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Longitudinal Study of Oropharyngeal Dysphagia in Preschool Children with Cerebral Palsy

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Running Head: Dysphagia in Children with Cerebral Palsy

Title: Longitudinal Study of Oropharyngeal Dysphagia in Preschool Children with Cerebral Palsy

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Longitudinal Study of Oropharyngeal Dysphagia in Preschool Children with Cerebral Palsy

Abstract

Objective: To determine changes in prevalence and severity of oropharyngeal dysphagia (OPD) in children with cerebral palsy (CP) and relationship to health outcomes.

Design: Longitudinal cohort study.

Setting: Community and tertiary institutions.

Participants: 53 children with confirmed CP diagnosis assessed first at 18-24 months (Ax1 mean age 22.9 months c.a. (SD=2.9), 33 males, Gross Motor Function Classification System (GMFCS) I=22, II=7, III=11, IV=5, V=8) and at 36 months (Ax2).

Interventions: none

Main Outcome Measures: OPD was classified using the Dysphagia Disorders Survey (DDS) and signs suggestive of pharyngeal dysphagia. Nutritional status was measured using Z-scores for weight, height, and body mass index (BMI). Gross motor skills were classified on GMFCS and motor type/ distribution.

Results: Prevalence of OPD reduced from 62% to 59% between ages. 30% of children had an improvement in severity of OPD (>smallest detectable change), and 4% had worse OPD. Gross motor function was strongly associated with OPD at both assessments, on the DDS (Ax1 OR=20.3, p=0.011; Ax2 OR=28.9, p=0.002), pharyngeal signs (Ax 1 OR=10.6, p=0.007; Ax2 OR=15.8, p=0.003), and OPD severity (Ax1 β=6.1, p<0.001; Ax2 β=5.5 p<0.001). OPD at 18-24 months was related to health outcomes at 36 months: low Z-scores for weight (adj β=1.2, p=0.03) and BMI (adj β=1.1, p=0.048), increased parent stress (adj OR=1.1, p=0.049).

Conclusions: Classification and severity of OPD remained relatively stable between 18-24 months and 36 months. Gross motor function was the best predictor of OPD. These findings contribute to developing more effective screening processes which consider critical developmental transitions which are anticipated to present challenges for children from each of the GMFCS levels.

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Key words: deglutition disorders, dysphagia, feeding, cerebral palsy, longitudinal

Abbreviations:

CP – Cerebral Palsy; DDS – Dysphagia Disorders Survey; GMFCS – Gross Motor Function Classification System; GNPA – Growth, Nutrition and Physical Activity (study); OPD – Oropharyngeal Dysphagia; OR – Odds Ratio

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Oropharyngeal dysphagia (OPD) is common in approximately 85% of preschool children with cerebral palsy (CP),[1] although this estimate may be lower when accounting for feeding limitations associated with typical development.[2] CP is a lifelong disability of central origin influencing motor control, including that needed for effective and efficient eating, drinking and saliva control.[3] Impaired feeding, or OPD, is characterised by difficulties in one or more phases of swallowing (including oral-preparatory, oral propulsive, or pharyngeal).[4] A number of important health outcomes have been associated with OPD, such as restricted growth and nutrition, compromised respiratory health, and increased parental stress during mealtimes.[5-7]

Children's feeding skills typically undergo a series of important changes through the preschool years, from suckle-feeding in infancy, to the rapid oropharyngeal skill changes and encephalization during transitional feeding (4-36 months), and finally a period of skill consolidation (3-6 years).[8-10] The range of food textures and fluid utensils children can safely, efficiently and independently manage are gradually expanded owing to a range of influences, particularly the development of children's oropharyngeal sensorimotor systems. By 18-24 months, children can typically ingest firm and dual-textured foods[10,11] and from 24-36 months they can regularly drink from an open cup.[11] These periods of feeding development may present varied challenges for children with CP, as more complex textures, greater volumes of intake, more challenging utensils and increased mealtime independence/ routines place additional requirements on their oral sensorimotor, swallow-respiratory and cognitive systems.

Previous research supports the supposition that much of the OPD in children with CP has persisted since infancy, including reports of early difficulties with sucking, swallowing, or transition to solid foods.[12-15] Despite this, OPD may emerge during childhood in children with normal feeding in infancy,[12] and those presenting with difficulties in infancy may proceed to have typical feeding in childhood.[15-17] There has been limited exploration of

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longitudinal changes to feeding during the preschool years in children with CP. The feeding skill progression of 23 children with CP was explored in a study by Clancy and colleagues, collecting information through parent-report from 4-7 years.[18] This study found significant differences in the proportion of impaired feeding skills between OPD severity groups (except for coughing/ choking), but only coughing reduced longitudinally.[18] Clancy's study emphasised the need for longitudinal research in children younger than four years in order to facilitate earlier intervention. The aim of the present longitudinal study, therefore, was to explore change in OPD prevalence and patterns in children with CP between two critical time points, 18-24 months and 36 months. Further, we aimed to understand whether feeding at 18-24 months could predict health outcomes (nutritional, respiratory and parent stress) at 36 months. Before evaluating change in OPD classification and severity, the test-retest reproducibility of measures had to be established. It was hypothesised that children with ambulatory CP may have delayed feeding at 18-24 months, but by 36 months fewer children would be classified as having OPD.

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Methods

This longitudinal cohort study of preschool-aged children with CP was conducted in Queensland, Australia between April 2009 and April 2013. It is part of two larger studies exploring relationships between growth, nutrition and physical activity[19] and brain structure and motor function in children with CP.[20] All caregivers consented for their child to participate with relevant institutional ethics gained.[19-21]

Participants

Children with a confirmed diagnosis of CP, aged 18-24 months corrected age (c.a.) at initial assessment, and born in Queensland between 2006-2009, were invited to participate. Only children returning for assessment at 36 months c.a. were included in this paper. Children with neurodegenerative conditions were excluded.

Forty children participated in the reproducibility sub-study, aged between 18-36 months c.a. and having a confirmed diagnosis of CP and (n=4 per GMFCS level per age band, stratified to 18-24 and 30-36 months). This sample was recruited primarily through the main study sample, and additional children recruited through the CP Health Service, Royal Children's Hospital, Brisbane.

Measures

Measures of Oropharyngeal Dysphagia

Three standardized clinical measures of OPD were selected following systematic review of measure psychometrics (Dysphagia Disorders Survey -- Pediatric (DDS), Schedule for Oral Motor Assessment and Pre Speech Assessment Scale.[21,22] A subsequent reproducibility and validity study resulted in the selection of the DDS with modified cut-points as the best

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available measure of OPD for research in preschool children with CP.[2] The DDS part 2 consists of a series of binary judgments of feeding competency on eight ingestion functions for puree, chewable food and fluid giving a maximum impairment raw score out of 22.[23,24]

Observation of 16 clinical signs suggestive of pharyngeal phase impairment was included in the determination of OPD classification, as the DDS provides insufficient detail on this phase of swallowing.[1] As this was a population-based study, Videofluoroscopic Swallow Study was not feasible as the standard evaluation due to ethical considerations. As such, observations of clinical signs suggestive of pharyngeal phase impairment using a standardised clinical tool were a proxy for direct assessment of the pharyngeal phase. OPD classification was based on presence of one or more signs, with the exception of a single cough on thin fluids.[25]

Two secondary measures of OPD were included as early predictors of health outcomes. Feeding efficiency was calculated from average intake (grams) and time (minutes), recorded on a three day weighed diet record completed by parents at home.[19] Challenging behaviours demonstrated regularly during feeding (at least once daily) were reported by parents using the CP Child Feeding Questionnaire (CPFQ, Supplementary 1, Question 6). The total number of challenging behaviours, out of 16, was used to indicate possible sensory or behavioural feeding difficulties.

Risk Factors for Oropharyngeal Dysphagia

Children were classified on the Gross Motor Function Classification System (GMFCS) according to their age using the <2 years and 2-4 year age bands.[26] Motor type (spasticity, dyskinesia, hypotonia/ ataxia) and distribution (number of limbs) were also classified.[27,28]

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Socioeconomic status (SES) was measured using the Socio-Economic Indexes for Areas (SEIFA), Index of Relative Socio-Economic Disadvantage[29] which assigns families to a decile rank (from 1= most disadvantaged to 10= least disadvantaged) based on family's postcode of residence. Preterm status was indicated for births with gestational age less than 37 weeks (time between first day of the last menstrual period and child's date of birth).[30] Presence of epilepsy was collected from parents during the initial physician interview.[20]

Measures of Health Outcomes

Nutritional status was indicated by gender- and age-referenced Z scores for height, weight and body mass index (BMI).[31] Height or length (depending on children's ability to stand) was measured to the last completed millimetre by a length board (Shorr Productions, Maryland USA). Height was estimated using published equations from knee length or upper-arm length[32] measured with an anthropometer (Holtain Ltd, UK) when direct measures were not possible. Weight was measured to the nearest 100 grams using chair scales (Seca, Germany), and BMI calculated as weight/ height (metres)^2.

Children's feeding method was reported by parents on a five-point ordinal scale on the CPFQ (from total oral intake to total tube-feeding; SI1, Question 12). Parent stress associated with feeding their child was self-reported on a five-point ordinal scale on the CPFQ (SI1, Question 7a). Respiratory illness was indicated by a hospitalisation for chest infection, diagnosis of pneumonia and/ or respiratory infection in the six months prior to assessment.[19-21]

Procedures

Children attended the hospital for anthropometry, mealtime and gross motor function assessments. During the mealtime assessment (videoed for rating by a paediatric speech

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pathologist), three standardized presentations of four textures (puree, lumpy, chewable and fluid) were given by the carer, using their regular utensils.[33] Growth anthropometry was measured by trained researchers, and gross motor function classifications conducted by two physiotherapists.

Reproducibility Sub-Study

For the test-retest reproducibility sub-study, children were seen twice within a month for mealtime assessment. On both occasions the same procedures were followed and the same battery of tests conducted. The time, location and foods were kept as consistent as possible. Reproducibility was analysed using percentage agreement, kappas (binary) and Intra-Class Correlation Coefficients (ordinal scales >five groups). The smallest detectable change (SDC) was calculated for the DDS raw score to determine score change that constituted true change in OPD (classification or severity). Clinical signs with agreement <80% were excluded from the definition of change for pharyngeal phase OPD.

Statistical Analysis

Participant characteristics, including OPD prevalence, were presented descriptively for both assessments, and change reported as a percentage and using McNemar's Test (binary), Wilcoxon Matched Pairs Test (ordinal) and paired T-test (continuous). Potential OPD risk factors (age, gender, GMFCS (collapsed I-II, III, IV-V), BMI Z score, preterm status, epilepsy, SES) were explored through mixed effects logistic regression for the presence of OPD outcomes (on the DDS and pharyngeal signs) and using mixed effects linear regression for OPD severity (DDS raw score). All models included 'participant' as a random effect to account for within-participant dependence across the two assessment points, and 'appointment' and 'GMFCS' as interaction terms. First, univariate models were run, then

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multivariate models, using the above-listed risk factors as fixed effects. Association between OPD variables at 18-24 months and health outcomes at 36 months (nutritional status, introduction of supplementary feeding/ gastrostomy, parent stress and hospitalisation for chest infection) were explored using logistic regression (binary outcomes) and linear regression (continuous outcomes). These models were adjusted for collapsed GMFCS at 36 months and gender. All analyses were performed using Stata 10.0 (Statacorp 2007), with significance set at $p<0.05$.

Results

Sample Characteristics

There were 53 children who participated, aged 22.9 months (SD=2.9) at initial assessment (see Supplementary Information 2 for recruitment pathways and missing data). Sample characteristics at each assessment and change between assessments are reported in Table 1. The sample's motor type distribution was not significantly different from the Australian CP Register at both assessments (Ax1: p=0.81; Ax2: p=0.37, chi-square test), although GMFCS classification differed at the second assessment (Ax1: p=0.09; Ax2: p=0.001, chi-square test).

Test-Retest Reproducibility

Reproducibility of the DDS overall was strong, and for clinical signs was moderate, as shown in Supplementary Information 3 (including data from SOMA and PSAS). Using the modified cut-points,[2] reproducibility for the DDS improved, with 90% agreement (kappa=0.8, $p<0.001$). The variability within the child's performance between mealtimes was greater than that attributable to intra-rater variability[2] (Figure 1 for measurement error and SDC). Coughing was the most variable sign between mealtimes, with 60% agreement (kappa=0.2, p=0.10).

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Prevalence of OPD

The prevalence of OPD reduced from 62% (n=33) at 18-24 months to 59% (n=31) at 36 months, as shown in Figure 2 (see SI3 for information on change based on the SOMA, PSAS and unmodified scoring). Four children changed from having OPD at 18-24 months to having no OPD at 36 months (all GMFCS I), and two children gained a classification of OPD at the second assessment (one each from GMFCS I and III). Decline in OPD status was related to the presence of clinical signs suggestive of pharyngeal phase impairments at assessment 2.

The change in DDS scores overall, and on specific items (according to gross motor function) is shown in Figure 1 and Supplementary 4, respectively. Fourteen children (30%) had an improvement in DDS score greater than that attributable to the test-retest SDC, and two children (4%) had a greater decline in scores.

Risk factors for oropharyngeal dysphagia and association with health outcomes

Gross motor function was the only risk factor for OPD that persisted between assessment 1 and 2 (Table 2). Age and epilepsy were also related to certain OPD outcomes and at certain assessment points. The relationship between OPD variables at 18-24 months and associated health outcomes at 36 months are reported in Table 3.

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Discussion

The classification and severity of OPD remained relatively stable between 18-24 months and 36 months, when removing classification error based on intra-child variability and limitations associated with typical development. The marginal reduction in OPD was seen as children with ambulatory CP (GMFCS I) matured. The modified OPD classification[2] accounts for the degree of maturation associated with typical development in the measure scores. Considering this, the change in OPD classification on the DDS may reflect later maturation of oral sensorimotor feeding skills in children with CP (particularly GMFCS I) compared to children with typical development.

The presence of an OPD classification did not change for children from GMFCS II-V following their second birthday, although OPD severity reduced in almost a third of children. The greatest and most frequent improvement in OPD severity was seen in children from GMFCS IV (on average 4.3 points). This may in part be due to small numbers in this group (n=4 with a DDS raw score), but may also relate to their heterogeneity in feeding skills.[34] Children from GMFCS IV also showed the greatest improvement of specific ingestion functions, which was particularly evident on pureed foods.

Children from GMFCS V appeared to reach their ceiling of performance for purees by 18-24 months (with all children impaired on all items, and no change between assessments). Interestingly more children from GMFCS V showed impairment on ingestion functions for chewable foods at 36 months compared to 18-24 months. This is perhaps due to the introduction of more challenging chewable foods between these ages for children from GMFCS V. Similarly, more children from GMFCS III were impaired on fluid items ‘containment’ (fluid loss) and ‘post-swallow’ (coughing or wet respiration/ phonation) at 36 months. This may be explained by more children from this group using modified utensils at

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18-24 months, but graduating to open cups or consecutive fluid swallows by 36 months. The developmental trajectories described in the gross motor literature,[35] suggest that children with poorer gross motor function will reach their functional capacity earlier than those with better gross motor function, which was reflected in our data. Gross motor function remained the best predictor of OPD classification and severity, being the only risk factor associated with each OPD outcome and at both assessment points.

During the twelve to eighteen months between assessments, there were minimal changes in health outcomes. Regarding feeding method, only one child who was fed orally (with modifications) at 18-24 months regressed onto tube feeding, and one who was predominately tube-fed transitioned to total tube-feeds. By three years, 9% of our sample received tube-feeding (a third of children from GMFCS IV-V), which was similar to average rates reported in a large multi-register study across six European countries (11%).[36] Regarding growth measures, on average children's weight- and BMI-for-age Z scores reduced marginally by the second assessment, but height increased.

In order to facilitate earlier health management for children with CP, we were interested in understanding associations between OPD at 18-24 months and health outcomes at 36 months. Weight and BMI Z scores were related to the presence of OPD on the DDS (using modified cut-points). This supports the construct validity of the DDS as a measure which is detecting children at risk of later poor nutritional status. There were 27 children in our sample identified as having OPD on the DDS who were not underweight (BMI≤2SD), and three children without OPD who were underweight. Hence the DDS cannot be used in isolation from a comprehensive mealtime and nutritional assessment for indicating children at risk of poor growth. Children of parents who experienced stress during mealtimes (at 36 months) demonstrated a significantly greater number of challenging behaviours during meals (at 18-24 months), but this was not related to OPD on the DDS. Children's active resistance to

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mealtimes perhaps increases the likelihood of stressful mealtimes for parents, rather than the child's motor difficulty during ingestion.

Study Limitations

This study is the first, to our knowledge, to explore changes to OPD prevalence and severity in transitional feeders with CP. It also contributed novel information regarding risk factors for OPD, and the relationship between early OPD and later health outcomes. This study had some limitations which may have influenced the interpretation of findings. The measurement of OPD using the DDS has been strengthened through conducting validation against children with typical development, and testing its reproducibility, in particular test-retest reliability. This provided information regarding the margin of error associated with repeated measures, as well as between-mealtime child variability in scores. While our findings were reported accounting for these differences, it is possible that the measurement error obscured some of the sensitivity of the DDS to detect change in feeding performance, and as such may represent a more conservative estimate of change.

Exploring OPD in 18 month-old children with CP restricted our sample size, as many participants only entered the study at 30-36 months, with CP diagnosis on average only occurring at 13.3 months.[37] Many of the health outcomes of interest, such as gastrostomy feeding and hospitalisation for chest infection were present in only a small subset of the sample. While a strength of this study was our ability to explore relationships with a direct OPD measure, future register-based studies may strengthen our preliminary clinical findings in understanding risk between early OPD and later health outcomes.

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Conclusion

The GMFCS remained a strong risk factor for OPD presence and severity. Raising awareness of this relationship for early intervention clinicians may assist in earlier screening and referral to feeding/ nutritional interventions. A more conservative monitoring approach should be taken for children classified as GMFCS I with apparent OPD before two years, as many of these children's skills appear continue to mature up to three years. OPD classification remained consistent between 18-24 and 36 months for most children from GMFCS III-V. Many children from GMFCS III-IV showed improvements in OPD severity, suggesting this group may be prioritised for feeding interventions from as young as 18 months, even if OPD is mild. Children classified as GMFCS V tended to show minimal change after 18-24 months, and as such, approaches focusing on safety and nutritional efficiency should be prioritised. These findings may also facilitate more appropriately targeted nutritional/ feeding interventions considering their influence on health. The presence of OPD at 18-24 months had the greatest influence on nutritional status at 36 months, but OPD severity did not. This suggests improving feeding skills alone may be insufficient to influence growth outcomes, and as such, interventions should holistically consider dietary intake in addition to oral sensorimotor skill development.

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Figure 1: Change in oropharyngeal dysphagia severity (Dysphagia Disorders Survey raw score) between assessment 1 (18-24 months) and assessment 2 (36 months), according to gross motor function (GMFCS) at assessment 1

Key: Dashed line indicates Smallest Detectable Change for test-retest (measurement error=1.4, smallest detectable change=3.8); solid line represents smallest detectable change for intra-rater,2 (measurement error=1.0, smallest detectable change=2.8); mean change for GMFCS I=1.6 (SD=4.1), II=2.2 (SD=1.2), III=2.3 (SD=2.4), IV=4.3 (SD=3.3), V=1.3 (SD=2.9), but these differences were not significant on linear regression (p=0.67)

Figure 2: Change in oropharyngeal dysphagia classification between assessment 1 (18-24 months) and assessment 2 (36 months), according to gross motor function (GMFCS) at assessment 1

Fig. 1A OPD classification on DDS, Fig. 1B OPD classification on pharyngeal signs; Box indicates children who were reclassified; OPD classification based on modified cut-points2; GMFCS shown is classification at 18-24 months; Different numbers of children had a DDS score calculable between their 18-24 month assessment and 36 month assessment (n=1 GMFCS II, n=2 GMFCS III)

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Table 1. Characteristics of Preschool-aged Children with Cerebral Palsy in the Longitudinal Oropharyngeal Dysphagia Study

	18-24 months n(%)	**36 months n(%)**	**n (%) change***	**Statistic (p value)†‡§**
Gender, males:	33 (62%)	n/a	n/a	
GMFCS level:			10 (19%)	(1.00)†
I	22 (42%)	26 (49%)	0 (0%)	
II	7 (13%)	1 (2%)	6 (86%)	
III	11 (21%)	11 (21%)	2 (18%)	
IV	5 (9%)	7 (13%)	1 (20%)	
V	8 (15%)	8 (15%)	1 (13%)	
Primary motor type:			6 (11%)	(0.55)†
Spasticity	47 (88%)	47 (88)	5 (11%)	
Dyskinesia	2 (4%)	4 (8%)	0 (0%)	
Ataxia	3 (6%)	0 (0%)	3 (100%)	
Hypotonia	1 (2%)	2 (4%)	0 (0%)	
Motor distribution			5 (9%)	(0.18)†
Unilateral	18 (34%)	16 (30%)	2 (11%)	
Diplegia	10 (19%)	10 (19%)	2 (20%)	
Triplegia/ Quadraplegia	25 (47%)	27 (51%)	1 (4%)	
Preterm birth (<37 weeks)	28 (53%)	n/a	n/a	n/a
Epilepsy	9 (19.0%)	n/a	n/a	n/a
Socio-Economic Status (SEIFA)		n/a	n/a	n/a
Least disadvantaged (8-10)	14 (26%)			
Moderate disadvantage (5-7)	27 (51%)			
Most disadvantaged (1-4)	12 (23%)			
Tube/ supplementary feeding				(0.31)†
Full oral	34 (64%)	40 (75%)	3 (9%)	
Supplementary	15 (28%)	8 (15%)	10 (67%)	
Partial tube (mostly oral)	0 (0%)	0 (0%)	0 (0%)	
Partial tube (mostly tube)	3 (6%)	3 (6%)	1 (33%)	
Non-oral	1 (2%)	2 (4%)	0 (0%)	
Height for age Z score (mean, SD)	-0.9 (1.9)	-0.7 (1.2)	0.2	1.1 (0.28)‡
Weight for age Z score (mean, SD)	-0.4 (1.6)	-0.6 (1.5)	-0.2	**-1.9 (0.07)‡**
BMI Z score (mean, SD)	0.0 (1.9)	-0.2 (1.5)	-0.2	-0.7 (0.49)‡
Respiratory illness				
Hospitalization for chest infection	4 (8%)	5 (9%)	7 (13%)	0.14 (0.71)§
Pneumonia	3 (6%)	2 (4%)	5 (10%)	0.2 (0.66)§
Respiratory infection	30 (57%)	23 (44%)	21 (40%)	2.3 (0.13)§

*Percentage change calculated based on number of children reclassified between assessment 1 and 2, divided by number in original group (assessment 1); †Wilcoxon Matched Pairs Test; ‡Paired t test; §McNemars Test; BMI Body Mass Index; GMFCS indicates Gross Motor Function Classification System; n/a not applicable or available; SD Standard Deviation; SEIFA Socio-Economic Indexes for Areas

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Table 2. Comparison of Oropharyngeal Dysphagia Risk Factors at 18-24 months c.a. (Assessment 1) and 36 months c.a. (Assessment 2)

	Assessment at 18-24 months	**Assessment at 36 months**
OPD on DDS (modified)	**OR (95% CI); p value**	**OR (95% CI); p value**
GMFCS (collapsed)	**20.3 (2.0, 208.4); 0.011**	**28.9 (3.4, 248.8); 0.002**
I-II	ref	ref
III	8.1 (0.6, 117.7); 0.13	**23.5 (1.3, 418.5); 0.032**
IV-V	NC	NC
Motor type (collapsed)	NC	NC
Spasticity	ref	ref
Dyskinetic	2.3 (0.1, 69.6); 0.64	NC
Hypotonia/ ataxic	NC	NC
Body Mass Index Z score	1.0 (0.5, 1.7); 0.89	0.7 (0.3, 1.5); 0.35
Preterm	0.4 (0.0, 6.4); 0.48	0.4 (0.2, 5.9); 0.49
Age	1.0 (0.7, 1.7); 0.86	0.2 (0.0, 1.3); 0.09
Gender (ref male)	0.8 (0.1, 14.9); 0.90	0.5 (0.0, 8.8); 0.66
Socio-economic status	1.0 (0.6, 1.8); 0.97	1.3 (0.7, 2.3); 0.46
Epilepsy	NC	NC
OPD severity (DDS raw score)	**β (95% CI); p value**	**β (95% CI); p value**
GMFCS (collapsed)	**6.1 (4.6, 7.6); <0.001**	**5.5 (4.0, 7.0); <0.001**
I-II	ref	ref
III	**4.1 (1.0, 7.2); 0.009**	2.5 (-0.6, 5.5); 0.11
IV-V	**12.9 (10.0, 15.9); <0.001**	**11.7 (8.8, 14.5); <0.001**
Motor type (collapsed)	0.3 (-1.6, 2.2); 0.77	1.7 (-0.4, 3.8); 0.10
Spasticity	ref	ref
Dyskinetic	1.2 (-4.7, 7.1); 0.69	2.7 (-1.6, 7.0); 0.22
Hypotonia/ ataxic	0.4 (-3.7, 4.6); 0.83	3.0 (-2.0, 8.0); 0.23
Body Mass Index Z score	0.0 (-0.5, 0.5); 0.98	-0.4 (-1.0, 0.3); 0.24
Preterm	-0.4 (-4.4, 3.6); 0.86	-1.1 (-5.1, 2.9); 0.58
Age	-0.1 (-0.4, 0.3); 0.70	-0.78 (-1.9, 0.4); 0.18
Gender (ref male)	-0.8 (-5.0, 3.3); 0.70	-4.1 (-4.5, 3.7); 0.85
Socio-economic status	0.1 (-0.7, 0.9); 0.82	0.3 (-0.5, 1.1); 0.48
Epilepsy	**8.1 (3.3, 12.8); 0.001**	**7.5 (2.7, 12.2); 0.002**
OPD of pharyngeal phase	**OR (95% CI); p value**	**OR (95% CI); p value**
GMFCS (collapsed)	**10.6 (1.9, 59.2); 0.007**	**15.8 (2.5, 99.8); 0.003**
I-II	ref	ref
III	**8.6 (0.7, 105.3); 0.09**	3.4 (0.3, 34.1); 0.31
IV-V	NC	NC
Motor type (collapsed)	1.9 (0.3, 11.1); 0.46	2.5 (0.3, 24.1); 0.42
Spasticity	ref	ref
Dyskinetic	NC	NC
Hypotonia/ ataxic	2.3 (0.1, 69.6); 0.64	1.3 (0.0, 176.6); 0.93
Body Mass Index Z score	1.5 (0.8, 2.6); 0.18	1.0 (0.5, 2.0); 0.90
Preterm	0.8 (0.1, 7.3); 0.87	1.1 (0.1, 10.0); 0.91
Age	**0.7 (0.4, 1.0); 0.06**	0.4 (0.1, 1.5); 0.19
Gender (ref male)	2.0 (0.2, 19.1); 0.55	1.3 (0.1, 12.4); 0.81
Socio-economic status	1.2 (0.8, 1.9); 0.47	1.3 (0.8, 2.0); 0.31
Epilepsy	**52.7 (1.1, 2433.9); 0.04**	43.0 (1.0, 1901.6); 0.052

BMI Body Mass Index; DDS Dysphagia Disorders Survey; GMFCS Gross Motor Function Classification System; OPD Oropharyngeal Dysphagia; NC Not calculable as exposures predict outcome perfectly; OR Odds Ratio; SEIFA Socio-Economic Indexes for Areas

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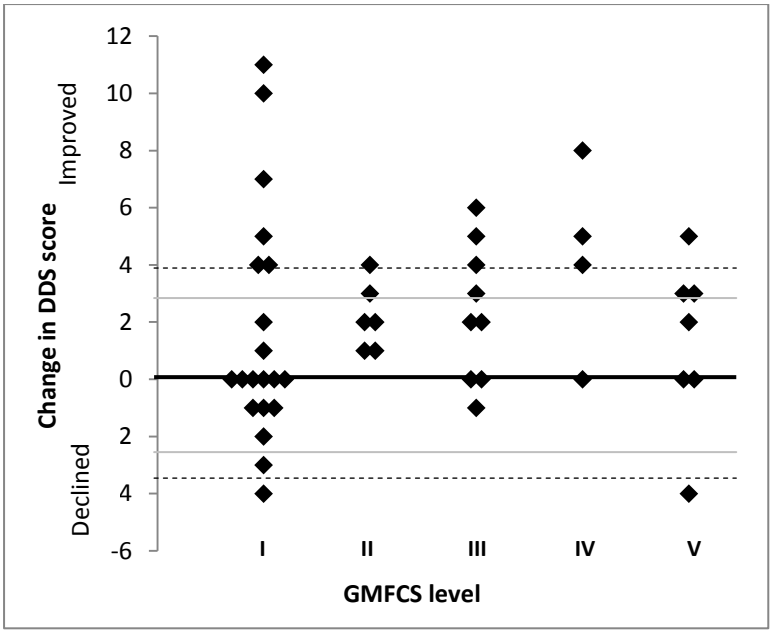
Table 3. Prediction of health outcomes in children with cerebral palsy at 36 months c.a. based on oropharyngeal dysphagia at 18-24 months c.a.

	Nutritional status					
	Height for Age Z score		**Weight for Age Z score**		**BMI Z score**	
	Crude B (p value)	**Adjusted B (p value)** ‡	**Crude B (p value)**	**Adjusted B (p value)** ‡	**Crude B (p value)**	**Adjusted B (p value)** ‡
OPD on DDS	-0.3 (0.56)	0.01 (0.98)	0.4 (0.50)	0.8 (0.19)	0.9 (0.11)	1.1 (0.054)
Modified	-0.04 (0.92)	0.5 (0.26)	0.3 (0.50)	**1.2 (0.03)***	0.5 (0.30)	**1.1 (0.048)***
OPD severity†	-0.01 (0.64)	0.04 (0.29)	-0.03 (0.39)	0.03 (0.58)	-0.03 (0.34)	-0.01 (0.78)
Feeding efficiency	0.05 (0.36)	0.1 (0.36)	0.1 (0.11)	0.1 (0.09)	0.1 (0.10)	0.1 (0.09)
Challenging behaviours	-0.2 (0.61)	-0.02 (0.71)	-0.01 (0.84)	-0.01 (0.92)	0.01 (0.83)	0.01 (0.85)
Pharyngeal phase	0.1 (0.80)	0.4 (0.31)	0.3 (0.57)	0.6 (0.21)	0.3 (0.56)	0.4 (0.37)
Modified	0.1 (0.70)	0.6 (0.13)	0.4 (0.38)	**1.0 (0.048)***	0.4 (0.38)	0.7 (0.16)
	Introduction of nutritional intervention					
	Supplementary feeding		**Gastrostomy feeding**			
	Crude OR (p value)	**Adjusted OR (p value)** §	**Crude OR (p value)**	**Adjusted OR (p value)** §		
OPD on DDS	2.6 (0.40) ||	0.3 (1.0)	1.5 (0.76) [d]	n/c ||		
Modified	**8.1 (0.04)[d]***	0.7 (1.0)	5.2 (0.13) [d]	n/c ||		
OPD severity†	**1.2 (0.02)***	1.0 (0.79)	2.1 (0.056)	n/c		
Feeding efficiency	1.1 (0.52)	1.2 (0.37)	0.9 (0.39)	0.8 (0.26)		
Challenging behaviours	1.0 (0.66)	1.0 (1.00)	1.0 (0.73)	0.9 (0.31)		
Pharyngeal phase	3.9 (0.23)	0.8 (0.88)	3.5 (0.26) [d]	0.8 (1.00) ||		
Modified	7.3 (0.07)	1.4 (0.79)	6.3 (0.08)	0.8 (1.00) ||		
	Parent Stress		**Hospitalisations for chest infection**			
	Crude OR (p value)	**Adjusted OR (p value)** ‡	**Crude OR (p value)**	**Adjusted OR (p value)** ‡		
OPD on DDS	1.1 (0.90)	0.5 (0.40)	1.5 (0.76) ||	0.6 (1.0) ||		
Modified	2.3 (0.14)	1.2 (0.83)	3.0 (0.34)	1.0 (0.98)		
OPD severity†	**1.1 (0.02)***	1.0 (0.52)	1.1 (0.09)	1.1 (0.60)		
Feeding efficiency	0.9 (0.32)	0.9 (0.38)	1.0 (0.78)	1.1 (0.58)		
Challenging behaviours	1.1 (0.054)	**1.1 (0.049)***	1.1 (0.54)	1.1 (0.59)		
Pharyngeal phase	2.8 (0.07)	2.0 (0.29)	2.0 (0.55)	0.8 (0.86)		
Modified	2.2 (0.12)	1.4 (0.58)	3.7 (0.26)	1.7 (0.67)		

*Significantly related; †Severity indicated by DDS raw score; ‡Model adjusted for GMFCS (collapsed I-II, III, IV-V, at 36 months), gender; §Model adjusted for GMFCS (at 36 months), BMI and gender; ||Predicts perfectly, therefore calculated using Exact Logistic Regression; BMI Body Mass Index; DDS Dysphagia Disorders Survey; inf infinity; n/c not calculable; OPD Oropharyngeal Dysphagia; PSAS Pre-Speech Assessment Scale; SOMA Schedule for Oral Motor Assessment



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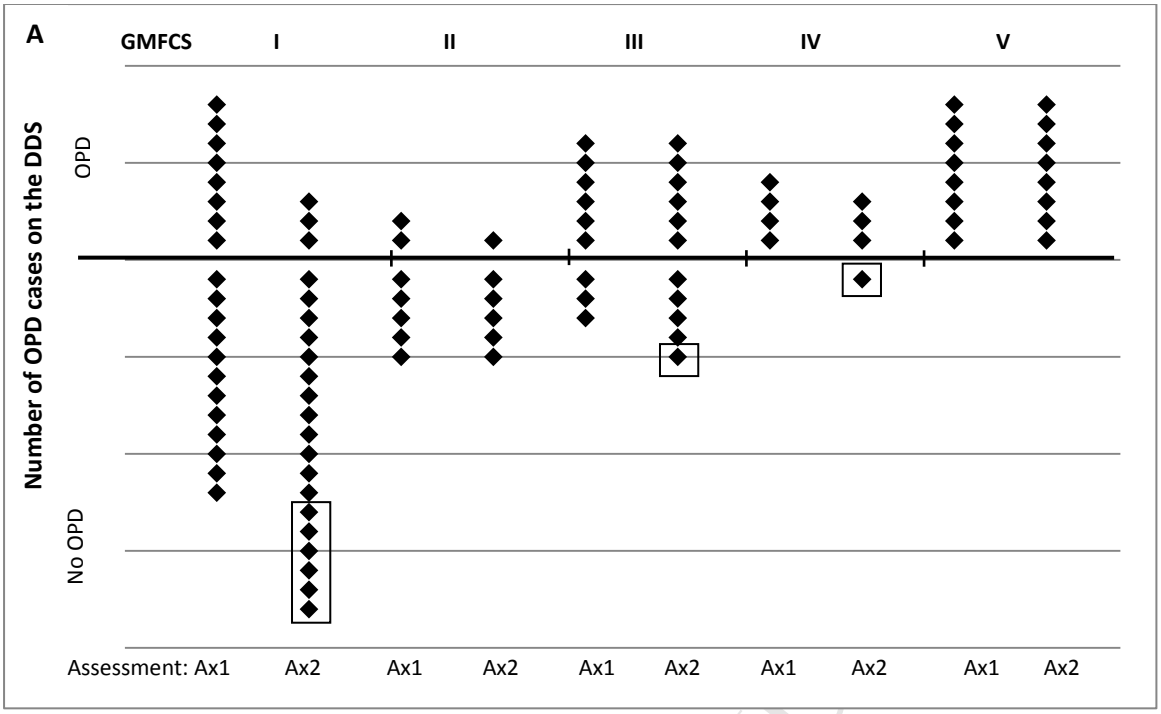
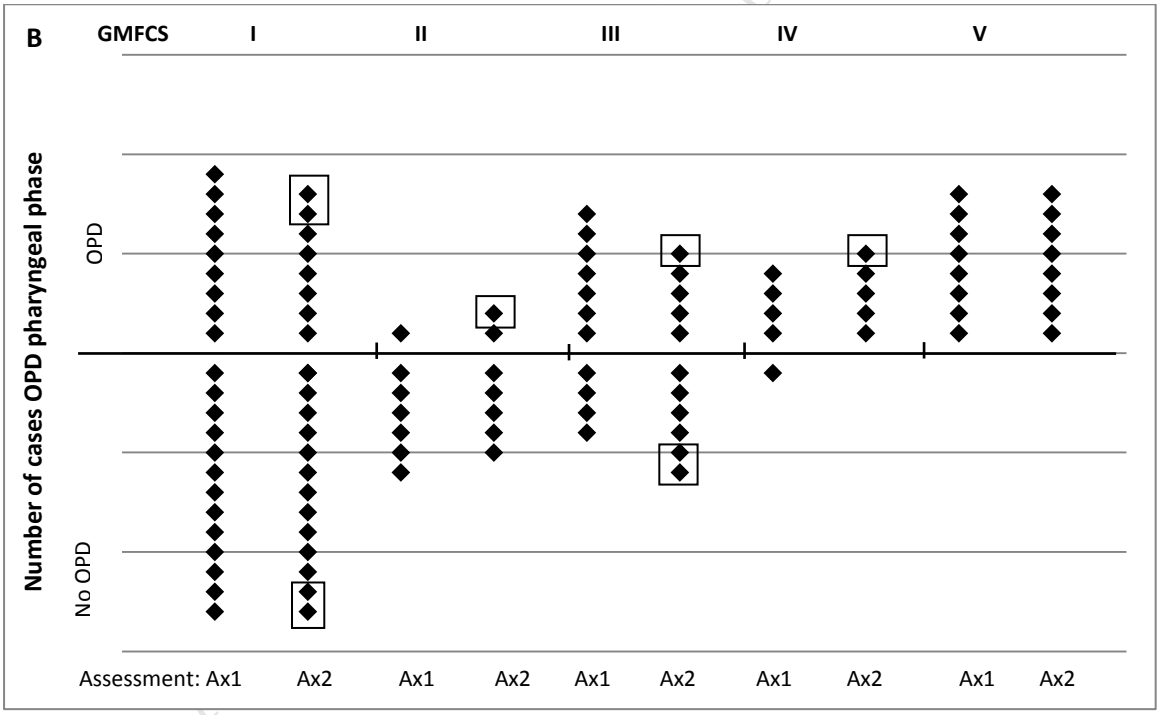

A
GMFCS
I
II
III
IV
V
Number of OPD cases on the DDS
OPD
No OPD
Assessment: Ax1
Ax2
Ax1
Ax2
Ax1
Ax2
Ax1
Ax2
Ax1
Ax2


B
GMFCS
I
II
III
IV
V
Number of cases OPD pharyngeal phase
OPD
No OPD
Assessment: Ax1
Ax2
Ax1
Ax2
Ax1
Ax2
Ax1
Ax2
Ax1
Ax2