symptoms on quality of life in patients with symptomatic \textit{de novo} detrusor instability (patients No 5, 6 and 7 not included as asymptomatic). Severity of incontinence is quantified subjectively on a scale of 0 to 100 (King’s Health Questionnaire, appendix 12). Other urinary symptoms (voiding dysfunction or persistent GSI) which may affect quality of life are shown. Means are obtained using quantitative data illustrated in appendix 15.

<table>
<thead>
<tr>
<th>No</th>
<th>Impact of all urinary symptoms on quality of life*</th>
<th>Severity of incontinence</th>
<th>Other factors</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre-op</td>
<td>3m</td>
<td>12m</td>
</tr>
<tr>
<td>1</td>
<td>A lot</td>
<td>A little</td>
<td>Moderate 75</td>
</tr>
<tr>
<td>2</td>
<td>Moderate</td>
<td>A little</td>
<td>None</td>
</tr>
<tr>
<td>3</td>
<td>Moderate</td>
<td>Moderate</td>
<td>Moderate 58.3</td>
</tr>
<tr>
<td>4</td>
<td>A lot</td>
<td>A little</td>
<td>A little 66.6</td>
</tr>
<tr>
<td>5</td>
<td>A lot</td>
<td>None</td>
<td>A little 75</td>
</tr>
<tr>
<td>6</td>
<td>Moderate</td>
<td>A little</td>
<td>A little 66.6</td>
</tr>
<tr>
<td>7</td>
<td>A lot</td>
<td>A little</td>
<td>A little 91.6</td>
</tr>
<tr>
<td>8</td>
<td>A lot</td>
<td>None</td>
<td>A little 66.6</td>
</tr>
<tr>
<td>9</td>
<td>Moderate</td>
<td>A little</td>
<td>None 91.6</td>
</tr>
<tr>
<td>Mean</td>
<td>81.4</td>
<td>33.3</td>
<td>29.6</td>
</tr>
</tbody>
</table>

* King’s Health Questionnaire (how much do you think your bladder problem affects your quality of life?).

The impact of urinary symptoms on quality of life three months and one year after colposuspension in women with symptomatic \textit{de novo} detrusor instability was reported as improved by all women except one. In all women severity scores were lower. Improvement was reported also between three months and one year, as shown by mean scores (although in individual cases slight worsening occurred) (table 54).
Forty-three of 44 women with stable bladders at three months underwent clinical and urodynamic assessment one year after surgery. One patient declined to attend but was interviewed over the phone.

At three months 17 women had symptoms of urge incontinence despite having stable bladders (38.6%). Symptoms were mild in 13 cases, moderate in one and severe in three.

At one year two more women were found to have unstable bladders and both had moderate symptoms of urge incontinence (one had been symptomatic also at three months). Eight of the remaining 41 women with persisting stable bladders had symptoms of urge incontinence (19.5%). Symptoms were mild in seven cases and moderate in one (figure 19).

![Diagram](image)

**Fig. 19.** History of women with stable bladders three months after colposuspension. Symptoms of urge incontinence (UI) and late development of detrusor instability.

In summary, this suggests that at one year, the majority of these patients have an improvement in urge incontinence.
C) SPECIAL CASES

The three women who complained of severe urge incontinence at three months and had stable bladders constitute special cases, which were difficult to classify:

Case 1: This patient had transient and severe urge incontinence for two months after surgery that had settled by the time she had cystometry at three months. She remained asymptomatic afterwards and declined to have urodynamics at one year. She was started on antidepressants by her GP.

Case 2: This patient went on to develop detrusor instability at one year. Urge incontinence at one year was described as moderate. This patient did not experience an improvement in quality of life as compared to before surgery (although the impact of her urinary symptoms on her quality of life was low). However, considerably lower incontinence severity scores were reported post-operatively.

Case 3: This patient complained of severe urge incontinence at three months and symptoms were moderate at one year. No stress incontinence. No unconscious leakage or enuresis. Cystometry showed a stable bladder at three months and one year, and this was confirmed by ambulatory urodynamics. Extended pad test confirmed urinary leakage. Interestingly, genuine stress incontinence was demonstrated at three months but not at one year, despite repeated provocation. Cystoscopy was negative. A urogram was normal (no evidence of fistula). Surprisingly, the impact of her symptoms on quality of life was low (as it had been before colposuspension). However, she had high incontinence severity scores which did not improve after surgery.
Retrospective [15-17] and prospective [18-20] studies indicate that the colposuspension operation for genuine stress incontinence has a high success rate of up to 85-90%, and that the beneficial effect tends to be long-lasting [17]. As a consequence, this operation is often taken as the ‘gold standard’ against which new procedures are measured. Despite the high success rate, complications occur which may adversely affect quality of life. A recent survey on the impact of incontinence surgery on morbidity found that only 68% were satisfied and would recommend surgery to a friend, while 26% reported worsening of mental health, and 11% reported feeling worse than before surgery [41]. The development of new problems after surgery or the worsening of pre-existing conditions may lead to a deterioration in quality of life despite curing genuine stress incontinence.

Post-operative voiding dysfunction and detrusor instability have been reported to occur in a mean of 12.5% and 9.6% of patients respectively [15]. Due to their frequency, their potential to become chronic, and the difficulties of treatment, they are the complications more likely to adversely affect quality of life in the long term.

Most studies on incontinence surgery focus on surgical technique and outcome relates mostly to success or failure in the treatment of genuine stress incontinence. No prospective studies to date have specifically addressed the issue of morbidity after incontinence surgery. There is therefore no reliable data on the incidence, natural history, and impact on quality of life of complications such as voiding dysfunction and detrusor instability.

The causes of voiding dysfunction and detrusor instability are largely unknown. A number of retrospective studies have looked at pre-operative clinical and urodynamic risk factors for the development of voiding dysfunction (chapter 6, section 5) and detrusor instability (chapter 7, section 2). Results are generally inconclusive. Surgical risk factors such as amount of bladder neck elevation and urethral compression have been postulated.
but little researched, and remain unproven (chapter 6, sections 5a and 5b). The causation of these complications is likely to be multifactorial, but no prospective studies to date have investigated possible pre-operative, operative and post-operative variables all together.

This study has investigated prospectively voiding dysfunction and detrusor instability after the operation of colposuspension. Complications were identified and followed-up objectively by means of serial urodynamic studies after surgery. The impact of these complications on quality of life was assessed using specific questionnaires. The development of complications was correlated to pre-operative variables and to a number of anatomical and functional changes caused by surgery. Anatomical changes were identified mainly by imaging the bladder neck with MRI, while functional changes were identified by means of urodynamic studies.

Results in relation to voiding dysfunction and detrusor instability will be discussed separately.
DISCUSSION:

1- THE CAUSES OF VOIDING DYSFUNCTION

As expected, voiding dysfunction after colposuspension was found to be multifactorial, related partly to intrinsic individual features (age and detrusor contractility) and partly to surgical technique (amount of bladder neck elevation and urethral compression). Past history of hysterectomy, previous bladder neck surgery, menopausal state, weight, psychological factors, use of epidural for post-operative analgesia, bowel function, bacteriuria and post-operative pain, were not found to be associated with post-operative voiding function.

Increasing age has already been reported as a cause of voiding dysfunction after colposuspension [27,303,304,309,342]. After multiple regression analysis, we found increasing age as the most important predictive factor of the day of catheter removal. Despite the lack of large population based studies on normal women, and the absence of longitudinal data, there is growing evidence that the incidence of voiding dysfunction in women increases with age and that the process of ageing may decrease detrusor contractility and increase urethral rigidity (chapter 6, section 5e). Increased urethral rigidity may increase the likelihood of causing obstruction during anti-incontinence surgery, and reduced detrusor contractility may impair the ability to cope with any obstruction. It is not clear whether the menopause has an effect on voiding which is distinct from age (chapter 6, section 5f). There are no studies showing that the menopause per se has a deleterious effect on voiding function and there are no reports suggesting that the menopause constitutes a risk factor for postoperative voiding dysfunction, which is independent of age. In accordance with observations made by others [27], we did not find menopausal status to be a significant independent risk factor.
Detrusor contractility, in combination with urethral resistance, is a key factor in the process of bladder emptying. The operation of colposuspension introduces an element of obstruction, as shown by the significant increase in detrusor pressure and decrease in peak flow rate which occurs post-operatively (chapter 5, section 2). This has also been confirmed by pressure/flow data in this study (chapter 9, section 10a). An intrinsically weak detrusor may be unable to cope with even the slightest increase in outflow resistance.

**Poor detrusor contractility** (and risk of post-operative voiding dysfunction) may be detected pre-operatively using pressure-flow studies and observing voiding patterns.

Pressure-flow studies are commonly used in an attempt to predict subjects at risk of voiding dysfunction due to an intrinsically weak detrusor. However, the predictive value of urodynamic variables of pressure and flow is controversial (chapter 6, section 5c).

In agreement with some studies [21,29,32], but in disagreement with others [27,304], we found the peak flow rate to be predictive of voiding performance (day of catheter removal). It is of interest that when using the Liverpool nomograms [179], 28.5% of women in our study had pre-operative values below the 5th centile, thus supporting the view of those who consider women with genuine stress incontinence to have lower peak flow rates than normal women [286] (chapter 6, section 2). Although the cause is not known, lower peak flow rates could mean that women with GSI suffer a degree of voiding impairment.

We did not find detrusor pressure during voiding (maximum detrusor pressure and detrusor pressure at maximum flow) to be associated with post-operative voiding performance, despite the presence of a considerable number of patients with low detrusor voiding pressures (p$_{\text{det, max}}$ < 15 cmH$_2$O in 19.6% of women). This finding is in agreement with some studies [27,304], but in disagreement with others [24,200,290].
We found urethral resistance to be predictive of voiding performance, but this variable was associated with the day of catheter removal only after performing regression analysis in the group of women who had bladder neck elevation beyond the ‘safe’ level of 26 mm. Previous studies had found urethral resistance to be predictive of ‘late’ but not ‘early’ post-operative voiding dysfunction [24].

The inconsistent predictive value of pressure-flow studies is likely to be due to methodological differences. In most studies, patients are grouped as having ‘low detrusor pressure’ as opposed to ‘normal detrusor pressure’, on the basis of whether they void with pressures of less than 15 cmH2O [24,200,290], or less than 20 cmH2O [27,32]. Grouping of patients using arbitrary values may be inappropriate, as normal values of voiding pressure have not been defined. While studies of normal continent women suggest that voiding always occurs in the presence of a detrusor contraction greater than 15 cmH2O (chapter 6, section 1), it has been shown that many women with stress incontinence void without a detrusor contraction (chapter 6, section 2). Also rigid definitions of voiding dysfunction result in arbitrary grouping of patients. As a consequence, a patient that resumes spontaneous voiding eight days after surgery would be classed as normal in one study [21] and abnormal in another study [290]. In this study the use of definitions and arbitrary grouping of patients were avoided. Also, for the first time, the predictive value of urodynamic variables was assessed in combination with other pre-operative and operative variables using regression analysis.

Voiding patterns in women vary. While normal women void with a detrusor contraction, many women with genuine stress incontinence have been shown to void by pelvic relaxation without a detrusor pressure rise (chapter 6, section 2). In addition to pelvic relaxation and detrusor contraction, women may perform an unconscious but sustained Valsalva manoeuvre during voiding. This has been shown to occur in 66-73% of normal
women [272,277] and it occurs more frequently in women with stress incontinence, perhaps as a compensatory mechanism for their weaker detrusor contraction [288].

A combination of voiding features (voiding with and without a detrusor contraction and with or without abdominal straining) has been used to predict post-operative voiding dysfunction, but studies have been inconsistent (chapter 6, section 5c).

In this study, 50.6% of women voided with a detrusor contraction alone, 31.1% voided with a detrusor contraction and abdominal straining, 10.3% voided without a detrusor contraction and with abdominal straining, and 7.7% voided without a detrusor contraction and without abdominal straining (pelvic relaxation alone). These proportions are similar to those reported by others [289]. In agreement with Milani et al [289], but in disagreement with other studies [22,318], the type of pre-operative voiding pattern was not found to be predictive of post-operative voiding performance.

The inconsistency of these findings may be partly explained by the lack of a clear definition of the concept of ‘voiding with abdominal straining’. Most studies define abdominal straining simply as an abdominal pressure rise greater than 10 cmH2O [22,24,289]. The number, size, and timing of these pressure rises may need to be defined in order to be meaningful, as a single pressure rise at the beginning or end of voiding may not have the same importance as repeated straining throughout voiding. We defined abdominal straining very strictly as a rise in intra-abdominal pressure of at least 10 cmH2O occurring throughout voiding (either intermittently or continuously). When analysing this variable in isolation (irrespective of whether there was a detrusor contraction), an association with poor voiding performance was found.

Detrusor contractility has been assessed by measuring the detrusor pressure that is generated when a voluntary contraction of the external sphincter interrupts the flow, the so-called ‘stop test’ [319]. However, clinical studies have not found the ‘stop test’ predictive-
of post-operative voiding dysfunction [32]. This may be due to reflex detrusor inhibition induced by voluntary striated sphincter activation and to inability of some patients to stop the flow of urine due to a weak pelvic floor or the presence of detrusor instability. The presence of an ‘after-contraction’ at the end of micturition has been shown to occur in 60% of normal women [272] and may be considered a form of physiological ‘stop test’. To our knowledge, this observation has never been assessed in relation to post-operative voiding dysfunction. An after contraction was seen towards the end of voiding in 54.5% of our patients (mean magnitude 75 cmH2O, range 24-230 cmH2O). We found the absence (and decreasing intensity) of after-contractions to be weakly correlated with a poorer voiding performance. However this variable was not found to have predictive value after multiple regression analysis.

Enhanced detrusor contractility has been reported in women with mixed incontinence as compared to women with pure genuine stress incontinence [404]. The presence of a coexistent overactive bladder could in theory be protective in relation to voiding dysfunction. However, women with mixed genuine stress incontinence and detrusor instability were not found to differ from women with pure genuine stress incontinence in relation to post-operative voiding performance.

The overall evidence from our study suggests that intrinsically poor detrusor contractility contributes to post-operative voiding dysfunction, although its role may have been overstated in the past. Women with genuine stress incontinence void with significantly lower detrusor pressures than normal continent women [277]. Also, in contrast to normal women who invariably void with a detrusor contraction (with or without abdominal straining), up to 11-15% of women with genuine stress incontinence have been shown to void without a contraction [277,289]. Although this may be due to a lower mean urethral
closure pressure, women with genuine stress incontinence may have a real impairment of detrusor muscle function and may be intrinsically predisposed to voiding dysfunction.

The development of new methods to assess detrusor contractility may help to improve the prediction of voiding dysfunction. The detrusor shortening velocity [320], which reflects the speed of detrusor muscle shortening, aims to assess detrusor function independently of urethral resistance. It can be obtained using a mathematical formula based on urodynamic parameters, which include the peak flow rate and the maximum detrusor pressure in the absence of flow (recorded prior to voiding or after the stop-flow test). A low detrusor muscle shortening velocity has been found to be associated with post-operative voiding dysfunction [302].

A continuous occlusion test has been proposed as a measure of detrusor contractility [321]. An isovolumetric detrusor contraction is obtained by occluding the outlet with a balloon catheter positioned at the bladder neck before the initiation of a voiding contraction. Simultaneous electromyography has shown that detrusor inhibition due to reflex sphincter contraction does not occur. This test has not yet been applied to the prediction of post-operative voiding dysfunction.

In the immediate post-operative period, many reversible factors may contribute to voiding dysfunction [306]. Atropine and other anticholinergic anaesthetic reversal agents (some with a half-life of three to four days) may reduce detrusor contractility, bladder overfilling may depress detrusor contractility, opiates may reduce bladder sensation, and pain may inhibit perineal relaxation. In addition, bruising and oedema might depress bladder contractility and cause temporary obstruction.

Whether **temporary detrusor failure** occurs in the immediate post-operative period is not known, as no pressure-flow studies have been performed in the immediate post-operative period after the operation of colposuspension. Drugs have been extensively used in the past
to enhance detrusor contractility in the belief that such a phenomenon occurs, with disappointing results (chapter 6, section 8). Urodynamic studies performed in the postoperative period after endoscopic bladder neck suspensions do not show detrusor failure and are more in keeping with an obstructive mechanism [322].

Our own urodynamic studies four days after colposuspension have shown that nine women (12.3%) had a detrusor pressure of less than 15 cmH2O, with all of them having had values of 15 cmH2O or greater prior to colposuspension. On the other hand, 64.3% of women had a detrusor pressure rise greater than 35 cmH2O on day four, which suggests that obstruction (as opposed to poor detrusor function) is more relevant in the immediate postoperative period [359].

While decreasing detrusor pressure values four days after colposuspension were found to be correlated with a poorer voiding performance, this association disappeared after multiple regression analysis. None of the women with poor detrusor pressure values in the immediate post-operative period required a catheter for more than 19 days (range 8-19) (chapter 9, table 24). Thus temporary detrusor failure in the post-operative period occurs but is uncommon and does not significantly impair post-operative voiding function.

Excessive elevation of the bladder neck and proximal urethra is commonly thought to be the main cause of voiding dysfunction after bladder neck suspension procedures [28,36,269,307]. It has been suggested that overelevation causes voiding dysfunction due to obstruction [36] or interference with the normal voiding mechanisms that include bladder neck descent at the commencement of voiding [54]. However, there is no clear evidence to support the theory that overelevation causes voiding dysfunction.

Dwyer and Stanton [29] assessed elevation subjectively during surgery by observing the degree of approximation of paravaginal fascia to the ilio-pectineal ligament (amount of
suture 'bow-stringing') and were unable to correlate the amount of elevation to the likelihood of post-operative voiding dysfunction. The subjective assessment of elevation during surgery may be inaccurate as the bladder neck is not visible and no account is taken of bladder neck position prior to elevation.

We made an attempt to make this assessment more accurately by expressing it in millimetres (to the nearest multiple of five). However, we were also unable to correlate this measure of elevation with post-operative voiding performance. In addition, measures of suture 'bow-stringing' did not correlate with the 'gold standard' MRI measures of elevation.

Direct assessment of elevation using videocystourethrography has been performed [21,30], and no correlation was found between amount of bladder neck elevation and voiding dysfunction (chapter 6, section 5a). However these studies cannot be considered conclusive, as reproducibility studies of measures of elevation were not included. Also, the use of a lateral bone landmark (such as the ischial tuberosity) in relation to a medial structure (such as the bladder neck), in the study by Dundas et al [30], may result in a significant loss of accuracy when measuring bladder neck elevation: a slight lateral tilt of the pelvis is likely to cause a large variation in measures of elevation.

We have used MRI to measure bladder neck elevation objectively. Unlike radiography, MRI is radiation free and does not require instrumentation. Although no studies have formally compared MRI to radiography and ultrasonography in relation to imaging of the bladder neck, the high quality of image obtained suggests that MRI may be more reproducible and less operator dependent. Our reproducibility studies have shown very high and positive correlations. In this study, a strong association between elevation and post-operative voiding performance was found. There was also a 'threshold' elevation of
around 26 mm below which prolonged catheterisation did not occur. The implications of this finding in relation to prevention of voiding dysfunction will be discussed later.

Elevation was found to be an important predictive factor of voiding dysfunction after interim results on 42 women. It could be argued that this knowledge could have resulted in bias in the second part of the study, with surgeons unconsciously performing limited elevation. However, the mean elevation before and after interim studies was the same.

One of the questions is why elevation is so variable despite the use of the same operative technique, with women having bladder neck elevation during colposuspension ranging from 10 to 48 mm. The main determining factor of the amount of bladder neck elevation achieved by surgery is the position of the bladder neck before surgery: the lower the position, the higher the elevation. This clearly reflects the amount of stretching sustained by paravaginal fascia before surgery (which may depend on collagen properties).

**Urethral compression** may presumably occur as a consequence of misplacement of sutures, causing kinking of the urethra and peri-urethral scarring. When assessing selected groups of women with post-operative voiding dysfunction after incontinence surgery using VCU and / or urethroscopy, some degree of urethral deformity or distortion has been reported in 11-45% of these patients [36,307,310].

Subtle differences in the technique of colposuspension may lead to different degrees of urethral compression. The Marshall-Marchetti-Krantz procedure, where stitches are placed in close proximity to the urethra, has been found to induce a later resumption of spontaneous voiding as compared to the Burch colposuspension [201]. Tanagho’s modification of the Burch colposuspension, where stitches are deliberately placed as far away from the urethra as possible (chapter 5, section 1), has been claimed to lead to earlier
resumption of voiding without affecting success rates [31], but so far there is no evidence to support this claim.

To date, no objective measures have been proposed to quantify subtle degrees of urethral compression and no studies have assessed urethral compression in relation to post-operative voiding dysfunction.

In this study, an attempt was made to do this by measuring: (1) the distance between the back of the pubic bone and the bladder neck using MRI after surgery, (2) the space between the back of the pubic bone and the urethra after tying the sutures using two Hegar dilators during surgery (similarly to Tanagho who used two fingers instead), and (3) the distance between the medial stitches during surgery (at paravaginal and pectineal insertions separately). All measures were highly reproducible. However, the three measures of urethral compression were not comparable. MRI measures were not correlated to the intra-operative measures, suggesting that they measured urethral compression differently. The distance between the back of the pubic bone and the urethra as measured with MRI is likely to represent 'anterior' urethral compression (i.e. compression against the pubic bone). Measures with Hegars and the distance between medial stitches (which were correlated to each other) probably represent 'lateral' compression (i.e. direct pressure exercised by the stitches laterally).

We found urethral compression to be predictive of post-operative voiding performance, as shown by MRI measures ('anterior' compression) and also by Hegar measures ('lateral' compression). While the distance between the medial stitches (both at ilio-pectineal and paravaginal insertion) was not found to be predictive of voiding dysfunction, the correlation between Hegars and the distance between the medial stitches suggests that a
more lateral placement of the stitches should leave more space for larger Hegars. This finding lends some support to Tanagho’s claim.

The relevance of these findings in relation to prevention of voiding dysfunction will be discussed later.

Differences in urethral rigidity may predispose some women to urethral compression and obstruction. Attempts have been made to quantify the elastic properties of the urethra by using complex mathematical models based on pressure-flow curves [315], and by measuring urethral distensibility mechanically using balloons [316] or probes of progressively increasing diameter [317]. These methods have yet to find a useful clinical application and have never been used for the prediction of post-operative voiding dysfunction.

It has been suggested that hysterectomy may affect voiding function, but clinical and urodynamic studies have found no evidence of increased voiding dysfunction after vaginal hysterectomy and after abdominal hysterectomy (chapter 6, section 5g). In agreement with other studies [27], we found no difference in the incidence of voiding dysfunction after colposuspension in patients with a past history of hysterectomy.

It is controversial whether a past history of bladder neck surgery predisposes to voiding dysfunction after colposuspension. Surgical dissection during incontinence surgery has the potential to cause increased urethral rigidity and nerve damage. Considering that the greater concentration of terminal nerves has been shown to be in the 4 o’clock and 8 o’clock positions [49,389], vaginal dissection should be more likely to cause nerve damage than abdominal dissection [390]. Published reports so far have been inconsistent (chapter 6, section 5g). We found no increased risk of post-operative voiding dysfunction in women
with a past history of bladder neck surgery. However most women had a history of previous anterior colporrhaphy. Only two women had a history of previous colposuspension and no conclusions can be made on whether previous retropubic surgery constitutes a risk factor.

**Psychosocial disturbances** have been reported to cause or to contribute to voiding dysfunction [269,292,323-325]. Urinary retention may develop secondary to centrally mediated, unconscious inhibition of either detrusor contraction or pelvic floor relaxation or both [323]. In a small preliminary study, Diazepam was found to be beneficial in preventing voiding delay after colposuspension [326]. To date, no prospective studies using psychological measures have looked at psychological factors in relation to post-operative voiding dysfunction. Our study population had an incidence of anxiety and depression comparable to that of patients attending other medical clinics [421]. We were unable to show an association between anxiety and depression, whether pre or post-operative (as measured with the HAD questionnaire), and the development of post-operative voiding dysfunction.

We have also assessed various other factors, which may contribute to voiding dysfunction after colposuspension. It is commonly thought that constipation aggravates voiding dysfunction, but there was no correlation between post-operative bowel and bladder emptying. Pain can be expected to inhibit micturition by causing spasm of the pelvic floor, but no correlation was found between pain scores using the McGill scale and post-operative voiding performance. Commonly bladder emptying is depressed during epidural anaesthesia, and the effect may persist for several hours afterwards. There was no correlation between use of epidural for post-operative analgesia and voiding performance. The presence of infection in the urine may aggravate voiding dysfunction. Significant
bacteriuria was found in 12.9% of women on day four despite the use of prophylactic antibiotics. There was no detectable effect on voiding function.

While scarring or nerve damage due to previous hysterectomy or bladder neck surgery, psychological factors, constipation, pain, epidural anaesthesia, or urinary tract infection, may be aggravating factors in individual cases, their role (if any), is likely to be secondary.
CAN VOIDING DYSFUNCTION BE PREVENTED?

Of the factors found to be predictive of post-operative voiding dysfunction, only urethral compression and bladder neck overerelevation are susceptible to modification and therefore amenable to prevention.

The finding that increasing Hegar size is associated with better voiding performance, and that the distance between the medial stitches is correlated to Hegar size, has some practical implications in relation to prevention of post-operative voiding dysfunction. A more lateral placement of stitches should lead to more free space around the urethra, thus leaving room (after tying the sutures) for the insertion of larger Hegars (or two fingers) between urethra and the medial stitches, thus supporting Tanagho’s suggestion [31]. This should reduce the risk of ‘lateral’ urethral compression.

However, there was no correlation between MRI measures of urethral compression (distance bladder neck to back of pubic bone) and any of the intra-operative measures. It is therefore not possible to reduce the risk of ‘anterior’ urethral compression, which in this study was the most important compression factor. Placing sutures more laterally might only make a modest impact on the incidence of post-operative voiding dysfunction.

The finding that prolonged catheterisation is unlikely in women with bladder neck elevation of 26 mm or less at colposuspension has more important implications in relation to the prevention of voiding dysfunction. Should the bladder neck be elevated by 26 mm or less (‘limited elevation’ colposuspension) in all women? The principle of ‘limited elevation’ is justifiable only if it does not decrease the cure rate for GSI. It is only practical if elevation can be measured at the time of surgery.
The amount of bladder neck elevation required to achieve successful treatment of genuine stress incontinence is not known. The position of the bladder neck in relation to the pubic bone following surgery has been studied in relation to success and failure, but studies have shown conflicting results (chapter 5, section 2). Fixation of the bladder neck behind the pubic bone is a recognised aim of the colposuspension operation, and may be more important than elevation as a determinant of success. This is supported by DeLancey’s anatomical studies that suggest that continence depends on the integrity of the hammock-like layer that stabilises the urethra [54]. The importance of bladder neck fixation for the successful treatment of genuine stress incontinence is also highlighted by a number of studies that have assessed post-operative bladder neck mobility in relation to success and failure [19, 125, 128, 222, 224] (chapter 5, section 2).

The problems of whether bladder neck elevation is more important than fixation, and how much elevation is needed to achieve fixation, have not been specifically addressed by our study. However, it was important to establish whether there was a correlation between amount of bladder neck elevation and likelihood of success, and whether ‘limited’ bladder neck elevation (of 26 mm or less) has a similar success rate to that associated with elevation greater than 26 mm. Since there is no standardised definition of ‘success’ or ‘failure’ after incontinence surgery, the definitions used in our study were arbitrary and similar to those subsequently suggested by the ICS.

Subjective ‘success’ was defined as absence of symptoms of stress incontinence, and objective ‘success’ as absence of leakage during stress at cystometry. Women with ‘limited’ bladder neck elevation had an objective and subjective success rate comparable to that of women with bladder neck elevation greater than 26 mm. While there was no correlation between amount of elevation and objective outcome, women with symptoms of stress incontinence (subjective ‘failures’) had a significantly higher mean bladder neck elevation as compared to women without symptoms. Excessive elevation may actually be
detrimental to success (perhaps by increasing the tension and therefore the likelihood of ‘tearing’ of the stitches).

It is of interest that patients with ‘subjective’ and ‘objective’ failure were not the same. Only one patient with ‘subjective’ failure was also shown to be an ‘objective’ failure. This discrepancy has already been noted by others [424-426], and highlights the importance of assessing surgical outcomes using several measures. It is possible that our objective method of assessment (coughing while standing at capacity during cystometry) was unphysiological. Also, some women may mistake vaginal discharge or sweating as urinary incontinence.

The subjective assessment of suture ‘bow-stringing’ is commonly used by surgeons to assess empirically the amount of bladder neck elevation. This did not correlate with MRI measures of elevation. An alternative method for the objective intra-operative assessment of elevation is therefore needed.

Perineal ultrasound has been shown to be a reproducible method for the assessment of the bladder neck [118]. Intra-operative ultrasound has been suggested as a method to assess elevation during surgery [427], but no objective studies have been performed to correlate measures to outcome or complications. A pilot study has shown good correlation between intra-operative measures of elevation using perineal ultrasound and MRI measures [428] (appendix 16). Perineal ultrasound seems to be a promising method for the objective measurement of bladder neck elevation during surgery, but requires further study.

Given that prolonged catheterisation is unlikely when the bladder neck is elevated by 26 mm or less, that the success rate in women with ‘limited’ elevation is not adversely affected, and that bladder neck elevation may be measured with reasonable accuracy using perineal ultrasound intra-operatively, voiding dysfunction after colposuspension could be
prevented by performing ‘limited elevation’ colposuspension in all patients. This however may be impractical. Not all surgeons may be familiar with ultrasound imaging of the bladder neck. Ultrasound equipment may not be available in the operating theatre. Intra-operative ultrasound is also likely to increase operative time.

In addition, no long-term data exist in relation to the success rate of ‘limited’ elevation colposuspension.

Further studies are needed to assess the reproducibility of perineal ultrasound in the intra-operative setting. Also a more accurate prediction of subjects at risk is needed. This could be based on the data presented in chapter 9 (section 7) (which can explain up to 43% of the variability in voiding performance in subjects with elevation greater than 26 mm), and perhaps also on new ways of assessing detrusor contractility (using the methods described above).

‘Limited elevation’ colposuspension could be offered to subjects deemed to be at risk, and surgery in these patients could be performed in tertiary centres with facilities and experience in bladder neck imaging with intra-operative ultrasound.
DISCUSSION:

2- THE NATURAL HISTORY OF VOIDING DYSFUNCTION

No studies have specifically addressed prospectively the natural history of voiding dysfunction after incontinence surgery, although it is common experience that a number of cases improve with time (appendix 1). The impact of prolonged voiding dysfunction on quality of life has also not been studied.

Studies that have used outcome measures and an operative technique, which are comparable to this investigation, report a mean number of days of catheterisation ranging from 6.3 to 15 days (chapter 6, table 7). The incidence of ‘early’ post-operative voiding dysfunction in our study was at the upper end of this range, with a mean number of days of catheterisation of 14.9 days. This may be due in part to cautious catheter management (appendix 8), and in part to surgical technique (as discussed before).

In our patients the incidence of voiding dysfunction at three months and at one year (defined as the presence of a residual urine volume of 100 ml or more in at least two occasions) was 7.7% and 2.5% respectively. Comparisons with others studies are difficult as ‘late’ voiding dysfunction is often not defined, or relies on low peak flow rates or symptoms alone (appendix 1). Using the same definition, in the short term, Milani et al [289] reported an incidence of 4.7% at one month, and Iosif [231] reported no cases at three months. In the longer term, Stanton et al [37] reported an incidence of 12% at 6-30 months, and Rizvi et al [241] reported an incidence of 3% at 2-3 years.

Six women in our study were found to have ‘late’ voiding dysfunction at three months. Only two of them had experienced difficulties since surgery requiring prolonged catheterisation and the need to learn CISC. The other four women had their catheters removed after 16-27 days (chapter 9, table 42). This supports the concept that some
patients with 'late' voiding dysfunction are different from those with 'early' dysfunction, as previously suggested [24]. The number of women with voiding dysfunction at three months was considered too low for meaningful statistical analysis exploring possible causative factors as compared to the remaining patients.

The cause of post-operative voiding dysfunction was obstruction (as defined by Massey and Abrams) [362] in five cases, but also the sixth patient had urodynamic parameters which were suggestive of obstruction (chapter 9, table 43). Colposuspension is an obstructive procedure (chapter 5), and this is confirmed by the mean pressure flow values at three and twelve months (chapter 9, table 39), and by the high number of women who could be classified as being 'obstructed' (18.6% and 12.5% at three and 12 months respectively).

In five patients with 'late' voiding dysfunction, bladder neck elevation was greater than 26 mm as measured with MRI, while in one case elevation was 26 mm. This suggests that elevation by 26 mm or less (which seems to prevent 'early' post-operative voiding dysfunction), may not be protective in relation to the development of 'late' voiding dysfunction. Progressive scarring is a possible explanation in these cases.

The natural history of voiding dysfunction after colposuspension, whether of 'early' or 'late' onset, was characterised in this small series by gradual improvement in two thirds of cases, with only two patients having residuals greater than 100 ml at one year (and only one requiring CISC). This improvement is mirrored by the changes of pressure/flow values between three months and one year, with an increase in mean peak flow rates and a decrease in mean detrusor pressures (chapter 9, table 39).
Women with 'pure' voiding dysfunction, requiring CISC but with no associated symptoms, enjoyed a considerable improvement in their quality of life. Rather than the need to self-catheterise, other factors seemed to cause poor quality of life in patients with voiding dysfunction. One patient developed recurrent urinary tract infections, while in another patient the pre-existent 'irritative' symptoms failed to improve. Both women were considered to have normal voiding at twelve months but it appears they still had problems. Whether their symptoms were due to 'borderline' voiding dysfunction is not known.

Current urodynamic definitions of 'voiding dysfunction' and 'obstruction' have limitations. The two women in this group whose quality of life remained significantly impaired when seen at one year, could not actually be urodynamically classified as having voiding dysfunction or obstruction. Cases of 'atypical' voiding dysfunction may also occur (as shown by our 'special case'), which are difficult to classify, but where quality of life does not improve and patients consider the operation to have been a 'failure', despite relief from stress incontinence.
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<td>• Voiding dysfunction occurs frequently after colposuspension but prolonged dysfunction is uncommon.</td>
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<td>• Patients who voided promptly after surgery may develop ‘late’ voiding dysfunction.</td>
</tr>
<tr>
<td>• The development of prolonged voiding dysfunction does not necessarily impair quality of life.</td>
</tr>
<tr>
<td>• Voiding dysfunction can be difficult to diagnose using present urodynamic criteria.</td>
</tr>
<tr>
<td>• Voiding dysfunction after colposuspension is multifactorial and is due to patient related factors (increasing age and poor pre-operative detrusor contractility) and to operative factors (increasing bladder neck elevation and urethral compression).</td>
</tr>
<tr>
<td>• Detrusor contractility may be transiently impaired after colposuspension but voiding performance is not significantly affected.</td>
</tr>
<tr>
<td>• Pre-operative urodynamic factors predictive of post-operative voiding dysfunction are a decreasing peak flow rate, increasing urethral resistance and the presence of abdominal straining during voiding.</td>
</tr>
<tr>
<td>• Prevention of voiding dysfunction may be possible, as there is a ‘threshold’ level of elevation (26 mm) below which prolonged voiding dysfunction does not occur.</td>
</tr>
<tr>
<td>• ‘Limited elevation’ colposuspension (26 mm or less) is not associated with an increased failure rate at one year.</td>
</tr>
</tbody>
</table>
DISCUSSION:

3- THE CAUSES OF DE NOVO DETRUSOR INSTABILITY

De novo detrusor instability, like voiding dysfunction, was also found to be multifactorial, related partly to individual features (age and previous bladder neck surgery) and partly to surgical technique (amount of bladder neck elevation and urethral compression).

The pre-operative detection of risk factors for the development of de novo detrusor instability has so far been unsuccessful [35].

It is not clear from previous reports whether a past history of bladder neck surgery predisposes to the development of de novo detrusor instability (chapter 7, section 2). A significant trend towards the development of de novo detrusor instability with an increasing number of previous bladder neck operations has been reported [34]. However, Sand et al [35] and Cardozo et al [391] did not find previous incontinence surgery to be a risk factor for the development of de novo detrusor instability.

In this study, a past history of bladder neck surgery (present in 19.6% of patients) was found to be associated with the development of de novo detrusor instability. All women with previous bladder neck surgery had previous anterior colporrhaphies. No conclusions can be drawn from this study in relation to a history of retropubic surgery (due to small numbers). Vaginal surgery, while not directly causing de novo detrusor instability, may have left patients vulnerable to subsequent damage. The greater concentration of terminal nerves around the bladder neck has been shown to be in the 4 o’clock and 8 o’clock positions [49,389], and these may be damaged during vaginal dissection [390]. Discrepancies with other reports may be due to the low number of cases of de novo detrusor instability in some reports (only five in the study by Sand et al [35]), or to
differences in patient selection (e.g. Cardozo et al [391] excluded from their study patients ‘at risk’ by using ambulatory urodynamics and measurements of bladder wall thickness).

In contrast to previous reports [34,35], we found increasing age to be associated with the development of de novo detrusor instability. This may be explained by the higher mean age in our study. Increasing age is reported by epidemiological studies to be a risk factor for the development of symptoms of urge incontinence [2]. It is however not clear why increasing age should increase the risk of post-operative detrusor instability. Older women might have latent nerve damage or might be more susceptible to nerve damage.

**Bladder outflow obstruction** has been associated with detrusor instability in men. The occurrence of instability in men has been correlated with the degree of obstruction [380], and relief of obstruction has resulted in reversion from unstable to stable detrusor behaviour in up to two thirds of patients [381]. The colposuspension operation has been shown to be an obstructive procedure (chapter 5), but pressure/flow studies before and after surgery do not support an obstructive theory for de novo detrusor instability in women [33] (chapter 7, section 2). Also in this study, no urodynamic evidence of obstruction was found when comparing women with and without de novo detrusor instability, and when comparing the amount of variation in pressure/flow parameters before and after surgery in both groups of patients.

However, surgical variables suggestive of obstruction such as increasing bladder neck elevation and increasing urethral compression (as measured with MRI), were found to be associated with the development of de novo detrusor instability.

In favour of an obstructive hypothesis are also the findings of Bump et al [387], who found higher and greater than 100% pressure transmission ratios in patients with post-operative detrusor instability as compared to patients without post-operative detrusor instability and patients with pure detrusor instability that did not undergo surgery. They thought that
surgery had overcorrected the urethral position and caused relative obstruction and postulated that post-operative detrusor instability is a condition that differs from idiopathic detrusor instability. In addition, clear evidence of obstruction (using VCU) has been reported in five of 17 women (29%) with post-operative detrusor instability [307], and surgical treatment of this condition with take-down procedures (and relief of obstruction) has been shown to be effective (chapter 7, section 6).

Obstructed women, unlike men, may not necessarily respond with a detrusor pressure rise (chapter 6, table 10), and pressure/flow studies may not be sensitive enough to detect minor degrees of obstruction in women.

We found the presence of a symptom of urge incontinence before surgery to be associated with urge incontinence post-operatively (but not predictive of de novo detrusor instability). It is of interest that 44.6% of women in our study reported symptoms of urge incontinence three months after colposuspension and that cystometry showed detrusor instability in only a third of them. The correlation of symptoms with cystometric findings is complex. Patients with isolated symptoms of urgency and urge incontinence have been found to have detrusor instability on cystometry in only 51-77% of cases [5,6,9], thus suggesting a high false negative rate. This may be even higher when symptoms occur after incontinence surgery, although the reason for this is not clear. The low incidence of detrusor instability in post-operative patients with ‘irritative’ symptoms has also been reported by others [23,36,311,313,364,365] (chapter 7, table 13).

It has been suggested that detrusor instability that develops after surgery may be present pre-operatively and that low outflow resistance may make pre-operative diagnosis difficult. The presence of unstable detrusor contractions on ambulatory urodynamics in patients with a stable bladder on static cystometry has been shown to predict the development of post-operative symptoms of detrusor instability, and the method has been proposed as a means
to diagnose occult pre-operative detrusor instability [384,385]. Increased bladder wall thickness measured on ultrasound may be a marker of detrusor instability [383] and a pre-operative bladder wall thickness greater than five millimetres has been shown to predict the development of irrititative symptoms after colposuspension [386]. It is therefore possible that a number of our patients with urge incontinence and stable bladders before and after surgery in fact had detrusor instability. Alternatively, women with urge incontinence and stable bladders may have an unstable urethra or may be affected by a different unknown pathology (e.g. sensory urgency leading to urinary incontinence).

The finding that women with higher pain scores in the immediate post-operative period (using the McGill questionnaire) were more likely to have symptoms of urge incontinence post-operatively (but not de novo detrusor instability) is difficult to explain, but might support the hypothesis of nerve trauma as a possible cause.

It is of interest that while voiding dysfunction and de novo detrusor instability share a number of causative factors (age, elevation and urethral compression), they do not seem to occur in the same patients, as shown by the lack of association between the number of days of catheterisation and the development of de novo detrusor instability. Only one patient developed both conditions. It is possible that individual bladders respond differently to obstruction and that individuals have intrinsically ‘overactive’ or ‘underactive’ bladders. Alternatively, the determining factor may be the degree of obstruction, with greater degrees resulting in voiding dysfunction and lesser degrees in de novo detrusor instability.
As *de novo* detrusor instability was found to be associated with the amount of bladder neck elevation and urethral compression, it might be expected that avoiding bladder neck overelevation and urethral compression, might help to reduce the incidence of this bothersome complication. It is however doubtful whether this can be achieved using the methods described in relation to voiding dysfunction.

As no cases of prolonged catheterisation were seen when the bladder neck was elevated by 26 mm or less, we have explored the possibility of whether this amount of elevation was also ‘safe’ in relation to the development of *de novo* detrusor instability. While the incidence of this complication was only 10% in the group of women with bladder neck elevation of 26 mm or less, as opposed to 21.8% in those with elevation greater than 26 mm, the difference was not found to be statistically significant. This may have been due to low numbers, as unfortunately three of the patients with *de novo* detrusor instability did not have MRI measures of elevation. A large prospective study is therefore required to test the hypothesis that a bladder neck elevation of 26 mm or less might help to reduce the incidence of this complication. A total of 230 women equally distributed into two groups (with bladder neck elevation of 26 mm or less or greater than 26 mm) would give a power of 80%.
DISCUSSION:

4- THE NATURAL HISTORY OF DE NOVO DETRUSOR INSTABILITY

No studies have specifically addressed prospectively the natural history of de novo detrusor instability. Only one cohort of patients with this condition has been reviewed [33,34]. Due to lack of urodynamic data, the only conclusion that can be drawn is that more than half of affected women are likely to be symptomatic in the long-term (chapter 7, section 4).

The impact of de novo detrusor instability on quality of life (as compared to pre-operative quality of life) has also not been studied, although detrusor instability is known to affect quality of life to a greater extent than genuine stress incontinence [378].

De novo detrusor instability occurred in 21.4% of our patients, at the upper end of the reported range of 3.2% to 27% (chapter 7, table 12). More than half (66.6%) of women with de novo detrusor instability were symptomatic. On review at one year, objective resolution had occurred in 58.3% of cases and symptoms had resolved in 50% of symptomatic cases. Quality of life reports (table 54) show that despite the development of de novo detrusor instability, the impact of symptoms on quality of life had improved after colposuspension in 88.8% of cases, and incontinence severity had improved in all cases. Scores at one year were also better than scores at three months, thus confirming a natural trend towards improvement.

Late cases of detrusor instability were seen at one year in two women who had stable bladders at three months. In these cases it is difficult to establish a clear link with surgery.
Symptoms of urge incontinence were also reported by 38.6% of women with stable bladders at three months and by 9.5% at one year. The high percentage of patients with post-operative irritative symptoms and stable bladders on cystometry has already been reported in the literature (chapter 7, table 13). This finding is difficult to explain and these women may be affected by a condition which is different from detrusor instability.

Although in most cases symptoms were mild and spontaneous resolution occurred, some of these women had persistent severe symptoms and posed difficult management problems (chapter 10, section 7e).
Key points

- *De novo* detrusor instability was diagnosed in 21.4% of women at three months follow-up.
- Despite the development of this complication, quality of life was improved in most cases.
- Objective and subjective resolution was seen in half of cases after one year.
- *De novo* detrusor instability is multifactorial and is due to patient related factors (increasing age and a past history of bladder neck surgery) and to operative factors (increasing bladder neck elevation and urethral compression).
- There was no urodynamic evidence of obstruction in women with *de novo* detrusor instability, but pressure-flow studies may not be sensitive enough to diagnose obstruction in women.
- The symptom of urge incontinence was reported by 38.6% of women with stable bladders at three months follow-up. Women with similar pre-operative symptoms are predisposed. Most cases resolved at one-year follow-up.
CONCLUSIONS

Prolonged catheterisation after colposuspension was common, with 69% of women requiring a catheter for more than seven days, and 28% for longer than 14 days. Improvement gradually occurred in most cases, with only 7.7% and 2.5% of them likely to need catheterisation at three months and one year respectively. Women distressed by their inability to void in the immediate post-operative period can therefore be reassured that improvement will gradually occur in most cases. Despite the low incidence of long-term voiding dysfunction, our study supports the concept that all women undergoing colposuspension should be counselled in relation to prolonged catheterisation and the need to perform CISC.

'Pure' voiding dysfunction (i.e. not associated with other symptoms) in women who performed CISC did not adversely affect quality of life. Cases of 'atypical' or 'borderline' voiding dysfunction occurred which were difficult to diagnose and classify; symptoms were difficult to explain and posed difficult management problems.

Voiding dysfunction after colposuspension was found to be multifactorial. It was caused by pre-existing patient-related factors, such as age and detrusor contractility (as assessed indirectly by reduced pre-operative peak flow rates, straining during voiding and increased urethral resistance). It was also caused by operative factors, such as the amount of bladder neck elevation and urethral compression.

There was a 'threshold' level of elevation of 26 mm, below which prolonged catheterisation did not occur. The success rate at one year in women with bladder neck elevation of 26 mm or less was equivalent to that of women with elevation greater than 26 mm.
De novo detrusor instability occurred in 21% of women at three months follow-up, and was symptomatic in 66% of cases. Objective and subjective resolution was seen in half of cases at one-year follow-up. Quality of life after colposuspension improved in most cases despite the development of this complication. Women who develop this complication may be reassured by these findings.

Symptoms of urge incontinence were reported by 38% of women with stable bladders postoperatively. Symptoms were usually mild and improved with time, but persistent and atypical cases occurred, where symptoms were difficult to explain, and which posed difficult management problems.

The frequency of post-operative symptoms of urge incontinence (whether or not associated with an unstable bladder) indicate that patients should be specifically warned during pre-operative counselling.

De novo detrusor instability was also found to be multifactorial, due to patient related factors (age and past history of bladder neck surgery) and to operative factors (amount of bladder neck elevation and urethral compression). While neurogenic factors may be present and some patients may have had undiagnosed detrusor instability, the operative factors (above) lend support to the possibility that de novo detrusor instability is due to obstruction, despite no urodynamic evidence.

Surgical treatment of voiding dysfunction and symptoms of urge incontinence (with and without detrusor instability) after colposuspension, by means of take-down procedures, should be undertaken only in exceptional cases, given the relatively benign course of these complications, and the uncertain outcome of these procedures (chapter 6, table 11, chapter 7, table 14).
FUTURE RESEARCH

No definite conclusions can be made on the long-term natural history of voiding dysfunction and *de novo* detrusor instability and their effects on quality of life, due to the low number of cases studied and limited follow-up. Larger cohorts of patients need to be evaluated over a longer period of time.

Due to low numbers, this study has not answered the question of whether prior suprapubic incontinence surgery constitutes a risk factor for the development of these complications.

The specific causes of 'prolonged' or 'late onset' voiding dysfunction remain unknown and a larger number of women with this complication need to be investigated. This may be difficult due to the infrequency of this condition.

Our findings open interesting research opportunities in relation to prediction and prevention of these complications, which still carry considerable short-term morbidity. A more accurate prediction of subjects at risk is needed and could be based on the data presented in this study. Further studies are needed to assess the value and reproducibility of perineal ultrasound in the intra-operative setting for the measurement of bladder neck elevation. A large prospective study is required to test the hypothesis that 'limited' bladder neck elevation (of less than 26 mm) might prevent voiding dysfunction and *de novo* detrusor instability.
APPENDIX 1. Reports on voiding dysfunction after colposuspension. Number of women undergoing surgery (n.), definitions (not available in report if not specified) and incidence.

<table>
<thead>
<tr>
<th>n.</th>
<th>Definitions and incidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Burch 1968 [39]</td>
<td>143 No long term subjective voiding difficulty</td>
</tr>
<tr>
<td>Morgan 1973 [73]</td>
<td>51 ‘Difficulty initiating voiding’: 24% ‘Persistent residual urine &gt; 100 ml’: 2%</td>
</tr>
<tr>
<td>Stanton et al 1976 [37]</td>
<td>40 Average time for spontaneous micturition: 14 days Range: 6-37 days Residual &gt; 100ml at follow-up (6-30 months): 12%</td>
</tr>
<tr>
<td>Stanton et al 1978 [21]</td>
<td>80 Difficulty in establishing normal micturition: 17% Voiding delay &gt; 21 days: 12%</td>
</tr>
<tr>
<td>Cowan &amp; Morgan 1979 [294]</td>
<td>77 Mean catheter requirement: 4.3 days Range: 2-20 days</td>
</tr>
<tr>
<td>Stanton &amp; Cardozo 1979 [191]</td>
<td>180 Average time for spontaneous micturition: 12.6 days</td>
</tr>
<tr>
<td>Dundas et al 1982 [30]</td>
<td>54 Post-operative Qmax &lt; 15ml/s: 37%</td>
</tr>
<tr>
<td>Mundy 1983 [18]</td>
<td>26 All patients voiding well within 18 days 12% had ‘low’ Qmax after 1 year</td>
</tr>
<tr>
<td>Hilton &amp; Stanton 1983 [199]</td>
<td>25 Qmax &lt; 15ml/s after 3 months: 36%</td>
</tr>
<tr>
<td>Iosif 1983 [231]</td>
<td>136 Residual &gt; 50 ml at 3 months: none</td>
</tr>
<tr>
<td>Bhatia &amp; Bergman 1984 [22]</td>
<td>30 Catheter required for &gt; than 7 days (residual &gt; 50ml): 20%</td>
</tr>
<tr>
<td>Gillon &amp; Stanton 1984 [38]</td>
<td>35 Mean catheter requirement (residual &gt; 100 ml): 15 days. Range: 4-35 days. No voiding difficulties at 3-5 year follow-up</td>
</tr>
<tr>
<td>Verges et al 1984 [295]</td>
<td>137 ‘Early’ (undefined) voiding delay: 1.4%</td>
</tr>
<tr>
<td>Iosif 1985 [296]</td>
<td>195 Catheter required for &gt; than 7 days: 27.6% Range 8-28 days</td>
</tr>
</tbody>
</table>
Bhatia & Bergman 1986 [290] 43 Catheter required for > than 7 days (residual > 50ml): 18.6%
Mean catheter requirement (residual > 50ml): 7 days
Range 3-24 days

Dwyer & Stanton 1986 [29] 187 Qmax < 15ml/s at 3 month: 37%
Qmax < 15ml/s at mean follow-up of 2.7 years: 20%

Lose et al 1987 [24] 80 Acute retention or residual of > 50mls after catheter removal
(3-8 days after surgery): 25%
70 Symptoms and ‘urodynamic evidence’ of poor flow at follow-up
(4-72 months): 20%
Both early and late dysfunction: 5%

Galloway et al 1987 [23] 50 ‘Prolonged’ voiding delay: 16%


Rizvi et al 1988 [241] 62 Inability to void on day 4 (when the catheter was clamped): 80%
59 ‘Low’ Qmax and residual > 100 ml at 2-3 year follow-up: 3%

van Geelen et al 1988 [214] 34 Micturition delayed by 3 or more days after catheter removal
(5-7 days after surgery): 5.8%

Korda et al 1989 [257] 174 Mean catheter requirement: 10 days
Range: 5-60
Catheter required > 10 days: 24%.
On permanent intermittent self catheterization: 1%

Bathia et al 1989 [200] 48 Catheter required > 7 days (residual > 50 ml): 20%

Mean catheter requirement: 4 days (range:1-7)

Eriksen et al 1990 [25] 91 Residual > 100ml on day 5: 15%
76 Residual > 100ml at 5 year follow-up: 3.9%

Milani et al 1991 [289] 129 Catheter required > 7 days (residual > 50ml): 30%
Mean catheter requirement: 6.3 days (range: 3-38 days)
Residual of > 100ml one month after surgery: 4.7%

Hosker et al 1991 [297] 22 No voiding difficulties at 3 months follow-up

Vinagre et al 1991 [298] 227 ‘Prolonged’ voiding delay: 3.9%

Guerinoni et al 1991 [216] 173 Mean catheter requirement: 9 days
Prolonged voiding delay requiring surgical intervention
(21 urethral dilatations, 1 take-down): 12.7%
114 ‘Late’ (at 3-11 year follow-up) voiding difficulties
(Qmax < 20ml/s and void lasting > 60s): 11.6%
<table>
<thead>
<tr>
<th>Study</th>
<th>Table Number</th>
<th>Findings &amp; Observations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kiilholma et al 1993 [192]</td>
<td>186</td>
<td>Mean catheter requirement: 6 days (range: 5-28 days)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>‘Early’ urinary retention: 13.9%</td>
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<tr>
<td></td>
<td></td>
<td>‘Mild’ (subjective) voiding difficulty at 1 year follow-up: 9%</td>
</tr>
<tr>
<td>Feyereisl et al 1994 [26]</td>
<td>87</td>
<td>‘Late’ (at 5-10 year follow-up) voiding difficulties: 4.6%</td>
</tr>
<tr>
<td>Colombo et al 1994 [201]</td>
<td>40</td>
<td>Mean catheter requirement: 8.5 days (range: 1-15)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Long lasting difficulties (subjective): 8%</td>
</tr>
<tr>
<td>Toniazzi et al 1994 [299]</td>
<td>29</td>
<td>Catheter required for more than 6 days: 45%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Catheter required for more than 10 days: 25%</td>
</tr>
<tr>
<td>Bergman &amp; Elia 1995 [20]</td>
<td>33</td>
<td>No voiding difficulties at 5 year follow-up</td>
</tr>
<tr>
<td>Chinegwundoh &amp; Nayeam 1995</td>
<td>61</td>
<td>‘Prolonged’ voiding delay: 20%</td>
</tr>
<tr>
<td>[300]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kremer &amp; Freeman 1995 [27]</td>
<td>51</td>
<td>Catheter required &gt; 7 days (residual &gt; 100ml): 43%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Catheter required &gt; 10 days: 23%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Mean catheter requirement (from day of clamping): 7.4 days</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Range: 2-32 days</td>
</tr>
<tr>
<td>Kinn 1995 [247]</td>
<td>153</td>
<td>Retention for more than 1 week: 4.5%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Residual &gt; 200ml at 2 month follow-up: none</td>
</tr>
<tr>
<td>Ross 1995 [234]</td>
<td>30</td>
<td>Mean time to void to completion: 3.2 days</td>
</tr>
<tr>
<td>Wang 1996 [301]</td>
<td>294</td>
<td>Voiding dysfunction (residual &gt; 20% of volume voided 3 weeks after catheter removal): 9.5%</td>
</tr>
<tr>
<td>Boos et al 1996 [302]</td>
<td>100</td>
<td>Voiding dysfunction (inability to void or residual &gt; 100ml on two occasions): 20%</td>
</tr>
<tr>
<td>Walters et al 1996 [303]</td>
<td>43</td>
<td>Mean catheter requirement: 7.1 days</td>
</tr>
<tr>
<td>Enzelberger et al 1996 [197]</td>
<td>36</td>
<td>Mean catheter requirement: 7 days</td>
</tr>
<tr>
<td>Heit et al 1997 [304]</td>
<td>70</td>
<td>Mean catheter requirement: 12.6 days</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Catheter required for &gt; 7 days (residual &gt; 100 ml): 48.6%</td>
</tr>
</tbody>
</table>
APPENDIX 2

Patient Consent Form

Name of Trial: Complications of Colposuspension for Urinary Incontinence

Have you read the Patient Information Sheet?  Yes / No

Have you had an opportunity to ask questions and discuss this study?  Yes / No

Have you received satisfactory answers to all your questions?  Yes / No

Have you received enough information about the study?  Yes / No

Who have you spoken to? Dr/Mr/Mrs

Do you understand that you are free to withdraw from the study:
- At any time
- Without having to give a reason for withdrawing
- And without affecting your future medical care  Yes / No

Do you agree to take part in this study?  Yes / No

Signed ................................................................. Date ........................................

(Name in block letters) .................................................................
We invite you to be part of a study that investigates the after effects of surgery for stress incontinence.

We know that the operation of Colposuspension has a high success rate. However, after surgery, some problems occasionally occur. Patients may find it difficult to pass water for a while and, very rarely, this inability persists.

Some patients may develop symptoms of frequency and urgency to pass water, due to overactivity of the bladder muscle; if these were already present before surgery, they may persist or worsen.

Some patients may become prone to develop a vaginal prolapse in later years.

Doctors are not sure of how often these problems occur and why they occur. This is what our study aims to investigate. The idea is to eventually find ways of preventing these complications from happening.

Should you wish to be part of the study, in addition to the routine investigations, you will be offered a pelvic MRI scan; this is non-invasive, does not require internal examinations,
and carries no risks. The scan will be repeated after surgery, in order to check the new position of the bladder neck.

Cystometry, which is the routine urodynamic investigation done before surgery, will also be repeated afterwards (once before discharge and twice after); this helps to understand the behaviour of the bladder muscle.

You will also be asked to complete questionnaires that provide information about levels of anxiety and quality of life before and after surgery.

All personal information will be treated as strictly confidential and will not be made publicly available.

We are inviting you to take part in this study, but if you do not wish to do so, then please feel free to refuse. This will not affect your further treatment in any way whatsoever, and certainly not incur our displeasure. Should you wish to withdraw at any time, then you are entitled to do so and again without any displeasure or penalty. You do not have to give a reason for your refusal to participate or for your wish to withdraw.

Finally, if you do wish to take part, you can ask for more information at any time; you will be given a telephone number to contact one of us directly should there be any concerns.

Yours faithfully,

Dr. Luigi Bombieri

Research Registrar to Mr. Freeman
Dear Doctor,

Your patient, Mrs.
is to undergo the operation of Colposuspension to treat her stress incontinence.

She has agreed to take part in a study that aims to assess the incidence and the causes of complications that may follow bladder neck surgery. Namely, voiding difficulties and detrusor instability.

Full explanation of the project has been given to her, including an information leaflet.

In addition to routine investigations, she will have:

- An MRI scan before and after surgery to assess the position of the bladder neck.
- Urodynamic investigations in the post-operative period.
- The completion of quality of life questionnaires.

The study should not carry additional risks or discomfort and the patient will be given a contact number should there be any concerns.

Yours faithfully,

Dr. Luigi Bombieri

Research Registrar

Obstetrics and Gynaecology Department

Derriford Hospital
APPENDIX 5. Standard history sheet for patients participating in the study.

HISTORY

URINARY SYMPTOMS:

INCONTINENCE:

Duration (worse for how long):

Severity: 1- Drops/damp
2- Wet pants
3- Pads for safety
4- Pads of necessity

Number of pads: -Day
               -Night

Incapacity: 1- Social restriction (embarrassment)
            2- Physical restriction (sports)
            3- Housebound

PREVIOUS TREATMENT: 1- Pelvic floor exercises
                     2- Tampons
                     3- Drugs (specify)
                     4- Physiotherapy/interferential
                     5- Surgery (specify)

STRESS INCONTINENCE: 1- Yes
                      2- No

Frequency of stress incontinence: 1- Daily
                                 2- One to six times / week
                                 3- Rarely

Provoking factors:

URGENCY: 1- Yes
          2- No

URGE INCONTINENCE: 1- Yes
                   2- No

Frequency of stress incontinence: 1- Daily
                                 2- One to six times / week
                                 3- Rarely

OTHER INCONTINENCE:  - Unconscious
                      - Intercourse
                      - Enuresis
HESITANCY:
1- No
2- Yes occasionally
3- Yes always
4- Yes only when full

URINARY STREAM:
1- Normal
2- Decreased
3- Decreased only on full bladder
4- Interrupted
5- Decreased and interrupted

RETENTION:
1- Never
2- Acute spontaneous
3- Acute after childbirth
4- Acute after surgery

DYSURIA:
1- No
2- Occasionally
3- Frequently

UTI (proven):
1- Never
2- Yes frequent (3 or more per year). Specify number past 12 months:
3- Yes infrequent

POST MICTURITION SYMPTOMS:
1- None
2- Pain
3- Feeling of incomplete emptying
4- Dribbling (post micturition incontinence)

FREQUENCY/VOLUME CHART:
- Maximum volume voided: ml.
- Average volume voided: ml.
- Day frequency
- Night frequency
- Number of leakage episodes/week

FLUID INTAKE: litre/24h

MSU:
1- Normal
2- Abnormal (specify)
HI S TORY - OT HE R

AGE:

PARITY:

CONTRACEPTION:

HORMONAL STATUS: 1- Premenopausal
2- Post menopausal

DELIVERY: 1- Forceps
2- Breech
3- Caesarean section
4- Perineal trauma (to sphincter at least)

WEIGHT OF HEAVIEST BABY: 1- <4 Kg.
2- >4 Kg.

CONSTIPATION (straining to evacuate): 1- Yes
2- No

FAECAL INCONTINENCE: 1- None
2- Flatus
3- Soiling
4- Liquid
5- Solid

SMOKE: 1- Yes
2- No

CHRONIC RESPIRATORY DISEASE: 1- Yes (specify)
2- No

NEUROLOGICAL DISEASE: - No
- Yes (specify)

OTHER MEDICAL PROBLEMS:

OPERATIONS:

PRESENT DRUG THERAPY:

EXAMINATION

ABDOMINAL EXAMINATION:
1-Normal
2-Abnormal (specify)

NEUROLOGICAL EXAMINATION (S234):
1-Normal
2-Abnormal (specify)

VAGINAL EXAMINATION:

Atrophic change: 1- None
2- Mild (dryness, erythema)
3- Moderate (petecchiae, reduced rugae)
4- Severe (contact bleeding, absent rugae)

Demonstrable stress incontinence: 1- Yes
2- No

Pelvic floor strength: 1- Normal
2- Decreased
3- Absent

Uterine size:

Pelvic masses:

Prolapse (traditional classification):
1- Normal
2- Bladder neck descent
3- Cystocele (grade)
4- Cervical (vault) descent (grade)
5- Enterocoele (grade)
6- Rectocoele (grade)

Grade 1: descent at least half-way but not to hymen
Grade 2: descent to hymen
Grade 3: descent beyond hymen
Grade 4: complete eversion

Prolapse (ICS classification):

1-Stage 0
2-Stage 1
3-Stage 2
4-Stage 3
5-Stage 4
ICS pelvic organ prolapse descriptive system (ICS, 1994). Summary of details.

Principles:

- The plane of the hymen is zero.
  
  Points above the hymen have the sign - (minus).
  
  Points below the hymen have the sign + (plus).

- The points Aa and Ap are fixed points 3 cm above the hymen anteriorly and posteriorly respectively. Their position can be between minus 3 and plus 3 during straining.

- The points Ba and Bp are not fixed and are defined as the most dependent (prolapsed) points of the upper vagina (anteriorly and posteriorly) during straining.

- Point C is the most distal part of the cervix (or vault) during straining.

- Point D is the position of the posterior fornix during straining (omit if cervix absent).

- 'Total vaginal length' is the maximum length after correction of prolapse.

- 'Genital hiatus' is the distance from the external urethral meatus to the fourchette.

- 'Perineal body' is the distance from fourchette to mid-anus.
Reporting of measures: A grid-like system is suggested:

<table>
<thead>
<tr>
<th>Aa</th>
<th>Ba</th>
<th>C</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Genital hiatus</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Perineal body</td>
</tr>
<tr>
<td>Ap</td>
<td>Bp</td>
<td>Total vaginal length</td>
</tr>
</tbody>
</table>

Staging of prolapse:

- **Stage 0**: No prolapse. Points Aa, Ap, Ba and Bp are all at -3 cm. Points C or D are not seen to descend by more than 2 cm beyond the total vaginal length.

- **Stage 1**: The most distal portion of the prolapse is 1 cm above the level of the hymen.

- **Stage 2**: The most distal portion of the prolapse is 1 cm or less proximal to or distal to the plane of the hymen.

- **Stage 3**: The most distal portion of the prolapse is more than 1 cm below the level of the hymen but there is no complete eversion.

- **Stage 4**: Complete eversion.
APPENDIX 8. Post-operative catheter regime after colposuspension.

Start this regime on Day 2.

1. Clamp the catheter at 0800 and leave clamped until 2100 hrs.

2. Encourage the patient to void every two hours - if voiding vol. less than 100 ml do a residual, if voiding greater than 100 ml - no residual until 2200 hrs.

3. If the patient cannot void after 4 hours, or if she complains of any discomfort, pain or feelings of wanting to pass urine but can’t, before the four hours is up, then release the catheter for 15 min and then re-clamp.

4. Then leave the catheter on drainage over night, and re-clamp again in the morning.

5. When voiding volumes of 200 ml or over and residuals are less than 100 ml, then clamp the catheter for 24 hrs. The patient can void when they want to, but it is important to ensure that they are voiding regularly and good amounts. At this stage the catheter can be left clamped over night, but the patient must be got out of bed twice during the night to void. If the residual in the morning is less than 100 ml the catheter can be removed providing the volume voided is more than 200 ml.
APPENDIX 9. The Hospital Anxiety and Depression scale [421].

HAD Scale

Name: ___________________________ Date: _______________________

Doctors are aware that emotions play an important part in most illnesses. If your doctor knows about these feelings he will be able to help you more.

This questionnaire is designed to help your doctor to know how you feel. Read each item and place a firm tick in the box opposite the reply which comes closest to how you have been feeling in the past week. Don’t take too long over your replies; your immediate reaction to each item will probably be more accurate than a long thought-out response.

Tick only one box in each section

I feel tense or ‘wound up’:
- Most of the time
- A lot of the time
- Time to time, Occasionally
- Not at all

I feel as if I am slowed down:
- Nearly all the time
- Very often
- Sometimes
- Not at all

I still enjoy the things I used to enjoy:
- Definitely as much
- Not quite so much
- Only a little
- Hardly at all

I get a sort of frightened feeling like ‘butterflies’ in the stomach:
- Not at all
- Occasionally
- Quite often
- Very often

I have lost interest in my appearance:
- Definitely
- I don’t take so much care as I should...
- I may not take quite as much care
- I take just as much care as ever

I feel restless as if I have to be on the move:
- Very much indeed
- Quite a lot
- Not very much
- Not at all

I look forward with enjoyment to things:
- As much as ever I did
- Rather less than I used to
- Definitely less than I used to
- Hardly at all

I get sudden feelings of panic:
- Very often indeed
- Quite often
- Not very often
- Not at all

I can sit at ease and feel relaxed:
- Definitely
- Usually
- Not often
- Not at all

I can enjoy a good book or radio or TV programme:
- Often
- Sometimes
- Not often
- Very seldom

APPENDIX

9. The Hospital Anxiety and Depression scale [421].
9. What Does Your Pain Feel Like?

Some of the words I will read to you describe your present pain. Tell me which words best describe it. Leave out any word-group that is not suitable. Use only a single word in each appropriate group—the one that applies best.

<table>
<thead>
<tr>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
</tr>
</thead>
<tbody>
<tr>
<td>2. Quivering</td>
<td>2. Flashing</td>
<td>2. Boring</td>
<td>2. Cutting</td>
</tr>
<tr>
<td>6. Pounding</td>
<td></td>
<td>5. Lancing</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>6</td>
<td>7</td>
<td>8</td>
</tr>
<tr>
<td>5. Crushing</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>10</td>
<td>11</td>
<td>12</td>
</tr>
<tr>
<td>2. Sore</td>
<td>2. Taut</td>
<td>2. Exhausting</td>
<td>2. Suffocating</td>
</tr>
<tr>
<td>3. Hurting</td>
<td>3. Rapping</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4.aching</td>
<td>4. Splitting</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Heavy</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>14</td>
<td>15</td>
<td>16</td>
</tr>
<tr>
<td>1. Fearful</td>
<td>1. Punishing</td>
<td>1. Wretched</td>
<td>1. Annoying</td>
</tr>
<tr>
<td></td>
<td>4. Vicious</td>
<td></td>
<td>4. Intense</td>
</tr>
<tr>
<td></td>
<td>5. Killing</td>
<td></td>
<td>5. Unbearable</td>
</tr>
<tr>
<td>17</td>
<td>18</td>
<td>19</td>
<td>20</td>
</tr>
<tr>
<td></td>
<td>5. Tearing</td>
<td></td>
<td>5. Torturing</td>
</tr>
</tbody>
</table>

No pain (tick if appropriate)
WHY DO WOMEN HAVE VOIDING DIFFICULTY AFTER
COLPOSUSPENSION?

L Bombieri and RM Freeman
Urogynaecology Unit, Derriford Hospital, Plymouth, UK

Objective: To study the possible causes of early voiding difficulty after Colposuspension for Genuine Stress Incontinence.

Methods: A prospective observational study of 42 women. The following possible factors were investigated: 1) Bladder neck elevation was assessed using MRI before and after surgery. 2) Urethral compression was assessed by measuring bladder neck approximation to the pubis using MRI before and after surgery; by measuring the distance between medial stitches with a ruler during surgery; by measuring the width of the urethro-pubic space with Hegar dilators. 3) Detrusor contractility was assessed pre-operatively and post-operatively on day 4 by voiding CMG. 4) Patient related factors: anxiety and depression were assessed with the HAD scale; post-operative pain was measured with the McGill's pain questionnaire; other factors such as age, menopausal status, weight, etc. were also considered.

All factors were correlated to: A) first day of void (i.e. volume greater than 50 mls). B) day of catheter removal.

Data was analysed using F test, t-test and multiple regression analysis.
**Results**: The following variables were found to be independent risk factors:

A) **First day of void**: elevation by 26 mm or more ($P=0.003$); increasing age ($P=0.0016$); decreasing Hegar size (i.e. increasing urethral compression) ($P=0.046$); higher anxiety score ($P=0.031$).

B) **Day of catheter removal**: elevation by 26 mm or more ($P=0.0001$); increasing age ($P=0.0152$); increasing pain score ($P=0.00063$); pre-operative voiding pattern (voiding +/- detrusor contraction +/- abdominal strain) ($P=0.0001$)

Post operative (day 4) detrusor pressure was not correlated to voiding performance.

**Conclusion**: The main factor associated with voiding difficulty appears to be bladder neck elevation. Other independent factors (e.g. age, pain, anxiety, pre-operative voiding pattern, urethral compression) might explain why some patients with bladder neck elevation of more than 26 mm do not have voiding difficulty.
KING'S HEALTH QUESTIONNAIRE
1993

Name ____________________________
Age ________ years
Todays date ________/______/1993

How would you describe your health at present?

Please tick one answer

- Very good
- Good
- Fair
- Poor
- Very poor

How much do you think your bladder problem affects your life?

Please tick one answer

- Not at all
- A little
- Moderately
- A lot

Please turn the page
We would like to know what your bladder problems are and how much they affect you. From the list below choose ONLY THOSE PROBLEMS that you have at present.

<table>
<thead>
<tr>
<th>Problem</th>
<th>A Little</th>
<th>Moderately</th>
<th>Alot</th>
</tr>
</thead>
<tbody>
<tr>
<td>FREQUENCY; going to the toilet very often.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NOCTURIA; getting up at night to pass urine.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>URGENCY; a strong and difficult to control desire to pass urine.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>URGE INCONTINENCE; urinary leakage associated with a strong desire to pass urine.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>STRESS INCONTINENCE; urinary leakage with physical activity eg coughing, sneezing, running.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NOCTURNAL ENURESIS; wetting the bed at night.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>INTERCOURSE INCONTINENCE; urinary leakage with sexual intercourse.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FREQUENT WATERWORKS INFECTIONS;</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BLADDER PAIN;</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>OTHER SPECIFY:</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Please turn the page

Office use

King's Health Questionnaire, Version 7
Below are some daily activities that can be affected by bladder problems. How much does your bladder problem affect you? We would like you to answer every question. **Simply tick the circle that applies to you.**

### ROLE LIMITATIONS

<table>
<thead>
<tr>
<th>Question</th>
<th>Not at all</th>
<th>Slightly</th>
<th>Moderately</th>
<th>Alot</th>
</tr>
</thead>
<tbody>
<tr>
<td>To what extent does your bladder problem affect your household tasks (e.g. cleaning, shopping etc)?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Does your bladder problem affect your job, or your normal daily activities outside the home?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### PHYSICAL/SOCIAL LIMITATIONS

<table>
<thead>
<tr>
<th>Question</th>
<th>Not at all</th>
<th>Slightly</th>
<th>Moderately</th>
<th>Alot</th>
</tr>
</thead>
<tbody>
<tr>
<td>Does your bladder problem affect your physical activities (e.g. going for a walk, run, sport, gym etc)?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Does your bladder problem affect your ability to travel?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Does your bladder problem limit your social life?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Does your bladder problem limit your ability to see/visit friends?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### PERSONAL RELATIONSHIPS

<table>
<thead>
<tr>
<th>Question</th>
<th>Not applicable</th>
<th>Not at all</th>
<th>Slightly</th>
<th>Moderately</th>
<th>Alot</th>
</tr>
</thead>
<tbody>
<tr>
<td>Does your bladder problem affect your relationship with your partner?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Does your bladder problem affect your sex life?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Does your bladder problem affect your family life?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Please turn the page

Office use

Kings’s Health Questionnaire, Version 7
## EMOTIONS

<table>
<thead>
<tr>
<th>Question</th>
<th>Not at all</th>
<th>Slightly</th>
<th>Moderately</th>
<th>Very much</th>
</tr>
</thead>
<tbody>
<tr>
<td>Does your bladder problem make you feel depressed?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Does your bladder problem make you feel anxious or nervous?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Does your bladder problem make you feel bad about yourself?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

## SLEEP / ENERGY

<table>
<thead>
<tr>
<th>Question</th>
<th>Never</th>
<th>Sometimes</th>
<th>Often</th>
<th>All the time</th>
</tr>
</thead>
<tbody>
<tr>
<td>Does your bladder problem affect your sleep?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Do you feel worn out / tired?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

## Do you do any of the following?:

If so how much?

<table>
<thead>
<tr>
<th>Question</th>
<th>Never</th>
<th>Sometimes</th>
<th>Often</th>
<th>All the time</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wear pads to keep dry?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Be careful how much fluid you drink?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Change your underclothes when they get wet?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Worry in case you smell?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Office use

[ ] [ ] [ ] [ ] [ ] [ ] [ ]

THANKYOU, NOW CHECK THAT YOU HAVE ANSWERED ALL THE QUESTIONS

King’s Health Questionnaire, Version 7.
**APPENDIX 13.** Quality of life of 77 women before colposuspension and after three and twelve months, using King’s Health Questionnaire [378]. Results of Wilcoxon tests for comparing before, three months (3m) and twelve months (12m) quality of life scores. Scores range from 0 (ideal outcome) to 100 (worse possible outcome). Reduction of median score values equates to improvement.

<table>
<thead>
<tr>
<th>Quality of life domains</th>
<th>Reduction of median score values</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Health 3m</td>
<td>-12.50</td>
<td>0.001</td>
</tr>
<tr>
<td>Health 12m</td>
<td>-12.50</td>
<td>0.005</td>
</tr>
<tr>
<td>Symptoms impact 3m</td>
<td>-50.00</td>
<td>0.001</td>
</tr>
<tr>
<td>Symptoms impact 12m</td>
<td>-66.60</td>
<td>0.001</td>
</tr>
<tr>
<td>Role limitations 3m</td>
<td>-41.60</td>
<td>0.001</td>
</tr>
<tr>
<td>Role limitations 12m</td>
<td>-41.65</td>
<td>0.001</td>
</tr>
<tr>
<td>Physical limitations 3m</td>
<td>-58.30</td>
<td>0.001</td>
</tr>
<tr>
<td>Physical limitations 12m</td>
<td>-58.30</td>
<td>0.001</td>
</tr>
<tr>
<td>Social limitations 3m</td>
<td>-27.75</td>
<td>0.001</td>
</tr>
<tr>
<td>Social limitations 12m</td>
<td>-27.75</td>
<td>0.001</td>
</tr>
<tr>
<td>Personal relationships 3m</td>
<td>-16.65</td>
<td>0.001</td>
</tr>
<tr>
<td>Personal relationships 12m</td>
<td>-16.65</td>
<td>0.001</td>
</tr>
<tr>
<td>Emotions 3m</td>
<td>-38.85</td>
<td>0.001</td>
</tr>
<tr>
<td>Emotions 12m</td>
<td>-38.85</td>
<td>0.001</td>
</tr>
<tr>
<td>Sleep / energy 3m</td>
<td>-16.65</td>
<td>0.001</td>
</tr>
<tr>
<td>Sleep / energy 12m</td>
<td>-16.65</td>
<td>0.001</td>
</tr>
<tr>
<td>Severity 3m</td>
<td>-54.15</td>
<td>0.001</td>
</tr>
<tr>
<td>Severity 12m</td>
<td>-58.30</td>
<td>0.001</td>
</tr>
</tbody>
</table>
APPENDIX 14: Quality of life domains (as reported in King’s Health Questionnaire) of six women with ‘late’ voiding dysfunction, before surgery (1) and three (2) and twelve (3) months post-operatively.

Each patient is listed separately (with median values in bold).
Scores range from 0 (ideal outcome) to 100 (worse possible outcome).

Post-operative factors (de novo detrusor instability and persistent stress incontinence) which may also affect quality of life are detailed below.

<table>
<thead>
<tr>
<th>Health</th>
<th>Health</th>
<th>Health</th>
<th>Impact</th>
<th>Impact</th>
<th>Impact</th>
<th>Role</th>
<th>Role</th>
<th>Role</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 25</td>
<td>2 25</td>
<td>3 25</td>
<td>1 66.6</td>
<td>2 100</td>
<td>3 66.6</td>
<td>1 66.6</td>
<td>3 33.3</td>
<td>0 0</td>
</tr>
<tr>
<td>2 0</td>
<td>0 0</td>
<td>0 25</td>
<td>0 66.6</td>
<td>0 100</td>
<td>0 100</td>
<td>0 100</td>
<td>0 100</td>
<td>33.3</td>
</tr>
<tr>
<td>3 0</td>
<td>0 25</td>
<td>0 66.6</td>
<td>0 100</td>
<td>0 100</td>
<td>0 100</td>
<td>0 100</td>
<td>0 100</td>
<td>33.3</td>
</tr>
<tr>
<td>4 50</td>
<td>25 25</td>
<td>50 66.6</td>
<td>0 100</td>
<td>0 33.3</td>
<td>0 33.3</td>
<td>50 0</td>
<td>0 0</td>
<td>0 0</td>
</tr>
<tr>
<td>5 25</td>
<td>25 50</td>
<td>25 100</td>
<td>0 33.3</td>
<td>3 33.3</td>
<td>0 83.3</td>
<td>16.6</td>
<td>16.6</td>
<td>0 0</td>
</tr>
<tr>
<td>6 25</td>
<td>25 25</td>
<td>25 100</td>
<td>0 33.3</td>
<td>3 33.3</td>
<td>0 83.3</td>
<td>16.6</td>
<td>33.3</td>
<td>0 0</td>
</tr>
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</table>

<table>
<thead>
<tr>
<th>Physical</th>
<th>Physical</th>
<th>Physical</th>
<th>Social</th>
<th>Social</th>
<th>Social</th>
<th>Personal</th>
<th>Personal</th>
<th>Personal</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 50</td>
<td>2 33.3</td>
<td>3 0</td>
<td>1 0</td>
<td>2 0</td>
<td>3 0</td>
<td>1 0</td>
<td>2 0</td>
<td>3 0</td>
</tr>
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APPENDIX 15: Quality of life domains (as reported in King’s Health Questionnaire) of nine women with symptomatic de novo detrusor instability, before surgery (1) and three (2) and twelve (3) months post-operatively.

Each patient is listed separately (with median values in bold).

Scores range from 0 (ideal outcome) to 100 (worse possible outcome).

No patients had additional post-operative problems (voiding dysfunction or persistent stress incontinence), which may have adversely affected quality of life, except patient no.5, who had voiding dysfunction in addition to de novo detrusor instability.

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Appendix 16.

Can Bladder Neck Elevation Be Objectively Assessed During Colposuspension?


Objective:

Overerelevation of the bladder neck may cause voiding dysfunction after colposuspension.

The aim of this study was to evaluate intra-operative methods for the assessment of elevation during colposuspension.

Methods:

Three methods that had the potential to measure the amount of bladder neck elevation were assessed. Measurements obtained using these methods were compared to the 'gold standard' MRI measures of elevation, using Pearson’s correlation coefficient.

The methods studied were:
1) The amount of change in Q-tip angle in 56 women.

Fig. 20. Intra-operative measurement of Q-tip angle (instruments used).
2) The amount of suture tension using a spring scale in 38 women (as suggested by Kondo et al) [308]

Each of the medial sutures was attached to a Spencer-Wells forceps that was hooked to a spring scale. The amount of tension required to lift the paravaginal fascia to the desired level was recorded in ounces and later converted to grams. The mean of two sides was used for analysis.

Fig. 21. Intra-operative measurement of suture tension using a spring scale.
3) Elevation measured by perineal ultrasound in 28 women. The position of the bladder neck was determined using a system of co-ordinates as suggested by Schaer et al [118].

Fig. 22. Intra-operative perineal ultrasound (arrow indicates bladder neck).
Results:

Suture tension did not correlate with MRI.

The change in Q-tip angle had a weak but significant \((p<0.05)\) positive correlation with MRI \((0.331)\).

Perineal ultrasound measures of elevation had a significant \((p<0.01)\) positive correlation with MRI \((0.50)\). The ultrasound measures of bladder neck position \((Dx)\) vs MRI before elevation showed a correlation of \(0.1\) (not significant) and after elevation of \(0.65\) (significant).

Discussion:

Despite a significant correlation with MRI, ultrasound underestimated elevation (as compared to MRI) by almost one centimetre. When comparing ultrasound and MRI measures separately before and after elevation, there was no correlation between pre-operative measures and a better correlation of \(0.65\) after elevation. This was surprising as we would have expected the opposite (quality of image is better before bladder neck elevation than afterwards, when the bladder neck disappears partially behind the pubic bone). Our baseline ultrasound measures had been made after dissection and suture placement, which by themselves may have caused some bladder neck elevation. This probably explains the discrepancy between MRI and ultrasound measures.

Conclusion:

Of the methods studied, perineal ultrasound has been shown to be the most promising for the objective measurement of bladder neck elevation during surgery. Better correlations could be obtained if the baseline bladder neck position is determined pre-operatively or at least prior to dissection and suture placement. Reproducibility studies of intra-operative perineal ultrasound should be performed.
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<td>Detrusor Instability</td>
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