# Managing patients with meconium-stained amniotic fluid

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***Meconium-stained amniotic fluid might signify underlying acute or chronic fetal hypoxia with adverse perinatal outcome, especially if associated with cardiotocographic abnormality. Management requires awareness of this potential risk, appropriate intrapartum care and a combined obstetric-neonatal approach. Amnioinfusion can be an effective preventative measure.***

Meconium-stained amniotic fluid (MSAF) has been traditionally taken as a sign of fetal distress, as it is associated with higher incidence of intrapartum stillbirths, increased risk of neonatal deaths, significantly lower 1- and 5-minute Apgar scores and various other neonatal morbidities in otherwise healthy term infants. The condition occurs in clinical obstetric practice in 7–20% of live births (Katz and Bowes, 1992). It is more common in certain populations (36% in a South African referred population) (Mitri et al, 1987). Of these deliveries, 2–4% of neonates will develop meconium aspiration syndrome (MAS), more than 90% of which are associated with thick MSAF (Benny et al, 1987). The resulting mortality rate ranges from 5 to 10% (Behrman et al, 2000).

## PATHOPHYSIOLOGY OF MECONIUM PASSAGE

Meconium first appears in the fetal ileum between the 10th and 16th week of gestation (Houlihan and Knuppel, 1994). It is a viscous, greenish-brown liquid composed of undigested debris of swallowed lanugo and vernix together with various products of secretions, excretions (such as bile, pancreatic juice, mucus) and desquamation of the gastrointestinal tract. Meconium has a bilirubin concentration of 1 mg/dl and its typical hue is caused by the pigment biliverdin.

The quantity of meconium in the fetal gut is small during the first two trimesters, but increases rapidly during the third (Wenstrom and Parsons, 1989). Because the internal and external anal sphincters are usually closed during fetal life the amniotic fluid ordinarily remains clear. However, various stimuli are known to cause relaxation of the sphincter tone and subsequent passage of meconium into the amniotic fluid. Any insult resulting in fetal hypoxia releases arginine vasopressin from the fetal pituitary which stimulates the smooth muscles of the colon. The induced hyperperistalsis then causes relaxation of the fetal anal sphincter. Increased vagal (parasympathetic) activity from in-utero stresses, e.g. cord compression may stimulate meconium passage without concomitant hypoxia.

Meconium passage may occur as a result of spontaneous gastrointestinal motility that reflects physiological maturation of the fetal gut as MSAF is uncommon before 38 weeks but increases after 40 weeks. Very occasionally, MSAF may be the result of in-utero bilious vomiting secondary to fetal intestinal obstruction (Akindale, 1994), intrauterine infection, e.g. *Listeria monocytogenes* (Valkenburg et al, 1988), or a rare condition of congenital chloride diarrhoea (Holmerg et al, 1977).

## GRADING OF MSAF

Traditionally, MSAF has been graded into three categories: grade 1 or light meconium-stained liquor which is translucent and light yellow-green in colour; grade 3 or thickly meconium-stained liquor that is opaque and deep green in colour with visually identified particulate matter (the 'pea soup' liquor) and grade 2, i.e. moderate meconium-stained liquor, opalescent with the colour in between that of grades 1 and 3 (Arulkumaron et al, 1985). However, grading the meconium staining by visual assessment may have a poor accuracy and precision and a wide interobserver variation.

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## CLINICAL SIGNIFICANCE OF MSAF

The most significant effect of MSAF on the neonate is MAS (see below). Other minor and major effects have also been described, e.g. meconium contamination of neonatal middle ear (Piza et al, 1989), seizures in term infants (Patterson et al, 1989) and renal proximal tubular dysfunction (Cole et al, 1985). Intrapartum MSAF has been described as a risk factor for microbial invasion of the amniotic cavity and preterm labour with a significantly higher postpartum infectious morbidity and endometritis in the mother. Exposure to meconium also decreases the stress tolerance with increased friability of chorioamniotic membranes, thereby causing difficulty in their removal after spontaneous vaginal deliveries complicated by MSAF.

When thick MSAF of early and late labour was compared with matched controls it was shown that a combination of late passage of meconium and other intrapartum signs, particularly abnormalities in the cardiotocograph (CTG), may indicate a fetus at risk when neither sign alone is predictive (Meis et al, 1978).

However, passage of meconium even early in labour combined with low fetal pH correlated with increased perinatal morbidity with Apgar score <7 at 1 minute (Starks, 1980). This risk is greater with thick meconium than when meconium is thin. Khatree and Mokgokong (1979) have found meconium in the latent phase of labour to be more ominous than during the active phase. Rosegger (1983) distinguished three variants of MAS: late MAS in non-asphyxiated infants, late MAS in asphyxiated infants and MAS because of continuous passage of meconium throughout labour in non-asphyxiated infants. He considers the second group to be at most risk.

## DIAGNOSIS

MSAF is clinically diagnosed after antepartum or intrapartum spontaneous rupture of membranes or at amniotomy during labour. Occasionally, MSAF is found at caesarean section performed for fetal distress or other indications. There has been no controlled evaluation of the role of routine amniotomy as a screening test to identify the fetus at risk (Grant, 1993).

Attempts at antenatal diagnosis of MSAF by routine amnioscopy have not been a success because of both high false-positive and false-negative rates. Ultrasonographic features of a diffuse (i.e. homogenous) echogenic pattern throughout the amniotic cavity, a clear contrast between the amniotic fluid and the umbilical cord, and layering in the more dependent areas, can be confused in the third trimester with a similar picture representing vernix instead of meconium.

Recently, very sensitive optical systems have been developed for continuous assessment of the amount of meconium in the amniotic fluid during labour (Genevier et al, 1993). A specially shaped fibreoptic-tipped probe is inserted into the uterine cavity. Light reflected from the amniotic fluid returns to a spectrum analyzer and the meconium concentration can be displayed in real time by computer. This system is claimed to be the first to be able to measure meconium concentration continuously during labour, is not invasive to the fetus, and is simple to use as a conventional intrauterine pressure catheter. Also its accuracy is greater at high meconium concentrations which are probably most significant. It will be useful if meconium is present but not clinically detectable when CTG abnormalities may be misinterpreted and labour may be managed inappropriately. In addition, the probe may enable monitoring of amnioinfusion (see below). However, despite its potential, the device has not been so popular in clinical practice as expected (P Danielian, personal communication, 2000).

## MECONIUM ASPIRATION SYNDROME

Once meconium has been passed, regardless of the stimulus, any episode of intrauterine fetal gasping can result in aspiration of meconium into the fetal trachea and lungs before delivery. Although hypoxia is the commonest trigger, there may be other mechanisms such as deep breathing in utero (Houlihan and Knuppel, 1994). Normal fetal respiration is the shallow type, in which the net flow of amniotic fluid is from the lungs to the amniotic cavity. Physiologically, this lung fluid contributes to the amniotic fluid volume each day. The fetus may breathe deeply into the lungs for up to 10% of its breaths in utero (Dawes, 1972). The amount of deep breathing increases as gestational age advances and during periods of fetal hypercarbia (Ritchie and Lakhane, 1980).

Once meconium is aspirated, the fetus/infant can be affected by either the direct effect of the meconium on the fetal lungs with its consequences, or this combined with the concomitant effect of hypoxia which triggered the event in the first place (*Figure 1*).

The particulate matter in meconium can cause partial or complete obstruction of the small respiratory passages, resulting in air trapping and/or atelectasis. The components of meconium

may incite chemical pneumonitis which may be further complicated by infection. The free fatty acids in the meconium may strip away the alveolar surfactant (Moses et al, 1991).

Hypoxia in utero as well as meconium itself can cause pulmonary vasospasm and necrosis, possibly as a result of a prostaglandin-like substance. The damage thus inflicted renders the fetal/neonatal lungs unable to clear the meconium. In most severe cases, even right-to-left shunting may ensue with occasional persistence of fetal circulation resulting in serious consequences on fetal/neonatal outcome, even death. Residual lung problems in the survivors are rare, but include symptomatic cough, wheezing, and persistent hyperinflation for up to 5–10 years. Ultimate prognosis depends on the extent of CNS injury from asphyxia and presence of associated problems such as pulmonary hypertension (Behrman et al, 2000).

Thick meconium and abnormal CTG are more common in severe MAS. It is more likely if the mother is a cigarette smoker, hypertensive, anaemic and of particular ethnic origin, e.g. Maori and Pacific Island ethnicity, or black Americans. Fewer than five antenatal care visits, pregnancy > 42 weeks, oligohydramnios, prolonged labour (> 15 hours) and inadequate suctioning of the airway at birth are other reported risk factors (Benny et al, 1987). Some studies have shown an association of MAS with primiparity (Urbaniak et al, 1996), while others suggest maternal cocaine use during pregnancy may be a risk factor (Hadeed and Siegel, 1989).

## MANAGEMENT

Because MSAF is associated with an increased risk of perinatal mortality and morbidity its presence is a concern to both obstetricians and

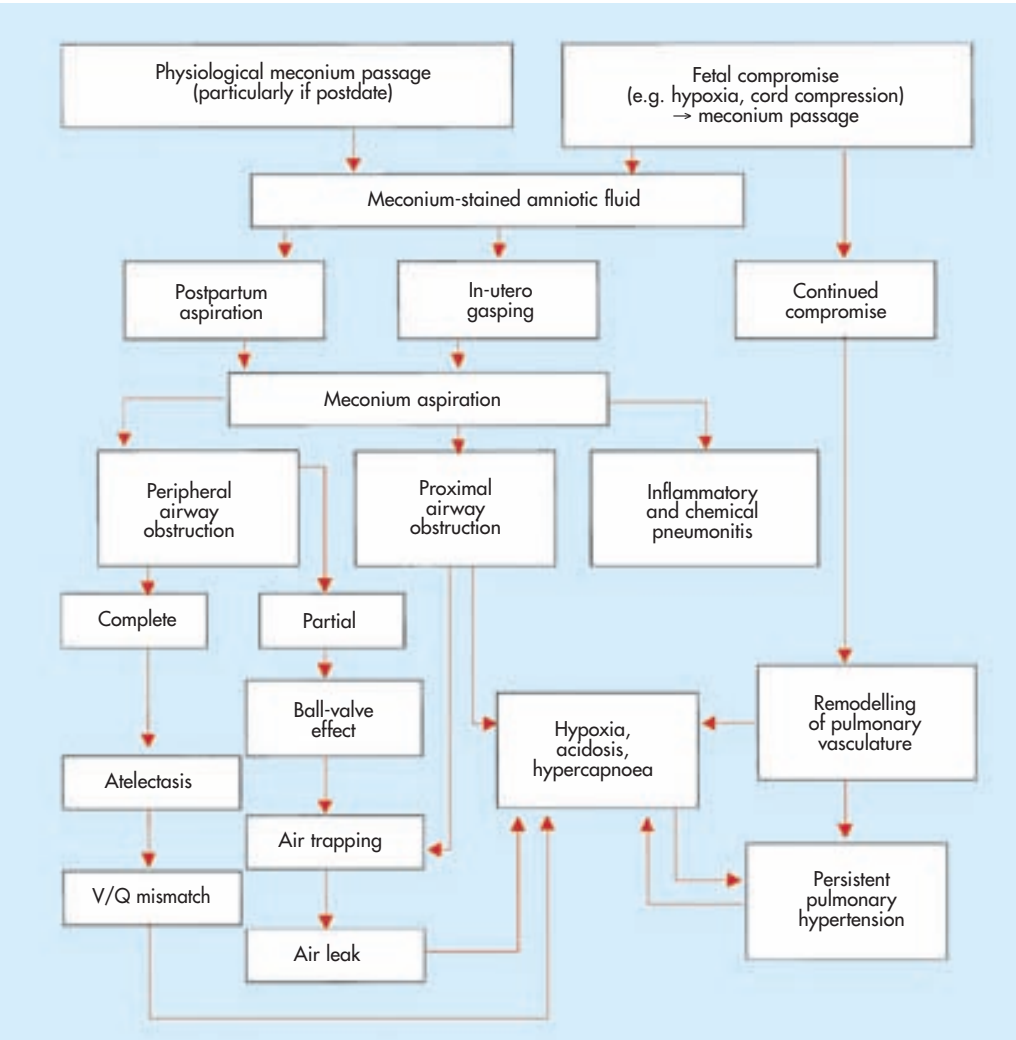


*Figure 1. Pathophysiology of meconium aspiration syndrome. Adapted from Wiswell and Bent (1993). V/Q = ventilation–perfusion.*

neonatologists. Arulkumaron et al (1985) have shown the relative safety of thin meconium-stained liquor amnii (grade 1) in presence of normal fetal heart rate tracing. Thick meconium itself, on the other hand, has been shown to be a risk factor for poor neonatal outcome, especially if associated with fetal heart rate abnormality and low umbilical artery pH. Therefore, these patients should be managed by continuous intrapartum CTG monitoring and if needed, fetal scalp blood sampling during labour, and optimal care at delivery (Arulkumaron et al, 1985).

Carson et al (1976) pioneered a 'combined obstetric and paediatric approach' which rapidly became the standard of care in obstetrics in the USA. Their protocol called for nasopharyngeal and oropharyngeal aspiration by the obstetrician before delivery of the shoulders or as the mouth is visualized through the uterine incision at caesarean section, followed by suction of the trachea under laryngoscopic visualization by the paediatrician.

Aspiration of MSAF from the mouth and pharynx above the level of vocal cords is easy, safe and can be performed even by the inexperienced. Its effectiveness in preventing severe MAS, however, is questionable. Routine intratracheal intubation and suction, on the other hand, is a potentially hazardous procedure with the risk of trauma, hypoxia, bradycardia, hypotension, infection and occasionally intracranial haemorrhage, especially in preterm infants (Linder et al, 1988).

This requires considerable expertise and training, although the incidence of morbidity is no different between an experienced paediatric resuscitator and an occasional performer (Falciglia, 1988). There is evidence from a randomized control trial that this practice only reduces the severity but not the incidence of MAS (Linder et al, 1988). It would seem unwise to suggest routine intubation in the infants who are not depressed and have no meconium below the vocal cords, simply because they have been born after meconium passage in utero. A more selective approach, as suggested by Cunningham (1990), would be appropriate .

The stomach of the newborn should be also emptied to prevent further meconium aspiration. Ventilation should be instituted if indicated. The policy of preventing meconium aspiration by early elective induction of labour has not gained favour in clinical practice because it does not consistently reduce the incidence of meconium aspiration.

## AMNIOINFUSION

Although the aggressive 'combined obstetric–paediatric approach' as described above has significantly decreased the occurrence of MAS, all cases of MAS cannot be eliminated by this policy (Falciglia, 1988). This may be caused by intrauterine aspiration, an event difficult to predict or prevent.

Amnioinfusion was initially proposed as a method to correct concurrent oligohydramnios, reduce cord compression, and thereby decrease vagal stimulation and meconium passage (Wenstrom and Parsons, 1989). Furthermore, by dilution, amnioinfusion also diminishes the toxic effects of aspiration should it occur. Subsequently, this has been carried out in many centres all over the world (Wenstrom et al, 1994; Mahomed et al, 1998).

Normal saline or Ringer's lactate, at room temperature, is infused transcervically via an intrauterine catheter in women in labour at >37 weeks of gestation with singleton cephalic presentation and moderate or heavy MSAF, or transabdominally through a spinal needle when the membranes are intact. The rate of infusion is 15 ml/min for 800 ml, then 3 ml/min for the duration of labour (Mahomed et al, 1998).

A systemic review of the randomized trials on amnioinfusion has shown it to be associated with an overall reduction in the incidence of caesarean sections and improvement in perinatal outcome, particularly in situations where facilities for intrapartum and perinatal surveillance are limited (Hofmeyr, 2000). Amnioinfusion is simple, inexpensive and technically an easy procedure to perform.

Complications include uterine hypertonia, CTG abnormality, amnionitis, uterine rupture, maternal cardiac or respiratory failure and even maternal deaths because of amniotic fluid embolism (Wenstrom et al, 1994). The systemic review of trials conducted to date is too small to address these rare but serious maternal adverse effects of amnioinfusion. Larger trials with fewer exclusions are needed to address the risk:benefit ratio of amnioinfusion conclusively.

## CONCLUSION

Meconium staining of amniotic fluid occurs in clinical obstetric practice in 7–20% of live births. Of these deliveries, 2–4% of neonates will develop MAS, more than 90% of which are associated with thick MSAF and CTG abnormalities with a resulting mortality rate of 5–10%. Diagnosis is mainly clinical during labour. Although not always very accurate to specify the grades. Sophisticated fibreoptic

instruments can bring promise in future. While MSAF may be physiological in a mature but otherwise normal fetus, more often it is a response to acute or chronic fetal hypoxia in utero.

Obstetricians should be aware of this fact and be very vigilant in intrapartum management. A selective combined obstetric–paediatric approach reduces the incidence and severity of MAS. No effective preventive measures to date have been identified, but amnioinfusion has been shown to improve perinatal outcome although the number of trials is too small at present to comment on maternal complications of the procedure. HM

*Conflict of interest: none.*


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## KEY POINTS

- Meconium staining of amniotic fluid (MSAF) occurs mainly by three distinct mechanisms: as a physiological maturational event, as a response to acute hypoxic event or underlying chronic intrauterine hypoxia.
- MSAF is usually diagnosed intrapartum: antenatal diagnosis by routine amnioscopy, amniotomy or ultrasonography has not been rewarding.
- Fetal and neonatal risks are more with moderate to thick MSAF and cardiotocographic abnormalities than in thin MSAF with normal cardiotocography.
- Patients with grade 3 MSAF with cardiotocographic abnormalities in labour should have continuous intrapartum cardiotocographic monitoring, fetal scalp blood sampling, and optimal care at delivery.
- Selective 'combined obstetric–paediatric approach' of management significantly decreases the severity of meconium aspiration syndrome.
- Systemic review of randomized controlled trials on amnioinfusion shows improved perinatal outcome.