Prevention and management of meconium aspiration syndrome

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INTRODUCTION — Meconium aspiration syndrome (MAS) is defined as respiratory distress in newborn infants born through meconium-stained amniotic fluid (MSAF) whose symptoms cannot be otherwise explained [1]. MAS can present with varying degrees of severity from mild respiratory distress to life-threatening respiratory failure. Coordination of care between the obstetric and neonatal team is important to reduce the incidence of MAS, and to identify and provide emergent therapy in those who develop MAS to reduce morbidity and mortality [2].

The prevention, management, and outcome of MAS will be reviewed here. The pathophysiology, clinical features, and diagnosis of MAS are discussed separately. (See "Clinical features and diagnosis of meconium aspiration syndrome".)

PREVENTION — Because of the potential for poor outcome, the best approach for managing MAS is prevention.

Intrapartum management — Intrapartum care to reduce the incidence of MAS includes:

- Prevention of fetal hypoxia, which is thought to be an important contributor to the pathogenesis of MAS (see "Clinical features and diagnosis of meconium aspiration syndrome", section on 'Pathophysiology')
- Prevention of postmature (>41 or 42 weeks gestation) delivery

Prevention of fetal hypoxia — Continuous or periodic intrapartum fetal heart rate (FHR) monitoring has become a standard of care in the United States, particularly in pregnancies thought to be at higher risk for intrapartum fetal hypoxemia (eg, postterm pregnancy, intrauterine growth restriction, preeclampsia). Evaluation and interventions are implemented in cases with abnormal tracings indicative of fetal stress. Although the combination of a nonreassuring FHR tracing and thick meconium in amniotic fluid has been associated with an increased risk of MAS, the value of intrapartum fetal monitoring to predict fetal hypoxemia and subsequent intervention in cases with abnormal traces in preventing MAS has not been proven. (See "Intrapartum fetal heart rate assessment").

Prevention of postmature delivery — Because the risk of MAS is greatest in infants with a gestational age greater than 41 weeks, preventing delivery after 41 weeks gestation reduces the incidence of MAS. For women greater than 41 weeks of gestation, we suggest induction of labor rather than expectant management. Evidence demonstrating a decreased incidence of MAS due to a reduction in the number of postmature births is discussed separately. (See "Clinical features and diagnosis of meconium aspiration syndrome", section on 'Epidemiology', and "Postterm pregnancy", section on 'Management' and "Postterm infant", section on 'Neonatal complications'.)

Amnioinfusion — Amnioinfusion, the instillation of isotonic fluid into the amniotic cavity, has been advocated to improve neonatal outcome in women laboring with thick meconium in the amniotic fluid. The proposed benefits of amnioinfusion include dilution of thick clumps of meconium by the instilled fluid, and possible prevention or relief of cord compression. However, amnioinfusion is not beneficial in reducing
meconium-related neonatal morbidity, with the possible exception of settings with limited facilities to monitor the fetus during labor [3]. As a result, amnioinfusion is not recommended as a routine approach for mothers with meconium-stained amniotic fluid (MSAF). (See "Amnioinfusion: Technique").

Delivery room management

Obstetrical care — In the presence of MSAF, it had been common practice for obstetrical care providers to aspirate the upper airways on the perineum in an attempt to reduce the risk of MAS [4-7]. However, evidence from a large randomized controlled trial did not find a benefit of intrapartum suctioning in infants with MSAF [8,9].

In this study, 2514 infants of at least 37 weeks gestation with cephalic (vertex) presentation and MSAF of any consistency were randomly assigned to suctioning of the oropharynx, nasopharynx, and hypopharynx or no suctioning before delivery of the shoulders. Suctioning was performed with a 10- to 13-Fr suction catheter connected to negative pressure of 150 mmHg. Postnatal delivery room management was in accordance with the concurrent 2000 Neonatal Resuscitation program guidelines [5] and provided by clinicians blinded to the allocation group. The following findings were noted:

- The incidence of MAS did not differ between groups (4 percent in both).
- There were no significant differences between the control and section groups detected in any of the secondary outcomes: the need for mechanical ventilation for MAS (1 versus 2 percent), mortality (51 percent in both groups), duration of mechanical ventilation (four versus five days), duration of oxygen therapy (five versus six days), or length of hospital stay (eight versus nine days).
- No complications of suctioning were noted.

The lack of benefit from intrapartum suctioning may be because events leading to MAS occur in-utero or before delivery of the shoulders, and thus are not affected by oropharyngeal suctioning by the obstetrician during delivery. (See "Clinical features and diagnosis of meconium aspiration syndrome", section on 'Pathophysiology'.)

Neonatal care — After delivery, the guidelines from the International Liaison Committee on Resuscitation (ILCOR), American Academy of Pediatrics (AAP), and the American Heart Association (AHA) do not recommend suctioning in the vigorous infant with MSAF, as it does not improve outcome and may cause complications [10,11].

- This was illustrated in a trial in which 2094 infants ≥37 weeks gestational age, born through MSAF of any consistency, and apparently vigorous at birth, were randomly assigned to have tracheal intubation and suctioning or to be managed expectantly [4]. Rates of MAS (3.2 versus 2.7 percent) and other respiratory disorders (3.8 versus 4.5 percent) did not differ between groups. Of the 1098 infants who were successfully intubated, including 64 in the expectantly managed group, 3.8 percent had complications that usually were transient. These included bradycardia, hoarseness or stridor, and laryngospasm.
- A meta-analysis reported similar results that tracheal suctioning was not shown to be superior to routine resuscitation in vigorous term meconium-stained infants [12].

Additional evidence also suggests that endotracheal suctioning of nonvigorous neonates with MSAF is not beneficial. Two Indian trials of nonvigorous meconium-stained term infants found no additional benefit to infants who were assigned to endotracheal suctioning versus those who were assigned to no suctioning [13,14]. In these two studies, there were no differences between the two groups in either the incidence of MAS or mortality. The 2015 updated guidelines from the AHA, AAP, and ILCOR do not recommend routine endotracheal suction for nonvigorous infants with MSAF [10,11]. The care of these infants should be guided by the same general principles for further intervention including endotracheal intubation, which are based on inadequate respiratory effort (gasping, labored breathing, or poor oxygenation) or heart rate (<100 beats/min) (algorithm 1). These guidelines were revised due to the emphasis of preventing harm (eg, delay in providing
ventilation and preventing complications of intubation) and insufficient evidence that routine endotracheal suction is beneficial in this setting. (See "Neonatal resuscitation in the delivery room", section on 'Meconium stained amniotic fluid' and "Clinical features and diagnosis of meconium aspiration syndrome", section on 'Clinical features'.)

**Post-delivery** — Infants who develop MAS exhibit signs of respiratory distress immediately after birth. In a study of 394 term infants with MSAF, MAS developed in 18 of 96 patients with Apgar scores <8 (19 percent) and in 1 of 298 patients with an Apgar score ≥9 (0.3 percent) [15]. All 19 patients with MAS had signs of respiratory distress within 15 minutes of birth, which required ventilatory support in 16 of the infants. These results demonstrate that full-term infants with MSAF without any sign of respiratory distress or depression immediately after birth are unlikely to develop MAS.

As a result, in our practice, infants with MSAF who exhibit signs of respiratory distress in the delivery room are observed in the NICU or Special Care Nursery for four to six hours to ensure they transition successfully. Asymptomatic infants with Apgar scores ≥9 can be admitted to the normal nursery without additional monitoring or intervention.

**MANAGEMENT** — Although the management of MAS is largely supportive, the prompt identification and care of affected patients have decreased morbidity and mortality, especially in patients with severe disease. Coordination of care between the obstetric and neonatal team is crucial to initiate effective management of infants who develop MAS [2]. (See 'Outcome' below.)

The general approach to care includes:

- Maintenance of adequate oxygenation and ventilation
- Maintenance of adequate blood pressure and perfusion
- Correction of any metabolic abnormality including hypoglycemia and acidosis, which increase oxygen consumption
- Empirical antibiotic therapy
- Care in a neutral thermal environment (unless there are signs of hypoxic ischemic encephalopathy, which is treated with hypothermia) (see "Clinical features, diagnosis, and treatment of neonatal encephalopathy", section on 'Therapeutic hypothermia')
- Minimal handling of the infant to avoid agitation, which exacerbates persistent pulmonary hypertension of the newborn (PPHN), if present (see "Persistent pulmonary hypertension of the newborn")

**Respiratory management** — Respiratory management is focused on maintaining optimal oxygenation and ventilation, especially as hypoxemia, acidosis, and hypercapnia may increase pulmonary vascular resistance and contribute to the development of PPHN. Hyperventilation, respiratory alkalosis, and air-trapping should be avoided.

Supplemental oxygen therapy is usually adequate in patients with mild or moderate disease. In patients with severe disease, interventions may include mechanical ventilation, high frequency ventilation, surfactant therapy, and/or inhaled nitric oxide therapy. In patients with respiratory failure who have failed to respond to other interventions, extracorporeal membrane oxygenation (ECMO) may be a life-saving intervention.

**Oxygen therapy** — Supplemental oxygen therapy should be initiated to keep the infant well saturated (SaO₂ >99 percent) while diagnostic tests are performed. When the diagnosis is established, arterial PO₂ should be maintained in the range of 55 to 90 mmHg (SaO₂ >90 percent) to provide adequate tissue oxygenation and avoid lung injury that may result from continued administration of high concentrations of oxygen. Hypoxemia should be avoided because it contributes to pulmonary vasoconstriction and possibly PPHN. Umbilical arterial and venous (multiple lumen) catheters are used to monitor arterial blood gases and blood pressure, and infuse fluids and medications. (See "Continuous oxygen delivery systems for infants, children, and adults" and "Oxygen monitoring and therapy in the newborn").
**Assisted ventilation** — Assisted ventilation is used when gas exchange is not adequate with spontaneous breathing. When FiO₂ exceeds 0.4 to 0.5, continuous positive airway pressure (CPAP) may improve oxygenation. CPAP should be used cautiously in infants with hyperinflation as it may exacerbate air trapping.

Approximately 30 percent of patients with MAS require mechanical ventilation due to respiratory failure [16,17]. The goal for assisted ventilation is to achieve optimal gas exchange with minimal respiratory trauma. In infants with MAS, we typically target PaCO₂ levels between 50 to 55 mmHg and arterial PO₂ between 55 to 90 mmHg (SaO₂ >90 percent). We consider using high frequency oscillatory ventilation in infants who fail to respond to conventional mechanical ventilation, and in those who fail mechanical ventilation and pharmacologic treatment, extracorporeal membrane oxygenation (ECMO) therapy. (See "Mechanical ventilation in neonates", section on 'Indications for ventilation' and "Mechanical ventilation in neonates", section on 'High-frequency ventilation' and 'Extracorporeal membrane oxygenation (ECMO)' below.)

**Sedation** — Infants with MAS may breathe out of synchrony with the ventilator, which may aggravate agitation. Agitation may be associated with catecholamine release, increased pulmonary vascular resistance, right-to-left shunting, and hypoxemia.

The goal of sedative therapy is to maintain effective and safe sedation to achieve optimal gas exchange during the acute phase of the illness and allow for controlled weaning from assisted ventilation.

In these patients, we use an opioid analgesic for sedation and analgesia including the following agents. (See "Prevention and treatment of neonatal pain", section on 'Systemic analgesia'.)

- Intravenous morphine sulfate (loading dose of 100 to 150 mcg/kg over one hour, followed by a continuous infusion of 10 to 20 mcg/kg per hour)
- Intravenous fentanyl (1 to 5 mcg/kg per hour)

If dyssynchronous breathing persists and a specific cause cannot be identified (eg, airway obstruction or air leak), we may use neuromuscular blockade with pancuronium (0.1 mg/kg IV push per dose). However, we limit this intervention as much as possible because of potential adverse effects.

**Surfactant** — Surfactant may reduce the severity of respiratory disease and reduce the need for ECMO in mechanically ventilated infants with MAS [18,19]. This was illustrated in a meta-analysis of four trials that included 326 infants, which was reassessed in 2014 [18]. The following findings were noted:

- There was no difference in mortality rate between infants who received surfactant compared with placebo (relative risk [RR] 0.98, 95% CI 0.41-2.39).
- In two studies that enrolled 208 infants, surfactant reduced the need for ECMO therapy (RR 0.64, 95% CI 0.46-0.91).
- Surfactant compared with placebo did not reduce the risk of pneumothorax (three studies), pulmonary interstitial emphysema (one study), chronic lung disease (one study), air leaks (one study), duration of mechanical ventilation (three studies) or oxygen therapy (two studies), or intraventricular hemorrhage (two studies).

We do not routinely administer surfactant to all patients with MAS. However, we will administer surfactant (150 mg/kg [6mL/kg]) to patients with severe disease who are mechanically ventilated and require high FiO₂ (>0.5) and high mean airway pressure (>10 to 12 cmH₂O) [20]. Surfactant may also be helpful in infants with radiographic evidence of surfactant dysfunction (eg, low lung volumes and homogeneous pulmonary parenchymal disease that is similar in appearance to respiratory distress syndrome [RDS]).

Data are insufficient to determine whether lung lavage with diluted surfactant is beneficial. A meta-analysis that included three trials reported no differences in the two separate outcomes of mortality and the use of...
ECMO for patients treated with surfactant lavage and controls, but the lavage group had a better outcome with a composite outcome of death or the use of ECMO [21]. However, this study was limited by methodological issues due to the small number of patients, and the differences in study design (eg, severity of disease, volume of lavage, and use of subsequent bolus of surfactant). As a result, we do not routinely recommend tracheobronchial lavage with surfactant. The use of this modality may be warranted in patients with severe disease in centers without ECMO capabilities and who are experienced in performing this intervention [22,23]. However, lung lavage with surfactant in ventilated infants is technically demanding and is associated with serious complications. Further studies confirming the efficacy of this therapy in larger randomized control trials is required before lavage with surfactant can be routinely recommended for infants with severe MAS.

**Nitric oxide** — Inhaled nitric oxide (iNO) is a selective pulmonary vasodilator that may improve oxygenation in patients with associated PPHN. (See "Persistent pulmonary hypertension of the newborn", section on 'Inhaled nitric oxide'.)

Another pulmonary vasodilator agent used in the treatment of PPHN is sildenafil, a phosphodiesterase inhibitor. (See "Persistent pulmonary hypertension of the newborn", section on 'Other vasodilatory agents'.)

**Extracorporeal membrane oxygenation (ECMO)** — ECMO may be life-saving in infants who do not respond to mechanical ventilation, surfactant therapy, and/or iNO [24-26]. ECMO provides cardiopulmonary support while awaiting resolution of the underlying pulmonary disease process without further risk of injury from volutrauma from mechanical ventilation and the use of high concentrations of supplemental oxygen.

Both venovenous and venoarterial ECMO have been used in infants with meconium aspiration [27]. Although both methods are invasive compared with the medical treatments for MAS, venoarterial ECMO requires ligation of the carotid artery and may be associated with complications of pulmonary emboli from the ECMO circuit and increased left ventricular afterload. The addition of other therapeutic modalities, such as surfactant and iNO, has allowed the successful use of venovenous ECMO in infants with MAS, thus avoiding the more invasive venoarterial procedure [27].

**Circulatory support** — Therapeutic measures that insure adequate cardiac output and tissue perfusion include:

- Maintaining sufficient intravascular volume. Volume expansion using normal saline may be needed in infants with low blood pressure and inadequate tissue perfusion. Enteral feeds are not provided during severe respiratory illness. In patients with adequate circulation, parenteral fluid management during the first 24 hours of life is restricted to a volume of 65 mL/kg using a solution that consists of 5 percent dextrose without additional electrolytes. Subsequently, the volume is adjusted based on the needs of the infant, and sodium intake is limited to minimize peripheral and pulmonary edema. (See "Fluid and electrolyte therapy in newborns", section on 'Fluid requirements'.)

- Transfusion of packed red blood cells may be required to optimize tissue oxygen delivery, especially in patients with marginal oxygenation. In general, we maintain hemoglobin concentration above 15 g/dL (hematocrit above 40 to 45 percent) in severe MAS.

- Vasopressor support often is needed to maintain adequate blood pressure. We begin with a continuous intravenous infusion of dopamine (2.5 to 10 mcg/kg per min IV) and increase the infusion rate as necessary to maintain normal mean arterial blood pressure. Blood pressure may need to be higher in infants with PPHN to minimize right-to-left shunting. (See "Persistent pulmonary hypertension of the newborn".)

**Antibiotics** — It is uncertain whether or not antibiotics are beneficial in infants with MAS. Data are sparse and include an open-label randomized trial conducted in India that reported antibiotics did not reduce the incidence of culture-proven sepsis [28]. However, this study has several limitations. Clinicians were not blinded to the assigned intervention. In addition, the study may have been underpowered, as the overall
incidence of suspected sepsis was 9.6 percent, and the number of patients (n = 250) may be insufficient to demonstrate a potential benefit.

As a result, until there are data that conclusively demonstrate no benefit, we begin broad-spectrum antibiotics (ampicillin and gentamicin) while awaiting the results of blood cultures because of the risk of infection and the difficulty of distinguishing between meconium aspiration syndrome and bacterial pneumonia. (See "Clinical features and diagnosis of meconium aspiration syndrome", section on 'Infection' and "Management and outcome of sepsis in term and late preterm infants").

Corticosteroid — Although corticosteroid therapy has been proposed to reduce the severity of MAS, there is no evidence of its effectiveness in infants with MAS [29,30]. As a result, we do not recommend the use of corticosteroid therapy in patients with MAS unless future control trials demonstrate significant benefit from its use.

OUTCOME — The overall outcome of MAS has improved with advances in neonatal care [16,17].

In a retrospective review from a multicenter study from the United States that included 162,075 term infants born between 1997 and 2007, 1.8 percent of patients developed MAS [17]. The following findings were noted:

- Of the 7518 infants with MAS, 82 percent were discharged home from a nonintensive care setting, 7.9 percent required transfer for intensive care, and 1.2 percent died (n = 88).

- Extracorporeal membrane oxygenation (ECMO) treatment was performed in 61 neonates (1.4 percent) including three infants who died.

- Multivariate analysis showed mortality was independently associated with an Apgar score less than 3 (odds ratio [OR] 7.5, 95% CI 4.6-12.2), need for ventilatory support within the first 48 hours of life (OR 4.1, 95% CI 2.1-8.1), repeated doses of vasopressive agents (OR 3.8, 95% CI 2.2-6.4), presence of a major congenital anomaly (OR 2.1, 95% CI 1.4-3.4), and the use of cefotaxime (OR 2.1, 95% CI 1.4-3.4).

- The short-term morbidity of survivors included oxygen supplementation at 28 days of life (5 percent) and seizures (5 percent), and four patients developed necrotizing enterocolitis.

In this study, the mortality rate of 1.2 percent was lower in comparison to 4.2 percent that was reported in a large retrospective study from the United States of 176,790 infants born between 1973 and 1987 [16].

Pulmonary sequelae — Pulmonary morbidity, especially reactive airway disease, appears to be common in patients who had MAS, as illustrated by three small follow-up studies [31-33]:

- Pulmonary outcome was evaluated in 35 infants with MAS and 70 controls [33]. During the first six months after birth, the infants with MAS were significantly more likely to have one or more episodes of wheezing and/or coughing lasting ≥3 days (49 versus 20 percent) and receive bronchodilator therapy (23 versus 3 percent) compared with controls.

- Pulmonary function testing was performed at eight years of age in 11 children who had MAS and 9 controls [32]. The MAS group was more likely to have mild airway obstruction, hyperinflation, and increased closing volumes compared with controls, and had more exercise-induced bronchospasm (four versus zero children). However, during graded exercise stress tests, MAS children had normal maximal oxygen consumption and anaerobic threshold (also known as lactate threshold) without significant hypoxemia or hypercarbia. (See "Exercise physiology").

- Respiratory symptoms, pulmonary function tests, and chest radiographs were evaluated in 18 children ages 6 to 11 years who had MAS [31]. Seven children had recurrent cough and wheezing consistent with asthma, and five of these had exercise-induced bronchospasm that responded to bronchodilators. Of the 11 asymptomatic children, two had mild expiratory airflow limitation, one had exercise-induced
bronchospasm, and eight had normal pulmonary function. Chest radiographs were normal in all the children.

**Neurologic outcome** — There are limited data on the neurodevelopmental outcome of patients with MAS. Small observational case series report that about 20 percent have significant neurodevelopmental impairment [34]. However, birth depression occurs in 20 to 30 percent of patients with MAS, and it is likely intrauterine asphyxia and/or infection that are the major underlying pathologic factors resulting in poor neurodevelopmental outcome. (See "Clinical features, diagnosis, and treatment of neonatal encephalopathy").

**SUMMARY AND RECOMMENDATIONS** — Meconium aspiration syndrome (MAS) is defined as respiratory distress in newborn infants born through meconium-stained amniotic fluid (MSAF) whose symptoms cannot be otherwise explained. In severe cases, MAS can be life threatening. As a result, it is important to reduce the incidence of MAS and provide emergent therapy to reduce the mortality in those who develop MAS.

- Interventions used to prevent MAS include the following:
  - We suggest that intrapartum fetal heart rate (FHR) monitoring to detect episodes of fetal hypoxia be used in pregnancies with meconium-stained amniotic fluid (Grade 2C). Although fetal hypoxia is thought to contribute to the pathogenesis of MAS, the value of intrapartum fetal monitoring and subsequent intervention in cases with abnormal traces in preventing MAS has not been proven. (See 'Prevention of fetal hypoxia' above.)
  - For women ≥41 weeks of gestation, we recommend induction rather than expectant management (Grade 1B). (See 'Prevention of postmature delivery' above and "Postterm pregnancy", section on 'Management'.)
  - Dilution of thick meconium with amnioinfusion has not been shown to be effective in reducing the incidence of MAS. As a result, we do not suggest amnioinfusion as a routine intervention for mothers with MSAF unless there are coincident variable fetal heart rate decelerations (Grade 2B). (See 'Intrapartum management' above.)
  - At delivery, we do not recommend intrapartum suction for MSAF (Grade 1B). (See 'Obstetrical care' above.)
  - After delivery, we do not recommend endotracheal intubation and suctioning of newborn infants born through MSAF (Grade 1B). This recommendation includes both vigorous and nonvigorous infants. (See 'Neonatal care' above.)
  - Patients who develop MAS exhibit signs of respiratory distress immediately after birth. In our practice, infants with MSAF who exhibit signs of respiratory distress in the delivery room are typically observed in the neonatal intensive care unit (NICU) or Special Care Nursery for four to six hours to ensure that they transition successfully. Asymptomatic infants with Apgar scores ≥9 are admitted to the normal nursery without additional monitoring or intervention. (See 'Post-delivery' above.)

- The management of MAS is supportive. In our practice, we suggest the following approach (Grade 2C):
  - Maintenance of adequate oxygenation and ventilation – Respiratory management is focused on maintaining adequate oxygenation and ventilation, especially as hypoxemia, acidosis, and hypercapnia may increase pulmonary vascular resistance and contribute to the development of persistent pulmonary hypertension (PPHN). Supplemental oxygen therapy is usually adequate in patients with mild or moderate disease. In patients with severe disease, interventions may include mechanical ventilation, surfactant therapy, and/or inhaled nitric oxide (INO) therapy. In patients with respiratory failure who have failed to respond to other interventions, extracorporeal membrane oxygenation (ECMO) may be a life-saving intervention. (See 'Respiratory management' above.)
- Maintenance of adequate blood pressure and perfusion with sufficient vascular volume, and in some patients, the use of vasopressor agents, such as dopamine. Transfusion of packed red blood cells is also required to replace blood lost from sampling and to optimize tissue oxygen delivery. In our practice, the hemoglobin level is generally maintained at 15 g/L. (See 'Circulatory support' above.)

- Correction of any metabolic abnormality including hypoglycemia, acidosis, and/or electrolyte derangements.

- The administration of empirical antibiotic therapy while awaiting the results of blood cultures because it is difficult to differentiate MAS from bacterial pneumonia and sepsis. (See 'Antibiotics' above.)

- Care in a neutral thermal environment for infants without evidence of hypoxic-ischemic encephalopathy.

- Minimal handling of the infant to avoid agitation, which may exacerbate PPHN, if present. (See "Persistent pulmonary hypertension of the newborn".)

- The mortality rate of MAS has improved because of advances in neonatal care. In survivors, long-term morbidity includes pulmonary sequelae, particularly reactive airway disease, and neurodevelopmental impairment. However, it is likely that intrauterine hypoxia and chronic infection, which are possible contributors to MAS, are the major underlying factors that result in poor neurodevelopmental outcome in patients with MAS. (See 'Outcome' above.)

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Neonatal resuscitation algorithm: 2010 guidelines

- Term gestation? Breathing or crying? Good tone?
  - Yes, stay with mother
  - No
  - Warm, clear airway if necessary, dry, stimulate

- HR below 100, gasping, or apnea?
  - Yes
  - PPV, SpO2 monitoring
  - No

- HR below 100?
  - Yes
  - Take ventilation corrective steps
  - No
  - HR below 60?
    - Yes
    - Consider intubation
    - Chest compressions
    - Coordinate with PPV
  - No
  - Take ventilation corrective steps

- Intubate if no chest rise!
  - Consider:
    - Hypovolemia
    - Pneumothorax

- HR below 60?
  - Yes
  - IV epinephrine

- Targeted preductal SpO2 after birth
  - 1 min: 60-65 percent
  - 2 min: 65-70 percent
  - 3 min: 70-75 percent
  - 4 min: 75-80 percent
  - 5 min: 80-85 percent
  - 10 min: 85-95 percent

CPAP: continuous positive airway pressure; HR: heart rate; IV: intravenous; min: minute; PPV: positive pressure ventilation; SpO2: oxygen saturation measured by pulse oximetry.


Graphic 80621 Version 14.0
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