

CME

Neonatal and Maternal Temperature Regulation During and After Delivery

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An important goal of obstetric anesthesia is to provide for the safety and comfort of the mother as well as to optimize physiologic outcomes for the neonate. Markers of neonatal physiologic outcome include cord umbilical artery pH and Apgar scores. Neonatal temperature has often been overlooked by anesthesiologists as an important physiologic outcome measure, but it may be significantly affected by operating room conditions and obstetric anesthesia technique at cesarean delivery. There is a dose-dependent increase in mortality with decreasing body temperature as well as an increased likelihood for more severe early respiratory distress. Multiple neonate-focused strategies have been shown to decrease the incidence of neonatal hypothermia. Because fetal temperature is affected by maternal temperature, strategies to mitigate maternal hypothermia at the time of delivery may also be important in preventing neonatal hypothermia. This focused review will examine the importance of neonatal temperature and discuss its relationship to maternal temperature as well as strategies for maintaining neonatal normothermia after delivery. (Anesth Analg 2016;123:168–72)

An important goal of obstetric anesthesia is to provide for the safety and comfort of the mother as well as to optimize physiologic outcomes for the neonate. Markers of neonatal physiologic outcome include cord umbilical artery pH and Apgar scores. Neonatal temperature has often been overlooked by anesthesiologists as an important physiologic outcome measure, but it may be significantly affected by operating room (OR) conditions and obstetric anesthesia technique at cesarean delivery. Neonatal hypothermia is categorized by the World Health Organization into 3 stages based on core temperature, prognoses, and action required.¹ These stages include cold stress: 36.0 to 36.4°C, moderate hypothermia: 32.0 to 35.9°C, and severe hypothermia: <32.0°C. This focused review will examine the importance of neonatal temperature and discuss its relationship to maternal temperature as well as strategies for maintaining neonatal normothermia after delivery.

TEMPERATURE REGULATION IN THE NEONATE

Body temperature is tightly controlled, with little variability in heat loss or gain, and finely tuned to maintain body temperature in the desired range for optimal metabolism, growth, and survival. Control of body temperature can only be accomplished over a narrow range of ambient conditions in the absence of external inputs of heat and/or aids to preserve heat. In the unclothed resting adult, the lower limit of the thermoneutral zone, defined as the ability to maintain

normal body temperature without needing to use energy above and beyond normal basal metabolic rate, is 26 to 28°C at 50% relative humidity; in the naked full-term newborn infant, it is much higher (32–35°C), and in small premature infants, the lower limit of the thermoneutral zone may be as high as 35°C.²⁻⁴ For >40 years, hypothermia has been recognized as an independent risk factor for death in newborns.⁵⁻⁹ Hypothermia is also associated with morbidities, including late-onset sepsis and respiratory complications.^{8,10,11} Thus, deliberate management strategies are essential for maintaining infant temperature in an optimal range (36.5–37.5°C) in the immediate postdelivery period at all gestational ages. Thermoregulatory controls during the early neonatal period, especially in the premature infant, are immature. At birth, the infant's core temperature can decrease rapidly because of evaporative losses from the wet body, a large surface area/body mass ratio (particularly prominent in the premature infant compared with the term infant and adult), and a cold delivery room environment resulting in large radiant and convective heat loss.⁵⁻⁷ The premature infant has high insensible water losses, in part, because of poorly developed stratum corneum that facilitates free movement of water. In the absence of thermal protection, the newborn at all gestational ages may lose considerable heat, resulting in a decrease in the infant's body temperature. Indeed, a naked, wet, term infant immediately after delivery placed on an open table with a room temperature of 25°C will lose up to 4°C in skin temperature and 2°C in core body temperature within 30 minutes.¹² As a consequence of body cooling, there will be an increase in metabolic stress with concomitant increase in oxygen consumption.² If gas exchange is compromised, a higher oxygen requirement may result.

ADVERSE NEONATAL EFFECTS OF HYPOTHERMIA

Hypothermia is an independent risk factor for death in newborns.⁵⁻⁷ Several observational studies have demonstrated an inverse relationship between each additional degree of hypothermia <36.5°C measured at birth and subsequent neonatal death. Specifically, in 1 multicenter U.S. study of

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infants weighing <1500 g, for every 1°C decrease in temperature <36.5°C, the risk of death increased by 28%.⁸ In a second study in a resource-limited setting, mortality increased significantly with moderate hypothermia between 35 and 36.0°C (relative risk [RR], 4.66; 95% confidence interval [CI], 3.47–6.24) and severe hypothermia (<35.0°C) (RR, 23; 95% CI, 4.31–126); the adjusted mortality risk was significantly higher among preterm infants (RR, 12; 95% CI, 6.23–23.18) compared with term infants (RR, 3.12; 95% CI, 1.75–5.57).⁹ Stressed and acutely hypoxic infants appear limited in their ability to use the normal mechanisms of heat production and are less able to maintain body temperatures.^{12,13}

Although the mechanisms predisposing to the increased mortality are not clear, it has been postulated that alterations of normal metabolic functions associated with hypothermia predispose to hypoxia, impaired fluid balance, hypoglycemia, hyperkalemia, or an accumulation of toxic metabolic by-products that may not be compatible with life.^{5–7,12} A common finding in hypothermic infants, particularly in fatal cases, is massive pulmonary hemorrhage in the absence of infection. Moreover, impaired kidney function and hypoglycemia are common. Nonetheless, autopsy studies consistently fail to indicate any specific findings accounting for neonatal death.

Pulmonary Manifestations

Adverse pulmonary manifestations of hypothermia may be mediated via several mechanisms (Table 1). First, hypothermia induces changes in pulmonary mechanics and surfactant distribution. Experimental studies in excised rabbit lungs demonstrate markedly limited surface adsorption and spread of surfactant, with a resultant reduction in compliance under hypothermic conditions.¹⁴ In an observational clinical study, it was shown that hypothermic infants with respiratory distress syndrome were less likely to exhibit improvement in respiratory status after surfactant replacement therapy.¹⁵ Second, hypothermia also modulates pulmonary vascular changes. Pulmonary venous constriction increases as temperature decreases, resulting in increased pulmonary resistance and pulmonary venous pressure, coupled with a decrease in the left atrial pressure.^{16,17} Finally, the biochemical changes induced by cold stress in depressed infants may compromise these infants' ability to recover from metabolic acidosis caused by intrauterine hypoxia.¹⁸ In a subsequent series of term infants, recovery from birth asphyxia was influenced by cold stress. Normothermic infants were able to achieve

and maintain a relatively normal pH by increasing carbon dioxide elimination in response to a developing metabolic acidosis.¹⁸ Infants depressed for even a brief period at birth were unable to maintain their pH and developed a more pronounced metabolic acidosis when maintained in a cold environment rather than in a warm environment.

STRATEGIES TO MAINTAIN NEONATAL HYPOTHERMIA

Strategies to minimize heat loss upon delivery are listed in Table 2. In the healthy infant, maternal to infant skin contact (skin-to-skin care) is strongly recommended, coupled with creating a warm environment in the OR.^{10,19} A randomized trial compared the incidence of hypothermia in neonates who were placed skin-to-skin on mothers' chests 5 minutes after delivery.²⁰ Infants were dried and heads were covered with a cotton cap; mother-baby dyads were randomly assigned to be covered with a forced-air warming blanket or a cotton blanket. The incidence of neonatal hypothermia (<36.5°C) was significantly greater in the cotton blanket group (5% vs 81%; $P < 0.0001$).

All infants requiring more than basic resuscitation after delivery are placed under a radiant warmer; the following strategies may be implemented singularly or in combination. *Occlusive wrapping*, which reduces evaporative heat loss from the baby but allows external heat to penetrate, was evaluated in 4 studies. Findings indicate that plastic wraps were effective in reducing heat losses in infants <28 weeks of gestation, with a temperature difference of 0.68°C (95% CI, 0.45–0.91), but this salutary effect was not observed in infants between 28 and 31 weeks of gestation.^{19,21} *Exothermic warming mattresses*, when placed under the neonate, provide an external source of heat and help maintain body temperature. Several observational studies indicate that the use of warming mattresses coupled with occlusive wrap and radiant heat, in comparison with wrap and radiant heat alone, reduces the number of preterm infants with moderate hypothermia upon admission to the Neonatal Intensive Care Unit (NICU).^{10,22,23} When an exothermic warming mattress is used in combination with occlusive wrap, the risk of hyperthermia (>37.5°C) is increased although this has been inconsistently observed.^{10,22,23}

Evidence from 1 observational study suggests that *heating and humidifying inspired gases*, presumably by maintaining respiratory tract temperature, in addition to occlusive wrap, reduced the number of preterm infants (25–32 weeks of gestational age) with moderate hypothermia upon admission to the NICU compared with the use of exothermic warming mattress and occlusive wrap or exothermic warming mattress alone.²⁴

Warmed polyethylene caps, by avoiding heat loss from the relatively large surface area of the scalp, have been shown to reduce the number of infants with a NICU admission temperature <36°C.²⁵ The International Liaison Committee on Resuscitation has suggested that the *delivery room temperature* should be maintained at 26°C, and the World Health Organization has suggested that the temperature should be 25°C.^{1,26} However, an even lower temperature (21°C) appears adequate to meet the desired goals, and it is more readily tolerated by providers in the OR.¹⁰ These

Table 1. Potential Mechanisms of Neonatal Pulmonary Injury Due to Hypothermia

Mechanism	Downstream effects	References
Poor adsorption and spread of surfactant	Reduction in lung compliance	14
Increased pulmonary venous resistance	Decreased left atrial pressure	15–16
Inadequate respiratory reserves	Inability to compensate for metabolic acidosis, decreased pH, increased base deficit, and increased ratio of lactate to pyruvate	17

Table 2. Strategies to Prevent Neonatal Hypothermia

Study	Impact	Risks	References
Occlusive wrapping	Reduces heat loss	None identified	19,21
Exothermic warming mattress	Maintains body temperature by providing warmth	Neonatal hyperthermia when combined with occlusive wrap	22,23
Warmed humidified resuscitation gases	Avoid cooling of the airway	None identified	24
Polyethylene caps	Prevents heat loss from the scalp	None identified	25
Raising delivery room temperature	Ideal room temperature is unclear (range 21–25°C)	Neonatal hyperthermia	1,10,26

strategies, either in isolation or combination, have met with varying success. Importantly, none of these studies has demonstrated an improvement in neonatal mortality or long-term morbidity despite minimizing the degree of hypothermia.

We recently implemented a practice plan (use of occlusive wrap, exothermic thermal mattress, and maintaining operating and delivery room temperatures between 21 and 23°C [72–74°F]) applied to all premature infants <35 weeks of gestation.¹⁰ This approach resulted in a significant increase in the delivery room axillary temperature of infants from 36.06 ± 0.65 to $36.61 \pm 0.56^\circ\text{C}$ ($P < 0.0001$) and NICU admitting temperature from 36.02 ± 0.81 to $36.70 \pm 0.56^\circ\text{C}$ ($P < 0.0001$) at baseline and implementation, respectively. The number of infants with a NICU admission axillary temperature $<36^\circ\text{C}$ decreased from 55% to 6% (RR, 0.18; 95% CI, 0.10–0.34; $P < 0.0001$).¹⁰ Importantly, the number of infants with tracheal intubation at 24 hours decreased significantly from 39% to 18% (RR, 0.53; 95% CI, 0.33–0.83; $P = 0.005$). This reduction was most prominent in infants ≤ 28 weeks of gestational age (a decrease from 88% to 53%; $P = 0.005$) but was less evident in larger premature infants (33–34 weeks) (a decrease from 21% to 8%; $P = 0.12$). This improvement in early respiratory status parallels observations noted in a large observational study of premature infants. In this study, a 2-fold increased likelihood of requiring supplemental oxygen at 40 weeks of postconceptual age was observed in infants born at <26 weeks of gestation admitted to the NICU with hypothermia (admission temperature $<35^\circ\text{C}$). This effect was independent of gestational age or body weight.²⁷ Several clinical reports describe an association between early hypothermia and/or low inspired gas temperatures and subsequent abnormal respiratory status in premature infants, including an increased duration of oxygen therapy and/or a significantly higher incidence of pneumothorax and more severe lung disease.^{28,29}

Delayed cord clamping for up to 60 seconds in the infant not requiring resuscitation is rapidly becoming the standard of care, particularly for the premature infants. The available evidence suggests that for vaginal deliveries, the basic steps of drying the baby and placing a cap on the head after delivery and prior to cord clamping are sufficient to ensure that infants are able to maintain temperature in a normal range during this brief transitional stage.³⁰

Therapeutic hypothermia to prevent ongoing injury after intrapartum hypoxia-ischemia when implemented within 6 hours after delivery has been shown to reduce the extent of brain injury after intrapartum hypoxia.³¹ Candidacy for therapeutic hypothermia is rarely evident until an evaluation by the neonatologist is complete. Consequently, the same principles outlined previously should be applied in

the delivery room to infants believed to be at risk for intrapartum hypoxia-ischemia.

ROLE OF MATERNAL TEMPERATURE AT DELIVERY

In utero, fetal temperature is 0.5°C higher on average than maternal temperature. When maternal temperature increases or decreases, fetal temperature follows correspondingly. Although the fetus develops in a warm and thermostable environment, its basal heat production is about double that of adult heat production.³² The placenta dissipates approximately 85% of the fetal heat to the maternal tissues. The fetal-maternal gradient increases further when the placental circulation is impaired or artificially occluded, which may mitigate the impact of maternal hypothermia on neonatal temperature.

Maternal hypothermia is common during cesarean delivery and may impact neonatal temperature. Despite warming with forced air and/or fluids, most women will experience a decrease in temperature of approximately 0.5°C during cesarean delivery over the first 30 minutes (Fig. 1).^{33–38} Numerous case reports describe severe maternal hypothermia after cesarean delivery under spinal or combined spinal-epidural with spinal morphine.^{39–45} The decrease typically observed is approximately 1.0°C per hour and may continue long after delivery. This intrathecal morphine-related hypothermia appears to be reversible with naloxone or lorazepam. Several routine conditions around the time of cesarean delivery contribute to maternal hypothermia (Table 3).^{46–48} Shivering often occurs during cesarean delivery performed under neuraxial block but does not appear to be effective at mitigating hypothermia.³³

Early decreases in maternal temperature during cesarean delivery are modest, and there is no documentation of adverse neonatal outcomes with maternal hypothermia. Nonetheless, maternal hypothermia developing in a cold OR may contribute to neonatal hypothermia, both before delivery and after delivery when skin-to-skin contact is initiated. As temperature monitoring during neuraxial anesthesia is often inadequate, maternal hypothermia frequently goes undetected.⁴⁹ Multiple strategies to warm mothers before and during cesarean delivery have been studied, with mixed results.^{50,51} Warming before initiation of neuraxial anesthesia appears to be the most effective, as shown with both forced-air heating and warmed IV colloid.^{37,38} OR temperature standards may be the most effective strategy to maintain both maternal and neonatal normothermia. No study has yet documented that prevention of maternal hypothermia by itself is associated with improved neonatal outcome.

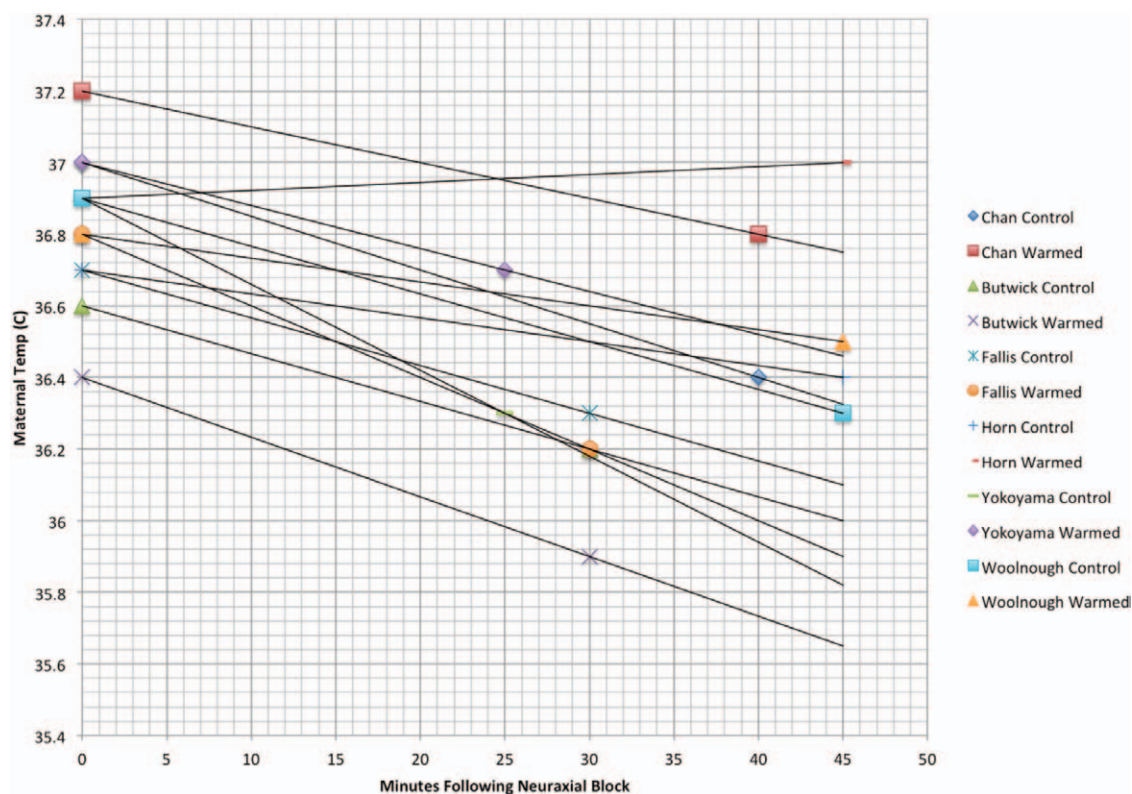


Figure 1. Temperature changes with and without warming after neuraxial block for cesarean delivery.^{33–38}

Table 3. Contributors to Hypothermia During Cesarean Delivery

Contributor	Primary mechanism	References
Neuraxial blockade	Sympathectomy and heat redistribution from core to periphery	46
Intrathecal opioids	Altered hypothalamic temperature regulation	47
Exposure to ambient temperature	Radiation, convection, and conduction	48
Room temperature IV fluids	Direct cooling	48

CONCLUSIONS

In conclusion, the newly born is extremely vulnerable to decreases in body temperature unless active warming strategies are implemented. There is a dose-dependent increase in mortality with decreasing body temperature as well as an increased likelihood for more severe early respiratory distress. Multiple neonate-focused strategies have been shown to decrease the incidence of neonatal hypothermia. However, none of these strategies has been shown to reduce neonatal mortality or morbidity. Because fetal temperature is affected by maternal temperature, further study of the contribution of maternal temperature to neonatal hypothermia is warranted. ■■

DISCLOSURES

Name: Jeffrey Perlman, MBChB.

Contribution: This author helped write the manuscript.

Attestation: Jeffrey Perlman approved the final manuscript.

Name: Klaus Kjaer, MD.

Contribution: This author helped write the manuscript.

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