

Prevalence of Periodontal Disease in the Primary Dentition of Children with Cerebral Palsy

Renata de Oliveira Guare, DDS, MS Ana Lidia Ciampioni, PhD, MSD, DDS

ABSTRACT

Purpose: The aim of this study was to evaluate periodontal disease prevalence in the primary dentition of children with cerebral palsy (CP).

Methods: The experimental group consisted of 100 children with CP and the control group, 100 healthy children. The indices used in the oral examination were: debris index, calculus index, gingival index, Simplified Oral Hygiene Index (OHI-S), and gingival hyperplasia index.

Results: It was observed that the mean values for the debris index, OHI-S, and gingival index were higher in the children with CP than in the control group. In the CP group, the percentage of children who had their teeth brushed by their parents or another person was greater than in the control group.

Conclusions: From this study, it was concluded that children with CP had greater prevalence of periodontal disease in the primary dentition than children in the control group. (*J Dent Child.* 2004;71:27-32)

KEYWORDS: CEREBRAL PALSY, SPECIAL HEALTH CARE NEEDS, PERIODONTAL DISEASE

Nonprogressive infantile chronic encephalopathy, also known as cerebral palsy (CP), is the name of a group of nonprogressive motion disorders subject to variations due to a cerebral lesion during the first stages of an infant's development.¹

The causes of CP may be perinatal anoxia, prematurity, prenatal infection, such as rubella, toxoplasmosis, cytomegalovirus, or postnatal infection, such as meningoencephalitis. According to the type of motion alteration presented by the child, various types of CP can be observed: spastic, athetoid, ataxic, and mixed.¹⁻³

The presence of periodontal disease in CP children typically is due to bacterial plaque accumulation caused by their inability to correctly clean their own teeth, difficulties in chewing and swallowing food, and improper movements of masticatory and tongue muscles.⁴ Their extreme difficulty in maintaining satisfactory oral hygiene is the main factor responsible for their high incidence of periodontal disease, as well as malocclusion, bruxism, and den-

tal caries. It is also worth remembering that in these patients, some predisposing factors, such as pasty consistency of ingested food and dietary restriction of vitamin C, may aggravate periodontal problems.⁵⁻⁸

METHODS

This study was conducted after approval by the Ethics Committee of the Faculdade de Odontologia da Universidade de São Paulo (FOUSP). The sample consisted of 100 noninstitutionalized CP boys and girls with complete primary dentition between 30 and 73 months old. They were examined at the Lar Escola São Francisco, Central of Rehabilitation in São Paulo. The control group consisted of 100 healthy boys and girls with complete primary dentition between 30 and 71 months who were selected from 2 schools in São Paulo. Examinations for all children were performed after written consent by their parents.

The calibrated examiner participated in all steps of this study. The CP children were examined at the dental office of the Lar Escola São Francisco under appropriate light-reflector illumination in a dental chair. Children belonging to the control group were examined at school. It must be emphasized that as schools did not have a dental office, the examiner used a portable compressor and fixed a flash-light to his forehead.

Dr. de Oliveira Guare is a masters student, and Dr. Ciampioni is professor, Department of Orthodontics and Pediatric Dentistry, School of Dentistry, University of São Paulo, São Paulo, Brazil.

Correspond with Dr. de Oliveira Guare at renataguare@uol.com.br

General information was reported for each patient regarding name, birth date, and clinical diagnosis regarding the type of CP, medications, and respective dosage. This data were obtained by consulting the patients' health chart or interviewing the parents. Information regarding frequency of tooth-brushing and who performed the oral hygiene for each child was obtained by questioning responsible adults accompanying children.

For the periodontal examination, a group of teeth was chosen as proposed by Santos⁹ and Rodrigues¹⁰: upper right first molar (54); upper left central incisor (61); lower left second molar (75); and lower right lateral incisor (82). When an examination of these specific teeth was not possible due to extraction, caries, or restoration, they were substituted by the subsequent element.

The status of oral hygiene was evaluated using the above described teeth and according to criteria used in the Simplified Oral Hygiene Index (OHI-S) proposed by Greene and Vermillion.¹¹ The OHI-S is a combination of debris index, (ie, plaque and calculus). Scores for debris and calculus vary from 0 to 3, according to the following criteria:

- 0 absence of debris or extrinsic stain;
- 1 soft debris covering not more than one-third of the tooth surface being examined or presence of extrinsic stains without debris regardless of the surface area covered;
- 2 soft debris covering more than one-third, but not more than two-thirds of the exposed tooth surface; the presence of extrinsic stain may occur or not;
- 3 soft debris covering more than two-thirds of the examined tooth surface.

Scores for the calculus index:

- 0 absence of supra- or subgingival calculus;
- 1 presence of supragingival calculus covering not more than one-third of the examined surface;
- 2 presence of supragingival calculus covering more than one-third, but not more than two-thirds of the examined surfaces, or presence of small portions of subgingival calculus around the cervical area of the tooth;
- 3 presence of supragingival calculus covering more than two-thirds of the examined surface or a continuous region of subgingival calculus along the cervical area of the tooth, or both.

The debris and calculus indices were calculated separately by summing the attributed scores and then dividing results by the number of examined surfaces. Results obtained using the simplified OHI-S were represented by the indices of debris and calculus.

The gingival index was used to quantify and evaluate gingivitis, as proposed by L  e and Silness.¹² The presence or absence of bleeding was observed by means of pressure stimulus (probing) against papillae, according to a 0-3 scale, so that:

- 0 normal tissue;
- 1 mild inflammation, with slight color alterations and discrete edema, absence of bleeding during probing;

Table 1. Results Obtained According to Kruskal-Wallis Test for Plaque, Calculus, OHI-S, Gingival and Gingival Hyperplasia Indices Among the Diagnosis of Spastic Diparesis, Spastic Hemiparesis, and Spastic Tetraparesis in the CP Group

Variable	chi-square	P value
Plaque index	0.896	.6 NS
Calculus index	0.990	.6 NS
OHI-S	0.984	.6 NS
Gingival index	9.550	.008*
Gingival hyperplasia index	4.119	.1 NS

*Statistically significant ($P < .01$).

NS: not significant.

- 2 moderate inflammation, rubor, edema with shiny surface, presence of bleeding during probing;
- 3 severe inflammation, intense rubor and edema, ulcerated tissue with tendency to spontaneous bleeding.

Care was taken to evaluate tissues around each tooth (ie, distal buccal papillae, buccal margin, mesial buccal papillae, and lingual gingival margin).

The Angeolopoulos and Goaz¹³ measurement was used to evaluate the presence of gingival growth (gingival hyperplasia), which measured the distance between the cement-enamel junction to the free gingival margin, according to the following criteria:

- Level 0 no hyperplasia, normal gingivae;
- Level 1 gingival hyperplasia covering the cervical third or less than one-third of the anatomic crown of anterior teeth;
- Level 2 gingival hyperplasia extending to two-thirds of the anatomic crown of anterior teeth;
- Level 3 gingival hyperplasia covering more than two-thirds of the anatomic crown of anterior teeth.

RESULTS

After evaluation of CP children (experimental group) and healthy children (control group), data were reported and statistically analyzed. No statistically significant differences were found between the evaluated groups regarding age or gender, and the sample was considered homogeneous. Regarding the diagnosed type of CP, the spastic type presented as the most prevalent, so that tetraparesis was found in the highest number of these cases.

Regarding periodontal disease, data presented in Table 1 were submitted to Kruskal-Wallis test to verify any differences in the mean values of plaque index (PI), calculus index (CI), OHI-S, gingival index (GI), and hyperplasia index among the diagnosis of CP found with highest frequency: spastic diparesis, spastic hemiparesis, and spastic tetraparesis.

The GI mean values obtained in the group of children with spastic hemiparesis were 0.842, and 1.293 in the group with spastic tetraparesis. After application of the Mann-Whitney test, it can be observed in Table 2 that significant differences were found among GI mean values, so that

Table 2. Mann-Whitney Test Obtained for the Gingival Index

Comparison of diagnosis	<i>z</i>	<i>P</i> value
Diparesis and Hemiparesis	-0.632	.5 NS
Diparesis and Tetraparesis	-1.423	.2 NS
Hemiparesis and Tetraparesis	-3.413	.001*

*Statistically significant ($P < .01$).

NS: not significant.

children with spastic tetraparesis showed a higher mean gingival index than children with spastic hemiparesis.

The Mann-Whitney test (Table 3) was performed to verify the existence of significant differences in the mean values of PI, CI, OHI-S, GI, and gingival hyperplasia index between the CP and control groups. After statistical analysis, statistically significant differences were observed in the mean values of PI, OHI-S, and GI, so that they were higher in the CP children.

Regarding oral hygiene, after evaluating data contained in Figure 1 and respective statistical analysis, it was observed that the percentage of CP children who had their teeth cleaned by parents or any other person was higher than in the control group (Table 4 and Figure 1).

The distribution of patients per diagnosis and daily frequency of tooth-brushing in the CP group can be observed in Figure 2.

According to data presented in Figure 3, it can be observed that statistically significant differences were found between groups in the percentages of children according to frequency of daily brushing.

DISCUSSION

Most previous studies that evaluated periodontal disease in handicapped children did not analyze this considered age group, whether patients were institutionalized, the type of presented disease, or the periodontal survey method, thus making it difficult to make a direct comparison with previous data. Up to the present moment, no research was specifically concerned with the prevalence of periodontal disease in the deciduous dentition of CP children. Hence, data presented in this study was

Table 4. Results Obtained According to Mann-Whitney Test for Plaque, Calculus, OHI-S, Gingival and Gingival Hyperplasia Indexes: Test Among Groups

Variable	<i>z</i>	<i>P</i> value
Plaque index	-5.879	.001†
Calculus index	-0.015	1.0 NS
OHI-S	-5.809	.001†
Gingival index	-2.955	.003†
Gingival hyperplasia index	-2.481	.01*

*Statistically significant at 5% ($P < .05$).

†Statistically significant at 1% ($P < .01$).

NS: not significant.

compared with others that evaluated CP children presenting deciduous dentition as an integral part of the entire study.

According to the statistical analysis in Table 1, statistically significant differences were observed in the GI among the different diagnosis. According to Löe and Silness,¹² the GI with values between 0.1 and 1.0 was considered as minimal inflammation; 1.1 to 2.0, moderate inflammation, and 2.1 to 3.0 as severe inflammation. Hence, it can be said that the group presenting spastic hemiparesis presented minimal clinically detectable inflammation (GI = 0.842), and the group with spastic tetraparesis (GI = 1.293), moderate inflammation.

Regarding periodontal disease, data presented in Table 3 were submitted to the Mann-Whitney test, which is a nonparametric test and does not follow a normal distribution. It could explain why some measures had standard deviations exceeding the means.

When the data presented in Table 3, regarding the mean values of GI for both groups was evaluated, it was observed after the Mann-Whitney test (Table 4) that although statistically significant differences were found, values demonstrated in the GI presented a difference of only 0.242 for the CP group. Mean values of GI obtained for CP patients were 1.036 and 0.794 for the control group. As they were inferior to 1.1, they were considered to be representative of minimal clinical inflammation, according to Löe and Silness.¹²

The presence of gingivitis in CP children was reported by several authors. Weisman¹⁴ observed that 80% of CP children

Table 3. Mean Values, Standard Deviations, Minimum and Maximum Values for Plaque, Calculus OHI-S, and Gingival Hyperplasia Per Group Indices

Variable	Group of CP children				Group of healthy children (control group)			
	Mean values	Standard deviations	Minimum	Maximum	Mean values	Standard deviations	Minimum	Maximum
Plaque index	1.262	0.716	0	3	0.682	0.629	0	2.750
Calculus index	0.015	0.111	0	1	0.008	0.058	0	0.500
OHI-S	1.277	0.754	0	4	0.690	0.634	0	2.750
Gingival index	1.036	0.585	0	2	0.794	0.522	0	1.875
Gingival hyperplasia index	0.060	0.239	0	1	0.000	0.000	0	0.000

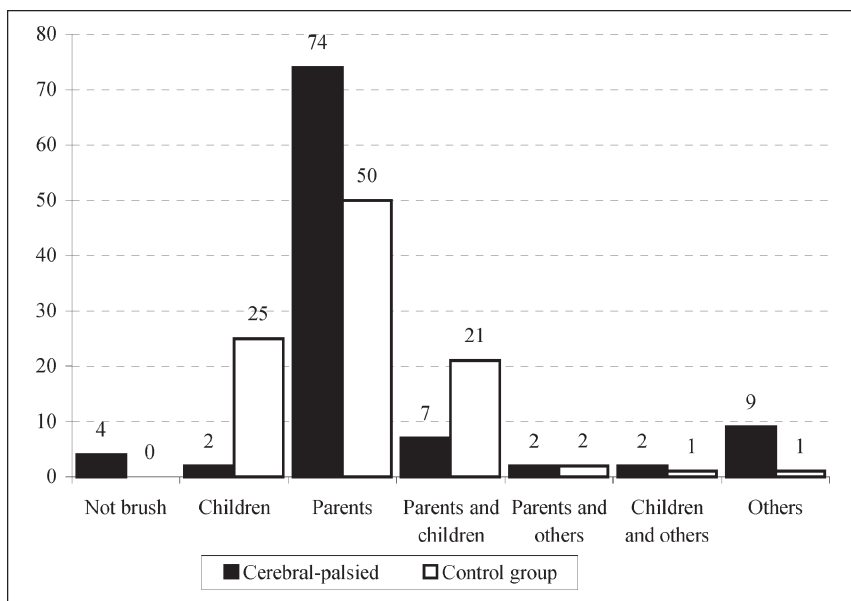


Figure 1. Distribution of patients according to the person who performs their oral hygiene.

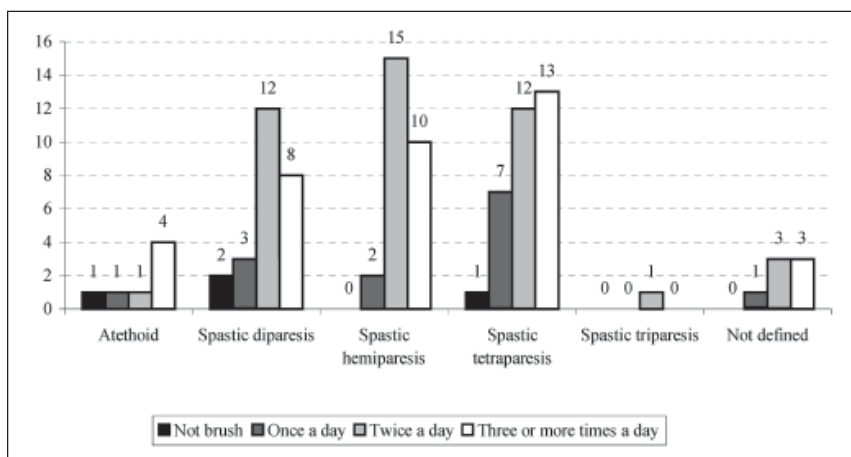


Figure 2. Distribution of patients per diagnosis and daily frequency of tooth-brushing: Cerebral-palsied group.

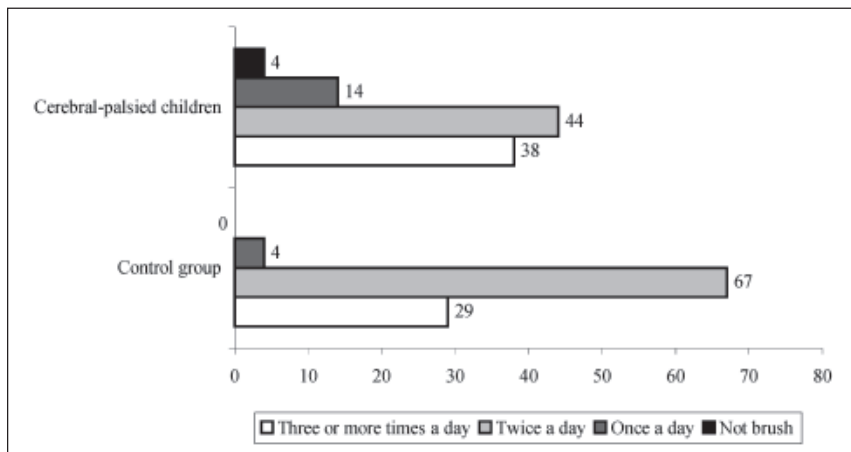


Figure 3. Distribution of patients per group and daily frequency of tooth-brushing.

between 6 and 18 years old presented with gingivitis and that for both genders the prevalence of gingival disease increased from 50% at 6 years old to 90% at 10 years old. The high incidence of gingivitis (3 times higher than the control group) was also observed by Magnusson and Val⁶ when they evaluated 76 Swedish CP children between 3 and 15 years old so that 16 (21%) were between 3 and 5 years old. Fishman et al¹⁵ observed that the severity of periodontal disease in CP children between 4 and 18 years old was significantly greater than in the group of healthy patients. However, this clinician used the Periodontal Index, which is commonly used for adult populations. Sznajder and Feniak⁷ observed the presence of mild to moderate gingivitis without bone loss in all evaluated CP patients (13 children) between 5 and 14 years old. Kaneko⁴ observed the presence of gingivitis in 76% of the 43 CP children between 1 and 17 years old.

However, other studies, such as the one presented by Shmarak and Bernstein¹⁶ with 81 CP children between 3 and 15 years old (37% were between 3 and 6 years old), demonstrated the presence of gingival inflammation in only 12% of the sample. On the other hand, Swallow¹⁷ observed that the prevalence of periodontal disease in CP children was similar to healthy children.

Regarding data obtained using the PI, as presented in Table 3, it can be observed that mean values obtained were 1.26 for the CP group and 0.68 for the control group. According to Wei and Lang,¹⁸ oral hygiene is considered as 'good' when plaque indices range between 0.3 and 0.6, 'regular' when they are between 0.7 and 1.8, and 'poor' between 1.9 and 3.0. Hence, it can be considered that in the authors' control group, oral hygiene was considered 'good' and 'regular' in the CP group.

Regarding the OHI-S, as presented in Table 3, it was observed that the CP group presented superior values (approximately 2 times) than the control group, and they were statistically significant. Kaneko⁴ evaluated 43 CP children between 1 and 17 years old and obtained superior mean values of OHI-S (1.74) to those obtained in the present study (1.277).

However, Fishman et al¹⁵ evaluated the oral health of 203 CP children and their healthy brothers or sisters (335), between 4 and 18 years old, and observed that patients

between 4 and 6 years old presented similar OHI-S, either for the CP or control group. Another study performed in England evaluated the presence of bacterial plaque in the buccal surface of upper and lower incisors of CP children between 5 and 16 years old, and those authors stated that CP children presented better oral hygiene than healthy patients.¹⁷

Gingival growth has been reported in users of anticonvulsant drugs, such as diphenylhydantoin, sodium valproate, and phenobarbital, calcium channel blockers, such as diltiazem, nifedipine, and verapamil, and immunosuppressive agents, such as cyclosporine.¹⁹⁻²⁵

Diphenylhydantoin (DPH) induced gingival growth and was mostly denominated as dilantinic gingival hyperplasia, which may have presented different incidences in the user population. According to Esterberg and White,²⁶ the dilantinic hyperplasia occurred in 54% of the patient users of this drug between 3 and 69 years old. Shmarak and Bernstein¹⁶ observed that 5% of the patients between 3 and 15 years old presented dilantin gingival hyperplasia. Angelopoulos and Goaz¹³ demonstrated an incidence of 53% (between 14 and 72 years old) and in 47% of all cases Pugliesi et al²⁷ showed the presence of gingival hyperplasia in users of the drug (age varied from 10 to 16 years old).

The possible influence of a rigorous oral hygiene on the occurrence of DPH induced gingival growth, thus preserving the gingival health in many cases was emphasized by Esterberg and White,²⁶ Angelopoulos and Goaz¹³ and Pugliesi et al,²⁷ so that Hassel et al²⁸ showed a positive correlation between the severity of DPH induced gingival growth and gingival inflammation, pocket depth, calculus accumulation, and plaque index. Maisonneuve²⁹ observed that hyperplastic lesions were found in areas safe from local irritating agents and absent in areas where local irritating agents were found in great quantity, thus suggesting a genetic susceptibility in the referred population or an individual variation of the drug metabolism.

Other anticonvulsant drugs may cause gingival growth, such as phenobarbital. According to Panuska et al,¹⁹ phenobarbital was also associated with gingival hyperplasia, although to a lesser extent than DPH. Besides this anticonvulsant drug, Syrjänen and Syrjänen²⁰ observed that sodium valproate may have caused gingival hyperplasia.

According to data obtained in the present study, 6 cases of gingival growth (gingival growth level 1, according to the GI by Angelopoulos and Goaz,¹³) was observed in the group of CP children, whereas no case was found in the control group.

From the 6 observed cases of gingival growth in CP children, only 1 patient used DPH with 2 months of treatment, thus being probably related to the presence of gingival hyperplasia, as reported by other authors.^{6,13,16,26,27} In another case of gingival hyperplasia presented in this study, the child was treated with fenobarbital and antispasmodic drugs. Perhaps, according to Panuska et al,¹⁹ phenobarbital may have been the responsible agent for the gingival growth. Sodium valproate, as according to Syrjänen and Syrjänen,²⁰ may have been the responsible agent for the gingival growth presented by another child who used this drug associated with a benzodiazepine. Similarly, according to Panuska et al¹⁹ and Syrjänen

and Syrjänen,²⁰ the treatment with phenobarbital and sodium valproate presented by a child in the present study may have been the responsible factor for the observed gingival growth.

However, 2 cases were observed in which both children used antispasmodic drugs and presented gingival growth. In these cases, the responsible factor for this gingival growth may have been buccal breathing presented by the patient. According to Wessels⁵, buccal breathing was also frequently noticed in CP patients and contributed to a worsened anterior upper gingival health, normally local and in which gingival hyperplasia appeared in higher prevalence.¹⁹

It must be emphasized that the presence of gingival growth depends on a series of interrelated factors, and that the use of drugs is only 1 more factor that leads to the development of the problem.

After evaluating data contained in Figure 1 and respective statistical analysis, it was observed that the percentage of CP children who had their teeth cleaned by parents or any other person was higher than in the control group. On the contrary, the percentage of children who brushed their teeth by themselves without any help was greater in the control group. Magnusson and Val⁶ observed that from 76 Swedish CP children between 3 and 15 years old, 38 (50%) depended on their parents or nurses for their oral hygiene, so that only 11 from the control group (13%) did the same, although this difference was not statistically significant.

The distribution of patients per diagnosis and daily frequency of tooth-brushing in the CP group can be observed in Figure 2. The number of patients who did not brush their teeth was higher in the group of children with spastic diparesis; those who brushed once a day occurred in higher number among patients with spastic tetraparesis. The group of patients with spastic hemiparesis presented a greater number of patients who cleaned their teeth twice a day, and the group with spastic tetraparesis presented the highest number of patients who had their teeth brushed 3 or more times a day. It is probable that the number of patients who presented a determined diagnosis of CP influenced the achievement of these results. Tetraparesis was observed in the highest number of patients (33%), particularly in those who brushed their teeth 3 or more times a day, even if their arms and legs made daily brushing difficult.

According to data presented in Figure 3, it was observed that the group of CP patients presented a higher percentage of children who never brushed their teeth or who brushed once a day. On the other hand, this group also presented those with the highest frequency of brushing (3 times or more a day), whereas the control group presented twice a day as the highest frequency. Data regarding no tooth-brushing among individuals belonging to the CP group (4%) seemed to reflect the lack of information by some parents about the necessity of oral hygiene in handicapped children. However, at the same time, it was contradictory that in this same population an elevated frequency of tooth-brushing of 3 or more times a day was presented in 38% of all patients. It must be observed that there were several studies that evaluated the frequency of tooth-brushing, demonstrating that twice a day was appropriate, and that small benefit was achieved in brushing more frequently,³⁰

whereas others³¹ believed that the quality of the oral hygiene was more important than the frequency of tooth-brushing.

CONCLUSIONS

1. The mean values of plaque, OHI-S, and GIs in the deciduous dentition of CP children were higher than in the control group.
2. Statistically significant differences were observed in the mean values of GI for the deciduous dentition among children presenting spastic hemiparesis and tetraparesis.
3. The percentage of children in the CP group who had their oral hygiene performed by parents or other persons was higher than in the control group. By the same token, the percentage of children who brushed their teeth by themselves was greater in the control than the CP group.

REFERENCES

1. Diamant A. Encefalopatias crônicas da infância (paralisia cerebral). In: Diamant A, Cypel S. *Neurologia infantil*. 3rd ed. São Paulo: Atheneu; 1996:781-798.
2. Piovesana AMS. Paralisia cerebral: Contribuição do estudo por imagem. In: Souza AMC, Ferrareto I. *Paralisia cerebral: Aspectos práticos*. 1st ed. São Paulo: Memnon; 1998:8-32.
3. Souza AMC. Prognóstico funcional da paralisia cerebral. In: Souza AMC, Ferrareto I. *Paralisia cerebral: Aspectos práticos*. 1st ed. São Paulo: Memnon; 1998:33-37.
4. Kaneko Y. Oral condition of the institutionalized severely handicapped children. *Bull Tokyo Dent Coll*. 1978;17:27-44.
5. Wessels KE. Oral conditions in cerebral palsy. *Dent Clin North Am*. 1960;4:455-467.
6. Magnusson B, Val RD. Oral conditions in a group of children with cerebral palsy. *Odontol Revy*. 1963;14:385-402.
7. Sznajder N, Feniak R. Hallazgos periodontales em niños com parálisis cerebral infantil. *Rev Asoc Odontol Argent*. 1967;55:126-129.
8. Gugushe TS. Dental caries experience and periodontal status of handicapped institutionalized black high school pupils in Soshanguve, Pretoria. *J Dent Assoc S Afr*. 1991;46:67-69.
9. Santos MTBR. *Estudo da importância das variáveis clínicas, microbiológicas e salivares de crianças portadoras de paralisia cerebral e de crianças normais na etiopatogenia da cárie dental*. Tese (Doutorado em Reabilitação): Escola Paulista de Medicina, Universidade Federal de São Paulo, São Paulo; 1999.
10. Rodrigues CRMD. *Simplificação do índice de cárie, do índice gengival e do índice de higiene bucal, nas faixas etárias de 4 a 6 e de 7 a 10 anos: Correlação entre os índices*. Dissertação (Mestrado em Odontopediatria), Faculdade de Odontologia, Universidade de São Paulo, São Paulo; 1987.
11. Greene JC, Vermillion JR. The simplified oral hygiene index. *J Am Dent Assoc*. 1964;68:7-13.
12. Loe H, Silness J. Periodontal disease in pregnancy. *Acta Odontol Scand*. 1963;21:533-551.
13. Angeolopoulos AP, Goaz PW. Incidence of diphenylhydantoin gingival hyperplasia. *Oral Surg Oral Med Oral Pathol*. 1972;34:898-905.
14. Weisman EJ. Diagnosis and treatment of gingival and periodontal disorders in children with cerebral palsy. *J Dent Child*. 1956;23:73-80.
15. Fishman S, Young WO, Haley JB, Sword C. The status of oral health in cerebral palsy children and their siblings. *J Dent Child*. 1967;34:219-227.
16. Shmarak KL, Bernstein JE. Caries incidence among cerebral palsy children: A preliminary study. *J Dent Child*. 1961;28:154-156.
17. Swallow JN. Dental disease in cerebral palsied children. *Dev Med Child Neurol*. 1968;10:180-189.
18. Wey SHY, Lang NP. Periodontal epidemiological indices for children and adolescents: II. Evaluation of oral hygiene; III. Clinical applications. *Pediatr Dent*. 1982;4:64-72.
19. Panuska HJ, Gorlin RJ, Bearman JE, Mitchell DE. The effect of anticonvulsant drugs upon the gingiva: A series of analyses of 1048 patients. *J Periodontol*. 1961;32:15-28.
20. Syrjänen SM, Syrjänen KJ. Hyperplastic gingivitis in a child receiving sodium valproate treatment. *Proc Finn Dent Soc*. 1979;75:95-98.
21. Lucas RM, Howell LP, Wall BA. Nifedipine-induced gingival hyperplasia: A histochemical and ultrastructural study. *J Periodontol*. 1985;56:211-215.
22. Barak S, Engelberg IS, Hiss J. Gingival hyperplasia caused by nifedipine. *J Periodontol*. 1987;58:639-642.
23. Bowman JM, Levy BA, Grubb RV. Gingival overgrowth induced by diltiazem. *Oral Surg Oral Med Oral Pathol*. 1988;65:183-185.
24. Fattore L, Stablein M, Bredfeldt G, Semla T, Moran M, Doherty-Greenberg JM. Gingival hyperplasia: A side effect of nifedipine and diltiazem. *Spec Care Dentist*. 1991;11:107-109.
25. Guaré RO, Franco VB. Hiperplasia gengival em crianças: Uso de anticonvulsivantes e higiene oral. *Rev Odontol Univ São Paulo*. 1998;12:39-45.
26. Esterberg HL, White PH. Sodium dilantim gingival hyperplasia. *J Am Dent Assoc*. 1945;32:16-24.
27. Pugliesi NS, Silva FT, Saleh ER, Biagini VS, Marin de Los Rios C, Rode SM. Hiperplasia gengival dilantínica: Avaliação do paciente com relação a dosagem de anticonvulsivante, grau de higiene bucal e extensão da lesão. *Rev Faculdade Odontol FZL*. 1989;1:97-103.
28. Hassel T, O'Donnel J, Pearlman J, Tesini D, Murphy T, Best H. Phenytoin induced gingival overgrowth in institutionalized epileptics. *J Clin Periodontol*. 1984;11:242-253.
29. Maisonneuve C. La plaque dentaire et l'hyperplasie gingivale au dilantin. *J Dent Que*. 1985;22:55-57.
30. Macgregor IDM, Balding JW, Regis D. Motivation for dental hygiene in adolescents. *Int J Paediatr Dent*. 1997;7:235-241.
31. Bellini HT, Arneberg P, Von Der Fehr FR. Oral hygiene and caries. *Acta Odontol Scand*. 1981;39:257-265.

Copyright of Journal of Dentistry for Children is the property of American Society of Dentistry for Children and its content may not be copied or emailed to multiple sites or posted to a listserv without the copyright holder's express written permission. However, users may print, download, or email articles for individual use.

Copyright of Journal of Dentistry for Children is the property of American Academy of Pediatric Dentistry and its content may not be copied or emailed to multiple sites or posted to a listserv without the copyright holder's express written permission. However, users may print, download, or email articles for individual use.