Urological problems in pregnancy

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Summary
During pregnancy the urinary tract undergoes extensive anatomical and physiological changes. These changes can result in many symptoms and pathological conditions that may affect the mother and fetus. It is well documented that childbirth may result in urinary tract damage which may predispose to postpartum symptoms. This review describes the physiological and pathological consequences of pregnancy and delivery on the urinary tract, and how these may be minimized.

Introduction
During pregnancy the urinary tract undergoes extensive anatomical and physiological changes. These changes may be further influenced by alteration in renal function and intercurrent pathology in pregnancy, and changes resulting from labour and delivery.

Anatomical and physiological changes

The upper urinary tract
In normal pregnancy the kidneys elongate by ≈1 cm because of the increase in vascular volume and interstitial space. Dilatation of the renal pelvis and ureter can be seen as early as 7 weeks of gestation and is thought to be secondary to a muscle-relaxant effect of progesterone and mechanical obstruction from the gravid uterus. It is more marked on the right than the left side, because of dextrorotation of the uterus and a protective effect of the sigmoid colon on the left ureter. There is a 40–50% increase in GFR and a 60–80% increase in the effective renal plasma flow [1]. Because of this the plasma creatinine, urea and urate values are lower than the normal ranges for nonpregnant women.

The lower urinary tract
The elevated levels of oestrogen and progesterone cause the bladder and urethral mucosa to become hyperaemic and congested, and the urethral transitional epithelium more squamous. The detrusor muscle hypertrophies under the elevated levels of oestrogen but the increased levels of progesterone lead to a relative bladder hypotonia and increased bladder capacity.

The bladder is drawn upwards anteriorly as the uterus enlarges, becoming more of an abdominal than a pelvic organ by the third trimester. The base of the bladder enlarges and the trigone becomes more convex than concave. Radiological studies in pregnancy show that the bladder is distorted by the fundus, and in labour the bladder neck is displaced forwards and becomes more funnelled [2].

Iosif et al. [3] performed urethral pressure profilometry and simultaneous urethrocystometry in 14 healthy continent primiparae in the first and late third trimester, and again 5–7 days postpartum, and reported an increase in total and functional urethral length, with an increase in intravesical pressure from 9 to 20 cmH₂O, and a corresponding increase in urethral closure pressure.

Problems in pregnancy

The upper urinary tract
UTI/pyelonephritis: UTIs are a common complication in pregnancy and the standard definition is a colony count of >10⁵ c.f.u./mL of urine; however, counts as low as 10² may represent active infection in pregnancy [4]. The prevalence of asymptomatic bacteriuria is similar to that in a nonpregnant population, at 5–10%, but there is a 3–4-fold higher progression rate, with ≈30% developing symptomatic infection [5]. Acute pyelonephritis complicates 1–2% of pregnancies. The development of acute pyelonephritis is associated with maternal and fetal morbidity and mortality. This typically presents with fever, costovertebral angle tenderness, cystitis symptoms and feeling systemically unwell. In severe cases sepsis and respiratory distress occur. Other reports of obstetric problems include preterm labour, pre-eclampsia and low birthweight, so it is advisable that all pregnant women should be screened frequently in pregnancy for asymptomatic bacteriuria and if positive, treated. Factors that increase the risk of pathogenicity of bacteriuria in pregnancy include the decrease in bladder tone and increased ureteric volume, as well as a facilitatory effect

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of oestrogen on upper tract infection, especially with the main pathogenic organism, *Escherichia coli*. Treatment should be aggressive, consisting of rehydration, analgesia and intravenous antibiotics.

Recommended antibiotics for use in pregnancy for asymptomatic bacteriuria include amoxycillin, an oral cephalosporin, nitrofurantoin, or nalidixic acid. Sulphonamides can also be used, but not in late pregnancy when they can cross the placenta and increase the risk of kernicterus by displacing bilirubin bound to albumin. Tetracyclines cause bone and teeth dysplasia and discoloration, and should be avoided. The antifolate agent trimethoprim should not be used in early pregnancy as it may affect neural tube development. Pyelonephritis should be treated with a second- or third-generation cephalosporin or a short course of an aminoglycoside. Prolonged courses of the latter may result in eighth nerve damage to the fetus.

Postpartum bacteriuria is twice as common in women who have been catheterized in labour (9.1% vs 4.7%). This risk increases to 25% where an indwelling catheter is used, and so ideally all women should be encouraged to void spontaneously in labour. This bacteriuria also needs treatment.

**Urinary calculi:** Despite the increase in urinary stasis, infection and obstruction in pregnancy, which generally predisposes to the formation of calculi, the incidence of renal calculi is ~0.3% and similar to that in a nonpregnant population [6]. Pregnancy also does not increase the risk of stones in known ‘stone formers’. Presentation is usually after 20 weeks’ gestation when ureteric dilatation is most marked. These stones are mainly composed of calcium, with an increase in struvite stones also reported. They may present with pain and tenderness, mimicking acute pyelonephritis, but without fever. Haematuria and colic may be absent because of the physiological hydrourery. Ultrasonography should be used and occasionally a plain abdominal X-ray may be required; renal function assessment and urine microscopy should also be undertaken. Initial treatment should be conservative, as 60% of stones will pass spontaneously. Definitive surgery should be delayed until at least 4 months postpartum if possible. The safety of lithotripsy in pregnancy has not been fully validated and drugs used to prevent stone formation, e.g. thiazides, xanthine oxidase inhibitors and d-penicillamine, are best avoided.

**Renal failure:** Acute renal failure may occur secondary to complications in pregnancy from pre-eclampsia, uterine sepsis, pyelonephritis and acute fatty liver of pregnancy. With improvements in the management of obstetric haemorrhage and pre-eclampsia, acute renal failure is seen less often in the West. Effective management of these clinical situations will often result in a return to normal renal function.

In women with chronic renal disease, pregnancy may have an adverse effect on renal function, which is linked to the degree of functional impairment before pregnancy and the presence of hypertension. With severe renal disease patients are often infertile and if pregnancy occurs, a live birth is uncommon. In those with moderate or severe renal insufficiency (serum creatinine >133 μmol/L) further loss of function will occur in half, with 10% of women progressing to end-stage renal failure [7].

**The lower urinary tract**

LUTS are very common in pregnancy; there are several large epidemiological studies which have assessed the prevalence of dysfunctional urinary symptoms in pregnancy, most focusing on the symptom of stress incontinence. Most of these symptoms may be a consequence of the normal anatomical and physiological changes that occur in pregnancy, but superimposed on these changes may be further pathological changes as a consequence of tissue damage either from pregnancy or labour, resulting in persistent symptoms. The distinction between normal physiological changes and transient or permanent pathophysiology is often not clear.

**Frequency and nocturia:** These are the commonest and earliest symptoms to develop in pregnancy. Normal non-pregnant women void four to six times per day and rarely at night. Francis [8], using a definition of frequency as at least seven daytime voids and one night-time void, studied the voiding habits in 400 healthy women during and after an uncomplicated pregnancy, and compared them with 50 normal nonpregnant patients of a similar age. Frequency was reported by 59% in early pregnancy, 61% in mid-pregnancy and 81% in late pregnancy. Parboosingh and Doig [9], defining nocturia as at least three night-time voids, questioned 873 healthy antenatal patients and found that nearly 66% experienced nocturia by the third trimester. Stanton et al. [10] reported that frequency was the commonest symptom. The onset of frequency may be as early as the first trimester. Cutner [11] assessed LUTS in 47 women undergoing termination of pregnancy at 6–15 weeks gestation, and found that 40% complained of frequency and 23% of nocturia. The cause of frequency was unrelated to bladder capacity or the effect of posture, but ascribable to the polydypsia and polyuria of pregnancy [8]. Both fluid intake and output increase in the first trimester, remaining constant until the third trimester, when a decrease in sodium output leads to a decrease in urine output. However, despite this, frequency persists, related to the
pressure on the bladder by the uterus. Cutner [12] found a correlation between the maximum voided volumes, first sensation to void and maximum bladder capacity, which was in turn related to the presence of low compliance. There was no correlation between the maximum voided volumes and diurnal frequency and nocturia. Parboosingh and Doig [9] measured mean urine flow and solute excretion in 24-h and overnight collections, in 100 normal and nonpregnant women. An increase in sodium excretion was the major reason for increased night-time voiding, as well as the mobilization of dependant oedema at night when in the recumbent position.

Voiding difficulties: Urinary hesitancy may occur in up to 27% of patients in the first two trimesters [10]. Fischer and Kittel [13] assessed flow rates in 290 women during pregnancy and found that there were significantly higher peak flow rates in the second and third trimester than in controls and early pregnancy, but these higher flow rates were associated with larger voided volumes. Nomograms have been produced to assess flow rates according to volumes voided, as low volumes may result in an artificially low peak flow rate [14]. Cutner [12] assessed flow rates in pregnancy, adjusted for volume voided, and found no difference in women complaining of hesitancy or incomplete emptying compared with pregnant women with normal voiding.

Urinary retention can occur in pregnancy, associated with the enlargement of a retroverted uterus with subsequent entrapment of the fundus below the sacral promontory [15]. Other causes include an enlarging fibroid, or a pelvic mass. This retention usually resolves by 16 weeks’ gestation as the uterus grows out of the pelvis, and can be managed meanwhile with either bladder drainage or intermittent self-catheterization. Alternatively, the uterus can be reduced manually, or a Hodge pessary may be inserted to maintain uterine position and relieve the obstruction on the bladder neck.

Postpartum urinary retention is common, with a reported incidence of 1.7–17.9% [16]. Risk factors include a first labour, instrumental delivery and epidural analgesia, and a longer duration of labour (≥800 min) [17,18]. Khullar and Cardozo [19] found that the bladder can take up to 8 h to regain sensation after the last dose of an epidural, and during this period overdistension of the bladder may occur, leading to permanent detrusor dysfunction. Tapp et al. [20] reported that in a group of six women with acute retention postpartum, two had permanent voiding difficulties, two required intermittent self-catheterization and two had altered bladder sensation. Because of this, women with these risk factors should be watched carefully after delivery to ensure that voiding is adequate, and if necessary catheterized to avoid overdistension.

Urgency and urge incontinence also increase in pregnancy: Cutner et al. [21] found that 62% of women complained of urgency and 18% complained of urge incontinence in pregnancy. In another study of 549 nulliparae, 2.2% reported urgency before pregnancy, with 22.9% and 7.8% reporting urgency in pregnancy and 12 weeks postpartum, respectively. In this same group of women, 0.5%, 8.0% and 2.2% reported urge incontinence before pregnancy, in pregnancy and 12 weeks postpartum, respectively [22].

Cutner et al. [21] reported that 23% of patients had detrusor instability, lower than the prevalence of women with irritative urinary symptoms. Calila et al. [23], in a prospective study of 161 nulliparae investigated by urodynamics in the third trimester, and then 12 weeks postpartum, found a prevalence of detrusor instability of 8.1% and 6.8%, respectively. In this group of women, only 4.9% reported urge incontinence postpartum. This suggests that the aetiology of irritative urinary symptoms is only partly explained by the development of detrusor instability and may be also a consequence of low compliance, as shown by Cutner et al. [21], or urethral instability.

Stress incontinence is a common symptom associated with pregnancy and has been reported in up to 85% of women [8,24,25]. Francis [8] found that in the first trimester of pregnancy 16% of women complained of stress incontinence, with 34% doing so in the second half of pregnancy. Stanton et al. [10] assessed the prevalence of both stress and urge incontinence at 32 weeks’ gestation and found an incidence of 36% and 13%, respectively. Both studies found that stress incontinence rarely appears for the first time after birth without previous antenatal symptoms, and is commoner in multiparae than nulliparae.

Viktrup et al. [25] interviewed 305 primiparae and found that 39% had stress incontinence before, during or after pregnancy, and 7% developed de novo stress incontinence after delivery. In those with onset of stress incontinence in pregnancy only 3% had persisting symptoms at 1 year after delivery, whereas in those with onset after delivery, 24% had symptoms at 1 year. In the 120 patients who had onset of stress incontinence before, during or after pregnancy, 21 (18%) had new onset of symptoms postpartum. There was no relationship between the presence of symptoms at 1 year and obstetric risk factors. In another study by Viktrup and Lose [26], there was a significant correlation between the length of the second stage of labour and the development of stress incontinence. Wilson et al. [27], in a postal questionnaire study of 2134 women 3 months
after delivery, found that 34.3% admitted to some degree of urinary incontinence, with 3.3% having daily or more frequent leakage. The risk was significantly less in those women, especially primiparous, who had a Caesarean section, whether elective or in labour, suggesting it is vaginal delivery not pregnancy that predisposes to stress incontinence. The prevalence of incontinence was similar in those women having three or more Caesarean sections (38.9%) to those delivered vaginally (37.7%). Some have proposed that in this situation incontinence may be secondary to nerve damage from bladder dissection during Caesarean section. This is supported by a study of women who had undergone elective Caesarean section, of whom 17% reported stress incontinence and 51% had severe symptoms that occurred for the first time in pregnancy or the puerperium [28].

Chaliha et al. [22] interviewed 549 nulliparous women to assess the prevalence of urinary incontinence before, during and after delivery. Pregnancy and delivery resulted in a significant increase in symptoms of urinary frequency, incontinence and urgency. Stress incontinence was the most common form of incontinence and reported by 17 (3.1%), 196 (35.7%) and 68 (12.4%) women before, during and after pregnancy, respectively (Table 1). The low prevalence of incontinence before pregnancy in nulliparous women agrees with previous data linking parity to incontinence [27,29,30].

### Changes in the lower urinary tract and pelvic floor related to stress incontinence

The exact causes of stress incontinence are unclear and probably multifactorial, related to nerve damage, and/or physiological and structural changes of the lower urinary tract. There also may be a group of women at increased risk of postpartum incontinence because of abnormalities of collagen.

#### Functional changes

Urethral pressure profilometry has been used to evaluate urethral sphincter function in normal pregnancies and those complicated by incontinence. Iosif and Ulmsten [31] compared urethral pressure profile measurements in pregnant women with stress incontinence with continent healthy women from an earlier study [3]. The absolute urethral length increased by a mean of 6.7 mm and the functional urethral length by 4.8 mm. The maximum urethral closure pressure increased to 93 cmH₂O at 38 weeks and then decreased to 69 cmH₂O (the value before pregnancy) after delivery. These changes were not apparent in women complaining of incontinence and are postulated to be a mechanism whereby continence is maintained despite an increase in intra-vesical pressure in pregnancy. This agrees with other studies showing evidence of low urethral pressure in nonpregnant women with stress incontinence [32,33]. Van Geelen et al. [34] reported similar work in 43 pregnant women; they found an increase in maximum urethral pressure, urethral length and bladder pressure, but no significant differences in maximum urethral closure pressure or functional urethral length. However the lack of difference may be because their study group included continent and stress incontinent women. After delivery the urethral length and pressure were significantly lower in those women who had vaginal deliveries but not in those delivered by Caesarean section. These changes after vaginal delivery were unrelated to the duration of the second stage of labour, episiotomy, or fetal birthweight. This increase in urethral closure pressure may be a result of an increase in urethral sphincter volume from increased blood flow, and there is an increase in the amplitude of vascular pulsations recorded from the urethral wall, especially in the first 16 weeks of pregnancy, which may be related to an increase in blood volume in pregnancy. Pregnant women with genuine stress incontinence had a lower amplitude of vascular pulsations in the periurethral plexus than had continent women, suggesting that this affects urethral closure pressure [35].

Further work suggesting that childbirth may lead to permanent damage to the urethral sphincter mechanism was reported by Tapp et al. [36], who assessed two groups of women, one with competent urethral sphincter mechanisms and one with genuine stress incontinence. The latter group had a negative correlation between the number of vaginal deliveries and the pressure transmission ratios in the distal quarter of the urethra; more vaginal deliveries were associated with poor function of the distal urethral sphincter mechanism. As continence is thought to be maintained by the action of the proximal and not distal sphincter
mechanism, the authors concluded that damage to
the distal sphincter mechanism may result in stress
incontinence in women with impaired proximal sphi-
nter function.

Cystometry in pregnancy has been used by several
groups; Cutner et al. [21] used cystometry in early preg-
nancy and found a normal first sensation and bladder
capacity. Kerr-Wilson et al. [37] assessed women uro-
dynamically immediately after delivery and then again
4 weeks afterward, finding a significant decrease in
maximum cystometric capacity at 4 weeks, and a sig-
nificant decrease in primiparous. However, all values were
within normal limits and there was no evidence of
bladder hypotonia. In a large prospective study of 161
nulliparous women in the third trimester and then again
at 12 weeks postpartum, Chaliha et al. [23] found a high
prevalence of genuine stress incontinence and detrusor
instability; 8.7% and 8.1%, respectively, in the ante-
natal period and 5.0% and 6.8%, respectively, after
delivery. The mean values for urodynamic variables
in the third trimester and after delivery were lower
than values defined in a nonpregnant population and
unrelated to obstetric or neonatal variables (Table 2).
After delivery there was an increase in first sensation
and strong sensation to void, and maximum bladder capacity.
However, despite the high prevalence of symptoms, there
was a poor correlation between symptoms and urody-
namic findings, which agrees with data in nonpregnant
women [38]. Therefore, these observed changes in
bladder function were consistent with a pressure effect
of a gravid uterus and unrelated to the mode of delivery
and neonatal factors.

Nerve damage

Patients with genuine stress incontinence have been
shown to have abnormal conduction in the perineal
branch of the pudendal nerve which innervates the
perirethral striated muscle and pubococcygeus
muscle [39–41]. Several reports also show injury to
the nerve supply after childbirth, but these studies
often do not relate objective damage to symptoms.
Snooks et al. [42] found prolongation of pudendal
nerve terminal motor latencies 48–72 h after delivery
in 42% of those delivered vaginally, but not those
delivered by Caesarean section. The degree of pudendal
nerve damage was greater in multiparous women and
correlated with the use of forceps and a longer second
stage of labour. In 60% of women with evidence of nerve
damage, the pudendal nerve latency had returned to
normal at 2 months after delivery. The authors suggested
that vaginal delivery results in pudendal nerve damage
probably from a combination of direct and traction injury
during delivery, and that this may lead to the develop-
ment of stress incontinence. However, that study was not
prospective and included multiparous women who may
have sustained previous nerve damage. Furthermore, the
study assessed innervation of the striated anal sphincter,
which may not reflect striated urethral sphincter
innervation, and there was a poor correlation between
abnormal latencies and symptoms. Allen et al. [43], using
concentric needle EMG and pudendal nerve conduction
tests, found evidence of denervation injury in 80% of
women after delivery. Those women with a long (active)
second stage of labour (> 56.7 min) and a large baby
(> 3.41 kg) showed greater nerve damage.

EMG of the right and left pubococcygeus muscle has
shown that childbirth induces both qualitative and
quantitative changes, such that sphincter weakness
was caused by not only loss of motor units but also
asynchronous activity in those that remained [44].

Structural changes

Trauma from vaginal delivery may result in muscular
and connective tissue damage. Gainey [45] examined
1000 women after delivery, finding evidence of urethral
detachment in 18% and damage to the pelvic floor

<table>
<thead>
<tr>
<th>Urodynamic variable, mean (sd)</th>
<th>Before</th>
<th>After</th>
<th>After/before (95% CI)*</th>
<th>P*</th>
<th>Vaginal (n = 89)</th>
<th>Instrumental (n = 41)</th>
<th>Caesarean (n = 31)</th>
<th>P†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sit SDV</td>
<td>111 (70.1)</td>
<td>148 (83.0)</td>
<td>1.34 (1.17–1.54) &lt; 0.001</td>
<td>150 (83)</td>
<td>138 (87)</td>
<td>153 (80)</td>
<td>0.5</td>
<td></td>
</tr>
<tr>
<td>Sit SDV</td>
<td>188 (91.3)</td>
<td>217 (94.3)</td>
<td>1.17 (1.07–1.28) &lt; 0.001</td>
<td>225 (96)</td>
<td>203 (96)</td>
<td>214 (87)</td>
<td>0.3</td>
<td></td>
</tr>
<tr>
<td>Sit MXCC</td>
<td>301 (146.1)</td>
<td>299 (114.4)</td>
<td>1.03 (0.96–1.12) 0.4</td>
<td>300 (114)</td>
<td>309 (126)</td>
<td>281 (101)</td>
<td>0.8</td>
<td></td>
</tr>
<tr>
<td>Stand SDV</td>
<td>96 (69.3)</td>
<td>139 (90.2)</td>
<td>1.48 (1.30–1.69) &lt; 0.001</td>
<td>96 (68)</td>
<td>162 (115)</td>
<td>140 (75)</td>
<td>0.2</td>
<td></td>
</tr>
<tr>
<td>Stand SDV</td>
<td>167 (102.0)</td>
<td>209 (105.0)</td>
<td>1.29 (1.18–1.42) &lt; 0.001</td>
<td>200 (93)</td>
<td>231 (131)</td>
<td>205 (98)</td>
<td>0.7</td>
<td></td>
</tr>
<tr>
<td>Stand MXCC</td>
<td>239 (136.4)</td>
<td>271 (123.6)</td>
<td>1.17 (1.08–1.27) 0.001</td>
<td>264 (14)</td>
<td>290 (146)</td>
<td>265 (119)</td>
<td>0.9</td>
<td></td>
</tr>
<tr>
<td>MVP [n]</td>
<td>38 (22.0 [125]</td>
<td>32 (17.2 [118]</td>
<td>0.85 (0.71–1.01) 0.07</td>
<td>33 (19 [70]</td>
<td>30 (13 [27]</td>
<td>33 (17 [28]</td>
<td>0.7</td>
<td></td>
</tr>
<tr>
<td>Peak flow rate [n]</td>
<td>28 (16.3 [153]</td>
<td>23 (14.4 [157]</td>
<td>0.83 (0.75–0.92) &lt; 0.001</td>
<td>24 (18 [85]</td>
<td>22 (8 [38]</td>
<td>23 (9 [30]</td>
<td>0.8</td>
<td></td>
</tr>
</tbody>
</table>

FDV, first desire to void (mL); SDV, strong desire to void (mL); MXCC, maximum cystometric capacity (mL); MVP, maximum voiding pressure (cmH2O); Peak flow rate (mL/s); *Paired t method on log-transformed data; †F-test on log-transformed data, adjusted for antenatal values.
muscles in 31%. More recently, Peschers et al. [46] evaluated bladder neck position and mobility using perineal ultrasonography at 8 weeks after delivery. Bladder neck position was significantly lower and bladder neck mobility greater after vaginal delivery than in women who had an elective Caesarean section and in nulligravid controls. This agrees with Meyer et al. [47] who reported a significant increase in bladder neck mobility after vaginal delivery in primiparae, but the bladder neck position was only lowered after forceps delivery. Thus vaginal delivery seems to alter urethral support, which may result in stress incontinence.

It has been suggested that there may be a group of women at an inherent increased risk of developing incontinence because they have abnormalities in collagen [48], as it is the collagenous component of the connective tissue that contributes to structural support of the bladder neck. In pregnancy the tensile properties of the connective tissue are reduced with a decrease in total collagen content and increase in glycosaminoglycans [49]. Changes in collagen may result in greater mobility of the bladder neck, resulting in stress incontinence. This was suggested by King and Freeman [50] who used perineal ultrasonography in 128 primigravidae antenatally and then again 10–14 weeks after delivery. They found an increase in bladder neck mobility antenatally in those women who subsequently developed postpartum stress incontinence. If collagen abnormalities increase the risk of incontinence, and if these can be detected antenatally to define women at risk of pelvic floor dysfunction, this will allow them to be counselled and preventative measures instituted if appropriate, e.g. offering elective Caesarean section. Chaliba et al. [22] evaluated the role of antenatal history and physical markers suggestive of collagen weakness, e.g. striae, hernia, varicose veins and joint mobility, in predicting postpartum incontinence. In the third trimester, 549 nulliparae were interviewed using a standardized urinary and bowel symptom questionnaire, and examined physically to assess these markers of collagen weakness. Postnatal anal and urinary incontinence was not related to race, antenatal body-mass index, the presence of striae, hernia, varicose veins, piles or a family history of incontinence, prolapse or collagen weakness. Higher joint mobility scores were associated with incontinence of flatus but not fecal urgency or urinary symptoms. Therefore, although collagen weakness has been implicated in the pathogenesis of incontinence, physical markers of collagen weakness as used in that study could not predict postpartum incontinence. Perhaps these markers were not representative of collagen weakness, or a larger study with a longer follow-up is required.

**Urinary tract trauma during delivery**

During labour and delivery the bladder is particularly vulnerable to trauma. Cystoscopy after delivery shows changes such as mucosal congestion, submucosal haemorrhage and capillary oozing, particularly around the trigone [51]. Even Caesarean delivery does not eliminate the risk of bladder trauma, as the position of the bladder as an abdominal organ exposes it to risk at the time of surgery. The incidence of bladder trauma at Caesarean section is <1% [52] but increases in situations where a Pfannenstiel incision, lower segment incision, previous Caesarean section, prolonged second stage of labour and Caesarean hysterectomy have occurred. The dome of the bladder is the site most frequently injured and should be repaired in two layers, whereas injuries extending into the trigone or ureteric orifices may require ureteric implantation or stenting. The incidence of ureteric injuries is ≈0.1% [53]. The diagnosis of bladder injury is usually immediate, whereas that of ureteric injury is often delayed, and should be suspected if the patient presents with flank tenderness and unilateral hydronephrosis [54]. Urinary tract injury has also been reported after instrumental deliveries, particularly mid-pelvic forceps, but even after ventouse delivery [55].

Vesicovaginal fistulae as a result of delivery are reported in 0.7% of patients undergoing Caesarean section [56]. Currently this complication is rarely seen in the West, but is common in developing countries in obstructed labour where there is necrosis of the anterior vaginal wall and the bladder. These fistulae typically present with continuous incontinence 7–14 days after delivery and if suspected should be investigated with cystoscopy and IVU.

**Conclusions**

Pregnancy and delivery is a time of major anatomical and physiological changes to the urinary tract which may result in an alteration in urinary tract function, most commonly manifested by the development of urinary symptoms. It is well documented that vaginal delivery results in anatomical, neural and structural damage to the pelvic floor, and these changes may result in permanent urinary tract dysfunction. However, Caesarean section may not totally prevent postpartum urinary incontinence and overall may subject a woman to greater morbidity and mortality. Further work is required to determine the pathophysiology of childbirth-related injuries to the urinary tract, and how labour can be managed to minimize them.
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