

# Cold Stress in Emergency Room Leads to Adverse Outcome

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## Fact pattern

A 28-year-old G3P1 patient at 34 weeks gestation developed contractions and was admitted for premature labor with intact membranes. Her Group B streptococcus (GBS) status was unknown at admission but a vaginal/rectal culture collected 7 hours after admission was positive. She received penicillin 3 hours before delivery. She made normal progress in labor and delivered spontaneously 1 hour after membranes were ruptured. The membranes were artificially ruptured 1 hour prior to delivery. A 34-week female infant with a birthweight of 2170 grams was delivered and received Apgars scores of 7 and 8, at 1 and 5 minutes, respectively. The baby was cared for in the Newborn Intensive Care Unit (NICU). The physical exam was unremarkable and was considered appropriate for gestational age (AGA). She received an evaluation for sepsis, including a lumbar puncture and two days of antibiotics. She remained asymptomatic, and all laboratory values were unremarkable, including blood and cerebral spinal fluid cultures. She was discharged after eight days. The placental examination was normal.

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A week after discharge, she visited her pediatrician for a well-baby check and was doing well. However, 12 days after that visit, when she was a month old, she stopped eating, became listless, developed pallor, and had fewer wet diapers and diminished crying compared to previously. She was brought to the Emergency Department (ED), where examination revealed a 2.7 kg child with poor muscle tone, flat to sunken fontanelles, dry mucous membranes, and poor air movement. Her heart rate (HR) was 200 beats per minute (bpm), her respiratory rate (RR) was 56 breaths per minute, and her temperature was **96.5°F**. Her oxygen saturation (SaO<sub>2</sub>) was 95%. A peripheral intravenous venous line was started with a bolus of normal saline. Studies were ordered, including bilirubin, blood culture, and urine analysis,

and about an hour after admission, she was started on ampicillin and cefotaxime. A chest radiograph showed non-specific bilateral perihilar interstitial prominence. The white blood cell count (WBC) revealed a low count of **2.9 x 10<sup>9</sup>/L**, the hematocrit was 46%, and the platelet count was 302 x10<sup>9</sup>/L. The electrolytes and urinalysis were unremarkable. An hour and a half after admission, the baby was transported to the radiology department for computerized axial tomography (CT) of her head. Following removal from the scanner, the child was noted to have profound bradycardia with severe desaturation. Upon her immediate return to the ED, the baby was pale and not breathing, and cardiopulmonary resuscitation was instituted immediately. A review of the events revealed that the nurse accompanying the baby had not assessed either the monitoring strips or the baby during the trip to the radiology department, during the procedure, or even shortly after departure from the Radiology Department. The radiologist called the ED to inform the physician that the CT was negative.

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After the baby's normal HR was restored, non-meningitic doses of ampicillin and cefotaxime were administered. A normal saline bolus was given, and a blood gas was drawn, which showed a pH of 7.13, a PCO<sub>2</sub> of 77mmHg, a pO<sub>2</sub> of 71 mmHg, and a base deficit of 3. The ED physician decided at this point to do a lumbar puncture (LP). After five attempts, the transport team arrived, and attempts to obtain cerebral spinal fluid were abandoned. The baby's HR was 160 bpm, the SaO<sub>2</sub> was 100%, and her temperature was **93.9°F** (33.8°C). At this point, she was apneic and was being manually ventilated by the respiratory therapist. Her blood pressure (BP) was 88/56 with a mean of 67 mmHg. This was her only BP in the ED. Five hours after being admitted to the ED, the baby was transported to a higher-level medical facility. The transport team slowly and appropriately warmed the baby during transport.

At the referral center, the baby continued to be lethargic and had decreased movement. Her temperature was 36.80F, HR 188 bpm, BP 79/50 mmHg, and SaO<sub>2</sub> 100% on arrival. Ampicillin,

cefotaxime, and vancomycin were given at meningitic doses, to which were added Acyclovir, bicarbonate, packed red blood cell transfusion, two normal saline boluses, and Lasix. Lactic acid was moderately elevated and increased as perfusion improved; liver function tests were moderately high. The blood culture drawn at the ED 12 hours earlier was positive for GBS. An LP performed that day when the baby was stable was also positive for GBS. She developed seizures, central diabetes insipidus, and adrenal insufficiency, all ascribed to her GBS meningitis during this hospitalization. The magnetic resonance imaging (MRI) done later that evening was consistent with an acute hypoxic-ischemic injury with abnormal signals in the deep gray matter. Several days later, another MRI and an MR venogram (MRV) showed diffuse pachymeningitis and cerebritis with acute infarcts involving the bilateral occipital and temporoparietal lobes, right thalamus, left lentiform nucleus, bilateral deep white matter, and bilateral frontal lobes as well as extra-axial fluid collections.

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On follow-up evaluations, the child continued to have seizures that were difficult to control. She had global developmental delays, cortical blindness, central hypothyroidism, cerebral palsy, constipation, gastroesophageal reflux, rumination, and sleep distance.

#### **Opinions and allegations**

1. The baby was in critical condition upon entering the ED. The history of lethargy, poor feeding, poor output, and her abnormal physical findings of low temperature (96.5), tachycardia, dry mucous membranes, and flat to sunken fontanels required the physicians and nurses to evaluate and stabilize the baby immediately.
2. The history, physical examination, and leukopenia all pointed to infection. Sending the baby to CT was not only detrimental but was unlikely to have diagnostic or therapeutic implications. Inexplicably, she was not monitored during this time and became profoundly hypothermic and hypoxemic and suffered a neurological injury.
3. Thus, it was below a reasonable standard of care

- i. To fail to place the baby immediately under a radiant warmer,
- ii. To fail to insert two peripheral intravenous lines (one for antibiotics and one for fluid support),
- iii. To fail to maintain close vigilance of vital signs, including BP, temperature, HR, and SaO<sub>2</sub>,
- iv. To fail to obtain additional laboratory examinations, including polymerase chain reaction (PCR) for herpes and blood gas,
- v. To fail to immediately (within 30 minutes) provide doses of antibiotics sufficient to cover potential meningitis as well as Acyclovir,
- vi. Fail to make immediate plans for transport to a higher center of care even while the evaluation was performed,
- vii. Further, the LP should have been deferred until the baby was stable (and abandoned before five failed attempts!). Similarly, the baby should not have been sent for CT scan. The management of this baby at the ED was reckless.

The defense's position was that the baby had GBS meningitis upon entry to the ED and that the standard of care was met.

#### **Causation**

Babies born at 34 weeks gestation are more vulnerable to infection than term babies. Preterm babies are deprived of normal amounts of maternal immunoglobulins transferred from the mother through the placenta to the fetus during the last trimester.

The respiratory arrest was secondary to iatrogenic hypothermia. The baby entered with a temperature of 96.5°F because of sepsis. There was no effort to maintain normothermia during the 5 hours in the ED. She decompensated because of the hypothermic stress, which led to a cardiopulmonary arrest leading to hypoxic-ischemic encephalopathy, while her GBS sepsis (inadequately treated) advanced to meningitis. The antibiotics administered could not adequately combat the GBS since her circulation was profoundly compromised.

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The case was settled against the ED.

## Discussion

Thermal management of the newborn infant is a cornerstone of neonatal care. Globally, hypothermia contributes to neonatal mortality and morbidity, and the potential impact of optimal thermal care provision on infant health is potentially considerable. Physicians caring for newborn infants have appreciated this important concept for over a century, and neonatologists have made considerable strides in fine-tuning thermal control in the delivery rooms and Newborn Intensive Care Units. However, the infant's thermal care in Emergency Rooms may not have been managed appropriately. In general, adequate thermal management depends at least as much on sound knowledge as on the use of high-tech devices such as incubators, radiant warmers, heat lamps, and heated mattresses, administration of warmed fluids, and the need to closely monitor an infant's temperature. In the ED, assessment, and procedures often require the exposure of a large portion of the infant's body surface, increasing the difficulty of maintaining the infant's temperature.

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Premature infants are at greater risk for hypothermia than older children and adults for several reasons: greater ratio of skin surface to body weight, less subcutaneous and brown fat, decreased ability to increase heat production through shivering, and limited glycogen stores to support heat production. An infant with a coexisting vulnerability is particularly susceptible to cold stress.

The above case described a baby with GBS sepsis whose temperature was 96.5°F upon entering the ED, but because the staff did not appropriately manage her thermal needs, the temperature dropped to 93°F. She developed apnea and bradycardia and required full resuscitation. The antibiotics eventually given to her could not adequately enter his circulation because of profound perfusion issues.

The neutral thermal environment (NTE) has been defined as maintaining the infant's temperature with a stable metabolic state and minimal oxygen and energy expenditure. It is not a fixed temperature but instead varies with gestational age, birthweight,

and the age of the newborn. Effective thermoregulation requires adequate energy stores (primarily glucose), insulation (fat deposits), hypothalamic function, and muscle tone. When environmental temperatures fall below the NTE, metabolic demands increase, increasing oxygen consumption as the infant attempts to restore NTE. If this deterioration continues, compensatory mechanisms are exhausted, and eventually, the infant's temperature decreases further. Increased glucose utilization results in exhaustion of glycogen stores, decompensation of the cardiac and respiratory systems, and death. In order to ensure the NTE is maintained in infants who are unable to achieve this with their physiological measures, it is essential to provide environmental thermostability by blocking avenues of heat loss and applying adequate warmth and adequate temperature monitoring.

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### Suggested reading:

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