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REVIEW

The umbilical coiling index, a review of the literature

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Abstract
Our aim was to review the literature on umbilical cord coiling. Relevant articles in English published between 1966 and 2003 were retrieved by a Medline search and cross-referencing. The normal umbilical cord coiling index (UCI) is 0.17 ($\pm$ 0.009) spirals completed per cm. Abnormal cord coiling, i.e. UCI $<$ 10th centile ($<$ 0.07) or $>$ 90th centile ($>$ 0.30) is associated with adverse pregnancy outcome. Hypocoiling of the cord is associated with increased incidence of fetal demise, intrapartum fetal heart rate decelerations, operative delivery for fetal distress, anatomic-karyotypic abnormalities and chorio-amnionitis. Hypercoiling of the cord is associated with increased incidence of fetal growth restriction, intrapartum fetal heart rate decelerations, vascular thrombosis and cord stenosis. It is not clear whether abnormal coiling is actually a cause of pathology, or merely one of the sequelae, or both. We discuss the theories involving the cause of cord coiling, and the consequences of the degree of cord coiling on blood flow through the umbilical vessels. In the future ultrasonographic evaluation of the umbilical cord and the UCI may become an integral part of fetal assessment in high-risk pregnancies.

Keywords: *Umbilical cord, coiling, umbilical coiling index*

Introduction

The umbilicus, actually a scar, is the only visible memento of our close connection with our mother before birth. This was by means of the umbilical cord, which determined not only our welfare, but our very existence. Together with the placenta it is the only organ that dies when life begins. Although the umbilical cord is one of the most intriguing of the human organs, it is one of the least investigated.

The most distinctive feature of the umbilical cord, the helical pattern of its vessels, was first recorded in 1521 by Berengarius, as reported by Edmonds [1]. A voluminous literature accumulated in the early 1900s. However, after the turn of the twentieth century the interest in the cord declined, and from the period hereafter only occasional reports on the helical structure of the cord appeared. This is mainly because most of the perinatal complications involving the umbilical cord were only detected after birth, since the cord was inaccessible antenatally. With modern ultrasound techniques it has now become possible to search for abnormalities of the cord before birth. As a consequence there is a renewed interest, and a number of publications have appeared in recent years about abnormalities in cord coiling. Our aim was to review the literature on umbilical cord coiling.

Methods

We identified studies in the English literature regarding umbilical cord coiling we obtained from a Medline search from 1966 through March 2003. Search terms were umbilical cord coiling, chirality, umbilical coiling index. Additional information was obtained through cross-referencing.

Anatomy and embryology of the umbilical cord

At term the umbilical cord has an average length of 55 cm (usual range 30–100 cm) [2]. Leonardo da

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Vinci postulated the rule of thumb that the umbilical cord at any gestational age is on average as long as the fetus itself.

The umbilical cord consists of an outer layer of epithelium from the amnion, with an internal mesodermal mass, the Wharton's jelly. In this jelly there are two endodermal ducts: the allantois and the vitelline duct, and the umbilical vessels. The umbilical cord is formed at 4 to 6 weeks post-conception. At 18 days post-conception the connecting stalk develops, which connects the early embryo to the trophoblast. In this connecting stalk lies the transitory allantois, the primitive extra-embryonic urinary bladder [3]. The primary yolk sac is lined with endoderm and forms the central portion of the embryonic gut [4]. After contributing to the embryonic gut, the remains of the primary yolk sac elongate ventrally, thereby narrowing the connection to the midgut. The connection forms the ductus vitellinus (Figure 1). In early gestation it is accompanied by vitelline arteries and veins. In humans, the secondary yolk sac is small and rudimentary. At 4 weeks post-conception the connecting stalk and the yolk sac duct merge, forming the umbilical cord. In humans the yolk sac is a rudimentary organ, that probably has a nutritive function only very early in pregnancy.

The development of the vascular system starts with the formation of blood islands in the mesoderm of the yolk sac, connecting stalk and chorion at the beginning of 3 weeks post-conception. Two days later angiogenesis begins in the intra-embryonic mesoderm. The 'allantoic' arteries appear 3 weeks post-conception as ventral branches of the paired dorsal aortas. Portions of the allantois will give rise to the urinary bladder, from which the urachus extends as a tiny duct, accompanied by the allantoic arteries. They course to the umbilical ring and into the

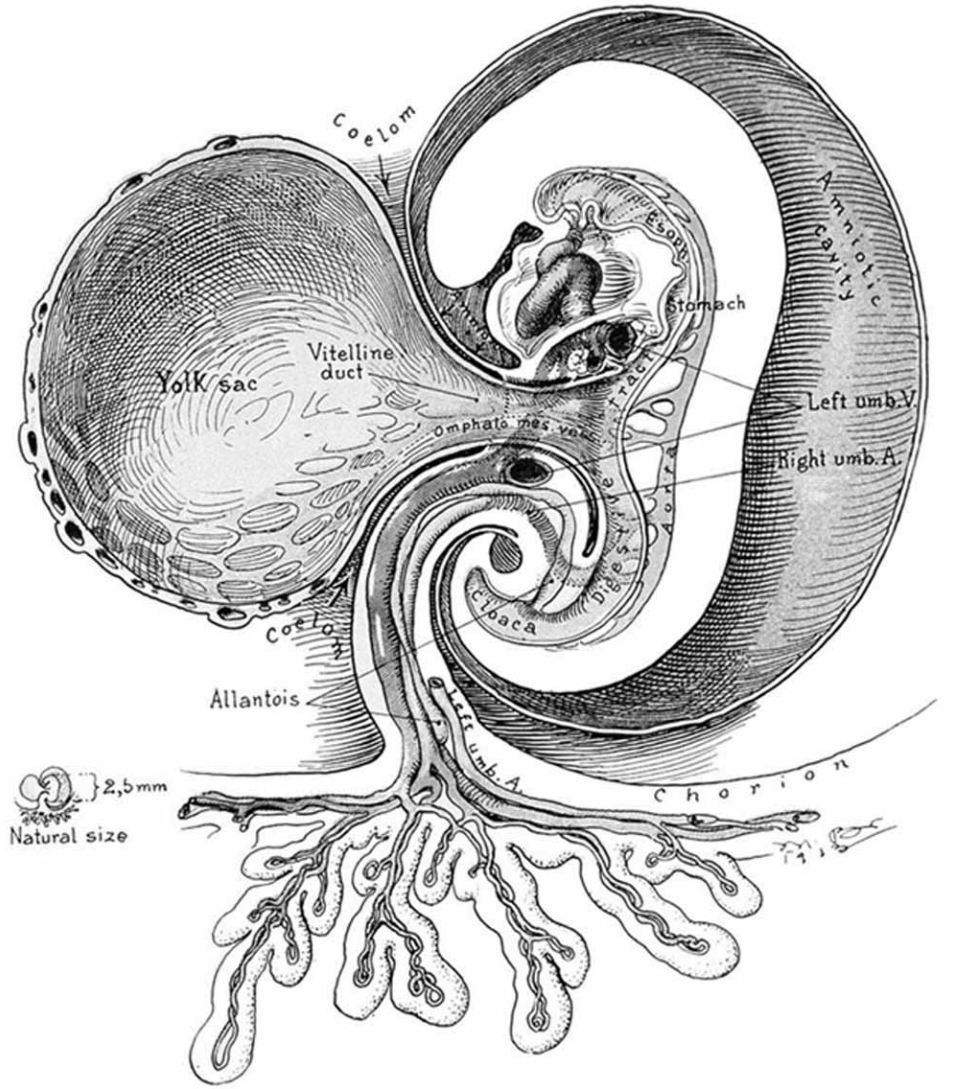

Figure 1. A composite picture showing the formation of the umbilicus in an embryo 2.5 mm long [31].

umbilical cord. When the aortas fuse the definitive arteries appear as lateral branches originating from its caudal end [5], and they ultimately become the umbilical arteries. The embryonic circulation is effective at 22–23 days post-conception, when the umbilical arteries have fused with the internal iliac arteries, and the umbilical vein with the ductus venosus, which enters the hepatic vein. One of the umbilical veins atrophies during the second month of pregnancy [3].

Until 11 weeks post-conception there are intestines in the umbilical cord, giving it a swollen appearance (Figure 2). Thereafter the intestines have retracted into the abdominal cavity [6]. The allantois, ductus vitellinus and vessels of the yolk sac obliterate, and all that remains in the umbilical cord are the umbilical vessels, surrounded by Wharton jelly.

In a normal umbilical cord there are two umbilical arteries, and one vein (the right vena umbilicalis usually obliterates) [2]. The two arteries are smaller in diameter than the vein. In 96% of all umbilical cords there is an anastomosis or, in 3%, even fusion of the two umbilical arteries within 1.5 cm of the placental insertion site (Hyrtl anastomosis) [3, 7]. This warrants an equalization of flow and pressures between the two arteries and a uniform distribution of blood to the different lobes of the placenta.

One of the most common vascular anomalies in humans is the absence of one umbilical artery, occurring in about 1% of umbilical cords, in most cases as an isolated abnormality. The umbilical vessels lack vasa vasorum [3]. Small nerve fibres seem to be present in the cord near the fetal end, but they are absent in the middle and placental segment of the human umbilical cord [8].

The helical course of the umbilical vessels can be observed as early as 28 days post-conception, and is clearly visible from 7 weeks post-conception in 95% of all fetuses.

The origin of the coiling is unknown. The hypotheses include fetal movements, active or passive torsion of the embryo [1], differential umbilical vascular growth rates and fetal hemodynamic forces, and, as mentioned before, the muscular fibers in the arterial wall [9]. Possibly there is a genetic factor, although in a small series of monozygotic twins no uniform concordance in the umbilical coiling index was found [10]. Fetuses with fixation of their bodies (due to amniotic bands) not only have relatively short cords, but also only few or no umbilical helices [3]. The same is true for species with elongated fetuses in elongated uterine horns

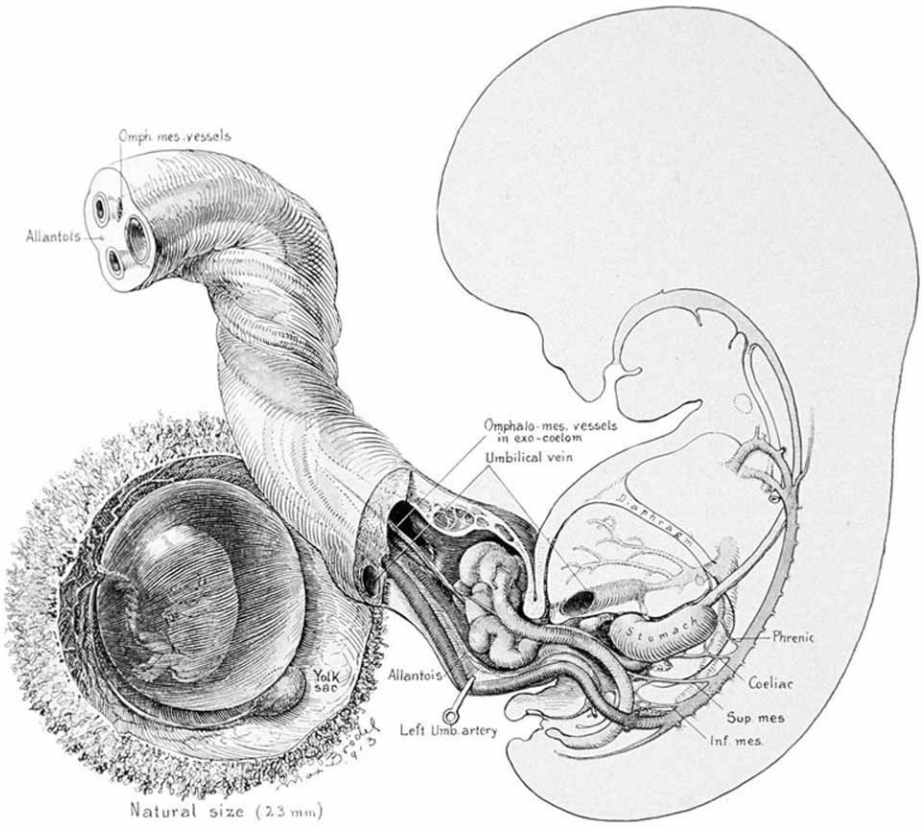

Figure 2. Sagittal section of the umbilical region in a human embryo 23 mm in length [31].

(e.g. whales), a situation that hinders embryonic rotation [3].

According to Roach [11], the coiling is caused by muscular fibers in the arterial walls. There are four different muscles in the arterial wall: an inner circular layer, regulating flow, an inner longitudinal layer, which closes the artery post-partum, a large coiling muscle, which has an intrinsic twist that makes the cord coil, and a small coiling muscle, which makes the arteries coil [11]. The large helical muscle has a long pitch which is comparable to the pitch of the coils of the cord itself. Attachments of the coiling muscle of the artery to the cord substance are responsible for coiling of the cord itself. When there is enough hydrostatic pressure, the cord coils in the direction opposite to the direction of the fibers in the helical muscle.

The cord is gradually covered by the amniotic membrane from 4 weeks post-conception onwards. The amniotic cavity continues to enlarge and the amnion sheathes the umbilical cord in the direction of the placenta [3].

Wharton's jelly, derived from mesenchyme, and formed by myofibroblasts, consists of collagen and hyaluronic acid, some muscular fibers, and water. This material seems to be responsible for the strength of the umbilical cord. It provides mechanical support and structural protection for umbilical vessels, and has angiogenic and metabolic roles for the umbilical circulation [12]. The osmotic environment is of utmost importance to the Wharton's jelly. Changes in osmolarity of 5 to 10 milliosmol cause evident swelling or shrinking of the cord. Wharton jelly has thyxotropic properties, i.e. this semi solid gelatinous substance liquefies due to pressure. The amount of Wharton jelly is a good predictor of perinatal complications: evidence cumulates that an umbilical cord with a diameter $<$ 10th centile is an early marker for the delivery of a small for gestational age infant and the occurrence of intrapartum complications [13].

Umbilical cord coiling index

An umbilical coil is defined as one complete spiral of 360° of the umbilical vessels around each other. The coiling makes the umbilical cord a structure which is both flexible and strong, and provides resistance to external forces which could compromise blood flow. Both sinistral and dextral spirals occur. In a sinistral spiral held vertically, the portions of the spiral anterior to the axis and therefore visible, will appear to slant from a point above on the left to a point below on the right. In other words, the course of the anterior portion of a sinistral spiral will parallel the left-hand limb of a V, while the anterior portion of a dextral spiral will parallel the right-hand limb [1]. It makes no difference from which side one looks at the cord: a sinistral course viewed from the fetal side is also sinistral if viewed from the placental insertion site.

Sinistral spiralling is four to eight times more common than dextral spiralling, and sometimes there is a mixed pattern of coiling. It is not clear why sinistral coiling is much more common. There is no relationship with left- or right-handedness in both fetus and mother, and both on the northern and southern hemisphere sinistral spiralling is most common (In contrast to the difference in 'bathtub vortex') [14]. In approximately 2–5% of umbilical cords there is no coiling at all.

According to Malpas and Symonds, the cord does not grow in length by any clear increase in the number of twists but by a progressive increase in the length of the pitch of the primary helix, making it clear that the cord grows uniformly at every point throughout its length and not from one growing point [9]. This conclusion is cited in many articles [5, 10, 14, 19, 21, 22, 23], but was only based on only six early pregnancy cases.

It has been observed that 30% of non-coiled cords still became coiled after a gestational age of 20 weeks, whereas loss of coiling has never been observed [15]. Cords tend to have more spiral turns towards the fetal end than towards the placental end [17].

Edmonds [1], in 1954, was the first to describe a method for quantification of cord coiling. He called it the index of twist, which was the ratio of twists to the length of the cord, giving positive and negative values to the twists if the direction of coiling changed from left to right, where sinistral turns counterbalance dextral turns. Strong [16], in 1994, was the first to simplify this method. He developed the umbilical coiling index, which is the ratio of twists to the length of the cord, irrespective of the direction of coiling. In an earlier paper we provided reference values of the umbilical coiling index (UCI) [17]. These were determined in a group consisting exclusively of uncomplicated pregnancies. The frequency distribution of the UCI appeared to be skewed to the right. The mean (SD) UCI was 0.17 (0.009) coils/cm. The 10th and 90th centiles for the UCI were 0.07 and 0.30 coils/cm.

Obstetrical complications associated with umbilical cord coiling abnormalities

Abnormally coiled cords have been reported to be more frequently present in cases of adverse perinatal outcome (Table I).

Strong et al. [18] first compared pregnancy outcome of fetuses born with non-coiled umbilical vessels with fetuses with coiled vessels, irrespective of the UCI. They reported a significantly increased

Table I. Studies on the relationship of (abnormal) umbilical coiling with adverse pregnancy outcome.

	UCI	% Fetal demise	% Variable fetal heart rate decelerations	% Premature delivery <37 weeks	% Aneuploidy/ fetal anomalies	% Intra-uterine growth restriction	% Operative delivery for fetal distress	% Meconium staining of amniotic fluid	% Maternal cocaine use
Strong 1 [18]	Non-coiled vs coiled		50/8.3	55.9/33.1	11.8/3.2	8.8/4.3 *	35/6.7	26.3/6.2	
n = 894			$p < 0.00005$	$p = 0.006$	$p = 0.03$		$p < 0.00005$	$p = 0.007$	
Strong 2 [16]	$<p$ 10 vs $>p$ 10			10/8*	20/0	10/2*	30/8	50/9	
n = 100					$p = 0.04$		$p = 0.03$	$p = 0.03$	
	$<p$10 and $>p$ 90 vs normal		25/5						
			$p = 0.03$						
Rana [20]	$<p$ 10 vs normal		28.6/15.9	15.9/12.0*	4.8/3.5*	6.4/4.7*		20.6/23.2	0/3.3*
n = 635			$p = 0.01$						
			15.9/17.5*	33.3/12.0	6.4/3.5*	7.9/4.7*	19.0/7.1	22.2/23.2*	12.7/3.3
	$>p$ 90 vs normal			$p < 0.0001$			$p < 0.002$		$p = 0.0006$
Ezimokhai [21]	Non-coiled vs normal	5/0.8		15.0/11.7*	0/2*	0/0.8*	55/3.2	15/4.5	
n = 657		$p < 0.0001$					$p < 0.0001$	$p < 0.001$	
	$<p$ 10 vs normal	0/0.8*		11.2/11.7*	3.2/2*	1.6/0.8*	6.5/3.2*	3.2/4.5*	
	$>p$ 90 vs normal	0.2/0.8*		19.3/11.7*	0/2*	4.4/0.8	16.1/3.2	10.3/4.5	
						$p < 0.05$	$p < 0.05$	$p < 0.001$	
Machin [22]	$<p$ 10 vs normal	35/8				15/1.5	21/10		
n = 1329		$p < 0.0005$				$p < 0.0005$	$p < 0.0005$		
	$>p$ 90 vs normal	37/8				13 vs 1.5	25/10		
		$p < 0.0005$				$p < 0.0005$	$p < 0.0005$		

* = non-significant. Fisher exact test, student's t test and Chi square test were used when appropriate.

incidence of intra-uterine death, preterm delivery, intrapartum fetal heart rate decelerations, operative delivery for fetal distress, meconium staining, and anatomical-karyotypic abnormalities in the group with non-coiled cords.

In a following study Strong et al. [16] reported a significantly greater incidence of karyotypic abnormalities, meconium staining, and operative intervention for fetal distress among those whose umbilical coiling index values were below the 10th centile. For those whose UCI was either $\leqslant$ 10th centile or $\geqslant$ 90th centile there was a significantly greater incidence of variable fetal heart rate decelerations. The values for 10th and 90th centile were derived from the UCI of 100 consecutive births (UCI 0.21 ± 0.07), in a high-risk pregnancy population, which may have caused selection bias. Moreover, the groups with an abnormal coiling index were so small ($n = 10$ for each group!) that the results may be explained by chance alone.

Rana et al [19] reported a significantly greater incidence of fetal heart rate disturbances and operative delivery among those whose umbilical coiling index values were below the 10th centile. The subjects with UCI values above the 90th centile had a higher rate of preterm delivery and a higher incidence of maternal cocaine use. The authors did not differentiate between spontaneous preterm delivery and iatrogenous preterm delivery. Their values for the 10th and 90th centile were also derived from a high risk population ($n = 635$). In this study no association was found between an abnormal UCI and fetal aneuploidy and/or fetal anomalies, low Apgar scores or meconium staining. However, not a single confirmed case of aneuploidy was found in this study, so it cannot be concluded from these data that there is no association of an abnormal UCI with aneuploidy.

Atalla et al. [20] assessed the relation between umbilical cord morphology and intrapartum fetal status and umbilical cord blood gases at birth. They found statistically significant positive linear correlations between umbilical venous and arterial pH and number of vascular coils ($r = 0.27$ and 0.17, respectively), and a negative linear relation between PCO_2 and the umbilical coiling index. In this study no association was found between the UCI and intrapartum fetal heart rate decelerations, meconium staining and interventional delivery for fetal distress. Unfortunately they only calculated mean UCI's of cords with or without each of these outcomes. This approach is questionable: if an adverse outcome is associated with both hyper- and hypocoiling, a normal *mean* UCI may be found.

Ezimokhai et al. [21] identified maternal risk factors for an abnormal UCI. For hypercoiling risk factors were extremes of age, for non-coiling risk factors were obesity, gestational diabetes mellitus and pre-eclampsia. Hypercoiling and non-coiling were significantly associated with meconium staining, adverse perinatal outcome and emergency cesarian delivery. In this study these outcomes were not associated with hypocoiling. Hypercoiling was associated with fetal growth restriction. Diminished placental blood supply underlies several of the aforementioned conditions. Therefore, the appropriate statistical method to analyse these data is multivariate analysis. Unfortunately, the authors omitted to do this.

Machin et al. [22] studied the umbilical coiling index of all placentas referred to their placental pathology services. In this selected group (possibly causing selection bias), abnormal cord coiling was associated with fetal demise, fetal intolerance to labor, intrauterine growth retardation and chorioamnionitis. It was associated with thrombosis of chorionic plate vessels, umbilical venous thrombosis and cord stenosis.

In all the studies mentioned above the used reference values were calculated from a population including complicated pregnancies.

To confirm that fetuses with an abnormal coiling index do indeed more often have an unfavourable outcome than those with normally coiled cords, a prospective study of an unselected population, using reference values from exclusively uncomplicated pregnancies, is currently under way at our institution.

Prenatal diagnosis of umbilical cord coiling abnormalities by ultrasonography

Degani et al. [23] showed that the UCI can be determined prenatally using ultrasonography and that the ultrasonographically determined UCI correlates well with the index measured after birth, when measured within 24 h before birth. The intrauterine UCI was higher than the postnatal UCI (0.44 ± 0.11 vs 0.28 ± 0.08, $r = 0.71$, $p < 0.001$, association formula: antenatal UCI = 0.1775 + 0.9622 x postnatal UCI). The antenatal UCI was calculated by taking the reciprocal of the average distance between a pair of coils, measured at three different segments. A possible explanation for the higher UCI in utero is that post-partum the measured UCI lacks the part of the cord closest to the child, which tends to be more coiled than the part near the placental insertion. Furthermore, antenatally the cord is more filled with blood, which makes the helix more dense due to the intrinsic twist in the vessels [9]. Torsion of the cord influences the ultrasonographic measurements in the ante partum period, but not any more post-partum after the cord is cut. Although the direction of coiling seems of no clinical importance, it can be deter-

mined by scanning near the surface of the cord closest to the observer [24].

Qin et al. [25] performed ultrasonographic measurement of the umbilical coiling index in the second trimester of pregnancy. They found that the UCI can be measured easily and reliably in the second trimester, but these measurements do not accurately reflect the UCI at term after birth (also contradicting that umbilical coiling does not alter after the initial formation of coils in the first trimester, as stated by Malpas and Symonds).

Degani et al. found a correlation between flow in the umbilical vein and the coiling index, with a linear trend ($r = 0.59$, $p < 0.001$). No significant correlation was found between UCI and Doppler characteristics in the umbilical arteries.

Discussion

How is optimal flow in the umbilical cord vessels achieved?

The umbilical vein is the only lifeline that transports oxygen and nutrients to the fetus. A merely passive process, caused by the pressure gradient between the umbilical vein and the fetal inferior vena cava, may not be sufficient to transport the necessary amount of blood back to the fetus.

The anatomy of the umbilical cord is such that flow is optimized, by the coils in the cord. The arteries, adjacent to the vein, cause alternating increases and decreases in the venous pressure with their pulsations. Umbilical arterial and venous pressure pulsations are 180° out of phase [26]. The arteries in the cord stretch in length during pulsations. This decreases the diameter of the arteries and thereby increases the diameter of the vein, causing a relative negative pressure in the vein. This way the venous blood is pulsed forward. When there are more coils, the effect of the pressure-pulsations of the arteries on the vein will increase, and as a consequence venous flow will increase [27]. Is this possibly the evolutionary reason that one of the umbilical veins obliterates? If there were two veins, the arteries could not completely spiral around the vein, and their effect on the two veins would be considerably smaller. Nevertheless, the arrangement of vessels is different in many other species. For example, two arteries and two veins are found in sheep, and cats have four vessels of each type [3] (so in cats both the embryonic vitelline and allantoic vessels persist).

The more coils there are, the less straight is the course the blood has to follow. The more the blood comes into contact with the vessel-wall, the more turbulence occurs, which slows the blood flow. Probably there is an optimal coiling index which provides maximum flow. Hypercoiling may also make the arteries compress the vein, compromising flow. As shown by Degani et al., the coiling index may influence venous flow velocity. Decreased coiling is associated with reduced flow indices in the umbilical vein. Increased coiling is associated with a pulsatile pattern of the umbilical venous flow velocity waveforms similar to those seen with abnormalities of the fetal central venous flow secondary to severe circulatory compromise [28].

It is unlikely that the coiling index has a great influence on normal arterial flow, since this is an active process. The literature supports this assumption: the coiling index does not significantly influence arterial Doppler measures. Arterial blood flow however, can be compromised by the occurrence of thrombosis, which is observed more often in hypercoiled cords [22].

Since it is not known whether the umbilical coiling index may change during pregnancy, it is not known if blood flow to the fetus can be increased by adapting of the coiling index. If adaptation is possible, it might explain the association of higher mean umbilical cord index in pregnancies complicated by fetal growth restriction. During parturition the coiling index is important for yet another reason. It should resist occlusion during contractions by torsion, kinking and compression, and occlusion due to traction on the cord. The coils may make the cord more resistant to kinking and compression, but under a tight encirclement force, the opposite was found. In an experiment by Georgiou et al., in which venous perfusion was measured in cords subjected to a standardized tight encirclement force, a significant inverse correlation was found between coiling index and the minimum weight required to occlude venous perfusion [29]. So whilst hypocoiled cords are strongly associated with nuchal cords [30], they seem to be more resistant to one of the problems caused by nuchal cords, i.e. occlusion of the cord when stretching around the fetal neck. However, the most important problem of tight nuchal cords is probably not compression of the cord itself, but compression of the fetal carotid arteries.

Other factors that may influence the susceptibility of the cord to vascular occlusion are the thickness, and degree of hydration. A significant inverse relation was found between hydration index and minimum weight required to occlude venous perfusion, although the relation was less strong than with the coiling index. There was no correlation with the mass index [29]. The mass index was calculated using the dry weight of the umbilical cord.

Conclusions

Abnormal coiling of the vessels of the umbilical cord is associated with adverse pregnancy outcome. It is

not clear if abnormal coiling is actually a cause of pathology, or merely one of the sequelae or both.

Determination of the umbilical coiling index should be a routine part of placental examination post-partum.

Antenatal evaluation of the UCI requires further study. An abnormal UCI may be predictive of later intra-uterine growth restriction and warrant intensified fetal monitoring. In the future evaluation of the umbilical cord and the UCI may become an integral part of fetal assessment in high-risk pregnancies.

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