

# Dental Anomalies: Foundational Articles and Consensus Recommendations, 2021

**Adekoya-Sofowora CA.** Natal and neonatal teeth: a review. *Niger Postgrad Med J* 2008;15:38-41

Al-Ani AH, Antoun JS, Thomson WM, Merriman TR, Farella M. Hypodontia: An Update on Its Etiology, Classification, and Clinical Management. *Biomed Res Int.* 2017;9378325. doi.org/10.1155/2017/9378325.

Anthonappa RP, King NM, Rabie AB. Aetiology of supernumerary teeth: A literature review. *Eur Arch Paediatr Dent.* 2013;14:279-88.

**Dashash, M. Yeung CA, Jamous I, Blinkhorn A.** Interventions for the restorative care of amelogenesis imperfecta in children and adolescents. *Cochrane Database Syst Rev* 2013;6:CD007157.

**Gallacher A, Ali R, Bhakta S.** Dens invaginatus: diagnosis and management strategies. *Br Dent J* 2016;221:383-7.

**Gill DS, Barker CS.** The multidisciplinary management of hypodontia: a team approach. *Br Dent J* 2015;218:143-9.

**Khalaf K, Miskelly J, Voge E, Macfarlane TV.** Prevalence of hypodontia and associated factors: a systematic review and meta-analysis. *J Orthod.* 2014; 41:299-316.

**Lammi L, Arte S, Somer M, Javinen H, et al.** Mutations in AXIN2 cause familial tooth agenesis and predispose to colorectal cancer. *Am. J. Hum. Genet.* 2004, 74:1043–1050.

**Marvin ML, Mazzoni S, Herron CM, Edwards S, et al.** AXIN2-associated autosomal dominant ectodermal dysplasia and neoplastic syndrome. *Am J Med Genet A.* 2011, 155 898–902.

**Seow WK.** Developmental defects of enamel and dentine: Challenges for basic science research and clinical management. *Aust Dent J* 2014;59:143-54.

**Shields ED, Bixler D, El-Kafrawy AM.** A proposed classification for heritable human dentine defects with a description of a new entity. *Arch Oral Biol* 1973;18:543-53.

**Smail-Faugeron V, Picou Rollin J, Muller Bolla M, Courson F.** Management of non-syndromic dens evaginatus affecting permanent maxillary central incisors: a systematic review. *BMJ Case Rep* 2016. doi: 10.1136/bcr-2016-216672.

**Smith CEL, Poulter J, Antanaviciute A, Kirkham J, et al.** Amelogenesis imperfecta; Genes, Proteins, and Pathways. *Front Physiol* 2017. doi.org/10.3389/fphys.2017.00435.

**Witkop CJ Jr.** Amelogenesis imperfecta, dentinogenesis imperfecta and dentin dysplasia revisited: Problems in classification. *J Oral Pathol* 1988;17:547-53.

**Witkop CJ Jr.** Hereditary defects of dentin. *Dent Clin North Am* 1975;19:25-45.

# IAPD Consensus Recommendations

## AMELOGENESIS IMPERFECTA

Amelogenesis imperfecta (AI) refers to a group of genetic disorders affecting dental enamel which may be inherited as autosomal dominant (AD), autosomal recessive (AR), or X-linked patterns. Diagnosis of AI is commonly based on phenotype, medical and family histories. Clinical features associated with AI may include: sensitivity, poor oral hygiene, calculus, post eruptive breakdown, aesthetic concerns, reduction of vertical dimension, difficulties eating, increased caries risk, reduced bond strength, delayed eruption or failure of eruption, pre-eruptive coronal resorption and anterior open bite. Management of AI may include the following:

1. Prevention following national and international guidelines for individuals with high caries risk.
2. Desensitizing agents to reduce the sensitivity of exposed dentin.
3. Tooth bleaching, microabrasion, infiltrative resin, opaque composite resin or indirect composite veneers, and ceramic veneers or crowns.
4. There is some evidence supporting the use of sodium hypochlorite prior to etching and bonding to achieve better bonding to composite/compomer restorations.
5. Early interventions, composite restorations, veneers, and gold or ceramic crowns to prevent loss of vertical dimension.

## DENTINOGENESIS IMPERFECTA

Dentinogenesis imperfecta (DI) is a group of autosomal dominant conditions characterised by defective formation of dentine, affecting the primary and permanent dentitions. Prevalence is thought to be approximately 1 in 6,000-8,000, in a population in the USA, while the prevalence reported in European studies ranged from 0.002% to 0.057%. Shield's classic classification defined this group of disorders into: Type 1: DI associated with osteogenesis imperfecta (OI); Type 2: DI not associated with OI; Type 3: rare DI with thin coronal and radicular dentine and severe attrition leading to pulpal abscess. Multidisciplinary management is often required, including the following:

1. Prevention following national and international guidelines for individuals with high caries risk.
2. Early intervention with composite restorations, gold or ceramic crowns, and overdentures to prevent loss of vertical dimension.
3. Aesthetics treatment including whitening and composite or ceramic veneers.

## DENTIN DYSPLASIA

Dentin dysplasia is an autosomal-dominant trait, affecting both the primary and secondary dentitions. Shields et al., proposed the classification: type I, or "radicular dentin dysplasia," and type II or "coronal dentin dysplasia." Both primary and permanent dentitions are affected. The affected teeth are characterized by short or total absence of roots, obliterated pulp chambers, and peri-apical radiolucencies. Type I teeth exhibit extreme mobility and are commonly exfoliated prematurely. Radiographically, primary teeth show total pulp obliteration and permanent teeth show thistle-tube pulp configuration and pulp stones in pulp chambers. Type II dentin dysplasia should be considered as a milder form of dentinogenesis imperfecta (DI type 2). Management may include:

1. Endodontic treatment is contraindicated in teeth with total obliteration of root canals and pulp chambers. Extraction has been suggested as an alternative treatment.
2. Oral rehabilitation with removable prosthetics after extractions may be necessary. Due to early exfoliation and consequent bone resorption, treatment with a combination of bone grafting and a sinus lift may be required for implant placement.

## HYPODONTIA

Hypodontia is defined by the absence of one or more teeth, with exception of the third permanent molars. Prevalence varies between 1.5% -1.8% in the primary dentition and 6.4% in the permanent dentition. Associated features may include conical teeth, microdontia, delayed eruption, ectopic eruption, ankylosis and submerged primary teeth, tooth wear of primary teeth and reduced alveolar development. Associated issues often include

aesthetics, compromised chewing function, and negative psychosocial impact. Management of hypodontia may include:

1. Aggressive prevention following national and international guidelines for individuals with high caries risk.
2. Composite restorations, ceramic or metallic crowns and veneers to manage conical or microdont teeth and tooth wear.
3. Management of submerged or ankylosed primary molars may be required.
4. Orthodontic treatment is often required to optimize space. Orthognathic surgery may be necessary in certain cases to improve occlusion.
5. Removable prosthetics, resin retained bridges, over dentures, tooth autotransplantation and/or dental implants are often necessary for the replacement of missing teeth.

### **SUPERNUMERARY TEETH**

Supernumerary teeth are teeth or tooth-like structures in addition to the normal number of primary and permanent teeth. Prevalence varies between 0.3-0.8% in the primary dentition and 0.1-3.8% in the permanent dentition, with a 2:1 male to female ratio. Associated features may include: delayed or failed eruption of permanent teeth, crowding, rotation or ectopic position of permanent teeth, root malformations, cyst formation of unerupted supernumerary teeth. Management may include:

1. Monitoring with annual radiographic exam, if no associated complications and if orthodontic treatment is not planned.
2. Removal of supernumerary tooth (teeth) with or without surgical exposure and orthodontic intervention of the unerupted permanent tooth (between 49% and 91% of supernumerary teeth erupt spontaneously in the presence of adequate space).

### **DENS EVAGINATUS**

Dens evaginatus are tooth cusp-like formations that contain enamel, dentine and occasionally pulp. They are most common in permanent incisors and premolars and have a prevalence, between 0.06 to

7.7%, with marked worldwide variation. Management may include:

1. Monitoring and placement of fissure sealant, if there is no associated complications.
2. Selective progressive reduction of the dens evaginatus.
3. Excision of dens evaginatus if complications are present. Clinicians must monitor the tooth (teeth) for the likelihood of pulp exposure and manage this complication.

### **DENS INVAGINATUS**

Dens invaginatus is defined by growing of the enamel, dentine and pulp complex into the pulpal space. Prevalence varies between 0.3 and 10%, with population variations. The upper lateral incisor is the most commonly affected tooth. Management may include:

1. Placement of fissure sealants and monitoring if tooth is vital.
2. Caries removal and adequate restoration if carious.
3. Root canal treatment if tooth becomes necrotic.

### **NATAL & NEONATAL TEETH**

Natal teeth are present at birth and neonatal teeth erupt within the first 30 days of life. The majority of natal and neonatal teeth represent the early eruption of normal primary teeth with less than 10% being supernumerary. Natal teeth are often smaller, conical and yellowish, and have hypoplastic enamel and dentin with poor or absent root formation. Complications include discomfort during suckling causing irritation and trauma to infants' tongue, sublingual ulceration, laceration of the mother's nipples and aspiration of the teeth. Management may include:

1. If possible, a radiographic exam to differentiate the premature eruption of a primary tooth from a supernumerary tooth.
2. Extraction if the tooth is supernumerary, excessively mobile, or interfering with breastfeeding.
3. Covering the incisal portion of the tooth with composite resin or smoothing the incisal edge may be an option if it interferes with breastfeeding.