There are times in our medical careers where we see a shift in thought that leads to a completely different way of doing things. This happened with episiotomy in the last few decades. Most recently trained physicians cannot imagine doing routine episiotomy with every delivery, yet it was not so long ago that this was common practice.

Episiotomy was supported in Medline indexed publications as early as the 1920s(1), and many publications followed in support of this procedure. But by as early as the 1940s, publications began to appear that argued that episiotomy was not such a good thing (2). Over the years the mix of publications changed, now the vast majority of recent publications on episiotomy focus on the problems with the procedure, and lament why older physicians are still doing them (3) (4). And over all this time, practice began to change.

It took a long time for this change to occur, and a lot of data had to accumulate and be absorbed by young inquisitive minds before we got to where we are today, with the majority of recently trained OBs and midwives now reserving episiotomy only for rare indicated situations.

Though this change in episiotomy seems behind us, there are many changes that are ahead of us. One of these changes, I believe, is in the way obstetricians handle the timing of cord clamping.

For the majority of my career, I routinely clamped and cut the umbilical cord as soon as it was reasonable. Occasionally a patient would want me to wait to clamp and cut for some arbitrary amount of time, and I would wait, but in my mind this was just humoring the patient and keeping good relations. After all, I had seen all my attendings and upper level residents clamp and cut right away, so it must be the right thing, right?

Later in my career I was exposed to enough other-thinking minds to consider that maybe this practice was not right. And after some research I found that there was some pretty compelling evidence that indeed, early clamping is harmful for the baby. So much evidence in fact, that I am a bit surprised that as a community, OBs in the US have not developed a culture of delayed routine cord clamping for neonatal benefit.

I think that this is a part of our culture that should change. This evidence is compelling enough that I feel like a real effort should be made in this regard. So to do my part in this, I am blogging about it.
As this is Academic OB/GYN, of course I am going to lay out this evidence I speak of. But before I do that, I want to present some logical ideas under which this evidence ought to be considered.

Prior to the advent of medical delivery, and for all time in animals, it has been the natural way of things for a baby to stay on the umbilical cord for a significant period of time after delivery. Depending on culture and situation, the delay in cord separation could be a few minutes or even a few hours. In some cultures the placenta is left on for days, which of course I find excessive and gross (5). But whatever the culture and time on cord, the absence of immediate cord clamping allows fetal blood that was previously in the placenta to transfuse back into the baby. Studies have demonstrated that a delay of as little as thirty seconds between delivery and cord clamping can result in 20-40 ml*kg-1 of blood entering the fetus from the placenta (6).

Considering this data, I have to think about evolution and function. I am a strong believer in evolution, but even under creationist thinking I have to believe that if the system meant for babies to have been phlebotomized of 50-100 cc of blood at birth, we would have been born with higher hemoglobins. Clearly the natural way of things is for this not to happen.

So does this mean that early cord clamping is necessarily harmful? Absolutely not. But what it means is that the burden of proof is on us to prove that early cord clamping, which amounts to planned fetal phlebotomy, is a beneficial thing. Otherwise, all things being equal we ought to give the tykes a few minutes to soak up what blood they can from the placenta before we cut’em off.

So the question is whether or not there is strong data either way.

It is easy to imagine a randomized study of immediate vs. delayed cord clamping, with quantitative analysis of fetal lab values and clinical outcomes. So easy in fact, that it has been done many times – and in just about every study, there is a clear benefit to delaying cord clamping, even if it is just for 30 seconds after delivery. These benefits include important outcomes such as decreased rates of intraventricular hemorrhage and necrotizing enterocolitis in preterm neonates. Furthermore, aside from some intermittent reports of clinically insignificant polycythemia and hyperbilirubinemia in term infants, there appears to be no harm that can be linked to delayed cord clamping. It feels like being a doctor 10-15 years ago looking to see if there is any data about episiotomy, and finding that there’s a lot, and it says we’ve been doing it wrong for awhile now.

So here’s the data:

**Delayed cord clamping in very preterm infants reduces the incidence of intraventricular hemorrhage and late-onset sepsis: a randomized, controlled trial(7)**

Randomized 72 VLBW infants (< 1500 grams) to immediate or delayed cord clamping (5-10 vs. 30-45 seconds). Delayed cord clamp infants had significantly less IVH (5/36 in delayed group vs. 13/36 in immediate group, p = 0.03) and less late onset sepsis (1/36
The Influence of the Timing of Cord Clamping on Postnatal Cerebral Oxygenation in Preterm Neonates: A Randomized, Controlled Trial (8)

Randomized 39 preterm infants to immediate clamping vs. 60-90 second delay, and examined fetal brain blood flow and tissue oxygenation. Results showed similar blood flow between groups, but increased tissue oxygenation in the delayed group and 4 and 24 hours after birth.

Effect of timing of umbilical cord clamping on iron status in Mexican infants: a randomized controlled trial(9)

Randomized 476 infants to immediate or 2 minute delayed clamping and followed them for 6 months. Delayed clamped babies had higher MCVs (81 vs. 79.5), higher ferritins (50.7 vs. 34.4), and higher total body iron. Effects were greater in infants born to iron deficient mothers. Delayed clamping increased total iron stores by 27-47mg. A follow up study showed that lead exposed infants with delayed clamping also had lower serum lead levels than immediate clamped infants, likely due to iron mediates changes in lead absorption.

A randomized clinical trial comparing immediate versus delayed clamping of the umbilical cord in preterm infants: short-term clinical and laboratory endpoints(10)

Infants delivering at 30 to 36 weeks gestation randomized to immediate vs. 1 minute delay. Delayed group had higher RBC volumes (p = 0.04) and hematocrits (p < 0.005), though there was no difference in RBC transfusions. There was a small increase in babies requiring phototherapy in the delayed group (p = 0.03) but no difference in bilirubin levels between groups.

Immediate versus delayed umbilical cord clamping in premature neonates born < 35 weeks: a prospective, randomized, controlled study (11)

Randomized 60 infants to clamping at 5-10 seconds vs. 30-45 seconds. Delayed clamping infants had higher BPs and hematocrits. Infants < 1500 grams with delayed clamping needed less mechanical ventilation and surfactant. Trend towards more polycythemia in delayed group, but not statistically significant.

And that’s just some of it. I’ll be happy to send you an Endnote file with a pile more of you’d like it. If the burden of proof is on us to prove that immediate clamping is good, that burden is clearly not met. And furthermore, there is strong evidence that delaying clamping as little as 30 seconds has measurable benefits for the infant, especially in premature babies and babies born to iron deficient mothers.

So basically, we should be doing this. I’m going to try to effect some change in my department, but there are a lot of things that need to happen for us to change as a general culture. It can’t just be the OBs. L and D nurses and pediatricians need to buy
Some people will argue that premature babies need to be brought to the warmer right away for resuscitation. I don’t know the answer to this, but it’s worth study. One might think that it is important to intubate a very premature baby right away, but I have to wonder if that intact cord will be better at delivering oxygen to the baby for 30-60 seconds than the premature lungs. Particularly in cases of fetal respiratory acidosis, there is strong logical argument that a baby might be better resuscitated by unwrapping the cord and letting it flow a bit than trying to oxygenate it through its lungs. Until that placenta is detached, you have a natural ECMO system. Why not use it? Certainly there are exceptions to this logical argument, abruption being the biggest one, and perhaps even severe pre-eclampsia and other poor feto-maternal circulation states.

I wonder at times why delayed cord clamping has not become the standard already; why by and large we have not heeded the literature. It is sad to say that I believe it is because the champions of this practice have not been doctors, but midwives, and sometimes we are influenced by prejudice. Clearly, midwives and doctors tend to have some different ideas about how labor should be managed, but in the end data is data. We championed evidence-based medicine, but tend to ignore evidence when it comes from the wrong source, which is unfair. It is fair to critique the research and the methods used to write it, but it shouldn’t matter who the author is. In this case, Mercer and other midwives have done the world a favor by scientifically addressing this issue, and their data deserves serious consideration.

To quote Levy et al (12) “Although a tailored approach is required in the case of cord clamping, the balance of available data suggests that delayed cord clamping should be the method of choice.” We ought to heed this advice better. Like episiotomy, this change in practice may take awhile, but we should get it started. I’m going to work on it myself. How about you?


Link for article on next page:
The cord clamp: A monument to stupidity.

(Third draft: refs not properly included. To get Professor Morley to check this, and give attribution to parts of his work used.)

In 1981, our birth plan included in writing, that our son’s umbilical cord and placenta remain attached until all pulsing had stopped and the cord had shrunk. It was NOT to be clamped. The doctor had noted our request, but as soon as our son was put on my stomach, she immediately clamped the cord right by my vagina where I couldn’t see it. However, I noted the direction of the midwife’s glance: It was she who confirmed the cord had been clamped. I was furious, but could do nothing, as everyone’s focus was on the damage their episiotomy had caused, in order to facilitate a face presentation.

Like so many other mothers who suffered under “active” (aggressive) third stage management, our son’s placenta was manually removed.

First, let’s talk definitions.

**Immediate cord clamping**: This is when a clamp is put on the cord immediately, amputating a functioning placenta and depriving the baby of around 50% of its total blood volume.

There is no excuse for this. Neonatologists say that immediate cord clamping allows them to take the baby to a resuscitation table quicker, but there is absolutely no reason why this cannot be done right there with the mother, so that the baby can still get blood that is rightfully its own.

**Delayed cord clamping**: This varies according to arbitrary paediatric “rules” such as “Wait a minute”, two minutes (Hutton et al, 2007) and if you’re lucky with staff day-dreaming, or a bit busy, maybe three minutes.

**Physiological (normal) cord closure**: This is when the baby clamps the umbilical vein and arteries at various locations inside its own body after an optimal blood volume has been transfused into the baby. This is the same process that occurs in all other mammals born with an umbilical cord.

A baby’s umbilical cord should not be clamped, even under exceptional circumstances such as caesarean sections, placenta previa or any other “excuse”. Should a caesarian or surgical intervention be necessary, the baby, cord and placenta should be removed as one, and the placenta hung up like a blood transfusion bottle, in order that the baby can obtain what is rightfully its, via gravity.

Normal cord closure physiology does not obey man’s clock. It is controlled by other parameters which vary according to circumstances. The transition for a baby, from life inside the womb, to life outside the womb is complex. A lot of crucial physiological changes take place unseen and interfering with this process can have serious consequences. Yet reading textbooks about how babies transfer from a fetal two chamber heart to an adult four chamber heart, you are told that a clamp stops the placental circulation. That much is true. Cord clamping stops circulation! However, a clamp is not part of the
physiological design and should not be used. The only book I found, to recognise that birth for the human mammal, (like other mammalian birthing) doesn’t require a cord clamp and pair of scissors, is Grey’s anatomy.

As Dr George Morley would say¹, “Man is the only mammal to routinely injure its newborns with a cord clamp.”

What happens between the head crowning, through the first five to ten minutes of life, with the blood in the placenta and cord, and why? Below, red blood is highly oxygenated, and blue blood has much less oxygen in it.

Inside the mother, up until the baby comes out of the vagina, a baby’s heart functions as a two chamber heart with the two sides of the heart working in parallel. The baby’s lungs act as an organ of excretion, producing around 400 mls of amniotic fluid a day. During labour, the baby releases

¹ http://www.bmj.com/cgi/eletters/335/7615/312#175640
catacholamines (a hormone), which results in lung fluid being absorbed, so that once a baby is born the lung alveoli (air sacs) can expand, allowing breathing to begin.

Blood from the placenta has an oxygen saturation of about 80%. It flows into the baby’s inferior vena cava, by passing the liver, mixing with the blood returning from the lower part of the body. The blood flows into the right atrium, and passes through into the left atrial septum. Both atria act as ONE chamber, because the foramen ovale is OPEN as a flap inside the left atrium. The foramen ovale lets the blood from the right atrium flow into the left atrium, then down into the left ventricle. It turns around, and the oxygenated blood goes up into the aorta.

Blood from the brain (“blue”) comes down the superior vena cava and is pulled into the right atrium at the same time as “red” blood is pumped through the foramen ovale. A small amount of “red” blood from the placenta is pulled down with “blue” blood from the upper body. This blood goes down into the right ventricle, turns around, and goes up the pulmonary artery. That blood splits three ways. Two-thirds of this blood goes up through the ductus arteriosus to join blood in the aorta (from the left ventricle), and one-third goes left and right to the lungs.

Because the lungs require very little blood, that blood quickly comes back via the pulmonary veins, into the left atrium, to join the oxygenated blood from the placenta, which came through the foramen ovale and joined the bulk that went up the aorta. The blood in the aorta which is now mixed, has about 58% oxygen saturation. That blood travels into the lower part of the body, and is returned to the mother carrying out waste, and ready to be re-oxygenated.

The ventricles also act as ONE chamber. So the heart is a TWO chambered pump in utero.
At the moment of birth a normal newborn is circulating a mixture of “red” and “blue” blood. The lips and tongue of a healthy newborn at birth is the same pinkish purple colour as the foetus has been for nine months in utero.

As a baby descends, the baby’s head comes out looking like a mango with cranial plates overlapping caused by considerable moulding of the baby’s head. Once the head comes out, these plates spring out, increasing the head circumference; the skin smooths and the brain is ready to receive more blood.

So long as the cord is still pulsating and exchanging oxygen in the baby, the tongue and lips will maintain this colour until the baby breathes on its own.

That first gasp and cry, cause the diaphragm to contract, and the intercostal muscles to increase chest volume and create a negative intra-thoracic pressure and the needed suction to help pull in the needed blood. This one action, results in around half of the placental blood being forced into the baby in less than a minute\(^2\). At the peak of this transfusion, not only the liver, but also the vena cava, the heart, the aorta and the pulmonary vessels become distended. Once air is drawn into the lungs, another reflex is triggered relaxing the pulmonary arterioles, which causes an enormous increase in blood flow to the lungs.

The baby cries, partly as a response to the wet skin cooling (cold pressor reflex), thus raising the blood pressure in the aorta. The pressure reverses the blood flow through the ductus arteriosis causing more placental blood to be transferred to the capillaries that supply the lungs. More oxygen in the ductus arteriosus triggers the duct to close, but it takes 15 hours to close completely. Structural closure is usually complete by two months of age.

Haemoglobin in the lung alveoli, releases CO2, which triggers the need for more oxygenation of the blood. The hepatic portal vein opens, allowing more blood into the liver, distending it and causing pain. This is thought to create a “safe backup”, when air temperatures are high, as the pain in the liver substitutes for the cold crying reflex. So if your baby is born in hot water, or a hot room, the liver distention may be the trigger for the first cry.

\(^2\) Mercer 2001 J midwifery and women’s health, figure 1 quoting Yao Lancet 69)
The start of breathing initiates various vessels to open or close. With the ductus arteriosus shutting off, the pulmonary arterioles open right up, causing more blood to flow through into the lungs, and more lung alveoli to expand. This high pressure perfusion of placental blood into the vena cava, heart and lungs “erects” the lung alveoli (“Jaykka” effect) allowing breathing to become efficient. It takes around five breaths for all the alveoli in the lung to expand. After that, proper oxygen transfer is established.

**The baby will now have a very large amount of blood flowing into the left atrium of the heart from the lungs.** This raises the left atrial pressure considerably, distending the left atrium and pressing the one-way foramen ovale flap valve, called the septum primum, back against the central wall of the heart, stopping blood flowing into the left atrium. Functional closure of the foramen ovale flap is usually completed quickly after the first breath, but structural closure (knitting together of flesh) takes longer.

The ductus arteriosus is properly closed within a day, and with the closed foramen ovale, the heart changes from a TWO chamber “fetal” parallel pump, to a FOUR chamber “adult” pump working serially (see left), with the right ventricle (“blue” blood) going to the lungs to be oxygenated, and the left ventricle output (“red” blood) going through the body. For this process to complete correctly, functional circulation from the placenta, pumping in all that blood is vital.

**Once breathing is properly established a second stage of shutting down the cord begins** in a two step process. The cord is also cooling, and could be considered a well designed refrigerator with no skin or blubber to keep it warm. It is only covered with a watery gel and a single layer of cells, the amnion. Water evaporation cools it rapidly, causing the vessels to constrict, helping to raise systemic blood pressure and reversing the ductus arteriosis flow. The now high arterial blood oxygen concentration triggers the umbilical arteries to close **next to the descending aorta, and behind the belly button.**

The placental vein continues to pump blood into the baby in a very measured and controlled manner as the peripheral circulation improves, helping push more blood flow through the lungs, and the rest of the body. **After the baby has received its full amount of blood,** high central venous pressure causes the placental vein flow to slow down markedly. The umbilical vein starts to close with a sphincter-like action, in portions of the vessel INSIDE the baby’s abdomen. The ductus venosus, where the umbilical vein joined the inferior vena cava, closes off, and the next contraction from the mother causes a slight blood surge, with a backwash, the pressure from which triggers for the umbilical to close completely inside the belly button. **The umbilical cord will by then look mostly empty, whitish and hard.**
Once natural umbilical closure is complete, the cord can later be detached within a few inches of the umbilicus without any form of clamp. This routinely happens in all mammals. Nothing is required for cord care other than an occasional clean with saline solution if required. Anything else prolongs the natural tissue break-down process by which the cord end “drops” off. Iodine or antibiotic gels are totally unnecessary, and counterproductive to the natural “drop-off” mechanisms.

No blood loss will occur from the baby’s cord stump, but some warm blood will drain from the placental portion of the umbilical vein showing that a bit of back-up blood in the placenta would have been there if required. The cord can be tested by stripping the cord away from the baby towards the placenta. The vein should stay empty.

The transferral of this blood from the placenta to the baby, not only provides total “life support” for the baby, and completes the baby’s normal physiological blood volume, but it also shrinks the surface of the placenta slightly, helping to initiate placental separation from the wall of the uterus. The natural transition from fetal to adult circulation via cord physiology, is co-ordinated and controlled within the baby, and is part of a carefully orchestrated process which should be left well alone. These complex mechanisms have been designed into the blue print of the human genome, to cover birth difficulties, and prevent and relieve any resultant birth asphyxiation. No chance evolution here!

According to Buckets (1965) the average total amount of blood in the placenta and cord is 166 mls of which some 115 mls belong to the baby. This amounts to around 30% of the baby’s final blood volume and 60% of the red cell volume. That is a huge amount of red cells that are needed for efficient oxygen saturation. For a neonatologist, resuscitating a baby whose cord was clamped immediately at birth will always be a struggle. After all, whoever clamped that cord just amputated 60% of the red blood cells that the neonatologist needs urgently to maintain respiration and oxygenation. In 1957, Gunther measured early clamping blood deprivation as between 100 – 200 mls.

Comparing the arbitrary three minute “delay” with immediate cord clamping, the difference in blood volume for a 4000 gram baby is 360 versus 280 mls.

So what is the total volume of blood the clamped baby is deprived of, when compared with the baby whose umbilical cord is allowed to close by itself? We don’t know. None of the studies on cord clamping included controls which were allowed to complete the process themselves without intervention.

This changeover from fetal to adult circulation is efficiently achieved by the use of this 120 - 200 mls of blood (depending on the author you believe) which is crucial to allow the lungs, liver, brain and extremities to have full blood volume and effective life support.

According to Hasselhorst et al (1938), 51 – 78% of placental blood is transferred after one minute and 79 – 82% within five minutes. Nelson NM (1975) in “Neonatology” said however that only 15 – 20 mls transfused within 3 minutes. A baby delivered with the mother squatting, and received on a warm towel, while kept lower than the uterus, can complete this process in about three minutes. The
standard horizontal hospital “normal” delivery slows this process markedly. Taking the baby up to the breast immediately slows down this process further.

While a clamp can be used to stop any static blood from dribbling out the placental end, the cord clamp represents a pompous monument to obstetric arrogance and …scientific ignorance. Humans have become the only birthing mammal with a serious defect: a clamp and scissor deficiency.

After cord vessel closure is complete, the placental transfusion of blood inside the baby is decreased over the space of four hours, as the baby removes fluid from the blood into the tissues, and is excreted via the kidneys and urine. The blood thickens a bit (becomes more viscous), which increases pressure in the circulation, and hematocrit values and albumin levels rise. This increases plasma colloid osmotic pressure is a survival strategy, which helps keep the lungs dry. Wet nappies soon after birth, shows that the baby has had a decent placental transfusion.

This extra fluid can also provide a survival buffer if the mother is unable to immediately provide pre-collostral fluid to the baby.

In hospitals where “routine” is paramount and breast feeding and lactation establishment is delayed for three or more days, weight loss is common in this period. This is explained away by saying that this excess fluid from placental blood is being excreted. However, why is it that homebirth babies whose cords close naturally, and who have breastfeeding established very quickly, do not lose as much weight?

So now let’s look at what happens when someone comes along with a clamp, and amputates a functioning placenta creating a “crisis” situation.

The heart size of a baby whose cord is not clamped is slightly larger than that of a baby whose cord is clamped immediately. (Buckels 65)

Babies whose cords are clamped immediately are pale, compared to the very pink babies who get all their blood. They have reduced blood thickness, much lower blood pressure, (47/62 mm Hg compared versus 65/78 mm Hg for late clamped babies) which means their bodies lack the grunt required to complete the heart closure. They have a much faster and more turbulent flow rate through the patent ductus arteriosis. They have a very high rate of heart murmurs with a soft blowing character, because the fast reversal of blood flow needed to close the ductus arteriosis and left atrial high pressure to shut the foramen ovale does NOT take place. Babies whose cords are not clamped are reported to rarely have heart murmurs.

The earlier the cord is clamped, the more blood is prevented from going into the baby and the greater the chance of serious damage. Reading through the medical literature, you can’t help but be alarmed by how many babies, with cerebral palsy, had hypovolemia (low blood pressure, oliguria); ischaemia (proven with an MRI brain scan) and anaemia, requiring blood transfusions. Immediately clamping off a functional placental circulation blocks placental oxygen exchange and the ability of the mother to
“breathe” for the baby, creating a crisis situation which results in a sudden redirection of blood flow. This is the equivalent of a massive instant haemorrhage in an adult.

The most potent stimulus that will make an immediately-clamped baby cry, is the shock of asphyxiation, caused by the inability of the baby to access that 60% of red blood cells that is lost in the amputated placenta and umbilical cord. The quick build up of carbon dioxide causes this baby to try to force the lungs to work before a lack of oxygen becomes dangerous. (Try holding your breath for two minutes!) Clamping the cord before breathing is fully established may cause asphyxia and force the child to breathe using lungs with little blood flow, and that are not ready to breathe.

Depending on how fast the cord is clamped, the baby will experience mild/moderate or severe hypoxia at birth. Even prolonged partial hypoxia at birth can cause subtle neuronal or synaptic damage in the brain resulting in subtly impaired cognitive function later in life. Babies who have low APGAR scores at birth but who do not develop encephalopathy are likely to have reduced IQ’s even if they remain healthy in the neonatal period. (Odd, David Lancet 2009)

The baby will try to get more blood into the lungs, and because blood is not available as it should be from the cord and placenta, the baby’s body constricts down all the arteries and veins, as a red alert situation prioritises where blood is most needed.

Some physicians still maintain that certain babies need to have their cords clamped and cut immediately. Perhaps it is due to the way some hospital births are still medical managed without staff giving any thought to the physiological changes taking place for the babies’ wellbeing.

Babies born asphyxiated because of cord compression during birth do not respond to pain, cold or carbon dioxide. Acidosis will result in increased CO2 tension, respiratory depression and reduced bicarbonate concentration. Cutting the cord, is exactly the same as leaving it compressed. If you leave the cord intact albeit compressed and get the baby out, the pressure will be relieved, the cord will open, and placental oxygenation will correct the asphyxia.

We experienced this first hand with our second son who was born with a double nuchal cord. Fortunately, the delivery was a home birth: The cord was unwrapped, replaced back up in the vagina and kept warm in order to prevent it from contracting.

The baby also had shoulder dystocia and it took a while longer to get him out. He tried to breathe just before he got out and with gravity, contractions, and leaving the placenta alone, his Apgar scores were 9 at one minute and 10 at 5 minutes. The doctor and midwife made sure to let him get all the blood he needed allowed him to self clamp his own cord on the inside.

**NEVER clamp the cord of a baby presenting with cord compression, or shoulder dystocia.** These infants shift blood to the placenta because of the tight compressive squeeze of the body in the birth canal, and
therefore can be born hypovolemic\(^3\) (without enough blood volume). Immediate cord clamping of these babies maintains that hypovolemic state by preventing the blood in the placental from readily returning to the baby. This blood loss caused by immediate clamping and rushing the baby to ICU to be “resuscitated” initiates an inflammatory response leading to seizures, hypoxic-ischemic encephalopathy and brain damage or death. Any baby who experiences hypovolemia or hypoxia, requires all of those huge quantities of CD34\(^+\) cells and stem cells present in the placenta and cord blood, which can repair any damage caused by the asphyxia. A cord-clamped baby is totally deprived of those repairing stem cells.

In an elective caesarean where there are no uterine contractions, the baby should not be delivered into the mother’s thighs above the level of the placenta. You will see the cord vessels become very full and distended. The baby should be held below the level of the uterus, until the placenta can be hung like a “unit of blood” for the baby to complete cord closure itself. If the baby is placed above the placenta, gravity can force blood back through the umbilical vein which has no valves, into the placenta in a flaccid uterus. The pulsating umbilical arteries will also contribute to engorgement of the placenta.

Babies whose cords are clamped quickly show a sudden sharp increase in cerebral pressure and flow which is a hallmark of hypoxic ischaemic encephalopathy. The inferior colliculi midbrain auditory pathway is most susceptible to damage than any other part of the brain, and hypoxic ischaemic encephalopathy can result in various degrees of mental retardation. Immediate cord clamping in premature babies causes intraventricular haemorrhaging in the brain.

Furthermore, babies whose cords are clamped immediately, have a very high risk of becoming anaemic, because there would be no breakdown of excess red blood cells with the body storing the removed iron.

Many medical people who resist returning obstetrics back to what is normal physiological function, state that delayed cord clamping results in polycythaemia without symptoms. The supposed “worry” about polycythaemia is thicker blood than normal or hyperviscosity. But given that most babies with polycythaemia never have problems, might that not be a normal state for a baby? Why are hospitals not full of polycythaemic homebirth babies?

Who defined the “normal” haematocrit, or the “normal thickness” of babies’ blood? And using what protocol? Are these so-called “normal” ranges solely the ranges seen in babies clamped between 1 and 2 minutes after delivery?

In 1965, Peltonen considered polycythaemia an oxygen reserve provided against physiological hypoxia.

Babies born naturally without a clamp, have a much higher haematocrit than those whose cords are clamped immediately or at one minute.

\(^3\) Mercer J Med Hyp 2009.
If the medical profession used actual values seen only in babies who close their own cords, to determine the baseline volume and haematocrit, babies whose cords are clamped immediately, would have thin blood, circulation compromise and anemia; they would have potentially suffered the equivalent of an iatrogenic 4 pint blood loss in an adult.

Perhaps what is considered polycythemia, is actually “normal” and necessary physiology.

Immediate cord clamping can cause Grade 1 intraventricular haemorrhage without symptoms, yet this is NOT considered to be a problem by paediatricians. I would suggest that bleeding in the brain, when it would not normally happen with no cord clamping at all, is indeed a problem. And I would suggest that the logic of the above two comparative reasonings is totally inconsistent.

Have you ever wondered why, when a baby’s cord is clamped immediately in theatre, they put two clamps on, and tell the husband to cut it between the two clamps? That’s because if there weren’t two clamps, all that blood in the long part of the cord and placenta, which belongs to the baby would gush out all over the table! Maybe the Dad would wonder where that blood really belonged or maybe he wouldn’t. Yet it amazes me that maternity staff can’t see the barbaric nature of this practice. Why is their knowledge of physiology so little that they can’t see what Darwin’s grandfather Erasmus Darwin wrote in 1801:

“Another thing very injurious to the child, is the tying and cutting of the navel string too soon; which should always be left till the child has not only repeatedly breathed but till all pulsation in the cord ceases. As otherwise the child is much weaker than it ought to be, a portion of the blood being left in the placenta, which ought to have been in the child.”

He wrote this, because immediate cord clamping started when male midwives took over from the women, who were considered witches. He could see then that it was a bad thing.

Consider how we progressed and then decide what you should do!

From 1773 most medical texts advocated leaving the cord alone until pulsation ceased.

Around 1913, immediate cord cutting became fashionable, because drugs disrupted normal delivery markedly. Early types of delivery anaesthesia caused the uterus to relax, and mostly anaesthetised the baby while analgesics such as pethidine and chloral hydrate prolonged cord pulsing. Because women were prevented from giving birth standing, or squatting, and were drugged and laid on their backs with legs up in stirrups, siphonage and arterial loss became common and ocytoxin was given to counteract this. This was the era of knocking women out, using forceps and basically interfering with natural delivery in any possible way. It was only logical that obstetricians would then take over cord management as well.

4 (Zoonomia, volume 11 page 321.)
Up until 1970, most texts still talked about leaving the cord alone until pulsation stopped.

Around 1976, textbooks stated that the cord should be clamped immediately or 30 seconds after delivery, the theory into line with the practice that had been going on for decades.

By 1986, a textbook stated that the optimal time for clamping wasn’t known and in 1994, a baby was usually immediately suctioned then the cord was clamped.

Now while a few lone voices campaign for doctors to see sense and return to natural physiological cord vessel closure, many obstetricians remain unwilling to stop the practice of immediate cord clamping. They do this despite knowing that there is no scientific rational for it. How many paediatricians or neonatologists have actually seen a baby delivered without a cord clamp?

The ultimate irony is that some physicians actually consider delayed cord clamping an “intervention” and immediate cord clamping “normal”.

Then of course, there are the cord-blood banks trying to persuade you to pay them to keep your baby’s blood for you, because all that blood, after the immediate cord clamp, is “wasted”; so why not keep it in case your baby gets cancer or something and you need all those stem cells which your baby should have got at birth.

What is now considered “normal medical practice” practice is a travesty of justice for the baby, when compared to natural physiological cord closure designed to give a baby the best possible start in life ... before the “clamp”. Disregarding these amazing design features built in to the new born baby is child abuse of the first degree. Yet within the medical system, it is allowed to occur every day without penalty. Babies and parents have to suffer the consequences.

Should not this unnecessary use of cord clamping be classified as complicity in a criminal offence?! Why is a clamp considered an enlightened medical “advance”? Why is it assumed to be a better way, to control (through interference) the sequence of the transition steps necessary to enable the new-born baby to safely adapt to its surroundings outside the womb?

The medical profession conditions parents to believe that, “You can trust us. We know what we’re doing and if anything goes wrong, we have the means whereby we can fix it.” Then again, parents like us, believe that when your written birth plan includes the sentence that the cord is not to be clamped or cut in any way, and that is agreed to verbally, that that is what happens.

Straight away, when I realised that the cord clamp had been put on the cord of our first son, I pulled up the cord to remove it. Only to find that it had already been cut beyond the clamp.

It’s not enough to write a birth plan, or even have your wishes supposedly accepted. Don’t ever be beguiled into thinking that what you want is what they want. In a hospital, you’ve got to be prepared to step in and forcefully remind them that they have no right to control that process. Hospitals think what
they want, is what you want. It’s important for parents to stand up to the system, and put them on notice. Until they do, the system will amble on knowing that they have control by default because everyone stayed silent.

Yes, some in the system consider parents who have convictions based on good facts, a threat. In 1984, management of childbirth in general was far too aggressive for us. I’m not confident that all that much has changed, from the stories I hear told today.

The aggressive management of our first son’s birth and neonatal care, and our anger at the incident of the early cord clamping, were the reasons why we had a home birth when our second son was born and when Joan Donley was the midwife and John Hilton the doctor. They understood why we felt betrayed the first time, and they believed the importance and value of a baby’s physiological cord closure design. This time, we knew that we wouldn’t have to fight for what we wanted, because both doctor and midwife really wanted what we wanted so much.

Obviously, that is the way it should be. Having to beg or “fight” for what you want during labour and what you know is best only creates tension and a toxic atmosphere which in itself has the potential to ruin the wonderful natural progression of a birth.

We know!

We’ve been there and done that!
This section provides an introduction to the human umbilical cord and its development, composition and function in supporting the growth of the fetus in utero. The umbilical cord is formed by the fifth week of pregnancy and therefore some of the text makes reference to the embryonic phase of fetal development in the early weeks of pregnancy. A glossary has been included at the end of this section to aid understanding and support your learning.

The umbilical cord - function, composition and development

The human umbilical cord, which is also sometimes referred to as the funiculus umbilicalis or birth cord, connects the developing embryo/fetus to the placenta and literally acts as a ‘life line’ supplying the fetus with oxygen and nutrients that support its growth and development throughout the duration of pregnancy.

The umbilical cord originates from the same zygote as the fetus, comprising remnants of the yolk sac and allantois. It develops during the fifth week of pregnancy and replaces the yolk sac as the provider of the nutrients that are required by the developing embryo/fetus. The exterior surface of the cord is dull white in colour and moist, and normally comprises two umbilical arteries and one umbilical vein which are continuous with the blood vessels in the chorionic vili of the placenta. These vessels are encased in a protective, gelatinous substance known as Wharton’s jelly (a form of connective tissue), which is covered by amnion (Tiran 2003, Cunningham et al 2005, [Fig 1]).

In some cases, examination of the umbilical cord will reveal that there are only two blood vessels present - one vein and one artery. This is an important observation and can be suggestive of congenital anomalies in up to 25% of pregnancies (Hill et al 2001, Cunningham et al 2005); however, it may also prove to be an entirely harmless characteristic.

The umbilical cord is attached to the placenta which transfers oxygen, nutrients and waste products, such as carbon dioxide (CO₂) to and from the maternal blood circulatory system without any direct contact between fetal and maternal blood. The blood vessels in the umbilical cord operate differently from what would normally be expected, with the umbilical vein providing the fetus with a supply of oxygenated blood and nutrients (which it carries to the fetal heart), and the umbilical arteries carrying away the deoxygenated and nutrient-depleted blood [Fig 2]. The only other example of this within human physiology occurs with the pulmonary veins and arteries which connect the lungs to the heart.

In the full-term healthy neonate the cord has a spiral twist and is normally around 50-60 cm in length, with a diameter of approximately 1-2 centimetres (although this diameter reduces significantly once the cord inserts itself into the
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Fig. 2

Uterus
Placenta

From mother
To mother

Waste products from baby to placenta
Nutrients from placenta to baby

The umbilical cord and fetal circulation

The umbilical cord enters the developing fetus through the lower abdominal wall, at the level which, following cord separation, becomes the umbilicus or navel. Once inside the fetus, the umbilical vein continues towards the transverse fissure on the visceral surface of the liver (ie where the portal vein and hepatic artery enter and the hepatic ducts leave). At this point it separates into two branches; the first joins with the hepatic portal vein, connecting to its left branch. The other, which is known as the ductus venosus, allows the majority of the incoming blood (around 80% of blood volume) to bypass the liver and flow via the left hepatic vein into the inferior vena cava, which carries blood towards the fetal heart. The two umbilical arteries branch from the internal iliac arteries, passing on each side of the urinary bladder of the fetus before joining the umbilical cord.

fetal surface of the placenta). The length of the umbilical cord enables the baby to pass down the birth canal and deliver vaginally without any traction being applied to the placenta. Where the umbilical cord is of an above average length, although not of clinical significance, there is an increased risk that it could become wrapped around the fetal body/neck, prolapse, or become knotted (known as a true knot). A ‘true knot’ results from active fetal movements, where the fetus moves through a loop of its cord, so that it literally forms a knot, which can be clearly seen on examination of the cord at birth. The obstetric concern where the cord becomes knotted or is compressed, relates to the potential for the blood vessels to become blocked and deprive the fetus of sufficient oxygen in utero, especially during labour and birth (Collins & Collins 2000, Sornes 2000, Cunningham et al 2005, Vance 2009). By contrast, a ‘false knot’ is caused by varicosities of the umbilical vessels and/or insignificant lumps of Wharton’s jelly that cause additional twists and protrusions on the surface of the cord.

A cord may also be distinctly short, or relatively shortened due to the extent of spiralling present; a cord that is less than 30 cm in length is considered to be abnormally short (Cunningham et al 2005). Where this is the case, it can be associated with fetal growth restriction, congenital malformations, early separation of the placenta from the uterine wall, fetal distress and in the worst case, fetal death (Krakowiak et al 2004).
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Umbilical cord insertion
In the majority of cases the umbilical cord is inserted at, or close to, the centre of the fetal surface of the placenta. However, abnormalities in the development and site of insertion of the umbilical cord can cause problems which have the potential to affect maternal and fetal health and well-being (Collins & Collins 2000, Cunningham et al 2005, Vance 2009).

Battledore insertion — this is where the cord is attached at the very edge of the placenta and can separate during delivery of the placenta and membranes by controlled cord traction (CCT).

Velamentous insertion — in around 1% of cases, the cord originates in membranes that are distanced from the placental margin, so the cord vessels run through the membranes to reach the placenta. There is a danger that spontaneous rupture of the membranes (SRM) can be accompanied by tearing of a cord vessel which will lead to severe haemorrhage and fetal exanguination (Fung & Lau 1998, Cunningham et al 2005).

Vasa praevia — this is associated with velamentous insertion where some of the fetal blood vessels in the membranes lie across the cervical os below the fetal presenting part. These are at risk of rupture when the fetal membranes supporting them rupture (Cunningham et al 2005).

Susceptibility of the umbilical cord
The umbilical cord fulfils a vital role in pregnancy (Ferguson & Dodson 2009), but because of its inherent characteristics it is susceptible to entanglement, prolapse, compression and occlusion. While some events tend to be of less concern, others can have serious implications for both short and longer-term perinatal morbidity and mortality.

- Nuchal cord — occurs in around 20-30% of births (Mercer et al 2005); this is where the cord is wrapped 360° around the baby's neck. A tight nuchal cord is associated with some short term fetal morbidity (Reed et al 2009); however, it is an uncommon cause of antepartum fetal mortality or neurological morbidity (Clapp et al 2003).

Physiological changes post birth
Soon after the baby's birth, the exposure of the umbilical cord to the 'outside' birth environment and the associated drop in temperature, instigates a physiological process which causes the Wharton's jelly to swell and compress the blood vessels buried within it. This process creates a natural 'physiological' clamping effect that curbs the flow of blood, and, where it is allowed to continue without intervention, will take between 5-20 minutes to completely halt umbilical blood flow. However, where birth takes place during immersion in warm water, the temperature of the water can be equal to inside the maternal body and so normal pulsation of the umbilical cord may be felt for five minutes or longer.

Following the baby's birth, the umbilical vein and ductus venous seal themselves and degenerate down into fibrous remnants known as the round ligament of the liver and the
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Ligamentum venosum. Similarly, part of each umbilical artery closes up and degenerates down into what are referred to as the medical umbilical ligaments; the remaining sections however, are retained to form part of the neonatal blood circulatory system.

Artificial clamping of the cord

Modern obstetric and midwifery practice has favoured active management of the third stage of labour for decades, i.e. early clamping/cutting of the umbilical cord, administration of a uterotonic drug and controlled cord traction (CCT) (NCCWCH 2007). Clamping of the cord can be undertaken as early as one minute post birth, or even sooner with studies demonstrating that this procedure is frequently undertaken within 10-30 seconds of birth (Airey et al 2008). Similarly, immediate clamping/cutting of the cord is often necessary in true obstetric emergency situations and/or prematurity, where neonatal resuscitation needs to be instigated without delay. However, as an alternative to the usual ‘cut and run’ approach, cord milking is suggested as an effective method of facilitating the flow of blood from the placenta to the baby. This is where the cord is milked two to four times in the direction from the woman’s introitus (placental end) towards the infant’s umbilicus. Nine clinical studies involving 746 term infants, demonstrated that cord milking was associated with a benign acceleration of blood and red cells transfer to the infant. These infants were also found to have significantly higher haematocrit and haemoglobin levels (Mercer & Erikson-Owens 2010). Similarly, a study of 24-28 week preterm infants found that cord milking and the resulting increased circulatory volume was associated with increased blood pressure and improved organ perfusion. Preterm infants who received cord milking at birth were also found to require significantly less red blood cell transfusions and circulatory and respiratory support (Hosono et al 2008).

However, women with uncomplicated pregnancies may choose a more natural approach and might request to have this procedure delayed by five minutes or longer, or elect to have artificial clamping omitted altogether in favour of physiological closure, as described earlier. Delayed clamping and the associated health implications have been discussed widely and continue to be a topic of debate (Mercer et al 2006, Selkirk et al 2008). A meta-analysis has shown that delayed clamping of the umbilical cord in term neonates, for at least two minutes post birth, improved the haematocrit (packed cell volume (PCV) - the percentage of blood cells to plasma), the iron status quantified by ferritin concentration (the iron-apoferritin complex; one of the forms in which iron is stored in the human body) (Tiran 2003) and stored iron, and reduced the risk of anaemia (relative risk, 0.53; 95% CI, 0.40-0.70). These benefits to the newborn were found to extend into infancy and although there was an increased incidence of polycythemia in infants who had delayed cord clamping, this appeared to be harmless (Hutton & Hassan 2007).

Similarly, a Cochrane Review has found that delayed umbilical cord clamping is associated with higher neonatal haemoglobin measurements at two months postpartum, although this effect did not persist beyond six months of age. The review also showed a statistically increased risk of neonatal jaundice requiring phototherapy where cord clamping was delayed in excess of 60 seconds post birth. However, assessment of clinical jaundice that was not treated by phototherapy failed to show any significant difference between delayed and early cord clamping practices (McDonald & Middleton 2008).

‘Lotus birth’/ Umbilical non-severance

This refers to the practice whereby the umbilical cord is not severed at birth and is left entirely intact and attached to the placenta. The cord is left to dry naturally and tends to separate and fall off between the third to tenth day postpartum, leaving a healed umbilicus (Crowther 2006). The use of oils, herbs and salt to help keep the placenta sweet smelling and to help it desiccate are recommended (Buckley 2003 & 2006).
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Cutting the cord

Where clamping of the umbilical cord is undertaken, this is then followed by cutting. This does not inflict any pain on the neonate because there are no nerves within the cord itself. The gelatinous quality of the Wharton’s jelly however, means the texture of the cord has a thick, tough sinewy quality that requires suitably sharp cord scissors [Fig 3] to enable it to be severed effectively. Where the umbilical cord is cut after it has ceased pulsating (usually 5-20 minutes post birth), there should be no significant loss of cord blood (venous or arterial) associated with the procedure.

![Fig. 3](image)

Once the cord has been clamped and cut, the neonate is left with a plastic clip in situ which allows the compressed section of the cord to dry out and seal. This should always be assessed to ensure that the cord vessels have been effectively ligated between the teeth of the cord clamp and that there is no active bleeding (Seilkirk et al 2008).

The remaining umbilical stump becomes dry and following a process of aseptic necrosis, normally separates and falls off around 7-10 days postpartum (Tiran 2003). Colonisation of the umbilical stump and surrounding skin by non-pathogenic bacteria is common, but there is no evidence to support the topical application of antiseptic preparations (Seilkirk et al 2008). Parents should be advised to maintain good hand hygiene and ensure minimal handling of the cord; where cleaning is required (in the event of soiling), this should be undertaken using plain water and cotton wool, after which the cord should be dried carefully (Trotter 2003 & 2009).

Inflammation of the skin surface around the cord and/or an offensive discharge may be an indication of infection, which should be investigated (swabbed) and treated as per local Trust guidelines. Unexplained spontaneous bleeding from the umbilicus can be suggestive of vitamin K deficiency bleeding (VDD), formerly known as haemorrhagic disease of the newborn (HDN) (Sutor 2003) and any suspected cases should be appropriately referred.

Glossary

**Allantois** – a membranous sac that develops from the posterior part of the embryonic alimentary canal and is important in the formation of the umbilical cord and placenta.

**Blastocyst** – this is an early pregnancy approximately one week following conception.

**Chorion** – the outer of the two membranes that enclose the fetus in utero; the inner membrane is known as the Amnion.

**Chorion frondosum** – refers to the part of the chorion that is covered by villi during the early weeks of development before the placenta has formed.

**Chorionic villi** – minuscule finger-like projections emerging from the trophoblast and continuing into the chorion frondosum.

**Conception** – the onset of pregnancy following fertilisation of the ovum by a spermatozoon that produces a viable zygote.

**Embryo** – refers to the first eight weeks following conception; after this time, the term fetus is used.

**Ovum** – the female reproductive cell or ‘egg’.

**Materna** – the afterbirth.

**Segmentation** – refers to the division of the fertilised ovum into two cells, then four etc.

**Spermatozoa** – the male reproductive cell or ‘sperm’.

**Trophoblast** – the outer covering of the blastocyst that the placenta and chorion develop from.

**Yolk sac** – this is one of the two spaces that occurs within the inner cell mass of the trophoblast; the second is the amniotic cavity.

**Zygote** – this is the fertilised ovum prior to segmentation/cell division.
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References


Additional reading


