Interpreting Umbilical Cord Blood Gases: Cord Occlusion with Terminal Fetal Bradycardia: Part 1

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There is widespread belief that severe fetal acidosis and resultant hypoxic-ischemic encephalopathy are most often associated with late decelerations and absent variability. In my experience, fetal asphyxia, as reflected in severe derangements in umbilical cord blood gases, is often associated with progressively severe variable decelerations that result in terminal, prolonged fetal bradycardia. Cord compression, especially if the umbilical vein continues to be occluded while umbilical arterial blood flow is restored, impedes return of blood from the placenta to the fetus while maintaining outflow from the fetus to the placenta. This may result in fetal hypovolemia and augment the consequences of decreased fetal cardiac output and ischemia. In this situation, the umbilical blood gases may reflect striking differences between the vein and the artery.

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In addition, ischemia is known to be a much more potent cause of brain injury than hypoxia alone.[1] In experiments with fetal sheep made severely hypoxic for two hours, the added presence of hypotension resulted in marked lactic acidosis and severe brain damage, whereas the presence of severe hypoxia alone did not.[2] Similarly, human fetuses exposed to severe hypoxia *and* concurrent "blood loss" (to the placenta) as typically occurs during terminal bradycardia following cord compression, are more likely to suffer brain damage than when hypoxia occurs alone.

Be aware that variable decelerations associated with a slow return to baseline, a rise in baseline, or absence of variability in the baseline between decelerations, are not reassuring.[3] Cord compression resulting in terminal bradycardia is a worrisome development and a potential harbinger of both fetal acidosis and neurological injury – irrespective of whether the cause of impaired umbilical cord blood flow is stretch as the baby descends into and through the birth canal or by external compression (see Table below). Accordingly, in order to give appropriate emphasis to the importance of impaired cord blood flow, this section contains ten case histories and is the longest section.

Umbilical Blood Flow – Mechanism of Impairment
Stretch
Short cord
A relatively short cord with fundal implantation
Cord around the neck or other structure(s) (functionally short cord)
A true knot in the cord (minimal stretch)
Descent of fetus
Shoulder dystocia* (possible)
Breech delivery with a trapped head (stretch and compression)*
* Sudden stretch may result in umbilical vessel spasm
Compression
Kinking of cord (especially with decreased Wharton's jelly)
A true knot in the cord
Torsion of cord
Entwining of cords (monoamniotic twins)
Hematoma of cord (may also cause vessel spasm)
Cysts of cord
Prolapsed cord (overt or occult)
Breech delivery with a trapped head (stretch and compression)
Stricture of cord
Shoulder dystocia (probable)

Table: Mechanism of impairment of umbilical cord blood flow

Whether or not the umbilical cord becomes stretched is determined by a number of factors: absolute cord length, distance of placental implantation from the cervix, location of cord insertion on the placental disc, functional cord shortening by wrapping around one or more fetal structures (most commonly the neck), true knot in the cord (causing minor functional cord shortening and self-compression), and descent of the fetus into and through the birth canal. Delivery of the head, followed by shoulder dystocia, causes functional cord shortening by the length of the fetal head, a length that may be sufficient to cause vascular occlusion by stretching. Breech delivery with a trapped head also causes a sudden and large functional cord shortening by the distance from the fetal breech to the fetal chin.

Stretching of the umbilical cord occludes the umbilical vessels in much the same way as does the simple Chinese finger puzzle in which a finger from each hand is placed in each end of a woven bamboo cylinder. When one attempts to remove their fingers, the result is a tightening of the woven tube. As the cylinder is stretched, thereby increasing its length, its diameter is decreased, trapping the fingers. Another way to think about this is to realize that the umbilical cord has a fixed volume, i.e., a cross-sectional area multiplied by length. If the umbilical cord is stretched, making it longer, the cross-sectional area must be decreased in order to maintain

a constant volume. It is this decrease in the cross-sectional area that can occlude the umbilical vessels. The fetal sheep tolerates experimental clamping of the umbilical cord without penalty, unless prolonged or very repetitive.[4]

The anatomic relationship between the umbilical vein and the umbilical arteries is quite varied. However, looking at the most common configuration (as depicted in an 1870 drawing by Joseph Hyrtl[5] on the front cover of *Interpreting Umbilical Cord Blood Gases, 2nd ed*[6]), one can see that if the umbilical cord were to be stretched, the more easily compressed vein would come into juxtaposition with the harder to compress arteries in many locations.[7] In some variants, the arteries are not wound around the vein at all, or only minimally so. In theory, certain configurations should be more resistant to venous occlusion by stretch than others.

External compression of the cord is influenced by the thickness of the cord, which in turn is determined by the amount of Wharton's jelly. The umbilical venous muscular coat is thinner than that of the umbilical arteries. Typically, infants of diabetic mothers whose diabetes was not well controlled during pregnancy have relatively thick cords, and infants with intrauterine growth restriction have relatively thin cords. It is easier to compress a thin cord. The presence of oligohydramnios (frequently associated with decreased Wharton's jelly, which is mostly composed of water) also increases the vulnerability of the cord to compression.

In the presence of oligohydramnios, the cord more commonly migrates to a position where it is at increased risk of compression, rather than safely "floating" in amniotic fluid. Classically, external compression of the cord occurs in association with cord prolapse. In this situation, typically, the cord is compressed between the presenting part of the baby and the lower uterine segment. Each subsequent uterine contraction further compresses the cord by wedging the fetus more tightly into the birth canal. External cord compression also occurs in breech presentation with entrapment of the aftercoming head.

Additionally, the cord may be compressed and partially or totally occluded when it doubles back on itself (kinked) or when there is a true knot in the cord. Usually, a true knot remains loose even as the baby descends into the pelvis as the cord is generally not critically short (either anatomically or functionally); however, fetal movement may lead to a "cord accident" by tightening of the knot at any time. This mechanism comes into play more frequently with a "thin" cord with decreased Wharton's jelly. A knot in such a cord is more easily tightened.

Cord compression typically results in variable decelerations. The quick decrease in the FHR is not caused by hypoxemia, but rather is a reflex response to a sudden increase in fetal blood pressure[8] when a major source of blood runoff, the umbilical arteries, is lost by cord compression.

Finally, occlusion of the umbilical arteries by either stretching or external compression becomes easier as fetal arterial pressure falls. As hypoxemia leads to asphyxia, this mechanism further facilitates occlusion of the umbilical arteries.

Case 9: Virtual Total Umbilical Cord Occlusion: A Thought Experiment

The mother was a 21-year-old, gravida 1, para 0, aborta 0, with an intrauterine pregnancy of 39 2/7 weeks' gestation by good dates. The pregnancy was uncomplicated. Membranes were intact. The FHR pattern was entirely normal with the presence of accelerations, absence of decelerations, and moderate variability. In this thought experiment, the umbilical cord was double clamped ("virtually" without entering the uterus). The clamp instantaneously and entirely interrupted all blood flow in the umbilical cord. The fetus was delivered 40 minutes later.

Questions (1-word answers preferred):

1) Describe the fetus, now newborn.

2) Describe the cord blood gases.

Interpretation:

Answers:

1) Dead

2) Normal

Once the umbilical cord is clamped, no blood flows through it. With no ingress of oxygen from the umbilical vein, the fetus will rapidly develop severe respiratory and metabolic acidosis. Over 40 minutes, the fetus will die. Blood gas values in the stagnant blood in the umbilical cord, however, remain approximately the same as they were prior to umbilical cord clamping. As the fetus had a perfectly normal and reassuring FHR tracing prior to being clamped, one would expect the umbilical cord blood gases to be entirely normal. Over time, probably in excess of one hour, the blood gases will degrade.

Total umbilical cord occlusion has been well studied by Myers in the anesthetized term monkey fetus.[9] In the first 90 seconds following umbilical cord clamping, the FHR decreased rapidly along with an abrupt increase in blood pressure due to the sudden increase in peripheral vascular resistance. After the first 15 to 20 seconds following cord clamping, the elevated fetal blood pressure began to decline, precisely at the time when the first oxygen-poor blood reached the myocardium. Then, at approximately 60 seconds following cord clamping, the beginnings of a "major, long-lived augmentation in blood pressure occurred during which both the systolic and the diastolic pressures were restored to values that, 150 seconds into the asphyxia, compared to values present prior to the asphyxia. After this secondary blood pressure increase reached its zenith at three to four minutes, the pressure again declined slowly until, after 12 to 15 minutes of asphyxia, the pressure recorded within the thoracic aorta approached that of the surrounding tissue." At this time, blood flow through all parts of the fetal body ceased.

Interestingly, although the fetal heart stopped performing mechanical work, it continued to register a QRS complex at 60-70 beats per minute, i.e., electromechanical dissociation, or pulseless electrical activity. Blood sampled from the fetal thoracic aorta demonstrated a linear decrease in pH, a linear increase in the base deficit, and an almost linear increase in Pco_2 over 12.5 minutes. The pH declined from 7.29 to 6.81, or approximately 0.038 per minute. The base deficit increased from 2.8 to 17 mEq/L, or approximately 1.1 mEq/L/minute, and the Pco_2 rose from 52 to 132 mmHg or approximately 6.4 mmHg/minute. It is important to note that further linear trends would not be expected to continue as blood flow to the fetal peripheral tissue had ceased, and peripheral lactic acid

would not be fully reflected even in the fetal aorta until circulation had been restored (reperfusion acidosis). Po_2, on the other hand, fell rapidly to low levels following cord occlusion and remained there for the length of the experiment.

> ***" Almost always in terminal fetal bradycardia resulting from cord occlusion, the vein remains occluded while blood flow in the umbilical arteries is restored for varying periods of time."***

Although Meyers' model is quite instructive, it does not represent what actually occurs during the great majority of cases of terminal cord occlusion. I have seen only rare instances in which I thought that umbilical venous and arterial occlusion happened almost simultaneously as a terminal event. Almost always in terminal fetal bradycardia resulting from cord occlusion, the vein remains occluded while blood flow in the umbilical arteries is restored for varying periods of time. This combination is more challenging for the fetus, as progressive hypoxia and varying degrees of hypovolemia occur simultaneously.Key Point

- When umbilical blood vessels are occluded during terminal fetal bradycardia, a blood gas sample taken from either an umbilical vein or artery will only reflect the blood gas status prior to the occlusion. Over time, probably in excess of one hour, the blood gas samples will degrade.

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