Secondary retention of molars

Gerry M. Raghoebar

SECONDARY RETENTION OF MOLARS

STELLINGEN

behorend bij het proefschrift

SECONDARY RETENTION OF MOLARS

Groningen, 26 juni 1991

GERRY M. RAGHOEBAR

1 Vroege herkenning van een eruptiestoornis van eerste en tweede molaren is van groot belang voor de preventie van ernstige malocclusie.

Dit proefschrift

2 Ankylose is kenmerkend, doch niet pathognomonisch voor secundaire retentie van een gebitselement.

Dit proefschrift

3 Percussie van secundair geretineerde molaren is geen betrouwbare methode om ankylose vast te stellen.

Dit proefschrift

4 Het is een misvatting te denken dat secundaire retentie van gebitselementen slechts voorkomt bij molaren.

5 Onverdraagzaamheid ten opzichte van een andere cultuur is terug te voeren op onvoldoende kennis van de eigen aardigheden daarvan.

6 Kinderen worden altijd, direct of indirect, het zwaarst getroffen tijdens door volwassenen gecreëerde oorlogen.

7 Een stap terug doen om vooruit te komen getuigt van zowel moed als inzicht.

8 Erosieve lichen planus van het mondslijmvlies is een schrijnend probleem.

9 Het beoefenen van de orale implantologie in zijn totaliteit vergt meer kennis en kunde dan de tandarts in zijn basispakket heeft.

10 Ontkenning van de noodzaak van samenwerking tussen kaakchirurg en tandarts in de orale implantologie vormt een bedreiging voor de ontwikkeling van dit vakgebied.

11 Als de natuurlijke wortel met evenveel zorg werd omgeven als de kunstwortel zou deze laatste totaal overbodig zijn.

12 Tijdens arthroscopie van het kaakgewricht gaat er een wereld voor je open.

13 Sinds voetbalvandalisme noopt tot het nemen van maatregelen, is 'vele goeden moeten het met de kwaden ontgelden' zeker van toepassing op voetbalsupporters.

Cover: H. Flanderijn
Typesetting: STYX Publications

RIJKSUNIVERSITEIT GRONINGEN

SECONDARY RETENTION OF MOLARS

PROEFSCHRIFT

ter verkrijging van het doctoraat in de Geneeskunde
aan de Rijksuniversiteit Groningen
op gezag van de Rector Magnificus Dr. L. J. Engels
in het openbaar te verdedigen op woensdag 26 juni 1991
des namiddags te 2.45 uur precies
door

GERRY MAX RAGHOEBAR

geboren op 29 november 1959
te Paramaribo

DRUKKERIJ VAN DENDEREN B.V.
Groningen
1991

Promotores	Prof. Dr. G. Boering Prof. C. Booy
Referenten	Dr. A. Vissink Dr. H. W. B. Jansen

Promotiecommissie	Prof. Dr. G. J. Kusen Prof. Dr. A. K. Panders Prof. Dr. I. van der Waal

Paranimfen	Mr. M. J. Bakker Drs. B. Stegenga

Aan mijn ouders
Voor Helga

ISBN 90–9004103–6

Contribution to the printing costs of this thesis was made by:
J. Sanders Enz-fonds, The Netherlands

CONTENTS

VOORWOORD

Bij het tot stand komen van dit proefschrift heb ik van velen medewerking en steun ondervonden, waarvoor mijn hartelijke dank. Zonder anderen tekort te willen doen, wil ik een aantal personen met name bedanken.

Prof. Dr. G. Boering, hooggeachte promotor, beste professor. U heeft reeds gedurende mijn opleiding tot kaakchirurg mijn interesse gewekt voor het verrichten van wetenschappelijk onderzoek. Dit heeft geleid tot dit proefschrift. Voor uw voortdurende belangstelling, gecombineerd met de voor U zo kenmerkende prettige begeleiding, heb ik veel respect gekregen.

Prof. C. Booy, hooggeachte promotor, beste professor. Uw kundige beoordeling, ervaring en enthousiasme hebben dit manuscript zeer verrijkt. Ik ervaar het dan ook als een voorrecht dat U bereid was om als promotor op te treden.

Dr. A. Vissink, zeer geachte referent, beste Arjan. Zonder jouw tomeloze inzet en nauwgezetheid was mijn onderzoek waarschijnlijk nog niet voltooid. Onze samenwerking was uiterst plezierig en je was altijd bereid, zelfs 's avonds, om met mij achter de computer plaats te nemen.

Dr. H.W.B. Jansen, zeer geachte referent, beste Henk. Ik ben je zeer erkentelijk voor het beschikbaar stellen van je expertise bij het histologische deel van dit onderzoek en voor het gebruikmaken van de faciliteiten van het laboratorium.

De leden van de promotiecommissie dank ik voor de accurate en vlotte wijze waarop zij zich van hun taak hebben gekweten.

Drs. B. Stegenga, beste Boudewijn. Tijdens het schrijven van dit proefschrift stond je altijd klaar met raad en daad. Jouw nuchtere kijk op de problematiek en stimulerende wetenschappelijke interesse voor dit onderzoek hebben mij tijdig voor de nodige dwalingen behoed.

Prof. Dr. L.P. ten Kate, beste professor. Hartelijk dank dat u mij wegwijs hebt willen maken in uw vakgebied en mij hebt willen ondersteunen bij het onderzoek naar de mogelijke rol van genetische factoren bij secundaire retentie van blijvende molaren.

Drs. F.A. van Koldam, beste Winnie. Bedankt voor het screenen van vele orthodontische patiënten op het vóórkomen van secundaire retentie.

Drs. G. Abma, beste Gerrit. Jouw scriptie over hetzelfde onderwerp heeft in belangrijke mate bijgedragen aan de inventarisatie van de relevante literatuur en daarmee aan het tot stand komen van dit proefschrift.

Dr. W.L. Jongebloed en Dhr. D. Kalicharan, beste Wim en Ruby. Dank voor het vervaardigen van de vele scanning-electronenmicroscopische opnamen. Ze zijn niet alleen een lust voor het oog maar van grote diagnostische waarde.

Mw. N. Brouwer en Mw. C.A.A.M Nooren, beste Nieske en Corina. Door de hoge kwaliteit van de histologische preparaten hebben jullie een belangrijke wetenschappelijke bijdrage aan dit onderzoek geleverd. De foto's van deze preparaten hebben bovendien mijn proefschrift aanzienlijk verfraaid.

Drs. C.J.F. Schoots, beste Coen. Vele uren heb je vrijgemaakt om met mij de dentale pathologie van secundaire retentie door te spreken. Hiervoor mijn hartelijke dank.

Drs. C.A.M. Hazenberg. Hartelijk dank voor het vaststellen van de bloedgroepen van een aantal patiënten met secundaire retentie.

Dr. V. Fidler. Mijn dank voor uw adviezen met betrekking tot het statistisch verwerken en interpreteren van de gegevens.

Dr. S. Gooda en Mw. M.A. de Jong, beste Steve en Mirabelle, respectievelijk bedankt voor de correctie van het Engels, en van de Nederlandse samenvatting.

De vele fraaie foto's zijn vervaardigd door de medewerkers van de afdeling fotografie van de Faculteit Tandheelkunde en de vakgroep Histologie en Celbiologie. Met name wil ik Dick Huizinga bedanken voor de vele uren die hij met mij in de donkere kamer heeft doorgebracht.

De heren H. Flanderijn en E.G.C. van Ommen, beste Henk en Erik. Dank voor het op professionele wijze vervaardigen van de illustraties.

Drs. Th.J. Brouwer, beste Theo. Met jou heb ik altijd met veel plezier samengewerkt, met name in het kader van de preprothetische chirurgie en implantologie. Van jou heb ik nooit een wanklank vernomen wanneer ik in verband met het onderzoek niet aanwezig kon zijn op de polikliniek. Hiervoor mijn dank.

Drs. G. Steensma, beste Gauke. Bedankt voor het kritisch doorlezen van de zetproeven. Zonder jouw inzet zouden vele zet- en typfouten niet zijn ontdekt.

Mw. K. Wolthuis, Mw. G. Boezerooy en Mw. E. van Drooge, beste Karin, Gerda en Ellen. Bedankt voor jullie administratieve en persoonlijke ondersteuning.

Alle medewerkers van de afdeling Mondziekten, Kaakchirurgie en Bijzondere Tandheelkunde, die hebben bijgedragen aan een aangename werksfeer en mij de directe of indirecte steun hebben gegeven waardoor dit proefschrift tot stand kon komen, dank ik hartelijk.

Alle patiënten die hun medewerking verleenden aan dit onderzoek en alle tandartsen, orthodontisten en kaakchirurgen die hun patiënten aan onze zorg toevertrouwden, mijn hartelijke dank.

Mijn ouders, jullie dank ik voor de warme belangstelling, de steun en de aanmoediging die jullie mij altijd hebben gegeven. Jullie hebben mij geleerd door te zetten. Daarvan heb ik veel profijt gehad gedurende dit onderzoek en het schrijven van dit proefschrift. Jullie zijn voor mij van onschatbare waarde.

Helga, jou wil ik heel hartelijk danken voor de wijze waarop je mij gedurende dit onderzoek hebt gesteund en gestimuleerd. Jij hebt mij altijd de mogelijkheid geboden om 's avonds en in het weekeinde tijd voor het uitwerken van de manuscripten te vinden.

CHAPTER 1

INTRODUCTION AND AIM OF THE INVESTIGATION

INTRODUCTION

Eruption refers to the axial or occlusal movement of the tooth from its developmental position within the jaw to its functional position in the occlusal plane.[1] Eruption continues throughout life to compensate for occlusal wear and growth of the jaws. Eruption is important for the maxillofacial development during the growth of the jaws because of the increase of the intermaxillary distance especially in the posterior region. During the growth and development of the face, compensatory changes in the path of eruption occur, leading to positional changes between the jaws. If such compensation is insufficient, or if it does not occur at all, malocclusion and positional anomalies due to lack of space may result.[2]

The development and eruption of individual permanent teeth are delayed occasionally. Third molars and maxillary canines are most frequently involved, while eruption disturbances of first and second molars are relatively rare.[3,4] Although the incidence of eruption disorders of first and second molars is low, they have a considerable impact when they do occur. First and second molars are of great importance for the normal development of the dentition and coordination of the facial growth. Their function is to provide sufficient occlusal support for undisturbed mastication. A disturbed eruption of molars may cause a number of complications such as tilting of neighbouring teeth, over-eruption of antagonists, loss of arch length, and cessation of alveolar bone growth.[4-6] The affected molars themselves can undergo resorption, may become involved in pericoronitis, and can cause formation of follicular cysts and odontogenic tumors.[4,6] In some cases of cysts or tumors it is difficult to determine whether or not these conditions have caused the eruption disturbance.

In molars, three types of eruption disturbances can be distinguished, namely *impaction, primary retention and secondary retention*. Impaction refers to cessation of the eruption of a molar due to a physical barrier in the eruption path or an abnormal position.[6,7] In the case of retention cessation of eruption of a molar occurs without a physical barrier in the eruption path nor as a result of an abnormal position. Primary retention occurs before emergence, while secondary retention occurs after emergence.[8] Thus secondary retention is cessation of eruption of a tooth after emergence neither due to a physical barrier in the eruption path nor as a result of an abnormal position. The latter eruption disturbance is also often described in the literature as submerging or ankylosis.[5,9] This thesis deals with secondary retention, because of its great clinical significance. If secondary retention is not treated at the right time, it may give rise to severe malocclusion, loss of the affected tooth and

neighbouring teeth due to caries or periodontal disease, and cessation of alveolar bone growth in the affected area.[5] Removal of secondarily retained permanent molars is often difficult, and can in severe cases even result in a fracture of the jaw.[10]

AIM OF THE INVESTIGATION

The information available in the literature about the clinical signs and symptoms of secondary retention in permanent molars is predominantly limited to case reports,[3,9-25] while some extended studies are available about secondary retention in the deciduous dentition.[26-33] A major difference between the clinical significance of secondary retention in the deciduous and permanent dentition is that the complications observed by secondarily retained deciduous molars were usually temporarily and had resolved in most cases spontaneously after eruption of the permanent successors.[26] Secondary retention of a permanent molar is rarely reversible. The extent of infraocclusion and the relating complications in general increase with time.

The criteria used for diagnosing secondary retention in permanent molars are confusing and often not reliable. The etiology of secondary retention is essentially unknown and there are no proper guidelines for the treatment of this eruption disorder. Furthermore, it has been suggested in the literature that ankylosis may play a role in the etiopathogenesis of secondary retention. Familial occurrence suggests a hereditary contribution.

The aim of this study was to obtain insight in the present knowledge of secondary retention and other eruption disturbances of molars, to state factors that may contribute to the etiopathogenesis, to state reliable diagnostic criteria, and to recommend guidelines for proper treatment. Third molars were excluded from this study as differentiation between primary retention, secondary retention and impaction is mostly not reliable. This is largely due to the fact that, contrary to first and second molars there is no proper time frame for third molars as far as eruption is concerned. The former goals were achieved by:

- a review of the literature regarding secondary retention (chapter 2);
- a study of the clinical, radiographic, and histological characteristics and recommendations for proper treatment of secondary retention in the deciduous dentition (chapter 3);
- a description of the eruption disturbances underlying impaction, primary retention and secondary retention in the permanent dentition (chapter 4);
- a study of the clinical and radiographic characteristics of secondary retention of permanent molars (chapter 5);
- a study of the hereditary factors of secondary retention of permanent molars (chapter 6);
- a study of the histological characteristics of secondary retention of permanent molars (chapter 7);

- an evaluation of the outcomes of different approaches of treatment and a proposal for guidelines for proper treatment of secondary retention of permanent molars (chapter 8).

REFERENCES

1 Ten Cate AR. Oral histology. Development, structure and function. 3rd ed. St Louis: CV Mosby, 1989:275–98.
2 Björk A, Skieller V. Facial development and tooth eruption: an implant study at the age of puberty. Am J Orthod 1972;62:339–83.
3 Farman AG, Eloff J, Nortjé CJ, Joubert JJV. Clinical absence of the first and second permanent molars. Br J Orthod 1978;5:93–7.
4 Pindborg JJ. Pathology of the dental hard tissues. Copenhagen: Munksgaard, 1970:225–55.
5 Biederman W. The problem of the ankylosed tooth. Dent Clin North Am 1968;12:409–24.
6 Shafer WG, Hine MK, Levy BM. A textbook of oral pathology. 4th ed. Philadelphia: WB Saunders, 1983:66–9.
7 Regezi JA, Sciubba JJ. Oral pathology. Clinical-pathologic correlations. Philadelphia: WB Saunders, 1989:469–71.
8 Dixon DA. Submerged teeth. Br Dent J 1954;97:325–7.
9 Sterrett DS. Report of a case of a submerging first molar. Am J Orthod Oral Surg 1940;26:681–3.
10 Adams D. An impacted permanent first molar. Oral Surg Oral Med Oral Pathol 1975;40:702–3.
11 Brenchley ML. Submerged first permanent molars. Dent Pract 1960;11:105–8
12 Dayan D, Littner MM, Gonshorowitz J, Mersel A. Reimpaction of a first permanent maxillary molar due to an obscure idiopathic etiology. Clin Prevent Dent 1983;5:22–4.
13 Dixon DA. Impactions of the first permanent molar. Br Dent J 1959;106:281–3.
14 Faulkner JR. Arrested eruption of first and second permanent molars with consequent impaction of second molar. Br Dent J 1939;67:450.
15 Hall GM, Reade PC. Quadrilateral submersion of permanent teeth. Brief review and case report. Aus Dent J 1981;26:73–6.
16 Israel H. Early hypercementosis and arrested dental eruption: heritable multiple ankylodontia. J Craniofac Genet Dev Biol 1984;4:243–6.
17 Jalili VP, Bobra K. Submerged molar teeth. J Indian Dent Assoc 1986;58:209, 236.
18 Kaban LB, Needleman HL, Hertzberg J. Idiopathic failure of eruption of permanent molar teeth. Oral Surg Oral Med Oral Pathol 1976;42:155–63.
19 Kapoor AK, Srivastava AB, Singh BP. Bilateral posterior open-bite. Oral Surg Oral Med Oral Pathol 1981;51:21–2.
20 Oliver RG, Richmond S, Hunter B. Submerged permanent molars: four case reports. Br Dent J 1986;160:128–30.
21 Sian JS. An impacted mandibular first permanent molar: report of case. J Dent Child 1982;49:39–40.
22 Skolnick IM. Ankylosis of maxillary permanent first molar. J Am Dent Assoc 1980;100:558–60.
23 Stoy PJ. Submerged permanent molars. The Dental Record 1951;71:12–4.
24 Tratman EK. Impacted first mandibular permanent molar. Br Dent J 1938;67:196–7.
25 Binder KH. Einstell- und Durchbruchsstörungen von Sechsjahrmolaren. Zahnärztl Welt 1959;60:736–9.
26 Kurol J. Infraocclusion of primary molars: an epidemiological, familial, longitudinal, clinical and histological study. Dissertation. Dept of Orthodontics, University of Göteborg, 1984.
27 Brearley LJ, McKibben DH. Ankylosis of primary molar teeth. I. Prevalence and characteristics. II. A longitudinal study. J Dent Child 1973;40:54–63.
28 Darling AI, Levers BGH. Submerged human deciduous molars and ankylosis. Arch Oral Biol 1973;18:1021–40.
29 Dixon DA. Observations on submerging deciduous molars. The first Chapman prize essay. Dent Prac 1963;13:303–15.
30 Henderson HZ. Ankylosis of primary molars: a clinical, radiographic, and histologic study. J Dent Child 1979;46:117–22.
31 Kula K, Tatum BM, Owen D, Smith RJ, Rule J. An occlusal and cephalometric study of children with ankylosis of primary molars. J Pedod 1984;8:146–59.

32 Rune B. Submerged deciduous molars. Odontol Revy 1971;22:257–73.
33 Rygh P, Reitan K. Changes in the supporting tissues of submerged deciduous molars with and without permanent successors. Odont T 1964;72:345–62.

CHAPTER 2

SECONDARY RETENTION OF MOLARS: REVIEW OF THE LITERATURE

INTRODUCTION

Eruption refers to the axial or occlusal movement of the tooth from its developmental position within the jaw to its functional position in the occlusal plane.[1] Once in occlusion, eruption continues throughout life to compensate for occlusal wear of teeth and for growth of the jaws.

Eruption disturbances of deciduous and permanent teeth are rather common. They may occur during any of the six eruptive stages. These stages include follicular growth, pre-emergent eruptive spurt, postemergent eruptive spurt, juvenile occlusal equilibrium, circumpubertal occlusal eruptive spurt, and adult occlusal equilibrium.[2] The disturbances may originate from systemic or local factors. Systemic factors are present in patients with certain developmental syndromes,[3,4] e.g. cleidocranial dysostosis, and as a consequence usually multiple teeth are affected (Fig. 1). By contrast, in patients with a local eruption disturbance at most a few teeth are affected, for example impacted lower third molars and permanent maxillary canines (Fig. 2). Eruption disturbances of first and second deciduous and permanent molars occur less frequently.[5,6] Occasionally deciduous or permanent molars are observed where the occlusal surface is below the occlusal plane of the neighbouring teeth. This cessation of the eruption is neither due to a physical barrier in the eruption path nor the result of an abnormal position. Cessation of eruption of these teeth may start even after occlusal contact with the antagonists has been established initially. This unusual eruption disorder is called *secondary retention* in this thesis. Although secondary retention may involve any molar and can be seen at any age, it is most frequently diagnosed in the mixed dentition.[7]

In the literature, secondary retention is also described as ankylosis,[8] arrested eruption,[9] depression,[10] Halbretention,[11] impaction,[12] infraocclusion,[13,14] infraposition,[15] nonocclusion,[16] reimpaction,[5] reinclusion,[17] retention,[18] and submerging.[19] Ankylosis and submerging are the terms most commonly used, while impaction is normally used to describe cessation of eruption of a tooth due to a physical barrier in the path of eruption or an abnormal position.[4] The term submerging in our opinion has to be considered a misnomer as it has the connotation of active depression.[8,20-22] The affected tooth is, however, not subjected to any depressive movement and does not slowly sink into the alveolar process, but remains static while the other teeth continue their occlusal movement. The term ankylosis reflects the most common explanation for the inability of teeth to maintain their position in the occlusal plane, particularly in a growing child, and is generally not used specifically to refer to this disorder. The term secondary retention gives a

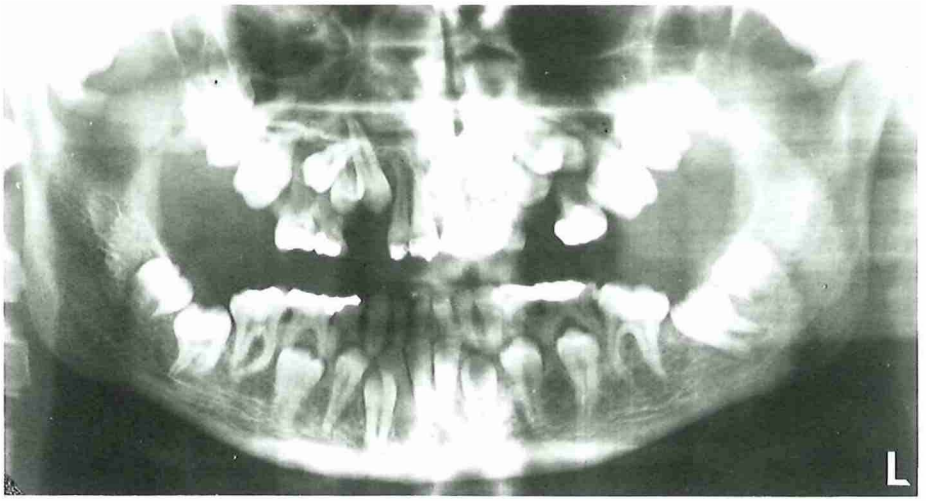


Fig. 1 Orthopantomogram of a 16-year-old man with cleidocranial dysostosis. Multiple unerupted teeth are present in the mandible and maxilla.

Fig. 2 Orthopantomogram of a 26-year-old woman showing impaction of both upper canines and of the lower left third molar.

good description of the clinical picture without consideration of the etiology. The term 'secondary' is added to indicate that the retention occurs secondarily to the postemergent eruptive spurt or during a later phase of the eruptive process. Until the phase in which secondary retention starts the eruptive process appears to be normal.

Secondary retention of deciduous molars has been extensively described in the literature.[24-31] A similar phenomenon in the permanent dentition is predominantly

reported in case reports.[6,9,12,19,32-45] Secondary retention is not recognized or not properly diagnosed by many dentists, orthodontists and oral and maxillofacial surgeons.[21,46] It may, however, cause serious malocclusion of the dentition if not recognized and treated on time. Apparently, there is a lack of knowledge concerning this eruption disorder, especially in the permanent dentition.

Secondary retention of deciduous and permanent molars have much in common, but the clinical significance of secondary retention in permanent molars is usually more severe. In this review the etiology, clinical features, histological findings, and treatment of secondary retention of both deciduous and permanent molars are summarized.

ETIOLOGY OF SECONDARY RETENTION

The biological mechanism of secondary retention is not fully understood. We assume that the etiopathogenesis of secondary retention of deciduous and permanent molars is largely the same. Most authors presume that secondary retention is somehow a result of ankylosis.[20-22,25,27] Others think that ankylosis is secondary to, for example, a deficient eruptive force, thus assuming that secondary retention is a failure of the normal eruptive process that can result in ankylosis.[26] However, if ankylosis is secondary to ceased eruption, ankylosis should be common in impacted or unerupted teeth too. Because this is not the case, the latter theory is less probable.[20-22,25,29]

Ankylosis may occur if the periodontal ligament fails to separate cementum and dentin from bone.[47] This may be due to a congenital defect in the periodontal ligament, or due to a local disturbance of metabolic or traumatic nature.[8,20-22,40,48] When this occurs, cementum and dentin may be resorbed and replaced by bone, resulting in fusion of the root with alveolar bone.[49] Ankylosis can also be a result of a disturbance in the interaction between normal root resorption and hard tissue repair.[26,27,50] If bone and not cementum is deposited in the resorption lacunes during the repair process, the result is ankylosis. In deciduous and permanent teeth, superficial root resorption and repair by cementum is a normal physiologic process.[51,52] Ankylosis can also originate from hypercementosis[38,53] and pulpo-periodontal canals.[30] Hypercementosis may lead to delayed tooth eruption, arrested tooth eruption, and ankylosis in permanent molars. This has not been reported for deciduous molars. Pulpo-periodontal canals exist in deciduous and permanent molars.[54-58] Rune assumed that superficial root resorption can start at these canals and may lead to ankylosis.[30]

Schulze suggested that secondary retention is the combined result of ankylosis and pressure from the adjacent teeth.[10] However, pressure from a distal tooth is not thought to be an important factor as the affected molar is erupted before emergence of the distal tooth. Moreover in any case, the secondarily retained teeth were in normal occlusion before they became affected.[25,30]

Adamson[59] and Graber[60] reported that excessive traumatic masticatory forces can cause secondary retention, assuming that the periodontal ligament is not capable to sustain the occlusal forces in adolescents. However, ankylosis can not be induced experimentally in animals by occlusal trauma or by direct mechanical and chemical injury to the root.[61,62] In addition, secondary retention is less frequently seen in permanent teeth, even though masticatory forces are greater in adults. Also, the fact that antagonists are normally not affected refutes this theory.[24,37,48] It was suggested that a combination of local trauma and local metabolic disturbances may be on the basis of secondary retention.[63] Alexander et al. found that the enzyme activity which is partly responsible for the deciduous root resorption was not detectable in the periodontal ligament of ankylosed deciduous teeth.[63]

Dixon[26] suggested tongue pressure as a causative factor. This pressure is believed to cause a disturbance of the blood supply of the periodontium and thus might induce metabolic disturbances.[64,65] Tongue-interposition is, however, often considered as the result of malocclusion.[66]

A local disturbance of vertical bone growth of the alveolar process has been suggested as a possible cause of secondary retention.[45,67] This is not very probable as removal of a deciduous secondarily retained molar was followed by local vertical bone growth and eruption of the succeeding premolar.[30] If the successor was agenetic, the edentulous space closed spontaneously as a result of mesial drift of the distal molar and sufficient bone was formed to enable this. Otherwise it could be closed orthodontically. The deficient vertical bone growth around the affected molar is probably the result of secondary retention and not the cause.[20-22]

Infection has been suggested as a possible cause of secondary retention,[30,32,41,59] but in the majority of the cases, secondary retention is observed in teeth lacking a clinical history of dental disease.[24]

An association between secondary retention and absence of the successor has been reported,[68] but this result could not be confirmed in another study.[69]

Several authors suggest a hereditary component in secondary retention of deciduous[13,23,70] and permanent molars.[14,17,38,53,71-75] In a linkage study of 5 pedigrees with secondary retention of permanent molars, bloodgroup P_1 was suggested to be linked to the secondary retention gene,[17] but in a second report by the same research group the lod scores were too low to corroborate the findings.[71] From the latter study it was concluded that secondary retention of permanent molars is inherited as an autosomal dominant disorder with vertical transmission and no skipping of generations. The exact mode of inheritance is not known.

CLINICAL CHARACTERISTICS OF SECONDARY RETENTION

Secondary retention is observed in the deciduous dentition from three years of age[13,24,30,76] and in the permanent dentition from nine years of age.[16] The disorder is not sex linked,[17,23,24,47,71] although in the deciduous dentition it is more frequently observed in girls than in boys.[24,77] The deciduous to permanent molars ratio

Table 1 Clinical impact of secondarily retained permanent molars

Secondarily retained molar:
– surgical removal is often necessary
– increased difficulty of removal in time
– bone growth and alveolar development is inhibited in the affected area
– high risk of caries, periodontal disease, and inflammation
Neighbouring teeth:
– increased susceptibility to caries and periodontal disease
– loss due to caries or periodontal disease
– tilting of the teeth into the diastema
Occlusion:
– development of malocclusion
– over-eruption of the antagonist
– loss of arch length
– abnormal tongue function
– impaired masticatory funtion
Miscellaneous:
– increased risk for oroantral communication on removal
– increased risk for damage to the inferior alveolar nerve on removal
– increased risk for pain due to infection and sinusitis
– food impaction in the diastema

for secondary retention is 10 to 1.[48] Secondary retention may be observed in deciduous and permanent molars within the same patient,[33] it is uncommon in multiple quadrants, and can lead to a bilateral posterior open bite.[30,37,41] Secondary retention of deciduous molars is not related to any particular type of malocclusion.[31,78] The reported prevalence of secondary retention in deciduous molars varies from 1,3% to 24,8%,[13,23,24,26,47,76-78] thus it seems that secondary retention is a relatively common disorder of tooth eruption in the deciduous dentition.

The clinical impact of secondarily retained permanent molars is summarized in Table 1. The clinical significance of secondarily retained deciduous molars is identical, but less severe. Additionally, exfoliation and eruption of the successor may be delayed or disturbed.[15,79,80]

The major clinical symptom of a secondarily retained tooth is infraocclusion. A proper criterion to consider a deciduous tooth in infraocclusion is the observation that the occlusal surface of the affected tooth is 1 mm or more cervical to the occlusal plane of the fully erupted neighbouring teeth for at least one year.[13,23,81] In case of permanent molars, the second or third molar is often not (fully) erupted at the time that secondary retention of the first or second molar is diagnosed, therefore definition of secondary retention of permanent molars has to be changed. In this thesis both deciduous and permanent molars were considered to be secondarily retained if the occlusal surface was at least 1 mm below the occlusal level of the other teeth in the relevant dental arch at an age at which the tooth normally would have been in occlusion. In case of severe infraocclusion the affected molar is barely visible in the oral cavity due to covering of the crown by surrounding

structures,[8,20-22,82,83] but there is always an epithelial tube connecting the pericoronal space of the affected molar and the oral cavity. Absence of such a tube proves that cessation of eruption has occurred before emergence and, as a consequence, the affected molar is not secondarily retained. Attrition marks, caries and restorations are typical for secondary retention and may facilitate differentiation from primary retention.[5,34,84,85]

An altered percussion sound has been suggested as a diagnostic aid.[7,49,68,86,87] Ankylotic teeth have a solid, clear sound on percussion, while normal teeth produce a dull percussion sound.[4,24,28,49,86,88] The accuracy of the percussion test has not yet been assessed.[24] The size of the ankylotic area may be a factor in determining the type of percussion sound.[89]

Partial absence of the periodontal ligament space is used to diagnose ankylosis radiographically and has also been suggested as an diagnostic aid in secondary retention.[4,87] Again, small ankylotic areas are not easy to detect radiographically and thus may lead to false negative results.[15,24,27,44] The diagnostic value of this radiographic criterion has yet to be assessed as well.

HISTOLOGICAL CHARACTERISTICS OF SECONDARY RETENTION

It is generally accepted that secondary retention is associated with ankylosis. Several histological studies of extracted secondarily retained deciduous molars revealed a presence of ankylosis in 80 up to 100% of the cases.[25-27,30,31,49,88,90] A possible explanation that ankylosis was not observed in all molars might be that fractures of roots are common in removal of these teeth, especially in the deciduous dentition. The fragment of the root that was left behind in the jaw may show signs of ankylosis.[88] Furthermore, the ankylotic areas are often small and thus may be easily missed during routine examination.[15] Therefore, it is important to make serial sections of each molar to detect ankylosis.[26,30,88] In most cases, ankylosis was observed at the bifurcation and the interradicular root surface.[25,26,30,31,49,88]

In secondarily retained permanent molars ankylosis was only reported to be present in two case studies.[11,91]

TREATMENT OF SECONDARY RETENTION

Treatment of secondary retention is directed toward prevention of malocclusion, caries and periodontal disease.[46] In the *deciduous* dentition, extraction is generally accepted to be the treatment of choice,[4,7,8,30,31,49,78] occasionally followed by space maintaining to preserve the diastema for the eruption of the premolar.[27,47,79,86,92] Removal of deciduous molars is often difficult[25,90,93] and it has therefore been argued that early extraction is necessary because the extent of infraocclusion is progressive and the molars thus become more difficult to remove with time.[47,78] Some authors recommend a non-extraction therapy to let the affected molar serve as

a space maintainer.[15,49,76,79,80,94,95] They indicate extraction in case of severe infraocclusion, severe occlusion disturbances and dislocation of the successor. If secondary retention starts at a later age, progression of infraocclusion is usually slow and root resorption is slight. In such cases the occlusal surface of the affected molar can be build up. The main factors to consider when planning therapy are the severity of infraocclusion, the age of the child related to possible future occlusal disturbances, and the presence or absence of the premolar. In summary, a secondarily retained deciduous molar is either left untreated, restored, or removed. Although delayed exfoliation will occur, normal eruption of the successor is often observed in cases that are left untreated.[80,96] The affected molar may be build up to restore occlusal contacts. The objectives are to restore function, and to prevent tilting of neighbouring teeth and over-eruption of antagonists. Gold crowns, stainless steel crowns, cast crowns and direct bonding have been used for build ups.[79,97]

In the *permanent dentition* extraction of the affected molar is usually recommended. In cases with only a slight infraocclusion a build up of the molar is the proper treatment.[20-22] Occasionally an alveolar segment osteotomy has been performed to move the affected tooth into occlusion.[65,98] Such a small segment osteotomy may easily result in a tooth which is 1 to 2 mm out of occlusion because of retraction of the segment during healing. It is generally accepted that secondarily retained teeth cannot be moved by orthodontic forces.[42,65,87,98] Luxation of the affected molars was sometimes successful.[20-22,95] This luxation may be followed by application of orthodontic force in an occlusal direction by means of elastics or springs.

CONCLUSION

Secondary retention of deciduous and permanent molars seems to be an eruption disturbance which can lead to major clinical problems such as malocclusion and loss of the neighbouring teeth. In addition to infraocclusion, percussion tests and radiographs have been used as clinical tools to diagnose secondary retention. The reliability of the latter methods are unknown. It is, however, important to have reliable diagnostic tools in addition to infraocclusion, particularly in doubtful cases, as the treatment of secondary retention differs from that of other eruption disorders. Furthermore, the etiopathogenesis of secondary retention is unknown. Particulary the contribution of ankylosis to secondary retention of permanent molars has not yet been fully investigated. Finally, there is no general agreement on the treatment of secondary retention, especially in the permanent dentition.

REFERENCES

1 Ten Cate AR. Oral histology. Development, structure and function. 3rd ed. St Louis: CV Mosby, 1989:275–98.

2 Steedle JR, Proffit WR. The pattern and control of eruptive tooth movements. Am J Orthod 1985;87:56–66.

3 Gorlin RJ, Pindborg JJ, Cohen MM. Syndromes of the head and neck. 2nd ed. New York: Mc Graw-Hill, 1976:180–4.
4 Shafer WG, Hine MK, Levy BM. A textbook of oral pathology. 4th ed. Philadelphia: WB Saunders, 1983:65–9.
5 Pindborg JJ. Pathology of the dental hard tissues. Copenhagen: Munksgaard, 1970:225–55.
6 Farman AG, Eloff J, Nortjé CJ, Joubert JJV. Clinical absence of the first and second permanent molars. Br J Orthod 1978;5:93–7.
7 Herman E. Evaluation and management of ankylosed teeth. NY State Dent J 1964;30:327–33.
8 Biederman W. Tooth ankylosis. Ann Dent 1953;12:1–15.
9 Faulkner JR. Arrested eruption of first and second permanent molars with consequent impaction of second molar. Br Dent J 1939;67:450.
10 Schulze Ch. Über Retention und Reinklusion(Depression) erster und zweiter Molaren. Dtsch Zahn Mund Kieferheilkd 1962;37:338–76.
11 Korkhaus G. Die Halbretention des ersten Molaren. Dtsch Zahnärztl Z 1952;7:1205–12.
12 Tratman EK. Impacted first mandibular permanent molar. Br Dent J 1938;67:196–7.
13 Kurol J. Infraocclusion of primary molars: an epidemiologic and familial study. Community Dent Oral Epidemiol 1981;9:94–102.
14 Theuerkauf J. Die Infraokklusion der Milchmolaren und Sechsjahrmolaren. Dtsch Zahnärztl Z 1960;15:1358–68.
15 Kloeppel JG. Die Infraposition einzelner Zähne des Milch- und bleibenden Gebisses. Dtsch Zahn Mund Kieferheilkd 1959;30:308–20.
16 Kessler W, Zur Frage der Häufigkeit von Nonokklusion von Milchmolaren und bleibenden Molaren. Stoma 1964;17:178–83.
17 Bosker H, Nijenhuis LE. Possible linkage between a gene causing reinclusion of a molar I and blood group P. Cytogenet Cell Genet 1975;14:255–6.
18 Brabant J, Brabant H. La rétention partielle des molaires. Schweiz Mschr Zahnhk 1950;994–1007.
19 Sterrett DS. Report of a case of a submerging first molar. Am J Orthod Oral Surg 1940;26:681–3.
20 Biederman W. The ankylosed tooth. Dent Clin North Am 1964;8:493–508.
21 Biederman W. The problem of the ankylosed tooth. Dent Clin North Am 1968;12:409–24.
22 Biederman W. Etiology and treatment of tooth ankylosis. Am J Orthod 1962;48:670–84.
23 Via WF. Submerged deciduous molars: familial tendencies. J Am Dent Assoc 1964;69:127–9.
24 Brearley LJ, McKibben DH. Ankylosis of primary molar teeth. I. Prevalence and characteristics. II. A longitudinal study. J Dent Child 1973;40:54–63.
25 Darling AI, Levers BGH. Submerged human deciduous molars and ankylosis. Arch Oral Biol 1973;18:1021–40.
26 Dixon DA. Observations on submerged deciduous molars. The first Chapman prize essay. Dent Pract 1963;13:303–15.
27 Henderson HZ. Ankylosis of primary molars: a clinical, radiographic, and histologic study. J Dent Child 1979;46:117–22.
28 Kula K, Tatum BM, Owen D, Smith RJ, Rule J. An occlusal and cephalometric study of children with ankylosis of primary molars. J Pedod 1984;8:146–59.
29 Kurol J. Infraocclusion of primary molars: an epidemiological, familial, longitudinal, clinical and histological study. Dissertation. Dept of Orthodontics, University of Göteborg 1984.
30 Rune B. Submerged deciduous molars. Odontol Revy 1971:22:257–73.
31 Rygh P, Reitan K. Changes in the supporting tissues of submerged deciduous molars with and without permanent successors. Odont T 1964;72:345–62.
32 Adams D. An impacted permanent first molar. Oral Surg Oral Med Oral Pathol 1975;40:702–3.
33 Brenchley ML. Submerged first permanent molars. Dent Pract 1960;11:105–8.
34 Dayan D, Littner MM, Gonshorowitz J, Mersel A. Reimpaction of a first permanent maxillary molar due to an obscure idiopathic etiology. Clin Prevent Dent 1983;5:22–4.
35 Dixon DA. Impactions of the first permanent molar. Br Dent J 1959;106:281–3.
36 Binder KH. Einstell- und Durchbruchsstörungen von Sechjahrmolaren. Zahnärztl Welt 1959;60: 736–9.
37 Hall GM, Reade PC. Quadrilateral submersion of permanent teeth. Brief review and case report. Aus Dent J 1981;26:73–6.
38 Israel H. Early hypercementosis and arrested dental eruption: heritable multiple ankylodontia. J Craniofac Genet Dev Biol 1984;4:243–6.
39 Jalili VP, Bobra K. Submerged molar teeth. J Indian Dent Assoc 1986;58:209, 236.

40 Kaban LB, Needleman HL, Hertzberg J. Idiopathic failure of eruption of permanent molar teeth. Oral Surg Oral Med Oral Pathol 1976;42:155–63.
41 Kapoor AK, Srivastava AB, Singh BP. Bilateral posterior open-bite. Oral Surg Oral Med Oral Pathol 1981;51:21–2.
42 Oliver RG, Richmond S, Hunter B. Submerged permanent molars: four case reports. Br Dent J 1986;160:128–30.
43 Sian JS. An impacted mandibular first permanent molar: report of case. J Dent Child 1982;49: 39–40.
44 Skolnick IM. Ankylosis of maxillary permanent first molar. J Am Dent Assoc 1980;100:558–60.
45 Stoy PJ. Submerged permanent molars. The Dental Record 1951;71:12–4.
46 Albers DD. Ankylosis of teeth in the developing dentition. Quintessence Int 1986;17:303–8.
47 Krakowiak FJ. Ankylosed primary molars. J Dent Child 1978;45:288–92.
48 Biederman W. The incidence and etiology of tooth ankylosis. Am J Orthod 1956;42:921–6.
49 Noyes F. Submerging deciduous molars. Angle Orthod 1932;2:77–87.
50 Kronfeld R. Histopathology of the teeth and their surrounding structures. 4th ed. Philadelphia: Lea and Febiger, 1955:293–5.
51 Brown WAB. Resorption of permanent teeth. Br J Orthod 1982;9:212–20.
52 Henry JL, Weinmann JP. The pattern of resorption and repair of human cementum. J Am Dent Assoc 1951;42:270–90.
53 Humerfelt A, Reitan K. Effects of hypercementosis on the movability of teeth during orthodontic treatment. Angle Orthod 1966;36:179–89.
54 Moss SJ, Addelston H, Goldsmith ED. Histologic study of the pulpal floor of deciduous molars. J Am Dent Assoc 1965;70:372–9.
55 Woo RK, Miller J. Accessory canals in deciduous molars. J Int Assoc Dent Child 1981;12:51–7.
56 Vertucci FJ, Williams RG. Furcations canals in the human mandibular first molar. Oral Surg Oral Med Oral Pathol 1974;38:308–14.
57 Vertucci FJ, Anthony RL. A scanning electron microscopic investigation of accessory foramina in the furcation and pulp chamber floor of molar teeth. Oral Surg Oral Med Oral Pathol 1986;62:319–26.
58 Gutmann JL. Prevalence, location, and patency of accessory canals in the furcation region of permanent molars. J Periodontol 1978;49:21–6.
59 Adamson KT. The problem of impacted teeth in orthodontics. Aust J Dent 1952;56:74–84.
60 Graber TM. Orthodontics. Principles and practice. 3rd ed. Philadelphia: WB Saunders, 1972: 391–2.
61 Parker WS, Frisbe H E, Grant TS. The experimental production of dental ankylosis. Angle Orthod 1964;34:103–7.
62 Rubin PL, Weisman EJ, Bisk F. Experimental tooth ankylosis in the monkey. Angle Orthod 1984;54:67–72.
63 Alexander SA, Swerdloff M. Identification and localization of a mucopolysaccharidase in human deciduous teeth. J Dent Res 1980;59:594–601.
64 Pytlik W. Das abwegige Verhalten der Zunge beim Schlucken als Ursache der Ankylose und Zahndurchbruchsbehindering. Fortschr Kieferorthop 1982;43:380–93.
65 Proffit WR, Vig KWL. Primary failure of eruption: a possible cause of posterior open-bite. Am J Orthod 1981;80:173–90.
66 Hoffmeister H. Zungendruck oder vererbte Störanfälligkeit, Hauptursache der Infraposition und Inklusion von Milchmolaren. Fortschr Kieferorthop 1983;44:316–25.
67 Glucksman DD. Localized vertical growth disturbance. J Am Dent Assoc 1942;29:184–7.
68 Vorhies JM, Gregory GT, McDonald R E. Ankylosed deciduous molars. J Am Dent Assoc 1952;44: 68–72.
69 Steigman S, Koyoumdjisky-Kaye E, Matrai Y. Submerged deciduous molars and congenital absence of premolars. J Dent Res 1973;52:842.
70 Helpin ML, Duncan WK. Ankylosis in monozygotic twins. J Dent Child 1986;53:135–9.
71 Bosker H, Ten Kate LP, Nijenhuis LE. Familial reinclusion of permanent molars. Clin Genet 1978;13:314–20.
72 Gysel C. Un cas familial de rétention partielle de la deuxième molaire inférieure. Rev Belg Stomat 1955;52:103–11.
73 Reid DJ. Incomplete eruption of the first permanent molar in two generations of the same family. Br Dent J 1954;96:272–3.

74 Pelias MZ, Kinnebrew MC. Autosomal dominant transmission of ankylosed teeth, abnormalities of the jaws, and clinodactyly. Clin Genet 1985;27:496–500.
75 Brady J. Familial primary failure of eruption of permanent teeth. Br J Orthod 1990;17:109–13.
76 Steigman S, Koyoumdjisky-Kaye E, Matrai Y. Submerged deciduous molars in preschool children: an epidemiologic survey. J Dent Res 1973;52:322–6.
77 Koyoumdjisky-Kaye E, Steigman S. Submerging primary molars in Israeli rural children. Community Dent Oral Epidemiol 1982;10:204–8.
78 Lamb KA, Reed MW. Measurement of space loss resulting from tooth ankylosis. J Dent Child 1968;35:483–6.
79 Messer LB, Cline JT. Ankylosed primary molars: results and treatment recommendations from an eight-year longitudinal study. Pediatr Dent 1980;2:37–47.
80 Kurol J, Thilander B. Infraocclusion of primary molars and the effect on occlusal development, a longitudinal study. Eur J Orthod 1984;6:277–93.
81 Rule JT, Zacherl WA, Pfefferle AM. The relationship between ankylosed primary molars and multiple enamel defects. J Dent Child 1972;39:29–35.
82 Maréchaux SC. The problems of treatment of early ankylosis: report of case. J Dent Child 1986;53:63–6.
83 Adams TW, Mabee ME, Browman JR. Early onset of primary molar ankylosis: report of a case. J Dent Child 1981;48:447–9.
84 Hughes CL. Reimpaction of deciduous tooth: report of a case. J Am Dent Assoc 1972;85:912–3.
85 Pilo R, Littner MM, Marshak B, Aviv I. Severe infraclusion ankylosis: report of three cases. J Dent Child 1989;56:144–6.
86 Eichenbaum IW. Tooth eruption and ankylosis. J Prev Dent 1977;4:39–45.
87 Mitchell DL, West JD. Attempted orthodontic movement in the presence of suspected ankylosis. Am J Orthod 1975;68:404–11.
88 Kurol J, Magnusson BC. Infraocclusion of primary molars: a histologic study. Scand J Dent Res 1984;92:564–76.
89 Andersson L, Blomlöf L, Lindskog S, Feiglin B, Hammarström L. Tooth ankylosis. Clinical, radiographic and histological assessments. Int J Oral Surg 1984;13:423–31.
90 Thornton M, Zimmerman ER. Ankylosis of primary teeth. J Dent Child 1964;31:120–6.
91 Sims MR. Tooth ankylosis in man and the oxytalan fibre meshwork. Aust Orthod J 1980;6:147–53.
92 Alexander SA, Donne DD, Swerdloff M, Caputo L. Multiple ankylosed teeth. J Pedod 1980;4: 354–9.
93 Dixon DA. Submerged teeth. Br Dent J 1954;97:325–7.
94 Kurol J, Koch G. The effect of extraction of infraoccluded deciduous molars: a longitudinal study. Am J Orthod 1985;87:46–55.
95 Moyers RE. Handbook of orthodontics. 4th ed. Chicago: Yearbook medical publ, 1988:388–91.
96 Nazif MM, Zullo T, Paulette S. The effects of primary molar ankylosis on root resorption and the development of permanent successors. J Dent Child 1986;53:115–8.
97 Bonin M. Simplified and rapid treatment of ankylosed primary molars with an amalgam and composite resin. J Dent Child 1976;43:159–62.
98 Piret JL, Wackens G. Eruptiestoornissen van definitieve molaren. Acta Stomatol Belg 1988;85: 289–97.

CHAPTER 3

SECONDARY RETENTION OF DECIDUOUS MOLARS

Parts of this chapter have been published in Nederlands Tijdschrift voor Tandheelkunde 1988;95:389–92, Journal of Dentistry for Children 1991;58:17–22, and Oral Surgery Oral Medicine Oral Pathology (in press). Articles are reprinted with permission of the publisher.

ABSTRACT

Secondary retention refers to cessation of tooth eruption after emergence. The aim of this study was to evaluate the clinical, radiographic, and histological characteristics of secondary retention as well as the treatment results in a group of 34 patients with 77 secondarily retained deciduous molars. The most important clinical and radiographic criterion for diagnosing secondary retention was infraocclusion. Percussion turned out to be an unreliable diagnostic tool. Secondary retention in the deciduous dentition may predispose to a similar disorder in the permanent dentition. In many cases, occlusal disturbances and underdevelopment of the alveolar process disappeared spontaneously after eruption of the permanent successor unless a similar disorder was observed in the permanent dentition. Active therapy is necessary only in case of severe infraocclusion, severe malocclusion, dislocation or agenesis of the successor.

INTRODUCTION

Secondary retention refers to the cessation of eruption of a tooth after emergence neither due to a physical barrier in the path of eruption nor as a result of an abnormal position.[1] Other terms used in the literature are submerging, ankylosis and infraocclusion.[2-4] As the neighbouring teeth continue to erupt simultaneously with the growth of the alveolar bone, the affected tooth becomes secondarily in infraocclusion. The extent of infraocclusion is related to the rate of facial growth.[2] Secondary retention is observed in first and second molars. Deciduous molars are affected more frequently than permanent molars.[5]

Several hypotheses regarding the etiology have been proposed, including deficient eruptive force, disturbed metabolism of the periodontal ligament, squeezing, trauma, deficient local vertical bone growth, local inflammation, and disturbance in interaction between normal resorption and hard tissue repair.[2,5-9] An hereditary component has also been suggested, based on the observation of secondary retention in several members of the same family.[4,10,11] The exact biological mechanism of secondary retention is still unknown. It is often suggested that limited areas of ankylosis play an important role in the development of secondary retention.

Consequences of secondary retention of deciduous molars are delayed exfoliation, malocclusion, increased susceptibility to dental caries and periodontal disease of both the neighbouring teeth and retained molar, and dislocation of the successor.[5,12-15] Early recognition of secondary retention, proper treatment and a thorough follow up are important to prevent or minimize these consequences. In this study the clinical, radiographic, and histological characteristics of secondarily retained deciduous molars were investigated and the results of different treatment methods were evaluated.

MATERIAL AND METHODS

Material. A deciduous molar was considered to be secondarily retained if the occlusal surface was at least 1 mm below the occlusal level of the other teeth in the relevant dental arch at an age at which the tooth normally would have been in occlusion. Based on this definition, 15 female and 19 male patients (mean age: 13.9 ± 5.2 years) with 77 secondarily retained deciduous molars were selected. The patients all had been referred to the Department of Oral and Maxillofacial Surgery of the University Hospital Groningen by their dentist, orthodontist or oral and maxillofacial surgeon.

Clinical examination. The following variables were recorded:

a the number and location of the secondarily retained molars.
b the extent of infraocclusion: the distance (mm) between the occlusal surface of the retained molar and the occlusal plane of the relevant dental arch.
c the percussion sound of the retained molar: dull sound (normal tooth) or a solid, clear sound (ankylosis).
d the degree of tilting of the adjacent teeth: no tilting, moderate tilting (less than half of the surface of the retained molar covered) and severe tilting (more than half of the retained molar surface covered).

Radiographic examination. Orthopantomograms and intra-oral periapical radiographs were taken of all patients. The following variables were recorded:

a the relationship between the retained molar and its successor.
b the presence or absence of root resorption.
c the presence or absence of a periodontal ligament space.
d the vertical development of the alveolar process in the area of the secondarily retained tooth.

Treatment. Secondarily retained molars were treated in three different ways, namely:

a extraction of the retained molar (n=25).
b extraction of the retained molar in combination with orthodontic treatment to create or hold space for the successor (n=16).
c no treatment (n=36).

Histological examination. All removed molars (n=41) were examined by light microscopy. After removal, the molars were fixed in a 10% buffered formalin solution for two weeks, washed with tap water and demineralized in a solution of 25% formic acid and 10% sodium citrate in water. After embedding in paraffin, sections (7μm) were cut in a mesio-distal direction, parallel to the vertical axis of the molar and stained with haematoxylin and eosin. The examination was especially focused on the detection of ankylotic areas. Ankylosis was defined as direct contact of the alveolar bone with root cement or dentin.

Follow up. New cases of secondary retention in the same group were recorded. The effect of the different treatment methods on the eruption of the successor (if

Table 1 Distribution of secondarily retained deciduous molars

	m1	m2	n
Mandible	11	35	46
Maxilla	5	26	31
n	16	61	77

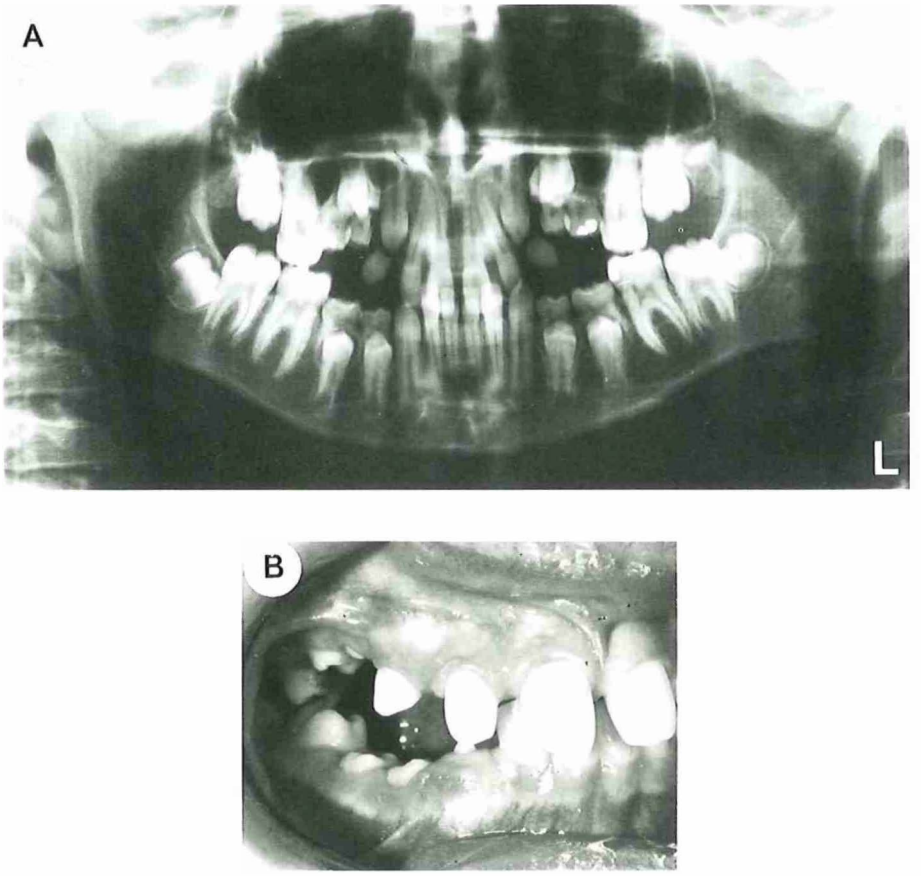


Fig. 1 A Orthopantomogram of a 10-year-old boy with secondary retention of all deciduous molars. **B** Clinical view of the right side.

present), on the height of the alveolar process, and on the occlusion were evaluated. The extent of infraocclusion was measured in cases in which no treatment was carried out.

Table 2 Distribution of secondarily retained permanent molars

	M1	M2	n
Mandible	5	–	5
Maxilla	5	1	6
n	10	1	11

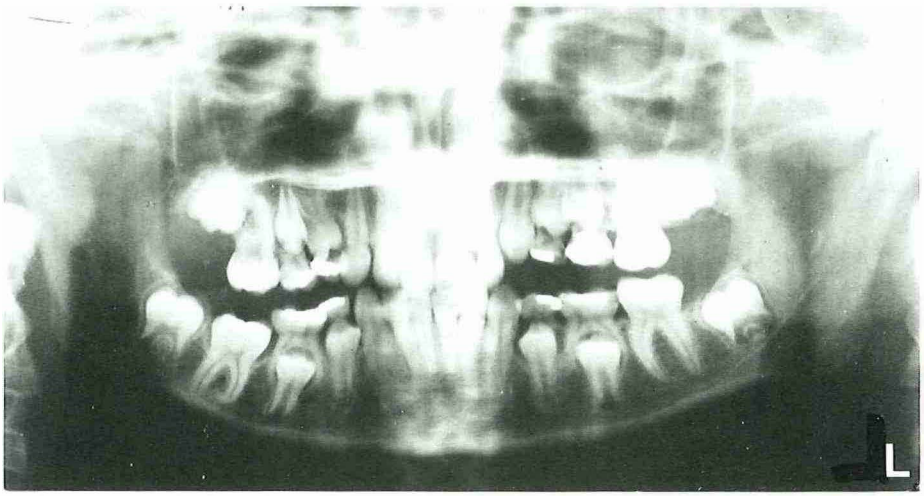


Fig. 2 Orthopantomogram of a 12-year-old girl showing secondary retention of the left first and second deciduous molars and right first permanent molar in the mandible.

RESULTS

Unless otherwise stated, the data given below all concern deciduous molars.

Clinical features. The distribution of the secondarily retained molars is given in Table 1. There is a definite preference for mandibular and maxillary second molars. In one patient all molars were retained (Fig. 1). Secondary retention of permanent molars (Table 2; Fig. 2) was observed in 9 patients (mean age: 13,5 years, range 11–17 years).

The mean infraocclusion was 4.1 ± 1.9 mm (median 4 mm, range 2 – 10 mm). In three cases the extent of infraocclusion was so severe that the retained molar was covered almost totally by the surrounding tissues (Fig. 3). In these cases, a narrow transepithelial connection between the occlusal surface of the retained tooth and oral cavity could be detected.

On percussion in 21 molars a solid, clear sound suggesting ankylosis was recorded, while in 48 molars a dull sound was heard. In 8 cases it was not possible to perform a reliable percussion test, because the retained molar was too obscured by

Table 3 Treatment results of secondarily retained deciduous molars in relation to eruption of the successor

	n	Normal eruption	Impaction	Agenetic
Extraction	25*	10	6	5
Extraction and orthodontic post-treatment	16**	14	–	–
No therapy	36***	30	1	2

* Four premolars were removed, because of abnormal position.
** Two premolars can erupt yet and have enough space.
*** Three premolars can erupt yet and have enough space.

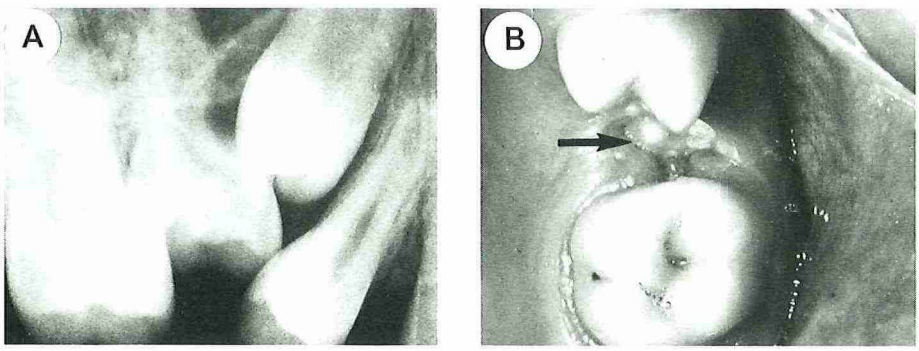


Fig. 3 A Intra-oral periapical radiograph of a 13-year-old girl with secondary retention of the maxillary left second deciduous molar. **B** Clinical view of the same patient, the retained molar being covered by the surrouding tissues.(arrow)

surrounding structures (severely tilted neighbouring teeth).

No tilting of the neighbouring teeth was observed in 48 secondarily retained molars, moderate tilting in 21 cases and severe tilting in 8 cases. In most cases, tilting was limited to the distal tooth and mainly observed, if infraocclusion was 4 mm or more and apparently insufficient space was available for the potential successor.

Radiographic features. The successors of 7 secondarily retained molars were agenetic. In all cases in which the premolar was present, a delay of root resorption of the retained molar in comparison with the normal deciduous molars was observed. Four successors had an abnormal position in the jaw.

Partial absence of the periodontal ligament space, suggestive for the presence of ankylosis, was seen in six cases. In five of these cases, a solid, clear percussion sound was recorded.

In all cases a decrease in vertical dimension of the alveolar bone was noted in the region of the retained molar.

Histological features. In 32 molars (78%), local areas of ankylosis were observed in the interradicular root area (Fig. 4). In two cases, ankylosis was also observed at

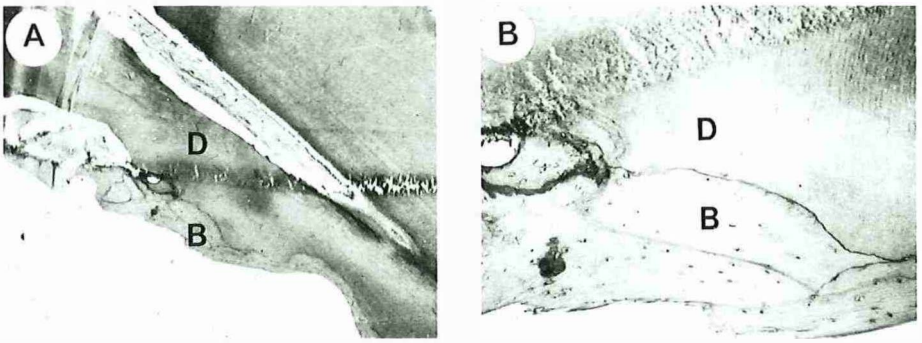


Fig. 4 A Histological section of a secondarily retained maxillary second deciduous molar. Bifurcation area. Dentin (D) in contact with bone (B). H-E, × 16. **B** Detail of bifurcation area. H-E, ×64.

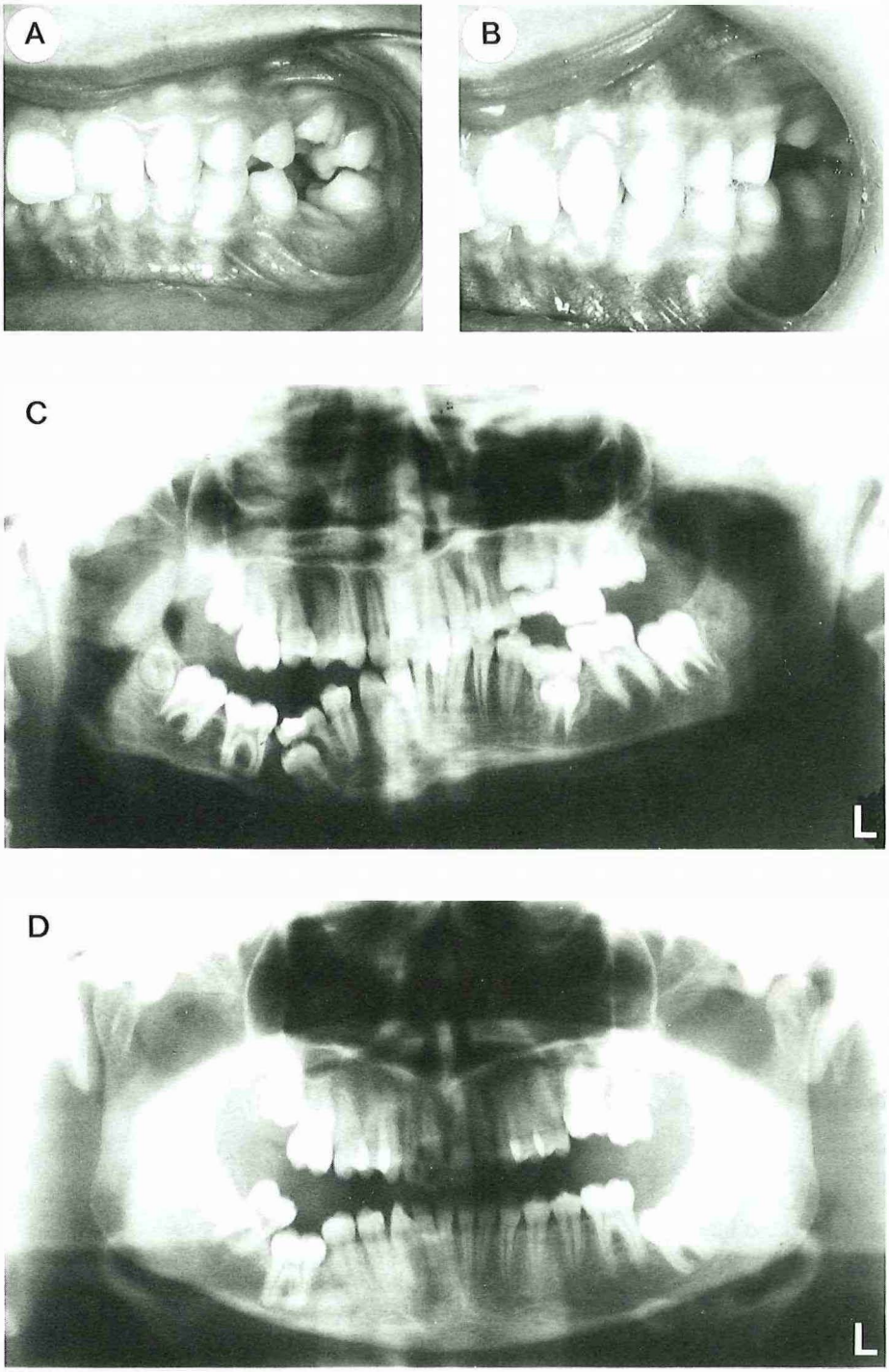
the outer root surface. From the 17 removed molars with a solid, clear percussion sound, 15 showed ankylosis histologically. The radiographic diagnosis of ankylosis (n=6) was supported by histological data.

Follow up. During the follow up period (6 months – 6 years), four new cases of secondarily retained molars were diagnosed, namely two deciduous and two permanent molars (Fig. 5). The treatment results are given in Table 3 and illustrated in Figures 6, 7, and 8. The extent of infraocclusion was progressive in most cases. All deciduous molars with successors that were left in place exfoliated, although with some delay. No re-eruption of the molars was observed. Eruption of a premolar after shedding of the retained molar was always associated with a recovery of the height of the alveolar process (Fig. 6). Impaction of a successor (n=7) was observed due to lack of sufficient space for this tooth to erupt. Five secondarily retained molars without a successor were removed (Fig. 7), because of the severe infraocclusion, tilting of the neighbouring teeth, or the extreme or progressive root resorption. The two remaining molars without a successor did not show any of these characteristics and were, therefore, left in place (Fig. 8). A prosthetic build up was made in both cases.

DISCUSSION

From the results of the present study, proper treatment of the disorder in the deciduous dentition and a careful follow up usually ensure a normal development of the permanent dentition. Furthermore, it appears that the occurrence of secondary retention in the deciduous dentition may predispose to a similar disorder in the permanent dentition.

Different nomenclature is used for the eruption disorder studied. We prefer the term 'secondary retention' because of its neutrality with respect to the still unknown etiology. Submerging is a misnomer, because the involved molar does not sink into the alveolar process, but remains static while the other teeth continue


A
B
C
L
D
L

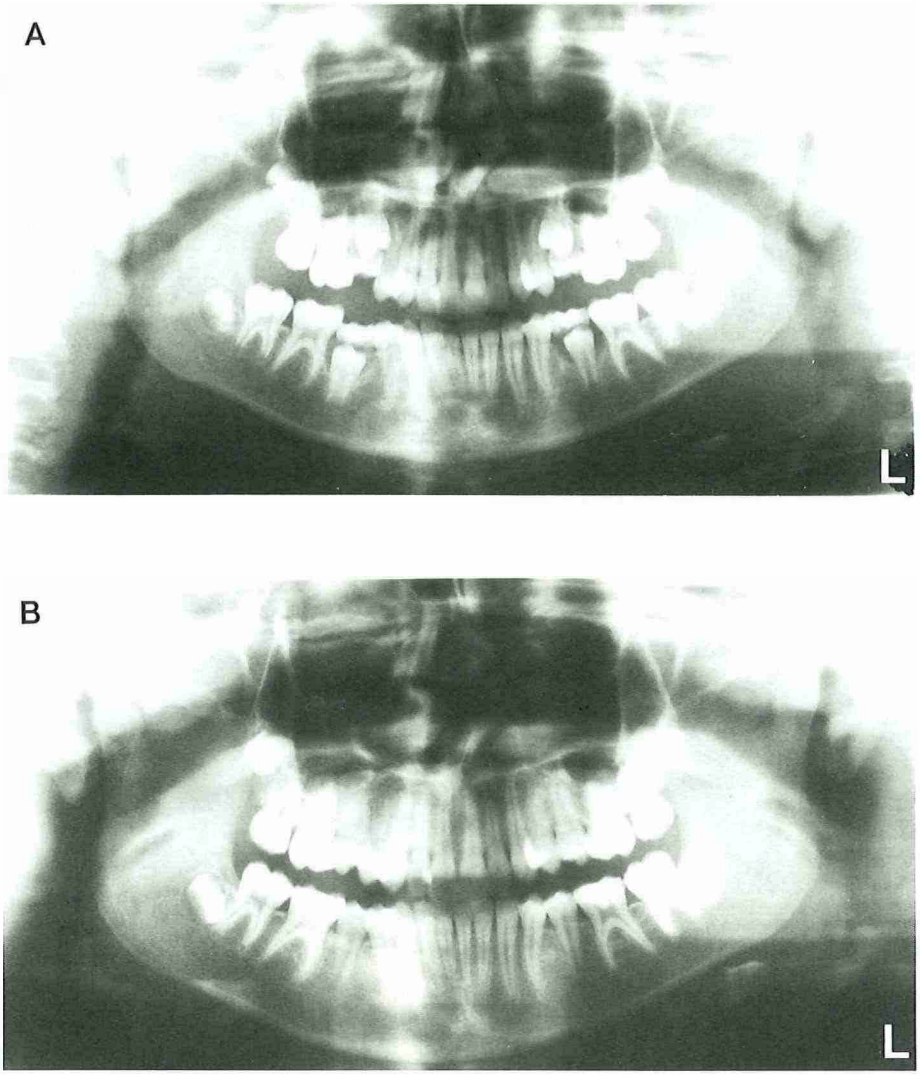


Fig. 6 A Orthopantomogram of a 12-year-old girl showing secondary retention of the maxillary second deciduous molars and mandibular right second deciduous molar. **B** Orthopantomogram of the same patient after 11 months without any active treatment. The premolars have erupted normally and the alveolar process had recovered its height.

Fig. 5 (page 22) **A** Clinical view of a 10-year-old girl showing secondary retention of the second deciduous molars in the mandible and maxilla on the left side. **B** Clinical view of the same patient after four years. The maxillary left first permanent molar showed secondary retention. **C** Orthopantomogram of the 10-year-old girl also showing secondary retention of the mandibular right second deciduous molar. The mandibular right first permanent molar is suspected of secondary retention. **D** Orthopantomogram of the same patient after four years. The mandibular right first permanent molar and maxillary left first permanent molar showed secondary retention.

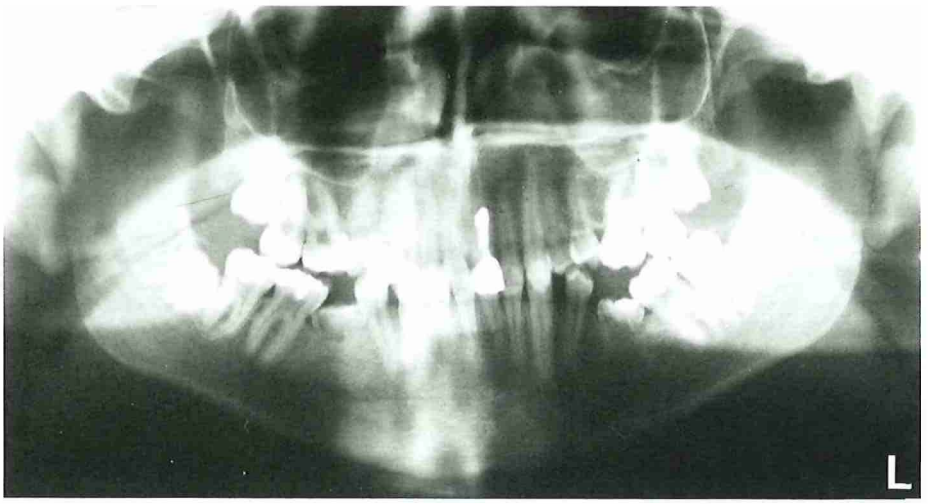


Fig. 7 Orthopantomogram of a 14-year-old girl showing secondary retention of both mandibular second deciduous molars without a successor. These molars were removed, because of tilting of the neighbouring teeth and root resorption of the retained molar.

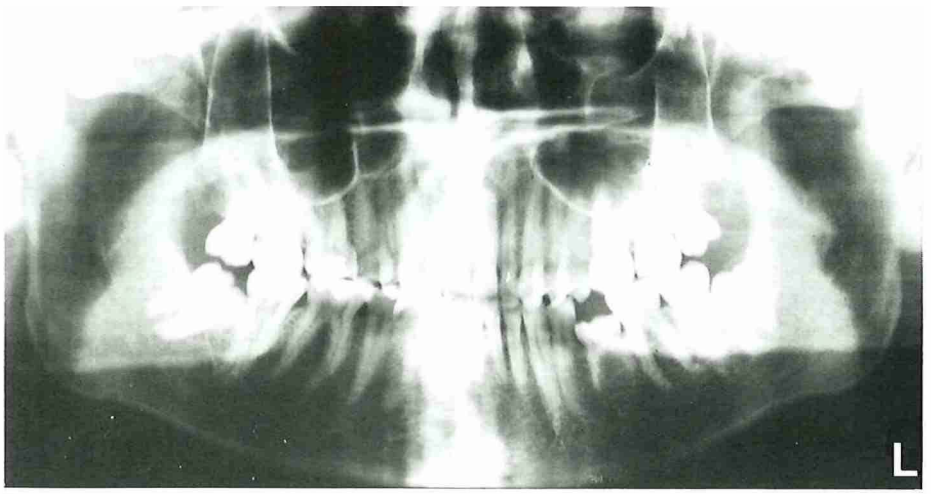


Fig. 8 Orthopantomogram of a 25-year-old man showing secondary retention of the mandibular left second deciduous molar without a successor. No treatment was performed.

to erupt.[5] Ankylosis only reflects one common exponent of the cessation of eruption. This is stressed by the fact that both clinically and histologically, ankylosis cannot be detected in all cases.[9,13] Infraocclusion is only a description of the clinical situation,[4] and is easily confused, therefore, with other abnormalities of the dentition causing infraocclusion.

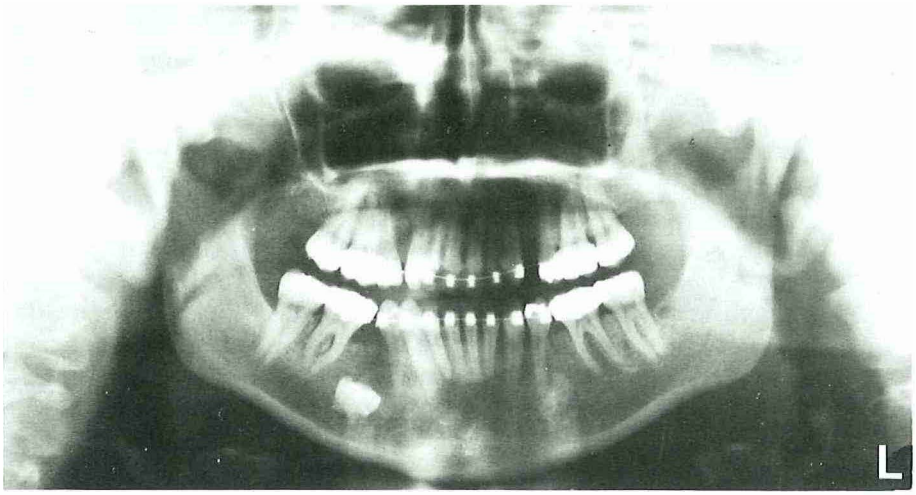


Fig. 9 Orthopantomogram of a 23-year-old woman showing an unerupted mandibular deciduous molar on the right side.

We call the disorder 'secondary' retention to distinguish it from 'primary' retention. In contrast to secondary retention, a primarily retained tooth has never shown tendency to erupt into the mouth. The tooth remains unerupted in the jaw beyond the time at which it should have been erupted. This eruption disturbance of deciduous molars is relatively uncommon (Fig. 9).[16,17] Secondary retention usually occurs in the deciduous molars in the mandible.[2,4,5,12,13] In this study, the mandibular second deciduous molar was most frequently affected, which is in accordance with the literature.[5,18-20] However, some authors reported that the mandibular first deciduous molar was the most frequently retained.[13,14] In a study, it was shown that after the age of 9-year the second mandibular deciduous molar was more frequently affected.[4] This molar may not exfoliate on time and still be evident in older children.[13]

The prevalence of secondary retention has been found to be age-related, with a maximum in 8- and 9-year old children.[4] Patients participating in our study were predominantly between 10 and 20 years of age. A possible explanation for this relatively old age-group might be that the oral and maxillofacial surgeon is usually confronted with the more severe and longer existing cases of secondary retention.

The most reliable clinical and radiographic characteristic of secondary retention seems to be infraocclusion. In addition, the percussion test is used to assess ankylosis in relation to secondary retention.[3,14,21] A percussion test may easily result in false negative findings, because of its subjectivity[13] and because of the fact that minor areas of ankylosis may fail to give a change in resonance.[22] It is, therefore, not a reliable diagnostic tool. In this study, two molars with a solid, clear percussion sound showed histologically no ankylosis.

Radiographically, ankylosis was observed in a minority of cases, while according to the literature and to the results of our study, areas of ankylosis were observed

histologically in 78–100% of the cases.[9,18,20,21] The low number of cases in which ankylosis was detected radiographically may be the result of the projection of the pictured molar in a flat plane and the preferential location of ankylosis in the interradicular area of the roots.[9,21,23-25] Both circumstances result in small areas of ankylosis not usually being diagnosed on radiographs.[18] Spontaneous re-eruption of the secondarily retained molar occasionally occurs due to resorption of the area of ankylosis.[20] This was not observed, however, in our study.

In the management of secondary retention, early recognition and diagnosis as well as proper treatment and careful follow up are very important. The indication for treatment depends on the age of the patient, the extent of infraocclusion, the extent of tilting of the neighbouring teeth, and the presence and location of the successor. Usually the recommended treatment is to wait for normal shedding under the supervision of an adequate follow up.[26] Extraction of the retained molar and the use of a space maintainer are indicated in cases of severe infraocclusion, severe occlusal disturbances, and dislocation of the crypt of the successor. Whenever possible, extraction should be deferred until the first permanent molar erupts. In case of an agenetic successor, the retained molar can be left in place if infraocclusion is slight, the adjacent teeth show no tilting, and the progression of root resorption is slow. In such a case, the occlusal, labial and lingual surfaces can be build up with composite resin in order to prevent tilting of the neighbouring teeth and over-eruption of the antagonist.[27] As the eruption of the neighbouring teeth continues, however, a new build up or removal of the retained molar may be necessary. Particularly in the mandible, it is advisable to treat the resulting space orthodontically or by restoration in order to prevent tilting of the neighbouring teeth.

The presence of secondary retention in the deciduous and in the permanent dentition may have the same etiology, and is probably the result of degenerative changes in the periodontal ligament of a multi-rooted tooth. Further investigation is necessary to detect the precise cause.

REFERENCES

1 Raghoebar GM, Abma G, Boering G, Stegenga B. Secundaire retentie van melkmolaren. Ned Tijdschr Tandheelkd 1988;95:389–92.
2 Dixon DA. Observations on submerging deciduous molars. The first Chapman prize essay. Dent Pract 1963;13:303–15.
3 Eichenbaum IW. Tooth eruption and ankylosis. J Prev Dent 1977;4:39–45.
4 Kurol J. Infraocclusion of primary molars: an epidemiologic and familial study. Community Dent Oral Epidemiol 1981;9:94–102.
5 Biederman W. The incidence and etiology of tooth ankylosis. Am J Orthod 1956;42:921–6.
6 Schulze Ch. Über Retention und Reinklusion (Depression) erster und zweiter Molaren. Dtsch Zahn Mund Kieferheilkd 1962;37:338–76.
7 Herman E. Evaluation and management of ankylosed teeth. NY State Dent J 1964;30:327–33.
8 Glucksman DD. Localized vertical growth disturbance. J Am Dent Assoc 1942;29:184–7.
9 Rune B. Submerged deciduous molars. Odontol Revy 1971;22:257–73.
10 Via WF. Submerged deciduous molars: familial tendencies. J Am Dent Assoc 1964;69:127–9.
11 Helpin ML, Duncan WK. Ankylosis in monozygotic twins. J Dent Child 1986;53:135–9.
12 Messer LB, Cline JT. Ankylosed primary molars: results and treatment recommendations from an eight-year longitudinal study. Pediatr Dent 1980;2:37–47.

13 Brearley LJ, McKibben DH. Ankylosis of primary molar teeth. I. Prevalence and characteristics. II. A longitudinal study. J Dent Child 1973;40:54–63.
14 Kula K, Tatum BM, Owen D, Smith RJ, Rule J. An occlusal and cephalometric study of children with ankylosis of primary molars. J Pedod 1984;8:146–59.
15 Pilo R, Littner MM, Marshak B, Aviv I. Severe infraclusion ankylosis: report of three cases. J Dent Child 1989;56:144–6.
16 Pindborg JJ. Pathology of the dental hard tissues. Copenhagen; Munksgaard, 1970:248–251.
17 Tsukamoto S, Braham RL. Unerupted second primary molar positioned inferior to the second premolar: clinical report. J Dent Child 1986;53:67–9.
18 Thornton M, Zimmermann ER. Ankylosis of primary teeth. J Dent Child 1964;31:120–6.
19 Lamb KA, Reed MW. Measurement of space loss resulting from tooth ankylosis. J Dent Child 1968;35:483–6.
20 Darling AI, Levers BGH. Submerged human deciduous molars and ankylosis. Arch Oral Biol 1973;18:1021–40.
21 Kurol J, Magnusson BC. Infraocclusion of primary molars: histologic study. Scand J Dent Res 1984;92:564–76.
22 Andersson L, Blomlöf L, Lindskog S, Feiglin B, Hammarström L. Tooth ankylosis. Clinical, radiographic, and histological assessments. Int J Oral Surg 1984;13:423–31.
23 Rygh P, Reitan K. Changes in the supporting tissues of submerged deciduous molars with and without permanent successors. Odont Tidsk 1964;72:345–62.
24 Henderson HZ. Ankylosis of primary molars: a clinical, radiographic, and histologic study. J Dent Child 1979;46:117–22.
25 Theuerkauf J. Die Infraokklusion der Milchmolaren und Sechsjahrmolaren. Dtsch Zahnärzt Z 1960;15:1358–68.
26 Kurol J, Thilander B. Infraocclusion of primary molars and the effect on occlusal development: a longitudinal study. Eur J Orthod 1984;6:277–93.
27 Bonin M. Simplified and rapid treatment of ankylosed primary molars with an amalgam and composite resin. J Dent Child 1976;43:159–62.

CHAPTER 4

ERUPTION DISTURBANCES OF PERMANENT MOLARS

This chapter has been accepted for publication in the Journal of Oral Pathology and Medicine and is reprinted with permission of the publisher.

ABSTRACT

Eruption disturbances of permanent molars may become clinically and radiographically manifest as impaction, primary retention or secondary retention. This may result in clinical problems such as malocclusion and loss of neighbouring teeth due to caries and periodontal disease. Which of these disorders will develop, depends primarily on the eruptive stage. Factors that may interfere with the eruptive stages (i.e. follicular growth, pre-emergent eruptive spurt, postemergent eruptive spurt, juvenile occlusal equilibrium, circumpubertal occlusal eruptive spurt, adult occlusal equilibrium) and the clinical consequences of that interference are reviewed. Treatment recommendations are given.

INTRODUCTION

Eruption refers to the axial or occlusal movement of the tooth from its developmental position within the jaw to its functional position in the occlusal plane.[1] Once in occlusion, eruption continues throughout life to compensate for occlusal wear of teeth and for growth of the jaws. Tooth eruption is important in the maxillofacial development during growth and development of the face. Compensatory changes in the path of eruption occur resulting in positional changes between the jaws. If the compensation is insufficient or does not occur at all, malocclusion and other dental anomalies may develop.[2]

Disturbances of eruption may depend on systemic or on local factors. Systemic factors are present in patients with certain developmental syndromes[3,4] and as a consequence usually multiple teeth are affected. By contrast, in patients with a local eruption disturbance at most a few teeth are affected, predominantly lower third molars and maxillary canines. Eruption disturbances of first and second permanent molars are relatively rare,[5,6] but they have considerable clinical impact when they occur. This is due to the fact that first and second permanent molars are particularly important for the normal development of the dentition and coordination of facial growth. Another function is to provide sufficient occlusal support for undisturbed mastication. Disturbed eruption of permanent molars can result in a lower facial height.[7] Other consequences may be formation of a follicular cyst, pericoronal inflammation, tilting of neighbouring teeth, resorption of the roots of neighbouring teeth and malocclusion.[6,8] It is important to diagnose and treat eruption disturbances as early as possible, because treatment at a later stage is usually more complicated due to the tendency of the clinical deviation to increase with time and of the reduced ability of the remaining dentition to adjust.

In order to understand eruption disturbances of permanent molars, knowledge of their normal eruption is mandatory.[9] In this article the major facets of normal eruption and eruption disturbances of first and second permanent molars are reviewed. The clinical manifestations of these disturbances are emphasized and treatment recommendations are given.

ERUPTION OF PERMANENT MOLARS

The eruption of permanent molars differs from that of other permanent teeth in the way that they do not have preceeding deciduous teeth. The tooth germs of permanent molars develop from the backward extension of the dental lamina.[1] Their development is sequentially initiated in the tuberosity of the maxilla and at the junction of the ascending and horizontal ramus of the mandible. As a result of the growth of the jaws, the relative position of the first molar shifts anteriorly at the time of the development of the second molar. At first, the occlusal surface of the mandibular molars is mesially and that of the maxillary molars distally inclined. During growth of the jaws the crowns gradually move to an upright position.

Four mechanisms explaining the occlusal movement of a tooth during eruption have been mentioned in the literature.[1] These are root growth, hydrostatic pressure, selective bone deposition and resorption, and the pulling force of cells or fibers (or both) of the periodontal ligament (PDL). The first three mechanisms are not sufficiently supported by experimental data,[10,11] while there is evidence that the PDL and the dental follicle, from which the ligament develops, provide the force required for tooth eruption.[12] At present the origin of this force is not completely understood. It seems likely that the principal cells of the PDL, i.e. the fibroblasts, are responsible for the eruptive movement although it has also been suggested that the collagen fibrils provide the contractile force.[1] One must, however, keep in mind that eruption is a multifactorial process in which the loss of one factor can be successfully compensated by an other.[12]

Eruption can be subdivided into three prefunctional stages, i.e. follicular growth, pre-emergent eruptive spurt and postemergent eruptive spurt, and three postfunctional stages i.e. juvenile occlusal equilibrium, circumpubertal occlusal eruptive spurt and adult occlusal equilibrium.[13] During the pre-emergent eruptive spurt, the rate of tooth movement is determined by the generative and adaptive capacity of the dental follicle as well as by resorption of the overlying bone and the gingival tissues. The presence of a dental follicle is a prerequisite for bone resorption in the eruption path as well as for formation of a characteristic type of trabecular bone below the roots of the erupting tooth.[10,11,14] PDL is probably not required for the pre-emergent eruptive stages, because eruption before emergence also proceeds in the absence of root formation, and thus without PDL.[10,11]

After emergence, the bone and gingiva are no longer decelerating factors.[13] The tooth can erupt at maximal rate towards its functional occlusal position, protected from masticatory and soft tissue forces by the adjacent fully erupted teeth. The PDL eruption mechanism becomes active after the tooth perforates the roof of the follicle. The moment at which this actually starts is yet unclear. If cessation of eruption after emergence occurs without a physical barrier in the eruption path, this disorder is probably due to a disturbance of the PDL.

ERUPTION DISTURBANCES OF PERMANENT MOLARS

Impaction is cessation of the eruption of a tooth caused by a clinically or radiographically detectable physical barrier in the eruption path or due to an abnormal position of the tooth.[8,15] If no physical barrier can be identified as an explanation for the cessation of eruption of a normally placed and developed tooth germ before emergence, the tooth is considered to be primarily retained. Cessation of eruption of a tooth after emergence neither due to a physical barrier in the path of eruption nor as a result of an abnormal position is called secondary retention.[16] In the literature impaction and retention are often used synonymously.[17] The etiology of the three disorders (impaction, primary retention, secondary retention) is, however, different as is the treatment approach for these disorders. The possible clinical features are summarized in Table 1.

Table 1 Possible clinical features of eruption disturbances of first and second permanent molars

Consequence	Impaction		Primary retention	Secondary retention
	before emergence	after emergence		
Affected molar:				
Abnormal position	+	+	–	–
Physical barrier	+	+	–	–
Caries	–	+	–	+
Restoration	–	+	–	+
Attrition marks	–	–	–	+
Epithelial connection between molar and oral cavity	–	–	–	+
Periodontal disease	–	+	–	+
Pericoronal inflammation	–	+	–	+
Infraocclusion	–	+	–	+
Resorption	+	+	+	+
Reduced vertical dimension of alveolar bone	–	+	–	+
Neighbouring teeth:				
Tilting	+	+	+	+
Resorption	+	+	+	+
Caries	–	+	–	+
Periodontal disease	–	+	–	+
Occlusion:				
Malocclusion	+	+	+	+
Over-eruption of antagonist	+	+	+	+
Loss of arch length	+	+	+	+
Impaired masticatory function	+	+	+	+

\+ may be present
– absent

Impaction. In the majority of the cases, impaction is the result of a physical barrier in the path of eruption (Fig. 1). This barrier is a factor that is independent of the

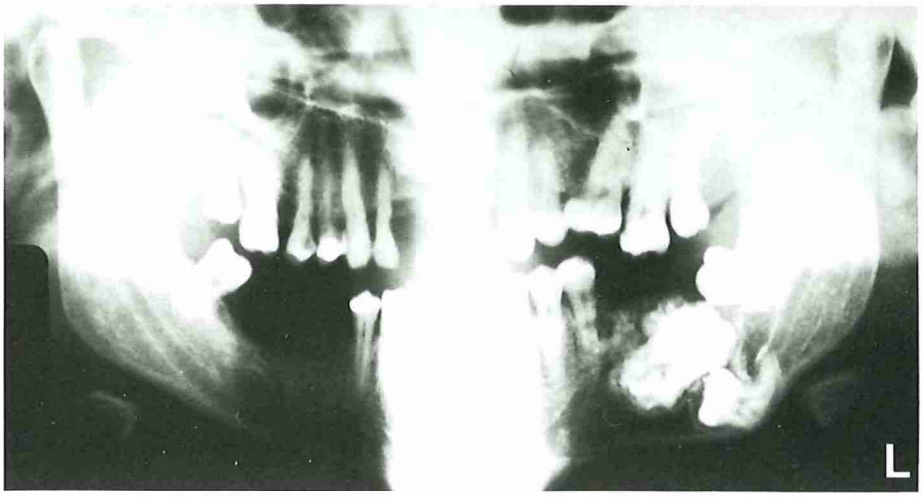


Fig. 1 Orthopantomogram of a 46-year-old male showing impaction of the mandibular left second molar. A complex odontome was found as a possible physical barrier in the eruption path.

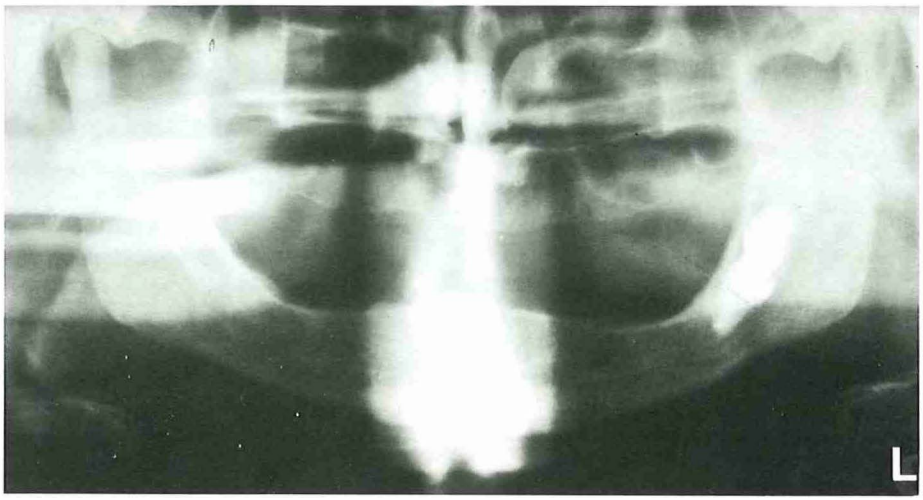


Fig. 2 Orthopantomogram of a 54-year-old male showing distal horizontal impaction of the second molar and mesial horizontal impaction of the third molar in the mandible on the left side.

eruption process as such and is in all cases present before full occlusal contact with the antagonists occurs. Impaction may also be due to an abnormal eruption path, presumably because of an unusual orientation of the tooth germ (Fig. 2).[6,15,18]

When an impacted molar is clinically visible, its long axis is always angulated when compared to that of the neighbouring teeth. If impaction has occurred before emergence, the radiograph shows that the long axis of the impacted molar is not parallel to the normal eruption path. This angulation of, for example, an

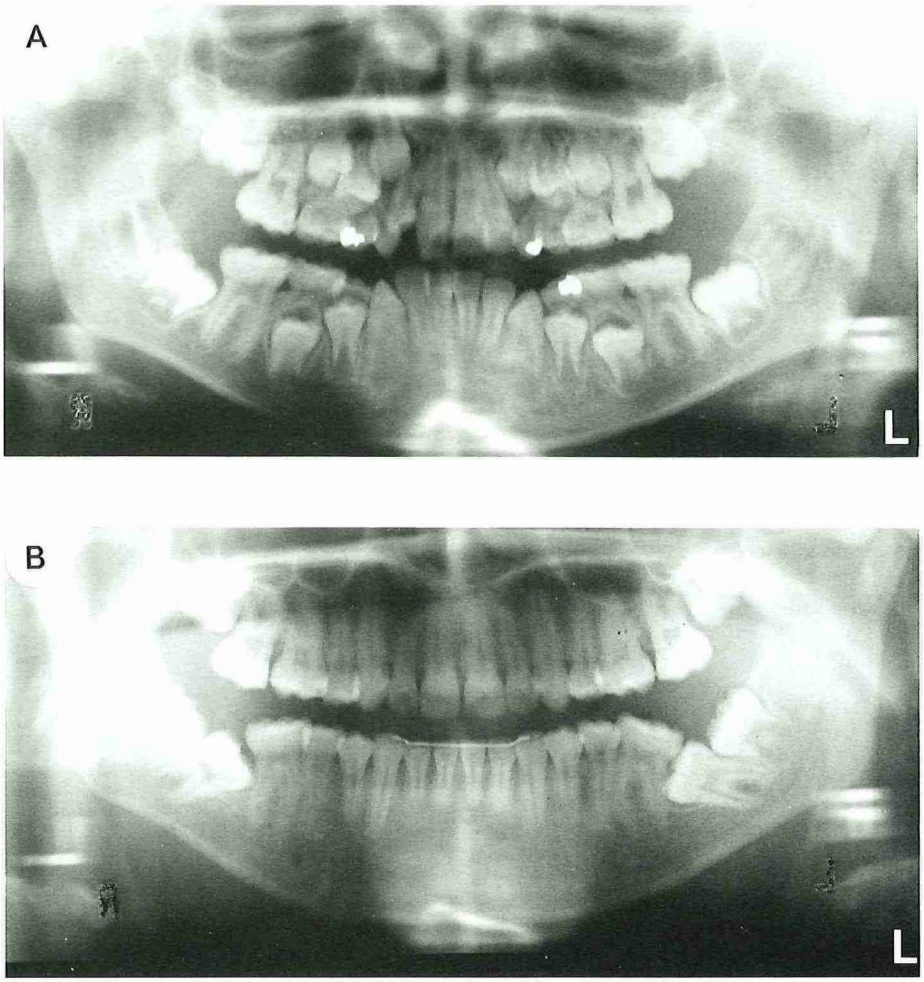


Fig. 3 A Orthopantomogram of a 10-year-old female showing normal position of the germs of the second molars in the mandible. **B** Orthopantomogram of the same female 5 years later showing impaction of the second molars in the mandible.

impacted lower molar may be the result of failure of the molar to upright from its mesial inclination during eruption (Figs. 3A, 3B). Other factors to account for impaction that have been suggested are inadequate arch length, supernumerary teeth, odontogenic tumors (eg. dentigerous cysts, odontomas), and idiopathic factors (Fig. 1).[19-22]

Impaction of first molars is often diagnosed as ectopic eruption. An ectopic molar erupts at a mesial angle to the normal path of eruption.[23] The result is cessation of eruption and atypical resorption of the distal surface of the neighbouring second deciduous molar. In most cases it affects maxillary first molars.[9]

Impaction of a second molar is usually associated with an arch length deficiency.[24]

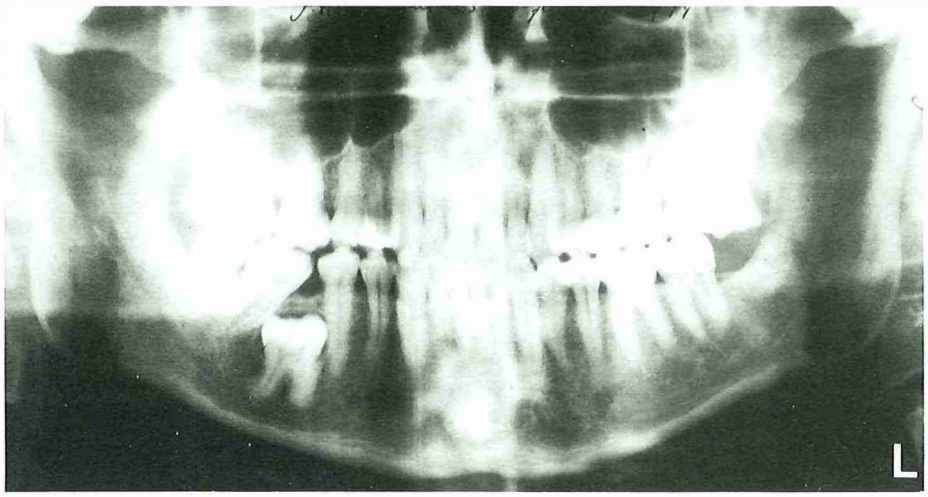


Fig. 4 Orthopantomogram of a 32-year-old male showing primary retention of the mandibular right first molar. The crown is covered by bone.

If the increase in arch length does not synchronize with the eruption of the second molar, an environment favouring impaction will be the result. This occurs more commonly in the mandible than in the maxilla.

Pericoronitis is a frequently observed clinical complication of impacted molars.[17] It particularly occurs when the follicle of the impacted tooth communicates with the oral cavity and becomes infected. It is frequently observed in cases of impacted lower third molars. Furthermore, impacted teeth may cause root resorption of the neighbouring teeth because of their location and their tendency to erupt in an abnormal position. An impacted molar may even undergo resorption.[6,8] Carious lesions are often observed in the neighbouring teeth. Finally, cystic or neoplastic changes may occur in the follicle of a non emerging impacted molar.[17]

Primary retention. Primary retention of molars is the cessation of eruption before emergence, without a physical barrier in the eruption path nor being the result of an abnormal position (Fig. 4). The nonresorbing bone occlusally of a primarily retained molar should be considered as a normal barrier in the eruption path. Primary retention is synonymous with "unerupted" and "embedded".[8] When eruption of a permanent tooth is at least two years behind schedule, primary retention should be suspected. A follow up of at least six months is necessary to detect radiographically whether the tooth is showing any eruptive movement or not.[25] Primary retention may be caused by a defect in the eruption mechanism similar to that found in developmental conditions like cleidocranial dysostosis. At present, there appears to be consensus that eruption failure of normal permanent teeth in cleidocranial dysostosis is associated with a disturbance in the resorption of overlying bone and is not due to an abnormality of the periodontal ligament.[4,26] The approach to treatment primarily retained molars should be according to the same

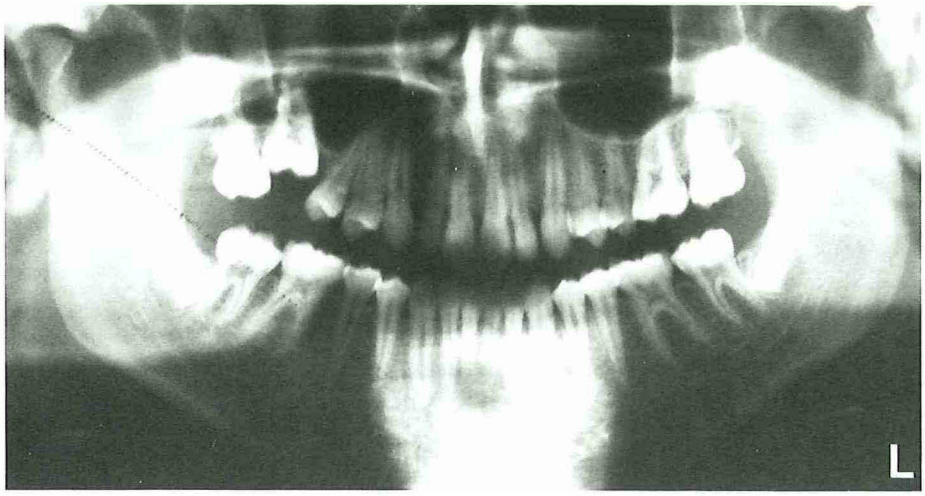


Fig. 5 Orthopantomogram of a 16-year-old male showing secondary retention of the maxillary right first molar.

principles as non emergent teeth in case of cleidocranial dysostosis.[27,28] Primary retention is probably caused by a disturbance in the dental follicle that fails to initiate the metabolic events responsible for bone resorption in the eruption traject.[29]

As a primarily retained molar has not passed the pre-emergent eruptive spurt, it is not visible within the oral cavity. Radiographically, the molar is normally oriented in its eruption path, the crown is covered by bone and mucosa (Fig. 4), and root elongation leads to bone resorption around the radicular portion of the tooth. In case of normal eruption mechanism, a period of rapid eruption in the occlusal direction is associated with continued root formation,[30] while the growing apex of the root remains at the same distance relative to the inferior alveolar canal. In primary retention, the deeply situated roots are sometimes completely formed. This implies that growth of the root has occurred in apical direction and, therefore, seems not to be related to the eruption.

Secondary retention. Secondary retention refers to cessation of eruption after emergence neither due to a physical barrier in the eruption path nor as a result of an abnormal position (Fig. 5).[16,31] Other terms used in the literature are submerged, Halbretention, reimpaction, reinclusion and ankylosis.[6,32-34] The term ankylosis has not been specifically used to refer to this disorder, but it is probably a main factor in its development. Clinically secondary retention is usually suspected when a molar is in infraocclusion at an age when the tooth would normally be in occlusion. The involvement of ankylosis might be detected with the percussion test and radiographic evidence of the periodontal ligament obliteration. These diagnostic tools are, however, not very reliable as the ankylosed area in secondary retention is usually too small to be detected with these tools.[16]

The etiopathogenesis of secondary retention is unknown. As already mentioned, ankylosis has been suggested as the main factor.[6,32] In a histological study of 26

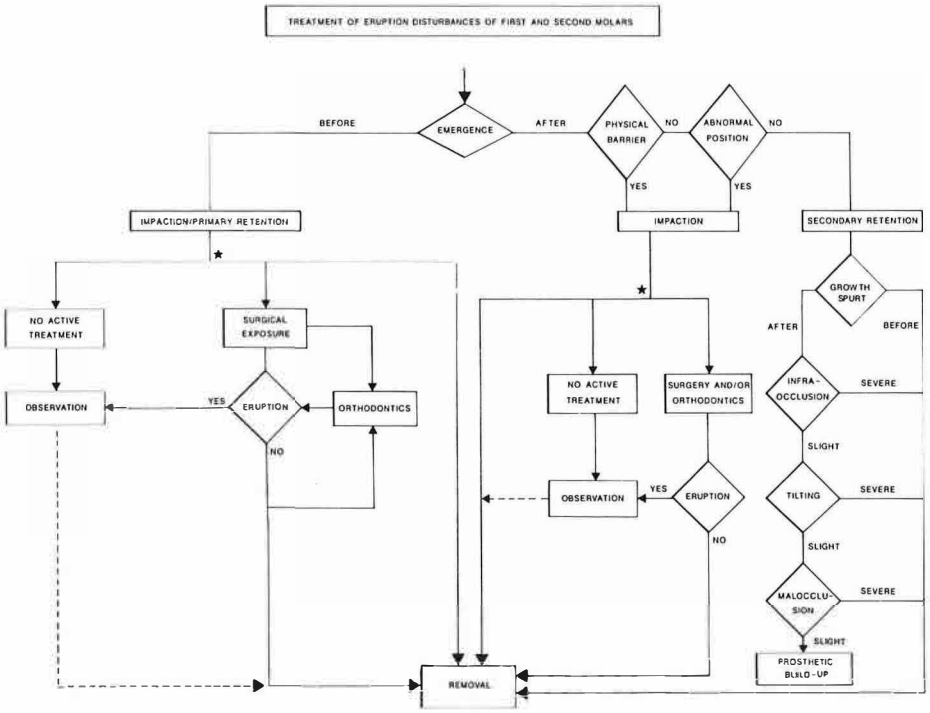


Fig. 6 Treatment survey of eruption disturbances of first and second molars. Asterisks indicates a choice of treatment depending on, presence of a pathologic process, age of the patient and position of the molar.

secondarily retained molars, all roots showed local areas of ankylosis.[16] Trauma, infection, disturbed local metabolism, and genetic factors have been suggested as causative factors.[32-34] Once ankylosis between the tooth and alveolar bone has developed, the eruption as well as the growth of the alveolar process in the affected area stop. The neighbouring teeth continue to erupt and consequently the affected tooth is in infraocclusion. This may cause malocclusion, especially after tilting of the neighbouring teeth towards the diastema.

TREATMENT OF ERUPTION DISTURBANCES OF PERMANENT MOLARS

Treatment approach of eruption disturbances before emergence is independent of the specific diagnosis. By contrast, after emergence treatment of impaction is different from treatment of secondary retention (Fig. 6).

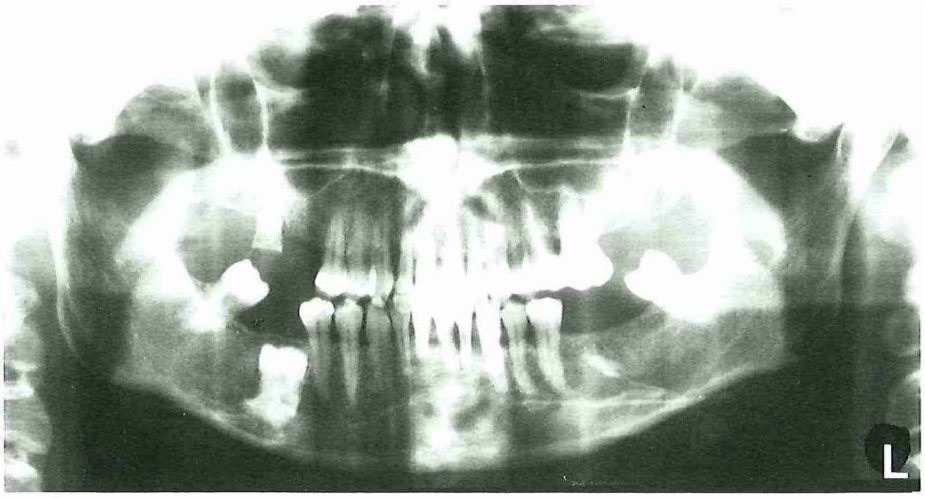


Fig. 7 Orthopantomogram of a 36-year-old male showing primary retention of the mandibular right first molar with resorption of the crown and root.

Before emergence: **primary retention or impaction**. The treatment of choice depends on the presence of a pathologic process, the age of the patient, the position of the molar in the jaw relative to other structures, and the willingness of the patient to undergo treatment. In most cases, early surgical exposure of the crown is the best initial treatment.[32,36,37] It is frequently followed by postoperative orthodontic treatment to create sufficient space and to move the molar into a normal position in the dental arch. Regular observation (e.g. biannual) will reveal if eruption is proceeding. When surgery is performed prior to completion of root formation, spontaneous eruption and continuation of root development can be expected.[38] Eruption often does not occur when the roots have formed a hook[21,29] or are ankylotic.[32] Luxation of the molar after exposure has also been suggested to promote eruption[38,40], but it is probably unnecessary as exposure alone is generally sufficient to enable spontaneous eruption.[37] Eruption can occur because the PDL and other eruptive mechanisms are basically normal. Removal of the molar is indicated if exposure and orthodontic treatment are not followed by eruption or when a pathologic process is present. A deeply located molar symptomless and not interfering with eruption of the neighbouring teeth may be left undisturbed.[32] Removal of such a molar can be complicated and hazardous to the surrounding structures, such as neighbouring teeth, maxillary sinus, blood vessels or inferior alveolar nerve.[39,41] These molars may start to resorb spontaneously and become ankylotic (Fig. 7).[8] Clinical inspection and radiographic observation on a regular basis is necessary,[39] as migration of such a molar has been reported.[42]

After emergence: **impaction**. In case of impaction, early removal of the physical barrier increases the likelihood of spontaneous eruption of the tooth.[37] An abnormal position of the germ of a third molar may form a barrier causing impaction of

the second molar.[43,44] The recommended treatment is removal of the third molar at the age of eleven to fourteen years[44] in combination with a thorough follow up of the eruption of the second molar. A second molar impacted against the first molar can be treated by surgical or orthodontic repositioning with or without removal of the adjacent third molar. Some clinicians advocate removal of the second molar allowing eruption of the third molar at the position of the second molar, or transplantation of the third molar into the socket of the extracted second molar.[24] For the choice of the treatment approach, the position and quality of the third molar are decisive.

Several orthodontic and surgical methods for uprighting impacted second molars have been reported.[24,45-49] The optimal moment for uprighting is when two-thirds of the roots have been formed.[24,50] If uprighting is carried out too early, root formation stops before the roots are fully formed.[50] Molars with fully formed roots have a poor prognosis.[5,24] In these cases surgical tipping procedures were found to be less likely to develop pulpal necrosis than bodily movement does.[48] Contraindications to upright molars are extreme horizontal impaction and widely divergening roots.[50] Surgical or orthodontic uprighting is important to provide a good occlusion and to reduce the risk of caries and periodontal disease by diminishing the risk of food-impaction and stagnation.[46] In many cases, a combination of surgical and orthodontic treatment is adequate (Fig. 6). If no eruptive movement can be established, removal of the second molar may be necessary.

Active treatment of ectopic permanent molars is not always necessary as occasionally these molars free themselves and erupt into their normal position. If no spontaneous correction occurs, orthodontic treatment with or without extraction of the neighbouring deciduous molar is indicated.[23,47,51]

After emergence: **secondary retention**. The treatment of secondary retention depends on the age of the patient, and the extent of infraocclusion and malocclusion. Spontaneous eruption of a secondarily retained molar may occur, but is extremely rare.[31] Orthodontic movement of the affected molar is not possible because of an abnormal periodontal ligament.[52] If secondary retention develops prior to the growth spurt, immediate removal of the affected molar followed by orthodontic alignment of the neighbouring teeth is the proper treatment. When secondary retention develops during the growth spurt, the molar should be observed at 6 month intervals. During or after the growth spurt, no active treatment other than a build up is recommended if the neighbouring teeth do not show tilting and the extent of infraocclusion is minor and stable.[53] In all other cases, the affected molar should be removed (Fig. 6). The resulting edentulous space can close spontaneously or has to be closed orthodontically or prosthetically. Another possibility is to transplant a developing third molar into the diastema. Before undertaking transplantation, the available space in the dental arch should be increased because some excess is required to improve the prognosis.[54]

Luxation of the secondarily retained molar has been described[32,54] and used with success,[32] but in our hands it is not a successful treatment in case of secondary retention.[53] In a histological study of secondarily retained molars, local areas of

ankylosis were observed in the bifurcation and interradicular root surface in 81% of the cases.[16] Luxation of these molars will not easily result in breaking of these interradicular bony connections, because the rotation point during luxation is through the bifurcation area. On the contrary, it will tend to promote new areas of ankylosis after healing. The discrepancy between the good treatment results reported in the literature[32] and our experience is probably based on no proper differentiation between impaction and secondary retention.

DISCUSSION

The diagnostic characteristics of eruption disorders are different but the treatment approaches are in some cases identical. Primary and secondary retention of permanent molars reflect disturbances in a particular stage of the eruptive process, while impaction is due to a physical barrier or an abnormal position of the tooth and thus not directly related to a particular eruptive stage. It is important to distinguish between these three phenomena in order to understand the clinical features (Table 1) and to choose a proper treatment (Fig. 6). The major treatment concern of secondarily retained molars is that these molars can not be moved orthodontically due to an abnormal periodontal ligament.[16,52] In contrast, orthodontics is a major modality in treatment of impacted teeth as these molars often have an abnormal position in the eruption path and mostly reveal an unchanged PDL. Primarily retained molars can also be moved orthodontically, but this is often not necessary because of the normal position in the eruption tract.

The clinical and radiographic characteristics of eruption disorders are usually sufficient to differentiate between impaction, primary retention and secondary retention,[19] although in some cases differentiation is difficult.[6] Primary and secondary retention can be differentiated considering the stage during which the molar ceases to erupt. Attrition marks, restorations, and an epithelial tube between the tooth and the oral cavity are characteristic for secondary retention and are an additional tool to distinguish between primary and secondary retention (Table 1).[6,32] These signs show that the tooth has been present in the oral cavity. An abnormal position of the molar is a usual phenomenon of impaction, but before emergence a position of the molar buccally or lingually from the normal eruption path is difficult to detect on radiographs. In these cases, the initial diagnosis likely would be primary retention, while after surgical exposure the diagnosis would have been corrected to impaction. Although this diagnostic problem can be anticipated by making a second radiograph in another plane, it has no treatment consequences as in both conditions surgical exposure, in combination with orthodontic treatment if indicated, is the treatment of choice. Furthermore, it has been suggested that impaction can be caused by tilting and pressure of the second molar over the first molar.[33] This can occur only due to cessation of eruption of the first molar, because at the time when the second molar starts to erupt the first molar normally is already in occlusion. Thus the proper diagnosis in such a case is secondary retention.

The diagnosis of eruption disorders involves clinical and radiographic examination followed by tentative classification. The diagnosis and treatment planning should be placed into the perspective of the patient's age, i.e. the stage of eruption, as well as of factors like the patient's needs and self-image. In this scope, early recognition of eruption disturbances is mandatory in order to start treatment at the optimal moment and to minimize complications.

REFERENCES

1 Ten Cate AR. Oral histology. Development, structure, and function. 3rd ed. St Louis: CV Mosby, 1989:275–98.
2 Björk A, Skieller V. Facial development and tooth eruption: an implant study at the age of puberty. Am J Orthod 1972;62:339–83.
3 Gorlin RJ, Pindborg JJ, Cohen MM. Syndromes of the head and neck. 2nd ed. New York: Mc Graw-Hill, 1976:180–4.
4 Jensen BL, Kreiborg S. Development of the dentition in cleidocranial dysplasia. J Oral Pathol Med 1990;19:89–93.
5 Farman AG, Eloff J, Nortjé CJ, Joubert JJV. Clinical absence of the first and second permanent molars. Br J Orthod 1978;5:93–7.
6 Pindborg JJ. Pathology of the dental hard tissues. Copenhagen: Munksgaard, 1970:225–55.
7 Proffit WR. Equilibrium theory revisited: factors influencing position of the teeth. Angle Orthod 1978;48:175–86.
8 Shafer WG, Hine MK, Levy BM. A texbook of oral pathology. 4th ed. Philadelphia: WB Saunders, 1983:66–9.
9 Moyers RE. Handbook of orthodondics. 4th ed. Chicago: Yearbook medical publ, 1988:111–9.
10 Cahill DR, Marks SC. Tooth eruption: evidence for the central role of the dental follicle. J Oral Pathol 1980;9:189–200.
11 Marks SC, Cahill DR. Experimental study in the dog of the non-active role of the tooth in the eruptive process. Arch Oral Biol 1984;29:311–22.
12 Berkovitz BKB. The effect of root transection and partial root resection on the unimpeded eruption rate of the rat incisor. Arch Oral Biol 1971;16:1033–43.
13 Steedle JR, Proffit WR. The pattern and control of eruptive tooth movements. Am J Orthod 1985;87:56–66.
14 Cahill DR, Marks SC, Wise GE, Gorski JP. A review and comparison of tooth eruption systems used in experimentation: a new proposal on tooth eruption. In: Davidovitch Z. eds. Biological mechanism of tooth eruption and root resorption. Birmingham: EBSCO Media, 1988:1–7.
15 Regezi JA, Sciubba JJ. Oral pathology. Clinical-pathologic correlations. Philadelphia: WB Saunders, 1989:469–71.
16 Raghoebar GM, Boering G, Jansen HWB, Vissink A. Secondary retention of permanent molars: a histologic study. J Oral Pathol Med 1989;18:427–31.
17 Van der Waal I, Van der Kwast WAM. Oral pathology. Chicago: Quintessence, 1988:122–4.
18 Watkins JJ, Tucker GJ. An unusual form of impaction of two permanent molars: a case report. J Dent 1977;5:215–8.
19 Grover PS, Lorton L. The incidence of unerupted permanent teeth and related clinical cases. Oral Surg Oral Med Oral Pathol 1985;59:420–5.
20 Lapeer GL. Impaction of the first permanent molar. Can Dent Assoc J 1988;54:113–4.
21 Mellor TK. Six cases of non-eruption of the first adult lower molar tooth. J Dent 1981;9:84–8.
22 Mehta DS, Ramchandra CS, Mehta MJ. Impacted first permanent molar at the lower border of the mandible. J Indian Dent Assoc 1986;58:129.
23 Kurol J, Bjerkin K. Treatment of children with ectopic eruption of the maxillary first permanent molar by cervical traction. Am J Orthod 1984;86:483–92.
24 Johnson JV, Quirk GP. Surgical repositioning of impacted mandibular second molar teeth. Am J Orthod Dentofacial Orthop 1987;91:242–51.
25 Kracke RR. Delayed tooth eruption versus impaction. J Dent Child 1975;42:371–4.
26 Chapnick LA, Main JH P. Cementum in cleidocranial dysostosis. Can Dent Assoc J 1976;42:139–42.

27 Smylski PT, Woodside DG, Harnett BE. Surgical and orthodontic treatment of cleidocranial dysostosis. Int J Oral Surg 1974;3:380–5.
28 Hall RK, Hyland AL. Combined surgical and orthodontic management of the oral abnormalities in children with cleidocranial dysplasia. Int J Oral Surg 1978;7:267–73.
29 Oliver RG, Richmond S, Hunter B. Submerged permanent molars: four case reports. Br Dent J 1986;160:128–30.
30 Darling AI, Levers BGH. The pattern of eruption of some human teeth. Arch Oral Biol 1975; 20:89–96.
31 Raghoebar GM, Van Koldam WA, Boering G. Spontaneous reeruption of a secondarily retained permanent molar and an unusual migration of a lower third molar. Am J Orthod Dentofacial Orthop 1990;97:82–4.
32 Biederman W. Etiology and treatment of tooth ankylosis. Am J Orthod 1962;48:670–84.
33 Schulze Ch. Über Retention und Reinclusion (Depression) erster und zweiter Molaren. Dtsch Zahn Mund Kieferheilkd 1962;37:338–76.
34 Bosker H, Ten Kate LP, Nijenhuis LE. Familial reinclusion of permanent molars. Clin Genet 1978;13:314–20.
35 Brady J. Familial primary failure of eruption of permanent teeth. Br J Orthod 1990;17:109–13.
36 Gono C. Delayed eruption due to overlying fibrous connective tissue. J Dent Child 1987;54:359–60.
37 Jacobs SG. The surgical exposure of teeth: simplest, safest and best? Aust Orthod J 1987;10:5–11.
38 Kaban LB, Needleman HL, Hertzberg J. Idiopathic failure of eruption of permanent molar teeth. Oral Surg Oral Med Oral Pathol 1976;42:155–63.
39 Di Salvo NA. Evaluation of unerupted teeth: orthodontic viewpoint. J Am Dent Assoc 1971;82: 829–35.
40 Skolnick IM. Ankylosis of maxillary permanent first molar. J Am Dent Assoc 1980;100:558–60.
41 Conklin WW. Impacted mandibular first molar. Oral Surg Oral Med Oral Pathol 1972;33:847–8.
42 Jasmin JR, Jonesco-Benaich N, Terestri P, Giudicelly J. Migration of an impacted mandibular second molar. Oral Surg Oral Med Oral Pathol 1989;67:478–9.
43 Levy I, Regan D. Impaction of maxillary permanent second molars by the third molars. J Paed Dent 1989;5:31–4.
44 Ranta R. Impacted maxillary second permanent molars. J Dent Child 1985;52:48–51.
45 Vig KWL. Some methods of uprighting lower second molars II. Br J Orthod 1976;3:39–44.
46 Reynolds LM. Uprighting lower molar teeth. Br J Orthod 1976;3:45–51.
47 Lang R. Uprighting partially impacted molars. J Clin Orthod 1985;19:646–50.
48 Laskin DA, Peskin S. Surgical aids in orthodontics. Dent Clin North Am 1968;12:509–24.
49 Wellfelt B, Varpio M. Disturbed eruption of the permanent lower second molar: treatment and results. J Dent Child 1988;55:183–9.
50 Graber TM. Orthodontics Principles and Practice. 3rd ed. Philadelphia: WB Saunders, 1972:748–57.
51 Kennedy DB, Turley PK. The clinical management of ectopically eruption first permanent molars. Am J Orthod Dentofac Orthop 1987;92:336–45.
52 Proffit WR, Vig KWL. Primary failure of eruption: a possible cause of posterior open-bite. Am J Orthod 1981;80:173–90.
53 Raghoebar GM, Boering G, Booy C, Vissink A. Treatment of the retained permanent molar. J Oral Maxillofac Surg 1990;48:1033–8.
54 Van der Linden FPGM. Problems and procedures in dentofacial orthopedics. Chicago: Quintessence, 1990:297–307.

CHAPTER 5

SECONDARY RETENTION OF PERMANENT MOLARS: CLINICAL AND RADIOGRAPHIC CHARACTERISTICS

This chapter is based on articles published in Journal of Dentistry (in press; section 5.1), Quintessence International (in press; section 5.2) and American Journal of Orthodontics and Dentofacial Orthopedics 1990;97:82–4 (section 5.3). Articles are reprinted with permission of the publisher.

5.1 CLINICAL AND RADIOGRAPHIC CHARACTERISTICS OF SECONDARY RETENTION OF PERMANENT MOLARS

ABSTRACT

Secondary retention refers to the cessation of eruption of a tooth after emergence neither due to a physical barrier in the path of eruption nor as a result of an abnormal position. In this study, the clinical and radiographic phenomena of 81 secondarily retained permanent molars in a group of 53 patients were evaluated. Retained molars that were removed for therapeutical reasons (n=38) were histologically studied to detect possible ankylotic areas. The first molars in the mandible and maxilla were most frequently affected. The mean infraocclusion at the patients' first visit was 4.3±1.9 mm. After six months, infraocclusion had increased in adolescents and seemed to be stable in adults. Tilting of the adjacent teeth was observed in 39 cases of secondary retention. A solid, clear percussion sound and a partial absence of the periodontal ligament space on radiographs were noted in less than one fifth of the affected molars, while histological evaluation revealed that local areas of ankylosis were present in all removed molars. From the histological, percussion and radiographic data of the 38 removed molars a sensitivity of the percussion test and radiographs to detect ankylotic areas of, respectively, 29% and 21% was calculated. During a period of four years, six new cases of secondary retention were observed in the same population. From this study it is concluded that secondary retention of permanent molars seems to be associated with focal ankylosis and that the percussion test and radiographs are not reliable to exclude the presence of ankylotic areas. The presence of ankylotic areas and tendency of infraocclusion to increase in adolescents and to be rather stable in adults have major implications for therapy.

INTRODUCTION

Eruption refers to the axial or occlusal movement of the tooth from its developmental position within the jaw to its functional position in the dental arch.[1] Normally this process is in phase with the growth of the alveolar process and continues throughout life to compensate for masticatory wear and the growth of the jaws. Secondary retention refers to the cessation of the eruption of a tooth after emergence neither due to a physical barrier in the path of eruption nor as a result of an abnormal position.[2] In the literature, this disturbance of eruption is also known as submerged, ankylosis, reimpaction, Halbretention, and reinclusion.[3-7]

Infraocclusion of a molar at an age when the tooth would normally be in occlusion is the common clinical characteristic of secondary retention. A solid, clear percussion sound and absence of the periodontal ligament space have been suggested as additional characteristics of secondary retention,[8-10] but there is no general agreement on the diagnostic value of these clinical and radiographic phenomena. Because timely treatment of secondary retention is necessary to prevent severe malocclusion,[11] it would be of great clinical value to have reliable diagnostic parameters in addition to infraocclusion. This particularly counts for doubtful

cases as the treatment of secondary retention is essentially different from treatment of other eruption disorders of permanent molars.[12] The aim of this study was to evaluate the clinical, radiographic and histological characteristics of secondary retention and to detect whether, besides infraocclusion, percussion tests and radiographs are reliable complementary tools for diagnosing secondary retention.

MATERIAL AND METHODS

Material. A molar was considered to be secondarily retained if the occlusal surface was 1 mm or more below the occlusal level of the other teeth in the relevant dental arch,[13] at an age at which the tooth normally would have been in occlusion and that this is not the result of an abnormal position or physical barrier in the eruption path. Molars with a disturbed morphology due to extensive carious lesions or restorations were excluded. A group of 25 female and 28 male patients (mean age 21.5 ± 11.9 years, median 17.5 years, range 9–62 years) with one or more secondarily retained molars participated in this study. The patients were referred by dentists, orthodontists and oral and maxillofacial surgeons to the Department of Oral and Maxillofacial Surgery of the University Hospital of Groningen. All subjects considered themselves to be in good health, and had no history of apparent medical problems, trauma, inflammation of the relevant molar area, or congenital anomalies.

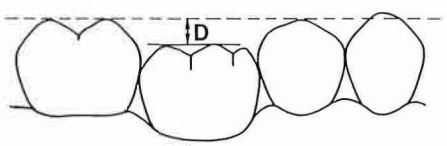


Fig. 1 Schematic illustration of a secondarily retained first molar in the mandible. Infraocclusion is the distance (D) between the occlusal plane of the retained molar and the occlusal surface of the relevant arch.

Clinical examination. A standardized assessment of the dental condition of all patients was performed, included the following items:

a the number and distribution of the secondarily retained molars.

b the extent of infraocclusion (mm). The infraocclusion was measured as the distance between the occlusal surface of the retained molar and the occlusal plane of the relevant arch (Fig. 1). Distances were measured with a periodontal probe.

c the sound on percussion of the retained tooth compared to that of the neighbouring teeth; scored as a dull sound (normal tooth) or a solid, clear sound (ankylosis).[9,10,14]
d the extent of tilting of the adjacent teeth; scored as no tilting, moderate tilting (less than half of the molar's occlusal surface covered) and severe tilting (more than half of the molar's occlusal surface covered).
e the presence of dental caries and restorations.

Radiographic examination. Intra-oral periapical radiographs of the affected molar and an orthopantomogram were taken of each patient. The radiographs were kept separate from the clinical data and were analyzed in random order with regard to:

a the vertical development of the alveolar process in the area of the secondarily retained tooth.
b the absence of a periodontal ligament space (PDLS).
c the presence of root resorption of the secondarily retained tooth.
d the presence of hypercementosis of the secondarily retained tooth. Hypercementosis was defined as thickening and apparent blunting of the roots on the radiograph. The roots lose their typical 'sharpened' or 'spiked' appearance and exhibit rounding of the apex.[14]
e abnormal curvature of the roots (presence of dilaceration). Dilaceration was defined as an abrupt deviation of the long axis of the crown or root portion of the tooth.[15]

Histological examination. Thirty-eight secondarily retained molars were removed for therapeutic reasons. Eight unaffected molars that had been removed for orthodontic reasons served as a control. After removal, the molars were fixed in a 10% buffered formalin solution for 2 weeks, washed with tap water and demineralized in a solution of 25% formic acid and 10% sodium citrate in water. After embedding in paraffin 7 μm thick sections were cut in the mesio-distal direction parallel to the vertical axis of the molar and stained with hematoxylin-eosin. The sections were examined by light microscopy. Histological examination was focused on the presence of ankylotic areas on the root surface, i.e. direct contact of the alveolar bone with root cementum or dentin and hypercementosis.

Follow-up. All patients were advised about the implications of their dental disorder and agreed to participate in a follow-up study. At six month-intervals the patients were re-examined clinically and radiographically, according to the protocol given above, until treatment was started. Special attention was paid to any possible eruptive movement of the retained molar as spontaneous re-eruption has been reported.[16]

Statistical and mathematical analysis. All data were separately analyzed for the presence of one, two, three and four secondarily retained molars, because the number of retained molars within one patient may possibly be related to the extent or progression of the disease. No weighting factors are known for these conditions permitting an overall analysis. Furthermore all statistical analyses were separately

applied for first and second retained molars. The following analyses were performed:

a the distribution of the secondarily retained molars (binominal distribution; null-hypothesis: equal frequencies for first and second retained molars).
b the extent of infraocclusion in molars with and without tilting of neighbouring teeth, abnormal sound on percussion, and absence of PDLS (t-test; null-hypotheses: extent of infraocclusion is not related to tilting, percussion sound, absence of PDLS).
c the progression of infraocclusion with time (matched t-test; null-hypothesis: infraocclusion does not increase with time).

Table 1 Distribution of secondarily retained permanent molars

Number of retained molars in one patient	Number of patients	M1	M2
1	34	26	8
2	12	19	5
3	5	13	2
4	2	7	1
Total	53	65	16

Sensitivity (S) and specificity (Sp) of the clinical and radiographic diagnostic methods, when compared with the histological results, were calculated according to $S=TP/(TP+FN)\times 100\%$ and $Sp=TN/(TN+FP)\times 100\%$ in which TP is the number of true positives, FN the number of false negatives, TN the number of true negatives and FP the number of false positives. The level of rejection was 0.05 in all cases.

RESULTS

Clinical features. A total number of 81 permanent molars in retention varying from 1 to 4 teeth per person was examined. The distribution of the secondarily retained permanent molars is given in Table 1. The first permanent molars in the mandible ($n=36$) and maxilla ($n=29$) were more frequently affected than second permanent molars ($p<0.05$). In five patients the adjacent molar and in 13 patients the antagonist was also affected. In one patient secondary retention was observed in all quadrants. The mean infraocclusion of the retained molars was 4.3 ± 1.9 mm. The extent of infraocclusion of three molars was so severe that the teeth were almost covered by the surrounding tissues (Fig. 2). In these cases a narrow trans-epithelial connection between the occlusal surface of the retained tooth and the oral cavity could be detected. As a result of the covering, it was not possible to perform the

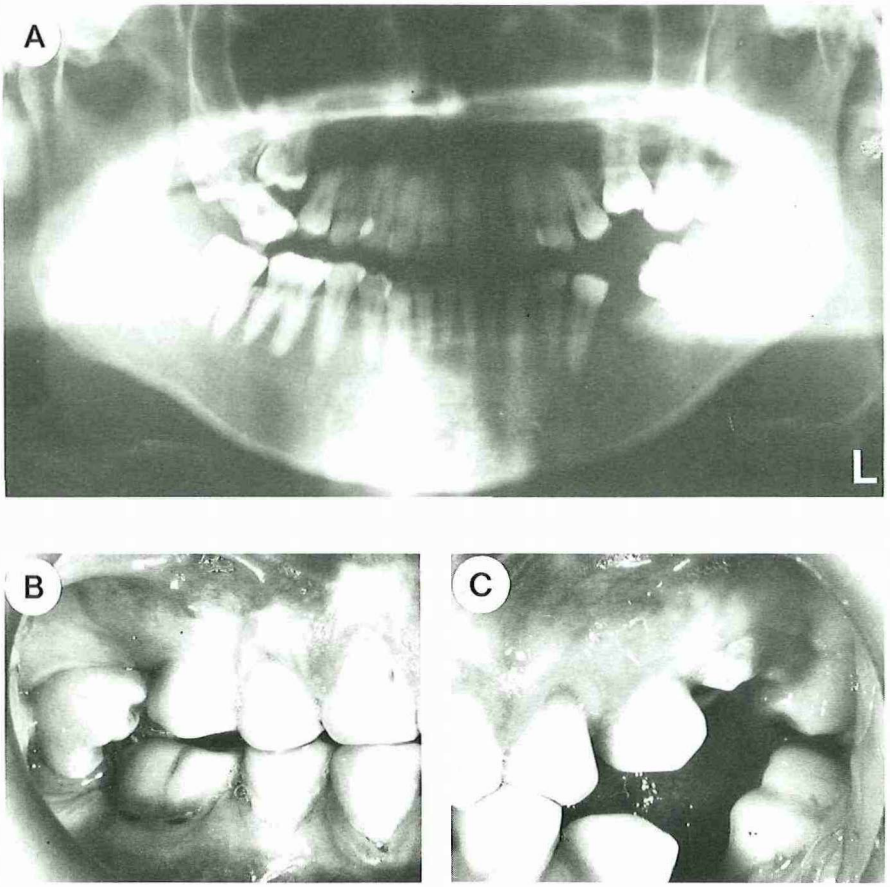


Fig. 2 A Orthopantomogram of a 26-year-old female with secondary retention of the first molars in the maxilla. The right first molar was almost completely covered by surrounding tissues. The neighbouring teeth showed severe and moderate tilting. **B** Clinical view of the right side. **C** Clinical view of the left side.

percussion test in these three cases. In 16 teeth a solid sound characteristic of ankylosis was recorded, while in 62 teeth a dull sound was heard. In 38 cases of secondary retention no tilting of the neighbouring teeth was observed, while 36 cases displayed moderate tilting (Fig. 2C), and three cases severe tilting (Fig. 2B). Four molars did not have neighbouring teeth. Twenty-four retained molars were caries free, while the other 57 teeth studied were carious or restored.

In patients with one first retained molar, infraocclusion was more pronounced if the neighbouring teeth were tilted ($p<0.05$). Furthermore, a percussion sound suggestive for ankylosis, and tilting of neighbouring teeth, was more frequently observed in older patients. None of these relationships could be shown in patients with more than one secondary retained molar ($p>0.05$). Notwithstanding

the number of secondarily retained molars in one patient, the presence of caries or restorations was not dependent on the extent of infraocclusion.

Radiographic features. In all cases of secondary retention a decrease in the vertical dimension of the alveolar bone was observed. The margin of the alveolar bone followed the cemento-enamel junction of the retained molar (Fig. 3). Partial absence of the PDLS, with areas of apparent blending between the root and alveolar bone, was observed in 14 cases. External resorption of the root was noted in two molars (Fig. 3) and hypercementosis in one case. Curvature of the roots was noted in five molars.

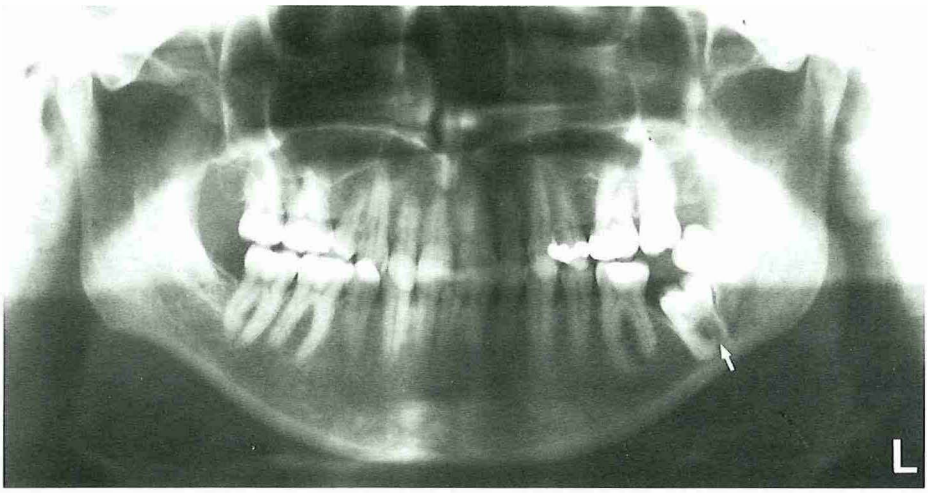


Fig. 3 Orthopantomogram of a 34-year-old female with secondary retention of the mandibular left second molar. The margin of the alveolar bone followed the cemento-enamel junction. The distal root of the retained molar shows signs resorption (arrow) which was proven after removal.

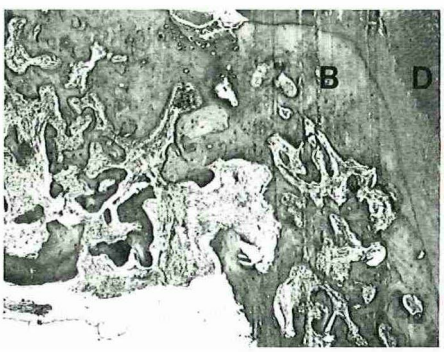


Fig. 4 Histological section of a secondary retained mandibular first molar. Bifurcation area. Dentin (D) in contact with bone (B). H-E, ×60.

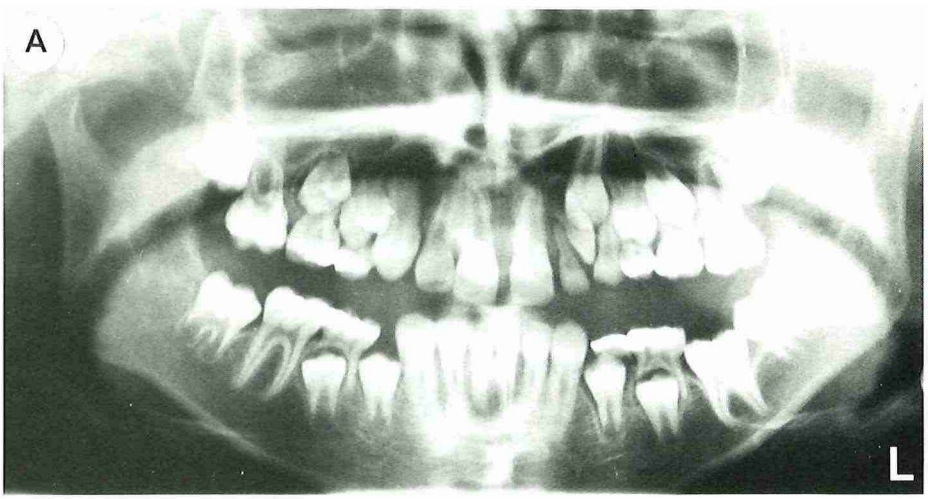
Table 2 Mean and standard deviation of infraocclusion in adolescents at their first visit (INF_0) and after six months (INF_6) in mm

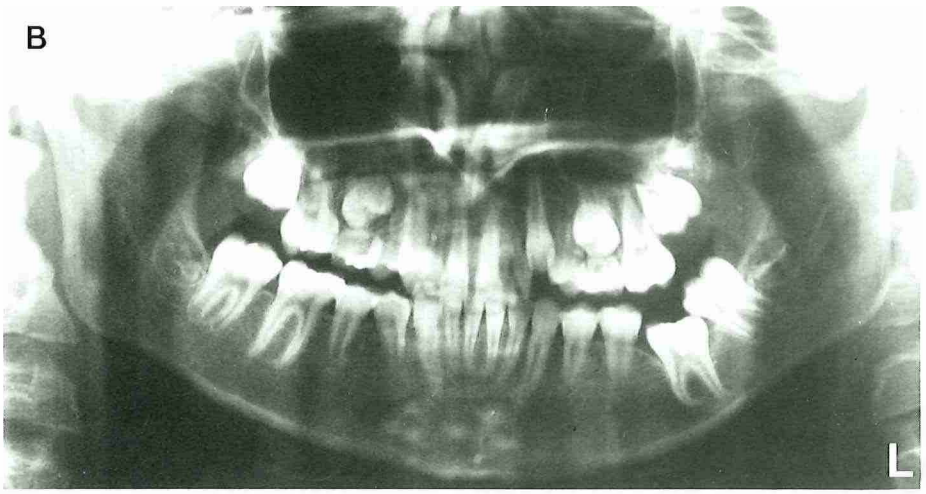
Number of retained molars in one patient	M1				M2			
	INF_0	INF_6	n	p	INF_0	INF_6	n	p
1	4.2±1.7	4.9±1.9	21	<0.05	4.3±1.9	4.6±1.7	3	NS
2	3.1±1.1	3.5±1.2	14	<0.05	4.0±0.8	4.3±0.9	2	NS
3	3.0	3.0	1	–	5.5±1.5	6.0±2.0	2	NS
4	3.3±1.6	3.3±1.6	7	NS	10.0	10.0	1	–
Total	3.7±1.6	4.1±1.8	43	–	5.1±2.3	5.4±2.3	8	–

NS: not significant

Histological features. Local areas of ankylosis were observed in all 38 secondarily retained molars. In 20 cases ankylosis was localized at the bifurcation and interradicular surface of the root only (Fig. 4). In 12 cases the bifurcation, interradicular and outer surface was involved and in the remaining 6 cases ankylosis was detected at the outer surface only. Hypercementosis was observed in 17 cases and was localized on the tip of the root. In the normal molars no histological indications for ankylosis were detected.

Follow-up. After a period of at least six months of observation, 38 molars were removed surgically. The teeth were removed because of increasing infraocclusion or





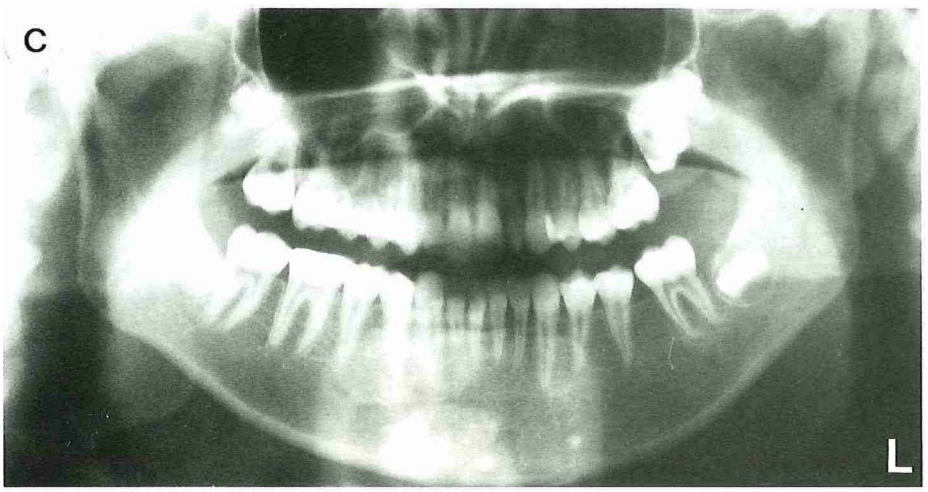


Fig. 5 A (see page 50) Orthopanthomogram of a 10-year-old girl with secondary retention of the mandibular left first molar. **B** Orthopantomogram of the same patient after 1 year. The infraocclusion had increased. **C** Orthopantomogram of the same patient after 3 years. The maxillary left second molar was now secondarily retained. The diastema in the mandible, due to the removal of the secondarily retained left first molar was closed by means of orthodontic treatment.

orthodontic or restorative treatment. An increase of the mean infraocclusion was observed in adolescents ($p<0.05$) (Table 2; Figs. 5A, 5B). In adults no increase of infraocclusion was observed ($p>0.05$). None of the retained molars showed eruptive movement. In the adolescents, two new cases of tilting were observed. No changes in percussion sounds and PDLS were observed in comparison with earlier visits. The external root resorption was progressive in one of the two cases.

Table 3 Relationship between percussion and radiological data in the removed secondarily retained molars. All molars showed ankylotic root areas

	absence of PDLS	presence of PDLS
solid, clear sound	8	3
dull sound	0	27

During the follow-up period of four years, six new cases of secondary retention were observed (Fig. 5C) in four patients (mean age 14.5 ± 1.7 years, median 14.0 years, range 13–17 years).

Comparison of clinical, radiographic and histological features. A decrease in the vertical dimension of the alveolar bone always accompanied an arrest of both the molar and the growth of the alveolar process in the affected area. Of the 16 molars with a solid sound, twelve showed partial absence of the PDLS suggestive of ankylosis. In contrast, of the 62 molars with a dull sound partial absence of the PDLS was observed in only two cases. Four molars with normal periodontal ligament had a solid, clear percussion sound. The extent of infraocclusion was not related to a partial absence of the PDLS or a solid, clear percussion sound ($p>0.05$). Table 3 shows an overview of the percussion sounds and radiographic findings (PDLS) in the 38 removed molars. When comparing the percussion and radiographic data with the histological data, the sensitivity and specificity of the percussion test and radiographs can be calculated. From the histological study it is apparent that ankylosis was present in all retained molars, so the FP is 0. A score suggestive for ankylosis on a percussion test or a radiograph was observed in 11 (TP=11, FN=27) and 8 molars (TP=8, FN=30), respectively. Thus the sensitivity of the percussion test and radiographs is 29% and 21%, respectively. The specifity of both diagnostic tools was 100%.

DISCUSSION

The eruptive process can be divided into six phases: three prefunctional stages of individual tooth eruption (follicular growth, pre-emergent eruptive spurt and postemergent eruptive spurt) and three postfunctional stages of the eruption of the entire dentition (juvenile occlusal equilibrium, circumpubertal occlusal eruptive spurt, and adult occlusal equilibrium).[17] In our opinion, the best term for the phenomenon whereby the eruption of a molar spontaneously stops during or after the postemergent eruptive spurt without any evidence of a physical barrier in the eruption path, is secondary retention. The term 'secondary' is added to indicate that the retention occurs secondarily to the postemergent eruptive spurt or a later phase of the eruptive process. Until the phase in which secondary retention occurs the eruptive process seems to be normal. By contrast, an impacted molar has often

never passed the pre-emergent eruptive spurt phase and remains unerupted in the jaw beyond the time at which it should normally be erupted.[6] The term submerged is a misnomer because it has the connotation of depression although the molar is not subjected to an actual apical movement but to a relative apical movement; the molar remains static while the neighbouring teeth continue to erupt.[12] The term ankylosis is not specifically used to refer to this disorder. In addition, using this term suggests ankylosis to be the cause of the disorder.

The etiopathogenesis of secondary retention is unknown, but ankylosis has been suggested as the main factor.[5,6,9,10,18,19] Other related factors reported in the literature are hypercementosis,[20,21] primary failure of eruption,[22] localized growth disturbance of the jaws,[3,23] and genetic factors.[7] The biological mechanism involved in the development of ankylosis in case of secondary retention has never been demonstrated, but a genetically determined or congenital gap in the periodontal ligament, a disturbance in the local metabolism, trauma or injury and tongue pressure during swallowing have been suggested.[5,8,12,25,26] A disturbance in the interaction between normal root resorption and hard tissue repair in deciduous molars has been suggested as a cause of ankylosis.[27] In permanent teeth, localized root resorption is a normal process.[28]

The histological diagnosis of ankylosis was supported by both clinical (percussion test) and radiographic (PDLS) observations in 11 cases only. Percussion tests seemed to be more accurate than radiographs. Andersson et al.[29] showed that a solid percussion sound can be recorded if over 20% of the root is ankylosed. Although we did not calculated the total area of the root which was ankylosed, we recorded a solid sound in cases of extreme ankylosis. Radiographic examination may be even less useful for the detection of ankylosis in multi-rooted teeth,[30,31] because small areas of ankylosis may not be visible because of overlapping structures. Thus, infraocclusion of a molar at an age when it normally would be in occlusion is the only reliable clinical criterion.

The observation that all removed secondarily retained molars are ankylotic has a significant clinical implication as molars with an abnormal periodontal ligament can not be treated orthodontically.[10,22] Based on an evaluation of treatment results[11] we recommend prompt removal of the affected molar in young patients, if necessary followed by orthodontic treatment of the neighbouring teeth to close the diastema. During the growth spurt no invasive treatment is indicated when infraocclusion is slight and non-progressive and the neighbouring teeth are not tilted, otherwise a similar treatment as for young patients is advisable. In adults, a build up of the retained molar to restore the occlusal height is a proper treatment in the case of slight infraocclusion as the extent of infraocclusion is relatively stable. Notwithstanding the age of the patient, molars with severe infraocclusion should be removed, because of the high incidence of tilting, carious destruction and periodontal disease of neighbouring teeth.

The age of onset of secondary retention is not clear. In our patient group new cases of secondary retention were observed between the ages of 13 and 17. This may indicate that the disorder develops between 6 (emergence of the first molar)

and 18 years (end of the adolescent growth spurt). Both in the literature and in our study the youngest patient was nine years old.[32] It appears that the later the onset, the less severe the infraocclusion is likely to be.

The increase of infraocclusion observed in adolescents may be explained by the eruption of the neighbouring teeth and the growth of the alveolar process during the growth spurt. In adults, no increase of infraocclusion was detected during the follow-up period.

From this study it is concluded that secondary retention of permanent molars seems to be associated with focal ankylosis and that percussion tests and radiographs are not reliable to exclude the presence of ankylosis. Infraocclusion of a permanent molar at an age that the tooth would normally be in occlusion is the only reliable criterion for diagnosing secondary retention. If there is any doubt about the diagnosis a follow-up of six months is recommended, especially in young patients. When observing infraocclusion of permanent molars without a solid percussion sound and without partial absence of the PDLS, one should keep in mind that there may be an underlying ankylotic process that may interfere with treatment.

REFERENCES

1 Ten Cate AR. Oral histology. Development, structure and function. 3rd ed. St Louis: CV Mosby, 1989:275–98.
2 Raghoebar GM, Boering G, Jansen HWB, Vissink A. Secondary retention of permanent molars: a histologic study. J Oral Pathol Med 1989;18:427–31.
3 Stoy PJ. Submerged permanent molars. The Dental Record 1951;71:12–4.
4 Korkhaus G. Die Halbretention des ersten Molaren. Dtsch Zahnärztl Z 1952;7:1205–12.
5 Biederman W. The incidence and etiology of tooth ankylosis. Am J Orthod 1956;42:921–6.
6 Pindborg JJ. Pathology of the dental hard tissues. Copenhagen: Munksgaard, 1970:241–52.
7 Bosker H, Ten Kate LP, Nijenhuis LE. Familial reinclusion of permanent molars. Clin Genet 1978;13:314–20.
8 Biederman W. Etiology and treatment of tooth ankylosis. Am J Orthod 1962;48:670–84.
9 Herman E. Evaluation and management of ankylosed teeth. NY State Dent J 1964;30:327–33.
10 Mitchell DL, West JD. Attempted orthodontic movement in the presence of suspected ankylosis. Am J Orthod 1975;68:404–11.
11 Raghoebar GM, Boering G, Booy C, Vissink A. Treatment of the retained permanent molar. J Oral Maxillofac Surg 1990;48:1033–8.
12 Biederman W. The problem of the ankylosed tooth. Dent Clin North Am 1968;12:409–24.
13 Kurol J. Infraocclusion of primary molars: an epidemiologic and familial study. Community Dent Oral Epidemiol 1981;9:94–102.
14 Shafer WG, Hine MK, Levy BM. A textbook of oral pathology. 4th ed. Philadelphia: WB Saunders, 1983:69, 335.
15 Andreasen JO. Traumatic injuries of the teeth. Copenhagen: Munksgaard, 1981:275.
16 Raghoebar GM, Van Koldam WA, Boering G. Spontaneous reeruption of a secondarily retained permanent molar and an unusual migration of a lower third molar. Am J Orthod Dentofacial Orthop 1990;97:82–4.
17 Steedle JR, Proffit WR. The pattern and control of eruptive tooth movements. Am J Orthod 1985;87:56–66.
18 Schulze Ch. Über Retention und Reinklusion (Depression) erster und zweiter Molaren. Dtsch Zahn Mund Kieferheilkd 1962;37:338–76.
19 Albers DD. Ankylosis of teeth in the developing dentition. Quintessence Int 1986;17:303–8.
20 Humerfelt A, Reitan K. Effects of hypercementosis on the movability of teeth during orthodontic treatment. Angle Orthod 1966;36:179–89.

21 Israel H. Early hypercementosis and arrested dental eruption: Heritable multiple ankylodontia. J Craniofac Genet Dev Biol 1984;4:243–6.
22 Proffit WR, Vig KWL. Primary failure of eruption; a possible cause of posterior open-bite. Am J Orthod 1981;80:173–90.
23 Gorlin RJ, Goldman HM. Thoma's oral pathology. 6th ed. St Louis: CV Mosby, 1970:153–4.
24 Adams D. An impacted permanent first molar. Oral Surg Oral Med Oral Pathol 1975;40:702–3.
25 Kapoor AK, Srivastava AB, Singh BP. Bilateral posterior open-bite. Oral Surg Oral Med Oral Pathol 1981;51:21–2.
26 Pytlik W. Das abwegige Verhalten der Zunge beim Schlucken als Ursache der Ankylose und Zahndurchbruchsbehinderung. Fortschr Kieferorthop 1982;43:380–93.
27 Dixon DA. Observations on submerged deciduous molars. The first Chapman prize essay. Dent Pract 1963;13:303–15.
28 Brown WAB. Resorption of permanent teeth. Br J Orthod 1982;9:212–20.
29 Andersson L, Blomlöf L, Lindskog S, Feiglin B, Hammarström L. Tooth ankylosis. Clinical, radiographic and histological assessments. Int J Oral Surg 1984;13:423–31.
30 Kloeppel JG. Die Infraposition einzelner Zähne des Milch- und bleibenden Gebisses. Dtsch Zahn Mund Kieferheilkd 1959;30:308–20.
31 Dayan D, Littner MM, Gonshorowitz J, Mersel A. Reimpaction of a first permanent maxillary molar due to an obscure idiopathic etiology. Clin Prevent Dent 1983;5:22–4.
32 Kessler W. Zur Frage der Häufigkeit von Nonokklusion von Milchmolaren und bleibenden Molaren. Stoma 1964;17:178–83.

5.2 SECONDARY RETENTION AS A POSSIBLE CAUSE OF IMPACTION OF PERMANENT MOLARS IN THE SAME DENTITION

ABSTRACT

Secondary retention refers to cessation of eruption of a tooth after emergence. Three cases of secondary retention of the first permanent molar and impaction of the adjacent second molar are reported. The clinical, radiographic and histological characteristics are described. In all three cases, the retained molars were in severe infraocclusion and they may represent the physical barrier for the impacted second molar. Early recognition of secondary retention by the family dentist is of great importance for a proper interceptive treatment as treatment of this disorder in children and adolescents is relatively simple. If secondary retention of permanent molars is not recognized before adulthood, often severe malocclusion has developed and removal of the affected molar is more difficult.

INTRODUCTION

Secondary retention refers to cessation of eruption of a tooth after emergence neither due to a physical barrier in the path of eruption nor as the result of an abnormal position.[1] Other terms commonly used for this phenomenon are ankylosis, submerging molar and reimpaction.[2-4] Clinically and radiographically, the tooth affected is in infraocclusion. As the neighbouring teeth and alveolar process continue their vertical development the involved molar gradually becomes more and more in infraocclusion. This may cause malocclusion, especially after tilting of the neighbouring teeth towards the diastema and over-eruption of the antagonist. The ultimate extent of malocclusion depends predominantly on the stage of development of the dentition and facial growth at the moment the retention occurs. The etiology of this disorder is not fully understood, but ankylosis seems to be a major factor.[5,6]

Successful treatment of secondary retention requires a multidisciplinary approach, including the dentist, the orthodontist, and the oral and maxillofacial surgeon. Early diagnosis is important for adequate treatment. In the case of severe infraocclusion of a retained molar that is not recognized early, there is a risk of jaw fracture when removing the tooth.[7]

This report describes three cases of secondary retention of the permanent first molar to illustrate some of the clinical problems. Particularly impaction of a second molar due to secondary retention of a first molar has, to the best of our knowledge, not been described before.

CASE REPORTS

Case 1

A 14-year-old girl was referred by her dentist for removal of the mandibular left third molar, because of referred pain in the mandible on the left side. Clinical examination revealed normal occlusion on the right side but severe malocclusion with

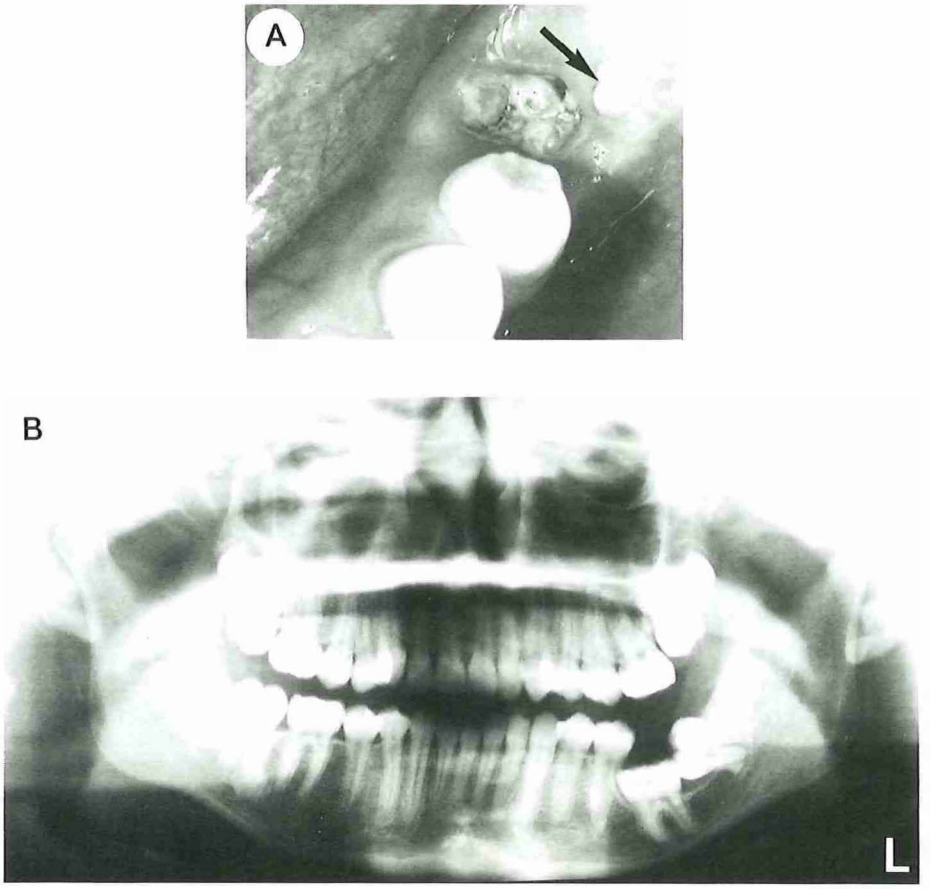


Fig. 1 A Clinical view of the mandible of a 14-year-old girl with secondary retention of the left first permanent molar. Note that only the distal surface of the impacted second molar is visible (arrow). **B** Orthopantomogram of the same patient showing secondary retention of the first molar and horizontal impaction of the second mandibular molar on the left side.

disruption of the plane of occlusion on the left side. The first mandibular left molar was in severe infraocclusion and its occlusal surface was deeply carious (Fig. 1A). Only the distal surface of the second molar was visible. A panoramic radiograph (Fig. 1B) revealed severe infraocclusion of the first molar and mesioangular impaction of the second molar on the left side. A decrease in the vertical dimension of the alveolar bone in that region was observed. The referred pain was not caused by her third molar but by deep caries in the retained first molar. This retained molar was surgically removed by a buccal approach after sectioning of the clinical crown and separation of the roots in the bifurcation. Histological examination showed the presence of local areas of ankylosis on the roots of the retained molar (Fig. 2). Orthodontic treatment was initiated to reposition the second molar and to allow the development of a proper occlusion.

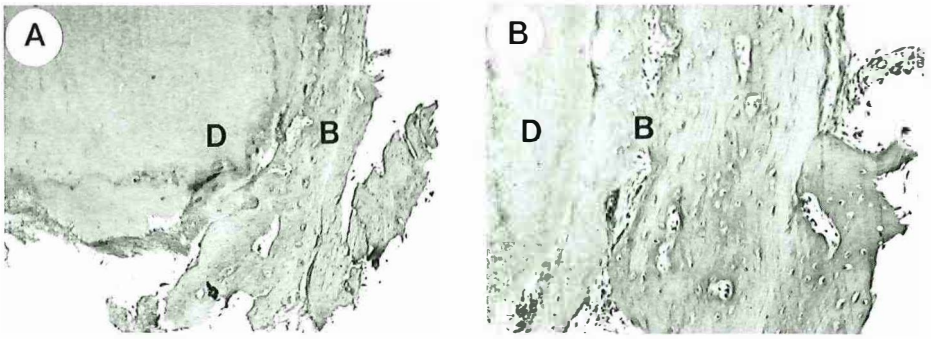


Fig. 2 A Histological section of the apical third of the root of the secondarily retained molar (Fig. 1). Dentin (D) is in direct contact with bone (B) H-E, ×45. **B** Detail of the apical third of the root. Dentin (D) is continuous with bone (B) H-E, ×100.

Case 2

A 36-year-old man was referred to our department complaining about pain in his lower jaw on the right side. Clinical and radiographic examination revealed a posterior open bite with severe infraocclusion of the mandibular first molar and maxillary second molar on the right side. The occlusal and buccal surface of the mandibular first molar were carious. The mandibular second molar was horizontally impacted (Fig. 3). Radiographs showed a decrease in vertical dimension of the alveolar bone in that region. The mandibular right first and second molar, the maxillary right second molar, and all four third molars were surgically removed under general anaesthesia. Histological examination revealed the presence of localized areas of ankylosis on the roots of the retained molars while the roots of the impacted second molar showed no signs of ankylosis.

Case 3

A 51-year-old man was referred by his dentist for removal of the second and third mandibular molars. He suffered from pain due to an abscess on the mandible on the right side. Regular dental care had been lacking for several years. Clinical and radiographic examination revealed malocclusion on the right mandibular side with infraocclusion of the first molar and impaction of the second and third molar (Fig. 4). On the radiographs a considerable alveolar bone deficiency was shown in that area. After abscess drainage the symptoms decreased. The first, second and third molars were surgically removed. A complicating factor was extensive ankylosis of the first molar. Histologically, ankylosis was confirmed. Partial dentures were made.

DISCUSSION

All three cases illustrate some of the serious clinical problems of late recognized of secondarily retained permanent molars such as aspecific pain, abscess formation

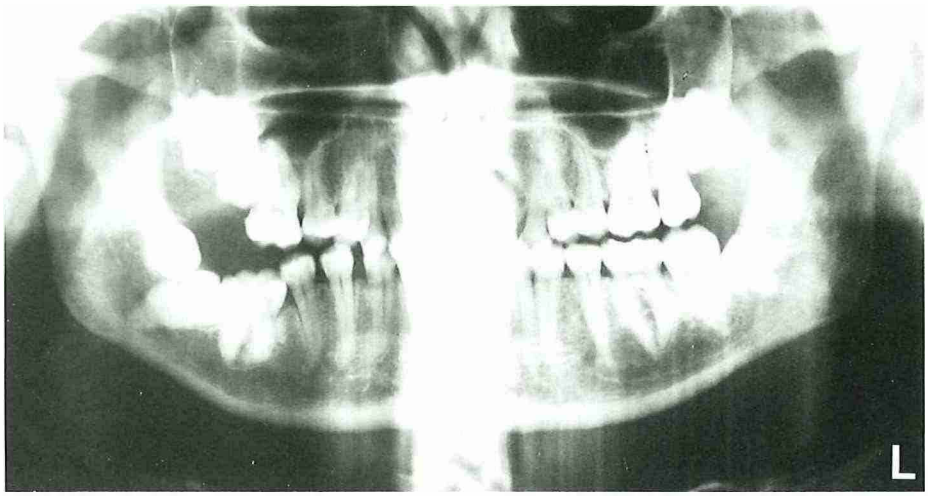


Fig. 3 Orthopantomogram of a 36-year-old man showing secondary retention of the mandibular right first molar and maxillary right second molar, and horizontal impaction of the mandibular right second molar.

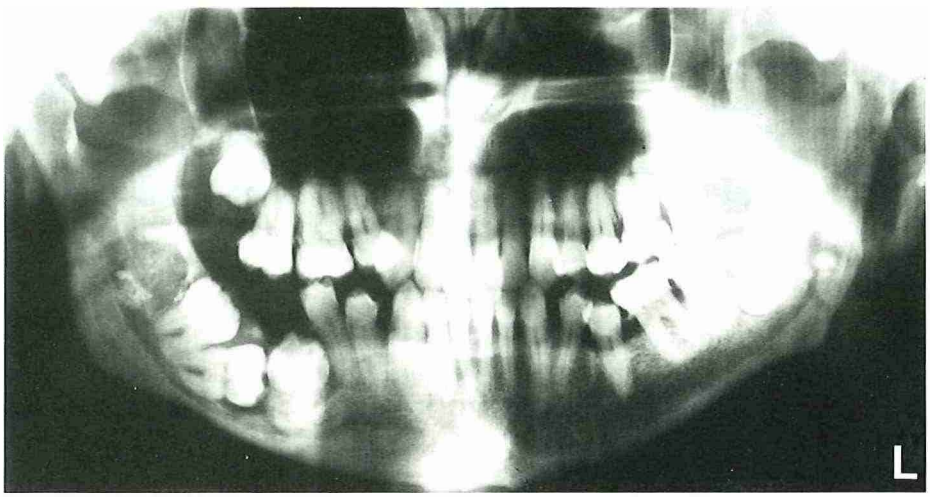


Fig. 4 Orthopantomogram of a 51-year-old man showing secondary retention of the mandibular right first molar and horizontal impaction of the second molar. The vertical dimension of the alveolar bone at the spot of the retained molar is decreased.

and impaction of adjacent molars. None of the subjects had a history of apparent medical problems and all considered themselves to be in good health.

Impaction is cessation of eruption due to a physical barrier in the eruption path.[8] Normally the occlusal surfaces of the mandibular molars are first mesially inclined and during the growth of the jaws the crowns gradually move into an upright position. Severe infraocclusion of the first molar can block the eruption of the second

molar, thus leading to its impaction.

The biological mechanism involving secondary retention is unknown. Ankylosis as a result of a defect in the periodontal ligament, trauma, or local disturbances in metabolism is generally accepted as the main factor.[2,4] It is apparent that some defect, failure, or alteration of the periodontal ligament must precede ankylosis, because ankylosis will not occur in case of a normal periodontal ligament.[6]

The most reliable criterion to diagnose secondary retention is infraocclusion at an age when this tooth would normally be in occlusion. The diagnosis is dependent on routine clinical periodic examination by the family dentist to notice the cessation of eruption of the affected molar.[9]

Secondary retained molars do not respond to orthodontic treatment, because of an abnormal periodontal ligament.[10] Luxation of secondarily retained molars to break the ankylotic area and to allow further eruption or orthodontic movement has been reported.[2] In a histological study of secondarily retained permanent molars, ankylosis was observed in all cases and in 81% in the interradicular and bifurcation areas.[6] Thus, luxation of these molars will not easily result in a disturbance of the bony connections in these areas because the rotation point during luxating is the bifurcation.[11] Furthermore, luxation may promote ankylosis rather than to eliminate the ankylotic area.

Treatment of secondary retention of permanent molars is based on the condition of the tooth, the jaw relationship and occlusion, the dental age of the patient at the time of diagnosis, and the patient's attitude about undergoing treatment.[11] Before the growth spurt starts, secondarily retained molars have to be removed after orthodontic consultation for closure of the diastema. During the growth spurt, no invasive treatment is indicated when infraocclusion is slight and nonprogressive, and the neighbouring teeth are not tilted; otherwise, the molar has usually to be removed. In adult patients, in case of slight infraocclusion a build up of the retained molar to restore the occlusal height is the proper treatment. In other cases removal is indicated.[11] The proper time for treatment of impacted molars is early in the adolescence, generally in the 11- to 14-year range.[12] This treatment usually consist of surgical and/or orthodontic uprighting of the impacted molar.[12,13]

Early detection of secondary retention followed by timely treatment greatly minimize the progressive negative effects of this dental condition such as the development of severe malocclusion and impaction of the neighbouring molar. Eruption disturbances of permanent molars must not be considered as a rare curiosity, but should be a substantial component of the dentist factual knowledge.

REFERENCES

1 Raghoebar GM, Van Koldam WA, Boering G. Spontaneous reeruption of a secondarily retained permanent lower molar and an unusual migration of a lower third molar. Am J Orthod Dentofacial Orthop 1990;97:82–4.

2 Biederman W. Etiology and treatment of tooth ankylosis. Am J Orthod 1962;48:670–84.

3 Sterrett DS. Report of a case of a submerged first molar. Am J Orthod Oral Surg 1940;26:681–3.

4 Pindborg JJ. Pathology of the dental hard tissues, Copenhagen: Munksgaard, 1970:248–52.

5 Korkhaus G. Die Halbretention des ersten Molaren. Dtsch Zahnärztl Z 1952;17:1205–12.
6 Raghoebar GM, Boering G, Jansen HWB, Vissink A. Secondary retention of permanent molars: a histologic study. J Oral Pathol Med 1989;18:427–31.
7 Adams D. An impacted permanent first molar. Oral Surg Oral Med Oral Pathol 1975;40:702–3.
8 Shafer WG, Hine MK, Levy BM. A texbook of oral pathology, 4th ed. Philadelphia: WB Saunders, 1983:66–9.
9 Albers DD. Ankylosis of teeth in the developing dentition. Quintessence Int 1986;17:303–8.
10 Proffit WR, Vig KWL. Primary failure of eruption: a possible cause of posterior open-bite. Am J Orthod 1981;80:173–90.
11 Raghoebar GM, Boering G, Booy C, Vissink A: Treatment of the retained permanent molar. J Oral Maxillofac Surg 1990;48:1033–8.
12 Johnson JV, Quirk GP. Surgical repositioning of impacted mandibular second molar teeth. Am J Orthod Dentofac Orthop 1987;91:242–51.
13 Wellfelt B, Varpio M. Disturbed eruption of the permanent lower second molar: treatment and results. J Dent Child 1988;55:183–9.

5.3 SPONTANEOUS RE-ERUPTION OF A SECONDARILY RETAINED PERMANENT MOLAR AND AN UNUSUAL MIGRATION OF A LOWER THIRD MOLAR

ABSTRACT

Secondary retention refers to cessation of eruption of a tooth after its emergence. It occurs mainly in the deciduous dentition, but it is also observed in the permanent dentition. Although ankylosis has been suggested as the main etiologic factor, the exact biologic mechanism of secondary retention is not yet known. This paper presents a case of spontaneous re-eruption of a secondarily retained permanent molar, a phenomenon which is very rare. It also describes an unusual migration of a lower third molar in the same patient.

INTRODUCTION

Secondary retention refers to the cessation of eruption of a tooth after emergence neither due to a physical barrier in the path of eruption nor as a result of an abnormal position. As the adjacent teeth continue to erupt vertically, the involved tooth comes more and more into infraocclusion. This disturbance in eruption is also known in the literature as submerging, ankylosis, reimpaction, and reinclusion.[1-6] In contrast to secondary retention, primary retention is characterized by absence of any eruption tendency.

Eruption may also be disturbed because of interference by another tooth or tissue. This is called impaction. Primary retention or impaction is seen mostly in lower third molars and upper canines. Secondary retention of molars is more often seen in the deciduous dentition, then in the permanent dentition. It is generally accepted that a secondarily retained permanent tooth will not re-erupt. The involved tooth also never seems to respond normally to orthodontic forces.[1,2]

In the case report that follows spontaneous re-eruption of a secondarily retained molar is described. In addition, an unusual migration of a lower third molar in the same patient is shown.

CASE REPORT

In August 1971, a 20-year-old male was referred by his dentist to the Department of Orthodontics, University of Groningen, for evaluation of the abnormal position of the lower left first permanent molar. The patient had no complaints, and his dentist had restored the tooth 2 weeks earlier. There had been no trauma or inflammation involving the tooth.

The medical history was unremarkable, and no other family members had secondarily retained teeth.

Clinical examination showed infraocclusion of the relevant molar of about 4 mm compared to the fully erupted neighbouring teeth (Fig. 1). Responses to pulp

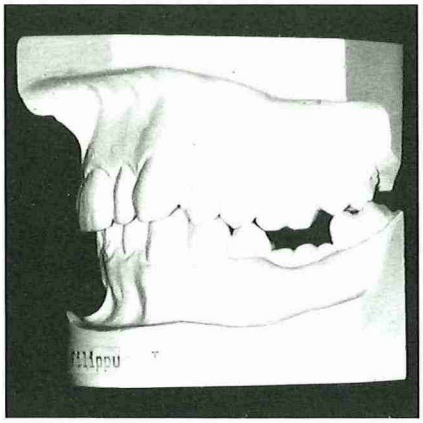


Fig. 1 Cast model of a 20-year-old male patient. The lower left molar is in infraocclusion and has no occlusal contact.

vitality tests were positive, and there were occlusal attrition marks. None of the third molars was visible on inspection.

The orthopantomogram showed distinct infraocclusion of the left first molar in the mandible, in combination with underdevelopment in height of the alveolar crest in this area (Fig. 2). The periodontal ligament space around the roots of the first molar seemed to be normal. No signs of ankylosis or root resorption could be seen. All four third molars were unerupted and showed a vertical position.

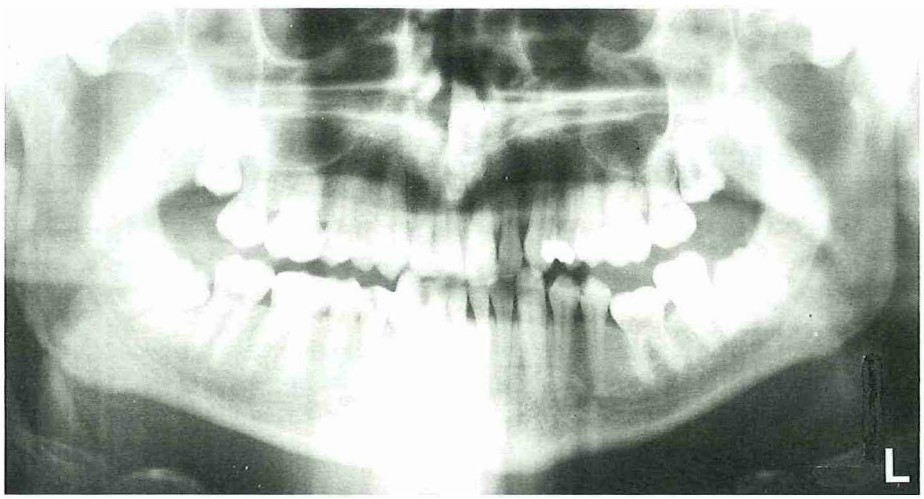


Fig. 2 Orthopantomogram of patient presented in Fig. 1. The infraocclusion of the lower left first molar is clearly visible. The lower right third lower molar has an upright position.

The diagnosis was secondary retention of the lower left first molar. It was expected that orthodontic treatment of a secondarily retained molar would not be successful. Therefore it was decided to leave it undisturbed. At the request of the patient, the impacted third molars were not removed.

In May 1988, (i.e., 17 years after the first consultation) the patient was referred to the Department of Oral and Maxillofacial Surgery of the Groningen University Hospital for consultation regarding the lower third molars. To our surprise, the secondarily retained molar had re-erupted spontaneously into normal occlusion with the opposing teeth (Fig. 3). The orthopantomogram showed a normally erupted molar with normal periodontal ligament space and an undisturbed occlusal curve (Fig. 4). The alveolar process had reached normal height. Tracings of the mandible, made on lateral head plates and superimposed on the external controls, also showed a complete re-eruption of the lower molar (Fig. 5). The most recent orthopantomogram showed that the right mandibular third molar, which initially had stood in an upright position, was now completely horizontally impacted. The crown was not in contact with the distal surface of the second molar. Clinically, there was no communication between the third molar and the oral cavity.

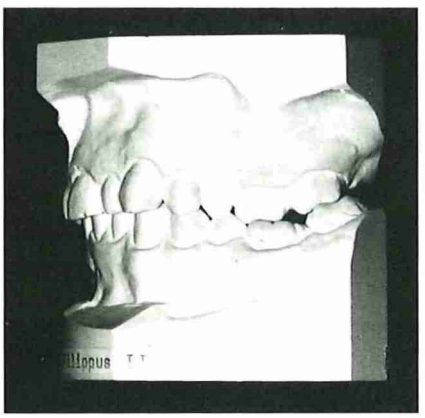


Fig. 3 Cast model of the dentition of same patient, now 37 years of age. The lower left first molar is in occlusal contact with its antagonist after 17 years of follow-up.

DISCUSSION

The cause of secondary retention remains unclear. This condition of failure or reduction of the eruptive force of a molar seems to be the result of abnormalities in the periodontal ligament. Localized ankylosis resulting from a local defect in the periodontal ligament is held responsible for this disturbance, but the real cause is not fully understood. In some cases trauma, infection, local disturbance of metabolism, or a genetic influence has been considered.[1,6] Histological investigations have shown that in secondary retention, ankylosis is often limited to a few

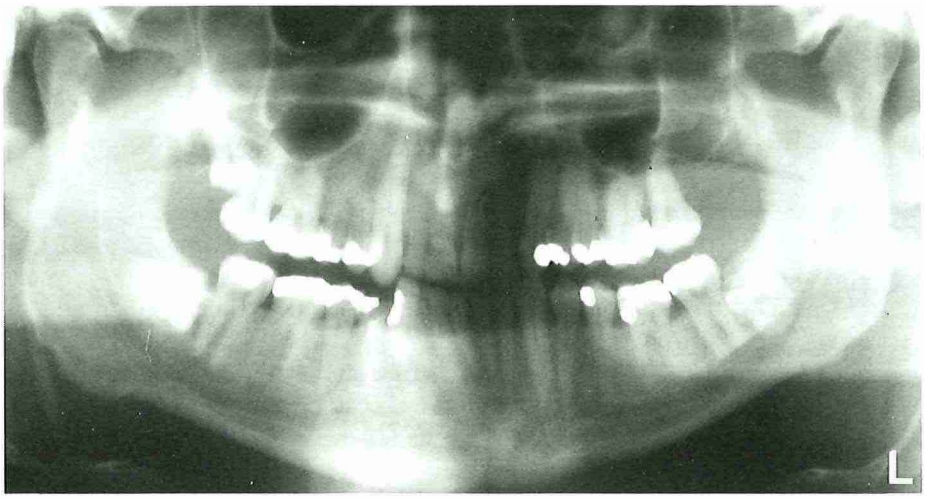


Fig. 4 Orthopantomogram of patient presented in Fig. 3 showing normal eruption of the lower left first molar. The lower right third molar is now lying horizontally without contacting the second molar.

small areas of the root surface. This could imply that, because of a constant bone remodelling of the alveolar process, this bony union could be resorbed. That would lead to release of the secondarily retained molar, and it could again reach its normal occlusal level in contact with its antagonist, probably because of the continuous eruption process that normally takes place when no contact with an antagonist exists. Another possible cause is a primary failure of eruption. Nonankylosed teeth can fail to erupt fully or partially because of malfunction of the eruption mechanism, probably caused by some disturbance in metabolism or blood flow changes in the periodontal ligament.[7] In our case, there was no evidence of that.

Re-eruption of secondarily retained deciduous molars has been reported previously.[8] These molars were released as a result of further resorption and resumed their occlusal movement, returning to the normal occlusal level before they were shed.

In the present case, re-eruption was probably favored by the absence of tilting of the neighbouring teeth. The possible role of the poorly distally extended occlusal restoration in the first molar (Fig. 2) merits some special consideration. This amalgam restoration was made 2 weeks before the orthopantomogram was taken. Since the first molar was in infraocclusion at that time the restoration cannot have been an etiologic factor. What possible role correction of the restoration shortly after the first orthopantomogram might have played in the re-eruption (Fig. 4) is not clear.

The local growth disturbance of the alveolar process is to be considered a secondary phenomenon.

This case report demonstrates that spontaneous re-eruption of a secondarily retained molar is possible. This is in accordance with the theory that secondary retention is caused by local spots of ankylosis that can be resorbed during the normal

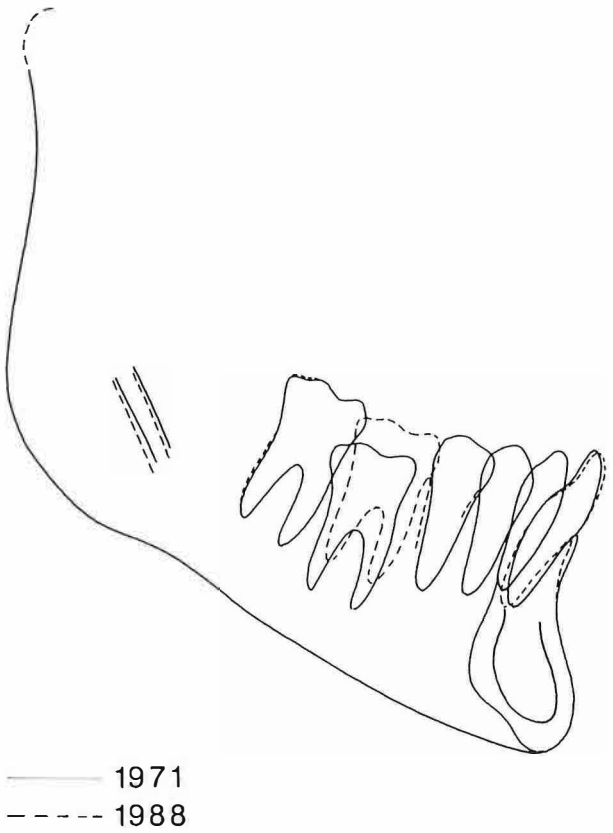


Fig. 5 Superimposed cephalometric tracings of the mandible showing re-eruption of lower molar.

remodelling process.

To the best of our knowledge, no previous report of re-eruption of a permanent molar has ever been published. The tilting of a lower third molar that initially showed an upright position is also a rare phenomenon. We have no explanation for this unusual phenomenon.

REFERENCES

1 Biederman W. The incidence and etiology of tooth ankylosis. Am J Orthod 1956;42:921–6.
2 Mitchell DL, West JD. Attempted orthodontic movement in the presence of suspected ankylosis. Am J Orthod 1975;68:404–11.
3 Stoy PJ. Submerged permanent molars. Dent Record 1951;71:12–4.
4 Pindborg JJ. Pathology of the dental hard tissues. Copenhagen: Munksgaard, 1970:248–52.
5 Dayan D, Littner MM, Gonshorowitz J, Mersel A. Reimpaction of a first permanent maxillary molar due to an obscure idiopathic etiology. Clin Prevent Dent 1983;5:22–4.
6 Bosker H, Ten Kate LP, Nijenhuis LE. Familial reinclusion of permanent molars. Clin Genet 1978;13:314–20.

7 Proffit WR, Vig KWL. Primary failure of eruption: a possible cause of posterior open-bite. Am J Orthod 1981;80:173–90.
8 Darling AI, Levers BGH. Submerged human deciduous molars and ankylosis. Arch Oral Biol 1973;18:1021–40.

CHAPTER 6

SECONDARY RETENTION OF PERMANENT MOLARS: HEREDITARY FACTORS

This chapter has been submitted to the Journal of Craniofacial Genetics and Developmental Biology.

ABSTRACT

Secondary retention is cessation of the eruption of a tooth after emergence neither due to a physical barrier in the path of eruption nor as a result of an abnormal position. The etiopathogenesis of secondary retention is not fully understood, but heredity is involved in at least some cases. In this study first degree relatives of 52 patients with secondary retention of permanent molars were screened for the presence of the same phenomenon in their dentition. Familial occurrence could be shown in five families. The pedigrees are compatible with autosomal dominant inheritance. HLA phenotypes and bloodgroups ABO, rhesus and P_1 were studied in two families. The lod scores for linkage with secondary retention were added to previously reported information. The lod score for linkage for bloodgroup system P increased from +0.940 to +1.475 at a recombination fraction of 5%. We conclude that secondary retention of permanent molars is an etiologically heterogeneous condition in which some cases are caused by the presence of an abnormal autosomal dominant gene.

INTRODUCTION

Secondary retention is the cessation of eruption of a tooth after emergence neither due to a physical barrier in the path of eruption nor as a result of an abnormal position.[1] This disturbance in eruption is also described in the literature as submerging, ankylosis, Halbretention, infraocclusion and reinclusion.[2-5] We prefer the description secondary retention, because the retention occurs secondarily to the postemergent eruptive spurt of a later phase of the eruptive process. Permanent molars are less frequently affected than deciduous molars.[2] The major characteristic of a secondarily retained molar is infraocclusion. This may result in malocclusion because of tilting of the neighbouring teeth and over-eruption of antagonists. The disorder can also result in loss of the retained molar and neighbouring teeth due to caries and periodontal disease, and in deformation of the facial skeleton.[6] Secondary retention has occasionally been described in combination with a posterior open-bite, reduction in the height of the lower face, mandibular prognathism, and bilateral clinodactyly of the fifth fingers.[7]

The etiopathogenesis of secondary retention is not fully understood. Local areas of ankylosis on the root surfaces have been suggested to be a significant factor.[1] Other proposed factors are trauma, local infection, excessive masticatory pressure, disturbed local metabolism, disturbed root resorption and hard tissue repair, and a congenital defect in the periodontal ligament.[1,2] None of these suggestions is supported by clear data. Familial occurrence in secondary retention of deciduous[4,5] and permanent[8-13] molars has been described. In a linkage study of 5 pedigrees with secondary retention of permanent molars using blood- and serum groups as genetic markers, bloodgroup system P was suggested to be linked to the secondary retention gene,[6] but in a second report by the same group the lod scores were too low to corroborate the finding.[3] In the latter study it was concluded that secondary retention in the permanent dentition is inherited as an autosomal dominant disorder with vertical transmission and no skipping of generations. In deciduous molars

it has been suggested that secondary retention is inherited on a multifactorial, polygenic basis.[5]

An hereditary component in secondary retention would imply the necessity of examinating the patient's family in order to diagnose and treat this eruption disorder as early as possible to prevent complications such as malocclusion. Here we describe the results of a study of familial occurrence of secondary retention among first degree relatives of 52 patients with secondary retention of permanent molars and report five new families compatible with autosomal dominant inheritance. Bloodgroups ABO, rhesus and P_1 and HLA phenotypes were determined in two families.

MATERIAL AND METHODS

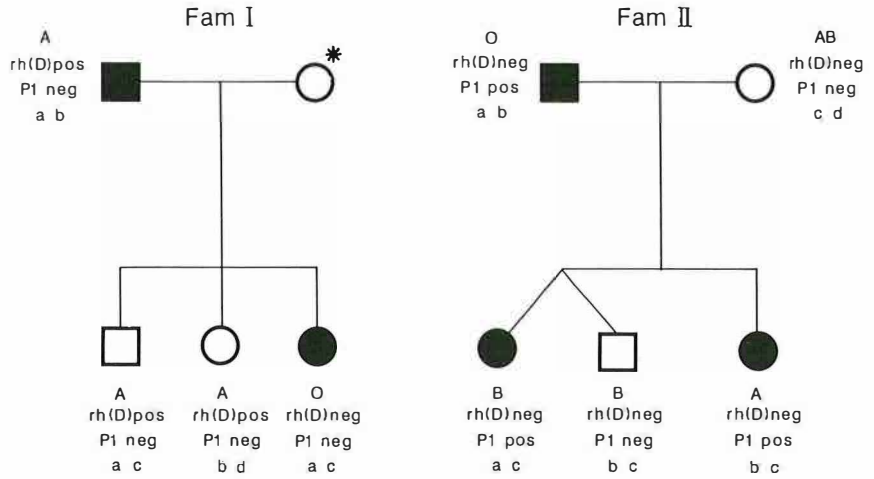
Definition. A molar was considered to be secondarily retained if the occlusal surface was 1 mm or more below the occlusal level of the other teeth in the relevant dental arch at an age at which the tooth normally would have been in occlusion. The molars included in this study were retained without inclination and had enough space in the arch to erupt.

Patients. During a five year period, 52 patients (mean age 18.2 years) with secondary retention of permanent molars were referred to the Department of Oral and Maxillofacial Surgery of the University Hospital by their dentist, orthodontist, or oral and maxillofacial surgeon. All subjects considered themselves in good general health. The parents, brothers and sisters of these 52 patients were screened for the presence of secondary retention in their dentition. The families were clinically examined and radiographs were taken only in cases of secondary retention. A family was characterized as positive if secondary retention was proven in the dental histories of at least one of the first degree relatives. The distribution and number of the affected molars was documented. Other dental anomalies such as enamel defects, aplasia, impaction, and root shortening were noted. Complete dental and family histories were obtained in 39 families (39 fathers, 39 mothers and 78 sibs). The data of the other 13 families (4 fathers, 9 mothers and 11 sibs) were not included in the results because of incomplete dental or family histories. In these families no familial occurrence was observed.

Linkage study. In two families, 20 ml of venous blood was collected in heparinized test tubes from each subject for linkage analysis involving HLA A, B, and C phenotypes and bloodgroups ABO, rhesus and P_1. The other 3 families did not consent to the procedure.

RESULTS

Frequency of familial occurrence. Familial occurrence was observed in five families. In all positive families one of the parents has secondary retention. Three mothers,



a) A1 B12(44) Bw4 DR7 DRw53 DQw2
b) Aw19(29) B5(51) Bw4 DR3 DRw52
c) A3 B7 Bw6 DR2 DQw1
d) Aw19(w33) B14 Bw6 –

a) Aw19(32) B40(w61) Bw6 Cw2 DR5 DRw52 DQw3
b) Aw19(33) B12(44) Bw4 DR7 DRw53 DQw2
c) Aw9 (24) B18 Bw6 DR3 DRw52 DQw2
d) A1 B8 Bw6 –

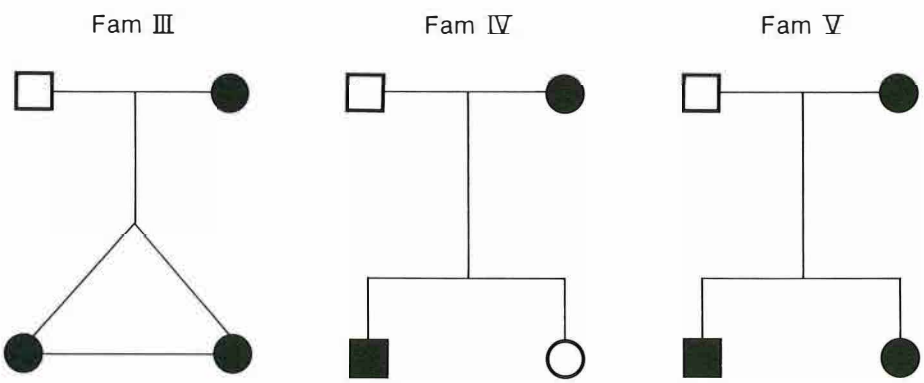


Fig. 1 Pedigrees of the five families with secondary retention. In two families HLA phenotypes and bloodgroups ABO, rhesus and P_1 were investigated. In the case of asterik no blood was available.

2 fathers and 3 out of 7 sibs, one being an identical twin sister, were affected. The pedigrees of the five families are shown in Fig. 1, and the distribution of the retained molars in the affected family members is given in Table 1. Female to female, female to male and male to female transmission were observed. The frequency of families with parental occurrence in this study is 13% (5 out of 39 families).

Table 1 Distribution of secondary retention of deciduous and permanent molars in five families

		Mandible		Maxilla	
		M1	M2	M1	M2
Family I	Father	2		1	
	Daugther	1	1		1
Family II	Father				1
	Daughter	1		2	
	Daughter	1			
Family III	Mother		1		
	Daughter	1			1*
	Daughter		1*	1	1*
Family IV	Mother			1	
	Son	2			2*
Family V	Mother			2	
	Daughter	1	1*	1	1*
	Son		1		

* deciduous molar

Description of families. **Family I.** The father and one daughter both had three secondarily retained molars, two in the mandible and one in the maxilla (Figs. 2, 3). One of the affected teeth of the daughter was a deep seated second molar with an amalgam restoration. She also had an impacted maxillary canine on the right side (Fig. 2B). In the past the fathers secondarily retained first molars of the mandible on the left side and of the maxilla on the right side had been removed (Figs. 3A, B). Two sibs were unaffected.

Family II. The father and two children were affected. The fathers maxillary second molar was secondarily retained, while both daughters had secondarily retained mandibular first molars. One of the affected daughters, part of a non identical twin, also showed two retained maxillary molars. Her twin brother was unaffected.

Family III. The mother and an identical female twin were affected. The twin showed secondary retention in the deciduous and permanent dentition (Fig. 4). One twin had secondary retention of the maxillary second deciduous molar and mandibular first permanent molar on the left side (Fig. 4A). Her sister showed secondary retention of the mandibular left second deciduous molar and the maxillary second deciduous molar and first permanent molar on the right side (Fig. 4B). The mother had a secondarily retained left mandibular second permanent molar.

Family IV. The mother and a son were affected. The mother had secondary retention of the maxillary left first molar (Fig. 5A). The son showed secondarily

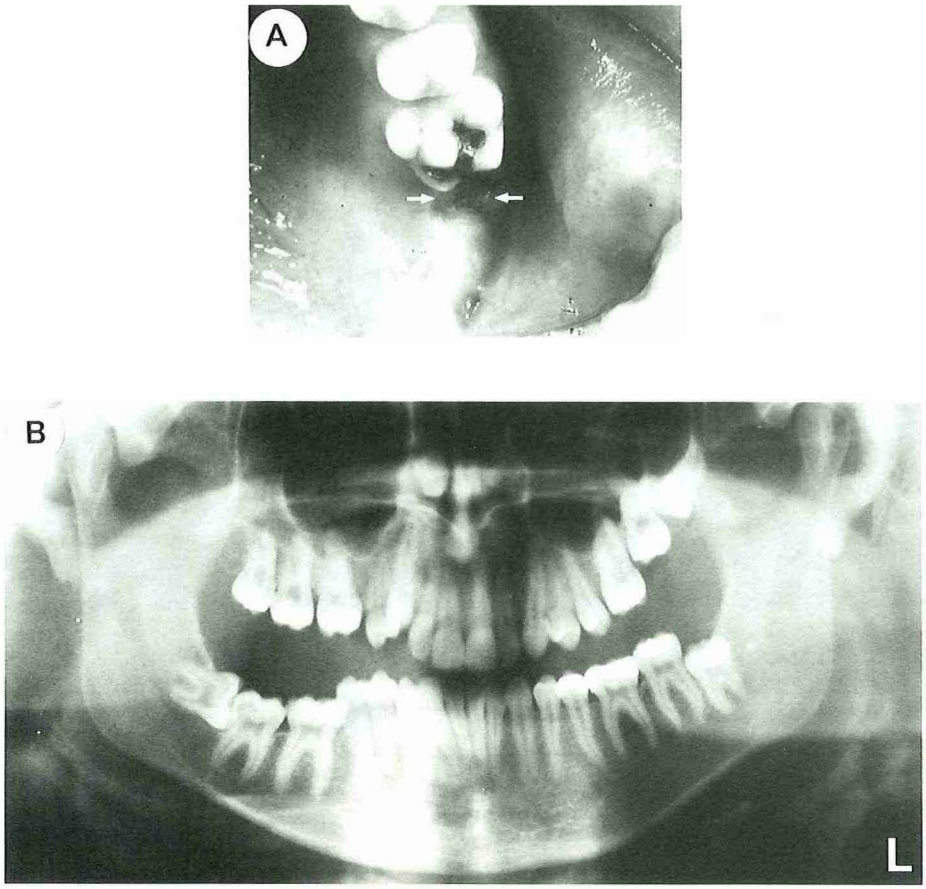


Fig. 2 A Intraoral view of a 15-year-old girl with secondary retention of the maxillary left second molar (arrows). **B** Orthopantomogram of the same patient showing severe infraocclusion of the maxillary left second molar. The mandibular first and second molar on the right side are also affected. Note the amalgam restoration in the maxillary left second molar indicating that the molar had been emerged into the oral cavity.

retained maxillary second deciduous molars (Fig. 5B). During a follow-up period of 4 years his mandibular left and right first permanent molar also became secondarily retained (Fig. 5C). A female sib was unaffected.

Family V. The mother, a son and a daughter were affected. The mother had secondary retention of the maxillary first molars. The daughter had secondary retention of the mandibular and maxillary second deciduous molars on the left side. The mandibular right first permanent molar was also affected. During a follow-up period of 5 years the maxillary left first molar also became affected. The son had secondary retention of the mandibular right second permanent molar.

Linkage studies. Results of bloodgroup and HLA studies are shown in Figure 1 (pedigree 1 and 2). Lod scores are shown in Table 2. They represent the combined scores of a previous report[3] and the present study. HLA phenotypes have not been

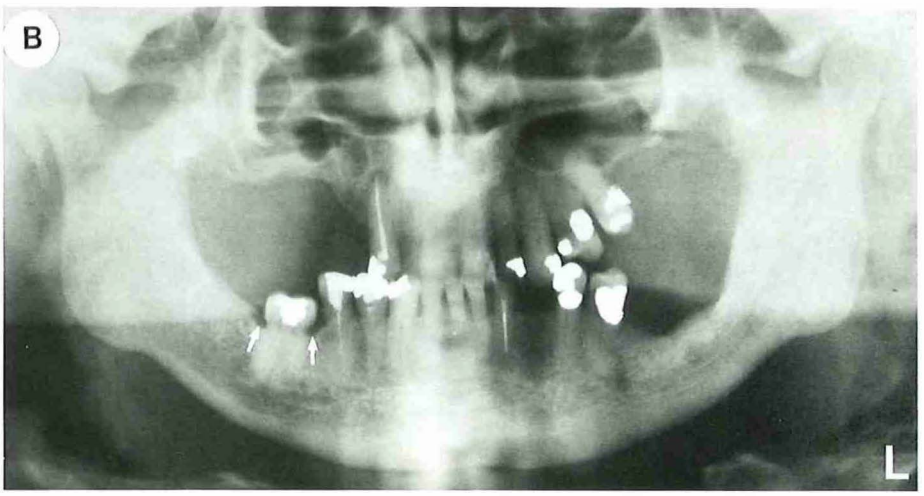


Fig. 3 A Intraoral view of her 52-year-old father with secondary retention of the right mandibular first molar. **B** Orthopantomogram of the father. Note the decreased margin of the alveolar bone in the area of the affected first molar (arrow).

reported on before. The lod score of bloodgroup system P increased from +0.940 to +1.475 at $\theta=0.05$.

DISCUSSION

This report confirms the presence of familial occurrence in secondary retention of permanent molars. The observation that in all familial cases one of the parents of the patient was affected, agrees with the suggested autosomal dominant inheritance pattern of the disorder.[3,4,7] One monozygotic twin was affected. Of the remaining 6 sibs, two were affected. No male to male inheritance was observed in this small sample. In an earlier study male to male transmission was observed 18 times.[3]

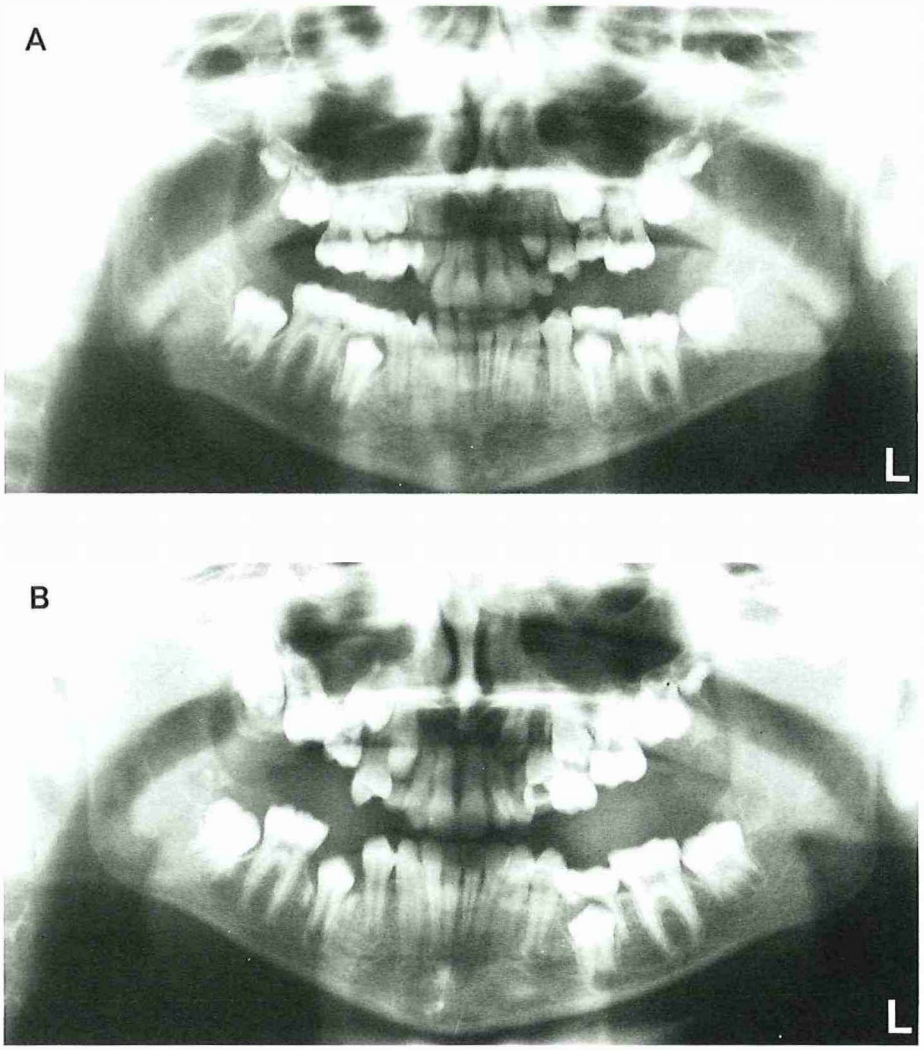


Fig. 4 A Orthopantomogram of an 11-year-old twin showing secondary retention of the maxillary second deciduous molar and mandibular first permanent molar on the left side. **B** Orthopantomogram of the other twin showing secondary retention of the mandibular left second deciduous molar, and the maxillary second deciduous molar and first permanent molar on the right side.

The phenotype distribution of bloodgroup system P in one family is compatible with linkage of bloodgroup P_1 gene with the autosomal dominant secondary retention gene, although the evidence still is inconclusive (a lod score of less than 3 is not regarded as convincing evidence). All other markers studied so far have negative lod scores.

It seems that the occurrence of secondary retention in the deciduous dentition may predispose to a similar disorder in the permanent dentition.[14] We postulate,

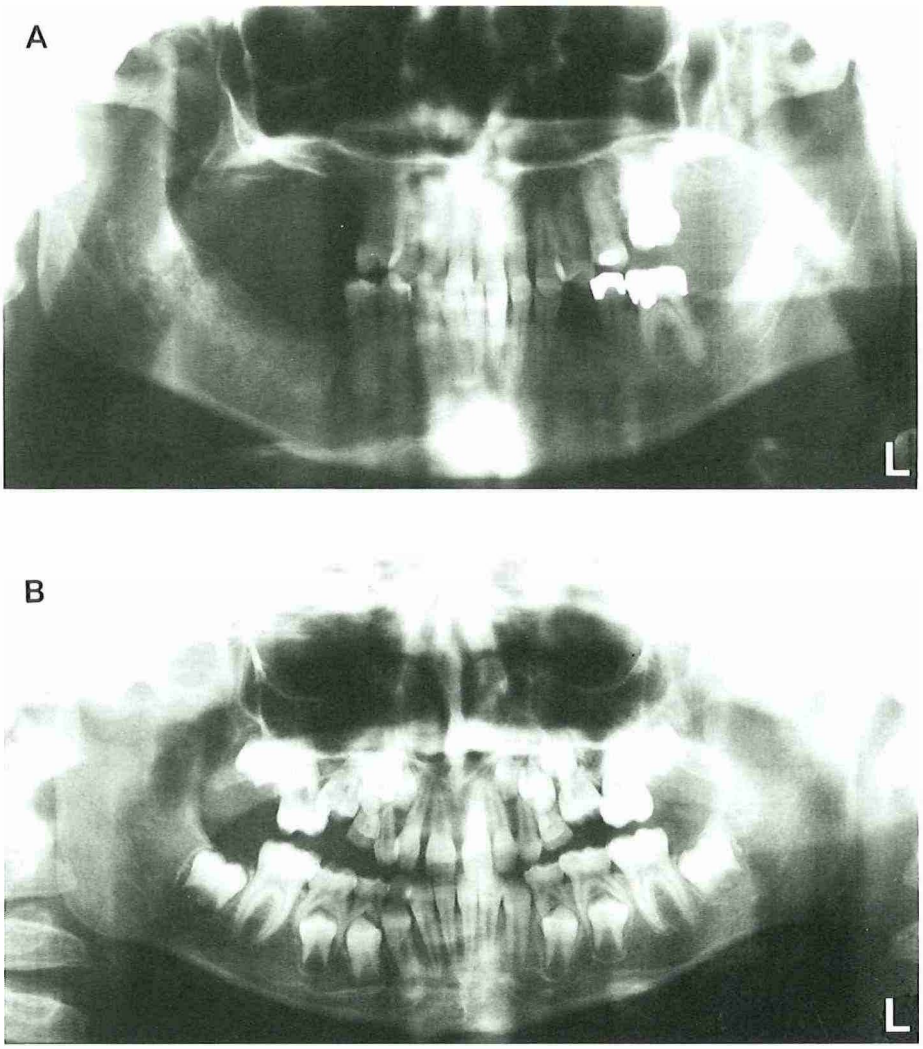


Fig. 5 A Orthopantomogram of the mother showing secondary retention of the maxillary left first molar. **B** Orthopantomogram of her 10-year-old son showing secondary retention of the maxillary second deciduous molars.

because secondary retention was observed in deciduous and permanent molars in several of our patients, that both phenomena can be pleiomorphic manifestations of the same gene. The most remarkable feature of a case report of a monozygotic twin with secondary retention was the nearly identical pattern in the distribution and severity of infraocclusion of the affected molars.[15] The twins in our study did not show the same concordance of features. Affected members of the same family showed much variability as to which molars were affected, indicating that variability of expression is the rule in hereditary secondary retention of permanent

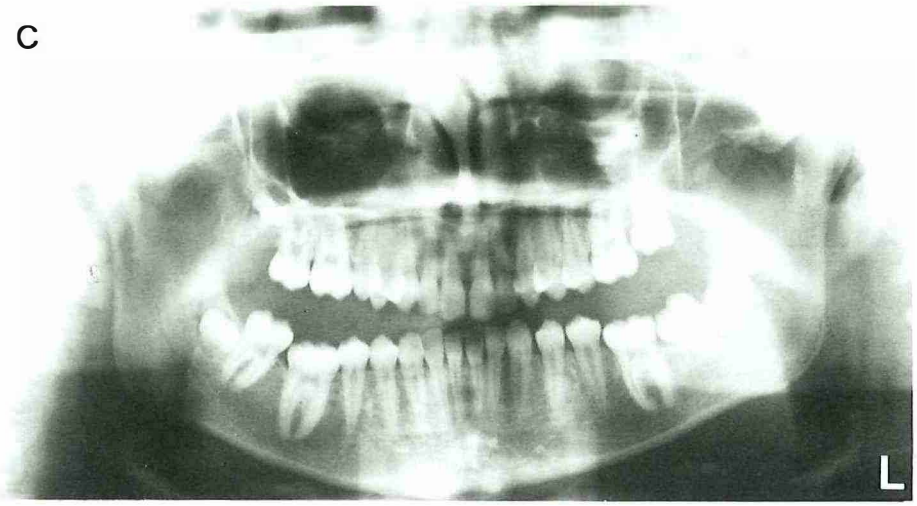


Fig. 5 C Orthopantomogram of the same patient after 4 year showing secondary retention of the mandibular right and left first permanent molar.

Table 2 Updated lod scores for linkage with selected bloodgroups and HLA phenotypes.*

Marker	Recombination fraction (θ)					
	0.00	0.05	0.10	0.20	0.30	0.40
ABO	$-\infty$	− 1.888	− 1.160	− 0.550	− 0.236	− 0.064
P_1	$-\infty$	+ 1.475	+ 1.459	+ 1.095	+ 0.620	+ 0.195
Rh	$-\infty$	− 5.130	− 3.036	− 1.287	− 0.568	− 0.237
HLA	$-\infty$	− 1.442	− 0.887	− 0.388	− 0.151	− 0.035

* Previous lod scores were published by Bosker et al.[3], who also included the markers MNS, Fy, Gc, Hp, Gm, and Km (all negative).

molars. Several authors have suggested that a genetic defect, which leads to secondary retention, might also be responsible for the development of other dental anomalies.[16,17] No such anomalies, however, were present in our families.

In an investigation of 138, 3–12 year old siblings, 109 children with secondary retention of deciduous molars the prevalence of secondary retention was found to be 18.1%.[5] When compared with the prevalence of this disorder in a group of randomly chosen children (8.9%), the difference was significant.[5] This supports the hypothesis that there is also a familial occurrence in deciduous molars. Kurol[5] suggested that the prevalence was lower than would be expected on a simple unifactorial basis and he assumed that secondary retention of deciduous molars is inherited as a multifactorial, polygenetic trait. However, in that study no account was given to the possibility of genetic heterogeneity. Therefore his results are not in

contradiction with autosomal dominant inheritance in the familial cases as data on parents and other family members are lacking. If we assume that secondary retention of deciduous molars is etiologically heterogeneous and that, as in secondary retention of permanent molars, the familial cases are due to a dominant gene, than we can estimate from Kurol's data that about 25% of his cases belonged to this genetic group. It is not clear at the moment whether familial secondary retention of permanent molars and familial secondary retention of deciduous molars are separate disease entities or not. Our data on secondary retention of permanent molars suggest that this condition is etiologically heterogeneous, and that an autosomal dominant gene is responsible for about 13% of the cases.

Because of the familial occurrence reported in over 10% of the cases, we advise investigation of family members, especially the younger ones, of each patient with secondary retention to detect this disorder as early as possible, in order to enable adequate treatment to take place.

REFERENCES

1 Raghoebar GM, Boering G, Jansen HWB, Vissink A. Secondary retention of permanent molars: a histologic study. J Oral Pathol Med 1989;18:427–31.
2 Biederman W. Etiology and treatment of tooth ankylosis. Am J Orthod 1962;48:670–84.
3 Bosker H, Ten Kate LP, Nijenhuis LE. Familial reinclusion of permanent molars. Clin Genet 1978;13:314–20.
4 Via WF. Submerged deciduous molars: familial tendencies. J Am Dent Assoc 1964;69:127–9.
5 Kurol J. Infraocclusion of primary molars: an epidemiologic and familial study. Community Dent Oral Epidemiol 1981;9:94–102.
6 Bosker H, Nijenhuis LE. Possible linkage between a gene causing reinclusion of molar I and bloodgroup P. Cytogenet Cell Genet 1975;14:255–6.
7 Pelias MZ, Kinnebrew MC. Autosomal dominant transmission of ankylosed teeth, abnormalities of the jaws, and clinodactyly: a four-generation study. Clin Genet 1985;27:496–500.
8 Gysel C. Un cas familial de rétention partiellè de la deuxième molaire inférieure. Rev Belg Stomat 1955;52:103–11.
9 Reid DJ. Incomplete eruption of the first permanent molar in two generations of the same family. Br Dent J 1954;96:272–3.
10 Israel H. Early hypercementosis and arrested dental eruption: heritable multiple ankylodontia. J Craniofac Genet Dev Biol 1984;4:234–6.
11 Humerfelt A, Reitan K. Effects of hypercementosis on the movability of teeth during orthodontic treatment. Angle Orthod 1966;36:179–89.
12 Theuerkauf J. Die Infraokklusion der Milchmolaren und Sechsjahrmolaren. Dtsch Zahnärztl Z 1960;15:1358–68.
13 Brady J. Familial primary failure of eruption of permanent teeth. Br J Orthod 1990;17:109–13.
14 Raghoebar GM, Abma G, Boering G, Stegenga B. Secundaire retentie van melkmolaren. Ned Tijdschr Tandheelkd 1988;95:389–92.
15 Helpin ML, Duncan WK. Ankylosis in monozygotic twins. J Dent Child 1986;53:135–39.
16 Roberts M W. Multiple familial dental anomalies: report of a case. J Dent Child 1973;40:482–3.
17 Rule JT, Zacherl WA, Pfefferle AM. The relationship between ankylosed primary molars and multiple enamel defects. J Dent Child 1972;39:29–35.

CHAPTER 7

SECONDARY RETENTION OF PERMANENT MOLARS: HISTOLOGICAL CHARACTERISTICS

This chapter is based on articles published in Journal of Oral Pathology and Medicine 1989;18:427–31 (section 7.1) and submitted to British Journal of Oral and Maxillofacial Surgery (section 7.2). Articles have been reprinted with permission of the publisher.

7.1 SECONDARY RETENTION OF PERMANENT MOLARS: A HISTOLOGICAL STUDY

ABSTRACT

The etiology of secondary retention is not well understood, but ankylosis is often considered to be an important factor in primary molars. Data concerning the mechanism of secondary retention in the permanent dentition are insufficient, although a possible role of ankylosis has been suggested. In order to analyze the frequency of occurrence and localization of ankylosis in secondarily retained permanent teeth, 26 secondarily retained molars and for comparison six normal molars were studied histologically. These data were compared with the clinical and radiographic findings. Areas of ankylosis were observed along the roots of all secondarily retained molars. In 81% of the cases these areas were located at the bifurcation and interradicular root surface. No signs of ankylosis were found in normal molars. When these results were compared with the clinical and radiographic data, it was obvious that the latter gave many false negative results, because the areas of ankylosis were often too small to be detected clinically or radiographically.

INTRODUCTION

Secondary retention refers to the cessation of eruption of a tooth after emergence neither due to a physical barrier in the path of eruption nor as a result of an abnormal position. This disturbance in eruption is also known in literature as submerging, ankylosis and reimpaction.[1] Permanent molars are less frequently affected than primary molars.[2] The exact biological mechanism of secondary retention is unknown, although ankylosis has been suggested as the main etiologic factor.[3,4,5]

Ankylosis is defined as a fusion of cementum or dentin with alveolar bone.[1] Due to the loss of the periodontal ligament in some areas the tooth is incapable to continue the eruption and hence is unable to follow the normal vertical development of the neighbouring part of the dentition and alveolar process.[6] In primary molars ankylosis was histologically demonstrable at the interradicular root surface of most of the secondarily retained molars.[7]

In secondarily retained permanent molars two case studies showed that ankylosis can be detected also.[4,8] The cause of this ankylosis is not fully understood. Secondary retention is also considered to be the result of a disturbed local metabolism,[9] infection,[10] genetic factors,[8] deficient eruptive force,[11] deficient vertical alveolar bone growth,[12] trauma,[6] hypercementosis[13] or pulpo-periodontal canals.[14]

We are not aware of data available in the literature in which the histological changes in the secondarily retained permanent molar and its surrounding tissues are studied systematically. The aim of this study was to determine the occurrence of ankylosis in secondarily retained permanent molars histologically and to compare the results with clinical and radiographic findings.

MATERIAL AND METHODS

Material. A molar was considered to be secondarily retained if the occlusal surface was 1 mm or more below the level of the occlusal level of the other teeth in the relevant dental arch at an age at which the tooth normally would have been in occlusion (Figs. 1A, B). Twenty-six secondarily retained molars were studied after removal in a group of 11 girls and 9 boys (mean age 16.2 ± 3.9 years, median 17 years, range 9–27 years). As controls served six normally erupted molars which were removed for orthodontic or prosthetic reasons in five patients. None of the retained or normally erupted molars had a history of trauma or inflammation.

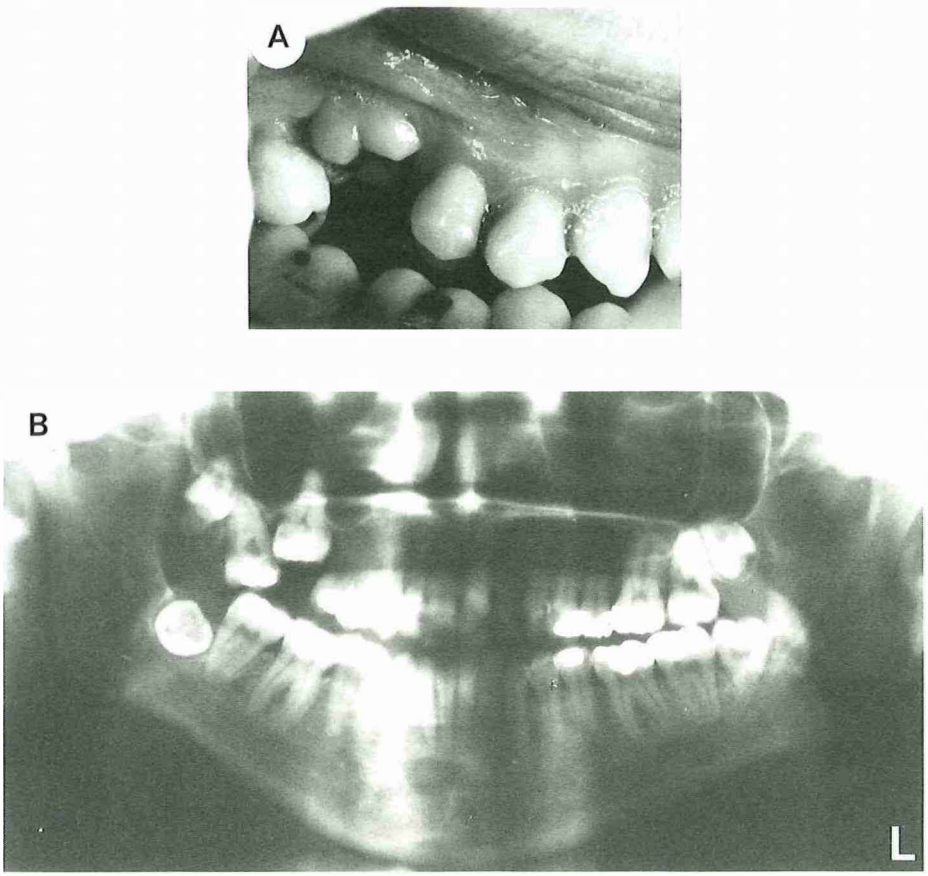


Fig. 1 A Clinical view of the maxilla of a 27-year-old woman showing secondary retention of the first molar. **B** Orthopantomogram of the jaws of same patient

Clinical and radiographic examination. The extent of infraocclusion was defined as the distance in mm between the occlusal surface of the retained molar and the occlusal plane of the relevant arch. A test for percussion sound was done on each

molar (dull sound: normal tooth; solid, clear sound: ankylotic tooth). The presence of dental caries and restorations was scored. Intraoral periapical radiographs and orthopantomograms were taken to detect the presence or absence of a periodontal space and hypercementosis.

Histological examination. After removal, the molars were fixed in a 10% buffered formalin solution for two weeks, washed with tap water and demineralized in a solution of 25% formic acid and 10% sodium citrate in water. After embedding in paraffin 7 μm thick sections were cut in the mesio-distal direction parallel to the vertical axis of the molar and stained with haematoxylin and eosin. Four molars were cut horizontally in bucco-lingual direction. The sections were examined by light microscopy. Histological examination was focused on the location and extent of ankylosis (the direct contact of the alveolar bone with root cement or dentin) and hypercementosis.

Statistical and mathematical analysis. Sensitivity (S) and specificity (Sp) of the clinical diagnostic methods, when compared with the histological results, were calculated according to:

$$S = \frac{TP}{TP + FN} * 100\%$$

$$Sp = \frac{TN}{TN + FP} * 100\%$$

in which TP is the number of true positives, FN the number of false negatives, TN the number of true negatives and FP the number of false positives.

A matched t-test was used to analyze all data at a level of rejection of 0.05.

Table 1 Distribution of secondarily retained permanent molars

	M1	M2	n
Mandible	12	2	14
Maxilla	10	2	12
n	22	4	26

RESULTS

Clinical and radiographic results. The distribution of the secondarily retained molars over the jaws is given in Table 1. The mean infraocclusion was 4.5 ± 2.0 mm (median 3.5 mm, range 2–10 mm). In six secondarily retained molars a solid, clear sound characteristic of ankylosis was recorded. The remaining 20 retained molars and all normal molars revealed a normal dull sound. Partial absence of the periodontal space was observed in three of the six cases with a solid, clear sound

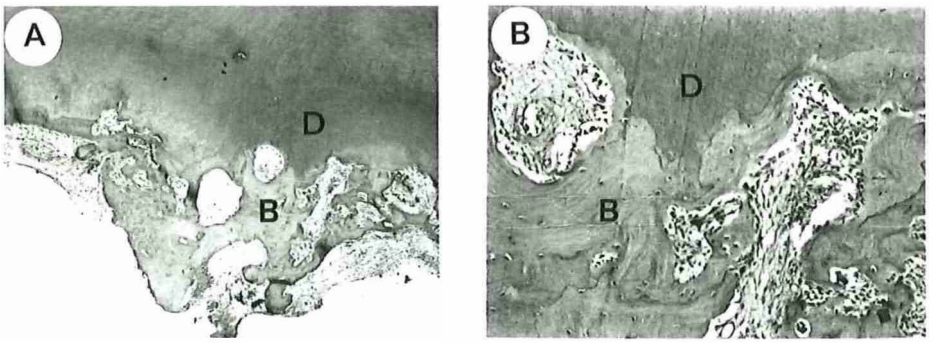


Fig. 2 A Histological section of secondarily retained mandibular first molar. Bifurcation area. Dentin (D) in contact with bone (B). H-E, ×15. **B** Detail of bifurcation area. Resorption lacunae are seen in dentin (D). H-E, ×60.

and in none of the molars with a normal dull sound. Thus ankylosis was clinically suspected in six out of 26 retained molars. Hypercementosis was observed in one secondarily retained molar. Twelve retained molars were free from caries and restorations. All normal molars were restored.

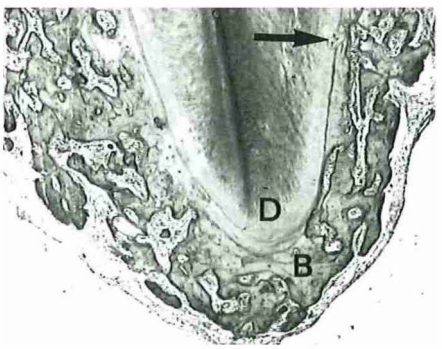


Fig. 3 Histological section of secondarily retained maxillary first molar. Inner and outer surface of apical third of the root. Note small resorption lacuna (arrow) in dentin (D). This part of the root is surrounded by bone (B). H-E, ×15.

Histological results. Partial ankylosis was observed in all secondarily retained molars. In 14 cases ankylosis was located at the bifurcation and interradicular surface of the roots only. In seven cases the bifurcation, interradicular and outer surface were involved and in the remaining five cases ankylosis was detected at the outer surface of the roots only (Figs. 2A, B). In most cases the area of resorption of dentin at the spot of ankylosis was only of a minor extent (Fig. 3). In none of the 26 secondarily retained molars the complete root surface showed ankylosis. Active resorption as indicated by the presence of osteoclasts was not often seen. Repair of a small area of root resorption with cementum was seen in six retained molars (Fig. 4). This repair was mostly located at the outer surface. Hypercementosis was observed in 14 cases on the tip of the root (Fig. 5). Pulpo-periodontal canals were

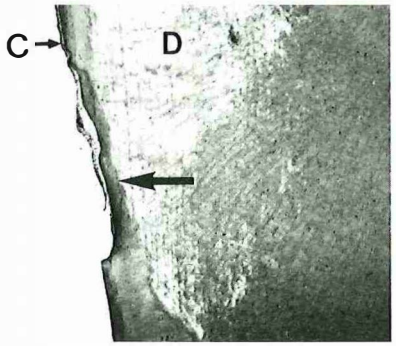


Fig. 4 Histological section of secondarily retained mandibular first molar. Cementum (C) and dentin (D) have been resorbed locally. Reparative cellular cementum (arrow) is deposited in lacuna. H-E, ×25.

seen in two cases, but not in proximity to an area with ankylosis. No pulpal changes were seen, although occasionally calcifications were found (Fig. 6). These pulpal calcifications were mostly located in a root canal.

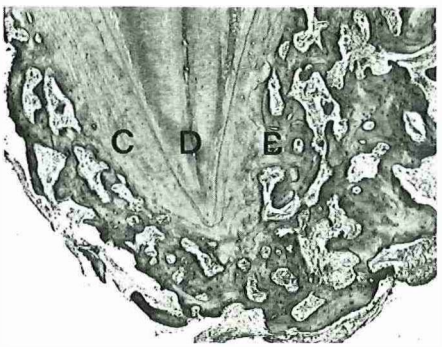


Fig. 5 Histological section of secondarily retained mandibular first molar. Inner and outer surface of apical third of the root. Cementum (C), in this case hypercementosis, in contact with bone (B) (D=dentin). H-E, ×15.

In the normal molars no histological indications for ankylosis were detected. Repair of a resorption lacuna was detected two times in this group. The outer surface of the cementum was intact. The resorption lacuna extending into the dentin was completely filled with reparative cellulair cementum and the shape of the root was recontoured (Fig. 7).

Analysis of clinical and histological results. No statistical relationship existed between the extent of infraocclusion and age ($r=0.004$, $p>0.05$), while there was a significant relationship between the score on the percussion test and the findings of ankylosis on the radiographs ($r=0.659$, $p<0.05$). When comparing the clinical results with the histological findings the sensitivity (S) and specificity (SP) of the clinical diagnostic methods can be calculated. From the histological study it is apparent that ankylosis was present in all retained molars, so FP is 0. A clinical score

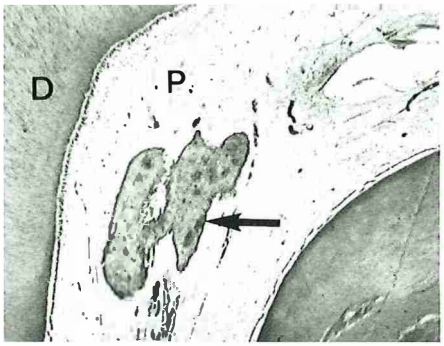


Fig. 6 Histological section of secondarily retained maxillary first molar showing pulp chamber (P). Calcification in pulp (arrow) (D=dentin). H-E, ×15.

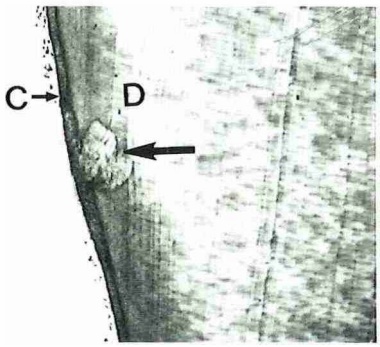


Fig. 7 Histological section of normal mandibular first molar. Deposition of cementum (C) in a former local resorption lacuna extending into dentine (D) (arrow). H-E, ×60.

suggestive of ankylosis was observed in six molars, so TP is 6 and FN is 20. From these data a sensitivity of 23% and a specificity of 100% was calculated.

DISCUSSION

In this study, a group of 26 secondarily retained molars were histologically classified as ankylotic, which supports the theory that a relationship exists between secondary retention and ankylosis. The molars with localized areas of ankylosis seem to be unable to continue eruption in harmony with the growth of the jaw. Nevertheless the question remains, whether the mechanism of eruption is disturbed before or after ankylosis occurs (in other words: the famous question of which was first, the chicken or the egg).

It is apparent that some defect, failure, or alteration of the periodontal ligament must precede ankylosis, because no ankylosis will occur in case of a normal periodontal membrane. Any tooth,[2] either primary or permanent, may become ankylotic during eruption. The exact biological mechanism of this ankylosis is still un-

known. Traumatic injuries resulting in a defective periodontal ligament are known to cause ankylosis.[15] However, the patients included in this study had no history of trauma. For secondarily retained primary molars, it has been suggested that ankylosis occurs as a result of a disturbance in the interaction between normal root resorption and hard tissue repair.[16,17] In permanent teeth, local root resorption is a physiological process.[18] This was also observed in the unaffected molars included in this study. Usually root resorption is repaired by formation of cementum. If this process is disturbed and the cementoblasts do not reappear but are replaced by osteoblasts, depositing osteoid material in the resorption lacunae, the result is probably ankylosis. Usually molars show the largest number of resorption areas,[19] which may account for the large number of molars involved in secondary retention. The etiological factor may be a local developmental disturbance of the periodontal ligament. The latter is supported by reports of a definite familial tendency, suggesting the cause to be inheritable.[8]

Other causative factors reported in the literature are deficient vertical alveolar bone growth,[12] hypercementosis[13] and pulpo-periodontal canals.[14] The growth of bone is related to the eruption of teeth. So if the eruption of a tooth ceases then the vertical bone growth will also stop. Hypercementosis may cause a delayed tooth eruption in young persons. This can result in ankylosis between alveolar bone and root surface with or without root resorption. Our histological specimens revealed that in all cases there was localized root resorption and the lacunae were filled with bone. Because eruption is usually not delayed as long as there is a normal periodontal ligament, hypercementosis can as well be the result of secondary retention. Pulpo-periodontal canals have been demonstrated to occur in the bifurcation area in 28–76 per cent of all permanent molars.[20,21] Rune[14] discussed a possible etiological factor in ankylosis where the resorption leading to ankylosis originates in such canals. The results of our study do not support this hypothesis as we could only demonstrate the presence of two pulpo-periodontal canals in our series.

The clinical and radiographic diagnostic method only supported the histological diagnosis ankylosis in six cases. Percussion test revealed ankylosis more accurately than radiographs.[15] Andersson et al.[23] showed that a solid percussion sound can be recorded if over 20% of the root is ankylotic. Although we did not calculate the total area of the root which was ankylotic, we recorded a solid sound in case of extreme ankylosis. Radiographic examination may be even less useful for the detection of ankylosis of multi-rooted teeth,[23] because small areas of ankylosis may not be visible because of overlapping structures. Furthermore, early hypercementosis can not always be detected on radiographs as in this study only in one case signs of hypercementosis were observed radiographically. Infraocclusion is the most important and reliable clinical criterion.

The present study has shown that the majority of secondarily retained permanent molars show areas of ankylosis histologically. The molars cease to erupt as soon as ankylosis occurs and cannot move or be moved for example orthodontically. Treatment recommendations must be based on this fundamental principle.

Diagnostic methods such as percussion sound and partial absence of the periodontal space on radiographs result in many false negative results, because they depend on the extent of the ankylotic area. A positive clinical test is strongly suggestive for ankylosis, but in case of a negative test ankylosis can not be excluded. Further studies are needed to clarify the biological mechanism of secondary retention.

REFERENCES

1 Pindborg JJ. Pathology of the dental hard tissues. Copenhagen: Munksgaard, 1970:248–52, 362–6.
2 Biederman W. The incidence and etiology of tooth ankylosis. Am J Orthod 1956;42:921–6.
3 Biederman W. Tooth ankylosis. Ann Dent 1953;12:1–15.
4 Korkhaus G. Die Halbretention des ersten Molaren. Dtsch Zahnärztl Z 1952;7:1205–12.
5 Herman E. Evaluation and management of ankylosed teeth. NY State Dent J 1964;30:327–33.
6 Biederman W. The ankylosed tooth. Dent Clin North Am 1964;8:493–508.
7 Kurol J, Magnusson BC. Infraocclusion of primary molars: a histologic study. Scand J Dent Res 1984;92:564–76.
8 Sims MR. Tooth ankylosis in man and the oxytalan fibre meshwork. Aust Orthod J 1980;6:147–53.
9 Sterrett DS. Report of a case of a submerging first molar. Am J Orthod Oral Surg 1940;26:681–3.
10 Shafer WG, Hine MK, Levy BM. A textbook of oral pathology. 4th ed. Philadelphia: WB Saunders, 1983:540–1.
11 Proffit WR, Vig KWL. Primary failure of eruption: a possible cause of posterior open-bite. Am J Orthod 1981;80:173–90.
12 Gorlin RJ, Goldman HM. Thoma's oral pathology. 6th ed. St Louis: CV Mosby, 1970:153–4.
13 Humerfelt A, Reitan K. Effects of hypercementosis on the movability of teeth during orthodontic treatment. Angle Orthod 1966;36:179–89.
14 Rune B. Submerged deciduous molars. Odontol Revy 1971;22:257–73.
15 Andreasen JO. Traumatic injuries of the teeth. 2nd ed. Copenhagen: Munksgaard, 1981:211–8.
16 Dixon DA. Observations on submerged deciduous molars. The first Chapman prize essay. Dent Pract 1963;13:303–15.
17 Henderson HZ. Ankylosis of primary molars: a clinical, radiographic, and histologic study. J Dent Child 1979;46:117–22.
18 Brown WAB. Resorption of permanent teeth. Br J Orthod 1982;9:212–20.
19 Henry JL, Weinmann JP. The pattern of resorption and repair of human cementum. J Am Dent Assoc 1951;42:270–90.
20 Gutmann JL. Prevalence, location, and patency of accessory canals in the furcation region of permanent molars. J Periodontol 1978;49:21–6.
21 Burch JG, Hulen S. A study of the presence of accessory foramina and the topography of molars furcations. Oral Surg Oral Med Oral Pathol 1974;38:451–5.
22 Andreasen JO. Periodontal healing after replantation of traumatically avulsed human teeth. Assessment by mobility testing and radiography. Acta Odontol Scand 1975;33:325–35.
23 Andersson L, Blomlöf L, Lindskog S, Feiglin B, Hammarström L. Tooth ankylosis. Clinical, radiographic and histological assessments. Int J Oral Surg 1984;13:423–31.

7.2 SECONDARY RETENTION OF PERMANENT MOLARS: AN ASSESSMENT OF ANKYLOSIS BY SCANNING ELECTRON AND LIGHT MICROSCOPY

ABSTRACT

Secondary retention refers to the cessation of eruption of a tooth after emergence. This may be the result of pathological changes in the periodontal ligament. The aim of this study was to describe the morphological and histological aspects of the radicular surface of secondarily retained permanent molars. The roots of 12 secondarily retained molars and two control molars, were examined by means of scanning electron microscopy (SEM) and light microscopy (LM) in order to analyze the occurrence and localization of ankylosis. With SEM it was observed that the root surface of retained molars showed local areas covered with bonelike tissue. LM of these areas showed that this tissue was bone in direct contact with the root surface (i.e. ankylosis). In 11 cases, the areas of ankylosis were observed in the bifurcation area and at the interradicular root surface. In the remaining case, ankylosis was located at the outer root surface. The results of this study endorse the assumption that focal ankylosis is an important factor in secondary retention. Treatment recommendations must be based on this fundamental principle, because orthodontic movement of ankylotic molars is not possible.

INTRODUCTION

Secondary retention refers to the cessation of eruption of a tooth after emergence neither due to of a physical barrier in the path of eruption nor as a result of an abnormal position.[1] This disturbance in eruption is also known in literature as submerging, ankylosis or reimpaction.[2,3,4] Local areas of ankylosis have been suggested to be an important etiopathologic factor.[1-3] Other proposed factors are a disturbed metabolism,[4] genetic factors,[5] deficient eruptive force,[6] deficient vertical alveolar bone growth,[7] hypercementosis,[8] and pulpo-periodontal canals.[9]

Ankylosis is defined as a fusion of cementum or dentin with alveolar bone.[2] Due to the loss of the periodontal ligament in the ankylotic area the tooth is incapable to continue eruption and hence is unable to follow the normal vertical development of the neighbouring teeth and alveolar process.[3] This makes that the molar gradually attains an infraposition in comparison with its neighbours. In both primary and permanent molars ankylosis was predominantly seen histologically at the interradicular root surface.[1,10,11] As ankylosis is probably a major factor in the development of secondary retention it is worthwhile to study this phenomenon in greater detail. Light microscopy is a proper tool to prove direct contact between root surface and surrounding bone (ankylosis), while scanning electron microscopy is an excellent tool to investigate the topography of the root surface in order to detect the spread of the ankylotic areas.[12] The aim of this study was to describe the morphological and histological aspects of the radicular surfaces of secondarily retained permanent molars.

MATERIALS AND METHODS

Clinical data. A molar was considered to be secondarily retained if the occlusal surface was 1 mm or more below the level of the occlusal level of the other teeth in the relevant dental arch at an age at which the tooth normally would have been in occlusion (Figs. 1A, B). A total of 12 secondarily retained molars were removed from six girls and five boys (mean age 15.3 ± 3.2 years, median 16 years, range 9–25 years) for therapeutic reasons. The distribution of the affected molars over the jaws is given in Table 1. The mean infraocclusion of the retained molars was 4.4 ± 2.1 mm. A percussion sound suggestive for the presence of ankylosis (solid, clear sound) and a partial absence of the periodontal ligament space on radiographs was observed in five retained molars only. None of the retained molars had a history of trauma or inflammation. A first and second permanent molar, that were in occlusion, and had to be removed for orthodontic reasons in the same patient group, served as a control.

Scanning electron microscopy (SEM). After removal, the molars were cleaned ultrasonically in a 0.9% w/v NaCl solution for 5 minutes to remove blood residues and tissue debris, whereafter they were treated with a 30% w/v H_2O_2 solution for two hours to remove adhering tissue and cleaned ultrasonically in buffer solution. Subsequently the teeth were fixed by immersion in a 2% w/v glutardialdehyde solution in 0.1 M cacodylate buffer, pH 7.4, for 24 hours. The specimens were then immersed in a mixture containing 2% w/v arginine-HCl, 2% w/v glycine, 2% w/v sodiumglutamate and 2% w/v sucrose in water for 16 hours, followed by immersion in a solution containing 2% w/v guanidine-HCl and 2% w/v tannic-acid in water for 8 hours, and washing in destilled water (6×). Finally, the samples were immersed in a 2% w/v OsO_4 solution in water for 8 hours at 4°C, washed again in destilled water (6×), dehydrated in an ethanol series up till 100% ethanol and critical point dried with liquid CO_2. Unless stated otherwise all procedures were performed at room temperature. The specimen were studied in a JEOL scanning electron microscope, operated at 15–25 kV.

Light microscopy (LM). After SEM, the samples were demineralized in a 25% v/v formic acid solution containing 10% w/v sodium citrate. The demineralized samples were embedded in paraffin. Seven μm serial sections were cut through the areas suspected for the presence of ankylosis or resorption on SEM and stained with hematoxylin and eosin. Light microphotographs were taken with a Zeiss photomicroscope.

RESULTS

Secondarily retained molars. With SEM local areas of bonelike tissue could be detected in all 12 secondarily retained permanent molars. LM of these areas showed that the bonelike tissue was in direct contact with dentin or cementum. In one molar, parts of the outer surface of the root were affected, while in the other 11

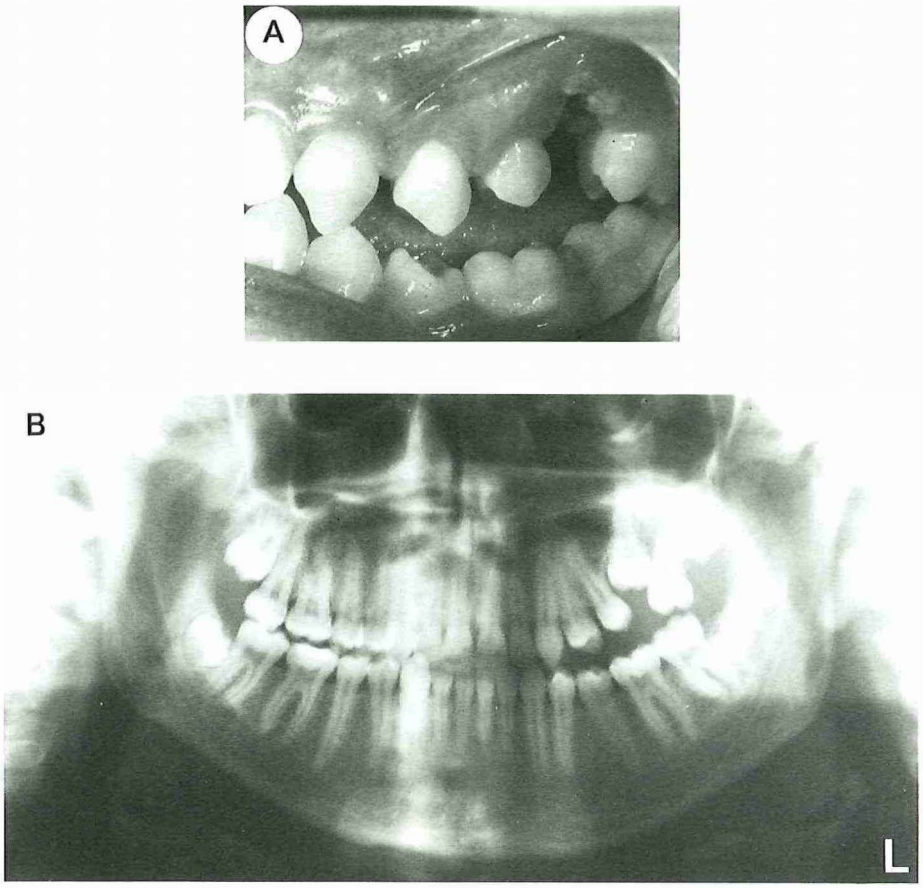


Fig. 1 A Clinical view of the maxilla of a 14-year-old girl showing secondary retention of the left first molar. **B** Orthopantomogram of the same patient showing the position of the retained molar and reduction of height of the alveolar process in the relevant area. Note the presence of a restoration in the crown, indicating that the molar once was in occlusion.

Table 1 Distribution of secondarily retained permanent molars

	M1	M2	n
Mandible	6	1	7
Maxilla	4	1	5
n	10	2	12

retained molars the apical, interradicular and/or bifurcation area were involved. Figs. 2A and 2B show localized deposits of bonelike material in the bifurcation area. Lightmicroscopically, these areas revealed deposits of bone in direct contact

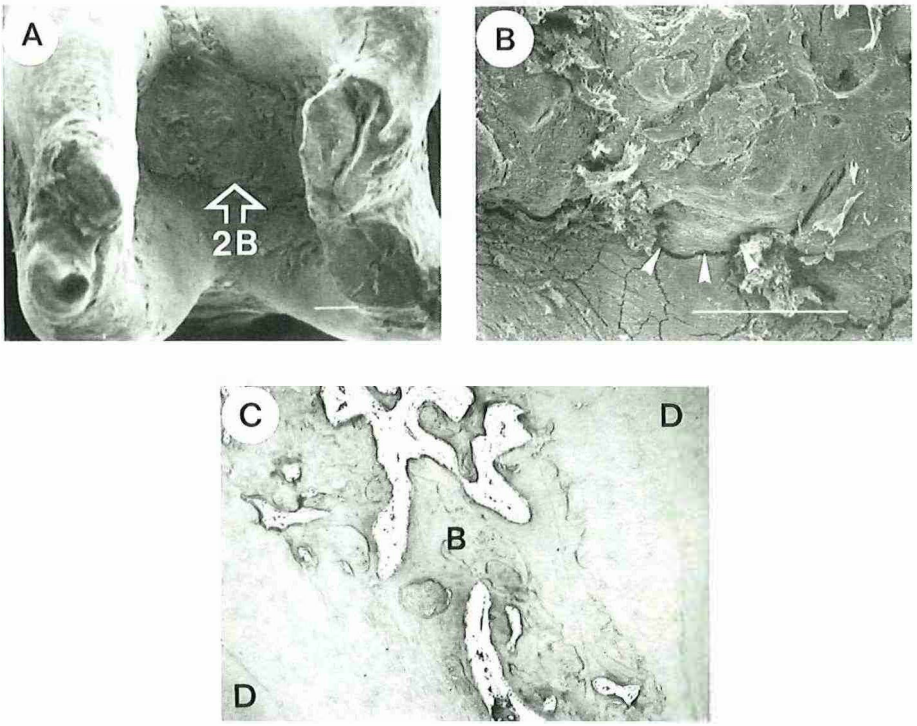


Fig. 2 A SEM micrograph showing deposit of bonelike tissue in the bifurcation. Bar=1mm. **B** Detail of the bifurcation area (arrow in Fig. 2A). Bony deposits are indicated by arrows. Bar=100 μm. **C** LM photomicrograph showing cross section through the bifurcation. Bone (B) is in direct contact with dentin (D). H-E, ×110.

with dentin indicating ankylosis (Fig. 2C). The periodontal ligament was absent in these areas. Fig. 3 shows an ankylotic apical area. The apex is covered with bonelike material. The borderline between the bonelike tissue and the root surface can be easily recognized (Fig. 3A). Cross sections of this area revealed hypercementosis, and the bone was in direct contact with the cementum. No periodontal ligament was observed between hypercementosis and bone (Fig. 3B). In six specimens, broken bridges of bone were detected (Figs. 4A, 4B). Cross sections through these bone bridges revealed bone in direct contact with dentin and presence of resorption lacunes (Fig. 4C). Hypercementosis was observed next to these bridges of bone (Figs. 4B, 4D). In the cementum many pits were observed (Figs. 4A, 4B). In two molars widely spread areas with resorption of the root surface were observed (Fig. 5). These areas were predominantly observed in the apical region (Figs. 5A, 5B). Repair with cellulair cementum was also observed (Fig. 5C). In the bifurcation ankylosis could be detected (Figs. 5A, 5D). Pulpo-periodontal canals were observed but not adjacent to the areas covered with bonelike tissue. In none of the 12 secondarily retained molars was the whole root surface ankylotic. The areas of ankylosis covered 10 to 60% of the whole root surface. More than 40% of the root

surface was ankylotic in the five molars that were clinically suspect for ankylosis (solid, clear sound and partial absence of the periodontal ligament space).

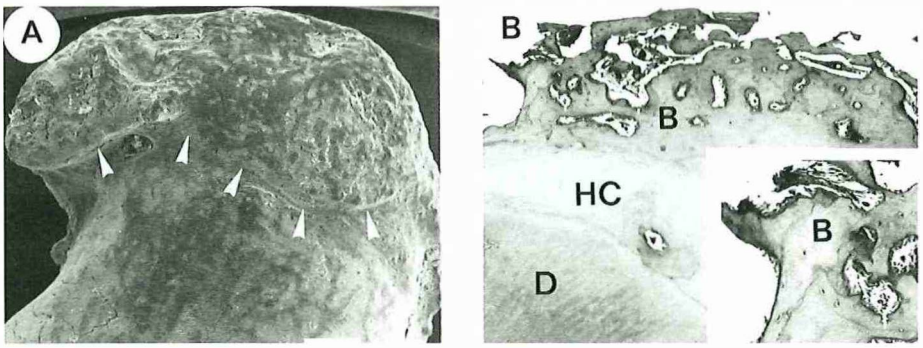


Fig. 3 A SEM micrograph showing a root apex covered with bonelike tissue. The borderline between the bonelike tissue and the rootsurface is indicated by arrows. Bar=1 mm. **B** LM photomicrograph showing cross section through the area. Hypercementosis (HC) is in direct contact with bone (B). H-E, ×25. The insert shows a detail of the bone. ×60.

Control molars. Neither by SEM nor by LM, signs of ankylosis of the root surface could be detected in control molars.

DISCUSSION

The roots of all twelve examined secondarily retained permanent molars showed local areas of ankylosis. Such areas were not observed in both normally erupted control molars. These results support the assumption that ankylosis is involved in secondary retention. It gives the impression that due to the ankylotic areas the retained molar is incapable to continue its eruption and hence comes in infraocclusion that may increase with time. The question remains, however, whether the eruption mechanism is disturbed before or after ankylosis begins.

In general, root resorption and ankylosis are frequently observed after injury to the periodontal ligament. This is probably the explanation for the fact that infection and trauma have been suggested to be substantial factors in the development of ankylosis in secondarily retained teeth.[2] In none of the retained molars in this study, however, there was a history of trauma or infection. We assume that defects in the periodontal ligament allow ingrowth of connective tissue different from that of the periodontal ligament leading to a bony union between the alveolar bone and root surface. This may occur during repair of the physiologically occurring areas of resorption, a phenomenon that is common in normal teeth.[13] In case of misrepair a bony union between alveolar bone and root surface may develop, instead of new cementum being laid down. This is in contrast to root resorption for example observed during orthodontic movement of teeth using high forces.[14,15] In these cases, the resorption defects are repaired with cellular cementum.[14]

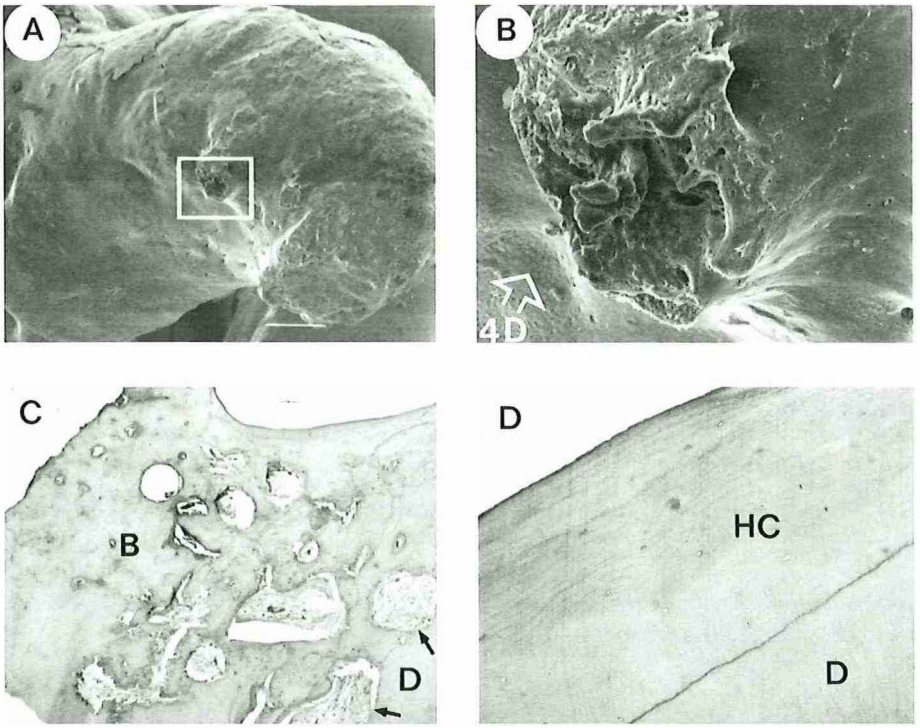


Fig. 4 A SEM micrograph showing covering of a root apex covered by bonelike tissue. The square highlights a broken bridge of bone. Bar= 1mm. **B** Detail of the broken bridge of bone. Bar= 100μm. **C** LM photomicrograph showing cross section through the broken bridge of bone. Dentin (D) is in direct contact with bone (B). Resorption lacunes are present (arrows). H-E, ×100. **D** LM photomicrograph showing cross section through the area immediately next to the bridge of bone (arrow in Fig. 4B). This area also shows hypercementosis (HC). H-E, ×100.

Pulpo-periodontal canals have been demonstrated normally to occur in 48–56% of all permanent molars.[16] Resorption areas are often located near these canals. Harry and Sims[17] assumed that there is a relationship between these zones of resorption and the tissue components, such as blood vessels passing through these openings. Rune[9] suggests that these resorption areas are a possible etiological factor in secondary retention, because resorption leading to ankylosis can originate from such canals. This hypothesis is not supported by Kurol's[11] and our data.

According to Humerfelt and Reitan[8], hypercementosis may cause a delayed tooth eruption in young persons. They assume that delayed eruption can result in ankylosis between alveolar bone and root surface with or without root resorption. We think that it is more probable that hypercementosis is a result of secondary retention as eruption is usually not delayed in case of a normal periodontal ligament.

As ankylosis seems to be a major phenomenon in secondary retention, it has many impacts on therapy. A molar with an abnormal periodontal ligament can not usually be moved by orthodontic force.[18,19] According to literature, luxation of a secondarily retained molar with ankylosis has been used with some success to move

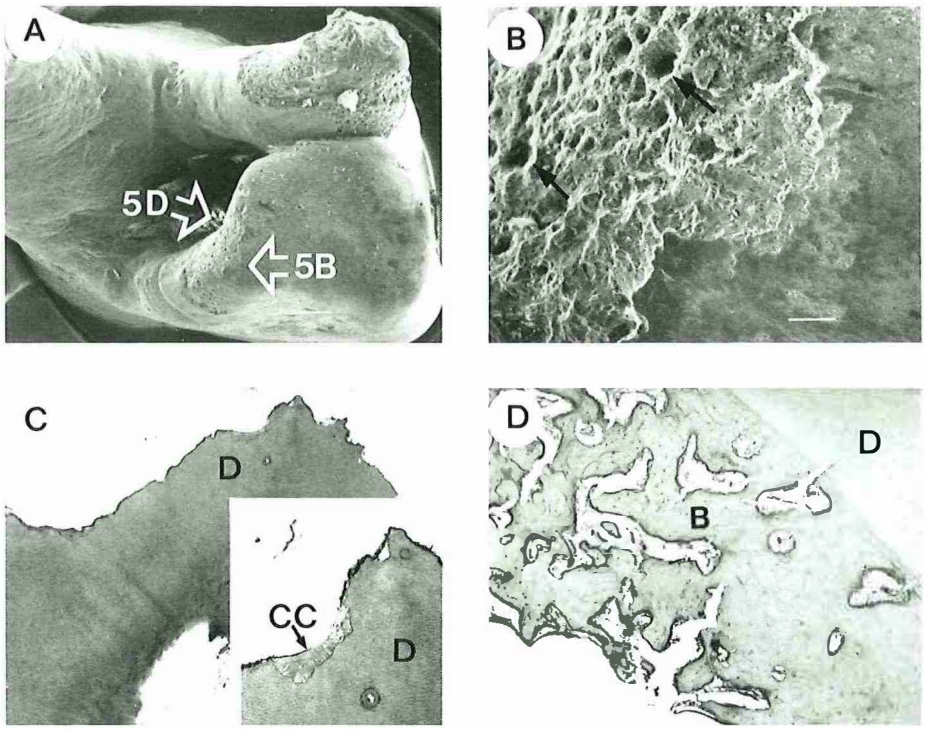


Fig. 5 A SEM micrograph showing widely spread areas with resorption in the apicial region. Bar=1mm. **B** Magnification of a resorption area (arrow in Fig. 5A). Bar=100μm. **C** LM photomicrograph showing cross section through a resorption area. H-E, ×25. The insert shows that at certain sites cellular cementum (CC) is deposited. ×60. **D** LM photomicrograph showing cross section through the interradicular root surface (arrow in Fig.5A). This area shows bone (B) in direct contact with dentin (D). H-E, ×100.

the tooth.[20] The luxation technique assumes that if the periapical blood supply can be preserved, a new fibrous ligament will be formed in the ankylotic region because of local inflammation due to the mechanical disturbance of that region. The luxation technique, however, is not thought to be successful in most cases of secondary retention,[21] because histologically in the majority of these cases the ankylotic areas are found at the bifurcation and interradicular root surfaces.[1] These ankylotic spots can not be easily broken by luxation, because the rotation point during luxation of the molars is at its bifurcation. In our opinion, it is more probable in these cases that luxation promotes further ankylosis rather than eliminates the ankylosis. Assuming that orthodontic movement of a secondarily retained molar is not possible we recommend the following treatment approach:[21]

- before the growth spurt has started: immediate removal of the retained permanent molar followed by orthodontic treatment to close the diastema.
- during the growth spurt: observation of the affected tooth at 6-month intervals to evaluate whether the infraocclusion remains minor and stable. In case of progression, an extraction and orthodontic treatment is advisable.

- after the growth spurt: in cases of slight infraocclusion, a prosthetic build up of the retained molar to restore the occlusal height is the proper treatment; in cases of severe infraocclusion, removal is recommended in order to prevent caries and periodontal disease of the neighbouring teeth.

The morphological and histological observations outlined in this study make it quite probable that local areas of ankylosis between the root and alveolar bone are a significant factor in secondary retention of permanent molars.

REFERENCES

1 Raghoebar GM, Boering G, Jansen HWB, Vissink A. Secondary retention of permanent molars: a histologic study. J Oral Pathol Med 1989;18:427–31.
2 Pindborg JJ. Pathology of dental hard tissues. Copenhagen: Munksgaard, 1970:248–52.
3 Biederman W. Tooth ankylosis. Ann Dent 1953;12:1–15.
4 Sterrett DS. Report of a case of a submerging first molar. Am J Orthod Oral Surg 1940;26:681–3.
5 Bosker H, Ten Kate LP, Nijenhuis LE. Familial reinclusion of permanent molars. Clin Genet 1978;13:314–20.
6 Dixon DA. Observations on submerging deciduous molars. The first Chapman prize essay. Dent Pract 1963;13:303–15.
7 Gorlin RJ, Goldman HM. Thoma's oral pathology. 6th ed. St Louis: CV Mosby, 1970:153–4.
8 Humerfelt A, Reitan K. Effects of hypercementosis on the movability of teeth during orthodontic treatment. Angle Orthod 1966;36:179–89.
9 Rune B. Submerged deciduous molars. Odontol Revy 1971;22:257–73.
10 Korkhaus G. Die Halbretention des ersten Molaren. Dtsch Zahnärtzl Z 1952;7:1205–12.
11 Kurol J, Magnusson BC. Infraocclusion of primary molars: a histologic study. Scand J Dent Res 1984;92:564–76.
12 Boyde A, Lester KS. Electron microscopy of resorbing surfaces of dental hard tissues. Z Zellforsch 1967;83:538–48.
13 Henry JL, Weinmann JP. The pattern of resorption and repair of human cementum. J Am Dent Assoc 1951;42:270–90.
14 Langford SR, Sims MR. Root surface resorption, repair and periodontal attachment following rapid maxillary expansion in man. Am J Orthod 1982;81:108–15.
15 Barber AF, Sims MR. Rapid maxillary expansion and external root resorption in man: a scanning electron microscope study. Am J Orthod 1981;79:630–52.
16 Vertucci FJ, Anthony RL. A scanning electron microscopic investigation of accessory foramina in the funcation and pulp chamber floor of molar teeth. Oral Surg Oral Med Oral Pathol 1986;62:319–26.
17 Harry MR, Sims MR. Root resorption in bicuspid intrusion: a scanning electron microscope study. Angle Orthod 1982;52:235–58.
18 Proffit WR, Vig KWL. Primary failure of eruption: a possible cause of posterior open-bite. Am J Orthod 1981;80:173–90.
19 Mitchell DL, West JD. Attemped orthodontic movement in the presence of suspected ankylosis. Am J Orthod 1975;68:404–11.
20 Biederman W. Etiology and treatment of tooth ankylosis. Am J Orthod 1962;48:670–84.
21 Raghoebar GM, Boering G, Booy C, Vissink A. Treatment of the retained molar. J Oral Maxillofac Surg 1990;48:1033–8.

CHAPTER 8

SECONDARY RETENTION OF PERMANENT MOLARS: TREATMENT

This chapter has been published in Journal of Oral and Maxillofacial Surgery 1990;48:1033–8, and has been reprinted with permission of the publisher.

ABSTRACT

The effect of five different treatments for secondary retention in permanent molars was evaluated in 59 patients to develop a rational guideline for adequate therapy. The results showed that a prosthetic build up is the proper treatment if secondary retention develops after the growth spurt, because in these cases the extent of infraocclusion is slight and relatively stable. If secondary retention develops before the growth spurt, immediate removal of the retained molar followed by orthodontic treatment gives maximal success. When secondary retention develops during the growth spurt, the affected tooth has to be observed at 6-monthly intervals. In such cases, no active treatment is indicated if the neighbouring teeth show no tilting and the extent of infraocclusion is minor and stable. In all other cases the affected teeth have to be removed, followed by orthodontic closure of the diastema. Finally, all patients must be regularly screened, as in this investigation, new cases of secondary retention were observed relatively frequently.

INTRODUCTION

Secondary retention is the cessation of eruption of a tooth after emergence neither due to a physical barrier in the path of eruption nor as a result of an abnormal position.[1] Other terms commonly used for this phenomenon are ankylosis, submerging molar, and reimpaction.[2-5] Secondary retention has been observed far more frequently in deciduous molars than in permanent molars.[6] If eruption of a tooth ceases, the continuing vertical development of the neighbouring teeth and the alveolar process leaves that tooth behind, so that it appears submerged. Clinically and radiographically, the tooth affected is in infraocclusion. Occlusal contact is lost and tilting of the neighbouring teeth may occur, leading to loss of arch length. The extent of malocclusion depends predominantly on the stage of development of the dentition and facial growth at the moment secondary retention begins. The etiology of this phenomenon is not fully understood. Histological examination shows that localized areas of ankylosis are present between the molar roots and the alveolar process,[5,7] but ankylosis is not suspected in all cases.[8,9]

Treatment of secondary retention is difficult because there is a periodontal, a restorative and an orthodontic problem, and in the case of deeply retained molars there is a risk of jaw fracture when removing the tooth.[10] Successful treatment requires a multidisciplinary approach, including the dentist, the orthodontist, and the oral and maxillofacial surgeon. Because of the low incidence of this disorder, different methods of treatment are predominantly reported in case reports.[9,11-14] To the best of our knowledge, no reports that study and compare the results of different treatment approaches for secondary retention of permanent molars are available. Because of this lack of data, we evaluated the clinical results of five different treatment approaches to work out a guideline for treatment of this disorder under various clinical conditions.

MATERIAL AND METHODS

A molar was considered to be secondarily retained if the occlusal surface was 1 mm or more below the occlusal level of the other teeth in the relevant dental arch at an age at which the tooth normally would have been in occlusion (Fig. 1). Molars with a disturbed morphology due to extensive carious lesions or restorations were excluded. The condition had to be in existence for a period of at least six months in order to be sure that there was no tendency for eruption.

Twenty-six female and 33 male patients (age: 22.7 ± 12.5 years, median 18.7 years, range 9 to 62 years) participated in this study. The patients were referred by dentists, orthodontists, and oral and maxillofacial surgeons. All patients were healthy and had no history of dental trauma, as indicated by a review of their medical history. In these patients, a total number of 88 molars was diagnosed to be secondarily retained. The distribution of these molars is given in Table 1.

Table 1 Distribution of secondarily retained permanent molars

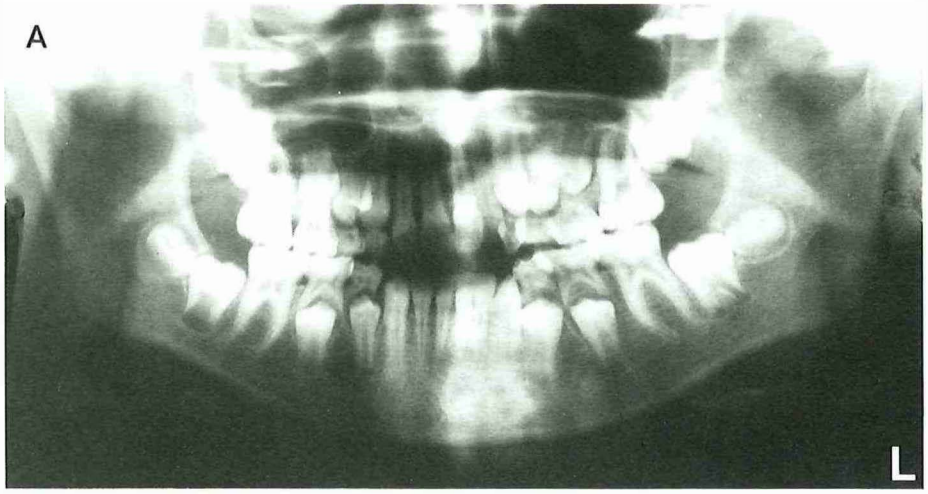
	M1	M2	n
Mandible	41	12	53
Maxilla	29	6	35
n	70	18	88

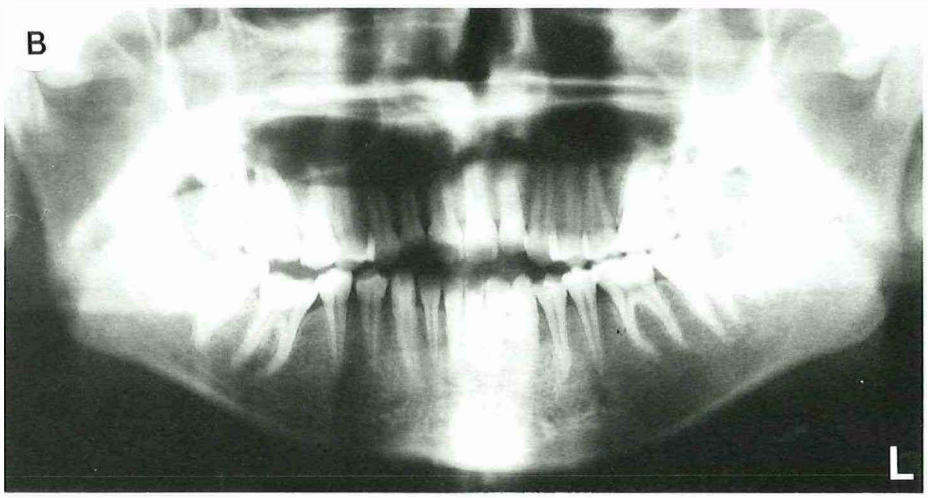
Infraocclusion was measured as the distance between the occlusal surface of the retained molar and the occlusal plane of the relevant dental arch. Tilting of the neighbouring teeth was scored on a three-point scale (1, no tilting; 2, moderate tilting, less than half of the occlusal surface of the retained molar is covered by neighbouring teeth; 3, severe tilting, more than half of the occlusal surface of the retained molar is covered by neighbouring teeth). Orthopantomograms and intraoral periapical radiographs were made of all patients to study the vertical development of the alveolar process in the area of the retained tooth and to detect other possible abnormalities. Caries and restorations were noted.

Five modes of treatment were applied.

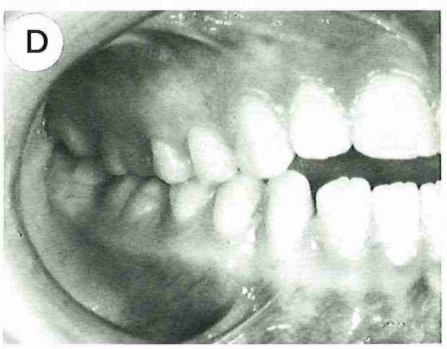
a *No treatment* (n=19). These patients refused treatment because they had no symptoms. They agreed to participate in the follow up study.

b *Surgical removal* (n=62). Removal of affected molars was recommended if these teeth were severely carious, were in severe infraocclusion, or if there was tilting of the neighbouring teeth. Eighteen molars were extracted with forceps. The other 44 molars had to be removed surgically (16 molars after removal of the buccal cortical bone, 28 molars after sectioning of the clinical crown and separation of the roots in the bifurcation). In seven cases, the removal of a maxillary molar resulted in a perforation of the antral floor because of the high anatomical position of the retained molar in the superior alveolar process. The


A
L


B
L


C


D

oroantral opening was closed with a buccal flap. In one case, the inferior alveolar nerve was injured. Orthodontic treatment of neighbouring teeth was performed in 11 cases. In 17 cases, orthodontic treatment was indicated, but was refused by the patients. In five cases, bridges were made to close the diastema.

c *Prosthetic treatment* (n=3). A composite resin build up was made in adult patients if there was no tilting of the neighbouring teeth and infraocclusion was less than 4 mm.

d *Luxation* (n=3). Luxation to break the ankylotic areas was used when the retained molar was not locked in infraposition by other teeth. Under local anesthesia, the retained molar was gently rocked in a buccolingual direction using the appropriate forceps.[12] The axis of the arc of movement was through the apices. The patient and the parents or guardians were informed that this form of treatment might fail to move the tooth (extensive areas of ankylosis) and might result in endodontic treatment or even loss of the tooth. In two cases, orthodontic treatment was started immediately after the luxation of the tooth. In the third case, the retained second molar only was luxated.

e *Orthodontic treatment* (n=1). In this case, there was no tilting of the neighbouring teeth and infraocclusion was 3 mm.

The follow up period varied from 6 months to 8 years after treatment. Special attention was paid to the dimension of the postextraction diastema, tilting of the neighbouring teeth, the relationship with opposing teeth (vertical interdigitation), development of the jaws, and result of treatment. In addition, new cases of secondary retention in the same dentition were recorded.

RESULTS

Clinical observations before treatment

The first permanent molars in the mandible and maxilla were the teeth most often affected (Table 1). In one patient, secondary retention was found in all four quadrants. The mean infraocclusion of the molars was 4.4 ± 2.3 mm (median 3.5 mm, range 2 to 12 mm). In 40 cases, no tilting of the neighbouring teeth was observed, 38 cases displayed moderate tilting, and in four cases severe tilting occurred (Fig. 2). Six molars had no neighbouring teeth. The vertical dimension of the alveolar bone in the area of the retained molar was less in all cases. Twenty-six retained molars were caries free; the other 62 teeth showed caries and/or had been restored.

Fig. 1 (see page 102) **A** Orthopantomogram of an 11-year-old girl showing all first permanent molars in occlusion. **B** Orthopantomogram of the same girl 5 years later showing secondary retention of the mandibular right first molar. Secondary retention started during the growth spurt. **C** Clinical view of the secondarily retained molar. **D** Clinical view after prosthetic build up of the retained molar (3 years after application).

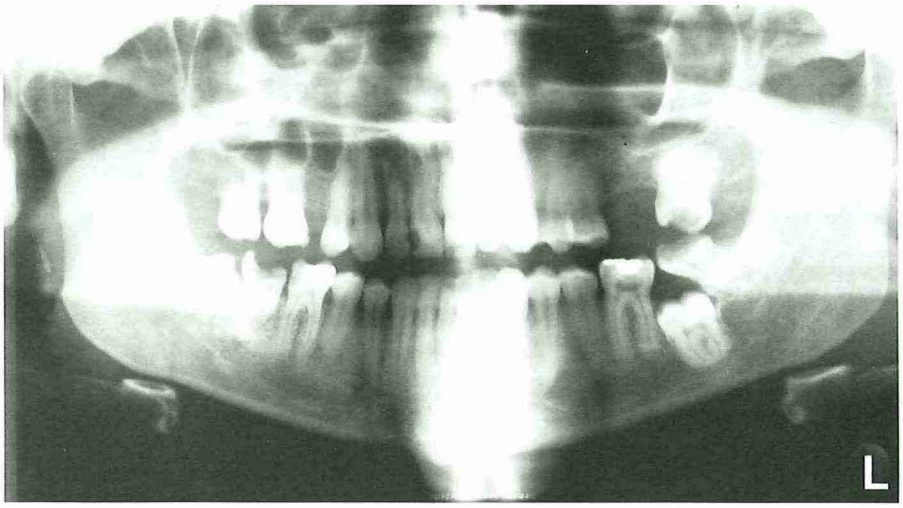


Fig. 2 Orthopantomogram of a 26-year-old man showing secondary retention of the mandibular left second molar and severe tilting of the third molar. Both the retained tooth and third molar were removed.

Table 2 Diastema and vertical interdigitation after treatment of secondary retention

Treatment	n*	Closure of diastema		Vertical interdigitation**	
		Yes	No	Yes	No
No treatment	19	0	19	0	19
Surgical removal in maxilla followed by:					
– no treatment	9	7	2	6	3
– orthodontic treatment	3	3	0	3	0
– bridge	2	2	0	2	0
Surgical removal in mandible followed by:					
– no treatment	8	4	4	2	6
– orthodontic treatment	8	8	0	8	0
– bridge	3	3	0	3	0
Prosthetic build up	3	3	0	3	0
Luxation	3	0	3	0	3
Orthodontic treatment	1	0	1	0	1

* In 29 cases, including second molars, no diastema existed or closure was not possible due to removal or agenesia of neighbouring teeth. These molars are not included.

** The occlusal relationship between the secondarily retained molar, the neighbouring teeth and its opposing teeth was assessed by the quality of interdigitation (good/acceptable contacts = Yes, and poor contacts = No).

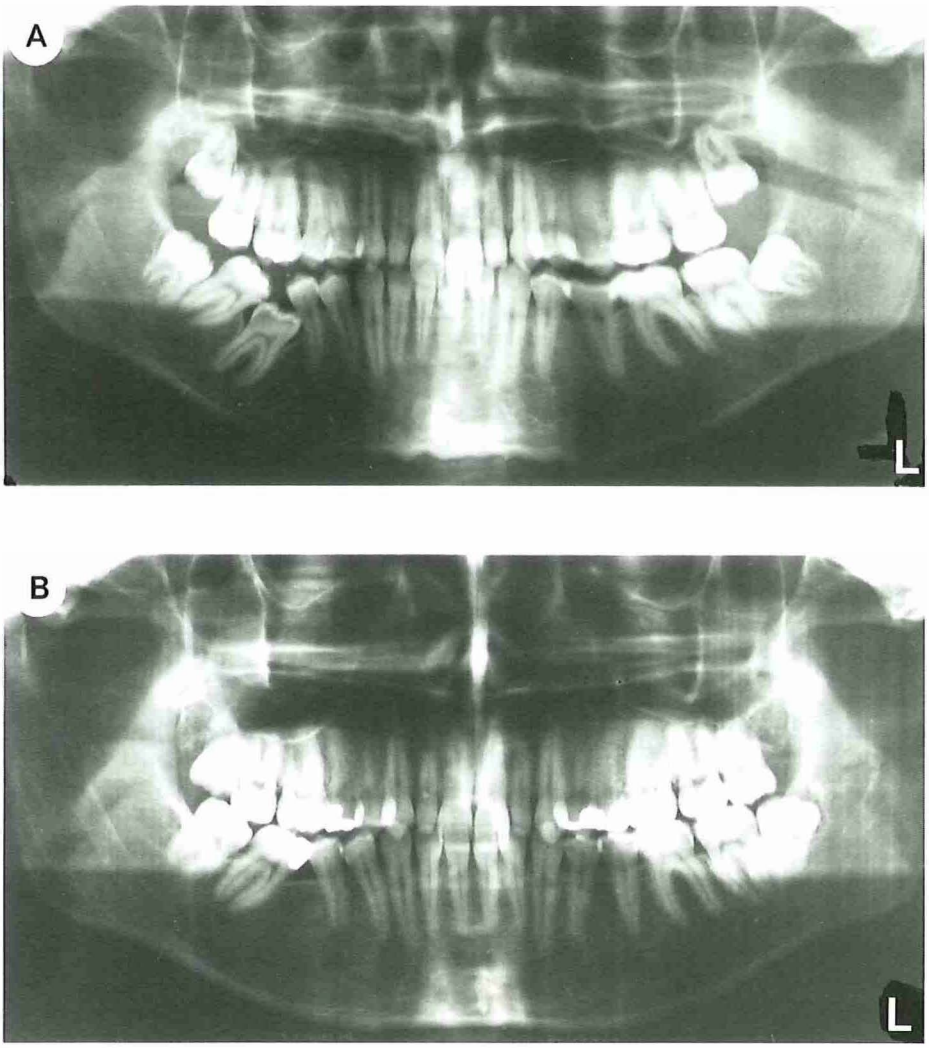


Fig. 3 A Orthopantomogram of a 17-year-old boy showing secondary retention of the mandibular right first molar. **B** Orthopantomogram of the same patient 4 years after removal of the retained molar. The right second molar has tilted toward the second premolar.

Treatment results (Table 2)

No treatment. In 17 of 19 cases in which no treatment had been applied, the extent of infraocclusion remained unchanged (all patients were older than 17 years). In the other two cases, infraocclusion increased by 2 mm in 2 years (patients were 12 and 13 years, respectively, at the time of diagnosis). One of these molars was removed.

Surgical removal. In four of eight cases in which the mandibular first molar had been removed without any further treatment, the diastema between the second

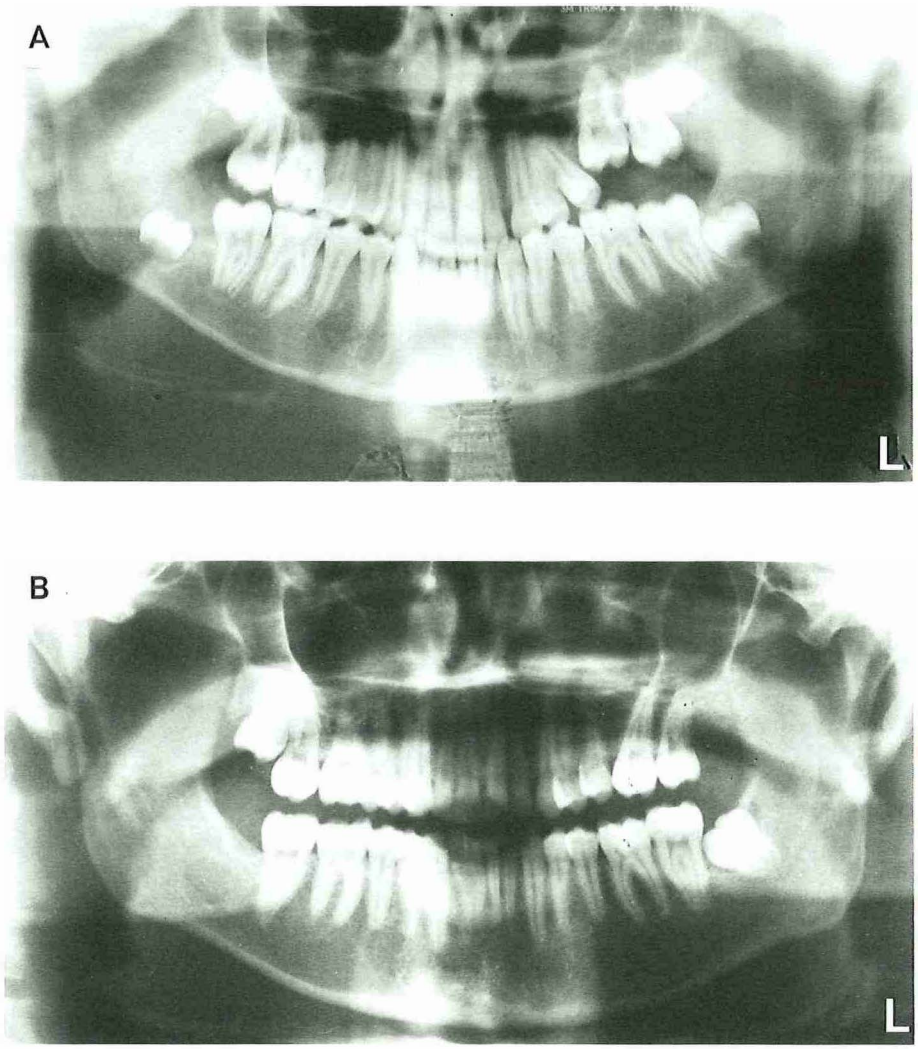


Fig. 4 A Orthopantomogram of a 14-year-old boy showing secondary retention of the maxillary left first molar. **B** Orthopantomogram of the same patient 5 years after removal of the retained molar. The left second and third molars erupted in contact to the second premolar.

molar and second premolar persisted because of the interdigitation with the opposing teeth. In the other four cases, the diastemas closed spontaneously because of mesial and distal migration of the neighbouring teeth (Fig. 3). In the maxilla, the diastema closed spontaneously in seven cases because of tilting and rotation of the neighbouring teeth (Fig. 4). In two cases the diastema persisted. In these cases, the neighbouring molar occluded in a good vertical interdigitation with its antagonist. Surgical removal of a secondarily retained molar followed by orthodontic treatment resulted in closure of the diastema and a reasonably normal occlusion in

all cases (Fig. 5). In five cases, the diastema was closed prosthetically by means of a fixed bridge. No teeth were available for migration in 29 cases, including second molars, because the mesial and/or distal tooth were also removed or were congenitally absent. The accidentally opened antrum healed in all cases without complications. The injured inferior alveolar nerve regenerated within 1 year. The normal second and third molars showed a tendency to erupt earlier after removal of the retained first molar.

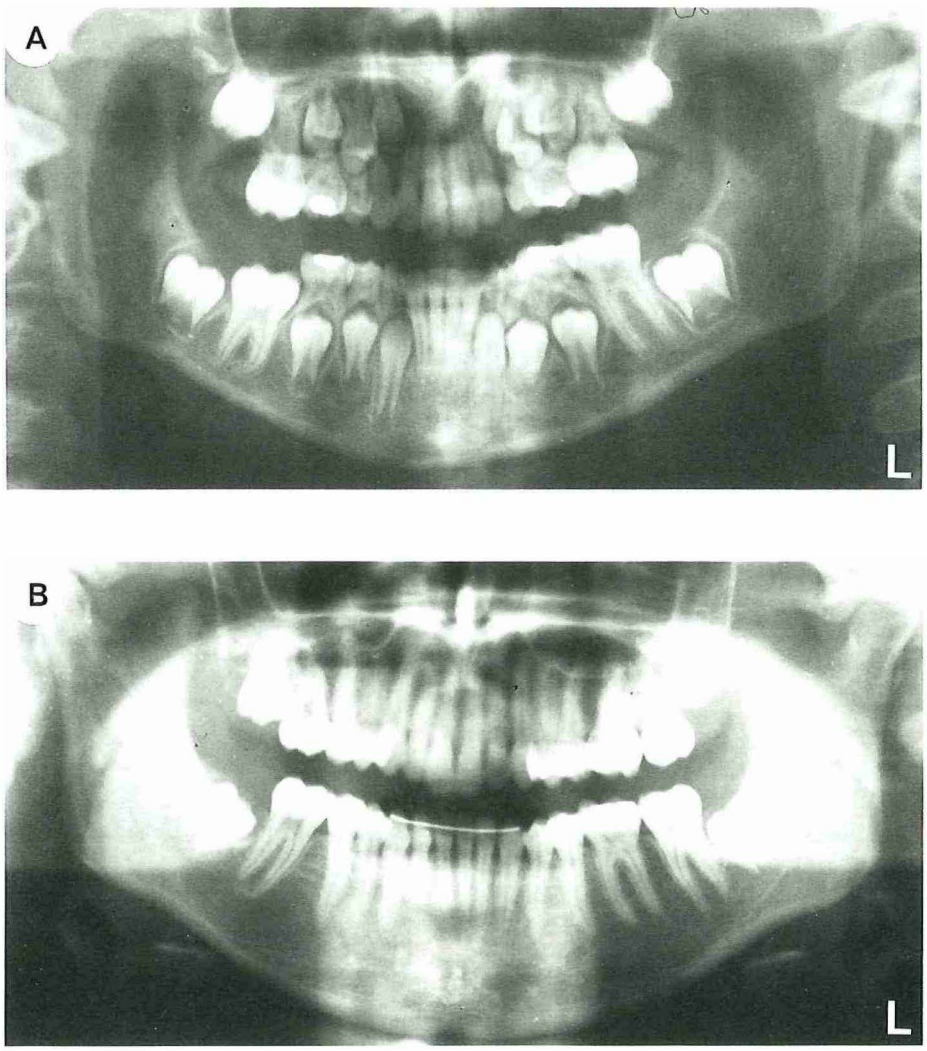


Fig. 5 A Orthopantomogram of a 10-year-old girl showing secondary retention of the mandibular right first molar. **B** Orthopantomogram of the same patient 6 years after removal of the retained molar and its antagonist. The diastemas in both the maxilla and mandible were closed by orthodontic treatment.

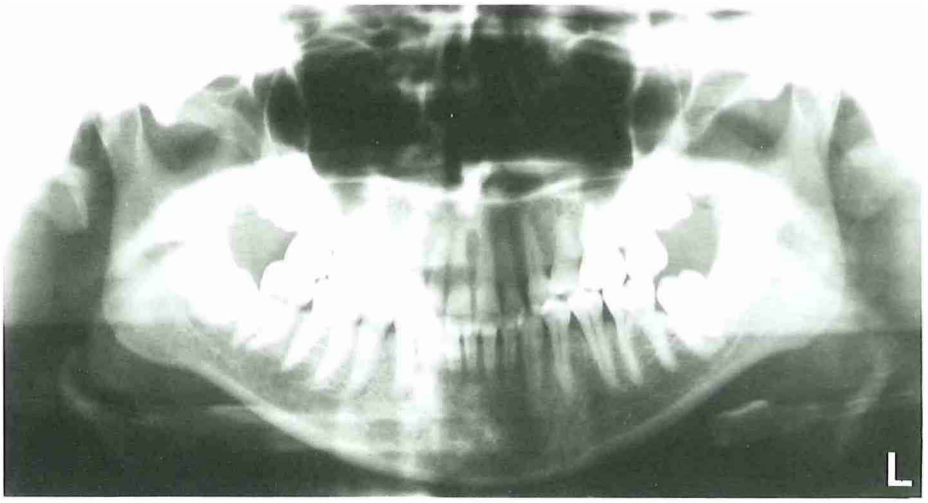


Fig. 6 Orthopantomogram of an 18-year-old girl showing secondary retention of the mandibular left second molar. Two years before the retained molar had been luxated after removal of the third molar. No eruption was observed.

Prosthetic treatment. The build up functioned well and remained in occlusion (Fig. 1D). The follow up period ranged from 1 to 3 years.

Luxation. After luxation by forceps and applying extrusive force, no orthodontic displacement was observed during the follow up period. After 6 months, luxation was repeated in one of these cases. Two luxated molars were eventually removed due to lack of success. The third luxated molar showed no eruptive movement and was left in place (Fig. 6).

Orthodontic treatment. A 1-year period of orthodontic treatment did not result in eruption of the retained molar. The molar was subsequently removed.

New cases. Seven new cases of secondary retention were noted in these patients during the observation period. These patients were between 13 and 17 years of age.

DISCUSSION

From this study it is obvious that treatment of secondarily retained molars should be based on the condition of the tooth, the jaw relationship and occlusion, the dental age of the patient at the time of diagnosis of the disorder, and the patient's attitude about undergoing treatment. If secondary retention develops after the growth spurt, the extent of infraocclusion and its progression is limited. Because of these factors, the retained molar can be build up to restore occlusal and proximal contacts to prevent tilting of neighbouring teeth and over-eruption of the antagonists. Because eruption is a continuous process, it is possible that eventual wear of the prosthetic surface will again result in infraocclusion, necessitating a new build up.

Prompt treatment at the time of discovery appears to be the method of choice when secondary retention develops before the growth spurt. In the majority of the young patients, removal of a retained first molar in the maxilla resulted in spontaneous closure of the diastema owing to mesial migration of the second molar. In the mandible, however, removal of the retained first molar resulted in a permanent diastema or severe tilting of the neighbouring teeth, even though the lower premolars have a greater tendency to migrate distally than upper premolars.[15] The observed jaw-related differences in closure of the diastema may be related to differences in the development of the second molars in the maxilla and in the mandible.[16] The crowns of the second and third mandibular molars are oriented mesially and lingually before emergence and they elevate into a more vertical position during normal eruption. If the first molar is removed before emergence of the second molar, this movement occurs to a lesser degree and can result in a mesial tilting of the second molar. In the maxilla, however, the crowns of the second and third molars are distally and buccally angulated before emergence. After removal of the first upper molar, the normal mesial eruption movement of the second molar does not essentially change or may even be exaggerated. Removal of the first molar leads to an earlier emergence of the second and third molar.[17] This was also observed in our study.

During the growth spurt, management of secondary retention depends on the extent of infraocclusion and tilting of the neighbouring teeth (Figs. 1A, 1B). In case of slight infraocclusion and absence of tilting, treatment is limited to a 6-month observation intervals. If infraocclusion is progressive or tilting of the neighbouring teeth occurs, removal of the retained molar and subsequent orthodontic treatment are indicated.

An alternative for closing the diastema is transplantation of the third molar into the socket of the removed molar. Transplantation of a molar, however, can result in root resorption and ankylosis, particularly in older patients. An additional complication is the minor vertical dimension of the alveolar process in the region of the retained molar. If the third molar is unerupted, an autotransplant probably has a greater chance of success.[18] A small segment alveolar bone osteotomy has been reported, but this may result in a tooth that is 1 mm to 2 mm out of occlusion because of retraction of the segment during healing.[9,13] In our study, no segment osteotomy was performed because in our cases the neighbouring teeth were tilted or there was a high risk of damaging the inferior alveolar nerve. In our opinion, this technique is only indicated for secondarily retained maxillary second molars.

According to literature, the luxation technique has been used with considerable success.[12] However, if timed correctly, extraction is a more conservative treatment in case of a secondarily retained first molars.[19] The luxation technique assumes that if the periapical blood supply is preserved, a new fibrous ligament in the ankylotic area will be formed in the ankylotic region because of local inflammation due to disturbance of that region. The biological mechanism underlying this technique is unknown. In our opinion, luxation may promote further ankylosis rather than eliminate the ankylotic area. In a histological study of secondarily retained perma-

nent molars, ankylosis was observed in the interradicular and bifurcation area in 81% of the cases.[7] Luxation of these molars does not easily result in a disturbance of the bony connections in these areas because the rotation point during extracting is the bifurcation.

Secondarily retained teeth do not respond to orthodontic treatment because of an abnormal periodontal ligament.[9,20,21] This is in accordance with the case reported in this study.

On the basis of the results presented it can be concluded that treatment of secondary retention deserves a careful evaluation. In young patients (before the growth spurt), secondarily retained permanent molars should be removed after orthodontic consultation. During the growth spurt, no invasive treatment is indicated when infraocclusion is slight and nonprogressive, and the neighbouring teeth are not tilted; otherwise, a similar treatment as used for young patients is advisable. In adult patients (after the growth spurt), a prosthetic build up is often the proper treatment.

REFERENCES

1 Raghoebar GM, Abma G, Boering G, Stegenga B. Secundaire retentie van melkmolaren. Ned Tijdschr Tandheelkd 1988;95:389–92.
2 Biederman W. Tooth ankylosis. Ann Dent 1953;12:1–15.
3 Sterrett DS. Report of a case of a submerging first molar. Am J Orthod Oral Surg 1940;26:681–3.
4 Dayan D, Littner MM, Gonshorowitz J, Mersel A. Reimpaction of a first permanent maxillary molar due to an obscure idiopatic etiology. Clin Prevent Dent 1983;5:22–4.
5 Korkhaus G. Die Halbretention des ersten Molaren. Dtsch Zahnärtzl Z 1952;7:1205–12.
6 Biederman W. The incidence and etiology of tooth ankylosis. Am J Orthod 1956;42:921–6.
7 Raghoebar GM, Boering G, Jansen HWB, Vissink A. Secondary retention of permanent molars: a histologic study. J Oral Pathol Med 1989;18:427–431.
8 Bosker H, Ten Kate LP, Nijenhuis LE. Familial reinclusion of permanent molars. Clin Genet 1978;13:314–20.
9 Proffit WR, Vig KWL. Primary failure of eruption; a possible cause of posterior open-bite. Am J Orhtod 1981;80:173–90.
10 Adams D. An impacted permanent first molar. Oral Surg Oral Med Oral Pathol 1975;40:702–3.
11 Hall GM, Reade PC. Quadrilateral submersion of permanent teeth. Brief review and case report. Aus Dent J 1981;26:73–6.
12 Biederman W. Etiology and treatment of tooth ankylosis. Am J Orthod 1962;48:670–84.
13 Piret JL, Wackens G. Eruptiestoornissen van definitieve molaren. Acta Stomatol Belg 1988;85: 289–97.
14 Albers DD. Ankylosis of teeth in the developing dentition. Quintessence Int 1986;17:303–8.
15 Plint DA. The effect on the occlusion of the loss of one or more permanent molars. Trans Eur Orthod Soc 1970;46:329–35.
16 Van der Linden FPGM. Problemen en procedures in de orthodontie. Alphen aan den Rijn: Samsom Stafleu, 1986:278–83.
17 Fanning EA. Third molar emergence in Bostonians. Am J Phys Anthrop 1962;20:339–46.
18 Hernandez SL, Cuestas-Carnero R. Autogenic tooth transplantation: a report of ten cases. J Oral Maxillofac Surg 1988;46:1051–55.
19 Moyers RE. Handbook of orthodontics. 3rd ed. Chicago: Yearbook Med Publ, 1974:530.
20 Oliver RG, Richmond S, Hunter B. Submerged permanent molars: four case reports. Br Dent J 1986;160:128–30.
21 Mitchell DL, West JD. Attempted orthodontic movement in the presence of suspected ankylosis. Am J Orthod 1975;68:404–11.

SUMMARY

Eruption is the axial or occlusal movement of the tooth from its developmental position within the jaw to its functional position in the occlusal plane. It continues throughout life to compensate for growth of the jaws and occlusal wear. Eruption disturbances of molars can lead to severe malocclusion, and to loss of the affected tooth and neighbouring teeth due to plaque accumulation followed by caries or periodontal disease. *Secondary retention* refers to the cessation of eruption neither due to a physical barrier in the path of eruption nor as a result of an abnormal position. The criteria currently used to diagnose secondary retention are confusing and often not reliable. The etiology of secondary retention is essentially unknown, but ankylosis has been suggested as being the main factor. There are no proper guidelines for treatment of this eruption disorder, especially when it occurs in the permanent dentition.

The aim of this study was to obtain insight in the present knowledge of secondary retention and other eruption disturbances of molars, to state factors that may contribute to the etiopathogenesis, to state reliable diagnostic criteria, and to recommend guidelines for proper treatment. These goals were achieved by:

- a review of the literature regarding secondary retention (chapter 2);
- a study of the clinical, radiographic and histological characteristics and recommendations for proper treatment of secondary retention in the deciduous dentition (chapter 3);
- a description of the eruption disturbances underlying impaction, primary retention and secondary retention in the permanent dentition (chapter 4);
- a study of the clinical and radiographic characteristics of secondary retention of permanent molars (chapter 5);
- a study of the role of hereditary factors of secondary retention of permanent molars (chapter 6);
- a study of the histological characteristics of secondary retention of permanent molars (chapter 7);
- an evaluation of the outcomes of different approaches of treatment and a proposal for guidelines for proper treatment of secondary retention of permanent molars (chapter 8).

In **chapter 2**, a review of the literature regarding secondary retention in the deciduous and permanent dentition is presented. Secondary retention is less frequently observed in permanent than in deciduous molars, but it can lead to severe malocclusion and loss of neighbouring teeth, particularly in the permanent dentition. The etiology of secondary retention in both the deciduous and permanent dentition is probably the same. The factors causing secondary retention are most likely of local origin and are probably due to remodelling disturbances in the periodon-

tal ligament. On the basis of histological studies in deciduous molars, ankylosis is generally accepted as an important factor in the etiopathogenesis of secondary retention. Although there are two case studies reported in the literature stating that ankylosis is also involved in secondary retention of permanent molars, this aspect must be studied in more detail. Clinically, percussion tests and partial absence of the periodontal ligament space on radiographs have been suggested to be valuable additional tools to the observation of infraocclusion in the diagnosis of secondary retention. There seems to be no consensus concerning the treatment of secondary retention of permanent molars in particular. Orthodontic movement of the affected molar is not possible.

In a group of 34 patients with 77 secondarily retained *deciduous molars*, the clinical, radiographic, and histological features of secondary retention and its treatment are evaluated (**chapter 3**). Thirty-two of the 41 deciduous molars studied histologically, showed small local areas of ankylosis. The clinical signs of ankylosis (solid, clear percussion sound, and partial absence of periodontal ligament space) turned out to be unreliable to detect ankylosis (many false negative results). In nine patients, secondary retention was observed in both deciduous and in permanent molars in the same dentition. Therefore, secondarily retained deciduous molars probably may predispose to a similar disorder in the permanent dentition. The recommended treatment policy, which is in accordance with the view of Kurol, is to wait for normal eruption of the permanent successor, because in most cases occlusal disturbances will recover spontaneously. A follow up is necessary to observe the occlusal development and to detect new cases of secondary retention in the same patient. Active therapy in the deciduous dentition, for example extraction, is only indicated in case of severe infraocclusion, severe malocclusion, and dislocation or agenesis of the successor.

In **chapter 4**, the eruption disturbances reported in *permanent molars* are analyzed. Eruption disturbances of permanent molars may become clinically and radiographically manifest as *impaction, primary retention, or secondary retention*. Which of these disorders develops, depends primarily on the eruptive stage. *Primary retention* is the cessation of eruption before emergence neither due to a physical barrier in the eruption path nor as a result of an abnormal position. *Impaction* is cessation of eruption caused by a clinical and radiographically detectable physical barrier in the eruption path or due to an abnormal position of the tooth. Treatment recommendations of these three eruption disturbances are given. The clinical features and the guidelines for treatment as derived from the data presented in chapters 5–8 and reported in literature are summarized in Table 1 (p.32) and Fig. 6 (p.37).

In **chapter 5**, the clinical and radiographic aspects of secondary retention in *permanent molars* are described. In a large group of 53 patients with 81 secondarily retained permanent molars these features were examinated (**section 5.1**). First molars turned out to be affected most frequently. The mean infraocclusion at the

patients' first visit was 4.3 mm. After six months, infraocclusion had increased in adolescents. It seemed to be stable in adults. Tilting of adjacent teeth was observed in 39 cases of secondary retention. A solid, clear percussion sound and a partial absence of the periodontal ligament space on radiographs were noted in less than 20% of the affected molars, while histological examination of 38 removed molars revealed that local areas of ankylosis were present in all cases. From the histological, percussion and radiographic data of the 38 removed molars a sensitivity of the percussion test and of radiographs to detect ankylotic areas of, respectively, 29% and 21% was calculated. This implies that percussion tests and judgement from radiographs result in many false negative outcomes and are thus non-reliable diagnostic tools. During a follow up period of four years, six new cases of secondary retention were observed in the same population.

In **section 5.2**, three cases of secondary retention of the first permanent molar and impaction of the adjacent molar in the mandible are reported to elucidate some of the clinical problems. Histologically, the roots of the secondarily retained molars showed the presence of local areas of ankylosis, while these were completely absent on the roots of the impacted molars. This difference has not been described before in the literature and supports the concept described in chapter 4. Early detection of secondary retention followed by prompt treatment greatly minimizes the progressive negative effects of this dental condition such as the development of severe malocclusion and impaction of the adjacent molar (chapter 8).

Spontaneous re-eruption of a secondarily retained permanent molar is a very rare phenomenon and has never been reported in the literature before (**section 5.3**). The case described is in accordance with the theory that secondary retention is caused by local areas of ankylosis and that these areas can be resorbed during remodelling of the bone of the alveolar process.

In **chapter 6**, first degree relatives of 52 patients with secondary retention of *permanent molars* are screened to study the familial occurrence. Familial occurrence could be demonstrated in five families. The pedigrees agreed with autosomal dominant inheritance as suggested in the literature. HLA phenotypes and bloodgroup ABO, rhesus and P_1 were studied in two positive families. The lod scores for linkage with secondary retention were added to previously reported information. The lod score for linkage for bloodgroup P_1 increased from +0.940 to +1.475 at a recombination fraction of 5%. The HLA phenotypes and bloodgroup ABO and rhesus showed negative lod scores. It was concluded that secondary retention of permanent molars is an etiologically heterogeneous condition of which some cases are caused by the presence of an abnormal autosomal dominant gene. Because of a familial occurrence over 10%, it is advisable to examine family members, especially the younger ones, of each patient with secondary retention to detect this disorder as early as possible in order to enable adequate treatment.

In **chapter 7**, the histological features of secondary retention of *permanent molars* are described. Twenty-six secondarily retained permanent molars and six normal

molars were examined histologically in order to detect the frequency of occurrence, extent and localization of ankylosis (**section 7.1**). Local areas of ankylosis were observed along roots of all secondarily retained molars. In 81% of the cases these areas were located at the bifurcation and interradicular root surface. No signs of ankylosis were found in control molars of the same dentition that had to be removed also. The roots of 12 other affected molars and two normal molars were examined by means of scanning electron microscopy (SEM) and light microscopy (LM) (**section 7.2**). With SEM it was observed that the root surface of retained molars showed local areas covered with bonelike tissue. LM of these areas showed that this tissue was bone indeed, that was in direct contact with the root surface. Ten to 60% of the whole root surface was ankylotic. In the five molars that were clinically suspected to be ankylotic (solid, clear percussion sound and partial absence of the periodontal ligament space) more than 40% of the root surface was ankylotic.

The morphological and histological observations outlined in this chapter, make it quite probable that local areas of ankylosis between the root and alveolar bone are a significant factor in secondary retention of permanent molars.

In **chapter 8**, the efficacy of five different treatment modalities for secondary retention of *permanent molars* is evaluated in 59 patients with 88 affected molars. The aim of this study was to develop guidelines for treatment of this disorder as the current knowledge is mainly based on case reports. The results showed that a prosthetic build up is the proper treatment if secondary retention develops after the growth spurt. In these cases the extent of infraocclusion is slight and relatively stable. If secondary retention starts before the growth spurt, immediate removal of the retained molar followed by orthodontic treatment to close the diastema gives maximal success. When secondary retention develops during the growth spurt, the tooth affected has to be observed at six monthly intervals. In such cases, no active treatment is indicated if the neighbouring teeth do not show tilting and if the extent of infraocclusion is minor and stable. A prosthetic build up can be made to avoid tilting of the neighbouring teeth and over-eruption of the antagonist. In all other cases the affected molar should be removed, followed by orthodontic closure of the diastema. Orthodontic treatment and luxation of the affected molar was not successful.

Eruption disturbances of molars cannot be considered as a curiosity. Therefore the knowledge about these phenomena should be a substantial component of the dentist's, orthodontist's and oral and maxillofacial surgeon's basic knowledge. Early detection of secondary retention followed by timely treatment can minimize progressive negative effects of these eruption disturbances, such as the development of a severe malocclusion.

SAMENVATTING

Onder de eruptie van een gebitselement wordt verstaan de continue beweging van dit element vanaf de plaats van aanleg in de kaak tot het maken van contact met de antagonist. Na volledige doorbraak gaat de eruptie in vertraagd tempo door ter compensatie van de groei van de kaken en de occlusale afslijting. Gestoorde eruptie van een molaar kan leiden tot een stoornis in de gebitsontwikkeling, occlusie en articulatie. De buurelementen kunnen kippen naar het diasteem en de antagonist kan uitgroeien. De molaar met een eruptiestoornis en de buurelementen hebben een grotere kans om vroegtijdig verloren te gaan als gevolg van cariës en/of parodontitis.

Onder *secundaire retentie* wordt verstaan het stoppen van de eruptie van een gebitselement na aanvankelijke doorbraak in de mondholte zonder dat er sprake is van een duidelijk aanwijsbare fysische barrière in het eruptietraject of van een abnormale ligging van dit element. Diagnostische criteria voor secundaire retentie zijn moeilijk aan te geven en zijn niet geheel betrouwbaar. De etiologie is nog onbekend. Men vermoedt dat ankylose tussen wortel en kaakbot een belangrijke rol speelt in de etiopathogenese. Er bestaan geen eenduidige richtlijnen voor een adequate behandeling van secundaire retentie in met name het blijvend gebit.

Het doel van dit onderzoek was het verwerven van kennis betreffende eruptiestoornissen van gebitselementen, het opsporen van etiologische factoren voor het onstaan van secundaire retentie, het vaststellen van betrouwbare diagnostische criteria en het opstellen van richtlijnen voor een adequate behandeling (**hoofdstuk 1**).

Deze doelstellingen werden nagestreefd door:

- het inventariseren en compileren van de literatuur over etiologie, klinische kenmerken en behandeling van secundaire retentie van melk- en blijvende molaren (hoofdstuk 2);
- het bestuderen van de klinische, röntgenologische en histologische kenmerken, en het opstellen van richtlijnen voor een adequate behandeling van secundaire retentie van melkmolaren (hoofdstuk 3);
- het beschrijven van de eruptiestoornissen in het algemeen zoals impactie, primaire retentie en secundaire retentie van blijvende molaren (hoofdstuk 4);
- het bestuderen van de klinische en röntgenologische kenmerken van secundaire retentie van blijvende molaren (hoofdstuk 5);
- het bestuderen van de eventuele rol van erfelijke factoren bij secundaire retentie van blijvende molaren (hoofdstuk 6);
- het bestuderen van de histologische kenmerken van secundaire retentie van blijvende molaren (hoofdstuk 7);

- het evalueren van de resultaten van de verschillende behandelingsmogelijkheden en het opstellen van richtlijnen voor een adequate behandeling van secundaire retentie van blijvende molaren (hoodstuk 8).

In **hoofdstuk 2** wordt een overzicht gegeven van de literatuur over secundaire retentie van melk- en blijvende gebitselementen. Secundaire retentie wordt vaker in het melkgebit gezien dan in het blijvend gebit. Vooral in het blijvend gebit kan secundaire retentie leiden tot ernstige occlusie- en articulatiestoornissen. Het verhoogde risico op het ontstaan van cariës en parodontitis dat hieruit voortvloeit, kan leiden tot vroegtijdig verlies van het desbetreffende gebitselement en zijn buurelementen.

De etiologie van secundaire retentie in het melkgebit en het blijvend gebit is zeer waarschijnlijk identiek. De oorzaak is vermoedelijk een locale stoornis in de continue ombouw in het parodontale ligament. Bij histologisch onderzoek van secundair geretineerde melkmolaren krijgt men sterk de indruk, dat locale gebieden met ankylose een belangrijke etiopathologische factor vormen. Het is echter niet duidelijk of de ankylose vóór of ná het stoppen van de eruptie optreedt. Ook erfelijke factoren spelen waarschijnlijk een rol bij het ontstaan van deze afwijking.

Het belangrijkste klinische criterium voor het stellen van de diagnose secundaire retentie is infrapositie. Het occlusale vlak van de molaar bevindt zich 1 mm of meer beneden het niveau van het occlusievlak van de gebitsboog op een leeftijd waarbij dit gebitselement in occlusie zou moeten staan. Sommige auteurs gebruiken de percussietest en het plaatselijk ontbreken van de weergave van het parodontale ligament op de röntgenfoto om ankylose van de betreffende molaar te bewijzen, waardoor de diagnose secundaire retentie met meer zekerheid zou kunnen worden vastgesteld. Deze laatste twee hulpmiddelen kunnen met name van diagnostische waarde zijn bij het maken van een onderscheid tussen secundaire retentie en primaire retentie of impactie.

De richtlijnen voor de behandeling van secundaire retentie van zowel melkmolaren als blijvende molaren zijn niet eenduidig. Deze omvatten geen behandeling (observatie), het occlusaal opbouwen van het aangedane gebitselement tot occlusieniveau of extractie. Orthodontische verplaatsing van een secundair geretineerd element wordt als onmogelijk beschouwd.

In een groep van 34 patiënten met 77 secundair geretineerde *melkmolaren* werden de klinische, röntgenologische en histologische kenmerken van secundaire retentie bestudeerd (**hoofdstuk 3**). De gevolgen van secundaire retentie voor de gebitsontwikkeling werden in deze groep geëvalueerd en gerelateerd aan de ingestelde therapie. Uit histologisch onderzoek bleek dat 32 van de 41 geëxtraheerde melkmolaren ankylose vertoonden. De door sommige onderzoekers voorgestelde criteria voor ankylose (een hogere percussietoon dan een niet-ankylotisch element en het plaatselijk ontbreken van het parodontale ligament op de röntgenfoto) bleken onvoldoende betrouwbaar te zijn. Bij negen patiënten werden behalve secundair geretineerde melkmolaren ook secundair geretineerde blijvende molaren gecon-

stateerd. Mogelijk bestaat er een predispositie voor het ontstaan van een vergelijkbare afwijking in zowel het melkgebit als in het blijvend gebit.

De meest adequate behandeling van secundaire retentie in het melkgebit is een terughoudend extractiebeleid. De wisseling wordt weliswaar vertraagd, maar verloopt in de meeste gevallen normaal zonder blijvende gevolgen voor de rest van het gebit. Een normale eruptie van blijvende gebitselementen werd waargenomen in 97% van de gevallen waarin geen behandeling plaatsvond en in alle gevallen waarbij na extractie van het geretineerde gebitselement het dichtgaan van het diasteem werd verhinderd met behulp van een spacemaintainer. Alleen extractie van het geretineerde element, zonder openhouden van het diasteem, resulteerde daarentegen in slechts 63% van de gevallen in een normale eruptie. Het regelmatig controleren van de patiënt is noodzakelijk om de ontwikkeling van de occlusie te beoordelen en om nieuwe gevallen van secundaire retentie tijdig op te sporen. In deze studie werden twee nieuwe gevallen van secundaire retentie in zowel het melkgebit als in het blijvend gebit waargenomen.

In **hoofdstuk 4** wordt een overzicht gegeven van de eruptiestoornissen van *blijvende molaren*. Deze eruptiestoornissen kunnen op grond van klinische en röntgenologische bevindingen worden onderverdeeld in *primaire retentie, secundaire retentie* en *impactie*. Afhankelijk van het stadium van eruptie, waarin de stoornis optreedt, wordt deze benoemd. *Primaire retentie* is het stoppen van de eruptie van een gebitselement vóór doorbraak in de mondholte. In dit geval is er géén sprake van een fysische barrière in het eruptietraject of van een abnormale ligging van dit element. *Secundaire retentie* is reeds eerder gedefinieerd. *Impactie* is het stoppen van de eruptie van een gebitselement door een klinisch en röntgenologisch waarneembare fysische barrière in het eruptietraject of door een abnormale ligging van dit element.

De klinische kenmerken en de verschillende behandelingsmogelijkheden, ontleend aan de gegevens beschreven in de hoofdstukken 5-8 en gerapporteerd in de literatuur, zijn schematisch weergegeven in Tabel 1 (p.32) en Fig. 6 (p.37).

In **hoofdstuk 5** zijn de klinische en röntgenologische kenmerken van secundaire retentie in het *blijvend gebit* beschreven. In totaal werden 53 patiënten met 81 secundair geretineerde molaren onderzocht (**paragraaf 5.1**). In 65 gevallen was de eerste molaar secundair geretineerd. De mate van infraocclusie bedroeg bij het eerste bezoek van de patiënten gemiddeld 4.3 $\pm$ 1.9 mm. Na zes maanden was er toename van de infraocclusie bij adolescenten, terwijl de infraocclusie bij volwassenen gelijk was gebleven. In 39 gevallen bestond een matige tot ernstige kipping van de buurelementen. Een percussietoon suggestief voor ankylose en het plaatselijk ontbreken van het parodontale ligament op de röntgenfoto werden slechts bij ongeveer 20% van de secundair geretineerde gebitselementen waargenomen. Histologisch werden bij de 38 onderzochte geretineerde molaren locale gebieden met ankylose aangetoond. Op grond van deze gegevens werd een sensitiviteit van 29% voor de percussietest en van 21% voor het plaatselijk ontbreken van het pa-

rodontale ligament op röntgenfoto's berekend. Dit betekent dat, bij verdenking op secundaire retentie, een normale percussietoon en een continu parodontaal ligament op een röntgenfoto geen bewijs vormen voor het afwezig zijn van een ankylotisch proces. Waarschijnlijk is in een groot aantal gevallen de omvang van de locale gebieden met ankylose te gering om door middel van percussie en röntgenfoto's te kunnen worden aangetoond. Tijdens het prospectief observeren van de patiënten over een periode van gemiddeld vier jaar werden zes nieuwe gevallen van secundaire retentie waargenomen.

In **paragraaf 5.2** zijn drie patiënten met secundaire retentie van de eerste blijvende molaar in de onderkaak en impactie van de tweede molaar in hetzelfde kaakgedeelte beschreven ter illustratie van de klinische problemen die kunnen ontstaan. Locale gebieden met ankylose werden bij alle drie secundair geretineerde elementen waargenomen. Dergelijke gebieden werden niet bij de geïmpacteerde elementen waargenomen.

Spontane reëruptie van een secundair geretineerde blijvende molaar is een zeldzaam verschijnsel (**paragraaf 5.3**). Er wordt één patiënt beschreven waarbij dit optrad. Dit is in overeenstemming met de opvatting, dat secundaire retentie berust op een gestoord eruptiemechanisme, veroorzaakt door locale gebieden met ankylose. Deze gebieden zouden mogelijk tijdens de fysiologische ombouw van de processus alveolaris kunnen worden geresorbeerd.

In **hoofdstuk 6** zijn de resultaten beschreven van een onderzoek naar het voorkomen van dezelfde afwijking in het gebit van eerstegraads bloedverwanten van 52 patiënten met secundaire retentie van *blijvende molaren*. Uit eerdere publicaties van onze afdeling en uit klinische waarnemingen werd de indruk gewekt dat secundaire retentie in het blijvend gebit familiair voorkomt. Een positieve familiaire samenhang werd bij vijf van de 53 patiënten geconstateerd. Uit de stambomen van deze vijf patiënten komt naar voren dat de overerving autosomaal dominant verloopt. HLA fenotypen en bloedgroepen A, B, O, rhesus en P_1 werden in twee positieve families bestudeerd. De lodscore werd opgeteld bij de in de literatuur bekende gegevens waardoor de lodscore van bloedgroep P_1 toenam van +0.940 tot +1.475 met een recombinatie fractie van 5%. De HLA fenotypen en bloedgroepen A, B, O en rhesus vertoonden een negatieve lodscore.

Uit dit onderzoek kan worden geconcludeerd, dat secundaire retentie van blijvende molaren een heterogene conditie is met in sommige gevallen een abnormaal autosomaal dominant gen dat verantwoordelijk is voor de afwijking. Het voorkomen van secundaire retentie in de onderzochte families bleek meer dan 10% te bedragen. Het advies wordt daarom gegeven om bij elke patiënt met secundaire retentie, ook de familieleden en in het bijzonder de jongere kinderen, te onderzoeken. Eventuele afwijkingen kunnen op deze manier zo vroeg mogelijk opgespoord worden.

In **hoofdstuk 7** zijn de histologische kenmerken van secundaire retentie van *blijvende molaren* beschreven. Zesentwintig secundair geretineerde en zes normale

molaren werden histologisch onderzocht op de aanwezigheid en de localisatie van ankylose (**paragraaf 7.1**). Locale gebieden met ankylose werden bij alle secundair geretineerde molaren geconstateerd. In 81% van de gevallen waren deze gebieden gelocaliseerd in de bifurcatie en/of op het interradiculaire wortelоppervlak van de molaren. Bij de normale molaren, welke geëxtraheerd waren bij patiënten uit dezelfde groep, werd geen ankylose aangetroffen.

De wortels van 12 andere secundair geretineerde blijvende molaren en twee normale molaren werden met behulp van scanning electronen microscopie (SEM) en licht microscopie (LM) onderzocht (**paragraaf 7.2**). Het SEM beeld van de wortels van de secundair geretineerde molaren vertoonde locale gebieden op het wortelоppervlak die bedekt waren met op bot gelijkend materiaal. Met behulp van LM kon worden aangetoond, dat in deze gebieden bot in direct contact stond met het wortelоppervlak (ankylose). Deze ankylotische gebieden bestreken 10% tot 60% van het totale wortelоppervlak. In de vijf gevallen die klinisch suspect waren voor de aanwezigheid van ankylose (scherpe, hoge percussietoon en het plaatselijk ontbreken van het parodontale ligament op de röntgenfoto) was meer dan 40% van het totale wortelоppervlak ankylotisch.

Uit bovenstaand morfologisch en histologisch onderzoek werd sterk de indruk verkregen dat locale gebieden met ankylose een belangrijke rol spelen in de etiopathogenese van secundaire retentie van blijvende molaren.

In **hoofdstuk 8** zijn vijf verschillende behandelingswijzen voor secundaire retentie in het *blijvend gebit* geëvalueerd bij 59 patiënten met 88 secundair geretineerde molaren. Op grond hiervan konden richtlijnen worden opgesteld voor een adequate behandeling van deze afwijking. Indien secundaire retentie optreedt **ná** de groeispurt en de infrapositie gering is, dan kan het element vaak worden behouden, omdat de toename van infrapositie meestal gering tot nihil is. De opbouw van het occlusale vlak met behulp van composiet of met een kroon is in deze gevallen een goede therapie. Het kippen van de buurelementen en het uitgroeien van de antagonisten kan hierdoor worden voorkomen. Als secundaire retentie optreedt **vóór** de groeispurt, zal een zo vroeg mogelijke verwijdering van het geretineerde element meestal het beste klinische resultaat opleveren. In dit stadium neemt de infrapositie vaak sterk toe met als gevolg kipping van buurelementen of uitgroei van de antagonisten. Het uiteindelijke gevolg kan een ernstige occlusiestoornis zijn. Deze ernstige afwijkingen kunnen door vroegtijdige verwijdering worden voorkomen. Orthodontische vervolgbehandeling voor het sluiten van het extractiediasteem is, in het bijzonder in de onderkaak, aan te bevelen. Als secundaire retentie optreedt **tijdens** de groeispurt en de mate van infrapositie gering is, kan worden volstaan met periodieke observatie. Bij toenemende infrapositie en/of kippen van de buurelementen moet het desbetreffende gebitselement alsnog worden verwijderd. In geen van de beschreven fasen was orthodontische behandeling, eventueel voorafgegaan door luxatie van het geretineerde gebitselement, succesvol.

Eruptiestoornissen van molaren dienen niet te worden beschouwd als een curiositeit. Kennis van deze stoornissen zal ertoe leiden dat de tandarts, de orthodontist en de kaakchirurg deze afwijkingen vroegtijdig herkennen, of hiermee in hun differentiële diagnose rekening moeten houden. Vroegtijdige herkenning van secundaire retentie, gevolgd door adequate behandeling op het juiste tijdstip, kan een cruciale rol spelen bij de preventie van de nadelige effecten van deze eruptiestoornis op de gebitsontwikkeling, de occlusie en de articulatie.

LIST OF PUBLICATIONS

1 Raghoebar GM, Abma G, Boering G, Stegenga B. Secundaire retentie van melkmolaren. Ned Tijdschr Tandheelkd 1988;95:389–92.

2 Raghoebar GM, Boering G, Jansen HWB, Vissink A. Secondary retention of permanent molars: a histologic study. J Oral Pathol Med 1989;18:427–31.

3 Raghoebar GM, Van Koldam WA, Boering G. Spontaneous re-eruption of a secondarily retained permanent lower molar and an unsual migration of a lower third molar. Am J Orthod Dentofacial Orthop 1990;97:82–4.

4 Raghoebar GM, Boering G, Booy C, Vissink A. Treatment of the retained permanent molar. J Oral Maxillofac Surg 1990;48:1033–38.

5 Raghoebar GM, Boering G, Stegenga B, Vissink A. Secondary retention in the primary dentition. J Dent Child 1991;58:17–22.

6 Raghoebar GM, Boering G, Vissink A. Clinical, radiographic and histological characteristics of secondary retention of permanent molars. J Dent (in press).

7 Raghoebar GM, Boering G, Vissink A, Stegenga B. Eruption disturbances of permanent molars. A Review. J Oral Pathol Med (in press).

8 Raghoebar GM, Boering G. An unerupted deciduous molar. Oral Surg Oral Med Oral Pathol (in press).

9 Raghoebar GM, Vissink A, Boering G. Secondary retention a possible cause for impaction of permanent molars in the same dentition. Quintessence Int (in press).

10 Raghoebar GM, Ten Kate LP, Hazenberg CAM, Boering G, Vissink A. Secondary retention of permanent molars. A report of five new families. J Craniofac Genet Dev Biol (submitted).

11 Raghoebar GM, Jansen HWB, Jongebloed WL, Boering G, Vissink A. Secondary retention of permanent molars: an assessment by scanning electron and light microscopy. Br J Oral Maxillofac Surg (submitted).

CURRICULUM VITÆ

Gerry Max Raghoebar werd op 29 november 1959 geboren te Paramaribo, Suriname. In 1978 behaalde hij het atheneum B diploma aan de Algemene Middelbare School te Paramaribo. In hetzelfde jaar begon hij met de studie Tandheelkunde aan de Rijksuniversiteit Groningen, waar hij in maart 1984 het tandartsdiploma behaalde. Van september 1984 tot september 1988 werd hij opgeleid tot specialist in de Mondziekten en Kaakchirurgie aan de Afdeling Mondziekten, Kaakchirurgie en Bijzondere Tandheelkunde (Hoofd: Prof. Dr. G. Boering) van het Academisch Ziekenhuis Groningen. Vanaf september 1988 is hij als kaakchirurg verbonden aan deze afdeling waar hij zich in het bijzonder toelegt op de preprothetische chirurgie en orale implantologie.

Address for correspondence:
Gerry M. Raghoebar
Dept. of Oral and Maxillofacial Surgery
University Hospital Groningen
Oostersingel 59
P. O. Box 30.001
9700 RB GRONINGEN
The Netherlands