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Neonatal Resuscitation at Birth with Intact Placental Circulation

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Abstract

Neonatal asphyxia at birth is a major cause of mortality and long term disability. However the clinical diagnosis is imprecise and largely subjective. At the moment of birth it is usually the obstetrician or midwife who has to make the diagnosis within the first 60 seconds. Logically positive pressure ventilation is the treatment of the apneic neonate but in order for this to be carried out on the roomside resuscitation trolley, another intervention, the intervention of cord clamping is required. Cord clamping is therefore performed, by definition, before neonatal breathing is established, and it is now known that clamping the cord at this stage shocks the cardiovascular system. The shock of early cord clamping results in bradycardia, hypoxaemia, hypovolemia, reduced cerebral circulation and reduced muscular tone, and reduced activity of the respiratory centre. Permanent injury and long term disability is possible if these adverse factors cannot be reversed quickly during resuscitation after birth. Being prepared for and then providing effective ventilation close to the mother with an intact cord can prevent all these adverse events and also initiate a physiological transition from placental to pulmonary respiration.

Keywords: Neonatal resuscitation; Birth; Placental circulation

Introduction

Asphyxia and hypovolemia

How accurate is the diagnosis of asphyxia at birth? In medicine, the success of any treatment requires an accurate diagnosis of the condition being treated. If the diagnosis is inaccurate then the treatment is not expected to work. Thus the treatment of apneic asphyxia at birth is positive pressure ventilation PPV) until the asphyxia is reversed sufficiently to restore the function of the respiratory centre. If hypovolemia is the cause of the apnoea, or even a contributory cause, then ventilation alone will not be effective until the hypovolemia is reversed and the neonatal blood volume restored [1].

Bradycardia

Neonatal bradycardia results in a fall in cardiac output which is reflected in pallor and cyanosis in the skin, and in addition poor circulation of all the major organs. Thus bradycardia in the neonate not only reduces the cerebral and coronary circulation, it also reduces the potential for a normal pulmonary circulation. At a time when respiration is transferring from the placenta to the lungs, the new pulmonary circulation may be critically reduced as a result of bradycardia.

It is clear that at birth neonatal hypovolemia and bradycardia must be avoided if possible, and the cause of the apnoea and diagnosis of asphyxia must be as accurate as possible.

Narcotic analgesia

Supression of the respiratory centre by narcotic analgesia should be an unusual cause of apnoea at birth now a days. The treatment is to reverse the narcosis with an opioid receptor antagonist, and until the reversal of narcosis is effective, ventilation should prevent any long term brain injury.

Cord clamping at birth

Early cord clamping at birth is well recognized to have a number of adverse effects on the neonate [2]. As a result of the known harm of the intervention of early cord clamping, ILCOR, NICE and WHO do not recommend early cord clamping at birth [3,4]. The degree of harm of early clamping is greatest in preterm neonates [5] but the harm extends to term babies where it has been shown to lead to anaemia, reduced iron stores and poorer brain development [6-10].

Early cord clamping

Early cord clamping has a number of adverse effects on the neonatal circulation and respiration [11]. Clamping the cord quickly after birth immediately cuts off the placental circulation which consist of 40% of the combined output of both ventricles. This leads to a sudden shock to the circulation with increased blood pressure, followed quickly by a reduction of 40% of the preload and a fall in cardiac output. Further the oxygenated blood returning from the placenta is lost before there has been any opportunity for oxygenation within the new neonatal pulmonary circulation [12].

In contrast during a physiological transition the closure of the placental circulation is gradual and as the vascular resistance of the umbilical arteries increases, the vascular resistance of the pulmonary circulation falls. Thus the blood is redirected from the placenta to the lungs. The umbilical vein remains open and a significant proportion of the blood volume in the placenta is redistributed into the neonatal compartment of the circulation [13]. Some of this blood volume fills the newly opened pulmonary circulation, while the rest of the blood volume increases the circulation of the bowel, respiratory and skeletal muscles, while maintaining the cerebral circulation. This redistribution of blood volume is termed the placental transfusion. Loss of the placental transfusion results in reduced blood volume at birth and thus

a reduction in the circulation of the lungs, bowel and brain. In the longer term there is anaemia, iron deficiency and brain development is reduced. In the preterm neonate the disruption to the circulation is more critical, leading to IVH and necrotizing enterocolitis, and a lower cerebral oxygen [14,15]. This is shown in a systematic review showing that preterm neonates subjected to early cord clamping at birth have a higher mortality and a higher risk of intraventricular hemorrhage and NEC [16,17].

When there is expected to be compromise in the function of the lungs or heart, avoiding early cord clamping, assisting transition with an intact cord is feasible and improves outcomes [18,19].

Early cord clamping, APGAR score and neonatal resuscitation

Early cord clamping has well recognised effects on some of the parameters used to determine the Apgar score and therefore impacts on the diagnosis of birth asphyxia and the need for neonatal resuscitation. Many years ago early cord clamping was shown to lead to neonatal bradycardia [20]. While colour is no longer included in the ILCOR criteria for determining the need for PPV, it is included in the Apgar score. The Apgar score is still used to broadly define and document the condition of the neonate at birth. Hypovolemia caused by early cord clamping will thus make pallor more likely. In addition the reduced blood volume and reduced cardiac output will impact on cerebral circulation, and increase the risk of a depressed respiratory centre. The muscular tone of the neonate is dependent upon a normal brain circulation which may be compromised by early cord clamping. Thus three of the five Apgar parameters are likely to be reduced by early cord clamping, and thus increase the need for neonatal resuscitation based on clinical assessment.

Traditional diagnosis and treatment of birth asphyxia

An observational study showed that for neonates apparently needing resuscitation, the outcome is inversely related to the interval before cord clamping [21]. It is well recognized that a number of neonates thought to be asphyxiated at birth recover quickly after being moved away from the mother to the room-side neonatal resuscitation trolley. Cleary the diagnosis of asphyxia was inaccurate. However not only is the diagnosis of asphyxia far from accurate, the treatment of neonatal asphyxia by positive pressure ventilation has never been subjected to a randomised controlled trial. Treatment of asphyxia is partly justified by animal studies and partly on logical and traditional practice [22]. The identification of the neonate needing ventilation at birth was pioneered by the anesthetist Virginia Apgar [23]. In her studies the umbilical cord had already clamped soon after birth. The neonates had all been delivered by caesarean section under general anesthetic. It was argued that immediate clamping at birth would reduce the transfer of anaesthetic agents to the neonate.

Nowadays the majority of caesarean births are under regional anesthesia so this argument does not apply. Some obstetricians are concerned that if care of the neonate remains close to the open uterine incision there may be excessive maternal blood loss if the obstetrician is less able to deal with bleeding express from the uterus. However for elective caesarean section there is now evidence that waiting several minutes before clamping and cutting the cord to remove the neonate away does not increase maternal blood loss [24]. Birth asphyxia at elective caesarean section of a normal term neonate should be quite rare, but it will be common in many emergency caesarean births when the indication for the surgical birth is fetal distress. There is usually no difference surgically between the two, so emergency caesarean births should not be any more susceptible to excessive blood loss than elective surgery. However neonates already compromised by intrapartum asphyxia are very much more likely to be harmed by early cord clamping.

Method

The accurate diagnosis of asphyxia is also important both for clinical audit and for medico-legal reasons and currently depends on the cord blood gases. The values of the cord blood gases are considered to represent the extent of hypoxia at the end of labour and determine whether or not appropriate clinical care during labour was provided. The normal range of cord blood gases is based on a cohort of healthy neonates who had early cord clamping. During neonatal transition with the cord intact the blood circulating through the cord and placenta becomes increasingly acidotic. The change is minor but could sometimes be of medico-legal significance when a threshold is crossed. The increased acidosis is thought to be due to the release into the neonatal circulation of lactate trapped in poorly perfused parts of the fetal circulation, a so called “hidden acidosis”. After early clamping this acidotic blood is hidden in the newborn and remains hidden unless there is the rare need for a neonatal blood gas test within minutes of early cord clamping. Not only is this acidosis hidden in the neonatal blood after early clamping, it is also redistributed into a smaller volume of blood as a result of loss of the placental transfusion. So if acidosis contributes to brain injury (in addition to hypoxia) then the brain injury of hypoxia and hypovolemia is likely to be exacerbated [25].

Chest compression

The ILCOR algorithm for neonatal resuscitation recommends that chest compression should be commenced if the heart rate falls below 60 bpm. The logic for this intervention is to increase the cardiac output. In children and adults chest compression is recommended when there is asystole. When there is asystole there will be zero cardiac output so chest compression, resulting in heart compression, is expected to increase cardiac ouput above zero. However if there is already some cardiac activity, cardiac compression is only likely to increase the output if the compressions coincide with systole or perhaps half way through prolonged periods of asystole. At other times in the cardiac cycle chest compression may actually interfere with cardiac output [26].

Early cord compression leads to bradycardia and in some neonates will therefore result in the heart rate falling below the threshold for recommending external cardiac compression (Chest compression). Thus, what seems to be a trivial intervention, may have marked consequences to a neonate with mild compromise, increasing the compromise from the loss of oxygen [27], from the hypovolemia, from the bradycardia and the disruption of cardiac output caused by chest compression [28].

Motherside neonatal resuscitation with an intact cord

The only way of avoiding the damaging intervention [29] of early cord clamping, which in some neonates will directly cause a need for resuscitation, is to be prepared to provide ventilation at all births close enough to the mother to maintain an intact cord [30-32]. This mother-side neonatal resuscitation needs to be fully planned and practiced. It requires closely coordinated team work and may well require different

approaches for the different modes of birth [33]. For example, at a normal birth, when the need for resuscitation is unlikely, it may be sufficient for the midwife to have immediate access to an Ambu bag and mask and, when needed, can provide positive pressure ventilation right by the mother with the cord intact [34,35]. At the other end of the spectrum, at emergency caesarean section for fetal distress, specialized or modified equipment will be needed so that a platform is available for the baby and more sophisticated ventilation and monitoring equipment available [36,37]. This equipment is now available to provide resuscitation at the side of the mother with an intact cord [38,39].

Discussion and Conclusion

Optimal care during labour ideally results in delivery of the neonate before a critical threshold of asphyxia is reached. The normal range of cord blood gases is based on measurement of the cord blood after immediate cord clamping [40]. If there is an adverse outcome for the neonate, cord blood gases within the acceptable range will relieve the obstetric team for responsibility of the outcome. However if early cord clamping has been carried out, with all the known adverse consequences of early clamping, the obstetric team might actually have been responsible for neonatal injury, but a normal cord blood gas result will disguise this responsibility [41].

Hypovolemia is probably the most serious harm of early cord clamping and is greatest when there has been a degree of cord compression. Cord compression affects the low pressure blood flow returning to the neonate more than the high pressure arterial outflow. Thus more blood is pumped out into the placenta than returns from the placenta and there is a net increase in blood volume in the placenta and a net reduction in the fetal compartment. Cord compression results in variable decelerations seen on the CTG. If the cord is clamped quickly after birth the normal redistribution of blood volume back into the neonate (the placental transfusion) is prevented and cannot be reversed after cord clamping. This may result in a significant neonatal hypovolemia at the moment of birth [42,43] and provide an explanation for the significantly poorer prognosis for a neonate with increasing acidaemia in the first two hours after birth [25].

A nuchal cord is a common cause of cord compression [44], and current advice is to clamp and cut the cord if it cannot be brought over the neonates head. The somersault manoeuvre [45], which allows the delivery of the baby's body to deliver with the cord intact, is not described in any RCOG guidelines [46]. Cutting and clamping a nuchal cord represents the earliest form of early cord clamping possible. If there is shoulder dystocia then the hypovolemia of early clamping is exacerbated by increased hypoxia [47]. Use of the somersault manoeuvre with delayed cord clamping and when necessary, mother-side neonatal resuscitation, will avoid all these adverse events. The hypoxia of early cord clamping may increase the risk of hypoxic ischemic encephalopathy and a change in medical practice in the delivery room is required [48]. Attributing adverse outcomes to early cord clamping and the expectation that avoiding this intervention will always avoid the markedly hypoxic ischemic neonates requiring hemodynamic support [49] is too simplistic, however only totally abandoning cord clamping at birth can provide a figure on just how many neonates will no longer need the extensive rescue treatments developed over the last 30 years.

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