An investigation into the role of pregnancy in the development of stress incontinence of urine.

Thesis submitted to The University of London for the Degree of Doctor of Medicine

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Abstract

An investigation into the role of pregnancy in the development of stress incontinence of urine.

Methods:

For the prospective observational study, 250 women were recruited from the antenatal clinics of University College London Hospital and The Whittington Hospital at their booking appointment. All women were less than 20 weeks pregnant at booking. The patients were interviewed with a standard questionnaire at booking, 28 weeks, 34 – 36 weeks of pregnancy and at 6 and 12 weeks postpartum. At the first interview patients were asked about incontinence prior to the pregnancy. The patients then completed a frequency volume voiding chart for each visit except the first. They were asked to attend the clinic with a full bladder and performed a standing stress test at the 28 and 34 – 36 week visit. The delivery details were collected at the first postpartum interview.

For the retrospective study 300 primiparous patients who had delivered at The Whittington hospital were sent a questionnaire three months after delivery.

Results:

181 women completed the prospective study. The reported frequency of micturition increased during pregnancy and declined after delivery. The frequency of micturition recorded on the charts showed a similar pattern. The total volume voided per day increased during pregnancy and declined after delivery whereas the mean volume voided at each micturition decreased in pregnancy compared to postpartum. There was no difference
in the mean volume voided in the women who reported incontinence compared to those women who were dry.

The numbers of women reporting incontinence increased in pregnancy to 44.8% at 28 weeks and then declined after delivery to 12.2% at 12 weeks postpartum.

58.1% of the retrospective questionnaires were returned. 32.9% had some form of incontinence at the time of completing the questionnaire whereas 31.8% had incontinence in pregnancy.
Table of Contents

ABSTRACT......................................................................................................................... 2
DECLARATION......................................................................................................................... 8
ACKNOWLEDGEMENTS.......................................................................................................... 9
PRESENTATIONS.................................................................................................................. 10
DEFINITIONS....................................................................................................................... 11

CHAPTER 1 - INTRODUCTION ............................................................................................ 13
1.1 THE USE OF ORDINAL SCALES TO DESCRIBE QUANTITATIVE DATA....................... 14
1.2 THE USE OF LONGITUDINAL STATEMENTS FOR CROSS SECTIONAL DATA ............ 15
1.3 CORRELATION, CAUSATION AND COINCIDENCE................................................... 15
1.4 DATA DREDGING......................................................................................................... 15

CHAPTER 2 - REVIEW OF THE LITERATURE ................................................................... 16
2.1 THE LOWER URINARY TRACT; STRUCTURE AND FUNCTION.................................... 17
2.2 URINARY INCONTINENCE.......................................................................................... 20
  2.2.1 Causes.................................................................................................................... 20
  2.2.2 Prevalence of incontinence................................................................................... 22
  2.2.3 Impact of incontinence......................................................................................... 25
  2.2.4 Management of incontinence by general practitioners........................................ 26
2.3 THE LOWER URINARY TRACT IN PREGNANCY....................................................... 28
  2.3.1 Frequency of micturition and nocturia................................................................. 28
  2.3.2 Incontinence......................................................................................................... 31
  2.3.3 Incontinence postpartum...................................................................................... 34
  2.3.4 Incontinence during pregnancy ......................................................................... 38
  2.3.5 Detrusor instability and pregnancy .................................................................... 41
  2.3.6 Prospective studies of stress incontinence during pregnancy and after delivery... 43
2.4 PATHOPHYSIOLOGY OF INCONTINENCE IN RELATION TO PREGNANCY AND CHILDBIRTH...... 48
  2.4.1 Neurological sphincter damage......................................................................... 48
  2.4.2 Primary muscle damage to the urethral sphincter.............................................. 53
  2.4.3 Hormonal effects............................................................................................... 55
  2.4.4 Vascular pulsations............................................................................................ 56
  2.4.5 Total and functional urethral length.................................................................... 57
  2.4.6 Loss of urethral support...................................................................................... 58
2.5 FAECAL INCONTINENCE ............................................................................................ 60
  2.5.1 The anal sphincters and maintenance of continence........................................ 61
  2.5.2 Faecal incontinence and childbirth.................................................................... 62
  2.5.3 Constipation and childbirth.............................................................................. 63
  2.5.4 Relevance of bowel symptoms to project......................................................... 63

CHAPTER 3 - METHODS OF INVESTIGATION...................................................................... 65
3.1 INVESTIGATION TECHNIQUES..................................................................................... 66
Table of Tables

Table 1: Studies of Prevalence of Incontinence ................................................................. 23
Table 2: Studies of Incontinence in Pregnancy ................................................................. 32
Table 3: Studies of Incontinence Postpartum .................................................................. 35
Table 4: Prospective Studies of Stress Incontinence during Pregnancy & Postpartum ...... 43
Table 5: Prevalence of Detrusor Instability during Pregnancy & Postpartum (Cutner) ...... 46
Table 6: Continuation Rate for the Whole Sample ............................................................ 96
Table 7: Continuation Rate by Hospital ........................................................................... 97
Table 8: Racial Distribution ............................................................................................ 98
Table 9: Mode of Delivery .............................................................................................. 99
Table 10: Stress Incontinence - All ................................................................................ 109
Table 11: Frequency of Defaecation ............................................................................... 111
Table 12: Frequency Volume Voiding Charts Completed by Gestation ......................... 115
Table 13: Mode of Delivery for retrospective questionnaire ......................................... 125
Table 14: Mode of Delivery Respondants vs Non-Respondants ..................................... 126
Table 15: Summary of Replies from retrospective questionnaire .................................. 129
Table 16: Frequency of Micturition in the Three Groups ............................................... 132
Table 17: Nocturia in the Three Groups ......................................................................... 133
Table of Figures

<table>
<thead>
<tr>
<th>Figure</th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Figure 1</td>
<td>Q-Q Plot of $P_{\text{OCT, CLOSE}}$</td>
<td>88</td>
</tr>
<tr>
<td>Figure 2</td>
<td>$P_{\text{OCT, CLOSE}}$ in women with and without genuine stress incontinence</td>
<td>90</td>
</tr>
<tr>
<td>Figure 3</td>
<td>$P_{\text{OCT, CLOSE}}$ in women with and without symptoms of stress incontinence (on coughing or sneezing)</td>
<td>90</td>
</tr>
<tr>
<td>Figure 4</td>
<td>$P_{\text{OCT, CLOSE}}$ in women with and without symptoms of urge incontinence</td>
<td>91</td>
</tr>
<tr>
<td>Figure 5</td>
<td>$P_{\text{OCT, CLOSE}}$ in women with symptoms of urgency</td>
<td>91</td>
</tr>
<tr>
<td>Figure 6</td>
<td>Racial distribution</td>
<td>98</td>
</tr>
<tr>
<td>Figure 7</td>
<td>Mode of delivery</td>
<td>100</td>
</tr>
<tr>
<td>Figure 8</td>
<td>Daytime micturition frequency by visit (from questionnaire)</td>
<td>102</td>
</tr>
<tr>
<td>Figure 9</td>
<td>Nocturia frequency by visit (from questionnaire)</td>
<td>103</td>
</tr>
<tr>
<td>Figure 10</td>
<td>Incontinence during the day</td>
<td>104</td>
</tr>
<tr>
<td>Figure 11</td>
<td>Comparison of incontinence in patients completing the study and those dropping out early</td>
<td>105</td>
</tr>
<tr>
<td>Figure 12</td>
<td>Urgency</td>
<td>106</td>
</tr>
<tr>
<td>Figure 13</td>
<td>Urge incontinence</td>
<td>106</td>
</tr>
<tr>
<td>Figure 14</td>
<td>Stress incontinence - cough or sneeze</td>
<td>107</td>
</tr>
<tr>
<td>Figure 15</td>
<td>Stress incontinence - walking or standing</td>
<td>108</td>
</tr>
<tr>
<td>Figure 16</td>
<td>Incontinence - no feeling</td>
<td>110</td>
</tr>
<tr>
<td>Figure 17</td>
<td>Frequency of defaecation</td>
<td>111</td>
</tr>
<tr>
<td>Figure 18</td>
<td>Urgency to defaecate</td>
<td>112</td>
</tr>
<tr>
<td>Figure 19</td>
<td>Faecal incontinence</td>
<td>113</td>
</tr>
<tr>
<td>Figure 20</td>
<td>Micturition frequency (7 day data) by visit (from frequency volume charts)</td>
<td>116</td>
</tr>
<tr>
<td>Figure 21</td>
<td>Micturition frequency – comparing volume measured days against unmeasured days</td>
<td>118</td>
</tr>
<tr>
<td>Figure 22</td>
<td>Total daily voided volume by visit</td>
<td>119</td>
</tr>
<tr>
<td>Figure 23</td>
<td>Mean voided volume by visit (2 day measured data)</td>
<td>120</td>
</tr>
<tr>
<td>Figure 24</td>
<td>Daily micturition frequency by visit (from frequency volume data) – comparing wet and dry women at that visit (determined from questionnaire)</td>
<td>122</td>
</tr>
<tr>
<td>Figure 25</td>
<td>Mean voiding volume by visit (from frequency volume data) – comparing wet and dry women at that visit (determined from questionnaire)</td>
<td>123</td>
</tr>
<tr>
<td>Figure 26</td>
<td>Frequency of micturition in the whole group</td>
<td>127</td>
</tr>
<tr>
<td>Figure 27</td>
<td>Nocturia in the whole group</td>
<td>128</td>
</tr>
<tr>
<td>Figure 28</td>
<td>Frequency of micturition in the three groups</td>
<td>131</td>
</tr>
<tr>
<td>Figure 29</td>
<td>Nocturia in the three groups</td>
<td>132</td>
</tr>
</tbody>
</table>
Declaration

The work contained in this thesis was carried out at The Obstetric Hospital of University College London Hospitals Trust and The Whittington Hospital between August 1997 and August 1999.

All of the work is my own. None of the data forms part of any other thesis. The study was approved by the Joint UCL/UCLH Committees on the Ethics of Human Research, Camden and Islington Community Health Services NHS Trust Research Ethics Committee and The Whittington Hospital Ethics Committee. All patients gave informed consent prior to their involvement in the study.

Victoria A.M. Cook
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Last but not least I would like to thank my husband Michael Gadsdon who has provided encouragement, child care and computer support throughout my time as a research fellow and during the writing up of this thesis.
Presentations

The work from this thesis has been presented to the following learned societies:

*An investigation into the role of pregnancy in the damage to the urethral sphincter mechanism that leads to stress incontinence of urine.*

Presented to The London Urodynamic Club, January 1998

*Pregnancy and not delivery associated with postpartum incontinence in primigravid women*

Presented to The International Continence Society, Denver, August 1999
Also presented to The London Urodynamic Club, January 2000

*Lower urinary tract symptoms in pregnancy and after delivery*

Presented to The International Continence Society (UK), Manchester April 2001

*Lower urinary tract symptoms in pregnancy and after delivery*

Presented to The London Urodynamic Club, May 2001
Definitions

The following definitions apply throughout this thesis and, where appropriate comply with the ‘Standardisation of terminology of lower urinary tract function’ recommended by The International Continence Society.

Incontinence is ‘a condition of involuntary urine loss that is a social or hygienic problem and is objectively demonstrable.’

Stress incontinence indicates the patient's statement of involuntary loss of urine during physical exertion.

Genuine stress incontinence is the involuntary loss of urine when intravesical pressure exceeds the maximum urethral closure pressure in the absence of detrusor activity. It is a diagnosis made by urodynamics, it is characterised by the symptom of stress incontinence and is caused by a failure of one or more components of the urethral sphincter mechanism.

Urgency is a strong desire to void.

Urgent incontinence is the involuntary loss of urine associated with a strong desire to void.

Detrusor instability is a detrusor that is shown objectively to contract, spontaneously or on provocation, during the filling phase of cystometry whilst the patient is attempting to inhibit micturition. These contractions may or may not result in leakage of urine. Detrusor instability is characterised by symptoms of frequency, urgency, urge incontinence and nocturia.
**Frequency** is the number of voids during waking hours. Often an arbitrary value is assigned to denote abnormal frequency, for example voiding greater than seven times per day.

**Nocturia** is the number of times the patient has to wake from sleep to void.

**Nocturnal enuresis** is the involuntary passage of urine during sleep.

The **overactive bladder** is characterised by symptoms of frequency and urgency with or without urge incontinence.
Chapter 1 - Introduction
Urinary incontinence is common and its prevalence increases with age and parity. The development of stress incontinence is often assumed to be due to vaginal delivery, particularly operative vaginal delivery. Some women and obstetricians are now beginning to discuss the option of elective caesarean section in order to prevent the development of urinary incontinence. However before this becomes widespread practice it is vitally important to establish whether or not there is a link between mode of delivery and the development of urinary stress incontinence. It is with this in mind that we have performed the following studies.

I would like to start by reviewing the literature in order to then illustrate the reasons as to why we performed the study and the methods that we have used.

In order to simplify some of the criticisms of previous studies I will first discuss some errors which are commonly made in published research.

1.1 The use of ordinal scales to describe quantitative data

Commonly arbitrary ordinal scales are applied to continuous variables which is inappropriate. For example in the description of frequency of micturition a figure is chosen, above which the frequency of micturition is abnormal, the data set is then described as normal or abnormal, rather than using statistical methods appropriate for the description of such quantitative data. The use of ordinal scales in such circumstances will introduce error.
1.2 The use of longitudinal statements for cross sectional data

The data obtained from longitudinal studies is different from that obtained from cross sectional studies and so appropriate statistical tests and statements should be made in each case.

1.3 Correlation, causation and coincidence

It is important to remember the differences between correlation and causation. Correlation means that variables are associated whereas causation means that one variable has caused the other. It should always be remembered that findings may be due to coincidence. In order to avoid error it is vital to start with an appropriate null hypothesis and then use appropriate methods to prove or disprove the null hypothesis. If the results are then found to be significant then they are unlikely to be due to coincidence.

1.4 Data dredging

It is unfortunate that much research employs this technique. Data is collected, often retrospectively, and is then examined for any correlations. If a correlation is found it is then often interpreted as a causation. This method of research should only be used in the preliminary stages of a study, so that the results are then used to design an experiment to prove or disprove causation.
Chapter 2 - Review of the Literature
2.1 The lower urinary tract; structure and function

The bladder is a muscular reservoir designed for the storage of urine. It comprises three layers. The outer serous layer, the peritoneum, is only present over the fundus. The muscular layer, the detrusor muscle, is made up of three layers of smooth muscle, contraction of this muscle results in reduction in the dimensions of the bladder in all directions simultaneously. The inner layer is made up of transitional cell epithelium. The trigone is the triangular area of the bladder between the two ureteric orifices and the urethral orifice, it's embryological origin is different and so the detrusor muscle in the trigone comprises only two layers.\(^3\)\(^4\)

The female urethra is around 4cm long, it lies between the internal meatus in the bladder and the external meatus in the vestibule of the vagina. The lining of the upper urethra is made up of transitional cell epithelium, this gradually changes to non-keratinizing stratified squamous epithelium as it approaches the external urethral meatus. The submucosa contains a vascular plexus which is thought to assist in forming a water-tight closure of the mucosa. The muscular elements of the urethra are rather complex. There is smooth muscle at the level of the bladder neck known as the internal sphincter, this is made up of a ring of smooth muscle and elastin, known as the trigonal ring, and a loop of detrusor muscle, known as the detrusor loop. The function of these muscles is not fully understood, it is thought that they may facilitate opening and closure of the bladder neck. A circular ring of striated muscle, the rhabdosphincter, runs from 20 to 80% of the length of urethra, it is thickest anteriorly, thins laterally and is virtually absent posteriorly. This muscle contains slow twitch fibres and is thought to be important in the maintenance of continence at rest.\(^3\)\(^4\)
Continence has been thought to be maintained by a combination of factors. In addition to the rhabdosphincter and the vascular plexus already mentioned, the fibres of the levator ani muscle form a ring around the urethra just below the level of the rhabdosphincter. These are mostly made up of fast twitch fibres and so can contract very efficiently but for short periods of time, thereby helping to maintain continence at times of increased abdominal pressure. The final factor in the maintenance of continence is the support of the bladder neck and upper urethra maintaining these structures within the abdominal cavity. Any increase in abdominal pressure is therefore transmitted equally to the bladder and to the upper urethra, the urethral pressure is maintained higher than bladder pressure and so continence results.\(^3\):\(^4\)

This concept has been challenged by DeLancey.\(^5\)-\(^7\) He realised that the traditional view did not explain the clinical situations in which some women with significant anterior wall prolapse, and hence prolapse of the bladder neck outside of the abdominal cavity, remained continent whereas other women with a bladder neck supported within the abdominal cavity suffered from incontinence.

DeLancey’s theory is dependant on the urethral support mechanisms which comprise the arcus tendineus fasciae pelvis, the levator ani muscles and the endopelvic fascia around the urethra and vagina. The arcus tendineus fascia pelvis, otherwise known as the white line, is a fibrous band which lies on either side of the pelvis, extending from the pubic bone anteriorly to the ischial spine posteriorly. The urethra and the anterior vaginal wall are intimately connected by the endopelvic connective tissue. The endopelvic connective tissue between the proximal urethra and vagina is adherent to the white line. Connective tissue around the vagina interpenetrates the fibres of levator ani. The connections between urethra, vagina, levator ani and white line are responsible for the position and mobility of the proximal
urethra and bladder neck. The levator ani muscle contains slow twitch fibres which maintain a constant tone maintaining the high position of the bladder neck at rest.

Increases in abdominal pressure, for example with a cough, will compress the proximal urethra against the anterior vaginal wall and endopelvic fascia as long as they remain attached to the levator ani and the white line. Thus provided these connections remain intact the mechanism will continue to function even if the anterior vaginal wall prolapses outside of the abdominal cavity.

Anterior to the bladder neck there is a thickening of the endopelvic connective tissue which contains smooth muscle, the pubovesical ligament. The pubovesical ligament is attached to the white line and is thought to contract at the time of voiding, pulling anteriorly on the bladder neck and facilitating its opening.

Continence is said to be maintained by several mechanisms, the urethral rhabdosphincter, the urethral support mechanism and the vascular plexus causing a water-tight seal of the epithelial lining. When incontinence occurs it can be because any one, or all of these mechanisms has failed. In many cases, for instance after childbirth, it is probable that one or more of these factors has failed but continence is maintained by compensation of the other mechanisms.
2.2 Urinary Incontinence

The International Continence Society has defined incontinence as ‘a condition of involuntary urine loss that is a social or hygienic problem and is objectively demonstrable.’

2.2.1 Causes

Continence is maintained by a combination of factors, as discussed above. The bladder is a low pressure reservoir for the storage of urine, the complex urethral sphincter mechanism maintains a high pressure in the urethra and so prevents leakage of urine. In order for incontinence to occur there has to be a disturbance of neurological or psychological control, a disturbance of muscle function or structural abnormalities.

The major causes of incontinence are:

- Genuine stress incontinence
- Detrusor instability
- Overflow incontinence
- Fistulae (vesicovaginal, ureterovaginal, urethrovaginal)
- Congenital (e.g. epispadias, ectopic ureter)
- Urethral diverticulum
- Temporary (e.g. Urinary tract infection, faecal impaction, drugs)
- Functional (e.g. immobility)

When considering incontinence in pregnancy not all the above causes are relevant. Urinary retention in pregnancy is uncommon, most often it occurs
with an impacted retroverted uterus at about sixteen weeks of pregnancy. As a result overflow incontinence is not a common problem in pregnancy. In developed countries the most common cause of fistulae is gynaecological surgery such as abdominal hysterectomy and so fistulae are not a cause of incontinence in pregnancy in this country. Congenital causes of incontinence and urethral diverticulum are both rare and so do not significantly contribute to incontinence in pregnancy. Temporary causes by their very nature do not cause ongoing incontinence through pregnancy. Functional problems leading to incontinence will be confined to groups such as the elderly, the handicapped and those with psychiatric problems and so again will not be a major cause of incontinence in pregnancy in the general population. As a result only genuine stress incontinence and detrusor instability will be considered further.

Genuine stress incontinence is defined by The International Continence Society as the involuntary loss of urine when intravesical pressure exceeds the maximum urethral closure pressure in the absence of detrusor activity. It is a diagnosis made by urodynamics, it is characterised by the symptom of stress incontinence and is caused by a failure of one or more components of the urethral sphincter mechanism.

Detrusor instability is defined by The International Continence Society as a detrusor that is shown objectively to contract, spontaneously or on provocation, during the filling phase of cystometry whilst the patient is attempting to inhibit micturition. These contractions may or may not result in leakage of urine. Detrusor instability is characterised by symptoms of frequency, urgency, urge incontinence and nocturia. The pathophysiology of detrusor instability is poorly understood and so most cases are classified as idiopathic detrusor instability.

Genuine stress incontinence and detrusor instability can occur together.
2.2.2 Prevalence of incontinence

There have been a number of large studies examining the prevalence of incontinence in the community. These have been conducted either as postal questionnaire studies or as interviews (on the telephone or face to face). These have demonstrated that incontinence is common, 14% to 60% of women admitted to some form of incontinence. The advantage of these studies is that it is easy to obtain data from large numbers of women, however there are several disadvantages. It is difficult to ensure a random population as those women who suffer incontinence are more likely to participate in the study than those without symptoms. The range of prevalence in these studies is very wide (14% - 60%) and this highlights another of the problems of questionnaire studies. Many different questionnaires have been developed and used in these studies with no validation as to the accuracy of their results. In order to assess the reliability of a questionnaire the data that it produces should be compared to some objective data, such as urodynamics in the case of incontinence, in order to validate the questionnaire prior to its use in a study.

The results of the major studies into prevalence of incontinence will be presented in a table form and then will be considered individually. It can be seen from this table that it is difficult to directly compare data from these studies as the questions asked differ as do the definitions of incontinence.
Table 1 Studies of Prevalence of Incontinence

<table>
<thead>
<tr>
<th>Authors</th>
<th>Type of study</th>
<th>Number of patients</th>
<th>Any incontinence</th>
<th>Regular incontinence</th>
<th>Sought help for incontinence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thomas et al</td>
<td>Postal questionnaire</td>
<td>18 084</td>
<td>11.2-21.9%</td>
<td>4.0-16.2%</td>
<td></td>
</tr>
<tr>
<td>Yarnell et al</td>
<td>Interview at home</td>
<td>1 000</td>
<td>45%</td>
<td>2.6%</td>
<td>9%</td>
</tr>
<tr>
<td>Holst and Wilson</td>
<td>Telephone interview</td>
<td>851</td>
<td>31%</td>
<td>17%</td>
<td>35%</td>
</tr>
<tr>
<td>Brocklehurst</td>
<td>Interview at home</td>
<td>2 124</td>
<td>14%</td>
<td></td>
<td>47%</td>
</tr>
<tr>
<td>Jolleys</td>
<td>Postal questionnaire</td>
<td>833</td>
<td>41%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Harrison and Memel</td>
<td>Postal questionnaire</td>
<td>314</td>
<td>53%</td>
<td></td>
<td>13%</td>
</tr>
</tbody>
</table>

In the study by Thomas et al incontinence in women increased with age and with parity. 4% of 15-24 year olds reported regular incontinence whereas 11.8% of 45-54 year olds and 16.0% of 75-84 year olds complained of regular incontinence. The numbers were greater but the trend the same for those women complaining of occasional incontinence. Incontinence was reported less commonly by nulliparous than multiparous women at all ages.

Of the women interviewed by Yarnell et al 45% of the women admitted to some degree of incontinence, stress incontinence was reported by 22% and urge incontinence by 10% of women and 14% complained of mixed symptoms. 3% of the women reported that the incontinence interfered with their social or domestic life yet only half of these women had sought medical help for their problem.
Holst and Wilson interviewed 851 women, 31% of the women had some degree of incontinence and 17% had regular incontinence. Only one third of the women with incontinence had sought medical help, reasons for not seeking help were that incontinence was not perceived as abnormal (81%) or low expectation of benefit from treatment (10%). The more socially debilitating the incontinence the more likely it was that the women would seek help.

Brocklehurst analysed a MORI poll of 4007 randomly selected adults, 2124 of the sample were women. Of the women questioned 14% admitted to some incontinence and as has been seen in previous studies the prevalence of incontinence increased with age. 80% of those with incontinence had consulted their general practitioner at some time.

Jolleyes found that the prevalence of incontinence increased to age 54 and then decreased, 31% of women age less than 25, 60% of 45-54 year olds and 20% of 75-84 year old women complained of incontinence. Rates of incontinence were higher in multiparous than nulliparous women, incontinence was not related to type of delivery.

Harrison and Memel found that the overall prevalence of incontinence was 53%, 46% of these women complained of stress incontinence, 8% complained of urge incontinence and 43% had mixed incontinence. Once again the prevalence increased with increasing parity and there was no relation between incontinence and mode of delivery. Those women who reported incontinence on the first questionnaire were sent a second questionnaire, only 46.7% were returned. Of these women 34.6% admitted to worrying about their incontinence but only 26.9% of these had spoken to their general practitioner. Reasons given for not consulting the doctor were that the incontinence was a minor inconvenience, they thought that
nothing could be done, incontinence is a usual women’s complaint and embarrassment.

2.2.3 Impact of incontinence

It is difficult to measure the impact of incontinence on quality of life as it is a very subjective issue. Different authors have tackled the problem in a variety of ways. Any questionnaire study will have the same problems as those which have already been discussed, in particular all questionnaires used should be properly validated. It is also important to consider the group that is being questioned, many of the studies have questioned women who either are attending for investigation of their incontinence or those who have been investigated and are undergoing treatment. From the data which has already been examined many incontinent women do not present for help and so how representative are the groups that are being studied? Some authors have questioned women about how their incontinence has affected their lives but without any validation of the questionnaires. Quality of life questionnaires are an excellent method of assessing the impact of incontinence on a patient’s life as they take into account the consequences of the incontinence from the patients point of view. There are a number of quality of life questionnaires that have been fully validated for use in relation to incontinence

Norton et al questioned 201 women, attending for initial assessment at an urodynamic clinic, about their urinary symptoms. Two thirds of the women described urinary incontinence as their worst symptom, of these women 57% complained of stress incontinence and 43% of urge incontinence. Two fifths of the women consulted a doctor within a year of noticing incontinence, a third delayed for up to five years and a quarter delayed for more than five years. Reasons given for delay were that they hoped symptoms would resolve, embarrassment, symptoms were thought to be
normal and fear of surgery. When questioned about the effect of incontinence on their daily lives 60% avoided going away from home and 58% felt their work was affected, significantly more than the women who were continent. When questioned about stigma attached to their incontinence 47% felt less attractive as a result of their symptoms, again significantly more than in the continent group.

Clark and Romm sent questionnaires to 90 patients who had been seen in their urodynamic clinic to ask about the effect of urinary incontinence on sexual activity in women. Unfortunately only 48 (53%) of the questionnaires were returned, 22 women had stress incontinence, 15 had detrusor instability and 7 had mixed incontinence. Clinically significant incontinence occurred in all diagnostic groups. Only 3 of 16 women reported decreased frequency of sexual intercourse however the patients did complain of decreased spontaneity as a result of concerns about odour and the need to dispose of wet pads. The numbers in this study were rather small due to the poor response rate.

Kobelt et al have reviewed the use of several quality of life questionnaires in patients with the overactive bladder (SF36, EuroQol, the Incontinence QoL Index and the King’s Healthcare Questionnaire). They found that those patients with the symptoms of frequency and incontinence had lower scores compared to the normal population. The scores improved with treatment indicating that quality of life improves as symptoms resolve.

### 2.2.4 Management of incontinence by general practitioners

Even when women do present to their general practitioner complaining of urinary incontinence they may not be managed appropriately. Briggs and Williams undertook a postal survey of general practitioners to question
about referral to the continence advisory service. They found that of 101 practitioners, 42 had never used the continence advisory service for their patients over 65 years of age. The continence advisory service, led by a trained nurse continence advisor had been operating in that area for several years and was accessible to general practitioner referrals.

In the study by Brocklehurst that has already been discussed the women were asked about the actions of their general practitioners when they consulted them. On the whole the actions were limited to taking a urine sample, referring to a specialist or prescribing tablets. Only in a minority of cases was an abdominal, rectal or vaginal examination performed therefore suggesting that medication is prescribed without clinical examination and without a diagnosis being made. Fewer than 5% were referred to a nurse or continence clinic.

Grealish and O’Dowd studied general practitioners awareness of urinary incontinence in women and their attitude to management of the incontinence. They interviewed 11 male and 9 female GPs. The GPs were aware of the prevalence and under-reporting of urinary incontinence. Many of the doctors were unhappy with their own management of incontinence. The male doctors on the whole were reluctant to perform a vaginal examination. The GPs mentioned teaching of pelvic floor exercises and bladder drill but expressed little enthusiasm about their success rate. Medication was used frequently but was generally considered ineffective or intolerable. The services of incontinence nurses were under-utilised as many GPs did not know of their availability.
2.3 The Lower Urinary Tract In Pregnancy.

2.3.1 Frequency of micturition and nocturia.

Although it has been stated over many years that frequency of micturition is very common in pregnancy, there have been few trials that have examined frequency and nocturia during pregnancy. The ideal method to study any urinary symptoms during pregnancy would be to collect a cohort of women and then follow them prospectively though pregnancy in a longitudinal study, however this would take many months due to the length of human gestation. Many of the studies that have been conducted have either been cross-sectional, using different groups of women at different gestations, or retrospective questioning women in the postpartum period, both these methods introduce bias. Another criticism of these studies is that there is no standard definition of frequency or nocturia. The data obtained in these studies is quantitative data yet instead of treating it as such the authors have used ordinal scales by applying arbitrary definitions of normal and abnormal frequency or nocturia. Some of the authors have compared the values to those of non-pregnant women which is a superior method compared to the setting of arbitrary values however it would be more appropriate to use quantitative statistics. Finally any study which includes objective data, such as in the form of a frequency volume voiding chart, is better than a purely questionnaire based study.

One of the first people to study frequency and nocturia prospectively though pregnancy was Francis. She studied 400 healthy pregnant women (nulliparous and multiparous) through pregnancy and 50 normal non-pregnant women. She found that the non-pregnant women voided up to six times during the day and only occasionally during the night whereas in the pregnant group 81% voided seven times or more by day and once or more at night. The symptoms tended to commence in the first trimester and
gradually worsen though pregnancy. Having asked 100 women to complete a record of their total fluid intake and output over a 72 hour period, 40 in the first trimester, 30 in the second trimester and 30 in the third trimester, her conclusion was that the frequency was due to polydypsia and polyuria. The women were selected ‘according to their intelligence and willingness to co-operate’ which of course could have introduced bias. Twenty of the women recorded their intake and output in all three trimesters so the study is partially longitudinal and partially cross-sectional, however the longitudinal data is limited by the small numbers.

Paboosingh and Doig studied nocturia in pregnancy with direct questioning and frequency volume charts, they defined nocturia as nighttime voiding on three nights in a one week period and so introduced error by incorrectly using ordinal scales for quantitative data. They found that 58% of women complained of nocturia in the first trimester, 57% in the second trimester and 66% in the third trimester, this was a cross sectional study. The frequency volume charts confirmed the findings of Francis, that the urine output was increased.

Stanton et al questioned 181 healthy women prospectively though pregnancy about various urinary symptoms. They defined frequency as being seven or more voids during the day and nocturia as voiding more than once at night, thus introducing error as has already been discussed. The group consisted of 83 nulliparous and 98 multiparous women. Frequency of micturition was more common in the nulliparous group and the incidence increased in both groups until term, the incidence had returned to the pre-booking level at the postpartum visit. The incidence of nocturia increased in the same way, however diurnal frequency was more common than nocturia in both groups.
Cutner recruited 165 women at 20 weeks gestation and followed them at 28 and 36 weeks gestation and 7 weeks postnatal, 119 women (71 nulliparous and 48 multiparous) completed the study. The women completed symptom questionnaires at each visit and a frequency volume chart, 58 women completed all four charts. Frequency of micturition increased during pregnancy and declined after delivery but not quite to the pre-pregnancy level. Nocturia also increased during pregnancy but did return to the pre-pregnancy level by the postnatal visit. When the relationship between the number of stated voids on direct questioning and the number on the frequency volume chart was examined it was found that the women tended to overestimate the number of voids on direct questioning.

Viktrup et al questioned 305 primiparous women on the third to fifth postpartum day. Frequency was defined as voiding seven or more times during the day and nocturia was defined as voiding twice or more during the night, this again introduces error by treating quantitative data in an ordinal way. They found that frequency and nocturia increased during pregnancy and returned to pre-pregnancy levels by three months postpartum.

Rasmussen et al sent questionnaires to 207 women, up to 18 months postpartum, and received 180 replies. The aim of their study was to examine if obesity was a predictor for postpartum urinary symptoms. They defined frequency as voiding 10 or more times per day and nocturia as voiding twice or more at night. They found that before pregnancy none of the women suffered from frequency whereas 2.1% of the normal weight women and 3.5% of the obese women suffered from nocturia. During pregnancy 30.9% of normal and 31.8% of obese women complained of frequency and 34% of normal and 50.8% of obese complained of nocturia. 6 to 18 months postpartum the frequency had decreased to 4% of normal and 1% of obese women and the nocturia had decreased to 3.2% of normal
and 5.8% of obese women. The fact that their definition of frequency is more stringent than the other studies may explain the lower rates of frequency in the women of normal weight however it would have been more appropriate to use quantitative statistics.

2.3.2 Incontinence

There have been many retrospective studies which have questioned women many years after childbirth in order to try to find the link between pregnancy, childbirth and incontinence. Once again these are all questionnaire studies with all the disadvantages that have already been discussed. As these were retrospective studies the data obtained was cross-sectional and so should be analysed as such. The studies varied in the time period between pregnancy and the time of the study, recall bias will worsen with a longer time interval. The population varied between studies, some chose a random population whereas others (for example Beck and Hsu) recruited patients attending a gynaecology clinic which would introduce bias. The response rates to the questionnaires varied greatly, the significance of the results from the studies with low response rates should be questioned. Finally the definition of incontinence varied between studies with some authors including any episode of incontinence whereas others include only regular incontinence. None of the studies used the International Continence Society definition that has already been mentioned.

The relevant studies are summarised in the following table and are then discussed in detail below.
### Table 2 Studies of Incontinence in Pregnancy

<table>
<thead>
<tr>
<th>Authors</th>
<th>Number of patients</th>
<th>Current incontinence</th>
<th>Incontinence during pregnancy</th>
<th>Incontinence after delivery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beck and Hsu</td>
<td>1000/1000</td>
<td>31.3%</td>
<td>20.2%</td>
<td>4.4%</td>
</tr>
<tr>
<td>Iosif and Ingemarsson</td>
<td>204/246</td>
<td>17.2%</td>
<td>7%</td>
<td>9%</td>
</tr>
<tr>
<td>Foldsprang et al</td>
<td>2,631/3,114</td>
<td>17.1%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Milsom et al</td>
<td>7,460/10,000</td>
<td>12.1 - 24.6%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ryhammer et al</td>
<td>242/304</td>
<td></td>
<td></td>
<td>3.3 - 6.8%</td>
</tr>
<tr>
<td>Thom et al</td>
<td>939/1,922</td>
<td>72.6%</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

In 1965 Beck and Hsu questioned 1000 women, who attended their outpatient clinic, about the presence or absence of stress incontinence, pregnancy and menstrual status and about the circumstances surrounding the onset of the incontinence.\(^{30}\) They found that 31.3% of the group complained of stress incontinence with 5% of the group describing the incontinence as a major problem. 32.9% of the incontinent group first developed incontinence whilst pregnant and continued to be incontinent after delivery, a few of this group developed permanent incontinence following the first delivery but more commonly the incontinence became gradually worse during successive pregnancies until it became permanent. 14.1% of the incontinent women first developed stress incontinence after delivery and all of this group had permanent symptoms.

Iosif and Ingemarsson sent questionnaires to 246 women who had been delivered by elective caesarean section up to seven years previously.\(^ {31}\) 77% of the women returned the questionnaires. 7% of women had had stress incontinence during the pregnancy only whereas 9% of the women had developed permanent stress incontinence with onset either during the pregnancy or in the puerperium. Although the authors state that the women
had been delivered by elective caesarean section due to a narrow pelvis, they do not state whether or not any of the women had laboured in any of their pregnancies, which could have affected the development of incontinence.

In 1992 Foldspang et al sent a questionnaire to 3114 women selected at random, 85% were returned. 17.1% of the women had experienced some type of incontinence during 1987 (the year that they were questioned about) of which 14.8% was stress incontinence. 8.6% was urge incontinence and 7.1% was mixed incontinence. The prevalence of incontinence was found to be associated with parity. Among the types of incontinence stress incontinence consistently showed the strongest association with parity. It should be remembered that this is cross-sectional data and so the findings should be confirmed with a longitudinal study.

Another study was published by Milsom et al in 1993. They sent questionnaires to 10,000 random women and had a 74.6% response rate. They found that there was an association between increasing prevalence of incontinence and increasing age. There was a linear association between prevalence of incontinence and increasing parity. The greatest increase in prevalence occurred after the birth of the first child. Once again the limitations of cross-sectional retrospective data should be remembered.

Ryhammer et al sent questionnaires to 304 women who had delivered in their department without a complete tear of the anal sphincter. This group was selected to act as a control group for another study involving women with complete tear of the anal sphincter and so the women selected were those that had delivered vaginally just before and after those women with anal sphincter tears. 80% of the women responded to the questionnaire. 3.3% of the women became permanently incontinent after their first delivery, 1% after their second delivery and 6.9% after the third delivery.
Although the selection of patients was appropriate for a study about anal sphincter tears, it is not random.

Thom et al published a postal questionnaire study in 1997. They sent out questionnaires to 1922 randomly selected women, of age 60 or over, 49% were returned fully completed. 682 women reported any incontinence over the previous year, 115 (12.3%) reported daily incontinence and 205 (30.2%) reported weekly incontinence. Of those women who were incontinent 23.3% complained of stress incontinence, 32.8% complained of urge incontinence and 32.5% complained of mixed incontinence. Women with incontinence reported more pregnancies and births. Parturition factors were examined in the 791 women with at least one live vaginal birth, only prolonged labour and use of oxytocin were significantly associated with incontinence. These final results are limited by the fact that they are a result of data dredging and so could be due to coincidence.

2.3.3 Incontinence postpartum

There have been a number of studies which have questioned women in the postpartum period. Once again these were retrospective questionnaire studies with no longitudinal data. Due to the nature of the data the results can show correlation but not causation. The time interval between delivery and questioning varied from study to study as did the definition of incontinence. These studies are summarised in the following table.
### Table 3 Studies of Incontinence Postpartum

<table>
<thead>
<tr>
<th>Authors</th>
<th>Number of patients</th>
<th>Interval since delivery</th>
<th>Incontinence at time of study</th>
<th>Factors influencing incontinence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schuessler et al</td>
<td>424</td>
<td>6 months</td>
<td>6%</td>
<td>Epidural ↓</td>
</tr>
<tr>
<td>Viktrup and Lose</td>
<td>208</td>
<td>3 months</td>
<td>13-27%</td>
<td>Epidural ↑</td>
</tr>
<tr>
<td>Viktrup et al</td>
<td>305</td>
<td>3-5 days 3 months</td>
<td>13% 4%</td>
<td>LSCS ↓</td>
</tr>
<tr>
<td>Glazener et al</td>
<td>1249</td>
<td>1 week 8 weeks 12-18 months</td>
<td>2% 6% 8%</td>
<td>LSCS ↓</td>
</tr>
<tr>
<td>Wilson et al</td>
<td>2134</td>
<td>3 months</td>
<td>3.3-34.3%</td>
<td>LSCS ↓</td>
</tr>
<tr>
<td>Rasmussen</td>
<td>207</td>
<td>6-18 months</td>
<td>11-25%</td>
<td>Obesity ↑</td>
</tr>
<tr>
<td>Brown and Lumley</td>
<td>1336</td>
<td>6-7 months</td>
<td>10.4-10.9%</td>
<td>Forceps ↑ LSCS ↓</td>
</tr>
</tbody>
</table>

In 1988 Schuessler et al questioned 424 primiparous women six months after delivery. They found that none of the women were incontinent prior to pregnancy and none of the women who were delivered by caesarean section (58) became incontinent however 6% of those who had a vaginal delivery were incontinent at six months. They found no significant connection between incontinence and birth weight, large head circumference, prolonged second stage of labour, instrumental vaginal delivery and maternal age. The women who had had an epidural for analgesia during labour had a lower incidence of incontinence. It may be expected that the women with epidurals would have had a higher incidence of complex labours and so a higher rate of incontinence. The authors explanation of the results is that the epidural relaxes the pelvic floor musculature and so prevents the damage that leads to stress incontinence.
but there is no evidence for this and the results may be just due to coincidence.

Viktrup and Lose followed up this study with one in which 208 primiparous patients were questioned three months postpartum. The group selected initially was all the primiparous patients who delivered in a six month period, however as the aim of the study was to study the effect of epidurals on the incidence of incontinence, the number of patients who had an epidural for analgesia in this original group was too low, so all primiparous women who delivered having had an epidural for analgesia in labour in the subsequent 25 months were also included. This method of selecting patients could well have introduced errors as the two groups of women did not deliver in the same time period. They found that 27% of women with an epidural developed incontinence de novo after delivery compared to 13% of women who delivered without an epidural so the hypothesis that epidural analgesia relaxes the pelvic floor and so protects against damage leading to stress incontinence was not supported.

In another study by Viktrup et al 305 primiparous women were questioned three to five days after delivery. They were questioned about urinary symptoms prior to and during pregnancy and after delivery. 293 women were followed up at three months postpartum. None of the women delivered by caesarean section developed incontinence after delivery whereas 13% of those who had had a vaginal delivery were incontinent immediately postpartum and 4% remained incontinent at three months.

In 1995 Glazener et al published a questionnaire survey of 1249 postnatal patients at one and eight weeks postpartum and 12 to 18 months after delivery. The women were questioned about many aspects of postnatal morbidity including incontinence. The women were selected at random, the response rates to the three questionnaires were 89.8%, 90.1% and 86.1%.
However the third questionnaire was only sent to half the original sample of women, those delivering in the second six months of the study. 2% of women complained of incontinence in the immediate postpartum period, eight weeks after delivery 6% of women complained of incontinence and by 18 months after delivery 8% of women complained of incontinence. Urinary incontinence was the morbidity which was least often treated. The rising rate of incontinence as time elapsed after delivery may be explained by the fact that women with morbidity are more likely to return the questionnaires therefore introducing error also the women who were sent the third questionnaire were not the whole sample which might have introduced error. Another explanation for the increasing rates of incontinence over time may be that the women were becoming more active over time, age may play a part however whether ageing over eighteen months is a significant factor in the development of incontinence is not known. There is no mention of breastfeeding rates in the study and there may be a connection with breastfeeding and incontinence.

Wilson et al published a postal questionnaire survey in 1996 they sent questionnaires to 2134 women at three months postpartum, 70.5% were returned. They found that 34.3% of women complained of some incontinence and 3.3% of women complained of daily incontinence. Those women delivered by caesarean section had a significantly lower prevalence of incontinence compared to those that had delivered vaginally, however there was no significant difference between those women delivered by elective caesarean section and those who had caesarean sections during the first or second stage of labour. Those women who had had three or more caesarean sections had a prevalence of incontinence similar to those that had delivered vaginally. As this is retrospective data is shows a correlation but not causation.
Rasmussen et al. studied 207 women six to eighteen months after delivery in order to investigate the relationship between pre-pregnancy obesity and urinary symptoms prior to and during pregnancy and postpartum. Prior to pregnancy, 10.6% of the obese women complained of stress incontinence whereas 1% of the women of normal weight had stress incontinence. During pregnancy these values increased to 39% and 23% respectively and six to eighteen months postpartum, 25% of obese women and 11% of normal weight women remained incontinent.

In 1998 Brown and Lumley published a postal survey of 1336 women who had delivered six to seven months previously. The aim of the study was to determine the prevalence of maternal physical and emotional health problems after delivery. 10.4% of primiparae and 10.9% of multiparae complained of urinary incontinence. When the rates of incontinence were compared to mode of delivery, the results were as follows: 10.9% after vaginal birth, 18.2% after forceps or vacuum extraction, 6.8% after emergency caesarean section and 2.4% after elective caesarean section.

2.3.4 Incontinence during pregnancy

There have been a number of studies, conducted in the postpartum period, enquiring into incontinence prior to, during pregnancy and postpartum. Iosif questioned 1500 women in the immediate postpartum period, 1411 completed the questionnaire, those women who complained of urinary incontinence were then referred to an urologist. Six to twelve months after delivery, all those women who had complained of incontinence on the initial questionnaire were sent a further questionnaire, there was a 90% response rate. A third questionnaire was sent to 300 women selected at random from those who had not been incontinent from the initial questionnaire. All three questionnaires were different. 22.7% of women complained of incontinence in the first questionnaire, 95% of these women
replied to the second questionnaire. 8.5% of these women were incontinent prior to pregnancy, 72.5% became incontinent during the pregnancy and 19% were incontinent after delivery of which 11% were still incontinent six to twelve months after delivery. Of the 300 women who were sent the third questionnaire 88% responded, 6% described mild stress incontinence one to three months after delivery. The data during pregnancy and prior to pregnancy is all retrospective in this study which introduces recall bias, also all three questionnaires that were used were different which could add error.

Viktrup et al studied 305 primiparous women on the third to the fifth postpartum day, they were then questioned again on the telephone three months postpartum and those that reported stress incontinence at the second interview were interviewed again at one year. The same questionnaire was used each time. Twelve women were lost to follow-up at three months and one further woman was lost to follow-up at one year. 4% of women were incontinent prior to pregnancy, 32% became incontinent during pregnancy, 19% were incontinent during the puerperium, 6% remained incontinent at three months and 3% were incontinent at one year postpartum. Among the women that were not incontinent prior to or during pregnancy, of the 35 that were delivered by caesarean section none became incontinent postpartum compared to 13% of those who had a vaginal delivery, at three months 4% remained incontinent. Once again the problem with this study is that it relies on retrospective data.

Marshall et al distributed questionnaires to 2062 women two to three days after delivery, they then mailed a follow-up questionnaire at three months postpartum to a cluster of 335 women who had delivered during a random month, of these 152 (45%) were returned. Finally a further questionnaire was sent to a random sample of 180 women randomly selected from those multiparous women who had complained of incontinence on the first
questionnaire, 72 (40%) replied. 59% of women experienced some leakage of urine immediately postpartum, the incidence increased with parity. 31% of the multiparous women and 11% of the primiparous women were incontinent prior to pregnancy. At three months postpartum 63% of the women that returned the questionnaire were still leaking. The final questionnaire revealed that 33% of the women that replied were still leaking. The high rates of incontinence continuing postpartum may well be due to the low rates of response to the second and third questionnaire.

Chiarelli and Campbell performed a structured interview on 304 women on the postnatal ward. They questioned the women about incontinence in the last month of pregnancy and about any advice they had been given about bladder control during pregnancy. They found that 64% of women reported incontinence during pregnancy. Those women who had had regular bouts of coughing during the pregnancy were more likely to report incontinence. Only 23% of the women reported having spoken to a healthcare professional regarding loss of bladder control. As this study did not separate the women according to parity and also did not question about incontinence prior to pregnancy the results are rather meaningless. It is the long term morbidity associated with incontinence following childbirth that is important rather than the short term incontinence during pregnancy, this study did not address incontinence postpartum at all.

In 1998 Marshall et al published a questionnaire study involving 7771 women who were questioned on day two or three postpartum. 1.4% of women were incontinent prior to but not during the pregnancy. The women were divided into groups according to parity: primiparous, multiparous (2-4) and multiparous (5+). The incidence of incontinence before pregnancy increased from 5% in the primiparous group to 21.4% in the multiparous (2-4) group to 32.2% in the multiparous (5+) group. The onset of incontinence during pregnancy was 50%, 44.5% and 36.1% respectively.
Once again this study did not address the issue of postpartum incontinence presumably because the questionnaire was issued so soon after delivery, however by missing out on such important data it looses its value.

2.3.5 *Detrusor instability and pregnancy*

Up to this point the studies discussed have focussed on incontinence of urine generally in relation to pregnancy and childbirth. The number of studies examining the relationship between pregnancy and genuine stress incontinence and detrusor instability are very limited. Few authors have performed urodynamics in pregnancy and so as a result the number of studies with proven genuine stress incontinence or detrusor instability are very small. Some authors have instead asked about symptoms such as frequency, urgency and urge incontinence in order to estimate the prevalence of detrusor instability during pregnancy. In those studies that have done urodynamic studies in pregnancy there has been poor correlation between urodynamic findings and symptoms.

In 1991 Cutner et al published a study of 47 women who underwent urodynamics prior to termination of pregnancy. 26 of the women were nulliparous and 21 were parous. 62% complained of urgency (28% mild, 15% moderate, 19% severe) 18% complained of urge incontinence (6% mild, 6% moderate, 6% severe) and 15% complained of stress incontinence (11% mild, 4% moderate). 23% of the women had phasic detrusor contractions on cystometry. Detrusor instability appeared to be more common in women who complained of urgency and urge incontinence but these differences were not significant. The urodynamic findings did not correlate with symptoms. Their conclusions were that although urgency is a very common symptom in early pregnancy it does not vary with the urodynamic findings of detrusor instability. They were unable to explain the high incidence of detrusor instability. The numbers in this study are
small. The results are limited as they only refer to the first trimester of pregnancy.

Viktrup et al questioned 305 primiparous women on day three to five postpartum and at three months postpartum, about urinary symptoms during pregnancy and after delivery. The incidence of urge incontinence was much lower than stress incontinence throughout the study. 6 women had urge incontinence prior to pregnancy, 30 women developed it during pregnancy and 12 women developed urge incontinence after delivery. In the group of women who did not have urge incontinence prior to or during pregnancy, of those delivered by caesarean section none developed urge incontinence whereas of those with a vaginal delivery 6% developed urge incontinence, this difference is not significant. Once again this study relies on retrospective data and so suffers from recall bias, also it is purely a questionnaire study and so does not have any means to correlate between the symptoms of urgency and urge incontinence and detrusor instability.

In the study of Rasmussen et al that has already been mentioned, 1.1% of normal and 10.6% of obese women complained of urge incontinence prior to pregnancy, 6.7% of normal and 16.5% of obese women complained of urge incontinence during pregnancy and none of the normal and 4.7% of the obese women complained of urge incontinence six to eighteen months after delivery. As previously stated this is a retrospective study and so has inherent error.

From these studies it would seem that detrusor instability is much less of a problem during pregnancy and after delivery compared to stress incontinence of urine.
2.3.6  **Prospective studies of stress incontinence during pregnancy and after delivery**

In comparison to the number of retrospective studies on this subject there are very few prospective studies though pregnancy. Most of these studies have included multiparous and primiparous women, they also vary widely as to how often the women were questioned during pregnancy and for the length of follow up postpartum. These studies are summarised in the following table and will then be discussed in further detail:

**Table 4 Prospective Studies of Stress Incontinence During Pregnancy & Postpartum**

<table>
<thead>
<tr>
<th>Authors</th>
<th>No. of patients</th>
<th>Primips or Multips</th>
<th>Incontinence in pregnancy</th>
<th>Length of follow-up</th>
<th>Incontinence postpartum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Francis</td>
<td>400</td>
<td>Both</td>
<td>53% primips</td>
<td>6 months</td>
<td>9%</td>
</tr>
<tr>
<td>Stanton et al</td>
<td>181</td>
<td>Both</td>
<td>38.6% primips 40.4% multips</td>
<td>7 weeks</td>
<td>5.8% primips 10.6% multips</td>
</tr>
<tr>
<td>Dimpfl et al</td>
<td>350</td>
<td>Both</td>
<td>53.5%</td>
<td>12 weeks</td>
<td>6% primips 7% multips</td>
</tr>
<tr>
<td>Cutner</td>
<td>119</td>
<td>Both</td>
<td>39%</td>
<td>7 weeks</td>
<td>9%</td>
</tr>
<tr>
<td>Sampselle et al</td>
<td>72</td>
<td>Primips</td>
<td>77%</td>
<td>12 months</td>
<td>67%</td>
</tr>
</tbody>
</table>

The first prospective study was by Francis. She questioned 400 unselected patients attending an antenatal clinic. She found that 53% of primiparous women and 83% of multiparous women complained of stress incontinence during pregnancy. Of the primiparous women 42% of them had occasional incontinence prior to pregnancy. Of the multiparous women 13% had developed incontinence whilst not pregnant, 47% had developed the incontinence during a previous pregnancy and 40% had developed it.
during the current pregnancy. None of the patients developed stress incontinence for the first time in the puerperium and of the women that were incontinent during the pregnancy, 91% found that the incontinence resolved postpartum with only 9% complaining of stress incontinence at follow up six months postpartum. Once present the incontinence (if not permanent) reappeared in a gradually worsening degree with each subsequent pregnancy. The onset of incontinence was quite evenly distributed between the trimesters, 30% in the first, 31% in the second and 39% in the third, once present the severity of incontinence gradually worsened until term. 20 of the women who had complained of stress incontinence during pregnancy were delivered by caesarean section, none of these remained incontinent postpartum.

Francis concluded that those women who complain of stress incontinence in middle life are destined to do so from an early age, and it is pregnancy rather than labour that reveals an intrinsic defect in the sphincter mechanism. The numbers of primiparous women who were incontinent prior to pregnancy (42%) in this study seems rather high. This study made a major breakthrough as it was the first prospective study through pregnancy, however by studying multiparous women as well as primiparous women the issues become rather complex as the multiparous women will be influenced by their previous pregnancies and deliveries.

Stanton et al questioned 181 women, attending the antenatal clinic, prospectively through pregnancy. The questions were asked and recorded by a nurse at visits to the antenatal clinic at booking (average gestation at booking 11.8 weeks for primiparous and 12.4 weeks for multiparous women), 32, 36, 38, 40 weeks gestation and at the postnatal visit. Both frequency of micturition and stress incontinence increased during pregnancy and reduced after delivery. The greatest incidence of stress incontinence was at 38 weeks of pregnancy (38.6% primiparous, 40.4%
multiparous women), the incidence had fallen to 5.8% for primiparous and 10.6% for multiparous women at the postnatal visit.

The problem with this study is that it again included both primiparous and multiparous women. Although the women were questioned in the first trimester they were not questioned at all through the second trimester. As the last questions were asked at the postnatal visit, which is usually at six weeks (the timing of the visit was not stated in the paper), the postpartum follow up is not really long enough. There has not been any longitudinal work to appraise the appropriate time interval for assessment of incontinence post partum, however in the study by Glazener already discussed the incidence of incontinence increased over eighteen months post partum.

Dimpfl et al questioned 350 unselected women attending the antenatal ward during the last three months of pregnancy.48 These women were followed up at six and twelve weeks postpartum. All women with incontinence prior to pregnancy were excluded. 60 women were delivered by caesarean section, none of these were incontinent postpartum, these women were excluded from further analysis. 53.5% of women developed incontinence during pregnancy and 6.2% remained incontinent at three months postpartum. Of those that were continent though pregnancy 3.7% became incontinent postpartum. The differences between primiparous women with persisting incontinence (6%) and multiparous women (7%) were slight. The incidence of persisting incontinence after a spontaneous vaginal delivery (6.1%) was lower than after an operative delivery (10% after forceps and 9.1% after vacuum extraction).

Once again this study investigated primiparous and multiparous women, also the women were not recruited until quite late in pregnancy and so data from earlier in pregnancy was missed.
Cutner studied 119 women prospectively through pregnancy at 20, 28 and 36 weeks gestation and 7 weeks postnatal with questionnaires, frequency volume charts and urodynamics. Urodynamics was performed on 127 occasions, 37 women had urodynamics at 28 weeks, 26 at 36 weeks and 32 postnatal, 21 women had the test performed at all three visits. They found that one woman had genuine stress incontinence demonstrated at 28 weeks, two women at 36 weeks and none of the women had genuine stress incontinence at the postpartum visit. The prevalence of detrusor instability is shown in the following table:

**Table 5 Prevalence of Detrusor Instability During Pregnancy & Postpartum (Cutner)**

<table>
<thead>
<tr>
<th></th>
<th>28 weeks gestation</th>
<th>36 weeks gestation</th>
<th>7 weeks postpartum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phasic detrusor contractions</td>
<td>3</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>low compliance</td>
<td>6</td>
<td>7</td>
<td>5</td>
</tr>
<tr>
<td>Combined (detrusor contractions and low compliance)</td>
<td>4</td>
<td>2</td>
<td>1</td>
</tr>
</tbody>
</table>

None of the changes are significant but there is a trend towards improvement in the postpartum period. The numbers of women undergoing urodynamics in this study is very small.

Sampselle et al performed a prospective study in which 72 primiparous women were questioned through pregnancy, and for twelve months postpartum, about symptoms of stress incontinence, in addition every woman underwent a standing stress test at each visit, only 59 women completed the study. Unfortunately they excluded all women who were delivered by Caesarean section (19%) from the postpartum analysis which therefore included only 48 women. The prevalence of stress incontinence
increased from 20 weeks gestation to a maximum prevalence at 35 weeks gestation and then declined after vaginal delivery. At all stages the numbers of women who reported symptoms but did not have demonstrable incontinence on stress testing was much greater than the number of women who had reported incontinence and had demonstrable stress incontinence.

The numbers of women in this study are rather small, also it is unfortunate that they excluded women who had been delivered by caesarean section as these provide a group in which the effect of pregnancy rather than vaginal delivery can be studied.

In summary there are five prospective studies examining the relationship between pregnancy, delivery and incontinence. These studies have found that the prevalence of incontinence increases in pregnancy and decreases after delivery. There is a group of women who remain permanently incontinent after delivery, it is not clear whether this is a result of the pregnancy or of the delivery. In order to assess the influence of pregnancy, rather than delivery on the onset on incontinence it is necessary to exclude all multiparous women as their previous deliveries will influence the results, to date there has only been one prospective study of primiparous women with several questionnaires through pregnancy. This study was rather small as only 59 women completed the study, also all women who were delivered by caesarean section were excluded, it may be that this group could have provided information as to the effect of pregnancy rather than delivery on the continence mechanism.
2.4 Pathophysiology of incontinence in relation to pregnancy and childbirth

There are several different theoretical mechanisms as to why stress incontinence develops in relation to pregnancy and delivery \(^{50}\), these include:

1. neurological urethral sphincter damage

2. primary muscle damage to the urethral sphincter

3. hormonal effects from the increased progesterone levels in pregnancy leading to relaxation of pelvic ligaments and smooth muscle

4. a reduction in the normal increase in the vascular pulsations in the periurethral plexus during pregnancy

5. a reduction in the normal increase in the total or functional urethral length seen in pregnancy

6. loss of urethral support \(^{51}\)

Each of these will now be considered in greater detail.

2.4.1 Neurological sphincter damage

The evidence for neurological damage to the urethral sphincter comes from studies of the pelvic nerves. Much of this work involved the use of spinal, perineal and pudendal nerve motor latencies. It was assumed that increased pudendal nerve motor nerve latency was an indicator of damage to the nerve supply of the urethral striated sphincter muscle and so was an appropriate test in the investigation of the cause of incontinence. It is now known that tests of nerve conduction velocity are of limited value as they
measure conduction of the nerve fibres rather than levels of innervation and so they are a poor measure of damage to the urethral sphincter. A prolonged motor latency is used as a measure of denervation, however what is assessed is the conduction of the fastest conducting fibres and not the integrity of the nerve as a whole. The latency is prolonged if all the fast-conducting fibres are damaged. To further complicate matters there is controversy as to whether the urethral rhabdosphincter is innervated by the pelvic splanchnic nerves or the pudendal nerve and so the most appropriate test of neurological damage to the rhabdosphincter would be EMG studies of the sphincter muscle itself. There have been concentric needle EMG and single fibre EMG studies performed in pregnancy, these studies will be discussed below, it should be noted however that none of the studies have performed needle EMG of the urethral rhabdosphincter itself presumably as it is small and not very accessible. Instead the more accessible pubococcygeus and external anal sphincter muscles are studied but this assumes the same innervation as the urethral rhabdosphincter which is in doubt. As these muscles are anatomically and functionally different from the urethral sphincter these results must be treated with caution.

Snooks and Swash have studied spinal, perineal and pudendal nerve motor latencies in patients with faecal and urinary incontinence. They interpreted that the increased latencies, that they found in patients with incontinence, indicated damage to the nerve supply of the urethral sphincter. They then studied women after delivery and found that the pudendal nerve terminal motor latency was prolonged 48-72 hours after vaginal delivery (but not after caesarean section), but in 60% of these women it had returned to normal at two months post delivery. Five years later the pudendal nerve terminal motor latency was again prolonged. Single fibre EMG studies of the external anal sphincter were performed
two months and five years after delivery and revealed increased mean fibre 
density in the sphincter muscle, compared to the antenatal value, indicating 
damage to the pudendal nerve. They interpreted that damage to the 
pudendal nerve indicated damage to the innervation of the striated urethral 
sphincter however as has been discussed there is doubt as to whether or not 
the sphincter is innervated by the pudendal nerve.

Smith et al measured the pudendal nerve conduction time to the striated 
urethral sphincter in women with stress incontinence, genitourinary 
prolapse or both and compared them with women with normal urinary control. 
Women with stress incontinence, with or without prolapse, had a 
significantly prolonged conduction time to the striated urethral sphincter 
compared with women with normal urinary control. The nerve conduction 
times to the pubococcygeus muscle and to the external anal sphincter were 
also increased in women with stress incontinence or prolapse or both. This 
work was interpreted as meaning that neurological damage to the urethral 
sphincter is important in the development of stress incontinence of urine. 
This study relates the neurophysiological findings to urodynamic findings 
in an attempt to relate neurology to function, however it relies on 
conduction studies.

Tetzschner et al measured pudendal nerve terminal motor latency in 
women through pregnancy and after delivery. They measured the 
pudendal nerve terminal motor latency at 14, 30 and 36 weeks of 
pregnancy and 12 weeks after delivery, the women were also questioned 
about any faecal or urinary incontinence. There was no significant change 
in the pudendal nerve terminal motor latency during pregnancy however it 
was significantly prolonged after delivery. No difference in pudendal nerve 
terminal motor latency between continent and incontinent women was 
found. The authors concluded that the increase in pudendal nerve terminal 
motor latency after delivery indicated damage at the time of delivery. As
there was no significant relationship with incontinence they concluded that factors other than pudendal nerve damage are important for the development of incontinence during pregnancy and after delivery. This may well be true as we now know that pudendal nerve damage does not necessarily indicate urethral sphincter damage.

Gilpin et al. studied muscle biopsies from the pubococcygeus of asymptomatic women and those with stress incontinence of urine, those women with incontinence all had abnormal single fibre EMG recordings. They found that in the symptomatic women there was a significant increase in the number of muscle fibres showing pathological damage indicating partial denervation of the pelvic floor. There was a difference between the anterior and posterior parts of the pubococcygeus, the significant differences in incontinent and dry women were in the posterior part of the muscle. They then concluded that the changes in the muscle combined with the single fibre EMG results confirmed that nerve fibre damage contributes to the symptom of stress incontinence of urine. As the muscle damage was apparent in the posterior part of the muscle they then hypothesised that this may be due to damage at the time of childbirth. Further work done around the time of delivery would be need to confirm this. The different findings in different areas of the muscle leads to concerns about EMG studies as the results could be affected by which part of the pubococcygeus is studied.

Smith et al. performed single fibre EMGs of the pubococcygeus muscle in 69 asymptomatic women and 105 women with stress incontinence or genitourinary prolapse or both. There was an association with increased fibre density and increasing age, it was also significantly greater in the women with stress incontinence or prolapse or both compared to asymptomatic women indicating partial denervation of the pelvic floor with subsequent reinnervation. As this is a cross sectional study it is not possible
to definitively link denervation of the pelvic floor and the development of incontinence.

Allen et al studied the effect of childbirth on the nerves of the pelvic floor of nulliparous women. They performed an assessment of pelvic floor muscle strength using a digital perineometer and concentric EMG of the pubococcygeus muscle at 36 weeks of pregnancy, 2-5 days following delivery they measured pudendal nerve conduction to the urethral sphincter, pubococcygeus and anal sphincter and repeated the assessment of the pelvic floor strength. Finally at two months following delivery they repeated all three tests: pelvic floor strength, pudendal nerve conduction and concentric EMG. There was a wide variation in the power of the pelvic floor between women however there was a significant reduction in power after delivery with some recovery at two months but not to the original antenatal level. The concentric EMG was normal in all women antenatally, at two months post delivery it was abnormal in four women all of whom complained of stress incontinence. Analysis of the individual motor units potentials showed a significant increase in the duration of the motor unit potential and in the proportion of polyphasic potentials postnatally indicating damage to the innervation of the pelvic floor muscles, the three women delivered by elective caesarean section had no change in their EMG values. There was no significant difference between the pudendal nerve conduction tests done immediately postpartum and two months later. These findings support the theory that childbirth causes damage to the pelvic nerves, hence damaging the urethral support from the pelvic floor and perhaps also direct damage to the innervation of the urethral sphincter. However as has already been discussed the tests used are not appropriate for assessing neurological damage to the urethral rhabdosphincter.

Mallett et al followed up the work of Allen, they were able to contact 76 of the original 96 women and followed them as a longitudinal cohort study.
31 of the subjects agreed only to give a history and the remaining 45 underwent physical examination, Q-tip test, concentric needle EMG, pudendal nerve latencies and perineometer measurements. 60% of the patients complained of some stress incontinence and 26% felt that their incontinence was a problem. The incontinent women had significant longer measurements of motor unit duration, urethral sphincter latencies and less muscle strength on perineometry. Further childbearing had no effect on outcome measures suggesting that the majority of damage occurs at first delivery. Once again this work is limited by the choice of tests used. The high percentage of women complaining of incontinence may be due to a bias in those followed up, however as this follow up study took place six years after the original study it may be that increasing age contributed to the increased incontinence. The original study took place eight weeks after delivery when women would probably not reached full normal activity, it therefor may be this study had a high percentage of incontinence as the women were very active.

2.4.2 Primary muscle damage to the urethral sphincter

Primary muscle damage, caused by pregnancy or delivery, in the urethral sphincter is almost impossible to study directly as the sphincter muscle is too small to biopsy. Tapp et al attempted to study the effect of vaginal delivery on the urethral sphincter indirectly by comparing urethral pressure profile measurements in two groups of women. The first group had competent urethral sphincter mechanisms, shown by absence of leakage of contrast medium per urethra at videocystourethrography, the second group of women complained of stress incontinence and had proven genuine stress incontinence at videocystourethrography. There was no difference between the two groups in respect of parity, number of vaginal deliveries or birth
weight of the heaviest baby. In the group with competent urethral sphincter mechanisms there was significant negative correlation between the resting and stress areas, under the urethral pressure profile curve distal to the point of maximal urethral pressure, and the number of vaginal deliveries. There was a positive correlation between transmission pressure ratios in the proximal ¼ of the urethra and the number of deliveries and a negative correlation in the distal ¼ of the urethra. The authors interpreted this as meaning that an increasing number of vaginal deliveries is associated with poorer function of the distal urethra but there is proximal compensation in women who are not incontinent. They did not carry out similar statistical analysis of the urethral pressure profile results in the women with incontinence but the implication is that women with incontinence do not have the proximal compensation for the damage to the distal urethral sphincter mechanism caused by increasing numbers of vaginal deliveries. Urethral pressure profile measurements are now known to be of very limited value in assessment of urethral sphincter function because of lack of reproducibility and accuracy. In particular urethral pressure profiles have been found to be particularly poor in the diagnosis of genuine stress incontinence.

Khullar et al used three-dimensional perineal ultrasound to study the urethral rhabdosphincter. They found that in women with stress incontinence the rhabdosphincter was significantly smaller and contained hypoechoic areas indicative of damage. Athanasiou et al used three-dimensional perineal ultrasound to measure the volume of the urethral sphincter, they found that the urethral sphincter was significantly smaller in women with stress incontinence. Both of these studies would indicate that there is damage to the urethral sphincter mechanism however they do not differentiate between neurological damage or direct muscle damage. The use of perineal ultrasound has not been validated with post-mortem studies.
Toozs-Hobson et al have used three-dimensional ultrasound to measure the urethral sphincter prior to, and six months after delivery. Of 113 primiparous women recruited, 78 completed their six month follow up. 44% were incontinent during pregnancy and 27% remained incontinent after delivery. There was no difference in the sphincter volume antenatally or postnatally, in women who had no antenatal symptoms regardless of postnatal symptoms. Women who remained incontinent after delivery had significantly smaller antenatal sphincter volumes compared to those whose incontinence resolved after delivery. This would imply that there is a group of women in which damage occurs to the urethral sphincter during pregnancy which then makes them at much higher risk of incontinence after delivery, however it may be that this group of women had inherently smaller sphincters before they became pregnant. Work has been done to correlate intraurethral ultrasound with urethral histology at post-mortem but this work has not been performed using three-dimensional ultrasound.

2.4.3 Hormonal effects

The hormonal changes in pregnancy, and in particular the increased progesterone levels may lead to relaxation of the pelvic ligaments and smooth muscles and so to stress incontinence. A direct cause and effect relationship has not been demonstrated.

Landon et al studied the mechanical properties of fascia during pregnancy by sampling the rectus sheath at the time of caesarean section and comparing it to rectus sheath from non pregnant women at the time of gynaecological surgery. They found that pregnancy was associated with a significant reduction in stiffness compared to the non pregnant controls, this change may be as a result in the change in hormones during pregnancy. The control patients were considerably older than the pregnant patients which could have a significant effect on the results.
They have continued this work by comparing fascia from pregnant patients with and without stress incontinence. They found that the ultimate tensile strength was reduced, the stress required for a 10% extension was reduced and tissue thickness was increased in the incontinent women compared to the continent women. All of this work is limited by the fact that measurements are performed on tissue taken from the rectus sheath rather than the pelvic fascia or muscles, which are actually the tissues that are involved in the maintenance of continence. The assumption is that the pelvic tissues will respond to pregnancy in the same way as the rectus sheath but there is no evidence as to the truth of this assumption. Even taking this into account, a further assumption is that the changes in the mechanical properties of the fascia are due to hormonal changes of pregnancy.

King and Freeman assessed bladder neck mobility with perineal ultrasound prospectively during pregnancy and at 10 – 14 weeks after delivery in 116 nulliparous women. They found that the women with postpartum urinary stress incontinence had a greater antenatal bladder neck mobility than those women who were continent postpartum. There were no significant differences in any labour or delivery variables in the two groups. The increase in bladder neck mobility postpartum above the antenatal levels was similar in the continent and incontinent groups, suggesting that delivery did not cause greater tissue trauma in the incontinent group. They concluded that there is evidence for a constitutional risk factor (eg defective pelvic floor connective tissue) in the development of postpartum stress incontinence.

2.4.4 Vascular pulsations

During pregnancy the amplitude of transmitted vascular pulsations recorded from the periurethral plexus are increased, this may be due to the
physiological increase in blood volume. Pregnant women with stress incontinence have a significant decrease in the amplitude of vascular pulsations in the periurethral plexus compared to pregnant continent women. It may be that women who do not manifest the increase in the amplitude of the vascular pulsations may experience some degree of defective pressure transmission and so be predisposed to genuine stress incontinence.

2.4.5 Total and functional urethral length

Work has been done using urethral pressure profiles in pregnancy and after delivery with conflicting results. Iosif et al studied urethral pressure profiles in 14 primiparous women through pregnancy and after delivery, they found that the functional length of the urethra and the urethral closure pressure both increased through pregnancy. Iosif and Ulmsten then studied 12 women with stress incontinence through pregnancy and after delivery and found that there was no increase in urethral length during pregnancy compared to the increase previously seen in continent pregnant women. The incontinent women had a low urethral closure pressure at rest which did not increase sufficiently through pregnancy to compensate for the increase in bladder pressure through pregnancy. Both of these studies are limited by their small size. Iosif et al then performed urethral pressure profiles postpartum in 62 women and found that the women with troublesome stress incontinence had a shorter urethral length and lower urethral resting pressure than women who had only occasional leakage.

In contrast to these results van Geelen et al studied 43 primiparous women through pregnancy and after delivery. They found that the urethral closure pressure and the functional urethral length did not change in pregnancy or in those women who were delivered by caesarean section.
Those women who delivered vaginally did have a decreased urethral closure pressure and reduced functional urethral length postpartum.

Wide variation has been found in urethral pressure profile measurements in women with genuine stress incontinence which would explain the inconsistencies between the studies. Urethral pressure profile measurements are not thought to be an accurate test for the diagnosis of genuine stress incontinence. In fact urethral pressure profiles have been found to have significant inter- and intraindividual variation making them inaccurate and of no value in the assessment of urethral sphincter function.

2.4.6 Loss of urethral support

Finally the evidence for loss of urethral support as a cause for stress incontinence. Klutke et al studied the paraurethral and bladder neck areas with MRI in patients with stress incontinence and normal controls. They identified direct musculofascial attachments originating from the levator muscle to the bladder neck and proximal urethra, the urethropelvic ligaments. In the continent women these ligaments supported the bladder neck and proximal urethra. In the women with stress incontinence the urethropelvic ligaments extended downwards, no longer supporting the bladder neck and proximal urethra. The accuracy of MRI studies of the pelvic tissues has been validated with cadaver studies.

Other studies have shown that urethral mobility is increased following vaginal delivery and so this loss of urethral support may lead to stress incontinence following pregnancy and delivery. Peschers et al studied 25 primigravidae, 20 multiparae and 10 women due to have elective caesarean section. Each woman was examined at 36 to 42 weeks of pregnancy and 6 to 10 weeks after delivery, vesical neck position at rest, during Valsalva and with maximal pelvic floor contraction was measured with perineal...
ultrasound. They found that the bladder neck was significantly lower at rest in women who had had a vaginal delivery compared to nulligravid controls and those who had had a caesarean section. The use of perineal ultrasound has not been validated with post-mortem studies.

Toozs-Hobson et al studied 76 primigravid women in the third trimester and at six weeks postnatal, with transperineal and transvaginal ultrasound. They found a significant difference in the incidence of damage to the levator ani in those that had delivered vaginally. The displacement of the vaginal sulci laterally and vertically was shown to be significantly different between the two routes of delivery. Once again this method of investigation has not been validated with post-mortem studies.
2.5 Faecal Incontinence

Faecal incontinence occurs on a daily or weekly basis in 2% of the adult population and in 7% of healthy independent adults over the age of 65\textsuperscript{82}. It is difficult to obtain accurate figures for the prevalence of faecal incontinence as it is a subject that women are ashamed to talk about, any figures quoted are probably an underestimation\textsuperscript{83,84}. Leigh and Turnberg questioned 76 patients with diarrhoea, attending a gastrointestinal clinic, about faecal incontinence\textsuperscript{85}. They found that 51% of the patients suffered from faecal incontinence however of these only 49% had spontaneously mentioned the incontinence. This was a very select group of patients with relatively high risk for faecal incontinence, however it does illustrate the point that patients are reluctant to present with symptoms of faecal incontinence.

Thomas et al conducted a survey of 16,631 patients of 12 general practitioners\textsuperscript{86}. Each patient who admitted to faecal incontinence on the postal questionnaire was then interviewed directly to confirm their symptoms, 65% attended the interview. In the group of women aged 15 to 64, 63 women (total 6205) reported incontinence in the postal survey, 24 women were interviewed and only 4 of those women were confirmed to have faecal incontinence. The authors explained the high discrepancy between the postal questionnaire and the interview by the fact that they felt that the questions about faecal incontinence had been misinterpreted by the responders.

Johanson and Lafferty surveyed individuals attending either their primary care physician or a gastroenterologist\textsuperscript{87}. 881 individuals were questioned, 586 by their primary care physician and 295 by the gastroenterologists. 18.4\% of the patients admitted to some form of faecal incontinence, 2.7\% had incontinence daily, 4.5\% weekly and 7.1\% at least once a month. The
prevalence of faecal incontinence increased with age. Only 33.8% of those patients with faecal incontinence had ever consulted a doctor about their problem.

2.5.1 The anal sphincters and maintenance of continence

The anal sphincter comprises the internal anal sphincter (smooth muscle) and the striated external anal sphincter. The combined internal and external sphincter complex is nearly cylindrical, encircling the anal canal, it is on average 28mm long and 18.3mm thick. 54% of the anterior thickness is made up of the internal sphincter the remainder is the external sphincter. The external sphincter comprises three parts; the subcutaneous, the superficial and the deep. The deep external anal sphincter is inseparable from the puborectalis muscle posteriorly. The internal anal sphincter is a continuation of the circular smooth muscle of the bowel.

The internal sphincter is responsible for 50-85% of the resting tone of the sphincter, the external sphincter accounts for 25-30% and the remaining 15% is attributed to expansion of the anal cushions. When faeces passes into the rectum the anorectal reflex causes the internal sphincter to relax and so allowing the contents of the rectum to come into contact with the sensitive zone of the anoderm. If faeces is present (rather than flatus) then the external anal sphincter contracts and the faeces is propelled back into the rectum until a suitable time for defaecation.

Damage to the smooth muscle of the internal sphincter will usually result in passive faecal incontinence or soiling whereas damage to the striated muscle of the external sphincter will result in faecal urgency and urge incontinence of faeces.
2.5.2 Faecal incontinence and childbirth

Faecal incontinence can occur as a result of vaginal delivery particularly if the anal sphincter is ruptured at delivery. Snooks et al concluded that faecal incontinence following vaginal delivery can involve a combination of direct sphincter division and pudendal nerve injury.

Sultan et al studied 202 women during pregnancy and after delivery with questionnaires, endoanal ultrasound, anal manometry, pudendal nerve terminal motor latencies and perineometry. They found that 13% of primiparous women who delivered vaginally had symptoms of faecal urgency or incontinence. Of the primiparous women, none of them had any evidence of anal sphincter damage during pregnancy but 35% of them had evidence of damage post delivery. Of the multiparous women 40% had evidence of anal sphincter damage prior to delivery and 44% had evidence of damage after delivery, those women delivered by caesarean section had no evidence of anal sphincter damage. There was a strong association between sphincter defects and bowel symptoms. Sultan et al studied 20 women during pregnancy and after caesarean section with anal endosonography, and anal manometry, they found no significant difference between the pre and post delivery results indicating that it is vaginal delivery rather than pregnancy that damages the anal sphincter.

Snooks et al studied 51 women in pregnancy and 122 postpartum with single fibre EMG and pudendal nerve terminal motor latencies, they found that the fibre density in EMG was increased and the pudendal nerve terminal motor latencies were prolonged in women who delivered vaginally but not in those women delivered by caesarean section. This would indicate that vaginal delivery can cause neurological damage to the anal sphincter in addition to the physical disruption seen on endoanal
ultrasound, however the limitations of pudendal terminal motor latencies has already been discussed.

2.5.3 **Constipation and childbirth**

Marshall et al have studied 7771 women with a short questionnaire administered in the immediate postpartum period. They divided their replies into three groups, those of primiparous women, multiparous (2-4) and multiparous (5+). They found that 37.5% of all women developed constipation at some stage in their pregnancy (35.3% of primiparous, 39.1% multiparous (2-4) and 42.3% multiparous (5+)). It is of interest that the prevalence of constipation prior to pregnancy increased with increasing parity: 9.6% of primiparous, 13.3% of multiparous (2-4) and 16.2% of multiparous (5+). This may mean that there is some form of permanent damage as a result of pregnancy or childbirth which leads to long term constipation. Constipation is known to be important in the long term as it can be associated with stress incontinence and uterovaginal prolapse.

2.5.4 **Relevance of bowel symptoms to project**

Traditionally the pelvis has been divided into the separate territories of the urologist, the gynaecologist and the colorectal surgeon. Pelvic floor dysfunction will often present with a combination of urinary or faecal incontinence or uterovaginal prolapse. Patients will often present to the doctor complaining of only one symptom, such as stress incontinence because they are too embarrassed to mention other symptoms such as faecal incontinence. It is not until we approach the pelvic floor, and its associated organs, as a whole that we will be able to give these patients the best possible care.

The pathophysiology of faecal and urinary incontinence may well be connected, particularly in relation to pregnancy and childbirth. It has
already been discussed that pudendal nerve damage at the time of delivery is thought to lead to both urinary and faecal incontinence. There may be traumatic damage to the sphincter muscles at the time of delivery and also damage to the pelvic floor muscles with concomitant loss of ligamentous integrity all leading to urinary and faecal incontinence. Tetzschner et al questioned 72 women who had sustained an obstetric third degree tear two to four years previously. They found that 42% had faecal incontinence, 32% had urinary incontinence and 18% had both urinary and faecal incontinence. This illustrates the point that an injury which would be expected to lead to faecal incontinence can also lead to urinary incontinence in a significant number of women.

With a common interest in pan-pelvic floor problems doctors in different specialities can combine forces in order to further research the aetiology of these problems and not only improve treatment but also prevention of these distressing problems.
Chapter 3 - Methods of Investigation
3.1 Investigation Techniques

There are many different techniques that have been used in the investigation of urinary incontinence, however these have often subsequently been found to be inappropriate. I shall discuss the most important methods here and include the reasoning underlying my choice of method.

3.1.1 Symptom questionnaires

Symptom questionnaires are straightforward methods for collecting subjective data about urinary incontinence. Provided the questionnaire is kept reasonably simple it is a quick and easy way of collecting data therefore suitable for large numbers of patients. A questionnaire can either be given to patients for them to complete by themselves or the questions can be asked by an investigator. If the patient answers the questions alone it will save time for the investigator, patients may feel less embarrassed about their symptoms and so may be more honest in their answers, however it is thought by some that self-administered questionnaire are less reliable as they depend on the patient’s full understanding of the terms used. It is important that questionnaires are well validated against an objective measurement. The bladder has been described as “an unreliable witness”, meaning that there is poor correlation between symptoms and urodynamic diagnosis. Jensen et al performed a literature search to obtain publications relating to clinical evaluation of urinary incontinence between 1975 and 1992. They found 19 articles in which they were able to compare patient history with urodynamic diagnosis. They found that the clinical history of stress incontinence when compared with urodynamic diagnosis showed a sensitivity of 0.906 and a specificity of 0.511, a clinical history suggestive of detrusor instability showed a sensitivity of 0.735 and a
specificity of 0.561. They concluded that patient history is a poor predictor of the final urodynamic diagnosis. The complaint of stress incontinence is sensitive in predicting the diagnosis of genuine stress incontinence but low specificity leads to a high false positive rate in the diagnosis of genuine stress incontinence. However these conclusions are predicated to the assumption that urodynamics provides an accurate diagnosis. This has yet to be proved.

Ryhammer et al compared questionnaire assessment of urinary incontinence with a 24 hour pad test \(^{105}\). They found no difference in the weights of the pads between those women that reported incontinence and those that did not. They did not state whether their questionnaire had been previously validated. The questions referred to incontinence during the previous 12 months. They did not explain why such a history should be concordant with a 24 hour pad weighing test.

The aim of this study was to assess the prevalence of urinary incontinence, stress incontinence in particular, through pregnancy and after delivery. Sample size estimates indicated a need for large numbers so it was decided that a simple questionnaire would be used as the mainstay of the data collection. The questions were based on those used in the urodynamic clinics at St Pancras Hospital and The Whittington Hospital. These data were collected onto computers so it was possible to validate the study questionnaire from the urodynamic clinical data set (see section 3.6). All questions were asked by the author (Victoria Cook). In order to maximise compliance it was decided that the study should be easy and convenient for women to participate in. All questions were asked when the women attended the antenatal clinic or on the telephone at home. In addition to the questions relating to urinary symptoms patients were asked three questions relating to bowel function. These questions were not validated.
The same questionnaire was used in the retrospective study. The questionnaires were posted to patients. Those that did not respond were telephoned and asked to answer the questionnaire on the telephone.

3.1.2 **Frequency volume voiding charts**

It has been found that patients tend to exaggerate their urinary symptoms on direct questioning, frequency volume voiding charts therefore prove useful in validating urinary symptoms. Frequency volume voiding charts vary in the data collected and in their duration. In most cases the volume at each void is recorded together with any episodes of incontinence, in some cases the oral intake is also recorded but this is thought to be unnecessary. A frequency volume voiding chart provides data about usual functional bladder capacity, mean volume voided, total voided volume and diurnal distribution of micturitions. Data can also be obtained about the number of episodes of incontinence. The advantage of frequency volume charts is that they provide data representative of what happens in the home environment rather than the clinic or laboratory. It has been suggested that the labour involved in the completion of a frequency volume chart may influence the results, although these have proved reproducible on re-test analysis. There is no standard length of time for a frequency volume voiding chart, however reproducibility increases with longer observation periods.

It was decided to use a seven day frequency voided chart with volumes measured on two days so as to make it as easy as possible for the women participating in the study and so increase compliance. By recording frequency for seven days any discrepancies on the days in which volumes were measured should be easily apparent. The method of chart maintenance was explained at the time of recruitment and additionally the instructions were written on each chart.
3.1.3 Pad test

A simple investigation to try to quantify incontinence is the perineal pad test. An absorbent pad is weighed before and after use and the increase in weight is thought to quantify the amount of urine lost. The time that the pad is worn for varies, different groups have used 1, 2, 24 and 48 hour tests. It is now accepted that the one hour testing should be done at standard bladder volumes, which requires catheterisation or assessment with ultrasound. Although the ICS has attempted to standardise the one hour test, none of the pad tests have been properly validated and there is still doubt as to their reproducibility. In view of the lack of validation it was decided not to use the pad test in this study.

3.1.4 Cough stress test

This test can be performed in the standing or lithotomy position. With a comfortably full bladder the woman is asked to cough and any leakage of urine is noted. This is a very simple test which is commonly used in clinical practice as an objective measure of incontinence.

Several authors have used the cough stress test to measure incontinence, Norton and Baker compared the urine lost (by measuring increase in weight of a perineal pad) with the cough stress test in four different standing positions. They found that the amount of urine lost was greatest when the women stood with their legs shoulder distance apart, and the amount lost was least when the women stood with crossed legs. Wall et al compared simple bladder filling with a cough stress test to subtracted cystometry. Of the 28 women who were found to demonstrate stress incontinence on simple bladder filling, 22 were found to have genuine stress incontinence on subtracted cystometry. Of the remaining patients, one demonstrated detrusor instability, one had no incontinence and four
had mixed incontinence on subtracted cystometry. They concluded that the clinical demonstration of stress incontinence with cough stress tests during simple bladder filling was predictive of the presence of genuine stress incontinence during subtracted multichannel provocative cystometry. Both these studies used urethral catheterisation to fill the bladder.

Miller et al measured the wetted area on a paper towel to quantify the urine lost following three coughs in the standing position. They asked the women involved to attend the experiment with a comfortably full bladder, at the end of the experiment they measured the volume voided and found it to be between 100 and 400ml. They performed several calibration experiments and then tested eight women twice two weeks apart. Each woman attended with a comfortably full bladder. Holding the paper towel against the perineum they coughed three times, a fresh piece of paper was then used and the coughs repeated. The process was then repeated at the next visit. The mean of the leakage on the two paper towel test measures at the first visit was 22.02cm² and at the second visit was 31.14cm², this equates to less than 1ml. No significant differences were found either within or across visits. They concluded that the cough stress test with paper towel is a simple and reliable measure providing immediate visual and quantitative feedback of cough related urine loss typical of mild to moderate stress incontinence. There was no standardisation of bladder volume except the instruction to come with a full bladder, despite the urine volume varying between 100 and 400mls this test provides good qualitative and quantitative data about urinary leakage and so would be a useful, non-invasive test in the clinical setting.

In view of the results of these studies we decided that the cough stress test is a simple test that could easily be performed in the antenatal clinic as an objective measure of incontinence. Vowles et al have demonstrated that stress leakage of urine can vary according to bladder volume and in...
particular some women will not leak at maximum cystometric capacity despite leakage at smaller volumes, and so there is not an ideal bladder volume at which to demonstrate stress incontinence. As a result of this we did not standardise bladder volume prior to the test. The women were asked to attend for their appointment with a comfortably full bladder, and volume voided was measured after the test. The test was performed in the standing position with legs apart and a blue paper towel held against the perineum. Each woman was asked to cough, with maximal effort, five times and the towel was then inspected for any urine loss. The result was recorded as positive or negative.

3.1.5 Urodynamics

Urodynamics is a commonly used term which covers a number of investigations of lower urinary tract function. These tests include urine flowmetry, measurement of residual urine, urethral profilometry, cystometry and pressure flow voiding studies. There is some variation in the ways that these tests are performed, these include:

- the filling medium (urine, saline, contrast medium, air or carbon dioxide)
- the pressure transducers (externally mounted, patient mounted or catheter tip transducers)
- the position in which the patient is placed during filling (lying, sitting or standing) flow rate during filling (slow – less than 10ml/min, medium – 10-100ml/min, fast – greater than 100ml/min)
- provocative tests used to demonstrate incontinence (coughing, jumping, sound of running water, washing hands)
Due to the different methods which can be employed to perform these tests it is vitally important to carefully document the precise details of the methods used\textsuperscript{113}. In addition to the difference already discussed, these tests can be performed using x-ray screening, synchronous pressure/flow videocystourethroscopy or video urodynamics, or ambulatory urodynamics.

Urodynamics is commonly referred to as the “gold standard” investigation of urinary incontinence, however it is not without its problems. The tests are time consuming to perform and therefore expensive. As the patient needs to be catheterised the tests are invasive and disliked by patients\textsuperscript{102}.

Even if performed cleanly and competently it is accepted that urodynamic studies are associated with urinary tract infection. This rate has been quoted as low as 2%\textsuperscript{114} however in the urodynamic unit at St Pancras Hospital the rate is 15%. It is accepted that in pregnancy urinary tract infection can involve the upper urinary tract in a substantial proportion of cases, acute pyelonephritis occurs in 1-2% of pregnancies. Acute pyelonephritis is associated with an increased incidence of premature labour and possibly intrauterine growth retardation and fetal death\textsuperscript{115}.

Although it has commonly been used in studies into urinary incontinence, urethral profilometry is now thought to have limited use in the investigation of urinary incontinence\textsuperscript{65; 66}

Few authors have performed urodynamic studies in pregnancy, Cutner only performed urodynamic studies on small numbers of women through pregnancy\textsuperscript{27}. He found that urinary symptoms are common in pregnancy but they cannot be explained by urodynamic findings in most women\textsuperscript{116}. Chaliha et al performed urodynamics on 286 pregnant women and found a poor correlation between symptoms and urodynamic diagnosis. Only 56% of their sample returned for follow up urodynamic testing after pregnancy, illustrating the poor compliance associated with such an invasive test\textsuperscript{117}.
For the purposes of this study urodynamics was rejected because the size of the sample necessary to test the hypothesis meant that it would not be feasible to accomplish the test in so many patients. It was felt that given the data from previous studies, there were grounds for believing that the probability of identifying meaningful differences urodynamically was too low to justify the risk of infection.
3.2 Methods

3.2.1 The prospective Study

The women in our study were recruited from the antenatal booking clinics of The Whittington Hospital and The Obstetric Hospital of University College Hospital. The study had full local ethical committee approval. All women were seen throughout by Victoria Cook. All women eligible for the study (women booking in their first pregnancy at any gestation less than 20 weeks) were offered the opportunity to participate in the study 73% of those asked agreed to take part. Women were excluded from the study if they did not understand English sufficiently to give informed consent or if they were under age 16 or older than age 40. The age of 40 was chosen as an arbitrary cut off as we were seeking to avoid the inclusion of excessive outliers. The numbers of primiparous women over the age of 40 who book at the hospitals involved in the study are small. All suitable women were given an information sheet about the study and the study was explained in detail. If they decided to participate they signed a consent form and were asked the questions from the first questionnaire. This questionnaire included questions relating to incontinence prior to pregnancy.

At The Obstetric Hospital it was routine for the patients to return to the antenatal clinic at 20, 28 and 34/36 weeks gestation. The patients were then seen at 20 weeks to ensure that they wanted to continue with the study and to explain how to complete the frequency volume voiding chart. The women were given a measuring jug and the frequency volume voiding chart at this visit. At their subsequent visits the women either saw the usual antenatal staff for their antenatal visit and then saw VC to complete the questionnaire and perform the standing stress test or VC completed their antenatal check as well as the questionnaire and standing stress test.
The standing stress test was performed as follows: each woman was asked to attend clinic with a comfortably full bladder. She was then asked to place a piece of blue paper towel against her perineum and to cough three times. The towel was then inspected for wetness and the result reported as positive or negative. The patient then emptied her bladder into a measuring jug and the amount voided was recorded.

At The Whittington Hospital it was routine for the patients to be cared for in the community following the booking appointment. Those participating in the study attended the antenatal clinic at 28 and 36 weeks to see VC who completed their antenatal check as well as the questionnaire and standing stress test. The women were given the measuring jug and first frequency volume voiding chart at the booking visit.

At both hospitals the women were asked to complete the frequency volume voiding chart for the week prior to their appointment. If they failed to do this they were asked to complete it for the week following the visit.

The women were asked to attend the hospital antenatal clinic for their six week postnatal visit. The details of delivery were collected at this visit in addition to the questionnaire. If the patient did not want to attend the hospital for this visit the details and the questionnaire were collected on the telephone. The final questionnaire was completed on the telephone at twelve weeks postpartum. Each women was asked if she was still breast feeding and if she was pregnant. The patients then posted the final frequency volume voiding chart back to the hospital.

3.2.2 The Retrospective Study

300 consecutive primiparous women were selected from the delivery register at The Whittington Hospital. All these women delivered between 5.8.97 and 29.10.97. These women were sent a questionnaire with a letter
explaining the study and a reply paid envelope 12 weeks following their delivery. Those women who did not return the questionnaire were telephoned and asked if they would answer the questions on the telephone. Two attempts were made to contact each woman if possible at different times of day. The delivery details were obtained from the delivery register.
3.3 The null hypothesis

It is delivery rather than pregnancy that is the major factor in the evolution of post partum incontinence.
3.4 Statistics

All data were stored on computer using Microsoft Access 97 and were analysed using the statistical package SPSS for Windows 10.0.5. The level of significance employed in this thesis was 5% (p <0.05).

The data derived from this study were not appropriate for parametric tests as they did not meet the requirements of interval measurement, normal distribution and homogeneity of variance. The exception to this is the data used for the validation of the questionnaire. The Kolmogorov-Smirnov test was used to measure normality for this data set.

Prior to commencing the study a sample size calculation was performed. From the literature it was estimated that 35% of women would be incontinent in pregnancy and that 17% would remain incontinent at three months postpartum. In order for the study to have a power of 80% with \( \alpha = 0.05 \) it was calculated that 180 women would need to complete the study if these two groups were to be identified as representing different populations.

Based on the results of the questionnaire data a power calculation was performed. With a sample size of 250 the study would have power of exceeding 99.9% to yield a statistically significant result. However as not all 250 patients completed the study the calculation was repeated using the 181 patients that did complete the study. With 181 subjects the study still had a power of exceeding 99.9% to yield a statistically significant result.

To compare sets of unpaired data a Mann Whitney U Test was used. When more than two sets of unpaired data were compared a Kruskal – Wallis Test was used.
Box plots were used to demonstrate some of the data, in all cases the 25–75 percentiles are drawn as a shaded box with the median as a line within, the 10–90 percentiles are drawn as a line with the outliers as individual dots.
3.5 Patient Questionnaires and Consent Forms

This chapter contains examples of the paperwork used in this study. In order are an example of the patient information sheet, consent form, questionnaire and frequency volume voiding chart used in the prospective study.

Finally there are examples of the letter and the questionnaire that were sent to each patient for the retrospective study.
An Investigation into the role of pregnancy in the damage to the urethral sphincter mechanism that leads to stress incontinence of urine

Up to 49% of women complain of leakage of urine when they cough or sneeze (stress incontinence) at some stage in their life. It is believed that often this incontinence is caused by pregnancy or childbirth. We are planning to look at the relationship between pregnancy and delivery and the development of stress incontinence of urine. The hope is that in the long run we will be able to change the way women are cared for during pregnancy and labour so that the incidence of incontinence is reduced.

We want to follow a group of women through their pregnancy and for three months after the baby is born. We would co-ordinate our research with your antenatal clinic appointments so during the pregnancy you would not need to make any extra visits to the hospital. We would plan to see you three times during the pregnancy and twice after the baby is born.

Prior to each visit we would ask you to complete a chart of every time you go to pass water and any episodes of incontinence for seven days and, in addition, for two of those days we would ask you to measure the volume of urine passed.

At the visit to the hospital Dr Victoria Cook will ask you some simple questions about your bladder and any incontinence you may have. We would then like to perform some simple tests:

Firstly we will ask you to cough five times with a full bladder, to see if you leak urine.

Secondly we would ask you to empty your bladder into a special commode which measures the flow of the urine. Once passing your water you will then be asked to stop mid-flow.

The questions and these simple tests would be repeated three times during your pregnancy and six weeks after the baby is born.

Three months after the baby is born we would see you for the final time. This appointment would be at a hospital convenient for you. In addition to the questionnaire we may wish to perform a test called urodynamics, you would be under no obligation to have this test. If we wish to perform this test we will discuss it with you, in detail, nearer the time and you would be able to decide about this then.

You do not have to take part in this study if you do not want to. If you do decide to take part you may withdraw at any time without having to give a reason. Your decision whether to take part or not will not affect your care and management in any way.

All proposals for research using human subjects are reviewed by an ethics committee before they can proceed. This proposal was reviewed by the Camden & Islington Community Health Ethics Committee and the Joint UCL / UCLH Committees on the Ethics of Human Research.

Victoria Cook
The United Elizabeth Garrett Anderson Hospital and Hospital For Women Soho
144 Euston Road
London NW1 2AP
0171 380 9294
CONSENT FORM

An investigation into the role of pregnancy in the damage to the urethral sphincter mechanism that leads to stress incontinence of urine

Miss Victoria Cook

To be completed by the patient

Delete as necessary

1. I have read the information sheet about this study YES / NO
2. I have had an opportunity to ask questions and discuss this study YES / NO
3. I have received satisfactory answers to all my questions YES / NO
4. I have received sufficient information about this study YES / NO
5. Which health professional have you spoken to about this study ..................
6. I understand that I am free to withdraw from this study:
   *at any time
   *without giving a reason for withdrawing
   *without affecting my future medical care YES / NO
7. Do you agree to take part in this study YES / NO

Signed .................................................. Date........

Name in Block Letters..........................................

Health Professional..........................................
Patient ID
Prospective
Trimester
Date

Weeks:

How many times do you pass urine in the day?
How many times do you pass urine at night?
Are you ever incontinent of urine during the day?
Do you wet your bed at night?
Do you have any urgency which is a strong desire to void caused by discomfort of any kind?
Do you experience incontinence preceded by a strong desire to void?
Is there any urine loss with coughing or sneezing?
Is there any urine loss with walking or running?
Is there any urine loss on standing after sitting?
Is there any urine loss on standing after lying?
Do you experience incontinence with no feeling that your bladder is full?
Do you suffer urgency to pass stool such that you have to rush to the toilet?
Do you have any incontinence from the back passage?
Did you have any problems with urinary incontinence prior to becoming pregnant?
Did you have any problems with urinary incontinence whilst pregnant?
Did you have any problems with faecal incontinence prior to becoming pregnant?
Did you have any problems with faecal incontinence whilst pregnant?
Frequency - Volume Voiding Chart

Instructions: Choose 7 normal days. For 5 of the days, record every time you pass water (but you do not need to measure the quantity) & any leakage. For the other 2 days, measure the volume of urine every time you pass water & any leakage. All items should be recorded on the chart below according to the time. For leakage, record according to the severity, using the scale given at the bottom of the chart. If you urinate more than once in an hour, then put all measurements for that hour in the same box.

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| Totals |       |       |       |       |       |       |       |

Leakage scale:

- Slight leak
- - Damp underwear
- - - Wet clothes
Dear Ms «Surname»,

I am a doctor doing research at The Whittington Hospital and at University College Hospital.

Up to 49% of women complain of leakage of urine when they cough or sneeze (stress incontinence) at some stage in their life. It is believed that often this incontinence is caused by pregnancy or childbirth. We are planning to look at the relationship between pregnancy and delivery and the development of stress incontinence of urine. The hope is that in the long run we will be able to change the way women are cared for during pregnancy and labour so that the incidence of incontinence is reduced.

As part of our research we are interested in the symptoms of incontinence that women have during their pregnancy and after delivery and the relationship between these symptoms and the type of delivery that they have had. As part of this study we should be very grateful if you could complete the enclosed questionnaire and return it in the stamped addressed envelope provided.

Many thanks for your help.

Yours sincerely

Miss Victoria Cook
QUESTIONNAIRE
An investigation into the role of pregnancy in the damage to the urethral sphincter mechanism that leads to stress incontinence of urine

<<First_Name>> <<Surname>>

Hospital number <<Hospital_Number>>

Date Date of birth

A.) Within the last month

1. How many times do you pass urine in the day? 0 2 4 6 8 10 12 14 16 18 20 22 24
2. How many times do you pass urine at night? 0 2 4 6 8 10 12 14 16 18 20 22 24
3. Are you ever incontinent of urine during the day? Yes / No
4. Do you wet your bed at night? Yes / No
5. Do you have any urgency which is a strong desire to void caused by discomfort of any kind? Yes / No
6. Do you experience incontinence preceded by a strong desire to void? Yes / No
7. Is there any urine loss with coughing or sneezing? Yes / No
8. Is there any urine loss when walking or running? Yes / No
9. Is there any urine loss on standing after sitting? Yes / No
10. Is there any urine loss on standing after lying? Yes / No
11. Do you experience incontinence with no feeling that your bladder is full? Yes / No
12. How often do you open your bowels?
   1-2 / day 3-4 / day 3-4 / week 1-2 / week less than 1 / week
13. Do you suffer urgency to pass stool such that you have to rush to the toilet? Yes / No
14. Do you have any incontinence from the back passage? Yes / No

B.) Before you had your baby

15. Did you have any problems with urinary incontinence prior to becoming pregnant? Yes / No
16. Did you have any problems with urinary incontinence whilst pregnant? Yes / No
18. Did you have any problems with faecal incontinence prior to becoming pregnant? Yes / No
19. Did you have any problems with faecal incontinence whilst pregnant? Yes / No

C.) Finally

20. Are you pregnant? Yes / No
21. Are you breastfeeding? Yes / No
3.6 Validation of the Questionnaire

The questionnaire used in the study was based on the questions asked at the urodynamic clinics at St Pancras Hospital and The Whittington Hospital, making it possible to relate the answers to the questions and the urodynamic results. A test retest analysis was not performed as it was not appropriate for this study, what was germane was the relationship of the results of a single application of the questionnaire to the findings obtained during a subsequent cystometrogram. These data were used to explore the relevance of a cystometrogram to the study, which was the subject of this thesis. The data, presented below, demonstrated a high level of probability that a cystometrogram would have been redundant. This was consoling because urodynamics was of logistical impossibility given the sample size required.

As has already been discussed the urethral pressure profile is not a useful test in the diagnosis of genuine stress incontinence. An alternative tool in the diagnosis of sphincter problems is the analysis of the voiding pressure-flow plot. The pressure is plotted against flow rate during voiding and the intercept on the pressure axis $P_{\text{det.close}}$ is identified. This variable has been shown to be inversely related to the presence of stress incontinence.

Patients undergoing urodynamics at St Pancras and The Whittington Hospitals always have a voiding pressure flow plot measured and stored where possible. This data was used to validate the questionnaire. This analysis is based on 1018 urodynamic studies done on female patients over the age of 18. Of these 445 patients had sufficient data for a pressure-flow plot. An analysable pressure flow plot requires considerable operator skill and the coincidence of a test unassociated with technical or human problems. These facts explain why 445 out of 1018 patients produce sufficient data for analysis. It must be understood that this could cause a
surrogate bias although the nature of such a bias is not immediately evident. The ages of the women ranged from 18 to 93 with a mean of 53 years. This chapter addresses an experiment which was designed to test the predictive power of a questionnaire. Intuitively you wouldn’t expect age to influence the outcome, or if it did the effect would be sufficiently small that very high sampling would be necessary to detect it with confidence.

The data has a normal distribution as demonstrated in the following Q-Q plot of $P_{\text{det.close}}$. This is confirmed with the Kolmogorov-Smirnov test (statistic=0.101, df=445, p=0.0001). This data is therefore suitable for parametric analysis.

**Figure 1 Q-Q Plot of $P_{\text{det.close}}$**

Firstly the relationship between the symptom of stress incontinence on the questionnaire and the demonstration of genuine stress incontinence at urodynamics was examined using the contingency tables. There was found to be a significant correlation between the symptom of stress incontinence (leakage on sneezing) and the demonstration of genuine stress incontinence on urodynamic investigation ($\chi^2=108, \text{df}=1, p=0.0001$). However when the correlation between the symptom of urge incontinence and the
demonstration of genuine stress incontinence was examined there was also found to be a significant correlation ($\chi^2=4.061$, $df=1$, $p=0.04$). There was no significant correlation between the symptom of continuous incontinence and the demonstration of genuine stress incontinence ($\chi^2=1.556$, $df=1$, $p=0.2$).

Many women who attend for urodynamical study complaining of stress incontinence do not have genuine stress incontinence demonstrated at the time of the test. As the pressure-flow plot is a measure of urethral sphincter function it may be a better method of distinguishing between those women with and without a sphincter lesion. The results of the pressure-flow plot (P_{det.close}) were compared with the elucidation of the symptom of stress incontinence and the demonstration of stress incontinence at urodynamics, using the Mann-Whitney U Test. In applying omissions erasure to the reporting of the results it was decided to describe $P_{det.close}$, since that is the most physiologically stable variable, and include the entire data set. Screening analysis of $P_{det.open}$ and age differences failed to identify any hint of an effect, so these aspects were not included in the report of the thesis since the screening analysis were inevitably underpowered. It is a principle of newtonian mechanics that the resistance of any conduit is analysed in terms of pressure and flow. The use of these data in characterising female outflow function has been the subject of earlier publications which validated the interpretation and method\textsuperscript{119}. This study used the method to assess the power of a questionnaire not the urodynamic method.

When the $P_{det.close}$ was compared in those women who did and did not demonstrate genuine stress incontinence there is found to be a significant difference ($W=36 \times 10^3$, $p=0.0001$). A parametric comparison is demonstrated in the ensuing figure which uses means and 95% confidence intervals.
When the $P_{\text{det,close}}$ is compared in women with and without the symptom of stress incontinence there is again a significant difference ($W=68 \times 10^3$, $p=0.0001$). The differences are similarly demonstrated in the following graph.

**Figure 2** $P_{\text{det,close}}$ in women with and without genuine stress incontinence

**Figure 3** $P_{\text{det,close}}$ in women with and without symptoms of stress incontinence (on coughing or sneezing)
However when the \( P_{\text{det.close}} \) is compared in women with and without the symptom of urge incontinence there is no significant difference (\( W=31 \times 10^3, p=0.5 \)) this is demonstrated in the following graph.

**Figure 4** \( P_{\text{det.close}} \) in women with and without symptoms of urge incontinence

![Graph showing \( P_{\text{det.close}} \) in women with and without symptoms of urge incontinence](image)

When the \( P_{\text{det.close}} \) is compared to symptoms of urgency using the Kruskal-Wallis Test there is no significant difference with any degree of urgency (\( \chi^2=2, df=3, p=0.5 \)) this is demonstrated in the following graph.

**Figure 5** \( P_{\text{det.close}} \) in women with symptoms of urgency

![Graph showing \( P_{\text{det.close}} \) in women with symptoms of urgency](image)
These data justify the use of the symptoms as a reliable surrogate for the physiological demonstration of a sphincter lesion. This can be amply demonstrated by using these data to calculate the probabilities of ordinal classification diagnoses associated with urodynamics.

The problem with the cystometrogram is that it produces an ordinal assessment of the pathology, i.e. demonstration of stress incontinence or not. A less arbitrary classification could use a measured variable that is known to reflect the urethral sphincter, define an arbitrary threshold and classify patients as normal or abnormal according to the value of the variable, in them, related to the threshold. A parametric analysis using $P_{\text{det.closing}}$ shows that females who describe stress incontinence have significantly lower pressures, this then supports the contention that they are describing a true experience of their symptoms. If an investigation is to be used as an arbiter in judging the veracity of symptoms it should first established that an a priori probability that a woman is misrepresenting her experience in what she describes. However without reference to such an arbitrary value judgement these data have been used to calculate the principal event probabilities using urodynamics interpreted on an ordinal scale.

Firstly calculating probabilities from all the cystometrograms with or without a pressure flow plot:

<table>
<thead>
<tr>
<th>Patients describing stress incontinence</th>
<th>752</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients demonstrated to have stress incontinence</td>
<td>413 (A)</td>
</tr>
<tr>
<td>Patients not demonstrated to have stress incontinence</td>
<td>339 (B)</td>
</tr>
</tbody>
</table>

$P(A) = 0.55$

$P(B) = 0.45$
So the probability of a woman who describes stress incontinence, then having a cystometrogram which demonstrates leakage with a cough is 0.55 whereas there is a 0.45 probability that leakage will not be demonstrated.

If the probabilities are calculated for the subgroup who had a pressure flow plot performed the results are much the same

Patients describing stress incontinence 326
Patients demonstrated to have stress incontinence 171 (A)
Patients not demonstrated to have stress incontinence 155 (B)
P(A) 0.52
P(B) 0.48

Taking the subset of 445 patients who provided measurement of $P_{\text{det.close}}$ and using an arbitrary cut-off value of Median $P_{\text{det.close}}$ the probabilities are calculated below

Patients without SI and $P_{\text{det.close}}$ above median 74 (A)
Patients without SI and $P_{\text{det.close}}$ below median 45 (B)
Patients with SI and $P_{\text{det.close}}$ above median 151 (C)
Patients with SI and $P_{\text{det.close}}$ below median 175 (D)
P(A) 0.17
P(B) 0.10
P(C) 0.34
P(D) 0.39

When the subset of 326 patients with stress incontinence who provided measurement of $P_{\text{det.close}}$ are analysed the results are again similar

Patients with SI and $P_{\text{det.close}}$ above median 151 (A)
Patients with SI and $P_{\text{det.close}}$ below median 175 (B)
P(A) 0.46
P(B) 0.54
From these results it can be concluded that the use of ordinal classification of diagnoses associated with urodynamics is not reliable and so further justifies the use of symptoms as a reliable surrogate for the physiological demonstration of a sphincter lesion. The probabilities that have been calculated indicate that the data obtained from a pressure flow plot and treated in an ordinal manner prove significantly incompetent at shedding light on the nature of the symptoms described by the patient. Indeed they suggest that the measured urodynamic variables allocate a diagnosis based on chance, given this perspective it would seem wisest to focus on what the women says is happening to her rather than attempting to cross check this using a method that is producing perplexing results. In doing this we adopt a conservative principal of empirical science.
4.1 Questionnaire Study

There are 2800 deliveries per year at UCH of which 49% are primiparous. There are 3300 deliveries per year at The Whittington of which 49% are primiparous.

250 Women were recruited to the study and 181 women completed all the questionnaires, the continuation rate by visit is as follows

Table 6 Continuation rate for the whole sample

<table>
<thead>
<tr>
<th>Trimester</th>
<th>Number</th>
<th>Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st Trimester</td>
<td>250</td>
<td>100%</td>
</tr>
<tr>
<td>2nd Trimester</td>
<td>210</td>
<td>84%</td>
</tr>
<tr>
<td>3rd Trimester</td>
<td>195</td>
<td>78%</td>
</tr>
<tr>
<td>6 weeks postpartum</td>
<td>186</td>
<td>74%</td>
</tr>
<tr>
<td>12 weeks postpartum</td>
<td>181</td>
<td>72%</td>
</tr>
</tbody>
</table>

As the nature of the visits was slightly different for the two hospitals the continuation rate for each hospital was examined separately and the results are in the following table. Any patient that failed to attend an appointment was contacted by telephone and asked if she wished to continue the study, if she did another appointment was made for her. A few women were followed up by telephone rather than attending the hospital either because they moved away or because they found it inconvenient to travel to the hospital. A number of women found the completion of the frequency volume voiding chart inconvenient and difficult. Rather than letting such women drop out of the study they were encouraged to continue with the questionnaire part of the study but did not complete the frequency volume voiding charts.
Table 7 Continuation Rate By Hospital

<table>
<thead>
<tr>
<th>Trimester</th>
<th>Hospital 1</th>
<th>Hospital 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st Trimester</td>
<td>147/147</td>
<td>100%</td>
</tr>
<tr>
<td></td>
<td>Whittington 103/103 100%</td>
<td></td>
</tr>
<tr>
<td>2nd Trimester</td>
<td>129/147</td>
<td>88%</td>
</tr>
<tr>
<td></td>
<td>Whittington 81/103 79%</td>
<td></td>
</tr>
<tr>
<td>3rd Trimester</td>
<td>121/147</td>
<td>82%</td>
</tr>
<tr>
<td></td>
<td>Whittington 74/103 72%</td>
<td></td>
</tr>
<tr>
<td>6 weeks postpartum</td>
<td>119/147</td>
<td>81%</td>
</tr>
<tr>
<td></td>
<td>Whittington 67/103 65%</td>
<td></td>
</tr>
<tr>
<td>12 weeks postpartum</td>
<td>114/147</td>
<td>78%</td>
</tr>
<tr>
<td></td>
<td>Whittington 67/103 65%</td>
<td></td>
</tr>
</tbody>
</table>

Each woman was asked her race at booking, the distribution is demonstrated in the following chart together with the different racial distribution for each hospital.
Table 8 Racial Distribution

<table>
<thead>
<tr>
<th></th>
<th>Study</th>
<th>Study %</th>
<th>UCH</th>
<th>Whittington</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asian, other</td>
<td>2</td>
<td>0.8%</td>
<td></td>
<td>0.3%</td>
</tr>
<tr>
<td>Bangladeshi</td>
<td>2</td>
<td>0.8%</td>
<td>2%</td>
<td></td>
</tr>
<tr>
<td>Black, African</td>
<td>14</td>
<td>5.6%</td>
<td>10%</td>
<td>14.2%</td>
</tr>
<tr>
<td>Black, Caribbean</td>
<td>7</td>
<td>2.8%</td>
<td>2%</td>
<td>7%</td>
</tr>
<tr>
<td>Black, other</td>
<td>1</td>
<td>0.4%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chinese</td>
<td>3</td>
<td>1.2%</td>
<td>2%</td>
<td>3.5%</td>
</tr>
<tr>
<td>Indian</td>
<td>4</td>
<td>1.6%</td>
<td>3%</td>
<td>5%</td>
</tr>
<tr>
<td>Mediterranean</td>
<td>19</td>
<td>7.6%</td>
<td></td>
<td>6.3%</td>
</tr>
<tr>
<td>Other</td>
<td>12</td>
<td>4.8%</td>
<td>12%</td>
<td>12.8%</td>
</tr>
<tr>
<td>White</td>
<td>186</td>
<td>74.4%</td>
<td>63%</td>
<td>50.9%</td>
</tr>
</tbody>
</table>

Figure 6 Racial Distribution
The wide distribution reflects the population of the two hospitals involved in the study. However the proportion of white women participating in the study is greater than in the normal population of each hospital, this probably reflects the need to be able to understand English in order to participate in the study.

Each woman was questioned at the six week visit about the mode of delivery the distribution is displayed in the following table, for comparison the mode of delivery rates for all women delivering at each hospital are displayed.

**Table 9 Mode of Delivery**

<table>
<thead>
<tr>
<th></th>
<th>Study</th>
<th>Study %</th>
<th>UCH</th>
<th>Whittington</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breech</td>
<td>0</td>
<td>0%</td>
<td>&lt;1%</td>
<td>&lt;1%</td>
</tr>
<tr>
<td>Elective Caesarean Section</td>
<td>25</td>
<td>13%</td>
<td>13.5%</td>
<td>6.7%</td>
</tr>
<tr>
<td>Forceps</td>
<td>8</td>
<td>4%</td>
<td>3%</td>
<td>6.1%</td>
</tr>
<tr>
<td>Caesarean Section in Labour</td>
<td>40</td>
<td>22%</td>
<td>13.5%</td>
<td>15.5%</td>
</tr>
<tr>
<td>Rotational Forceps</td>
<td>2</td>
<td>1%</td>
<td>&lt;1%</td>
<td>&lt;1%</td>
</tr>
<tr>
<td>Spontaneous Vaginal Delivery</td>
<td>75</td>
<td>40%</td>
<td>59%</td>
<td>60%</td>
</tr>
<tr>
<td>Ventouse Extraction</td>
<td>36</td>
<td>19%</td>
<td>10%</td>
<td>10%</td>
</tr>
</tbody>
</table>

On scrutinising the figures there is a difference between UCH and The Whittington which may introduce error but this can't be explored further as any subanalysis would be underpowered.
There were five cases in which the ventouse extraction failed and the baby was delivered by forceps and one case in which the forceps failed and the baby was delivered by ventouse.

There were five sets of twins, the mode of delivery of the first baby is included in the figures above. Of the twin deliveries one had an elective caesarean section, two had a caesarean section in labour and two had spontaneous vaginal deliveries of both babies.

The rate of ‘caesarean section in labour’ and of ventouse extraction is higher than would normally be expected from the rates at UCH or The Whittington and as a result the spontaneous vaginal delivery rate is lower. The figures given for the two hospitals are for the total deliveries rather than just women in their first pregnancy. At UCH the caesarean section rate for a standard primiparous patient (nulliparous, singleton, cephalic presentation, >37 weeks gestation in spontaneous labour) is 16% which is higher than for the population as a whole, there are no similar figures for ventouse rates in primiparous patients.
The mean age of the mother at the time of delivery was very similar for both groups at UCH it was 29.7 years with a standard deviation of 4.7 years whereas at The Whittington it was 29.2 years with a standard deviation of 4.3 years. The mean age of women delivering at UCH is 31 years, at The Whittington 24% of the primiparous patients lie in the age rate of 25 – 29 and 24% lie in the age range 30 – 34.

The women were asked at the time of their final questionnaire as to whether or not they were breastfeeding, 82 (72%) of the UCH group and 44 (67%) of The Whittington group were still breastfeeding at 12 weeks postpartum.
4.1.1 Questionnaire – Urinary Symptoms

4.1.1.1 Frequency of micturition and nocturia

The frequency of micturition increased during pregnancy and then declined again after delivery. The mean number of voids per day increased from 8 at booking to 9 at 28 weeks and 10 at 34 – 36 weeks, after delivery it declined to 6 at both 6 and 12 weeks postpartum. These changes are significant ($\chi^2 = 262$, df = 4, p = 0.0001) This is illustrated in the box plot

Figure 8 Daytime Micturition Frequency by Visit (from questionnaire)
In contrast the number of times that the women passed urine at night remained stable at 1 at all visits except the 34 – 36 week visit when it increased to 2. These results are significant ($\chi^2 = 128$, df = 4, p = 0.0001). This is illustrated in the box plot.

**Figure 9 Nocturia Frequency by Visit (from questionnaire)**

![Box plot showing nocturia frequency by visit](image)

**4.1.1.2 Incontinence**

In reply to the question ‘Are you ever incontinent of urine during the day?’ the numbers of women with a positive reply increased during pregnancy and declined again after delivery. The maximal rate of incontinence occurred at 28 weeks. 26.8% of the women reported incontinence at the
booking visit, this increased to 44.8% at 28 weeks and was 42.6% at 34-36 weeks. 14.5% of the sample reported incontinence at 6 weeks and 12.2% at 12 weeks postpartum. These results are significant ($\chi^2 = 87$, df = 4, p = 0.0001)

**Figure 10 Incontinence During The Day**

![Bar chart showing incontinence percentages at different stages: Booking: 26.8%, 28 weeks: 44.8%, 34-36 weeks: 42.6%, 6 weeks PP: 14.5%, 12 weeks PP: 12.2%]

There were only two women who reported incontinence in the postpartum period who had not reported antenatal incontinence, one was incontinent at both postpartum visits and the other was incontinent only at 12 weeks postpartum.

There were 14 women who reported incontinence at both postpartum visits, 11 of these were incontinent at the 28 week visit.

10% of the women reported urinary incontinence prior to pregnancy.

Only three women reported nocturnal enuresis with a positive answer to the question ‘Do you wet your bed at night’. Two reported nocturnal enuresis at just one visit during pregnancy, the other woman complained of nocturnal enuresis throughout pregnancy but was dry after delivery. Interestingly this woman also reported symptoms of urgency and urge incontinence and her symptoms had predated the pregnancy. These results are not significant ($\chi^2 = 3.2$, df = 4, p = 0.5).
The results from this study have been analysed on an ‘intention to treat’ basis so all the questionnaire and frequency volume voiding chart data has been included, including those of the patients that dropped out during the study. In order to examine whether or not this may have introduced bias into the results, the question ‘Are you ever incontinent of urine during the day?’ was examined, separating the positive replies from the women who completed all five visits and those who dropped out during the course of the study. The results are demonstrated in the following graph. It can be seen that during pregnancy the women who dropped out had a higher incidence of incontinence compared to those who completed the study, 33.3% v 24.3% at booking, 51.7% v 43.6% at 28 weeks and 47.1% v 42.1% at 34 – 36 weeks. In the postpartum period none of the women who answered the 6 week questionnaire but did not answer the 12 weeks questionnaire were incontinent.

Figure 11 Comparison of Incontinence in Patients Completing the Study and Those Dropping Out Early

![Graph showing percentage incontinence during pregnancy and postpartum](image_url)

4.1.1.3 Urgency and Urge Incontinence

The numbers of women reporting urgency of micturition remained stable though pregnancy and declined after delivery. In reply to the question ‘Do you have any urgency which is a strong desire to void caused by discomfort
of any kind' 34% reported urgency at booking, 36.7% at 28 weeks, 33.8% at 34-36 weeks and 17.2% at 6 weeks postpartum which fell to 13.9% at 12 weeks postpartum, this is significant \( \chi^2 = 44, \text{df} = 4, p = 0.0001 \).

**Figure 12 Urgency**

![Bar chart showing urgency percentages](image)

The pattern is similar for the question relating to urge incontinence but the numbers are smaller, 15.6% at booking, 18.6% at 28 weeks, 17.9% at 34-36 weeks, 9.1% at 6 weeks postpartum and 7.2% at 12 weeks postpartum, these changes are significant \( \chi^2 = 17, \text{df} = 4, p = 0.002 \).

**Figure 13 Urge Incontinence**

![Bar chart showing urge incontinence percentages](image)
There were eight women who reported postpartum urge incontinence who had not reported antenatal incontinence but only three of these had symptoms at both 6 and 12 weeks.

Eight women reported urge incontinence at both postpartum visits, as previously stated three of these had not been incontinent in pregnancy, however the remaining five were all incontinent at 28 weeks.

4.1.1.4 Stress Incontinence

The numbers of women with a positive answer to the question ‘Is there any urine loss with coughing or sneezing’ increased in pregnancy with the greatest prevalence at 28 weeks and then declined after delivery. 16.4% were incontinent at booking, 31.4% at 28 weeks, 30.3% at 34-36 weeks which fell to 10.2% at 6 weeks postpartum and 9.4% at 12 weeks postpartum. These results are significant ($\chi^2 = 10$, df = 4, p = 0.0001).

Figure 14 Stress Incontinence – Cough or Sneeze

4 Women reported incontinence for the first time in the postpartum period but none of them were incontinent at both of the postpartum visits. 10 women reported incontinence at both 6 and 12 weeks postpartum, they were all incontinent at 28 weeks, these women represented 3.7% of the total sample.
The results for the question ‘Is there any urine loss with walking or running’ shows a similar pattern to the previous question except that the greatest incidence is at 34 – 36 weeks, this is just significant ($\chi^2 = 10$, df = 4, $p = 0.03$). However the questions ‘Is there any urine loss on standing after sitting’ and ‘Is there any urine loss on standing after lying’ show a different pattern, the incidence of incontinence is low at booking, rises during pregnancy and returns to a low level again postpartum. The percentages of women giving a positive reply to these questions is demonstrated in the following graph and table. These results are also significant ($\chi^2 = 12$, df = 4, $p = 0.01$) for incontinence on standing after sitting and ($\chi^2 = 31$, df = 4, $p = 0.0001$) for incontinence on standing after lying.

Figure 15 Stress Incontinence - Walking or Standing

![Graph showing percentages of incontinence](image)

The numbers with a positive answer to all three questions is much smaller than those who report incontinence with coughing and sneezing. These results are compared in table 10.
<table>
<thead>
<tr>
<th></th>
<th>Leak on coughing or sneezing</th>
<th>Leak on walking or running</th>
<th>Leak on standing from sitting</th>
<th>Leak on standing from lying</th>
</tr>
</thead>
<tbody>
<tr>
<td>Booking</td>
<td>41</td>
<td>16</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>16.4%</td>
<td>6.4%</td>
<td>1.6%</td>
<td>1.2%</td>
</tr>
<tr>
<td>28 weeks</td>
<td>66</td>
<td>16</td>
<td>13</td>
<td>12</td>
</tr>
<tr>
<td></td>
<td>31.4%</td>
<td>7.6%</td>
<td>6.2%</td>
<td>5.7%</td>
</tr>
<tr>
<td>34 – 36 weeks</td>
<td>59</td>
<td>17</td>
<td>10</td>
<td>16</td>
</tr>
<tr>
<td></td>
<td>30.3%</td>
<td>8.7%</td>
<td>5.1%</td>
<td>8.2%</td>
</tr>
<tr>
<td>6 weeks postpartum</td>
<td>19</td>
<td>8</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>10.2%</td>
<td>4.3%</td>
<td>2.7%</td>
<td>1.1%</td>
</tr>
<tr>
<td>12 weeks postpartum</td>
<td>17</td>
<td>3</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>9.4%</td>
<td>1.7%</td>
<td>1.1%</td>
<td>0%</td>
</tr>
</tbody>
</table>

There were six women who reported incontinence on walking or running for the first time in the postpartum period, only one woman was incontinent at 6 and 12 weeks postpartum.

Seven women reported incontinence on standing after sitting in the postpartum period, none of these women reported the symptom in the antenatal period and all of them reported it only once in the postpartum period.

Only two women reported incontinence on standing from lying in the postpartum period, one had reported it in the antenatal period and the other had not.

The final question relating to urinary symptoms was ‘Do you experience incontinence with no feeling that your bladder is full’ The incidence of
incontinence in these circumstances increased from 9.2% at booking to
20.5% at 28 weeks, it reached a maximum of 21.5% at 34 – 36 weeks and
dropped again after delivery to 7.0% at 6 weeks and 5% at 12 weeks
postpartum. These results are significant ($\chi^2 = 42, \text{df} = 4, p = 0.0001$). Three women reported this symptom for the first time after delivery but only one of them reported it at both of the postpartum visits.

Figure 16 Incontinence - No Feeling

As the numbers of women reporting incontinence for the first time in the
postpartum period are so small no analysis was performed comparing mode
of delivery to symptoms.

4.1.2 Questionnaire – Bowel Symptoms

4.1.2.1 Frequency of defaecation

Each woman was asked at each visit about the frequency of defaecation, the answers were grouped as follows

- >4 per day
- 3 – 4 per day
- 1 – 2 per day
- 3 – 4 per week
• 1 – 2 per week
• less than 1 per week

The results by gestation are demonstrated in the following graph and table

**Figure 17 Frequency of Defaecation**

![Graph showing frequency of defaecation by gestation stages.]

<table>
<thead>
<tr>
<th>gestation</th>
<th>&gt;4 per day</th>
<th>3-4 per day</th>
<th>1-2 per day</th>
<th>3-4 per week</th>
<th>1-2 per week</th>
</tr>
</thead>
<tbody>
<tr>
<td>Booking</td>
<td>0.8%</td>
<td>4.0%</td>
<td>65.2%</td>
<td>23.2%</td>
<td>6.8%</td>
</tr>
<tr>
<td>28 Weeks</td>
<td>0%</td>
<td>5.2%</td>
<td>66.2%</td>
<td>22.4%</td>
<td>6.2%</td>
</tr>
<tr>
<td>34-36 Weeks</td>
<td>1.0%</td>
<td>4.6%</td>
<td>71.8%</td>
<td>19.0%</td>
<td>3.6%</td>
</tr>
<tr>
<td>6 Wks PP</td>
<td>0%</td>
<td>0.5%</td>
<td>71.5%</td>
<td>18.8%</td>
<td>9.1%</td>
</tr>
<tr>
<td>12 Wks PP</td>
<td>0%</td>
<td>1.1%</td>
<td>76.1%</td>
<td>18.3%</td>
<td>4.4%</td>
</tr>
</tbody>
</table>
It can be seen that the frequency of defaecation remains stable through pregnancy and for the first twelve weeks after delivery. There is no significant change through pregnancy ($\chi^2 = 6$, df = 4, p = 0.2).

4.1.2.2 Urgency to defaecate

The numbers of women reporting this symptom remained quite stable throughout the study and in particular the numbers declined slightly following delivery. 12.4% of the women reported urgency at booking, this rose to 15.2% at 28 weeks and 18.5% at 34-36 weeks. 16.1% of women complained of urgency at 6 weeks postpartum and 14.4% at 12 weeks postpartum. These results are not significant ($\chi^2 = 3$, df = 4, p = 0.5).

Figure 18 Urgency to Defaecate

There were nine women who reported faecal urgency postpartum having not reported it in the antenatal period however only one of these women reported it at both 6 and 12 weeks postpartum.

4.1.2.3 Faecal Incontinence

The numbers of women reporting this symptom are very small and there is quite an even spread across all visits, 2% reported this symptom at
booking, 1.9% at 28 weeks, 1.0% at 34-36 weeks and 2.2% at 6 weeks postpartum which fell to 0.6% at 12 weeks postpartum. These results are not significant ($\chi^2 = 2$, df = 4, $p = 0.6$).

**Figure 19 Faecal Incontinence**

There were three women who reported faecal incontinence after delivery with no antenatal incontinence. Most women reported faecal incontinence on isolated visits. No women reported incontinence at both of the postpartum visits. Only one woman was known to have a third degree tear at the time of delivery.

4 women (1.6%) reported faecal incontinence prior to pregnancy.
4.2 Standing Stress Test

The standing stress was not performed at the booking visit as women had not been asked to attend with a full bladder. Some of the women elected to perform the 6 week postpartum visit by telephone and all the 12 week postpartum questionnaires were asked on the telephone so as a result the standing stress test was not performed at either of the postpartum visits. The results presented are therefore for the 28 and 34 – 36 week visits.

At 28 weeks 182 standing stress tests were performed (87% of women). At 34 – 36 weeks 165 standing stress tests were performed (85% of women). The remaining women had their questionnaires done on the telephone.

Although the women were asked to attend their appointment with a full bladder 74% of the women who performed the test had an empty bladder.

At 28 weeks 14% of stress tests were positive, at 34 – 36 weeks 13% of stress tests were positive. As such a high proportion of the standing stress tests were performed with an empty bladder the results have not been analysed further.
4.3 Frequency Volume Voiding Chart

The women completed frequency volume voiding charts at all visits except the initial booking visit. The women often found it inconvenient to complete the charts and so if they were unhappy about continuing to complete the charts they were encouraged to continue with the questionnaire study without the frequency volume voiding chart. The numbers of charts completed at each gestation are presented below.

Table 12 Frequency Volume Voiding Charts Completed by Gestation

<table>
<thead>
<tr>
<th>Gestation</th>
<th>Completed</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>28 Weeks</td>
<td>171/210</td>
<td>82%</td>
</tr>
<tr>
<td>34 – 36 Weeks</td>
<td>138/195</td>
<td>71%</td>
</tr>
<tr>
<td>6 Weeks Post Partum</td>
<td>97/186</td>
<td>52%</td>
</tr>
<tr>
<td>12 Weeks Post Partum</td>
<td>79/181</td>
<td>44%</td>
</tr>
</tbody>
</table>

The frequency of micturition was recorded for seven days whereas volumes were measured for two days per chart.
When the data for frequency of micturition is examined for the seven day data it can be clearly seen that the frequency is increased in pregnancy compared to the postpartum data, this is a similar pattern as for the reported frequency of micturition in the questionnaire data. These results are significant ($\chi^2 = 802, \text{df} = 3, p = 0.0001$).

**Figure 20 Micturition Frequency (7 day data) by Visit (from frequency volume charts)**

In order to examine the effect of breastfeeding on frequency, the total frequency was compared in those women who were and were not breastfeeding at 6 and 12 weeks post partum. There was no significant difference between the 2 groups. At 6 weeks post partum 87 women were breastfeeding and the mean number of voids over 7 days was 49.5, whereas 10 women were not breastfeeding and their mean number of voids over 7 days was 45.9 ($W=449, p=0.6$). At 12 weeks post partum 63 women were still breastfeeding and the mean number of voids over 7 days was 39.9,
whereas the 16 women who were not breastfeeding had a mean number of
voids over 7 days of 40.3 (W=2516, p=0.96).
In order to ensure that the frequency on days in which volumes were measured were the same as on the remaining five days of the chart the two were compared.

**Figure 21 Micturition Frequency – Comparing volume measured days against unmeasured days**

It can be seen from this box plot that the median frequency remains the same but the range is statistically different ($W = 1459 \times 10^3$, $p = 0.004$).
When the volume voided per day is examined it is found that the total volume voided increased in pregnancy compared to the measurements in the postpartum period. This is significant ($\chi^2 = 32$, df = 4, p = 0.0001).

**Figure 22 Total Daily Voided Volume by Visit**

![Box plot showing total daily voided volume by visit](image)
However the when the results for the mean volume voided are plotted in a similar manner it becomes clear that the mean volume voided is decreased in pregnancy compared to the postpartum values. This is significant ($\chi^2 = 19$, df = 3, $p = 0.0001$).

**Figure 23 Mean Voided Volume by Visit (2 day measured data)**
In order to determine whether women are leaking urine during pregnancy due to an overactive bladder or due to stress incontinence it is necessary to study the results from the frequency volume charts in the wet and dry women. If women leak in pregnancy due to an overactive bladder rather than a sphincter lesion it could be proposed that those with overactive bladder would have a lower bladder capacity compared to those without. Since all studies have consistently demonstrated low bladder capacity with detrusor instability and overactive bladder it could be reasonably proposed that if the incontinent women were so affected as their bladders were overactive then their capacities should be lower than those who are dry. If the bladder volumes are similar this is an observation that would mitigate against a causal role for bladder overactivity but it is by no means proof of the pudding.

When the frequency data is examined in the form of a box plot there is no significant difference between the two groups. (Analysis comparing the two groups at each visit using the Mann Whitney U test, at 28 weeks W = 7855, p = 0.1, at 34 – 36 weeks W = 5763, p = 0.9, at 6 weeks post partum W = 4095, p = 0.4, at 12 weeks postpartum W = 2706, p = 0.1).
Similarly the mean voided volume does not vary between the two groups, at 28 weeks (W = 4638, p = 0.08), at 34 – 36 weeks (W = 3121, p = 0.6), at 6 weeks postpartum (W = 3752, p = 0.9), at 12 weeks postpartum (W = 2496, p = 0.9).
Figure 25 Mean Voiding Volume by Visit (from frequency volume data) – Comparing wet and dry women at that visit (determined from questionnaire)
4.4 Comparison between the questionnaire and frequency volume voiding chart data

It would be possible to compare the declarations of frequency of micturition, nocturia and incontinence in the questionnaire data and the recorded frequency and episodes of incontinence in the frequency volume voiding chart data. However in practice, during the study, the women would often refer to their charts when replying to the questions about frequency and nocturia. They did not refer to the charts in order to answer the questions about incontinence. In addition to this the charts did not record when the patients went to sleep and so it is not possible to accurately separate the frequencies of micturition in daytime and night time voiding. As a result of this no comparison of the frequencies of micturition between the questionnaire data and the frequency volume voiding chart data has been performed.

The reported incontinence on the questionnaire was compared to the episodes of incontinence recorded on the frequency volume voiding chart as follows: the episodes of incontinence recorded on the seven days of each chart were summed and compared to whether or not the woman reported incontinence on the questionnaire at each visit.

For those women who reported incontinence on the questionnaire the median number of recorded episodes of incontinence was two with an interquartile range of six. However those women who denied incontinence on the questionnaire had a median number of incontinence episodes of zero with an interquartile range of zero. This is an significant result ($\chi^2 = 215$, df = 1, $p = 0.0001$).
4.5 Retrospective questionnaire data

The women for the retrospective questionnaire were selected from the delivery register at the Whittington Hospital. 300 consecutive women having their first baby were selected over a three month period. When the questionnaires were returned it became apparent that two of the women were not primiparous and so their replies were not included in the analysis. Despite trying to contact those women who did not reply by telephone, 125 (41.9%) of the total did not answer a questionnaire.

The mean age of the women was 28 years with a range of 16 to 42 years.

The mode of delivery was as follows:

<table>
<thead>
<tr>
<th></th>
<th>Study</th>
<th>Whittington</th>
</tr>
</thead>
<tbody>
<tr>
<td>SVD</td>
<td>54.4%</td>
<td>60%</td>
</tr>
<tr>
<td>Ventouse</td>
<td>15.8%</td>
<td>10%</td>
</tr>
<tr>
<td>Forceps</td>
<td>5.4%</td>
<td>6.1%</td>
</tr>
<tr>
<td>Emergency LSCS</td>
<td>17.8%</td>
<td>15.5%</td>
</tr>
<tr>
<td>Elective LSCS</td>
<td>6.7%</td>
<td>6.7%</td>
</tr>
</tbody>
</table>

There were five sets of twins, they were delivered as follows:

1. Ventouse / Forceps
2. Ventouse / Breech
3. Emergency LSCS
4. Elective LSCS
5. Forceps / Forceps

The mode of delivery of the first baby is included in the table above.
In view of the large percentage of women who did not return their questionnaires a comparison was made between the ages and mode of delivery of those women who did and did not return the questionnaires. Those women who did return their questionnaire had a mean age of 29 years with a range of 16 to 42 years whereas those that did not return their questionnaire had a mean age of 26 years with a range of 17 to 40 years. The mode of delivery for the two groups is demonstrated in the following table

**Table 14 Mode of delivery respondants vs non-respondants**

<table>
<thead>
<tr>
<th></th>
<th>Responders</th>
<th>Non responders</th>
</tr>
</thead>
<tbody>
<tr>
<td>SVD</td>
<td>52%</td>
<td>58.1%</td>
</tr>
<tr>
<td>Ventouse</td>
<td>19%</td>
<td>15.3%</td>
</tr>
<tr>
<td>Forceps</td>
<td>5.8%</td>
<td>2.4%</td>
</tr>
<tr>
<td>Emergency LSCS</td>
<td>16.8%</td>
<td>16.9%</td>
</tr>
<tr>
<td>Elective LSCS</td>
<td>6.4%</td>
<td>7.3%</td>
</tr>
</tbody>
</table>

The frequency of micturition as declared on the questionnaire is demonstrated in the following graph
From this it can be seen that most of the women were passing urine between 4 and 8 times per day. 2.9% passed urine twice daily, 30.6% 4 times, 31.2% 6 times, 21.4% 8 times, 6.4% 10 times, 3.5% 12 times, 1.2% 14 times and 0.6% 20 times per day. 2.3% did not record their frequency of micturition.

The number of times that the women passed urine at night is demonstrated in the following graph.
From this it can be seen that 57.2% of women did not have nocturia, 1.2% passed urine once, 36.4% twice, 4% four times and 1.2% six times per night.

Of the total sample 32.9% were incontinent of urine at the time of answering the questionnaire. 28.3% had incontinence on coughing or sneezing. 12.7% complained of urgency and 8.7% complained of urge incontinence. 8.7% of the women had had some form of urinary incontinence prior to pregnancy and 31.8% had been incontinent of urine in pregnancy.

2.3% had faecal incontinence prior to becoming pregnant, 2.9% complained of faecal incontinence during pregnancy but this doubled postpartum so that 5.8% had faecal incontinence at the time of answering the questionnaire. 28.9% of the respondents reported urgency to pass stool such that they needed to rush to the toilet.

1.7% (3 women) of the respondents were pregnant at the time of completing the questionnaire. 53.8% of the women were breastfeeding.

For further comparison the data is divided into the following three groups:
1. Women who were dry in pregnancy and incontinent postpartum.
2. Women who were incontinent in pregnancy and incontinent postpartum.
3. Women who were incontinent in pregnancy and dry postpartum.

The results for these groups are compared to the group as a whole in table 14 below.

**Table 15 Summary of replies from retrospective questionnaire**

<table>
<thead>
<tr>
<th></th>
<th>Total Sample</th>
<th>Wet PP, dry in pregnancy</th>
<th>Wet PP, wet in pregnancy</th>
<th>Dry PP, wet in pregnancy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Are you incontinent?</td>
<td>32.9%</td>
<td>100%</td>
<td>100%</td>
<td>0%</td>
</tr>
<tr>
<td>Do you wet your bed?</td>
<td>1.2%</td>
<td>3.1%</td>
<td>4%</td>
<td>0%</td>
</tr>
<tr>
<td>Do you have urgency?</td>
<td>12.7%</td>
<td>25%</td>
<td>24%</td>
<td>10%</td>
</tr>
<tr>
<td>Do you have urge incontinence?</td>
<td>8.7%</td>
<td>28.1%</td>
<td>24%</td>
<td>0%</td>
</tr>
<tr>
<td>Do you leak with walking?</td>
<td>8.1%</td>
<td>34.4%</td>
<td>8%</td>
<td>3.3%</td>
</tr>
<tr>
<td>Do you leak with coughing?</td>
<td>28.3%</td>
<td>84.4%</td>
<td>76%</td>
<td>10%</td>
</tr>
<tr>
<td>Do you leak on standing from sitting?</td>
<td>8.1%</td>
<td>28.1%</td>
<td>20%</td>
<td>0%</td>
</tr>
<tr>
<td>Do you leak on standing from lying?</td>
<td>6.9%</td>
<td>25%</td>
<td>16%</td>
<td>0%</td>
</tr>
<tr>
<td>Do you leak with no feeling?</td>
<td>12.1%</td>
<td>28.1%</td>
<td>44%</td>
<td>3.3%</td>
</tr>
<tr>
<td>Do you have urgency to pass stool?</td>
<td>28.9%</td>
<td>31.3%</td>
<td>32%</td>
<td>26.7%</td>
</tr>
<tr>
<td>Do you have faecal incontinence?</td>
<td>5.8%</td>
<td>9.4%</td>
<td>8%</td>
<td>6.7%</td>
</tr>
<tr>
<td></td>
<td>Total Sample</td>
<td>Wet PP, dry in pregnancy</td>
<td>Wet PP, wet in pregnancy</td>
<td>Dry PP, wet in pregnancy</td>
</tr>
<tr>
<td>--------------------------------------</td>
<td>--------------</td>
<td>--------------------------</td>
<td>--------------------------</td>
<td>--------------------------</td>
</tr>
<tr>
<td>Did you have urinary incontinence prior to becoming pregnant?</td>
<td>8.7%</td>
<td>6.3%</td>
<td>20%</td>
<td>16.7%</td>
</tr>
<tr>
<td>Did you have urinary incontinence whilst pregnant?</td>
<td>31.8%</td>
<td>0%</td>
<td>100%</td>
<td>100%</td>
</tr>
<tr>
<td>Did you have faecal incontinence prior to becoming pregnant?</td>
<td>2.3%</td>
<td>3.1%</td>
<td>4%</td>
<td>0%</td>
</tr>
<tr>
<td>Did you have faecal incontinence whilst pregnant?</td>
<td>2.9%</td>
<td>0%</td>
<td>8%</td>
<td>3.3%</td>
</tr>
<tr>
<td>Number of women pregnant at time of questionnaire</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Total number of women</td>
<td>173</td>
<td>32</td>
<td>25</td>
<td>30</td>
</tr>
</tbody>
</table>

It can be seen from this table that in the two groups with postpartum urinary incontinence the numbers of women complaining of nocturnal enuresis, urgency and urge incontinence are much greater than in the sample as a whole. The numbers of women complaining of stress incontinence are greatest in the group with postpartum incontinence but who were dry in pregnancy.

Of the women with urinary incontinence during pregnancy who remain incontinent postpartum, 20% had urinary incontinence prior to pregnancy compared to 6.3% in the women who were dry in pregnancy and 8.7% in the total sample. Interestingly these women also reported 4% faecal
incontinence prior to pregnancy and 8% during pregnancy compared to 2.3% and 2.9% in the whole sample.

Those women who became incontinent in pregnancy but who were dry at the time of answering the questionnaire complained of urgency in only 10% of cases compared to 12.7% in the total sample. Interestingly 16.7% of this group were incontinent prior to pregnancy compared to 8.7% in the total sample. The rates of faecal incontinence were similar to the whole group.

The declarations of frequency of micturition for the three groups are displayed in the graph and table below. It can be seen that frequency remains similar to the group as a whole except for those women who are incontinent in pregnancy and postpartum, in these women the frequency is increased.

**Figure 28 frequency of micturition in the three groups**
Table 16: Frequency of micturition in the three groups

<table>
<thead>
<tr>
<th></th>
<th>2</th>
<th>4</th>
<th>6</th>
<th>8</th>
<th>10</th>
<th>12</th>
<th>14</th>
<th>20</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wet in pregnancy / dry PP</td>
<td>0%</td>
<td>30%</td>
<td>30%</td>
<td>30%</td>
<td>6.7%</td>
<td>0%</td>
<td>0%</td>
<td>0%</td>
</tr>
<tr>
<td>Wet in pregnancy / wet PP</td>
<td>4%</td>
<td>28%</td>
<td>24%</td>
<td>20%</td>
<td>8%</td>
<td>12%</td>
<td>0%</td>
<td>4%</td>
</tr>
<tr>
<td>Dry in pregnancy / wet PP</td>
<td>3.1%</td>
<td>25%</td>
<td>31.3%</td>
<td>15.6%</td>
<td>12.5%</td>
<td>3.1%</td>
<td>3.1%</td>
<td>0%</td>
</tr>
</tbody>
</table>

The declarations of nocturia for the individual groups are displayed in the graph and table below, most of the women did not pass urine more than twice at night which is comparable to the group as a whole.

Figure 29: Nocturia in the three groups
Table 17 Nocturia in the three groups

<table>
<thead>
<tr>
<th></th>
<th>0</th>
<th>2</th>
<th>4</th>
<th>6</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wet in pregnancy / dry postpartum</td>
<td>53.3%</td>
<td>36.7%</td>
<td>3.3%</td>
<td>6.7%</td>
</tr>
<tr>
<td>Wet in pregnancy / wet postpartum</td>
<td>52%</td>
<td>48%</td>
<td>0%</td>
<td>0%</td>
</tr>
<tr>
<td>Dry in pregnancy / wet postpartum</td>
<td>50%</td>
<td>46.9%</td>
<td>3.1%</td>
<td>0%</td>
</tr>
</tbody>
</table>

When a comparison is made between those women who reported incontinence in pregnancy and whether or not they remain incontinent after delivery the results are as follows. 45.5% of the women who were incontinent in pregnancy remained incontinent postpartum whereas 54.5% of them were dry postpartum. Of those women who did not have incontinence in pregnancy 72.9% were dry postpartum and 27.1% complained of incontinence postpartum. These results are significant \( \chi^2 = 5.7, \text{df} = 1, \text{p} = 0.02 \).
Chapter 5 - Discussion
This is the largest prospective study of primiparous women to date, following them longitudinally from booking to three months postpartum. The women who participated in this study did so for approximately nine months, in the circumstances a continuation rate of 72% is impressive. The greatest number of women dropped out after the first visit, this was usually because they had decided against participating in the study. As they were recruited and completed the first questionnaire at the first visit it is not surprising that a number of the women then changed their minds. There is quite a difference in the continuation rates between the two hospitals, this was probably due to the fact that at UCH the women were attending the hospital for the visits routinely whereas at The Whittington Hospital the women would not usually attend the hospital between booking and 41 weeks gestation. Although they had an antenatal check when they came for their appointment they often had already had a recent antenatal appointment with their GP or midwife and so their total number of visits during the pregnancy was increased which some may have found inconvenient. As both hospitals serve an inner city population, by its nature the population is mobile. When expecting their first child many people move house in order to accommodate the expanding family, in London it is often necessary to move further out of the city in order to afford a bigger house. Several of the women in this study moved away from the area, some of them were happy to continue the study by telephone (the furthest being New York) however a common reason for women not continuing the study after the second visit was due to a move. When those women who dropped out were compared to those that completed the study, those that dropped out had a higher incidence of incontinence at each visit. It may be that women were embarrassed by their incontinence and so did not want to continue to answer questionnaires about it.
The racial distribution of the participants of the study reflects the wide range of racial groups that attend the two hospitals. The proportion of white women participating in the study is greater than in the hospital populations, this was probably because of the need to understand and speak sufficient English to take part in the study.

The mode of deliveries of the women taking part in the study were similar to those for the hospital populations. However the spontaneous vaginal delivery rate was lower in the study patients whilst the ventouse rate and caesarean section rate was higher. The figures given for the hospitals include both primiparous and multiparous women. It would be expected that the instrumental delivery rate would be greater in primiparous patients compared to multiparous patients which may account for some of the differences between the study group and the hospital populations.
5.1 Frequency and Nocturia

The findings of previous studies that frequency of micturition and nocturia increase in pregnancy and then decline in the postpartum period have been confirmed by this study with the use of quantitative statistics rather than the application of arbitrary values for normal and abnormal. Both the questionnaire data and the frequency volume voiding chart data have demonstrated a significant increase of micturition in pregnancy compared to postpartum. These results are confirmed by Chaliha et al who studied 549 nulliparous women with questionnaires at 34 weeks gestation and at three months postpartum. They defined frequency as seven or more voids per day or the need to void more often that every two hours, and nocturia as two or more voids at night. They found that both frequency and nocturia increased during pregnancy and declined after delivery. Declared frequency prior to pregnancy was 28% which increased to 81.1% at 34 weeks gestation and declined to 22.4% at three months postpartum, nocturia was reported by 5.1% of women prior to pregnancy, 67.6% of women at 34 weeks of pregnancy and 4.4% of the women at three months postpartum. Neither study has addressed why the frequency changes during pregnancy, this could be due to a reduced bladder capacity, increased fluid intake or increased urine output due to the increased renal blood flow in pregnancy. Francis concluded that the increased frequency was due to polyuria which was related to increased intake of fluid but that it was not known which was cause and which was the effect.

As the results of reported frequency on the questionnaire and recorded frequency on the frequency volume voiding chart showed a similar change throughout the period of the study it can be concluded that women are accurate in the reporting of their voiding habits.
The total volume voided per day, as recorded on the frequency volume voiding charts, increased in pregnancy and declined after delivery which is agreement with the results from Francis and Cutner. However previous studies have concluded that there is no change in the mean volume voided in pregnancy or puerperium which is in contrast to the results of this study which found that the mean volume voided in pregnancy was less than that voided postpartum. The bladder volume may be reduced by compression of the bladder by the pregnant uterus resulting in a reduction in mean volume voided in pregnancy, however this theory has been rejected by other studies. Thorp et al performed a prospective study through pregnancy of lower urinary tract symptoms combined with frequency volume chart data. 123 pregnant (primiparous and multiparous) women completed a weekly voiding diary, this included the recording of the time of micturition and the volume voided together with a series of questions related to urinary symptoms for 24 hours each week through pregnancy and the puerperium. Women were recruited at different points during pregnancy. Mean daily urine output increased significantly as pregnancy progressed, it then fell again in the puerperium. The mean number of voids per day increased in pregnancy but the volume voided at each void did not change significantly. These results are in agreement with previous studies but not our study.

Another explanation as to our results of a decrease in mean volume voided during pregnancy may be the development of an overactive bladder during pregnancy. However when the results of the frequency volume voiding charts are compared in those women with and those without incontinence there is no difference in the frequency or mean volume voided. From this it could be proposed that the incontinence is not due to an overactive bladder but must result from a sphincter deficiency. It is possible that the decrease in mean volume voided during pregnancy is a response to fear about leaking with stress incontinence, however if this was the case we would
expect to find a difference in mean volume voided and frequency in those women who were incontinent and those who were dry.
5.2 Incontinence

Reported incontinence, stress incontinence and urge incontinence increased during pregnancy and declined in the postpartum period, these results are in agreement with previously published studies. Chaliha et al performed a questionnaire study involving 549 primiparous women at 34 weeks gestation and at three months postpartum. The questions related to urinary and faecal symptoms prior, during and after pregnancy. They found that 3.1% had stress incontinence prior to pregnancy, 35.7% during and 12.4% after delivery. 0.5% had urge incontinence prior to pregnancy, 8.0% during pregnancy and 2.2% after delivery. This study only includes data from the last few weeks of pregnancy, the figures for stress incontinence are very similar to our study but the figures for urge incontinence are much smaller, this may be due to differences in the questionnaires or Chaliha’s figures may be affected by questioning only women in the last trimester.

Nocturnal enuresis is uncommon in pregnancy or postpartum. The numbers of women who reported urge incontinence were smaller than those reporting stress incontinence at any time.

The numbers of women who developed incontinence of any type for the first time after delivery were very small making a comparison between delivery and postpartum incontinence meaningless in this sample. In order to perform such an analysis a study with very large numbers of women would be needed in order for sufficient women to have developed postpartum incontinence. When the data for those women who remained incontinent at both postpartum visits was examined, most of the women were incontinent by the 28 week visit. From these data it can be stated that there is an association between the development of incontinence in the second trimester of pregnancy and incontinence continuing postpartum. Meyer et al studied 149 primiparous women once during pregnancy and
again at nine weeks postpartum, with questionnaires, clinical examination, perineal sonography, urethral pressure profiles and recording of intravaginal and intra-anal pressures during pelvic floor contraction. They found that 31% of women developed stress incontinence during pregnancy, 5.5% remained incontinent after spontaneous vaginal delivery whereas 36% of women delivered with forceps were incontinent after delivery. Of those women who were incontinent during pregnancy 22% remained incontinent afterwards. This was more than in our study but the women were questioned at 9 weeks which may have made a difference. Additionally, these results were divided according to mode of delivery, the incontinence rate for the group as a whole was not quoted in the paper. Chaliha et al assessed 161 nulliparous patients at 12 weeks postpartum with a questionnaire and cystometry. They found that postpartum bladder dysfunction was common and correlated poorly with urodynamic diagnosis. They found that a high proportion of those women who had delivered by caesarean section had reported symptoms of incontinence. Digesu et al recruited 114 primiparous women in the third trimester and followed them for six months postpartum with questionnaires. They found that there were no significant differences between delivery mode and urinary symptoms. They concluded that caesarean section cannot be justified in the prevention of stress incontinence, and those women with antenatal symptoms can be reassured that in majority of cases the symptoms will resolve in the long term.

The reported incontinence on the questionnaire data compared well to the recorded incontinence on the frequency volume voided chart data. From this it can be concluded that women are accurate in reporting incontinence. Thorp et al performed a prospective study that has already been discussed. 123 women were recruited at different gestations, they completed a weekly voiding diary which included the recording of the time...
of micturition and the volume voided together with a series of questions related to urinary symptoms for 24 hours each week through pregnancy and eight weeks postpartum. The frequency of episodes of urinary incontinence and stress incontinence and severity of urine loss worsened steadily through pregnancy and improved after delivery. The frequency and severity of incontinence was worse among postpartum women than in the non-pregnant controls. This was a well planned study with objective measurements of urinary frequency and volumes voided however there was no objective measurement of incontinence. No direct comparison was made between the reported symptoms and the recorded data from the bladder diaries. The results were in agreement with those from our study.

Chaliha et al studied 286 nulliparous women with a questionnaire and cystometry at 34 weeks gestation, 161 women attended for a repeat of the investigations at three months postpartum. 4.2% of women reported stress incontinence prior to pregnancy, 45.8% during pregnancy and 19.2% at three months postpartum. At cystometry 9.1% were found to have a diagnosis of genuine stress incontinence during pregnancy and 5.0% postpartum. 3.1% of women complained of urgency prior to pregnancy, 22.7% had urgency during pregnancy and 14.3% had urgency postpartum whereas 0.3% of women complained of urge incontinence prior to pregnancy, 8.7% during and 4.9% complained of urge incontinence after delivery. At urodynamics they found that of the antenatal patients 8.4% had detrusor instability, 1.7% had mixed detrusor instability and stress incontinence and 2.8% had poor compliance. After delivery 6.8% had detrusor instability, 0.6% had mixed detrusor instability and stress incontinence and 0.6% had low compliance. Their conclusions were that lower urinary tract symptomatology does not correlate with urodynamic findings in pregnancy. Their results were broadly similar to ours.
Foldspang et al repeated their postal questionnaire study. They sent out questionnaires to an age stratified random sample of 6240 women, 75.5% were returned. The women were asked about episodes of incontinence over the past year in addition to their past obstetric history. 17.7% of women had experienced incontinence, 15.1% had stress incontinence, 8.7% urge incontinence and 6.8% mixed incontinence. The prevalence of incontinence increased with age. 62.5% of the women had had at least one vaginal delivery and the prevalence of incontinence was higher in this group, 22.9% compared to 9.0% in those who had not had a vaginal delivery. 7.6% of the women reported incontinence during pregnancy and 9.0% reported incontinence immediately after delivery, these women had a higher prevalence of incontinence at the time of the questionnaire. Various factors were examined and the strongest risk factors for stress urinary incontinence were age 40 years or more at the second childbirth, urinary incontinence immediately after childbirth and incontinence during pregnancy. More than two thirds of the women who reported incontinence during pregnancy of after vaginal delivery also reported incontinence at the time of the questionnaire. This study agrees with the other studies discussed that incontinence dating from pregnancy is associated with postpartum incontinence and it may be that this incontinence is permanent in some women, however the results should be interpreted with caution as they rely on retrospective data.

The high caesarean section rate in our prospective study may have influenced the post partum incontinence rate, however this is unlikely as our results were in agreement with other studies. The mode of delivery and incontinence rates were not compared as the numbers were too small to make any such analysis meaningful. There was a difference between the results of the prospective and retrospective studies this will be discussed in section 5.5
5.3 Standing Stress Test

When the women were recruited into the study they were asked to attend their appointments with a full bladder in order to perform the standing stress test. However at both the hospitals it was routine practice for the patients to be asked to produce a specimen of urine for dipstix analysis by the receptionist as they arrived for their appointments. Despite the fact that the receptionists were asked to tell the women not to void, 74% of the standing stress tests were performed with an empty bladder. This may be because the women become indoctrinated during pregnancy that they needed to produce a sample of urine for each visit, or it may be because the receptionists did not remember to pass on the message to the study patients, as they were usually seen during a normal antenatal clinic in which the majority of patients were asked to produce a sample of urine.

In view of the high proportion of our patients who performed the standing stress test with an empty bladder it is impossible to state whether or not this is a useful test for incontinence in pregnancy. Certainly the women found it an acceptable test to perform.
5.4 Bowel Symptoms

This study has found no change in reported frequency of defaecation, faecal urgency or faecal incontinence through pregnancy or the first 12 weeks postpartum. This is in contrast to the other studies already discussed. Chaliha et al studied 286 nulliparous women during pregnancy and three months postpartum with a questionnaire, anorectal sensation and manometry. Endoanal ultrasound was performed at the postpartum visit. They found that 7% of women had some form of anal incontinence in pregnancy whereas 13% were incontinent postpartum, these were much higher than the rates of incontinence in our study, however they included incontinence to flatus whereas we did not. Vaginal delivery was associated with a significant reduction in maximum squeeze pressure. 40% of women had a sphincter defect on endoanal ultrasound scan. The presence of a defect was associated with a lower maximum resting pressure and maximum squeeze pressure and a decrease in anal electrosensitivity. Sphincter defects were associated with symptoms of faecal incontinence but not faecal urgency. Unfortunately only 161 (56%) of the women returned for the postnatal investigations which may reduce the significance of the results. Endoanal ultrasound has been found to be accurate in identifying anal sphincter injuries in women with known sphincter defects, it has been found to falsely identify sphincter defects in 5 to 25% of normal anal sphincters. The use of anal manometry in the identification of anal sphincter defects has been found to be non-specific but when used in conjunction with endoanal ultrasound it has reduced the incidence of false sphincter defects identified on ultrasound. This may explain why the numbers of women with sphincter defects were so much greater than those with symptoms.
In contrast to these results are the results of Zetterstrom et al who questioned 349 women in the immediate postpartum period and again at five and nine months about anal incontinence. 80% of the women completed all of the questionnaires. They found that 1% of women had symptoms of faecal incontinence before pregnancy, 2% five months after delivery and 1% at nine months postpartum, these results were comparable to ours. 13.7% of the women had a sphincter tear identified at the time of delivery, even in this group the incidence of faecal incontinence at five months was only 3% and none of the women remained incontinent at nine months postpartum. At all stages the incidence of incontinence to flatus was much greater than incontinence to faeces. The same authors then studied 38 primiparous patients during and after delivery. They performed endoanal ultrasound, anal manometry, and pudendal nerve terminal motor latency at both visits. Clinical sphincter tears occurred in 15% of the patients. Endoanal ultrasound revealed disruptions to the external sphincter in six patients but no damage to the internal sphincter. Of the patients with damaged sphincters the left pudendal latency increased after delivery and manometry decreased. Three of these patients had a third or fourth degree tear at the time of delivery.

Varma et al studied 159 women with a questionnaire, endoanal ultrasound and anal manometry within five days of delivery. One patient developed faecal urgency and none developed faecal incontinence. These results can be compared to ours in which 3 women reported faecal incontinence for the first time in the post partum period but none of these women reported the incontinence at both the post partum visits. Anal sphincter damage was detected on ultrasound in 6.8% of the primiparous patients. Manometry confirmed the damage with significant reduction in maximum squeeze pressures in the patients with a disrupted anal sphincter. They concluded that a symptom questionnaire is inadequate to identify anal
sphincter injuries and so the incidence of sphincter injury has been overestimated in relation to vaginal delivery in previous published work. The true incidence is 8.7% overall (primiparous and multiparous patients) and symptoms of sphincter dysfunction are uncommon.

Faltin et al studied 150 women following their first delivery\textsuperscript{132}. All the women had delivered vaginally and did not have an anal sphincter tear diagnosed clinically. Endoanal ultrasound was performed prior to suturing of the perineum, the obstetrician and patient were not informed of the ultrasound result. All patients were sent a symptom questionnaire three months after delivery. 28% of the women had an anal sphincter tear diagnosed on ultrasound. At three months 15% of women had some anal incontinence but mostly incontinence to flatus. There was an association between undiagnosed anal sphincter tears and incontinence at three months postpartum. The rate of incontinence at three months was much higher than that reported in our study but the difference could be accounted by the inclusion of incontinence of flatus.

From these studies it would seem that symptoms of anal sphincter damage are not as common as previously published. The three questions in our questionnaire relating to bowel function were not validated which may have affected our results. We did not question the women about incontinence of flatus. When our results are compared to those studies that do not include incontinence of flatus the results were similar. Although previous studies have found an increase in the incidence of constipation in pregnancy\textsuperscript{45, 95} we found no change in frequency of defaecation through pregnancy, however we did not question the women directly about the symptom of constipation.

Although the actual numbers of women complaining of anal sphincter dysfunction were small, these symptoms can be very detrimental to quality
of life. All health professionals involved with the care of women after delivery should be aware of the need to directly question women about symptoms of anal sphincter dysfunction as women will not readily admit to such symptoms. The one woman in our study with a third degree tear was incontinent to faeces at six weeks postpartum, however although she knew me well she did not admit to any problems until directly asked the questions on the questionnaire.
5.5 The Retrospective Questionnaire

Only 58.1% of the questionnaires were returned, compared to 72% for the prospective study. The mean age of those who responded was higher than those that did not (29 years compared to 26 years). The mode of delivery was similar for the two groups. The population used for the retrospective questionnaire was similar to that for the prospective questionnaire with similar mean age at delivery and delivery rates.

Although it was planned that all patients that did not return a questionnaire were to be followed up by telephone, in practice many patients did not have a telephone number recorded on the hospital records or they were no longer at the address when rung. The questionnaires were sent to the patients three months after delivery in the hope that the relevant information was obtained at a similar time as the last questionnaire in the prospective study. However in practice there was a greater delay in the retrospective study, the mean time interval between date of delivery and answering the questionnaire was 91 days (range 73 to 181 days) for the prospective group whereas it was 148 days (range 85 to 342 days) for the retrospective group.

The declarations of frequency of micturition and nocturia were comparable for the prospective and retrospective questionnaire data. However the reported incontinence and reported stress incontinence were greater in the retrospective questionnaire group (32.9% reported incontinence in retrospective group compared to 12.2% in prospective group at 12 weeks postpartum and 28.3% reported stress incontinence in the retrospective group compared to 9.4% in the prospective group). The reported urgency and urge incontinence were similar in the two groups (12.7% of the retrospective group reported urgency compared to 13.9% in the prospective group and 8.7% of the retrospective group reported urge incontinence compared to 7.2% of the prospective group). The numbers of women
reporting incontinence prior to pregnancy was similar in the two groups (8.7% in the retrospective group compared to 10% in the prospective group). The numbers of women reporting incontinence in pregnancy was much greater in the prospective groups (31.8% in the retrospective group compared to 44.8% at 28 weeks and 42.6% at 34 – 36 weeks in the prospective group).

The incidence of bowel symptoms was also greater in the retrospective group compared to the prospective group. Incontinence of faeces prior, during and after pregnancy was reported in 2.3%, 2.9% and 5.8% of cases in the retrospective group compared to 1.6%, 2.0% and 0.6% of cases in the prospective group. 28.9% of the retrospective group reported faecal urgency compared to 14.4% of the prospective group.

The differences between the two groups could be due to recall bias in the retrospective group, particularly the difference between the incontinence rates during pregnancy, however most of the questions on the retrospective questionnaire related to current symptoms. Only 58% of the retrospective questionnaires were returned compared to 72% of the prospective questionnaires which could affect the results. The women who dropped out of the prospective study had a higher incidence of incontinence during pregnancy which may have given a falsely low incidence of incontinence in this study, however the differences were only small and so could not account for the differences between the prospective and retrospective groups. An other difference between the two groups was that the prospective questionnaire was completed at interview whereas the retrospective questionnaire was a postal questionnaire. It has already been discussed that there are advantages and disadvantages of both methods, however it is generally accepted that an interview is better than a postal questionnaire. The retrospective questionnaires were answered at a longer time interval from delivery compared to the prospective questionnaire.
(mean interval 91 days for the prospective study compared to 148 days for the retrospective study). There has not been any longitudinal work to appraise the appropriate time interval at which to question women about incontinence post partum. It may be that incontinence gets worse as time passes after delivery as the woman becomes more active, or that it improves with time as tissues heal. Those studies that have looked at incontinence over a longer time period (up to 18 months) mostly show an improvement with time or, it was only the postal questionnaire data of Glazener that showed a worsening of incontinence with time (2% at 1 week, 6% at 8 weeks, 8% at 12-18 months.) and the work of Mallet et al which followed up women six years after delivery and showed increased incidence of incontinence and evidence of worsening of pelvic floor physiology over time which was not related to further child bearing. This last study is particularly interesting because of its long follow up compared to the others.

When the data from the retrospective questionnaire was divided into groups some differences become apparent. When the women who reported incontinence postpartum were compared to the group as a whole there was a greater incidence of urgency (24-25% v 13%, \( \chi^2 = 30.4, \text{df} = 1, p = 0.0001 \)), urge incontinence (24-28% v 9%, \( \chi^2 = 66.8, \text{df} = 1, p = 0.0001 \)), stress incontinence (76-84% v 28%, \( \chi^2 = 211.8, \text{df} = 1, p = 0.0001 \)) and faecal incontinence (8-9% v 6%, \( \chi^2 = 40.1, \text{df} = 1, p = 0.0001 \)). All these symptoms except that of stress incontinence are associated with an overactive bladder. Chaliha et al found the incidence of genuine stress incontinence and detrusor instability to be similar in both the vaginal delivery and caesarean section groups. They concluded that the aetiology of postpartum stress incontinence may be detrusor instability rather than genuine stress incontinence. However the results from the frequency volume voiding data in the prospective group do not support this.
When the women who reported incontinence during pregnancy and postpartum were compared to the group as a whole, they were found to have an increased declaration of incontinence prior to pregnancy (20% v 9%, $\chi^2 = 19.7$, df = 1, $p = 0.0001$). Those women who were incontinent during pregnancy but who became continent after delivery also had a much higher incidence of incontinence prior to pregnancy compared to the total sample (20% v 9%, $\chi^2 = 9.4$, df = 1, $p = 0.02$).

Despite the fact that the results from the retrospective questionnaire are not as reliable as the prospective questionnaire there is still good correlation between the development of incontinence in pregnancy and postpartum incontinence. Retrospective questionnaires have commonly been used in previous publications, however in comparing the same questionnaire in the same population with a prospective and retrospective study we have demonstrated the retrospective questionnaire to be wanting.
5.6 Criticisms of the study

This was a well planned prospective longitudinal study of primiparous patients through pregnancy and postpartum. However we were restricted to starting the study at the booking appointment and so the prospective data collection did not start until the end of the first trimester of pregnancy. Likewise due to time constraints the data collection ceased relatively soon after delivery, it would have been ideal to continue to follow the women for much longer after delivery, however due to the number of women recruited this was not possible.

The urinary questions on the questionnaire were well validated however the bowel questions were not validated which may have affected the results. In asking the questions about incontinence the ICS definition of incontinence was not used. Instead the questions were kept very simple and were clarified only if the patient did not understand the question. We could be criticised for not using the ICS definition.

The questionnaire data was compared to objective data in the form of the frequency volume voiding charts. Although the charts were designed to be easy to complete, particularly as the volumes only had to be measured for two days of each chart, the women found them inconvenient and so the numbers of women completing the charts at each visit declined during the study. Unfortunately only 44% of the women completed a chart at the last visit. The low numbers of women completing the charts later in the study may have influenced the results.

The numbers of women completing the study were impressive in the circumstances. The only way to improve on these figures would be to perform a similar study in an area with a very stable population.
The results from the standing stress test are meaningless as such a large proportion of the tests were performed with an empty bladder, as a result it is not possible to state whether or not this is a useful test to perform in pregnancy. Despite the fact that the women were told to attend their appointment with a full bladder, and were reminded not to empty their bladders on arrival by the receptionist, they still had empty bladders. The obvious way to rectify this would either be to wait for the bladder to fill naturally or to catheterise prior to the test. Neither of these options were practical in the antenatal setting. An alternative would be to perform a standing stress test at the time that women attend for an ultrasound scan. Women are requested to attend for such scans with a full bladder and the bladder volume could be assessed by ultrasound before performing the standing stress test.
Chapter 6 - Conclusions
The questionnaire used in this study has been validated against urodynamic data and has been found to be a good tool in the investigation of lower urinary tract symptoms in pregnancy. Likewise the frequency volume voiding chart used has been found to be a simple and practical method of obtaining data from large numbers of women in pregnancy. The results correlate well with the questionnaire data, so women are accurate in the reporting of their symptoms prospectively. However some people might say that as there is no correlation between urodynamics and the questionnaire and that the problem is not with the instrumentation but with the patients psychological or cognitive function in relation to the questionnaire and so questionnaire data cannot be trusted.

As suspected prior to the study the collection of retrospective data is less accurate than prospective data. The results from our prospective study are more comparable to other published work than those of the retrospective study. The completion rate was greater for the prospective study (72% v 58%)

Frequency of micturition increases in pregnancy and declines after delivery. The total volume voided per day also increases however the mean volume voided at each micturition decreases slightly in pregnancy compared to postpartum.

Incontinence is common in pregnancy and declines significantly after delivery. The numbers of women developing incontinence for the first time after delivery are very small. It is therefore possible to reject the null hypothesis ‘It is delivery rather than pregnancy that is the major factor in the evolution of post partum incontinence’ and state that incontinence is due to pregnancy rather than delivery. There is no difference in the frequency or the mean volume voided in women with and without incontinence and so the incontinence is not due to an overactive bladder. It
is possible to conclude that the incontinence is due to a sphincter deficiency. The results of this study agree with that of Francis \(^{47}\) so we can also conclude as she did, ‘that those women who complain of stress incontinence in middle life are destined to do so from an early age, and it is pregnancy rather than labour that reveals an intrinsic defect in the sphincter mechanism’. It may be that in a small number of women damage occurs at delivery which contributes to postpartum incontinence.

6.1 Further Research

It is the first time that this questionnaire has been used in such a study, as has already been discussed the questionnaire was validated except for the bowel questions. It would be possible to develop this questionnaire further to include the ICS definitions and to fully validate the bowel questions. The reliability of the questionnaire on a test retest analysis would be easy to perform if it was felt necessary for further studies.

It would be ideal to follow a cohort of women from prior to conception, through pregnancy and then for many years postpartum to see if the women that are incontinent in pregnancy are those that appear in the urodynamic clinics many years later. It would also be possible to examine the effect of further pregnancies on the development of incontinence. There has been no work to date examining the ideal time to question women about post partum incontinence, it would ideal to study women prospectively after delivery to examine whether incontinence improves or worsens and what factors may be influencing such changes.

In any longitudinal study the follow up rates are important, our study was performed using an inner city population. It would be useful to repeat such a study in a more static population. It would also be useful to investigate why the patients in our study dropped out in order to improve follow up in
the future. The completion rate for the frequency volume charts could be improved by making them shorter or by eliminating the need for measuring the urine volume voided.

No attempt has been made to investigate why urine output changes during pregnancy and after delivery in this study, this would be a subject of further studies.

Further work is needed into the mechanisms by which women become incontinent in pregnancy. Following this it may be possible to elucidate whether the incontinence of pregnancy is different to incontinence postpartum.
Reference List


   Ref. Type: Thesis/Dissertation


91. Snooks SJ, Henry M, Swash M. Faecal incontinence due to external anal sphincter division in childbirth is associated with damage to the


Ref. Type: Conference Proceeding


