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# UMBILICAL CORD LENGTH AND ADVERSE OBSTETRIC OUTCOMES: PLACENTAL DEVELOPMENT, CORD ACCIDENTS, AND ANTENATAL ULTRASOUND

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## Abstract: -

**Background:** Umbilical cord length is routinely recorded after delivery but seldom considered before it, despite growing evidence that both short and long cords carry obstetric risk.

**Objective:** To review the association between umbilical cord length and adverse obstetric outcomes, examine the biological mechanisms underlying it, and assess whether cord length can be evaluated antenatally.

**Methods:** A narrative review of the literature on cord length, morphometry, coiling and cord accidents, drawing on population registries, cohort and autopsy series, and ultrasound and artificial-intelligence studies, appraised for definitions, measurement timing and confounding.

**Result:** Adverse outcomes cluster at both extremes rather than rising linearly. Short cords were associated with caesarean delivery (38.8%), fetal distress (29.9%) and neonatal intensive care unit (NICU) admission (16.4%), with unplanned caesarean risk increasing progressively as length falls and placental abruption raised in term births (OR 1.98, 95% CI 1.72–2.73). Long cords act chiefly through mechanical opportunity, increasing wrapping, looping and knotting. Cord accidents accounted for 10% of possible or probable stillbirth causes, more commonly beyond 32 weeks, though published estimates vary widely. Lean cords occurred in 73.8% of IUGR fetuses versus 11.3% of normally grown fetuses. Absolute length is measurable only in the first trimester ($r^2 = 0.89$); three-dimensional estimation does not predict it, whereas coiling, diameter and insertion are assessable in routine examinations.

**Conclusion:** Cord length is best understood as a marker of fetal movement, placental development and the intrauterine environment rather than a single pathological variable. Routine measurement is not yet justified; a composite assessment of cord morphology, supported by standardised definitions and prospective cohorts, offers the more realistic path forward.

**Keywords:** umbilical cord length; short umbilical cord; long umbilical cord; adverse obstetric outcomes; umbilical coiling index; cord accidents; antenatal ultrasound; stillbirth.

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# 1. INTRODUCTION

## 1.1 The Background of the Study

The umbilical cord is the lifeline of the fetus. It is a fetal, not placental, structure, developmentally derived from the "primitive ridge" of the embryo.[1] A normal cord contains a single umbilical vein and two umbilical arteries embedded within Wharton's jelly, a specialised mucous connective tissue that cushions the vessels against compression and torsion.[2] The vessels have a characteristic twisted, rope-like appearance, and loss of this twisting is often associated with decreased fetal movement and poor pregnancy prognosis.[1] Because the cord is the sole route of oxygen and nutrient delivery and of waste removal, obstruction or disruption of flow through the umbilical vessels can produce any degree of fetal injury, from acute transient hypoxia to fetal death.[3] The cord is therefore best understood as the functional interface between fetal physiology and placental function, and its morphology carries information about both.

The central hypothesis of this review is that umbilical cord length may represent not merely an anatomical characteristic but a marker of fetal movement, placental development and the intrauterine environment, while extreme cord lengths may predispose to mechanical complications. Importantly, the review does not assume that cord length causes adverse outcomes. Instead it attempts to investigate whether cord length acts as (1) an independent risk factor for cord complications, (2) a marker of abnormal fetal or placental development, (3) a mediator of cord complications, or (4) simply an incidental anatomical finding. Distinguishing among these possibilities is one of the principal analytical tasks of this review.

## 1.2 Objectives of the Review

1. To examine the relationship between umbilical cord length and obstetric outcomes.
2. To explore the biological mechanisms underlying these associations.
3. To evaluate the potential role of antenatal ultrasound in identifying abnormal cord length and related pathology.

# 2. METHODS

## 2.1 Study Design

This is a narrative (non-systematic) review of the literature on umbilical cord length and its relationship to obstetric outcomes. A narrative design was chosen because the field is characterised by heterogeneous definitions, retrospective designs and inconsistent outcome reporting, which makes pooled quantitative synthesis of limited value; the aim is instead to map and critically appraise the evidence across mechanistic, epidemiological and imaging domains.

## 2.2 Search Strategy

A structured literature search was conducted in PubMed/MEDLINE, Scopus, Web of Science, Google Scholar and the Cochrane Library. The search combined terms relating to the exposure (umbilical cord length, short umbilical cord, long umbilical cord, umbilical coiling index, cord morphometry, cord diameter, nuchal cord, true knot, cord entanglement, cord prolapse) with terms relating to outcomes (adverse pregnancy outcome, caesarean delivery, fetal distress, intrauterine growth restriction, NICU admission, stillbirth, perinatal mortality) and to assessment (antenatal ultrasound, sonographic, 3D ultrasound, artificial intelligence). Reference lists of retrieved articles and relevant reviews were hand-searched for additional studies. The search was limited to English-language publications. The included evidence spans 1981 to 2025, reflecting both foundational embryological and anatomical work and recent imaging and machine-learning developments.

## 2.3 Eligibility Criteria

Studies were included if they reported original data or systematic synthesis on cord length, cord morphometry or cord coiling in human pregnancy, or on the antenatal sonographic assessment of the umbilical cord. Eligible designs included population-based registries and cohort studies, retrospective and pathology series, meta-analyses, large multicentre cohorts, and imaging and artificial-intelligence studies. Narrative and systematic reviews were used for contextual and mechanistic material. Case reports, animal studies and studies in which cord length was neither measured nor estimated were excluded, as were publications without sufficient methodological detail to appraise the reliability of reported associations.

## 2.4 Data Extraction and Appraisal

For each included study, the following were extracted where reported: study design and setting, sample size, definition and measurement method for short or long cord (absolute centimetres, gestational-age percentile, or umbilical coiling index threshold), timing of measurement (antenatal or postnatal), outcomes examined, and effect estimates with confidence intervals. Findings were grouped thematically and reported narratively, with effect estimates presented as published rather than pooled.

## 2.5 Limitations of the Method

The principal limitation of this approach is that study selection is not exhaustive and is subject to reviewer judgement; the review therefore describes the state of the evidence and identifies gaps rather than claiming an unbiased pooled estimate.

## 3. BIOLOGY AND DETERMINANTS OF UMBILICAL CORD LENGTH

### 3.1 Embryological Development

The definitive umbilical cord forms from the convergence of several embryonic structures, including the connecting stalk, the allantois, and the vitelline (omphalomesenteric) duct, together with the physiological herniation of the midgut into the extraembryonic coelom during early development.[4] As the amnion envelops these structures, the cord acquires its characteristic covering and its vascular core.[4] Because the cord is fetal in origin, its development is subject to paternal genetic influences on growth and development, and cord pathology is properly considered separately from placental pathology.[1]

### 3.2 Fetal Movement and Cord Elongation

One of the most influential hypotheses in this field is that cord elongation is driven by mechanical forces generated by fetal movement. The classic evidence comes from Miller and colleagues, who found short umbilical cords in newborns with evidence of early intrauterine constraint and in those with gross structural or functional limb defects that limited intrauterine movement. These findings were interpreted as showing that cord growth occurs in response to tensile forces relating to intrauterine space availability and fetal movement during early development such that a short cord may indicate diminished fetal movement from either early constraint or fetal limb dysfunction.[5] Consistent with this, it is suggested that sufficient space in the amniotic cavity for movement and the tensile force applied to the cord during fetal movements are the two main factors determining cord length.[6,7] More directly, a study using a fetal movement acceleration measurement recorder concluded that fetal movement near term is an important factor in determining whether cord length becomes relatively longer in normal pregnancies.[8]

Taken together, these observations support the concept that cord length may be partly a historical record of fetal movement during gestation.

### 3.3 Placental Factors

Placental characteristics plausibly influence cord length and morphology. Cord length appears to be influenced positively by amniotic fluid volume and fetal mobility, both of which are in turn related to placental function.[9] In intrauterine growth-restricted fetuses, all cord components, cross-sectional area, vein area, artery area, and Wharton's jelly area, are smaller than in appropriate-for-gestational-age fetuses, and the prevalence of lean cords is significantly higher (73.8% vs 11.3%).[10]

### 3.4 Fetal Factors

Potential fetal determinants of cord length include fetal size, gestational age, sex, congenital anomalies, fetal movement, and multiple pregnancy. Cord length is significantly related to birth weight.[6] In a retrospective cohort of 14,873 term singleton deliveries, birth weight and the rate of male infants were both increased in the long-cord group and decreased in the short-cord group ($p < 0.05$).[9] Excessively long cords have also been associated with fetal anomalies, vertex presentation, and male sex.[11] Congenital anomalies that restrict movement, particularly limb defects and neuromuscular conditions, are among the strongest recognized correlates of short cords.[5]

### 3.5 Maternal and Pregnancy Factors

Maternal factors associated with longer cords include increased maternal age and gravidity, while short cords are associated with lower values of both.[9] Maternal diabetes is associated with larger cords: the cord is significantly larger in fetuses of mothers with gestational diabetes than in the normal population, with the increase in width attributed to increased Wharton's jelly content.[12] Cords in patients with gestational diabetes are larger, and lean cords have been associated with preeclampsia.[13]

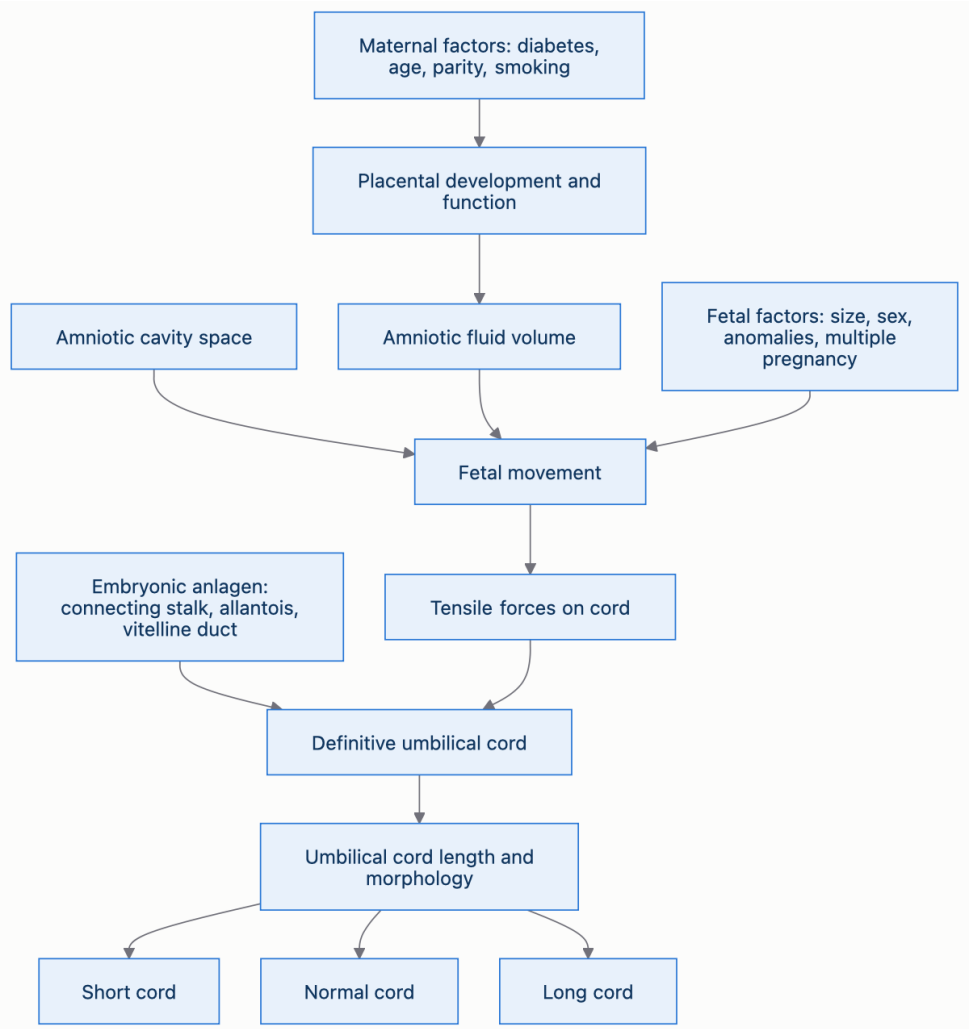



**Figure 1** summarises the developmental and determinant pathways discussed in this section.

## 4. SHORT UMBILICAL CORD

### 4.1 Definition

The most widely cited thresholds are a short cord of <35 cm and a long cord of >80 cm, with 35–80 cm regarded as normal.[9,14] However, other studies adopt quite different boundaries. One recent retrospective study classified cords of <45 cm as short, 46–90 cm as medium, and >90 cm as long.[15] Percentile-based approaches avoid arbitrary cut-offs altogether: in a cohort of 89,042 term deliveries, cord lengths of 33, 43, 48, and 63 cm corresponded to the first, 10th, 25th, and 75th percentiles.[16]

The practical unavoidable consequence of this inconsistency is that a "short cord" in one study may not meet the definition used in another**.** This directly affects prevalence estimates, effect sizes, and the comparability of findings across the literature.

### 4.2 Proposed Mechanisms

Because cord growth appears to occur in response to tensile forces generated by fetal movement, a short cord may itself be a marker of diminished fetal mobility from early constraint or limb dysfunction.[5] At delivery, a cord that is insufficiently long to accommodate the descent of the fetus may be placed under increased tension, transmitting traction to the placenta and potentially compromising umbilical blood flow.[17]

### 4.3 Complications of Short Umbilical Cord

Reported complications of excessively short cords include delay in the second stage of labour, retained placenta, placental abruption, rupture of the umbilical cord, inversion of the uterus, birth asphyxia, and cord herniation.[7] Potential vascular compromise arises both from direct traction and from the mechanical effects of a taut, poorly cushioned cord.

Short cords are associated with lower birth weights and reduced rates of male infants compared with normal and long cords.[9,6] In a cohort of 134 singleton pregnancies, short cords were associated with a 23.1% rate of low birth weight and a 9.7% rate of small-for-gestational-age status.[14] The direction of causality remains contested: a short cord may be a consequence of restricted fetal growth and movement, a contributor to it through vascular compromise, or both.

In the large Japanese registry analysis, statistically significant differences were observed in the rate of unplanned caesarean delivery for all three short-cord groups compared with controls, with a higher odds ratio for unplanned caesarean delivery as the umbilical cord became shorter.[16] Short cords have been associated with increased risk of non-cephalic presentation in both term and preterm births, and with increased risk of emergency and all-cause caesarean delivery in stratified analyses.[18] In a smaller cohort, short cords were associated with higher rates of caesarean delivery (38.8%) and fetal distress (29.9%) as well as delayed second stage labour.[7,14]

In a population-based study, pregnancies with a short cord carried an increased risk of placenta previa, placental abruption, and the need for manual removal of the placenta after birth.[18] The associated risk of placental abruption was observed particularly in term births (OR 1.98, 95% CI 1.72–2.73) but was also significant in preterm births (OR 1.35, 95% CI 1.12–1.62).[18] Retained placenta, placental abruption, and uterine inversion are all described among the complications of excessively short cords.[7]

Short cords have been linked to low Apgar scores, NICU admission, birth asphyxia, stillbirth, and perinatal mortality. In a cohort of 134 singleton pregnancies, short cords were associated with NICU admission in 16.4% of cases, with 14.9% low Apgar scores and 3.7% perinatal mortality overall. [7,8,10 14]

**Table 3** consolidates the short-cord associations discussed in this section.

## 5. LONG UMBILICAL CORD

### 5.1 Definition

As with short cords, definitions vary. Long cords are most commonly defined as >80 cm, but some studies use >90 cm, and others use percentile thresholds.[9,14-16]. Excessively long umbilical cords are found in approximately 4% of pregnancies and represent a recognised risk factor for nuchal cords and true knots.[19] In a cohort of 14,873 term singleton deliveries, 2.5% had long cords (>80 cm), 6.6% had short cords (<35 cm), and 90.9% had normal cords.[9]

### 5.2 Biological Mechanisms and Complications

A longer cord has greater freedom to move around the fetus, increasing the opportunity for the cord to wrap, loop, and knot, and thereby increasing susceptibility to entanglement and vascular compression.[7] Excessively long cords are significantly associated with cord entanglement and true knots in large pathological series.[11] The consequence of these mechanical configurations is potential vascular occlusion leading to asphyxia.[20]

### 5.3 Nuchal Cords

Nuchal cord is common. Reported incidences include 23.5% of deliveries (single loop 18.8%, multiple loops 2.8%, tight nuchal cord 1.9%) in a series of 2,530 deliveries, and approximately 22.7% at delivery in another report.[21,22] Nuchal cords are noted in almost one-fourth of uncomplicated pregnancies.[23]

Distribution of loops varies across series: in one cohort of 100 nuchal-cord cases, 56% had a single loop, 37% double, 4% triple, 2% four loops, and 1% eight loops; in another, 91% had one loop and 9% had two or more, with 82% loose and 18% tight.[22,24]

Long cord may increase the risk of nuchal cord formation.[19] The clinical significance of nuchal cord is contested. Some studies report an association with non-reassuring fetal heart rate patterns ($p = 0.000$) without corresponding differences in Apgar scores, resuscitation, respiratory morbidity, or ICU admission.[21] In a cohort of 4,426 term neonates comparing 0, 1, and ≥2 loops, there were no significant differences in mean birth weight, non-reassuring fetal heart rate patterns, operative vaginal delivery, or 5-minute Apgar <7. As a matter of fact, the caesarean rate was highest among women whose fetus had no nuchal cord ($p < 0.01$).[25] In a more recent retrospective cohort of 466 pregnancies, the presence of a nuchal cord had no effect on adverse perinatal outcomes overall, although a tight nuchal cord was associated with a 7.52-fold increased risk of Apgar <7 at 1 minute (OR 7.52, 95% CI 1.51–37.31, $p = 0.014$).[24] Vaginal delivery can be safely contemplated in well-monitored labours with nuchal cord.[22]

### 5.4 True Umbilical Knots

True umbilical cord knots occur when the cord twists around itself, forming a tight knot, with a reported incidence of 0.3–2%.[26] Formation is assumed to take place between 9 and 12 gestational weeks, and detection in utero can be challenging, so knots often go unnoticed until delivery.[26] Risk factors include a long umbilical cord, polyhydramnios, male fetus, gestational diabetes, and multiparity.[26] In a multicentre retrospective cohort of 283,055 deliveries, the incidence of true knot was 1.3%, and IUFD was significantly higher in the true-knot group (1.8% vs 0.3%), with 91% of these deaths occurring after 34 weeks and 72% after 37 weeks; multivariate analysis showed true knot was independently associated with IUFD (adjusted OR 6.07, 95% CI 4.68–7.86, $p < 0.01$).[27] Women with true knots also experienced higher rates of preterm premature rupture of membranes, induction of labour, meconium-stained amniotic fluid, vacuum-assisted delivery, and postpartum haemorrhage, with higher rates of SGA neonates, low Apgar scores, jaundice, hypoglycaemia, and intracranial haemorrhage.[27] Knots may be tight or loose, and although they have no clinical

significance in most cases, blood flow may potentially be compromised as the fetus moves and grows.[26]

A notable counterpoint concerns the interaction with cord length. In one analysis, true knots were associated with IUFD (adjusted OR 5.65, 95% CI 1.9–17, p = 0.02) and with excessively long cord (adjusted OR 8.26, 95% CI 6.0–11, p < 0.01), yet the incidence of IUFD in cases with excessively long cords was significantly lower than in those without (0% vs 4.2%, p = 0.04) and not measurably different from cases without true knots (0.3%, p = 1.00), leading the authors to propose that excessively long cords may contribute to a decreased risk of IUFD in singleton pregnancies delivered at ≥34 weeks.[28] This finding underscores how far the field remains from a coherent causal model.

### 5.5 Umbilical Cord Entanglement

Cord entanglement is most consequential in multiple gestations sharing a single amniotic sac. Cord entanglement is present in all monoamniotic twins when systematically evaluated by ultrasound and colour Doppler, and the rate diagnosed by ultrasound during pregnancy in monochorionic monoamniotic (MCMA) twins ranges between 55% and 100%.[29,30] In MCMA twins, fetal death risk after 24 weeks is nine times higher than in dichorionic twins (9% vs 1%), and historically perinatal mortality in monoamniotic twins has been reported as ranging from 30–70%, with umbilical cord accident the most significant contributor.[20,30]

However, newer evidence suggests entanglement may not be the main cause of poor outcomes. Perinatal mortality in monoamniotic twins appears to be mainly a consequence of conjoined twins, twin reversed arterial perfusion, discordant anomaly, and spontaneous miscarriage before 20 weeks; expectantly managed monoamniotic twins after 20 weeks have a very good prognosis despite the finding of cord entanglement.[29] A systematic review concluded that cord entanglement does not contribute to prenatal morbidity and mortality in monoamniotic twin pregnancies, and a series of 60 MCMA pregnancies found that prenatal ultrasound had a sensitivity of 58.1% and specificity of 82.8% for cord entanglement, with entanglement not contributing to prenatal mortality.[31,32]

### 5.6 Umbilical Cord Prolapse

Cord prolapse is a rare but high-stakes obstetric emergency. The overall prevalence is **0.1%–0.6% of all deliveries**, with higher rates in non-cephalic presentation, multiple gestation, polyhydramnios and preterm pregnancies.[33] In a cohort of 87 true prolapse cases, **14%** were twin gestations and **41%** were non-cephalic, with a mean gestational age at delivery of **34.0 ± 6.0 weeks** and mean birth weight of **2318 ± 1159 g**; **47%** of cases followed an obstetric intervention (amniotomy, scalp electrode application, intrauterine pressure catheter insertion, attempted external cephalic version, expectant management of PPROM, manual rotation of the fetal head, and amnioreduction).[34] Presentation-specific incidence has been reported as **9.6%** for transverse and **3.5%** for breech presentations.[35]

Outcomes can be severe: in the series of 87 true cord prolapses mentioned previously, 89% of infants were delivered by caesarean section, 33% had a 5-minute Apgar score <7, 34% had a cord pH <7.20, and there were 11 perinatal deaths.[34] Caesarean delivery decreases the risk of perinatal mortality among cases of cord prolapse.[36,37]

The link between cord prolapse and increased cord length is plausible but less firmly established than the link with entanglement and knots. In one cohort, long cords were more often linked to cord prolapse (7.5%) and abnormal labour progression (12.7%).[14] Given that prolapse is primarily determined by fetal presentation, membrane status, and obstetric intervention, cord length is best regarded as a possible contributing factor rather than a principal determinant.

**Table 4** consolidates the long-cord associations.

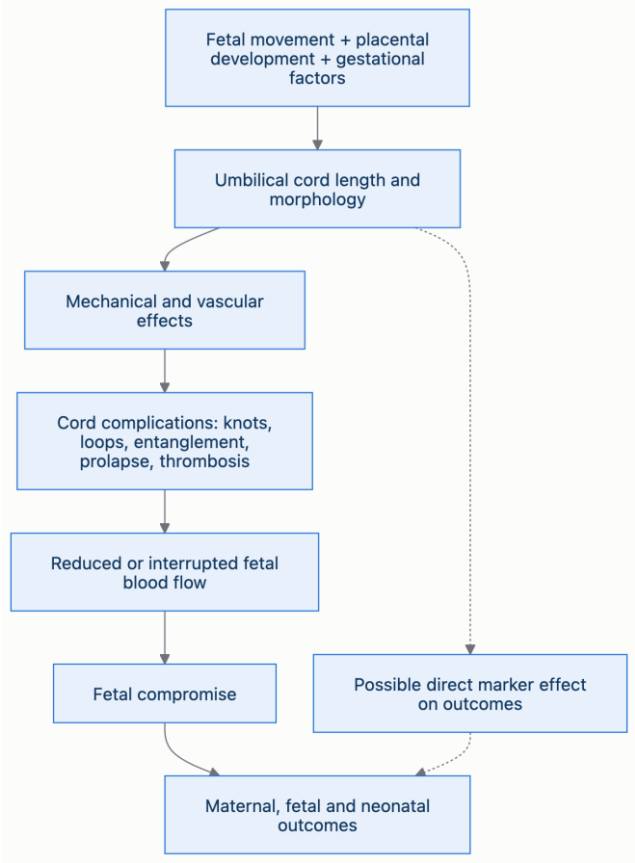



**Figure 2** presents the proposed relationship between abnormal cord length and adverse outcomes.

## 6. UMBILICAL CORD COILING

The normal cord completes roughly one coil per 5 cm, giving a normal umbilical coiling index of 0.17 (±0.009)

spirals per cm; values below the 10th centile (<0.07) define hypocoiling and above the 90th centile (>0.30) hypercoiling.[38]

Both directions of abnormality are associated with adverse outcomes. A meta-analysis of 24 studies involving 9,553 women found hypocoiled cords significantly associated with preterm birth <37 weeks, interventional delivery for fetal distress, meconium-stained liquor, Apgar <7 at 5 minutes, SGA, fetal anomalies, NICU admission, fetal heart rate abnormalities and fetal death, while hypercoiled cords showed a similar profile plus fetal growth restriction and fetal acidosis.[39] Hypocoiled cords have been identified as predictors of interventional delivery and intrapartum fetal heart rate disturbances, and hypercoiled cords with preterm labour, preterm birth and growth restriction.[4,40]

Coiling is clinically attractive because it is visible on ultrasound, and detection of the antenatal UCI during routine care has been recommended; automated measurement using a multi-task neural network has recently been developed.[41,42] Two caveats temper this: mechanical testing found uncoiled cords suffer no more ill effects from compression and twisting than normal cords, and it remains unclear whether abnormal coiling is a cause of pathology, a sequela, or both.[4,38]

## 7. CLINICAL SIGNIFICANCE: SHOULD CORD LENGTH BE PART OF ANTENATAL ASSESSMENT?

This section is deliberately critical rather than advocating routine assessment.

### 7.1 Why Antenatal Assessment is Difficult

The cord is a mobile, convoluted, three-dimensional structure that moves continuously within the amniotic cavity. High fetal mobility, excessive maternal abdominal wall thickness, and inter-observer variability all limit conventional ultrasound.[43] The cord's own coiling, its looping around the fetus and limbs, and variable fetal position make it difficult to follow the entire structure from placental to fetal insertion in a single view. Currently, measurements of umbilical cord length, except as a subjective assessment, are not widely applied in clinical obstetrics.[44]

### 7.2 Direct Ultrasound Measurement

Direct measurement is feasible in early gestation. Cord length has been measured sonographically between 6.1 and 11 weeks with a close linear relationship to menstrual age ($r^2$ = 0.89) and acceptable intra- and interobserver agreement.[45] Strikingly, in that study, 60% of fetuses (9 of 15) with intrauterine demise had cord lengths more than 2 SD below the expected value for menstrual age.[44,45] Beyond the first trimester, however, direct measurement of the whole cord becomes impractical as the structure elongates and coils.

Three-dimensional approaches have been explored. Using 3D ultrasound with VOCAL, mean umbilical cord length at autopsy was 55.8 cm (SD 7.6 cm) and mean sonographically estimated cord volume was 95.5 $cm^3$ (SD 80.6 $cm^3$); cord volume correlated negatively with cord length (Pearson correlation −0.351, p = 0.033; Spearman's rho −0.370, p = 0.024), and the authors concluded that estimated cord volume has no significance in the calculation of cord length and cannot be used as an admission test for delivery.[46] Segmental measurement strategies and 3D post-processing with rotation in X, Y, or Z planes have been proposed to obtain the full cord for measurement, but preliminary data show large variation and numbers remain too limited to produce a nomogram.[44] The fundamental problem remains the gap between estimated and actual cord length.

### 7.3 Indirect Ultrasound Markers

Indirect markers may ultimately be more clinically useful than absolute length. These include the umbilical coiling index, the number of visible loops, nuchal cord, cord insertion abnormalities, umbilical artery Doppler, umbilical vein abnormalities, cord diameter, Wharton's jelly characteristics, and free-floating cord characteristics.

Umbilical cord diameter has shown promise: in a cross-sectional study of 150 women at 20-27 weeks, cord diameter correlated positively with gestational age, femur length, biparietal diameter, head circumference, estimated fetal weight, and Apgar scores at 1 and 5 minutes, with a significant relationship to IUGR.[2] Cord morphometry and Wharton's jelly area, along with umbilical vein calibre, have been described as the most sensitive biometric parameters in IUGR, with worsening umbilical artery resistance in lean cords.[13] Umbilical cord evaluation with sonography includes the appearance, composition, location and size of the cord.[1]

### 7.4 Sonographic Estimation of Cord Length

A cord length index based on counting cord rings in each amniotic fluid quadrant has been proposed for use during routine amniotic fluid evaluation in full-term pregnancies. In 681 women beyond 37 weeks, mean index values for short, normal, and long cords were 2.96, 5.36, and 6.98, respectively; the cut-off index for a short cord was 3.75, with sensitivity and specificity of 67% and 97% for a value ≤3 and 92% and 80% for a value ≤4; the cut-off for a long cord was 6.25, with sensitivity and specificity of 75% and 78% for a value ≥6 and 85% and 64% for a value ≥6.5.[47]

A subsequent prospective cohort of 369 women at ≥37 weeks, however, found that the umbilical cord length index was not a statistically significant predictor of umbilical cord abnormalities, with no significant differences between cord problem groups in cord length (p = 0.42) or cord index (p =

0.28), though the frequency of neck loops was significantly higher among women with cord problems (44.2% vs 25.5%, p = 0.01).[48] This divergence between a promising derivation study and a subsequent validation attempt is characteristic of the field.

The central question remains: Can ultrasound identify a clinically meaningful "abnormally short" or "abnormally long" cord before delivery?

On current evidence, the answer is: not reliably, and not for absolute length. What ultrasound can do which includes detect nuchal loops, characterise coiling, assess cord insertion, measure cord diameter and Wharton's jelly, and evaluate Doppler is better framed as assessment of cord morphology than of cord length.

**Table 5** summarises the ultrasound approaches and their reported performance.

### 7.5 Arguments for Antenatal Assessment

The potential benefits of assessing cord morphology antenatally include risk stratification; identification of cord abnormalities; increased surveillance in selected pregnancies; better recognition of cord accidents; and a possible contribution to stillbirth prevention. Cord abnormalities account for 10% of possible or probable stillbirth causes and deserve further investigation as a potentially preventable cause of stillbirth.[23] Umbilical cord accidents are considered an important cause of stillbirth, and it is argued that screening for UCA could potentially avoid thousands of stillbirths worldwide.[1] Because abnormal coiling is associated with a broad range of adverse outcomes and is detectable antenatally, its detection during routine antenatal care has been recommended.[41] Vasa previa offers the clearest proof of principle that antenatal detection changes outcome: with antenatal diagnosis and planned caesarean delivery before membrane rupture, survival increases to 97–99%, compared with less than 50% when the diagnosis is made only at delivery.[49]

### 7.6 Arguments Against Routine Assessment

Against routine assessment are substantial methodological and clinical concerns. Measurement uncertainty is high: 3D ultrasound estimation of cord volume does not predict cord length, and a proposed cord length index failed validation as a predictor of cord abnormalities.[46,48] Reproducibility and standardisation are lacking, and there are no universally accepted thresholds.[9,14,16] Many cord abnormalities are clinically benign: nuchal cord is present in almost one-fourth of uncomplicated pregnancies and is not itself a cause of death, most nuchal cords have no effect on perinatal outcomes, and cord entanglement does not contribute to prenatal morbidity and mortality in monoamniotic twins.[23-25,31] False-positive findings would be frequent. Most critically, there is no evidence that intervention based on cord findings improves outcomes, and there are no reliable clinical risk factors to predict stillbirth due to cord abnormalities.[50] Diagnosis can also be deceptive and lead to unnecessary interventions, as illustrated by a case in which ambiguous ultrasound findings prompted a caesarean request for suspected fetal malformation.[19]

## 8. CONTROVERSIES AND LIMITATIONS IN THE EXISTING LITERATURE

I. Inconsistent definitions

II. Postnatal measurement: Because cord length cannot be reliably estimated before delivery, most associations in this literature are retrospective correlations rather than predictive relationships. This limits their clinical utility: a variable measured after the outcome cannot inform antenatal management.

III. Confounding: Potential confounders include gestational age, birth weight, fetal growth, placental abnormalities, fetal movement, and maternal disease. Cord length correlates with gestational age and birth weight, both of which independently influence outcomes. Many reported associations may therefore be partly or wholly explained by shared upstream determinants rather than by cord length itself.

IV. Retrospective studies: Much of the evidence base is retrospective, including large database studies and chart reviews. Retrospective design limits the ability to establish temporal sequence and to control for unmeasured confounding.

V. Difficulty establishing causality: The most important limitation is the difficulty of establishing causality. This distinction is central. For example: a nuchal cord is present in almost one-fourth of uncomplicated pregnancies and is not itself a cause of death; cord entanglement does not contribute to prenatal mortality in monoamniotic twins; and excessively long cords may even be associated with reduced IUFD risk in some knot cases.[23,28,31,32] It is also unclear whether abnormal coiling is a cause of pathology, a sequela, or both.[38] The literature therefore supports association far more strongly than it supports causation.

### 8.6 Future Research

The future research agenda rests on three priorities: establishing gestational-age-specific international reference ranges and standardising definitions of short and long cords and of hypo- and hypercoiling by anchoring them to population percentiles rather than arbitrary centimetre thresholds; developing reliable ultrasound techniques,

including 3D imaging and AI-assisted analysis, building on demonstrated volumetric segmentation of fetal structures and emerging 3D approaches to capture the full cord; and conducting prospective cohorts that follow pregnancies from antenatal ultrasound through to delivery, addressing the field's central limitation which is that cord length is usually known only after the outcome it is correlated with. Artificial intelligence is the most promising enabling technology, given that deep learning is already standard in fetal ultrasound analysis and cord-specific tools such as automated coiling index measurement are emerging.

## 8. CONCLUSION

Umbilical cord length is a readily measured but still poorly understood obstetric variable, and the evidence supports three broad conclusions.

First, adverse outcomes cluster at both extremes rather than rising linearly. Short cords are associated with higher rates of caesarean delivery, fetal distress and NICU admission, with unplanned caesarean risk increasing progressively as length falls and placental complications including abruption rising in term births. Long cords act chiefly through mechanical opportunity, increasing the chance of wrapping, looping and knotting, and excessively long cords are significantly associated with entanglement and true knots.

Second, cord length is better understood as a marker than a direct cause. Elongation follows tensile forces generated by fetal movement, so a short cord may partly record reduced fetal mobility.

Third, absolute cord length cannot be assessed antenatally, but cord morphology can. Direct measurement is reliable only in the first trimester; 3D volume estimation does not predict length, and a proposed cord length index was not a statistically significant predictor of cord abnormalities. By contrast, coiling index, diameter and insertion are assessable in routine examinations. The clinical implication is to move from measuring cord length toward a composite assessment of cord morphology and context, supported by standardised definitions, reference ranges and prospective studies linking antenatal imaging to delivery outcomes.

**TABLES**

**Table 1 — Definitions of umbilical cord length across studies**

| Category | Definition used | Source/study |
|---|---|---|
| Short | <35 cm | Berg & Rayburn, cited in Behera et al. [6] |
| Long | >80 cm | Berg & Rayburn, cited in Behera et al. [6] |
| Short / Normal / Long | <35 cm / 35–80 cm / >80 cm | Algreisi et al. [9]; Mehr et al. [14] |
| Short / Medium / Long | <45 cm / 46–90 cm / >90 cm | Rawal et al. [15] |
| Percentile-based | 1st = 33 cm; 10th = 43 cm; 25th = 48 cm; 75th = 63 cm | Yamamoto et al. [16] |

**Table 2 — Determinants of umbilical cord length**

| Factor | Proposed effect | Evidence |
|---|---|---|
| Gestational age | ↑ (positive correlation) | Cord length correlated with gestational age [6]; linear relationship $r^2 = 0.89$ in first trimester [45] |
| Fetal movement | ↑ | Tensile force from movement proposed as main determinant [5–7]; near-term movement influences relative length [8] |
| Fetal size / birth weight | ↑ | Cord length significantly related to birth weight [6]; birth weight higher in long-cord, lower in short-cord groups [9] |
| Male sex | ↑ | Rate of male infants increased in long-cord and decreased in short-cord groups [9]; male sex associated with ELUC [11] |
| Amniotic fluid volume | ↑ | Cord length influenced positively by amniotic fluid volume and fetal mobility [9] |
| Maternal age and gravidity | ↑ | Both increased in long-cord and decreased in short-cord groups [9] |
| Maternal diabetes | ↑ cord size | Cord significantly larger in gestational diabetes; increase attributed to Wharton's jelly [12,13] |
| Genetic factors | Uncertain | Cord length thought influenced by environmental and genetic factors [7]; mothers with ELUC at increased risk of a second long cord [11] |

**Table 3 — Short umbilical cord and obstetric outcomes**

| Outcome | Reported finding | Source |
|---|---|---|
| Unplanned caesarean delivery | Significant difference for all three short-cord groups vs control; odds ratio higher as cord shortened | Yamamoto et al. [16] |
| Emergency / all-cause caesarean | Increased risk in both term and preterm births | Linde et al. [18] |
| Caesarean delivery | 38.8% | Mehr et al. [14] |
| Non-cephalic presentation | Increased risk in term and preterm births | Linde et al. [18] |
| Fetal distress | 29.9% | Mehr et al. [14] |
| Placental abruption | OR 1.98 (95% CI 1.72–2.73) in term births; OR 1.35 (95% CI 1.12–1.62) in preterm births | Linde et al. [18] |
| Placenta previa; manual removal of placenta | Increased risk | Linde et al. [18] |
| Delay in second stage; retained placenta; birth asphyxia; cord rupture; uterine inversion | Reported complications | Njoku et al. [7] |
| NICU admission | 16.4% | Mehr et al. [14] |
| Low birth weight; SGA | 23.1%; 9.7% | Mehr et al. [14] |
| IUFD | 31.25% (vs 11.5% long, 2.7% medium; overall 5%) | Rawal et al. [15] |
| PPROM and preterm birth | Reduced risk when cord was short | Linde et al. [18] |

**Table 4 — Long umbilical cord and obstetric outcomes**

| Outcome | Reported finding | Source |
|---|---|---|
| Cord prolapse | 7.5% | Mehr et al. [14] |
| Abnormal labour progression | 12.7% | Mehr et al. [14] |
| IUFD | 11.5% | Rawal et al. [15] |
| Nuchal cords and true knots | ELUC found in ~4% of pregnancies; risk factor for both | Hanna et al. [19] |
| True knot | Excessively long cord in 46.9% of knot cases; adjusted OR 8.26 (95% CI 6.0–11) | Suzuki [28] |
| Cord entanglement; fetal anomalies; non-reassuring fetal status; respiratory distress | Significantly associated with ELUC | Baergen et al. [11] |
| Brain imaging abnormalities / abnormal neurological follow-up | Significantly increased risk in ELUC infants | Baergen et al. [11] |
| Birth weight; male sex | Increased in long-cord group | Algreisi et al. [9] |
| IUFD (true knot) | 1.8% vs 0.3%; adjusted OR 6.07 (95% CI 4.68–7.86) | Bas Lando et al. [27] |
| IUFD | 0% vs 4.2% without ELUC; not different from no-knot group (0.3%) | Suzuki [28] |

**Table 5 — Ultrasound assessment of umbilical cord morphology**

| Technique / marker | Reported performance | Source |
|---|---|---|
| First-trimester direct cord length | Linear relationship with menstrual age ($r^2$ = 0.89); 60% of demised fetuses >2 SD below expected | Hill et al. [45] |
| 3D ultrasound cord volume | Mean length 55.8 cm (SD 7.6); negative correlation with volume (−0.351, p = 0.033); not usable as admission test | Assimakopoulos et al. [46] |
| Cord length index (quadrant ring count) | Short cord cut-off 3.75: sensitivity/specificity 67%/97% (≤3) and 92%/80% (≤4); long cut-off 6.25: 75%/78% (≥6) and 85%/64% (≥6.5) | Şahin et al. [47] |
| Cord length index (validation) | Not a significant predictor of cord abnormalities (p = 0.42 length; p = 0.28 index); neck loops higher with cord problems (44.2% vs 25.5%, p = 0.01) | Shindeen et al. [48] |
| Umbilical cord diameter | Correlated with GA, FL, BPD, HC, EFW, and Apgar at 1 and 5 min; significant relationship with IUGR | Khalifa et al. [2] |
| Umbilical coiling index | Normal 0.17 ± 0.009 spirals/cm; abnormal <0.07 or >0.30 | de Laat et al. [38] |
| AI-based UCI measurement | +1.2 percentage points mAP@50 vs Deformable DETR-R50 (detection); +3.0 percentage points mAP@50 vs YOLOv11 (keypoints); 2,018 annotated images | Liu et al. [42] |

**Table 6 — Knowledge gaps and future research priorities**

| Gap | Research priority | Supporting evidence |
|---|---|---|
| Inconsistent definitions | Agree on short/long cord and hypocoiling/hypercoiling thresholds | 9,14,15,38,39 |
| No international reference ranges | Develop gestational-age-specific cord length charts | 16 |
| Cord length measured postnatally | Prospective antenatal cohorts linking imaging to outcomes | 46,48 |
| Unreliable antenatal estimation | Validate 3D, automated, and AI-assisted techniques | 42,46,51 |
| Uncertain causality | Distinguish risk factor, marker, mediator, and incidental finding | 28,31,38 |
| No reliable predictors of cord-related stillbirth | Identify clinical and imaging risk factors | 23,50 |
| No evidence intervention improves outcomes | Trial surveillance strategies against outcomes | 50 |

**FIGURES**

**Figure 1.** Development and determinants of umbilical cord length — see Section 3.

**Figure 2.** Proposed relationship between abnormal cord length and adverse obstetric outcomes — see Section 5.

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Note applicable

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