Neonatal Heart Rate in the Delivery Room

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At birth, a newborn's heart rate should be evaluated immediately to determine the need for resuscitation and stabilizing measures. As cardiopulmonary transition is affected by the cord management strategy, gestational age, and underlying condition, different heart rate intervention thresholds might be needed in different subgroups of newborn infants.

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1. Introduction

Heart rate (HR) is considered the gold standard clinical indicator of successful transition from intra- to extrauterine life and is used to guide delivery room resuscitation and stabilization [1]. International guidelines recommend an HR threshold of 100 beats per minute (bpm), below which, intervention should be considered. Current guidelines recommend neonatal HR assessment by cardiac auscultation, pulse oximetry, and/or electrocardiogram (ECG) [1]. However, studies indicate that some methods, including auscultation and palpation [2][3][4], may be inaccurate. This is important, as HR overestimation might result in interventions being delayed, while underestimation might result in unwarranted interventions [2]. Also, with contemporary methods for fast and reliable measurement, the HR threshold for intervention in different subgroups of infants, e.g., by the cord management method [5][6], delivery mode [7], and gestational age [7], has been challenged. As HR is dependent on blood oxygenation, changes in practice with regard to the use of supplementary oxygen in preterm and term infants may have affected the expected course of and prognostic value of HR in the first minutes after birth. A pulse oximeter continuously measures oxygen saturation and HR; the signal is delayed compared to dry-electrode ECG/newer generation ECG devices.

2. Factors, Including Measurement Method, That Influence HR in Newborn Infants Immediately after Birth

2.1. First Reports of Normal HR Immediately after Birth—Pulse Oximetry

During neonatal resuscitation, the use of pulse oximetry to obtain preductal oxygen saturation and HR is recommended [1]. In the 1980s, several studies published oxygen saturation data in the first few minutes after birth [8]. Dawson et al. reported HR [9] in addition to oxygen saturation [7] percentiles in the first 10 min of life in healthy

term and preterm infants. These studies demonstrated that HR starts out low and increases rapidly in the first minutes of life. There is a wide normal range and a slower HR increase in preterm vs. term infants, after cesarean vs. vaginal birth, and after maternal analgesia administration [9]. According to these data, the median (interquartile range, IQR) HR for term infants at one minute of age is 99 (66–132) bpm versus 96 (72–122) bpm in preterm infants. A plateau of 160 (range 130–180) bpm was, in most cases, reached before 10 min of age in healthy term babies. At one minute of age, 61% of healthy term infants had an HR < 100 bpm, decreasing to 21% and 7% at 2 and 3 min of age, respectively. However, still, at 10 min of age, 1% of healthy term infants had an HR < 100 bpm [7]. Singh et al. [10] and Kamlin et al. [11] investigated the accuracy of the pulse oximetry HR measurement in preterm infants (n = 30) in the Neonatal Intensive Care Unit (NICU) and late preterm and term infants (n = 55) in the delivery room, respectively. Both Singh et al. [10] and Kamlin et al. [11] found lower HRs with pulse oximetry compared to ECGs. On the other hand, pulse oximetry detected an HR < 100 bpm only 89% of the time [11].

2.2. Recent Reports of Normal HR Immediately after Birth—Electrocardiogram

With traditional ECGs, self-adhesive electrodes on clean and dry skin are attached to a monitor that measures and displays the heart's electrical activity. Two papers reported the time to a reliable HR display, comparing pulse oximetry with ECGs during the resuscitation of preterm (n = 46) and term (n = 20) infants [12][13]. Both studies concluded that the time to obtain an ECG-derived HR was half that of pulse oximetry (median time 28 s (ECG) vs. 62 s (pulse oximetry) [12]). Similarly, van Vonderen et al. [14] showed that the mean (standard deviation, SD) time from birth until an HR was detected using an ECG and pulse oximetry was 82 (26) s and 99 (33) s, respectively, in 53 vaginally- and caesarian section-delivered preterm and term infants. Bradycardia was diagnosed twice as often in the first two minutes of life via pulse oximetry vs. ECGs with a mean difference in HR between pulse oximetry and ECGs of −3 bpm in the first 10 min of life.

Kukka et al. [15] used a dry-electrode ECG, a technology not contingent on skin cleaning and drying prior to application, and made HR percentile charts based on 1155 crying and 54 non-crying but breathing infants ≥ 33 weeks' gestation. Similarly, Bjorland et al. [16] used the same technology to describe HR in the first 5 min of life in 898 vaginally delivered term infants. Both studies demonstrated that HR could be detected earlier (at 10 s and 5 s of life, respectively) than with traditional ECGs. The HR was consistently higher than the pulse oximetry-derived references by Dawson et al. [9].

Thus, studies indicate that an ECG provides an HR faster than pulse oximetry. However, potential limitations of ECGs include that pulseless electric activity may be misinterpreted as an HR and thus might delay resuscitative interventions [12][17][18]. The fact that recent studies using dry-electrode ECGs show an HR difference compared to pulse oximetry by far exceeding the 3 bpm found in studies with paired measurements [14] should be further explored.

2.3. Delayed Cord Clamping

While traditionally, HR has been seen as a pure marker of fetal and neonatal hypoxia, there is an increasing awareness that other factors, including cardiac preload, may play an important role [19]. When the umbilical cord is clamped, the infant is separated from the low-resistance placental circulation, systemic vascular resistance and blood pressure increase, and right-to-left ductal shunting decreases [20]. Lung aeration after birth results in reduced pulmonary vascular resistance and increased pulmonary blood flow. Thus, lung aeration prior to umbilical cord clamping ensures that the left ventricular preload and output are maintained when the umbilical cord is clamped [21].

Smit et al. [5] reported on the pulse oximetry-derived HR during 109 midwife-attended home deliveries and concluded that delayed cord clamping and/or skin-to-skin contact were associated with lower HRs and a slower HR increase compared to the reference ranges by Dawson et al. [9]. Pichler et al. [6] confirmed that the pulse oximetry-derived HR was lower in caesarian section-delivered preterm infants with delayed (for 30 s or 60 s) cord clamping vs. immediate cord clamping. In a study from Nepal by Kc et al. [22], the HR was 9 and 3 beats lower at 1 and 5 min, respectively, in late preterm and term infants after delayed vs. immediate cord clamping. Physiologically based cord clamping, i.e., cord clamping after established lung aeration, did not result in less bradycardia in infants ≥32 weeks needing resuscitation compared to early cord clamping [23].

2.4. The Trigemino-Cardiac Reflex

PPV with a facemask may cause a powerful vagal stimulus and result in reflex bradycardia [24]. In delivery room resuscitation, placing a facemask over the mouth and nose may activate the trigeminal nerve and stimulate the trigemino-cardiac reflex, which is characterized by blood pressure changes, bradycardia, and apnea [25]. In support of this, a cohort study showed that 54% of preterm infants had less spontaneous breathing after facemask placement [25]. In infants >34 weeks' gestation, Gaertner et al. [26] reported in a subgroup analysis of a previously conducted clinical trial that 10% of delivery room facemask applications were followed by apnea and bradycardia, suggesting that the trigemino-cardiac reflex is less pronounced in late preterm and term infants.

2.5. Heart Rate in Hypoxemia and Asphyxia

In asphyxiated infants, HR, in addition to oxygen saturation, Apgar scores, and time to first breath, has been shown to be different in infants with a poor prognosis [27], and an HR < 100 bpm is a major criterium for starting PPV immediately after birth. However, according to the Dawson references [9], it is quite clear that not all newborn infants with an HR < 100 bpm have suffered birth asphyxia and are in need of PPV. In addition to HR itself, it is therefore important to make a clinical assessment of the infant and assess the ventilatory drive and how vigorous it is before PPV is initiated.

In the classic resuscitation/oxygen studies in the 1990s and early 2000s, HR (auscultation) was in general <100 bpm in newly born infants given PPV. The Resair 2 study [28] included 609 term or near-term newly born infants in need of PPV and pseudorandomized to receive 100% or 21% oxygen. The major inclusion criterion was an HR < 80 bpm. At one minute of age, 2/3 had an HR < 100 bpm, with a median of 90 (95% CI 40–140) bpm. From 3 min

and onward, the HR was stable above 130 bpm [27]. Ramji et al. [29] found a mean (SD) HR of 94 (26) and 87 (28) bpm at one minute of age in infants in need of PPV given air and 100% oxygen, respectively [30]. In a study from Uganda, Pejovic et al. [31] found a mean HR of 82 at 30 s of life and 100 at 60 s of life for newly born infants needing bag and mask ventilation immediately after birth. Sixty-three percent had an HR < 100 bpm at 1 min of age [31].

In a study of 98 infants ≥30 weeks' gestation with inadequate respiration and therefore needing PPV at birth, Kibsgaard et al. [32] found that in ventilated infants, the median (IQR) first measured HR was 112 (80, 146) bpm recorded 30 (15, 52) s after birth by a dry-electrode ECG. The same group of investigators found an HR > 100 bpm in about 60% of the infants receiving PPV [33]. They concluded that an HR ≥ 100 bpm does not necessarily imply that the newborn is not in need of resuscitation.

Several studies have demonstrated no significant difference regarding the development of HR in the first minutes after birth both in term and preterm infants as related to initial FiO_2 [28][34]. The interaction between HR and hypoxemia and its effect on tissue oxygen delivery and oxygenation immediately after birth has, however, been insufficiently studied. A more thorough understanding of such a relationship might explain the interaction between hypoxemia and bradycardia in the course of perinatal transition. In preterm infants, Bresesti et al. [35] showed that the degree of bradycardia impacted oxygen saturation. Prolonged bradycardia (≥2 min) combined with an oxygen saturation <80% at 5 min of age was associated with a lower oxygen saturation, higher cerebral fractional tissue oxygen extraction, and higher FiO_2 in the first minutes of life. I.e., only in the case of bradycardia and hypoxemia did cerebral oxygenation drop. Infants with prolonged bradycardia had significantly lower oxygen saturation until 5 min compared to infants with no bradycardia and until 4 min compared to infants with brief bradycardia. After 5 min, there were no differences between the groups [35].

Badurdeen et al. [39] investigated the trigemino-cardiac reflex in late preterm and term infants and found that in initially depressed infants, the application of a facemask resulted in an increased not a decreased HR. These findings prompted the authors to speculate that the trigemino-cardiac reflex is suppressed in asphyxiated infants, i.e., those with a low or unstable baseline HR. In contrast, the infants that experienced an HR decrease upon facemask application had a higher baseline HR.

3. Delivery Room HR as a Prognostic Indicator in Different Subgroups of Newborns

3.1. The Golden Minute

McCarthy et al. [37] found a median (IQR) time to auscultation of the heart of 62 (40–79) s and that the first HR was available after 70 (57–89) s, whereas van Vonderen et al. [14] found that HR was detected at a mean (SD) time of 82 (26) s (ECG) and 99 (33) s (pulse oximetry) after birth. Thus, the principle of the golden minute, implying that HR assessment should be made within 60 s of birth, may not always be feasible with traditional methods of HR assessment. According to guidelines [38], HR assessment should be performed without delay so that bradycardia

can be diagnosed, and respiratory support be initiated within 60 s after birth. However, even with the increasing use of ECGs, which have been shown to provide an HR faster than pulse oximetry [32], studies indicate that respiratory support is being initiated later. Badurdeen et al. [39] showed that in late preterm and term infants with an anticipated need for resuscitation, respiratory support was initiated at a median (IQR) age of 63 (41–112) s. As there was a wide range of HRs when respiratory support was initiated, the authors speculated that insufficient respiratory efforts, rather than bradycardia, were common reasons for commencing positive pressure ventilation (PPV), which is in agreement with Kibsgaard et al. [32].

3.2. Prognostic Value and Significance of Delivery Room HR Assessment

Yam et al. [40] reported the HR rise during mask ventilation in infants with GA < 30 weeks (n = 27). They found a median time of 73 s and 243 s for the HR to reach >100 bpm and >120 bpm, respectively. Similarly, Palme-Kilander and Tunell [41] documented a rapid HR increase after the establishment of the gas exchange. Saugstad et al. [27] reported that a five-minute HR ≤ 60 bpm had an odds ratio (OR) of 16.5 (3.1–86.6) for dying, and those who had resuscitation failure defined as an HR < 100 bpm and/or cyanosis at 90 s of life had a 30% risk of dying the first week of life compared to 7.7% in those without resuscitation failure. Further, survivors of perinatal asphyxia had a significantly higher HR during the first 30 min of life compared to those who did not survive [27]. The initial HR in resuscitated infants >1000 g may therefore be a determinant of early neonatal death and moderate-to-severe brain damage in survivors [27]. Kapadia et al. [42] could demonstrate that in preterm infants <32 weeks GA, mortality increased in a linear fashion with the duration of bradycardia during the first 5 min of life. These authors [42] performed an individual patient data meta-analysis of eight studies that used pulse oximetry in preterm infants with GA < 32 weeks. Newborns with prolonged bradycardia (HR < 100 bpm for ≥2 min) had higher odds of hospital death and also when adjusting for potential confounders. Neonates with an additional oxygen saturation <80% at 5 min of life had higher odds of hospital death: OR 18.6 (4.3–79.7) [42]. Infants who were bradycardic in the delivery room were more premature and had lower birth weights compared to infants without bradycardia. There was no association between initial FiO_2 and bradycardia. More infants with delivery room bradycardia had an oxygen saturation <80% at 5 min after birth and low 1 min and 5 min Apgar scores. Also, as the bradycardia duration increased, the incidence of intraventricular hemorrhage, bronchopulmonary dysplasia, and in-hospital mortality increased. In another study, infants who did not reach an HR of 100 bpm by 5 min of life were at an increased risk of death (OR 4.57, 95% CI 1.62–13.98, $p < 0.05$) [36].

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