Katrin Bekes

Molar Incisor Hypomineralization

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Preface

"Chalk teeth: Dentists warn of new widespread disease MIH," "MIH: Are chalk teeth the new construction site in the mouth?", "The mysterious crumbling" – these or similar headlines appeared in the media in 2018. A press conference and press release of the German Society of Dentistry and Oral Medicine, in which attention was drawn to the increasing problem of molar incisor hypomineralization (MIH), started the media's attention to this mysterious oral health problem. An alarming trend had appeared, the incidence of the disease had already overtaken the incidence of caries in certain age groups.

Since then, media interest is unabated. Not only in scientific publications, but also in the general press, the topic is taken up again and again, both by the German Press Agency (dpa) and regional media outlets. However, the phenomenon of MIH is not new at all. As early as 2001, the term appeared in the literature. By definition, it is a qualitative enamel developmental defect in one or more 6-year molars with or without involvement of the permanent incisors. Characteristic features are opacities of the affected teeth with partially occurring posteruptive enamel breakdowns as well as a sometimes-occurring strong hypersensitivity of hypomineralized teeth.

MIH has become a much-discussed topic of interest that currently presents major challenges for dental professionals in practice and research worldwide and will likely continue to do so in the future.

This book serves as a comprehensive reference that critically appraises the controversies around MIH found in the scientific literature. Practical advice about diagnosis and treatment of the disease is provided, and the various clinical treatment options are explained in detail. The text is written for dental practitioners, postgraduate and undergraduate students and researchers, as well as anyone who would like to expand their knowledge of this contemporary topic in everyday clinical dentistry.

In the first chapters, the clinical appearance and structural features of hypomineralized enamel are described. In addition, current prevalence figures are presented and possible etiological factors are discussed. The following chapters focus on clinical aspects of the disease. Diagnostic and differential diagnostic criteria are explained, and classification options and treatment strategies are described. Subsequently, various treatment options are presented in detail – from pain control, desensitization, and preventive measures to different restorative approaches and extraction. In the last chapters, the clinical MIH picture in the primary dentition is highlighted and the possible association of MIH with the occurrence of caries is examined.

Beyond that, this book would like to show you further clinical perspectives in the handling of the disease and, based on that, resulting research needs. Ultimately, it is intended to sharpen your understanding of why we are still unable to conclusively answer all the questions that are of urgent interest to us. For example, the etiology of the disease – despite several attempts for explanation – is still unknown. Without knowing the necessary and sufficient causes of the disease, we are only able to make limited recommendations for prevention. Likewise, there is a need to conduct research on possibilities of "post-maturing" the enamel of inferior quality in the form of remineralization. Available clinical treatment concepts

need to be evaluated more thoroughly, especially taking dental patient-reported outcomes into account that capture the disease impact on the patient. Fortunately, it is anticipated that the dental community will make progress in the near future toward answering these questions.

Dear readers, please allow me to conclude this preface with a few personal lines. Many of you know that I have been dealing with the problem of MIH clinically, scientifically, and academically for many years. For this reason, it has been a special interest and a long-cherished wish of mine to write a book on this significant topic that is both scientifically sound and oriented toward clinical practice at the same time. I am pleased this could now be realized.

I would like to take this opportunity to express my gratitude. My thanks go to Dr. Richard Steffen (St. Gallen, Switzerland) and Priv.-Doz. Dr. Christian Kirschneck (University of Regensburg, Germany) for their contribution to the chapters on pain management and extraction therapy. Dr. Jorge Casián (Poza Rica de Hidalgo, Mexico) and Dr. Clarence Tam (Auckland, New Zealand) have provided valuable clinical case examples to make different treatment approaches even more illustrative. Hassan Shokoohi, M.D., Stefan Tangl, M.D., Anton Dobsak, and Cornelia Jungwirth, M.D. (Medical University of Vienna, Austria) assisted in preparing specimens for micro-CT images, SEM images, histological images, and graphs. Last but not least, I would of course like to sincerely thank all my MIH patients who continue to place their trust in me and who have also made themselves available for taking the photographs. Without them, this book would not have been possible in such form and detail.

Katrin Bekes, Vienna, May 2021

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Chapter 1
Introduction

Molar incisor hypomineralization (MIH) – also commonly known as chalk teeth – has been a concern of pediatric dentistry for many years. Initially almost dismissed as an incidental finding, this disease has now reached a high clinical relevance due to remarkable prevalence figures.

The present chapter provides information on the genesis of research on the disease, gives an insight into when hypomineralizations belonging to the MIH group were first mentioned scientifically, and how the term was coined.

1.1 First clinical mention of MIH

The phenomenon of MIH is not new in pediatric dentistry. As early as 2001, the term was introduced into the literature and a definition was developed.[1] Strictly speaking, however, even at that time it was not a new disease. Clinical attention was first drawn to the pathology at the end of the 1970s. At that time, dentists from the Public Dental Service in Sweden reported an unusually high and increasing number of children with extensive, demarcated, severe enamel hypomineralizations of unknown etiology of the first permanent molars and incisors (Fig 1-1). The enamel defects were difficult to treat and clean due to extreme sensitivity.[2] This gave rise to the initiation of a first epidemiological study, published by Koch et al in 1987.[3] In this investigation, the prevalence, extent and severity of the defects in 8- to 15-year-old children were analyzed. It was found that – depending on the birth cohort – the structural defects occurred in a frequency of up to 15%. Furthermore, it turned out that the most severely affected teeth were the first permanent molars. In addition, more than one molar was usually affected per child. Since the structural disorders did not fit into the conventional etiological classification at that time, they were described as "idiopathic enamel hypomineralizations of permanent teeth."[3] A reliable reason or solution for why there was a much higher prevalence of MIH-affected teeth in one of the birth cohorts in particular could not be found at that time either.

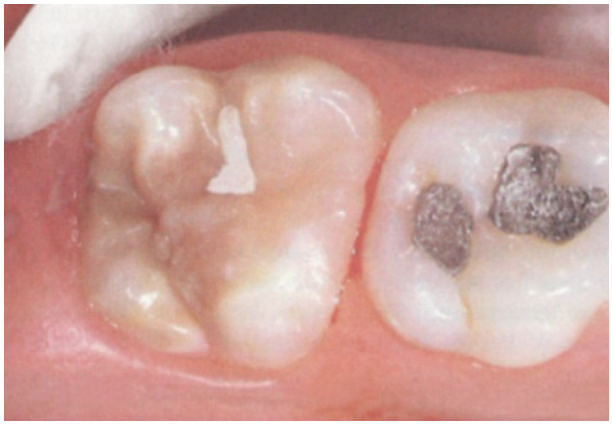

Fig 1-1 Patient case with demarcated hypomineralization of a first permanent molar, now attributable to MIH, from the 1987 Swedish study (source: Koch et al[3]; courtesy of Wiley).

1.2 Determination of terms

As mentioned above, the independent term "molar incisor hypomineralization" entered the literature in 2001.[1] This was preceded a year earlier by the 5th Congress of the European Academy for Paediatric Dentistry (EAPD) in Bergen, Norway. There, four abstracts from three working groups addressed enamel developmental defects of permanent first molars independently of each other.[4–7] The authors referred to the defects as "hypomineralized permanent first molars," "idiopathic enamel hypomineralization in the permanent first molars," "non-fluoride hypomineralization in permanent first molars," or "cheese molars."[1] These different terminological attributions motivated the working group to work together and find a uniform name for the novel pathology in order to make future studies and case reports comparable. Due to the unclear etiology, both the distribution pattern and the morphology were emphasized in the naming process.

MIH is currently defined as a (systemic) qualitative enamel defect of one or more 6-year molars with or without the involvement of the permanent incisors (Figs 1-2). The authors agreed on this definition to clarify two important points:

- The phenomenon of MIH always involves at least one molar.
- A combination of affected molars with demarcated opacities on the incisors is possible, but not required.[8]

Opacities occurring only on the incisors indicate a different cause and should therefore not be assigned to MIH. At the EAPD meeting in Athens 2 years later, it was reconfirmed to use the chosen term consistently. In addition, diagnostic criteria were named to ensure comparability in future epidemiological studies.[8] These are still valid today[9] and will be described and explained in more detail in Chapter 6.

1.3 Medieval finds

Since that time, not only the dental profession but also patients have been concerned with the question of whether MIH is really a new condition. This is not easy to answer, especially since the occurrence of hypomineralizations in molars and incisors was rarely the focus of research before the 20th century. However, findings from the 17th and 18th century from Broadgate Cemetery, UK, give reason to consider whether MIH may have occurred in earlier centuries.[10] Ogden et al examined skeletal remains from 45 sub-adults and interestingly found structurally compromised molars exhibiting enamel developmental dysplasia or MIH in 93.2% of the dentitions examined (Fig 1-3). A case report from the 15th century seems to confirm this.[11] Dental findings on examination of the skull showed enamel defects of molars 36 and 46 and small defects on the other first molars. Furthermore, streaks of enamel were found on the permanent incisors.

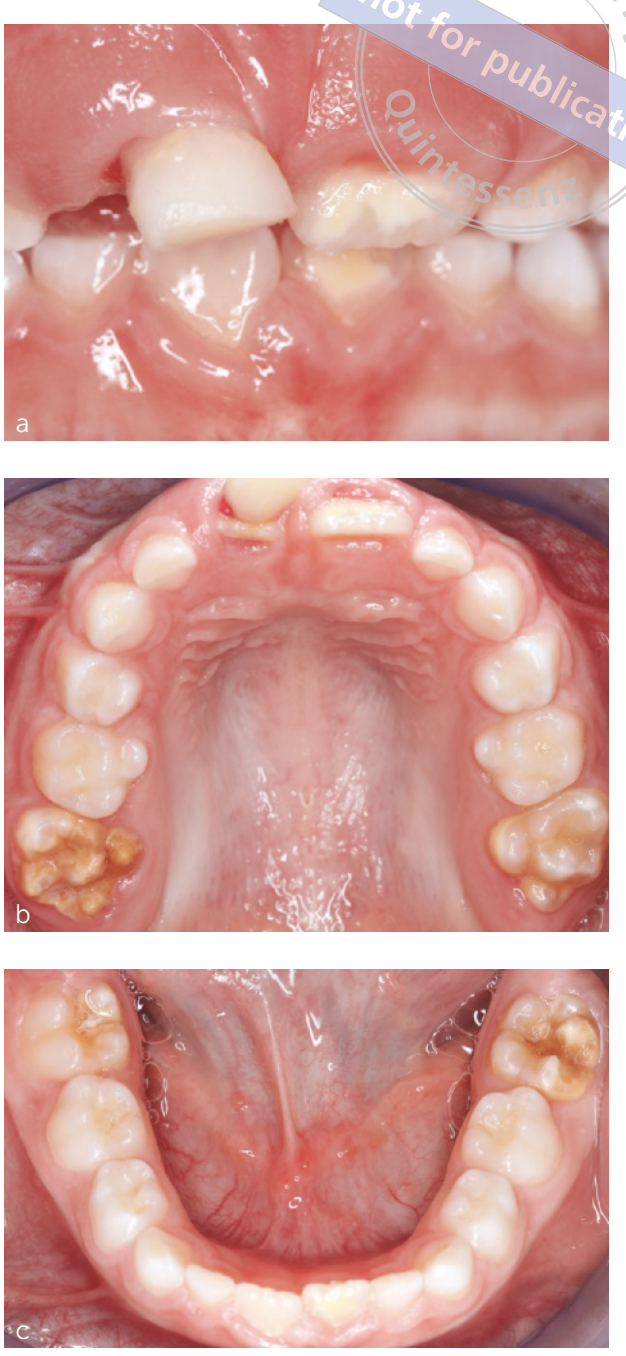

Fig 1-2 (a–c) MIH with qualitative enamel defects on several 6-year molars with involvement of the permanent incisors.

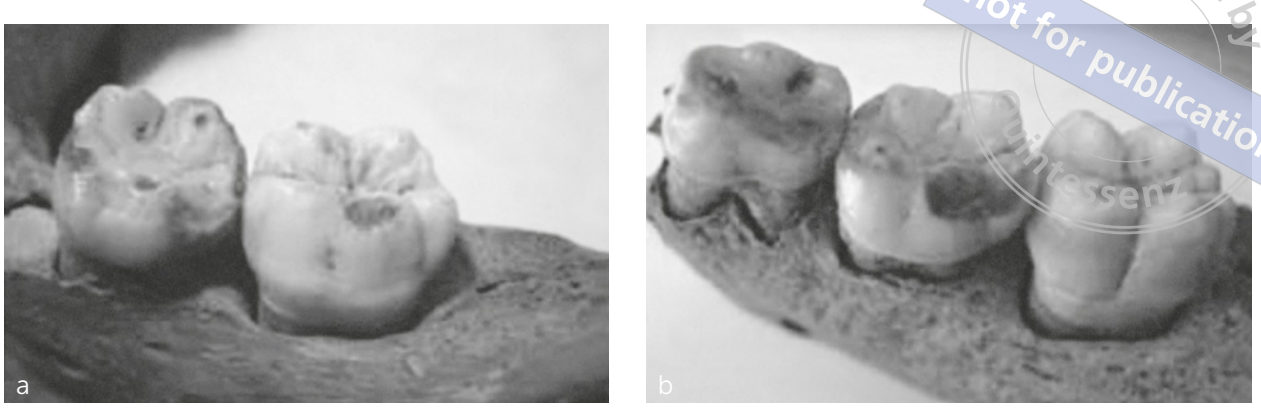

Fig 1-3 Medieval skeletal find: a) probable MIH at the cusp tip of the first mandibular molar; b) probable MIH of the distobuccal cusp of the mandibular left second primary molar (source: Ogden et al[10]; courtesy of Springer).

There are also empirical data for Germany in this context. However, these relativize the statement of the occurrence of MIH in the further past. A study of skeletons of the 12th to 16th centuries from Regensburg and the 16th to 18th centuries from Passau showed the existance of MIH, but only in a very small percentage (3.2%).[12,13] Of the total 309 skulls examined, seven (2.2%) showed at least one molar with a demarcated opacity, which could be assigned to MIH according to the EAPD criteria. Three additional skulls (1.0%) had at least one tooth with a posteruptive enamel breakdown. Based on the low prevalence, the authors concluded that MIH most likely did not exist, or at least existed only rarely, in the archaeological case series they studied. Thus, they suggested that MIH is a specific phenomenon and therapeutic problem of our century.

1.4 Outlook

Much has happened since MIH was first described and defined as a disease in its own terms. Media interest about the problem has increased in recent years, both in the professional and general press. Epidemiologically and etiologically oriented studies of MIH have appeared in large numbers in recent decades. This shows that research on the disease is being conducted worldwide. Because internationally standardized diagnostic criteria are available, prevalence data can be documented in a comparable manner.

However, there is still a great need for research in the areas of etiology and therapy. The causative factors of the disease remain unclear although numerous investigations have been carried out, some of them with partly controversial results. A major problem is that MIH only becomes clinically apparent when eruption of the affected tooth into the oral cavity begins. However, this does happen for several years after enamel formation and thus a long time after damage. Frequently published retrospective studies are therefore limiting and carry the risk of misinterpretation of results. Prospective randomized studies are currently lacking, but would be immensely important. Consequently, the goal in the coming years must be to further increase research in this area. With regard to the therapy of MIH, the required knowledge about the symptomatology of the disease – such as posteruptive enamel breakdowns or hypersensitivity – has greatly increased, and immense progress could also be made in the context of its treatment. Nevertheless, there is a great need for clinical studies to refine and appropriately enhance existing treatment concepts, including randomized clinical trials. In addition, the goal of basic research in the coming years must be to develop procedures and solutions for stabilizing the soft and porous tooth structure.

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Chapter 2

Clinical presentation and morphological features

By definition, molar incisor hypomineralization (MIH) is a systemic hypomineralization of one to four permanent first molars with or without involvement of the incisors.[1] A major clinical challenge is the variability of hypomineralization and the different degrees of severity that result.

Therefore, in this chapter, the different clinical manifestations of MIH are described and explained.

2.1 Teeth

At least one 6-year molar is affected. The permanent incisors may or may not be involved (Figs 2-1 and 2-2). Clinically, the first molars are usually affected more frequently and more severely than the incisors. If the incisors are also involved in a patient, then those in the maxilla seem to be affected more often.[2]

2.2 Color

MIH teeth are clinically characterized by a change in the enamel translucency. The hypomineralized enamel can vary in shade from white to yellow to brown (Figs 2-3 and 2-4). The margins or borders are always clearly visible, well-defined, and can be clearly distinguished from healthy enamel.

As a rule, the darker the color, the softer and more porous the enamel, and thus the higher the risk of posteruptive breakdown with dentin exposure.[3] These enamel breakdowns are usually found at the tooth cusps, but can also be localized in other areas (Figs 2-5 and 2-6).

2.3 Localization

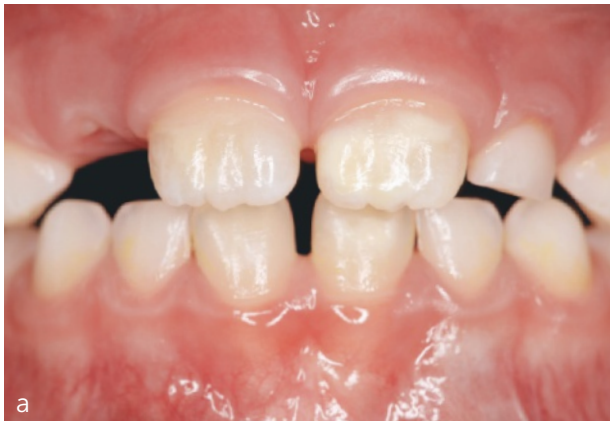
The mineralization disorder may be limited to a single cusp in the molar region or may extend over the entire smooth surface or fissure relief to the cervical area of the tooth[4] (Fig 2-7). If multiple molars are affected in a patient, variations may also occur. Therefore, it is possible that in one patient, small, intact opacities are found in one molar, while large parts of enamel breakdown can be seen shortly after eruption in another molar[5] (Fig 2-8).

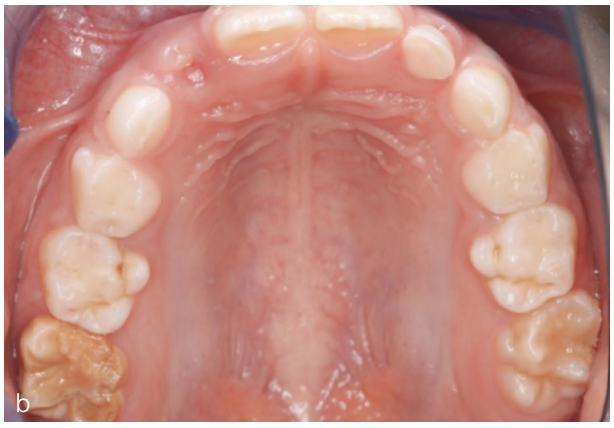
The incisors usually show hypomineralization in the buccal area. Here, many different manifestations are possible as well (Fig 2-9).

2.4 Characteristics

Mildly altered teeth tend to show white-yellow or yellow-brown, irregular discoloration, whereas severe forms of hypomineralization present chipped or missing enamel and/or dentin areas of varying extent.[4]

Especially under the influence of masticatory forces, initially intact but discolored enamel can easily chip off due to its porosity (Fig 2-10). A predictor for such posteruptive chipping of hypomineralized enamel seems to be the color of the opacity.[6] In permanent incisors, the affected enamel is usually less affected and probably less susceptible to breakdown due to the absence of masticatory forces. However, incisal enamel defects are usually quite extensive and most commonly found on the buccal tooth surfaces. This appearance often triggers irritation among parents – as documented by discussions with them – who claim cosmetic concerns in particular.





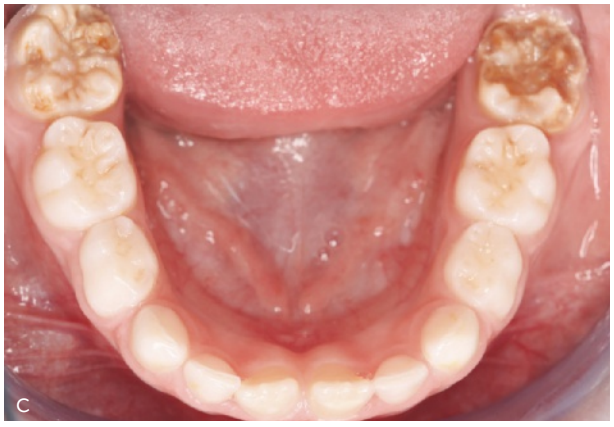
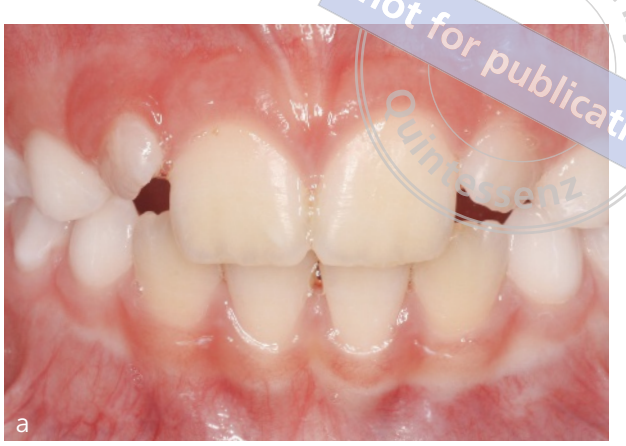
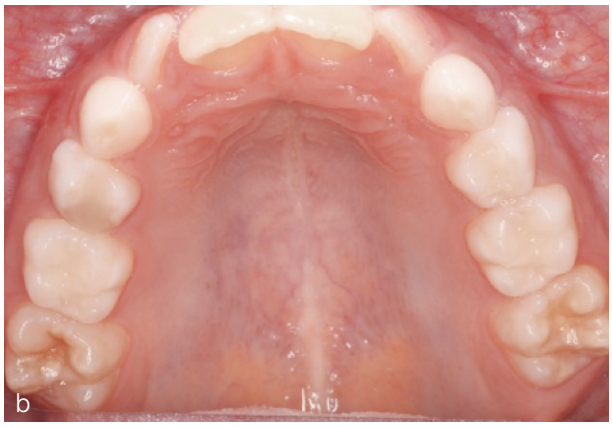


Fig 2-1 MIH in a 7-year-old female patient with molars and incisors being involved. a) Frontal view: teeth 11, 21, and 31 show white opacities. The maxillary lateral incisors are not yet evaluable. b) Maxillary view: tooth 16 shows posteruptive enamel breakdowns, tooth 26 shows sharply defined opacities. c) Mandibular view: teeth 36 and 46 are also characterized by posteruptive enamel breakdowns.





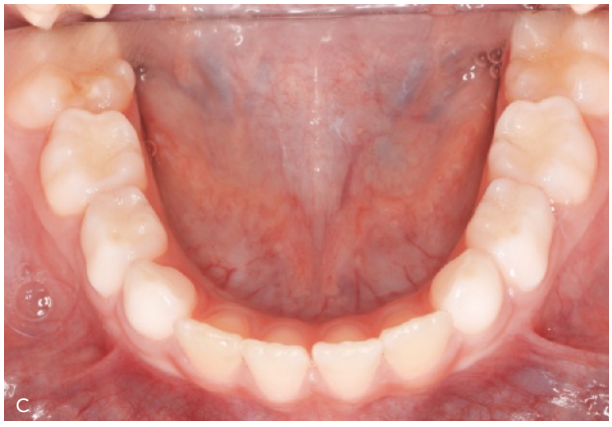


Fig 2-2 MIH in an 8-year-old patient without incisors being affected. a) Frontal view: the permanent incisors are healthy. b) Maxillary view: tooth 16 and tooth 26 show hypomineralizations in the occlusal relief. c) Mandibular view: tooth 36 is healthy, tooth 46 has a small white opacity in the occlusal-mesial area.

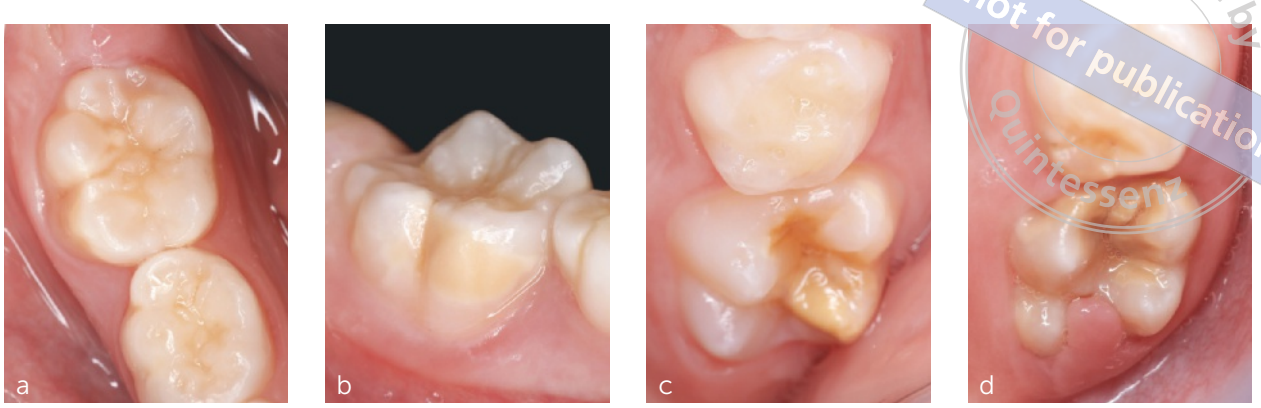


Fig 2-3 Opacities on MIH molars of different colors. a) Tooth 46 with white discoloration in the buccal region at the mesiobuccal cusp in occlusal view. b) Tooth 46 from Figure 2-3a in buccal view. c) Tooth 26 with a yellow opacity. d) Tooth 26 with a brown opacity, which changes to white at the margins.

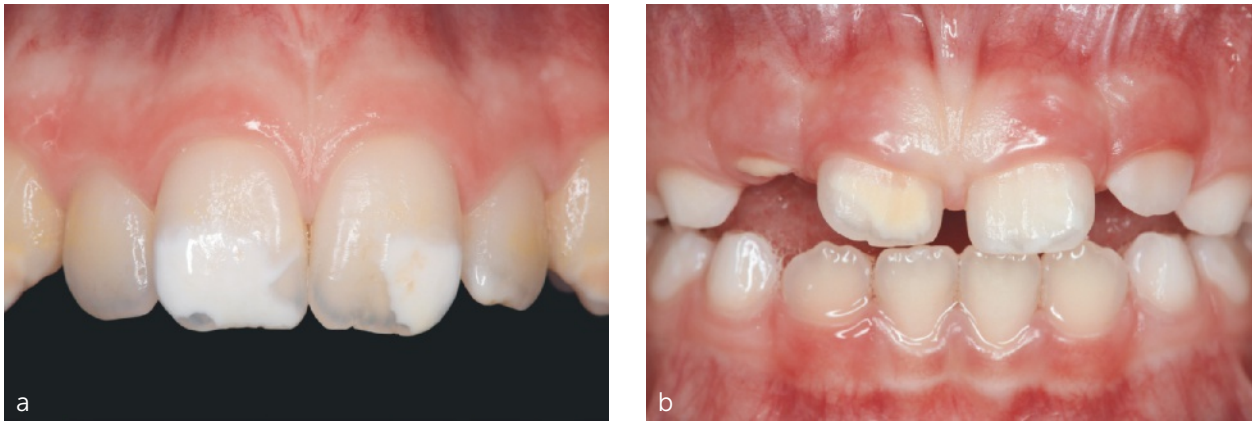
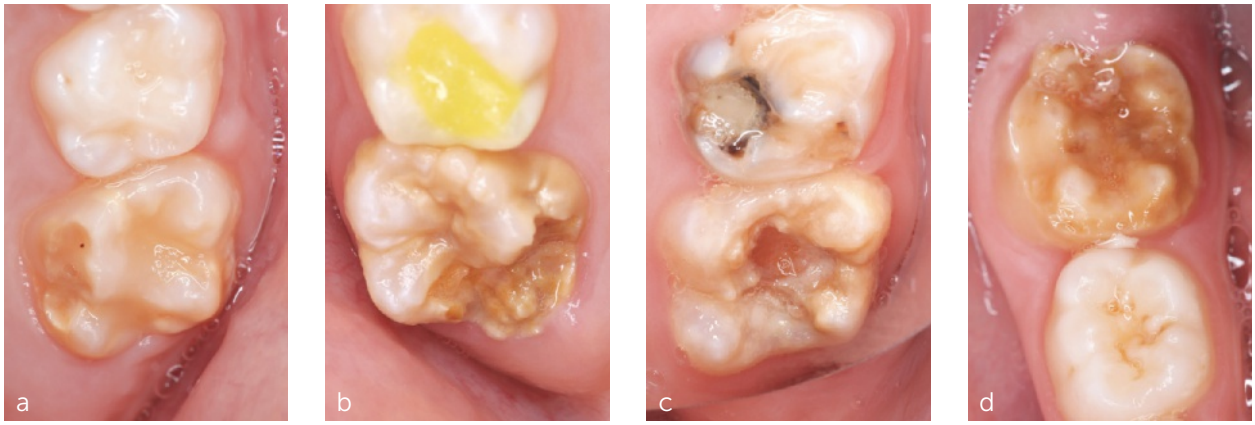


Fig 2-4 Opacities on MIH-affected incisors in different shades. a) Teeth 11, 21, and 22 with white opacities of varying degrees. b) Mid maxillary incisors with a white-yellow opacity on tooth 11 and a white opacity on tooth 21.



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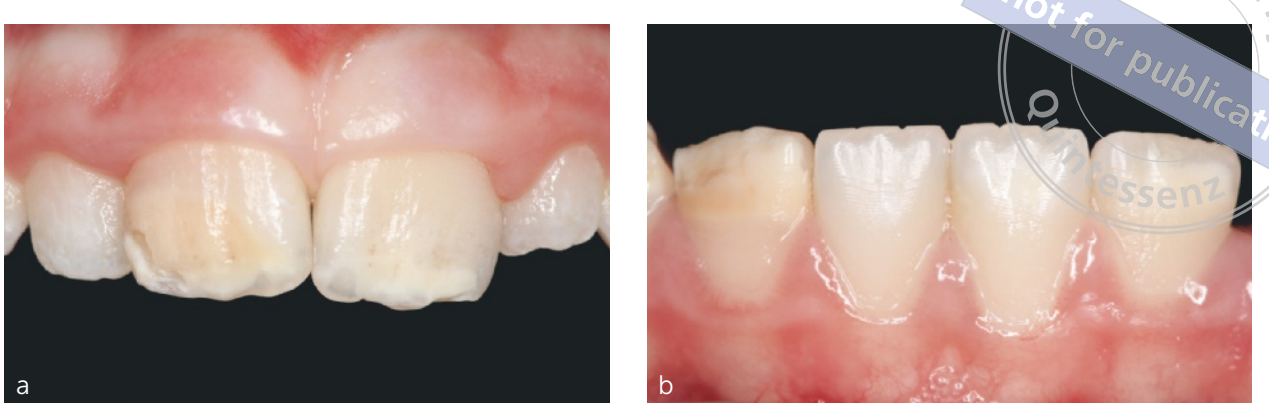


Fig 2-6 Posteruptive enamel breakdown of hypomineralized incisors. a) Substantial breakdown of tooth 11 in the distoincisal area. b) Enamel breakdown in the incisal area of tooth 42.

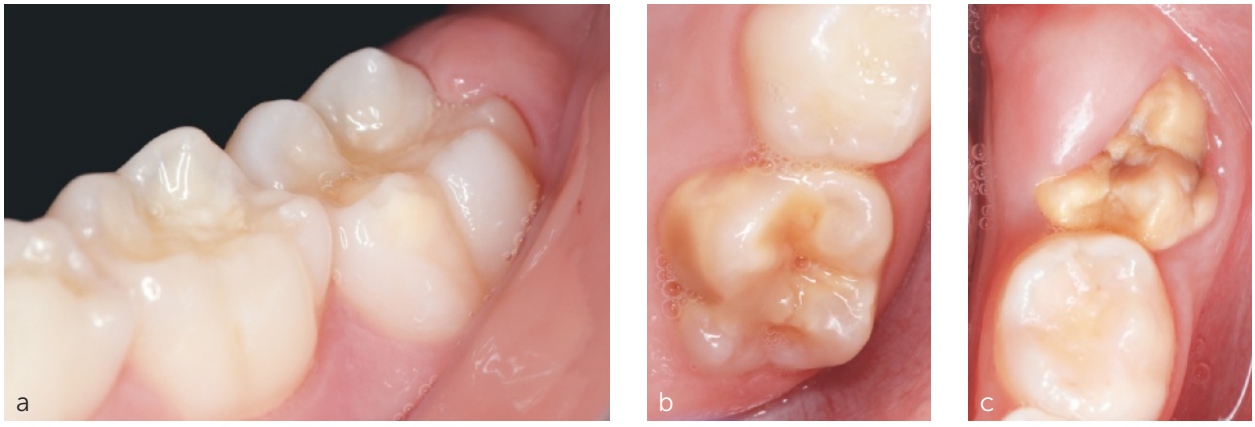


Fig 2-7 Different localizations of hypomineralization. a) Tooth 36 with a white opacity in the buccal area of the mesiobuccal cusp. b) Opacity in the fissure relief of tooth 26 and in the palatal area of the distopalatal cusp. c) Hypomineralization (as far as can be assessed) of tooth 36 in the occlusal area and spreading to the cusps.

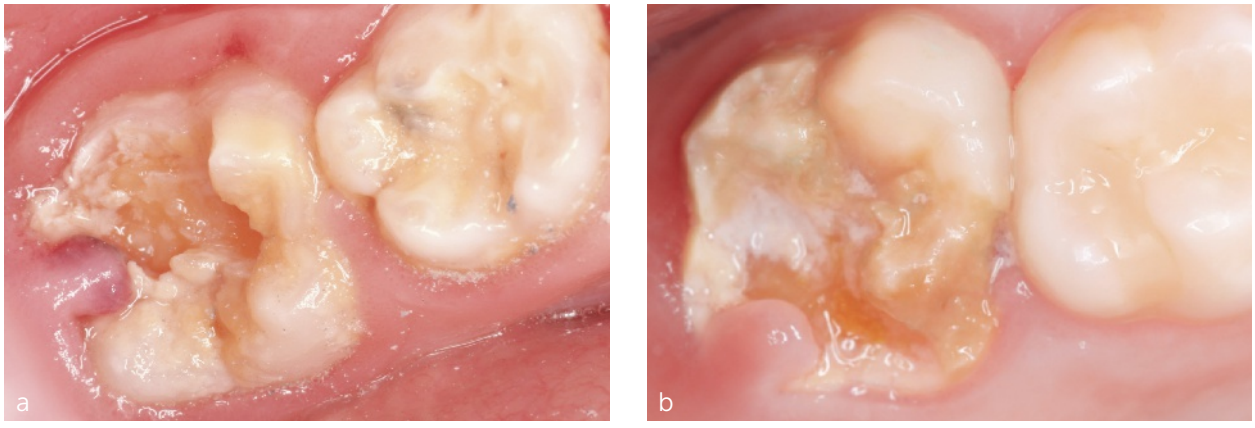


Fig 2-8 MIH molars with posteruptive breakdown shortly after eruption. a) A not yet completely erupted tooth 36 with small mucosa hood in the distal region, which already shows a loss of substance. b) Tooth 16 with enamel breakdown during eruption.

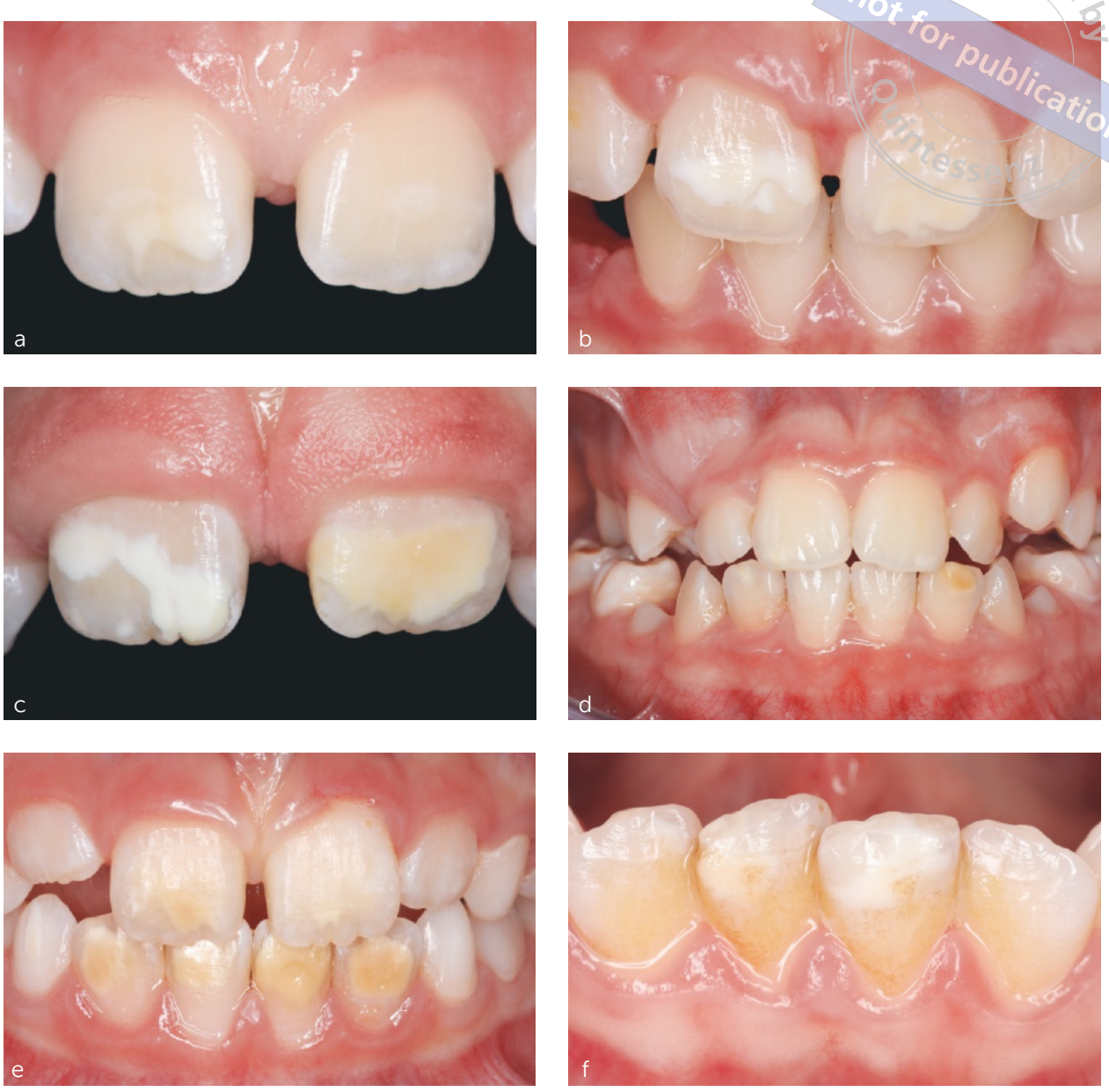


Fig 2-9 Incisors of MIH patients with hypomineralizations of varying localization and form. a) Small white opacities on teeth 11 and 21. b) Hypomineralizations of varying heights and colors in maxillary incisors 11 and 21. c) Opacities of variable heights and colors on maxillary central incisors. d) Hypomineralization of tooth 32 in the upper third. e) Opacities on maxillary central incisors and all four mandibular incisors occupying almost the entire buccal surface. f) Hypomineralization of the mandibular central incisors.

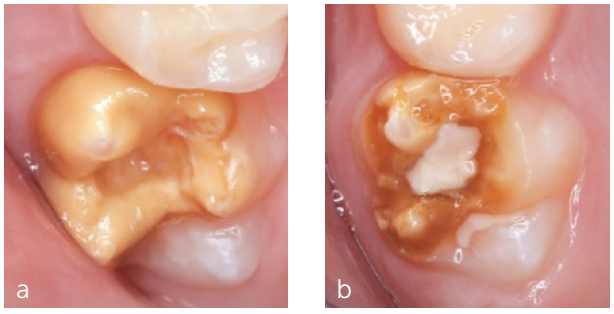
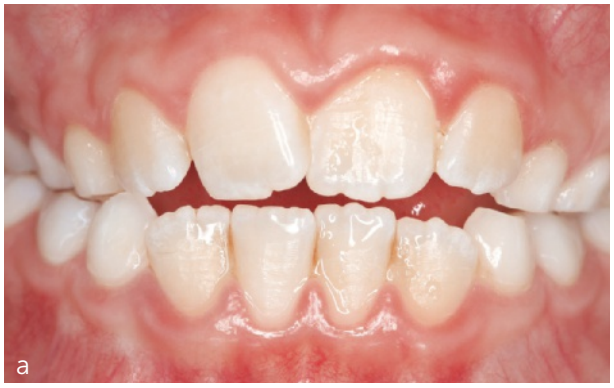
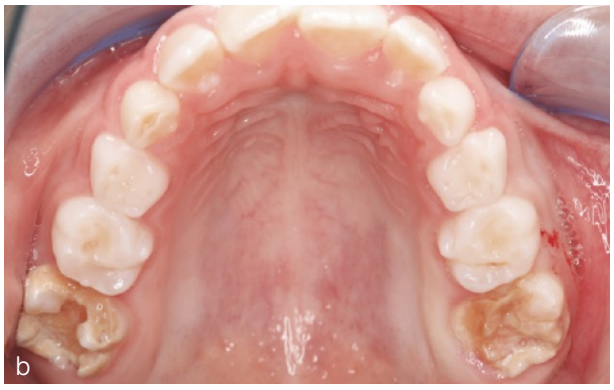


Fig 2-10 MIH-affected maxillary first molar at different time points. a) During eruption. b) 3 years later.





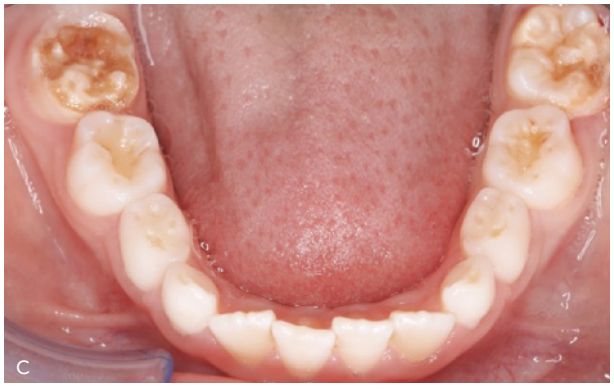
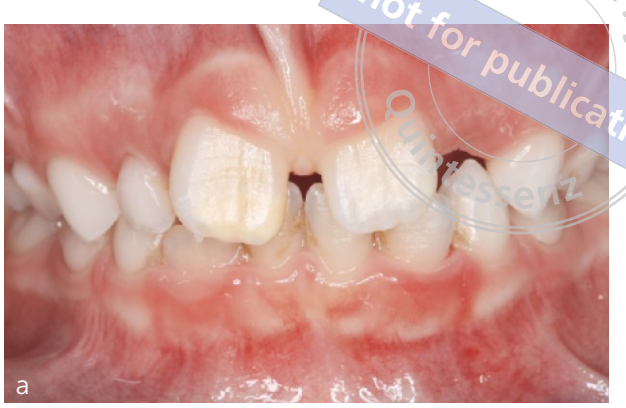
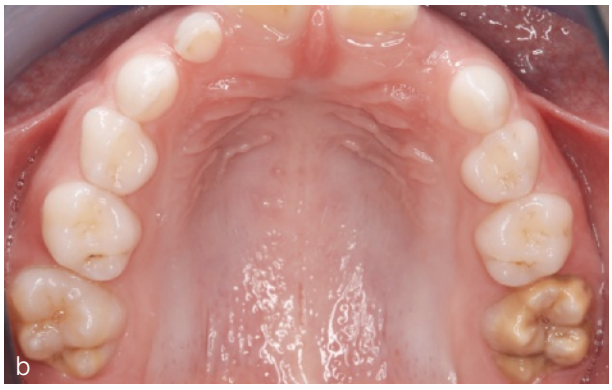


Fig 2-11 A 9-year-old female MIH patient with severe manifestations of MIH in all first permanent molars without involvement of the incisors. a) Maxillary view: no incisor shows an opacity. b) Upper view: both first molars already show extensive posteruptive enamel breakdowns. c) Mandibular view: the mandibular molars have already lost substance as well.





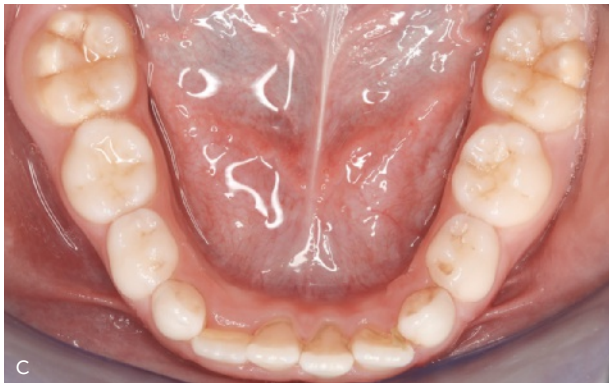
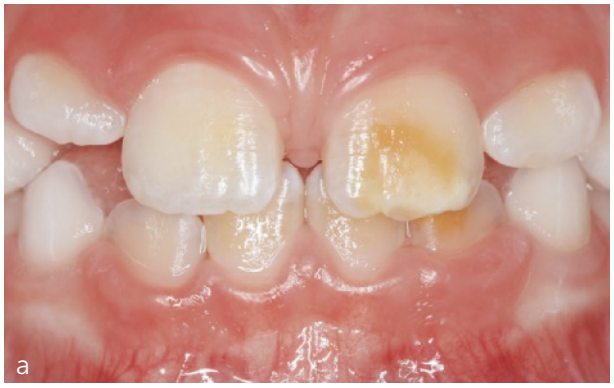
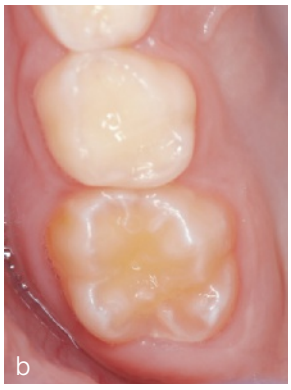
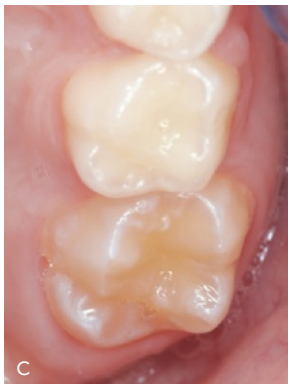


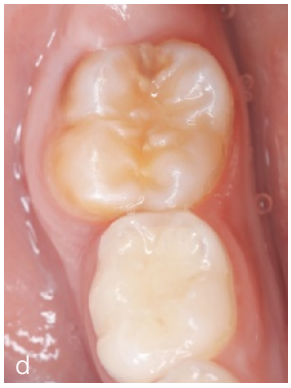
Fig 2-12 A 6.5-year-old MIH patient with opacities on the molars and both maxillary central incisors. a) Frontal view: teeth 11 and 21 with opacities of variable intensity. b) Maxillary view: opacities of different color on both molars. c) Mandibular view: white demarcated discolorations in the buccal region of each of the mandibular 6-year molars.

The severity of the defects of the molars does not necessarily correlate with the number and severity of the hypomineralizations of the incisors. On the one hand, all four molars may be severely affected in a patient, but the incisors may not be involved at all (Fig 2-11); on the other hand, it is possible that a mild form of MIH may occur in only one single molar combined with the involvement of several incisors (Figs 2-12 and 2-13). The different involvement of molars or incisors within one dentition is also conceivable (Figs 2-14 to 2-16).









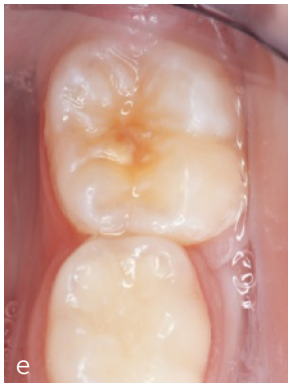
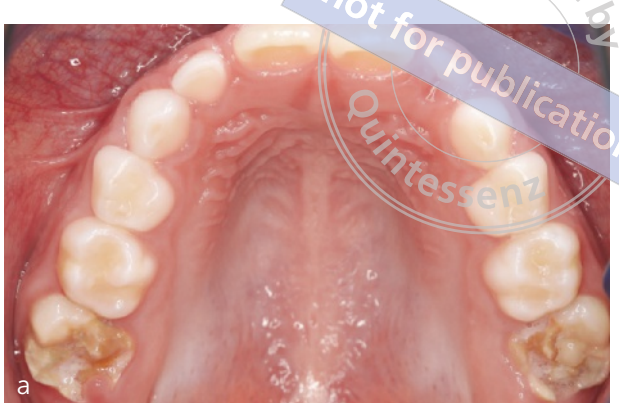


Fig 2-13 A 7-year-old female MIH patient with a prominent opacity on one maxillary and one mandibular incisor and mild appearance on the molars. a) Frontal view: tooth 21 and 32 with yellow hypomineralization. b) Tooth 16: no MIH visible. c) Tooth 26: no MIH identifiable. d) Tooth 46: MIH-affected molar with opacities in the distal fissure and on the distobuccal cusp in the buccal region. e) Tooth 36: MIH-affected molar with yellow hypomineralization in the occlusal fissure relief.



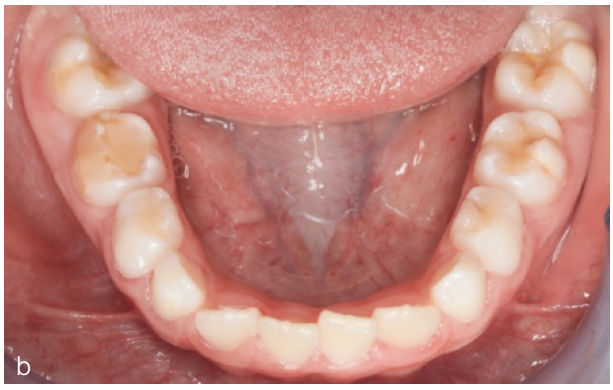
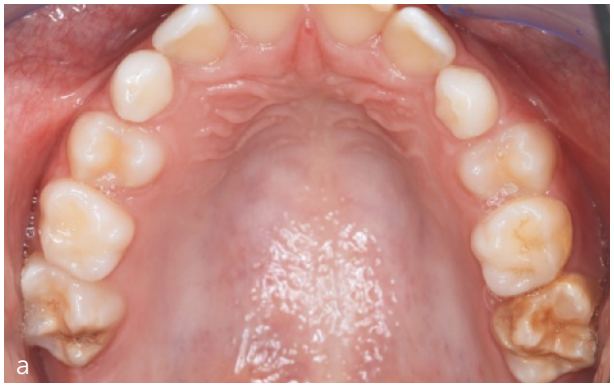


Fig 2-14 Different manifestations of MIH in the maxilla and mandible of a patient. a) Maxillary view: teeth 16 and 26 show posteruptive enamel breakdowns. b) Mandibular view: teeth 36 and 46 show only small opacities.

Therefore, early detection, intervention, and appropriate therapy are necessary to avoid severe complications and improve both masticatory function and esthetics of the teeth.

2.5 Hypersensitivity

In addition to the defect itself, the occurrence of hypersensitivity is an important and not negligible key symptom of MIH. MIH-affected molars can often be highly sensitive to temperature and tactile sensation. It has been hypothesized that the high porosity of the hypomineralized enamel favors the penetration of bacteria in the dentinal tubules, causing a subclinical pulpal inflammation[7–9] (see Chapter 3). This hypersensitivity has considerable



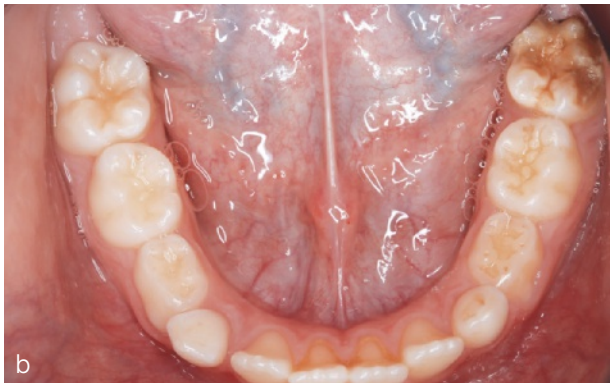
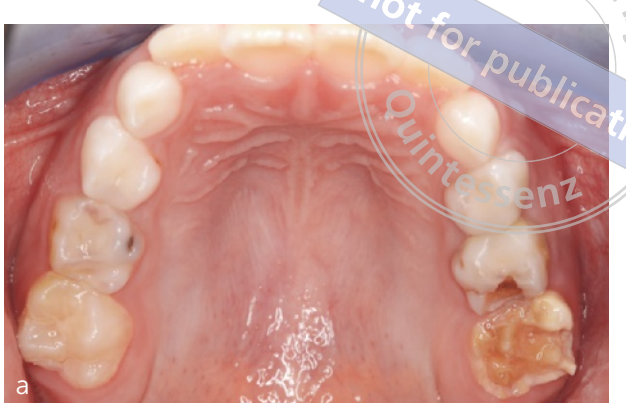


Fig 2-15 Heterogeneous MIH involvement of the maxillary and lower first molars of a female patient. a) Maxillary view: tooth 16 shows an opacity, tooth 26 already presents with loss of substance. b) Mandibular view: tooth 36 has also suffered enamel breakdown, tooth 46 is healthy.



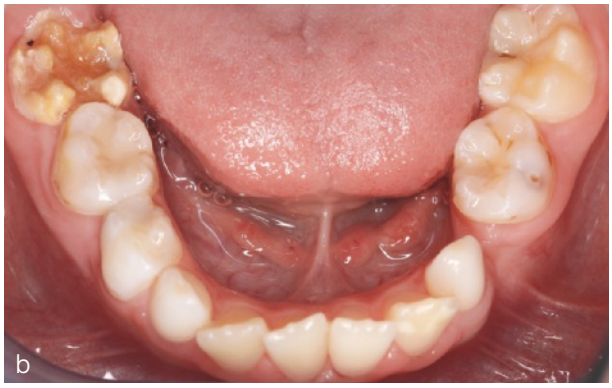


Fig 2-16 Different manifestations of hypomineralization in the maxillary and mandibular 6-year molars. a) Maxillary view: tooth 16 is healthy, tooth 26 is characterized by extensive loss of substance. b) Mandibular view: tooth 36 shows a small opacity in the buccal region, tooth 46 is characterized by a severe posteruptive enamel breakdown.

subsequent burdens for children: it restricts them in the consumption of cold and hot foods and in oral hygiene (Fig 2-17). It also interferes with the treatment of these teeth, partly because chronic pulpal inflammation can make successful local anesthesia difficult.[10] Therefore, premedication is often used in the treatment of hypersensitive molars, which will be discussed in more detail in Chapter 9.

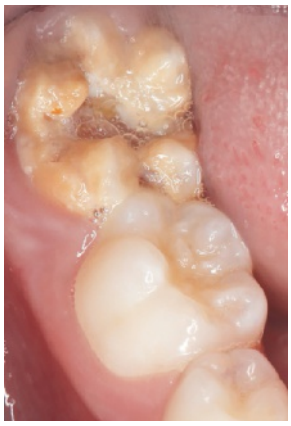


Fig 2-17 Hypersensitive MIH molar with posteruptive enamel breakdown. You can also see the plaque accumulations, which indicate a lack of oral hygiene due to pain.

2.6 Deviations from the classical definition

As described above, MIH is classically defined for the first permanent molars and incisors. In the meantime, MIH-characteristic defects have occasionally been observed in other permanent teeth (second permanent molars, premolars, canines).[11,12] These hypomineralizations may or

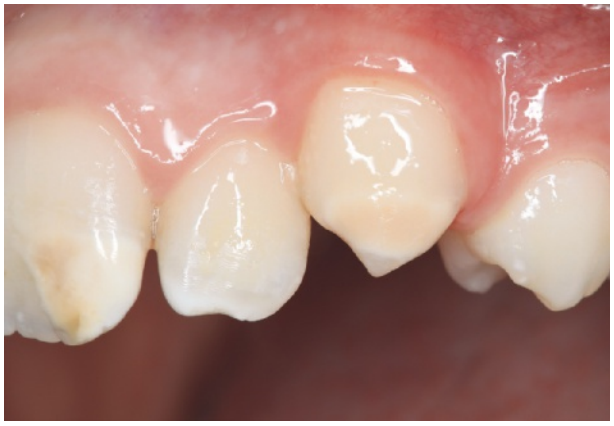


Fig 2-18 Additional hypomineralization in a maxillary permanent canine in a patient with MIH. The classic opacity can also be seen on tooth 21.

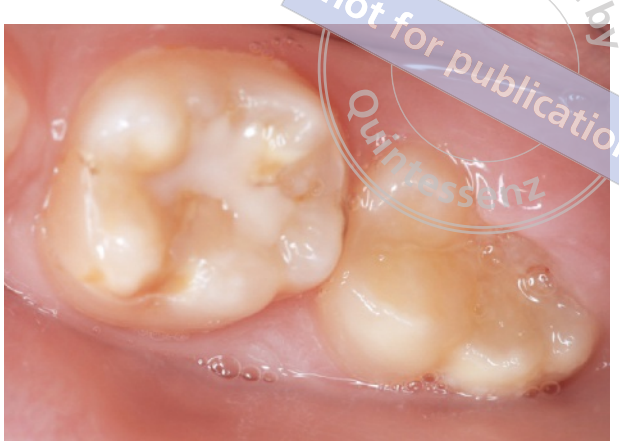


Fig 2-19 MIH patient showing a demarcated opacity on tooth 37 in addition to the involvement of tooth 36.

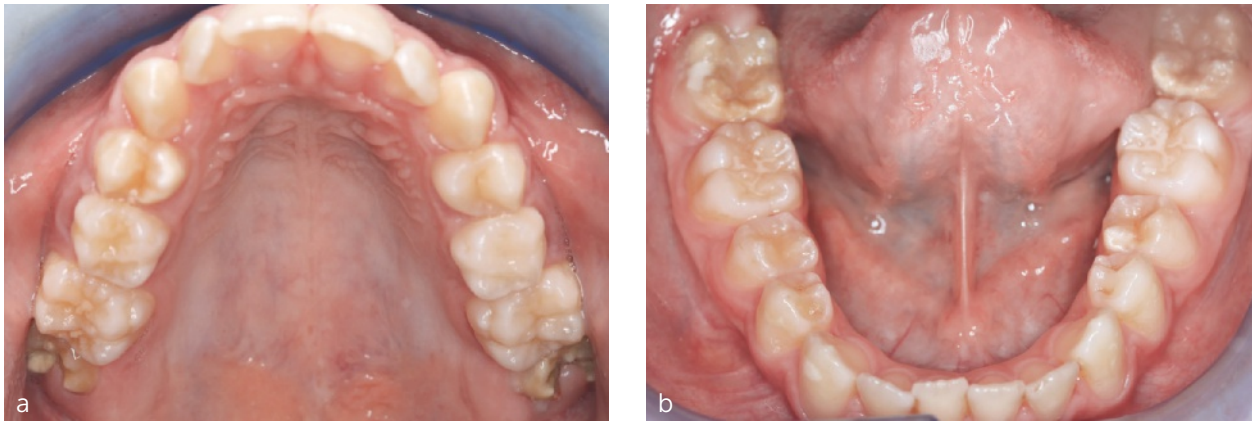


Fig 2-20 A 12-year-old patient with hypomineralized second molars in combination with the involvement of single premolars. a) Maxillary view: teeth 17, 27, and 14 with hypomineralizations. b) Mandibular view: teeth 37, 47, and 35 with hypomineralizations.

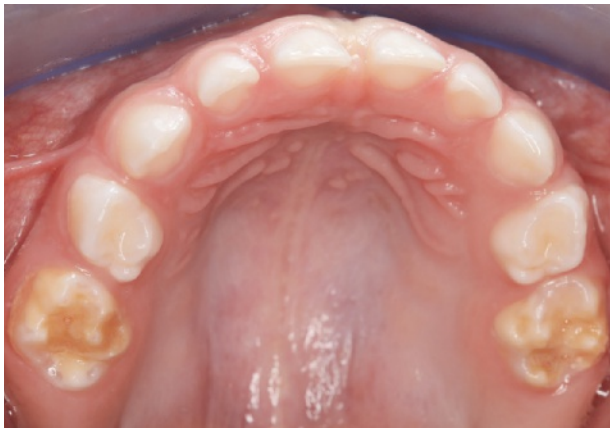


Fig 2-21 Hypomineralized second primary molars in the upper jaw of a 3-year-old female patient. In addition to opacities, the primary molars also show first posteruptive enamel breakdowns.

may not occur in combination with the classic manifestation of MIH. To date, however, scientific data on this are sparse.

In a Greek study[12] of 1156 14-year-old adolescents, it was shown that out of 244 (21.1%) MIH children, 117 (48.1%) had hypomineralizations in other permanent teeth, too. In addition, 16.2% out of the children not affected by classic MIH also showed hypomineralizations in other permanent index teeth. Their frequency by tooth type was as follows: second molar 33.7%; canine 25.7%; first premolar 23.6%; second premolar 17.0%.

Figures 2-18 to 2-20 show examples of the different variations described. Furthermore, hypo-

mineralization can also be found in deciduous teeth. The second primary molars (see Chapter 17) with/without involvement of the primary canines are then the most frequently affected (Fig 2-21). The structurally compromised teeth are also clinically characterized by a change in the translucency of the enamel. Due to their considerable prevalence worldwide, a separate term has been developed. Such findings are known as "hypomineralized second primary molars" (HSPM).

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Molar incisor hypomineralization (MIH) has become a much-discussed topic of interest that presents major challenges for dental professionals in practice and research. It is defined as a qualitative enamel defect that typically occurs on one or more first permanent molars with or without the involvement of the permanent incisors.

The book serves as a comprehensive reference that critically appraises the controversies around MIH found in the scientific literature. Practical advice about diagnosis and treatment of the disease is provided, and the various clinical treatment options are explained in detail.

The text is written for dental practitioners, postgraduate and undergraduate students and researchers, as well as anyone who would like to expand their knowledge of this contemporary topic in everyday clinical dentistry.

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