Novel Insights from Clinical Practice

Neonatology

Neonatology 2019;115:95–98
DOI: 10.1159/000493357

Received: April 17, 2018
Accepted after revision: August 28, 2018
Published online: October 23, 2018

# Pulseless Electrical Activity Complicating Neonatal Resuscitation

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**Established Facts**

- Detection of pulseless electrical activity (PEA) has been cited as a potential complication of electrocardiography use in the delivery room, but its occurrence in this setting has not been well described in the literature.

**Novel Insights**

- PEA may be observed during resuscitation of critically ill newborns, as described in this report. Incorporation of discrepant physical examination findings and nonpulsatile pulse oximetry waveforms with electronic heart rate monitoring may improve providers' ability to rapidly recognize PEA.

**Keywords**
Neonatology · Resuscitation · Delivery room · Neonatal Resuscitation Program · Chest compressions

**Abstract**
***Background:*** The most recent guidelines by the Neonatal Resuscitation Program recommend use of electrocardiography monitoring during advanced resuscitation. ***Objective:*** We describe a case in whom detection of pulseless electrical activity (PEA) on electronic heart rate monitoring complicated delivery room management of an extremely low birth weight infant and offer suggestions for the identification of PEA for neonatal providers. ***Conclusion:*** Further prospective studies are needed to determine the true incidence of PEA in the delivery room setting as well as its prognosis in newborns.



## Introduction

Integration of electrocardiography (ECG) into delivery room management is a recent development in neonatal resuscitation. Clinical studies have demonstrated that ECG is the most accurate assessment of newborn heart rate compared to palpation, auscultation, and pulse oximetry [1]. Of note, these studies were largely conducted in healthy term infants, and very few infants required advanced resuscitation, such as chest compressions [2, 3]. Despite clear advantages of ECG, limitations and challenges associated with this technology are now emerging during the resuscitation of critically ill neonates. A recent case report described a hydropic infant whose extensive chest wall edema prevented detection of a heart rate by ECG [4].



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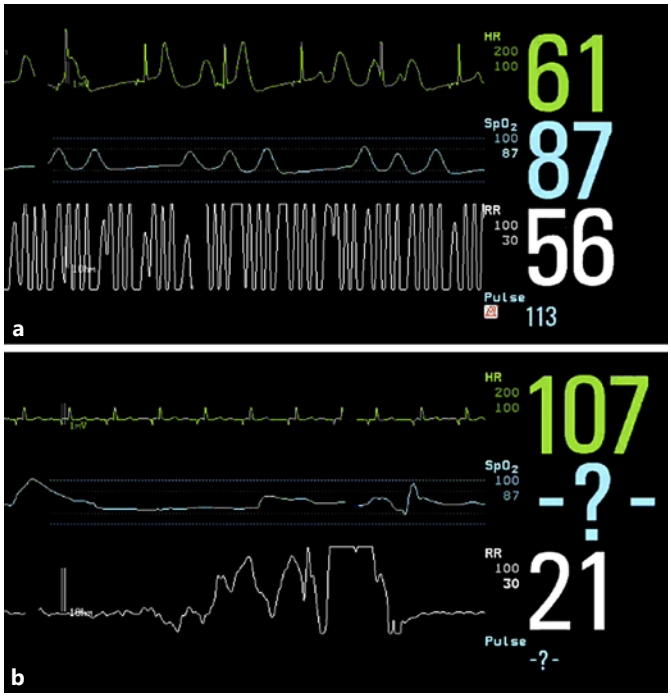


**Fig. 1.** Monitor output during resuscitation. **a** ECG monitoring showing a pulsatile waveform on pulse oximetry during chest compressions, confirming appropriate pulse oximeter placement. **b** ECG monitoring during PEA. There is electrical activity resembling normal sinus rhythm without a correlating waveform on pulse oximetry. No heart tones or pulses were detected on the infant's physical examination.

A frequently cited limitation of ECG use to guide delivery room resuscitation is the potential detection of pulseless electrical activity (PEA) that could be mistaken for a heart rate with associated cardiac output. However, to our knowledge no report of PEA in the delivery room setting currently exists in the literature. Here, we describe the resuscitation of a severely compromised, preterm neonate found to demonstrate PEA on ECG monitoring and share clinical pearls to facilitate appropriate diagnosis and direct management.

## Case Report

An extremely premature, extremely low birth weight infant was born precipitously following spontaneous preterm labor. Delivery was complicated by breech presentation and 10 min of head entrapment. On initial assessment, the infant was limp, apneic, and extensively bruised across the entire body. ECG and pulse oximetry sensors were placed immediately; concurrent auscultation of the chest revealed bradycardia, with a heart rate <60 bpm on initial presentation. The resuscitation team provided positive pressure ventilation by bag mask and proceeded to endotracheal intubation. An endotracheal tube was successfully placed on the second attempt with confirmation by colorimetric $CO_2$ detection, visualization of chest rise, and auscultation of bilateral breath sounds. After securing the breathing tube and providing positive pressure ventilation, heart rate remained <60 bpm and chest compressions were initiated. During compressions, there was a pulsatile waveform on the pulse oximeter monitor output (Fig. 1a). An emergent umbilical venous catheter was placed and epinephrine was administered.

One minute following epinephrine administration with ongoing chest compressions, the heart rate detected by ECG was >60 bpm and compressions were stopped. While the heart rate on ECG continued to rise (Fig. 1b), the infant showed no improvement in color or tone, made no spontaneous respirations or movements, and had no detectable pulse oximetry waveform in the absence of chest compressions. On physical examination, there were no heart tones on auscultation and no palpable pulses. At this point, the team leader expressed concern that the infant was demonstrating PEA and directed the team to resume chest compressions. Subsequent assessments by multiple providers during ongoing resuscitation confirmed the suspected diagnosis. The infant continued to show no improvement; physical examination did not suggest signs of endotracheal tube displacement, pneumothorax, or other correctable process contributing to the infant's arrest. Venous blood gas analysis revealed severe acidosis with a pH of 6.73, a carbon dioxide of 95 mm Hg, a bicarbonate of 8.8 mmol/L, and a base excess of –22.8 mmol/L, as well as a potassium of 8.6 mmol/L and a lactate of 13.8 mmol/L. Given the infant's degree of perinatal insult, extreme prematurity, and persistent PEA after 10 min of resuscitation, resuscitation interventions were discontinued.

## Discussion

PEA is a form of cardiac arrest characterized by organized electrical activity detected by ECG without meaningful ventricular contraction or cardiac output. PEA is commonly encountered in pediatric resuscitation, where it is classified along with asystole as a nonshockable rhythm [5]. In contrast, PEA is believed to be an unusual occurrence in neonatal resuscitation, though diagnosis relies on use of ECG and a precise incidence has never been reported in this population. Recent animal data by Patel et al. [6] identified PEA in nearly half of neonatal piglets following prolonged asphyxia. This finding suggests that PEA may be relatively frequent in asphyxiated newborns, such as the infant described in our case, but has been undetected prior to widespread adoption of ECG in the delivery room setting.

PEA occurs in the setting of severe respiratory failure, extreme metabolic derangements, or mechanical obstruction that uncouple the heart's electrical signaling and the functional contraction of cardiac myofilaments. The Hs and Ts mnemonic (Table 1) provides a cognitive aid to remember the diverse potential etiologies underly-

**Table 1.** Hs and Ts mnemonic

| Hs | Ts |
|---|---|
| Hypoglycemia | Toxins |
| Hypovolemia | Tamponade (cardiac) |
| Hypoxia | Tension pneumothorax |
| Hydrogen ions (acidosis) | Thrombosis (myocardial infarction) |
| Hyper-/hypokalemia | Thrombosis (pulmonary embolism) |
| Hypothermia | Trauma |

ing PEA [5]. Detected electrical activity may resemble normal sinus rhythm or may show a variety of abnormalities, including widening or narrowing of the QRS complex, T-wave abnormalities, or AV dissociation. The prognosis relative to other forms of cardiac arrest in neonates is uncertain; studies in the pediatric population report decreased survival following code events where PEA is the initial presenting rhythm, though this finding has not been consistent across the literature [7].

The latest Neonatal Resuscitation Program algorithm, published in 2016 [8], includes ECG monitoring during advanced resuscitation, such as intubation and chest compressions (class IIB recommendation). Other guidelines, such as the 2015 International Liaison Committee on Resuscitation consensus statement, have incorporated similar recommendations for ECG monitoring during neonatal resuscitation [9]. However, in the setting of PEA, reliance on ECG can mislead providers, particularly if ECG output is not confirmed by concurrent auscultation and assessment of pulses on examination. Heart rate assessment by Doppler ultrasound has been proposed as an alternative to ECG that would not be susceptible to detection of PEA, but this method has not been as robustly studied and is not widely available [1].

With rising use of ECG to guide delivery room resuscitation, familiarity with diagnosis and management of PEA is increasingly important for neonatal providers. Reliance on ECG monitor output for decision-making in the delivery room has the potential to introduce cognitive bias, especially if providers do not routinely confirm ECG output with physical examination findings. Pulse oximetry may not be reliable in the initial minutes of resuscitation due to peripheral vasoconstriction and difficulty acquiring a high-quality signal. Attributing lack of pulsatile activity on pulse oximetry to these limitations (rather than a pulseless state) during ongoing resuscitation is another potential source of cognitive bias. In our case, the fact that a consistent pulsatile waveform was seen on the pulse oximeter during chest compressions suggested that the pulse oximeter sensor was adequately positioned and the monitor was capable of detecting pulsatile blood flow in the extremities.

The study by Patel et al. [6] and our clinical experience described in this report suggest that a high index of suspicion may be warranted during resuscitation of infants following severe asphyxia or other extensive perinatal insults. Integrating physical examination findings and the pulse oximeter output, specifically the absence of a pulsatile waveform, may help clinicians quickly identify PEA. Given that PEA is an emerging diagnosis in the delivery room setting, effective team leaders should clearly communicate when PEA is suspected and guide the team as to appropriate next steps. As in asystole, management of PEA consists of providing high-quality chest compressions and epinephrine administration after establishing adequate ventilation [8]. Additionally, clinicians should consider evaluating for and treating additional underlying etiologies of PEA using the Hs and Ts mnemonic as indicated by the clinical scenario.

The infant in our case had multiple findings that may have contributed to the development of PEA, primarily hypoxia following prolonged perinatal asphyxia, which likely precipitated severe acidosis, hyperkalemia, and hypovolemia. Due to the infant's periviable gestational age and extensive preceding injury without response to resuscitative efforts, an exhaustive search for other causes was deferred by the resuscitating team; the infant's parents declined autopsy.

## Disclosure Statement

The authors have no competing interests to declare.

## Author Contributions

All of the authors contributed to the preparation of manuscript. J.R. James led the team for the resuscitation. L. Sillers and S.C. Handley participated in the resuscitation.

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