CHAPTER 16

Obstetrics and Pelvic Floor Disorders

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Introduction

There are many physiologic adaptations a woman undergoes during the course of a routine pregnancy. In most obstetric textbooks, much attention is focused on the physiologic changes in the cardiovascular, hematologic, renal, and immunologic systems, and anatomic changes in the uterus and the growing fetus and placenta. There are also anatomic and physiologic changes in the lower genitourinary tract and pelvic floor support. The subsequent process of both labor and delivery also impart significant changes, especially to the pelvic floor support.

To whom are these changes significant? Which woman will evidence the symptoms of pelvic floor disorders (PFDs), about which this textbook is dedicated? Nearly 100 years ago, (in his argument espousing more liberal use of forceps, it must be pointed out), DeLee wrote:

"Labor has been called, and still is believed by many to be, a normal function ... and yet it is a decidedly pathologic process. Everything, of course, depends on what we define as normal. If a woman falls on a pitchfork, and drives the handle through her perineum, we call that pathologic-abnormal, but if a large baby is driven through the pelvic floor, we say that it is natural, and therefore normal."

This chapter discusses the relevant changes that occur with pregnancy and following delivery, and how they may affect the development of PFDs.

Alterations of Pelvic Floor Anatomy and Physiology in Pregnancy

Pelvic Floor Support

As pointed out in Chapter 2, interactions between the pelvic floor muscles and connective tissue attachments provide the support to the pelvic organs. Probably as a result of hormonal changes, there are significant adaptations to prepare the pelvic floor and birth passage for the events of labor and delivery. Clinical evidence of this has been observed using the standardized pelvic organ prolapse (POPQ) system. O'Boyle et al. (2002) found that nulliparous pregnant women were more likely to have mild pelvic organ prolapse (POP) than their nulligravid counterparts.

Consistent with the clinical changes noted earlier, molecular and histologic studies of vaginal tissues have demonstrated increased amounts of extracellular matrix material.
in the fibromuscular layers. Collagen becomes less densely packed, and smooth muscle cells revert to noncontractile phenotypes (Daucher et al., 2007). The result of these changes is increased distensibility of the vagina, presumably to allow for genital stretching during parturition (Alperin et al., 2010).

The advent of genetically engineered knockout mouse models (LOXL-1 and FBN-5) have provided more insight for changes in elastin homeostasis during pregnancy, and the potential that the mechanisms responsible for the regeneration of the elastic properties of the vagina may be deranged in those animals who develop prolapse as well as other PFD postpartum.

### Urinary Tract in Pregnancy

The kidneys and urinary tract play a major role in maternal adaptation to pregnancy. Consequently, observed differences in function cannot be judged by nonpregnant standards. The renal system increases in size and capacity during pregnancy. Anatomic changes involving the urinary tract begin during the first trimester of pregnancy and can persist up to 16 weeks postpartum.

The most striking anatomic change in the urinary tract is dilation of the ureters (Fig. 16.1). Bilateral dilation of the calyces, renal pelvis, and ureters can be seen early during the first trimester and is present in 90% of women during the late third trimester or early puerperium. The changes are usually more prominent on the right and may persist for 3 to 4 months. In 11% of women, ureteral dilation persists indefinitely (Bailey et al., 1971). In addition, there is reduced ureteral peristalsis compared with the nonpregnant state.

Vesicoureteral reflux is a sporadic, transient occurrence during pregnancy and has been demonstrated radiologically in 3.5% of pregnant women. The enlarging uterus displaces the ureters laterally, and the intravesical ureters are shortened and enter the bladder perpendicularly rather than obliquely. Consequently, the ureterovesical junction is less efficient in preventing reflux. This increased incidence of reflux may explain the high incidence of pyelonephritis during pregnancy; however, this association has not been confirmed. The transitory nature of vesicoureteral reflux, and the necessary exposure to X-rays for study purposes, hinder adequate evaluation of the problem. Nevertheless, vesicoureteral reflux probably plays only a small role in symptomatic or asymptomatic urinary tract infection (UTI).

Renal blood flow is usually assessed by r-aminohippurate clearance, which measures effective renal plasma flow (ERPF). ERPF increases significantly during pregnancy. It reaches a peak increment during the mid trimester of 50% to 85% and then shows a small decline during the third trimester. ERPF and glomerular filtration rate (GFR) in pregnancy are affected markedly by posture, and are maximal when the pregnant woman lies on her side. Normal pregnancy is associated with plasma volume expansion and an increase in the GFR of 40% to 65% (measured by insulin clearance) and a decrease in GFR of approximately 15% to 20% late during the third trimester. The mechanisms responsible for the increase in GFR, plasma volume, and renal plasma flow rate are unknown.

Tests of renal function during pregnancy must be interpreted in relation to the changes in plasma volume, glomerular filtration, and tubular reabsorption that normally occur with advancing gestation. Many of the commonly used tests of function yield lower results in pregnancy than in the nonpregnant state. Consequently, values that may be regarded as normal during the nonpregnant state may well indicate renal dysfunction in pregnancy.

Uric acid, blood urea nitrogen (BUN), and serum creatinine levels are crude indices of renal function. In pregnancy, plasma uric acid usually decreases by 25% beginning during the first trimester, and increases during the third trimester. Upper normal limits of plasma uric acid levels are 5 to 5.5 mg/dL during pregnancy. Levels are influenced by race, multiple gestation, and time of day sampled, with higher levels in the morning. An indicator of renal filtration, the BUN normally decreases from nonpregnant levels of 12 mg/dL (4.3 mmol/L) to 9 mg/dL (3.2 mmol/L), and plasma creatinine levels decline from a nonpregnant mean value of 0.7 mg/dL (62 mmol/L) to 0.5 mg/dL (44 mmol/L). If the plasma creatinine level exceeds 0.9 mg/dL, or if the BUN is greater than 14 mg/dL at any stage during pregnancy, renal dysfunction should be suspected and more detailed investigation should be performed.

Twenty-four-hour creatinine clearance is the best clinical measurement of GFR. By week 8 of pregnancy, the creatinine clearance rate normally increases by 45% and remains elevated during the second trimester. During the final weeks of pregnancy, creatinine clearance usually declines to near nonpregnant levels.

Urinalysis is essentially unchanged during pregnancy. However, many variables can affect the results. Normal
kidneys should be able to concentrate urine to a specific gravity of 1.026 or more, and to dilute urine to a value less than 1.005. During pregnancy, posture affects urine concentration and specific gravity.

In addition to the ERPF, the capacity of the urinary tract increases during pregnancy. Bladder volume during pregnancy increases to 450 to 650 mL, compared with 400 mL in nonpregnant control subjects (Table 16.1). The hydronephrotic ureters can hold as much as 200 mL extra urine; however, no changes appear in the contraction patterns on retrograde-cystometry. Depending on maternal position, uterine size, and position of the fetus, the functional volume of the bladder and ureters is dynamic during the third trimester. This increased functional volume, coupled with high urine flows (especially with fluid mobilization at night), causes polyuria and nocturia in most pregnant women.

The etiology of ureteral and bladder dilation generates much discussion. Sharp termination of the ureteral dilatation at the pelvic brim seen on intravenous pyelogram (IVP) suggests an obstruction. When a woman is upright or supine, as during the filming of an IVP, the pregnant uterus compresses the ureter against the pelvic rim and its overlying iliac vessels. On the left side, the ureter is somewhat protected by the iliac arteries and sigmoid colon and, as a result, is usually less-dilated than the right ureter. Although mechanical obstruction plays a major role in ureteral dilation during pregnancy, the relative infrequency of ureteral obstruction by large ovarian tumors or fibroids in nonpregnant women suggests additional factors. In addition, high urine production, as occurs in diabetes insipidus or pregnancy, is also associated with urinary tract dilation.

In the past, the elevated progesterone levels that accompany pregnancy were thought to cause smooth-muscle relaxation and subsequent hypotonicity and hypomotility of the ureter—defects that would contribute to ureteral dilation. Contrary to the latter observation, the large doses of synthetic progesterone used in cancer chemotherapy do not cause ureteral dilatation. Measurements of ureteral tone during pregnancy reveal an increase in ureteral tone and no decrease in frequency or amplitude of ureteral contractions. Histologic study of the ureters of pregnant animals reveals smooth-muscle hypertrophy and hyperplasia of the connective tissue. Thus, progesterone probably plays a small role in ureteral dilation during pregnancy.

**Urinary Tract Conditions during Pregnancy**

**Urinary Retention**

Occasionally, the enlarging uterus completely obstructs both ureters and causes azotemia. Patients usually present during the early second trimester with a retroverted uterus (perhaps incarcerated), flank pain, and minimal signs of infection. The differential diagnosis includes pyelonephritis, renal calculi, or papillary necrosis. Serum creatinine is elevated (3.8-11.6 mg/dL), but urinary sediment does not indicate intrinsic renal disease or prerenal azotemia. The diagnosis is confirmed by IVP or renal ultrasound. In this case, treatment requires bimanual manipulation of the uterus out of the sacral hollow. Some have recommended the use of a Smith-Hodge pessary to maintain anteversion of the uterus. Other risk factors for obstruction include previous urologic surgery, unilateral absence of a kidney, polyhydramnios, multiple gestation, and ovarian or uterine neoplasia.

Occasionally, third-trimester retention can develop. Ultimately, delivery relieves the obstruction, and postpartum recovery is complete. In cases remote from term, fetal risk from preterm delivery outweighs the risks of urologic management. Conservative management for 12 to 24 hours is warranted before more aggressive therapy is initiated, including amniocentesis (in cases with polyhydramnios), cystoscopically-placed ureteral stents, or percutaneous nephrostomy under ultrasound guidance.

**Urinary Incontinence**

As shown in Table 16.1, the symptoms of frequency, nocturia, and incontinence all increase during in the first trimester, and increase during the course of the pregnancy. Part of this change is a result of the increase in glomerular flow described earlier. Stress incontinence is more common than urge-incontinence or overactive-bladder symptoms, but mixed symptoms are common. For those who do have incontinence rather than frequency alone, their quality of life is affected significantly more. The few urodynamic

<table>
<thead>
<tr>
<th>Table 16.1</th>
<th>Urologic Symptoms and Measurements in Pregnancy</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>First Trimester</td>
</tr>
<tr>
<td><strong>Symptom-Frequency</strong></td>
<td></td>
</tr>
<tr>
<td>Day ≥7</td>
<td>45%</td>
</tr>
<tr>
<td>Night ≥2</td>
<td>22%</td>
</tr>
<tr>
<td><strong>Incontinence</strong></td>
<td></td>
</tr>
<tr>
<td>Stress</td>
<td>30%</td>
</tr>
<tr>
<td>Urge</td>
<td>4%</td>
</tr>
<tr>
<td>Hesitancy</td>
<td>24%</td>
</tr>
<tr>
<td><strong>Measurement</strong></td>
<td></td>
</tr>
<tr>
<td>Urine output (mL)</td>
<td>1917</td>
</tr>
<tr>
<td>Bladder capacity (mL)</td>
<td>410</td>
</tr>
<tr>
<td>Functional urethral length (mm)</td>
<td>30.3 ± 4.6</td>
</tr>
<tr>
<td>Bladder pressure (cm H₂O)</td>
<td>9 ± 3</td>
</tr>
<tr>
<td>Closure pressure (cm H₂O)</td>
<td>61 ± 14</td>
</tr>
</tbody>
</table>

Asymptomatic Bacteriuria

The cohort of women with chronic, episodic asymptomatic bacteriuria is identified by routine screening of urine cultures at the first prenatal visit. The prevalence of asymptomatic bacteriuria (two or more cultures at ≥10^5 cfu/mL) is increased by prior renal or urinary tract disease, diabetes, sickle cell trait or disease, poor hygiene, high parity, increased age, and lower socioeconomic status. The overall prevalence varies between 1.9% and 11.8%, with the lowest prevalence in primiparous patients of the upper socioeconomic class and the highest among indigent multiparas. Although most women with asymptomatic bacteriuria are identified shortly after entering prenatal care, approximately 1% to 2% acquire bacteriuria later during pregnancy.

Because of the more significant concerns of worsening obstetric outcomes associated with an escalation to acute cystitis or pyelonephritis and because the positive predictive values of culture-independent tests (i.e., dipstick urinalysis) drop precipitously with low prevalence, they should not be used for diagnosis in the pregnant population. On the other hand, the negative predictive value is 98% or more and with any of these tests. In a low-risk population, urine testing for leukocyte esterase and nitrite on a clean-catch, first-void midstream specimen can supplant urine culture. In high-risk groups (Box 16.1), a culture should be obtained each trimester.

The association between preterm birth and asymptomatic bacteriuria was first identified by Elder, Kass, and others at Boston City Hospital between 1955 and 1960. This initial study reported that 32 of 179 (17.8%) patients with bacteriuria delivered low-birth weight (LBW) infants, whereas 88 of 1000 (8.8%) patients without bacteriuria delivered LBW infants. Romero et al. (1989) reviewed 19 studies that related bacteriuria to LBW infants. In these studies, 3610 pregnant women with bacteriuria delivered 400 (11%; range, 4.4%–23%) LBW infants. In these same studies, 31,277 women without bacteriuria delivered 2725 (8.7%; range, 3%–13.5%) LBW infants. In 1989, Romero et al. performed a meta-analysis on the relationship between asymptomatic bacteriuria and LBW. Eight randomized clinical trials of antibiotic therapy showed a significant reduction in the frequency of LBW after antibiotic therapy (typical relative risk (RR) of 0.56, with a 95% confidence interval (CI) of 0.43-0.73). It is unclear whether the benefit from antibiotics results from a reduction in asymptomatic or symptomatic pyelonephritis, or from beneficial changes in abnormal genital tract flora, which is associated with LBW.

The association between asymptomatic bacteriuria and other adverse pregnancy outcomes (hypertension, anemia, chronic renal disease, and fetal neuropathology) is controversial, being both supported and refuted by different cohort studies. Between 25% and 50% of pregnant women with asymptomatic bacteriuria have evidence of asymptomatic renal involvement, and these women are twice as likely to relapse within 2 weeks after therapy as women with bladder bacteriuria alone. Asymptomatic renal infection has been associated with decreased creatinine clearance, intrauterine growth retardation, and maternal hypertension. On the other hand, Gilstrap et al. (1981b) failed to note a difference in outcomes between asymptomatic women with and without renal infection, as defined by fluorescent antibody testing (Table 16.2).

A variety of antimicrobial agents and treatment regimens have been used to treat asymptomatic bacteriuria during pregnancy. Most community-acquired pathogens associated with asymptomatic bacteriuria during pregnancy are sensitive to sulfisoxazole (sulfonpyrazone, 1–3 g four times a day (qid) for 7–10 days), nitrofurantoin (100 mg twice daily for 7–10 days), or cephalosporins (cephalexin, 500 mg qid for 7–10 days). Ampicillin (500 mg qid for 7–10 days) is a time-honored, safe, and inexpensive therapy; however, there are a growing number of resistant Escherichia coli strains (Table 16.3). Other antibiotics should be used. A Cochrane Review

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**Box 16.1—Conditions That Place Patients at High Risk for Urinary Tract Infections during Pregnancy**

- Diabetes
- Sickle cell disease or trait
- Urinary tract abnormalities
- Müllerian duct abnormalities
- Renal disease
- Urolithiasis
- Hypertensive diseases
- Chronic analgesic use
- Genitourinary group B Streptococcus
- History of urinary tract infections
- Severe ureteral reflux
- Urinary infections as a child younger than 4 years old

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**Table 16.2 Incidence of Urinary Tract Infection during Pregnancy**

<table>
<thead>
<tr>
<th>Infection</th>
<th>Incidence (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asymptomatic bacteriuria</td>
<td>2–14</td>
</tr>
<tr>
<td>Acute cystitis</td>
<td>1–4</td>
</tr>
<tr>
<td>Acute pyelonephritis</td>
<td>1–2</td>
</tr>
</tbody>
</table>

**Table 16.3 Microbiology of Urinary Tract Infections in Pregnancy**

<table>
<thead>
<tr>
<th>Organism</th>
<th>Percentage</th>
</tr>
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<tbody>
<tr>
<td><em>Escherichia coli</em></td>
<td>60–80</td>
</tr>
<tr>
<td>Klebsiella pneumoniae-Intermedius</td>
<td>3–5</td>
</tr>
<tr>
<td>Proteus sp.</td>
<td>1–5</td>
</tr>
<tr>
<td><em>Streptococcus faecalis</em></td>
<td>1–4</td>
</tr>
<tr>
<td>Group B Streptococcus</td>
<td>4–8</td>
</tr>
<tr>
<td>Staphylococcus saprophyticus</td>
<td>1–3</td>
</tr>
</tbody>
</table>
supported the extended treatment duration rather than single-dose regimens (Widmer et al., 2011).

Between 20% and 30% of women who have bacteriuria during pregnancy will have bacteriuria on long-term follow-up cultures when not pregnant. Radiologic examination at follow-up of women who had bacteriuria during pregnancy revealed abnormalities in 316 of 777 women (41%; range, 5%-75%). Chronic pyelonephritis was the most common radiologic diagnosis (47% of abnormalities). The incidence of bacteriuria during first pregnancies was significantly greater in women with (47%) than without (27%) renal scarring from childhood urinary infections. Similar control subjects who had not had childhood urinary infections had an incidence of 2%.

**Acute Cystitis**

Acute cystitis occurs in 1% to 4% of pregnancies. The reported frequency is only minimally greater than the frequency of cystitis in sexually active nonpregnant women. Unfortunately, the diagnosis is more difficult to make during pregnancy. Most pregnant women have urgency, frequency, or suprapubic discomfort. Suprapubic discomfort in pregnancy often results from pressure on the presenting fetal part or early labor. Nevertheless, suprapubic discomfort from cystitis is unique, and most women with a history of acute cystitis can discriminate accurately between cystitis and pregnancy-related discomfort. The most reliable findings are dysuria and hematuria. Acute dysuria may also result from labial or perivaginal irritation from vaginitis, vulvitis, herpes simplex, condylomata acuminatum, or genital ulcers. Because of the separate pregnancy risks encumbered with these factors, an inspection of the vulva and vagina is warranted in patients with acute cystitis during pregnancy.

Preterm labor and threatening second-trimester loss often present with signs and symptoms similar to those of acute cystitis. As the lower uterine segment expands and the presenting fetal part descends, hesitancy, urgency, frequency, and suprapubic discomfort occur. A bloody vaginal discharge may contaminate and confuse urine testing, and may lead to misdiagnosis of UTI. Pelvic examination is warranted in patients presenting with signs and symptoms of lower UTI to rule out preterm labor.

The pathophysiology of acute cystitis is more similar to that of asymptomatic bacteriuria than pyelonephritis. Acute cystitis has sociodemographic and behavioral risk factors similar to those of asymptomatic bacteriuria: Enterobacteriaceae, especially *E. coli*, are the most common uropathogens. Acute cystitis is associated with a high prevalence of uropathogens in the periurethral flora. *E. coli* serotypes are associated with more epithelial cell adherence, hence virulence, than fecal strains. Antibody-coated bacteria, indicative of renal infection, are present in only 5% of acute cystitis, compared with 45% for asymptomatic bacteriuria and 65% for acute pyelonephritis. This difference may result from earlier identification and treatment of the patient with these latter conditions because of the intense discomfort that accompanies cystitis.

Treatment of acute cystitis is similar to that of asymptomatic bacteriuria: nitrofurantoin, a cephalosporin, or a sulfonamide. Because these patients are symptomatic, therapy is initiated as soon as a midstream, clean-catch urine culture has been obtained. A test-of-cure culture is obtained within 2 weeks after therapy is complete. Between 10% and 20% have a positive test-of-cure culture, representing a relapse. These women should be retreated with another antibiotic, as determined by bacterial sensitivities. After retreatment, these patients should be placed on suppressive antibiotic therapy. Without suppressive therapy, an additional 20% to 30% of women develop another UTI—a reinfection—during the remainder of their pregnancies and puerperium. Because of the risk of recurrence, patients with cystitis should be monitored intensively, with a urine screen biweekly for nitrite and leukocyte esterase.

**Acute Pyelonephritis**

Acute pyelonephritis is the most common serious medical complication of pregnancy. The modern incidence of pyelonephritis is 1% to 2%. Often, these patients present for prenatal care late during pregnancy with the signs and symptoms of pyelonephritis. Only 40% to 67% of cases of pyelonephritis occur in patients with a known history of asymptomatic bacteriuria. Three-fourths of women with pyelonephritis present in the antepartum period, 5% to 10% during labor, and 15% to 20% postpartum. Antepartum pyelonephritis occurs mainly after the first trimester: 10% to 20% during the first trimester, 45% to 70% during the second trimester, and 8% to 45% during the third trimester. The predominance of pyelonephritis during late pregnancy and the puerperium likely relates to the partial obstruction caused by the growing uterus and trauma or interventions at birth.

The diagnosis of acute pyelonephritis is based on clinical presentation: a temperature of 38°C or more, costovertebral angle tenderness, and either bacteriuria or pyuria. Enterobacteriaceae cause a majority of the cases of pyelonephritis: *E. coli*, 70% to 80%; *Klebsiella-enterobacter* spp., 5%; *Proteus* sp., 2% to 4%; and gram-positive organisms inducing group B *Streptococcus*, 10%. Blood cultures are positive in 17% of cases. Infection of the kidney may have an effect on function. Two percent have a serum creatinine greater than 1.1 mg/dL. This dysfunction is a direct result of endotoxic injury to both kidneys. After appropriate antibiotic treatment, renal function returns to normal by 3 to 8 weeks.

Endotoxins produced by Enterobacteriaceae have adverse consequences on multiple organ systems as well as the kidneys. The injuries include thermoregulatory instability (fever and chills), destruction of blood cells (leukocytopenia, thrombocytopenia, anemia), hypercoagulability (disseminated intravascular coagulation), endothelial injury (adult respiratory distress syndrome), cardiomyopathy (pulmonary edema), and myometrial irritability (preterm labor).

Overt septic shock or adult respiratory distress syndrome occurs in 1% to 2% of pregnant women with acute pyelonephritis. Clinical clues to the development of these life-threatening complications are leukocytopenia (<6000 cells/mL²), hypothermia (<35°C), elevated respiratory rate, and widened pulse pressure. During the late stages, hypothermia, mental confusion, and symptomatic hyperstimulation of the sympathetic nervous system (cold, clammy
extremities) herald a scenario that often leads to maternal or fetal death.

Most pregnant women with pyelonephritis (and all pregnant women greater than 24 weeks’ gestation) should be hospitalized because of the additional fetal and maternal risks of acute pyelonephritis in pregnancy. Appropriate broad-spectrum antibiotics should be initiated as soon as possible after urine and blood cultures are obtained. Antibiotic therapy should be continued until the patient is afebrile ($\leq 37.5^\circ$C) for more than 24 h. The patient should finish a 14-day course of antibiotics with oral medication. A test-of-cure urine culture should be performed 2 weeks after therapy. Re-infection is common in these patients; 20% have asymptomatic bacteriuria and 23% have recurrent pyelonephritis. Frequent surveillance (nitrite leukocyte esterase testing biweekly) or suppressive antibiotic therapy (nitrofurantoin, 100 mg every night at bedtime) is warranted. With either regimen, the risk of recurrent pyelonephritis is less than 10%.

Because many patients are dehydrated as a result of nausea and vomiting, careful rehydration is started. The degree of endothelial damage in the lungs may not be apparent, so careful attention to fluid intake and output, and vital signs, and especially respiratory rate, is imperative.

Endotoxins stimulate cytokine and prostaglandin production by decidual macrophages and fetal membranes. The ensuing preterm contractions raise concern for preterm birth. In the past, preterm labor and delivery were reported to be a common finding in women with pyelonephritis; however, data in 440 cases reported by Hill et al. (2005) found a rate of only 5%.

The differential diagnosis in patients with persistent fever and costovertebral angle tenderness at 72 h of therapy includes a resistant organism, urolithiasis, renal abscess, complete ureteral obstruction, or another source of infection (e.g., appendicitis or intra-amniotic infection). A radiologic evaluation of the urinary tract is warranted after re-examination of the patient and review of culture and sensitivity reports. Many radiologists have undue concern regarding the fetal dangers of IVPs during pregnancy and advocate renal ultrasound. A renal ultrasound is useful for evaluating renal abscess, but not for evaluating function or ureteral abnormalities—the more common issues associated with antibiotic failure. A “one-shot” IVP (no plain film and one 20-min film) is appropriate.

**Urolithiasis**

Urolithiasis occurs in 0.03% to 0.9% of pregnancies, usually in the last two trimesters. Between 20% and 40% of women with urolithiasis during pregnancy have a history of stone disease. Although pregnancy does not appear to increase the risk of urolithiasis during any 9-month period in susceptible persons, recurrent urolithiasis may indicate primary renal disease (medullary sponge kidney), transport diseases (renal tubular acidosis), or metabolic diseases (hyperparathyroidism). The fetal or maternal risk may reflect these systemic diseases rather than urolithiasis alone.

Most stones (70%) pass during the second or third trimester, with equal distribution between the right and left sides. The presentation is more obscure during pregnancy; the most common signs being severe flank pain (80%) with radiation to the groin or lower abdomen, nausea, and vomiting. Renal colic is less common after the first trimester because of ureteral dilation. Likewise, gross hematuria is less common (23%), but microscopic hematuria occurs in 60% to 90% of patients. Bacteriuria may be present in 80%.

The differential diagnosis includes premature labor, appendicitis, and, most commonly, pyelonephritis. Premature labor is diagnosed by contractions and cervical dilation. Urolithiasis is more likely than appendicitis when the patient has no fever, the abdominal pain is not localized to the right lower quadrant, and no peritoneal signs are present. The most difficult differentiation is between pyelonephritis and urolithiasis. Indeed, they may coexist.

**IVP** is the diagnostic technique of choice. In pregnancy, the protocol and frequency of IVP vary. The IVP should be limited to a 20-min film and, if there is delayed excretion, a 60-min film. Fluoroscopy is used only in very exceptional circumstances. An IVP is indicated when the patient has renal colic and gross hematuria, persistent fever or a positive culture after 48 h of parenteral antibiotic therapy, persistent nausea and vomiting after 48 h of conservative therapy, or evidence of a complete obstruction (e.g., increasing levels of BUN and serum creatinine).

Transabdominal or transvaginal ultrasound often is the first diagnostic choice of radiologists. Their concern is the 0.4 to 1 rad of radiation the fetus receives with a limited IVP. There is concern that doses this low may double childhood cancer rates. Although ultrasound is a good diagnostic tool for renal abnormalities and ureteral dilation, its sensitivity is 34%, with an 86% specificity for the detection of an abnormality in the presence of a stone in a symptomatic patient. In one study, renal ultrasound was used as the primary diagnostic test in 35 of 56 women, but calculi were detected in only 21 (60%) of these women (Butler et al., 2000). IVP is clearly more efficacious for diagnosis of distal stones.

Urolithiasis in pregnancy is treated conservatively with bed rest, hydration, and analgesics. Seventy percent of patients pass the stone spontaneously. Urolithiasis during pregnancy does not increase the likelihood of abortion, prematurity, or hypertension, but the incidence of symptomatic urinary tract disease is greater in pregnancies complicated by a history of urolithiasis (20%–65%), potentially leading to an increased rate of preterm rupture of membranes. Parenteral antibiotics (cefazolin 2 g IV q8h) are added to conservative management when infection is likely.

When conservative management is unsuccessful (complete obstruction, persistent pain, or sepsis), surgical intervention is indicated. The choice of procedure depends on the size and location of the stone. The usual procedures include basket extraction or retrograde stent placement at cystoscopy. Percutaneous nephrostomy under ultrasound guidance has also been used as a temporizing procedure. Rarely and with considerably more fetal and maternal morbidity, ureterolithotomy, pyelolithotomy/pyelotomy, or partial nephrectomy can be performed.

**Previous Urologic Surgery**

An increasing number of women are becoming pregnant who were born with urinary tract abnormalities that were corrected surgically. These operations include urinary diversion
procedures—(ileal conduit and ureterosigmoidostomy), augmentation cystoplasty, and ureteral reimplantation for vesicoureteral reflux. The changes in pelvic anatomy caused by the enlarging uterus create the potential for infection, obstruction, and trauma at cesarean section.

Pregnancy in patients with a urinary diversion is complicated by premature delivery, 20% to 50%; symptomatic UTIs, 15%; urinary obstruction, 10%; and intestinal obstruction, 10%. Cesarean delivery should be reserved for obstetric indications. Although less commonly performed, a ureterosigmoidostomy may be an indication to schedule a cesarean delivery to preserve the integrity of the anal sphincter.

During the past 20 years, the treatment of patients with abnormal urinary tracts has changed from cutaneous diversion to patients who have had a continent internal diversion—and augmentation cystoplasty, they may have also undergone vesical neck reconstruction or artificial sphincter placement. This may place the patient at risk for the development of incontinence after vaginal delivery. Hill et al. (1990) reviewed 15 pregnancies in 15 women after augmentation cystoplasty. Eight of thirteen were continent before, during, and after pregnancy. One patient who was continent before delivery became incontinent after vaginal delivery. Four patients became incontinent during the last trimester, but regained continence postpartum. The pregnancies were complicated by UTIs (60%), preterm labor (20%), and urinary obstruction (7%). Five cesarean deliveries were performed, three electively for vesical neck or artificial sphincter construction. One cesarean operation was complicated by extensive anterior uterine adhesions. Although stretching of the mesentery by the enlarging uterus has the potential risk of vascular compromise, this complication was not seen in the 15 patients.

Ureteral reimplantation has been performed routinely for severe primary vesicoureteral reflux for many years. Austenfeld and Snow (1988) reviewed 64 pregnancies in 34 women after ureteroneocystostomy for primary reflux. The overall infection rate before pregnancy was 48%. During pregnancy, 57% experienced a UTI. Pyelonephritis was more common during pregnancy (17%) than before pregnancy (4%). Of the 64 pregnancies, eight were lost between 9 weeks and 21 weeks, and six were associated with a UTI. The authors did not report the route of delivery and the difficulty of cesarean section.

The latter reviews of pregnancies in women with urinary tract surgery suggest the following obstetric management: close monitoring for preterm labor (patient education, frequent office visits, frequent pelvic examinations), suppressive antibiotic therapy (nitrofurantoin 100 mg every night at bedtime), monthly BUN and serum creatinine evaluation, vigilance for ureteral obstruction, vaginal delivery except for obstetric indications and patients who have undergone urinary diversion to the sigmoid and bladder neck/sphincter surgery, and urologic consultation at cesarean section for patients with a history of complex urologic surgery, especially augmentation cystoplasty.

Impact of Childbirth on Anatomy and Physiology

You may likely have witnessed countless times the miracle of childbirth and you may also have likely considered the “Ps”—that is, how the power pushes the passenger through the passage. In a classic review, Power (1946) described the mechanism by which the fetus negotiates the birth canal and is expelled through the pelvic diaphragm as follows:

As the flexed fetal head strikes the pelvic floor, the levator ani muscle segments are funneled from behind and forward. The ischiococcygeus muscle is the first to receive the impact, but the head is often preceded by a dilating wedge of amniotic fluid and membranes that transfers most of the pressure onto the front of the pubococcygeus muscle. The anococcygeal raphe is pushed down until it becomes vertical. The ischiococcygeus assumes a vertical plane and acts as a deflecting surface for the descending head, which is deflected downward and forward onto the iliococcygeus.

After the resistance of the ischiococcygeus is overcome, the head is shunted onto the pubococcygeus segment, which is stretched anteroposteriorly and peripherally. The perineal body is pushed downward as the head is propelled along the axis of the pelvic outlet. The rectovaginal septum fibers are stretched peripherally and longitudinally and often torn. As the outlet muscles—the bulbocavernosus, ischiocavernosus, transverse perinei, and periurethral muscles—are dilated, they are converted into a short muscular tube along the axis of the pelvic outlet. Finally, as the biparietal diameter of the fetal head reaches the transverse diameter of the pelvic outlet, the uterovaginal canal is converted into one continuous hiatus. The lateral ligaments of the cervix uteri (endopelvic fascia) are flattened peripherally and stretched vertically. The vagina is dilated spherically, and the pelvic diaphragm is changed from an oblique to a vertical plane.

Much of Power’s description was based on his experience and conjecture. During the past 15 years, advanced imaging and computer modeling have added an improved, but by no means complete, understanding of the changes that occur after labor and delivery (Fig 16.2).

Muscular Injury

Lien et al. (2004) used sophisticated imaging and engineering techniques to develop a biomechanical model to describe changes in levator ani muscles as the fetal head descends through the vagina (Fig. 16.3). The medial pubococcygeus muscle reached an impressive stretch ratio (defined as tissue length under stretch/original tissue length) of 3.26. According to the authors, this exceeds the greatest stretch ratio (1.5) seen in passive striated muscle of nonpregnant women by 217%. Increasing the fetal head diameter by 9% increased medial pubococcygeus stretch by the same proportion. This model suggests that the medial muscles of the levator ani complex have the greatest risk for injury of all the levator ani muscles during the second stage of labor. This supposition is supported by magnetic resonance imaging (Fig. 16.4) and three-dimensional/four-dimensional ultrasound (Fig. 16.5) studies that reveal abnormalities in this area in 20% to 36% of primiparous women after vaginal delivery (DeLancey et al., 2003; Dietz and Lanzarone, 2005).

This evidence, including the observations so well made by Power, is not a surprising consequence of pregnancy and delivery. But why do some women go on to have prolapse or incontinence whereas others are asymptomatic? It has been demonstrated recently that women with PFDs are more likely to have the levator ani injuries shown to be acquired after delivery—certainly a very strong piece of evidence against injuring this vital structure (Delancey et al., 2007; Dietz, 2007).

Some of the answers about why some women develop PFDs and others do not may be also provided by lessons learned from rodent knockout models. Several investigators have demonstrated that certain strains of mice are deficient in proteins responsible for collagen and elastin synthesis and homeostasis. Some of these strains will develop prolapse spontaneously, such as in the fibulin-5 knockout mouse (Wieslander et al., 2006). However, others, such as the LOXL-1 knockout mouse (Liu et al., 2006), often require a traumatic instigating event such as vaginal delivery to demonstrate the phenotype of prolapse. A defect in connective tissue homeostasis can worsen tissue damage that results from injury sustained during delivery resulting from an inability to repair postpartum. The requirement for effective repair and regeneration is likely applicable to the muscular and nervous system injuries that follow pregnancy and delivery, which generate a significant amount of interest in using stem cells to aid in that regeneration (Pathi et al., 2012; White et al., 2010).

Injury to Urinary Tract during Delivery

Injury to the urethra or bladder trigone from prolonged, obstructed labor or difficult operative deliveries is rare in the developed world, but is still quite common in sub-Saharan Africa, where it remains a scourge. On the other hand, the dramatic increase in the United States in cesarean deliveries from 5% to greater than 30% during the past 30 years has increased the rates of bladder dome and ureteral injury. The reported prevalence of injury to the bladder is 0.19% of primary and 0.6% of repeat cesarean deliveries. Most bladder injuries are associated with postsurgical (cesarean) adhesions between the bladder and the lower uterine segment. The risk of bladder injury is increased among patients with four or more uterine incisions (1.5%) and cesarean hysterectomy (1.7%).

Ureteral injury occurs in 0.09% to 0.6% of cesarean operations and usually occurs in association with late
second-stage dystocia; deep uterine, cervical, or vaginal lacerations; or cesarean hysterectomy. Two-thirds of urinary tract injuries are identified at the time of surgery. The technique and management of bladder and ureteral injury repair are described in Chapter 29.

**Postpartum Urinary Retention**

Postpartum urinary retention occurs in 1.5% to 14% (Yip et al., 2004) of deliveries, depending on the definition. Risk factors include nulliparity, long labor, operative vaginal delivery, and the use of regional anesthesia (Yip et al., 2005).
The inability to void spontaneously within 6 h after delivery deserves careful attention, such as considering an indwelling catheter or intermittent catheterization for 12 to 48 h. However, the question regarding whether to screen for elevated postvoid residuals after delivery remains unanswered, in part because of the challenges of defining abnormal residual volumes (50-200 mL) and determining a lasting effect in women who void spontaneously, but incompletely, during the early postpartum period.

**Injury to Anal Canal**

No documented cases are known of sonographically detected anal sphincter disruption following cesarean delivery. However, cesarean delivery after labor onset is not entirely protective against postpartum fecal incontinence resulting from some other mechanism, potentially pelvic nerve injury.

Injury to the anal sphincter during childbirth likely occurs either as a result of direct disruption of the muscles or because of injury to the pudendal nerves. The quoted incidence of sphincter laceration noted at the time of vaginal delivery ranges from 1% to 24%. The average incidence ranges from 6.5% (0.4%-23.9%) in women with midline episiotomy, 1.3% (0.5%-2.0%) in women with mediolateral episiotomy, and 1.4% (0%-6.4%) in women without episiotomy.

Because the perineal body anchors not only the superficial perineal space, but also the deeper perineal space and the anorectum itself (Woodman and Grady, 2002), it also serves as a barrier between the vagina and rectum. Perineal body length has been reported to be approximately 3.8 cm–4.6 cm during the third trimester (Dua et al., 2009; Rizk and Thomas 2000), and was demonstrated by O’Boyle that the length of the perineal body may actually change between early and late pregnancy in an adaptive manner (O’Boyle et al., 2002). Confirming the logical supposition, shorter perineal body length has been shown to be associated with greater risk of anal sphincter injury at vaginal delivery, with one group demonstrating a 10-fold greater risk when the perineal length decreased to less than 2.5 cm (Deering et al., 2004).

Methods of reducing the incidence of perineal body trauma have included supporting the perineum manually during vaginal delivery as well as perineal massage. In a Cochrane Review, regular antenatal perineal massage was shown to be an effective intervention, reducing the likelihood of perineal laceration or episiotomy by 10% (Beckmann and Stock, 2013). There has not been a similar benefit shown for any of the PFDs (Beckmann and Garrett, 2006). In a German randomized trial at four centers, investigators used an inflatable silicone balloon connected to a hand pump called the EPI-No birth trainer, and found a significant increase in an intact perineum (37.4% versus 25.7%; \( P = 0.05 \)) (Ruckhäberle et al., 2009).

The plight of the perineal body, and its relationship not only to the anal sphincter but also to the levator ani complex has been questioned recently. The untested theory is that the perineal body is a “ fusible link” and it undergoes preferential stretching (and/or tearing) that protects against the extreme stretching believed to occur in the other pelvic floor constituents, such as the levator ani attachments to the pubic bone (Ashton-Miller and Delancey, 2009). In a potentially relevant study germane to that question, nulliparous women were randomized to use the EPI-No birth trainer or not. Although the intervention showed a 50% decrease in sonographically detected levator ani avulsions, the result was not statistically significant because the proportions of levator trauma were 6% versus 13% \( ( P = 0.19 ) \), rendering the sample size with too little power to demonstrate the difference (Shek et al., 2011).

Endoanal ultrasound performed during the first months postpartum reveals that as many as 35% of primiparous women and up to 44% of multiparous women have evidence of sphincter disruption. Other anal function studies, such as anorectal manometry and anorectal sensation testing, have also demonstrated that vaginal delivery has an effect on anal function. Chaliha et al. (2001) showed that, compared with before delivery, vaginal delivery was associated...
with decreased anal squeeze pressures and resting pressures, whereas no change in anal sensation was noted. Risk factors for both overt and occult sphincter injuries include forceps, prolonged second stage, large birth weight, midline episiotomy, and occipitoposterior positions. Table 16.4 describes the typical adjusted odds ratios for predictors of anal sphincter disruption on multivariate analyses. In addition, Richter et al. (2002) reported that vaginal birth after cesarean delivery was an independent risk factor for anal sphincter disruption compared with previous vaginal delivery (RR, 5.46; 95% CI, 3.69-8.08), after adjusting for primiparity, birth weight greater than 4000 g, forceps delivery, vacuum delivery, shoulder dystocia, and episiotomy.

Randomized trials have also supported the association between episiotomy and anal sphincter disruption. Harrison et al. (1984) randomized 181 primigravid women to routine mediolateral episiotomy or to restricted use of mediolateral episiotomy. The episiotomy group sustained rectal injury in 5 of 89 cases (5.6%) compared with no cases of rectal injury in the restricted group. Sleep et al. (1984) randomized 1000 women to liberal use of mediolateral episiotomy (51% of patients) or restricted mediolateral episiotomy (10% of patients). A liberal policy toward episiotomy resulted in significantly more maternal vaginal trauma and more suturing.

The association between operative delivery and perineal trauma has also been studied in a randomized fashion. Yancey et al. (1991) randomly assigned uncomplicated term gestations at 2+ station in occipitoposterior position to routine outlet forceps or to spontaneous delivery. Among patients delivered by outlet forceps, the incidence of third- or fourth-degree lacerations was 30 of 165 (18%) versus 12 of 168 (7%) in women who delivered spontaneously. Midline episiotomy and outlet forceps were the only factors associated significantly with rectal trauma on multivariate analysis.

Incidentally, defects that appear to be traumatic sphincter injuries on ultrasound may not always be the case. Three-dimensional imaging and anatomic studies combined with magnetic resonance imaging have shown that both the female and male anal sphincters have a variable natural gap occurring along its anterior length. The natural gap seen on ultrasound can be distinguished from a traumatic one by its anterior uniform hypoechogenicity, smooth and uniform edges, and symmetry.

### Neurologic Injury

Earlier studies have suggested that labor and childbirth, but not pregnancy, are major risk factors for neurologic injury. For example, in a study by Sultan et al. (1994), the pudendal nerve terminal motor latency (PNTML) was not increased in pregnant nonlaboring women compared with nonpregnant women. However, after vaginal delivery, Snooks et al. (1986) found that 42% of women had a prolonged PNTML, but this eventually recovered in most women by 2 months postpartum. Single-fiber electromyography (EMG) of the anal sphincter revealed an increased fiber density 2 months postpartum. These findings were most striking in multiparous women (suggesting a cumulative effect of vaginal delivery on the pelvic floor) and women delivered by forceps. No changes in these parameters were seen in women who had delivered by cesarean section. Five years after delivery, Snooks et al. (1990) reported there was manometric and neurophysiologic evidence of weakness because of partial denervation of the pelvic floor striated sphincter musculature, with pudendal neuropathy, which was more marked in those women with incontinence. In a prospective study by Allen et al. (1990), concentric-needle EMG was performed on the levator ani before delivery as well as 2 and 5 days after delivery. After vaginal delivery, the duration of the motor unit potential was increased, consistent with nerve damage. Eighty percent of the women showed evidence of denervation with subsequent reinnervation after vaginal delivery. After elective cesarean delivery, however, the motor unit potentials were unchanged. In a more recent study, 12 weeks after a first routine vaginal delivery, quantitative EMG parameters of the external anal sphincter demonstrated evidence of denervation/reinnervation for the inferior hemorrhoidal (pudendal) nerve; however, those undergoing cesarean delivery were not evaluated (Gregory et al., 2004).

Some investigations are questioning whether cesarean delivery is truly protective. Investigators have found the PNTML to be prolonged after a cesarean delivery performed in late labor (cervical dilation, 8 cm or greater), but not in women delivered by cesarean before they reached 8 cm dilation (Fynes et al., 1998; Allen et al., 1990). More recently, though, South et al., (2009) demonstrated that 30% of previously nulliparous women demonstrated EMG evidence of denervation of the levator ani muscles 6 weeks after delivery, including those who underwent cesarean delivery.

### Impact of Childbirth on Specific PFDs

As outlined earlier, significant anatomic and physiologic changes occur as a result of pregnancy and delivery. Most of these findings have been made in the belief they are related to the onset of PFDs, either immediately after delivery or years after. In the following sections, we highlight some of the evidence about how childbirth, and the method of childbirth, affect the risk of developing signs or symptoms of these disorders.

### Urinary Incontinence

In the large, community-based 2005 to 2006 National Health and Nutrition Examination Survey, 1961 women reported PFDs; 6.5% of nulliparous women had urinary incontinence, but nearly 24% of women who had delivered more than three children leaked. There was a clear positive relationship between parity and increased prevalence of urinary incontinence. Nulligravid pregnant women leak

### Table 16.4 Predictors of Anal Sphincter Disruption after Vaginal Delivery

<table>
<thead>
<tr>
<th>Risk Factors</th>
<th>Adjusted Odds Ratios*</th>
</tr>
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<tbody>
<tr>
<td>Midline episiotomy</td>
<td>4.9-16.5</td>
</tr>
<tr>
<td>Nulliparity</td>
<td>2.5-4.0</td>
</tr>
<tr>
<td>Operative delivery</td>
<td>2.5-3.5</td>
</tr>
<tr>
<td>Birth weight ≥4000 g</td>
<td>1.5-2.5</td>
</tr>
<tr>
<td>Occipitoposterior presentation</td>
<td>1.2-1.8</td>
</tr>
</tbody>
</table>

*Numbers represent the minimum and maximum adjusted odds ratios reported in the medical literature.
Conflict evidence exists about the role of other obstetric variables (besides vaginal delivery) on the development of SUI. Although some studies reported more SUI with prolonged second stage, increased head circumference, increased birth weight, epidural anesthesia, and oxytocin use, others did not. In prospective randomized trials, episiotomy did not protect women from developing SUI after delivery. Episiotomy also does not seem to affect PNTML, urethral closure pressure, or pelvic muscle strength.

Only one randomized trial has assessed the difference in pelvic floor symptoms after planned elective cesarean delivery or planned vaginal birth: the Term Breech Trial, conducted by Hannah et al. (2002). Questionnaires were completed 3 months postpartum by 1596 women from 110 centers around the world. Although the conclusions are limited by the large number of women in the planned vaginal birth group who, instead, delivered by cesarean, the short-term results are of interest. Women in the planned cesarean delivery group reported less urinary incontinence than those in the planned vaginal birth group (4.5% versus 7.3%; RR, 0.62; 95% CI, 0.41-0.93). Other outcomes did not differ. The lower rate of incontinence in this study, compared with others that assessed postpartum urinary incontinence, may be because women were queried about urinary incontinence symptoms only during the week before taking the questionnaire. In addition, women from different countries may interpret symptom questionnaires differently.

**Fecal Incontinence**

In large, prospective cohort studies of pregnant or newly postpartum women, incontinence to both flatus and stool ranged from 6% to 25%. Vaginal delivery by forceps and, in some cases, vacuum delivery increased the odds of postpartum fecal incontinence. As discussed previously, midline episiotomy is highly associated with sphincter injury but does not appear, in most studies, to be an independent risk factor for fecal incontinence. Studies assessing the prevalence of fecal incontinence at various postpartum intervals in women with known sphincter tears reveal prevalence of incontinence of flatus of 17% to 59% and incontinence to liquid and solid stool of 3% to 27%.

Few studies have been designed to examine the difference in fecal incontinence after cesarean versus vaginal delivery, and the studies have contradictory findings. In a prospective study by Fynes et al. (1998), of 234 women attending a hospital antepartum clinic in Dublin, none of the 34 women who had a cesarean section reported fecal incontinence. Similarly, Abramowitz et al. (2000) reported that of 259 women who delivered consecutively at a single hospital, none of the 31 who delivered by cesarean reported fecal incontinence compared with 13% of primiparous women who delivered vaginally. In a large prospective study by Chalifa et al. (1999), of 549 nulliparous pregnant women, a greater proportion of women reported fecal urgency after vaginal delivery than after cesarean (7.3% versus 3.1%, respectively). In the Childbirth and Pelvic Symptoms study completed by the Pelvic Floor Disorders Network in 2006, 17% of women who had sustained a sphincter tear had fecal incontinence versus 8% of those who had a vaginal delivery without obvious sphincter...
involvement (Borello-France et al., 2006). Interestingly, in the cohort of women who had a cesarean delivery, the rate of fecal incontinence was no different than the vaginal delivery group, suggesting that cesarean delivery is not fully protective for the symptom of fecal incontinence, even if sphincter injury is spared.

The impact of delivery type on fecal incontinence appears to decline with age. In a retrospective study by Nygaard et al. (1997), the reported prevalence of fecal incontinence 30 years after delivery was similar, regardless of delivery type. Most studies have not examined separately the effect of a third-degree versus a fourth-degree laceration on future bowel control. In a retrospective, single-site study by Fenner et al. (2003), 831 women (29% response rate) completed a bowel questionnaire 6 months after their first delivery. The incidence of worse bowel control than before pregnancy was nearly 10 times greater in women with fourth-degree lacerations (30.8%) compared with women with third-degree lacerations (3.6%). Although this difference may be the result of increased participation from women with more symptoms, it is also possible that the greater incidence of fecal incontinence associated with fourth-degree lacerations may be the result of the loss of some of the internal anal sphincter muscle. Further study is needed on the role of the internal anal sphincter in continuity, and the best methods of repairing this muscle at the time of rupture.

Although some degree of fecal incontinence after sphincter disruption is likely related to impaired neurologic control of continence, separation of the repaired muscles may also play a role. Fitzpatrick et al. (2000) examined 154 women 3 months after primary repair of an anal sphincter defect. One-third had ultrasound evidence of a persistent, large (larger than one quadrant) anal sphincter defect; this was not influenced by whether the repair was done in an end-to-end manner or an overlapping fashion.

Pelvic Organ Prolapse (POP)

Pelvic organ support defects appear to occur during pregnancy but before delivery. O’Boyle et al. (2002) found that nulligravid pregnant women were more likely to have POP than their nulliparous counterparts.

Parity increases the risk for both POP and surgery for POP. In women participating in the Women’s Health Initiative, those who had borne at least one child were twice as likely to have uterine prolapse, rectocele, and cystocele as nulliparous women, after adjusting for age, ethnicity, body mass index, and other factors. Similarly, in a study of 487 Swedish women, Samuelsson et al. (1999) reported that 31% had some degree of POP on examination; parity and age increased the risk of POP after adjusting for other variables.

In a British cohort study conducted by Mant et al. (1997) of 17,000 women, parity was the variable related most strongly to surgery for POP. The risk increased with each child, but the rate of increased declined after women were delivered of two children. In a case–control study, Rinne and Kirkinen (1999) found that women younger than 45 years who underwent surgery for POP had more deliveries and heavier babies than age-matched control subjects operated for benign ovarian tumors.

In a more recent cohort study out of Johns Hopkins in which PFDs were compared with route of delivery, Handa et al. (2011) demonstrated in women who were a median of 7.4 years out from birth that 13% of the women who had spontaneous deliveries had prolapse on examination, but that only 3% of those same women had symptoms of prolapse on validated questionnaires. Table 16.5 shows more of the data for prolapse and other PFDs.

The Debate about Elective Primary Cesarean Delivery

No chapter about the effect of childbirth on PFDs is complete without mention of the ongoing debate about the role of elective primary cesarean delivery. Certainly, most serious are concerns about greater mortality associated with cesarean delivery. It is difficult to tease out morbidity and mortality related to an elective cesarean delivery done before labor versus that done not only after active labor, but also because of pregnancy complications that increase morbidity. Perhaps more important, minimal information exists that allows us to judge risk for a woman’s entire reproductive career; the information that does exist suggests strongly

<table>
<thead>
<tr>
<th>Pelvic Floor Disorder</th>
<th>All Births Cesarean before Active Labor (n = 192)</th>
<th>All Cesarean Births before Complete Cervical Dilation (n = 228)</th>
<th>At Least One Cesarean Delivery after Complete Cervical Dilation (n = 140)</th>
<th>No Operative Vaginal Births (n = 325)</th>
<th>At Least One Operative Vaginal Birth (n = 126)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stress incontinence</td>
<td>1 (Reference)</td>
<td>0.88 (0.4-1.9)</td>
<td>1.30 (0.57-2.95)</td>
<td>2.87 (1.49-5.52)</td>
<td>4.45 (2.14-9.27)</td>
</tr>
<tr>
<td>Overactive bladder</td>
<td>1 (Reference)</td>
<td>0.74 (0.32-1.73)</td>
<td>1.17 (0.47-2.91)</td>
<td>1.66 (0.80-3.48)</td>
<td>4.89 (2.23-10.74)</td>
</tr>
<tr>
<td>Anal incontinence</td>
<td>1 (Reference)</td>
<td>1.12 (0.55-2.29)</td>
<td>1.48 (0.70-3.11)</td>
<td>1.62 (0.85-3.10)</td>
<td>2.22 (1.06-4.64)</td>
</tr>
<tr>
<td>POP symptom</td>
<td>1 (Reference)</td>
<td>0.72 (0.12-4.42)</td>
<td>0.99 (0.16-6.13)</td>
<td>2.80 (0.73-10.81)</td>
<td>6.83 (1.68-27.80)</td>
</tr>
<tr>
<td>POP at or beyond hymen</td>
<td>1 (Reference)</td>
<td>0.53 (0.13-2.27)</td>
<td>0.73 (0.17-3.13)</td>
<td>5.64 (2.16-14.70)</td>
<td>7.50 (2.70-20.87)</td>
</tr>
</tbody>
</table>

POP, pelvic organ prolapse.

that severe complications increase dramatically after two cesarean deliveries. Those in favor of a policy of elective cesarean support their decision with factors such as patient autonomy, concern over long-term adverse outcomes thought to be associated with vaginal delivery, particularly PFDs; avoidance of the greater morbidity associated with cesarean delivery after the onset of labor; prevention of stillbirths that occur after 39 weeks by delivering preemptively via cesarean instead; and a decrease in the incidence of traumatic birth injuries in neonates.

Conversely, those opposed to allowing women a delivery choice cite the increased short-term morbidity, greater maternal mortality rate, greater neonatal respiratory complications, adverse effects on future pregnancies (in terms of both fertility and subsequent complications from repeated cesarean deliveries), greater cost associated with cesarean delivery, and dubious morality involved in turning a “natural” event into an invasive operation.

Since the last edition of this textbook was published, an National Institutes of Health State of the Science Conference brought experts and interested parties together to discuss the question of cesarean on maternal request, and its effect on neonatal and maternal outcomes, including the potential effect on the acquisition of PFDs. In summary, only urinary incontinence had “weak” evidence that supported the elective cesarean (over planned vaginal delivery). However, the duration of the effect was uncertain, and seemed to decrease over time, diminishing the potential benefit. There was not enough evidence to clarify for fecal incontinence or POP. In addition, even in those studies in which PFDs were demonstrated to be less frequent after cesarean only, the incidence was not zero, suggesting that perhaps pregnancy itself contributes to the pathophysiologic process that leads to these disorders. Although a randomized trial would provide the highest level of evidence for this particular question, it is possible that longitudinal natural history studies that include relevant data about PFDs could eventually provide missing data, especially given that the cesarean rate, as of this edition, is well over 30% in the United States, and even higher in other countries.

Fistulas

Vesicovaginal fistulas and rectovaginal fistulas are complications that can arise after difficult deliveries. The majority of obstetric vesicovaginal fistulas occur in the developing world as a result of obstetric labor, but can also occur after vaginal as well as cesarean deliveries (in particular, difficult repeat cesarean deliveries) in the developed world. Rectovaginal fistulas are seen most commonly after an obstetric injury, generally when anal sphincter disruption has occurred, as in a third- or fourth-degree laceration. These are extremely important problems that require significant attention from the pelvic surgeon. Chapter 33 is dedicated to rectovaginal fistulas and perineal breakdown; Chapter 41 discusses lower urinary tract fistulas.

Conclusion

Childbirth increases the risk of PFDs in young and middle-age women. In most studies, vaginal delivery increases this risk more than cesarean delivery. Fistulas are the most obvious, extreme example of the trauma to the pelvic floor. However, nulliparous women also develop PFDs, although not at as high a rate as parous women. Furthermore, most parous women do not have surgery for PFDs. Both urinary and fecal incontinence increase with age; indeed, studies of older nulliparous women find the same or greater rates of these disorders, as do studies of younger parous women. The fact that older women have high rates of urinary and fecal incontinence does not, however, mean that childbirth is not one of the causes of these disorders, but that, in older women, other factors (such as functional impairment, medical problems, and even age itself) assume much greater importance, thereby minimizing the role of childbirth. In younger and middle-age women, parity and vaginal delivery remain important risk factors for PFDs. Additional research is needed to identify the group of women most at risk for PFDs so that preventive efforts may be studied.

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Alterations of Pelvic Floor Anatomy and Physiology in Pregnancy


