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Meconium Aspiration Syndrome

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AUTHOR AND EDITOR INFORMATION

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[Author and Editor Disclosure](#)

Synonyms and related keywords: meconium aspiration syndrome, MAS, meconium aspiration syndrome, meconium-stained amniotic fluid, fetal hypoxic distress

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Background

The first intestinal discharge from newborns is meconium, which is a viscous, dark green substance composed of intestinal epithelial cells, lanugo, mucus, and intestinal secretions, such as bile. Intestinal secretions, mucosal cells, and solid elements of swallowed amniotic fluid are the 3 major solid constituents of meconium. Water is the major liquid constituent, making up 85-95% of meconium. Intrauterine distress can cause passage into the amniotic fluid. Factors that promote the passage in utero include placental insufficiency, maternal hypertension, preeclampsia, oligohydramnios, and maternal drug abuse, especially of tobacco and cocaine. Meconium-stained amniotic fluid may be aspirated during labor and delivery, causing neonatal respiratory distress. Because meconium is rarely found in the amniotic fluid prior to 34 weeks' gestation, meconium aspiration chiefly affects infants at term and postterm.

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Pathophysiology

In utero meconium passage results from neural stimulation of a mature GI tract and usually results from fetal hypoxic stress. As the fetus approaches term, the GI tract matures, and vagal stimulation from head or cord compression may cause peristalsis and relaxation of the rectal sphincter leading to meconium passage.

The effects of meconium in amniotic fluid are well documented. Meconium directly alters the amniotic fluid, reducing antibacterial activity and subsequently increasing the risk of perinatal bacterial infection. Additionally, meconium is irritating to fetal skin, thus increasing the incidence of erythema toxicum. However, the most severe complication of meconium passage in utero is aspiration of stained amniotic fluid before, during, and after birth. Aspiration induces hypoxia via 3 major pulmonary effects, which are airway obstruction, surfactant dysfunction, and chemical pneumonitis.

Airway obstruction

Complete obstruction of the airways by meconium results in atelectasis. Partial obstruction causes air trapping and hyperdistention of the alveoli, commonly termed the ball-valve effect. Hyperdistention of the alveoli occurs from airway expansion during inhalation and airway collapse around inspissated meconium in the airway, causing increased resistance during exhalation. The gas that is trapped, hyperinflating the lung, may rupture into the pleura (pneumothorax), mediastinum (pneumomediastinum), or pericardium (pneumopericardium).

Surfactant dysfunction

Several constituents of meconium, especially the free fatty acids (eg, palmitic, stearic, oleic), have a higher minimal surface tension than surfactant and strip it from the alveolar surface, resulting in diffuse atelectasis.

Chemical pneumonitis

Enzymes, bile salts, and fats in meconium irritate the airways and parenchyma, causing a release of cytokines and resulting in a diffuse pneumonia that may begin within a few hours of aspiration.

All of these pulmonary effects can produce gross ventilation-perfusion (V-Q) mismatch. To complicate matters further, many infants with meconium aspiration syndrome (MAS) have primary or secondary persistent pulmonary hypertension of the newborn (PPHN) as a result of chronic in utero stress and thickening of the pulmonary vessels. Finally, though meconium is sterile, its presence in the air passages can predispose the infant to pulmonary infection.

Frequency

United States

In the industrialized world, meconium in the amniotic fluid can be detected in 8-25% of all births after 34 weeks' gestation. Of those newborns with meconium-stained amniotic fluid, approximately 10% develop MAS.

Patient Education

Click [here](#) for patient education.

International

In developing countries with less availability of prenatal care and where home births are common, incidence of MAS is thought to be higher and is associated with a greater mortality rate.

Mortality/Morbidity

- The mortality rate for MAS resulting from severe parenchymal pulmonary disease and pulmonary hypertension is as high as 20%.
- Other complications include air block syndromes (eg, pneumothorax, pneumomediastinum, pneumopericardium) and pulmonary interstitial emphysema.

Race

No racial predilection exists.

Sex

MAS affects both sexes equally.

Age

MAS is exclusively a disease of newborns.

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History

- Presence of meconium in amniotic fluid is required to cause MAS, but not all neonates with meconium-stained fluid develop MAS.
- Green urine may be observed in newborns with MAS less than 24 hours after birth. Meconium pigments can be absorbed by the lung and excreted in urine.

Physical

- Severe respiratory distress may be present. Symptoms include the following:
 - Cyanosis
 - End-expiratory grunting
 - Alar flaring
 - Intercostal retractions
 - Tachypnea
 - Barrel chest in the presence of air trapping
 - Auscultated rales and rhonchi (in some cases)
- Yellow-green staining of fingernails, umbilical cord, and skin

Causes

- Factors that promote the passage of meconium in utero include the following:
 - Placental insufficiency
 - Maternal hypertension
 - Preeclampsia
 - Oligohydramnios
 - Maternal drug abuse, especially of tobacco and cocaine

- Maternal infection/chorioamnionitis
- Fetal gasping secondary to hypoxia
- Inadequate removal of meconium from the airway prior to the first breath
- Use of PPV prior to clearing the airway of meconium

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Other Problems to be Considered

Surfactant deficiency

Congenital heart disease with pulmonary hypertension

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Lab Studies

- Acid-base status
 - V-Q mismatch and perinatal stress are prevalent and assessment of acid-base status is crucial.
 - Metabolic acidosis from perinatal stress is complicated by respiratory acidosis from parenchymal disease and PPHN.
 - Arterial blood gases that measure pH, partial pressure of carbon dioxide (pCO₂), partial pressure of oxygen (pO₂), and continuous measurement of oxygenation by pulse oximetry are necessary for appropriate management.
- Serum electrolytes: Obtain sodium, potassium, and calcium concentrations when the infant with MAS aged 24 hours because the syndrome of inappropriate secretion of antidiuretic hormone (SIADH) and acute renal failure are frequent complications of perinatal stress.
- CBC
 - In utero or perinatal blood loss, as well as infection, contributes to postnatal stress.
 - Hemoglobin and hematocrit levels must be sufficient to ensure adequate oxygen-carrying capacity.
 - Thrombocytopenia increases the risk for neonatal hemorrhage.
 - Neutropenia or neutrophilia with left shift of the differential may indicate perinatal bacterial infection.

- Polycythemia may be present secondary to chronic and/or acute fetal hypoxia. Polycythemia is associated with decreased pulmonary blood flow and may exacerbate the hypoxia associated with MAS and PPHN.

Imaging Studies

- A chest radiograph is essential to do the following:
 - Determine the extent of intrathoracic pathology
 - Identify areas of atelectasis and air block syndromes
 - Assure appropriate positioning of the endotracheal tube and umbilical catheters.
- Later in the course of MAS when the infant is stable, imaging procedures of the brain, such as MRI, CT scan, or cranial ultrasound, are indicated if the infant's neurologic examination is abnormal.

Other Tests

- An echocardiogram ensures normal cardiac structure and assesses cardiac function, as well as the severity of pulmonary hypertension and right-to-left shunting.

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Medical Care

- Prevention
 - Prevention is paramount.
 - Obstetricians should monitor fetal status in an attempt to identify fetal stress.
 - When meconium is detected, administering amnioinfusion with warm sterile saline may be beneficial. This procedure dilutes meconium in the amniotic fluid; therefore, the severity of aspiration may be minimized.
 - Upon delivery of the head of the baby, careful suctioning of the posterior pharynx decreases the potential for aspiration of meconium. When aspiration occurs, intubation and immediate suctioning of the airway can remove much of the aspirated meconium.
 - No clinical trials justify suctioning based on the consistency of meconium. Do not perform the following harmful techniques in an attempt to prevent aspiration of meconium-stained amniotic fluid:
 - Squeezing the chest of the baby
 - Inserting a finger into the mouth of the baby
 - The American Academy of Pediatrics Neonatal Resuscitation Program Steering Committee has promulgated the following guidelines for management of the baby exposed to meconium:
 - If the baby is not vigorous (defined as poor muscle tone and little or absent respiratory effort): Suction the trachea immediately after delivery. Suction for no longer than 5 seconds. If no meconium is retrieved, do not repeat intubation and

suction. If meconium is retrieved and no bradycardia is present, reintubate and suction. If the heart rate is low, administer positive pressure ventilation and consider suctioning again later.

- If the baby is vigorous (defined as good respiratory effort, crying, good muscle tone, and good color): Clear secretions and meconium from the mouth and nose with a bulb syringe or a large-bore suction catheter. In either case, the remainder of the initial resuscitation steps should ensue: dry, stimulate, reposition, and administer oxygen as necessary.
 - The NRP guidelines are under continuous review and are revised as new evidence-based research becomes available.
- Intervention
 - Maintain an optimal thermal environment and minimal handling because these infants are agitated easily, which causes right-to-left shunting, leading to hypoxia and acidosis.
 - Continue respiratory care. Oxygen therapy via hood or positive pressure is crucial in maintaining adequate arterial oxygenation. If mechanical ventilation is required, make concerted efforts to minimize the mean airway pressure and to use as short an inspiratory time as possible. Surfactant therapy is now commonly used to replace displaced or inactivated surfactant and as a detergent to remove meconium. Studies are ongoing to evaluate the potential role of pulmonary lavage with surfactant.
 - Although conventional ventilation commonly is used initially, oscillatory, high-frequency, and jet ventilation are alternative effective therapies. Hyperventilation to induce hypocapnia and respiratory alkalosis is no longer a primary therapy for pulmonary hypertension because hypocarbia may result in decreased cerebral perfusion. Prolonged alkalosis has been shown to cause neuronal injury in the animal model, providing another reason to avoid alkalosis in these patients. Inhaled nitric oxide has replaced the use of most intravenous pulmonary vasodilators.
 - Pay careful attention to systemic blood volume and BP. Volume expansion, transfusion therapy, and systemic vasopressors are critical in maintaining systemic BP greater than pulmonary BP, thereby decreasing the right-to-left shunt through the patent ductus arteriosus.
 - Extracorporeal membrane oxygenation (ECMO) is employed if all other therapeutic options have been exhausted. Although effective in treating MAS, ECMO is associated with a high incidence of poor neurologic outcomes.

Surgical Care

- Although primary management of air block syndromes is achieved by thoracic drainage tubes inserted by a neonatologist, a pediatric surgical consultation may be necessary in severe cases.

Consultations

- A pediatric cardiology evaluation is necessary to perform an echocardiogram. This imaging technique ensures normal cardiac structure and assesses the severity of pulmonary hypertension and right-to-left shunting.
- A pediatric neurology evaluation is essential in the presence of neonatal encephalopathy or seizure activity.

Diet

- Perinatal distress and severe respiratory distress preclude feeding.
- Intravenous fluid therapy begins with adequate dextrose infusion to prevent hypoglycemia.
- Progressively add electrolytes, protein, lipids, and vitamins to ensure adequate nutrition and prevent

essential amino acid and essential fatty acid deficiencies.

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In addition to the treatments listed below, surfactant replacement therapy is frequently used. Natural lung extract is administered to replace the surfactant that has been stripped. Surfactant also acts as a detergent to break up residual meconium, thereby decreasing the severity of lung disease. Surfactant is used in patients with MAS, even though its efficacy, dosage regimen, and most effective product are not yet established.

Drug Category: *Respiratory gases*

Inhaled nitric oxide (NO) has the direct effect of pulmonary vasodilatation without the adverse effect of systemic hypotension. It is approved for use, if concomitant hypoxemic respiratory failure occurs.

Drug Name	Nitric oxide, inhaled (INOmax)
Description	Produced endogenously from the action of the enzyme NO synthetase on arginine. Exogenously inhaled NO is used in an attempt to decrease pulmonary vascular resistance and improve lung blood flow. It relaxes vascular smooth muscle by binding to the heme moiety of cytosolic guanylate cyclase, activating guanylate cyclase and increasing intracellular levels of cGMP, which then leads to vasodilation.
Pediatric Dose	20 ppm inhaled via respirator initially; not to exceed 80 ppm; most children respond at 20 ppm and can be weaned to lower doses; effect of pulmonary vasodilatation may still be observed at 5 ppm Must be delivered by a system that measures concentrations of NO in the breathing gas, with a constant concentration throughout the respiratory cycle and that does not cause generation of excessive inhaled nitrogen dioxide
Contraindications	Right to left shunting of blood; methemoglobin reductase deficiency
Interactions	Nitric oxide donor compounds (eg, nitroprusside, nitroglycerin) may increase risk of developing methemoglobinemia
Pregnancy	C - Safety for use during pregnancy has not been established.
Precautions	Toxic effects include methemoglobinemia and pulmonary inflammation resulting from reactive nitrogen intermediates; caution in thrombocytopenia, anemia, leukopenia, or bleeding disorders; monitor for PaO ₂ , methemoglobin, and NO ₂ ; abrupt withdrawal causes rebound pulmonary hypertension

Drug Category: *Systemic vasoconstrictors*

These agents are used to prevent right-to-left shunting by raising systemic pressure above pulmonary pressure. Systemic vasoconstrictors include dopamine, dobutamine, and epinephrine. Dopamine is the most commonly used.

Drug Name	Dopamine (Intropin)
Description	At lower doses, dopamine stimulates beta1-adrenergic and dopaminergic receptors (renal vasodilation, positive inotropism); at higher doses, it stimulates alpha-adrenergic receptors (renal vasoconstriction).

Pediatric Dose	5-20 mcg/kg/min IV
Contraindications	Documented hypersensitivity (rare in neonatal population); outflow tract obstructions such as subaortic stenosis
Interactions	Incompatible when admixed with acyclovir, amphotericin B, indomethacin, insulin, and sodium bicarbonate Phenytoin, alpha- and beta-adrenergic blockers, general anesthesia, and MAOIs increase and prolong effects of dopamine
Pregnancy	C - Safety for use during pregnancy has not been established.
Precautions	Adverse effects include tachycardia and arrhythmia; treat hypovolemia before infusion; promptly treat extravasation with SC phentolamine; administration through a central vein is recommended; do not use a systemic or umbilical artery for infusion; if dosages >20 mcg/kg/min are required, consider a different agent (eg, epinephrine, dobutamine) Monitor closely urine flow, cardiac output, pulmonary wedge pressure, and blood pressure during the infusion; before infusion, correct hypovolemia as indicated; monitoring central venous pressure or left ventricular filling pressure may be helpful in detecting and treating hypovolemia

Drug Category: Sedatives

These agents maximize efficiency of mechanical ventilation and minimize oxygen consumption.

Drug Name	Morphine
Description	Used for analgesia and sedation.
Pediatric Dose	0.05-0.2 mg/kg/dose IV over 5 min q2-4h prn
Contraindications	Documented hypersensitivity (rare in neonates); severe respiratory depression
Interactions	Any CNS depressant; phenothiazines may antagonize analgesic effects of opiate agonists; tricyclic antidepressants, MAOIs, and other CNS depressants may potentiate adverse effects of morphine; incompatible when admixed with furosemide, pentobarbital, phenobarbital, or phenytoin (forms precipitant)
Pregnancy	C - Safety for use during pregnancy has not been established.
Precautions	Caution in hypotension, respiratory depression, nausea, emesis, constipation, urinary retention, atrial flutter, and other supraventricular tachycardias; has vagolytic action and may increase ventricular response rate; may cause histamine release

Drug Name	Fentanyl (Sublimaze)
Description	Potent opioid used for analgesia, sedation, and anesthesia. Has a shorter duration of action than morphine.
Pediatric Dose	1-4 mcg/kg/dose IV slow push Infusion rate: 1-5 mcg/kg/h IV
Contraindications	Documented hypersensitivity (rare in neonates); hypotension or potentially compromised airway where it would be difficult to establish rapid airway control
	Barbiturates (eg, pentobarbital, thiopental) or other CNS depressants may have additive effects; phenothiazines

Interactions	may antagonize analgesic effects of opiate agonists; tricyclic antidepressants may potentiate adverse effects of fentanyl when both drugs are used concurrently
Pregnancy	C - Safety for use during pregnancy has not been established.
Precautions	May cause marked respiratory depression and hypotension; exercise caution with patients diagnosed with emesis, constipation, or urinary retention; idiosyncratic reaction (ie, chest wall rigidity syndrome) may require neuromuscular blockade to increase ventilation

Drug Name	Phenobarbital (Luminal)
Description	An anticonvulsant that may be used as a sedative. Suppresses the CNS from the reticular activating system (ie, presynaptic, postsynaptic).
Pediatric Dose	20 mg/kg IV as a single dose, administer slowly over 10-15 min
Contraindications	Documented hypersensitivity (rare in neonates); severe uncontrolled pain
Interactions	Incompatible when admixed with clindamycin, hydralazine, insulin, methadone, midazolam, morphine, ranitidine, and vancomycin; may cause respiratory depression if concurrently on CNS depressants (eg, benzodiazepines); decreases effectiveness of corticosteroids, theophylline, and beta-blockers
Pregnancy	D - Unsafe in pregnancy
Precautions	Rarely causes respiratory depression at this dose; do not administer IV administration faster than 50 mg/min; carefully monitor upon administration for hypotension, bradycardia, and arrhythmias because parental product contains 68% propylene glycol; paradoxical excitement and delirium may occur in infants experiencing pain

Drug Name	Pentobarbital (Nembutal)
Description	CNS sedative and hypnotic that acts primarily on the cerebral cortex and reticular formation through decreased neuronal synaptic activity.
Pediatric Dose	2-6 mg/kg IV slow push
Contraindications	Documented hypersensitivity (rare in neonates); severe uncontrolled pain
Interactions	Incompatible when admixed with cefazolin, cimetidine, clindamycin, fentanyl, hydrocortisone, insulin, midazolam, morphine, pancuronium bromide, phenytoin, ranitidine, or vancomycin; may cause respiratory depression with concurrent use of CNS depressants (eg, benzodiazepines); increased toxicity with CNS depressants and possibly phenobarbital
Pregnancy	D - Unsafe in pregnancy
Precautions	Caution with hypovolemic shock, CHF, hepatic impairment, chronic or acute pain, or renal dysfunction; may cause respiratory and cardiovascular depression; carefully monitor upon administration for hypotension, bradycardia, and arrhythmias because parental product contains 68% propylene glycol; paradoxical excitement and delirium may occur in infants experiencing pain

Drug Category: Neuromuscular blocking agents

These agents are used for skeletal muscle paralysis to maximize ventilation by improving oxygenation and

ventilation. They are also used to reduce barotrauma and minimize oxygen consumption.

Drug Name	Pancuronium (Pavulon)
Description	Neuromuscular blocker whose effects are reversed by neostigmine and atropine.
Pediatric Dose	Initial dose: 0.1 mg/kg (0.04-0.15 mg/kg) IV push Maintenance dose: 0.02-0.1 mg/kg/dose q30min to q3h prn
Contraindications	Documented hypersensitivity (rare in neonates)
Interactions	Dose-dependent increased toxicity with magnesium sulfate and furosemide (increase or decrease neuromuscular blockade); caution with coadministration with drugs that increase neuromuscular blockade (eg, aminoglycosides, inhaled anesthetics); avoid drugs that antagonize neuromuscular blockade or prolong muscular weakness (eg, corticosteroids, amphotericin B, phenytoin, verapamil)
Pregnancy	C - Safety for use during pregnancy has not been established.
Precautions	May cause hypoxemia (unlikely in a ventilated patient), tachycardia, BP changes, and excessive salivation; exercise caution in patients with preexisting pulmonary, hepatic, or renal disease; prolonged use may result in muscle delayed recovery of paralysis

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Further Inpatient Care

- Thorough cardiac examination is necessary to evaluate for cyanotic heart disease.
- Confirming the degree of pulmonary hypertension, prior to instituting therapy, is extremely important.

Further Outpatient Care

- Infants with MAS are at increased risk for adverse developmental outcomes and should be referred for developmental assessment as an outpatient.

Transfer

- Although initial stabilization is necessary at community hospitals, infants with MAS frequently require high-frequency ventilation, inhaled nitric oxide, or ECMO. Therefore, in the event of significant aspiration, transferring these infants to a regional neonatal intensive care unit as soon as possible is important.

Complications

- Children with MAS may develop chronic lung disease as a result of intense pulmonary intervention.
- Infants with MAS have a slightly increased incidence of infections in the first year of life because the lungs are still in recovery.

Prognosis

- Nearly all infants with MAS have complete recovery of pulmonary function.
- Events initiating the meconium passage may cause the infant to have long-term neurologic deficits, including CNS damage, seizures, mental retardation, and cerebral palsy.

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- Many infants who have experienced MAS have had prenatal and postnatal periods of hypoxia and acidosis; therefore, these individuals are at increased risk of significant CNS damage.
- Typically, medicolegal action is initiated by parents whose newborn develops long-term sequelae from significant perinatal hypoxia. Although the delivering physician is the primary focus of such a lawsuit, additional liability to other healthcare professionals may ensue from a poorly planned and executed resuscitation.
- Commonly, the providers of the tertiary intensive care are included in these lawsuits, which are usually due to complications of necessary complex and aggressive care. Although other organ systems may be damaged by the initial insult and subsequent therapy, they rarely are the basis of legal action.

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Media type: X-RAY

Media file 2: Acute atelectasis[View Full Size Image](#)

Media type: X-RAY

Media file 3: Pneumomediastinum from gas trapping and air leak



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Media file 4: **Left pneumothorax with depressed diaphragm and minimal mediastinal shift because of noncompliant lungs**



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Media file 5: **Diffuse chemical pneumonitis from constituents of meconium**



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