Resuscitation of the newborn

Lindsay FJ Mildenhall

Abstract

A newborn baby has innate reserves to withstand the physiological stress of labour and delivery. These reserves can become depleted and for a small proportion of newborns, assistance is required to aid transition from placental to extra-uterine pulmonary respiration. The compromised newborn arrest is a respiratory arrest and for the vast majority of those needing assistance with the transition, simple airway support manoeuvres will suffice. This article outlines the cascade of evidence-based interventions that include and follow on from airway support in those newborns requiring ongoing assistance. These comments are based around the International Liaison Committee of Resuscitation (ILCOR) 2010 and 2015 Consensus on Science statements and subsequent guideline reviews.

Keywords Asphyxia; cardiopulmonary resuscitation; neonatal; newborn life support; newborn resuscitation

Royal College of Anaesthetists CPD Matrix: 2B07

Learning objectives

After reading this article, you should be able to:

- have a fundamental understanding of the presumed mechanism for evolving perinatal hypoxic stress in a compromised birthing newborn
- recognize a newborn that is requiring assistance to transition
- institute a stepwise cascade of interventions based on the clinical response efficacy of resuscitation measures

Introduction

The paucity of quality research upon which neonatal resuscitation guidelines are based is alarming. A recent letter highlighted the issue with respect to the 2010 neonatal International Liaison Committee on Resuscitation (ILCOR) guidelines.[1] Current neonatology recommendations were based on 157 articles; only 15% were randomized controlled trials (RCTs) and 73% lacked a control group or were performed in a different population (usually animals) or mechanical models. Despite these challenges, the body of evidence upon which to build guidelines slowly grows. The ILCOR 2010 and 2015 reviews that make up the neonatal resuscitation algorithm have been subjected to evidence-based scrutiny or are in the process of being so.[2,3]

Terminology

There has been a move in neonatal circles to be very careful with terminology used to describe resuscitation procedures.[4] For example assisted ventilation does not deliver breaths as only living people and animals breathe, it delivers inflations. This terminology will be used here.

Physiology of the compromised transition

The transition at birth requires parallel anatomic and physiological changes to move from placental gas exchange to pulmonary respiration. The majority of newborns require little assistance to stabilize after birth and adapt to extra-uterine life. Approximately 10% of newborns born at term require some assistance to begin spontaneous breathing at birth. For the majority this involves drying, stimulation and head positioning. Approximately 3% will initiate breathing after positive pressure lung inflation and a further 2% after intubation. If resuscitation steps are implemented in a skilful and timely manner, most compromised newborns will improve. Only under 0.1% of all live-born deliveries need cardiac compressions and/or medications.

The fetus lives in a fluid-filled environment with the developing alveolar spaces themselves filled with lung fluid. Lung fluid production decreases as the fetus approaches term. With birth and as the baby takes its first spontaneous breaths the remainder of the lung fluid is absorbed into the pulmonary interstitial spaces or expelled from the mouth. These first breaths generate considerable intrathoracic pressure (approximately 50 cmH_2O); enough to fill the fluid-filled spaces with air. These air spaces, in the presence of pulmonary surfactant, do not completely collapse with expiration but maintain an air-filled volume (a functional residual capacity (FRC)). The air-filled volume increases with subsequent breaths. Lung compliance increases with each breath. The term baby has an ossified, non-compliant rib cage which helps generate the intrathoracic pressure and maintain the FRC. Preterm babies may have minimal surfactant, a very compliant incompletely ossified rib cage and an underdeveloped muscle mass that limits the efficiency of this process.

Our understanding of the physiology of birth asphyxia stems from classic studies conducted on fetal and newborn rhesus monkeys and lambs in the early 1960s.[5] This experimental model involved taking a pregnant female at term, incising the gravid uterus and surgically exposing the head of the fetus. The head was then enclosed in a bag of amniotic fluid or normal saline and the umbilical cord clamped at time zero. The healthy fetus in utero exhibits intermittent breathing movements as part of normal development. The hypoxic/acidotic physiological stress that cord clamping induces initiates a fetal gasping respiratory pattern. As the asphyxial insult continued this gasping after a few minutes ceases and a period of apnoea ensues (Figure 1). This period of initial breathing cessation is termed primary apnoea. If the asphyxial insult continues a second period of gasping respirations appears after 5–10 minutes followed by a second period of apnoea. This latter apnoea is termed secondary or terminal apnoea. After this second apnoea, spontaneous breathing will never recur unless the hypoxia is reversed. All these stages of asphyxia can be passed through in utero. If a baby is born in the

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Diagrammatic representations of the effect of hypoxia and intervention

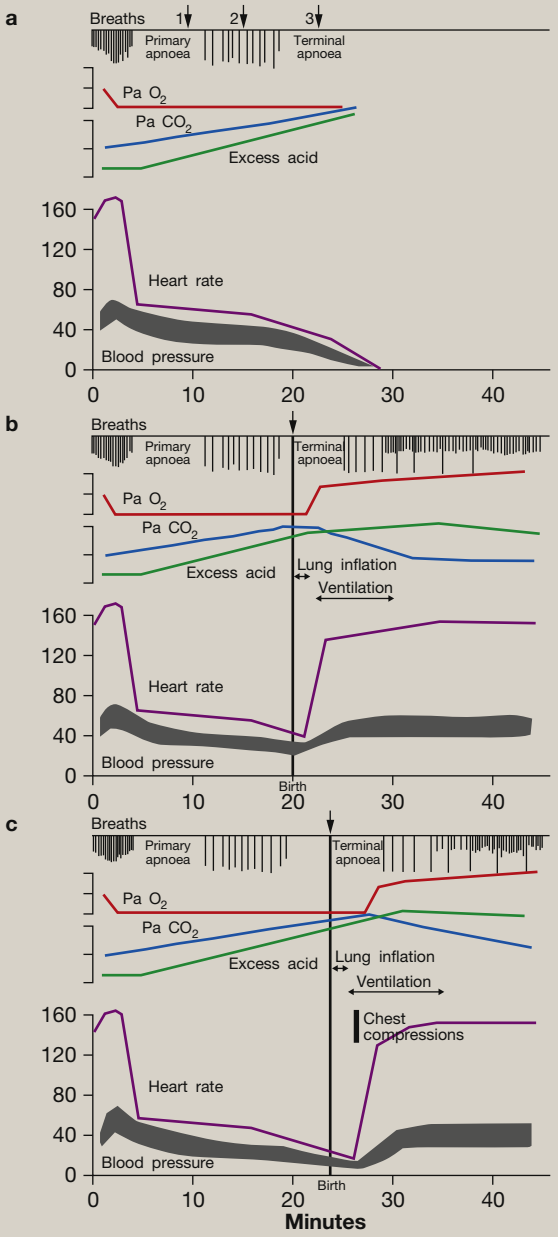

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Figure 1 (**a**) Primary and secondary apnoea following onset of total asphyxia at time zero. Arrows indicate points during process when babies might be delivered. 1: primary apnoea; 2: gasping phase; 3: terminal apnoea. (**b**) The physiological effect of lung inflation in a baby born in early terminal apnoea. (**c**) The physiological effect of chest compressions on a baby born in early terminal apnoea who did not respond to lung inflation.

period of primary apnoea, simple stimulation may be all the baby requires to start breathing again spontaneously. While the initial spontaneous breaths may be of a gasping nature a normal respiratory pattern will appear as the brainstem respiratory centre becomes oxygenated. If the baby is born during the period of terminal apnoea no amount of stimulation will initiate spontaneous respiration. This baby will need positive pressure lung inflation and oxygenation to establish breathing. Any depressed newborn may be in either of these stages of asphyxia. If a newborn response to stimulation is poor or non-existent one should assume this baby is in a period of terminal apnoea.

The cardiovascular system also attempts compensation mechanisms as asphyxia evolves. An initial rise in heart rate is followed by a fall paired with a rise in pulmonary vascular resistance and systemic resistance. The latter results in a redistribution of blood flow with increased flow to the head and the heart. Cardiac output is maintained and the while the oxygen content of the blood is lowered the amount of blood delivered to the heart and brain is optimised. The neonatal myocardium maintains a remarkable ability to keep functioning during periods of hypoxia. Neonatal myocardial cells have large stores of glycogen and this tolerance is presumed to be a result of higher rates of anaerobic glycolysis. Any reserve has limits however and in the presence of ongoing hypoxia the heart will begin to fail and then stop after 20–30 minutes. The first sign of recovery during neonatal resuscitation will be a rise in heart rate.

Temperature control

Hypothermia in newborns, especially those born premature, is associated with higher morbidity and mortality. However, *controlled* hypothermia is now a proven treatment in the term asphyxiated newborn. This treatment improves neurodevelopmental outcomes when implemented within 6 hours of the insult, especially in those with a moderate encephalopathy. It is very important that this group of babies are not rendered hyperthermic (>38.0) while awaiting assessment as to whether they meet the criteria for cooling. Overheating is associated with poorer neurodevelopmental outcome.

A newborn, non-asphyxiated infant should have a temperature maintained between 36.5 and 37.5°C. The act of drying a newborn with a warm towel and then replacing it with a warm clean one for cover has the dual function of limiting evaporative heat loss and providing stimulation as part of the initial assessment. Delivery rooms, including Caesarean theatres should ideally have an ambient temperature of between 22 and 26°C with the higher temperature preferred for the more premature newborns. These temperatures can be uncomfortable, especially for theatre staff. A warmed annex room next to theatre, where the baby can be stabilized after delivery, is used in some spheres. Delivery rooms should be devoid of draughts. Resuscitaires should have radiant or other heat sources.

The preterm baby presents special issues. Newborns less than 32 weeks should not be dried with a towel but be immediately enveloped in plastic wraps or plastic bags and placed under a radiant warmer. Assessment and resuscitation can be performed through the wrap. Other measures include placing a cap on the head and the careful use of thermal blankets. In resource-limited settings or deliveries away from a normal delivery environment, skin-to-skin care, called ‘kangaroo care’, with the mother is an effective measure while transfer is arranged.

Delayed cord clamping

Delayed cord clamping in the normal, well term newborn has become a practice returning to obstetric and neonatal practice.

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