Fetal and Placental Indications For Placental Examination: Absence or Minimal Umbilical Cord Twisting and Vascular Coiling

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Cord Twisting

The twist, spiral, helix of the umbilical cord is present as early as six weeks gestation and is well established by nine weeks. Twist direction is determined by looking at the stretched out cord (doesn't matter which way is up) and identifying the upward direction of the twist (see figure). Left twists (sinistral/counter clockwise from the placental aspect/ clockwise from the fetal aspect) outnumber right twists (dextral/clockwise from the placental aspect/counterclockwise from the fetal aspect) seven to one.

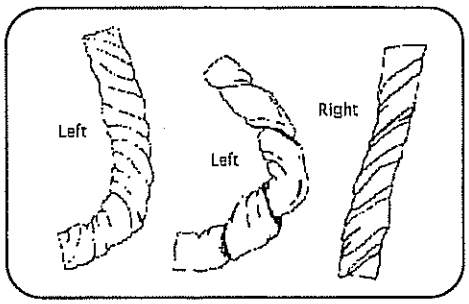
Typically only one twist direction is present in a cord, but both may be present. If a cord doesn't lay straight or appears "kinked," check for a change in twist direction.

Usually there are up to forty twists over the length of the umbilical cord but up to 380 (!) have been described. Their number is established early in pregnancy and increases only slightly in late gestation.

The cause of cord spiraling is unknown, despite much investigation. Because the seven-to-one ratio of left-to-right cord twisting is about the same distribution of "handedness," Lacro et al suggested cord twisting was a result of cerebral organization; they disproved this hypothesis showing there is no correlation between the cord twist direction and the handedness of mother or child. Others have suggested that the twists are governed by the earth's rotational forces (i.e. like the familiar bathtub vortex); this has also been disproved.

Vascular Coiling

Vascular coiling and cord twisting are not the same. According to Dr. Thomas Strong, Perinatologist in Phoenix, Arizona, "Coiling refers to the inherent helical configuration of the umbilical vessels created by the natural process of development. "Twists" refer to the torsion imparted to the entire cord by external forces such as fetal movement. To illustrate, think of a little girl with a braided ponytail - the braiding would represent umbilical coiling. Now imagine that someone comes up behind the girl and twists her ponytail - that would represent umbilical twisting. Once her ponytail is released, the twisting will disappear but the braiding (i.e. the "coiling") will remain. When studying a gross specimen of cord, I hold it vertically in the air at one end so as to allow the freely hanging cord to untwist itself. Then I count the number of coils." (9)



Dr. Strong and colleagues have studied the relationship between cord vascular coiling and neonatal outcome. In the early 1990's, along with Dr. John Elliot and Dr. Tari Rabin, he prospectively investigated whether straight noncoiled umbilical cords identified at delivery were associated with an increased risk of sub-optimal perinatal outcome. Thirty-eight (4%) neonates out of 894 were identified to have noncoiled umbilical cords during the study. Compared to the infants with coiled cords, these infants had a higher incidence of intrauterine death, preterm delivery, repetitive intrapartum fetal heart rate decelerations, operative delivery of fetal distress, and congenital anomalies. At the conclusion of the study, they recommended careful study of the umbilical vascular architecture during ultrasound.

Next, Dr. Strong et al developed the umbilical coiling index (UCI), a simple objective technique to quantitate the degree of umbilical vascular coiling: divide the number of complete vascular coils seen grossly at delivery by the total cord length in centimeters. They found a mean umbilical coiling index of 0.2 coils/centimeter (normal range considered 0.1 - 0.3 coils/cm). At term, most cords measure 55-66 cm in length, so the mean UCI would predict ten-twelve vascular coils per cord, consistent with the findings described for over sixty years that the typical cord has eleven vascular coils. They also again concluded that children born with noncoiled umbilical cords were at higher risk for sub-optimal outcome.

For the UCI to be meaningful, the technique had to be determinable antepartum (i.e. had to be identifiable on ultrasound). In 1992, Dr. Strong et al used ultrasonography to identify vascular cord coiling in utero. They confirmed that the UCI could be determined in utero, again identifying children at higher risk for sub-optimal outcome, suggesting the need for early and careful evaluation of fetal anatomy, fetal well-being, and possible early delivery. Interestingly, they also discovered that 30% of the cords not coiled in early gestation were coiled at follow-up ultrasound in later gestation, with no untoward outcomes in those fetuses.

Dr. Strong et al also observed that fetuses with poorly coiled cords had higher incidences of nuchal cord and more frequently required operative intervention for fetal distress during labor. Noncoiled umbilical vessels alone were not an independent risk factor for trisomy.

Of note, whether the arteries wind around the vein, or the vein around the arteries - both conditions may be seen in the same umbilical cord - is irrelevant.

Etiology of Cord Twisting and Vascular Coiling

Since it has already been proven that cord and vascular coiling are not a function of handedness (cerebral organization) and are not related to the earth's gravitation forces (i.e. analogous to the bathtub vortex), researchers are looking at the structure of Wharton's jelly and the blood vessels themselves, and their relationship with amniotic fluid.

The coiled, three-vessel arrangement of the umbilical cord has been recognized since

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the 16th century. The cord reaches structural and functional maturity relatively early in gestation, and once achieved, the cord configuration is thought to be unchangeable.

Some researchers suggest the incidence of coiled cords and preponderance of left twist are not random, but rather are inherited. Twin studies demonstrate less variation in cord lengths (cord lengths within 2 inches of each other) between monozygotic twins when compared to fraternal twins, leading researchers to conclude the umbilical cord structure is the result of the relationship between events at the cellular and gross morphologic levels. In other words, information at the molecular level is ultimately expressed using available materials, modified by growth and the structural response to external factors.

The asymmetry in size of the umbilical arteries may explain the etiology of cord twist as the result of unequal growth of the two arteries or the rotational torque resulting from the differential blood flow within the differently sized vessels. Researchers point out the fact that the right umbilical artery is usually larger than the left, which supports the observation that left twisting is more common than right twisting. This theory is also supported by the absence of twisting in many cords with only a single umbilical artery (two-vessel cords).

Recent studies put forth two fundamental theories for the etiology of cord twisting. Experimental models suggest the twist is inherent to the cord itself: hollow tubes with walls constructed of fibers with an inherent tendency to twist, when subjected to sufficient intraluminal pressure to induce swelling, exhibit the typical helical twist of the cord. The twist direction of the tubes was opposite to the twist direction of the fibers constituting the walls. Since the umbilical arterial wall is composed largely of smooth muscle that coils around the lumen of the vessel, the helical layer of muscle fibers may be the axis for umbilical coiling. Again, an inherited trait?

The second theory suggests that cord length is related to fetal movement and long cords tend to have more twists, suggesting twist severity may also be related to active or passive rotation by the fetus. This postulation is corroborated by the fact that cord twists are absent in cases where fetal movement has been restricted, such as fetuses fixed in place by amniotic bands, or where the fetus suffered from a neurologic or neuromuscular abnormality. Therefore, a poorly twisted cord or a cord with no twists suggests an inactive fetus, and clinically the question becomes, "why was the fetus inactive?"

Many researchers feel both theories are relevant, implying the inherent cord twist can be impeded by any factor that restricts normal fetal movement.

Straight cords likely result from a number of factors, possibly involving absence of umbilical arterial helical smooth muscle fibers, inadequate intravascular pressure, and/or decreased fetal activity.

Vascular coiling is also thought to be an intrinsic characteristic of the cord vessels, again due to an intrinsic twist in some or all of the constituent fibers of the umbilical cord, possibly within the media of the arteries.

Protective Function

As one author put it, the twisted, three-coiled vessel arrangement of the umbilical cord is likely the product of evolution and confers some advantage over other arrangements at variance with this form in order to ensure adequate placental-fetal blood flow through the umbilical vessels.

During intrauterine life, the umbilical cord is susceptible to many processes with the potential to compromise placental-fetal blood flow. Most such processes are external, against which amniotic fluid may be protective. In fact, amniotic fluid is well recognized for its ability to protect the umbilical cord from compression, as an inverse relationship seems to exist between amniotic fluid volume and frequency of variable fetal heart rate (FHR) decelerations: high amniotic fluid volume - lower frequency of FHR decels, low amniotic fluid volume - higher frequency of FHR decels. However, because amnioinfusion ("saline replenishment" of amniotic fluid volumes) does not always correct FHR decels associated with low amniotic fluid volumes and oligohydramnios does not always result in FHR decels, it has been suggested that additional mechanisms are involved to protect blood flow through the umbilical cord, namely cord twisting and vascular coiling, and Wharton's jelly.

Regardless of its origin, twisting appears to confer rigidity, producing a strong but flexible umbilical cord resistant to entanglement, snarling, and torsion. Consider the similar structure and characteristics of the telephone cord: semi-erect yet flexible and relatively resistant to stretch, compression, and tight knotting. Twisting also gives the cord a larger overall diameter, exposing less of the umbilical vascular surface area to the intrauterine environment, thereby allowing the cord to resist external forces, such as vascular stretching and compression, that might compromise placental-fetal blood flow through the cord. In addition, within the cord, the coiled blood vessels experience less turbulent flow since curved conduits tend to have higher thresholds for the onset of turbulence than do straight ones.

In comparison, less twisted umbilical cords tend to be more flaccid and more prone to prolapse after the extraplacental membranes rupture during labor and delivery.

Wharton's jelly itself, the actual volume of which appears to be related to the osmolality of the amniotic fluid, is also thought to protect placental-fetal blood flow through the cord. It has been observed that coiled cords with adequate Wharton's jelly have been associated with better neonatal outcomes, whereas neonates with reduced Wharton's jelly experience a higher incidence of intrapartum distress.

Data suggests the cord structure may be altered by prolonged, low-grade compression, specifically liquefaction of Wharton's jelly, hence its characterization as a thixotropic gel (a gel that becomes fluid when stirred or shaken and returns to a semisolid state upon standing). This is supported by the observation that knotted cord segments or cord segments entangled with the fetal neck or body tend to have less Wharton's jelly. It has been proposed that the pressure produced by entanglement allows for remodeling of the compressed segment secondary to jelly liquefaction, the extent of which may be a function of the duration and tautness of the entanglement. Functionally, such a change might allow direct pressure upon the umbilical vessels themselves, impeding placental-fetal blood flow through the cord. Some authors suggest the pattern of noncoiled segments of the cord may represent periods of time where significant entanglement or compression occurred.

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The Umbilical Pump

First proposed in the 1950's, studies suggest the coiled umbilical cord may act as a secondary cardiac pump that augments fetal cardiac preload by increasing umbilical venous return from the placenta.

Typically, intravascular pressure within arteries and veins is in-phase (i.e. synchronous, with circumferential distortion of the arteries contributing to the pulse). Research revealed that the systolic pressure pulsations in the arteries and vein of the umbilical cord are not in-phase, that they are in fact asynchronous, paradoxically alternating with one another. This observation was found to be related to longitudinal distortion of the umbilical arteries, which subsequently induced relative negative pressure in the umbilical vein during fetal systole and had the opposite effect during fetal diastole. In other words, the longitudinal distortion induced arterial and venous pressure variations 180 degrees out of phase with each other. The study also discovered this process may be modulated by the density of Wharton's jelly surrounding the umbilical vessels.

This system contains the elements of a peristaltic pump, a mechanical device familiar to engineers but found only in the arteries and vein of the umbilical cord.

Indirect evidence for the umbilical pump includes observations of significantly higher incidence of placental congestion among those with straight umbilical cords and a significant correlation between the umbilical coiling index and the rate of umbilical venous flow.

Interestingly, the umbilical pump may also play a role in the pathophysiology of the twin-twin transfusion syndrome. Assuming monochorionic twins share a common placental reservoir, discrepant degrees of umbilical coiling/differential umbilical pump activities between the two fetuses would create a net flow of blood to the twin with the more densely coiled umbilical cord. However, it remains a mystery why genetically identical twins would have such different degrees of umbilical coiling.

Optimal protection by the umbilical cord of the placental-fetal blood flow through the umbilical arteries and vein is likely a function of umbilical cord twisting, vascular coiling, Wharton's jelly, and amniotic fluid. Moreover, the degree of cord twisting/vascular coiling may impact fetal cardiovascular dynamics via the umbilical pump, which may in turn impact amniotic fluid volume/osmolality, which may in turn affect the volume and elasticity of Wharton's jelly, which may in turn modulate the function of the umbilical pump. In other words, the umbilical cord and its intrauterine environment may represent a complex system of interaction and feedback.

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