Urinary incontinence (UI) is an often under-reported and untreated medical condition that affects the lives of women of all ages. Although there are six major types of UI, stress urinary incontinence (SUI) is the type most often associated with women of childbearing age. Development of SUI has also been linked to the childbirth process itself. This article will discuss the types, characteristics, and diagnosis of UI, with a focus on SUI. Precipitating factors for SUI, including the influence of pregnancy, childbirth, and aging on its development, will be reviewed.

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Key Words: Urinary incontinence, pelvic floor disorder, stress urinary incontinence, pelvic organ prolapse, pregnancy, aging, childbirth.
pelvic floor muscles, as well as denervation of the PF during obstetric events, increases the likelihood of UI development (Altman et al., 2006; Morkved et al., 2003; Nygaard, 2005; Pantazis & Freeman, 2006; Wesnes et al., 2007). Damage to the pudendal nerve, such as that which can occur during childbirth, is associated with PFD (Peeker & Peeker, 2003; Viktrup et al., 2006). Childbirth injuries to the PF and their potential long-term sequelae are major public health concerns because the economic and psychological costs of managing UI and POP are significant (Heit, Mudd, & Culligan, 2001).

The causal association of UI with pregnancy and childbirth, particularly antenatal and pre-pregnancy UI, has become widely accepted over the last several decades. It is thought that childbirth-related UI results from trauma to the supportive tissue, ligaments, and pelvic floor muscles, as well as from denervation of the PF during obstetric events (Altman et al., 2006; Morkved et al., 2003; Pantazis & Freeman, 2006; Wesnes et al., 2007; Woldringh, van den Wijngaart, Albers-Heitner, Lycklama a Nijeholt, & Lagro-Janssen, 2007).

Vaginal delivery may increase the risk of PFD by two to three fold, and first delivery has a greater association with long-lasting SUI over first pregnancy (Viktrup et al., 2006; Wesnes et al., 2007; Nygaard, 2005; Sand et al., 1995). High parity and forceps deliveries are associated with an increased incidence of postnatal UI, which is a risk factor for long-term UI (Pantazis & Freeman, 2006; Peeker & Peeker, 2003).

Much of the literature that focuses on vaginal deliveries and their relation to PFD and UI are based on all types of vaginal deliveries. This includes instrumental deliveries as well as common second-stage interventions, including episiotomy and forced pushing. These types of interventions may increase the risk of PFD by compromising the integrity of the intact pelvic floor during birth (Leslie, 2004).

Recently, there has been a trend in primary, elective cesarean sections (CS) without medical indications. Instead of undergoing a trial of labor and subsequent vaginal delivery, women are opting for a surgical resolution to their pregnancies. Protection of the PF is one of the main arguments for this procedure (Bettes et al., 2007; Nygaard, 2006; Visco et al., 2006). However, most women who deliver vaginally do not have severe PFD (Nygaard, 2005). In addition, it is important to note that some women who never become pregnant or bear children develop PFD or UI (Buchsbaum et al., 2002).

**TYPES OF URINARY INCONTINENCE**

The six types of UI classifications are stress (SUI), urge (UUI), mixed (MUI), overflow, functional, and reflex (see Figure 1). The three most common types of UI are SUI, UUI, and MUI. SUI is the most common type of UI in women under age 60 and accounts for almost half of incon-

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**Figure 1. Major Classifications of Urinary Incontinence**

<table>
<thead>
<tr>
<th>Type of Urinary Incontinence</th>
<th>Definition</th>
<th>Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Stress Urinary Incontinence</strong></td>
<td>Involuntary leakage upon effort or exertion, or on sneezing or coughing; loss of urine upon increased abdominal pressure.</td>
<td>Urethral hypermobility due to weakened pelvic floor muscles, pregnancy, intrinsic sphincter deficiency, or aging.</td>
</tr>
<tr>
<td><strong>Urge Urinary Incontinence (Overactive Bladder in Between Overactivity)</strong></td>
<td>Involuntary leakage of urine immediately preceded or accompanied by urgency; sudden sensation of a need to urinate with inability to get to a toilet before involuntary leakage.</td>
<td>Overactive bladder; detrusor overactivity +/- spontaneous contraction, bladder infections, nerve damage from stroke, dementia, or multiple sclerosis.</td>
</tr>
<tr>
<td><strong>Mixed Urinary Incontinence</strong></td>
<td>Involuntary leakage associated with urgency as well as with exertion, effort, or increased abdominal pressure, causing symptoms of both conditions.</td>
<td>See above; usually the type causing the most bothersome symptoms will be treated first.</td>
</tr>
<tr>
<td><strong>Overflow Incontinence</strong></td>
<td>Condition where the bladder is full but not able to empty properly. This causes leaks, dribbles, and hesitancy. More common in men.</td>
<td>Urethral blockage hindering passage of urine from the bladder; weakened bladder contractions due to diabetes mellitus or neurologic disorder.</td>
</tr>
<tr>
<td><strong>Functional Incontinence</strong></td>
<td>Condition where the urinary system is normal, but sufferers have mental or physical conditions that preclude adequate or appropriate toileting.</td>
<td>Parkinson’s disease, Alzheimer’s disease, severe depression, severe arthritic conditions.</td>
</tr>
<tr>
<td><strong>Reflex Incontinence</strong></td>
<td>Involuntary loss of bladder control without warning; the bladder empties due to reflex activity with no control of urination.</td>
<td>Neurological impairment, such as quadriplegia or spinal cord lesions.</td>
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</table>
SUI results from ineffective closure of the bladder neck and the urethral sphincter, often due to loss of integrity of the vaginal musculofascial attachments (including the pubo-urethral ligaments and endopelvic fascia) that normally support the bladder neck and urethra in a retropubic position. This loss leads to hypermobility and descent of structures, which ultimately results in impaired intra-abdominal pressure transmission to the urethra (Bernier & Sims, 2009; Culligan & Heit, 2000; Lingam, 2001; Lowdermilk, 2004; McCool & Durain, 2004; Pantazis & Freeman, 2006).

This loss of bladder neck support is often attributed to nerve, muscle, and connective tissue damage during vaginal delivery. Changes in the urethrovaginal junction due to weakness of the periurethral muscles from childbirth or menopause cause general loosening of the PF, which can create a funneling effect of the bladder neck during exertion (Bernier & Sims, 2009; Culligan & Heit, 2000; McCool & Durain, 2004; Pantazis & Freeman, 2006).

Up to one-third of women experience SUI precipitated by physical activity and exertion (including laughing, coughing, and exercising) in the absence of bladder (detrusor) contraction (Culligan & Heit, 2000; Moore, 2001; Rousseau, 2004). Deficient PF support, obesity, urinary tract infection (UTI), chronic cough, or sphincter weakness (intrinsic sphincter deficiency) are contributory factors (Bernier & Sims, 2009; Culligan & Heit, 2000; McCool & Durain, 2004; Pantazis & Freeman, 2006). Intrinsic sphincter deficiency is usually a more severe form of SUI that is associated with advanced age, estrogen loss, previous vaginal surgery, obstetric trauma, urogenital prolapse, and certain neurological conditions (Culligan & Heit, 2000). There is no urge component with SUI (Peeker & Peeker, 2003).

UUI is an involuntary loss of urine associated with a strong desire to void without delay, whether or not the bladder is full. This is accompanied by the inability to tighten the urethral sphincter to inhibit voiding. It is sometimes referred to as overactive bladder (OAB) (Bernier & Sims, 2009; Culligan & Heit, 2000; McCool & Durain, 2004; Peeker & Peeker, 2003; Pantazis & Freeman, 2006).

MUI is identified when symptoms of both SUI and UUI are present. It is diagnosed by a combination of urodynamic conditions, such as urodynamic SUI and detrusor overactivity (Bump, Norton, Zinner, & Yalcin, 2003; Culligan & Heit, 2000). Treatment of MUI is based on the predominance, or most bothersome, symptoms (Culligan & Heit, 2000; McCool & Durain, 2004; Pantazis & Freeman, 2006; Peeker & Peeker, 2003). In the clinical setting, MUI symptoms are actually more common than mixed conditions, and women whose UI is more bothersome or is perceived to be more severe tend to be diagnosed with MUI. However, upon urodynamic testing, many of these women often show conclusive results for either SUI or UUI, not both (Bump et al., 2003). In a randomized, double-blind, placebo-controlled study, Bump and coauthors (2003) found that with treatment and as the severity of UI improves, mixed symptoms resolved. This suggests that those patients that are more severe may produce mixed symptoms (Bump et al., 2003), which may indicate that MUI symptoms often impede predict the true pathophysiological mechanism responsible for UI in individual patients (Bump et al., 2003; Lingam, 2001). Generally, pregnancy and childbirth are more likely to precipitate symptoms of UI. For these reasons, this article will focus on SUI.
research on the impact of childhood UI on UI in adulthood because it could make providers aware of women who are likely to develop UI whether or not they give birth. Connective tissue disorders have a clear association with UI and POP (Heit et al., 2001). Although abnormalities of collagen have been implicated in the pathogenesis of UI and POP, the cause or effect remains to be established (Sultan & Fernando, 2001). Vaginal deliveries do not cause, nor does CS prevent, connective tissue abnormalities. These abnormalities may occur after years of decreased pelvic support, progressive pudendal neuropathy, and hypoestrogenism (Heit et al., 2001). Primary connective tissue abnormalities occur with pregnancy, and women who present with POP may have a genetically weaker endogenous collagen type and composition (Sasso, 2006). Identifying antenatal markers of collagen weakness that can predict women who may develop UI requires further study (Sultan & Fernando, 2001).

High BMI (>30) is a significant risk factor for long-term SUI symptoms, especially during the antenatal period (Buchbaum et al., 2002; Danforth et al., 2006; Minassian et al., 2006; Moore, 2001; Peek & Peeker, 2003; Viktrup et al., 2006; Wenner, 2007). Certain medications, including opioids, sedatives, alcohol, antihistamines, hypotensive agents, beta-adrenergic blockers, and diuretics, can contribute to SUI, as can chronic disease and caffeine intake (Carroll, 2009; Kincade et al., 2007; Moore, 2001). Family history of SUI in the mother or any UI in multiple first-degree relatives are also predictors of SUI in individual women (Heit et al., 2001). Caucasian race is associated with an increase in UI risk, and structural and functional differences in the urethra and its support systems have been demonstrated between African-American and Caucasian women. African-American women have greater levator ani (LA) strength and greater urethral sphincter closure pressure (Danforth et al., 2006; Graham & Mallet, 2001; McIntosh, 2005; Moore, 2001; Pantazis & Freeman, 2006; Waetjen et al., 2003).

**Influence of Pregnancy**

Physical changes during pregnancy may play a contributory role in the process of fecal and urinary incontinence, and even the most non-traumatic birth can result in loss of pelvic muscle tone from pregnancy alone (Borello-France et al., 2006; Martin & Paisley, 2004; McCool & Durain, 2004). Pregnancy as a risk factor for UI development suggests that factors other than the delivery of a child can contribute to SUI (Nygaard, 2006; Peeker & Peeker, 2003; Wesnes et al., 2007; Wilson et al., 1996). Although UI has been reported in 34% of women at 3 months postpartum, peak incidence is during pregnancy (Sultan & Fernando, 2001).

In the first trimester, the increased weight of the uterine fundus presses directly on the bladder and can compress the ureters. The ureters, renal pelvis, and calices dilate, and increasing levels of progesterone may lead to smooth muscle relaxation. Renal blood flow increases 50% with accompanying increases in the glomerular filtration rate (Martin & Paisley, 2004). In the second trimester, the uterus rises up and out of the pelvis to become an abdominal organ, and pressure on the bladder lessens. In the third trimester, after the presenting part of the fetus descends into the pelvis, direct pressure on the bladder, coupled with decreased room for bladder distension, increases UI symptoms again (Varney, Krieks, & Gregor, 2004a).

Hormonal influences may also precipitate urinary changes in pregnancy leading to UI development (Viktrup et al., 2006; Woldringh et al., 2007). Though urinary frequency is often a non-

**Figure 2. Risk Factors for Stress Urinary Incontinence**

<table>
<thead>
<tr>
<th>Risk Factor</th>
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<tbody>
<tr>
<td>Female gender</td>
</tr>
<tr>
<td>Pregnancy</td>
</tr>
<tr>
<td>Vaginal delivery</td>
</tr>
<tr>
<td>Advanced age</td>
</tr>
<tr>
<td>BMI &gt; 30</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
</tr>
<tr>
<td>Smoking</td>
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<tr>
<td>Hysterectomy</td>
</tr>
<tr>
<td>Spinal cord trauma</td>
</tr>
<tr>
<td>Neurological disease</td>
</tr>
<tr>
<td>Chronic cough</td>
</tr>
<tr>
<td>Constipation</td>
</tr>
<tr>
<td>Pelvic floor injury</td>
</tr>
<tr>
<td>High impact exercise</td>
</tr>
<tr>
<td>Ethnicity (Caucasian race)</td>
</tr>
<tr>
<td>Pelvic surgery</td>
</tr>
<tr>
<td>Certain medications</td>
</tr>
<tr>
<td>Pelvic prolapse</td>
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</tbody>
</table>
pathological discomfort of pregnancy, onset of SUI during first pregnancy or the puerperal period carries the risk of long-term symptoms at 5 and 15 years post-first delivery (Dolan, Hosker, Mallett, Allen, & Smith, 2003; Varney et al., 2004a; Viktrup et al., 2006; Woldringh et al., 2007). Five years post-delivery, the risk of SUI is four times as high (Viktrup et al., 2006), and at 15 years post-delivery, it is twice as high (Dolan et al., 2003). Obesity before first pregnancy and delivery increases the risk of SUI; in this case, there is a risk that SUI will become chronic and continue up to 12 years postpartum (Viktrup et al., 2006; Wesnes et al., 2007).

Using data from the Norwegian Mother and Child Cohort Study, Wesnes et al. (2007) investigated the incidence and prevalence of UI during pregnancy and its associated risk factors. Of the 43,279 data sets examined, 26.2% of all women reported UI before pregnancy (15.4% of nulliparous women, 33% of primiparous women, and 40% of multiparous women). SUI was the most commonly reported (Wesnes et al., 2007). During pregnancy, 58.1% (25,121) of women reported UI, again with SUI the most common. Among women who were continent prior to pregnancy, incidence of any incontinence by week 30 of pregnancy was 45.6%. During pregnancy, 48% of nulliparous women and 66.6% of parous women experienced incontinence. In this study, prevalence of any UI was doubled compared with prevalence before pregnancy, with the increase most notable in the SUI component. UI both prior to and during pregnancy was found to be associated with parity, age, and BMI (Wesnes et al., 2007).

**Bony Pelvis**

The bony structure of the pelvis itself may indicate women at a higher risk for developing UI after pregnancy and childbirth, and may also provide further explanation for the racial differences in UI symptoms. Handa and coauthors (2003) performed a retrospective, record review, case-control study of women who had undergone pelvic MRI to examine differences between the bony pelvis structures of women with and without PFD. Fifty-nine subjects were in the case group (women with PFD) and 39 were in the control group (women without PFD). Of the 59 women with PFD, the most common complaints were SUI and POP. According to the demographics of the study cohort, women presenting with PFD were more likely to be white, older, and of higher parity.

Typical obstetric pelvic measurements that are adequate for vaginal delivery include:

- The transverse inlet is the measurement of the greatest distance between the linea terminalis on either side of the pelvis, and it should be 12 cm or more.
- The obstetrical conjugate is the measurement extending from the middle of the sacral promontory to the middle of the inner surface of the symphysis pubis, and should be 10 cm or more.
- The interspinous diameter is the measurement between the ischial spines and is the plane of least dimensions, making it the smallest area through which the fetal head must pass, and should be of 10 cm or more.

**Sources:** Lowdermilk (2004); Varney et al. (2004b).

**Table 1. Bony Pelvis Shapes**

<table>
<thead>
<tr>
<th>Bony Pelvis Shapes</th>
<th>Gynecoid</th>
<th>Android</th>
<th>Anthropoid</th>
<th>Platypelloid</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>“Female” pelvis</td>
<td>“Male” pelvis</td>
<td>Occurs in 40.5% non-white and 23.5% white women</td>
<td>Rare</td>
</tr>
<tr>
<td></td>
<td>Most common (41% to 42 % of women)</td>
<td>Occurs in 32.5 % white women, 15.7% non-white women</td>
<td>Oval shape, with anteroposterior(AP) diameter greater than the transverse</td>
<td>Not conducive to vaginal delivery</td>
</tr>
<tr>
<td></td>
<td>Ideal for childbearing</td>
<td>Heavy, heart-shaped pelvis, increased incidence of posterior fetal position</td>
<td>Well-rounded posterior and oval inlet</td>
<td>&lt; 3% occurrence in white and non-white women</td>
</tr>
<tr>
<td></td>
<td>Well-rounded anterior, lateral and posterior segments</td>
<td>Increased incidence of forceps delivery</td>
<td>Anterior segment narrower than posterior</td>
<td>Flattened gynecoid-type pelvis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Contracted midplane and outlet increase cesarean delivery</td>
<td>Favors posterior fetal position, but adequate for vaginal delivery</td>
<td>Wide transverse diameter and short AP diameter</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Widest of all pelvic types</td>
<td></td>
</tr>
</tbody>
</table>

**Bony pelvic measurements:**

Typical obstetric pelvic measurements that are adequate for vaginal delivery include:

- The transverse inlet is the measurement of the greatest distance between the linea terminalis on either side of the pelvis, and it should be 12 cm or more.
- The obstetrical conjugate is the measurement extending from the middle of the sacral promontory to the middle of the inner surface of the symphysis pubis, and should be 10 cm or more.
- The interspinous diameter is the measurement between the ischial spines and is the plane of least dimensions, making it the smallest area through which the fetal head must pass, and should be of 10 cm or more.
parity, PFDs were 7.2 times more likely in women with transverse inlets greater than 13.9 cm (p < 0.01). Platypelloid pelvic shapes (wide, ovoid inlet) have the highest chance of UI, while the anthropoid pelvic structure (heart-shaped, narrow transverse inlet, wide obstetrical conjugate, and narrow spines) has the lowest chance of UI. Statistically, African-American women are more likely to have the anthropoid pelvic structure, and they are also less likely to suffer from POP and SUI (Handa et al., 2003; Varney et al., 2004b).

The platypelloid pelvis is also associated with deep transverse arrest in labor. This can prolong the second stage of labor, predisposing women to neuropathy and increasing their risk of UI development (Handa et al., 2003; Pantazis & Freeman, 2006; Viktrip et al., 2006). The narrow obstetric conjugate may also increase trauma to structures along the anterior sacrum, including the origins of levator ani, uterosacral ligaments, and hypogastric nerve. Women with less than adequate pelvic features may be predisposed to neuromuscular and connective tissue injuries, thus leading to PFD because women without PFDs were less likely to have inadequate pelvimetry (Handa et al., 2003).

**Advancing Age**

Before menopause, estrogen receptors are present in female urethral and bladder tissues, as well as in the PF musculature (Herbruck, 2009; Chancellor, 2000; Moore, 2001; Rovner & Wein, 2004). Depletion of estrogen at menopause is associated with diminished urethral mucosa, vascularity, and thickness, all of which can cause the urethra to lose its ability to maintain a tight seal, especially when intra-abdominal pressure increases, such as with the Valsalva maneuver, coughing, or exercise (Bernier & Sims, 2009; Moore, 2001). Reduction of estrogen with aging also leads to vaginal dryness, loss of tissue elasticity affecting support in the perineal region, devascularization and thinning of mucosal and support tissues, urogenital changes (including decreases in collagen content), and atrophic changes in tissues of the pelvic floor (McIntosh, 2005; Rousseau, 2004; Sasso, 2006). Loss of estrogen also contributes to an increased tendency for PFD development in women with pelvises damaged by the childbirth process (McIntosh, 2005).

Independent predictors of UI in older women include Caucasian race, estrogen cream use, vaginal dryness, vaginal discharge, lifetime number of UTIs (> 6), and diabetic peripheral nephropathy. History of hysterectomy is predictive of severe incontinence (Jackson, Scholes, Boyko, Abraham, & Fihn, 2006). In some women, shrinking of the uterus, vulva, and distal portion of the urethra that accompanies aging can lead to urinary frequency, dysuria, ureteral prolapse, and SUI (Lowdermilk, 2004).

**Pelvic Organ Prolapse**

Pelvic organ prolapse (POP), a commonly occurring and distressing condition, is defined as protrusion or projection of pelvic organs into the vagina or outside the vagina that progresses over time (McIntosh, 2005; Sasso, 2006; Farkas & Radley, 2002). Vaginal anatomical models from DeLancey (1993) depict the support of pelvic structures to show that vaginal prolapse is a phenomenon of the failure of vaginal, not uterine, support (Delancey, 1999; Farkas & Radley, 2002). It is commonly thought that POP is caused by denervation of PF muscles and/or disruption of endopelvic fascia (Sasso, 2006). POP is associated with a distortion of the urethra and may cause voiding difficulties (Fernando, Thakar, Sultan, Shah, & Jones, 2006). Annual costs in the U.S. have been estimated to be around $10 billion annually for POP treatment and care (Romanzi, 2002). POP is often thought of as a silent disease because many women who suffer with the associated symptoms, including UI, rectal pressure, pain, and discomfort, often find it embarrassing to discuss it with their providers (Fernando et al., 2006; Sasso, 2006).

Risk factors for POP include age, parity, forceps delivery, neuromuscular damage of the PF, family history of prolapse, obesity, HRT use, menopause, hysterectomy, and connective tissue disorders (Bradley, Zimmerman & Nygaard, 2007; DeLancey et al., 2007; Farkas & Radley, 2002; Heit & Culligan, 2001; Sasso, 2006). Other contributing factors to POP include smoking, individual anatomy, constipation, neurological disease, long labor, instrument-assisted delivery, large birthweight babies, chronic respiratory disease, and Caucasian race (Buchsbaum et al., 2006; McIntosh, 2005; Vimplis & Hooper, 2005).

Vaginal birth confers a 4 to 11-fold increase in risk for developing POP among parous women and is the single most important modifiable factor for this condition (Delancey et al., 2007). Fifty percent of parous women over age 50 are affected by POP (Fernando, et al., 2006); however, many parous women never develop symptoms of POP. POP can also occur in women who have never given birth because uterine prolapse occurs in 2% of nulliparous women (Buchsbaum et al., 2006; McIntosh, 2005). Lukacz, Lawrence, Contreras, Nager, and Luber (2006) found that incidence of POP is the same in women who have had a CS after labor had begun and women who had vaginal delivery, but incidence was lower in women who had a CS without labor. This may indicate that it is the event of labor, rather than the pregnancy, that contributes more to POP development. Their study also found that women who had been pregnant but who had never delivered an infant had an increased risk in POP over nulliparous women.

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**Additional Notes**

- Pelvic organ prolapse (POP) is a common condition affecting women of all ages, with risk factors including age, parity, forceps delivery, neuromuscular damage of the PF, family history of prolapse, obesity, HRT use, menopause, hysterectomy, and connective tissue disorders.
- Risk factors for POP can be managed through lifestyle changes and medical interventions.
- The incidence of POP can be increased in women who have had a cesarean section after labor, but the exact mechanism remains unclear.
- Pelvic floor disorders (PFDs) are prevalent and can significantly impact quality of life, necessitating effective management strategies.
Bradley et al. (2007) performed a 4-year prospective observational study on 259 postmenopausal women to examine the natural history of POP and risk factors for changes in vaginal descent. They found that prolapse progresses and regresses in older women, with rates of vaginal descent progression slightly greater than regression. DeLancey et al. (2007) studied 286 women (151 with POP and 135 without POP) using MRI to determine and compare LA defects between the groups. LA muscles are unique striated muscles that play a critical role in pelvic organ support because they provide postural and upward support to the pelvic viscera. They found that there are strong associations between POP and major LA defects ($p < 0.001$). Women with POP were more likely to have major LA defects when compared to controls (55% to 16%), but were equally likely to have minor defects. Fifty-three percent of women in the study who delivered by forceps had major LA defects.

Although POP is a common disorder, the true incidence is not known. Some women who present with what would be considered “severe” POP have no symptoms, while some women with “mild” POP have debilitating symptoms (Sasso, 2006; Vimplis & Hooper, 2005). Patients with a major degree of POP rarely have SUI (Vimplis & Hooper, 2005; Ward, 2003).

Treatment and assessment of POP is based as much on symptomology as it is the extent of the prolapse. Symptom assessment must include the prolapse itself, as well as related PFDs, including urinary, bowel, and sexual functioning. The patient’s view of her symptoms, needs, and expectations are paramount to her treatment (Farkas & Radley, 2002). Symptoms generally relate to type and location of POP, but location and extent may not correlate well with symptom severity (Sasso, 2006). Treatment of POP strives to maintain urinary and bowel function, preserve sexual function, and decrease irritating symptoms (Farkas & Radley, 2002; Vimplis & Hooper, 2005). Symptoms generally relate to type and location of POP, but location and extent may not correlate well with symptom severity (Sasso, 2006).

Treatment of POP strives to maintain urinary and bowel function, preserve sexual function, and decrease irritating symptoms (Farkas & Radley, 2002; Vimplis & Hooper, 2005). Pessary use improves POP and associated pelvic organ dysfunction, including bladder, bowel, and sexual function. Pessaries may be an appropriate conservative treatment, and is a first-line treatment in women who are not good surgical candidates (Bernier & Sims, 2009, Fernando et al., 2006; Herbruck, 2009; Vimplis & Hooper, 2005). Sometimes pessary placement can unmask incontinence by straightening out urethral kinks that may have been causing some level of urinary retention (Walters, 2005). However, pessary placement, particularly when fitted with an incontinence ring, can help reduce symptoms of known SUI and UUI (Culligan & Heit, 2000; Fernando et al., 2006; McIntosh, 2005; Rovner & Wein, 2004).

Alternative treatment of POP may also include lifestyle changes, such as weight loss or resolution of constipation, to prevent worsening of POP (Vimplis & Hooper, 2005). Surgical treatment includes colposuspension of the anterior or posterior compartments. Newer techniques exist, including tension-free vaginal tape and other procedures that employ the use of synthetic materials. Ongoing studies aim to determine their effectiveness when compared to colposuspension (Farkas & Radley, 2002).

**SUI in the Nulliparous Woman**

With aging populations on the rise, greater numbers of nulliparous women are being identified with urogential prolapse and SUI (Sultan & Fernando, 2001). It is important to remember that nulliparous women experience SUI and that UI itself is not entirely related to childbirth (Lowdermilk, 2004). Additional factors other than childbirth that might contribute to UI development include heredity, menopause and the subsequent loss of estrogen, and pelvic surgeries (Altman et al., 2006; Buchsbaum et al., 2002; Nygaard, 2005).

Buchsbaum and colleagues (2002) performed a study examining the prevalence of UI among a group of 149 nulliparous nuns. Individual medical history, demographic data, and symptoms of UI were examined. This study concluded that prevalence of UI among nulliparous, postmenopausal nuns was similar to UI rates reported by parous, postmenopausal women. After multivariate logistic regression, BMI ($p = 0.001$), multiple UTIs ($p = 0.033$), and depression ($p = 0.022$) were significant statistics that correlated with UI in the study group (Buchsbaum et al., 2002).

Since parity and pelvic trauma are considered major risk factors for SUI, one might expect the occurrence of UUI to be higher than occurrence of UI in nulliparous postmenopausal women. However, in the Buchsbaum et al. (2002) study, they found that UI in elderly, postmenopausal, nulliparous women was associated with SUI more often than UUI, even in the absence of trauma of the pelvic floor due to pregnancy or childbirth. These results directly contradict the theories that nulliparity protects against SUI.

Trowbridge and colleagues (2007) performed a cross-sectional study involving a cohort of 82 nulliparous women to evaluate the effects of aging, independent of parity, on POP, urethral support and function, and levator function. Women 20 to 71 years of age underwent a pelvic examination. Additional tests performed were POP quantification (POP-Q measured at maximal Valsalva strain),
urethral angles by cotton-tipped swab, and multichannel urodynamics and uroflow. Vaginal closure was also quantified. In this study, increasing age was associated with decreasing maximal urethral closure pressure ($r = -0.758, p < 0.001$), averaging a 15-cm-$H_2O$ drop per decade. Maximal urethral closure pressure in the older women was 40% that of the younger women (Trowbridge et al., 2007). The changes in mean urethral close pressure may reflect histologic changes in urethral striated muscle, blood vessels, and connective tissue. Increasing age did not affect clinical measures of POP, urethral support, and levator function. They concluded that urethral closure pressure may be a factor in SUI in older, nulliparous women. This study delineated an association between aging and a declining continence mechanism, regardless of parity (Trowbridge et al., 2007).

**SUMMARY**

The effects of UI on women are wide-reaching and life altering. Costs in both financial and emotional/social realms burden women of all ages throughout their life cycle. Though studies continue to find associations between incontinence and childbearing, it is prudent to remember that there are other genetic and environmental factors that can play a roll in the establishment of UI and PFDs in any individual woman’s health history.

Future studies may investigate which exact biological and environmental factors play roles that may place certain women at higher risk for severe PFD and UI (Nygaard, 2007). Additional studies regarding relationships between the maternal bony pelvis and obstetrical injuries may also provide further insight into PF issues. Studies that examine modifications and improvements in labor and birth procedures (including management of the second stage of labor, positional changes and perineal management during delivery, use of episiotomy, instrumental/operative vaginal deliveries, and cesarean section) to impart the least damage all childbearing women. Studies designed to evaluate the natural history of incontinence over a woman’s lifetime have the potential to provide strong scientific evidence for a cause-and-effect relationship of an etiology, as well as a better understanding of the natural occurrence and progression of UI and PF issues (Handa et al., 2003; McFarlin, 2004).

**References**


the bony pelvis of women with and without pelvic floor disorders. *Obstetrics and Gynecology*, 102(6), 1283-1290.


