Novel Approaches to Neonatal Resuscitation and the Impact on Birth Asphyxia

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KEYWORDS

• Asphyxia • Resuscitation • Sustained inflation • Oxygen • Cord clamping • Cardiac compressions

KEY POINTS

- In neonatal asphyxia at birth, effective and timely resuscitative measures are vital to avoid prolonging the hypoxic and ischemic insult.
- Adequate lung aeration is the key to a prompt restoration of heart rate and cardiac function, for which a sustained inflation can be helpful in achieving this.
- Hyperoxia needs to be avoided, and the extra oxygen that may be needed to restore cardiac function and spontaneous breathing needs to be titrated based on measured oxygen saturations.
- When ventilation is given effectively, cardiac compression is seldom needed.

INTRODUCTION

Birth asphyxia accounts for a quarter of neonatal deaths worldwide[1]; although 5% to 10% of newborns require some assistance at birth, only approximately 1% of infants need more extensive resuscitative measures.[2,3] If newborns are asphyxic, these measures should be initiated in a timely and effective manner to avoid prolonging the hypoxic and ischemic insults, which may cause permanent injury. Most asphyxiated infants only require assistance in the form of effective ventilation to restore breathing and improve circulatory function. Although a small proportion of infants will need cardiac resuscitation (chest compression and adrenaline) in most of these cases, it is likely

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that the bradycardia only persists when the ventilation is inadequate.[4] For this reason, the emphasis on neonatal resuscitation is on administering effective ventilation.

With regard to resuscitation procedures, caregivers are guided by the international consensus on science that informs resuscitation guidelines every 5 years. These guidelines were largely based on dogma; but more recently, recognition that the first few minutes after birth are critical for determining neonatal outcomes has prompted renewed interest in studies performed in infants at birth. Most new data are derived from experimental studies, as it is very difficult or impractical to perform clinical trials because of the infrequent occurrence of birth asphyxia and the unpredictable need for extensive resuscitation. In this article, the authors review the current knowledge on the physiologic responses to asphyxia and the novel approaches for resuscitation.

PATHOPHYSIOLOGY OF PERINATAL ASPHYXIA

Asphyxia is a mixture of hypoxia, hypercapnia, and a combination of both respiratory and metabolic acidosis caused by a reduction in respiratory gas exchange as well as the ensuing metabolic stress response.[5] Perinatal asphyxia can result from multiple complications, including disruption of blood flow through the placenta, placental detachment, compression of the umbilical cord, prolonged labour,[5] and failure of the newborn to initiate pulmonary gas exchange after birth.[6]

Although adults respond to hypoxia/asphyxia with tachycardia and an increase in respiratory drive, the fetal response is very different as it induces apnea and bradycardia.[6] Our current understanding of the cardiorespiratory responses to birth asphyxia is primarily based on animal experiments conducted in the 1960s, which usually involved placing a water-filled bag over the newborn's head after delivery (**Fig. 1**).[7–9] With the onset of asphyxia, the first sign of compromise is cessation of respiratory efforts, commonly referred to as primary apnea, which is accompanied by a profound bradycardia (see **Fig. 1**; **Fig. 2**). This bradycardia is rapid in onset, mediated by vagal inputs, and is most strongly initiated by both hypoxia and acidosis in the fetus. Primary apnea is followed by a period of irregular gasping, which culminates in secondary or terminal apnea that eventually results in cardiac arrest if the compromise continues. Although the bradycardic response to asphyxia is immediate, arterial blood pressure tends to initially increase before gradually decreasing because of myocardial energy failure caused by the hypoxia (see **Fig. 1**). If ventilation of the lungs occurs before the blood pressure decreases to less than a critical level, both heart rate (HR) and blood pressure are rapidly restored but of concern can cause a rebound tachycardia and hypertension.[5,6,10,11]

The pathophysiology of fetal asphyxia has been well documented by animal studies; but the interpretation of these data has been overextrapolated, and the experimental context in which the data were collected has been overlooked. As a result, it is widely assumed that all bradycardic infants at birth are asphyxic and that reoxygenation is the key to birth asphyxia and perinatal resuscitation. Although oxygen is important, this is clearly an inaccurate assumption as a high proportion of normal term infants, considered normoxic, have a HR less than 100 within the first minute after birth.[12] Clearly there are other factors influencing HRs at birth, one of which is the timing of umbilical cord clamping (UCC).[13,14]

As the placenta receives a large proportion of fetal cardiac output (30%–50%), umbilical venous return provides a large proportion of preload for the fetal heart, particularly the left ventricle. In contrast to adults, fetal pulmonary blood flow (PBF) is low and so pulmonary venous return provides only a small proportion of left ventricular preload, with the majority coming from umbilical venous return.[15] This umbilical venous return