The cord clamp: A monument to stupidity.

Fourth Draft.

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 midwive'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.

Some articles consider "***early cord clamping***" to be around 1 minute.

Delayed cord clamping: This varies according from one article which considered "delayed" cord clamping to be at 30 seconds, to other 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.

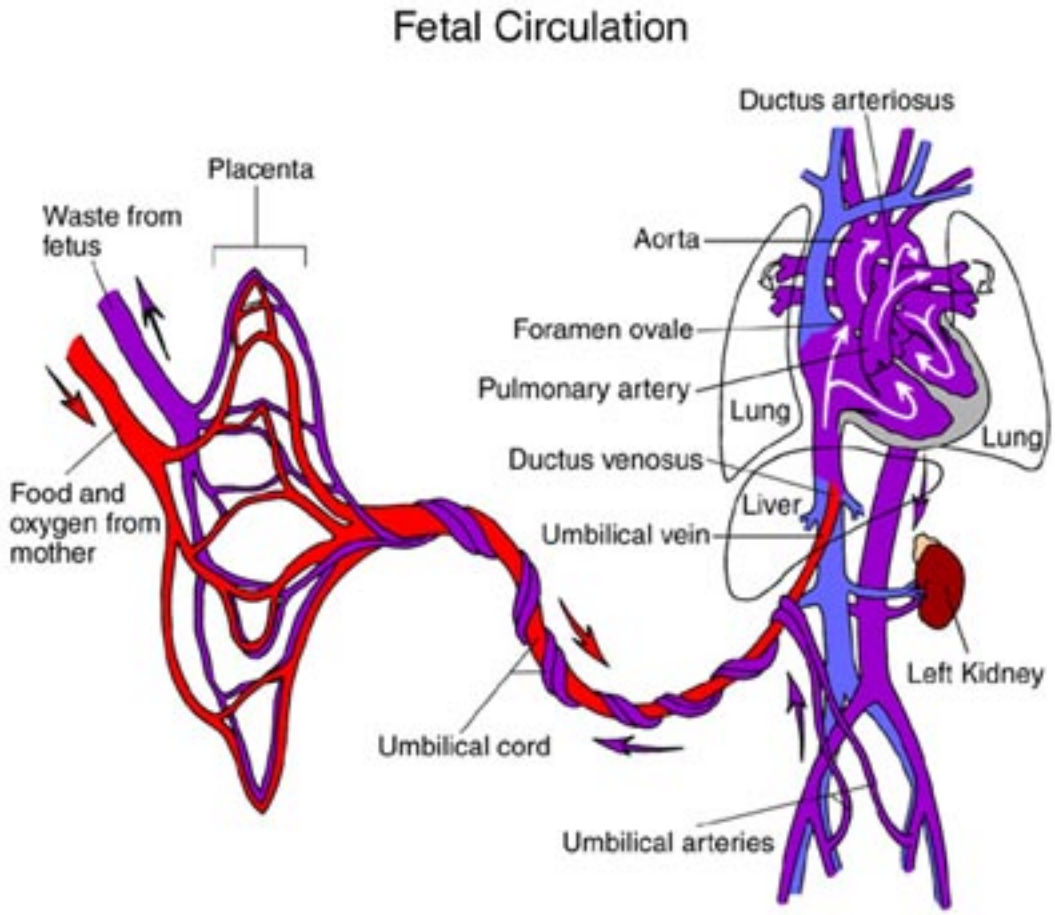
Physiological (normal) cord closure: This is when the baby clamps the umbilical vein and arteries at various locations inside its own body after an optimal blood volume has been transfused into the baby. However, you won't see this term used in many studies, because very few randomised trials actively describe a baby closing its own cord. 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 say[1], "***Man is the only mammal to routinely injure its newborns with a cord clamp***."

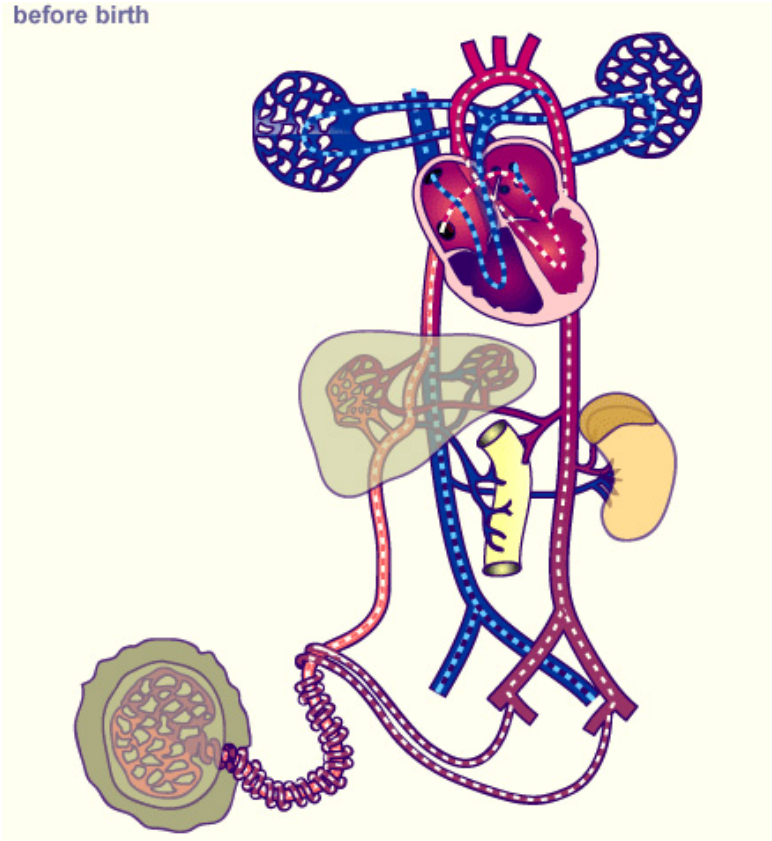


(A mother's blood does not mix with a baby's blood, in case the diagram above is confusing. There is a membrane which separates the two.) What happens between the head crowning, through the first five

[1] http://www.bmj.com/cgi/eletters/335/7615/312#175640 George Morley has a lot of very valuable medical material on his website at www.cordclamping.com

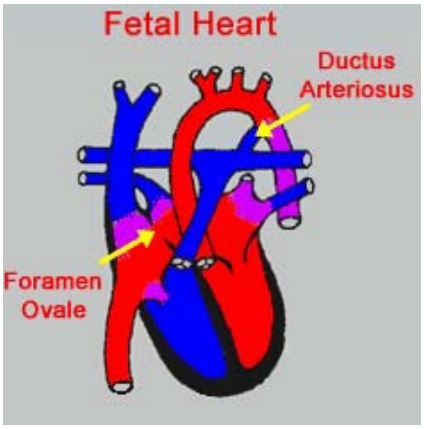
to ten minutes of life, with the blood in the placenta and cord, and why? Below, red blood is highly oxygenated, and blue blood has much less oxygen in it.

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 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.

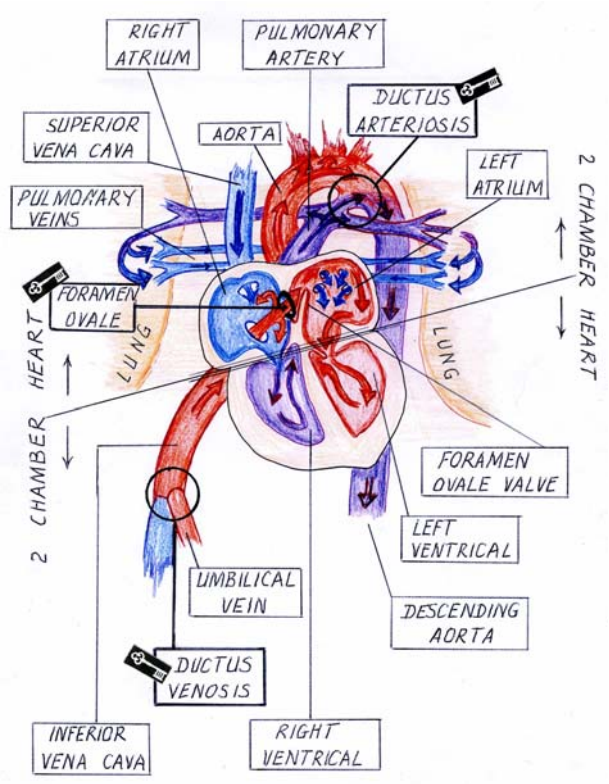
Blood from the brain ("blue") comes down the superior vena cava and is pulled into the right atrium at the same time as "red" blood is pumped through the foramen ovale. A small amount of "red" blood from the placenta is pulled down with "blue" blood from the upper body. This blood goes down into the 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 arteriosis 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.



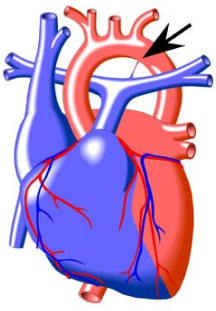
At the moment of birth a normal newborn is circulating a mixture of "red" and "blue" blood. The lips and tongue of a healthy newborn at birth is the same pinkish purple colour as the foetus has been for nine months in utero.

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 smoothes 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

Closed Ductus Arteriosus



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

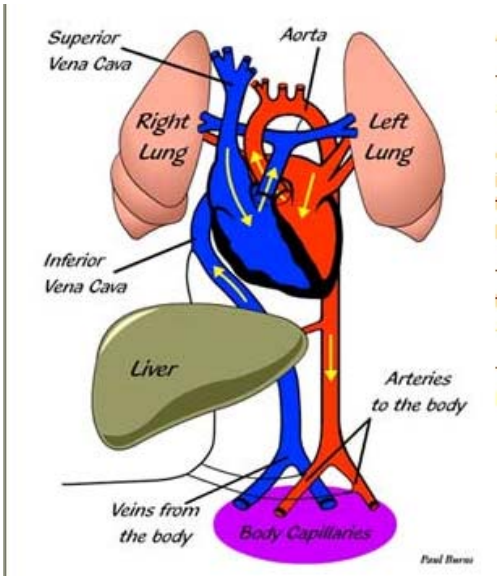
[2] Mercer 2001 J midwifery and women's health, figure 1 quoting Yao Lancet 69) Judith Mercer has a very valuable website with most of her articles on it, which can be found at http://www.cordclamping.info/publications/publications.htm

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.

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!

> ***"Delayed cord clamping to facilitate placental transfusion at birth offers an alternative to medication administration during resuscitation by avoiding hypovolaemia."***[3]

However, Wyllie only considers that to be around 20 – 40 mls! What does that mean? 10 second delay? 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.

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[3] Wyllie, J. 2008. "The role of resuscitation drugs and placental transfusion in the delivery room management of newborn infants." *Semin Fetal Neonatal Med*. 2008 Dec;13(6):416-23. Epub 2008 May 27. PMID: 18508418.

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.

At birth babies are born cyanotic[4], with the arterial oxygen sats being around 60%. In a study where very aggressively managed babies were clamped at birth, it took 5 minutes to for the baby to reach 80% sats and almost 10 minutes to read 90% oxygen sats. Caesarian babies had lower sats and took longer to stabilise. I can find no study looking at unclamped babies as a comparison.

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-colostral 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.

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[4] PMID: 16737866.

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.

In discussing immediate cord clamping, one[5] medical article says:

"… this practice truncates the normal redistribution of blood from the placenta to the newly born infant… immediate clamping of the umbilical cord results in relative hypovolaemia of the newly born infant. Conversely, facilitation of the physiological placental transfusion promotes euvolaemia and reversal of bradycardia with positive-pressure ventilation only. In theory, facilitation of placenta transfusion might reduce the need for exogenous fluid administration; either during delivery room resuscitation or during later stabilization."

What a novel idea. Allowing the placenta to do what it is designed to do. 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)hypoxia and anaemia, requiring blood transfusions. Most of the articles I read on neonatal encephalopathy went to great pains never to mention cord cutting or anoxia at birth, but to always pin the problem on something intrinsic in the labour itself[6]:

"The consequences of interruption of placental blood flow that occurs during labor, particularly when severe or prolonged can affect cerebral and system function." Other articles mention umbilical cord prolapse or mysterious baby heart going out of pattern… or postnatal (after birth) event, which has nothing to do with the actions of the staff. Professor George Morley[7] is the only obstetrician to come right out and state in plain words, that in his opinion, immediate cord clamping after perfectly normal labour, is behind a huge amount of all 'mental retardation' problems.

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[5] Wyllie, J. 2008. "The role of resuscitation drugs and placental transfusion in the delivery room management of newborn infants." *Semin Fetal Neonatal Med*. 2008 Dec;13(6):416-23. Epub 2008 May 27. PMID: 18508418.
[6] PMID: 18501692
[7] www.cordclamping.com

This issue will never be resolved until it becomes mandatory to specify in record exactly when a cord is clamped, and when and how breathing is established at birth. That information is never written into the records now.

In spite of medical articles denying that aggressive birth management using immediate cord clamping etc, has anything to do with brain damage from hypoxia, or brain haemorrhages, we read[8]:

"A recent randomized trial in brain hemodynamics demonstrated that delayed clamping (by 60 – 90 sec) improved cerebral oxygenation in the first 24 hours of life.. delaying cord clamping by 30 – 120 s seems to be associated with a lesser need for transfusion and less intraventricular haemorrhage."

Maybe if the babies had had all their blood, there would be no hypoxia, no need for transfusions, and no haemorrhages in the brain.

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 lack of oxygen to the brain, lower blood pressure, sudden redirection of blood flow with a whole cascade of worsening events. 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 breath 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.

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[8] Zaramella, P. et al 2008 "Early versus late cord clamping: Effects on peripheral blood flow and cardiac function in term infants." *Early Hum Dev.* Mar;84(3):195-200. PMID: 17513072.

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[9] (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.

[9] Mercer J Med Hyp 2009.

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 with no 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.

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 polycythaemia, 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: [10]

"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

[10] (Zoonomia, volume 11 page 321.)

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.

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", according to them. They want to harvest the stem cells, which are specialised cells with genetic blueprints which when taken to parts of the body which are injured or not working correctly, and give the cellular DNA instructions as to how to self-repair. Cord blood banks are saying, why not pay us to keep your baby's cord blood, it in case your baby gets cancer or something and you need all that waste? Frankly, all that "waste" should have gone into the baby at birth, to help the baby establish the best possible life. IF…

there is any left over, then maybe it can be harvested. (Perhaps one day, they will develop techniques to spin out static intracellular blood from the placenta and use that instead.

But first, the medical profession has to accept that what is now considered “normal medical practice” practice of immediate cord cutting, 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” was first used.

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.

Why is a clamp considered an enlightened medical “advance”? Why is a clamp assumed to be a better way, to control (through interference) the sequence of the transition steps to better 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.” The problem is, you can’t “fix” any serious damaged created by robbing a baby of blood and stem cells. If Cerebral Palsy is the result of that, the baby will live with that for the rest of it’s life.

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 will happen.

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. In a hospital, you’ve got to be prepared to step in and remind them that they have no right to control that process against your wishes. 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. Your husband, or someone there, who knows your wishes, needs to watch the point of birth like a hawk, because it’s that important. Silent observation should be all that’s necessary, but if a clamp comes out against your wishes, stop that staff member, and remind them that that will be considered assault. Until hospitals realise that parents mean business, the system will amble on knowing that they have control by default, because everyone stayed silent.

It’s a tragedy that 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 it has changed for the better, 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!

But we have to clamp the cord!!!

"Otherwise you will haemorrhage after the baby is born, and die!"

Second draft.

This will be the response from some medical people whose midwifery skills are primarily what they call "active management" (interference) and who don't understand either the physiological processes of the baby or mother well enough to feel confident to observe and implement appropriate passive management.

Haemorrhage was also uppermost in my head when I was pregnant with our second son, for three reasons.

Much of classical literature read to us in high school had women labouring in bed, and dying of haemorrhages, though interestingly enough, in our family tree from 1500, no deaths in childbirth were from haemorrhage. All were from puerperal fever, caused by doctors refusing to wash their hands.

After the fiasco with Ian's birth, and the damage caused by manual removal of the placenta, retained bits and rebound bleeding, I was concerned, because David's placenta was in exactly the same place. After Joan Donley and Dr John Hilton had reviewed my records, they both agreed that there was a chance that I would haemorrhage.

Our solution to this was two-fold. While we had a home birth with David, we rented a flat just down the road from Waitakere hospital where the doctor also did births. If anything went wrong, a quick transfer would be no problem. When I was 32 weeks pregnant, my haemoglobin was 11.2. Joan said, "I want a better buffer than 11.2 gives me", so I spent the last 8 weeks eating as much vitamin K and iron rich foods as possible. The week before I went into labour, my haemoglobin was 14.4.

I got David down in a supported squat, but as his head crowned I got thigh cramp, so lay back on some pillows. His head came out with no tearing, but then, the nuchal cord had to be unwrapped twice from around his neck and tucked back into the vagina to keep it warm. However, he also had shoulder dystocia, which proved problematic no matter which way Joan tried to shift his shoulders. Joan was getting concerned as two contractions went by, because he was a dusky shade, and trying to breathe, so I took a deep breath, let most of it out, and used my diaphragm and every gymnastic stomach muscle I had. He literally shot out like a cork out of a bottle, telling the world about it, full volume. He was put on my stomach, where he promptly covered me with wee and meconium. He stopped crying just as suddenly, and started looking around. I turned him over and looked at his large brown eyes. I backed up a bit and put him to the breast, which he took straight away, and got stuck in.

I couldn't help but notice how much pinker he was looking that Ian had ever got after birth. After about 20 minutes, there was more uterine pressure, and the placenta came out by itself. It was much larger than Ian's had been and was complete. And yes, I haemorrhaged. How much, I'm not sure. When Dr Hilton filled in the sheet he said to me, "I'll put 500 mls, so that you can have another home birth."

However, a blood test 5 days later showed my haemoglobin to be 10.4, so I suspect it was more than 500 mls!

Fact: haemorrhages can, and do happen. Fact: haemorrhages are one of the commonest causes of maternal deaths in the world. Fact: haemorrhages happen for many reasons. Anaemia in pregnancy is one very preventable reason. Every sensible mother will do what she can to make sure that she has as high a haemoglobin level at the end of pregnancy as possible. The doctor might say that because there is extra fluid in a pregnant woman's blood, a low haemoglobin doesn't matter, but I think it does. By sticking to good dietary advice, it's easy to do.

Another fact is that haemorrhage in subsequent babies can be cause by damage to the uterus wall from bad placenta removal the first time and a dilation and curette to get out the retained bits.

On of the problems when discussing haemorrhage, is that the medical profession can't agree on what a haemorrhage is. A variety of definitions abound. Pritchard[1] in 1962 defined normal blood loss as 500 cc for vaginal births and 1000 cc for caesarean births. WHO defined a haemorrhage as anything greater than 500 cc in the 24 hours after birth. Another proposed definition is a 10% decrease in haemoglobin. Combs in 1991 suggested a clinical definition of "need for blood transfusion". In 2008[2], it was suggested that a haemorrhage ***"may be best defined as excessive bleeding that makes the patient symptomatic (light headedness, vertigo, syncope) and/or results in signs of hypovolemia, hypotension, tachycardia or oliguria)".*** The only symptom I had was light headedness. According to this same article, blood loss exceeding 500 cc complicated up to 18% of all deliveries, and a blood loss of 1000 cc is estimated to complicate 1 – 5%.

But here's an interesting comment: ***"Despite significant improvements in surgical and medical techniques, and improved access to care, multiple countries within the developed world have reported an increase in rates of obstetric hemorrhage."***

Question. Is the increase in haemorrhage partly as a result of the current widespread practice of augmenting labour with Syntocinon, immediate clamping, more injected Syntocinon? When you read, ***Elimination of routine episiotomy is a simple intervention that has been demonstrated to result in a decreased blood loss at the time of delivery"*** you also have to ask what else causes bleeding. And since when is NOT doing an episiotomy, an "intervention"?

Devine also says, ***"The ability of a patient to cope with blood loss depends on multiple factors. Her previous health is critical. The ability to tolerate excessive blood loss is compromised in the setting of pre-existing anemia… because of increased blood volume of pregnancy, most patients can tolerate blood loss up to 1500 cc, provided they are in good health."***

Standard medical practice according to Devine, would appear to be active management of labour with cord clamping at 1 minute, and then a syntocinon injection, stomach massage and controlled traction

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[1] Pritchard J.A. et al. 1962. *"Blood volume changes in pregnancy and the puerperium."* Am J Obstet 84:1271-1282 (cited in Devine, but not traceable.)

[2] Devine, P.C. 2008. "Obstetric Hemorrhage." *Semin Perinatol* Apr;33(2):76-81. PMID: 19324235

while waiting for the placenta to separate. It's not a protocol I agree with, but they say it reduces post partum haemorrhage by 40%.

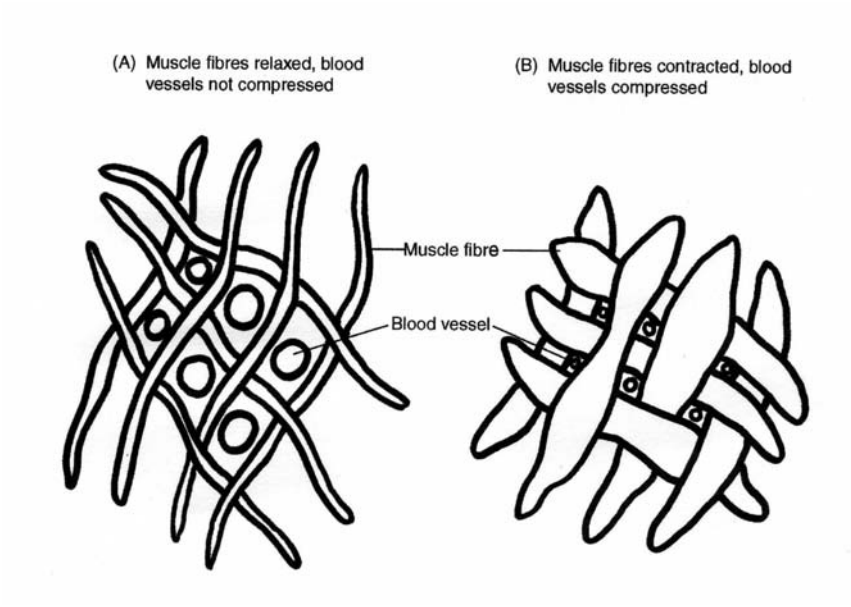
If you chose not to have active management, and if, as I did, you do haemorrhage, the most important thing to do is be prepared. Have plenty of good food prepared and ready, in the freezer for easy re-heating. Talk to your midwife. The local midwives here recommend a supplement based on molasses which reminds me of the old fashioned molasses mineral supplements farmers used to give cows. As a dairy herd tester in 1974, I would give a jar to the farmer, and ask him to decant me some.

If you are planning to NOT clamp the cord to give your baby the best start, then not only do you have to plan for the possibility of a haemorrhage, it also pays to know what your body is designed to do during third stage labour. Knowledge is power. It means you can prepare yourself really well, you know what is happening, and if you do start to haemorrhage, you will know what it feels like, and at what point intervention is necessary.

So here is what you need to know to know the process, so that you aren't frightened into letting them clamp the cord immediately, and possible compromise both your health and your babies health.

Just as the baby has specific mechanisms which allow it to complete the transition from foetal circulation, to "adult" circulation, the mother also has specific physiological mechanisms which allow her to quickly shut down the many blood vessels, called "uterine sinuses".

In the first and second stage of labour the flesh around the top part of the uterus wall, called the myometrium, has a job to do. This flesh is a mesh of criss-crossing fibres, which look like this:



During pregnancy, and early labour, the fibres are relaxed in order to allow the many blood vessels (uterine sinuses) to supply the placenta with food and oxygen.

In the first and second stage of labour the bulk of the myometrium contracts and retracts, causing extensive thickening of most of the myometrium.

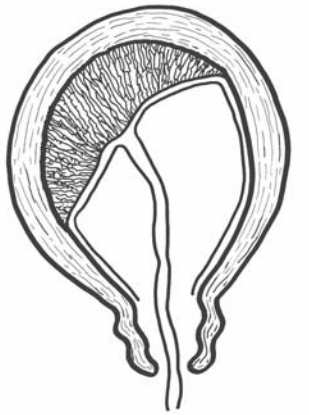
The area of the myometrium next to the placenta does not contract because it needs to continue supplying the placenta with oxygen and nutrients.

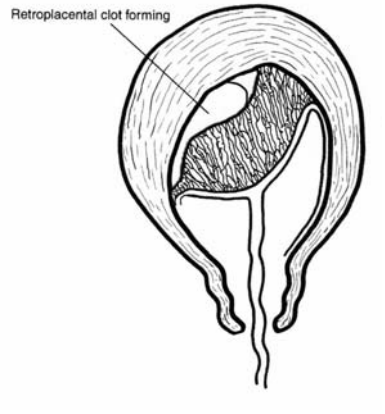
The key to effective myometrium contraction is NOT cutting the cord. The first breath results in a large dump of blood from the placenta into the cord. This blood dump is crucial in triggering separation,

because it reduces the surface area of the placenta slightly, and signals to myometrium which hasn't yet contracted to shut the blood vessels on the uterus wall.

As one midwifery text[3] says:

"Placental separation relies on the ability of the uterus to contract and retract. If the cord is clamped, a counter-resistance is set up in the placenta, preventing the transfer of blood to the baby. The size of the placenta does not reduce as much and this can inhibit contraction and retraction, resulting in a slower separation process. The result of this is …. Means there is a delay in sealing off the torn maternal vessels, resulting in retroplacental clot and increasing the risk of haemorrhage."



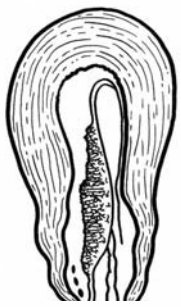


The slight reduction in the placenta size triggers the final stage of myometrium contraction. Imagine a postage stamp, stuck onto a stretched elastic. Imagine the elastic shrinking (the myometrium contracting). Imagine the stamp (placenta) shearing off the myometrium.

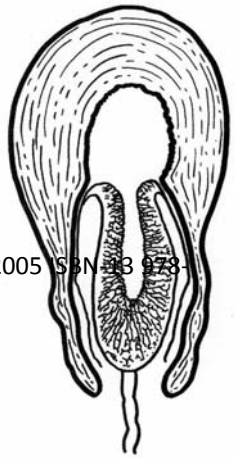
This diagram sequence above is the way a placenta shear off most of the time. It's known as the Schultze method:

See the myometrium really thicking and contracting? See the placenta shearing off?

Rarely, the placenta comes off the uterus this way, which is known as the Matthews Duncan method:



The Schultze method



Sometimes blood loss is greater with the Matthews Duncan removal and membranes are more likely to be left behind.

At the same time as placental separation, the sinuses and damaged edges of all those blood vessels which once supplied the placenta, release thrombokinase (a clotting factor), which converts prothrombin to thrombin. This combines with another clotting aid called fibrinogen to form fibrin, which results in clotting in the sinuses, and also causes any leaked blood to form a small retroplacental clot starts to form behind the placenta. Vitamin K, calcium and other nutrients are required for this bolus clotting effect to happen effectively. ***A mother planning a natural***

[3] "Skills for Midwifery Practice" Ruth Johnson and Wendy Taylor, Churchill Livingstone, 2005 ISBN-13 978-0443101280 Page 266

upright birth and placental delivery, is wise to make sure that her nutrition is A1, so that her body is able to work fast and efficiently.

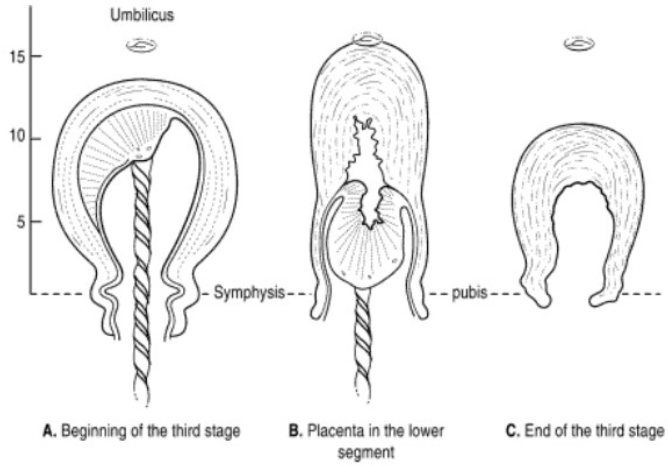
As the placenta moves down to the cervix in the Schultze method, the membranes peel away from the uterus wall and are pulled out with the placenta. The foetal surface comes out first, with any blood trapped behind. With an efficient physiological separation 30 – 60 mls of blood will trickle out. The uterus becomes globular, hard, high, mobile and is palpable.

It's interesting that "Skills for Midwifery practice" points out that cord clamping became popular once labouring women were confined to bed. Clamping was apparently seen as a way to keep bedding cleaner. What the people who started the clamping mania failed to see was that the key to effective placental delivery and minimal blood loss, is an upright delivery.

Gravity is a very useful partner in helping deliver the placenta. Physiologically, the best start to a baby's life, and placenta delivery, is where the mother stays upright. Before "labouring" horizontal in a bed became fashionable in western countries, the most common labouring position was upright. In societies where western medicine and obstetricians have never held sway, birthing is mostly done upright, because mothers automatically prefer to be mobile. Traditionally in western countries, a knotted rope was used from the ceiling of the bedroom, from which the mother hung at various heights according to her own comfort.

The bed became preferred by male midwives, to give the mother "rest". A more likely explanation was to put the mother in a position where she was no longer in control and the obstetrician was.

Upright delivery does two things. Gravity and efficient myometrium contraction, enables the blood transfer to the baby to be much faster than horizontal delivery, where the blood is pushed uphill against gravity.



With a fast efficient blood transfer into the baby, the myometrium contracts down very fast and the retroplacental clot (see left) will be smaller; the myometrium will close faster, and the placenta will be expelled quicker. Result? A nice hard clamped uterus:

The sequence on the left is what you would see from an efficient upright delivery.

In active management (interference) of third stage labour, the minute the baby's shoulders come out, the mother will be injected with Syntocinon, to clamp down the myometrium really fast.

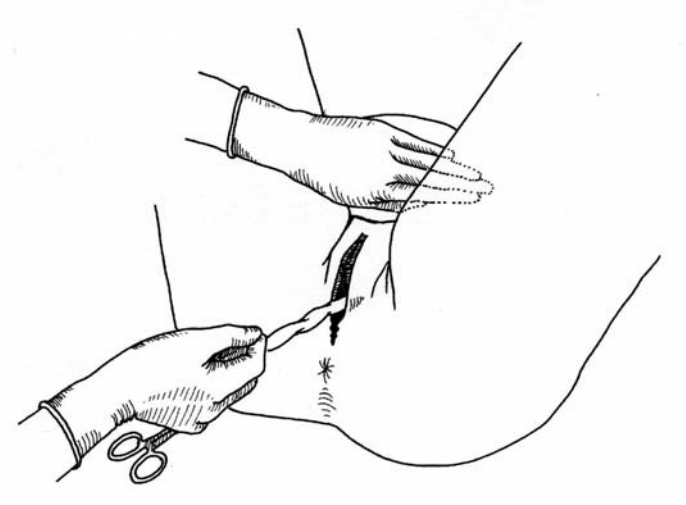
The baby's cord has to be immediately clamped, because if the Syntocinon gets into the baby it causes havoc in the baby's system, closing down its circulation.

As stated above, clamping the cord causes counter resistance from the placenta hindering its shrinkage, which in turn slows separation from the myometrium!

Active management of placenta delivery lying down, using Syntocinon, is counter intuitive to the physiology of good birthing, because in order to prevent a haemorrhage, (often caused in the first place by immediate cord clamping), the baby is deprived of a very large volume of blood. Both the baby and mother suffer from active management.

In a natural, physiological delivery, a mother is upright, either on a birthing stool, or supported squatting, so that the placenta comes out with gravity, combined with the mother using a contraction to push it out. By this time, the baby will have closed off its cord, and will already have been sucking for a few minutes. Sucking results in syntocinon being released from inside the mother, which further strengthens contractions, and speeds up the delivery of the placenta.

However, a mother in a stranded beetle position, will not have such an advantage. "Control" of placental delivery, may have been removed from her. She has no gravity. The syntocinon injection may have had a relaxation rebound effect, so she can't deliver her own placenta, even if she wanted to try to push it out "uphill". She will be told that because she can't do it, her placenta will have to be taken out of her. This process is called "manual traction". The left hand of the person removing the placenta is pushed into the stomach hard to hold back the uterus, so that it doesn't turn inside out and come out with the placenta. The doctor or midwife takes the clamped cord, makes a fist full of it, and slowly pulls out the placenta .



But what the doctor or midwife can't know is how well the placenta shears away from the uterus. There is a danger that pieces of the placenta can still be stuck, and may be left inside the uterus. While they inspect the placenta to see if that's happened, they can't know for sure. How do I know? They missed some bits left in my uterus, yet said it was fine! The bits dropped out later.

Active management is what happens when staff consider the physiological birth process to be defective. In actual fact, ***their actions*** may have rendered the process defective.

This is what happened to me in my first delivery. The message "active management" sent to me, was that the human body isn't capable of doing something it was designed to do. But I knew it was. I felt I had been prevented from doing it myself, because of aggressive active management.

We never put a birth notice in the paper. I just couldn't bring myself to do it. As I read the birth notices, I was struck by how many people put in such things as: "Grateful thanks to the doctor and nurses who delivered our babies." What they don't say is the additional words: "… in their way". Isn't birthing something we as mothers are designed to do ourselves… not have done to us?

In the years that have passed since our children have been born, I have done labour support for first and subsequent babies, for many friends, who wished to achieve natural, normal physiological birth. Not one of my many friends haemorrhaged the way I did. But then, not one of them had a first birth as messed up as mine was. One thing has stood out to me. All those who have achieved natural birth with the co-operation of midwives and doctors, are far more likely to breastfeed, and have a level of self confidence mothering, with an uncluttered ability to make their own well informed choices for their children. ***They*** have given birth, and are at peace with themselves. They don't feel like they have missed out on what is their "birthright". They achieved with their first birth, what I only achieved with my second, despite the complications.

A mother can cope with disappointment, if there is no other option. The end point is always to have a healthy baby and a functioning mother.

But there is a sense in which successful natural childbirth is unique. It has a psychological function. It "makes" a woman.

Delayed clamping causes Jaundice.

First draft.

"You see, Hilary, that's one reason why we clamp the cord immediately. If you leave it, you can get serious jaundice, which cases brain damage." Those words, said to me after I complained about an immediately clamped cord, rang through my head later, as the paediatrian stood there with the blood test results saying that he wanted to put my badly jaundiced baby "under lights". I had four words for him: "Over my dead body". I'd had enough of lies, betrayal, and being treated like an idiot.

So when David was born, I waited with interest to see whether or all that blood from the placenta, resulted in jaundice worse than Ian's. David's jaundice was milder, and cleared up far quicker than Ian's did. So much for the wisdom of the experts.

In 1993 this[1] caught my eye:

"Take neonatal jaundice. Babies with high levels of bilirubin in their blood look yellowish. Physicians put the babies under special lights that alter the bilirubin molecule so it can be excreted. But testing and treating neonatal jaundice exacts an emotional price on mother and baby and the cost is high.

"On night while my wife [Dr susan Niermeyer of the University of Colorado Health Sciences Centre] was cooking dinner, she wondered out loud, 'Why are so many babies jaundiced?' recalls anthropologist John Brett of the University of California at San Francisco.

"I said, 'Probably because they're supposed to be." "

He may be right. Bilirubin acts as a toxic avenger, sweeping up molecules that can damage lung, interesting, eye and other cells.

In older children and adults, enzyme systems do the biochemical housecleaning: in newborns, those systems are not up to it."

The Guardian, reporting the same item, said,

Dr Brett and Dr Niermeyer think this [lights] is at best pointless and at worst harmful. Bilirubin is extremely good at mopping up oxygen-based free radical – highly reactive molecules which can do a lot of damage if allowed to wander around on their own.

[1] "Medical Frontiers", 1993. "Scientists turn to the Flintstone diagnosis" New Zealand Herald, May 26, Section 2, page 4.

…believe that the high bilirubin levels in babies are a finely crafted evolutionary response to being pushed into the oxygen-rich air of the outside world after nine moths of cosseting in the womb. Any brain damage, they say, is a result of other disease that allow bilirubin to get into the brain."

The Economist, in their rendition said all of the above, but after the Guardian bit above, added:

"brain – a place where it has no right to be, and from which it is normally excluded."

If this is enough for you, read no more.

If you want chapter and verse, here goes:

Expanding on this in 1999[2] Dr Niermeyer says, ***"Jaundice of the newborn is one of the most common, complex, well-studied, and misunderstood phenomena in modern pediatric medicine. Despite thousands of studies spanning nearly 50 years, neonatal jaundice, or hyperbilirubinemia remains one of the most baffling and frustrating aspects of health in newborns. We suggest that the primary reason for this ongoing confusion is the inappropriate conceptualization of the role of bilirubin in the newborn period… bilirubin in the healthy newborn ceases to be a confusing and troubling aspect of the newborn period but rather and expected and valued part of the transition to extrauterine life if viewed from the stance of an evolutionary theory of medicine."*** (my emphasis)

If, as Niermeyer says, all newborns have bilirubin levels well above the adults norm, and the majority develop and get rid of visible jaundice within the first few weeks without a problem…, what is the problem?

How did "jaundice" become a "dangerous disease"? The whole issue of brain damage (kernicterus) was first concentrated on in the 50's with regard to Rh incompatibility, (probably caused by immediate cord clamping in Rh incompatible babies) and as part of that, the medical profession attempted to define what was normal. The guidelines which determined "sickness" in normal physiological jaundice were extrapolated from poorly controlled studies on a small number of sick babies with erythroblastosis fetalis. Yet there are pitifully few studies trying to figure out the physiological basis of jaundice in a healthy newborn with no underlying illnesses.

There is a huge difference between pathological jaundice, and physiological jaundice.

A baby with jaundice caused by abnormal biochemical processes, will have a skin colour more akin to a yellow green. These disoreder have names which will cross your eyes, like; tyrosinaemia; hypothyroidism; hepatitis syndromes like biliary artesia, cystic fibrosis, choledochal cyst and alpha-1 antitrypsin deficiency; rhesus incompatibility; PKU; Gilbert Syndrome; and G6PD deficiency. Other pathological causes are sepsis, disseminated intravascular coagulation and Crigler-Najjar syndrome.

[2] Trevathan, W. 1999. "Evolutionary Medicine" June 17. ISBN-13: 978-0195103557 (Pages 7 – 23)

Yet people with Crigler-Najjar syndrome maintain bilirubin levels of 19 mg/dL for 50 years or more with no detectable damage to their nervous system.

The skin colour of normal physiological jaundice will appear as a more bronzy sun-tanned look. The original studies which decreed that breastfeeding "caused" jaundice, were done in hospital, in the days when babies were regimented on 3 – 4 hourly feed; it was assumed that mothers didn't have breastmilk until after several days, so babies were given glucose water, and formula, "to give the mother her rest", which of course, further delayed establishment of breastfeeding.

Ironically,(and empirically), when milk comes in fast, like mine does, a well-fed fully breastfed baby can showed exaggerated jaundice colour (and an abundant supply of wet nappies) which indicates that the laying down of good gut flora and plenty of "fluid" has increased the speed of bilirubin clearance. That is a positive physiological sign, which may be interpreted as a "worsening" of "disease".

However, one of the earliest signs of insufficient milk can also be exaggerated jaundice (with very few wet nappies), because the small amount of breast milk doesn't block de-conjugation of bilirubin.

The fact that most mammal species exhibit similar metabolic processes in their newborn, argues against bilirubin as being some aberrant toxic waste just waiting to cause brain damage. Yet that's what the medical profession believes, so they constantly test bilirubin levels, and when they get to a certain point, the baby is blindfolded and put under lights (phototherapy).

Phototherapy came about because of two observations[3]. The first was a "careless accidental exposure of some blood to sunlight which lead to the finding that bilirubin degraded to biliverdin when exposed to light." The second was the astute incidental observation by a nurse (Sister J. Ward, that the skin of jaundiced infants becomes bleached on exposure to sunlight, whereas unexposed skin does not.

Based on these two empirical observations, phototherapy was started. According to Niermeyer, phototherapy is detrimental to both a mother's confidence in herself and bonding. Mothers whose babies are treated seek more medical attention for their babies, and are more likely to stop breastfeeding. The mother becomes uncertain, defers to the system, and considers that her own body (breastfeeding) isn't up to the job.

The why's and what's of jaundice aren't explained to the mother, seemingly, because there is so much difference of opinion on what jaundice is all about, that it's easier to follow protocols, than give mothers some facts.

Babies who do get kernicterus and brain damage, are not thoroughly studied, looking at genetic bases for bilirubin expression, factors that permit bilirubin to cross the blood-brain barrier, underlying liver and metabolic disorders, sepsis … or the impact of immediate cord clamping on a wide range of physiological parameters in the newborn. Studies of jaundice outcomes should be done, but must have

[3] PMID: 11803408

proper controls of babies who closed their own cords, and were successfully appropriately breastfed on demand.

This is not merely and intellectually selfish idea for self-justification. Debate has arisen in medical literature of late, that phototherapy of babies for jaundice is linked with type 1 diabetes, asthma[4] and that children who have been put under lights, develop "considerably higher numbers of common and clinically atypical nevi[5]" (moles). The authors say, ***"In view of the immaturity of the skin and immune system in newborns, intensive neonatal phototherapy could markedly influence melanocytes and nevus developments."***

Is an increased risk of skin cancers, acceptable, if indeed bilirubin is a neonatal anti-oxidant scavenger of critical importance to good survival? You could say, it would be simpler to go back to using the sun, but ironically the phototherapy technicians[6] now decrees that ***"At present there is insufficient evidence to support exposure to sunlight for treating neonatal jaundice."***

If bilirubin is an antioxidant, and an oxygen free radical scavenger, how quickly does it take for a baby to raise its oxygen levels and what is the impact of that in the baby's body?

An immediately clamped newborn baby hasn't yet developed its anti-oxidant enzyme system and in utero is about 45% oxygen saturated. This baby, doesn't reach 90% oxygen saturation until 10 minutes after birth. Caesarean babies take even longer. We don't know what the situation would be for an unclamped baby.

Within 15 minutes this immediately clamped baby is dealing with more than twice the amount of oxygen, in a circulatory system which is being re-educated. This increased oxygen will produce oxygen free-radicals, which could potentially damage protein, lipids and nucleic acids. Babies, both clamped and unclamped, develop jaundice. The enzymes superoxide dismutase, catalase and glutathione peroxidase are at much lower levels in newborns than in adults, and when these enzymes, and other antioxidant systems are at their lowest activity, bilirubin rises rapidly and falls slowly over several weeks.

A recent article[7] summarises this:

"Recently, some authors have suggested that unconjugated bilirubin is physiologically useful and can act as an antioxidant. During the oxidant stress, oxidants such as nitric oxide play an important role in the pathogenesis of human diseases, especially in the neonatal period. Neonates have limited antioxidant protective capacity against the circulating free radicals, and bilirubin is a potent antioxidant cytoprotectant. Increased oxidative stress in neonates may trigger hyperbilirubinemia and high serum bilirubin levels may protect the cells from oxidative damage."

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[4] PMID 18305267
[5] PMID 18536103
[6] PMID: 12697014
[7] PMID 18426853.

As I thought back to Ian, with his wonderfully bronze levels of jaundice which lasted over three weeks, and David's much milder and shorter form, there is no doubt in my mind, that jaundice has nothing to do with when the cord was clamped. To me, it's a programmed coping mechanism.

Many studies[8] have been done recently, which show that late clamping which markedly increases haematocrit, blood volume and tissue oxygen saturation, does not increase hyperbilirubinaemia, and there is no difference in either group.

The circumstance I believe caused Ian's more serious jaundice, were; Syntocinon[9] augmentation; and epidurals (especially bupivacaine which is what I was given). Those are just two amongst quite an array of drugs used during later, which can increase jaundice, supposedly through taking up space on the plasma sites which transport bilirubin to be excreted, leaving it free in the blood. However, I wonder how much "stress" aggressive management created inside him, "requiring" a higher anti-oxidant level of bilirubin, to meet his needs. By comparison, David's birth was a breeze and much more peaceful for both of us.

The study[10] which meshed Niermeyer's thoughts together in my mind, came out in 2004. This should be compulsory reading for neonatologists. In bird, reptiles and amphibians, the less toxic biliverdin is the end point of blood cell breakdown. In mammals, biliverdin is reduced to the seemingly more toxic "bilirubin". Sedlak asks the question, ***"Why have mammals evolved an energetically expensive and apparently unnecessary enzymatic step to converting the relatively innocuous biliverdin to the more toxic bilirubin? Moveover, why would nature develop a system that generates "elevated" bilirubin levels in a high proportion of all neonates?"*** Sedlak points out that looked at from the "why" point of view, biosynthesis of bilirubin does not seem to make sense.

But he said that if bilirubin protects against oxidation of lipids such as linoleic acid and vitamin A and had a greater antioxidant level than vitamin E, then maybe it does. If 10 nanomolar bilirubin can protect cultures from the oxidant stress of 10,000 times higher concentrations of hydrogen peroxide, that's worth having. When a molecule of bilirubin acts as an antioxidant, with biliverdin reductase, it is oxidized to biliverdin. Biliverdin reductase is an abundant and ubiquitous enzyme with a high turnover rate, so recycling biliverdin and bilirubin around and around, and using it as a cytoprotectant and antioxidant which is gradually degraded and later excreted, would ***"represent an elegant tour de force on the part of nature making use of bilirubin's antioxidant capacity but ensuring that tissues had low endogenous levels of bilirubin."*** He lists the many studies on various conditions in babies and adults where bilirubin protects from a vast array of disorders, and where people with high levels of bilirubin have a survival advantage.

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[8] Zaramella, P. et al 2008 "Early versus late cord clamping: Effects on peripheral blood flow and cardiac function in term infants." *Early Hum Dev.* Mar;84(3):195-200. PMID: 17513072.

[9] PMID: 12552319.

[10] Sedlak, T.W., et al. 2004. "Bilirubin Benefits: Cellular protection by a Biliverdin Reductase Antioxidant Cycle." Pediatrics. Jun;113(6): 1776-82, PMID: 15173506.
http://pediatrics.aappublications.org/cgi/reprint/113/6/1776?ck=nck

He also points out that in babies, serum bilirubin are 100 to 1000 times higher than intracellular values, but that 99% of that is bound to plasma and not available for intracellular action, but would be likely to have a direct therapeutic action in coping with oxidative stimuli wherever blood flows, but also feeding tissues. The tissue bilirubin would most likely function in diseases of specific organs. He also pointed out that scientists knew little about how bilirubin moved in and out of cells.

The whole article argued that bilirubin was a plausible survival strategy, and finished the article with the comment that, ***"uric acid was once regarded solely as a toxic metabolite responsible for gout, whereas it is now increasingly appreciated as an antioxidant. Similarly physiologic antioxidant roles for bilirubin, may detoxify its traditional nefarious reputation."***

So why is it so important to find out just what bilirubin does; why all mammals use it, and why physiological jaundice affects the majority of babies?

If you are going to look at what causes brain injury and such lesions as neonatal encephalopathy, you have to take into consideration that[11]: ***"Since oxygen free radicals are considered important in the genesis of ongoing injury following hypoxia-ischemia, therapies targeting the destruction of oxygen free radical have been developed…. Neuroprotection has only been shown when these agents have been administered several hours before the hypoxic-ischemic insult."***

Is that what bilirubin, the ultimate antioxidant is for? Yet most paediatricians consider physiological jaundice a toxic evil to be removed as fast as possible.

Cord clamping in this context is important, because while cord clamping doesn't affect whether or not a baby gets jaundice, not cord clamping is proven to protect babies from free radical brain damage:

"A recent randomized trial in brain hemodynamics demonstrated that delayed clamping (by 60 – 90 sec) improved cerebral oxygenation in the first 24 hours of life.. delaying cord clamping by 30 – 120 s seems to be associated with a lesser need for transfusion and less intraventricular haemorrhage. [12]***"***

Another study [13] ***says that the additional blood in preterm infants obtained by delayed cord clamping " helps stabilize cerebral blood flow, autoregulation, increase oxygen delivery to vulnerable tissues, prevent ischemia, and cytokine release, and provide additional stem cells to establish adequate immunocompetence". It also prevented late onset sepsis. "Delayed clamping of the umbilical cord… improved oxygen delivery to the tissues in increasing system blood volume. Increased blood volume was advocated to facilitate pulmonary adaptation with decrease for medical interventions particularly mechanical ventilation… this reserve potentially reduces the risk of hypoxic ischemic events to the brain..placentofetal transfusion may be beneficial to reduce the risk of disturbed cerebral***

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[11] PMID 15693398.

[12] Zaramella, P. et al 2008 "Early versus late cord clamping: Effects on peripheral blood flow and cardiac function in term infants." *Early Hum Dev.* Mar;84(3):195-200. PMID: 17513072.

[13] PMID: 17332197.

oxygenation. Our study demonstrates an increased tissue oxygenation in the neonatal brain after delayed cord clamping."

I find it hard to believe that delayed, or no cord clamping, ONLY does that in preterm babies!

And at the BMJ, the debate goes on[14] with many doctors calling for a halt to the barbaric practice of immediate cord clamping.

How could a baby be better set up for survival, than all the blood in the cord and placenta to increase oxygen levels, prevent brain damage etc, with an added dollop of bilirubin to mop up free radicals caused by the required increase in oxygen use.

What a unique system, designed specifically to bridge mammalian babies over that time period when their own antioxidant systems haven't yet got up to speed, and optimum survival of healthy babies without any pathological disease, is the name of the game.

Lights, anyone?

[14] http://www.bmj.com/cgi/eletters/335/7615/312 and http://www.bmj.com/cgi/eletters/334/7602/1027-f