Pregnancy and childbirth: the effects on pelvic floor muscles

26 February, 2009

Stress incontinence can follow childbirth as pelvic floor muscles are damaged

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ABSTRACT

This article examines the risks of damage to the pelvic floor that are associated with pregnancy and delivery. It will aim to dispel some myths about pelvic floor exercises and pregnancy and will look at the latest recommendations about pelvic floor muscle exercise.

During pregnancy there are outward signs that the body is changing but changes that occur to the pelvic floor muscles and the nerves and soft tissues that support them are less obvious.

In recent years, there has been an increasing interest in the effect of childbirth on continence.

Wall (1999) suggests that we now have an increased awareness of the effects of obstetric trauma because modern obstetric care has reduced maternal deaths and women now survive difficult or obstructed deliveries. As a result of their obstetric trauma, these women have an increased risk of developing pelvic floor muscle dysfunction.

The physiological changes occurring during pregnancy and the processes of childbirth have a detrimental effect on the structure and function of the muscles, nerves and fascial tissues (connective tissue) that make up the pelvic floor complex.

Dysfunction of the pelvic floor complex can result in a wide range of symptoms including urinary or anal incontinence. Symptoms of pelvic floor dysfunction are listed in Box 1.

Box 1. Symptoms of pelvic floor dysfunction

Lower urinary tract

• Urinary incontinence
• Urgency and frequency
• Slow or intermittent stream and straining
• Feeling of incomplete emptying

Bowel
Several epidemiological studies have shown that urinary incontinence, in particular stress urinary incontinence, is strongly associated with pregnancy and childbirth. The prevalence of stress urinary incontinence increases during pregnancy and declines after delivery (Morkved, 2007).

As the baby grows, the weight of the baby and the gravid uterus (pregnant uterus) produce anatomical changes to the bladder and urethra. Studies using ultrasound imaging techniques have shown that the angle between the bladder neck and the urethra increases, producing an increased opening of the bladder neck. There is also an increased mobility of the bladder due to the hormonal changes of pregnancy which also affect the pelvic floor complex.

A study of 184 first-time mothers who delivered by Caesarean section and 100 who delivered vaginally (Lal et al, 2003) found that there was no significant difference in the prevalence of symptoms of incontinence 10 months following delivery. This study supports the argument that pregnancy is the cause of incontinence for many women irrespective of their mode of delivery.

These findings confirmed those of MacLennan et al (2000) who reported the results of a survey of 4,400 households, randomly selected and questioned about pelvic floor disorders. This study concluded that women having Caesarean section were just as likely to have symptoms of incontinence than those having a vaginal delivery.

Lal et al (2003) also suggest that changes occur to the properties of collagen and other connective tissues during pregnancy. When these changes are combined with possible inherited susceptibilities, anal incontinence can occur as a result of the pregnancy alone, regardless of delivery mode.

In a prospective study of 363 women up to one year after delivery, Groutz et al (2004) found that a similar percentage of women who had vaginal deliveries and Caesarean section for obstructed labour developed stress urinary incontinence (10.3% and 12% respectively). A significantly lower percentage (3.5%) of women developed stress urinary incontinence following an elective Caesarean section with

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no trial of labour. Although this study demonstrates that Caesarean section protects the pelvic floor from injury, it also supports the theory that some changes to the function of the pelvic floor must occur as a result of pregnancy.

This view is also supported by van Brummen et al (2007) in a prospective study of 344 women expecting their first baby. This study compared the prevalence of postpartum stress urinary incontinence and urge incontinence in women who had had a Caesarean section with those who had had a vaginal delivery. They also noted that the presence of stress urinary incontinence in early pregnancy was a strong predictor of it persisting in the year following delivery in both groups.

When women are advised about mode of delivery, consideration should be given to the morbidity associated with Caesarean section such as increased risks of postpartum haemorrhage, infection, pulmonary embolism and ileus. Sultan and Stanton (1996) suggest that the risk of serious maternal morbidity is considerably lower following vaginal delivery compared with Caesarean section and that the risk of hysterectomy secondary to haemorrhage is 10 times higher following Caesarean section than with vaginal delivery.

Influence of general health

A pregnant woman’s general health may influence the likelihood of developing incontinence.

Obesity in pregnancy has been suggested as a possible risk factor for postpartum stress urinary incontinence (Ramussen et al, 1997; Wilson et al, 1996). However, in their study of 304 women following childbirth, Chiarelli and Campbell (1997), did not report a link to obesity.

In studies not specifically related to pregnancy there has been a definite link demonstrated between obesity and pelvic floor dysfunction (Kapoor and Freeman, 2008).

Chiarelli and Campbell (1997) suggest that women with a chronic cough during pregnancy could have four times the risk of developing incontinence. They also report that there is an fourfold increased risk of incontinence for pregnant women who have had a previous vaginal delivery and a 10-fold increased risk for those who have had a previous forceps delivery.

Initial strength of the pelvic floor muscles also influences the development of incontinence during pregnancy (Morkved et al, 2003).

The pelvic floor and childbirth

Muscles and fascia

The ability of the pelvic floor to contribute to the maintenance of both urinary and anal continence is undoubtedly threatened by the processes of vaginal delivery. As the baby emerges, the widest part of the babies’ head stretches the pelvic floor muscles, fascia and nerves – this stage of labour is referred to as the ‘crowning’ of the baby’s head.

It is likely that stretching and possible tearing of the endopelvic fascia and muscles and associated trauma to the pudendal nerves may cause pelvic floor dysfunction (Kapoor and Freeman, 2008).

The development of ultrasound techniques has enabled a better understanding of the nature of fascial injuries during delivery. Dietz and Lanzarone (2005) demonstrated that up to one-third of women following vaginal delivery experienced avulsion (tearing) of the fascia supporting the pelvic floor muscles which was associated with postpartum stress urinary incontinence three months following delivery.

Kapoor and Freeman (2008) suggest that fascial tears have the ability to heal but it is thought that the resulting connective tissue will not be as strong as the original and that the woman will be more likely to experience symptoms of pelvic floor dysfunction such as incontinence or pelvic organ prolapse. Such symptoms may become more evident later in life as a result of the ageing process and hormonal
changes at menopause that affect the already weakened fascia.

Some women may be predisposed to developing pelvic floor dysfunction because of a familial deficiency in their collagen type. Keane et al (1997) have shown that premenopausal nulliparous women with stress urinary incontinence have weaker pelvic floor collagen than control groups containing premenopausal nulliparous women without stress urinary incontinence. They suggest that there may be a group of women with congenitally weak connective tissue and fascia who might be at risk of stress urinary incontinence and pelvic organ prolapse as a result of pregnancy.

Injuries to the pelvic floor muscles have been classified by the Royal College of Obstetricians and Gynaecologists (RCOG, 2007). This guideline document on the management of third and fourth-degree tears following vaginal delivery, divides muscle tears into second, third and fourth degrees. (Box 2). First-degree tears are not included in this guideline because it concerns pelvic floor muscle injury whereas a first-degree tear involves only the skin covering the muscles.

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<td>• Second degree: injury to the perineum involving perineal muscles but not involving the anal sphincter</td>
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| • Third degree: injury to the perineum involving the anal sphincter complex (external anal sphincter (EAS) and internal anal sphincter (IAS)):
  3a: less than 50% of EAS thickness torn
  3b: more than 50% of EAS thickness torn
  3c: IAS torn |
| • Fourth degree: injury to perineum involving the anal sphincter complex (EAS and IAS) and rectal mucosa |

Source: RCOG (2007)

It is difficult to determine the exact prevalence of obstetric injuries because of the difficulty in diagnosing the degree of tear immediately following delivery because tissues are swollen and deformed.

**Nerve injury**

A number of studies have suggested that stress urinary incontinence following delivery is due to damage to the peripheral nerves (Snooks et al, 1990, 1984; Allen et al, 1990). Comparison of pudendal nerve function prior and post delivery suggests that the cause of denervation (loss of nerve supply) occurs during the second stage of labour (Sultan et al, 1994).

Allen et al (1990) demonstrated pudendal nerve denervation in 80% of women following their first vaginal delivery. In this study, the risk factors for this nerve injury were a prolonged active second stage of labour and heavier babies.

In many women, the nerve damage occurring at delivery may persist and the associated pelvic floor muscle dysfunction may become worse over time (Snooks et al, 1990).

Nerve injury may be down to the significant difference between the ability of nerve tissue to elongate compared with muscle or fascial tissues. Nerves have been shown to be able to elongate by 6–22% of their initial length before damage occurs whereas skeletal muscle has been shown to sustain distension by a factor of up to 200% (Morkved, 2007).

**Risk factors for obstetric trauma**

The update of the RCOG guidelines for the management of obstetric injuries has listed a number of risk
factors that may contribute to increased risk of obstetric anal sphincter injuries. These are (RCOG, 2007):

- Birthweight over 4kg;
- Induction of labour;
- Epidural analgesia;
- Persistent occipito-posterior position (baby positioned back to back with mother – commonly called posterior presentation);
- Episiotomy;
- Second stage of labour longer than one hour;
- Shoulder dystocia (delivery that requires additional obstetric manoeuvres to release the baby’s shoulders);
- Nulliparity (primigravida);
- Forceps delivery.

The predominant recurring risk factors in the literature are:

- Operative delivery (forceps);
- Nulliparity (primigravida);
- Birthweight over 4kg;
- Prolonged second stage of labour.

The length of the active second stage of labour has in the past been considered prolonged if it is greater than one hour. However, in a recent paper, Scheer et al (2008) suggested that an active second stage greater than 50 minutes is significant for obstetric and sphincter injuries (OASI). They also suggest that there is a strong relationship between OASI and symptoms of urinary incontinence.

Other risk factors highlighted in various studies but not thought to be as significant as those in the RCOG guideline are:

- Ventouse delivery;
- Women who have gone past expected date of delivery;
- Women who do not cooperate with instructions during delivery, in particular pushing when the head is crowning;
- Ethnic background – some minorities appear to be more predisposed to pelvic floor muscle dysfunction (Christianson et al, 2003; Gupta et al, 2003; Carroli and Belizan, 2000; Thacker and Banta, 1983).

The RCOG (2007) guideline acknowledges many of these risks, such as nulliparity, posterior presentation and induction of labour, cannot be avoided but suggests more research is needed to cut or prevent third-degree tears in at-risk patients.
Williams et al (2005) also acknowledges that some risks cannot be avoided and that, in many cases, OASI, is ‘neither predictable nor preventable’. They conclude that clinical effort would be best placed in using evidence-based practice to ‘optimise the outcome after repair’.

The relationship between birth position, perineal trauma and women’s experience is thought to be significant. Although somewhat tentative, there is evidence that certain birth positions (lateral) are better than others (squatting) in reducing the risks of trauma to the perineum (Hastings-Tolsma et al, 2007; Shorten et al, 2002). These papers suggest that women should be allowed to make decisions about delivery in partnership with midwives and obstetricians who are equipped with the knowledge of risks and benefits of options and skills to implement women’s choices for birth.

Dispelling old (mid)wives’ tales?

Midwives sometimes tell women that strong pelvic floor muscles can cause problems at delivery.

The evidence is that having healthy, strong pelvic floor muscles does not lengthen the second stage of labour (Agur et al, 2008; Morkved, 2007). It has been suggested that training the pelvic floor muscles in pregnancy can facilitate labour (Salvesen and Morkved, 2004), although this was not supported in the study by Agur et al (2008).

Stop mid-stream test

There is confusion about whether or not to advise women to ‘stop’ their urine midstream. While this may be considered a good method of identifying the correct action of the pelvic floor muscles, it is often confused with being an exercise for them.

Concerns have also been expressed that this interruption of the flow of urine could irritate the bladder (Bump et al, 1991). It can also cause incomplete emptying of the bladder which increases the risk of urinary tract infection.

Women should be taught to identify their pelvic floor muscles by palpating the skin over the perineum and feel for a movement lifting upwards and forwards towards the bladder when the muscles contract. Alternatively, they can insert a finger or thumb into the vaginal opening and feel the contraction of the muscles as they move upwards and forwards towards the bladder.

Conclusion

With a greater understanding of the function of the pelvic floor muscles and risk factors for trauma and damage as a result of pregnancy and birth, healthcare professionals will be better able to meet the needs of women in the childbearing year.

Recommendations for practice

- Pelvic floor muscles and their associated structures are at risk of becoming weakened during pregnancy or of experiencing trauma and damage during delivery

- This problem is identified by the NICE (2006) guidance Urinary Incontinence: the Management of Urinary Incontinence in Women. It states: ‘Pelvic floor muscle training should be offered to women in their first pregnancy as a preventive strategy for urinary incontinence. There is evidence that pelvic floor muscle training used during a first pregnancy reduces the likelihood of postnatal urinary incontinence’ (NICE, 2006)

- Incontinence is still a taboo subject and reluctance by both pregnant women and healthcare professionals to discuss it can make it difficult to fulfil the NICE recommendations

- PromoCon, a charity led by the Disabled Living Centre that focuses on the promotion of continence and management of incontinence, has developed a risk-assessment tool that can be
used to help midwives raise the issue of incontinence and pelvic floor muscle health at the first booking appointment (Pearl and Herbert, 2008)

- This tool can also be used following delivery to assess the risk of pelvic floor muscle dysfunction by considering antenatal risk factors in addition to factors occurring as a result of the delivery

References


Messelink, B. et al (2005) Standardization of terminology of pelvic floor muscle function and dysfunction: report from the pelvic floor clinical assessment group of the International Continence...


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