# Fetal heart rate patterns in cerebral palsy associated with umbilical cord troubles: Nationwide cohort study

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**Abstract**

Objective: To clarify the fetal heart rate (FHR) pattern evolution and estimate the timing of brain injury in infants with cerebral palsy (CP) according to each of the umbilical cord (UC) troubles. Design: Longitudinal cohort study using the nationwide registry. Setting: Children with CP who were approved for disability support by the Japan Obstetric Compensation System for CP. Population: 126 infants with severe CP caused by UC troubles with a birth weight of ?2000 g and ?33 weeks of gestational age. Methods: Observers classified the intrapartum FHR strips into five groups: (1) the persistent bradycardia (P-Brady); (2) the persistently non-reassuring (P-NR); (3) Hon's pattern; (4) the reassuring-prolonged deceleration (R-PD); and (5) the persistently reassuring (P-R). Main outcome measures: Timing and type of evolution patterns of abnormal FHR stratified by various UC troubles. Results: Of 126 cases, 32 were associated with umbilical cord prolapse, and 94 were with other UC morphological abnormalities. Overall, nearly half of the eligible cases had intrapartum onset with R-PD (43%). Meanwhile, around half of those associated with UC morphological abnormalities had an antenatal onset. In the intrapartum setting, most of the FHR deteriorations occurred during the first stage of labor, and there was a high incidence of Hon's pattern in the case with velamentous insertion and multiple entanglements. Conclusion: Infants with severe CP associated with the UC troubles were characterized as sudden onset during the first stage of labor. Around half of those due to the UC morphological abnormalities were presumed to have an antenatal onset.

## Introduction

Although intrapartum hypoxia-ischemia is associated with cerebral palsy (CP), many CP cases' etiology remains unknown.[1]A systematic review found that CP associated with intrapartum hypoxia-ischemia accounted for 14.5% of CP in developed countries, and the incidence of CP has persisted over the past 30

years.[1] Placental and umbilical cord abnormalities are likely to induce fetal hypoxia, and the association between CP development and these abnormalities has been previously reported.[2-4]

The pathology of umbilical cord abnormality might be associated with its vulnerability to the external force during uterine contraction, resulting in acute intrapartum hypoxia-ischemia. However, the pattern and extent of deterioration in umbilical blood flow might vary depending on the type of umbilical cord abnormalities. A recently published nationwide study showed that the umbilical cord troubles were responsible for one-fourth of CP in term or near-term infants.[5] This study also classified each CP case into five fetal heart rate (FHR) pattern categories. The FHR pattern evolution suggests the timing of fetal brain injury, which is expected to be critical considerations for the provision of safe delivery. Therefore, we focused on these notable features in elucidating CP associated with the umbilical cord troubles. We hypothesized that the FHR pattern evolution provides clues to prevent cord-related brain damage. The objective was to identify the FHR patterns in patients with CP associated with cord problems.

**Methods**

We performed a longitudinal cohort study using the nationwide registry. Details about our registry were summarized in our previous report.[4] In brief, the subjects included children with CP who were approved for disability support by the age of 5 after a review of the Operating Organization of the Japan Obstetric Compensation System for Cerebral Palsy (JOCSC). The JOCSC covers more than 99% of delivery institutions throughout Japan.

Patients eligible for inclusion in the present study were children born from January 2009 to July 2014, with a birth weight of [?]2000 g, gestational age of [?]33 weeks, and disability due to CP independent of congenital causes or factors during the neonatal period or later. We defined severe CP as the first- or second-degree of disability according to the definitions in the Act on Welfare of Physically Disabled Persons,[4] which are equivalent to level 3 to 5 of the Gross Motor Function Classification System-Expanded and Revised.[6] Multiple pregnancies and cases with poor FHR tracings were excluded from the present study. Among the eligible patients, those associated with umbilical cord troubles were retrieved from reports of the Operating Organization of the JOCSC.

In each case, the FHR strips were obtained and retrospectively analyzed by four authors (J.H., M.N., T.I., and E.J.). The FHR class was determined after careful discussions. The National Institute of Child Health and Human Development guidelines[7,8] were applied when interpreting the FHR patterns. We categorized all the FHR patterns into the following five groups based on the guidelines between the time of admission and delivery, as advocated by Phelan et al.[9]

When an abnormal FHR pattern was observed on admission for delivery or at labor onset, we defined the following two groups. (i) The Persistent bradycardia (P-Brady) group consists of fetuses with severe bradycardia (<80 beats/min) or absent variability with persistent severe decelerations on admission. (ii) The persistently non-reassuring (P-NR) group comprises fetuses with late or variable decelerations or decreased variability without bradycardia on admission test, which persisted until delivery.

In contrast, when the admission test showed a reassuring pattern (moderate variability on a normal baseline rate without late or variable decelerations), the following three groups were defined. (iii) Hon's pattern (Hon) group comprises fetuses with a reassuring FHR pattern on admission and subsequent recurrent severe decelerations with or without an increased baseline rate and decreased variability. Finally, prolonged deceleration (PD) or terminal bradycardia was observed before delivery. (iv) A reassuring-PD (R-PD) group consists of the fetuses with a reassuring FHR pattern on admission; however, an abrupt change to severe PD or bradycardia occurred before delivery. (v) A persistently reassuring group indicates fetuses with a reassuring FHR that remains within ordinary throughout the entire course (Figure 1).

Umbilical cord troubles analyzed in the present study included umbilical cord prolapse, marginal/velamentous cord insertion, multiple cord entanglement, a true knot, umbilical cord constriction, hyper-coiled cord, hypo-coiled cord, and a single umbilical artery. We assessed frequencies of the

FHR evolution patterns in patients with CP associated with umbilical cord troubles.

Definitions

The evidence of the umbilical cord troubles in the present study was obtained from medical records in the individual facilities. And definitions were used regarding the Glossary of Obstetrics and Gynecology published by the Japan Society of Obstetrics and Gynecology.[10]

*Velamentous and marginal cord insertion:* Velamentous or marginal cord insertion is characterized by membranous umbilical vessels at the placental insertion site. In velamentous insertion, the umbilical vessels diverge, surrounded by fetal membranes with no Wharton's jelly at the placental end of the cord. Marginal insertion is the umbilical cord located just at the edge of the placenta.

*Multiple umbilical cord entanglements:* Umbilical cord entanglement is defined when one or more loops of the umbilical cord are encircled around any part of the fetus. In the present study, we included cases with two or more entanglements.

*Umbilical cord constriction/thin cord:* Diagnosis of umbilical cord constriction is made when the umbilical cord has one or more narrow parts by macroscopic evaluation, including umbilical ring constriction, constriction in the free loop, and thin umbilical cord without Wharton's jelly.

*True knot:* A true knot of the umbilical cord is formed when the fetus passes through a loop of the umbilical cord while being active in the uterus.

*Abnormal umbilical coiling:* The umbilical coiling index is calculated by dividing the total number of coils by the cord's length in centimeters. Hyper- and hypo-coiled cord after delivery are defined with umbilical coiling indices of [?]0.3 coils/cm (>90% percentile) and <0.1 coils/cm (<10% percentile).[11]

*Single umbilical artery:* Diagnosis of a single umbilical artery is made macro- or microscopically when one umbilical artery is absent or obstructed.

*Umbilical cord prolapse:* The umbilical cord prolapse is diagnosed clinically when the umbilical cord's free loop protrudes into or outside the vagina.

Statistical analysis

All analyses were conducted using Stata version 16.0 (STATA Corporation, College Station, TX). Continuous or integer variables were reported as mean ± standard deviation or median and range, respectively. Categorical variables were reported as frequencies.

Ethical statement

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. The study protocol was approved by the Institutional Review Boards of the JOCSC (No. 26-01, Aug. 5, 2014). Informed consent was obtained from patients. Although the analysis was retrospective, data for the anonymized JOCSC database were collected in a normal clinical setting, and the confidentiality of the patients involved was protected. All patients' records/information was anonymized and de-identified prior to analysis.

## Results

The study flow diagram was demonstrated in Figure 2. Among 1017 cases of CP from the JOCSC database, 131 were considered to be associated with umbilical cord troubles in singleton pregnancies. Five cases were excluded because of uninterpretable or missing FHR data, and 126 cases were analyzed.

The characteristics of our study cohort were shown in Table 1. 24.6% were complicated with preterm premature rupture of membranes, the trans-cervical balloon catheter was used for cervical ripening in 17.5%, and most cases were delivered by the emergency cesarean section (63.5%). More than half in the umbilical

cord arterial pH was no less than 7.0 (57.8%). However, the Apgar scores were lower than normal in most CP cases (Apgar score <7: at 1 min, 96.8%; at 5 min, 83.9%, respectively)

The FHR classifications and those stratified by the umbilical cord troubles are shown in Figures 3 and 4. Overall, nearly half of CP caused by umbilical cord troubles occurred with a sudden evolution pattern during delivery (R-PD, 43%). On the contrary, approximately one-third of those showed abnormal FHR patterns on admission (P-Brady and P-NR, 33%). The FHR class analysis divided by umbilical cord prolapse and the other morphological abnormalities showed that nearly 90% of CP due to umbilical cord prolapse developed FHR deterioration during labor, particularly with R-PD. Meanwhile, approximately 40% of those associated with cord morphological abnormalities had already shown abnormal FHR patterns on admission, mostly persistently non-reassuring patterns.

Timing and type of evolution patterns of abnormal FHR stratified by umbilical cord troubles are demonstrated in Table 2. In cases of various umbilical cord abnormalities, such as velamentous insertion, true knots, constriction, and hyper-coiled cords, around half of the patients already had FHR alterations at the time of admission. In terms of the intrapartum onset, most FHR deteriorations occurred during the first stage of labor in all types of cord troubles. Furthermore, most patients complicated with velamentous insertion and multiple entanglements developed CP with Hon's pattern.

## Discussion

### Main findings

This study demonstrated that severe CP associated with the umbilical cord troubles were characterized as sudden onset during labor. Meanwhile, around half of those due to cord morphological abnormalities developed with an antenatal onset. Furthermore, most FHR deteriorations occurred during the first stage of labor, and there was a high incidence of Hon's pattern in the case with velamentous insertion and multiple entanglements.

### Strengths and limitations

The strength of this nationwide analysis highlighted the features of FHR evolution patterns in infants with severe CP associated with umbilical cord troubles. We also identified some factors that have the potential of preventing CP based on FHR pattern analysis.

Despite these strengths, the current research also has several limitations. First, this is a retrospective observational study without a control group. Furthermore, we collected only severe CP cases with a birth weight of [?]2000 g and at the gestational age of [?]33 weeks, and we analyzed only available data. Therefore, the present study may have a collection bias. Second, we were not able to infer the associations among CP, umbilical cord abnormalities, fetal growth restriction, and pathological findings of the placenta that were not sufficiently available in this cohort.

We need further analysis, including milder forms of CP cases, to generalize our result. Furthermore, larger case-control studies and prospective cohort studies will be needed to determine whether each umbilical cord trouble itself is associated with the specific FHR pattern.

### Interpretation

Concurring with the previous report,[5] severe CP caused by umbilical cord troubles, especially cord prolapse, was characterized by a sudden onset (R-PD) in the intrapartum. It is not surprising, given these configurations of the umbilical cord lead to vulnerability to compression or torsion during uterine contractions.[12,13] In contrast, we identified a high incidence of CP associated with cord morphological abnormalities developed in an antenatal period, mostly P-NR accounted for 36%. The hyper-coiled cord is reported to be more prone to torsion rather than compression or stretching.[14,15] Furthermore, several histological studies showed the association between umbilical cord abnormalities and placental circulatory stasis or thrombosis.[2,16,17] Such a cumulative effect on fetoplacental blood flow may result in antenatal etiology of brain injury.

We identified that most FHR deteriorations occurred during the first stage of labor and a high incidence of Hon's pattern in the case with umbilical cord structural lesions such as velamentous insertion and multiple entanglements. Several retrospective studies showed the association of variable decelerations during the first stage of labor with umbilical cord abnormalities such as velamentous cord insertion and hyper-coiled cord.[18,19] Moreover, a prospective study showed that morphological abnormalities developed in the earlier gestation were likely to coexist with placental abnormalities that affect the fetoplacental perfusion during labor.[20]Such cumulative distress described above might also arise during repetitive uterine contractions and could develop Hon's pattern. As this pattern evolves over several hours, we may be able to intervene and correct the FHR pattern before severe hypoxia occurs. Therefore, morphological abnormalities of the umbilical cord have the potential of predicting such FHR alterations, and ultrasound screening for them antenatally is crucial to predispose labor and delivery care providers to greater focus on Hon's FHR progression.

On the other hand, the prevention of CP in cases with R-PD might be difficult. A previous nationwide study on the prognosis of infants with umbilical cord prolapse found that the interval from the diagnosis of prolapse to delivery was significantly longer in infants with a poor outcome than those with intact survival (median, 30 vs. 24 minutes).[21] We would not reduce such CP cases unless obstetric facilities throughout the country can provide immediate cesarean delivery within 15 minutes. However, half of the deliveries are managed in a private clinic where the emergency cesarean section is not available.

Although electronic fetal monitoring (EFM) has not yet been shown to be a good predictor of fetal asphyxia,[22,23] previous studies suggested that optimal intrapartum care could offer a preventive opportunity of labor asphyxia in cases with normal admission FHR tracings.[24,25] Furthermore, a retrospective study suggested the combination of EFM with clinical risk factors performed better for screening CP than EFM alone.[26] As a risk screening of umbilical cord troubles before labor management, ultrasound screening, triage of pregnant women according to risks of emergency cesarean section, and appropriate intrapartum management of FHR combined with evolution patterns are recommended.[5,27,28] In case these factors are recognized, the parturients should be placed under continuous observation of FHR with preparing an emergency cesarean section for early intervention to abnormal FHR that may cause fetal brain damage.

Conclusion

Infants with severe CP associated with the umbilical cord troubles were characterized as sudden onset during the first stage of labor. Around half of those due to cord morphological abnormalities were presumed to have an antenatal onset. Prenatal ultrasound screening and meticulous FHR monitoring during the first stage of labor are crucial for early intervention. Focusing on Hon's pattern may help prevent CP in case velamentous insertion and multiple entanglements exist.

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**Conflict of interest:** We declare no competing interests.

**Author contributions statement:** JH, NM, TI, and TI conceived the study. JH wrote the initial protocol, analyzed the data, and wrote the first draft of the manuscript. All authors collected data and analyzed cases of CP. JH, MN, TI, and EJ coordinated the study and JH, MN, TI, and EJ produced the database. Statistical analyses were performed by ST and NT. All authors contributed to writing the manuscript. TI, SS, KI, ST, AN, KF, TM, ST, HS, MI and TI are the guarantors for the study. All authors had full access to all of the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis. The lead author affirms that this manuscript is an honest, accurate, and transparent account of the study being reported; that no important aspects of the study have been omitted; and that any discrepancies from

the study as planned have been explained.

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**Details of ethics approval:** Alll procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. The study protocol was approved by the Institutional Review Boards (IRBs) of the JOCSC (No.26-01, Aug. 5th 2014).

**Data sharing:** No additional data is available.

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**Table 1. Maternal and neonatal background of the study cohort**

| Variables | Variables | **Mean ± SD or median (range) or n (%)** |
|---|---|---|
| Maternal characteristics | Maternal characteristics | |
| | Age – mean ± SD | 31.6 ± 5.4 |
| | Height (cm) – mean ± SD | 157.4 ± 5.8 |
| | Weight at the beginning of pregnancy (kg) – mean ± SD | 52.5 ± 7.8 |
| | BMI (kg/m$^2$) – mean ± SD | 21.2 ± 2.9 |
| | Weight at delivery (kg) – mean ± SD | 62.6 ± 8.2 |
| | Weight gain (kg) – mean ± SD | 10.2 ± 3.9 |
| | Parity – median (range) | 0 (0–4) |
| | *In vitro* fertilization – n (%) | 6 (4.6%) |
| Delivery profiles | Delivery profiles | |
| | Premature rupture of membranes – n (%) | 22 (17.5%) |
| | Use of a cervical balloon catheter – n (%) | 22 (17.5%) |
| | Augmentation – n (%) | 45 (35.7%) |
| | Uterine fundal pressure – n (%) | 27 (21.4%) |
| *Mode* | *Mode* | |
| | Normal spontaneous – n (%) | 24 (19.0%) |
| | Instrumental – n (%) | 22 (17.5%) |
| | Elective CS – n (%) | 0 (0.0%) |
| | Emergency CS – n (%) | 80 (63.5%) |
| *Delivery at* | *Delivery at* | |
| | Hospital – n (%) | 74 (56.4%) |
| | Clinic – n (%) | 51 (38.9%) |
| | Midwifery home – n (%) | 1 (0.8%) |

| Variables | Variables | **Mean ± SD or median (range) or n (%)** |
|---|---|---|
| *Maternal transport after the onset of labor* – n (%) | *Maternal transport after the onset of labor* – n (%) | 9 (7.1%) |
| Neonatal outcomes | Neonatal outcomes | |
| | Gestational weeks – mean ± SD | 38.6 ± 1.9 |
| | Birth weight (g) – mean ± SD | 2861 ± 449 |
| | Birth weight (SD) – mean ± SD | -0.31 ± 0.99 |
| | Male – n (%) | 69 (54.8%) |
| | Apgar score at 1 min – median (range) | 1(0–10) |
| | [?]7 – n/total (%) | 4/126 (3.2%) |
| | 4–6 – n/total (%) | 22/126 (17.5%) |
| | [?]3 – n/total (%) | 100/126 (79.4%) |
| | Apgar score at 5 min – median (range) | 3 (0–10) |
| | [?]7 – n/total (%) | 20/124 (16.1%) |
| | 4–6 – n/total (%) | 40/124 (31.7%) |
| | [?]3 – n/total (%) | 64/124 (50.8%) |
| | Umbilical artery pH – mean ± SD | 7.04 ± 0.22 |
| | >7.20 – n/total (%) | 24/83 (28.9%) |
| | 7.00–7.20 – n/total (%) | 24/83 (28.9%) |
| | <7.00 – n/total (%) | 35/83 (42.2%) |

SD, standard deviation; BMI, body mass index; CS, cesarean section.

**Table 2. Timing and type of evolution patterns of abnormal fetal heart rate stratified by umbilical cord troubles**

| Umbilical cord troubles | Abnormal FHR on admission | Abnormal FHR on admission | Abnormal FHR on admission | Abnormal FHR during labor | Abnormal FHR during labor | Abnormal FHR during labor | Abnormal FHR during labor | Abnormal FHR during labor | Pe rea thr ou (P- |
|---|---|---|---|---|---|---|---|---|---|
| | | **P-Brady** | **P-NR** | | 1st stage | 2nd stage | **Hon** | **R-PD** | |
| Umbilical cord prolapse (n = 32) | **4 (13%)** | 100% | 0% | **28 (88%)** | 96% | 4% | 21% | 79% | **0 (0** |
| Marginal insertion (n = 33) | **10 (30%)** | 20% | 80% | **21 (64%)** | 95% | 5% | 33% | 67% | **2 (6** |
| Velamentous insertion (n = 10) | **4 (40%)** | 0% | 100% | **4 (40%)** | 75% | 25% | 75% | 25% | **2 (2** |

| Umbilical cord troubles | Abnormal FHR on admission | Abnormal FHR on admission | Abnormal FHR on admission | Abnormal FHR during labor | Abnormal FHR during labor | Abnormal FHR during labor | Abnormal FHR during labor | Abnormal FHR during labor | Pe rea thr ou (P- |
|---|---|---|---|---|---|---|---|---|---|
| Multiple cord entanglements (n = 28) | **10 (36%)** | 20% | 80% | **13 (46%)** | 69% | 31% | 54% | 46% | **5 (1** |
| True knots (n = 5) | **3 (60%)** | 0% | 100% | **1 (20%)** | 100% | 0% | 0% | 100% | **1 (2** |
| Cord constriction (n = 18) | **7 (39%)** | 29% | 71% | **10 (56%)** | 100% | 0% | 20% | 80% | **1 (6** |
| Hypercoiled cord (n = 20) | **14 (70%)** | 0% | 100% | **6 (30%)** | 83% | 17% | 0% | 100% | **0 (0** |
| Hypocoiled cord (n = 3) | **1 (33%)** | 0% | 100% | **2 (67%)** | 100% | 0% | 0% | 100% | **0 (0** |
| Single umbilical artery (n = 7) | **2 (29%)** | 0% | 100% | **4 (57%)** | 100% | 0% | 0% | 100% | **1 (1** |

P-Brady, persistent bradycardia; P-NR, persistently non-reassuring; R-PD, reassuring-prolonged deceleration; P-R, persistently reassuring

**Figure legends**

**Figure 1:** Study flow diagram

JOCSC, Japan Obstetric Compensation System for cerebral palsy; CP, cerebral palsy; FHR, fetal heart rate

**Figure 2:** Fetal heart rate evolution patterns and the estimated timing of brain injury (adapted from Parer JT, King TL, Ikeda T. Electronic fetal heart rate monitoring: the 5-tier system 3rd ed, Burlington, MA: Jones & Barlett Learning, 2018.)

P-Brady, severe bradycardia (<80 beats/min) or absent variability with persistent severe decelerations on admission; P-NR: recurrent decelerations or a decreased variability without severe bradycardia on admission and persisted until delivery; Hon, reassuring on admission and deteriorate gradually followed by a decline in baseline fetal heart rate; R-PD, reassuring on admission and an abrupt change to severe prolonged deceleration or bradycardia occurred before delivery; P-R, reassuring on admission and remained within ordinary pattern during the entire course.

P-Brady, persistent bradycardia; P-NR, persistently non-reassuring; R-PD, reassuring-prolonged deceleration; P-R, persistently reassuring

**Figure 3:** Analysis of fetal heart rate evolution patterns

P-Brady, persistent bradycardia; P-NR, persistently non-reassuring; R-PD, reassuring-prolonged deceleration; P-R, persistently reassuring

**Figure 4:** Analysis of fetal heart rate evolution patterns stratified by umbilical cord troubles

P-Brady, persistent bradycardia; P-NR, persistently non-reassuring; R-PD, reassuring-prolonged deceleration; P-R, persistently reassuring

**Figure 1.**

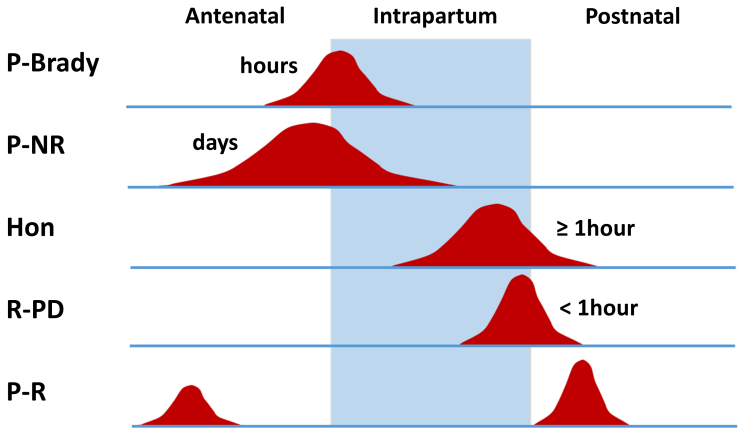


**Figure 2.**

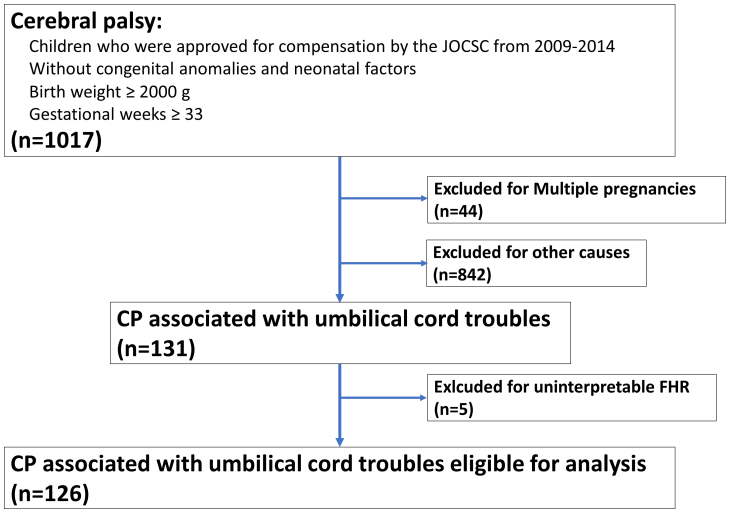

**Figure 3.**

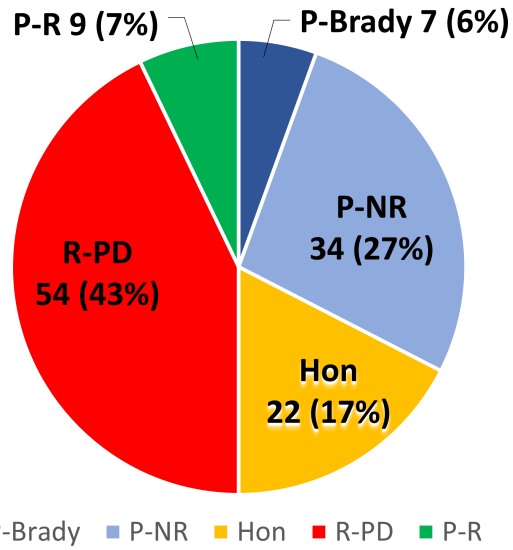


**Figure 4.**