Stomatologija, Baltic Dental and Maxillofacial Journal, 12: 67-72, 2010

Factors influencing permanent teeth eruption. Part one – general factors

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SUMMARY

Variation in the normal eruption of teeth is a common finding, but significant deviation from established norms should alert the clinician to take some diagnostic procedures in order to evaluate patient health and development. Disturbance in tooth eruption time could be a symptom of general condition or indication of altered physiology and craniofacial development.

The aim of this review is to analyze general factors that could influence permanent teeth eruption. The articles from 1965 to 2009 in English related to topic were identified. 84 articles were selected for data collection.

Although permanent teeth eruption is under significant genetic control, various general factors such as gender, socioeconomic status, craniofacial morphology, body composition can influence this process. Most significant disturbance in teeth emergence is caused by systemic diseases and syndromes.

Key words: permanent teeth eruption, general factors, influence.

INTRODUCTION

Tooth eruption is defined as the movement of the tooth from its site of development in alveolar bone to the occlusal plane in the oral cavity. The tooth eruption is a complex and tightly regulated process which is divided into five stages: preeruptive movements, intraosseous stage, mucosal penetration, preocclusal and postocclusal stages. Preeruptive movements occur during crown formation and are so small that they could only be observed by vital staining experiments [1]. Active eruption movements occur when root formation begins and therefore it was believed that eruptive force comes from periodontal ligament. Although tooth eruption mechanisms are still under debate, it was suggested that periodontal ligament provides eruption force after the tooth has pierced gingiva but not during intraosseous stage [2]. For active tooth eruption to begin eruption pathway by osteoclasts in alveolar bone must be formed. In succedeaneous dentition, this pathway follows the gubernacular canal above each tooth; i.e., bone resorption widens the canal to allow the crown to move through it and exit the alveolar bone [3]. From studies with the dogs it was shown that dental follicle (DF) plays major role during intraosseous stage of eruption as teeth didn't erupt if the DF had been removed. Simultaneously when the tooth in the DF was replaced with dental amalgam but the DF had been left intact, artificial tooth erupted [4]. Osteoclasts which create eruption pathway are formed from mononuclear cells which in turn are recruited to the DF by chemokines CSF-1 (functional colony-stimulating factor–1) and MCP-1 (monocyte chemotactic protein-1). Osteoblasts might also influence the process of eruption by activating osteoclasts. Formation of the tooth eruption pathway is a localized, genetically programmed event that does not require pressure from the erupting tooth. Putative eruption genes and their products are localized primarily in either the DF or stellate reticulum [5,6]. During intraosseous stage there is a coordinated translocation of the tooth into resorbed space, bone apposition at the DF fundus and simultaneous root elongation. Formation of the eruption pathway is completed soon after the cusps reach the alveolar crest and at this point the rate of eruption accelerates [7].

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The outer enamel epithelium of the tooth bud proliferates and fuses with oral epithelium creating the junctional epithelium on the tooth surface. Erupting tooth penetrates mucosa and preeoclusal eruption stage begins. As the root grows and bone forms at the base of the crypt, tooth reaches functional occlusion plane. Most of the postemerged eruption proceeds during night [1]. Once the occlusion is reached, tooth eruption speed drops dramatically but continues at a slow rate during life thus compensating tooth wear. If the antagonist tooth is lost, eruption rate increases.

Normal permanent teeth eruption into oral cavity occurs over a broad chronological age range and can be influenced by number of factors. These factors can be classified into local and general.

MATERIALS AND METHODS

To identify all studies that examined the relationship between general factors and permanent teeth eruption, a literature survey was performed using Medline database. Free text terms or in combination with controlled vocabulary were used for a search. Key words used in the search included "permanent teeth emergence", "impaired tooth eruption", "delayed tooth eruption", accelerated tooth eruption". The articles from 1965 to 2009 in English related to subject were identified. 84 articles were selected and analyzed. Most of the articles were case-control studies except for gender influence (longitudinal and cross-sectional population studies) and syndromes, which are so rare that the only information available is case reports.

RESULTS

Genetics

Genetic factors definitely controls tooth emergence as studies with monozygotic twins shows a concordance rate of 0.9 [8]. Dizygotic twins and siblings show a lower concordance rate but it is still higher than in unrelated individuals. Some authors stated that heritability is higher for tooth development then for tooth eruption [9,10].

Both longitudinal and cross-sectional studies reported differences in teeth emergence time among different races [11-16]. Permanent teeth emerge considerably earlier in African and American-African children than in Asians and Caucasians [12].

There are certain genetic disorders that affect teeth eruption. Most of them are reported to delay permanent teeth eruption, others are associated with complete failure teeth to erupt. Genetic disorders

Table 1. Genetic disorders influencing permanent teeth eruption

Amelogenesis imperfecta [43]	Gorlin syndrome [61]
Aarskog syndrome [44]	Hallermann-Streiff syndrome [62]
Acrodysostosis [6]	Hyperimmunoglobulinemia (Buckley syndrome) [53]
Albright Hereditary Osteodystrophy [6]	I-cell disease (mucolipidosis II) [63]
Apert syndrome [45]	Incontinentia pigmenti (Bloch-Sulzberger syndrome) [64]
Carpenter syndrome [46]	Mc-Cune-Albright syndrome (polyostotic fibrous dysplasia) [48]
Cherubism [47]	Menkes' kinky hair syndrome [65]
Chondroectodermal dysplasia [48]	Neurofibromatoses [66]
Cleidocranial dysplasia [1]	Oculoauriculo vertebral spectrum (Goldenhar syndrome/hemifacial microsomia) [67]
Cockayne syndrome [6]	Osteoglophonic dyspalsia [68]
Congenital hypertrichosis lanuginosa [49]	Osteopathia striata with cranial stenosis [68]
Dentin dysplasia [50]	Osteopetrosis (marble bone disease) Osteogenesis imperfecta [69]
Mucopolysaccharidosis (MPS) [48]	Otodental dysplasia [70]
Down syndrome [51]	Parry-Romberg syndrome (progressive hemifacial atrophy) [71]
Dyskeratosis congenita [52]	Progeria (Hutchinson-Gilford syndrome) [68]
Ectodermal dysplasia [53]	Rothmund-Thompson syndrome [68]
Ekman-Westborg-Julin syndrome [54]	Sclerosteosis [72]
Epidermolysis bullosa [55]	Shokier syndrome (hereditary anodontia spuria) [60]
GAPO syndrome (growth retardation, alopecia, pseudoanodontia, and optic atrophy) [56]	SHORT syndrome [68]
Gardner syndrome [57]	Singleton-Merten syndrome [73]
Gaucher disease [58]	VonRecklinghausen neurofibromatosis [74]
Gingival fibromatosis associated syndromes [59]	22q11 deletion syndrome [75]
Laband syndrome [60]	
Murray-Puretic-Drescher syndrome [60]	
Rutherford syndrome [60]	
Cross syndrome [60]	

can be divided into disorders that affect enamel formation and/or the tooth follicle (e.g. amelogenesis imperfecta, Hurler's syndrome, mucopolysaccharidosis VI) and disorders that interfere with osteoclastic activity (e.g. Cleidocranial dysplasia, osteopetrosis). Other syndromes are associated with deficient growth or teeth eruption can be delayed by multiple supernumerary teeth or gingival hyperplasia [7]. Genetic syndromes that affect teeth eruption are listed in Table 1.

Gender

There is an agreement from studies on teeth emergence that in girls permanent teeth erupt earlier than in boys [13,17-20]. Significant differences has been found for maxillary lateral incisors and canines [15,18,19] and mandibular canine [15,17-19]. The difference between eruption times on average is from 4 to 6 months, largest difference being for permanent canines. Earlier eruption of permanent teeth in females is attributed to earlier onset of maturation. Only one study reported earlier emergence of second molars in boys than in girls and explained this phenomenon as a catch-up development by the age of eruption of second molars because of later onset of puberty in the males [15]. Eruption sequence, especially during the second eruption phase, differs among genders: the classical orders of eruption appeared more frequently in males (20% upper, 17% lower) as compared to females (12% upper, 8% lower) [15,17,20,21]. In girls the maxillary canine can be expected before the second premolar, and the mandibular second premolar can be expected before second molar; in boys both orders are reversed [21].

Nutrition

Although data on nutrition influence on permanent teeth emergence is scarce, there is evidence that chronic malnutrition extending beyond the early childhood is correlated with delayed teeth eruption [22]. Although one study reported accelerated eruption of permanent first molars and incisors in 6 year old group of children with early childhood protein- energy malnutrition, but their sample was small and they failed to report nutritional status at the time of examination [23].

Preterm birth

According to World Health Organization (WHO) preterm birth is defined as birth occuring before 37 weeks of gestation or if the birth weight is below 2500 g. The incidence of preterm birth (PT) varies between populations and is reported to be from 5 to 13% in developed countries [24-27].

The weight of an infant at birth is usually accepted as the best index of prematurity.

Influence of preterm birth on teeth development and eruption has been investigated [28-30]. Most of the studies reported oral findings during primary dentition stage, while data on permanent dentition development, especially on permanent teeth eruption, is rare. Most of the studies reported that PT children have delayed primary and permanent teeth eruption, if emergence time was compared to chronological age. However some researches showed that if eruption time was related to corrected age (i.e. chronological age in weeks minus gestational age 40 weeks), no difference has been found between dental maturation and eruption times of PT and control children [28-30]. Some researches reported that the greatest delay was found in children younger than 6 years of age, whereas for those aged 9 years or older, there was no difference, indicating that a ''catch-up'' had occurred [29]. Results of another study showed that maturation of permanent dentition evaluated according Demirjan methodology did not differ among PT born children and controls [28]. One study even reported earlier eruption of permanent first molar and incisors in PT black and white children when compared to controls, although group of white children was small [31]. The authors suggested that various post-natal factors and an accelerated

Table 2. Systemic diseases associated with impaired dental eruption

Vitamin D-resistant rickets [48]	Renal failure [79]
Endocrine disorders [76]	Exposure to hypobaria [80]
Hypothyroidism (cretinism)	Idiopathic [1]
Hypopituitarism	Radiation damage
Hypoparathyroidism	Celiac disease [81]
Pseudohypoparathyroidism	Anemia [79]
Drugs: phenytoin [77]	Dysosteosclerosis [82]
Long-term chemotherapy [78]	Cerebral palsy [1]
Ichthyosis [53]	HIV infection [83]
Oral clefts [1]	Heavy metal intoxication [84]

growth period (catch-up growth) with related unknown factors may influence the eruption of teeth that goes through a sensitive period circumnatally (permanent incisors and first molars).

When evaluating factors affecting maturation and eruption of permanent dentition, one study concluded that neither low birth weight, nor gestational age nor pubertal stage had significant influence on dentition [28]. Associations has been found between maturity of primary dentition and permanent dentition in that a small number of primary teeth at 1 year of chronological age is associated with less mature permanent teeth at 9–11 years of age in children born preterm. Also positive correlation has been reported between PT children body height and permanent teeth maturation stage (the taller preterm child was at the time of evaluation, the Demirjan SDS was more advanced). The correlation between body height and permanent tooth emergence has been noted in another study in that stunted children at 6 months of age were more likely to have non-emerged first upper left and lower right permanent molars at 6 years of age.

Socioeconomic factors

In a number of studies it has been found that children from higher socioeconomic backgrounds show earlier tooth emergence than children from lower socioeconomic classes [32], while others did not supported this theory [33].

It is thought that children from higher socioeconomic class get better health care, nutrition and these factors influence earlier development of dentition.

Some researches found that permanent teeth eruption sequence is different among children from different socioeconomic classes. The first teeth to erupt in child's from higher backgrounds oral cavity is mandibular incisor as opposite to mandibular first molar in children from lower class [16,33].

Body height and weight

A positive correlation between body height and weight and teeth emergence has been established in the earlier studies [8,34]. The taller and heavier children are slightly advanced dentally while it is apparent that stunting (retarded linear growth) is more strongly associated with delayed tooth eruption.

Research on children obesity and dental development also showed a positive correlation: obese children mature earlier and teeth tend to erupt on average 1.2 to 1.5 year earlier as compared to children with normal body mass index [35].

Craniofacial morphology

Although tooth eruption has a poor correlation with general body and facial growth, it was speculated that permanent second molar eruption might be different in subjects with skeletal Class II and Class I malocclusion [36]. The study sample was rather small and they actually look at the eruptive position on panoramic radiograms but they concluded that in part study results suggests that the maxillary second molars may erupt earlier in patients with skeletal maxillary Class II malocclusions.

Several studies showed that formation and eruption of the maxillary teeth, especially molars, are delayed in skeletal Class III patients [37]. One study found the only teeth to be influenced by skeletal maxillary morphology (in particular maxillary retrusion) are maxillary second molars [38]. Authors concluded that variables influencing delayed eruption were palatal length and chronological age.

Several studies tried to investigate the influence of facial type on dental development. The first conducted study in this field concluded that skeletal open bite is associated with advanced dental maturity if compared to skeletal deep bite. The difference between dental ages was about 6 months [39]. However, the sample included in study was rather small and the two extreme groups were overlapping each other, which might have obscured results. Another study with a larger sample group failed to find any significant difference in permanent teeth development between the short face and the long face skeletal types [40].

Hormonal factors

Disturbance of the endocrine glands usually has a profound effect on the entire body, including the dentition. Hypothyroidism, hypopituitarism, hypoparathyroidism, and pseudohypoparathyroidism are the most common endocrine disorders associated with delayed permanent teeth eruption [41,42].

Accelerated dental development has been noted in association with increased adrenal androgen secretion, whereas the effect of an excess of growth hormone and thyroid hormones on dental development is less clear [8].

Systemic diseases

Most of the diseases reported in literature are associated with delayed tooth eruption, and only diabetes accelerates tooth eruption [1,7]. There are different mechanisms for the delay, usually it is associated with retained primary teeth, gingival hyperplasia, fibromatosis or hormonal changes which influences bone resorption rate. Diseases that interfere with tooth eruption are listed in Table 2.

CONCLUSION

Permanent teeth eruption is a complex process that can be influenced by a number of general factors: genetics, nutrition, preterm birth, socioeconomic factors, body height and weight, craniofacial morphology, hormonal factors and various systemic diseases.

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Received: 13 10 2009
Accepted for publishing: 23 09 1010