كلية : كلية الطب العام

القسم او الفرع : النسائية والتوليد

المرحلة: الرابعة

أستاذ المادة : م.د.آلاء شلال فرحان

اسم المادة باللغة العربية :التوليد

اسم المادة باللغة الإنكليزية : Obstetrics

اسم المحاضرة الثالثة عشر باللغة العربية: الولادة المبكرة وربط عنق الرحم

اسم المحاضرة الثالثة عشر باللغة الإنكليزية :Preterm labour and cerclage

محتوى الحاضرة الثالثة عشر

Preterm labour and cerclage

Preterm labour (PTL) is the onset of labour before 37 weeks' gestation. For reasons related to aetiology, outcome and recurrence risk, preterm births should be divided into three gestational periods:

1.Mildly preterm births at 32.0 to 36.6 weeks (incidence 6.1 per cent).

2.Very preterm births at 28.0 to 31.6 weeks (incidence 0.9 per cent).

3.extremely preterm births at 24.0 to 27.6 weeks (incidence 0.4 percent)

Epidemiology

Worldwide, 15 million babies are born preterm every year and of these 1.1 million will die. Of the survivors, many will be left with lifelong disability. The vast majority of these births occur in countries with emerging economies (where PTL is up to three times more common and the prognosis of those born preterm far worse. In high-income countries, a baby born at 24 weeks has a 50% chance of surviving. In low-income countries, a similar survival rate is not achieved until 34 weeks.

PTD can be categorized into spontaneous PTL, PPROM and delivery for maternal or fetal indications. Approximately 25% of PTDs are for maternal or fetal indications, 50% follow spontaneous PTL and 25% follow PPROM.

Iatrogenic or medically-indicated deliveries are typically for diagnoses such as pre-eclampsia, fetal growth restriction (FGR) and maternal cardiac or renal conditions. The risk of PTD is greater in teenagers and women with advanced maternal age, and there is a higher incidence of preterm deliveries in first pregnancies. Socioeconomic factors, marital status, environmental stress, cigarette smoking, illegal substance (i.e. cocaine) abuse, alcohol and poor nutrition have all been linked to an increased risk of preterm birth. Intervention studies have shown that smoking cessation programs can reduce the risk of preterm birth.

The incidence of PTDs is greater in African or Afro-Caribbean women, but it is difficult to differentiate between social deprivation and genetic variation.

Specific genetic polymorphisms have been recently linked with increased risk of PTL (rarely), suggesting that genetic as well as environmental factors can explain increased rates of preterm birth in specific ethnic groups.

The rate of preterm birth overall has risen progressively from the 1960s, when rates were in the region of 6%, to the early years of the new millennium, when the rates peaked at around 10%. More recently, rates have stabilized and even declined in some countries.

Neonatal outcomes have improved too, with better survival and reduced morbidity, but rates of neonatal mortality and morbidity remain considerable. Neonatal care will continue to advance, but to improve these figures substantially we have to work to prevent preterm birth.

Improvements in other areas of paediatric care mean that PTL is now not only the most important cause of perinatal morbidity and mortality worldwide, but also of infant mortality less than 5 years of age. The World Health Organization (WHO) stated in 2010 “The largest barrier to the development of diagnostic, treatment and prevention strategies for preterm birth and stillbirth is our inability to comprehend the biological processes of pregnancy and childbirth”. This statement is as true now is was then; substantial work needs to be performed to achieve an improvement in pregnancy outcomes worldwide.

Endocrinology and biochemistry of preterm labour

The mechanisms that control the length of human pregnancy and signal the onset of labour have been the subject of extensive research, but are not yet determined.

During pregnancy the uterus undergoes marked biochemical and physiological changes while expanding to accommodate the growing fetus and remaining quiescent. At the same time the cervix remains rigid and closed to retain the developing fetus within the uterus. Throughout pregnancy, ‘pro-pregnancy’ factors such as progesterone, relaxin, human chorionic gonadotrophin (hCG) and prorelaxation prostaglandins (PGs), such as prostacyclin, inhibit myometrial contractility.

The onset of labour involves the synchronization of myometrial activity through greater expression of gap junctions that connect myometrial cells. Labour onset is diagnosed by the occurrence of painful uterine contractions with changes in the structure of the cervix, leading to cervical dilatation and effacement. It is a gradual process that begins several weeks before delivery with changes in the lower pole of the uterus, which cause cervical ripening and effacement.

The changes in the cervix occur through breakdown of collagen, changes in proteoglycan concentrations, infiltration of leucocytes and macrophages and an increase in water content. Increased myometrial activity results from the activation of a ‘cassette of contraction-associated proteins’ (CAPs), which convert the myometrium from a quiescent to a contractile state. CAPs include gap junction proteins, oxytocin and prostanoid receptors, enzymes for PG synthesis and cell signalling proteins. The latter mediate the uterine response to receptor activation.

Progesterone maintains uterine quiescence

Progesterone is considered to play a major role in the maintenance of pregnancy.
In most pregnant mammals, the onset of parturition is associated with a decrease
in circulating progesterone through a variety of mechanisms including corpus
luteum lysis in rodents and a reduction in placental progesterone synthesis in the
sheep. However, in humans the levels of circulating progesterone remain elevated
throughout pregnancy, but the administration of the progesterone receptor antagonist RU486 can induce labour. This suggests that although progesterone is
involved in pregnancy maintenance, the onset of human labour either involves a
functional withdrawal of progesterone action or occurs independent of any loss in

Labour as an inflammatory process

Consistent with the theory that progesterone action is repressed by inflammation, several studies have shown that human labour is associated with a global increase in a number of proinflammatory factors including PGs, cytokines and chemokines.

Elevated levels of interleukin-1β (IL-1β), IL-6 and tumor necrosis factor-α (TNFα) have been shown in amnion, myometrium and choriodecidua. The influx of inflammatory cells such as neutrophils, macrophages and T-lymphocytes into the labouring uterine tissues may result in the increased levels of proinflammatory cytokines. In addition, inflammation has been strongly implicated in infection driven preterm labour.

The roles of oxytocin and prostaglandins

The oxytocin/oxytocin receptor (OTR) system within the pregnant uterus serves two distinct physiological functions, stimulation of contractions and production of PGs. There is no increase in the production of oxytocin associated with the onset or progression of labour; however, the sensitivity of the myometrium to oxytocin at term increases dramatically and this is mediated via increased expression of OTR. Activation of OTR triggers the release of intracellular Ca^{2+}, leading to a calmodulin-mediated activation of myosin light chain kinase, which phosphorylates myosin, promoting the interaction with actin and the onset of contractions.

PGs are derived from arachidonic acid (AA), which is found in cell membrane phospholipids. They play a key role in the onset and progression of human labour, promoting cervical ripening and myometrial contractility. Their importance is shown by the widespread use of different formulations of PGE to induce labour and the success of PG inhibitors in the inhibition of PTL. However, in the case of the latter, their use is limited by their adverse fetal effects (premature closure of the ductus arteriosus and renal impairment). The PG family is large with procontractile ($PGF_{2\alpha}$) and prorelaxatory (PGI_2) members.

Causes of preterm labour

PTL is not a single condition; it has multiple causes. Many risk factors have obvious biophysical correlates; multiple pregnancy is associated with prematurity through its effect on myometrial stretch. However, in many other cases this is not clear and more work needs to be performed to understand the mechanisms involved.

Cervical weakness

Cervical weakness is classically associated with painless premature cervical dilatation and is suggested by a history of painless second trimester pregnancy loss. There is almost certainly an overlap between cervical weakness and other factors such as ascending infection, as during pregnancy, the cervix not only acts as a physical obstacle, keeping the pregnancy in the uterus, but also as a barrier to ascending infection through the synthesis of a thick mucus plug in the cervical canal that has bactericidal properties. Several studies have demonstrated a strong relationship between cervical length and the risk for PTL, and a previous history of cervical surgery is a common risk factor for cervical weakness.

Infection

Infection of the fetal membranes, chorioamnionitis, is a major cause of preterm birth particularly in deliveries before <32 weeks' gestation. It is associated with a threefold increased risk of PTL with intact membranes, and a fourfold increased risk with ruptured membranes. In most cases, infection ascends from the vagina, although the route of infection may be transplacental or introduced during invasive procedures. Cervical weakness, resulting in early shortening, as described earlier, can predispose to ascending bacterial infection. However, it is possible for vaginal pathogens to ascend through a normal cervix.

Overall, 33% of all pregnancies delivered after PPROM are complicated by infection. This rate rises the earlier the PPROM occurs, with positive amniotic fluid cultures found in 83% of babies delivered weighing <1 kg, which approximates to a gestation of less than 28 weeks. Abnormal vaginal flora, for example bacterial vaginosis (BV), affects 16% of pregnant women and is associated with PPROM and PTL, with a greater risk the earlier in gestation it is identified. The relationship with PTL is not direct, since antibiotic treatment of BV does not consistently reduce the risk of PTL. Current recommendations are to screen high-risk women for BV and treat those found to be positive.

Chorioamnionitis not only drives PTL, but it is also associated with fetal brain damage, since intrauterine infection drives a fetal inflammatory response, involving a proinflammatory cytokinaemia and, morphologically, a vasculitis of the umbilical cord and/or the vessels of the chorionic plate. The release of inflammatory cytokines during maternal infection is harmful to the developing brain of the unborn infant, causing periventricular white matter damage also known as periventricular leukomalacia. Indeed, increased amniotic fluid IL-6 levels are associated with intraventricular haemorrhage (IVH) and PVL and high levels of cytokines have been found in brains of infants who die with evidence of PVL.

Multiple pregnancy and uterine distension

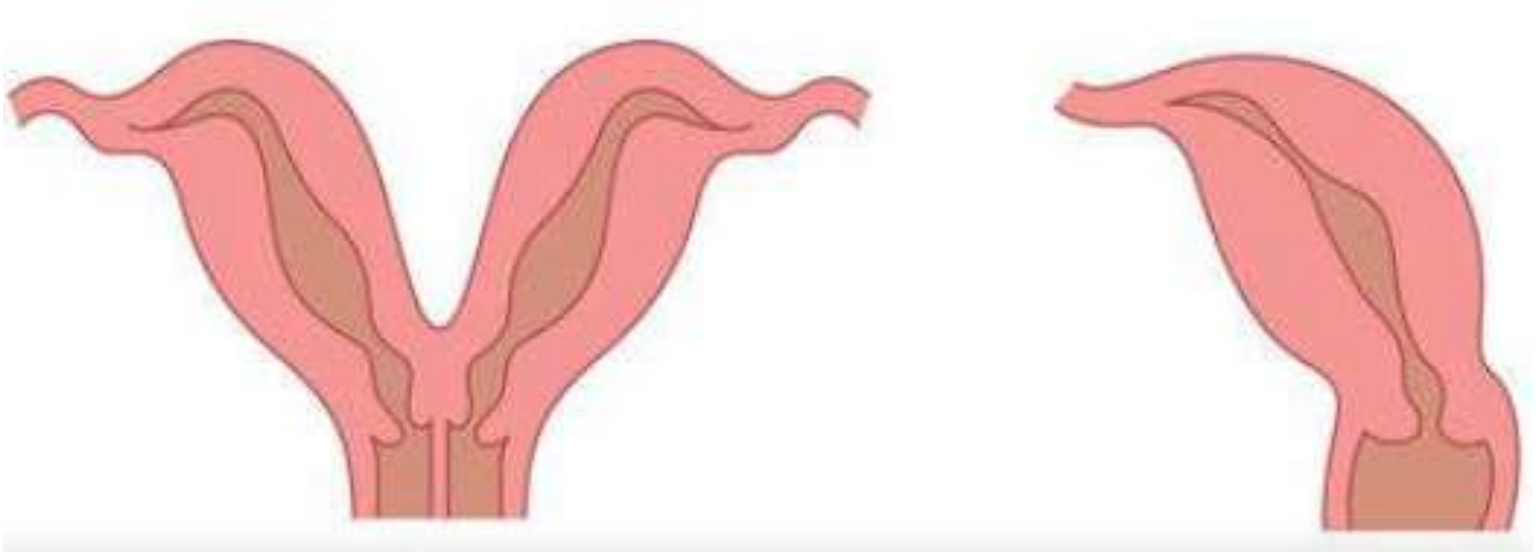
Overall, 56% of multiple births deliver before 37 weeks and 10–15% before 32 weeks. Consequently, although multiple pregnancies only make up 2% of the pregnant population, they contribute disproportionately to PTLs and, consequently, Neonatal Intensive Care Unit (NICU) admissions. The risk of PTL rises with fetal number, with triplets delivering on average at 32 weeks and quadruplets delivering at 28 weeks. Multiple pregnancies have an increased risk of pre-eclampsia, FGR and other medical complications of pregnancy, explaining the observation in one study that of the 54% of the twins delivered preterm, 23% were for medical reasons and 76% were after PTL or PPROM. Twins have a six to sevenfold increased risk of cerebral palsy and this rises to 100-fold if one twin dies antenatally.

Polyhydramnios, the presence of too much amniotic fluid, also increases the risk of PTL and PPROM, although the effect is not as great as with twins, with PTL occurring in between 7 and 25% of fetuses depending on the degree. Severe polyhydramnios can be managed with amnio-drainage, but this may itself precipitate PTL and/or PPROM. Alternatively, indomethacin, a non-steroidal anti inflammatory drug (NSAID), may be used as it reduces fetal urine production, but flow through the ductus arteriosus has to be closely monitored as the inhibition of PGE production by indomethacin may result in premature closure.

Uterine müllerian anomalies

Congenital müllerian anomalies are often unrecognized but are estimated to occur in up to 4% of women of reproductive age. They occur as a consequence of abnormal embryologic fusion and canalization of the müllerian ducts and result in an abnormally formed uterine cavity, which can range from an arcuate uterus, which results in minimal fundal cavity indentation, to complete failure of fusion resulting in uterine didelphys. They are associated with adverse pregnancy outcome in up to 25% of women, including first and second trimester miscarriage, PPROM, preterm birth, FGR, breech presentation and caesarean section.

Didelphic and unicornuate uterus



Haemorrhage

Antepartum haemorrhage and placental abruption may lead to spontaneous PTL.

The presence of a subchorionic haematoma in early pregnancy increases the risk of later PPROM, either through an effect of thrombin on membrane strength or through the occurrence of infection in the haematoma. Acute bleeding leads to the release thrombin that directly stimulates myometrial contractions. Placental abruption complicates 1% of all pregnancies and the maternal or fetal effects depend primarily on its severity and the gestational age when it occurs. Risk factors include pre-eclampsia and hypertension, previous abruption, trauma, smoking, cocaine use, multiple pregnancy, polyhydramnios, thrombophilias, advanced maternal age and PPROM. When an abruption involves 50% or more of the placenta it is frequently associated with fetal death.

Stress

There is growing evidence that either maternal or fetal stress may be associated with PTL. Major life events have an association with prematurity, as does the fetal stress of FGR. A link between maternal stress and PTL is suggested by its increased prevalence among poor mothers, as well as in stressful sociodemographic conditions (such as loss of employment, housing or husband). Prematurity is also more common among women reporting increased stress or anxiety. The biochemical pathway through which maternal and fetal stress promotes PTL is uncertain but may involve a premature increase in circulating corticotrophin-releasing hormone (CRH).

Management of preterm labour

Diagnosis:

1. Patient Symptoms

Early differentiation between true and false labor is difficult before there is demonstrable cervical effacement and dilatation. Uterine activity alone can be misleading because of *Braxton Hicks contractions*, described as irregular, nonrhythmical, and either painful or painless, can cause considerable confusion in the diagnosis of true preterm labor. Not infrequently, women who deliver before term have uterine activity that is attributed to Braxton Hicks contractions, prompting an incorrect diagnosis of false labor. Because uterine contractions alone may be misleading, the American Academy of Pediatrics and the American College of Obstetricians and Gynecologists had earlier proposed the following criteria to document preterm labor:

1. Contractions of four in 20 minutes or eight in 60 minutes plus progressive change in the cervix
2. Cervical dilatation greater than 1 cm
3. Cervical effacement of 80 percent or greater.

Currently, however, such clinical findings are now considered inaccurate predictors of preterm delivery. In addition to painful or painless uterine contractions, symptoms such as pelvic pressure, menstrual-like cramps, watery vaginal discharge, and lower back pain have been empirically associated with impending preterm birth. Such complaints are thought by some to be common in normal pregnancy and are therefore often dismissed by patients, clinicians, and nurses. The importance of these symptoms as a harbinger of labor has been emphasized by some but not all investigators.

2.Cervical Changes

A.Cervical Dilatation

Asymptomatic cervical dilatation after midpregnancy is suspected as a risk factor for preterm delivery, although some clinicians consider it to be a normal anatomical variant, particularly in parous women. Studies, however, have suggested that parity alone is not sufficient to explain cervical dilatation discovered early in the third trimester.

Although women with dilatation and effacement in the third trimester are at increased risk for preterm birth, detection does not improve pregnancy outcome. The investigators also reported that cervical examinations were not related to preterm membrane rupture. It seems, therefore, that prenatal cervical examinations are neither beneficial nor harmful.

B.Cervical Length

Vaginal-probe sonographic cervical assessment has been evaluated extensively over the past decade. The mean cervical length at 24 weeks was approximately 35 mm, and those women with progressively shorter cervices experienced increased rates of preterm birth.

The investigators correlated sonographic cervical length, funneling, and prior history of preterm birth with delivery before 35 weeks. Funneling was defined as bulging of the membranes into the endocervical canal and protruding at least 25 percent of the entire cervical length. A short cervix by itself was the poorest predictor of preterm birth, whereas funneling plus a history of prior preterm birth was highly predictive.

C.Incompetent Cervix

- *Cervical incompetence* is a clinical diagnosis characterized by recurrent, painless cervical dilatation and spontaneous midtrimester birth in the absence of spontaneous membrane rupture, bleeding, or infection.

3.fetal fibronectin (fFN)

Up to 70% of women who present with threatened PTL to the labour ward will not deliver during the current admission, and up to 50% will not deliver until term. Deciding who is and who is not in PTL has been helped by testing the cervicovaginal fluid levels of fetal fibronectin (fFN), a glycoprotein found in cervicovaginal fluid, amniotic fluid, placental tissue and in the interface between the chorion and decidua. It acts like ‘glue’ at the maternal–fetal interface and its presence in cervicovaginal fluid between 22 and 36 weeks’ gestation has been shown to be a predictor of PTL. Negative fFN testing has a very high negative predictive value, enabling most women with threatened PTL and a negative fFN test to be sent home. Those with a positive fFN test can be admitted for tocolysis and steroids for fetal lung maturation.

Treatment

1.Tocolytics

Tocolytics are used to delay delivery long enough for corticosteroid administration to improve neonatal lung function and, if necessary, for *in utero* transfer to a NICU. The current guidelines from the Royal College of Obstetricians and Gynecologists suggest that if tocolytics are administered for the medical treatment of PTL, the first choice should be a calcium channel blocker (nifedipine) or an OTR antagonist (atosiban). However, a recent review and trials of tocolysis found that PG inhibitors and calcium channel blockers are most likely the best therapy for PTL on the basis of delaying delivery by 48 hours, neonatal mortality, neonatal respiratory distress syndrome (RDS) and maternal side-effects. The different types of tocolytics are discussed below.

Beta-sympathomimetics

Beta-agonists (ritodrine, salbutamol and terbutaline) are predominantly β2 adreno-receptor agonists, which mediate myometrial relaxation by stimulating cyclic adenyl monophospate (AMP) production. They are effective in delaying delivery, but do not improve neonatal outcome or ultimate PTL rates. Further, they have significant maternal side-effects, which means that they are rarely used in the context of threatened PTL in the UK, although globally they are still widely used.

The most serious side-effect is pulmonary oedema, with an estimated incidence of 1:350–1:400 treated patients. Maternal deaths from acute cardiopulmonary compromise are described, with greater risks if beta-agonists are given in large fluid volumes, in multiple pregnancies and in women with cardiac disease.

Magnesium sulphate

Magnesium decreases the frequency of depolarization of smooth muscle by modulating calcium uptake, binding and distribution in smooth muscle cells and results in inhibition of uterine contractions. However, although magnesium sulphate is widely used as a tocolytic in the USA, there has only been one randomized trial comparing its effect to placebo and that failed to demonstrate a beneficial effect on the duration of pregnancy. Similarly, a study on magnesium sulphate tocolysis found no effect favouring magnesium sulphate over controls. However, the American College of Obstetricians and Gynecologists (ACOG) supports the use of magnesium sulphate for neuroprotection stating that “magnesium sulphate reduces the risk of cerebral palsy in surviving infants”.

Non-steroidal anti-inflammatory drugs

The first NSAID to be widely used in the management of PTL was indomethacin. It is a reversible, non-specific competitive cyclooxygenase (COX) inhibitor. In a series of studies, indomethacin has been shown to effectively delay delivery for 48 hours, 7–10 days and beyond 37 weeks, so reducing the incidence of low birthweight (<2,500 g). However, although PG inhibitors are effective in delaying PTL, they do have several adverse fetal effects. As mentioned above, PG synthesis is responsible for the maintenance of a patent ductus arteriosus and inhibition can lead to its premature closure.

This can occur as early as the late second trimester, with the incidence increasing dramatically from 32 weeks. This may lead to persistent pulmonary hypertension in the fetal circulation of the neonate. The effect is completely reversible with early identification and discontinuation of treatment. In addition, indomethacin use has been associated with an increased risk of necrotizing enterocolitis and neonatal renal dysfunction. The latter probably occurs because inhibition of fetal PG synthesis reduces renal perfusion and fetal urine output, resulting in reversible (after discontinuation of the drug) oligohydramnios.

Calcium channel blockers

The effects of calcium channel blockers in relaxing the contractions of the human myometrium have been known for several years. They exert their effect by binding to L-type channels, reducing intracellular levels of calcium and blocking the transmembrane influx of calcium ions into muscle cells. Comparing nifedipine with other tocolytics (including beta-sympathomimetics, NSAIDs, magnesium sulphate and OTR antagonist [OTR-A]), no significant reductions were shown in the primary outcome measures of birth within 48 hours of treatment or in perinatal mortality. However, adverse drug reactions, discontinuation due to side-effects, neonatal RDS, necrotizing enterocolitis, intraventricular haemorrhage and neonatal jaundice were least for OTR-A, intermediate for nifedipine and

Oxytocin receptor antagonists

OTRs play an important role in the onset and progression of labour as described above. The OTR-A atosiban is a competitive antagonist of oxytocin and vasopressin, binding to both the OTRs and the vasopressin V1a receptors within the myometrium. Administration of atosiban results in a dose-dependent inhibition of uterine contractility and oxytocin-mediated PG release. In pregnant women, atosiban is 46–48% plasma protein bound and only a small amount appears to cross the placenta into the fetal circulation. It has a similar efficacy as betasympathomimetics, but is much better tolerated. Compared to placebo, more atosiban-treated patients remained undelivered at 24 hours, 48 hours and 7 days.

2.Corticosteroid therapy

The administration of corticosteroids has the greatest influence on preterm neonatal outcome. Although the use of recombinant surfactant in neonates has also had a major impact on the incidence and consequences of RDS, antenatal
corticosteroids are still associated with significant reduction in neonatal mortality
principally through reduced rates of RDS and IVH. A study confirmed significant reductions in the risks of mortality, RDS and IVH in preterm infants of 31, 44 and 46%, respectively, after a single course of steroids.
Their mechanism of action is complex; they affect not only fetal lung maturation,
but also, fetal growth, organ system maturation, fetal brain development, immune
function and the fetal hypothalamic–pituitary–adrenocortical axis. Currently, betamethasone or dexamethasone are recommended; both are able to cross the
placenta in their active form and have comparable properties, but some dexamethasone preparations contain a sulphite preservative that has been linked

Interest for their use has been tempered by recent concerns, based on animal and some human data, that repeated antenatal doses could lead to a decrease in birthweight, brain size and abnormal neuronal development.

However, the long-term outcomes related to their use have been largely positive and overall, antenatal corticosteroid treatment has been associated with less developmental delay in childhood and a trend towards fewer children having cerebral palsy when compared with no corticosteroid treatment. While 30 years follow-up showed no clinical differences in adults who were exposed *in utero* to betamethasone, there are no comparable data for dexamethasone.

3.Antibiotics

Despite a clear link between bacterial infection and preterm birth, the results of antibiotic treatment as an attempt to prevent PTL have been disappointing. The trials focused on the use of antibiotics in PPROM and spontaneous PTL with intact membranes. These trials demonstrated that, in singleton pregnancies with PPROM, erythromycin improved neonatal outcomes, but that antibiotic treatment in women with intact membranes had no benefit. As a result of these trials, 10 days of erythromycin has been adopted as the treatment of choice for PPROM in many obstetric units in the UK.

However, concerns have been raised over widespread use of broad-spectrum antibiotics for all patients with PPROM.

Prediction of preterm labour

Since the current management of PTL has little impact on neonatal outcome, research has focused on detecting those women who are at high risk of PTL and intervening to reduce their risk. Risk scoring systems rely heavily on previous obstetric history and are therefore not useful in women having their first baby.

Past obstetric history:

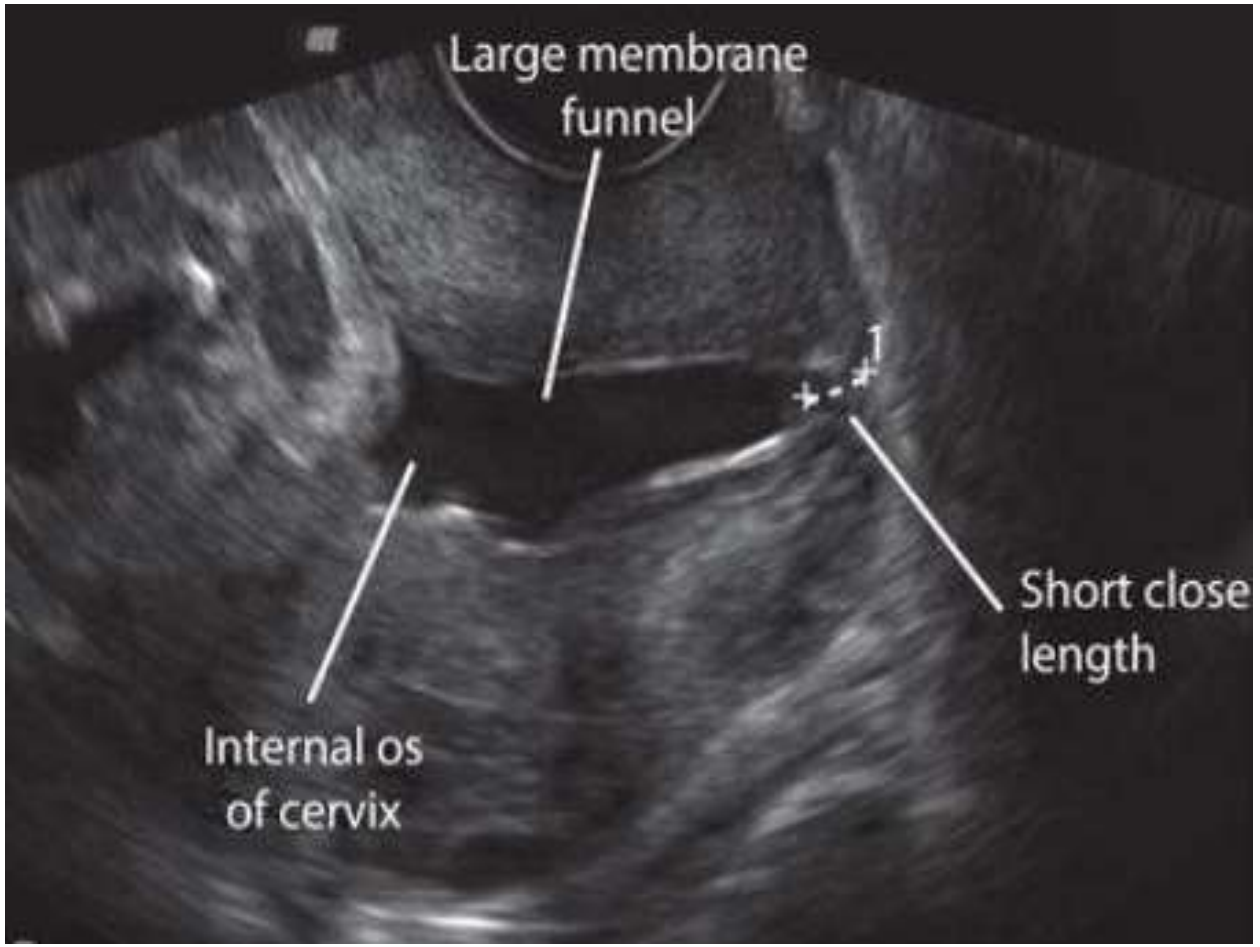
Having had a previous PTD increases the risk of PTL in a subsequent pregnancy four times in comparison to a woman who had a previous delivery at term.

Ultrasound measurement of cervical length

Cervical length measured by transvaginal ultrasound has been shown to be more accurate than transabdominal ultrasound or digital examination. There is a direct relationship between cervical length and the risk of PTL. Cervical length surveillance with serial measurement of cervical length throughout the second and early third trimester is now used to monitor women at high risk of PTD. The combination of cervical length and obstetric history can predict 80.6% of extremely early spontaneous PTD (10% screen-positive rate).

However, currently universal cervical length screening has not been approved as it has not been shown to be cost effective.

cervical length and funnelling on ultrasound.



Prevention of preterm labour

In those found to be at high risk of PTD, two interventions are currently available, progesterone and cervical cerclage.

Progesterone

Progesterone has been known to be important in maintaining pregnancy for more than 80 years and is thought to promote uterine quiescence and inhibit the production of proinflammatory cytokines and PGs within the uterus. In women with a previous preterm birth, there is some evidence that intramuscular hydroxyprogesterone caproate is effective in reducing the risk of recurrence.

Hydroxyprogesterone caproate is licenced in the USA for preterm birth prevention in women with a previous preterm birth. In women with a short cervix, some studies have suggested that vaginal progesterone may prevent preterm birth. Crucially, there is no evidence from any study that progesterone can reduce the longer-term adverse effects of preterm birth, that of neurodevelopmental disability and respiratory morbidity.

Cervical cerclage

Transvaginal cervical cerclage may be placed in three different circumstances:
1.Following multiple mid trimester losses or preterm deliveries (history indicated cerclage).

2.When the cervix shortens (usually <25 mm) in women with a history of cervical surgery or previous preterm birth (ultrasound indicated cerclage).

3.When the cervix is dilating in the absence of contractions (rescue cerclage).

The exact mechanism by which cerclage helps to prevent or delay PTL is not entirely understood. It is likely, however, that cerclage provides structural support to a weakened cervix, and enhances the cervical immunological barrier by improving retention of the mucous plug and preventing ascending infection by maintaining cervical length. Similar to progesterone, cervical cerclage does not appear to reduce the risk of PTL in multiple pregnancies.

Types of cerclage

1.McDonald transvaginal cerclage:

Transvaginal purse-string suture inserted at the cervicovaginal junction without bladder mobilization.

2.Shirodkar (high transvaginal)cerclage:

Transvaginal purse-string suture inserted following bladder mobilization, to allow insertion above the level of cardinal ligaments.

3.Transabdominal cerclage:

Suture inserted at the cervicoisthmic junction via laparotomy or laparoscopy. Transabdominal cerclages can either be inserted preconceptionally or in the first trimester of pregnancy.

A transabdominal cerclage is usually inserted following a failed vaginal cerclage or extensive cervical surgery. There are no randomized studies comparing the effectiveness of transabdominal cerclage with that of expectant management or transvaginal cerclage. Any potential benefits of transabdominal cerclage must be weighed against its increased operative risks. Patients must undergo two laparotomies during pregnancy, one for suture insertion and the other for caesarean section. Transabdominal cerclage should be therefore performed only by experienced operators and only for clear and defined indications.

Thank you