Meconium in labour

Jeffrey Unsworth

Sarah Vause

Abstract

Meconium staining of the amniotic fluid is a common occurrence during labour and although a large proportion of these pregnancies will have a normal neonatal outcome, its presence may be an indicator of fetal hypoxia and has been linked to the development of cerebral palsy, seizures and meconium aspiration syndrome. The management of the intrauterine passage of meconium has been controversial but appropriate intrapartum care with early detection and management of fetal hypoxia is important in minimizing the risk from meconium staining of the amniotic fluid. This review looks at the evidence for the potential mechanisms implicated in the passage of meconium; its intrapartum management and possible interventions available to reduce the risk of meconium aspiration. The neonatal complications and immediate delivery room management of the meconium-stained neonate are also discussed.

Keywords meconium; meconium aspiration syndrome; meconium liquor

Introduction

Meconium-stained amniotic fluid (MSAF) occurs as a result of the passage of fetal colonic contents into the amniotic fluid. It occurs in approximately 15–20% of term pregnancies but this number increases to 30–40% in the post-term pregnancy. MSAF has been linked with an increased risk of developing chorioamnionitis and is associated with adverse fetal outcomes including neonatal sepsis, cerebral palsy, seizures and in particular an increased risk of the neonate developing meconium aspiration syndrome, which in itself accounts for approximately 2% of perinatal deaths. The passage of meconium, in part, is thought to be due to physiological fetal gut maturation, thereby, explaining the higher incidence in the post-term pregnancy. It has also been suggested that fetal hypoxia and acidaemia may also be implicated in the pathogenesis of MSAF, thereby, acting as a potential sign of fetal distress. However, the exact relationship between fetal distress and MSAF is uncertain; passage of meconium may occur in the absence of any sustained hypoxia or increased neonatal morbidity or mortality and conversely fetal distress may occur in the absence of any meconium passage. For this reason it is controversial whether the *in utero* passage of meconium represents normal gut maturation or occurs as a result of hypoxic stress. It does seem evident; however, that in the preterm fetus, the passage of meconium, although a relatively rare event, is of significance as this group has a particularly high rate of perinatal morbidity and mortality when labour is associated with MSAF.

This review aims to discuss the theories behind the production of meconium, its implications during labour, the complications associated with its presence and the evidence for any potential therapeutic options available.

Jeffrey Unsworth *MB ChB is a Specialist Registrar in Obstetrics and Gynaecology at St Mary's Hospital, Manchester, UK. Conflicts of interest: none declared.*

Sarah Vause *MD MRCOG is a Consultant Obstetrician and Sub-specialist in Maternal and Fetal Medicine at St Mary's Hospital, Manchester, UK. Conflicts of interest: none declared.*

Formation and composition of meconium

The name meconium is derived from the name *meconium-arion*, meaning "opium-like", and has been linked with Aristotle's belief that it induced sleep in the fetus. It first appears within the fetal gastrointestinal tract at 70–85 days gestation as a viscous substance made up primarily of water (70–80%). Other constituents include intestinal epithelial cells, squamous cells, lanugo, amniotic fluid, bile acids and salts (giving the characteristic green colour), phospholipase A2, interleukin-8, mucus glycoproteins, lipids and proteases.

The main theories accounting for the passage of meconium before birth are based on those of fetal maturation and fetal stress.

Fetal maturation: although meconium appears very early in the gastrointestinal tract, MSAF rarely occurs before 34 weeks gestation and appears increasingly with advancing gestational age with its incidence increasing to 30–40% over 42 weeks. Motilin, an intestinal polypeptide which stimulates contraction of intestinal muscle, is found in higher concentrations in post-term than pre-term fetal gastrointestinal tracts. Furthermore, intestinal parasympathetic innervation and myelination also increase in later gestations implying that the increasing incidence may reflect the maturation of peristalsis in the fetal intestine. Therefore, at increasing gestations, particularly post-term, MSAF may be a physiological event, simply reflecting the maturation of fetal intestinal function.

Fetal stress: MSAF has also been attributed to a fetal response to intrauterine stress and hypoxia, with the passage of meconium occurring more frequently when umbilical vein oxygen saturations are below 30%. Furthermore, the degree of MSAF is related to the degree of hypoxia with "thick" stained MSAF being associated with lower oxygen concentrations than "light" stained MSAF. One theory to explain this is that of intestinal ischaemia, which is thought to result in relaxation of the fetal anal sphincter and increased gastrointestinal peristalsis, thereby, leading to the passage of meconium. It has been theorized, therefore, that during hypoxia, the fetal circulation shunts blood away from the bowel and directs it to the brain and heart, thereby contributing to intestinal ischaemia and subsequently MSAF. Conversely, in animal studies, term rabbits failed to pass meconium during a hypoxic insult, calling into question, whether this mechanism is a major cause for meconium passage in a hypoxic human fetus.

Vagally mediated gastrointestinal peristalsis in response to head or cord compression (the same reflex which initiates variable decelerations) may also be associated with meconium passage in the absence of fetal distress.

Meconium passage, which may be secondary to smooth muscle contraction in the fetal gastrointestinal tract, has also been linked to the use of misoprostol when inducing labour.

The exact mechanism for meconium passage in the human fetus is still not completely understood and may be a combination of all the above. It should be emphasized, however, that despite being complicated by the presence of MSAF, many pregnancies do not have any adverse outcome, and indeed fetal distress occurs frequently in the absence of the passage of meconium, therefore, more research is required for us to fully understand the relationship between MSAF and fetal distress.

Grading of meconium

For many years attempts have been made to correlate increased meconium thickness with a worse perinatal outcome, but due to the subjectivity of assessing meconium thickness, it makes it very difficult to compare studies with any scientific rigour. Indeed, it has been shown that inter- and intra-observer agreement on visual grading of MSAF thickness is poor (Table 1 illustrates a common grading system of meconium). However, there does appear to be a significant linear association between meconium thickness and abnormal fetal heart rate patterns during labour, low Apgar scores (Table 2) and risk for caesarean section delivery. There also appears to be a higher risk of neonatal intensive care admission in pregnancies with thick meconium as compared to those with clear amniotic fluid, suggesting that thick meconium, not thin, is associated with an increased risk for perinatal complications during labour and delivery. A system of measuring quantitative meconium concentrations using a "meconiumcrit" (percentage by volume of the solid component of meconium) was proposed in the 1990s, but this has not been adopted clinically, as a study investigating the value of measuring meconiumcrit showed no significant correlation with umbilical artery pH or Apgar score and no clinical benefit. However, it should be noted that two cases of meconium aspiration syndrome occurred within this study, both of which were from the "thick" meconium group. Although there is limited good quality evidence suggesting that the use of a system to grade meconium has any significant impact on neonatal outcome, most obstetricians would consider thick meconium a more ominous sign than thin and the National Institute of Clinical Excellence (NICE) recommends a standardized scoring system for the degree of meconium staining and its association with neonatal outcome. Further to this, accurately estimating the degree of meconium thickness is of importance as it helps determine the intensity of monitoring required following birth.

Standard grading system for meconium thickness

Grade 1 (thin)	Refers to light green or flecking of otherwise clear amniotic fluid
Grade 2	Brown but thin uniform staining of the amniotic fluid
Grade 3 (thick)	Bright green or brown thick uniform staining of the amniotic fluid

Table 1

The Apgar scoring system

	Score		
Apgar sign	2 Points	1 Point	0 Points
Activity (muscle tone)	Active movements	Arms and legs flexed	Absent
Pulse	>100	<100	Absent
Grimace (reflex irritability)	Sneeze, coughs	Grimace	Absent
Appearance (skin colour)	Entire body normal	Normal, except for extremities	Pale all over
Respiration	Good, crying	Slow, irregular	Absent

Table 2

Intrapartum management

Due to the mechanisms mentioned above, meconium passage may have different clinical significance at different gestations.

Meconium in the term fetus

As the fetal gut matures, meconium moves closer to the distal colon and rectum. Therefore, at term there is a close proximity between the meconium and the anal sphincter. For this reason, the amount of fetal stress required to initiate the release of meconium may be trivial and unsustained, such as occurs with cord compression during normal labour. However, in the absence of the stress of labour, meconium may be more significant. Risk factors for MSAF in the term fetus are shown in Table 3.

The presence of MSAF in early labour is associated with an increased rate of caesarean section delivery compared to the presence of clear amniotic fluid, due to the increased rate of fetal heart rate abnormalities observed in this group, in particular, the presence of early and variable decelerations, explainable by the theory of cord compression.

Numerous studies have utilized cardiotocographical fetal heart rate recordings, cord pH measuring and Apgar scores to try and ascertain the relevance of MSAF in the term fetus and its impact on neonatal outcome in order to determine any way of identifying the at-risk pregnancy. The evidence suggests that in

Risk factors for meconium passage

Risk factors for meconium-stained amniotic fluid
Post-term pregnancy
Placental insufficiency
Maternal hypertension and pre-eclampsia
Maternal diabetes
Fetal heart rate abnormalities
IUGR
Poor biophysical profile
Smoking
Oligohydramnios
Cocaine abuse

Table 3