Briefly Legal: An Avoidable Fetal/Neonatal Death

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A 24-year-old G2P1 patient presented for induction of labor at 39 1/7 weeks gestation. She had an uneventful course with a normal result to a glucose challenge at 28 weeks gestation. At 30 weeks gestation, she was hospitalized for ulcerative colitis and treated with steroids. She had a negative Group B streptococcus culture at 36 weeks gestation. Three days prior to admission, a non-stress test NST was interpreted as reactive (normal). On the biophysical profile, the fetus was active, but the amniotic fluid volume was moderately increased (polyhydramnios). An anatomical evaluation of the fetus revealed no apparent abnormalities. The mother reported normal fetal movements up to the time of admission.

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The patient had normal vital signs on admission, and the FHR pattern was reassuring (Category I). The induction was initiated with Intravenous Pitocin, and an epidural was provided early in labor for pain relief. With the onset of pushing in the 2nd stage of labor, the initially normal FHR tracing deteriorated with the appearance of several deep decelerations associated with frequent contractions and maternal pushing. Membranes were ruptured at this time with the egress of abundant, meconium-stained fluid. The 2nd stage lasted just over one hour. Relentless pushing coincided with frequent contractions and a deteriorating fetal heart rate pattern, including severe, prolonged variable decelerations with overshoot, rising baseline heart rate, and absent variability. Just prior to delivery, again in association with frequent contractions and relentless pushing, there was a prolonged deceleration lasting over 10 minutes with the nadir of 60 bpm. The monitor was disconnected several minutes prior to the spontaneous vaginal delivery.

At delivery, the 3351- gram female infant was lifeless – with a 1-minute Apgar score of 0. The arterial cord blood gas had a pH of 7.1, pCO_2 69 mmHg, pO_2 24.7 mmHg, and a base deficit (BD) of 9.8. The venous cord blood gas analysis revealed a pH of 7.34, pCO_2 37 mmHg, pO_2 50 mmHg, and a base deficit of 5. (Note the wide difference between the arterial and venous values.) The infant was brought to the radiant warmer, where she was dried, stimulated, and given continuous positive airway pressure (CPAP) by the labor and delivery nurse. No member of the neonatal resuscitation team was present at the delivery. After 1-2 minutes, the nurse began positive pressure ventilation (PPV). At 2 1⁄2 minutes, the HR was noted to be 63 bpm, the inspired oxygen level was increased from 30% to 100%, and the resuscitation team was finally paged. The oxygen saturation (SaO_2) levels were recorded from the pulse oximeter were noted to be 100% after the inspired oxygen was increased, but then shortly dropped to 88%. At 5 minutes after the birth, the resuscitation team arrived. At 6 minutes, the HR was 153 bpm. At 7 minutes, the HR was 80, and the O_2 saturation was 68%. At 7 minutes, an intubation attempt was made, unsuccessfully. At 11 minutes, the infant was finally intubated. At 14 minutes, the HR was 71 bpm, and the SaO_2 was 72%. HRs were determined by auscultation and pulse oximeter (documentation by nurses in the chart). A nasogastric tube was placed at 15 minutes.

At 18 minutes, the neonatologist began chest compressions, having determined that the pulse oximeter was not reliably determining the HR. He observed that the only time a HR was found was when a nurse was holding it in place. Several doses of epinephrine were given through the endotracheal tube (ETT) every 3-5 minutes. The HR recorded from 18 to 25 minutes ranged from 78-151 bpm, and the O_2 saturation ranged between 50 and 89%. At 25 minutes, an umbilical venous catheter was placed and could be flushed, but no blood could be drawn back. Resuscitation was stopped at 30 minutes, at which point the baby was pronounced dead.

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The following day the neonatologist annotated the chart to "provide clarification" of the events surrounding the resuscitation of the newborn. He averred that no HR or pulses were noted by any physician on examination at any point after delivery. Further, he stated that the wording in the chart "minutes of life" should have actually stated "minutes after birth," because the baby was not alive at birth; she never had a HR, respiratory effort, movement, or tone, and the death should be considered a fetal demise, not a neonatal death. The Apgar scores were changed and re-assigned after death as 0 at 1, 5, 10, 15, 20, and 25 minutes after birth.

On postmortem examination, the baby was without dysmorphology and appropriately grown with a birthweight of 3351-grams, the length was 50.8 cm, and the head circumference was 32 cm. Meconium was found in some of the alveolar spaces suggesting aspiration. The placental examination revealed a 459-gram placenta and 45 cm umbilical cord.

Plaintiff Allegations:

The resuscitation was below the standard of care

Despite the presence of meconium in the amniotic fluid and the profound deceleration, the resuscitation team was not present before delivery.

The resuscitation team did not arrive until 5 minutes after birth

The resuscitation process was egregiously below the standard of care; it failed to comply with Neonatal Resuscitation Guidelines.

After the fact assessment, the neonatologist's late entry was self-serving to avoid accountability for a mismanaged neonatal resuscitation.

Defense Allegations:

The baby was stillborn, and nothing could be done to save her, even if the resuscitation process had followed the NRP.

After many months of discovery, the case was dropped.

"In the 2016 American Academy of Pediatrics and American Heart Association Neonatal Resuscitation (NRP) Guidelines (7th edition) stated that if one cannot determine the HR by physical examination and the baby is not vigorous, it is necessary to connect a pulse oximetry sensor or electronic cardiac (ECG) monitor lead and evaluate the HR."

Discussion:

In the 2016 American Academy of Pediatrics and American Heart Association Neonatal Resuscitation (NRP) Guidelines (7th edition) stated that if one cannot determine the HR by physical examination and the baby is not vigorous, it is necessary to connect a pulse oximetry sensor or electronic cardiac (ECG) monitor lead and evaluate the HR. The Guidelines caution that pulse oximetry may not function if the baby's HR is low or if the baby has poor perfusion. In these situations, monitoring the HR with ECG becomes the preferred method. In unusual circumstances, an ECG monitor may show an electrical signal, although the heart is not pumping, a condition referred to as pulseless electrical activity (PEA). This condition should be treated the same as an absent HR (asystole). The 8th edition of the NRP (2021) continues to make this same recommendation for using a pulse oximetry sensor or ECG to assess HR and adds another option to include a handheld Doppler ultrasound or digital stethoscope. A combination of these modalities- auscultation, palpation, pulse oximetry, ECG, and perhaps the newer modalities will give the best assessment when a patient presents as described. In the above case, it seems likely that the baby would have responded favorably had timely and effective PPV been instituted. Even more probably, had proper and timely intrauterine resuscitation been forthcoming, the problems of neonatal resuscitation would have been avoided.

Pertinently, the obstetrical care provided by the physicians and the nurses, the real culprits, were not sued. It is important to consider that on admission; the fetus was demonstrably normal with a reassuring FHR pattern, normal growth, somewhat increased amniotic fluid volume, and normal anatomy (confirmed by ultrasound and subsequently at autopsy). With the onset of the 2nd stage of labor, the FHR tracing deteriorated, predictably, in relationship to the frequent contractions, compulsive pushing (pushing not modified as a response to the deteriorating FHR pattern) to the point of a profound hypoxic-ischemic event. The cause of the decelerations was likely umbilical cord compression given the modest BD and the wide differences in the umbilical artery and the vein values. (Pomerance)[1] These severe changes in the FHR, co-associated with maternal pushing, demanded reduction of the Pitocin, perhaps even tocolysis, and restraining the pushing with contractions. With reasonable medical probability, such maneuvers, applied in a timely fashion, would have restored fetal homeostasis and permitted vaginal delivery of a non-asphyxiated, healthy newborn.

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Suggested Reading

1. *American Academy of Pediatrics. Textbook of Neonatal Resuscitation, 8th ed, Weiner*
2. *GM (Ed), American Academy of Pediatrics, 2021.*
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