

Intensive Care Nursery House Staff Manual

Apnea and Bradycardia

DEFINITION: Pathologic apnea is a prolonged respiratory pause of ≥ 20 sec, or one associated with bradycardia or color change. The respiratory pause may be **central** (*i.e.*, no respiratory effort), **obstructive** (usually due to upper airway obstruction), or **mixed**. Short (≤ 5 sec) episodes of central apnea can be normal at all ages.

DIFFERENTIAL DIAGNOSIS:

1. Apnea of prematurity, most common in infants ≤ 34 weeks gestation. (**Note:** All following etiologies must be excluded or appropriately treated before a diagnosis of apnea of prematurity can be made).
2. Hypoxemia
3. Infection (sepsis, meningitis, pneumonia)
4. Necrotizing enterocolitis
5. Intracranial hemorrhage
6. Hydrocephalus
7. Seizures
8. Patent ductus arteriosus
9. Hypoglycemia
10. Anemia (**Note:** Apnea may improve after PRBC transfusion; however, infants who respond cannot be predicted by their Hct. Infants with a low Hct who respond to transfusion usually have an elevated blood lactate and elevated heart rate.)
11. Polycythemia/hyperviscosity
12. Atelectasis
13. Gastroesophageal reflux. Methylxanthines may exacerbate reflux. If reflux associated with apnea becomes a persistent problem, the head of the bed should be elevated 20 degrees. Metoclopramide (Reglan™: 0.1 – 0.2 mg/kg/dose, TID or QID), bethanechol, and H₂ blocking agents should be used only after reflux has been documented with pH probe analysis.
14. Feeding bradycardia (probably due to vagal stimulation from the NG tube or from gastric distension)
15. Following anesthesia or depressant drugs (This is frequently observed after repair of inguinal hernias in former preterm infants and can be minimized with perioperative caffeine administration. The susceptibility of preterm infants to apnea after anesthesia may persist until 50-60 post conceptional weeks.)
16. Maternal drug withdrawal
17. Elevated environmental temperature
18. Upper Airway obstruction (*e.g.*, due to nasal secretions, choanal atresia or stenosis, vocal cord paralysis)
19. Tracheal suctioning (due to hypoxemia and/or vagal stimulation)
20. PGE₁ infusion
21. Congenital Hypoventilation Syndrome (Methylxanthines are ineffective and these infants need mechanical ventilation.)

TREATMENT: If the infant has any of the above conditions, these should be treated appropriately.

- **Monitoring:** Infants at risk for apnea should have continuous monitoring of respiration and heart rate (because an apnea monitor alone will miss obstructive apnea) until they are at least 34 wks postconceptional age and have been free of apnea and bradycardia for one week.

-**Prevent hypoxemic episodes.**

-**Cutaneous stimulation** is effective with mild apnea.

-Maintain the infant in **prone position**.

-**Methylxanthines:** When idiopathic apnea of prematurity is severe enough to treat pharmacologically, the drug of choice is p.o. caffeine (even when the child is n.p.o. – except during necrotizing enterocolitis). A loading dose of caffeine citrate 20 mg/kg p.o. (10 mg/kg caffeine base) should be used, followed in 24 h by a daily maintenance dose of 5.0 mg/kg/d caffeine citrate. Therapeutic levels will usually be achieved within 30–120 minutes of the initial dose. Plasma caffeine levels are usually not measured, because some infants may require higher plasma levels for response and because caffeine toxicity is not generally a problem until plasma levels >50 mg/L. For infants who fail to respond to the above dosages, a second load of 10–20 mg/kg caffeine citrate can be given, followed by a maintenance dose of 7.5 mg/kg/d. If symptoms that could be attributable to methylxanthine toxicity occur (most commonly tachycardia, jitteriness and vomiting), theophylline concentrations as well as caffeine concentrations should be measured. Because of the long half-life of caffeine, therapeutic levels may persist for >7–14 d after discontinuing therapy. **Caffeine therapy needs to be discontinued well before discharge.**

-**Respiratory support:** Continuous air flow through nasal cannulae is useful in some infants, and others will respond to nasal CPAP. If apnea is severe, the infant may require mechanical ventilation.

FEEDING AND CONTROL OF BREATHING: Preterm infants have a 50% fall in minute ventilation during nipple feedings, which may progress to hypoxemia, apnea, and bradycardia. Feeding hypoxemia resolves with maturation. It is usually gone by 44 weeks postconceptional age, but occasionally may last longer. Infants are treated by frequent interruptions during a feed, by supplemental oxygen during a feed, or, in extreme cases, by gavage.

HOME APNEA MONITORING: Home monitoring is not indicated for normal infants or for asymptomatic preterm infants.

SUPINE POSITION FOR SLEEP: DISCHARGE RECOMMENDATIONS: **Supine positioning during sleep reduces the incidence of SIDS in normal infants.** Premature infants should be encouraged into the supine position prior to discharge and mothers should be advised of the advantages of the supine position. These recommendations do not apply to preterm infants with apnea in hospital (who are being monitored for apnea), or to those with Pierre Robin sequence, laryngomalacia, or gastroesophageal reflux.