Pelvic joint pain in pregnancy

The literature pertaining to pain in the symphysis pubis and sacroiliac joints during pregnancy is reviewed here. The signs and symptoms associated with symphysis pubis dysfunction are also discussed.

Pelvic joint pain is a common complaint for pregnant women and as its name suggests encompasses pain in any or all of the pelvic joints. There is very little work focusing on symphysis pubis pain during pregnancy, given that it is a significant problem for many women. Most of the literature addresses “peripartum pain” (pain antenatally, during childbirth or postnatally) and considers the primary problem to be posterior pelvic joint pain with or without anterior (symphysis pubis) pain.

A large number of different names have been used to describe joint pain in the pelvic region during the peripartum period, including symphysiolysis (Schwartz, Katz, & Lancet, 1985), symphysis pubis separation (Taylor & Sonson, 1986), symptomatic symphyseal separation (Kubitz & Goodlin, 1986), pelvic joint instability (Saugstad, 1991), posterior pelvic pain (Ostgaard et al., 1994), diastasis symphysis pubis (Scriven, Jones, & McKnight, 1995), peripartum pelvic pain (Mens, Vleeming, Stoeckart, Stam, & Snijders, 1996; Vleeming et al., 1997), and symphysis pubis dysfunction (Fry, 1999). Some of these acknowledge the involvement of all pelvic joints and others focus on the one which is most affected. The large variety in names probably suggests the diversity in symptoms experienced.

It is difficult to determine the prevalence of pelvic joint pain during pregnancy, as there are so many variations in names and definitions and such a small volume of literature available on antenatal women. The reported prevalence rate of pelvic pain (all types) during pregnancy ranges between 48% and 56% (Mantle et al., 1981; Mens et al., 1997; Bjorklund et al., 1999), with the rate of severe dysfunction ranging from 15% to 22%. In terms of symphysis pubis dysfunction during pregnancy, the only
reported rate was 1:300 (from one hospital) by Kubitz & Goodlin (1986). More recently MacLennan & MacLennan (1997) found that 31.7% of respondents in their retrospective survey of women with symptom-giving pelvic girdle relaxation of pregnancy had antenatal symphysis pubis pain.

The majority of literature pertaining to problems with the symphysis pubis is in the form of case reports of symphysis pubis diastasis (from 1cm to 5.8cm) during childbirth. This is a rare event and does not appear to be related to antenatal symphysis pubis dysfunction (Schwartz et al., 1985; Taylor & Sonson, 1986; Lindsey, Leggon, Wright, & Nolasco, 1988; Dhar & Anderton, 1992; Musumeci & Villa, 1994; Kowalk, Perdue, Bourgeois, & Whitehill, 1996; Kharrazi, Rodgers, Kennedy, & Lhowe, 1997; Snow & Neubert, 1997). Another rare condition is symphysis pubis rupture following a traumatic event during pregnancy (Luger, Arbel, & Dekel, 1995).

Fry (1999) defined the term symphysis pubis dysfunction as the inability of the symphysis pubis to effectively perform its role in pelvic stabilisation, thus altering pelvic biomechanics and impairing efficient weightbearing. The location of the symptoms may suggest that this is a problem of the symphysis pubis only, but as stated previously none of the pelvic joints can be considered in isolation. One theory for symptoms being felt only at this joint, despite the fact that there must be simultaneous involvement of the sacroiliac joints, has been suggested by Vleeming et al. (1992a) to be the result of an unbalanced relaxation (due to relaxin being at different levels) of the sacroiliac joints and the symphysis pubis. Fry (1999) and Sheppard (1997) have suggested that symphysis pubis dysfunction is generally underdiagnosed despite being a significant obstetric problem with consequences for the sufferer being underrated. Conversely Renckens (2000) believed that pelvic instability is greatly over-diagnosed and that symphysis pubis pain is transient and resolves spontaneously after delivery, and Ostgaard (1997) wrote that anterior pelvic problems are caused by posterior pelvic malfunction, and that a thorough examination of the pelvis will detect that symphysis pubis pain is always combined with posterior pain.

The lack of scientific studies investigating symphysis pubis dysfunction makes it difficult to define the type and area of pain experienced. In the National Clinical
Guideline for the Care of Women with Symphysis Pubis Dysfunction, Fry et al., (1997) described the types of symptoms women experience as being mild to severe pain in the pubic region, groin and medial aspect of thighs (unilateral or bilateral); frequently accompanied by sacroiliac, low back and supra-pubic pain. The pain is worst on weightbearing activities (particularly those which involve lifting one leg). Women may also hear or feel a clicking or grinding sensation in the joint, and there is often difficulty walking so a “waddling style” gait is adopted.

Mens et al. (1996) conducted a patient survey of 394 patients with pelvic joint pain including pain in the symphysis pubis region, the groin, the greater trochanter, the region of the sacroiliac joints or the lateral parts of the buttocks. Data were obtained by a questionnaire from members of the Dutch Association for Patients with Pelvic Complaints in Relation to Symphysiolysis, and from responses to an advertisement in a magazine. The findings showed that standing for more than 30 minutes was painful for 90% of respondents, and of 15 listed daily activities (including rolling over in bed, getting in and out of bed, carrying shopping, climbing stairs and walking for more than 30 minutes), 14 were painful in more than 45% of the women. Mens et al’s results showed that pelvic joint pain seriously interfered with many activities of daily living such as standing, walking and sitting. During a subsequent pregnancy, 85% of the women experienced a relapse of the same pain, and 72% experienced a relapse at the time of menstruation.

MacLennan & MacLennan (1997) completed a postal survey of 1115 women (79% response rate) with symptom-giving pelvic joint syndrome of pregnancy and/or postnatal pelvic joint syndrome. The women were all members of the Norwegian Association of Women Suffering from Pelvic Girdle Pain. The commonest sites of pain were the sacroiliac joints and the symphysis pubis but peripheral joints were also often affected. They found that the respondents considered this problem to be severely debilitating and that the problem often continued for many years (mean 6.25 years). The activities that were worst affected were found to be housework, exercise, activities with children, employment, leisure/hobbies and personal relationships or married life. Frequently these effects on the lives of women with symphysis pubis dysfunction were seen as major. Shepherd & Fry (1996) also described women being affected by adverse effects on relationships, decreased ability to care for their baby
and family, stress and depression. The length of time women suffer from this condition is variable, resolution may occur in weeks, months or years (MacLennan & MacLennan, 1997; Scriven et al., 1995).

**Causes of pelvic joint pain**

The primary cause of pelvic joint pain during pregnancy is thought to be instability of the pelvic ring due to hormone induced ligamentous laxity and continuous strain on the ligaments of the pelvis. This continuous strain is as a result of the biomechanical changes associated with the increased anterior weight of the growing foetus. These concepts are reviewed in the following paragraphs.

**The magnitude of symphyseal change**

Relaxation of the pelvic joints is an essential and normal part of pregnancy in order to allow the passage of the baby through the pelvis. X-ray studies in the 1930’s showed that there is normal symphyseal widening associated with pregnancy. Heyman & Lundqvist (1932) used x-ray techniques to show that the average width of the symphysis pubis in 144 asymptomatic pregnant women was 7-8mm. Roberts (1934) reported the average width of the symphysis pubis (from x-ray measurements) to be 2.6mm in both primiparous women (who had never had children, n=59) and multiparous women (who had previously had children, n=71). In pregnant women, the gap was found to be 4.2mm in primiparous women (n=77) and 5.0mm in multiparous women (n=75). Bjorklund, Bergstrom, Lindgren, & Ulmsten (1996) compared ultrasound and x-ray to measure symphysis pubis width in 15 non-pregnant women of fertile age and found measurements ranging from 1.2 to 4.6mm. Farbrot (1952) measured the vertical gap between the upper medial margins of the pubic bones using x-ray in standing on one leg and found that in 97.2% of 211 women who had given birth (often many years earlier) there was vertical stretching. The average distance was 3.1mm. Of the 37 women studied who had not had children there were only three women who showed any vertical displacement at all.
Bjorklund et al. (1999) used ultrasound on 49 pregnant women in a prospective cohort study to determine whether there is a relationship between pregnancy-related pelvic joint pain and degrees of symphyseal laxity. Measurements were taken at 12 and 35 weeks of pregnancy and five months post partum. They also assessed these women for pain in the pelvic joints. The mean width of the symphysis pubis during pregnancy was found to be 3.4mm (SD: 0.8mm) at 12 weeks and 4.8mm (SD: 1.6mm) at 35 weeks. At five months postpartum, the average width had returned to 2.8mm (SD: 1.2mm). There was no evidence that the 49% of women in the study who suffered from pelvic joint pain had wider symphyseal gaps, although the one woman who had a gap of more than ten mm did have pronounced pain. Bjorklund et al concluded that there is a pregnancy induced physiological increase in the laxity of the symphyseal soft tissue, however that there was no evidence that the degree of symphyseal distension determined the severity of pelvic joint pain during pregnancy or after childbirth. Severe dysfunction and pain commonly occur irrespective of clinical evidence of joint dysfunction, and conversely there may be radiological evidence of widening at the top end of the scale that is not associated with clinically severe symptoms (Bjorklund et al., 1999; Ostgaard, 1997).

**Hormonal effect on ligament laxity**

Goldsmith, Weiss, & Steinetz (1995) reported that the involvement of the hormones relaxin, progesterone and oestrogen is frequently cited as being responsible for ligamentous laxity in order to facilitate the opening of the pelvic ring while giving birth. Kristiansson, Svardsudd, & von Schoultz (1996) described relaxin as a peptide hormone of the insulin-like growth factor family, and stated that whilst knowledge of its precise role in humans was still unknown, it has been suggested to have a role in the development of pelvic joint pain in pregnant women by increasing laxity, leading to instability and therefore pain. Like Vleeming et al. (1992a), Wilson (1978) proposed that relaxin has its most marked effect on collagen tissues, and the fact that the dense ligaments of the pelvis are closely packed with collagen cells may explain the presence of relaxin having such a significant effect in this area. However Samuel, Coghlan, & Bateman (1998) showed in rats that the effects of relaxin on the
symphysis pubis were independent of the tissue or species type. These authors noted an increase in water content and a decrease in the overall tissue collagen content, thus allowing the tissue to expand and lose its rigidity.

Kristiansson et al. (1996) showed that serum relaxin levels were highest in early pregnancy, peaking at approximately 12 weeks, then declining until the 17th week and thereafter remaining stable at about 50% of the peak for the rest of the pregnancy. The same authors found that six months post delivery, serum relaxin was not detectable. Goldsmith et al. (1995) found that relaxin is detectable in human milk six weeks postpartum.

Several authors have attempted to correlate relaxin levels with the presence of laxity or pelvic joint pain during pregnancy. Calguneri, Bird, & Wright (1982) showed that ligamentous laxity was increased generally during pregnancy in a study based on finger extension. This was found to be maximal in second and subsequent pregnancies. Schaubeger et al. (1996) measured peripheral joint laxity during pregnancy. The measurements taken were knee hyperextension and anterior cruciate ligament excursion, elbow hyperextension, metacarpophalangeal joint hyperextension, index finger distal interphalangeal joint extension and thumb to forearm stretch. The findings showed that anterior cruciate ligament excursion, knee hyperextension, elbow hyperextension and metacarpophalangeal joint hyperextension had either significantly increased laxity or range of motion throughout the pregnancy with levels of 102 to 215 percent of baseline (which was measured prior to pregnancy) being recorded. These levels continued to rise until two weeks post partum, and had decreased (but not to normal, especially in the knee), by six weeks post partum. They were unable to relate these changes to serum relaxin levels.

Hansen, Jensen, Larsen, Wilken-Jensen, & Petersen (1996) showed no increase in serum relaxin concentrations at 30 and 36 weeks gestation and two and six months postnatally in patients with symphysis pubis pain versus controls. Conversely, in a study involving 35 women, MacLennan, Nicolson, Green, & Bath (1986) suggested that there is a positive relationship between high mean serum relaxin levels, joint instability and pelvic pain. Similar results were obtained by Kristiannson et al. (1996) who undertook a prospective cohort study with serial measurements of back
symptoms, clinical back status and hormone levels in pregnant women sampled from the general population of pregnant women. They found a positive correlation between relaxin levels and pain in the pelvic area, and found similar results from a further study (Kristiansson, Svardsudd, & von Schoultz, 1999).

Hence there is currently no agreement on the significance of relaxin in the development of pelvic pain. The onset of symptoms (which varies but is often in the second or third trimester) does not match the levels of hormone present, so it would seem that there are additional factors involved in the aetiology of pelvic joint problems, including symphysis pubis dysfunction, during pregnancy.

**Biomechanical changes**

In advancing pregnancy, the anterior growth of the foetus results in the centre of gravity of the mother moving anteriorly. Vleeming et al. (1995b) described the change in posture as follows: the pregnant woman tries to compensate for anterior weight increase by leaning backwards to regain equilibrium, thus there is anterior iliac rotation which results in relative counterrotation of the sacrum and hence instability. Due to the dorsal position of the trunk relative to the pelvic girdle, the function of the gluteal muscles is decreased and therefore the woman walks with a waddling gait (Abitbol, 1997).

The growing weight of the uterus and foetus combined with the effect of gravity can result in increased downward force on the pelvic ring. However, this also does not appear to be the primary cause of ligamentous laxity, as pain may begin in the first trimester and does not increase in incidence significantly towards the end of pregnancy as would be expected if the increasing weight of the foetus was the cause of the pain (Mens et al., 1996). Related to the possibility of increased weight of the foetus being a factor, Ostgaard (1997) reported from a review of postnatal literature that peripartum pelvic pain may be associated with the weight of the newborn, that is, heavier babies may lead to more pain.
Vleeming et al. (1995b) stated that nutation of the sacrum is painful when the symphysis pubis is painful because it involves compression superiorly and stretching inferiorly. These authors suggested therefore that when there is symphysis pubis pain, the woman may try to adopt a counternutated position in order to avoid nutation because of this inferior ligamentous stretching. This position is unstable and will thus result in further pain by increasing the instability. This is another reason put forward for the waddling style of gait being adopted. That is, it tends to further anteriorly rotate the ilia, thus enhancing counternutation of the sacrum (Vleeming et al., 1997).

**Changes in muscle during pregnancy**

It is commonly suggested in clinical settings (Gleeson & Pauls, 1988) that relaxin has an effect on muscle as well as ligamentous tissue, however a search of the literature has failed to find any evidence to support this conjecture. Very little research describes any muscular changes during pregnancy. The most obvious influence of pregnancy on muscle is the lengthening of the abdominal wall. Due to the location of this muscle group it is affected by the increasing size of the growing uterus, which, as it expands, is accommodated by gross changes in the abdominal wall anatomy (Bullock-Saxton, 1998). The postural changes discussed previously that are associated with pregnancy may also have an affect on the function of muscles. Gilleard & Brown (1996) investigated the structure and function of the abdominal muscles during pregnancy. These authors carried out a study of the abdominal muscles, using rectus abdominis as a representative abdominal muscle (assuming that the adaptation of one muscle does not occur in isolation from other muscles). In order to investigate structural changes, the variables measured were the separation width (diastasis), muscle length and angle of insertion at superior and inferior attachments. Three dimensional photography of abdominal skin markers was used to perform this procedure. Secondly, the functional abilities of the muscles were examined by the ability to perform a curl-up and a posterior pelvic tilt against resistance, and the relationship over time between abdominal muscle function and musculoskeletal adaptations to pregnancy determined. Six primigravid subjects volunteered to be involved in the study. The results showed increases in rectus abdominis muscle separation width, length and angles of insertion as the pregnancy progressed.
Abdominal muscle function decreased with time as the muscle lengthened. The maximum mean normalised length of rectus abdominis was 115% at 38 weeks gestation. These authors assumed that this was a reflection of muscle fibre length increase. In terms of functional ability, by 26 weeks gestation the ability to perform a curl-up exercise had diminished for all subjects and was impossible for all subjects by 38 weeks gestation. These authors also found a decreased ability to perform a posterior pelvic tilt against resistance as pregnancy progresses. Women in this study were fit and exercising throughout pregnancy, therefore results may not be able to be transferred to women who were not fit initially.

The effect of pregnancy on other skeletal muscle connective tissue in humans is unclear. Gillear & Brown (1996) showed that during pregnancy the abdominal muscle lengthening occurs over a period of approximately 22 weeks and therefore the stretch is applied over time. Williams & Goldspink (1971) showed in mice that skeletal muscle fibres add sarcomeres to their length when stretched over time. Conversely shortened muscles show a decrease in sarcomere numbers. These authors also showed that the maximum active tension increased in lengthened muscle and was generated at the new muscle length as the muscle grew. Herring, Grimm, & Grimm (1984) also reported that normal linear growth of vertebrate skeletal muscle fibres proceeds by the serial addition of sarcomeres, and that sarcomere number is highly adjustable (can either increase or decrease). Regulation of sarcomere number is generally considered an adaptation to functional conditions, particularly changes in muscle length. In light of this insight, Gillear & Brown (1996) suggested that the long term stretch in rectus abdominis during pregnancy may increase muscle length but at the same time maintain maximum active tension. They concluded that the increase in rectus abdominis muscle length was unlikely to greatly reduce the ability to produce tension within the muscle. Hence any functional deficits of the abdominal muscles during pregnancy may result from other factors such as altered line of action rather than from overstretched and thinning.

Fast, Weiss, Ducommun, Medina, & Butler (1990) investigated the effect of abdominal muscles and sit-up performance on low back pain during pregnancy. Three hundred and twenty-eight pregnant women were subjects in this study and all had their rectus abdominis length measured using the direct linear transformation
method for three dimensional photography to establish the position in space of a series of nine measurement points over rectus abdominis. All subjects were asked to perform one of three types of sit-up. They reported that abdominal muscles during the third trimester of pregnancy were weakened relative to the abdominal muscles of non-pregnant subjects. These authors found no statistically significant correlation between sit-up performance and back-ache.

Hence, there is some evidence, albeit limited, to suggest that muscles lengthen but that this lengthening is not necessarily responsible for a change in muscle function. There is a gap in the literature regarding the effect of pregnancy on skeletal muscles in general.

**Other factors causing pelvic joint pain**

Whilst it is generally agreed that the reasons for the temporary dysfunction of the ligamentous system during pregnancy are largely hormonal and related to the increased weight and altered centre of gravity, other contributing factors have been suggested. Mens et al. (1996) used a questionnaire to collect data from 394 members of the Dutch Association for Patients with Pelvic Complaints in Relation to Symphysiolysis. Of the women surveyed 69.9% had their pain labelled as symphysiolysis. They found that women with twin pregnancies were significantly more likely to have peripartum pelvic pain (3.3% of this group compared with 1.4% frequency of twins in the general population). Height and weight of women within the study were similar to that of other pregnant women, suggesting no effect of these factors on pain. The average age of all women with peripartum pelvic pain was 28.6 +/- 3.7 years, and of primiparous women with pain was 27.9 +/- 3.6 years, both of these being 0.5 years older than the values for the Dutch pregnant population.

In a survey of 153 members of the Norwegian Association for Women suffering from Pelvic Pain and Pelvic Joint Instability, Saugstad (1991) found that onset of pain was significantly earlier (16.3 weeks) in users of oral contraceptives than non-users (20.5 weeks). MacLennan & MacLennan (1997) surveyed 1609 women from the same association and suggested a strong family history of pelvic joint syndrome was also
apparent. These authors raised the possibility of genetic inheritance since the prevalence varied widely in different populations (although this may be related to diagnostic criteria).

Heiberg & Aarseth (1997) surveyed 5438 women in Norway between one and four days postpartum (87.2% of women who gave birth during the study period). They reported that women with one child had higher rates of peripartum pelvic pain than those with no previous children (47.7% compared with 32.9% for primiparous women). This increased to 54.4% for women with two previous children. They also suggested that the severity was worse in the women with more children. Fifteen percent of women with two previous children, and 11.1% with one child reported great difficulty with housework compared with 5.3% for women with no children. Furthermore, in women with no children, the onset of peripartum pelvic pain occurred later in the pregnancy.

REFERENCES


