6
Postpartum Problems and the Role of a Perineal Clinic

Ranee Thakar and Abdul H. Sultan

6.1 Introduction

Over the past century there has been a dramatic fall in maternal morbidity and mortality, especially in the developed countries. Women therefore have high expectations of pregnancy and childbirth and consequently feel disillusioned when complications occur in the postpartum period. Up to 87% of mothers experience at least one health problem in the first 8 weeks postpartum, reducing to 76% 8 weeks to 18 months after delivery.[1] MacArthur et al.[2] found that the majority of symptoms that lasted more than a year after birth were still present 1–8 years later, suggesting that problems arising after childbirth can have long-term consequences on women's health. Common postnatal health problems include tiredness,[1,3,4] headache,[1,3] haemorrhoids,[1,3,4] perineal pain,[1,5] breast conditions,[1] constipation,[1] dyspareunia,[4,6] backache,[7] and urinary[8–10] and anal incontinence.[11–14] However, the majority of women do not consult a health professional even if they feel that they need help.[3] These conditions are more prevalent in primiparas and more likely to occur following instrumental rather than normal vaginal delivery or caesarean section.[1]

In this chapter, management of problems pertaining to the perineum or pelvic floor during pregnancy and the early postpartum period will be discussed.

6.2 Perineal Problems

6.2.1 Perineal Pain

Perineal pain is a common symptom following vaginal delivery, regardless of the presence of perineal trauma. However, the severity of perineal pain is directly proportional to the severity of perineal trauma.[5,15] Perineal pain occurs in 42% of women immediately after delivery but significantly reduces to 22% and 10% at 8 and 12 weeks respectively. Compared to a normal delivery, perineal pain occurs more frequently and persists for a longer period after assisted delivery (forceps, vacuum delivery, vaginal breech delivery).[16] Risk factors include having any perineal stitches (not only episiotomy), primiparity, assisted delivery and using entonox for analgesia in labour.[16]

Perineal pain may be due to soft tissue trauma with or without suturing. The pain becomes exaggerated if there is an associated inflammatory process, which can range from mild inflammation, cellulitis and florid inflammation with wound breakdown to abscess formation. Precipitating factors include lack of aseptic techniques, poor surgical techniques, which can result in poor apposition and/or granulation tissue or a stitch placed inadvertently in the rectal mucosa.

6.2.1.1 Treatment of Perineal Pain

In addition to treating the underlying condition, treatment options used to relieve perineal pain may be divided into local and systemic. Perineal pain is often accompanied with dyspareunia. Figure 6.1 shows a pathway that can be followed for both conditions.

Local Treatment

Although perineal ice packs have been very popular, there is inconclusive evidence regarding their effectiveness. Bathing in water with salt additives (sitz baths) has been traditionally used to relieve perineal wound pain and promote healing. A three-arm randomised controlled trial ($n = 1{,}800$) examined the effectiveness of adding salt, a 25-ml sachet of Savlon or nothing to the bath water each day for the first 10 days following delivery.[17] At 10 days and at 3 months the prevalence of pain and pattern of wound healing were similar in all the groups. Although bath additives did not enhance healing or reduce pain or dyspareunia, most of the women reported that bathing did provide some relief of the discomfort. Lavender oil added to bath water has been evaluated in a randomised trial and found to be ineffective.[18] Therapeutic ultrasound has been suggested as a means to alleviate perineal pain, but a Cochrane review was inconclusive.[19]

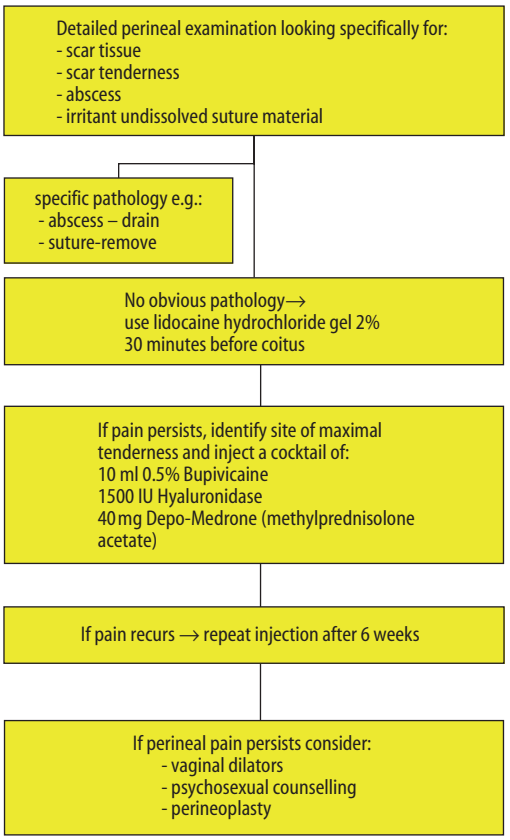

FIGURE 6.1. Suggested regimen for management of perineal pain/dyspareunia.

Local anaesthetics that can be applied directly to the perineum include lidocaine, which can be administered as a spray, gel or cream, and Epifoam (anti-inflammatory steroid-based foam containing 1% hydrocortisone acetate and 1% pramoxine hydrochloride). A recent Cochrane review indicated that the evidence for the effectiveness of topically applied anaesthetics is not compelling. The variety of topical anaesthetics used in the eight included trials limited the capacity to pool data, thus making interpretation difficult. The different methods of measuring pain also made comparisons between trials difficult. Furthermore, there has been no evaluation regarding any long-term effects of topically applied anaesthetics.[20]

Systemic Treatment

Oral Analgesics. Paracetamol (acetomenophen) is one of the most commonly used analgesics to relieve mild postnatal perineal pain. This is a sensible choice as it is relatively inexpensive, effective and free of side-effects to the mother and child.[17] As pain increases to moderate and severe levels, stronger analgesics such as opioid analgesics and non-opioid analgesics including non-steroidal anti-inflammatory drugs (NSAIDs) can be considered. However, one must be aware that opioids can cause constipation and NSAIDs can cause gastric irritation, renal failure and haematological problems. In addition, these drugs can be secreted in breast milk.

Suppositories. Rectal analgesia may be preferred in certain situations, e.g. nausea and vomiting and/or gastric irritation. The mother should be informed about the route of administration and verbal consent obtained. About 50% of the drug administered via the rectum will bypass the liver, resulting in faster pain relief and more local

action.[21] In a systematic review, which set out to assess the effectiveness of analgesic rectal suppositories for perineal pain after childbirth, Hedayati et al.[21] found that compared to placebo women were less likely to experience pain at or close to 24 hours after birth if they received NSAIDs in the form of suppositories such as diclofenac. They also required less additional analgesia in the first 24 hours after birth and this effect was still evident at 48 hours. The effect, if any, on longer-term pain relief and analgesia is not known.

Proven strategies to reduce perineal pain include use of continuous absorbable synthetic sutures[22] and antenatal perineal massage in women who have had a previous vaginal birth.[23]

6.2.2 Perineal Haematoma

Haematomas may be infralevator (vulval, perineal, vaginal) or supralevator (in the broad ligament or paravaginal area). They occur infrequently, with an incidence of between 1:500 and 1:900 vaginal deliveries. Although frequently related to an episiotomy due to an incomplete repair and obliteration of the "dead space", about 20% of cases occur even with an apparently intact perineum due to a concealed ruptured vessel.[24,25] A supralevator haematoma forms in the broad ligament and could be due to an extension of a tear of the cervix, vaginal fornix or uterus. As the haematoma distends, it displaces the uterus contralaterally and bulges into the upper vagina.

With both infralevator and supralevator haematomas, the patient often presents with pain and swelling in the perineal area immediately after delivery or in the postpartum period. However, with a supralevator haematoma, shock may ensue without any obvious swelling. The classical presentation is pain, restlessness, inability to pass urine and rectal tenesmus within a few hours after delivery. On examination there is usually an obvious tender swelling of the perineal area with purple and glistening overlying skin.

Management includes treatment of shock if present. Analgesics, ice packs and pressure dressings can be used if the haematoma is small (less than 5 cm) and not expanding. Surgical evacuation of the infralevator haematoma by incision and drainage should be carried out if the haematoma is large and expanding. The incision should preferably be made in the vagina to avoid scar formation. Often no obvious bleeding points are seen and deep sutures will need to be inserted at the base of the haematoma. It is advisable to leave a drain or pack for at least 24 hours. In contrast to an infralevator haematoma, the management of the supralevator haematoma is largely conservative and a blood transfusion may be necessary. Surgical exploration by laparotomy is usually very frustrating as the bleeding point cannot be identified and the ureter can be injured with the insertion of deep sutures. In this situation, options include evacuation of the clot and packing the cavity for 24 hours or performing an internal iliac artery ligation. The ideal approach when conservative management fails is not to operate, but to involve an intervention radiologist to perform angiographic embolisation of the bleeding vessel.[25]

6.2.3 Perineal Wound Infection

Delay in perineal wound healing due to infection can lead to discomfort, which can contribute to making the entire experience of pregnancy and childbirth very unpleasant. Furthermore, serious complications such as puerperal sepsis[26] and necrotising fasciitis[27] after perineal wound infection have been reported.

It is difficult to quantify the number of women that develop perineal wound infections as there is no standard classification for postpartum perineal wound infection and most studies quoting prevalence are retrospective, observational studies. Furthermore, perineal wound problems are managed in both primary and secondary care, making data collection unreliable. In a survey of 707 women who had spontaneous or assisted vaginal deliveries, Glazener et al.[28] found that 5.5% had perineal wound breakdown. Goldaber et al.[29] undertook a retrospective case review of 390 fourth degree tears in a hospital in Texas, USA and found that 5.4% had postpartum perineal morbidity (1.8% wound dehiscence alone, 2.8% infection and dehiscence, 0.8% infection alone). Women with morbidity were more likely to have experienced shoulder dystocia, endometritis and postpartum pyrexia.

Features of perineal infection include local pain, erythema, exudate, odour, oedema and pyrexia with or without wound breakdown. The care of such women should be transferred to the obstetrician, general practitioner or a specialist perineal clinic. Wound swabs should be taken for culture and sensitivities and antibiotics prescribed. Because there are no randomised studies to develop evidence-based management strategies, there is considerable controversy as to whether perineal wounds should be sutured as soon as possible or let to heal by secondary intention, as the wound may break down further if there is underlying wound infection. Uygur et al.[30] studied 37 women with episiotomy dehiscence (25 underwent early repair and 12 healed by secondary intention). They concluded that with adequate preoperative care such as primary wound cleansing (daily scrubbing and irrigation and intravenous antibiotics), early repair of episiotomy dehiscence is safe and effective. Similarly, in a small randomised study comparing the two methods, Christensen et al.[31] found no significant difference in healing time, hospitalisation and infection between the two groups. However, there was a need for vaginal plastic repair in three of the nine who underwent "open" treatment, i.e. healing by secondary intention.

When third or fourth degree tears break down, it is conventional practice to defer a second attempt at repair for 3–4 months. The delay is considered necessary to ensure adequate blood supply to the margins of the defect and restoration of tissue viability.[32] However, this delay is undesirable (persistent faecal incontinence, effect on sexual intercourse, muscle atrophy, etc.) and therefore early repair after outpatient wound debridement and preparation has been advocated. In a case series of 22 patients who developed wound breakdown within a week of repair of a fourth degree tear, all had a subsequent repair within an average of a further week. None experienced subsequent wound breakdown. One patient developed a small rectoperineal fistula that closed spontaneously after irrigation.[32] In another series of 23 patients (two third and 21 fourth degree tears), a second repair was performed within a mean of 7 days of outpatient debridement and all repairs were successful apart from one rectoperineal fistula that healed spontaneously after 3 months.[33] The authors concluded that forcing women to wait the traditional 3–4 months before repairing such defects may be both cruel and unnecessary and perhaps should be obsolete.

The risk of perineal infection can be minimised by adopting sound surgical principles, and emphasising the importance of good perineal hygiene such as changing sanitary pads regularly and frequent irrigation with a shower.

There are no randomised trials to establish the efficacy of peripartum antibiotics in preventing perineal wound infections.

6.3 Bowel Problems

6.3.1 Anal Fissure

Anal fissure is an ulcer in the squamous epithelium of the anus located just distal to the mucocutaneous junction; it usually occurs in the posterior midline.[34] The mucosa in this area is sensitive to pain due to its somatic nerve supply (see Chapter 1) and therefore can be extremely painful. Anal fissures are believed to be caused by trauma to the anal mucosa, usually after passage of hard, bulky stools. Constipation is the most common predisposing factor, although diarrhoea may also be a cause.[35,36] Atypical fissures (large, irregular, multiple and non-midline) may be caused by inflammatory bowel disease, local or systemic malignancy, venereal infection, trauma, tuberculosis or chemotherapy.[34]

In a prospective study before and after delivery of 163 consecutive women (84 primiparous), Abramowitz et al.[37] reported anal fissures in 15% during the first 2 months postpartum. Others have reported an incidence of 9% during a 6-week follow-up period of primiparous women.[35] Risk factors associated with the development of anal fissure include dyschezia (painful defaecation), heavier babies, long second stage of labour, anal incontinence after delivery,[37] primiparity, forceps deliveries and perineal damage.[38] Caesarean section did not appear to be protective against anal fissure.[35,37]

Anal fissures typically cause episodic pain that occurs during defaecation and for 1–2 hours afterwards. The diagnosis is suspected from the history and frequently confirmed by visual examination

of the anal margin. The most consistent finding in typical fissures is spasm of the internal anal sphincter, which is so severe that the pain caused by the fissure is thought to be due to ischaemia of the sphincter.[39] Relief of the spasm has been associated with relief of pain and healing of the fissure without recurrence.

Treatment is aimed at relieving constipation using a high fibre diet along with fibre supplements such as Psyllium seed supplement (Fybogel). Iron preparations should be avoided. Local application of lidocaine in the anal canal can provide effective analgesia. After defaecation, water (preferably a shower) should be used to clean the anus rather than wiping with toilet paper to avoid abrasions. Medical treatments are aimed at relaxing the internal anal sphincter. These include nitroglycerin ointment, calcium channel blockers, either given as tablets or applied topically, and injection of botulinum toxin.[34] In a systematic review, nitroglycerin ointment was found to have a healing rate of about 55%. Combining all analyses in which a placebo was used as the comparison group, the healing rate in the placebo group was found to be 35%. In comparisons of nitroglycerin ointment to botulinum toxin injection or calcium channel blockers, no significant difference in efficacy was found. However, in almost 40% of subjects, nitroglycerin ointment was associated with headache often severe enough to stop treatment.[40] Conservative treatment (stool softeners, laxatives and local anaesthetic cream) has been shown to be successful in 97% of postpartum women.[35,36] There are no data regarding the use of medical treatments for anal fissure during pregnancy. A suggested pathway for the management of anal fissures in postpartum women is shown in Figure 6.2.

Non-pregnant women who fail to respond to these conservative measures are candidates for surgical procedures. However, postpartum anal fissures are associated with low anal canal pressures, and surgical interference with the anal sphincter mechanism must be avoided.[35] For those patients requiring surgery for anal fissure, open and closed partial lateral internal sphincterotomy appear to be equally efficacious. It is less clear whether posterior sphincterotomy should be performed as the primary treatment of anal fissure.[41]

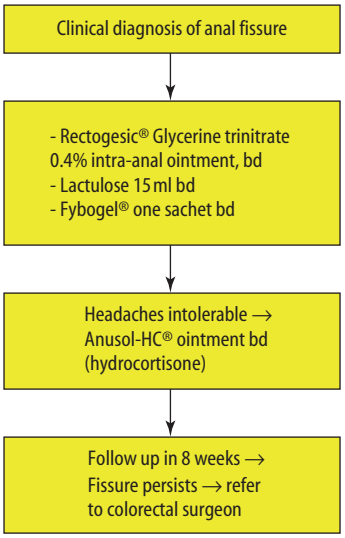

Figure 6.2. Management of anal fissures.

6.3.2 Haemorrhoids

Haemorrhoids (piles) are swollen veins at or near the anus. During bowel evacuation, normal haemorrhoids are compressed and drained, facilitating the emptying of the rectum. Haemorrhoids can become symptomatic if there is damage to their structure and/or alteration in function. Associated factors include straining at defaecation, constipation, vascular enlargement secondary to increased intra-abdominal pressure, erect posture and heredity. Several physiological changes during pregnancy may lead to development of haemorrhoids. Constipation is likely to be due to smooth muscle inhibition by high levels of circulating progesterone or by mechanical obstruction by the gravid uterus. Venous engorgement and dilatation may occur during pregnancy as the circulating blood volume is increased by 25–40%. Hormonal changes result in increased laxity of connective tissue, especially in the pelvis.[36] In the postnatal period haemorrhoids are possibly a consequence of pushing in the second stage of labour. Risk factors include instrumental delivery,[1–3] a longer second stage of labour[2] and vaginal delivery of a heavier baby.[2]

Haemorrhoids may be internal or external. Internal haemorrhoids originate from the internal haemorrhoidal plexus above the dentate line and external haemorrhoids originate from the

external plexus below the dentate line. Internal haemorrhoids may be classified according to the degree of prolapse, although this may not necessarily reflect the severity of the woman's symptoms.

Haemorrhoids are classified as follows:[42]

1. First degree: bleed but do not prolapse.
2. Second degree: prolapse on straining but reduce spontaneously.
3. Third degree: prolapse on straining and require manual reduction.
4. Fourth degree: prolapsed and incarcerated.

In an observational study of 11,701 women, MacArthur et al.[2] found that 8% reported haemorrhoids of more than 6 weeks' duration for the first time within 3 months of birth and an additional 10% reported these as ongoing or recurrent symptoms. Two thirds reported the presence of haemorrhoids 1–9 years after delivery. Glazener et al.[1] found that 17% of postnatal women reported haemorrhoids (new and recurrent) when questioned in hospital, 22% between delivery and 8 weeks postpartum and 15% after 2 months. The problem can manifest in a wide range of symptoms such as a burning sensation, itching, intermittent bleeding of the anus, varying degrees of leakage of mucus, faeces or flatus, sensation of fullness or a lump, perianal hygienic problems, discomfort and/or pain. The most common symptoms during pregnancy and the puerperium are intermittent bleeding from the anus and pain. Depending on the degree of pain, quality of life could be compromised, affecting the activities of everyday life, such as walking, sitting down, emptying bowels, sleeping, or caring for the family or a new baby. Assessment should include anoscopy and a digital examination in the left lateral position.

Treatment during pregnancy is mainly directed to the relief of symptoms, especially pain control. For many women, symptoms will resolve spontaneously soon after birth, and so any corrective treatment is usually deferred to some time after birth.

Complications of haemorrhoids (acute thrombosis, incarceration of prolapsed internal haemorrhoid, unremitting pain) require more aggressive treatment such as closed excisional haemorrhoidectomy under local anaesthetic.[43]

The treatment for haemorrhoids during pregnancy and the postpartum period should be tailored to the severity of the disease and duration of symptoms. External haemorrhoids require treatment only for acute thrombosis. Internal haemorrhoids are best managed conservatively. Management can be broadly classified as conservative, alternative or surgical.[44,45]

6.3.2.1 Conservative Management

Conservative treatment is based on: (a) dietary modifications (high fibre intake, high liquid intake, stool softeners); (b) stimulants or depressants of the bowel transit (depending on whether the woman has constipation or diarrhoea); (c) local treatments (sitz baths, creams, ointments or suppositories containing anaesthetics, anti-inflammatory drugs, steroids, etc., alone or in combination); (d) drugs of the flavonoid family such as rutosides that cause decreased capillary fragility, improving the microcirculation in venous insufficiency. Most minor haemorrhoidal symptoms can be treated this way.

6.3.2.2 Alternative Management

Alternative treatment is required if the haemorrhoids are severe and non-responsive to conservative treatment. It includes a number of ambulatory interventions that usually do not need anaesthetics, such as injection sclerotherapy, rubber-band ligation, cryotherapy, infrared photocoagulation, laser therapy, etc. Injection sclerotherapy has been used effectively during pregnancy. In one study, 86% of antenatal patients (24 of 28) became asymptomatic by means of injection of 5% phenol in almond oil.[45] No complications occurred and six patients required further injection during the ensuing 30 months.

6.3.2.3 Surgical Management

Surgical treatment includes excision surgery and stapled anopexy. These methods are usually a third-line therapy after other treatments have failed. There are no known trials that have specifically evaluated treatments for severe haemorrhoids during pregnancy and the postpartum period.

6.3.3 Anal Incontinence

The diagnosis and management of anal incontinence are covered in Chapters 8, 11, 12a and 12b).

6.3.4 Constipation

About 80% of people suffer from constipation at some time during their lives, and brief periods of constipation are normal. Widespread beliefs, such as the assumption that everyone should have a movement at least once each day, have led to overuse and abuse of laxatives. Some of the most commonly used definitions include infrequent bowel movements (typically fewer than two bowel actions per week), difficulty in defecation (straining at passing stools for more than 25% of bowel movements or a subjective sensation of hard stools), or the sensation of incomplete bowel emptying.

The prevalence of constipation in pregnancy is reported to be 11–38%.[46] The pathogenesis is not fully understood but It appears to be related to the effect of progesterone on gastrointestinal motility as there is an increase in gut transit time in the second and third trimester compared with both the first trimester and the postpartum period. Fibre supplements are effective, and raise no serious concerns about side-effects to mother or fetus. Stimulant laxatives are more effective than bulk-forming laxatives but are more likely to cause side-effects that reduce their acceptability to patients.

Women complaining of constipation in pregnancy can be treated effectively with daily dietary supplements of fibre in the form of bran or wheat fibre. If these are ineffective, stimulant laxatives may be used.

6.4 Bladder Problems

6.4.1 Postpartum Urinary Retention

There is no standardised definition that qualifies postpartum urinary retention. A commonly used symptom-based definition is the absence of spontaneous voiding of urine within 6 hours of delivery. After caesarean section, if a catheter is used, retention is defined as "no spontaneous voiding within 6 hours after removal of the indwelling catheter".[47] Another commonly used definition is based on the post-void residual bladder volume as estimated by ultrasound or catheterisation. Although most experts agree that residual volumes of less than 50 ml are normal and more than 200 ml are abnormal, little agreement exists on the intervening grey zone.[48] Postpartum urinary retention can be classified into covert and overt forms. The covert form is asymptomatic and recognised by demonstrating an elevated post-void residual measurement of more than or equal to 150 ml, with either ultrasound scanning or catheterisation. Clinically overt postpartum urinary retention refers to the inability to void spontaneously after delivery.[49] Risk factors include nulliparity,[50,51] instrumental delivery,[49,50,52] prolonged first and second stage of labour[51,53] and severe perineal trauma.[51,52] Although there is a lack of consensus in the literature regarding the effect of epidural analgesia on postpartum retention,[48] the Royal College of Obstetricians and Gynaecologists guidelines recommend catheterisation for at least 12 hours following regional anaesthesia.[54]

The incidence of postpartum urinary retention ranges between 0.5% and 14% and varies depending on the definition used.[48] The mother will present with an inability to pass urine associated with acute lower abdominal pain. Occasionally she may complain of continuous leakage of urine in the presence of a large palpable bladder. Other presenting symptoms may be hesitancy, slow or intermittent stream, straining to void and a sense of incomplete emptying. On examination of the abdomen, a suprapubic mass will be palpable. A diagnosis can be made after catheterisation or imaging techniques such as an ultrasound. There is no consensus on the minimal urine volume that constitutes postpartum urinary retention as measured on ultrasound.[55] Ultrasound estimation of post-void residual urine in postpartum women can be measured accurately and is not confounded by the enlarged postpartum uterus.

There is no consensus of opinion on the management of postpartum urinary retention[56] and various treatment regimens have been described.[48,49,51,52] Treatment includes general measures such as administration of oral analgesia, helping the woman to mobilise, ensuring privacy

during voiding and having a warm bath. None of the pharmacologic drugs have been studied systematically in postpartum women, as most women would be breastfeeding. If conservative measures fail, it is advisable to insert a urethral catheter and remove it after the bladder has been emptied. If spontaneous voiding fails to occur within 4 hours or if the voided volume is less than 150 ml and/or the post void residual urine is more than 150 ml a Foley catheter should be inserted. A trial without catheter can be attempted after 24–48 hours. The duration of catheterisation is empirical, and no standard has been agreed to. Burkhart et al.[57] found that no postpartum women with a residual urine volume of less than 700 ml required repeat catheterisation, but repeat catheterisation was required in 14% of patients with 700–999 ml of residual urine. If trial without catheter fails, the woman can be taught intermittent self-catheterisation every 4–6 hours until she is able to void and then until the residual is less than 150 ml. If this is not feasible, send her home with an indwelling catheter for 48 hours and repeat the voiding trial. A suggested regimen for management of postpartum urinary retention is shown in Figure 6.3.

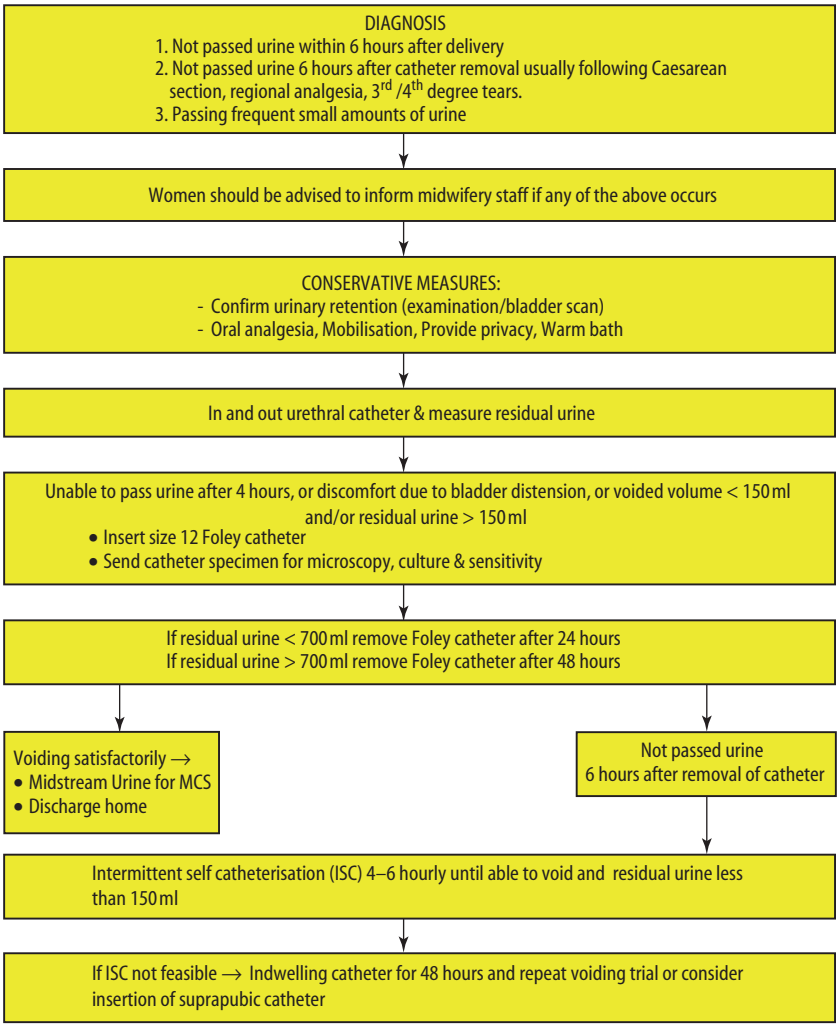

FIGURE 6.3. Management of postpartum urinary retention.

Suprapubic catheterisation may become necessary if the problem becomes chronic and the patient does not wish to per-form intermittent self-catheterisation. Other potential treatment modalities include acupuncture.[58]

Overdistension bladder injury in the postpartum period can be avoided by strict vigilance in ensuring that voiding occurs regularly. Women with potential risk factors, e.g. regional anaesthesia, instrumental delivery, obstetric anal sphincter trauma or severe perineal tears should be catheterised during labour and delivery.

There are very few studies on the sequelae of postpartum urinary retention but published data suggest that this condition returns to normal within a short period and specific treatment is not necessary.[49,50] Yip et al.[59] in a case-controlled study, found that women with postpartum urinary retention did not have a higher incidence of urinary incontinence, frequency nocturia or urgency.

6.4.2 Urinary Incontinence

Childbirth is an established risk factor for urinary incontinence.[60,61] Although vaginal delivery has been implicated as the main contributory factor, the exact mechanism is not known. Pregnancy itself may cause mechanical and/or hormonal changes that can lead to urinary incontinence.

There is evidence that the prevalence of urinary incontinence is highest during pregnancy and decreases in the puerperium.[62,63] Interestingly, stress or urge incontinence during the first pregnancy and puerperium predicts an increased risk of having the symptom 5 years later.[64] In a large community-based epidemiological study, designed to evaluate the risks of incontinence associated with caesarean section and vaginal delivery, Rortveit et al.[65] found that compared to nulliparous women, those who had caesarean section were at higher risk for any incontinence. Vaginal delivery was associated with a greater increase in risk. The risk of moderate or severe incontinence was also higher in the vaginal-delivery group compared to the caesarean section group. As compared with nulliparous status, caesarean section was associated with stress and mixed type of incontinence, whereas vaginal delivery further increased the risk of stress incontinence only. There was no difference in the prevalence of incontinence in women who underwent elective and emergency caesarean section. The authors suggest that this data should not be used as an argument for increased use of caesarean section, as a very large number of women would have to have a caesarean section to prevent urinary incontinence. Moreover, the decrease in incontinence after caesarean section would apply until 50 years of age, since there was no association of incontinence with mode of delivery in the older age groups.

Current evidence suggests that intensive supervised pelvic floor muscle training should be offered to antenatal women[66] and/or to postnatal high-risk women, i.e. instrumental delivery, large baby, prepregnancy and antenatal stress incontinence, persistent postnatal urinary incontinence, family history of incontinence, prepregnancy obesity, increased bladder neck mobility.[66,67] Unfortunately the early benefits do not seem to persist on long-term follow-up, probably due to reduced compliance.[68]

6.5 Sexual Problems

Recent work on women's sexual health after childbirth has shown that sexual health problems are common. In a cross-sectional study of 796 primiparous women over a 6-month period after delivery, Barrett et al.[6] found that 32% had resumed intercourse within 6 weeks of birth and the majority of respondents (89%) had resumed intercourse within 6 months. Sexual health problems, as recalled by women, increased significantly after childbirth. In the first 3 months, 83% had experienced sexual problems, which declined to 64% at 6 months, although not reaching prepregnancy levels of 38%. Dyspareunia in the first 3 months after delivery was significantly associated with vaginal delivery and previous experience of dyspareunia. At 6 months the association with the type of delivery was not significant. However, experience of previous dyspareunia persisted (fourfold) and current breastfeeding was associated with dyspareunia. This suggests that women with dyspareunia prior to pregnancy may have specific needs or issues that could be identified antenatally; appropriate advice and

help could be offered. The response rate of this study was 61%, suggesting that the prevalence of these problems is under-reported. In a further analysis of the same cohort of women, Morof et al.[69] found that women who were depressed were less likely to have resumed sexual intercourse by 6 months postpartum and more likely to report sexual health problems than women who were not depressed. However, sexual health problems were common after childbirth in both depressed and non-depressed women and therefore the authors suggest that postnatal sexual morbidity cannot be assumed to be simply a product of the depressed mental state. In a different study, Glazener[70] found that 53% of women experienced sexual problems in the first 8 weeks postpartum and 49% in the subsequent year. Women who reported perineal pain, depression and tiredness experienced problems related to intercourse more often than those who did not. Although sexual problems are common after childbirth, the proportion of women who ask for help or discuss their problem is low.[6,70] Health professionals should therefore include sexual well-being as a part of a women's overall assessment in the postnatal period, which may well extend beyond the traditional 6 weeks after delivery, as 60% of women may not have resumed intercourse at this stage.

Initial assessment involves a detailed history and examination. In the absence of a local pathology, if a psychosexual problem is suspected, referral to a psychosexual counsellor may be helpful. If localised tender scar tissue is identified, we advise women to have sexual intercourse after insertion of lignocaine gel in the vagina. If unsuccessful, a cocktail (10 ml of 0.5% bupivacaine, 1,500 iu of hyaluronidase and 1 ml of Depo-Medrone) is injected into the perineum at the site of maximal tenderness (Figure 6.1). Occasionally, women may present with a perineal skin web in the posterior fourchette (Figure 6.4) or fusion of the labia (Figure 6.5). These can be divided after infiltrating the area with a local anaesthetic injection. In women who are breastfeeding, dyspareunia may be due to vaginal dryness. New mothers could be alerted to this side-effect and given advice on the use of lubricants and oestrogen pessaries or creams if necessary, while being reassured about the benefits of breastfeeding.

Figure 6.4. Perinal skin tag causing dypareunia.

Figure 6.5. Fusion of the labia.

6.6 Pelvic Organ Prolapse

Injury to the pelvic floor during childbirth is incriminated in the development of pelvic organ prolapse. Other risk factors include defective collagen, race, advancing age, hysterectomy, certain medical illnesses, chronic raised intra-abdominal pressure and states. In a large study in the USA, Swift[71] found an association between gravidity, parity, number of vaginal deliveries, vaginal delivery of a macrosomic infant and an increased pelvic organ prolapse quantification (POPQ) system stage.

In a prospective study conducted to document pelvic organ prolapse throughout the pregnancy, O'Boyle et al.[72] found that the POPQ stage was significantly higher in the third than in the first trimester. They concluded that these findings probably represent normal physiological changes of the pelvic floor during pregnancy, but suggest

that significant changes may be objectively demonstrated prior to delivery. The effect of delivery on pelvic organ prolapse has been investigated by Sze et al.,[73] who found a prevalence of pelvic organ prolapse in 46% of women (26% had POPQ stage II) at the 36-week antepartum visit. Six weeks postpartum, 83% of women developed prolapse; 52% of these had stage II prolapse. This study directly links the development of prolapse to childbirth. However, this study was small and larger studies are needed to investigate the cause and effect of pregnancy and genital prolapse.

The woman usually presents with a feeling of a lump in the vagina, which may or may not be associated with urinary symptoms. In our practice we use the ring pessary as the first option to relieve the symptom of prolapse in the antenatal and postnatal woman with prolapse. If this is uncomfortable or not retained, we teach the woman to insert a cube pessary herself. There is not enough evidence to suggest the best mode of delivery when a pregnant woman presents with prolapse. Sze et al.,[73] in a small study, demonstrated that elective caesarean section is only partially effective in preventing pelvic organ prolapse. After delivery a decision can be made as to whether the woman wishes to continue with conservative management or plan surgery.

6.7 Perineal Clinic

6.7.1 Background

In a survey of 1,249 women in the postnatal period, Glazener et al.[1] found that all 85% of women who reported at least one health problem in hospital received help or treatment for it. Although just as many had problems on discharge home (87%), a small proportion (69%) were treated, and of the 75% who had a health problem after the first 2 months, just over half received treatment. Reasons for the apparent lack of treatment include that it was inappropriate to ask for help or that professional help was not available, unsatisfactory or ineffective.[1] Perhaps it was perceived that health professionals might only treat women with more severe problems[1] or women may feel that the doctors may be unable to treat this condition or that they can cope with the problem themselves.[74] The pattern of decreasing help might be due to the natural resolution of the problem or the decrease in severity with time, but might also reflect a lack of recognition of the impact of continuing maternal morbidity by health professionals and mothers themselves.

As these problems are usually of a sensitive nature, women should ideally be seen in a dedicated clinic instead of a busy general clinic. Furthermore, this environment would facilitate childcare and breastfeeding. The establishment of a dedicated one-stop clinic enables provision of evidence-based quality care, by experienced professionals. A dedicated perineal clinic also provides women with an opportunity to be given an explanation of the circumstances under which the perineal injury occurred, and appropriate counselling regarding mode of subsequent delivery. Due to time constraints and inappropriate expertise, explanation and counselling are often suboptimal in a busy postnatal or gynaecology clinic.

Although perineal clinics are now becoming very popular, there appear to be a variety of models of care. In 2002, Fitzpatrick et al.[75] published their experience of the perineal clinic held in the National Maternity Hospital in Dublin. This was a weekly clinic staffed by an obstetric registrar, continence nurse and medical technician and overseen by a consultant obstetrician and colorectal surgeon. Endoanal ultrasound and neurophysiological tests were reported at a later date by a radiologist and neurophysiologist. However, the clinic was not restricted to women of childbearing age, as women up to the age of 77 were included. In North Staffordshire Hospital (Kettle C, personal communication), the perineal clinic is led by a specialist clinical midwife with access to an obstetrician. Endoanal ultrasound and anorectal physiology tests are performed at a separate visit. Complicated cases are discussed at a multidisciplinary meeting (including midwife, colorectal surgeon, physiotherapist and obstetrician).

6.7.2 The Mayday Perineal Clinic

The perineal clinic at Mayday University Hospital, Croydon, UK is run by a consultant urogynaecologist (trained in anal manometry and ultrasound) and a trained nurse/midwife. We have easy access

Table 6.1. Reason (main symptom) for postnatal referrals ($n = 373$).

	No. of women	%
OASIS	245	65.7
Dyspareunia	17	4.6
Urinary incontinence	10	2.7
Anal incontinence and faecal urgency	23	6.2
Prolapse	10	2.7
Perineal pain	29	7.8
Haemorrhoids	2	0.5
Wound breakdown/infection	21	5.6
Miscellaneous	16	4.3
	373	100

to a physiotherapist, a continence nurse specialist, a colorectal nurse specialist, colorectal surgeons and a psychosexual counsellor. Integration of multidisciplinary professionals promotes a holistic approach to pelvic floor and perineal problems. Furthermore, in this clinic we accept self-referrals and are easily accessible to general practitioners and midwives to allow fast tracking. This clinic is restricted to childbirth-related problems and includes conditions such as dyspareunia, perineal pain, wound breakdown, infection, prolapse, and urinary incontinence that occur during pregnancy and up to 16 weeks after childbirth. Women who sustain obstetric anal sphincter injuries (OASIS) are seen within 3 months postpartum. In addition pregnant women with previous OASIS are evaluated and counselled regarding mode of delivery (see Chapter 4). The clinic is equipped with facilities for endoanal ultrasound scan and anal manometry to facilitate a one-stop approach.

There were 423 new referrals to the perineal clinic from July 2002 to July 2005. Tables 6.1 and 6.2 show the type of patients seen in the clinic.

Table 6.2. Reason (main symptom) for antenatal referrals ($n = 50$).

	No. of women	%
OASIS	39	78
Anal incontinence	8	16
Prolapse	1	2
Urinary incontinence	1	2
Other	1	2
	50	100

6.8 Conclusion

Postpartum problems are clearly an integral part of the care of childbearing women. Ideally, a team of healthcare providers with the knowledge and expertise to care for these women in a single clinic setting should be available to mothers. More research is needed in the management of perineal problems including perineal wounds, OASIS in subsequent pregnancies and perineal pain. A perineal clinic is an ideal setting to advance our knowledge, develop local experts and provide comprehensive care. We have described three models of perineal clinics and each has its merits and limitations. The value of a dedicated perineal clinic is unquestionable but the best model of care will be dependent on local expertise and resources.

References

1. Glazener CM, Abdalla M, Stroud P, Naji S, Templeton A, Russell IT. Postnatal maternal morbidity: extent, causes, prevention and treatment. Br J Obstet Gynaecol 1995;102:282–7.
2. MacArthur C, Lewis M, Knox E. Health after childbirth. London: HMSO, 1991.
3. Brown S, Lumley J. Maternal health after childbirth: results of an Australian population based survey. Br J Obstet Gynaecol 1998;105(2):156–61.
4. Schytt E, Lindmark G, Waldenstrom U. Physical symptoms after childbirth: prevalence and associations with self-rated health. BJOG 2005;112(2): 210–17.
5. Albers L, Garcia J, Renfrew M, McCandlish R, Elbourne D. Distribution of genital tract trauma in childbirth and related postnatal pain. Birth 1999;26(1):11–17.
6. Barrett G, Pendry E, Peacock J, Victor C, Thakar R, Manyonda I. Women's sexual health after childbirth. BJOG 2000;107(2):186–95.
7. Thompson JF, Roberts CL, Currie M, Ellwood DA. Prevalence and persistence of health problems after childbirth: associations with parity and method of birth. Birth 2002;29(2):83–94.
8. Hojberg KE, Salvig JD, Winslow NA, Lose G, Secher NJ. Urinary incontinence: prevalence and risk factors at 16 weeks of gestation. Br J Obstet Gynaecol 1999;106(8):842–50.
9. Foldspang A, Mommsen S, Djurhuus JC. Prevalent urinary incontinence as a correlate of pregnancy, vaginal childbirth, and obstetric techniques. Am J Public Health 1999;89(2):209–12.

10. Wilson PD, Herbison RM, Herbison GP. Obstetric practice and the prevalence of urinary incontinence three months after delivery. Br J Obstet Gynaecol 1996;103:154–61.
11. Sultan AH, Kamm MA, Hudson CN, Thomas JM, Bartram CI. Anal sphincter disruption during vaginal delivery. N Engl J Med 1993;329:1905–11.
12. Abramowitz L, Sobhani I, Ganansia R, Vuagnat A, Benifla JL, Darai E et al. Are sphincter defects the cause of anal incontinence after vaginal delivery? Results of a prospective study. Dis Colon Rectum 2000;43(5):590–6.
13. MacArthur C, Bick DE, Keighley MRB. Faecal incontinence after childbirth. Br J Obstet Gynaecol 1997;104:46–50.
14. Chaliha C, Sultan AH, Bland JM, Monga AK, Stanton SL. Anal function: effect of pregnancy and delivery. Am J Obstet Gynecol 2001;185(2): 427–32.
15. Macarthur AJ, MacArthur C. Incidence, severity, and determinants of perineal pain after vaginal delivery: a prospective cohort study. Am J Obstet Gynecol 2004;191(4):1199–204.
16. Glazener CM. Women's health after delivery. In: Henderson C, Bick D, eds. Perineal care: an international issue, 1st edn. Salisbury: Quay Books Division, MA Healthcare Ltd, 2005, pp 11–17.
17. Sleep J, Grant A. Relief of perineal pain following childbirth: a survey of midwifery practice. Midwifery 1988;4(3):118–22.
18. Dale A, Cornwell S. The role of lavender oil in relieving perineal discomfort following childbirth: a blind randomized clinical trial. J Adv Nurs 1994; 19(1):89–96.
19. Hay-Smith J, Herbison P, Morkved S. Physical therapies for prevention of urinary and faecal incontinence in adults (Cochrane Review). The Cochrane Library, Issue 3, 2004. Chichester, UK: John Wiley & Sons, Ltd.
20. Hedayati H, Parsons J, Crowther CA. Topically applied anaesthetics for treating perineal pain after childbirth. Cochrane Database Syst Rev 2005;2: CD004223.
21. Hedayati H, Parsons J, Crowther CA. Rectal analgesia for pain from perineal trauma following childbirth. Cochrane Database Syst Rev 2003;3: CD003931.
22. Kettle C. Perineal care. Update in Clin Evid 2002;8: 1461–74.
23. Labrecque M, Eason E, Marcoux S. Randomized trial of perineal massage during pregnancy: perineal symptoms three months after delivery. Am J Obstet Gynecol 2000;182(1 Pt 1):76–80.
24. Ridgway LE. Puerperal emergency. Vaginal and vulvar hematomas. Obstet Gynecol Clin North Am 1995;22(2):275–82.
25. Villella J, Garry D, Levine G, Glanz S, Figueroa R, Maulik D. Postpartum angiographic embolization for vulvovaginal hematoma. A report of two cases. J Reprod Med 2001;46(1):65–7.
26. Soltesz S, Biedler A, Ohlmann P, Molter G. [Puerperal sepsis due to infected episiotomy wound.] Zentralbl Gynakol 1999;121(9):441–3.
27. Hausler G, Hanzal E, Dagak C, Gruber W. Necrotizing fasciitis arising from episiotomy. Arch Gynecol Obstet 1994;255:153–5.
28. Glazener CMA. Investigation of postnatal experience and care in the Grampian. PhD Thesis, University of Aberdeen, 1999.
29. Goldaber KG, Wendel PJ, McIntire DD, Wendel GD Jr. Postpartum perineal morbidity after fourth-degree perineal repair. Am J Obstet Gynecol 1993;168(2):489–93.
30. Uygur D, Yesildaglar N, Kis S, Sipahi T. Early repair of episiotomy dehiscence. Aust N Z J Obstet Gynaecol 2004;44(3):244–6.
31. Christensen S, Andersen G, Detlefsen GU, Hansen PK. [Treatment of episiotomy wound infections. Incision and drainage versus incision, curettage and sutures under antibiotic cover – a randomized trial.] Ugeskr Laeger 1994;156(34):4829–33.
32. Hankins GD, Hauth JC, Gilstrap LC III, Hammond TL, Yeomans ER, Snyder RR. Early repair of episiotomy dehiscence. Obstet Gynecol 1990;75(1): 48–51.
33. Arona AJ, al-Marayati L, Grimes DA, Ballard CA. Early secondary repair of third- and fourth-degree perineal lacerations after outpatient wound preparation. Obstet Gynecol 1995;86(2):294–6.
34. Nelson RL. Treatment of anal fissure. BMJ 2003; 327(7411):354–5.
35. Corby H, Donnelly VS, O'Herlihy C, O'Connell PR. Anal canal pressures are low in women with postpartum anal fissure. Br J Surg 1997;84(1):86–8.
36. Medich DS, Fazio VW. Hemorrhoids, anal fissure, and carcinoma of the colon, rectum, and anus during pregnancy. Surg Clin North Am 1995;75(1): 77–88.
37. Abramowitz L, Sobhani I, Benifla JL, Vuagnat A, Darai E, Mignon M et al. Anal fissure and thrombosed external hemorrhoids before and after delivery. Dis Colon Rectum 2002;45(5):650–5.
38. Martin JD. Postpartum anal fissure. Lancet 1953; 1(6):271–3.
39. Schouten WR, Briel JW, Auwerda JJ, Boerma MO. Anal fissure: new concepts in pathogenesis and treatment. Scand J Gastroenterol Suppl 1996;218: 78–81.

40. Nelson R. Non surgical therapy for anal fissure. Cochrane Database Syst Rev 2003;4:CD003431.
41. Nelson R. Operative procedures for fissure in ano. Cochrane Database Syst Rev 2005;2:CD002199.
42. Nisar PJ, Scholefield JH. Managing haemorrhoids. BMJ 2003;327(7419):847–51.
43. Saleeby RG Jr, Rosen L, Stasik JJ, Riether RD, Sheets J, Khubchandani IT. Hemorrhoidectomy during pregnancy: risk or relief? Dis Colon Rectum 1991; 34(3):260–1.
44. Quijano C, Abalos E. Conservative management of symptomatic and/or complicated haemorrhoids in pregnancy and the puerperium. Cochrane Database Syst Rev 2005;3:CD004077.
45. Simmons SC. Ano-rectal disorders in pregnancy. J Obstet Gynaecol Br Commonw 1964;71:960–2.
46. Jewell DJ, Young G. Interventions for treating constipation in pregnancy. Cochrane Database Syst Rev 2001;2:CD001142.
47. Kermans G, Wyndaele JJ, Thiery M, De SW. Puerperal urinary retention. Acta Urol Belg 1986; 54(4):376–85.
48. Yip SK, Sahota D, Pang MW, Day L. Postpartum urinary retention. Obstet Gynecol 2005;106(3): 602–6.
49. Carley ME, Carley JM, Vasdev G, Lesnick TG, Webb MJ, Ramin KD et al. Factors that are associated with clinically overt postpartum urinary retention after vaginal delivery. Am J Obstet Gynecol 2002;187(2): 430–3.
50. Andolf E, Iosif CS, Jorgensen C, Rydhstrom H. Insidious urinary retention after vaginal delivery: prevalence and symptoms at follow-up in a population-based study. Gynecol Obstet Invest 1994;38(1): 51–3.
51. Ching-Chung L, Shuenn-Dhy C, Ling-Hong T, Ching-Chang H, Chao-Lun C, Po-Jen C. Postpartum urinary retention: assessment of contributing factors and long-term clinical impact. Aust N Z J Obstet Gynaecol 2002;42(4):365–8.
52. Glavind K, Bjork J. Incidence and treatment of urinary retention postpartum. Int Urogynecol J Pelvic Floor Dysfunct 2003;14(2):119–21.
53. Yip SK, Brieger G, Hin LY, Chung T. Urinary retention in the post-partum period. The relationship between obstetric factors and the post-partum post-void residual bladder volume. Acta Obstet Gynecol Scand 1997;76(7):667–72.
54. Royal College of Obstetricians and Gynaecologists. Operative vaginal delivery. Guideline no. 26. London: RCOG, 2005.
55. Yip SK, Sahota D, Chang AM. Determining the reliability of ultrasound measurements and the validity of the formulae for ultrasound estimation of postvoid residual bladder volume in postpartum women. Neurourol Urodyn 2003;22(3):255–60.
56. Zaki MM, Pandit M, Jackson S. National survey for intrapartum and postpartum bladder care: assessing the need for guidelines. BJOG 2004;111(8): 874–6.
57. Burkhart FL, Porges RF, Gibbs CE. Bladder capacity postpartum and catheterization. Obstet Gynecol 1965;26:176–9.
58. Yang DL. Acupuncture therapy in 49 cases of postpartum urinary retention. J Tradit Chin Med 1985;5(1):26.
59. Yip SK, Sahota D, Chang AM, Chung TK. Four-year follow-up of women who were diagnosed to have postpartum urinary retention. Am J Obstet Gynecol 2002;187(3):648–52.
60. Rortveit G, Hannestad YS, Daltveit AK, Hunskaar S. Age- and type-dependent effects of parity on urinary incontinence: the Norwegian EPINCONT study. Obstet Gynecol 2001;98(6):1004–10.
61. Chiarelli P, Brown W, McElduff P. Leaking urine: prevalence and associated factors in Australian women. Neurourol Urodyn 1999;18(6):567–77.
62. Viktrup L, Lose G, Rolff M, Barfoed K. The symptom of stress incontinence caused by pregnancy or delivery in primiparas. Obstet Gynecol 1992;79(6): 945–9.
63. Thorp JM Jr, Norton PA, Wall LL, Kuller JA, Eucker B, Wells E. Urinary incontinence in pregnancy and the puerperium: a prospective study. Am J Obstet Gynecol 1999;181(2):266–73.
64. Viktrup L, Lose G. Lower urinary tract symptoms 5 years after the first delivery. Int Urogynecol J 2000; 11:336–40.
65. Rortveit G, Daltveit AK, Hannestad YS, Hunskaar S. Urinary incontinence after vaginal delivery or cesarean section. N Engl J Med 2003;348(10): 900–7.
66. Adult conservative management [computer program]. Paris: Health Publications Ltd, 2005.
67. Freeman RM. The role of pelvic floor muscle training in urinary incontinence. BJOG 2004;111(Suppl 1):37–40.
68. Glazener CM, Herbison GP, MacArthur C, Grant A, Wilson PD. Randomised controlled trial of conservative management of postnatal urinary and faecal incontinence: six year follow up. BMJ 2005;330(7487): 337.
69. Morof D, Barrett G, Peacock J, Victor CR, Manyonda I. Postnatal depression and sexual health after childbirth. Obstet Gynecol 2003;102(6): 1318–25.
70. Glazener CM. Sexual function after childbirth: women's experiences, persistent morbidity and

lack of professional recognition. Br J Obstet Gynaecol 1997;104(3):330–5.
71. Swift SE. The distribution of pelvic organ support in a population of female subjects seen for routine gynecologic health care. Am J Obstet Gynecol 2000; 183(2):277–85.
72. O'Boyle ALO, O'Boyle JD, Ricks RE, Patience TH, Calhoun B, Davis G. The natural history of pelvic organ support in pregnancy. Int Urogynecol J Pelvic Floor Dysfunct 2003;14:46–9.
73. Sze EH, Sherard GB III, Dolezal JM. Pregnancy, labor, delivery, and pelvic organ prolapse. Obstet Gynecol 2002;100(5 Pt 1):981–6.
74. Bick DE, MacArthur C. Attendance, content and relevance of the six week postnatal examination. Midwifery 1995;11(2):69–73.
75. Fitzpatrick M, Cassidy M, O'Connell RO, O'Herlihy C. Experience with an obstetric perineal clinic. Eur J Obstet Gynecol Reprod Biol 2002;100: 199–203.