Arrhythmias and Cardiac Bedside Monitoring in the Neonatal Intensive Care Unit

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KEYWORDS

• Cardiac monitoring • Neonatal intensive care unit • Electrocardiography • Infants • Neonates

KEY POINTS

- It is essential to comprehend the cardiovascular changes that occur in an infant after birth and to understand important information provided on a neonate's bedside monitor.
- Caregivers must understand the physiology surrounding arrhythmias and the importance of reporting a variance from baseline.
- A specialized bedside monitor can offer early warning signs of late-onset sepsis using an algorithm and the subtle changes in heart rate and rhythm.

INTRODUCTION

In 1963, Patrick Bouvier Kennedy, son of President John F. Kennedy, died as a result of complications from being born 5 weeks early.[1] Today, 50 years later, he would have a greater than 95% chance of survival. Neonatal care impacts our country greatly because one in every 10 infants is born preterm in the United States.[2] Despite this remarkable improvement in patient outcomes, in 2010, the infant mortality rates in the United States remain 3 times higher than that of countries with the lowest mortalities (6.05 per 1000 live births).[3] The care provided to critically ill infants over the last 50 years has dramatically improved survival as a result of the advancements in knowledge and technology; however, we must continue our efforts to reduce infant mortality. One way to further reduce it is to improve our knowledge and surveillance of infants in the neonatal intensive care unit (NICU). Cardiac monitors provide information about respiration, heart rate variability, and arrhythmias; in addition, with the use of specialized bedside monitors, early signs of sepsis can be recognized. The purpose of this article is to review neonatal cardiac physiology, to examine neonatal arrhythmias

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visible on the bedside monitor, and to describe a specialized bedside monitor that provides data indicating early signs of sepsis.

THE PHYSIOLOGY OF A NEONATE: THE FIRST 28 DAYS OF LIFE

A neonate is a human newborn in the first 28 days of life. It is important to understand the transition from fetal to newborn circulation when performing a cardiac evaluation. Fetal circulation has high pulmonary pressures, which divert blood from the pulmonary beds and into the patent ductus arteriosus. Although not evident on the bedside monitor, high pulmonary pressure leads to right ventricular (RV) dominance and should be a consideration when interpreting the newborn electrocardiogram (ECG). This RV dominance is not always evident in preterm infants depending on their birth gestational ages: the closer to 40 weeks the infant is born, the more evident the RV dominance.[4] What is evident on the bedside monitors is the heart rate and a variance in heart rate, and that variance should be noted by the nurse. The median heart rate for newborn infants is 127 beats per minute (bpm); the rate increases over the first month of age to 145 bpm before decreasing over the next few years.[5] This rate can be lower when sleeping and higher with crying. Institutions have individual guidelines for low and high limits for neonatal bedside cardiac monitoring, which are based on the diagnoses an infant has on admission to the NICU. These limits can vary; for example, in a level II NICU, a limit of 80 bpm and 200 bpm may be acceptable if the unit does not have a large number of very premature infants (born before 27 weeks' gestation) and high acuity. Whereas, a level III NICU, with a large number of very premature infants with very low birth weight (fewer than 1500 g), may desire a level of 100 bpm and 250 bpm.

In the normal electrical conduction system, the impulse, which originates in the sinoatrial (SA) node located within the right atrium, activates the right and the left atrium, and the atria contract simultaneously. The atria contract and blood flows into the ventricles; the impulse continues to the atrioventricular (AV) node where it is delayed, which allows for filling of the ventricles. The impulse continues through the bundle of His, the bundle branches, and spontaneously terminates among the Purkinje fibers leading to an organized, simultaneous contraction of both ventricles. After the cardiac muscle contracts (depolarizes), there is a period of rest wherein the tissue cannot be excited to contract again (repolarization).

THE NORMAL ELECTROCARDIOGRAM OF THE NEONATE HEART

The neonatal sinus rhythm ECG has a P wave (atrial depolarization) before every QRS (ventricular depolarization), and this is followed by a T wave (ventricular repolarization). The QRS duration is shorter in infants than adults as a result of the reduced cardiac muscle mass.[6] The PR interval displays AV conduction and is the measurement from the onset of the P wave to the onset of the QRS complex (ventricular depolarization). The QT interval begins at the onset of the QRS and ends at the end of the T wave. The QT interval duration changes with heart rate, is age dependent, and is represented by the QT_c identifying the correction for heart rate.[7] The R-R interval is measured from the beginning of one QRS complex to the beginning of the next QRS complex. Many of these intervals are difficult to appreciate on a neonatal bedside monitor, especially with tachyarrhythmias because the heart rates are much faster than children and adults, which make the intervals much smaller.

The electrical axis is the direction the electrical impulse flows. Although there is a T and P wave axis, the QRS complex is the most important axis to determine. In a neonate with normal sinus rhythm, atrial depolarization occurs from the SA node