Colonic Transit Time and Constipation in Children With Spastic Cerebral Palsy

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ABSTRACT. Park ES, Park CI, Cho S-R, Na S, Cho YS. Colonic transit time and constipation in children with spastic cerebral palsy. Arch Phys Med Rehabil 2004;85:453-6.

Objectives: To evaluate colonic motility and to investigate contributing factors to colonic dysmotility in children with spastic cerebral palsy (CP).

Design: Cross-sectional study.

Setting: A university-based rehabilitation hospital.

Participants: Thirty-eight children with spastic CP.

Interventions: Not applicable.

Main Outcome Measures: Colonic transit time was measured by using a Sitzmarks. The nutrient intake during 3 consecutive days was analyzed by using the ESHA Food Processor program.

Results: A significant relationship between colon transit time and stool frequency was observed ($P<.05$). All children with constipation and 17 (60.8%) of 28 without constipation showed an abnormal segmental colon transit time in at least 1 segment of the colon. A transit time delay at the proximal segment of colon was remarkable in CP children with constipation. In children without constipation, a transit time delay was marked at the rectosigmoid colon only. Constipation and transit time delay were significantly related to ambulatory function ($P<.05$).

Conclusions: A transit time delay at total or segmental colon was frequently observed in children with CP. Constipation and colonic motility were related to ambulatory function.

Key Words: Cerebral palsy; Colon; Gastrointestinal motility; Rehabilitation.

CEREBRAL PALSY (CP) CONCERNS a group of chronic movement and posture disorders caused by nonprogressive lesion in the immature brain. Other than motor deficits, which are uniquely referred to as CP, accompanying features, such as mental retardation, seizure, oculomotor, and speech problems, may frequently be encountered. Apart from neuropsychiatric problems, a variety of gastrointestinal symptoms, such as dysphagia, gastroesophageal reflux, swallowing disorder, delayed gastric emptying, vomiting, and chronic constipation, are commonly reported in CP.[1-4] Constipation is a significant and common problem in neurologically impaired children and a manifestation of a probable underlying defect in gut innervation.[4] Colonic transit studies using radio-opaque markers proved to be useful in establishing the diagnosis of constipation in children and might be of value, in association with clinical symptoms, in assessing constipation severity.[5] Furthermore, constipation is associated with early satiety and is an additional reason for poor feeding in disabled children; moreover, it was found to be a contributory factor to undernutrition in 41% of cases in 1 series.[6] Otherwise, low fiber[7,8] and fluid intake[9,10] are widely accepted as risk factors of chronic constipation. However, the associations among constipation and dietary intake and colon transit time have only rarely been studied in children with CP. The objectives of our study were to evaluate colonic transit time and to determine the relationship among colonic transit time and stool frequency, nutrient intake, functional level, and brain magnetic resonance imaging (MRI) findings in children with spastic CP.

METHODS

The inclusion criteria of the subjects were spastic CP, no identified congenital malformation in the gastrointestinal tract, and not receiving medications affecting gastrointestinal motility. Thirty-eight children with spastic CP (25 boys, 13 girls) were recruited as subjects from among the patients who were admitted to our hospital for rehabilitation management. Their mean age ± standard deviation (SD) was 5.0±2.9 years. In terms of clinical spastic CP type, 19 children had quadriplegia, 11 had diplegia, 5 had hemiplegia, and 3 had monoplegia. According to the Gross Motor Function Classification System,[11] 3 children were level I, 17 level II, 11 level III, 4 level IV, and 3 level V. Ambulation status was classified into 2 groups: nonambulator (those who used wheelchairs exclusively) and ambulator (those who were able to walk indoors or outdoors independently). Constipation was defined as less than 3 stools per week.[1,12]

A clinician interviewed parents or a subject's caregiver to check clinical symptoms. A food record of 3 consecutive days was completed for each patient. These records were analyzed for nutrient composition by using ESHA Food Processor program, version 6.11,[a] and the energy and nutrient intakes of each subject were calculated.

Colon transit time was measured by the Metcalf method by using a Sitzmarks.[b] Each patient ingested 1 Sitzmarks capsule, containing 24 markers, on 3 successive days at 9:00 AM and a plain abdominal radiograph was taken at the same hour on the fourth day. Three segments of the large bowel—right colon, left colon, and rectosigmoid colon—were identified by referring to bony structures.[13,14] The location of right, left, and rectosigmoid colons were determined on the basis of their positions with respect to lines drawn from the midline to the L5 vertebra and from the L5 vertebra to each pelvic outlet.[15] The total and segmental colonic transit times, determined by using the single-film technique of Metcalf et al,[16] were calculated by

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Table 1: Total and Segmental Colon Transit Time

Transit Time (h)	Normal Controls[18] (n=51)	Children With CP (n=38)		
		Constipation	No Constipation	Total
Total colon	15.6±9.4	63.0±8.4*	17.3±16.0	37.1±21.4
Segmental colon				
Right colon	3.1±4.2	13.1±11.5*	4.8±5.5	6.9±8.2
Left colon	5.1±4.9	24.3±12.1*	5.8±8.8	11.0±12.6
Rectum	7.4±4.9	26.6±14.3*	16.7±11.9	19.5±12.8

NOTE. Values are mean ± SD.
*$P<.05$ by Mann-Whitney U test (constipation vs no constipation).

counting the markers from single abdominal film, taken on day 4. The normal reference was taken from the data of a previous report by the pediatric department of our university.[17] The normal range for total and segmental colon transit time was taken from mean plus 2 SDs.

RESULTS

Ten of the 38 children with spastic CP were constipated (hard stool consistency) and defecation was difficult in another 19 children. Eight of the 10 constipated children had both hard stool consistency and defecation difficulty. At least 1 of the symptoms was found in 23 children. Total and segmental colon transit times were significantly longer in CP children with constipation than in children without constipation (Mann-Whitney U test, $P<.05$; table 1). A significant negative correlation ($P<.05$) between colon transit time and stool frequency was evident for total colon ($r=-.77$), right colon ($r=-.486$), left colon ($r=-.551$), and rectosigmoid colon ($r=-.434$). All children with constipation and 17 (60.8%) of 28 without constipation showed an abnormal segmental colon transit time in at least 1 segment of the colon. Among children with constipation, a transit time delay at the rectosigmoid colon was noted in only 1 child (10%), and 9 children (90%) had a transit delay at the more proximal segments of the colon with or without a transit delay of the rectosigmoid colon. These findings differed significantly from the 12 of 18 children without constipation (Fisher exact test, $P<.05$), for whom the segmental colon transit time was abnormally prolonged at the rectosigmoid colon. The total colon transit time was significantly longer in the nonambulators than in the ambulators ($P<.05$; table 2), and constipation was more frequent in nonambulators (8/20 nonambulatory children vs 2/18 ambulatory children) (Fisher exact test, $P<.10$). However, the pattern of transit time delay at the colonic segment was not significantly dependent on ambulatory function. Nineteen (57.6%) of 33 children with abnormal findings on brain MRI showed symptoms related to constipation, and one third of these children were constipated. On the other hand, constipation, hard stool, or defecation difficulty was noted in none of the children with normal MRI findings. The colonic transit time of groups with normal and abnormal brain MRI findings did not differ significantly, except for their right colon transit times (Mann-Whitney U test, $P<.05$; table 3).

Fluid and fiber intakes were not significantly related to prolonged total colon transit time or constipation (table 4), and no significant differences in nutrient intakes between those with and without constipation were observed.

Table 2: Colon Transit Time According to Ambulatory Status

	Nonambulator	Ambulator
Total colon transit (h)	50.1±17.4*	27.1±20.1
Segmental colon transit		
Right colon transit (h)	9.4±8.5	5.5±8.0
Left colon transit (h)	15.5±13.9	8.4±10.7
Rectal transit (h)	25.2±10.7	13.7±13.4

NOTE. Values are mean ± SD.
*$P<.05$ by Mann-Whitney U test.

DISCUSSION

The measurement of colonic transit time is well tolerated by children and proved to be a reliable and useful test, with only small inter- and intraobserver variations.[5] Estimated colonic transit time based on calculations from single abdominal films correlate well with the average mean colonic transit time based on multiple films.[16] Thus, we used a simple assessment, based on a single film, and obtained a good correlation between the stool frequency and colon transit time in children with CP. Moreover, these results are in accordance with the results in neurologically unimpaired children with functional chronic constipation.[18,19]

An earlier study[3] showed that a colonic transit delay in children with severe brain damage, who had chronic constipation, was abnormal at both the left colon and the rectum, which contrasts with a transit time delay, predominantly at the rectum only, in children with functional fecal retention. In addition, another study[1] showed a colonic transit time delay at proximal segments of the colon in 52%, at the level of the left colon and rectum in 36%, and at the level of the rectum only in 12% of children with CP. Our study also showed transit time delays at the proximal segments of the colon in constipated children, which indicates that disruption of the neural modulation of colonic motility might be responsible for constipation in these children. On the other hand, half of the children without constipation showed a transit time delay at the rectosigmoid colon only. Although children with prolonged transit times at the rectosigmoid colon were not constipated by definition, it is nevertheless possible for them to have a substantial defecation problem. Previously, a prolonged transit time at the level of the rectum was noted in neurologically unimpaired children with long-standing constipation.[3,18] In these children, altered motor behavior of the striated muscle, rather than dismotility of the smooth muscle of the rectoanal region, was suggested as the cause of fecal retention. Moreover, a study[20] of anorectal manometry in children with CP suggested that anal sphincter and/or pelvic floor muscle incoordination resulted in defecation difficulties in children with CP. Thus, a transit delay at the rectosigmoid and at the proximal segment of the colon in our subjects suggests that both the motility of the smooth muscle of the colon and the activity in the striated muscles of the anal sphincter and/or of the pelvic floor are altered in children with spastic CP.

Table 3: Colon Transit Time and Brain MRI Findings

Brain MRI Findings	Colon Transit Time (h)			
	Total Colon	Right Colon	Left Colon	Rectal Colon
Normal findings (n=5)	25.4±20.7	1.6±2.5*	5.8±7.1	19.5±12.8
Abnormal findings (n=33)	38.9±21.2	7.8±8.5	11.8±13.1	19.7±12.3
Periventricular leukomalacia (n=11)	43.2±19.4	7.5±7.3	14.7±15.8	10.9±8.2
Cortical atrophy (n=5)	43.2±17.9	13.4±10.5	10.6±13.0	21.2±4.3
Cortical migration disorder (n=3)	64.0±9.6	17.3±15.3	18.7±13.0	28.0±11.8
Multicystic cephalomalacia (n=7)	25.4±22.1	4.6±3.9	5.1±5.8	15.7±20.0
Miscellaneous (n=7)	31.9±20.3	3.1±4.4	11.6±14.4	17.1±13.1

NOTE. Values are mean ± SD.
*$P<.05$ by Mann-Whitney *U* test (normal vs abnormal brain MRI findings).

Ten (26.5%) children with CP were constipated in our study. The frequency of constipation in our subjects was much lower than the 74% found by Del Giudice et al[1] and the 89.6% found by Agnarsson et al,[20] but similar to the 39% reported by Feldkamp et al.[21] These differences between studies might have been caused by clinical severity differences. Because of the small number of cases with normal brain MRI, the relationship between constipation and colonic transit time with brain MRI abnormalities could not be investigated. However, almost 80% of children with abnormal brain MRI findings showed a transit time delay in at least 1 segment of the colon, in contrast with no transit time delay along the entire colon in 80% of children with normal MRI findings. It is likely that brain MRI abnormalities may play a role in colonic transit time delay in these children. However, clarification through further study with a larger sample size is needed. Previously, a transit time delay at the proximal colon was frequent in chronically constipated children with a severe brain lesion, compared with constipated children with functional fecal retention.[3] Therefore, it may be possible that longer right colon transit times in children with abnormal brain MRI findings might reflect more dysfunction in the neural modulation of colonic motility due to a central nervous system (CNS) lesion than in those with normal brain MRI findings. However, these findings should be interpreted cautiously, because the extent of a CNS lesion cannot be simply estimated by brain MRI findings alone, and patients with identical brain MRI findings can have variable outcomes.[22] Moreover, the prolongation of colonic transit time was not significantly associated with any kind of abnormal brain MRI finding, which suggests that a transit time delay might not be related to specific brain lesions.

A few reports have shown a significant relationship between esophageal reflux or esophageal motility and the functional extent of CNS lesions.[2,23] In children with CP, ambulatory function might imply extent of a brain lesion and mobility. Thus, a longer total colon transit and more frequent abnormal transit time in at least 1 segment of colon in nonambulatory children with CP might be presumed to be a consequence of both neural modulation dysfunction of colonic motility, because of the presence of a severe CNS lesion, and poor mobility.

Increasing the amount of fiber and fluid intake in constipated children remains a commonly recommended intervention.[10,24] However, no significant effects of dietary fiber and fluid intake on colonic motility have been documented in children with chronic constipation.[2,19,25,26] Our study also did not reveal any significant relationship between fluid and fiber intake and constipation and colonic motility. The dietary intake is very reliably recorded by history in general. However, the validity of it as a reliable method to evaluate dietary intake in severely affected CP has been questioned.[27] Therefore, assessment of dietary intake using a more reliable and direct method will be

Table 4: Daily Dietary Nutritional Intakes

	Constipation		Total Colon Transit Time	
	Present (n=10)	Absent (n=28)	Prolonged (n=19)	Normal Ranges (n=19)
Fluid intake (mL)	419.3±221.6	436.5±213.9	435.8±233.8	382.9±196.0
Fiber intake (g)	4.1±3.2	6.7±4.1	5.2±3.9	4.1±3.4
Calorie (kcal)	971.7±10.6	956.0±339.9	923.0±334.4	998.2±285.8
Protein (g)	38.3±10.6	36.4±15.9	36.6±17.1	37.4±11.5
Vitamin A (μg)	407.6±202.6	250.3±161.2	225.4±142.6	367.7±249.2
Vitamin B_1 (mg)	0.6±0.3	0.5±0.2	0.5±0.3	0.5±0.2
Vitamin B_2 (mg)	0.8±0.3	0.8±0.3	0.7±0.3	0.9±0.4
Vitamin B_3 (mg)	10.6±7.8	8.1±6.8	8.4±7.6	9.2±6.7
Vitamin B_6 (mg)	0.8±0.6	0.8±0.7	0.9±0.8	0.7±0.3
Vitamin C (mg)	48.3±32.6	29.3±26.0	31.0±29.0	38.8±29.2
Vitamin D (μg)	1.6±1.0	2.4±2.0	2.3±1.5	2.1±2.1
Vitamin E (mg)	1.7±1.0	1.9±1.2	1.9±1.1	1.9±1.3
Calcium (mg)	424.4±144.9	505.3±231.1	451.1±203.5	511.9±219.2
Phosphorus (mg)	610.7±166.5	623.4±253.6	598.4±248.8	640.9±212.8
Iron (mg)	5.4±1.8	4.6±2.7	4.8±3.0	4.9±2.0
Zinc (mg)	4.2±1.2	4.4±2.2	4.4±2.3	4.3±1.6

NOTE. Values are mean ± SD.

necessary to obtain conclusive data regarding the relationship between dietary intake and colonic motility in children with CP. Moreover, it will be helpful to determine whether colonic dysmotility is associated with malnutrition in CP, by using a reliable tool based on directly measured body composition for evaluation of nutritional status.

CONCLUSIONS

A good relationship between bowel frequency and colonic transit time suggests that the colonic transit time can be used as a quantitative measure of constipation in children with CP. A transit time delay at the proximal segment of colon was a dominant finding in constipated CP children, which suggests that a disruption of the neural modulation of colon motility is responsible for their constipation. However, the overall prevalence of a transit time delay at the rectosigmoid colon in our subjects implies that altered motor behavior of muscles of the rectoanal region might also produce a substantial defecation problem. Constipation and colonic motility were found to be related to ambulatory function.

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Suppliers

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