Long-term consequences of vaginal delivery on the pelvic floor: A comparison with caesarean section in one-parae women

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For the times they are a-changin'...

Bob Dylan 1964

Abstract

Long-term consequences of vaginal delivery on the pelvic floor:
A comparison with caesarean section in one-parae women

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Urinary incontinence (UI), symptomatic pelvic organ prolapse (sPOP), and faecal and anal incontinence (FI and AI) are the three major sequelae of childbirth. It has as yet not been finally settled whether in the long term vaginal delivery (VD) is detrimental to pelvic floor function in comparison to caesarean section (CS). The aim of this thesis was to study the influence of childbirth on the long-term prevalence of these pelvic floor disorders (PFD) and their putative obstetric and non-obstetric risk factors by comparing two large cohorts of women after one VD compared to one CS in 2008, 20 years after childbirth. This national cohort study included 5 236 one-parae women who gave birth in 1985-1988 and returned a questionnaire on PFD in 2008. Self-reported information was linked to obstetric data from the Swedish Medical Birth Register.

The prevalence of UI**;** UI for more than 10 years; subtypes of UI; severe, significant and bothersome UI; sPOP; AI, severe AI; FI; were consistently higher after VD compared to CS. After one VD the prevalence of FI increased by about 4%, UI by 12% and sPOP by about 8%, compared to one CS. After a VD women with sPOP had an almost tripled prevalence of UI compared with CS. A $\geq 2^{nd}$ degree perineal tear was associated with an almost doubled prevalence of FI. Episiotomy during VD was protective for FI. BMI was second to VD the most important risk factor for PFD, which is important since it is modifiable.

In conclusion, one single VD was associated with an increased prevalence of all three of the most important pelvic floor disorders - UI, sPOP, and FI - 20 years after giving birth to one child.

Key-words: Vaginal delivery, caesarean section, urinary incontinence, subtypes, bothersome, severity, pelvic organ prolapse, anal incontinence, faecal incontinence, long-term, epidemiology, body mass index, episiotomy, perineal tear.

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List of publications

This thesis is based on the following papers, which will be referred to in the text by their roman numerals

I. The prevalence of urinary incontinence 20 years after childbirth: a national cohort study in singleton primiparae after vaginal or caesarean delivery. Gyhagen M, Bullarbo M, Nielsen TF, Milsom I. *BJOG. 2012 Mar 14. doi: 10.1111/j.1471-0528.2012.03301.x. [Epub ahead of print]*

II. Prevalence and risk factors for pelvic organ prolapse 20 years after childbirth: a national cohort study in singleton primiparae after vaginal or caesarean delivery. Gyhagen M, Bullarbo M, Nielsen TF, Milsom I. *BJOG. 2012 Nov 12.* doi: 10.1111/1471-0528.12020 . *[Epub ahead of print]*

III. A comparison of the long-term consequences of vaginal delivery versus caesarean section on the prevalence, severity and bothersomeness of urinary incontinence subtypes. Gyhagen M, Bullarbo M, Nielsen TF, Milsom I. (*Submitted*).

IV. Caesarean section and episiotomy during vaginal delivery protect women from faecal incontinence in later life. Gyhagen M, Bullarbo M, Nielsen TF, Milsom I. (*Submitted*).

Abbreviations

ACS	Acute Caesarean Section
AI	Anal Incontinence
BMI	Body Mass Index
CS	Caesarean Section
CI	Confidence Interval
EAS	External Anal Sphincter
ECS	Elective Caesarean Section
FI	Faecal Incontinence
IAS	Internal Anal Sphincter
ICS	International Continence Society
MBR	Medical Birth Register
MRI	Magnetic Resonance Imaging
MUI	Mixed Urinary Incontinence
NNT	Number Needed to Treat
OR	Odds Ratio
POP	Pelvic Organ Prolapse
POPQ	Pelvic Organ Prolapse Quantification
QoL	Quality of Life
sPOP	symptomatic Pelvic Organ Prolapse
SVD	Spontaneous Vaginal Delivery
SUI	Stress Urinary Incontinence
TFR	Total Fertility Rate
UI	Urinary Incontinence
UUI	Urge Urinary Incontinence
VD	Vaginal Delivery
VE	Vacuum Extraction

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Introduction

These days most women in welfare states live a major part of their active lives after giving birth to one or two children. The long-term effects of childbirth, which manifest themselves in mid- and upper midlife, are known to negatively affect the quality of life and professional careers of many women.[1] This thesis investigates the three most important long-term pelvic floor sequelae of childbirth, urinary incontinence, pelvic organ prolapse and faecal incontinence. The influence of one single pregnancy and vaginal delivery on pelvic floor function later in life is compared with one single delivery by caesarean section.

By necessity, the obstetrician's and a midwife's view on how vaginal birth affects the female pelvic floor is foremost based on real-time experience, while the urogynecologist's opinion about the relevance of a vaginal birth for pelvic floor dysfunction is retrospective emanating from a late *faite accompli*: *'Every urogynecologist has heard the basic scenario hundreds of ways and hundreds of times. There is an onset following the birth of a child, and some level of symptoms which progress and ultimately require physician intervention'.**

In the literature there is at present a divergence in perception as to the effects of childbirth on the pelvic floor which is illustrated by the large number of studies performed that combine obstetric and non-obstetric events in longitudinal, short-term follow-ups.[1] On the other hand there is a scarcity of more long-term longitudinal studies as they are time-consuming, very costly, and require a significant effort over a long time period. Randomised controlled trials in this field of investigation are either inconceivable from an ethical point of view or in fact impracticable.

* Linda Brubaker 1998

At present it may seem unrepresentative to study the consequences of giving birth to only one child. However UN data show that total fertility rates (TFRs) are rapidly declining globally and predicted TFRs indicate that in the middle of this century the mean TFR worldwide will be less than 2.0 children per mother and in many developed countries TFRs are already between 1.0 and 1.5.[2]

In the studies of this thesis it will be shown that middle-aged women after one single vaginal birth have an increased prevalence of FI by about 4%, of urinary incontinence by about 12%, and of symptomatic pelvic organ prolapse by about 8% compared to delivery by one caesarean section. But even if these differences do not seem very impressive, they constitute the effect of the first vaginal birth only. Globally, women today deliver on the average one or two more children, thereby adding further to these risks. Each year almost 50 million nulliparous women give birth to their first child but the total population at risk is almost 150 million women yearly, and more than every second delivery is performed under suboptimal conditions, without assistance from skilled personnel.[3] Furthermore, the outcome in terms of pelvic floor dysfunction, which in our rigorously toilet-trained society applies particularly to faecal incontinence can be disastrous socially, sexually, and devastating for a woman's self-esteem.

Prevention of pelvic floor dysfunction is reported to be the main cause for requesting caesarean section for non-medical reasons.[4,5] The very thought of becoming permanently incontinent to faeces or gas due to vaginal delivery has been shown to be the primary reason why some women contemplate caesarean section. This trend is also steadily rising and in several studies this attitude was shown to be most prevalent among female physicians. [4,5]

The starting position for the SWEPOP project, part of which constitutes this thesis, was that it has not yet been settled whether vaginal birth in the long term is detrimental to pelvic floor function or not. The State of the Science Conference on

Caesarean Delivery on Maternal Request of NIH in 2006 could not find any high quality data on pelvic floor outcomes supporting either type of delivery.[6]

Now that the picture of the long-term consequences of injuries due to vaginal birth is becoming increasingly clear, both perspectives must be taken into account – what is best for today and best for tomorrow – must guide both the woman and her physician. For the profession, giving priority to PFD alone, against all other maternal and neonatal outcomes, seems inappropriate. Prevention is however universally considered superior to treatment, and zero tolerance or nil sequelae, though in practice as unattainable as infinity, is a most useful target for our joint efforts.

The female pelvic floor – relevant functional anatomy

The structure of the female pelvis may be seen as a compromise between the preconditions for a regulated and controlled evacuation of urine and faeces but also allowing the passage of a fetus through the pelvic hiatus at vaginal delivery.

Viewed from above the pelvic floor is a hexagonally shaped entity consisting of striated muscles, connective tissue, and the organs that fill the lower pelvic cavity. The muscles of the pelvic diaphragm are collectively referred to as the levator ani muscles. These muscles form three regions. Region 1 is composed of m. ileococcygeus that builds a flat, horizontally oriented plate behind the anal canal from the arcus tendineus and the pelvic sidewall on one side to the opposite side passing in front of the sacrum in the midline. Region 2 encompasses the urogenital hiatus – through which the urethra and the vagina pass – and includes the pubovisceral muscles (the pubovaginal and puboanal muscle) that arise from either side of the symphysis and attaches to and encircles the urethra, the vagina, and the

perineal body. Contraction of these muscles closes the urogenital hiatus against the opening force of the intra-abdominal pressure. Region 3 consists of the puborectal muscle, which is a muscle sling that arises lateral to the pubovisceral muscles at the symphysis and fuses behind the recto-anal junction cephalad to the external anal sphincter (Figure 1). [7,8]

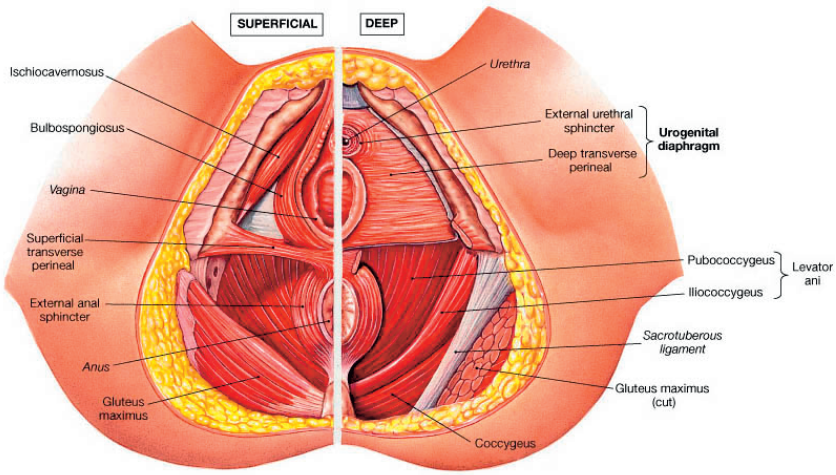

Figure 1.

In contrast to other striated muscles of the human body, the muscles of the pelvic floor exhibit continuous activity both at rest and during sleep. The tonic, continuous activity is considered to originate from a sacral spinal centre.[9] Postural changes such as movements of the body, abdominal straining, speaking, coughing, etc. transiently increase the tone of the pelvic floor muscles.[10] Phasic contractions of the

levator ani muscles are coordinated and exert a force in a ventro-cephalic direction, which keeps the urogenital hiatus closed against the intra-abdominal pressure. A voluntary contraction exerts an even stronger force to close the urogenital hiatus. The hydrostatic pressure carried by the pelvic floor in the upright position is about 40 cm H_2O and 140 cm H_2O during maximum cough.[11]

The endopelvic fascia attaches the bladder, uterus, and rectum to the pelvic walls. The uterosacral and cardinal ligaments are important fascial condensations involved in supporting the uterus and upper vagina. In the distal vagina its wall is directly attached to the surrounding structures and fuses with the urethra anteriorly and to the perineal body posteriorly.

The anal sphincter complex consists of three muscle components: the internal anal sphincter (IAS), the external anal sphincter (EAS), and the puborectal muscle sling. The EAS surrounds the IAS and extends more caudally. All three sphincter components exhibit constant tonic activity to close the anal canal.[12,13] The EAS and the levator ani complex are also responsible for voluntary contraction and phasic reflex contractions. During defecation and by distending the rectum, the IAS is relaxed, which is mediated by the recto-anal inhibitory reflex along the intramural myenteric plexus of the rectum.[14] The IAS is the strongest smooth muscle sphincter of the human body and is responsible for maintaining anal continence at rest (Figure 2). Pudendal nerve branches from the S2–S4 segments travel through Alcock's canal, where the nerve is fixed, into the ischiorectal fossa on both sides and innervate the levator ani muscles on their inferior surface. Direct branches from the S2–S4 segments innervate the cranial surface of m. levator.[15]

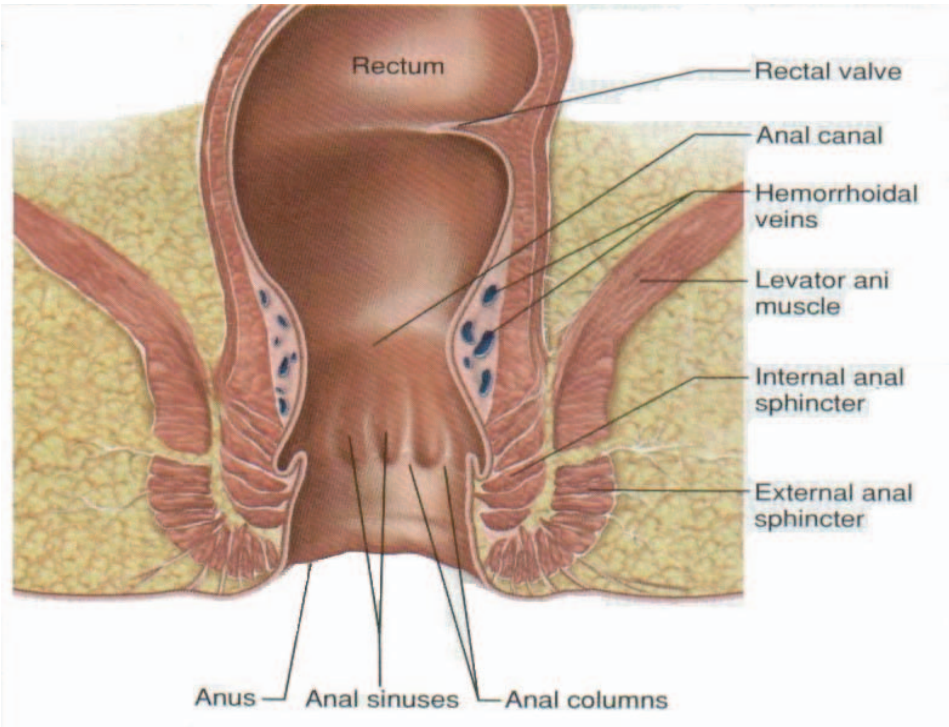

Figure 2.

The effects of vaginal delivery

During the second stage of labour damage to the pelvic floor, its muscles and nerves, and to the endopelvic fascia, may occur due to stretch, compression, and ischemia. The difficult vaginal delivery has typically been associated with a forceps delivery, a prolonged second stage of labour, and high infant birthweight.

Post partum MRI studies in one-parae women have revealed that levator ani muscle injury was found in 6-10% after spontaneous vaginal delivery, in 17-33% after vacuum extraction, and in 67-71% of women after forceps delivery, but was not identified in nulliparous women or after caesarean section.[16-18] The medial-most part of the pubovisceral muscle, nearest to the urethra, is exposed to the largest

stretch.[19] In an ultrasound study by Dietz et al. it was found that detachment of the levator muscle was found in 14 of 39 examined women after VD, which was associated with stress urinary incontinence 3 months post partum.[20] Opening of the genital hiatus and weakening of the pelvic plate have also been shown to occur more often in women with prolapse compared to women with normal pelvic support. [21]

The mechanism of nerve damage during vaginal delivery is thought to be stretch and compression in combination when the fetal head descends through the birth canal. This is considered to impair the blood flow by constricting intraneural vessels.[19,22] The association between vaginal delivery and pelvic nerve injury was first established in 1977 when Allen Parks found that anal sphincter denervation with faecal incontinence was found in women after vaginal delivery.[23] In a geometric model to predict the stretch of pelvic nerves during the second stage of labour it was found that the nerves innervating the anal sphincter were stretched beyond the 15% strain threshold which cause permanent damage to appendicular nerves.[19] Electrophysiological studies have demonstrated that neuropathic injury was observed in 42% of women after vaginal delivery. No evidence of pudendal nerve injury was seen after caesarean section. Two months after vaginal delivery recovery of pudendal nerve function was observed in 60% of women but persisted in the remainder.[24-26]

Pathogenesis and pathophysiology

Urinary incontinence

Urinary continence depends on the competence of the urethral sphincter and the compliance of the urinary bladder to allow low-pressure storage of urine. The urethral sphincter – especially its proximal one-third, which is situated in the pelvis

immediately behind the pubic bone – generates a high resting pressure due to tonic and phasic urethral activity. According to the 'hammock hypothesis' pressure transmission from the abdominal cavity onto the urethra, with the endopelvic fascia and the anterior wall of the vagina providing a backboard, assists in compressing the urethra further.[27] The stability of this part of the endopelvic fascia and its connection to the pelvic floor muscles is considered to be essential for urethral closing during stress.[28]

A number of factors during labour have been shown to contribute to *de novo* urinary incontinence after vaginal birth: pudendal nerve damage, rupture and stretching of the cardinal and sacro-uterine ligaments, vaginal injuries, and widening of the levator hiatus.[29] A longitudinal urodynamic study performed early during pregnancy and 8 weeks after delivery demonstrated a decrease in urethral closure pressure and a shorter functional urethra. This was not observed in healthy nulliparous women and in women after caesarean section.[30] Women delivered by VD also had diminished intra-abdominal pressure transmission during coughing. Ultrasound studies during cough have also demonstrated an increase of bladder neck mobility and poor bladder neck support after vaginal delivery, not observed after caesarean section.[31,32]

Pelvic organ prolapse

Normal pelvic organ support depends on the integrity of the endopelvic fascia, the pelvic floor muscles, and an adequate nerve supply. Vaginal birth can cause disturbances of all these components. Mechanical damage to the pelvic floor structures mainly occurs during the second stage of delivery when the fetal head distends and stretches the pelvic floor.[20] Pelvic organ prolapse is a rare condition in nulliparous women and in women after one or several caesarean sections indicating that mode of delivery is more important for the pathogenesis of POP than

pregnancy alone.[33] During pregnancy hormones and enzymes affect the connective tissue so that it is softened and able to stretch adequately during vaginal delivery.[34,35] Women with joint hypermobility and some rare connective tissue disorder syndromes are at higher risk of developing POP.[36,37]

Faecal and anal incontinence

Bowel continence is an extremely complex bodily function, the main components of which are the quality of colonic content, the integrity of the nervous and humoral control of intestinal motility and the endo- and exogenous secretory mechanisms of the gastro–intestinal tract, the sensory function of the rectum and the anal canal, and finally the functional status of the pelvic floor muscles.[38] The effect of vaginal birth affects however mainly the last component. To date, much research has been focused on sphincter injuries to explain incontinence in parous women, based on the implicit assumption that these damages and their putative risk factors alone explain bowel incontinence after childbirth. Injury to the anal sphincter during childbirth may be the result of a direct disruption of the anal sphincter muscles. Using anal ultrasound Sultan et al. found sphincter defects 6 weeks postpartum in 35% of primiparous women but no defects in women delivered by caesarean section.[39] In addition to sphincter muscle injury also neuropathy caused by stretch, compression, and ischemia in combination is thought to occur during the second stage of delivery.[40] The pudendal nerve is fixed at the ischial spine, which predisposes it to traction injury as the fetal head emerges in the birth canal. A prolonged second stage of labour has been associated with neuromuscular injury.[41] In nulliparous women an active second stage lasting for more than 1 hour was associated with a significantly higher risk of denervation injury, whereas a prolongation of the passive portion of the second stage was not associated with an increased risk of this damage.[41] In a study of women with advanced cervical

dilation (>8 cm) at the time of caesarean delivery, delayed pudendal nerve terminal motor latency (PNTML) and reduced anal squeeze pressures were demonstrated.[42]

Epidemiology

Urinary incontinence

The reported prevalence rates of UI in women vary considerably due to differences in target population, definitions, study designs and methods chosen for statistical analysis, which complicates the comparison of results.[1] The prevalence of UI in women varies during their lifetime also depending on the effect of age, weight, parity, lifestyle factors, and general health status. In population based studies the prevalence of UI (any) ranged between 5% and 69%, although most studies reported 25-45%. In most studies the prevalence of isolated stress UI was 10-39%, the prevalence of mixed UI 7.5-25%, and isolated urge UI in 1-7%.[1]

The prevalence of UI in nulliparous women of childbearing age has been reported to be 10-15%.[43-47] UI preceding pregnancy in nulliparous has been shown to be a strong indicator for increased prevalence of UI 4-12 years post partum.[48,49] Pregnancy in itself, independent of labour and delivery practices, seems to be a risk factor for postpartum UI,[50,51] especially if the incontinence started during the first trimester.[52] During pregnancy the prevalence of UI increases with gestational age[53] so that more than half of all women report UI during the third trimester.[44,54,55] Stress UI and mixed UI increased the most during pregnancy compared to before pregnancy whereas urge UI did not change during the same period.[56] During the first three months postpartum UI prevalence was 30% and most women had stress UI.[57] Urinary incontinence usually declines rapidly during the first 3 months following childbirth, indicating that most symptoms are part of an apparently normal course of childbirth.[53] Several studies have also demonstrated that

postpartum UI is a risk factor for UI after longer (7 months to 6 years) terms of follow-up.[51,58-60]

The first delivery is considered to increase the prevalence of UI the most, but several studies have also demonstrated a further increase for each delivery.[61-65] Many cross-sectional and longitudinal studies show a protective effect of CS for UI.[66] BMI is considered to be an established risk factor for UI,[64] whereas the association between UI and age is complicated by confounding.[67]

The prevalence of subtypes of UI is age-dependent. Pooled data from 14 studies showed that the prevalence of SUI peaks in the 4th decade and then declines gradually with increasing age, whereas MUI starts to increase in the 4th and UUI in the 6th decade. The prevalence of all three subtypes subsequently converges to approximately 30% in the 8th decade.[68]

Pelvic organ prolapse

Assessment of the prevalence of symptomatic pelvic organ prolapse in women without known PFD using a postal questionnaire are remarkably consistent, with a prevalence between 4% and 11%.[33,69-71] POP is a rare condition in nulliparous women and in women after one or several caesarean sections, indicating that mode of delivery is more important than pregnancy alone.[33,72] Vaginal delivery is associated with an increased risk for pelvic organ prolapse and increasing number of births is positively associated with increased risk of POP.[33]

The main risk factor of POP is vaginal delivery.[73] Even if most studies have shown BMI to be a risk factor for POP, data are conflicting.[74] Several obstetric interventions and events have been associated with POP. Data are however also in

this regard disparate.[72] There is no consensus regarding the effect of acute versus elective caesarean section, infant birth weight, episiotomy, instrumental delivery, and length of the second stage of labour on the development of POP. It has been concluded that these inconsistencies probably are due to lack of statistical power.[1]

Faecal and anal incontinence

In a recent cross-sectional study on faecal incontinence ≥ 1/month in adults (aged >18 years) the overall community prevalence of FI was 12.4%.[75] Many earlier studies have reported much lower prevalence rates,[76] but those figures are almost certainly too low, as it has been shown that more than every second person with FI do not report the symptoms to their doctor because of embarrassment or pessimism about its curability.[77]

Reported prevalence of AI and FI after childbirth vary considerably due to lack of consistency in definition, type of questionnaire, selected population, and mainly to length of follow-up.[78] During pregnancy AI was reported by 10% of nulliparous women, of which 90% had incontinence to gas only.[79] In another report on FI during the pregnancy of nulliparous women no increase of incontinence occurred during the second and third trimester.[80]

Faecal/anal incontinence is common during the first months postpartum,[39,40,81] but a majority of women with these early problems will recover.[82] To date there are no studies that have demonstrated any benefit of caesarean section over vaginal delivery with regard to the long-term prevalence of bowel incontinence.[82] In the study of MacArthur et al. women were followed for 12 years. The prevalence of FI among those who had a spontaneous vaginal delivery was 11.5% (n=213) compared to 14.1% (n = 24) women delivered exclusively by caesarean section.[83]

Age is widely considered to be a major risk factor for FI.[78] However, in parous women the association between FI and age is probably not linear, since FI usually has a late onset, in most women after the age of 40.[84,85] A number of studies have shown an association between BMI and faecal/anal incontinence.[86,87] Weight reduction has also been shown to result in improvement of FI.[88]

The association between sphincter injury at delivery and subsequent late incontinence to stool is complex. To date much attention has been focused on overt sphincter and levator ani injuries, but due to methodological deficiencies results are conflicting even between studies of women at a time remote from the date of birth.[89-91] In primiparous women, clinical sphincter injury has been demonstrated in about 7% (Swedish MBR data) and by endoanal ultrasonography in up to 35%.[39] The rate of occult defects in women with early symptoms that persist, and those with late onset incontinence has been reported to be 65% to 95%.[92,93] However, even if deficiencies in bowel continence are common during the first months postpartum,[39,40,81] a majority of women with early problems will recover,[94-98] within the first year.[99] There is also a poor correlation between the extent of sphincter injury and the severity of clinical symptoms[100-102] and many patients with occult injuries at ultrasound report no impairment of continence function.[103]

The role of episiotomy for subsequent bowel continence is uncertain. A systematic review in 2005 concluded that the effect of episiotomy on the development of pelvic floor disorders remains unknown.[104] Several studies have shown that midline episiotomy is an independent risk factor for sphincter injury,[96,105] but data on sphincter damage in association with medio-lateral episiotomy are conflicting. In a prospective study of 241 obstetric nulliparous women Andrews et al. found that mediolateral episiotomy was a risk factor for anal sphincter rupture.[106] Contrary to this, in the study of Poen et al. mediolateral episiotomy was shown to be protective for lacerations in nulliparous women.[107] It has been proposed that the use of an

inappropriate episiotomy technique may be one explanation for the diverging results.[108]

Combined symptoms – pelvic organ prolapse and urinary incontinence

The observation that prolapse is associated with a higher prevalence of urinary incontinence was first reported by Olsen et al. in 1997.[109] In a community-based study symptomatic pelvic organ prolapse was shown to be associated with an increased risk of having urge or stress incontinence or both compared to women without sPOP.[71] These findings have later been confirmed by the EPIQ cohort study of community-dwelling women aged 25-84 years. It was found that 11.3% of women had prolapse and 57.4% of these also reported isolated SUI or overactive bladder or a combination of both.[110]

Aims of the study

The overall objective of this thesis was to study the influence of childbirth on the long-term prevalence of pelvic floor dysfunctions and some of their putative obstetric and non-obstetric risk factors, by comparing one vaginal delivery with one caesarean section.

The specific aims were

Paper I To investigate the prevalence and risk factors for urinary incontinence and UI >10 years after vaginal delivery and caesarean section 20 years after one childbirth.

Paper II To determine the prevalence of symptomatic pelvic organ prolapse and concomitant urinary incontinence in women 20 years after one vaginal or one surgical delivery and to analyse the relative importance of some obstetric and non-obstetric risk factors for sPOP.

Paper III To investigate the effect of vaginal delivery compared to caesarean section on the prevalence, severity, and subjective perception of stress, urge, and mixed urinary incontinence in women 20 years after one birth.

Paper IV To investigate the prevalence and risk factors for anal and faecal incontinence and the severity of incontinence in women 20 years after one vaginal or one caesarean delivery.

Methods

Classifications/Definitions/Symptoms

Body mass index (BMI) was calculated from the height and weight of the women according to the formula kg/m^2. BMI was categorized as normal (<25), overweight (≥25-29.9) and obese (≥30) according to the WHO classification.[111] In this study BMI was registered in early pregnancy (week 8-10), at delivery, and 20 years after delivery in 2008 (current BMI). Early pregnancy and delivery BMI were registry data whereas current BMI was self-reported in the questionnaire.

Caesarean section done before the onset of labour was classified as an elective caesarean section and caesarean done during labour was denoted as an acute caesarean section. Only women with unequivocal information about ECS/ACS in the MBR were included in the study.

Perineal tears were classified according to the WHO International Classification of Diseases into four degrees. A first-degree tear involves the forchet, the perineal skin, vaginal epithelium but not the underlying fascia and muscles. A second-degree tear also involves the fascia, muscles, perineal body but not the anal sphincter. A third-degree tear involves the anal sphincter, but does not extend through the rectal mucosa. A fourth degree tear is defined as extending also through the rectal mucosa.[112]

Urinary incontinence was defined according to the International Continence Society and by the question 'Do you have involuntary loss of urine?'.[113] Participants reporting UI were classified as having stress urinary incontinence if there was involuntary loss of urine in connection with coughing, sneezing, laughing, or lifting heavy items. Urge urinary incontinence was present if loss of

urine occurred in connection with a sudden and strong urge to void and mixed urinary incontinence if both components were present.[113]

Severity of UI was measured by the Sandvik severity index and calculated by multiplying the reported frequency (four levels) by the amount of leakage (two levels). The resulting index value (1-8) was further categorised into slight (1-2), moderate (3-4), and severe (6-8). The severity index has been validated against a 48-hour pad-weighing test.[114,115] According to this validation, slight incontinence means a leakage of 6 g/24 hours (95% CI, 2–9), moderate incontinence means a leakage of 17 g/24 hours (95% CI, 13–22), and severe incontinence means a leakage of 56 g/24 hours (95% CI, 44–67). The severity index is thus a semi-objective and quantitative measure and does not include women's subjective perception of her leakage as being a problem or not.

Duration of UI was categorized into UI lasting less than 5 years (UI<5 years), UI lasting 5-10 years (UI 5-10 years), and UI lasting for more than 10 years (UI>10 years).

Bothersome UI was dichotomised to two levels: minor problem (no problem/a small nuisance) or bothersome (some bother/much bother/a major problem). A question about the impact of incontinence was also included. Significant urinary incontinence was defined as bothersome incontinence in women with moderate to severe incontinence according to the Sandvik severity index. 'Significant incontinence' in this context is thus not related to the statistical term.[116]

Symptomatic pelvic organ prolapse was defined by the affirmation of the symptom 'feeling a bulge' or affirmation of the combination of all four other pelvic floor symptoms: 'vaginal pain/discomfort' (often), 'worsening upon heavy lifting' (yes), 'need for manual reduction of the anterior vaginal wall' (often /sometimes

/infrequently), and 'urge urinary incontinence' (often/sometimes/infrequently) might also lead to a sPOP classification.[117]

Anal incontinence was defined as any involuntary leakage of liquid stool, solid stool or gas (any = isolated or in combination).

Faecal incontinence included any degree of leakage of liquid and/or solid stool only, but not gas leakage.

The severity of bowel incontinence was measured using the 5-item questionnaire of Jorge and Wexner[118] that denotes incontinence as either absent (never) or the presence for each symptom (solid, liquid and gas) and the frequency of episodes for each type of leakage on a scale from 0 to 4 (0 = absent; 1 = <l/month; 2 = >l/month but <l/week; 4 = ≥l/day). In this study the alternative 3 = ≥1/week but <1/day was however omitted for conformity reasons of the questionnaire. When coping items such as need to wear a pad or a diaper and the impact of incontinence on daily activities were included the overall severity score ranged from 0 (continent) to 20 (complete incontinence). Total scores of 1 to 3 were defined as minor, scores 4-8 as moderate, and scores 9-20 as severe. Scores ≥ 9 have been shown to indicate a significant impairment of quality of life. [119]

The Swedish Medical Birth Register

The Swedish Medical Birth Register was founded in 1973 and includes data on practically all deliveries (in the 1980s more than 98%) in Sweden. It is compulsory for every health care provider to report to the register and the information available is collected from medical records from the antenatal, delivery and neonatal care units. Data are collected prospectively, starting at the first antenatal visit and

contains information on maternal data such as height, weight, smoking habits, concomitant diseases, socio-demographic factors, parity, and complications during pregnancy. Obstetric parameters such as gestational age at delivery, infant birth weight and head circumference, perineal tears, instrumental delivery, induction of labour, and mode of delivery, etc. The register is validated annually against the National Population Register, using the mother's and infant's unique personal identification numbers. The robustness of the national database has been evaluated and published by the National Board of Health and Welfare (http://www.socialstyrelsen.se/publikationer2002/2002-112-4).

Misdiagnosis of 'parity' is predominantly related to immigration. The first birth in Sweden is sometimes misdiagnosed as the first ever. The MBR has reported that 10.9-11.9% (in 2002) of non-Swedish born women have an incorrect 'parity' in the registry and therefore we included a control question in the questionnaire about the total number of children delivered by the participant. Some other examples from the MBR quality declaration are:

1. In a sample of 440 women who gave birth, 43 of 1264 diagnoses obtained by reviewing hospital records were incorrect in the MBR (3.4%). 'Mode of delivery' was not among the 20 most common misclassified diagnoses.
2. In a sample of 451 women information about 'maternal length' in the maternity record was compared with registry data. Data were incorrect in 2 cases (0.4%) and missing in 11 cases (2.4%).
3. In a sample of 526 children 'infant birth weight' was incorrect in 1 case (0.2%) and missing in 3 (0.6%) cases.
4. MBR data about 'infant head circumference' were missing in 1.0-2.3% of all cases between 1973 and 1989.

When individual data for maternal body weights was initially examined in this project by plotting it was noted that the maximum registered body weight from

the MBR was 99 kg. It was by this means noted that due to lack of data storage capacity in the 1980s the MBR had decided to restrict registration of 'heavy women' by recording weight up to two digits only. We therefore reviewed the patient records of the 300 women recorded as having a body weight of 99 kg to obtain the correct weights of these women.

For the SWEPOP-study the following parameters were obtained from the registry: maternal age and date of birth, parity, weight gain during pregnancy, maternal BMI at delivery, infant birth weight and head circumference, mode of delivery, gestational weeks, instrumental delivery, episiotomy, perineal tear, etc. It was not possible to assess the importance of the length of the second stage of delivery, as this is unfortunately not documented in the MBR.

Selection of the study population

Women who participated in this study were obtained from the Medical Birth Registry at the Epidemiology Center of the National Board of Health and Welfare in Sweden. Inclusion criteria for participation in this study were primiparae with one single birth 1985-1988 and no further births. Exclusion criteria were multiparity and multifetal or on-going pregnancy.

Women with a BMI <25 at delivery and who had given birth to a child <4000g were chosen at random from the total population who had one single birth between 1985 to 1988 and no further births. All women with a BMI ≥25 and women who had given birth to a single child ≥ 4000g or had a recorded elective caesarean section during the same calendar period were included in order to obtain sufficient numbers of these groups.

We however deliberately chose to include all types of vaginal deliveries (breech presentation, instrumental delivery etc.) since the main consideration in this study was to compare vaginal delivery with caesarean section *in toto*. Women were thus

included regardless of maternal health status and maternal and fetal complications, for a greater generalisation of results and therefore a more realistic basis for consultation.

A total of 10 117 women who fulfilled these criteria were obtained from the MBR and addresses for 9 423 of these women could be traced in the Swedish Population Address Register (SPAR), which includes all persons who are registered as residents in Sweden. The difference, 694 women, was due to newly deceased women or women with unknown address or hidden personal identity.

A letter was thus sent to 9 423 women who were asked to provide written, informed consent to participate and to complete an enclosed questionnaire. After three mailing cycles during a four month period the questionnaire was returned by 6 148 women. Of the 6 148 that returned questionnaires 6 060 women were able to participate or gave their informed consent for participation in the study. A further 824 women were excluded from the study since they affirmed multiparity, multifetal, or on-going pregnancy in the questionnaire. In this study the following numbers of women were excluded: 716 due to multiparity; 43 due to multifetal pregnancy; 6 due to on-going pregnancy, and 59 due to missing data about parity in the questionnaire. Thus 5 236 women constituted the final study population (Figure 3).

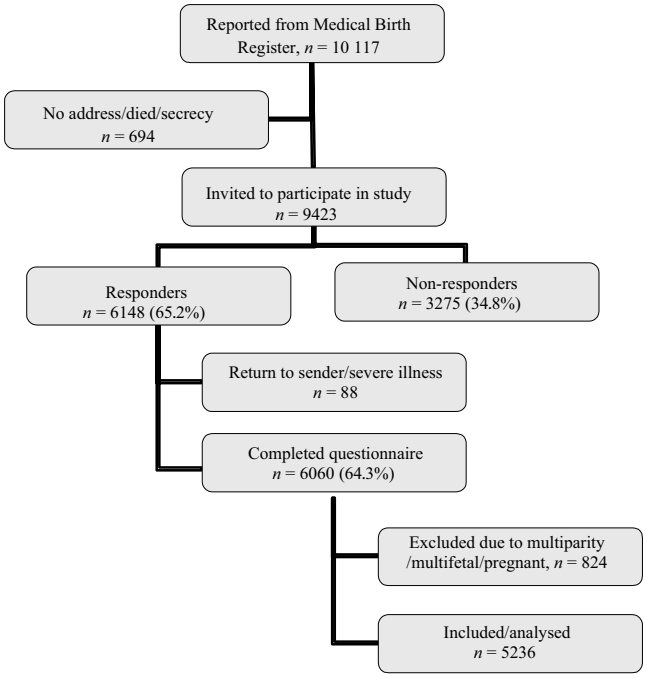

Figure 3. Flowchart of the women who gave birth to one child 1985–1988 identified from the Swedish Medical Birth Register (MBR).

Questionnaires

The 31-item questionnaire included questions about current height and weight, urinary incontinence, faecal and anal incontinence, and genital prolapse, menstrual status, hysterectomy, the menopause, hormone treatment, etc. The questionnaire was divided into three different sections serving both as a symptom inventory and a measure of degree of bother and distress caused by urinary, faecal and anal incontinence and symptoms of genital prolapse.

The first section of the questionnaire covered basic information about micturition habits. Those who confirmed UI gave further information about duration, frequency, and amount of leakage. The severity of leakage was estimated by using the severity index by Sandvik.[114]

The second section included a combination of five questions shown to predict the presence of sPOP reported by Tegerstedt et al.[117]

The third section included questions about bowel incontinence of gas, stool (solid and/or liquid), wearing pads, and impact on daily life according to the Wexner Continence Grading Scale.[118] This scale has become widely used for assessment of anal incontinence severity and evaluation of outcome after treatment. It is simple to use and easily understood by patients.[120]

The women were also asked about help-seeking behaviour, treatment, and impact on daily life (frustration, bother, impact on quality of life) for UI, sPOP and anal/faecal incontinence. Impact of UI, sPOP, and AI/FI on activities of everyday life was assessed using the short-form Incontinence Impact Questionnaire (IIQ-7), [121] which includes questions about physical and social activities, travel and emotional health. The specific questions in Swedish are shown in Appendix 1.

Characteristics of the sample population and the non-responders

Women delivered by CS were older ($p<0.001$) and gave birth to an infant with a lower birth weight ($p<0.001$) and at a lower gestational week ($p<0.001$) compared to women after VD. The proportion of women with an age at delivery ≥ 35 years was higher ($p<0.001$) in the CS group whereas the proportion of infants with a birth weight ≥ 3500 g was lower ($p<0.001$) in the CS group compared to the VD group. The mean current age when answering the questionnaire was 53.7 years (SD 6.3) in the CS group and 50.4 years (SD 5.6) in the VD group. The mean follow-up was 21.5 years (SD 1.5) in VD group and 21.8 years (SD 1.1) in the CS group. The proportion of missing data varied between 0% (age) and 15.9% (hysterectomy) in the cohort. There was little difference in the proportions of missing data between groups, e.g. the proportion of missing data for hysterectomy, which had the greatest proportion of missing data, was 15.5% (620/3995) in the VD-group and 17.0% (205/1204) in the CS-group. The non-responders were 1.6 years younger (49.6

years ± 5.9 vs. 51.2 years ± 5.9; p<0.001), and they were more often overweight or obese (37% vs. 27%; p<0.001 and non-responders more often delivered a child with a high birth weight (≥4000g) compared to responders (48% vs. 42%; p<0.003).

Selection bias

In the SWEPOP study the response rate was 65.2% after three mailing cycles during a four-month period and therefore some considerations about selection bias must be done. First, symptomatic women have been shown to be more predisposed to participate in studies and therefore symptoms of PFD might have been overestimated.[122] Secondly, the symptoms were self-reported. However, several studies have shown that self-reported symptoms are consistent and valid when they exist at the time of report.[63,123,124] Thirdly, it has been shown in a community based population[117] that sensitivity and specificity for the question 'feeling a bulge' to correctly identify genital prolapse according to the POPQ system were 66.5% and 94.2% respectively. However, it should be noted that although every third woman with prolapse was false negative, most of the missed cases (72.5%) were stage I prolapses only. In our study, as also in the study of Tegerstedt et al.,[117] all the women who reported a combination of symptoms indicating sPOP in fact also reported the single symptom 'feeling a bulge', indicating the discriminatory importance of this single symptom. Further, analyses of the non-responder group suggest a small selection bias on our results acting in both ways (younger women leading to overestimation of results; overweight/obesity among the non-responders to the opposite). We were not able to analyse if the non-responder group were equally distributed between the two modes of delivery. Whether there is a selection bias due to non-responders has however been challenged.[125]

Statistics

A major objective of the statistical analysis in this study was to create models that allowed calculation of prevalence rates, adjusted for all relevant cohort characteristics, risk factors and confounders to demonstrate the clinical relevance of the findings. Statistical analysis was performed with SAS 9.1 (SAS Institute Inc., Cary, NC, USA). Chi-square test was used to compare categorical variables and Student´s t-test to compare continuous variables. BMI, maternal age, infant birth weight, gestational week were treated as continuous variables and were sometimes stratified. Stratification was performed according to the following: Maternal age groups: <23; 23-29; 30-34; ≥35 years; BMI: Categories of BMI were the same as the WHO classification; Infant head circumference: Dichotomised to ≤ 35 cm or > 35 cm; Infant birth weight: The most commonly used stratification in the literature starting at <3000g and with intervals of 500g; Maternal height and infant birth weight: Maternal height was dichotomized into ≤160 cm or >160 cm and the birth weight into <4000g or ≥4000g.

Logistic regression analysis was used to demonstrate independent risk factors for UI, sPOP, and bowel incontinence while controlling for potential confounding factors. Potential risk factors used in the analysis were mode of delivery, maternal age at delivery, BMI, hysterectomy, hormone replacement therapy, gestational age, infant birth weight, and head circumference. Linear regression analysis was used to analyse risk factors for severity of UI. Potential risk factors were mode of delivery, infant birth weight, current BMI, maternal age, subtypes of UI, duration of UI, and sPOP. Odds ratios and their 95% confidence intervals were calculated from the model. The Wald odds ratio was used to test for multiple parameters simultaneously. Adjusted prevalence rates and odds ratios were calculated using a covariance analysis model and was performed for maternal age, infant birth weight, current BMI and infant head circumference (only for the subgroup analysis of the

VD group). These variables were considered potential confounders and effect modifiers on the basis of a combination of clinical considerations and the significance of risk factors identified in the logistic regression analysis. Because the regression models require that there are no missing data for the dependent and all of the independent variables, the number of women included in each analysis varied, and therefore usually decreased for each additional variable introduced in the analysis. A P-value of <0.05 was considered statistically significant. Non-linearity and possible threshold effects of the stratified variables (current BMI, infant birth weight and maternal age) were analysed for vaginal delivery and caesarean section separately. To address some specific obstetric events (vacuum extraction, disproportion, episiotomy) associated with vaginal delivery, subgroup analysis was performed and presented separately. The prevalence data permitted the calculation of the number of caesarean sections needed to avoid one case of UI, sPOP, AI and FI using the number needed to treat principle (NNT). The NNT was calculated as the inverse of the absolute risk reduction, where risk reduction was the difference of adjusted prevalences between vaginal delivery and caesarean section.

Ethical considerations

Ethical approval was obtained from the Regional and the National Ethic Review Boards (the Ethics Committee at Sahlgrenska Academy, Gothenburg University, and the National Board of Health and Welfare). All women received written information and gave their written consent before participation in the study.

Results

Paper I

The aim of this study was to investigate the prevalence and risk factors for urinary incontinence 20 years after one vaginal delivery or one caesarean section.

The odds ratio for UI was 67% higher (OR 1.67, 95% CI; 1.45-1.92) after a vaginal delivery (prevalence 40.3%) compared to women who had been delivered by caesarean section (prevalence 28.8%). Furthermore, the prevalence and risk increase of UI for more than 10 years almost tripled after VD compared to after CS. The prevalence of UI>10 years after VD was 10.1% compared to 3.9% after CS (OR 2.75, 95% CI: 2.02-3.75). There was however no significant differences in the prevalence of UI (27.1% vs. 24.4%, OR 1.15, 95% CI: 0.88-1.51) or UI for more than 10 years (6.5% vs. 5.1%, OR 1.30, 95% CI: 0.79-2.14) between women delivered by acute caesarean section or elective caesarean section respectively. Calculation of NNT showed that it is necessary to perform 8-9 caesarean sections to avoid one case of UI and 16 caesarean sections to avoid one case of UI> 10 years.

In the multiple regression analysis the following variables were found to be significant risk factors of UI. In order of degree of association these were: VD, current BMI, and maternal age. BMI at delivery, gestational age, infant birth weight, and head circumference, hysterectomy and oestrogen replacement therapy were not risk factors.

We found an 8% (range 6-10%) increased risk of UI per BMI unit increase and the increased rate of UI was apparent for both modes of delivery. The prevalence of urinary incontinence was higher after VD compared to CS for each current BMI class (BMI<25, BMI≥25-29.9, and BMI≥30) with differences ranging from 11 to 14%. The combined effect of BMI and mode of delivery was substantial. For example the adjusted frequency of UI after CS with a current BMI<25 was 24.7%

whereas the prevalence more than doubled to 54.8% after VD with a current BMI≥30. When using 'normal BMI' as reference the risk of UI increased significantly for both overweight and obese women after both modes of delivery. The risk increase of UI in obese women more than doubled in comparison to women with a normal BMI after VD (OR 2.50, 95% CI 2.10–2.98) and more than tripled after CS (OR 3.27, 95% CI 2.34–4.59). There seems to be a dose-response relationship between BMI and the rate of UI for both modes of delivery.

A higher maternal age was associated with an increased risk of UI (OR 1.03, 95% CI 1.02–1.04), which corresponds to an annual risk increase of 3% per year. The prevalence of UI increased by 7-10% from maternal age <23 years to age ≥35 for both modes of delivery. This means e.g. that childbirth at the age of 40 years in comparison with 20 years of age increases the risk by 81%.

The prevalence of UI was higher after VD compared to CS in all infant birthweight groups except for weights <3000 g. For women delivered vaginally rates of incontinence increased with increasing infant birthweight but this was not observed after CS. The logistic regression showed however no significant association with UI for infant birthweight (OR 1.00 95% CI 0.98–1.02).

Paper II

The aim of this study was to determine the prevalence of sPOP and concomitant UI in women 20 years after one single pregnancy terminating either in a vaginal or a surgical delivery and to analyse the relative importance of obstetric and non-obstetric risk factors for sPOP.

The overall prevalence of sPOP was 12.8% (663/5199). The number of women with sPOP in the VD group was 588/3995 (14.7%) and in the CS group [75/1204 (6.2%); 48/766 (6.3%) elective CS and 27/438 (6.2%) acute CS]. The adjusted prevalence of symptomatic POP was more than doubled after vaginal delivery compared with caesarean section [14.6% versus 6.3% (OR 2.55; 95% CI 1.98-

3.28)]. These results indicate that 12 caesarean sections need to be performed to avoid one case of sPOP. There was however no significant difference in the adjusted prevalence or odds of sPOP after acute caesarean section compared with elective caesarean section [6.0% versus 6.3% (OR 0.95; 95% CI 0.58-1.57)]. The prevalence of UI was higher in women with sPOP [61.8% versus 34.8% (OR 3.02; 95% CI 2.54-3.59)] and the prevalence of UI >10 years more than doubled in women with sPOP [16.7% versus 7.6%, (OR 2.43; 95% CI 1.93-3.07)] compared with those without sPOP. Vaginally delivered women with sPOP had an almost tripled prevalence of UI compared with caesarean section delivered women with sPOP [9.1% versus 2.8% (OR 3.48; 95% CI 2.41-4.99)]. Furthermore, women who were delivered vaginally and who had sPOP reported UI>10 years 5 times more often compared with women delivered by caesarean section with sPOP [2.6% versus 0.5% (OR 5.22; 95% CI 2.29-11.92)]. Of the 383 women with a combination of sPOP and UI 351(91.6%) had been delivered vaginally and of 110 women with the combination sPOP and UI>10 years 105 (95.5%) had been delivered vaginally. The prevalence of either sPOP or UI or sPOP and UI in combination was 45.5% in women who had a vaginal delivery. The corresponding prevalence of either sPOP or UI or sPOP and UI combined for women delivered by caesarean section was 33.2%. These results mean that 8 caesarean sections have to be performed to avoid one case of either UI or sPOP or the combination of UI and sPOP.

The logistic regression analysis showed that vaginal delivery, infant birthweight and current BMI were significant risk factors for sPOP. There were no effects demonstrated for gestational age, infant head circumference, maternal age, hysterectomy, or oestrogen replacement therapy. The odds of sPOP increased 3% (OR 1.03; 95% CI 1.01-1.05) for each unit increase of BMI. With normal current BMI as reference the odds of sPOP increased significantly for both overweight and obese women in the vaginal delivery group. The prevalence of sPOP increased from 12.4% in women with BMI<25 to 19.4% in women with BMI>30 after

vaginal delivery. In women delivered by caesarean section the corresponding prevalences were 4.7% to 7.4%.

There was also a 3% increased odds of sPOP (OR 1.03; 95% CI 1.01-1.05) for each 100 g increase of infant birthweight. The prevalence of sPOP showed a numerical trend towards higher rates with increasing infant birthweights, which was significant for a birthweight ≥4500 g compared to <3000g. The prevalence of sPOP was 11.8% in women who delivered a child <3000g vaginally and 23.8% after delivering a child ≥4500g. This effect of was not observed after caesarean section. For all infant birthweights the prevalence of sPOP was less than 8% after caesarean section.

The combined factor of birthweight and maternal height together for vaginally delivered women (disproportion) was found to be important for the prevalence and risk of sPOP. Mothers ≤160 cm who delivered a child with a birthweight ≥4000g had a higher prevalence of sPOP compared with short mothers who delivered a small child <4000g; (24.2%, n = 91 versus 13.4%, n = 484; OR 2.06; 95% CI 1.19-3.55). This effect of disproportion was not observed for mothers >160 cm regardless of infant birthweight. The prevalence and odds of concomitant sPOP and UI was doubled and the prevalence and odds of concomitant sPOP and UI>10 years was tripled after giving birth to a child with birthweight ≥4000g in women of short stature (≤160cm). The prevalence and odds of concomitant sPOP and UI>10 years was also doubled for mothers >160cm who gave birth to a child with birthweight ≥4000g. No effect of maternal height and infant birthweight was seen in the caesarean section group on prevalence of sPOP, sPOP and UI, and sPOP and UI>10 years.

None of the obstetric events, vacuum extraction, episiotomy, or ≥ 2nd degree lacerations was significantly associated with an increased prevalence or odds of sPOP. The prevalence of sPOP after spontaneous vaginal delivery was similar to the prevalence of sPOP after birth assisted by VE, between women who had an

episiotomy compared with those without episiotomy and between women who had ≥ 2nd degree lacerations and those without or with a <2nd degree laceration.

Paper III

The aim of this study was to investigate the effect of vaginal delivery compared to caesarean section on the prevalence, severity and subjective perception of stress, urge, and mixed urinary incontinence in women 20 years after one birth.

In the total cohort (n = 5118) SUI occurred in 15.3%, UUI in 6.1%, and MUI in 14.4%. The prevalence of all subtypes of urinary incontinence (SUI, UUI and MUI) was significantly higher after VD compared to CS and the increase was 4.4% for SUI, 2.8% for UUI, and 4.7% for MUI. The percentage of each subtype was however very similar in women after both modes of delivery (Figure 4).

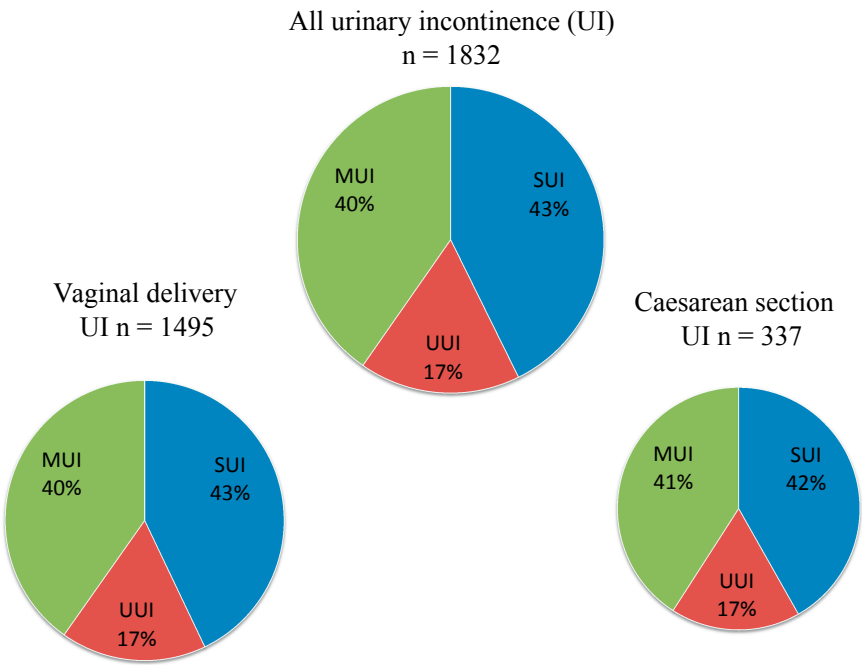

Figure 4.

Of all incontinent women (n = 1899) in this study, moderate to severe incontinence according to the Sandvik score (3-8) occurred in 68.1% of women with MUI, in

53.7% with UUI and 40.9% of women with SUI. The difference in severity between the three subtypes was significant, MUI being most severe and SUI least severe (Figure 5).

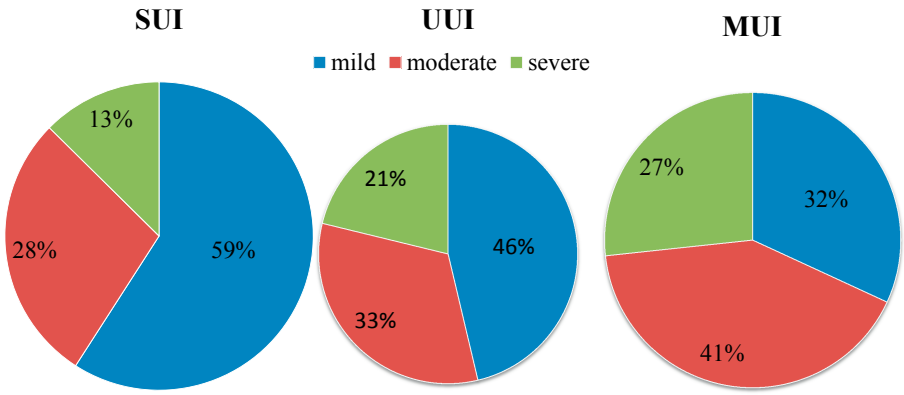

Figure 5. Severity of urinary incontinence and each subtype according to the Sandvik severity index (n = 1899)

The prevalence of moderate to severe incontinence was significantly higher for each subtype of incontinence after vaginal delivery compared to caesarean section and the difference in prevalence between the two modes of delivery was 7.8% (OR 1.72; 95% CI 1.44-2.07). In women with incontinence after both modes of delivery the proportion of moderate to severe incontinence (score 3-8) was however similar. The prevalence of bothersome UI was significantly higher after VD compared to CS (11.2% vs. 6.3%, OR 1.88; 95% CI 1.46-2.41). Of all the incontinent women (VD+CS), bothersome incontinence was reported by 27.2% (449+83/1954) and

13.2% (251/1899) had consulted a doctor. Of all incontinent women bothersome incontinence was 5.4% higher after VD compared to CS (OR 1.33; 95% CI 1.01-1.75). The percentage of bothersome incontinence was more than doubled (38.9%) for MUI compared to SUI (18.0%) and was 27.1% for UUI. Bothersomeness occurred significantly more often in vaginally delivered women having MUI compared to women delivered by caesarean section having MUI (10.1%; OR 1.56 95% CI 1.04-2.32). The prevalence of significant urinary incontinence was 9.7% after VD compared to 5.7% after CS (1.80; 95% CI 1.38-2.35). Linear regression analysis showed that urgency incontinence, duration of UI, sPOP, and BMI were risk factors of the severity of incontinence. Backward logistic regression analysis of risk factors for bothersome UI showed that sPOP was a significant risk factor for all three subtypes of UI; that duration of UI was a risk factor for UUI and MUI but not for SUI; and that BMI was a significant risk factor for MUI only.

Paper IV

The aim of this study was to investigate the prevalence and risk factors for anal and faecal incontinence and its severity in women 20 years after one vaginal or one caesarean delivery and to analyse the relative importance of obstetric and non-obstetric risk factors for AI and FI.

Regardless of mode of delivery, the prevalence of incontinence was high among middle-aged women after one pregnancy and delivery only: FI in 13.5% and AI in 47.0%. Any leakage (i.e. isolated or in combination) to solid stool occurred in 4.7%, to liquid stool in 12.7%, and to gas in 45.4%. Leakage of gas occurred as an isolated symptom in 71.4% (74.1% after caesarean section and 70.6% after vaginal delivery) of women with anal incontinence, whereas 96.3% of women incontinent to solid stool had at least one additional type of leakage. Mild symptoms (less than one episode/month) were most prevalent (liquid 75.5%, solid 66.1%, and gas

50.0%). The proportion of women having daily leakage was 4.7% for liquid, 11.4% for solid stool and 15.6% for gas respectively.

The prevalence of AI was significantly higher (5.5%) after vaginal delivery compared to caesarean section (OR 1.25; 95% CI 1.10-1.43) and also for FI (3.9%, OR 1.44; 95% CI 1.17-1.77). There was however no difference in the prevalence or odds of AI after acute compared to elective caesarean section (47.0% versus 43.0%, OR 1.17; 95% CI 0.92-1.49) or FI (12.7% versus 10.8% OR 1.20; 95% CI (0.83-1.73). These results indicate that 18 caesarean sections need to be performed to avoid one case of AI and 26 caesarean sections to avoid one case of FI.

For each quality of leakage (solid, liquid, and gas in any combination) the prevalence was higher after vaginal delivery compared to caesarean section, the difference being greatest for gas incontinence (5.1%, OR 1.23; 95% CI 1.08-1.41) and smallest for solid stool (1.7%, OR 1.56; 95% CI 1.10-2.22). However, isolated gas leakage did not differ statistically between the two modes of delivery. The proportion of vaginally delivered women having severe incontinence, according to the Wexner Continence Grading Scale, was higher compared to the proportion of women having severe incontinence after caesarean section (4.4% versus 2.8%, OR 1.86; CI 1.03-3.58).

The logistic regression analysis showed that vaginal delivery, current BMI, and maternal age were significant risk factors for AI and FI. There were no effects demonstrated for head circumference or infant birthweight.

For each unit increase of BMI there was an increased odds of AI by 3% (OR 1.03; CI: 1.02-1.04) and for FI by 6% (OR 1.06; 95% CI: 1.04-1.08). In a subgroup analysis of both modes of delivery it was found that BMI ≥30 in comparison with normal BMI was associated with an odds increase of approximately 50% (for caesarean section OR 1.55; 95% CI 1.15-2.09; vaginal delivery OR 1.41; 95% CI 1.19-1.67). Maternal age was a risk factor for both AI (4% yearly) and FI (3% yearly).

The prevalence of ≥ 2nd degree perineal tear was 4.2% (n = 166), of which 83.7% (n = 139) did not involve the anal sphincters. A ≥2nd degree perineal tear was associated with an almost doubled prevalence of FI compared to women without ≥2nd degree tears (22.8% versus 13.9%, OR 1.84; 95% CI 1.26-2.68). It was further calculated that the difference of ORs for FI between surgically and vaginally delivered women including or excluding perineal tear ≥2nd degree from the vaginal cohort resulted in a 5% decrease in odds of FI, from 44% to 39% (OR 1.39 95% CI 1.13-1.72). The prevalence of FI after episiotomy compared to vaginal delivery without episiotomy was 11.1% versus 14.7%, a risk reduction of 27% (OR 0.73; 95% CI 0.54-0.98). Vacuum extraction was not associated with an increased prevalence or odds of AI or FI. Disproportion between a mother ≤160 cm and infant birthweight ≥4000g compared to ≤160 cm and infant birthweight <4000 g was not a risk factor for neither AI (OR 1.01; 95% CI 0.64-1.58) nor FI (OR 0.81; CI 0.40-1.64).

General discussion

Study design

The ultimate goal of epidemiological research is the prevention of disease. If this principle is applied to the present study then the goal is to obtain information leading to the prevention of the unwanted consequences of childbirth on pelvic floor function. Such information would not only be useful to guide the evidence-based practice of doctors involved in delivery care but also provide useful information for women in their decision-making with regard to mode of delivery. Unfortunately, in this respect data currently available about the effect of vaginal birth versus caesarean section on the occurrence of pelvic floor disorders, is often inconclusive, and it is unlikely that randomized controlled trials can be applied to address this issue.

To date the majority of studies of the effect of childbirth on pelvic floor function have been done with short-term follow-up after an index birth. Long-term studies on the other hand are rare. The reason for this is obvious. A longitudinal study or a randomized controlled study is ethically, practically, and economically almost impossible to accomplish. Our study design is one method to circumvent this difficulty (the other being very large population studies) by combining historical registry data with current information from a questionnaire with current information on PFD.

The primary aim of this thesis was therefore not to examine the total impact of a woman's reproductive career on her pelvic floor but instead to quantify the effect of vaginal delivery in comparison with caesarean section on the prevalence of UI and subtypes of UI, sPOP, and on anal/faecal incontinence. The baseline outcome after

CS might be interpreted to represent the risk of pregnancy *per se* and the risk of VD to represent the risk of pregnancy plus VD and hence the difference being a measure of the vaginal birth trauma.

The population at risk in this study was a cohort of women fulfilling the inclusion criteria (primiparae) with one single birth 1985–1988. The index birth was therefore the only birth, thus avoiding the problem in many other studies, in which multiparity disrupts obstetric homogeneity. Since most risk factors also covariate with time/age also this will confound effect measures of the analysis. A further benefit of the design of this study was that all deliveries occurred within a short calendar period during the 1980s and also that the follow-up time was uniform (20 years). Definitions of outcomes followed internationally accepted and/or validated definitions of symptoms of UI, sPOP and bowel incontinence. The prevalence rates were consistently calculated by adjusting for relevant risk factors and the size of the cohorts of vaginally and caesarean delivered women were made large enough to detect also small differences between groups as could be expected for the prevalence of anal and faecal incontinence. Risk factors were identified for each of the three main outcomes and rated according to its degree of association. Stratification of risk factors was used to test non-linearity and possible threshold effects for vaginal delivery and caesarean section separately.

Methodological considerations

Internal validity

A study is internally valid if the conclusions of the study represent the truth for the individuals studied, i.e. it measures what it sets out to measure. Bias and systematic errors undermine the internal validity.[126]

Information bias

In this study only registry data from the MBR was used and this avoided recall bias about obstetric information. The robustness of the national database has been evaluated and has been published by the National Board of Health and Welfare (http://www.socialstyrelsen.se/publikationer2002/2002-112-4).

BMI is simple to measure and includes only the measurements weight and height in its calculation. It is highly reliable when measured and recorded by a midwife. However due to lack of data storage capacity in the 1980s the MBR had decided to restrict registration of "heavy women" by recording up to two digits only. We therefore reviewed the patient records of the 300 women recorded as having a body weight of 99 kg to obtain the correct weights of these women. However, current weight obtained from our questionnaire depended on the participants´ willingness and ability to measure and report correctly.

Information on parity in the registry is occasionally inaccurate, predominantly related to immigration (the first birth in Sweden is sometimes misdiagnosed as the first ever). Therefore subjects were excluded from the study, based on the answers in the questionnaire, if they affirmed multiparity.

Selection bias

Symptomatic women have been shown to be more predisposed to participate in studies and they therefore might be overrepresented.[122] It has also been shown that middle-aged women with severe incontinence have a higher response rate in surveys of UI compared to younger and elderly women.[127] This should however not influence the results of this study since the effect might be expected to influence both modes of delivery similarly.

Analysis of the non-responder group in this study suggests a small selection bias on our results however acting both ways (younger women and overweight/obesity to underestimation). We were not able to analyse if the non-responder group was equally distributed between the two groups of delivery. Whether non-responders cause selection bias in postal surveys on pelvic floor function has however been challenged.[125]

External validity

External validity refers to the generalizability of the findings. Papers I-IV were based on a random sample from a national database (MBR) of one-para women. For study efficiency reasons we over-sampled women recruiting all eligible women with a BMI>25 at delivery, women who delivered an infant with a birth weight >4000g, and women who had an elective caesarean section. This means that our results are generalizable for a population which is overrepresented with respect to mothers that are heavier, more often are delivered by caesarean section and give birth to heavier children, which in fact is what has been the trend in welfare states during the last decades and therefore is more representative for the current situation.

We deliberately chose to include all types of vaginal deliveries (all gestational weeks, abnormal presentations, instrumental delivery, etc.) since the main consideration in this study was to compare vaginal delivery with caesarean section *in toto*. Women were also included regardless of maternal health status, maternal and faetal complications for a greater generalisation of results and a more realistic basis for consultation.

The first delivery is considered to exert the greatest risk increase for pelvic floor dysfunction even if subsequent deliveries contribute to a further increase.[1,61] Our

finding cannot be generalised to women with multiple childbirths. The impact of additional childbirths on pelvic floor function will require further investigations. One single birth however probably represents the minimum impact that is further aggravated by each additional birth.

It might however also be of interest to consider that even if studying the consequences of giving birth to one child only may seem unrepresentative, UN data show that total fertility rates (TFR) are rapidly declining globally and the predicted TFR in the middle of this century is predicted to be less than 2.0 children/woman and in many developed countries the TFR is already between 1.0 and 1.5 (UN-data 2011.[2]

Strengths and limitations

The main strengths of this study are the large cohort of one-para women delivered vaginally or by caesarean section and the high response rate. To include also multipara would disrupt obstetric homogeneity of the cohorts. A constant follow-up time period of 20 years circumvents the fact that most risk factors covariate with time/age. Different follow-up periods (as in cross-sectional population studies) may confound effect measures of the analysis, which is of great importance in long-term follow-up studies, such as this study.

Pelvic floor symptoms were self-reported. However, several studies have shown that self-reported symptoms are consistent and valid when they exist at the time of report,[123,124] with respect to the type of UI,[128,129] and changes in incontinence severity over time,[130] which applies to our study. This study also lacks information on whether UI was present or not before or/and during pregnancy or started after delivery. However it is reasonable to presume that such symptoms before and during pregnancy are similarly distributed between the two modes of delivery. It is

also a shortcoming of this study that data on the length of the second stage of delivery is lacking because it was not documented in the MBR. A number of sensitive but maybe important questions about smoking, sexuality, and life-style factors were excluded from this survey. One reason for this was not to overload the questionnaire because such questions are known to significantly reduce the response rate.

Considerations of definitions and classifications

Validated questionnaires were used to identify pelvic floor dysfunction. When the study was designed in 2007, no validated complete questionnaire for all three major pelvic floor disorders was available. We therefore used three separate questionnaires – Sandvik's, Tegerstedt's, and Wexner's – combined into one.[114,117,118] The numbers and sequence of questions may have affected the answers, but if so, this bias ought to be equally distributed between the two major cohorts.

The determination of subtypes of UI from a questionnaire has been recommended to be included in epidemiology studies of UI,[1,129] since it has been shown that they are associated with different degrees of severity and bothersomeness.[116,131] Furthermore, Sandvik et al. has shown that clinical and urodynamic evaluations reveal that stress UI often is misdiagnosed as mixed UI in epidemiological surveys.[128] This circumstance does not however change the conclusions reached in this study, comparing the outcome after vaginal and caesarean birth, since it is reasonable to presume that this effect will affect our large cohorts in a similar way.

The high prevalence of UI in women obtained from epidemiology surveys has highlighted the need for some system of classification of severity to determine the relevance of the problem and to select patients for further evaluation and for treatment. Sandvik's severity index is such a semi-objective classification that

grades the severity of UI according to information collected by a postal questionnaire.[114] The subjective perception of the condition – if it causes bother or not, and to what degree – could be considered to be the crucial criterion as to whether the pelvic floor disorder carries enough relevance to motivate prevention and treatment.[1] This seems reasonable, provided that the symptom that causes the bother is not classified as mild. The concept 'significant' urinary incontinence is one way to increase the discriminatory power by considering women with moderate to severe UI only (as defined by the Sandvik severity index), and who at the same time state that their symptoms are troublesome. According to Hannestad et al., all women with significant UI should be regarded as potential patients and in need of treatment.[132]

The symptom of 'feeling a vaginal bulge' is the most strongly correlated threshold symptom for prolapse when the bulge protrusion reaches near the hymen.[133] For identification of symptomatic POP in this study the 5-item questionnaire from Tegerstedt et al. was used. Symptomatic prolapse was diagnosed according to a validated 5-item questionnaire and sPOP was defined by the key symptom 'feeling of a vaginal bulge' (often/ sometimes/infrequently). The positive predictive value was around 60%, while the negative predictive value was 97%. This means that approximately 40% of women classified as having sPOP, in fact did not have anatomical prolapse. Among the women classified as being without symptomatic prolapse no more than 3% were expected to have an anatomic prolapse. The effect of this misclassification would be to underestimate associations with risk factors. However the affect will be the same for vaginally and caesarean delivered women.[117]

In this study faecal incontinence was defined as the involuntary loss of solid or liquid faeces. Anal incontinence includes these events as well as the involuntary loss of flatus. We used the 5-item questionnaire of Jorge and Wexner[118] to classify incontinence as either absence (never) or the presence of each symptom of

incontinence. This scale does not rank the three modes of incontinence in terms of severity. The severity of incontinence was instead evaluated in terms of frequency of incontinence episodes for each type of leakage.

The lack of a standard definition as well as validated tools for the assessment of anal and faecal incontinence is a problem that hampers the ability to accurately compare prevalence rates.[134] Many studies (mainly based on telephone interviews) have reported a low prevalence of FI, but those figures are almost certainly too low as it is known that more than every second person with FI does not report the symptom to their doctors because of embarrassment or pessimism about its curability.[77]

Results and comments

Unspecified urinary incontinence and its subtypes (paper I and III)

The aetiology of UI is known to be multifactorial but female gender, obesity and aging as well as obstetric trauma during childbirth are known to be three of the most important risk factors.[1,135] Although several studies have demonstrated an association between UI and vaginal delivery in the short- and the medium long-term, the long-term effects of childbirth on the risk of UI remain controversial.[46,59,60] The high prevalence of UI in women obtained from epidemiology surveys has highlighted the need for some system of classification of severity to determine the relevance of the problem and to select patients for further evaluation and for treatment.[114] Determination of subtypes of UI from a questionnaire has been requested to be included in all epidemiology studies on UI,[1,129] since it has been shown that they are associated with different degrees of severity and bother.[116,131]

Vaginal delivery

In paper I we found an increased odds ratio for UI after VD amounting to 67-71% compared to after CS. The prevalence of UI lasting more than 10 years almost tripled after VD compared to CS. The prevalence of UI was 10.7 % higher after VD compared to CS and UI lasting more than 10 years more than doubled after VD compared to CS (9.6% vs. 4.5%). Several studies have demonstrated that postpartum UI is a risk factor for UI after varying terms of follow-up.[59,60,66] There is however still no general agreement as to whether or not the long-term maternal effects of the two delivery modes differ with regard to prevalence of UI. The prospective multicentre study of McKinnie et al. did not show any significant difference of risk for bothersome UI between women delivered by one or more VD compared to one or more CS.[136] Also the omnibus survey of MacLennan et al. could not demonstrate an increased risk for any type of UI after VD when compared to CS.[70] In these studies however the CS groups were relatively small and heterogeneous with respect to parity. In the EPINCONT study a 1.7-fold increase was demonstrated of UI after one or more VD compared with one or more CS.[46] Some later studies have also indicated an increased risk of UI following VD compared to CS.[137-139]

In paper III we found that the prevalence of SUI, UUI and MUI was higher after VD compared to CS. This was also the case for the severity of UI, the severity of each subtype of UI and bothersome UI. The percentage of moderate to severe incontinence according to the Sandvik severity index was higher after VD compared to CS. Symptomatic pelvic organ prolapse was found to be an important modifier of urinary incontinence with regard to its prevalence, duration, type and degree of bother.

Reduction of the prevalence and odds of SUI after caesarean section compared to vaginal delivery has been demonstrated in a number of cross-sectional and cohort

studies. Data on UUI and MUI are however conflicting. Most cross-sectional and cohort studies with short or 1-year and longer follow-up could not demonstrate a significant difference in the prevalence of UUI and MUI between the two modes of delivery.[66] One reason for this discrepancy may be lack of statistical power.[49,51,140] In the EPINCONT cross-sectional study the difference in prevalence between VD and CS was significant for SUI, but not for UUI and MUI. This may be attributed to heterogeneity of parity (1-4), a large proportion of younger women (55% <40 years), and the relatively small fraction (6%) of caesarean sections in that study.[46]

Body mass

Several studies have reported that a higher BMI is a risk factor for unspecified UI[1] and cross-sectional studies have confirmed this association in middle-aged women.[141,142] In a couple of recent large cross-sectional studies of an Asian population, waist circumference, but not BMI, was associated with SUI.[143,144] A majority of studies in other ethnic groups have however observed that higher BMI is a risk factor for UI.[145,146] In a cross-sectional study of Mommsen and Foldspang in women aged 30-59 years, BMI was positively associated with UI (4-10% risk increase per BMI unit).[147] Waetjen et al. followed middle-aged women for 2 years and found that the risk of developing UI was 2-7% per unit increase of BMI.[62] In the SWAN study of middle-aged women a 5 % (4 %-7%) increased risk of UI with each 1-unit increase of BMI was found.[142]

In our study maternal weight was an important risk factor and in the logistic regression analyses we found an 8% increased risk of UI per BMI unit increase and was apparent for both modes of delivery. The relationship between BMI and prevalence of UI in this study indicated that there was a dose-response relationship between BMI and UI. Current BMI was the most important BMI-determinant for UI. Prevalence rates of UI for stratified BMI in this study showed that UI prevalence increased from 24.3% and 34.1% respectively for CS and VD with

normal BMI <25 to 43.2% and 53.3% respectively for CS and VD in obese women with BMI ≥30. In the logistic regression analyses it was found that BMI was a risk factor for bothersome UI and bothersome mixed UI but not for bothersome stress UI or urge UI. Several cross-sectional studies have demonstrated a positive association between BMI and the severity of incontinence and that the association with BMI was greater for stress or mixed UI but was relatively modest for UUI.[148-150.]

Age

Since women in this study reported on urinary continence two decades after delivery the effect of age contains both the risk of UI due to biological aging in itself, i.e. current age at the time of measure and the risk of UI associated with the age at delivery. In our study higher maternal age was associated with an increased risk of UI with 3% yearly. Most risk factors for urinary incontinence co-vary with age[143,151,152] so even if aging per se has been shown to be strongly associated to UI in many studies, its impact, in the absence of adjustment for confounders (parity, co-morbidities, BMI, etc.), may be overestimated.

Rortveit et al. showed that UI was more than 2 times more frequent in nulliparae aged 40-49 years than in those aged 20-29.[46] A few studies have reported that older age at first birth is associated with an increased risk of later UI.[150,153-155] Persson et al. found that higher age at the first, uncomplicated vaginal delivery was associated with a significant higher risk of later incontinence surgery.[153] In the study of Kuh et al. of 48 year old women, those aged > 30 at first birth had an adjusted odds ratio for SUI of 3.1 (CI 1.5-6.0) after one or more VD with nulliparous as reference.[150] Rortveit et al studied 11 397 women after 1-4 VDs only. Prevalence of UI was significantly higher (about 5 %) in women who were older than 25 years at their first birth compared with women younger than 25 years.[154]

The prevalence of subtypes of UI is age-dependent. Pooled data from 14 studies showed that the prevalence of SUI peaks in the 4th decade and then declines gradually with increasing age, whereas MUI starts to increase in the 4th and UUI in the 6th decade. The prevalence of all three subtypes subsequently converges to approximately 30% in the 8th decade.[68] In the multiple regression analysis of this study it was found that age was not an independent risk factor for neither severity nor bother of unspecified UI. One interpretation of this finding may be that age is a surrogate measure for the effect of the progressive dominance of the symptom of urge incontinence that occurs with increasing age.

Infant birthweight

Several studies have reported that there is no association between high infant birthweight (>4000 g) and UI after vaginal delivery.[156-158] In one study high birth weight (≥4000 g) was however associated with stress urinary incontinence.[135]

We found that the prevalence of UI after VD was higher than after CS in all infant birthweight groups except for weights <3000 g. In the group of women who delivered vaginally UI prevalence increased with increasing infant birth weight but this trend was not observed after CS. We also found high infant birthweight ≥4500g to be a risk factor for bothersome UI.

Symptomatic pelvic organ prolapse as risk factor for UI (paper II)

The observation that POP is associated with a higher frequency of urinary incontinence was first reported by Olsen et al in 1997 and later confirmed by Buchsbaum et al and in the EPIQ study.[109,110,159] All three studies reported that about 60 % of women with POP also were diagnosed with UI. These findings are very close to our results that 61.8% of all women with sPOP also reported UI compared with 34.8% of women with no sPOP. In our study sPOP was a strong risk factor for concomitant UI and even stronger for women with UI of longer

duration (>10 years). The prevalence of sPOP in our study was more than twice as common in women after a vaginal delivery compared with caesarean section. In addition the prevalence of sPOP and concomitant UI was tripled after vaginal delivery compared with caesarean section. It should also be noted that the prevalence of UI>10 yr was more than five times higher after vaginal delivery compared with caesarean section in women with sPOP. This indicates a protective effect of caesarean section for the occurrence of sPOP and the protective effect of caesarean section was even greater for sPOP + UI and sPOP +UI> 10 years This may also indicate that sPOP after VD has a different pathogenesis and higher grade compared to sPOP in women after a ceasarean section. Data in the literature are missing for comparison on the association for VD and sPOP and concomitant UI.

Symptomatic pelvic organ prolapse (paper II)

Many conditions predispose to pelvic organ prolapse and its occurrence is thought to be multifactorial and in some cases also individual due to a genetic predisposition. Aging, chronic lung disorders,[160] constipation,[161] and other strenuous activities resulting in increased intra abdominal pressure that puts excessive strain on supporting structures and nerves of the pelvic floor[162] are thought to be of importance. It is still controversial whether pregnancy in itself, distinct from mode of delivery, alters the risk of pelvic organ prolapse.[151]

Vaginal delivery

The reported wide range (15% - 48%) in the prevalence of POP after childbirth is mainly due to differences in study populations and varying classification of POP.[1,72,163] The influence of specific obstetric events is difficult to interpret in the majority of studies due to the inclusion of several pregnancies. Information about the prevalence of sPOP after one single vaginal or surgical delivery is scarce.[164] Both epidemiological and observational cohort studies have shown that the main

risk factor of prolapse is vaginal delivery and that the risk increases for every additional birth.[33,73,165]

We found that the prevalence of late sPOP more than doubled after VD compared to CS (14.6% versus 6.3%). The risk increase after one vaginal delivery was 255% compared to one caesarean section. We did not find any significant difference in the prevalence of sPOP between women delivered by acute caesarean section (6.3%) and elective caesarean section (6.0%). We interpret these results to indicate that it is not until the final stage of delivery when the fetus passes through the pelvic hiatus during vaginal delivery (at the end of the second stage of delivery) that the structural damage to the pelvic floor occurs causing sPOP. This is contrary to two other studies that concluded that caesarean section does not provide a significant risk reduction in the long term for genital prolapse.[70,166]

Body mass

In our study current BMI was an independent but in comparison with mode of delivery a much less important risk factor with a weak effect on the odds increase for prolapse. In the logistic regression analysis the prevalence of sPOP increased by 3% for each unit increase of BMI. Whether current BMI is a risk factor for POP or not is still controversial. A positive association between BMI and the odds of sPOP has been shown in the study by Miedel et al. and in the Women´s Health Initiative.[167,168] Even if the analysis used in these two studies differed from our study, the estimated risk increase by each unit increase of BMI from the stratified data in these two studies was approximately in the same range as in the present study. However it should be noted that two other studies did not establish BMI as a significant risk factor for sPOP and clinical POP respectively.[33,169]

Age

Aging is widely accepted as an independent risk factor for genital prolapse.[166,168] Miedel et al. found a significant age dependent (parity adjusted) increase of sPOP for women of all age groups ≥50 years compared to women aged 30-39 years old after one or more deliveries.[167] Contrary to these findings we did not find age to be a significant risk factor for sPOP, neither in the logistic regression analysis nor in analysis of stratified age groups. Our findings may reflect the homogeneous cohort of women with a narrow age range (35-67 years), a fixed time interval remote from birth (20 years), generally good access to modern antenatal care and obstetric practices in the 1980´s, the more favourable health status among pregnant women in Sweden and the narrow age range of our cohort. Cross-sectional studies have included elderly women who gave birth earlier under perhaps less optimal birth conditions. In support of our results the Oxford FPA Study also concluded that age was a much less important risk factor, once parity and calendar period was taken into account.[73]

Fetal weight

Whether or not infant birthweight is a risk factor for prolapse is still controversial. In the study of Tegerstedt el al. birthweight was not a risk factor for sPOP after adjustment for age and parity, but the groups were probably too small.[72] Our results are however consistent with those of two other studies indicating that birthweight is a risk factor. In the cross-sectional population study of Samuelsson et al a strong correlation was found between odds of POP and maximum birthweight.[169] Timonen et al.[170] reported that POP was found in more than one third of women who delivered a child ≥4000g compared with 9.5% in the general population. Similarly the analysis of prolapse prevalence in relation to stratified infant birth weights in the present study showed that sPOP more than doubled for infants ≥4500g in comparison with infants <3000g after vaginal delivery. In contrast this was not observed after caesarean section. The logistic regression analysis confirmed a 3%

risk increase in the prevalence of sPOP for each 100g increase of birth weight. However, from the analysis of stratified infant birth weight the relationship did not seem to be linear and there seemed to be a threshold effect for a birth weight around 4500 g. This was not observed after CS. The logistic regression analysis confirmed a 3% odds increase in the prevalence of sPOP for each 100g increase of birth weight.

Disproportion mother vs. child

We also found a strong association for the prevalence of sPOP among shorter mothers (≤160 cm) who delivered an infant ≥4000g compared with an infant <4000g after vaginal delivery. This effect was not observed for mothers >160 cm or in the caesarean section group. This consequence of disproportion between mother and child may be the result of a prolonged second stage of delivery and therefore act as a surrogate measure. Data in the literature on the effect of incongruity between mother and child as a risk factor for sPOP are lacking for comparison.

Vacuum extraction, episiotomy, and perineal tear

The effect of specific obstetric risk factors for the development of prolapse remains controversial. In this study we found none of the obstetric events – vacuum extraction, episiotomy, or ≥ 2nd degree laceration – to be associated with an increased prevalence or risk of sPOP.

Risk for subsequent long-term development of POP that could be attributed to specific obstetric events during vaginal delivery is poorly analysed in the literature. Tegerstedt et al found that episiotomy or instrumental delivery (proportions of VE and forceps were not specified) was neither positively nor negatively associated with sPOP, which is consistent with the results of our study.[72] Handa et al. found a strong association between operative vaginal delivery and POP despite a low

prevalence rate (3%) of clinical POP in that study[164] Tegerstedt et al. also found that vaginal delivery in combination with "extensive vaginal rupture" more than doubled the odds of sPOP (in connection with at least one delivery).[72] The diagnosis however was based on womens recall. This is contrary to our finding that perineal tear $\geq$ 2nd degrees, after adjustment for infant birthweight, maternal age, current BMI and infant head circumference was not associated with sPOP.

Feacal and anal incontinence

Bowel continence is an extremely complex body function. Its main components are the quality of colonic content, the integrity of the nervous and humoral control of intestinal motility and the endo- and exogenous secretory mechanisms of the gastrointestinal tract, the sensory function of the rectum and the anal canal, and finally the functional status of the pelvic floor muscles.[38] The effect of pregnancy and vaginal birth on continence function for bowel content does however affect mainly the last component mentioned. To date, much research has been focused on sphincter injuries to explain incontinence in parous women based on the implicit assumption that these injuries and their putative risk factors alone explain bowel incontinence after childbirth. This hypothesis has been challenged in recent studies that have shown that the vast majority of community-dwelling women with feacal incontinence report that symptoms developed after 40 years of age[84] and that the main risk factors were not related to childbirth but instead to diarrhea, IBS, smoking, cholecystectomy and obesity.[85] Rather, these observations indicate that the 'multiple-hit hypothesis' is more relevant. According to this, obstetric anal sphincter injury (the initial 'hit') is compounded by other factors (e.g. pelvic muscle atrophy, pudendal nerve dysfunction, aging, etc.) manifesting itself as incontinence later in life.[84]

Vaginal delivery

Two reviews assessing whether caesarean section is protective or not against bowel incontinence both concluded that it is not protective.[82,171] However, according to the Cochrane review the primary studies were methodologically poor with insufficient statistical power, employed different assessment tools and definitions and had too short a follow-up time after delivery.[82]

We found that the prevalence of both AI and FI was higher after VD compared to CS. The difference between vaginal delivery and caesarean section was consistent for any incontinence (any = isolated + combinations) and for each modality of leakage (gas, solid and liquid). The difference between vaginal delivery and caesarean section was further confirmed by results using the Wexner Continence Grading Scale. Scores ≥9 have been shown to indicate a significant impairment of quality of life.[119] The odds increase for severe incontinence (score ≥9) was almost doubled after vaginal delivery compared to caesarean section. Only one other study has used the Wexner score to compare the outcome after caesarean section to that after vaginal delivery in the short-term conforming to our results.[172]

Sphincter tear

Incontinence is common during the first months postpartum,[39,40,81] but a majority of women with early problems will recover.[82] There is also a poor correlation between the extent of the sphincter injury and the severity of clinical symptoms,[102] and many patients with occult injuries at ultrasound report no impairment of continence function.[103]

Most probably there is an under-reporting of obstetric sphincter ruptures in our cohort of vaginally delivered women with ≥2nd degree tears, since recent studies have shown that 87% of midwives and 27% of junior doctors failed to recognize

3rd- 4th degree tears clinically[173] and that the rate of 3rd degree tears increased to 15% when all '2nd degree tears' were re-assessed by a second experienced person.[174] It has even been suggested that sphincter defects previously designated as 'occult' are unrecognized clinical injuries and that genuine occult sphincter damages may be less than 1% – if they exist at all.[106]

Our study showed that a ≥2nd degree perineal tear was associated with an almost doubled prevalence of FI compared to women without ≥2 nd degree tears. Calculation of the difference in odds ratio for FI between surgically and vaginally delivered women including or excluding a perineal tear ≥2 nd degree from the vaginal cohort resulted in a 5% decrease in odds of FI, from 44% to 39%, which meant that tears were responsible for only 11% of the total risk increase after vaginal delivery.

The association between perineal tears and subsequent late incontinence is complex and largely unknown.[175] In this study ≥2nd degree tears were associated with an 84% odds increase of FI. This is the first time that perineal tears have shown to be associated with long-term incontinence. It may be that lacerations ≥ 2nd degree are a marker for other multiple occult tissue injuries, the effects of which manifest later in life. But even if a perineal tear is a bad omen for future incontinence, the dominant factor leading to FI, is not tears or even sphincter injuries, but the trauma from the vaginal birth itself. This conclusion is to some extent supported by an electrophysiological study showing that neuronal damage to the pelvic floor may result from a normal vaginal delivery.[24] This finding of an association between ≥2nd degree tears and subsequent late incontinence is of particular clinical importance as the incidence of ≥2nd degree tears in Sweden has increased in primiparae from 3.1% in 1987 to a peak of 16.9% in 2003. The etiology of this sharp increase is still controversial. However, considering an almost doubled prevalence of late FI in association with ≥2nd degree tears shown in this study, such a trend is very

distressing. It has been suggested that changes in the conduct of labor during the last three decades may explain this trend.[108,176]

Episiotomy

Only four cohort studies have reported data on the association between episiotomy and self-reported symptoms of incontinence of stool and/or flatus close after birth (3-10 months).[177] None of these found episiotomy to be associated with reduced risk of incontinence. In our study, episiotomies were protective for FI resulting in a risk reduction of 27% and a prevalence of 11.1% for FI, which, interestingly, is close to 10.6% after caesarean section and lower then 14.7% after vaginal delivery without episiotomy. Assessment of continence at a time remote from childbirth in relation to episiotomy has not previously been presented. The rate of episiotomy in this study was 12.8% (n = 510), which by international standards is a low prevalence, but similar to the national episiotomy rate (16%) in Sweden for 1985 (MBR Sweden). This protective effect of episiotomy is important since episiotomy rates have been steadily decreasing in Sweden from 21% in 1975 to 7% in 2005 (MBR data). During the same time period the rate of perineal tears has increased dramatically because of a change in policy favoring spontaneous tears to avoid pain, dyspareunia and scars from episiotomy during the months following birth.[104] Studies from Finland have shown that a more restrictive use of medio-lateral episiotomy is associated with a higher incidence of sphincter injuries.[108,178] A more generous use of episiotomy, to lower the rate of spontaneous perineal tears in combination with the classic active monitoring to protect the perineum during the final stage of delivery, as proposed by Laine et al., should perhaps be reconsidered also for the prevention of late FI.[108]

Body mass and age

Age is widely considered to be a risk factor for FI.[78] This was confirmed in the present study, which demonstrated an annual increase of FI by 3% yearly. However, the association between FI and age is probably not linear, since FI usually has a late onset, with an incidence peak after the menopause at about 55 years of age,[84,85] which is close to the mean age of 51 years in this study.

Several studies have shown an association between BMI and incontinence, consistent with the findings in this study.[86-87] Weight reduction has also been shown to result in improvement of FI.[88] In our study the odds for each 5-unit BMI increase was 15% for AI and 30% for FI.

Conclusions

- Two decades after one childbirth, VD was associated with a 67% increased odds of UI, and UI >10 years increased by 275% compared to CS. Current BMI was the most important BMI-determinant for UI, which is important, since BMI is modifiable.

- The single most important risk factor for sPOP was delivery via the vaginal route. Birthweight above 4500g was a risk factor for sPOP after VD. sPOP was also an important risk factor for UI and UI >10 years.

- Vaginal delivery was associated with a higher prevalence of SUI, UUI, and MUI. Moderate to severe UI and all subtypes of UI occurred more often after VD compared to CS. After VD bothersome UI and bothersome MUI occurred more often compared to after CS. sPOP and UI >10 years were major risk factors for bothersome urinary incontinence.

- The prevalence of FI and AI was higher after VD compared with CS. Perineal tear >2nd degrees almost doubled the prevalence of FI. Episiotomy was protective against late faecal incontinence.

- The prevalence of UI, sPOP, and FI/AI did not differ between women delivered by acute compared to elective caesarean section, indicating that it is not until the fetus passes through the pelvic hiatus that the injuries occur that causes these pelvic floor disorders.

Swedish summary – Sammanfattning på svenska

Bakgrund: Bäckenbottendysfunktion i form av urin- och avföringsläckage och urogenitalt framfall är ett stort folkhälsoproblem som drabbar kvinnor över hela världen. I västvärlden är för närvarande risken för kvinnor att före 80 års ålder opereras med ett rekonstruktivt ingrepp p.g.a. framfall och urinläckage över 11%. Graviditet och förlossning är de viktigaste orsaksfaktorerna, men även åldrande och övervikt anses spela en viktig roll för uppkomsten. De subjektiva besvär som dessa dysfunktionella tillstånd ger upphov till är en känsla eller obehag av att något buktar ut ur slidan, skav och sårbildning i underlivet, urinträngningar, urinläckage och tömningssvårigheter, oförmåga/svårighet att hålla tätt för avföring och gas samt sexuell dysfunktion.
Andelen kvinnor som begär kejsarsnitt av icke-medicinska skäl har ökat under de senaste decennierna. Önskan att undvika det vaginala traumat i samband med en förlossning har angetts som en vanlig anledning. Om kejsarsnitt i det längre perspektivet verkligen skyddar bäckenbotten har emellertid ifrågasatts. Andra icke-obstetriska orsaker som övervikt och åldrande har av många forskare ansetts utjämna effekten av eventuella förlossningsskador.

Syftet med denna avhandling var att undersöka skillnaden i förekomsten av urin- och avföringsläckage och genital prolaps efter en enda vaginal förlossning kontra ett enda kejsarsnitt 20 år senare. Syftet var också att undersöka möjliga obstetriska riskfaktorer (fostervikt, klipp, vaginala bristningar, sugklocka etc.) och icke-obstetriska riskfaktorer (ålder, övervikt, östrogenbehandling etc.) för utveckling av senare bäckenbottendysfunktion.

Metod: Studien var en nationell kohortstudie som inkluderade 5236 kvinnor vilka fött ett barn mellan 1985-1988 och därefter inget ytterligare barn. Självrapporterade besvär från bäckenbotten inhämtades via en brevenkät 2008. Frågeformuläret

innehöll 31 validerade frågor för att fastställa förekomsten av urin- och avföringsläckage samt genital prolaps. Dessa uppgifter kopplades till obstetriska data hämtade från det svenska medicinska födelsregistret (MFR). Uppgifter om den gravida kvinnans vikt, längd, viktuppgång under graviditeten, fostervikt samt uppgifter om förlossningsförloppet finns dokumenterade i MFR. Uppgifter om kvinnans aktuella vikt, menstruationsstatus, östrogenintag, borttagande av livmoder, etc. inhämtades via enkäten.

Resultat: Förekomsten av urininkontinens och urininkontinens som varat mer än 10 år, subtyper av urinläckage (ansträngningsläckage, trängningsläckage och blandinkontinens), högre svårighetsgrad av urininkontinens och graden av subjektiva besvär som den ger upphov till, symtomatisk genital prolapse, förekomsten av anal och fekal inkontinens och svår anal inkontinens förekom oftare efter en vaginal förlossning jämfört med efter ett kejsarsnitt. Efter en vaginal förlossning ökade förekomsten av fekal inkontinens med ca 4%, urinläckage med ca 12% och symtomgivande prolaps med ca 8% jämfört med ett kejsarsnitt. Vaginalförlösta kvinnor med prolaps hade en nästan tre gånger så hög förekomst av urinläckage jämfört med kejsarsnittade kvinnor med prolaps. Förekomsten av avföringsläckage nästan fördubblades efter en vaginal förlossning som medfört en ≥2:a gradens perineal bristning. Klipp (episiotomi) reducerade risken för avföringsinkontinens. Den aktuella vikten (2008) var, näst efter att ha blivit förlöst vaginalt, den högst rankade riskfaktorn för förekomsten av bäckenbottenstörningar. Detta är särskilt betydelsefullt eftersom vikten kan påverkas.

Konklusion: 20 år efter en vaginal förlossning var förekomsten av de tre vanligaste förekommande bäckenbottenstörningarna hos kvinnor - urinläckage, avförings-läckage och genital prolaps - högre jämfört med efter ett kejsarsnitt.

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References

1. Milsom I, Altman D, Lapitan MC, et al. Epidemiology of urinary (UI) and faecal (FI) incontinence and pelvic organ prolapse (POP). In: Abrams P, Cardozo L, Khoury S, eds. Incontinence: 4th International Consultation on Incontinence. Plymouth, United Kingdom: Health Publications Ltd;2009:37-111.

2. United Nations Children's Fund. Total fertility rate (TFR). 2008 [updated 07/201003/02/2011]; Available from: http://data.un.org/Data.aspx?d=SOWC&f=inID%3A127.

3. http://www.prb.org/pdf11/2011population-data-sheet_eng.pdf (updated 07/20)

4. Al-Mufti R, McCarthy A, Fisk NM. Survey of obstetricians' personal preference and discretionary practice. Eur J Obstet Gynecol Reprod Biol. 1997;73:1-4.

5. Wax JR, Cartin A, Pinette MG, Blackstone J. Patient choice cesarean--the Maine experience. Birth. 2005;32:203-6.

6. National Institute of Health State of the Science conference statement. Cesarean delivery on maternal request. March 27-29, 2006. http://consensus.nih.gov

7. Oelrich TM. The striated urogenital sphincter muscle in the female. Anat Rec, 1983;205: 223-32.

8. DeLancey, J.O. Functional anatomy of the female lower urinary tract and pelvic floor. Ciba Found Symp, 1990:151;57-69; discussion 69-76.

9. Parks AG, Porter NH, Melzack J. Experimental study of the reflex mechanism controlling the muscles of the pelvic floor. Dis Colon Rectum 1962;5:407-14.

10. Melzack J, Porter NH. Studies of the reflex activity of the external sphincter ani in spinal man. Paraplegia 1964;1:277-296.

11. Ashton-Miller JA, Delancey JO. On the biomechanics of vaginal birth and common sequelae. Annu Rev Biomed Eng. 2009;11:163-76.

12. Ruskin AP, Davis JE. Anal sphincter electromyography. Electroencephalogr Clin Neurophysiol. 1969;27:713.

13. Shuster MM. Faecal incontinence. Treatment: Biofeedback. In: Coloproctology and the Pelvic Floor. Pathophyiology and management. Eds. Henry MM, Swash M. Butterworths, London 1985:268-281.

14. Monges H, Saducci J, Nandy B, et al. The electrical activity of the internal anal sphincter: a comparative study in man and cat. In. Gastrointestinal Motility. Ed. Christiansen J. New York, Raven Press 1980;495-501.

15. Percy JP, Parks AG. The nerve supply of the pelvic floor. Schweiz Rundschau Med 1981; 70:640-642.

16. Kearney R, Miller JM, Ashton-Miller JA, DeLancey JO. Obstetric factors associated with levator ani muscle injury after vaginal birth. Obstet Gynecol. 2006;107:144-9.

17. Kearney R, Fitzpatrick M, Brennan S, Behan M, Miller J, Keane D, et al. Levator ani injury in primiparous women with forceps delivery for fetal distress, forceps for second stage arrest, and spontaneous delivery. Int J Gynaecol Obstet. 2010;111:19-22.

18. Chan SS, Cheung RY, Yiu AK, Lee LL, Pang AW, Choy KW, et al. Prevalence of levator ani muscle injury in Chinese women after first delivery. Ultrasound Obstet Gynecol. 2012;39:704-9.

19. Lien KC, Morgan DM, Delancey JO, Ashton-Miller JA. Pudendal nerve stretch during vaginal birth: a 3D computer simulation. Am J Obstet Gynecol. 2005;192:1669-76.

20. Dietz HP, Lanzarone V. Levator trauma after vaginal delivery. Obstet Gynecol. 2005;106: 707-12.

21. Singh K, Jakab M, Reid WM, Berger LA, Hoyte L. Three-dimensional magnetic resonance imaging assessment of levator ani morphologic features in different grades of prolapse. Am J Obstet Gynecol. 2003;188:910-15.

22. Jou IM, Lai KA, Shen CL, Yamano Y. Changes in conduction, blood flow, histology, and neurological status following acute nerve-stretch injury induced by femoral lengthening. J Orthop Res. 2000;18:149-55.

23. Parks AG, Swash M, Urich H. Sphincter denervation in anorectal incontinence and rectal prolapse. Gut. 1977;18:656-65.

24. Snooks SJ, Setchell M, Swash M, Henry MM. Injury to innervation of pelvic floor sphincter musculature in childbirth. Lancet. 1984;2:546-50.

25. Snooks SJ, Swash M, Henry MM, Setchell M. Risk factors in childbirth causing damage to the pelvic floor innervation. Int J Colorectal Dis. 1986;1:20-4.

26. Snooks SJ, Swash M, Mathers SE, Henry MM. Effect of vaginal delivery on the pelvic floor: a 5-year follow-up. Br J Surg. 1990;77:1358-60.

27. DeLancey JO. Structural support of the urethra as it relates to stress urinary incontinence: the hammock hypothesis. Am J Obstet Gynecol. 1994;170:1713-20; discussion 20-3.

28. van Geelen JM. The urethral sphincter mechanism: where are we now? Int Urogynecol J Pelvic Floor Dysfunct. 2001;12:149-50.

29. Koelbl H, Nitti V, Baessler K, et al. Pathophysiology of Urinary Incontinence, Faecal Incontinence and Pelvic Organ Prolapse. In: Abrams P, Cardozo L, Khoury S, eds. Incontinence: 4th International Consultation on Incontinence. Plymouth, United Kingdom: Health Publications Ltd; 2009:37-111.

30. van Geelen JM, Lemmens WA, Eskes TK, Martin CB, Jr. The urethral pressure profile in pregnancy and after delivery in healthy nulliparous women. Am J Obstet Gynecol 1982;144:636-49.

31. Meyer S, Bachelard O, De Grandi P. Do bladder neck mobility and urethral sphincter function differ during pregnancy compared with during the non-pregnant state? Int Urogynecol J Pelvic Floor Dysfunct. 1998;9:397-404.

32. King JK, Freeman RM. Is antenatal bladder neck mobility a risk factor for postpartum stress incontinence? Br J Obstet Gynaecol. 1998;105:1300-7.

33. Rortveit G, Brown JS, Thom DH, Van Den Eeden SK, Creasman JM, Subak LL. Symptomatic pelvic organ prolapse: prevalence and risk factors in a population-based, racially diverse cohort. Obstet Gynecol. 2007;109:1396-403.

34. Miodrag A, Castleden CM, Vallance TR. Sex hormones and the female urinary tract. Drugs. 1988;36:491-504.

35. Wahl LM, Blandau RJ, Page RC. Effect of hormones on collagen metabolism and collagenase activity in the pubic symphysis ligament of the guinea pig. Endocrinology. 1977;100:571-9.

36. Norton PA, Baker JE, Sharp HC, Warenski JC. Genitourinary prolapse and joint hypermobility in women. Obstet Gynecol. 1995;85:225-8.

37. Carley ME, Schaffer J. Urinary incontinence and pelvic organ prolapse in women with Marfan or Ehlers Danlos syndrome. Am J Obstet Gynecol. 2000;182:1021-3.

38. Bharucha AE. incontinence. Gastroenterology. 2003;124:1672-85.

39. Sultan AH, Kamm MA, Hudson CN, Thomas JM, Bartram CI. Anal-sphincter disruption during vaginal delivery. N Engl J Med 1993;329:1905-11.

40. Kamm MA. Obstetric damage and faecal incontinence. Lancet 1994;344:730-3.

41. Allen RE, Hosker GL, Smith AR, Warrell DW. Pelvic floor damage and childbirth: a neurophysiological study. Br J Obstet Gynaecol 1990;97:770-9.

42. Fynes M, Donnelly VS, O'Connell PR, O'Herlihy C. Cesarean delivery and anal sphincter injury. Obstet Gynecol. 1998;92:496-500.

43. Alnaif B, Drutz HP. The prevalence of urinary and fecal incontinence in Canadian secondary school teenage girls: questionnaire study and review of the literature. Int Urogynecol J Pelvic Floor Dysfunct. 2001;12:134-7; discussion 8.

44. Eason E, Labrecque M, Marcoux S, Mondor M. Effects of carrying a pregnancy and of method of delivery on urinary incontinence: a prospective cohort study. BMC Pregnancy Childbirth. 2004;4:4.

45. Brown SJ, Donath S, MacArthur C, McDonald EA, Krastev AH. Urinary incontinence in nulliparous women before and during pregnancy: prevalence, incidence, and associated risk factors. Int Urogynecol J Pelvic Floor Dysfunct. 2010;21:193-202.

46. Rortveit G, Daltveit AK, Hannestad YS, Hunskaar S. Urinary incontinence after vaginal delivery or cesarean section. N Engl J Med. 2003;348:900-7.

47. Peyrat L, Haillot O, Bruyere F, Boutin JM, Bertrand P, Lanson Y. Prevalence and risk factors of urinary incontinence in young and middle-aged women. BJU Int. 2002;89:61-6.

48. Hvidman L, Foldspang A, Mommsen S, Nielsen JB. Postpartum urinary incontinence. Acta Obstet Gynecol Scand. 2003;82:556-63.

49. Fritel X, Fauconnier A, Levet C, Benifla JL. Stress urinary incontinence 4 years after the first delivery: a retrospective cohort survey. Acta Obstet Gynecol Scand. 2004;83:941-5.

50. Foldspang A, Hvidman L, Mommsen S, Nielsen JB. Risk of postpartum urinary incontinence associated with pregnancy and mode of delivery. Acta Obstet Gynecol Scand. 2004;83:923-7.

51. Schytt E, Lindmark G, Waldenstrom U. Symptoms of stress incontinence 1 year after childbirth: prevalence and predictors in a national Swedish sample. Acta Obstet Gynecol Scand. 2004;83:928-36.

52. Van Brummen HJ, Bruinse HW, Van de Pol G, Heintz AP, Van der Vaart CH. What is the effect of overactive bladder symptoms on woman's quality of life during and after first pregnancy? BJU Int. 2006;97:296-300.

53. Van Brummen HJ, Bruinse HW, van de Pol G, Heintz AP, van der Vaart CH. The effect of vaginal and cesarean delivery on lower urinary tract symptoms: what makes the difference? Int Urogynecol J Pelvic Floor Dysfunct. 2007;18:133-9.

54. Dimpfl T, Hesse U, Schussler B. Incidence and cause of postpartum urinary stress incontinence. Eur J Obstet Gynecol Reprod Biol. 1992;43:29-33.

55. Burgio KL, Zyczynski H, Locher JL, Richter HE, Redden DT, Wright KC. Urinary incontinence in the 12-month postpartum period. Obstet Gynecol. 2003;102:1291-8.

56. Morkved S, Bo K. Prevalence of urinary incontinence during pregnancy and postpartum. Int Urogynecol J Pelvic Floor Dysfunct. 1999;10:394-8.

57. Thom DH, Rortveit G. Prevalence of postpartum urinary incontinence: a systematic review. Acta Obstet Gynecol Scand. 2010;89:1511-22.

58. Casey BM, Schaffer JI, Bloom SL, Heartwell SF, McIntire DD, Leveno KJ. Obstetric antecedents for postpartum pelvic floor dysfunction. Am J Obstet Gynecol. 2005;192:1655-62.

59. Viktrup L, Lose G, Rolff M, Barfoed K. The symptom of stress incontinence caused by pregnancy or delivery in primiparas. Obstet Gynecol. 1992;79:945-9.

60. MacArthur C, Glazener CM, Wilson PD, Lancashire RJ, Herbison GP, Grant AM. Persistent urinary incontinence and delivery mode history: a six-year longitudinal study. BJOG. 2006;113:218-24.

61. Milsom I, Ekelund P, Molander U, Arvidsson L, Areskoug B. The influence of age, parity, oral contraception, hysterectomy and menopause on the prevalence of urinary incontinence in women. J Urol. 1993;149:1459-62.

62. Waetjen LE, Liao S, Johnson WO, et al. Factors associated with prevalent and incident urinary incontinence in a cohort of midlife women: a longitudinal analysis of data: study of women's health across the nation. Am J Epidemiol 2007;165:309-18.

63. Grodstein F, Fretts R, Lifford K, Resnick N, Curhan G. Association of age, race, and obstetric history with urinary symptoms among women in the Nurses' Health Study. Am J Obstet Gynecol. 2003;189:428-34.

64. Danforth KN, Townsend MK, Lifford K, Curhan GC, Resnick NM, Grodstein F. Risk factors for urinary incontinence among middle-aged women. Am J Obstet Gynecol. 2006;194:339-45.

65. Rortveit G, Hannestad YS, Daltveit AK, Hunskaar S. Age- and type-dependent effects of parity on urinary incontinence: the Norwegian EPINCONT study. Obstet Gynecol. 2001;98:1004-10.

66. Press JZ, Klein MC, Kaczorowski J, Liston RM, von Dadelszen P. Does cesarean section reduce postpartum urinary incontinence? A systematic review. Birth. 2007;34:228-37.

67. Tennstedt SL, Link CL, Steers WD, McKinlay JB. Prevalence of and risk factors for urine leakage in a racially and ethnically diverse population of adults: the Boston Area Community Health (BACH) Survey. Am J Epidemiol. 2008;167:390-9.

68. Minassian VA, Drutz HP, Al-Badr A. Urinary incontinence as a worldwide problem. Int J Gynaecol Obstet. 2003 ;82:327-38.

69. Fornell EU, Wingren G, Kjoelhede P. Factors associated with pelvic floor dysfunction with emphasis on urinary and fecal incontinence and genital prolapse: an epidemiological study. Act. Obstet. Gynecol. Scand. 2004;83:383-389.

70. MacLennan AH, Taylor AW, Wilson DH, Wilson D. The prevalence of pelvic floor disorders and their relationship to gender, age, parity and mode of delivery. BJOG. 2000;07:1460-70.

71. Tegerstedt G, Maehle-Schmidt M, Nyren O, Hammarstrom M. Prevalence of symptomatic pelvic organ prolapse in a Swedish population. Int Urogynecol J Pelvic Floor Dysfunct 2005;16:497-503.

72. Tegerstedt G, Miedel A, Maehle-Schmidt M, Nyren O, Hammarstrom M. Obstetric risk factors for symptomatic prolapse: a population-based approach. Am J Obstet Gynecol 2006;194:75-81.

73. Mant J, Painter R, Vessey M. Epidemiology of genital prolapse: observations from the Oxford Family Planning Association Study. British Journal of Obstetrics & Gynaecology. 1997;104:579-85.

74. Greer WJ, Richter HE, Bartolucci AA, Burgio KL. Obesity and pelvic floor disorders: a systematic review. Obstet Gynecol. 2008;112:341-9.

75. Sharma A, Marshjall RJ, MacMillan AK, Merrie AEH, Reid P, Bissett IP. Determining levels of fecal incontinence in the community: a New Zealand cross-sectional study. Dis. Colon Rectum. 2011;54:1381-7.

76. Nelson RL, Norton N, Cautley E, Furner S. Community based prevalence of AI. JAMA. 1995;274:559-562.

77. Whitehead WE, Norton NJ, Wald A. Advancing the treatment of faecal and urinary incontinence through research. Gastroenterology 2004;126:S1-2.

78. Pretlove SJ, Radley S, Toozs-Hobson PM, Thompson PJ, Coomarasamy A, Khan KS. Prevalence of anal incontinence according to age and gender: a systematic review and meta-regression analysis. Int Urogynecol J Pelvic Floor Dysfunct. 2006;17:407-17.

79. Solans-Domenech M, Sanchez E, Espuna-Pons M. Urinary and anal incontinence during pregnancy and postpartum: incidence, severity, and risk factors. Obstet Gynecol. 2010;115:618-28.

80. Elenskaia K, Thakar R, Sultan AH, Scheer I, Onwude J. Effect of childbirth on pelvic organ support and quality of life: a longitudinal cohort study. Int Urogynecol J. 2012 Sep 7. DOI 10.1007/s00192-012-1932-7.

81. Pollack J, Nordenstam J, Brismar S, Lopez A, Altman D, Zetterstrom J. Anal incontinence after vaginal delivery: a five-year prospective cohort study. Obstet Gynecol 2004;104:1397-402.

82. Nelson RL, Furner SE, Westercamp M, Farquhar C. Caesaren delivery for the prevention of anal incontinence. Cochrane Database Syst Rev 2010; 2: CD006756.

83. MacArthur C, Glazener C, Lancashire R, Herbison P, Wilson D. Exclusive caesarean section delivery and subsequent urinary and faecal incontinence: a 12-year longitudinal study. BJOG. 2011;118:1001-7.

84. Bharucha AE, Zinsmeister AR, Locke GR, Seide BM, McKeon K, Schleck CD, et al. Prevalence and burden of faecal incontinence: a population-based study in women. Gastroenterology 2005;129:42-9.

85. Bharucha AE, Zinsmeister AR, Schleck CD, Melton LJ, 3rd. Bowel disturbances are the most important risk factors for late onset faecal incontinence: a population-based case-control study in women. Gastroenterology 2010;139:1559-66.

86. Varma MG, Brown JS, Creasman JM, Thom DH, Van Den Eeden SK, Beattie MS, et al. Fecal incontinence in females older than aged 40 years: who is at risk? Dis Colon Rectum. 2006;49:841-51.

87. Erekson EA, Sung VW, Myers DL. Effect of body mass index on the risk of anal incontinence and defecatory dysfunction in women. Am J Obstet Gynecol. 2008;198:596 e1-4.

88. Cuicchi D, Lombardi R, Cariani S, Leuratti L, Lecce F, Cola B. Clinical and instrumental evaluation of pelvic floor disorders before and after bariatric surgery in obese women. Surg Obes Relat Dis. 2011 Aug 28. DOI 10.1016/j.soard.2011.08.013.

89. Mous M, Muller SA, de Leeuw JW. Long-term effects of anal sphincter rupture during vaginal delivery: faecal incontinence and sexual complaints. BJOG. 2008;115:234-8.

90. Nygaard IE, Rao SS, Dawson JD. Anal incontinence after anal sphincter disruption: a 30-year retrospective cohort study. Obstet Gynecol. 1997;89:896-901.

91. Faltin DL, Otero M, Petignat P, Sangalli MR, Floris LA, Boulvain M, et al. Women's health 18 years after rupture of the anal sphincter during childbirth: I. Fecal incontinence. Am J Obstet Gynecol. 2006;194:1255-9.

92. Burnett SJ, Spence-Jones C, Speakman CT, Kamm MA, Hudson CN, Bartram CI. Unsuspected sphincter damage following childbirth revealed by anal endosonography. Br J Radiol. 1991;64:225-7.

93. Karoui S, Savoye-Collet C, Koning E, Leroi AM, Denis P. Prevalence of anal sphincter defects revealed by sonography in 335 incontinent patients and 115 continent patients. AJR Am J Roentgenol. 1999;173:389-92.

94. Eason E, Labrecque M, Marcoux S, Mondor M. AI after childbirth. CMAJ. 2002;166:326-330.

95. Chaliha C, Kalia V, Stanton SL, Monga A, Sultan AH. Antenatal prediction of postpartum urinary and fecal incontinence. Obstet Gynecol. 1999;94:689-694.

96. Zetterstrom JP, Lopez A, Anzen B, Dolk A, Norman M, Mellgren A. Anal incontinence after vaginal delivery: a prospective study in primiparous women. Br J Obstet Gynaecol. 199;106:324-30.

97. Groutz A, Fait G, Lessing JB, David MP, Wolman I, Jaffa A, et al. Incidence and obstetric risk factors of postpartum anal incontinence. Scand J Gastroenterol. 1999;34:315-8.

98. Hall W, McCracken K, Osterweil P, Guise JM. Frequency and predictors for postpartum fecal incontinence. Am J Obstet Gynecol. 2003;188:1205-7.

99. Nazir M, Stien R, Carlsen E, Jacobsen AF, Nesheim BI. Early evaluation of bowel symptoms after primary repair of obstetric perineal rupture is misleading: an observational cohort study. Dis Colon Rectum. 2003;46:1245-50.

100. Starck M, Bohe M, Valentin L. The extent of endosonographic anal sphincter defects after primary repair of obstetric sphincter tears increases over time and is related to anal incontinence. Ultrasound Obstet Gynecol. 2006;27:188-97.

101. Nichols CM, Ramakrishnan V, Gill EJ, Hurt WG. Anal incontinence in women with and those without pelvic floor disorders. Obstet Gynecol. 2005;106:1266-71.

102. Voyvodic F, Rieger NA, Skinner S, Schloithe AC, Saccone GT, Sage MR, et al. Endosonographic imaging of anal sphincter injury: does the size of the tear correlate with the degree of dysfunction? Dis Colon Rectum 2003;46:735-41.

103. Damon H, Henry L, Bretones S, Mellier G, Minaire Y, Mion F. Postdelivery anal function in primiparous females: ultrasound and manometric study. Dis Colon Rectum 2000;43:4727.

104. Hartmann K, Viswanathan M, Palmieri R, Gartlehner G, Thorp J Jr, Lohr KN. Outcomes of routine episiotomy: a systematic review. JAMA 2005; 293:2141-48.

105. Shiono P, Klebanoff MA, Carey JC. Midline episiotomies: more harm than good? Obstet Gynecol. 1990;75:765-70.

106. Andrews V, Sultan AH, Thakar R, Jones PW. Risk factors for obstetric anal sphincter injury: a prospective study. Birth. 2006;33:117-22.

107. Poen AC, Felt-Bersma RJ, Dekker GA, Deville W, Cuesta MA, Meuwissen SG. Third degree obstetric perineal tears: risk factors and the preventive role of mediolateral episiotomy. Br J Obstet Gynaecol. 1997;104:563-6.

108. Laine K, Gissler M, Pirhonen J. Changing incidence of anal sphincter tears in four Nordic countries through the last decades. Eur J Obstet Gynecol Reprod Biol. 2009;146:71-5.

109. Olsen AL, Smith VJ, Bergstrom JO, Colling JC, Clark AL. Epidemiology of surgically managed pelvic organ prolapse and urinary incontinence. Obstet Gynecol 1997;89:501-6.

110. Lawrence JM, Lukasz ES, Nager CW, Hsu JWY, Luber KM. Prevalence and co-occurence of pelvic floor disorders in community-dwelling women. Obstet Gynecol. 2008;111:678-85.

111. World Health Organization. BMI classification. 2006 [updated 02/02/2011]; Available from: http://apps.who.int/bmi/index.jsp?introPage=intro_3.html.

112. World Health Organization. International classification of deseases, 9th revision, clinical modification (ICD-9-CM). Geneva, Switzerland 1996.

113. Abrams P, Cardozo L, Fall M, Griffiths D, Rosier P, Ulmsten U, et al. The standardisation of terminology in lower urinary tract function: report from the standardisation sub-committee of the International Continence Society. Urology 2003;61:37-49.

114. Sandvik H, Hunskaar S, Seim A, Hermstad R, Vanvik A, Bratt H. Validation of a severity index in female urinary incontinence and its implementation in an epidemiological survey. J Epidemiol Community Health. 1993;47:497-9.

115. Sandvik H, Seim A, Vanvik A, Hunskaar S. A severity index for epidemiological surveys of female urinary incontinence: comparison with 48-hour pad-weighing tests. Neurourol Urodyn. 2000;19:137-45.

116. Hannestad YS, Rortveit G, Sandvik H, Hunskaar S. A community-based epidemiological survey of female urinary incontinence: the Norwegian EPINCONT study. Epidemiology of Incontinence in the County of Nord-Trondelag. J Clin Epidemiol. 2000;53:1150-7.

117. Tegerstedt G, Miedel A, Maehle-Schmidt M, Nyren O, Hammarstrom M. A short-form questionnaire identified genital organ prolapse. J Clin Epidemiol. 2005;58:41-6.

118. Jorge JMN, Wexner SD. Etiology and management of fecal incontinence. Dis Colon Rectum 1993;36:77-97.

119. Rothbarth J, Bemelman WA, Meijerink WJHJ, et al. What is the impact of faecal incontinence on quality of life? Dis Colon Rectum 2001;44:67-71

120. Norton C, Christiansen J, Butler U, et al. Anal Incontinence. In: Abrams P, Cardozo L, Khoury S, eds. Incontinence: 4th International Consultation on Incontinence. Plymouth, United Kingdom: Health Publications Ltd; 2001

121. Uebersax JS, Wyman JF, Shumaker SA, McClish DK, Fantl JA. Short forms to assess life quality and symptom distress for urinary incontinence in women: the Incontinence Impact Questionnaire and the Urogenital Distress Inventory. Neurourol Urodyn. 1995;14:131-9.

122. Thom DH, van den Eeden SK, Ragins AI, Wassel-Fyr C, Vittinghof E, Subak LL, et al. Differences in prevalence of urinary incontinence by race/ethnicity. J Urol. 2006;175:259-64.

123. Diokno AC, Brown MB, Brock BM, Herzog AR, Normolle DP. Clinical and cystometric characteristics of continent and incontinent noninstitutionalized elderly. J Urol. 1988;140:567-71.

124. Herzog AR, Fultz NH. Prevalence and incidence of urinary incontinence in community-dwelling populations. J Am Geriatr Soc. 1990;38:273-81.

125. Dallosso HM, Matthews RJ, McGrother CW, Clarke M, Perry SI, Shaw C, et al. An investigation into nonresponse bias in a postal survey on urinary symptoms. BJU Int. [Research Support, Non-U.S. Gov't]. 2003;91:631-6.

126. Grimes DA, Schulz KF. An overwiev of clinical research: the lay of the land. Lancet 2002; 359:57-61

127. Sandvik H, Hunskaar S. Incontinence in women: different response rates may introduce bias in community studies of pad consumption. J Epidemiol Community Health. 1994;48: 419.

128. Sandvik H, Hunskaar S, Vanvik A, Bratt H, Seim A, Hermstad R. Diagnostic classification of female urinary incontinence: an epidemiological survey corrected for validity. J Clin Epidemiol. 1995;48:339-43.

129. Thom D. Variation in estimates of urinary incontinence prevalence in the community: effects of differences in definition, population characteristics, and study type. J Am Geriatr Soc. 1998;46:473-480.

130. Hanley J, Capewell A, Hagen S. Validity study of the severity index, a simple measure of urinary incontinence in women. BMJ. 2001;322:1096-7.

131. Monz B, Chartier-Kastler E, Hampel C, Samsioe G, Hunskaar S, Espuna-Pons M, et al. Patient characteristics associated with quality of life in European women seeking treatment for urinary incontinence: results from PURE. Eur Urol. 2007;51:1073-81; discussion 81-2.

132. Hannestad YS, Rortveit G, Hunskaar S. Help-seeking and associated factors in female urinary incontinence. The Norwegian EPINCONT Study. Epidemiology of Incontinence in the County of Nord-Trondelag. Scand J Prim Health Care. 2002;20:102-7.

133. Gutman RE, Ford DE, Quiroz LH, Shippey SH, Handa VL. Is there a pelvic organ prolapse threshold that predicts pelvic floor symptoms? Am J Obstet Gynecol. 2008;199:683 e1-7.

134. Macmillan AK, Merrie AE, Marshall RJ, Parry BR. The prevalence of fecal incontinence in community-dwelling adults: a systematic review of the literature. Dis Colon Rectum. 2004;47:1341-9.

135. Rortveit G, Dalveit AK, Hannestad YS, et al. Vaginal delivery parameters and urinary incontinence: The Norwegian EPINCONT study. Am J Obstet Gynecol 2003;189:1268-74.

136. McKinnie V, Swift SE, Wang W, Woodman P, O'Boyle A, Kahn M, et al. The effect of pregnancy and mode of delivery on the prevalence of urinary and fecal incontinence. Am J Obstet Gynecol. 2005;193:512-7; discussion 7-8.

137. Lukacz ES, Lawrence JM, Contreras R, Nager CW, Luber KM. Parity, mode of delivery, and pelvic floor disorders. Obstet Gynecol. 2006;107:1253-60.

138. Dolan LM, Hilton P. Obstetric risk factors and pelvic floor dysfunction 20 years after first delivery. Int Urogynecol J. 2010;21:535-44.

139. Leijonhufvud A, Lundholm C, Cnattingius S, Granath F, Andolf E, Altman D. Risks of stress urinary incontinence and pelvic organ prolapse surgery in relation to mode of childbirth. Am J Obstet Gynecol. 2011;204:70 e1-7.

140. Groutz A, Rimon E, Peled S, Gold R, Pauzner D, Lessing JB, et al. Cesarean section: does it really prevent the development of postpartum stress urinary incontinence? A prospective study of 363 women one year after their first delivery. Neurourol Urodyn. 2004;23:2-6.

141. Chiarelli P, Brown W, McElduff P. Leaking urine: prevalence and associated factors in Australian women. Neurourol Urodyn. 1999;18:567-77.

142. Sampselle CM, Harlow SD, Skurnick J, Brubaker L, Bondarenko I. Urinary incontinence predictors and life impact in ethnically diverse perimenopausal women. Obstet Gynecol. 2002 ;100:1230-38.

143. Zhu L, Lang J, Liu C, Han S, Huang J, Li X. The epidemiological study of women with urinary incontinence and risk factors for stress urinary incontinence in China. Menopause. 2009;16:831-36.

144. Han MO, Lee NY, Park HS. Abdominal obesity is associated with stress urinary incontinence in Korean women. Int Urogynecol J Pelvic Floor Dysfunct. 2006;17:35-9.

145. Hunskaar S, Arnold EP, Burgio K, Diokno AC, Herzog AR, Mallett VT. Epidemiology and natural history of urinary incontinence. Int Urogynecol J Pelvic Floor Dysfunct. 2000;11:301-19.

146. Subak LL, Richter HE, Hunskaar S. Obesity and urinary incontinence: epidemiology and clinical research update. J Urol. 2009;182(6 Suppl):S2-7.

147. Mommsen S, Foldspang A. Body mass index and adult female urinary incontinence. World J Urol 1994;12:319-22.

148. Hannestad YS, Rortveit G, Daltveit AK, Hunskaar S. Are smoking and other lifestyle factors associated with female urinary incontinence? The Norwegian EPINCONT Study. BJOG: An International Journal of Obstetrics & Gynaecology. 2003;110:247-54.

149. Brown JS, Grady D, Ouslander JG, Herzog AR, Varner RE, Posner SF. Prevalence of urinary incontinence and associated risk factors in postmenopausal women. Obstet Gynecol. 1999;94:66-70.

150. Kuh D, Cardozo L, Hardy R. Urinary incontinence in middle aged women: childhood enuresis and other lifetime risk factors in a British prospective cohort. J Epidemiol Community Health. 1999;53:453-8.

151. Bump RC, Norton PA. Epidemiology and natural history of pelvic floor dysfunction. Obstet Gynecol Clin North Am. 1998;25:723-46.

152. Parazzini F, Chiaffarino F, Lavezzari M, Giambanco V, VIVA Study Group. Risk factors for stress, urge or mixed urinary incontinence in Italy. BJOG 2003;110:927-33.

153. Persson J, Wolner-Hanssen P, Rydhstroem H. Obstetric risk factors for stress urinary incontinence: a population-based study. Obstet Gynecol. 2000;96:440-5.

154. Rortveit G, Hunskaar S. Urinary incontinence and age at the first and last delivery: the Norwegian HUNT/EPINCONT study. Am J Obstet Gynecol. 2006;195:433-8.

155. Leijonhufvud A, Lundholm C, Cnattingius S, Granath F, Andolf E, Altman D. Risk of surgically managed pelvic floor dysfunction in relation to age at first delivery. Am J Obstet Gynecol. 2012 ;207:303 e1-7.

156. Højberg KE, Salvig JD, Winsløw NA, Lose G, Secher NJ. Urinary incontinence: prevalence and risk factors at 16 weeks of gestation. Br J Obstet Gynaecol. 1999;106:842-50.

157. Connolly TJ, Litman HJ, Tennstedt SL, Link CL, McKinlay JB. The effect of mode of delivery, parity, and birth weight on risk of urinary incontinence. Int Urogynecol J Pelvic Floor Dysfunct. 2007;18:1033-42.

158. Thom DH, Brown JS, Schembri M, Ragins AI, Creasman JM, Van Den Eeden SK. Parturition events and risk of urinary incontinence in later life. Neurourol. Urodyn. 2011;30:1456-61.

159. Buchsbaum GM. Urinary incontinence and pelvic organ prolapse. Minerva Urol Nefrol. 2006;58:311-9.

160. Rinne K. What predisposes young women to genital prolapse? European Journal of Obstetrics, Gynecology, & Reproductive Biology. 1999;84:23-5.

161. Spence-Jones C, Kamm MA, Henry MM, Hudson CN. Bowel dysfunction: a pathogenic factor in uterovaginal prolapse and urinary stress incontinence. Br J Obstet Gynaecol. 1994;101:147-52.

162. Schaffer JI, Wai CY, Boreham MK. Etiology of pelvic organ prolapse. Clin Obstet Gynecol. 2005 ;48:639-47.

163. Slieker-ten Hove MC, Pool-Goudzwaard AL, Eijkemans MJ, Steegers-Theunissen RP, Burger CW, Vierhout ME. The prevalence of pelvic organ prolapse symptoms and signs and their relation with bladder and bowel disorders in a general female population. Int Urogynecol J Pelvic Floor Dysfunct. 2009;20:1037-45.

164. Handa VL, Blomquist JL, Knoepp LR, Hoskey KA, McDermott KC, Munoz A. Pelvic floor disorders 5-10 years after vaginal or cesarean childbirth. Obstet Gynecol. 2011;118:777-84.

165. Patel DA, Xu X, Thomason AD, Ransom SB, Ivy JS, DeLancey JO. Childbirth and pelvic floor dysfunction: an epidemiologic approach to the assessment of prevention opportunities at delivery. Am J Obstet Gynecol. 2006;195:23-8.

166. Risk factors for genital prolapse in non-hysterectomized women around menopause. Results from a large cross-sectional study in menopausal clinics in Italy. Progetto Menopausa Italia Study Group. Eur J Obstet Gynecol Reprod Biol 2000;93:135-40.

167. Miedel A, Tegerstedt G, Maehle-Schmidt M, Nyren O, Hammarstrom M. Nonobstetric risk factors for symptomatic pelvic organ prolapse. Obstet Gynecol 2009;113:1089-97.

168. Hendrix SL, Clark A, Nygaard I, Aragaki A, Barnabei V, McTiernan A. Pelvic organ prolapse in the Women's Health Initiative: gravity and gravidity. Am J Obstet Gynecol 2002;186:1160-6.

169. Samuelsson E, Ladfors L, Wennerholm UB, Gareberg B, Nyberg K, Hagberg H. Anal sphincter tears: prospective study of obstetric risk factors. BJOG. 2000;107:926-31.

170. Timonen S, Nuoranne E, Meyer B. Genital prolapse. Etiological factors. Ann Chir Gynaecol Fenn 1968;57:363-70.

171. Pretlove SJ, Thompson PJ, Toozs-Hobson PM, Radley S, Khan KS. Does the mode of delivery predispose women to anal incontinence in the first year postpartum? A comparative systematic review. BJOG 2008;115:421-34.

172. Pinta TM, Kylanpaa ML, Teramo KA, Luukkonen PS. Sphincter rupture and anal incontinence after first vaginal delivery. Acta Obstet Gynecol Scand. 2004;83:917-22.

173. Sultan AH, Thakar R, Fenner D. Diagnosis of perineal trauma, in Perineal and anal sphincter trauma: diagnosis and clinical management. Springer: London. 2007 p195.

174. Groom KM, Paterson-Brown S. Can we improve on the diagnosis of third degree tears? Eur J Obstet Gynecol Reprod Biol. 2002;101:19-21.

175. Bharucha AE, Fletcher JG, Melton LJ, 3rd, Zinsmeister AR. Obstetric trauma, pelvic floor injury and fecal incontinence: a population-based case-control study. Am J Gastroenterol. 2012;107:902-11.

176. Samuelsson EC, Victor FT, Tibblin G, Svardsudd KF. Signs of genital prolapse in a Swedish population of women 20 to 59 years of age and possible related factors. Am J Obstet Gynecol 1999;180:299-305.

177. Whitehead WE, Wald A, Norton NJ. Treatment options for fecal incontinence. Dis Colon Rectum 2001;44:131-42.

178. Raisanen S, Vehvilainen-Julkunen K, Gissler M, Heinonen S. High episiotomy rate protects from obstetric anal sphincter ruptures: a birth register-study on delivery intervention policies in Finland. Scand J Public Health. 2011;39:457-63.

Appendix

UNDERSÖKNING AV KVINNORS BÄCKENBOTTENBESVÄR EFTER BARNAFÖDANDE

Detta formulär innehåller frågor om symtom och besvär från bäckenbotten och hur det i så fall påverkar Dig i ditt dagliga liv. Sätt ett kryss i den ruta som Du anser stämmer bäst in på Dig. Även om det inte exakt beskriver hur Du upplever besvären, kryssa ändå i den ruta som känns mest riktig för Dig. Vi är tacksamma om Du fyller i frågorna även om Du inte har några besvär.

1. Hur lång är du? ☐☐☐
2. Hur mycket väger du? ☐☐☐
3. Hur många barn har du fött? ☐☐
4. Menstruerar du fortfarande? JA ☐ NEJ ☐

5. Om du inte menstruerar-

Är du gravid?	JA ☐ NEJ ☐
Är livmodern bortopererad?	JA ☐ NEJ ☐
Använder du hormonspiral?	JA ☐ NEJ ☐
Är du i klimakteriet?	JA ☐ NEJ ☐
Använder du östrogen?	JA ☐ NEJ ☐

BESVÄR IFRÅN URINVÄGARNA

6. Har du ofrivilligt urinläckage? JA ☐ NEJ ☐

Om du inte har besvär med urinläckage gå vidare till fråga 15

7. Hur ofta har du urinläckage?
   - mer sällan än en gång i månaden ☐
   - en gång i månaden eller mer ☐
   - en gång i veckan eller mer ☐
   - varje dag och/eller natt ☐

8. Hur mycket urin läcker Du vid varje läckagetillfälle?

- några droppar eller lite ☐
- små mängder ☐
- stora mängder ☐

9. Har du urinläckage när Du hostar, nyser, skrattar eller lyfter tungt? JA ☐ NEJ ☐

10. Har Du urinläckage i samband med plötsligt påkomna och starka urinträngningar? JA ☐ NEJ ☐

11. Hur länge har Du haft urinläckage?

-0-5 år ☐
-5-10 år ☐
- mer än 10 år ☐

12. Har Du sökt läkare på grund av ditt urinläckage? JA ☐ NEJ ☐

13. Hur påverkas Du av ditt urinläckage?

- inget problem ☐
- lite besvär ☐
- en del besvär ☐
- mycket besvär ☐
- mycket stort problem ☐

14. Har urinläckage påverkat din(a):

- förmåga att utföra hushållsarbete?

Inte alls ☐
Lite ☐
Måttligt ☐
Mycket ☐

- fysiska aktiviteter som promenader, simning osv?

Inte alls ☐
Lite ☐
Måttligt ☐
Mycket ☐

- nöjen som att gå på bio, konsert o dyl?

Inte alls ☐
Lite ☐
Måttligt ☐
Mycket ☐

- förmåga att åka bil eller buss mer än 30 min hemifrån?

Inte alls ☐
Lite ☐
Måttligt ☐
Mycket ☐

- medverkan vid sociala evenemang utanför hemmet?

Inte alls ☐
Lite ☐
Måttligt ☐
Mycket ☐

- mentala hälsa (nervositet, depression osv)

Inte alls ☐
Lite ☐
Måttligt ☐
Mycket ☐

- känsla av frustration?

Inte alls ☐
Lite ☐
Måttligt ☐
Mycket ☐

15. Har du fått någon behandling för urinläckage? JA ☐ NEJ ☐

16. Har din mor besvärats av urinläckage? JA ☐ NEJ ☐

BESVÄR IFRÅN SLIDAN

17. Har Du en känsla av att något buktar fram ur slidan?
- ja ofta ☐
- ibland ☐
- någon gång ☐
- nej aldrig ☐

18. Händer det att Du har skavningsbesvär i underlivet?
- ja ofta ☐
- ibland ☐
- någon gång ☐
- nej aldrig ☐

19. Händer det att Du behöver lyfta fram främre slidväggen för att kunna kissa?
- ja ofta ☐
- ibland ☐
- någon gång ☐
- nej aldrig ☐

Besvara endast fråga 20-21 om Du *har* besvär från slidan, om inte, gå till fråga 22

20. Om Du anstränger dig med tunga lyft blir dina besvär:
- oförändrade ☐
- bättre ☐
- sämre ☐

21. Har framfall påverkat din(a):

- förmåga att utföra hushållsarbete?
  - Inte alls ☐
  - Lite ☐
  - Måttligt ☐
  - Mycket ☐

- fysiska aktiviteter som promenader, simning osv?
  - Inte alls ☐
  - Lite ☐
  - Måttligt ☐
  - Mycket ☐

- nöjen som att gå på bio, konsert odyl?

Inte alls ☐
Lite ☐
Måttligt ☐
Mycket ☐

- förmåga att åka bil eller buss mer än 30 min hemifrån?

Inte alls ☐
Lite ☐
Måttligt ☐
Mycket ☐

- medverkan vid sociala evenemang utanför hemmet?

Inte alls ☐
Lite ☐
Måttligt ☐
Mycket ☐

- mentala hälsa (nervositet, depression osv)

Inte alls ☐
Lite ☐
Måttligt ☐
Mycket ☐

- känsla av frustration?

Inte alls ☐
Lite ☐
Måttligt ☐
Mycket ☐

22. Har du fått någon behandling för framfall? JA ☐ NEJ ☐

23. Har din mor besvärats av framfall? JA ☐ NEJ ☐

BESVÄR IFRÅN ÄNDTARMEN

24. Läcker Du fast avföring ofrivilligt?

- aldrig ☐
- mer sällan än 1 ggr i månaden ☐
- flera ggr i månaden, men inte varje vecka ☐
- alltid, varje dag ☐

25. Läcker Du lös avföring ofrivilligt?

- aldrig ☐
- mer sällan än 1 ggr i månaden ☐
- flera ggr i månaden, men inte varje vecka ☐
- alltid, varje dag ☐

26. Läcker Du gas ofrivilligt?

- aldrig ☐
- mer sällan än 1 ggr i månaden ☐
- flera ggr i månaden, men inte varje vecka ☐
- alltid, varje dag ☐

Om Du *inte* läcker gas eller avföring gå vidare till fråga 30

27. Använder Du skydd pga ofrivilligt läckage ifrån tarmen?

- aldrig ☐
- mer sällan än 1 ggr i månaden ☐
- flera ggr i månaden, men inte varje vecka ☐
- alltid, varje dag ☐

28. Påverkas din dagliga livsföring pga ofrivilligt läckage från tarmen?

- aldrig ☐
- mer sällan än 1 ggr i månaden ☐
- flera ggr i månaden, men inte varje vecka ☐
- alltid, varje dag ☐

29. Har gas eller avföringsläckage påverkat din(a):

- förmåga att utföra hushållsarbete?

Inte alls ☐
Lite ☐
Måttligt ☐
Mycket ☐

- fysiska aktiviteter som promenader, simning osv?

Inte alls ☐
Lite ☐
Måttligt ☐
Mycket ☐

- nöjen som att gå på bio, konsert odyl?

Inte alls ☐
Lite ☐
Måttligt ☐
Mycket ☐

- förmåga att åka bil eller buss mer än 30 min hemifrån?

Inte alls ☐
Lite ☐
Måttligt ☐
Mycket ☐

- medverkan vid sociala evenemang utanför hemmet?

Inte alls ☐
Lite ☐
Måttligt ☐
Mycket ☐

- mentala hälsa (nervositet, depression osv)

Inte alls ☐
Lite ☐
Måttligt ☐
Mycket ☐

- känsla av frustration?

Inte alls ☐
Lite ☐
Måttligt ☐
Mycket ☐

30. Har du fått någon behandling för gas eller avföringsläckage? JA ☐ NEJ ☐

31. Har din mor besvärats av gas eller avföringsläckage? JA ☐ NEJ ☐