

## The Broader Implications of Early Cord Clamping at Birth.

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### Introduction

Initially introduced as part of active management of the third stage of labour to reduce the risk of post-partum hemorrhage [1], the safety of early cord clamping was soon questioned [2] and investigated [3]. The finding that depriving the neonate of a volume of blood [4] resulted in anaemia and iron deficiency six weeks later [5] was not surprising but only recently this anaemia and iron deficiency has been shown to have more significant long term adverse consequences [6]. While a reduction in fine motor skill is not trivial, it is unlikely to be life-threatening. The consequences of blood loss is very much volume dependent. The average healthy adult can donate 500mls without anything more than mild inconvenience, and the cardiovascular system quickly compensates so that normal activity can be resumed. What is the maximum volume that a neonate can lose before the cardiovascular system is unable to compensate? If this volume is exceeded what are the likely consequences. In this article we use current evidence to implicate a number of adverse clinical outcomes resulting from the simple act of early cord clamping at birth.

### Definition of early cord clamping

The definition of early cord clamping is variable and loose in the same way there is no exact and consistent definition for delayed cord clamping. In principle, early cord clamping is interfering with a physiological transition in the neonate while delayed cord clamping is not interfering. The quicker the cord is clamped after birth the more the circulatory transition is disrupted. Once the placental circulation has closed naturally, cord clamping has no effect on the neonatal circulation. Closure of the placental circulation usually takes about 5 minutes.

### Loss of volume

The volume of the placental transfusion can be easily and accurately measured by continuously weighing the baby during transition after birth. The original work of Yao [7] was repeated recently using accurate and recording electronic scales [4] and they showed that maximum volume of the placental transfusion can be at least 205 mls. By implication cord clamping within seconds of birth will prevent that volume of blood reaching the neonate. All these babies were healthy and none were likely to have any significant cord compression. However cord compression leads to an increased volume of blood in the placenta and therefore a greater loss of blood volume from early cord clamping [8].

### Loss of cardiac output

Hypovolaemia leads to a fall in preload of the heart and a fall in cardiac output. Early cord clamping also leads to bradycardia [9] and

the combination leads to a marked fall in cardiac output and cerebral circulation. Brain injury [10] is the result of ischemia and the injury of subsequent reperfusion [11].

### Loss of oxygen

The blood in the umbilical vein returning from the placenta is remains oxygenated for at least 90 seconds after birth with an oxygen content of 3.77 kPa [12]. This is more than enough oxygen to keep a man functioning normally at the summit of Everest [13]. Early cord clamping prevents this supplemental source of oxygenated blood during transition. There is also the lost oxygen carrying capacity of this blood in the pulmonary circulation [14]. The combination of loss of oxygen and loss of cerebral perfusion may lead to catastrophic hypoxia and ischemia of the brain [15,16].

### Loss of stem cells

Early cord clamping results in a high residual volume of blood remaining within the placenta and this volume of blood contains the stem cells capable of repairing and restoring tissues anywhere in the body [17]. It is these stem cells which are stored for future transplant. However it is possible that an additional number of these stem cells at birth could initiate immediate repair of damaged tissue [18]. This approach is being researched to prevent the onset of cerebral palsy [19].

### Jaundice

Neonatal jaundice is the result of bilirubin production exceeding the capacity of the neonate's excretion system. Some jaundice is a part of physiology but is pathological in some circumstances when there is excessive production. A reduced excretion is also possible and hypoxia and ischemia could reduce the capacity of this system [20].

### PPHN

Persistent Pulmonary Hypertension of the Newborn (PPHN) is a condition where there is failure of the fall in pulmonary vascular resistance after birth [21]. In order to maintain an adequate pulmonary blood flow the pressure in the pulmonary system has to be much higher than normal. While there are some recognized causes of the high pulmonary vascular resistance, some are idiopathic. At birth the pulmonary vascular resistance falls dramatically with vasodilation of the vessels as a result of the high oxygen tensions in the lungs with the first breaths. Hypoxia is recognized to cause vasoconstriction in these vessels [22].

After birth, and the increased pulmonary blood flow, the blood returning in the umbilical vein can no longer pass through the foramen ovale to the left atrium since most of the atrial capacity is now

being met by oxygenated blood returning from the pulmonary veins. Thus the oxygenated blood from the umbilical vein is directed into the right atrium and pumped by the right ventricle into the pulmonary system. Now, instead of densely deoxygenated blood, the pulmonary system is receiving partially oxygenated blood. This may have a further vasodilatory effect. Any failure of respiration in these first 90 seconds after birth will not result in constriction of the pulmonary vessels, which may be the case if the cord was clamped.

### Patent ductus arteriosus (PDA)

In the fetus the ductus arteriosus (DA) plays a vital role in allowing the two sides of the heart to pump in parallel. Soon after birth it closes to allow the serial system of the neonatal circulation. A soon as the right and left ventricles are capable of pumping the same volume of blood, the ductus is redundant. There are a number of stimuli which lead to closure of the ductus arteriosus [23], including the rise in oxygen tension in the neonatal blood at birth. Until closure occurs the direction of the net blood flow across the DA depends on the net pressures at either end. Early cord clamping leads to a fall in right ventricular output [24] and when the right ventricle is pumping less than the left, reverse DA flow (L to R) is required. This may not occur when early cord clamping is completely avoided [25].

### Abrupt occlusion of the umbilical arteries

Early cord clamping results in sudden occlusion of the umbilical arteries which at the moment of birth are carrying about 40% of the combined cardiac output. It is inevitable that there will be a sudden rise in arterial pressure of the neonate [26] and the possibility of rupture of susceptible vessels leading to the well-recognized risk of intraventricular haemorrhage in preterm babies after early cord clamping [27].

### Conclusion

Often considered a trivial intervention, early cord clamping can have serious very serious consequences. The intervention has been tolerated by millions of babies without obvious harm for the past 50 years. Although a common intervention it is virtually never documented so any cohort study is impossible. Some of the harms described above have been confirmed through small randomized controlled trials or case studies while others remain hypothetical but based on a sound understanding of physiology. Only with a worldwide change in practice will it be possible to determine, in due course, the broader implications and harms described in this article.

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