Prematurity: Clinical Issues, Part One

Objectives:

• Define prematurity.
• Describe risk factors and long-term outcomes related to premature birth.
• Describe lung development and the role of surfactant.
• State the incidence, signs and symptoms, treatment options, and outcomes for several disease processes in the premature or term infant including RDS, CLD, PDA, and apnea of prematurity.
• Describe the thermoregulation management of the premature infant.

Classification by Age

Preterm (or premature) infant
- Infant born before 37 completed weeks of gestation

Late preterm infant
- Infant born between 34 and 36 weeks gestation

Moderately preterm infant
- Infant born between 32 and 36 completed weeks of gestation

Very preterm infant
- Infant born before 32 completed weeks of gestation

Extremely preterm infant
- Infant born before 25 weeks of gestation

Classification by Weight

Normal weight
- 2500 - 4000 grams at term

Low birth weight
- < 2500 grams at birth

Very low birth weight
- < 1,500 grams at birth

Extremely low birth weight
- < 1,000 grams at birth

High birth weight
- > 4000 grams at birth

Classification by Weight and Age

• SGA – small for gestational age
  - Birth weight less than 10th percentile

• AGA – appropriate for gestational age
  - Birth weight within 10th and 90th percentile

• LGA – large for gestational age
  - Birth weight greater than 90th percentile

The SGA Infant

• Infant with a birth weight below 10th percentile due to constitutional factors including:
  - Maternal height and weight
  - Ethnicity
  - Parity

• These infants are typically not at increased risk for perinatal mortality or morbidity
The LGA Infant

- **Appearance:**
  - Macrosomia
  - Large body with normal head circumference
  - Infant of diabetic mother (IDM)

- **Potential problems:**
  - Hypoglycemia and hyperinsulinemia
  - Birth trauma
  - RDS (6x more frequently in IDM than non IDM)
  - Polycythemia (↑glucose → ↓O\(_2\) tension→↑erythropoiesis)
  - Pulmonary hypertension
  - Poor feeding
  - Thermal instability
  - CHD (30%)

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LGA Newborn

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Risk Factors for Preterm Birth

- The best predictors of having a preterm birth are:
  - History of preterm labor/delivery
  - Multiple gestation
  - Uterine and/or cervical abnormalities

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Preterm Birth Survival Statistics

- 23 weeks = 30%
- 24 weeks = 50 – 60%
- 25 weeks = 75%
- 26 weeks = 86%
- 27 weeks = 88%
- 28 weeks = 92%
- 29 weeks = 95%

Additional Information: Late Preterm Infant Statistics
- 70 percent of premature babies are born between 34 and 36 weeks gestation
- More than 99% survive, though they are:
  - 6 times more likely than full-term infants to die in the first week of life (2.8 per 1,000 vs. 0.5 per 1,000)
  - 3 times more likely to die in the first year of life (7.9 per 1,000 vs. 2.4 per 1,000)
  - more than 3 times as likely to develop cerebral palsy and are slightly more likely to have developmental delays than babies born full term.

Additional Information: Preterm Birth Statistics – WA state
- 9.8% of live births – born preterm
- 7.0% of live births – born late preterm
- 1.4% of live births – born very preterm
- In an average week:
  - 164 babies are born preterm – < 37 weeks
  - 24 babies are born very preterm – < 32 weeks
  - 103 babies are born low birth weight – < 2,500 gm
  - 16 babies are born very low birth weight – < 1,500 gm
Total live births = 86,976 (based on 2011 statistics)

March of Dimes Premature Birth Report Card 2015


Morbidity of Prematurity
**VON NICUs**
- Respiratory distress syndrome (RDS) – 70%
- Chronic lung disease (CLD) – 22%
- Intraventricular hemorrhage (IVH) – 19%
- Grade III/IV hemorrhage – 4.8%
- Periventricular leukomalacia (PVL) – 2.6%
- Necrotizing enterocolitis (NEC) – 3.6%
- Patent ductus arteriosus (PDA) – 26.7%
- Late Onset Infection – 10.8%
- Retinopathy of prematurity (ROP) – 24.8%

Morbidity of Prematurity
**Long term issues**
- Chronic lung disease
- Cerebral palsy; motor and cognitive deficits
- Visual & hearing impairments; visual-motor issues
- Growth impairment; feeding difficulties
- Chronic kidney disease; hypertension
- Neuro-developmental lags
- Attentional problems; school difficulties
- Behavioral problems, psychological & psychosocial issues
- Special health care needs, increased re-hospitalization rates

Neurodevelopmental Outcomes

- **23 – 24 weeks GA**
  - Severe disability = 21 – 23%
  - Moderate disability = 26 – 33%
  - Mild disability = 30 – 39%
  - No disability = 13 – 16%

- **25 – 26 weeks GA**
  - Severe disability = 10%
  - Moderate disability = 28 – 34%
  - Mild disability = 33 – 44%
  - No disability = 17 – 23%

- **27 – 28 weeks GA**
  - Severe disability = 10%
  - Moderate disability = 10%
  - Mild disability = 35%
  - No disability = 45%


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Stages of Lung Development

<table>
<thead>
<tr>
<th>Embryonic phase:</th>
<th>Pseudoglandular phase:</th>
</tr>
</thead>
<tbody>
<tr>
<td>3 – 5 weeks gestation</td>
<td>6 – 16 weeks gestation</td>
</tr>
<tr>
<td>Respiratory bud forms</td>
<td>Bronchial tree from trachea to terminal bronchioles are formed.</td>
</tr>
<tr>
<td></td>
<td>Lung arterial and venous systems develop.</td>
</tr>
</tbody>
</table>

Embryonic: ![Embryonic Lung](image)

Pseudoglandular: ![Pseudoglandular Lung](image)

Canalicular Phase: 17 – 24 weeks

- Airways become tubular, increase in length & diameter
- Terminal sacs/primitive alveoli begin to form
- Pulmonary vasculature continues to develop
- Epithelial cells subdivide into type I (for gas exchange) and type II pneumocytes (for surfactant production)

Canalicular: ![Canalicular Lung](image)

Saccular Phase: 24 wks – term

- Intense vascularization of the lung
- Elastic fibers begin to develop.
- Close connections form between air spaces & pulmonary capillaries.
- Lymphatic capillaries are being developed.
- Surfactant production increases.

Saccular: ![Saccular Lung](image)

Alveolar Phase: 36 wks – term

- Terminal air sacs are refined and alveolar walls are formed
- Total # of airways is now complete
- One-fifth the # of alveoli of an adult
- Alveoli continue to increase in number, size, and shape throughout early childhood.

Alveolar: ![Alveolar Lung](image)
Premature Birth Interrupts Lung Development

Surfactant – What is it?

- Naturally occurring substance containing lipids (90%) & proteins (10%) – produced by Type II pneumocytes in the lung
- Detected as early as 22 weeks gestation in the form of a phospholipid – lecithin
- 'Mature' surfactant (main component – phosphatidylcholine) is prevalent after 34 weeks gestation

Factors that Impede Surfactant Production

- Maternal diabetes, chorioamnionitis
- 2nd born twin
- Male gender
- C-section without labor
- Meconium in the airway
- Prematurity, lower gestational ages
- Perinatal asphyxia
- Presence of hypoxia, acidosis, and hypothermia
- Surfactant protein B deficiency

Surfactant – What does it do?

- Lowers surface tension at the air-water interface in the alveolus, allowing expansion of alveolus and prevents deflation by maintaining surface pressure at end expiration.
- Overall, surfactant helps to increase lung compliance, provide alveolar stability, and decrease alveolar opening pressure.

Prematurity: Respiratory Conditions
Respiratory Distress Syndrome (RDS)

- Develops within the first few hours after preterm birth secondary to:
  - Surfactant deficiency
  - Pulmonary hypo-perfusion
  - Anatomic immaturity, weak musculature
  - Immature respiratory center

RDS - Incidence

- Inversely proportional to GA
- Affects 60% of infants born < 30 weeks gestation
- Higher incidence in males and infants of multiple gestation pregnancies
- Occasionally seen in term infants (e.g. diabetic mothers, birth asphyxia)

ELBW Infants – Lung Characteristics

- Stage of lung development at the time of preterm birth = Canalicular stage
  - Alveoli not truly present = terminal sacs, few in number compared to term infant
  - Pulmonary vasculature not well established
  - Distance between air spaces and pulmonary capillaries
- Surfactant deficiency -
  - Alveoli require much higher opening pressures
  - Alveoli collapse more readily on deflation

RDS - Characteristics

- Atelectasis/alveolar collapse, pulmonary edema, and cellular injury occur
  - ↓ FRC, alteration in ventilation perfusion ratio, and uneven distribution of ventilation, low surface area for gas exchange
  - Inadequate gas exchange, hypoxia, hypoventilation
- Ventilator-induced lung injury ensues:
  - ↑ levels of inflammatory cells, free radicals, and inflammatory cytokines

RDS – Clinical Signs

- Tachypnea
- Grunting, nasal flaring, and retractions
- Cyanosis
- Increasing O2 requirements
- Crackles, diminished breath sounds
- Tachycardia
- Within 24-48 hours:
  - Oliguria, generalized edema, heart murmur

Diagnostic Evaluation

- Arterial blood gas
- Chest x-ray
  - RDS:
    - low lung volumes
    - hazy lung fields
    - fine reticuloalveolar pattern of density
    - air bronchograms
Blood Gas & Oxygenation Thresholds for Intervention:

- Increased PCO2 - > 60 mmHg
- PaO2 < 50 mmHg in 100% oxygen
- Inability to maintain acceptable oxygen saturation levels despite high supplemental oxygen; rapidly increasing O2 requirement

Respiratory Support

- Nasal CPAP
  - Continuous positive airway pressure (nasal mask or prongs)
  - Goal is to prevent alveolar collapse at end expiration.
  - Allows supplemental oxygen to be delivered continuously.
  - Used for moderate respiratory distress and for recurrent apnea

NAVA Ventilation

Invasive & Noninvasive

- Neurally Adjusted Ventilatory Assist
  - Variable pressure ventilation – proportional to diaphragmatic effort and triggered by the diaphragmatic electromyography (EMG), called the Edi signal
  - Edi is measured using a naso-gastric catheter
  - Patient-controlled timing and magnitude of the respiratory cycle

MECHANICAL VENTILATION

Goal – provide the most effective gas exchange with the least amount of damage to the lungs

Inhaled Nitric Oxide

- Nitric oxide is an endogenous vasodilator.
- Treatment for infants with severe respiratory distress and hypoxemia
- Causes selective pulmonary vasodilation, thus lowering pulmonary vascular resistance and improving arterial oxygenation

RDS – Early Management

- Early administration of surfactant
- Sustain lung recruitment by utilizing appropriate ventilator strategies to prevent atelectasis.
- Achieve and maintain optimal lung inflation or functional residual capacity by providing appropriate tidal volumes/PIPs/PEEP
- Minimize oxygen toxicity
- Manage fluid & electrolytes and optimize nutrition
Goals for Blood Gases

- pH: 7.25 – 7.4
- PaCO2: 35-55 (preterm infant 40 – 55)
- PaO2: 50-75
- Keep PaCO2 in normal range particularly in first 72 hours – to keep cerebral blood flow within normal limits during time of maximal risk for IVH
  - low PaCO2 – lowers cerebral blood flow and is associated with ischemic brain injury (PVL)

Chronic Lung Disease

- Also known as Bronchopulmonary Dysplasia (BPD)
- Major cause of morbidity & mortality in preterm infants.
- Affects 37% of VLBW infants
- Generally a progression from RDS to chronic impairment in lung function once O₂ toxicity, barotrauma, and other factors have produced cellular, airway, and interstitial changes.

Chronic Lung Disease – CLD

CLD/BPD Definition

<table>
<thead>
<tr>
<th>Gestational age</th>
<th>Treatment with oxygen</th>
<th>Clinical criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;37 weeks</td>
<td>&gt; 30 days</td>
<td>Breathing room air, PAO₂ ≥ 50 mm Hg</td>
</tr>
<tr>
<td>&lt;30 weeks</td>
<td>&lt; 30 days</td>
<td>Breathing room air, PAO₂ ≥ 34 mm Hg</td>
</tr>
</tbody>
</table>

- Mild
  - Breathing room air, PAO₂ ≥ 50 mm Hg
- Moderate
  - Breathing room air, PAO₂ ≥ 34 mm Hg
- Severe
  - Breathing room air, PAO₂ ≥ 34 mm Hg

CLD – Multifactorial Etiology

- Prematurity
- Premature extra-uterine lung development
- Surfactant deficiency
- Mechanical ventilation
- Ventilator induced lung injury
- Oxygen toxicity
- Infection/inflammation
- PDA/excessive fluid intake
- Poor nutrition
- Arrested lung development- fewer and larger alveoli
- Vascular maldevelopment- fewer capillaries

CLD – Radiographic Evidence

- Chest x-ray showing lung collapse, fibrosis, cystic changes, and over-distention of the lungs.
Characteristics – CLD

- Tachypnea, retractions, and increased oxygen consumption secondary to hypoxia and elevated CO$_2$ levels
- Chronically compensated respiratory acidosis throughout the first year of life
- Cyanosis
- Wheezing
- Pulmonary edema secondary to capillary leak syndrome; crackles on auscultation
- Fluid issues (at risk for fluid overload)
- Mucous plugging

Characteristics – CLD

- Episodes of bronchospasm and airway obstruction
- Poor weight gain/growth
- Pulmonary hypertension
- Increased pulmonary resistance
- Limited surfactant production and function
- Pulmonary air leaks
- Tracheo-bronchial injuries

Management – CLD

- Oxygen therapy - promote adequate and stable oxygenation for growth and healing, prevent recurrent hypoxemia
- Anti-oxidant therapy (Vitamin A) to neutralize and clear free oxygen radicals
- Diuretics- counteract increased interstitial fluid and pulmonary edema
- Bronchodilators-
  - decrease pulmonary resistance, diaphragmatic fatigue, & pulmonary edema
  - increase compliance & minute ventilation

Management – CLD

- Nutritional support- promote body and lung growth, compensate for increased oxygen and caloric consumption
- Developmental enhancement- maximize the infant’s developmental potential and minimize the effects of stress on weight gain, respiratory function, and development

Long Term Consequences

- Prolonged oxygen therapy over many months
- Feeding problems
- Inguinal hernias
- Risk of RSV (respiratory syncytial virus)
- Risk for re-hospitalization due to respiratory illness
- Increased risk for neurodevelopmental problems

Prematurity: Patent Ductus Arteriosus

- The Ductus Arteriosus (DA) is the vascular connection or shunt between the pulmonary artery and the aortic arch, allowing the majority of blood to be diverted away from the fetal lungs.
- If the DA remains open beyond 72 hours of life, it is defined as ‘Patent Ductus Arteriosus’.
Fetal Circulation

- Typically occurs by 48-72 hours of age in the term neonate, variable in the preterm neonate
- As pulmonary vascular resistance (PVR) decreases, blood flow through the DA is from left to right and then ceases.
- Influenced primarily by oxygen levels and vasoconstrictive substances – PGE₂, prostaglandin synthetase, and bradykinin
- DA may reopen in response to hypoxia, acidosis, and increased PVR.

Anatomical Closure of DA

- Typically occurs by 2-3 months of age
- Involves endothelial destruction, proliferation of subintimal tissue, and formation of connective tissues
- Fibrous strand known as the ligamentum arteriosus

Patent Ductus Arteriosus

- Definition
  - DA remaining open beyond 72 hours of life

Preterm Infant Risk factors for PDA

- Less developed pulmonary arterial muscle as compared to term newborns
- Immature lung parenchyma
- Respiratory distress syndrome
- Episodes of hypoxia and acidosis – cause DA smooth muscle to relax/dilate
- DA is not as responsive to increased O₂ content as in term newborns

Physiologic Consequences

- Pulmonary blood flow
- Reduced systemic perfusion
- Deterioration in lung function and pulmonary edema
- Compromised brain, gut, and/or renal perfusion
- Biventricular failure & pulmonary hypertension
Moderate to Large PDA shunts

- Increased pulmonary venous congestion and interstitial edema, with subsequent reduced lung compliance
- Symptoms of CHF, mild cardiomegaly
- Inability to wean from ventilatory support – prolonged course due to PDA increases risk for development of chronic lung disease
- Increased oxygen requirement
- Increased CO2 retention

Other Clinical Features

- Tachypnea
- Tachycardia
- Widened pulse pressure, low diastolic pressure
- Bounding peripheral pulses
- Heart murmur – audible at the left upper sternal border
- Active precordium
- Apnea and bradycardia

PDA - Diagnosis

- Physical examination findings
- Increased ventilatory support/respiratory compromise
- Chest x-ray
  - Increased vascular markings
  - Cardiomegaly – late sign
- Echocardiogram

PDA - Treatment/Management

- Fluid restriction
- Diuretics
- Ventilatory support
- Prostaglandin synthetase inhibitor:
  - Indomethacin
  - Ibuprofen
- Surgical ligation if medical treatment fails

Indomethacin/Ibuprofen

Contraindications for use

- Bleeding, especially those with active IVH or GI bleeding
- Thrombocytopenia
- Coagulopathies
- Suspected or proven NEC
- Renal impairment

Apnea of Prematurity
Definition

- Cessation of breathing for 20 seconds or cessation of breathing for 10–15 seconds accompanied by bradycardia and/or cyanosis.
- If demonstrated within the first 24 hours of birth, may be considered pathologic.
- After 24 hours, often considered a condition related to immaturity.
- Commonly seen in premature infants and typically resolves by 36 weeks.

Incidence

- Most apnea occurs in healthy preterm infants without organic disease.
- Up to 80% infants weighing <1000 grams and 25% weighing <2500 grams at birth will have apnea during their neonatal course.
  (Miller & Martin, 1998)

Types of Apnea

- **Central Apnea**
  - Absence of airflow and respiratory effort
  - Cessation of chest wall motion due to loss of respiratory neural output
- **Obstructive Apnea**
  - Absence of airflow with continued respiratory effort
  - Associated with blockage of airway, positional issue, or anatomic abnormalities

Types of Apnea

- **Mixed apnea** – a combination of central and obstructive apnea, with obstructed respiratory efforts intermittently throughout the apnea
- 50–60% of neonatal apnea episodes are mixed.

Apnea of Prematurity

- Recurrent apnea seen in preterm infants who show no other abnormalities
- Diagnosis after exclusion of pathologic processes
- Onset – within the first week of life, usually at 24 – 48 hours
- More likely to be obstructive than central in the first 2 days of life

Pathophysiology

- **Immaturity of the brainstem**
  - Incomplete organization and connections of respiratory neurons
- **Chemoreceptor Response**
  - Premature infants are less responsive to hypoxia and increased CO2 – triggering increased ventilation
Other Causes of Apnea

• RDS
• Pneumonia
• Aspiration
• Acidosis
• Airway obstruction
• Atelectasis
• Pneumothorax
• Pulmonary hemorrhage
• Congenital anomalies of the upper airway
• Hypotension
• Arrhythmias
• Congestive heart failure
• PDA
• Sepsis
• NEC
• Seizures
• Asphyxia
• IVH
• Maternal drugs
• Narcotics
• Analgesics
• β-blocker antihypertensive agents
• Magnesium sulfate
• Neonatal drugs
• Narcotics
• Prostaglandin E
• Phenobarbital
• Metabolic derangements
• Polycythemia
• Anemia
• Gastroesophageal reflux
• Hyperthermia
• Hypothermia
• Rapid warming
• Stooling
• Painful stimuli

Management

• Treat underlying cause if determined
• Avoid triggering reflexes
• Maintain prone position when possible
• Maintain neck in neutral position
• Avoid vigorous manual ventilation
• Avoid painful stimuli, noise, noxious stimuli, potent odors

Thermoregulation

Not too hot…

Not too cold…

Getting it just right…

Vulnerability of ELBW & VLBW infants:

• Absent or immature heat conserving and production mechanisms
• Low muscle tone, extended positioning
• Decreased fat insulation
• Limited or absent metabolic stores, including brown adipose tissue
• Inadequate caloric intake
• Thin, permeable skin → high evaporative losses
Surface area-to-body mass ratio

- The term infant’s surface-to-body mass ratio is typically **3 times greater** than that of an adult.
- The preterm infant’s surface-to-body mass ratio may be up to **6 times greater** than that of an adult.

*Larger surface area-to-body mass ratio increases the rate & degree of heat transfer from within the body to the surface.*

Goals for Thermoregulation

- Maintain infant axillary temp 36.5 – 37.0°C
- Prevent heat loss and minimize fluctuations in infant’s temperature
- Rewarm the infant when necessary
- Minimize stress to the infant’s respiratory and metabolic systems
  - Maintain a neutral thermal environment – the range of ambient temperatures that support/maintain an infant’s core temperature at rest between 36.5°C-37.0°C, and the core and mean skin temperature changes less than 0.2°C-0.3°C/hour

Thermoneutral State

- State in which body temperature is maintained within a normal range and calorie expenditure and oxygen consumption are minimal
- Infant is neither gaining nor losing heat and the core-to-skin temperature gradient is small

Methods of Heat Loss or Gain

- **Conduction**
- **Convection**
- **Radiation**
- **Evaporation**

Conduction

- Transfer of heat between two solid objects that are in contact
  - Heat flux between infant’s body surface and solid surfaces

Convection

- Transfer of heat between a solid surface (the infant) and either air or liquid
  - Losing heat to the environment – air currents, drafts
  - Loss of heat during a bath
Radiation

- Transfer of radiant energy from the body surface (through absorbance and emission of infrared rays) to surrounding cooler or warmer surfaces
  - Walls, windows, heat lamps, light bulbs, etc

Radiant Heat

- “Greenhouse effect” can be created in incubators when sunlight or other radiant heat sources transmit heat through the Plexiglas
- Radiant heat loss is the most common type of heat loss for neonates
  - Exception: evaporative losses exceed radiant heat losses in VLBW infants in the first week of life - secondary to their almost transparent and gelatinous skin

Evaporation

- Loss of heat through water from the infant’s skin surface or respiratory tract
- Produces heat loss through the energy used in the conversion of water to its gaseous state

VLBW Infants at Risk...

- Evaporative heat losses alone are often greater than their heat producing ability
- May lose up to 120 mL/kg/day through evaporation in the first week of life
  - ↑ risk with the use of phototherapy, warming lights, radiant warmers
  - This represents a loss of 72 kcal/kg/day!

Physiologic Response to Cold Stress

- Norepinephrine release:
  - Pulmonary vasoconstriction, R – L. shunting with subsequent HYPOXEMIA
  - Peripheral vasoconstriction –
    - Decreased O2 delivery to tissues (HYPOXIA)
  - Increased metabolic rate –
    - Increased O2 consumption, HYPOXIA
  - Increased glucose utilization, HYPOGLYCEMIA

Physiologic response to cold stress

- If the infant is already experiencing respiratory distress, oxygenation may be significantly compromised – anaerobic metabolism will ensue in the absence of available O2, with subsequent lactic acid production (↓pH).
- With progressive hypothermia (below 36.0), the infant may exhibit decreased level of consciousness, hypoventilation, bradycardia, and hypotension.
Other signs of cold stress

- Lethargy or restlessness
- Pallor
- Cool skin
- Tachypnea
- Respiratory distress

Thermoregulation – First hours of life

- Effects of significant hypothermia in ELBW infants in the immediate postnatal period:
  - Pulmonary hypertension
  - Worsening respiratory distress
  - Hypoxia, respiratory & metabolic acidosis
  - Hypotension, hypovolemia
  - Altered cerebral blood flow
  - Increase risk of mortality

Delivery Room management for VLBW & ELBW infants

- Increase room temperature
- Pre-heat bed surface & linens
- Chemical heating mattress/pad
- Plastic wraps
- Flexed positioning & containment
- Hat
- Pre-warm equipment/objects
- Monitor axillary temperature every 30 minutes – 1 hour.

Ongoing thermal management

- Utilize servo-control on radiant warming bed. Compare infant’s temperature (thermometer reading) with skin/servo probe temperature – calculate “set-point” temperature accordingly.
- Move infant into incubator ASAP to diminish potential for evaporative, convective, and radiant heat losses occurring in the open environment.
- Humidity for ELBW infants 1-2 weeks of life

Guidelines for rewarming

- Rewarm slowly while monitoring:
  - Temperature
  - Heart rate and rhythm
  - Blood pressure
  - Respiratory rate and effort
  - Oxygen saturation
  - Blood glucose
  - Acid/base status

- Remove caps, plastic wraps, and heat shields to prevent them from interfering with heat gain.
- Incubator – set air temperature 1-1.5°C above core temperature OR use servo-control with skin temperature probe.
- Radiant Warming Bed – utilize skin probe/servo control; monitor both axillary and skin probe temperatures – adjust desired set point accordingly.
Guidelines for rewarming

Radiant warming bed or lamps – utilize caution: when radiant heat output is high, skin blood vessels may dilate rapidly and cause hypotension.

- Heating pad may be used – disposable warming mattresses should be approved for use in neonates; electric/water heating pads should be set no higher than 38°C.

References:

A. Rahmani, J. Khan, J. Corder, E. Almonte, F. Cheudi | Neurally Adjusted Ventilatory Assist in Neonates: Applications & Limitations. [NPM 2012; Volume 5; Number 2]


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