

The Conscious Neonate and the Neonatal “Faint”

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Abstract

The Resuscitation Council (UK) explains the pathophysiology of acute perinatal hypoxia. They explain that “if subjected to sufficient hypoxia in utero, or during passage through the birth canal, the fetus will attempt to breathe. If the hypoxic insult is continued the fetus will eventually lose consciousness. Shortly after this the neural centers in the brainstem which control these breathing efforts will cease to function because of lack of oxygen. Thus the fetus born after significant and prolonged intrapartum hypoxia will be unconscious and if the neural center of the brainstem is affected the neonate will make no respiratory efforts”. Therefore, the neonate is floppy and unresponsive from unconsciousness, and will make no respiratory efforts in response to the hypoxia as a result of depression of the respiratory center. This neonate requires ventilation to rescue it from increasing hypoxia. Previous researches described a simple scoring system to the neonate in the first minute after birth so that the healthy neonate requiring no attention could be differentiated from the neonate that required ventilatory assistance. The Apgar score measures muscle tone and responsiveness and the effectiveness of the cardiac output, circulation and respiration.

Keywords: Ventilatory assistance; Neonatal; Cardiac output

Introduction

The fully conscious term neonate is easy to recognize at birth. It will have its eyes wide open, have good tone and make muscular movements, and quickly after birth commence breathing and crying. Just like the neonate, the fetus undergoes cycles of sleep and wakefulness. At birth there is a large surge of noradrenaline which ensures the healthy neonate is fully awake at birth [1-3]. With a normal cardiac function and good circulation this provides an Apgar score of 9 and once pulmonary respiration is established a score of 10. On the other hand, the unconscious neonate has its eyes closed, is floppy and unresponsive and makes no attempt to breath. If the loss of consciousness is the result of poor cardiac output and reduced cerebral circulation, the Apgar score will be only 1 or 2. Thus it can be understood that the individual elements of the Apgar score are not usually independent but rely largely upon a good cardiac output and good cerebral circulation.

Causes of neonatal unconsciousness

Intrapartum hypoxia is a significant problem and it is always assumed to be the major reason why a neonate is unresponsive at birth. However, are there any other reasons why a neonate is unconscious at birth? If the mother has been given a narcotic sedative which has crossed the placenta, the fetal brain will be sedated. In modern obstetrics the risk of narcotic analgesia is well recognized and avoided if possible.

Other causes which affect consciousness in children and adults should also be considered to be possible in the neonate at birth. Fainting is the sudden but temporary loss of consciousness in a healthy individual is well recognized to occur as a result of vasovagal response by the heart and veins of the lower body. Pregnant women are

particularly susceptible to a vasovagal faint where the cardiac output suddenly falls due to pooling of blood in the lower part of the body leading to a low cardiac preload. This is immediately corrected when the individual loses consciousness and falls to lie prone on the ground.

Pregnant women are also susceptible to the supine hypotension syndrome if they lie flat on their back. The weight of the uterus can compress the inferior vena cava and this results in a reduced cardiac preload and cardiac output. In the 1960's this was first recognized to be of particular importance during caesarean section [4,5]. It is prevented by tilting the woman onto her left or right side and relieving the pressure of the uterus on the inferior vena cava [6].

Loss of consciousness from loss of perfusion of the brain is quite sudden, and the return of consciousness is also rapid when perfusion is restored. Thus the baby may have normal consciousness at the moment it is born but, if it then suffers a sudden fall in cerebral perfusion, a neonatal “faint”, it will become unconscious. It will be hypotonic and unresponsive and fail to commence breathing. If perfusion is then quickly restored, it would be expected to recover consciousness and to respond to the new environment as normal.

Anything which therefore leads to a significant fall in cardiac preload, resulting in a marked fall in cardiac output could lead to a loss of consciousness in the neonate at birth. The neonate that is unconscious from intrapartum hypoxia is initially indistinguishable from the neonate that is unconscious from a sudden fall in cardiac output and cerebral circulation. It is likely that this partly explains why some babies with low Apgar scores having no evidence of intrapartum hypoxia on cord blood gas tests [7].

Causes of sudden fall in cardiac output of neonate at birth

Intrapartum cord compression: Intrapartum cord compression results in compression of the low pressure umbilical vein and a loss of oxygenated blood returning to the fetus. This results in the typical

deceleration of the fetal heart seen on the cardiotocograph. Flow in the higher pressure umbilical arteries is not so readily affected and this leads to a greater volume of blood pumped out into the placenta then returns in the umbilical vein. However, the compression is usually intermittent and coincides with a uterine contraction so that between contractions the volume of blood in the placental compartment of the fetal circulation may return to normal.

The general compression of the fetal body within the pelvic canal and uterus maintains a high pressure in the peripheral vessels and helps to maintain the preload of the fetal heart. At birth the body compression is suddenly lost and the preload of the heart more dependent upon the placental return than normal. The pelvic pressure on the fetal body may lead to something similar to the prolonged immersion syndrome so that when the fetus is delivered there is a sudden fall in cardiac preload. This may lead to a sudden loss of cardiac output and in its most extreme form to cardiac arrest [8].

Cord compression from a nuchal cord presents a particular problem which can lead to severe neonatal hypovolemia [9]. It is often recommended that the nuchal cord should be clamped and cut if it is not loose enough to pass over the neonate's head. However, a nuchal cord will rarely prevent delivery of the body and if necessary the body can still be delivered using the Somersault manoeuvre without dividing the cord [10,11]. The risk of established hypovolemia is reduced and any loss of consciousness is short lived once the hypovolemia is corrected from the redistribution of the placental blood back into the neonate through the intact cord.

Vasa praevia hemorrhage: Vasa praevia results in large unsupported cord vessels running within the fetal membranes. Rupture and bleeding puts the fetus at very high risk and only rapid recognition and delivery will prevent fetal death from hypovolemia. Hypovolemia from hemorrhage can only be corrected by volume replacement after delivery of the neonate. Elective caesarean birth is generally recommended when a vasa praevia is detected. Even without rupture and bleeding the vessels are very susceptible to compression which can also lead to hypovolemia of the fetal compartment. Given the opportunity by relief of compression of the vasa praevia, the blood volume of the neonate will be restored from the blood accumulated in the placental compartment.

Vaso vagal response: At one minute after birth the normal range of heart rate of the 50% of healthy neonates with Apgar scores of 9 is considered to include the range of 50 bpm to 90 bpm [12]. This is well below the normal range for the fetus and also for the neonate ten minutes later. The bradycardia may be a vagal response of the neonate to birth [13].

Cord prolapse: Cord prolapse is associated with both compression of the cord between the presenting head and the pelvis and with constriction in the umbilical vessels on exposure of the umbilical cord to cold and handling. This is an obstetric emergency requiring relief of the cord compression and maintenance of the cord circulation while preparations are underway for an emergency delivery of the neonate. While most of the loss of consciousness in such a neonate is likely to be from hypoxia, it illustrates the recognized importance of maintaining the placental circulation until the neonate has had the opportunity to transition to pulmonary respiration.

Hypoglycemia: Hypoglycemia seems unlikely to be a cause of unconsciousness at birth unless the mother also has severe hypoglycemia.

Elevation of the neonate: Elevation of the neonate, usually at caesarean section to allow the mother to see her newborn child over the sterile drapes was common practice until recently. It is now recognized that raising the baby well above the placenta can lead to acute hypovolemia. The procedure was largely confined to healthy babies delivered electively and now with a modified approach the mother can still have the chance to see and touch her newborn baby [14].

Discussion

In what circumstances can we expect neonatal cardiac output and cerebral perfusion to be reduced? Clamping late maintains perfusion and cerebral perfusion and by definition sometimes perfusion may not be maintained after early cord clamping [15]. This is likely to be most significant when there has been a large redistribution of blood into the placental compartment occurring during cord compression. Cord compression differentially reduces the low pressure venous flow much more than the high pressure arterial flow. Thus more blood is pumped out into the placenta than returns during cord compression. When the cord compression is relieved, the venous flow will exceed the arterial flow and return a volume of blood to the fetal body compartment and the risk of hypovolemia and reduced cerebral circulation is avoided. Brady [13] showed a significant bradycardia occurred after early cord clamping and attributed this to a vasovagal effect. Early cord clamping may therefore be a cause of loss of cardiac output and a loss of consciousness in the neonate.

A recent study showed that cerebral circulation is linked to cerebral activity shown on the EEG [16]. The study found that nine out of 244 neonates had a lower than normal cerebral activity for a short time after birth. They showed that those with low cerebral activity during immediate transition after birth also had low cerebral regional oxygen saturation as measured by near-infrared spectroscopy (NIRS). There was however increased cerebral oxygen extraction and this is consistent with reduced cerebral blood flow. The timing of cord clamping was not documented but the effect of early cord clamping is highly consistent with the findings of Farrar who showed that one out of 26 babies would be subjected to a significant hypovolemia as a result of early clamping [17].

Conclusion

There is good evidence that the neonate may lose consciousness at birth due to sudden reduction in cerebral circulation similar to the vaso vagal collapse in the adult. The appearance of the neonate with an acute loss of consciousness at birth due to loss of cerebral circulation is indistinguishable from the neonate unconscious from significant intrapartum hypoxia. For the neonate that has had intrapartum cord compression there may be an additional degree of hypoxia, and clinical concern about the hypoxia and findings of the CTG during labor. Both will respond to ventilation but the neonates with additional hypovolemia will only respond and regain consciousness if the volume of blood accumulated in the placental compartment during cord compression is allowed to return to the neonate with an intact cord during transition. Resuscitation without clamping the cord is now feasible with modified mobile resuscitation equipment [18,19].

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