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Cerebral palsy and oral health

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Abstract

Cerebral Palsy (CP) is a disease characterized by posture, motor and movement disorders, which are caused by non -progressive but permanent damage of the fetal or infant brain in the early stages of life. In addition to these disorders abnormal motion patterns, spasticity and weakness, vision and hearing problems, epilepsy, cognitive function disorders may be accompanied. Neuromuscular problems due to CP adversely affect oral health. In addition to orofacial problems, the influence of the extremities prevents the independence of the individual affected in many areas from oral hygiene to nutritional failures. Sialorrhea, dental caries, periodontal disease, bruxism, malocclusion, dental erosion, traumatic dental injuries, enamel defects, temporomandibular joint disorders and delay in dental eruption are frequently experienced in patients with CP. The aim of this study was to examine the publications related to oral health in CP patients in the literature and to compile oral health problems and methods of managing these problems in patients with CP. 'Cerebral Palsy, Oral Health, Oral, Dental, Health, Care, Hygiene, Caries, Dysphagia, Malocclusion, Bruxism, Tooth Brushing 'and combinations of these words are scanned in Turkish and English in health databases (PubMed, Cochrane, Embase, Medline and others). Studies containing at least one subject for oral health in CP patients have been included in the examination. Existing approaches and standards were investigated. The populations examined in the studies are considered to be any subtypes of the CP, to be included in our study. The references of all scanned articles were also sought and examined. As a result, CP, one of the diseases with special requirements, brings many oral health problems. These problems adversely affect the quality of life as well as the general health of the affected individual. It is important that health professionals especially dentists, patients, patients' parents and caregivers should be aware of the oral health problems that may be encountered, should have information about protective and therapeutic methods and determine the risks and be encouraged to configure the correct treatment protocols.

Keywords: caries, cerebral palsy, dental hygiene, oral health

1. Introduction

Cerebral Palsy (CP) is a disease characterized by posture, motor and movement disorders, which are caused by non - progressive but permanent damage of the fetal or infant brain in the early stages of life (1-3). In addition to posture disorders, abnormal motion patterns, spasticity and weakness, vision and hearing problems, epilepsy, cognitive function disorders may be accompanied (2, 4, 5).

While CP is seen in an average of 2-2.5/1000 live births around the world (2,6) it has been reported to be 4.4 in Turkey (7). The high prevalence in Turkey is related with excess prevalence of consanguineous marriage, excessive infectious and febrile illnesses, inadequacy of nutrition in infants, negativity in birth conditions, inadequate baby care and diseases during pregnancy (8).

While the underlying etiological factors can be demonstrated in approximately half of the cases (9), the risk factor cannot be determined in approximately one third (10-12). While prenatal causes are held responsible in almost 70-80 %of patients (multiple pregnancies, intrauterine infections, cervical insufficiency, placenta anomalies, bleeding, intravenous clotting, pregnancy toxication, hyperthyroidism, drug use, iodine deficiency, genetic, hypertension, mental retardation, Metabolic and hormonal diseases such as epilepsy and diabetes mellitus), perinatal causes in 10-20 % (placenta infarction, vaginal bleeding, asphyxia, prematurity, placenta previa, low birth weight, corioamniotis, cord wandering, abnormal presentation, early membrane rupture, low score by APGAR scoring system (scoring by evaluating the appearance of the newborn, heart rate, reflex response, tonus and respiration) and postnatal causes (encephalopathies, polycythemia, hypoglycemia, CNS infection, intracranial bleeding, coagulopathy, convulsions, hyperbilirubinemia) in 10% of patients are held responsible (12, 13).

Tonus disorder in CP is at the forefront. According to the type of tonus disorder, CP can be classified as spastic, ataxic, dyskinetic and mixed types (14). Almost three quarters of cases are spastic types. It is characterized by increased muscle tone in the rapid angular movement of the joint in the extremity affected by the first motor neuron damage. This may cause problems such as impaired posture, limitation of

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movement, difficulty in coordination, joint contracture and deformity (6,15,16). Balance disorder and tremor are at the forefront in the ataxic type. It occurs as a result of damage to cerebellum. It has an unbalanced walking pattern with a wide gait surface and swinging. Explosive speech can be seen. Ataxia cannot be diagnosed until the patient starts to walk. The only symptom is hypotonia before starting to walk. It contains approximately 5 % to 10 % of patients with CP and usually affects all extremities and trunk. Dyskinetic type CP is the form of athetoid, choreic and choreoathetoid movements. Dystonia can also be seen (17). While these cases are usually hypotonic at birth, findings of extrapyramidal system are characterized by the hypotonia and hypertonia in which they are seen in fluctuations. (18). There are swings, curls and snake movements in the extremities. Dysarthric type speech disorder may occur. Extrapyramidal movements are also reflected in the tongue of wormy, and in these patients, the difficulty of swallowing, salivation problem, speech disorder, oral-motor dyskinesia are seen as severe (19). The mixed type contains the spastic, dyskinetic and ataxic form of CP. Approximately 10 %of the cases are this type. It takes place with the involvement of both pyramidal and extrapyramidal system (18). CP can also be classified according to the affected extremity. When one half of the body is affected, it can be mentioned as hemiplegic, monoplegic when a single extremity is affected, diplegic when the lower extremities are affected, tetraplegic/quadriplegic CP when both the upper and lower extremities are affected together (18, 20).

Additional problems can be seen in CP (21). Mental retardation is one of them. As the loss of motor function increases, mental retardation increases. Dyskinetic and hemiplegic types are at least affected by mental retardation, while 70 % of the quadriplegic type CP have mental retardation (22). Mental retardation with epilepsy is thought to be related. The frequency of epilepsy in all children is 2-12/1000 (23), while in spastic tetraplegic individuals, this frequency is 42-94 % and seizures are the generalized tonic clonus type. In hemiplegic individuals, this frequency is 30-65 %and seizures are of focal types. Dyskinetic type CP is at least risky in terms of epilepsy. Epilepsy treatment in children with CP is very difficult and combined drug use is required (24,25). In 42-81 % of CP cases, speech disorders such as difficulty in speech and sound producing, such as dysarthria or aphasia are seen. There is no conversation in the cases of tetraplegic cases with mental retardation (26). Oral problems are more in this group (27,28). Difficulties in carrying out related actions due to insufficient muscles that provide suction, swallowing and chewing can be encountered. Vision problems are 28-90 %in children with CP. While vision defects and strabismus are most common; blindness, glaucoma, nystagmus can also be seen (29). Hearing loss can be seen in 12 %. Hearing loss can be prevented by routine controls (29). Speech disorder is seen in 38 %of children with CP. Inclusion of intercostal muscles, tongue muscles and larynx muscles cause this situation. (6). Apnea, bronchitis, asthma, atelectasis, pneumonia may occur due to aspiration. It is very important because aspiration is the cause of mortality and morbidity (30).

Children with CP cannot get enough nutrients for reasons such as suction, chewing and swallowing disorders, loss of appetite, rejection of food and prolonged food time. Nutritional failure is associated with mortality and morbidity (31). GIS problems are seen in 80-90 %of children with CP (32). One of the most common GIS problems is gastroesophageal reflux (GER). It can cause discomfort, restlessness, irritability, vomiting, esophagitis and aspiration pneumonia in children (32). Aspiration, GER and constipation may cause the child to reduce the interest of food and reject food. (33). Children with ataxic and spastic dyskinetic type CP can be fed to themselves, while children with spastic tetraplegic and dyskinetic type CP cannot feed on themselves due to the severity of motor functional disorder (34). Due to the difficulty of chewing and swallowing, the eating time causes malnutrition. This leads to important problems such as retardation in growth development, deterioration of the immune system, delay of wound healing and weakness (34). While the parents of normal children spend the average of 0.8 hours a day to feed their children, the parents of CPs spend 3.5-7.5 hours. Natural gagging and cough reflexes may have never developed in individuals with CP. The absence of these reflexes may cause irritation and inflammation in the respiratory tract. Sometimes these reflexes may become severe enough to reject food intake during consumption of foods (33). In children with CP, the distress in making the food into a bolus (soft, chewed food mass) is due to chewing problems of insufficient tongue and side jaw movements. In particular, the limitation of the temporomandibular joint (TMJ) movement in spastic tetraplegic CP causes the chewing problem to become even more severe (35).

Neuromuscular problems due to CP affect oral health negatively (36-39). In addition to orofacial problems, the influence of the extremities prevents the independence of the individual affected in many areas from the provision of oral hygiene to nutritional failure (9, 28, 36, 40-41). As the intensity of the disease increases, oral problems also increase (6,42). Sialorrhea, dental caries, periodontal disease, bruxism, malocclusion, dental erosion, traumatic dental injuries, enamel defects, temporomandibular joint disorders and delay in dental eruption are among the most frequent oral health problems in CP patients (43-46). Some studies show that oral-dental health is not different from healthy children as a result of appropriate oral care in children with CP, while in some studies, if there is no oral hygiene motivation, it has been shown that oral-dental health is significantly affected (38-49)

The aim of this study was to examine the publications

related to oral health in CP patients in the literature and to compile oral health problems and methods of managing these problems in patients with CP.

2. Materials and Methods

Cerebral Palsy, Oral Health, Oral, Dental, Health, Care, Hygiene, Caries, Dysphagia, Malocclusion, Bruxism, Tooth Brushing ’and combinations of these words are scanned in Turkish and English in health databases (PubMed, Cochrane, Embase, Medline and others). Studies containing at least one subject for oral health in CP patients have been included in the examination. Existing approaches and standards were investigated. The populations examined in the studies are considered to be any subtypes of the CP, to be included in our study. The references of all scanned articles were also sought and examined.

3. Results and Discussion

3.1. Sialorrhea

In fact, sialorrhea can be seen as normal in infants and children under the age of 4 where coordination is not fully developed. More saliva occurs in the anterior part of the oral cavity. Deterioration of swallowing and coordination defect of orofacial muscles can cause sialorrhea in patients with CP (50-53). Sialorrhea is not socially accepted and can produce significant negative effects on the psychosocial health and quality of life (54). The incidence of sialorrhea in CP was reported as 10-58 % (52, 55-57). Sometimes sialorrhea is related to an irritating lesion, such as dental caries or throat infection, resulting in increased production of saliva. Severe sialorrhea may get worse with some antiepileptic drugs, such as clonazepam (55). Sialorrhea has been shown to be the most serious in tetraplegic spastic CP (52,55). Cosmetic problems, perioral redness, infection and even dehydration may occur as a result of sialorrhea (58-60).

Sialorrhea must be treated if it is at the level to wet the child's clothes and toys (61). While oral-motor treatments (speech therapy, physiotherapy, biofeedback treatment) are appropriate in mild to medium intensity cases, pharmacotherapy and more radical methods can be applied in severe cases and it is recommended to lie on the side to protect the patient from aspiration (59,61,62). Conservative treatments are preferred to maintain normal mouth, teeth and jaw development. In cases where these are not effective, radiotherapy, pharmacotherapy and surgical procedures may be preferred. In children with cooperation, behavioral therapies should be applied for 6 months before considering advanced treatment options (63). Rehabilitation applications for the mouth and face region are the most frequently applied treatment option. These methods are methods aimed at healing muscle tone with lip, jaw and tongue movements (64). Often lip shrinkage, blowing, cheek inflation, tongue suppressing, rounding, tongue to the nose and chin, kiss and laughing exercises are performed (1).

Biophunctional apparatuses (ISMAR (Innsbruck sensory motor activator and regulator) (65), Castillo Morales (66) and Dr. Hinz (67) apparatuses) can be used because they increase oral awareness, help to start swallowing and increase tongue movements. ISMAR apparatus is a combination of monoblocs and Frankel apparatus that improves eating and drinking functions rather than improvement of Sialorrhea control. It is used for 1 year or longer (65). Castillo Morales apparatus stimulates the tongue with the help of a stimulator in the palatal area and ensures its position in the mouth, while stimulating the upper lip with the acrylic part on the surface of the vestibule. It is stated that it can be used in preventing sialorrhea and fighting with nutrition and speech problems (66,68). In patients with CP, the vestibule plaque developed by Dr. Hinz can be used to control sialorrhea. This apparatus encourages breathing through the nose and prevents the external forces formed from the lips and cheeks to the teeth and activates the muscle tone of the lip. With the correct positioning of the tongue, the mouth remains open (67). These apparatuses are used before moving to advanced treatment methods in cases where physiotherapy and behavioral methods are insufficient. It is recommended to apply to children older than 6 years old (69).

The use of anticholinergic drugs such as benztropine, glycopyrrolate and benzhexol hydrochloride can reduce sialorrhea (70) however, it should be remembered that they have side effects such as dry mouth, increase in salivary viscosity, urinary retention, irritability, sedation, blurred vision, constipation and flushing (51,59,62,71).

Botulinum toxin A injection in the salivary glands is another treatment option, especially parotid (72,73). Side effects include a decrease in saliva, difficulty chewing, dry mouth and difficulty in swallowing (74). Side effects such as oral asymmetry, ecchymosis and ptosis may also be seen. In a study conducted for the frequency of application of Botulinum toxin, it was emphasized that the efficacy was 3 months and that it was necessary to repetition at intervals (75,76). Drug treatment with rehabilitation is the first choice of treatment, but botulinum toxin injection is more effective and it has been reported to have less side effects than pharmacological treatment (74).

Surgical interventions can be performed in order to prevent sialorrhea, but as a result of ripening oral muscles by increasing age sialorrhea may decrease and no surgical treatment is recommended until oral muscles complete the maturation (54,62). Submandibular gland excision, changing the location of the parotid/submandibular canal, ligation of parotid ductus, tympanic neurotomy or combinations of these methods can be applied for saliva control (62, 77).

3.2. Dental caries

Dental caries is a multifactorial disease caused by microorganisms fermenting sugar -containing foods that provides acids destructing the harsh tissues of the tooth (78). Differences in food form, prolongation of the time of food in

the mouth, difficulty in cooperation, structural disorders in the teeth may cause dmft-dmfs values to increase in children with CP (39,79). In dmft index, d: caries, m: missing teeth f: the number of filled teeth, while the dmfs index express the number of affected surfaces. In addition to the higher dmft-dmfs values, it has been found that children with CP have more drawn tooths and untreated tooths than healthy children (79-82). According to a study conducted among children with CP during the primary dentition, children with CP have higher percentage of dental caries than normal children and this result was reported to be independent of oral hygiene habits and diet (83).

Oral hygiene was found worse in CP (84,85). Other risk factors are the effect of the drugs used, oral respiration and enamel hypoplasia (86). In addition, saliva flow rate, acidity and buffering capacity is lower (87). In patients with CP, a strong relationship was found between increased saliva osmolarity and caries formation (50). More severe disease is related to more caries (88, 89). Severe motor incoordination affects the ability to perform adequate oral hygiene and cognitive deficits makes cooperation for effective oral care more difficult (90).

Patients and caregivers should be informed that drugs have sugar content and reduce saliva. Dietary habits should be arranged, frequent consumption of fruit juices and sugar -containing drinks, consumption of adhesive foods that have a karyogenic affect should be limited. Instead, snacks that can be easily cleaned from teeth should be preferred. In addition, foods such as daily milk, yogurt and cheese, vegetables, fruits, cereals and protein -containing eggs, meat, chicken and fish can be recommended (91). Sealants and fluorine applications can be applied as preventive treatment (92). Caregivers should inspect the mouth after each meal or dose of medicine and remove food or medicine from the mouth by rinsing with water, sweeping the mouth with a finger wrapped in gauze, or using a disposable foam applicator swab.

3.3. Periodontal disease

Inadequate in the removal of the dental plaque and the fact that the parent cannot comprehend the importance of oral hygiene, more gingivitis and other periodontal diseases are seen in children with CP compared to normal children (92). Several studies have shown that gingival hyperplasia and associated bleeding occurs with higher frequency in children with CP (3). This high frequency may be due to the same factors predisposing to dental caries and leading to biofilm buildup (3). Difficulties in conducting daily oral hygiene, intraoral sensitivity, malocclusion, and oro-facial motor dysfunction are the main contributing factors (84, 85, 93, 94). Another important factor is the use of antiepileptic drugs, particularly phenytoin (95). Gingival hyperplasia is predictive for periodontal diseases. Eighty percent gingivitis was detected in children with CP aged 6-8 years. This frequency is 50 %under 6 years of age, 90 %over the age of 9 (96). Higher gingival index scores are seen in the tetraplegic CP than hemiplegic CP (83). Saliva has an important place in the protection of oral hygiene with its mechanical and chemical cleaning ability. The osmolarity of saliva in patients with CP increases with the rise in proinflammatory cytokines and immunoglobulin A levels, thus increasing the incidence of gingivitis, tooth stone and plaque (97).

The mechanism and treatment of these diseases in CP is the same as in periodontal diseases seen in normal children (98, 99). CP patients should be provided with oral care daily in the prevention and treatment of periodontal disease. For this purpose, it can be ensured to achieve self -care with personal modifications in appropriate cases. The use of special toothbrush techniques and dental floss to the patient should be taught to the patient or those who provide care (95). It has been reported that the plaque removal increases by the personalized modifications such as connecting the toothbrush into the hand of the child and thickening of the handle of the brush. (100). Three -dimensional toothbrush was found to be more effective than traditional toothbrushes in reducing plaque and periodontal problems, as well as the use of three -dimensional brush in patients with CP, the time for brushing, vomiting reflex and gingival bleeding decrease (101). In addition, it may be recommended to use power sonic toothbrushes. There are studies that indicate that power toothbrushes can be an alternative to three -dimensional toothbrushes in plaque removal (102). necessary, the use of mouthwash should be encouraged. Antimicrobials such as chlorhexidine have been reported to be effective (95).

3.4. Bruxism

Bruxism, the habitual grinding of teeth, is a common problem in children with CP, particularly those with severe motor and cognitive deficits (56,103). Bruxism may lead to teeth abrasion, flattening of biting surfaces, damage to the periodontal tissues, alveolar bone resorption and TMJ dysfunction. The incidence of bruxism in individuals with CP is between 25-51 % (56,103-106). In healthy children, this frequency has been reported as 15.29 % (107).

Although the cause is not certain, neurological disorders, irregularities in myophunctional complex and inadequacies to control of the lower jaw, dopamine function, sleep disorders, anxiety, stress and neuroleptic drug use have been reported to have related. In addition, GER, which is frequently monitored in CP patients, has been reported to play a role in the etiology of bruxism (1, 108).

In the treatment of bruxism, the cause should be determined first. Methods such as release of parafunctional habits, splint administration, pharmacological treatment and regulation of occlusion can be used in order to eliminate the cause of bruxism. There are publications that cold application applied to the face or cheek during tooth squeezing significantly reduces bruxism, however, that pharmacological treatment has no effect (109). In addition, botulinum toxin

application in the temporalis and masseter muscles and in advanced cases maxillary osteotomy can be considered. Medications that improve the sleep-wake cycle, such as melatonin, should be used and may also result in improved daytime behavior (110).

3.5. Malocclusion

Malocclusion has been reported with increasing frequency in children with CP, most commonly over-bite and anterior open-bite (3). Increased muscle tone caused by excessive stimulation of motor neurons in children with CP causes malocclusion (111). These abnormalities have been reported to get worse with age. Mouth breathing, lip incompetence and long face are contributing factors (112). The disturbances of the facial, masticatory and tongue musculature cause abnormal facial growth and increase the incidence of malocclusion (113). Pseudo-bulbar palsy, orofacial incoordination and hypotonia could further add to the risk of developing malocclusion (3). Poor chewing efficiency is associated with few occlusal contacts (114).

Orthodontic treatment application for children with CP is useful but, it is thought to be unnecessary (115). Unfortunately, correcting malocclusion is almost impossible in people with moderate or severe cerebral palsy. Orthodontic treatment may not be an option because of the risk of caries and enamel hypoplasia. However, a developmental disability in and of itself should not be perceived as a barrier to orthodontic treatment. The ability of the patient or the caregiver to maintain good daily oral hygiene is critical to the feasibility and success of orthodontic treatment (116).

3.6. Dental Erosion

The loss of hard tissues of the tooth with chemical factors is called dental erosion (117). There is a consensus among the researchers that dental erosion is caused by acidic foods and beverages. (118,119). It has been reported that CP is not an etiological factor for erosion, but erosion may be increased in children with CP due to vomiting, chewing and swallowing problems (120). Increased intra-abdominal pressure, neuromotor insufficiency, long-term inpatient position, delay of stomach discharge, convulsions, a wide variety of drug use are also one of the causes that increase the risk of GER and indirectly the erosion in CP (121). In a study, 73% of CP patients with dental erosions had history of GER (122). Both primary and permanent teeth can be affected, most commonly the upper molars, lower molars and upper incisors (123). The maxillary central teeth's palatine surfaces are the most affected sites by the stomach content reaching the mouth (124). Other surfaces of the teeth are less affected by erosion as they are close to salivary glands (125). This influence starts from enamel level and can reach the dentin and pulp (126).

Active factors should be determined and eliminated for the prevention and treatment of erosion. It is important to provide oral hygiene and to regulate nutrition and diet. Foods with erosive potential such as, fruit juices, carbonated and sports beverages such as non -alcoholic beverages, fresh fruits, acetic acid foods, pickles, potato chips should be reduced in the diet. The use of soft -haired toothbrushes and the use of toothpastes with low erosive features with low fluoride ratio should be encouraged (1).

3.7. Traumatic dental injuries

Although they move less than healthy children, children with CP are exposed to more trauma because of the increased overjet, coordination disorder in walking and uncontrolled head movements (127,128). Children with CP presented four times greater probability of dental trauma (129). In a study, trauma was detected in 57 % of 68 children with CP. Sixty two percent of these traumas is the fracture of the enamel and dentine. The most affected tooth by trauma is maxillary central tooth. Females are exposed to more trauma than males (128).

Tooth-saving kits can be suggested for group homes. Caregivers should be emphasized that traumas require immediate professional attention. Also, caregivers should locate any missing pieces of a fractured tooth, and radiographs of the patient's chest may be necessary to determine whether any fragments have been aspirated (116).

3.8. Enamel defects

Children with CP are at an increased risk for having developmental enamel defects (44,130). Developing germs of the primary teeth are very sensitive to systemic problems and drugs used in the prenatal and postnatal period from the 4th month of intrauterine life to 1 years old. These problems may cause enamel defects (36).

3.9. Dysphagia

Dysphagia is frequently seen in CP (131). According to the literature, the prevalence of dysphagia in children with and without CP are 19-25% vs. 45-60% (132,133). Dysphagia causes feeding difficulty and increased feeding time that impairs the quality of life (134). Aspiration is associated with dysphagia, vomiting and regurgitation (135). Causes of dysphagia in children with CP are the presence of anatomical pathologies, spastic and hypotonic posture, undeveloped swallowing reflex, inadequacy in tongue, lips, jaw and oral functions, lack of body control, deep sensation loss, inability to communicate, no lip closing, inadequate tongue movements, inadequate tongue push, inadequate jaw push, decreased tone on the cheeks, presence of tonic biting reflex, oral hypersensitivity, existence of hyperactive gag reflex, limited upper lip movement (136,137).

The first step in the treatment of dysphagia is compensatory exercises. The aim is to ensure the safety of swallowing and to prevent aspiration (138). They include replacement of posture, changes in the volume and velocity of food intake, changes in food consistency, temperature and taste, change of auxiliary tools, techniques to increase the feeling of oral sense (139). Neuromuscular electrical stimulation can be used in pharyngeal dysfunction (141). In

cases where airway safety cannot be achieved and other methods are not successful enough, the transfer of nutrients to the stomach can be achieved by orogastric, nasogastric or percutaneous endoscopic gastrotomy to ensure adequate calorie intake. Even if one of these methods is used, oral motor therapy should be applied both to switch to oral nutrition and to prevent salivary aspiration (141).

3.10. Delay of the dental eruption

Delay of the dental eruption is associated with oral dysfunction. It is more common in children who cannot be fed oral (142). Delay of the dental eruption is frequent in children with CP (45,49,143). In children with CP, the lack of chewing function and gingival hyperplasia also cause delay of the dental eruption (144).

3.11. Temporomandibular joint disorders

Children with CP are at a significantly higher risk for developing signs and symptoms of TMJ disorders (145). Male gender, the presence and severity of any malocclusion, mouth breathing, and mixed dentition were all identified as risk factors for developing signs and symptoms of TMJ disorders in CP patients (28).

3.12. Dental management

CP patients' oral health is worse than non -CP, as well as the frequency of treatment was found to be low. The reason for this is that dentists do not have sufficient information and experience about this patient group that requires special interest, or that these patients may have difficulties to apply to the dentist.

Some practical challenges are commonly encountered when handling children with CP. These include apprehension, fear from strangers, and communication difficulties (146). Special seating and positioning adjustments are needed for children with abnormal posture. Assistance from the parents and dental assistant is often needed particularly for immobilization and during radiographic procedures (28). Sedation and anesthesia are frequently needed in non-compliant children (147).

When the CP patient comes to the dentist clinic, first, he should be evaluated in detail. The presence of additional problems should be questioned and consulted to the relevant doctors. Patient's cooperation should be achieved by applying patiently the tell-show-apply method (148).

As for all individuals, there should be preventive approaches for individuals with CP. The training of the family, caregiver and child, making oral hygiene habits a part of the daily life, regular examination of children by dentists, taking professional preventative measures are among the first step approaches (149,150).

CP, one of the diseases with special requirements, brings many oral health problems. These problems adversely affect the quality of life as well as the general health of the affected individual. It is important that health professionals, especially dentists, patients, parents and caregivers should be aware of the oral health problems that may be encountered, should be encouraged to have information about preventative and therapeutic methods and to determine the risks and to configure the correct treatment protocols.

Conflict of interest

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References

1. Kırzıoğlu Z, Bayraktar C. Serebral Palsili Çocuklarda Sık Rastlanan Oral Problemler, Ağız Dışına Salya Akışı ve Tedavi Önerileri. Sdü Sağlık Bilimleri Enstitüsü Dergisi. 2018; 9(2):156-62.

2. Bax M, Goldstein M, Rosenbaum P, Leviton A, Paneth N, Dan B, et al. Executive committee for the definition of cerebral palsy. Proposed definition and classification of cerebral palsy. Dev Med Child Neurol. 2005; 47: 571-6.

3. Jan MM. Cerebral palsy: comprehensive review and update. Ann Saudi Med. 2006; 26: 123-32.

4. Rosenbaum P, Stewart D. The World Health Organization International Classification of Functioning, Disability, and Health: a model to guide clinical thinking, practice and research in the field of cerebral palsy. Semin Pediatr Neurol. 2004; 11: 5-15.

5. Gokkaya NK, Caliskan A, Karakus D, Ucan H. Relation between objectively measured growth determinants and ambulation in children with cerebral palsy. Turk J Med Sci. 2009; 39: 85-90.

6. Sankar C, Mundkur N. Cerebral palsy-definition, classification, etiology and early diagnosis. Indian J Pediatr. 2005; 72(10): 865-8.

7. Serdaroglu A, Cansu A, Ozkan S, Tezcan S. Prevalence of cerebral palsy in turkish children between the ages of 2 and 16 years. Dev Med Child Neurol. 2006; 48: 413-6.

8. Livanelioglu A, Kerem Gunel M. Serebral palsi'de fizyoterapi. Ankara Yeni Ozbek Matbaası; 2009, p. 5-12.

9. Sehrawat N, Marwaha M, Bansal K, Chopra R. Cerebral palsy: a dental update. Int J Clin Pediatr Dent. 2014; 7(2): 109- 18.

10. Berker AN, Yalcin MS. Cerebral palsy: orthopedic aspects and rehabilitation. Pediatr Clin N Am. 2008; 55: 1209-25.

11. Shevell MI, Majnemer A, Morin I. Etiologic yield of cerebral palsy: a contemporary case series. Pediatr Neurol. 2003; 28(5): 352-9.

12. Jacobsson B, Hagberg G. Antenatal risk factors for cerebral palsy. Best Pract Res Clin Obstet Gynaecol. 2004; 18(3): 425-

36.

13. Nelson KB, Ellenberg JH. Children who "outgrew" cerebral palsy. Pediatrics. 1982; 69(5): 529-36.

14. Surveillance of cerebral palsy in Europe: a collaboration of cerebral palsy surveys and registers. Surveillance of Cerebral Palsy in Europe (SCPE). Dev Med Child Neurol. 2000; 42(12): 816-24.

15. Mukherjee S, Gaebler-Spira DJ. Cerebral Palsy. In: Braddom RL (Ed.). Physical Medicine & Rehabilitation 3th. Philedelphia: Saunders Elsevier; 2007, p.1243-67.

16. Diamond M, Armento M. Children with Disabilities. In: D. J. A. (Ed.). Physical Medicine & Rehabilitation Principles and Practice. 4th. Philedelphia: Lippincott Williams & Wilkins; 2005, p. 122.

17. Molnar GE, Alexander MA. Pediatric Rehabilitation. Philedelphia, USA: Hanley Belfus inc;1999.

18. Koman LA, Smith BP, Shilt JS. Cerebral palsy. Lancet. 2004; 363(9421): 1619-31.

19. Hoyer H, Limbrock GJ. Orofacial regulation therapy in children with Down syndrome, using the methods and appliances of Castillo-Morales. ASDC J Dent Child. 1990; 57(6): 442-4.

20. Yakut, A. Serebral Palside Yeni Gelişmeler. Türkiye Klinikleri. 2008;4(4): 127-38.

21. Russman BS, Ashwal S. Evaluation of the child with cerebral palsy. Semin Pediatr Neurol. 2004; 11: 47-57.

22. Sala DA, Grand AD. Prognosis for ambulation in cerebral palsy. Dev Med Child Neurol. 1995; 37(11): 1020-6.

23. Cowan LD. The epidemiology of the epilepsies in children. Mental Retardation and Developmental Disabilities Research Reviews. 2002; 8(3): 171-81.

24. Serdaroglu A, Ozkan S, Aydin K, Gucuyener K, Tezcan S, Aycan S. Prevalence of epilepsy in Turkish children between the ages of 0 and 16 years. Journal of Child Neurology. 2004;19(4): 271-74.

25. Zelnik N, Konopnicki M, Bennett-Back O, Castel-Deutsch T, Tirosh E. Risk factors for epilepsy in children with cerebral palsy. European Journal of Paediatric Neurology. 2010; 14(1): 67-72.

26. Odding E, Roebroeck ME, Stam HJ. The epidemiology of cerebral palsy: Incidence, impairments and risk factors. Disability and Rehabilitation. 2006; 28(4): 183-91.

27. Eltumi M, Sullivan PB. Nutritional management of the disabled child: the role of percutaneous endoscopic gastrostomy. Dev Med Child Neurol. 1997; 39: 66-68.

28. Jan BM, Jan MM. Dental health of children with cerebral palsy. Neurosciences (Riyadh). 2016;21(4):314-318.

29. Duman O, Imad FM, Kızılay F, Yucel I, Balkan S, Haspolat S. Serebral palsili hastaların işlevsel kapasitelerine göre görme sorunlarının değerlendirilmesi. Çocuk Sağlığı ve Hastalıkları Dergisi. 2005; 48: 130-5.

30. Fitzgerald DA, Follett J, Asperen PPV. Assessing and managing lung disease and sleep disordered breathing in children with cerebral palsy. Paediatr Res Rev. 2009; 10(1): 18-24.

31. Vega-Sanchez R, de la Luz Gomez-Aguilar M, Haua K, Rozada G. Weight-based nutritional diagnosis of Mexican children and adolescents with neuromotor disabilities. BMC Res Notes. 2012; 5: 218.

32. Ceriati E, Peppo FD, Cıprandı G, Marchettı P, Sılverı M, Rıvosecchı M. Surgery in disabled children: General gastroenterological aspects. Acta Paediatr. 2006; 95: 34-7.

33. Erkin G, Kacar S, Ozel S. Serebral palsili hastalarda gastrointestinal sistem ve beslenme problemleri. Türkiye Fiziksel Tıp ve Rehabilitasyon Dergisi. 2005; 51(4): 150-5.

34. Dahlseng MO, Finbraten AK, Juliusson PB, Skranes J, Andersen G, Vik T. Feeding problems, growth and nutritional status in children with cerebral palsy. Acta Paediatr. 2012; 101(1): 92-8.

35. Pelegano JP, Nowysz S, Goepferd S. Temporomandibular joint contracture in spastic quadriplegia: effect on oral-motor skills. Dev Med Child Neurol. 1994; 36(6): 487-94.

36. Bhat M, Nelson KB. Developmental enamel defects in primary teeth in children with cerebral palsy, mental retardation, or hearing defects: a review. Advances in Dental Research. 1989; 3:132-42.

37. Cohen MD, Diner H. The significance of developmental dental enamel defects in neurological diagnosis. Pediatr. 1970; 46:737-47.

38. Murray GS, Johnsen DC, Weissman BW. Hearing and neurologic impairment: insult timing indicated by primary tooth enamel defects. Ear and Hearing. 1987; 8:68-73.

39. Grzić R, Bakarcić D, Prpić I, Jokić NI, Sasso A, Kovac Z, et al. Dental health and dental care in children with cerebral palsy. Coll Antropol. 2011; 35: 761-4.

40. Bhowate R, Dubey A. Dentofacial changes and oral health status in mentally challenged children. Journal of Indian Society of Pedodontics and Preventive Dentistry. 2005; 23(2):71-3.

41. Altun C, Guven G, Basak F, Akbulut E. Altı-onbir yaş grubu çocukların ağız-diş sağlığı yönünden değerlendirilmesi. Gülhane Tıp Dergisi. 2005; 47:114-8.

42. Jones MW, Morgan E, Shelton JE. Primary care of the child with cerebral palsy: a review of system (Part II). J Pediatr Health Care. 2007; 21: 226-37.

43. Faulconbridge RVL, Tranter RM, Moffat V, Green E. Review of management of drooling problems in neurologically impaired children: a review of methods and results over 6 years at Chailey Heritage Clinical Services. Clinical Otolaryngology. 2001; 26(2): 76-81.

44. Lin X, Wu W, Zhang C, Lo EC, Chu CH, Dissanayaka WL. Prevalence and distribution of developmental enamel defects in children with cerebral palsy in Beijing, China. Int J Paediatr Dent. 2011; 21(1): 1-6.

45. Moslemi M, Vejdani J, Sadrabad ZK, Shadkar MM. A study on the eruption timing of permanent dentition in patients with cerebral palsy. Spec Care Dentist. 2013; 33(6): 275-9.

46. Inga CJ, Reddy AK, Richardson SA, Sanders B. Appliance for chronic drooling in cerebral palsy patients. Pediatric Dentistry. 2001; 23 (3):241-2.

47. de Carvalho RB, Mendes RF, Prado RR, Jr. Moita Neto JM. Oral health and oral motor function in children with cerebral palsy. Spec Care Dentist. 2011; 31(2): 58-62.

48. Katz CR. Integrated approach to outpatient dental treatment of a patient with cerebral palsy: a case report. Spec Care Dentist. 2012; 32(5): 210-7.

49. Rodrigues dos Santos MT, Masiero D, Novo NF, Simionato MR. Oral conditions in children with cerebral palsy. J Dent Child (Chic). 2003; 70(1): 40-6.

50. Santos MT, Ferreira MC, Mendes FM, de Oliveira Guaré R. Assessing salivary osmolality as a caries risk indicator in cerebral palsy children. Int J Paediatr Dent. 2014; 24(2): 84-9.

51. Fairhurst CB, Cockerill H. Management of drooling in children. Arch Dis Child Educ Pract Ed. 2011; 96(1): 25-30.

52. Hegde AM, Pani SC. Drooling of saliva in children with cerebral palsy-etiology, prevalence, and relationship to salivary flow rate in an Indian population. Spec Care Dentist. 2009; 29(4): 163-8.

53. Tahmassebi JF, Curzon ME. The cause of drooling in children with cerebral palsy - hypersalivation or swallowing defect? Int J Paediatr Dent. 2003; 13(2): 106-11.

54. Meningaud JP, Pitak-Arnnop P, Chikhani L, Bertrand JC. Drooling of saliva: a review of the etiology and management options. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2006; 101: 48-57.

55. Tahmassebi JF, Curzon ME. Prevalence of drooling in children with cerebral palsy attending special schools. Dev Med Child Neurol. 2003; 45(9): 613-7.

56. Ortega AO, Guimarães AS, Ciamponi AL, Marie SK. Frequency of parafunctional oral habits in patients with cerebral palsy. J Oral Rehabil. 2007; 34(5): 323-8.

57. Roberto LL, Machado MG, Resende VL, Castilho LS, Abreu MH. Factors associated with dental caries in the primary dentition of children with cerebral palsy. Braz Oral Res. 2012; 26(5): 471-7.

58. Dougherty NJ. A review of cerebral palsy for the oral health professional. Dent Clin N Am. 2009; 53: 329-38.

59. Hussein I, Kershaw AE, Tahmassebi JF, Fayle SA. The Management of Drooling in Children and Patients with Mental and Physical Disabilities a Literature Review. International Journal of Pediatric Dentistry. 1998; 8: 3-11.

60. Siegel L, Klingbeil M. Control of drooling with transdermal scopolamine in a child with cerebral palsy. Dev Med Child Neurol. 1991; 33: 1013-4.

61. Nunn JH. Drooling: review of the literature and proposals for management. J Oral Rehabil. 2000; 27(9): 735-43.

62. Lal D, Hotaling AJ. Drooling. Curr Opin Otolaryngol Head Neck Surg. 2006; 14(6): 381-6.

63. Van der Burg JJ, Didden R, Jongerius PH, Rotteveel JJ. Behavioral treatment of drooling: a methodological critique of the literature with clinical guidelines and suggestions for future research. Behav Modif. 2007; 31(5): 573-94.

64. Ruscello DM. Nonspeech oral motor treatment issues related to children with developmental speech sound disorders. Lang Speech Hear Serv Sch. 2008; 39(3): 380-91.

65. Johnson HM, Reid SM, Hazard CJ, Lucas JO, Desai M, Reddihough DS. Effectiveness of the Innsbruck Sensorimotor Activator and Regulator in improving saliva control in children with cerebral palsy. Developmental Medicine & Child Neurology. 2004; 46: 39-45.

66. Castillo-Morales R, Brondo J, Hoyer H, Limbrock GJ. Treatment of chewing, swallowing and speech defects in handicapped children with Castillo-Morales orofacial regulator therapy: advice for pediatricians and dentists. Zahnarztl Mitt. 1985; 75(9): 935-42, 947-51.

67. Hinz R. Habits and Their Prevention. Düsseldorf: HPSS-Gmbh; 1995.

68. Limbrock GJ, Hoyer H, Scheying H. Drooling, chewing and swallowing dysfunctions in children with cerebral palsy: treatment according to Castillo-Morales. ASDC J Dent Child. 1990; 57(6): 445-51.

69. Little SA, Kubba H, Hussain SS. An evidence-based approach to the child who drools saliva. Clin Otolaryngol. 2009; 34(3): 236-9.

70. Jongerius PH, van Tiel P, van Limbeek J, Gabreëls FJ, Rotteveel JJ. Ansystematic review for evidence of efficacy of anticholinergic drugs to treatndrooling. Arch Dis Child. 2003; 88(10): 911-4.

71. Bachrach SJ, Walter RS, Trzcinski K. Use of glycopyrrolate and other anticholinergic medications for sialorrhea in children with cerebral palsy. Clin Pediatr (Phila). 1998; 37(8): 485-90.

72. Hanagası HA. Hipersekretuvar Bozukluklar ve Botulinum Toksini. Nöropsikiyatri Arşivi. 2010; 47: 48-51.

73. Porte M, Chaléat-Valayer E, Patte K, D'Anjou MC, Boulay C, Laffont I. Relevance of intraglandular injections of Botulinum toxin for the treatment of sialorrhea in children with cerebral palsy: a review. Eur J Paediatr Neurol. 2014; 18(6): 649-57.

74. Chaléat-Valayer E, Porte M, Buchet-Poyau K, Roumenoff-Turcant F, D'Anjou MC, Boulay C, et al. Management of drooling in children with cerebral palsy: A French survey. Eur J Paediatr Neurol. 2016; 20(4): 524-31.

75. Koyuncuoglu HR, Demirci S. Application of botulinum toxin at a neurology clinic: an eleven-year experience. Turk J Neurol. 2016; 22: 8-12.

76. Gonzalez-L MD, Martinez C, Bori Y, Fortuny I, Suso-Vergara S. Factors in the efficacy, safety, and ımpact on quality of life for treatment of drooling with botulinum toxin type a in patients with cerebral palsy. Am J Phys Med Rehabil. 2017; 96(2): 68-76.

77. Reed J, Mans CK, Brietzke SE. Surgical management of drooling: a meta-analysis. Arch Otolaryngol Head Neck Surg. 2009; 135(9): 924-31.

78. Nguyen DH, Martin JT. Common dental infections in the primary care setting. Am Fam Physician. 2008; 77(6): 797-802.

79. Albert DA, Park K, Findley S, Mitchell DA, McManus JM. Dental caries among disadvantaged 3- to 4-year-old children in northern Manhattan. Pediatr Dent. 2002; 24(3): 229-33.

80. Cardoso AM, Gomes LN, Silva CR, Soares RD, De Abreu MH, Padilha WW, et al. Dental caries and periodontal disease in Brazilian children and adolescents with cerebral palsy. Int J Environ Res Public Health. 2014; 12: 335-53.

81. Botti MT, Biancardi M, Oliveira R, Jardim JR. Caries prevalence in patients with cerebral palsy and the burden of caring for them. Spec Care Dentist. 2010; 30:206-10.

82. Dini EL, Holt RD, Bedi R. Prevalance of caries and developmental defects of enamel in 9-10 year old children living in Brazil with differing water fluoride histories. British Dental Journal. 2000; 188:146-49.

83. Guare Rde O, Ciampioni AL. Prevalence of periodontal disease in the primary dentition of children with cerebral palsy. J Dent Child (Chic). 2004; 71(1): 27-32.

84. de Oliveira R, Ciampioni AL. Prevalence of Periodontal Disease in the Primary Dentition of Children with Cerebral Palsy. J Dent Child. 2004; 71:27-32.

85. Rennan Y, Colman McG, Cynthia K, Nigel M. Oral health in pres¬chool children with cerebral palsy: a case–control community-based study. International Journal of Paediatric Dentistry. 2010; 20:330-5.

86. Mani SA, Mote N, Kathariya M, Pawar KD. Adaptation and development of dental procedure in cerebral palsy. Pravara Med Rev. 2015; 7(4): 17-22.

87. dos Santos MT, Masiero D, Simionato MR. Risk factors for dental caries in children with cerebral palsy. Spec Care Dentist. 2002; 22(3): 103-7.

88. Santos MT, Guare RO, Celiberti P, Siqueira WL. Caries experience in individuals with cerebral palsy in relation to oromotor dysfunction and dietary consistency. Spec Care Dentist. 2009; 29: 198-203.

89. Dourado Mda R, Andrade PM, Ramos-Jorge ML, Moreira RN, Oliveira-Ferreira F. Association between executive/attentional functions and caries in children with cerebral palsy. Res Dev Disabil. 2013; 34: 2493-9.

90. Subasi F, Mumcu G, Koksal L, Cimilli H, Bitlis D. Factors affecting oral health habits among children with cerebral palsy: pilot study. Pediatr Int. 2007; 49: 853-7.

91. Jones MW, Morgan E, Shelton JE, Thorogood C. Cerebral palsy: introduction and diagnosis (Part I). J Pediatr Health Care. 2007; 21(3):146-52.

92. Kosif R, Gunalp Z, Safi Oz Z. Evaluation of the dental-gingival health in children with cerebral palsy in Zonguldak. The New Journal of Medicine. 2013; 30(1):37-41.

93. Gunel MK, Mutlu A, Tarsuslu T, Livanelioglu A. Relationship among the Manual Ability Classification System (MACS), the Gross Motor Function Classification System (GMFCS), and the functional status (WeeFIM) in children with spastic cerebral palsy. Eur J Pediatr. 2009; 168: 477-85.

94. Cokpekin F, Koymen G, Basak F, Akbulut E, Altun C. Engelliler okuluna devam eden çocukların ağız diş sağlığı yönünden değerlendirilmesi. Gülhane Tıp Dergisi. 2003: 45(3):228-32.

95. Mani SA, Mote N, Kathariya M, Pawar KD. Adaptation and development of dental procedure in cerebral palsy. Pravara Med Rev. 2015; 7(4): 17-22.

96. Weisman EJ. Diagnosis and treatment of gingival and periodontal disorders in children with cerebral palsy. J Dent Child (Chic). 1956; 23: 73-80.

97. Santos MT, Ferreira MC, Guaré RO, Diniz MB, Rösing CK, Rodrigues JA, et al. Gingivitis and salivary osmolality in children with cerebral palsy. Int J Paediatr Dent. 2016; 26(6): 463-70.

98. Bozkurt FY, Fentoglu O, Yetkin Z. The Comparison of Various Oral Hygiene Strategies in Neuromuscularly Disabled Individuals. J Contemp Dent Pract. 2004; 4(5): 23-31.

99. Mitsea AG, Karidis AG, Donta-Bakoyianni C, Spyropoulos ND. Oral health status in Greek children and teenagers, with disabilities. J Clin Pediatr Dent. 2001; 26(1): 111-8.

100. Damle SG, Bhavsar JP. Plaque removing efficacy of individually modified toothbrushes in cerebral palsy children. ASDC J Dent Child. 1995; 62(4): 279-82.

101. Yitzhak M, Sarnat H, Rakocz M, Yaish Y, Ashkenazi M. The effect of toothbrush design on the ability of nurses to brush the teeth of institutionalized cerebral palsy patients. Spec Care Dentist. 2013; 33(1): 20-7.

102. Dogan MC, Alacam A, Asici N, Odabas M, Seydaoglu G. Clinical evaluation of the plaque-removing ability of three different toothbrushes in a mentally disabled group. Acta Odontol Scand. 2004; 62(6): 350-4.

103. Santos MT, Duarte Ferreira MC, de Oliveira Guaré R, Guimarães AS, Lira Ortega A. Teeth grinding, oral motor performance and maximal bite force in cerebral palsy children. Spec Care Dentist. 2015; 35(4): 170-4.

104. Zarowski M, Mojs E, Gajewska E, Steinborn B, Samborski W. Prevelance of sleep problems in children with cerebral palsy. Preliminary study. Ann. Acad. Med. Stetin. 2008; 54: 59-64.

105. Miamoto CB, Pereira LJ, Ramos-Jorge ML, Marques LS. Prevalence and predictive factors of sleep bruxism in children with and without cognitive impairment. Braz Oral Res. 2011; 25(5): 439-45.

106. Abanto J, Ortega AO, Raggio DP, Bönecker M, Mendes FM, Ciamponi AL. Impact of oral diseases and disorders on oral-health-related quality of life of children with cerebral palsy. Spec Care Dentist. 2014; 34(2): 56-63.

107. Fonseca CM, dos Santos MB, Consani RL, dos Santos JF, Marchini L. Incidence of sleep bruxism among children in Itanhandu, Brazil. Sleep Breath. 2011; 15(2): 215-20.

108. Souza VA, Abreu MH, Resende VL, Castilho LS. Factors associated with bruxism in children with developmental disabilities. Braz Oral Res. 2015; 29: 1-5.

109. Lang R, White PJ, Machalicek W, Rispoli M, Kang S, Aquilar J, et al. Treatment of bruxism in individuals with developmental disabilities: a systematic review. Res Dev Disabil. 2009; 30(5): 809-18.

110. Jan MM. Melatonin for the treatment of handicapped children with severe sleep disorders. Pediatr Neurol. 2000; 23: 229-32.

111. Yoshida M, Nakajima I, Uchida A, Yamaguchi T, Nonaka T, Yoshida H, et al. Characteristics of lower-jaw-position sensation with respect to oral-jaw functions in patients with cerebral palsy. Pediatric Dental Journal. 2004; 14(1): 23-8.

112. Miamoto CB, Ramos-Jorge ML, Pereira LJ, Paiva SM, Pordeus IA, Marques LS. Severity of malocclusion in patients with cerebral palsy: determinant factors. Am J Orthod Dentofacial Orthop. 2010; 138: 394-5.

113. Franklin DL, Luther F, Curzon ME. The prevalence of

malocclusion in children with cerebral palsy. Eur J Orthod. 1996; 18: 637-43.

114. Henrikson T, Ekberg EC, Nilner M. Masticatory efficiency and ability in relation to occlusion and mandibular dysfunction in girls. Int J Prosthodont. 1998; 11: 125-32.

115. Becker A, Chaushu S, Shapira J. Orthodontic treatment for the special needs child. Seminars in Orthodontics. 2004; 10: 281-92.

116. Practical oral care for people with cerebral palsy. U.S. Department of Health and Human Services, National institutes of Health, National institute of Dental and Craniofacial Research. 2009. Available at https://www.nidcr.nih.gov/sites/default/files/2017-09/practical-oral-care-cerebral-palsy.pdf

117. Barron RP, Carmichael RP, Marcon MA, Sandor GK. Dental erosion in gastroesophageal reflux disease. J Can Dent Assoc. 2003; 69: 84-9.

118. Jarvinen VK, Rytomaa II, Heinonen OP. Risk factors in dental erosion. J Dent Res. 1991; 70(6): 942-7.

119. Millward A, Shaw L, Smith AJ, Rippin JW, Harrington E. The distribution and severity of tooth wear and the relationship between erosion and dietary constituents in a group of children. Int J Paediatr Dent. 1994; 4(3): 151-7.

120. Shaw L, Weatherill S, Smith A. Tooth wear in children: an investigation of etiological factors in children with cerebral palsy and gastroesophageal reflux. ASDC J Dent Child. 1998; 65(6): 484-6, 439.

121. Erkin G, Kacar S, Ozel S. Serebral palsili hastalarda gastrointestinal sistem ve beslenme problemleri. Türkiye Fiziksel Tıp ve Rehabilitasyon Dergisi. 2005; 51(4): 150-5.

122. Su JM, Tsamtsouris A, Laskou M. Gastroesophageal reflux in children with cerebral palsy and its relationship to erosion of primary and permanent teeth. J Mass Dent Soc. 2003; 52: 20-4.

123. Goncalves GK, Carmagnani FG, Correa MS, Duarte DA, Santos MT. Dental erosion in cerebral palsy patients. J Dent Child. 2008; 75:117-20.

124. Lussi A. Dental Erosion from Diagnosis to Therapy. 2nd ed. Vol 20. Switzerland, Karger; 2006, 36-41.

125. Polat Z, Akgun O, Turan I, Polat GG, Altun C. Evaluation of the relationship between dental erosion and scintigraphically detected gastroesophageal reflux in patients with cerebral palsy. Turk J Med Sci. 2013; 43:283-8.

126. Dahshan A, Patel H, Delaney J, Wuerth A, Thomas R, Tolia V. Gastroesophageal reflux disease and dental erosion in children. J Pediatr. 2002; 140(4): 474-8.

127. Yasui EM, Kimura RK, Kawamura A, Akiyama S, Morisaki I. A modified oral screen appliance to prevent self-inflicted oral trauma in an infant with cerebral palsy: a case report. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2004; 97(4): 471-5.

128. Holan G, Peretz B, Efrat J, Shapira Y. Traumatic injuries to the teeth in young individuals with cerebral palsy. Dent Traumatol. 2005; 21(2): 65-9.

129. Batista C, Ramos-Jorge ML, Coelho M, De Oliveira M, Gonçalves R, Silva L. Dental trauma in individuals with severe cerebral palsy: prevalence and associated factors. Braz Oral Res. 2011; 25:319-23.

130. Jaffe M, Attias D, Dar H, Eli I, Judes H. Prevalence of gestational and perinatal insults in brain-damaged children. Isr J Med Sci. 1985; 21(12): 940-4.

131. Dodrill P, Gosa MM. Pediatric dysphagia: physiology, assessment, and management. Ann Nutr Metab. 2015; 66(5): 24-31.

132. **132.** Lloyd Faulconbridge RV, Tranter RM, Moffat V, Green E. Review of management of drooling problems in neurologically impaired children: a review of methods and results over 6 years at Chailey Heritage clinical services. Clin Otolaryngol Allied Sci. 2001; 26: 76-81.

133. Lefton-Greif MA. Pediatric dysphagia. Phys Med Rehabil Clin N Am. 2008; 19: 837-51.

134. Sullivan PB, Lambert B, Rose M, Ford-Adams M, Johnson A, Griffiths P. Prevalence and severity of feeding and nutritional problems in children with neurological impairment: oxford feeding study. Dev Med Child Neurol. 2000; 42: 674-80.

135. Alsaggaf AH, Jan MM, Saadah OI, Alsaggaf HM. Percutaneous endoscopic gastrostomy (PEG) tube placement in children with neurodevelopmental disabilities: parents' perspectives. Saudi Med J. 2013; 34: 695-700.

136. Yılmaz S, Basar P, Gisel EG. Assessment of feeding performance in patients with cerebral palsy. Int Journal Rehabil Res. 2004; 27(4): 325-9.

137. Mathisen B, Skuse D, Wolke D, Reilly S. Oral-Motor Dysfunction and Failure to Thrive among Inner-City Infants. Developmental Medicine and Child Neurology. 1989; 31(3): 293-302.

138. Morasch HS, Bartolome G. Swallowing disorders: Pathophysiology and rehabilitation of neurogenic dysphagia. NeuroRehabilitation. 1998; 10: 169-89.

139. Logemann JA. Evaluation and treatment of swallowing disorders (2nd);1998.

140. Park JW, Oh JC, Lee HJ, Park SJ, Yoon TS, Kwon BS. Effortful swallowing training coupled with electrical stimulation leads to an increase in hyoid elevation during swallowing. Dysphagia. 2009; 24(3): 296-301.

141. Haberfellner H, Schwartz S, Gisel EG. Feeding skills and growth after one year of intraoral appliance therapy in moderately dysphagic children with cerebral palsy. Dysphagia. 2001; 16(2): 83-96.

142. Cumella S, Ransford N, Lyons J, Burnham H. Needs for oral care among people with intellectual disability not in contact with Community Dental Services. J Intellect Disabil Res. 2000; 44 (1): 45-52.

143. Pope JE, Curzon ME. The dental status of cerebral palsied children. Pediatr Dent. 1991; 13: 156-62.

144. Suri L, Gagari E, Vastardis H. Delayed tooth eruption: pathogenesis, diagnosis, and treatment. A literature review. Am J Orthod Dentofacial Orthop. 2004; 126(4): 432-45.

145. Miamoto CB, Pereira LJ, Paiva SM, Pordeus IA, Ramos-Jorge ML, Marques LS. Prevalence and risk indicators of temporomandibular disorder signs and symptoms in a pediatric population with spastic cerebral palsy. J Clin Pediatr Dent. 2011;

35: 259-63.

146. Jan MM. Neurological examination of difficult and poorly cooperative children. J Child Neurol. 2007; 22: 1209-13.

147. Wongprasartsuk P, Stevens J. Cerebral palsy and anaesthesia. Paediatr Anaesth. 2002; 12: 296-303.

148. Komerik N, Kırzıoglu Z, Efeoglu CG. Zihinsel Engele Sahip Bireylerde Ağız Sağlığı. J Dent Atatürk Uni. 2012; 22(1): 96-104.

149. Tesini DA, Fenton SJ. Oral health needs of persons with physical or mental disabilities. Dent Clin North Am. 1994; 38(3): 483-98.

150. Ferguson FS, Cinotti D. Home oral health practice: the foundation for desensitization and dental care for special needs. Dent Clin North Am. 2009; 53: 375-87.