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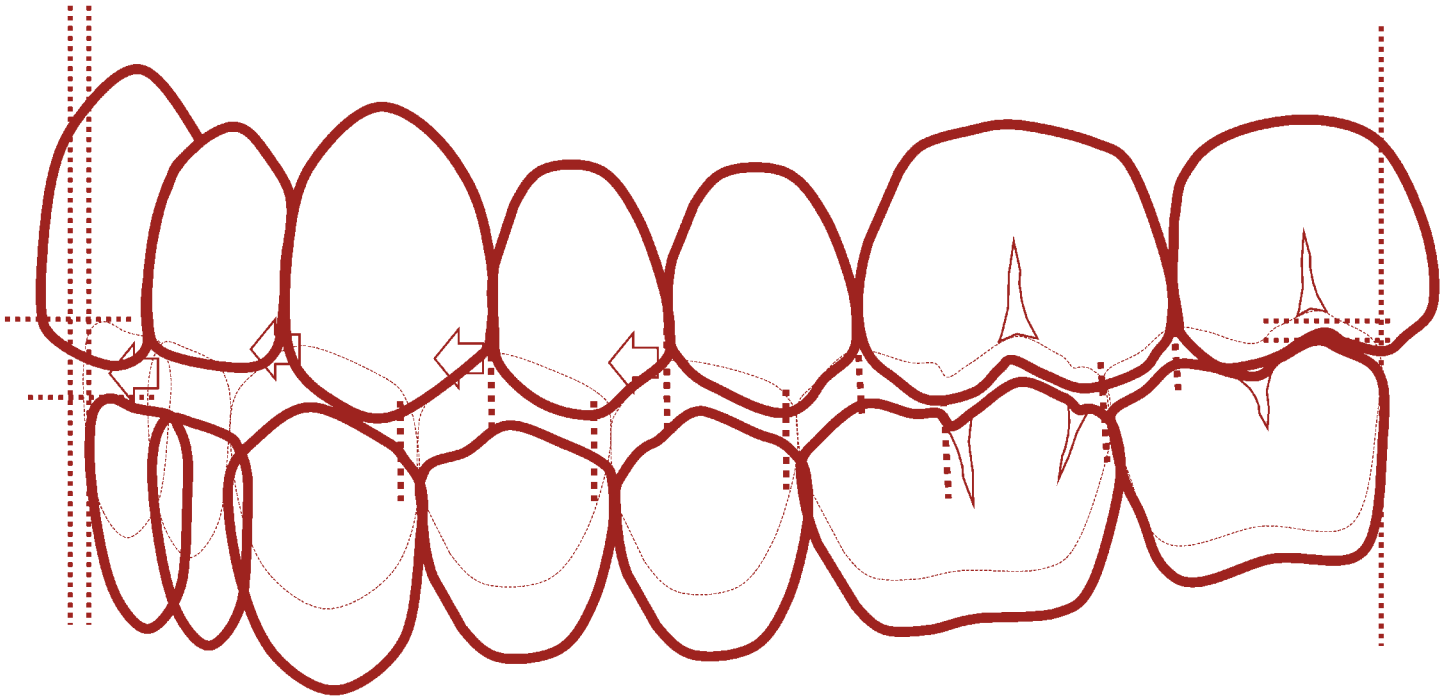
Interceptive treatment approach

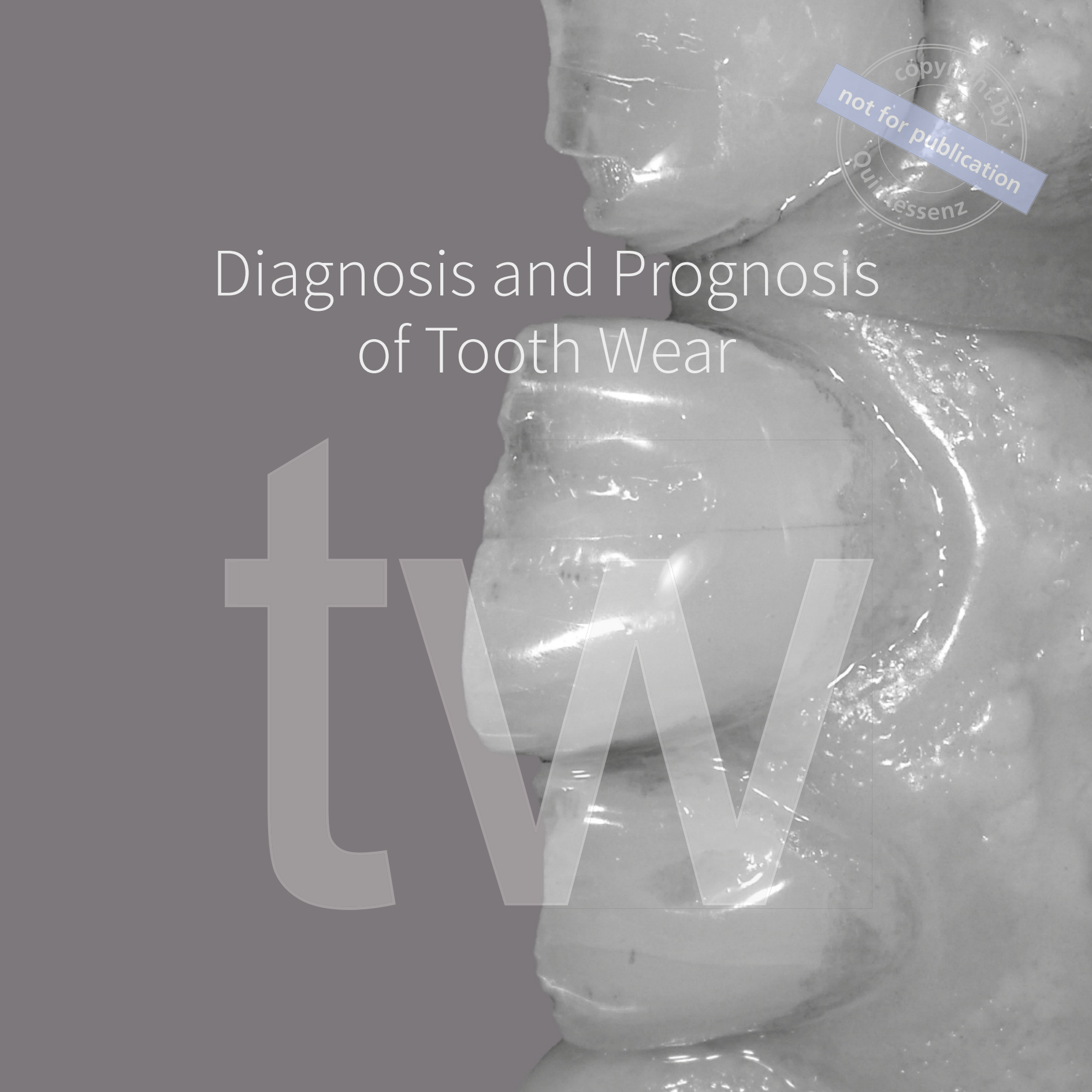
TOOTH WEAR

with minimally invasive protocols



QUINTESSENCE PUBLISHING

CHAPTER 1



Diagnosis and Prognosis of Tooth Wear

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No proper preventive or therapeutic measures can be installed before a diagnosis is performed; tooth wear is of course no exception to this basic rule, in particular due to its multifactorial nature. We need first to analyze the basic mechanisms (erosion, abrasion, and attrition) and then consider risk factors (eg, diet, occlusion, function and parafunctions, anatomy, patient's physical and psychologic health) and co-factors (eg, patient's age, compliance, socioeconomic conditions); this is important due to the complex interaction that occurs between the aforementioned risk factors and co-factors, leading to various degrees of hard tissue loss. Understanding basic tooth wear mechanisms and related criteria of diagnosis will help clinicians to safely and efficiently manage patients affected by tooth wear.

Wear mechanisms and their incidence

The first basic mechanism of tooth wear, erosion, is induced by extrinsic and intrinsic acids, fragilizing hard tissues mainly through mineral loss; fragilized surfaces then become prone to loosening during occlusion, mastication, and brushing. The second basic mechanism, abrasion-attrition (mechanical-frictional), is due to abnormal forces and contacts on hard tissues (through shear, flexural, and compressive strains), which lead to microscopic, structural disruption of enamel or dentin structures. Both phenomena happen from physiologic to various pathologic levels. Moreover, both mechanisms often appear to act synergistically, worsening and accelerating tooth wear. Clinically, both mechanisms induce typical wear patterns while when acting concomitantly, atypical lesions with hybrid patterns are observed.

Erosion

Erosion originates from extrinsic and intrinsic acid sources; extrinsic sources include beverages (carbonated drinks, fruits juices, salad dressing, spicy liquid condiments), many of them with low pH values (2.0–3.5), food (typically fruits and some vegetables like tomatoes and chilies), and a few medications (eg, chewing vitamin C). People who consume abnormal quantities of the aforementioned acidic products will then develop dental erosion. Unbalanced diet and food compulsive behavior have unfortunately become common problems in our modern society. If this etiology is identified, a basic approach is to ask the patient to monitor daily food and beverage intake; the dentist or nutritionist can then analyze the patient's weekly reports and recommend imperative changes. Needless to say, the dentist is not always in a very strong position to control food behavior. A final cause for dental erosion, although of lesser incidence, is the decreased salivary flow and dehydration resulting from athletic or strenuous physical activities, with added risk from regular consumption of sweetened sport drinks.

There is only one intrinsic source of erosion: the gastric acid produced by our stomach (main constituent being hydrochloric acid), with a pH varying from 1 to 3. Apart from letting food enter the stomach, the lower esophageal sphincter (LES) normally prevents gastric acid from entering the esophagus toward the mouth.

If the LES doesn't close all the way or if it opens too often, gastric acid can move up into the esophagus. An abnormal acid reflux is called *gastroesophageal reflux disease* (GERD), inducing symptoms like pain and heartburn, and if untreated

for too long, a Barrett's esophagus can develop with an elevated risk of adenocarcinoma. It of course also causes dental erosion, which contributes to diagnose the underlying condition. A common cause of acid reflux is related to a malfunction of the LES called *hiatal hernia*. The other conditions of abnormal gastric acid production and increased reflux episodes are linked to poor dietary habits like eating large and/or heavy meals, particularly before bedtime, and drinking in excess fizzy drinks, alcohol, coffee, and tea. Other physical and medical conditions such as obesity, pregnancy, or medications like pain killers, muscle relaxers, and blood pressure medications can also trigger acid reflux. The critical period in case of reflux is during sleep as salivary flow and swallowing are reduced. In case of suspected reflux, proximal esophageal pH-metry and/or upper GI endoscopy (gastrointestinal) or EGD (esophagogastroduodenoscopy) are recommended to, respectively, monitor the frequency, duration, and pH drop of reflux episodes and assess any anatomical anomalies or mucosa alteration.

The last causes for severe dental erosion are bulimia and anorexia (bulimia nervosa and anorexia nervosa); patients suffering from bulimia are known to eat excessive amounts of food in a short amount of time, then purge the consumed food through vomiting or the use of laxatives, diuretics, or stimulants. With anorexia, on the contrary, patients restrict their food intake but apply the same purge methods to lose weight, next to compulsive physical exercise. Bulimia and anorexia are frequently associated with depression and anxiety, with a distorted self-image. Apart from the severe dental implications of these diseases (rapidly evolving erosion), psychotherapy (psychopharmacologic and psychosocial treatments) is highly recommended as untreated patients likely worsen their conditions and can even put their life at risk.

Attrition and abrasion

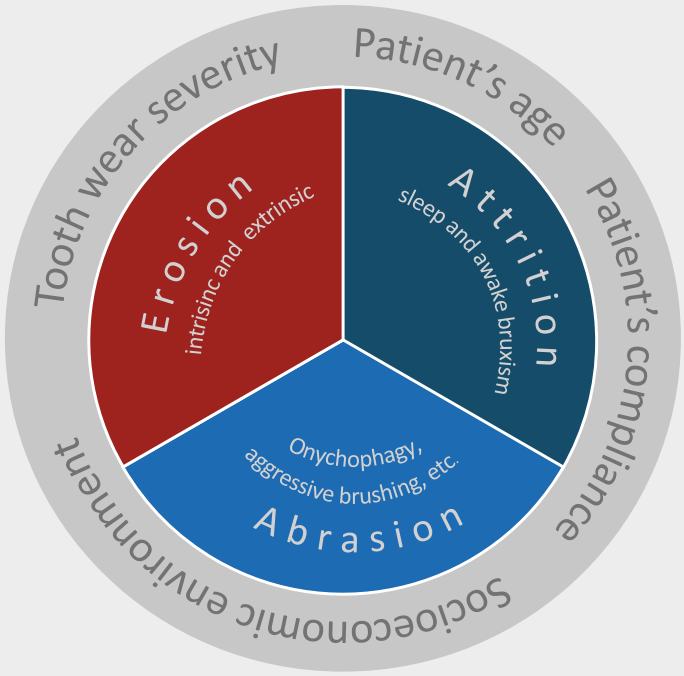
Semantically, *attrition* is related to the wear process resulting from tooth-to-tooth contacts, while *abrasion* results from the effect of a foreign object on dental tissues. Both phenomena lead to tooth wear by direct mechanical disruption of enamel and dentin. Attrition is physiologic up to a certain level but can be significantly elevated by abnormal occlusal forces and functional movements such as sleep and awake bruxism. Bruxism (parafunctions) is extremely complex as it involves not only the masticatory system itself and its regulation by the central nervous system but seemingly further musculoskeletal structures as well. Sleep bruxism is seen today as a sleeping disorder (rhythmic body movements actually involve many other zones than the jaws), while awake bruxism seems mainly a reaction to stress and anxiety. The role of occlusion still remains unclear, apart from the logical role of stress distribution by dental contacts; the more occlusal contacts, the more evenly functional stresses are distributed on teeth, and vice versa.

In light of recent evidence, occlusion seems neither related directly to the incidence nor the severity of parafunctions; this however remains a highly controversial topic that necessitates further research to confirm the complex interactions between occlusion and the many systems involved in parafunctional activities (read more on this in chapter 3). Apart from the minor contribution of regular, proper personal

dental hygiene, the causes of atypical abrasion include aggressive tooth brushing (using too much force and wrong movements, stiff toothbrush, and abrasive toothpaste), biting nails (onychophagy), and chewing on a smoking pipe mouthpiece or pen, to cite the most common damaging habits. Again, the simultaneous interplay of various causes of wear (erosion, abrasion, and attrition) is a very common situation that creates a vicious cycle for hard tissue integrity and also makes it more intricate for the dental team having to act against several risk factors.

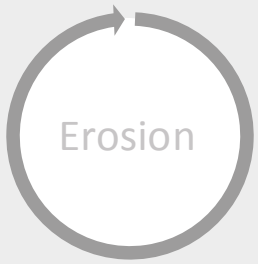
Abfraction

Abfraction lesions are non-carious cervical lesions (NCCLs) that result from tooth flexure arising from repeated, eccentric function. Tooth deformation deteriorates the thin cervical enamel prism structure, leading to dentin exposure above the cementoenamel junction (CEJ); this localization actually allows a differential diagnosis with other NCCLs, such as root abrasion induced by brushing in the presence of gingival recession. Abfraction lesions are likely to happen conjointly with other lesions like erosion and abrasion, lowering the occurrence of "mere" abfraction decays. However, the etiology and biomechanics of abfraction remain controversial in the literature. In fact, supposed abfraction lesions are not frequently observed.

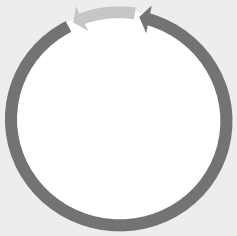


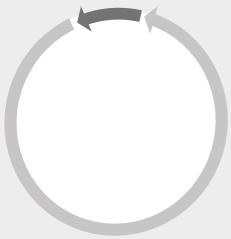
Interplay of wear mechanisms, risk factors, and co-factors

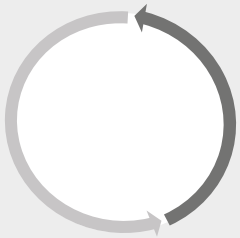
Unlikely scenarios suggesting an absolute predominance of one wear mechanism



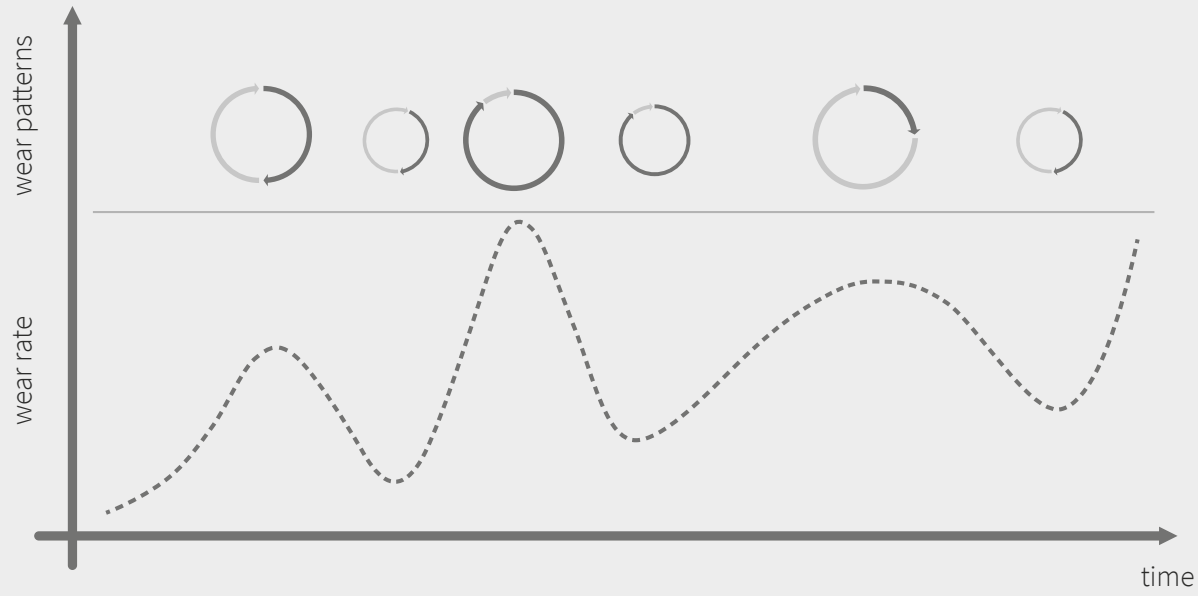
Most frequent and likely scenarios showing various levels of predominance or combinations of the two wear mechanisms







These diagrams suggest that the respective contributions of both wear mechanisms can greatly vary and are frequently associated; clinical observations confirm that this later tooth wear model is prevailing on the one of single mechanism. Moreover, risk factors are likely to vary over time while an alternate prevalence of wear mechanism and intensity is to be expected; this has a major implication in selecting the correct treatment approach. For instance, a patient can suffer from compulsory food behavior for a while and then, after controlling his diet, will return to a normal risk factor or on the contrary develop parafunctional activities if some underlying anxiety and stress factors are not controlled; the same "scenario" can obviously be reformulated with an infinite number of variations. Then, having to decide how to treat tooth wear while not knowing the true dynamics of the disease is a limiting factor for success. This is why an interceptive treatment approach using simple no-prep, chairside procedures with composite is best indicated for moderate and intermediate tooth wear, considering indirect restorations only for severe cases.



Incidence and prevalence of tooth wear

The incidence and prevalence of tooth wear seem to have increased over the last few decades, although not all authors agree on this hypothesis. Studies in various age groups and regions have however reported high incidence of tooth wear in all groups. Kreulen et al in their systematic review on tooth wear confirmed that lesions exposing dentin in deciduous teeth had a prevalence ranging from 0% to 82%, with significant correlation to age; in a systematic review and meta-analysis study, Salas et al estimated that the overall prevalence of tooth erosion in permanent teeth of children and adolescents was 30.4%; Bartlett et al reported a 29% incidence of substantial tooth wear in young European adults (age 18–35 years).

Kitasako et al found a prevalence of 26% tooth wear in Japanese adults; in another review by Van't Speiker et al, the prevalence of severe tooth wear appeared, logically, to increase from the ages of 20 years (3%) to 70 years (17%). Jaeggi and Lussi, when analyzing some literature data, also confirmed the high, increasing prevalence of tooth wear in younger age groups (mainly linked to the consumption of acidic drinks) and a correlation of tooth wear with age.

From a practitioner's perspective, it seems rather unquestionable that the occurrence of wear defects has increased in all age groups during recent decades.

Dental wear patterns and tooth wear diagnosis

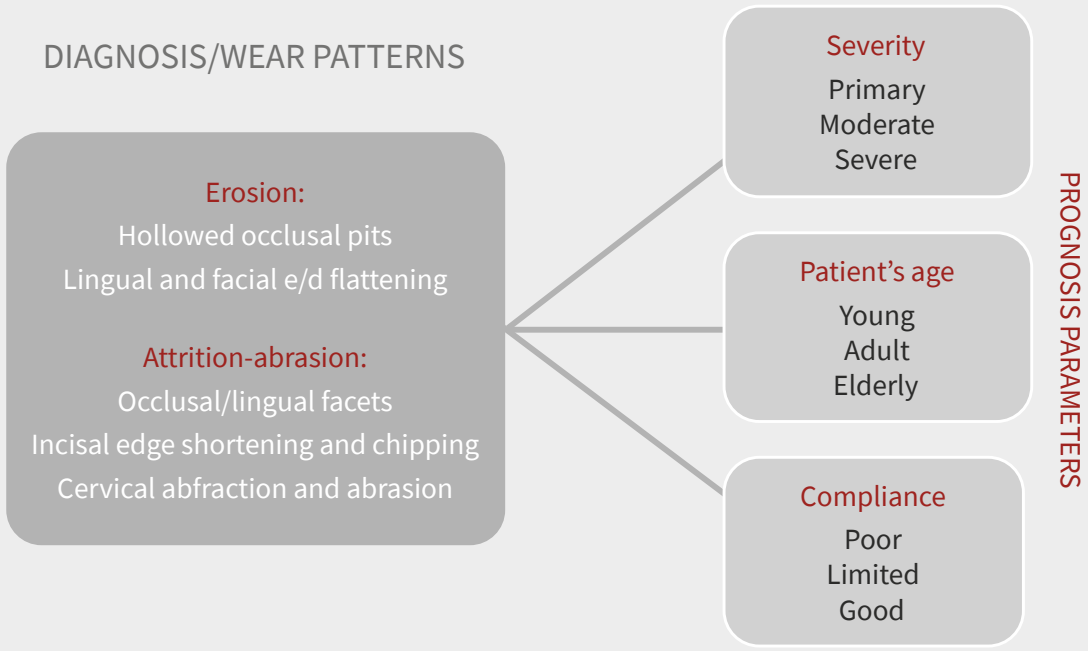
Depending on the type erosion process (extrinsic or intrinsic), one can observe a number of typical lesions:

- Occlusal hollows
- Smoothing of enamel surfaces, with prevalence in areas in direct contact with acidic food or beverage
- Complete loss of thin cervical enamel

For attrition and abrasion, loss of tooth structure appears more specifically in the form of wear facets or localized tooth fractures (enamel chipping) in these areas:

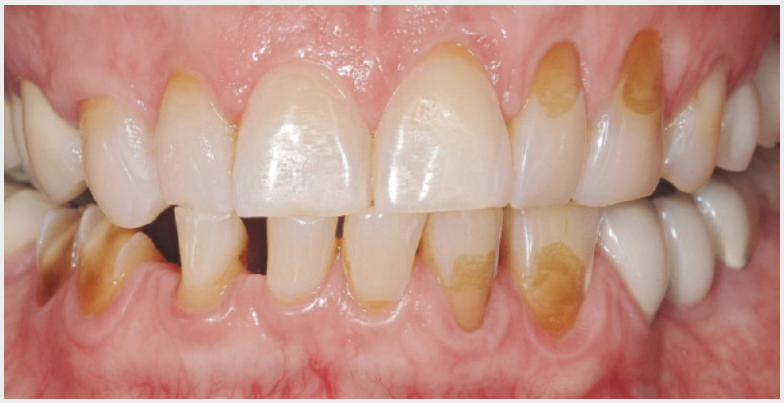
- Flattening of occlusal surfaces, starting with cusps
- Flattening and chipping of incisal edges and cuspid tips
- Flattening and indentation on palatal surfaces

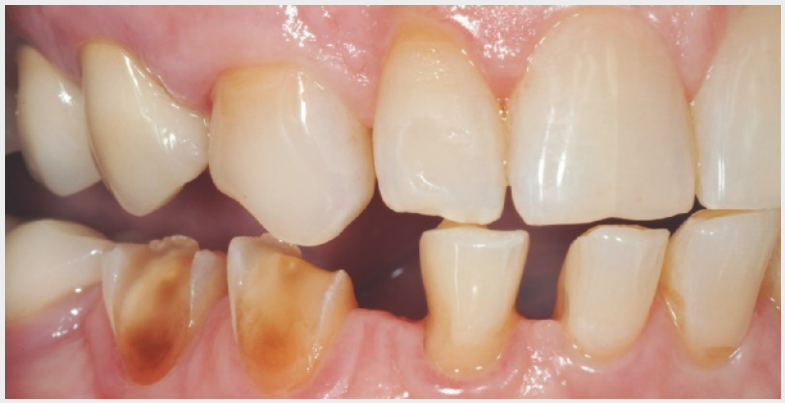
In combined cases (erosive and mechanical tooth wear), hybrid patterns or so-called “atypical forms” of tooth wear are observed. This is a very common finding, when one cause is not dominating, again because the majority of patients are exposed alternatively or simultaneously to both wear mechanisms.



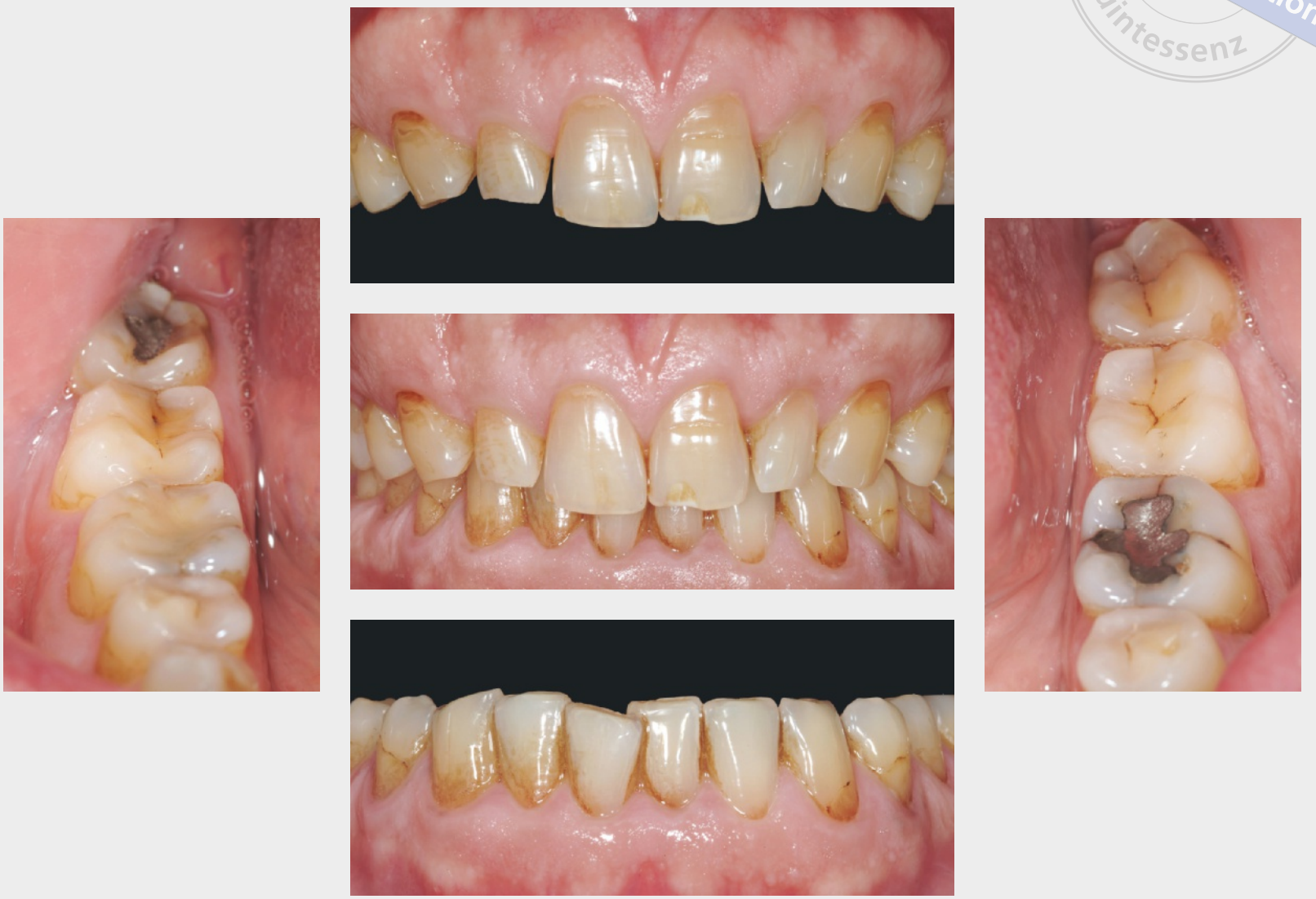
The extent of tooth wear considered physiologic and at what age related progression brings tooth structure damage to a pathologic level has been vastly and controversially discussed in the literature. Common sense and medical ethics however suggest to prevent wear as much as possible and install interceptive measures when prevention only is not enough to stop tooth wear progression. A few simple guidelines are then sufficient to help the practitioner toward the right decision between "acting" or just "observing." The diagnosis and prognosis parameters summarized in the previous diagram provide a simple base for monitoring and treating tooth wear cases. In short, when tooth wear impacts tooth esthetics or the biomechanical integrity of an individual or group of teeth or a full arch or mouth, our action is needed, involving preventive measures (in any case) up to more comprehensive restorative procedures.

Various wear indices have been recommended to study the incidence and severity of tooth wear at enamel and dentin levels; however, these indices are usually too detailed and then inappropriate for the general practice. Such research tools can be advantageously replaced by a simpler approach involving three prognosis parameters, next to the identified risk factors (see lower diagram of previous page). The first parameter is wear severity (locally or globally), and the other two relate to the patient's age and known (or expected) compliance with treatment and risk control recommendations. The relationship of tooth wear severity to the patient's age and compliance are good indicators of potential risk factor control, treatment complexity, and its likely outcome.

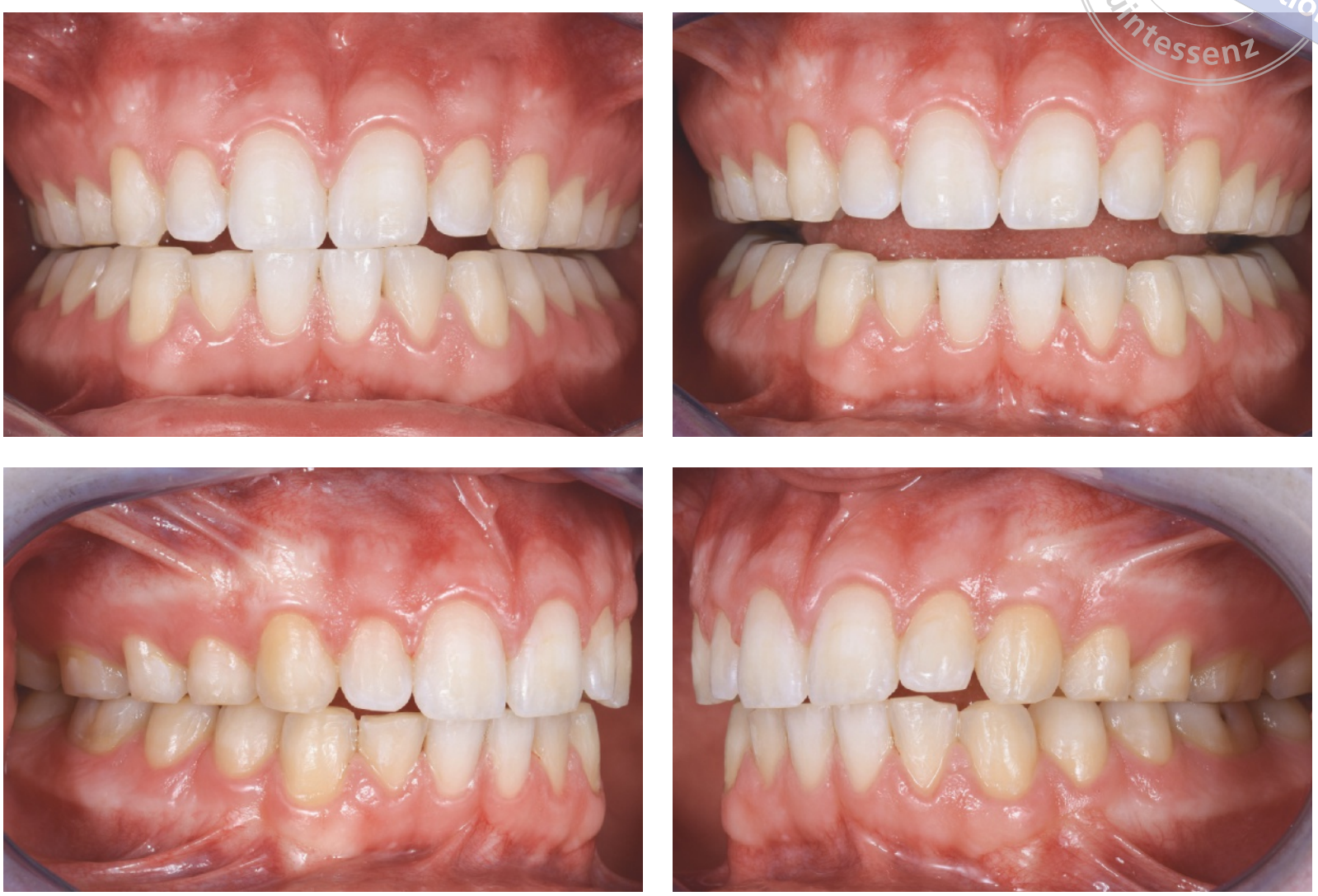




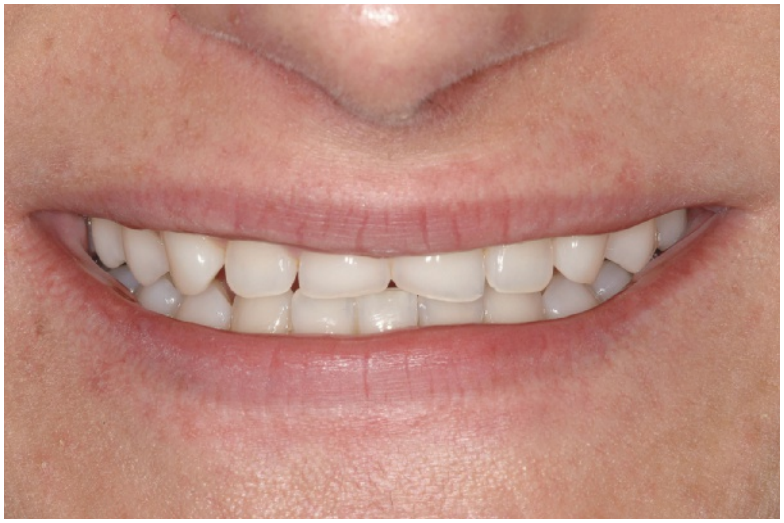
Multifactorial wear risk factors with combined patterns and varying, localized extent is a highly common finding (right page).

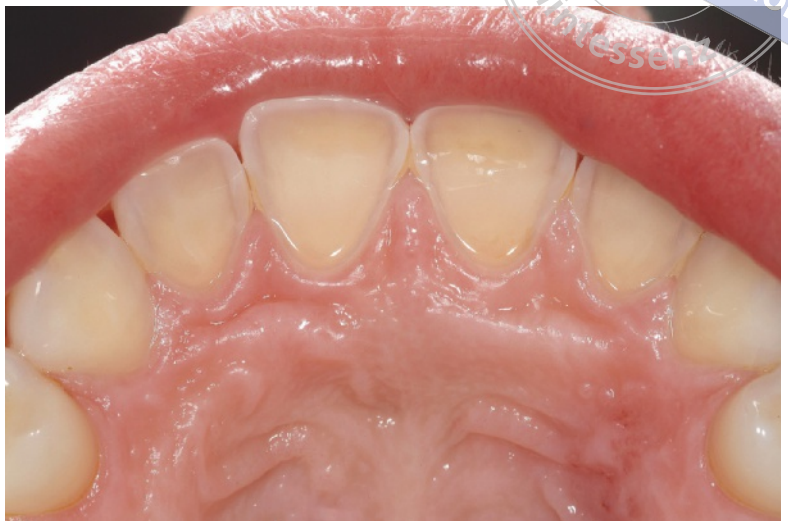


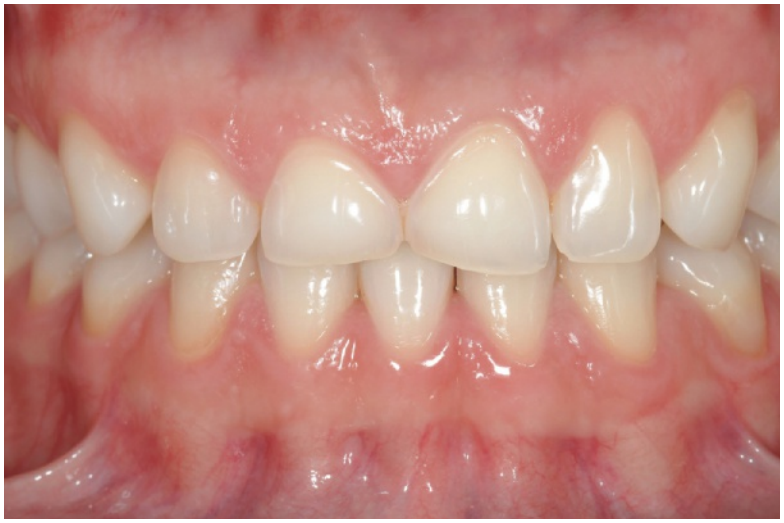
This 60-year-old patient had only the chief complaint of discoloration and unesthetic smile appearance. Clinical examination revealed moderate generalized tooth wear with evident signs of bruxism (shortened incisal edges and canine tips) and clenching (concave occlusal anatomy of molars with mesiodistal cracks). Such findings call for preventive/interceptive measures, despite the patient's age, which suggests a physiologic wear extent.

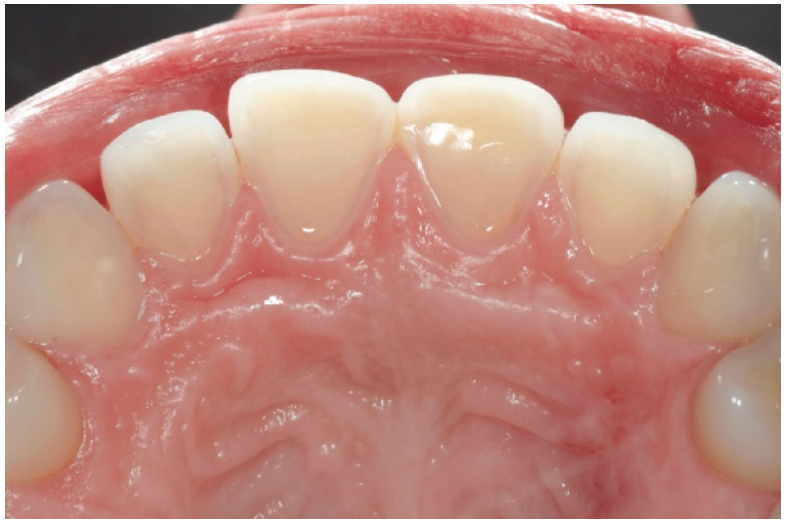


Young male patient (aged 17 years) showing advanced tooth wear from parafunctional activities; attrition is clearly the main wear mechanism involved here with flattening of cusps and wear facets. The risk factor (correlation between patient's age, wear severity, and potential progression) is considered high due to the patient's age.

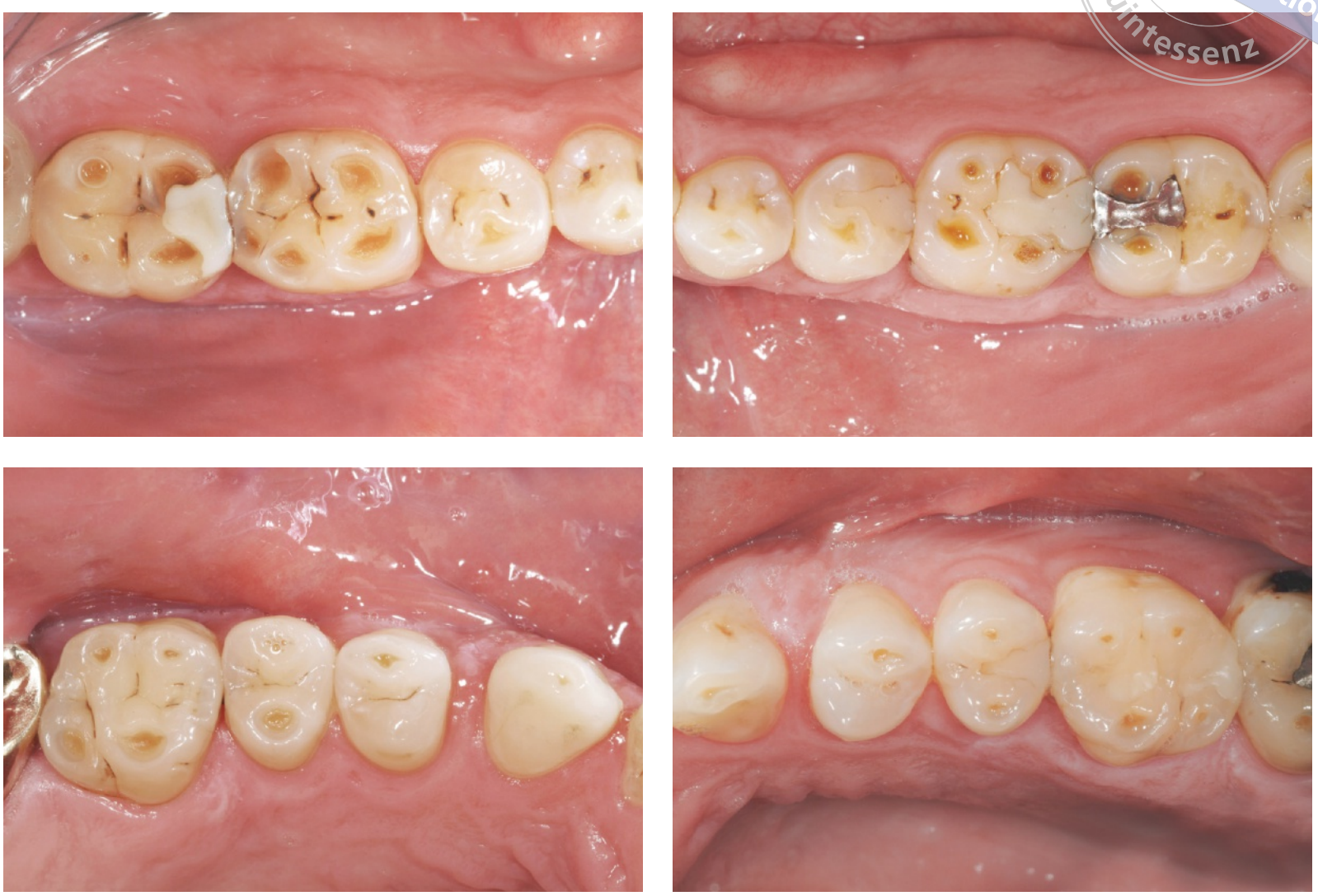




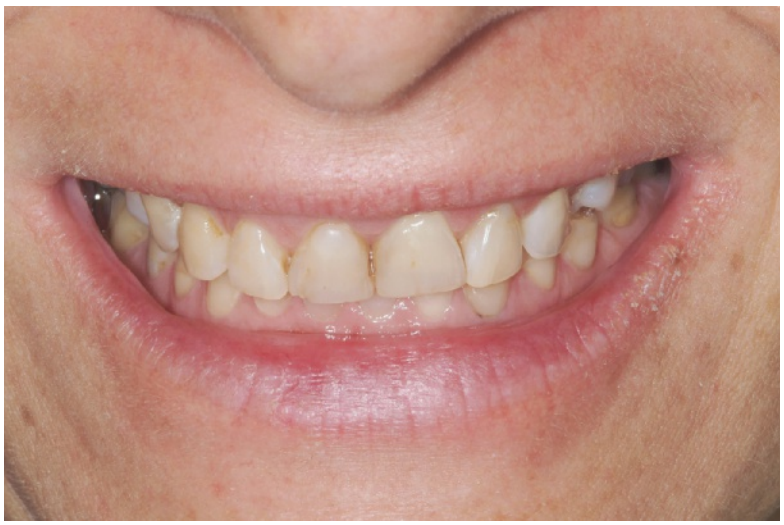
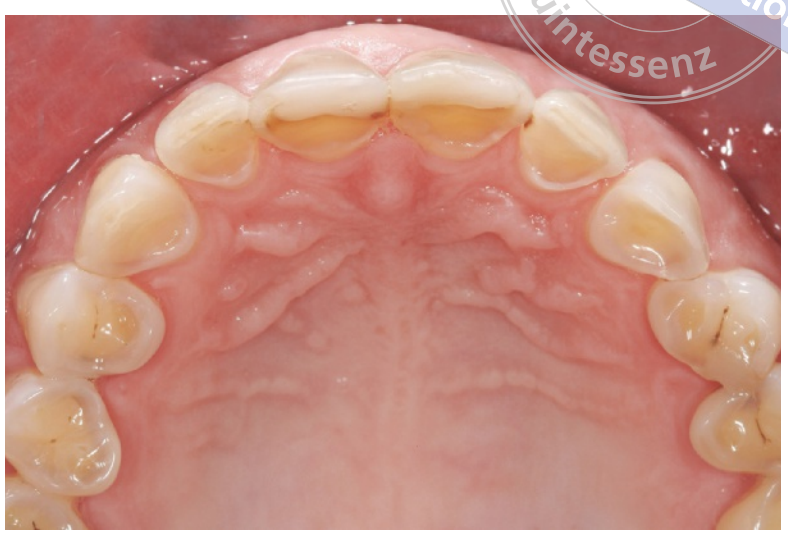
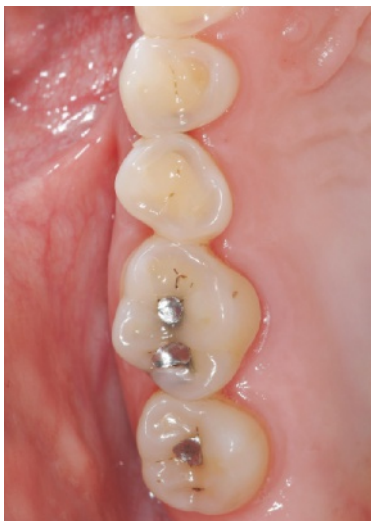
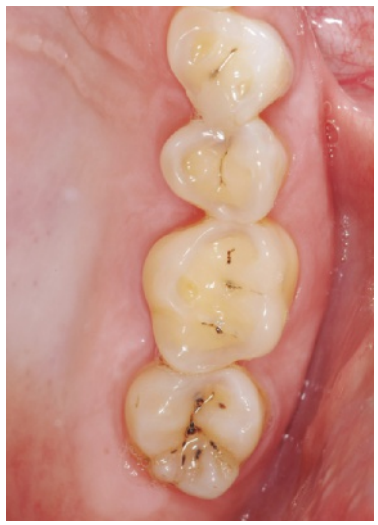
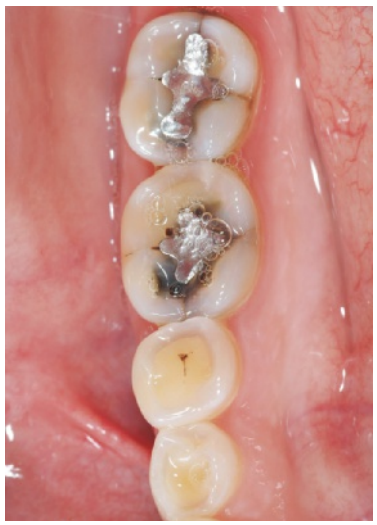
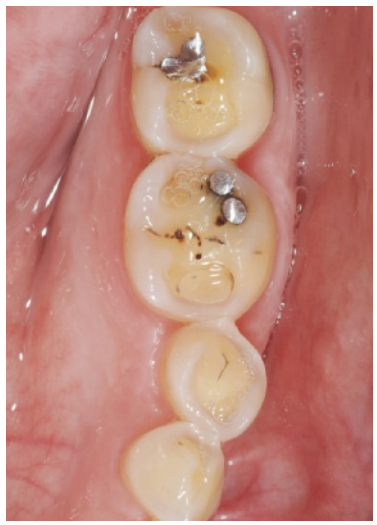




Young female patient (aged 19 years) with localized erosion of the four maxillary incisors due to compulsory consumption of fruits. Note the smooth edges of worn incisal edges, in contrast to attrition induced by bruxism that triggers enamel chipping. The risk factor is considered moderate as it is controllable with proper patient guidance and awareness.

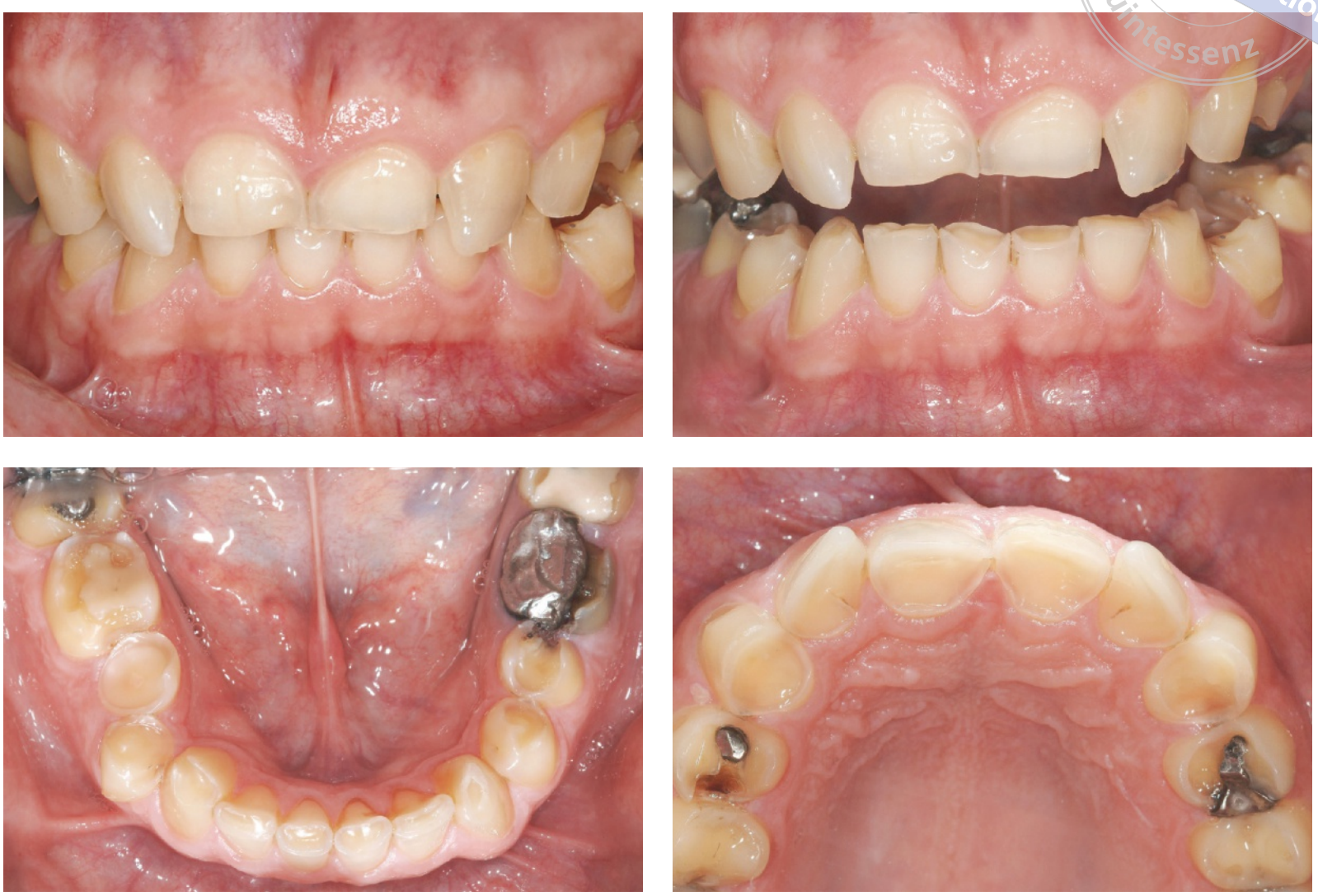


Male patient (aged 40 years) with typical hollowed occlusal lesions; erosion appears to be the predominant wear mechanism, although wear facets (attrition) are also present and suggest combined risk factors. The risk factor is moderate to intermediate.

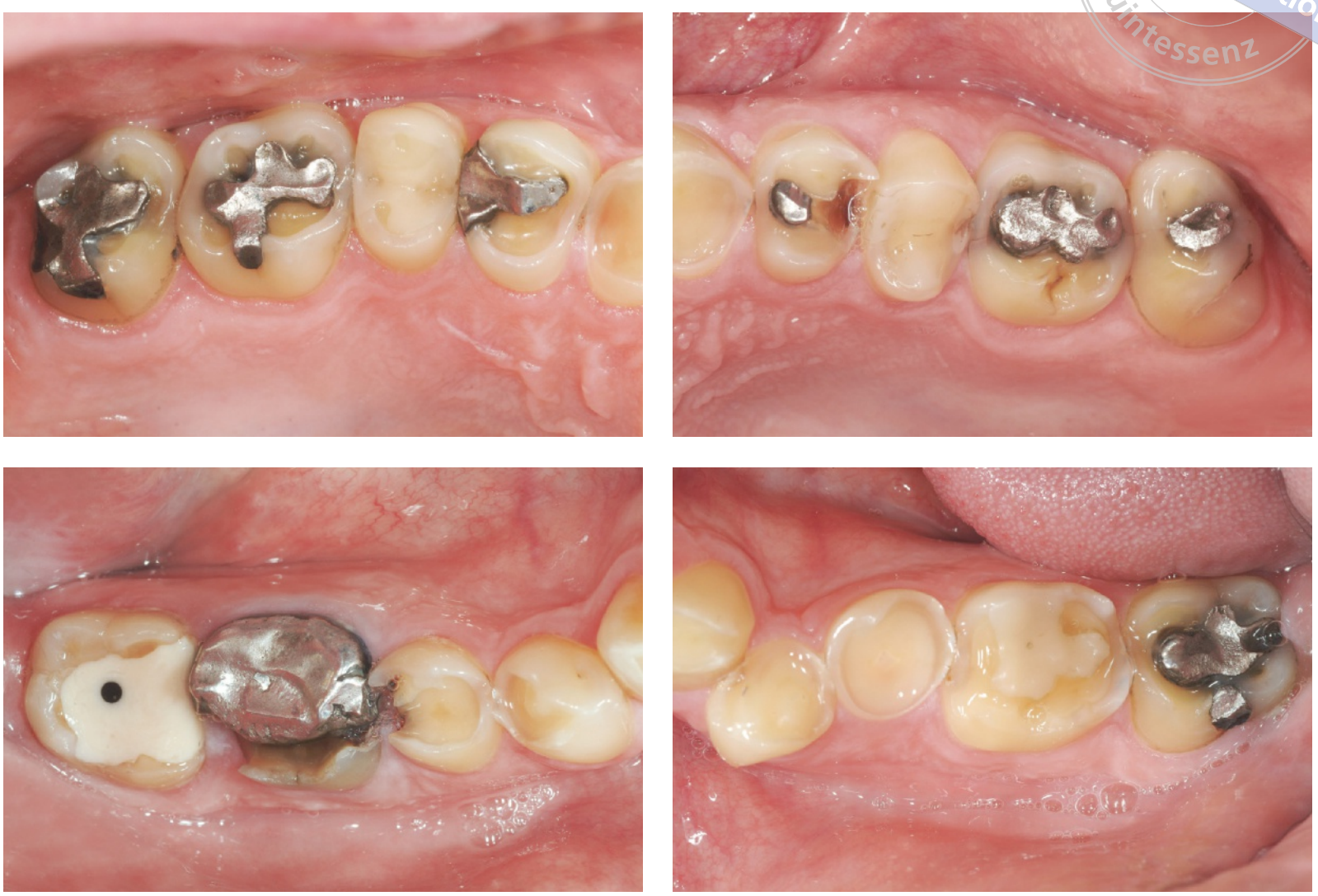







This female patient (aged 39 years) consulted because of the esthetic impact of tooth wear on her smile appearance (flattening of the smile line). The overall buccal examination revealed significant erosion of the palatal enamel of all anterior teeth and occlusal surfaces of mandibular and maxillary posterior teeth. This wear pattern is typical for erosion, keeping in mind the likely contribution of the additional attrition co-factor. The risk is considered intermediate for a middle-aged adult.

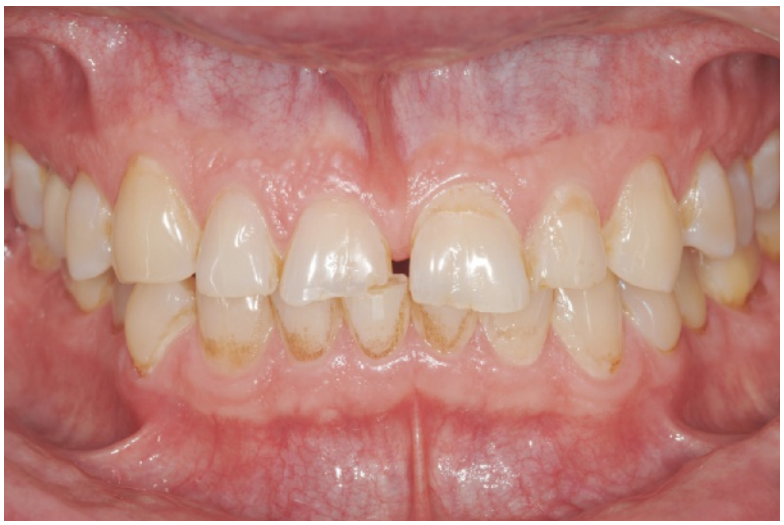
Severe erosion

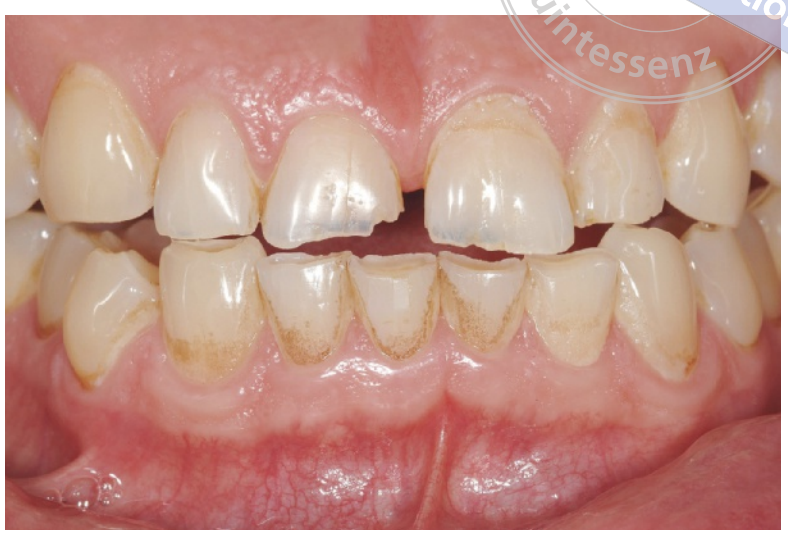


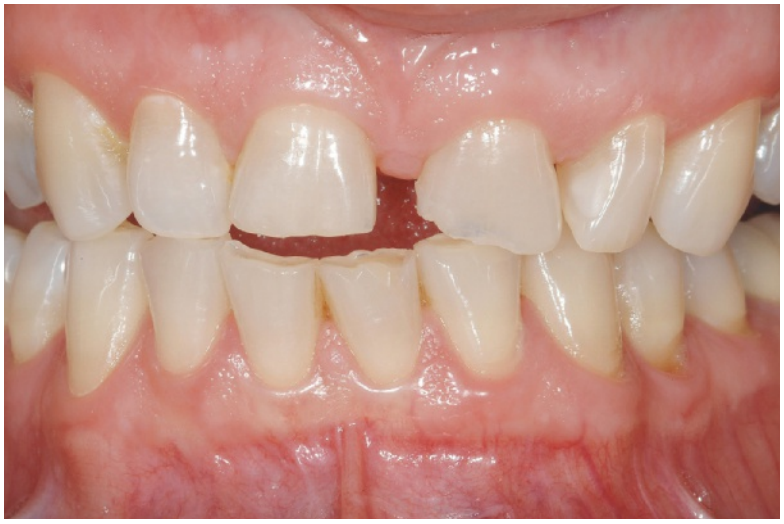
Male patient (aged 36 years) showing extensive signs of tooth wear with predominant erosion pattern; the contribution of attrition to the rapid loss of tooth structure was likely for this case. The patient is showing typical dental impact of bulimia nervosa.

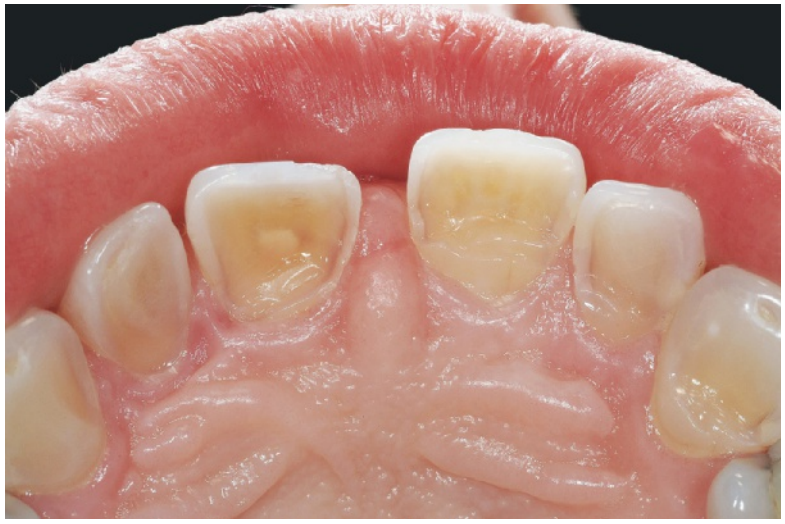


Some additional carious risk factor is present. The overall risk factor is extremely high in such cases (ie, bulimia and anorexia).

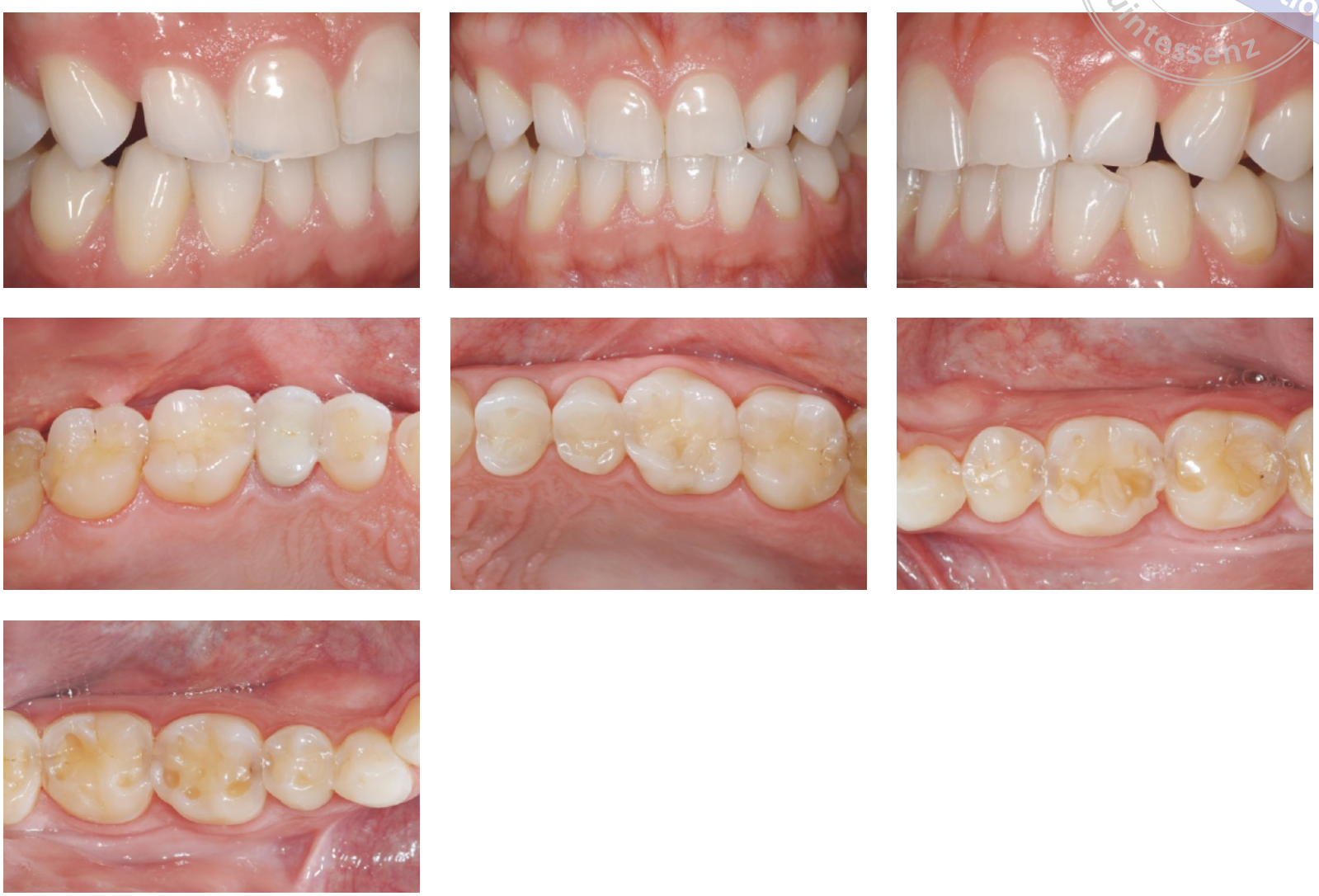






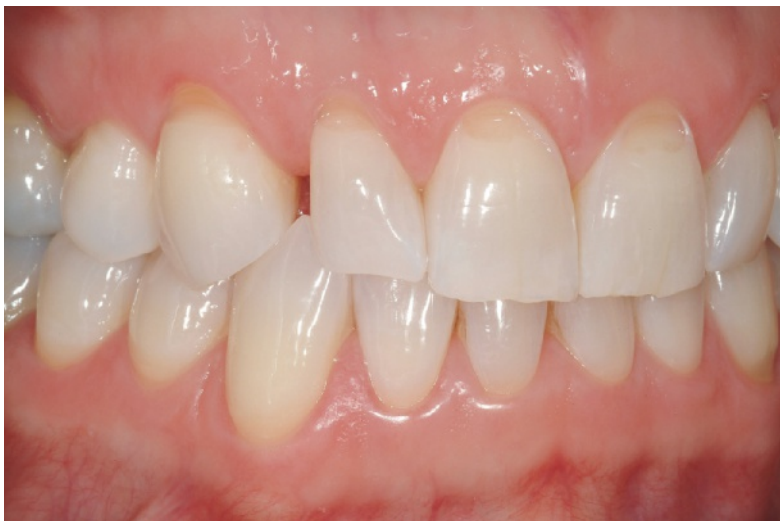


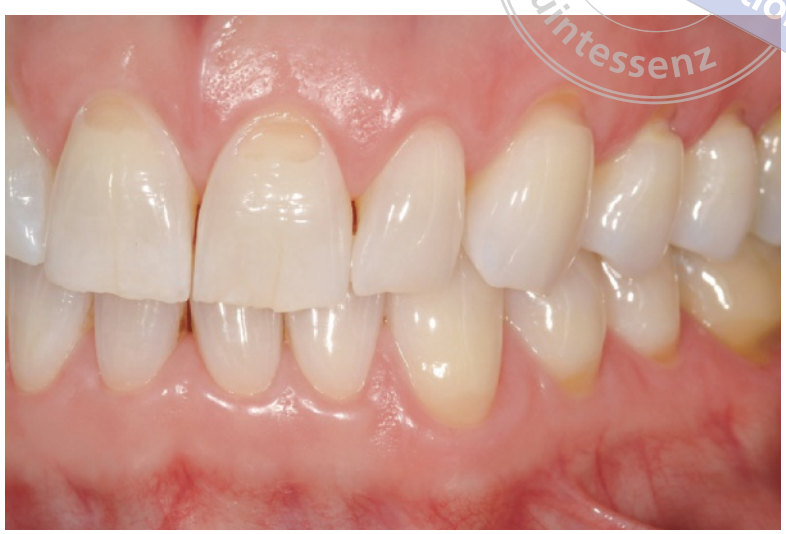
The two cases presented here (above, male patient aged 55 years, and below, female patient aged 48 years) show similar wear patterns induced mainly by attrition (bruxism); note the irregular, chipped incisal edges. Depending on the overbite extent, wear will involve more or less of the maxillary palatal surfaces. The risk factor for both cases is intermediate.



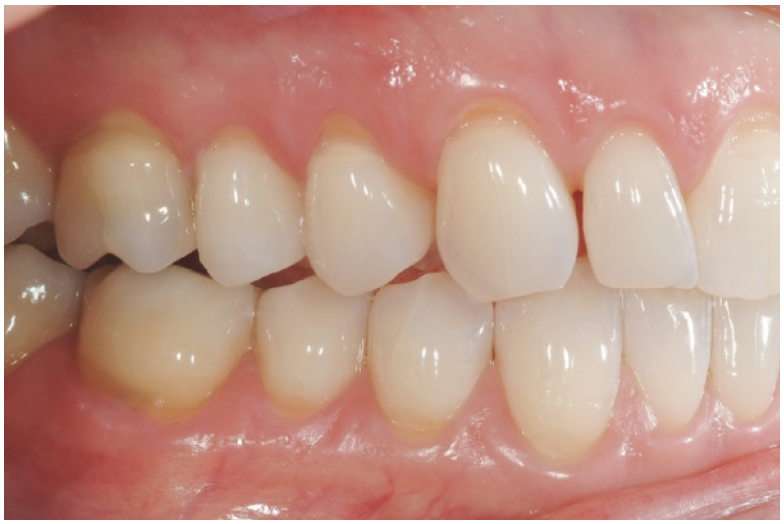
Male patient (aged 41 years) presents combined wear patterns with occlusal hollowed lesions and wear facets, as a result of excessive acidic beverage consumption and moderate parafunctions, without any protective means (ie, mouthguard). The incidence of combined erosion/attrition is very high and makes it the most prevalent condition. The risk factor is considered moderate to intermediate.

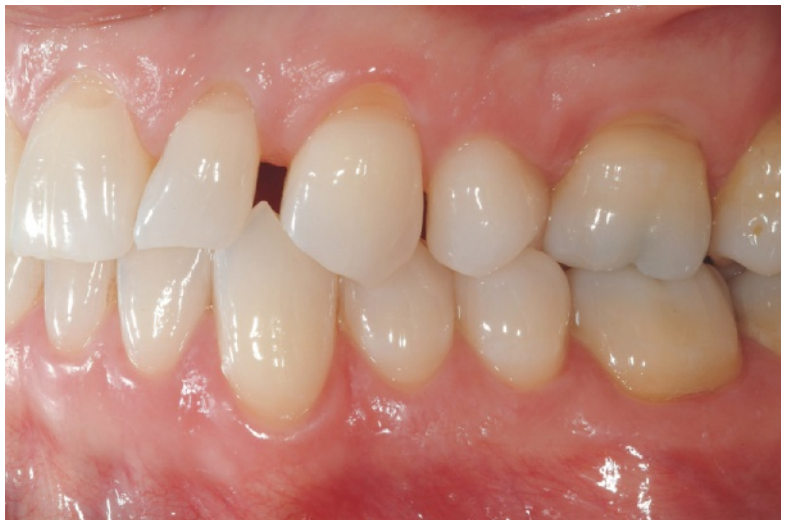
Abfraction

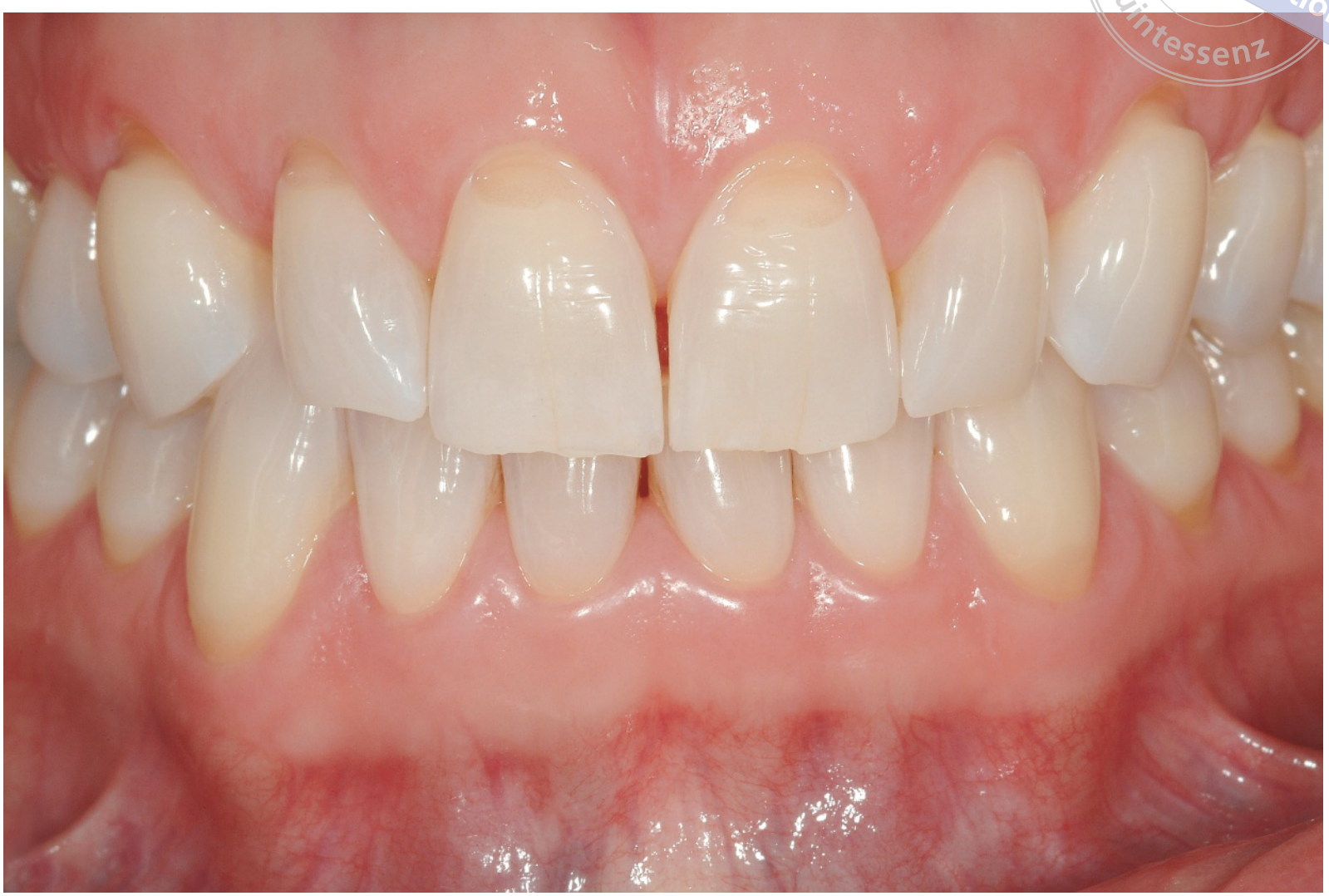




Abfraction is conceptually a mechanical disruption of cervical enamel through the action of excessive tooth flexure due to bruxism. Enamel prisms are then progressively lost either spontaneously or during tooth brushing of fragilized enamel (abrasion). As soon as dentin is exposed, erosion and abrasion can worsen hard tissue loss.

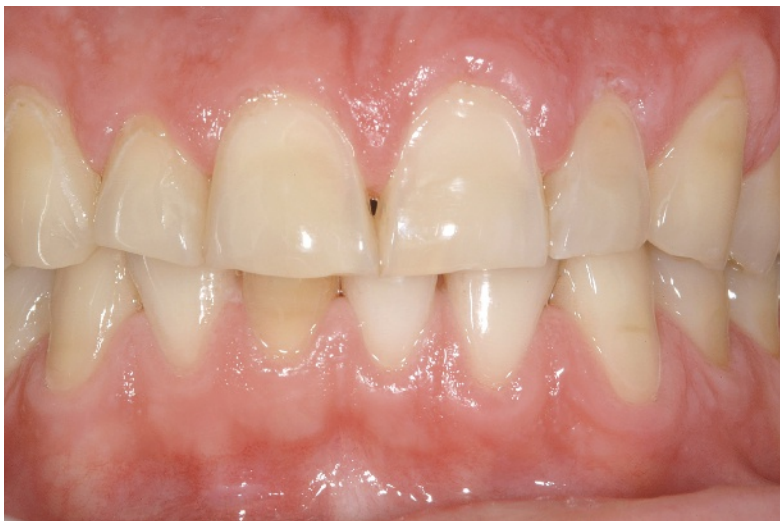


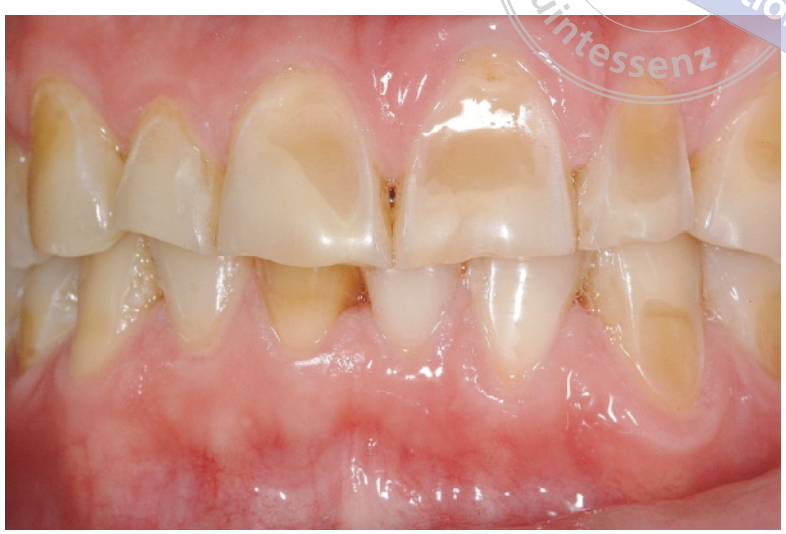


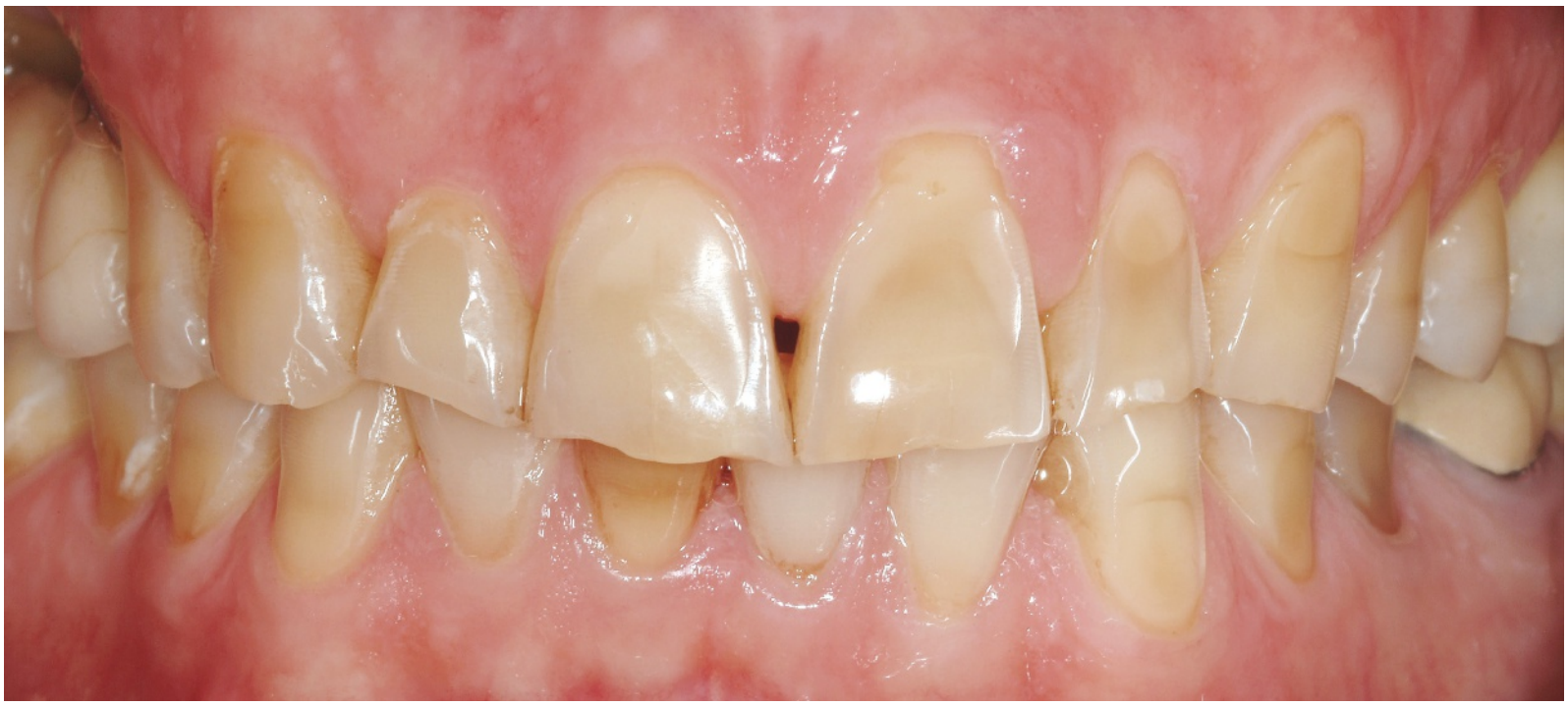


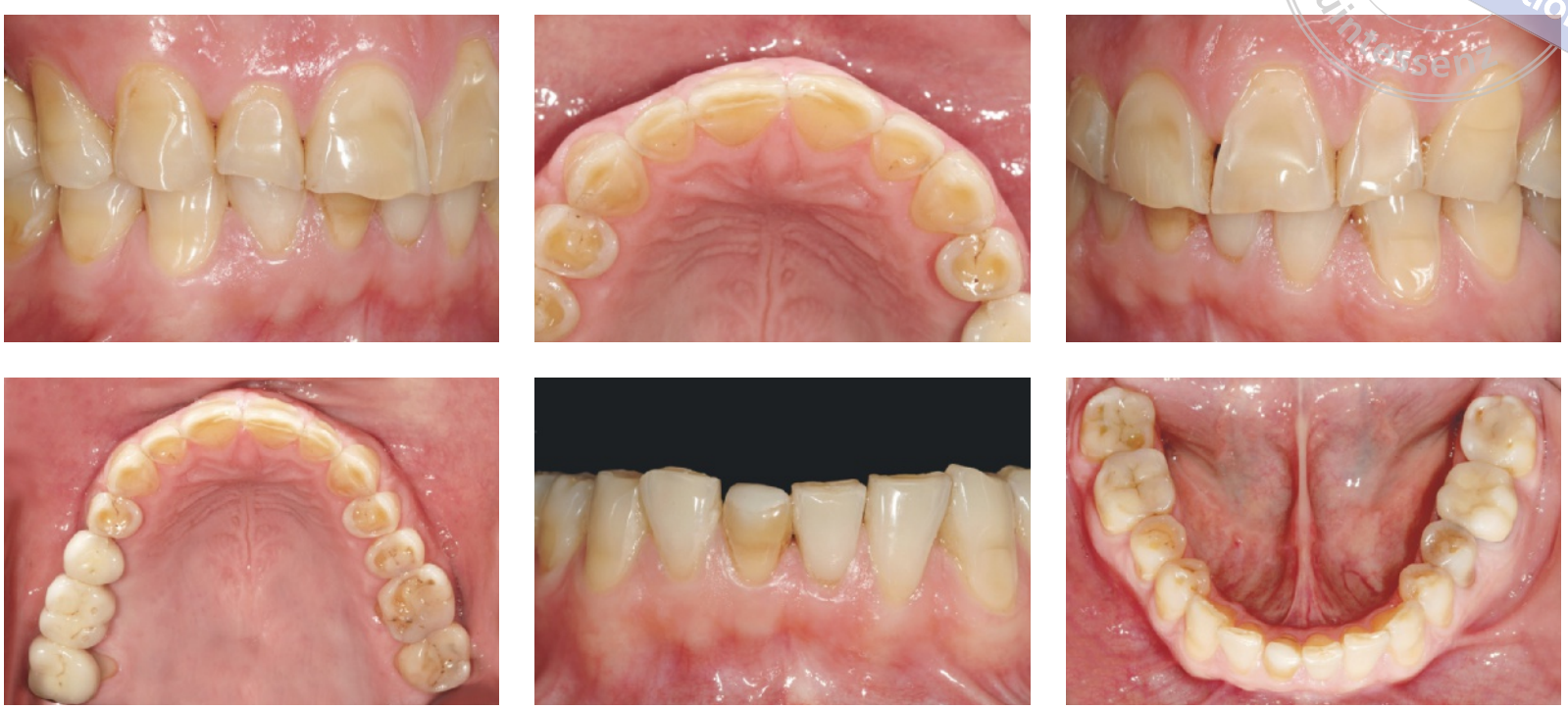
The anatomical peculiarity of abfraction (which still remains a controversial mechanism) is the localization of lesions, namely cervical enamel above the CEJ; this makes it possible to differentiate abfraction from abrasion of root dentin in case of gingival recessions. Abfraction lesions tend to affect teeth overloaded by excursive movements in bruxers (see here all maxillary anterior teeth, up to premolars). The nature of forces induced by parafunctional movements might explain why facial surfaces of maxillary teeth (submitted to compressive/shear forces) are more affected than mandibular teeth (submitted to tensile/shear forces).

Tooth wear progression



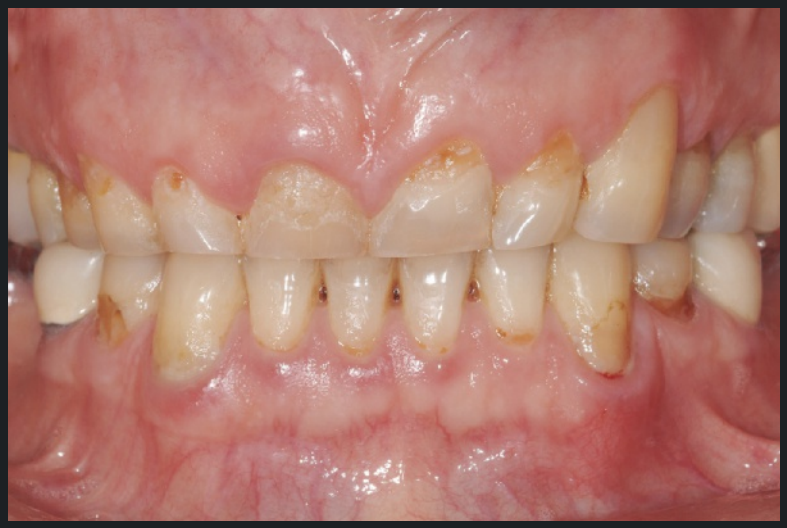


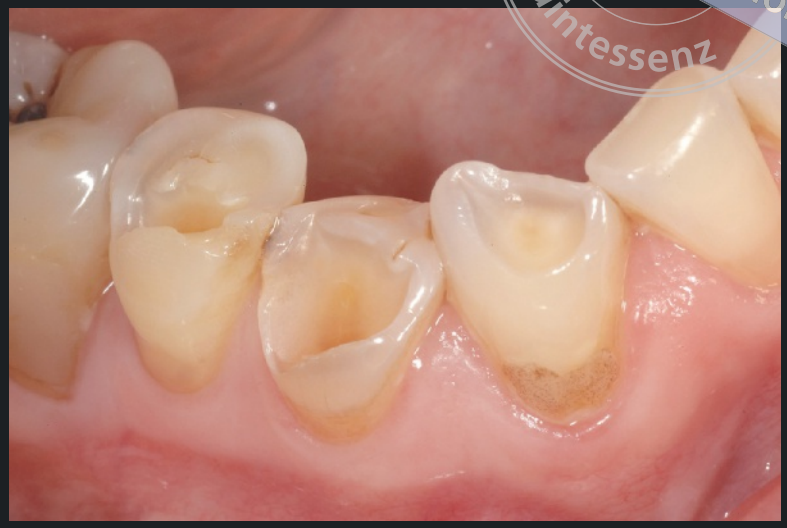




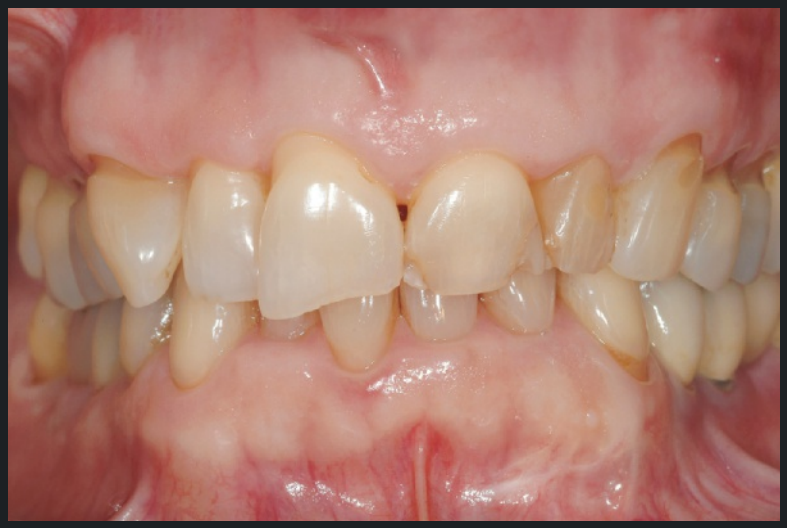
Evolution of erosive tooth wear in a male patient (aged 32 years) at the first visit (left page, upper left); the 8-year (left page, upper right) and 12-year (left page, lower image and above) follow-up visits show significant progression of the tissue loss. The patient denied having any abnormal diet and did not consider our suggestion for consultation with a gastroenterologist. As the wear more severely involves the facial aspect of the teeth, the extrinsic acidic origin was the privileged diagnosis, with likely contribution of attrition and abrasion (aggressive brushing). Without the patient's proper compliance, wear pathologies will likely evolve toward significant progression of tissue loss. Clinicians must be aware that patients occasionally refuse even any preventive or interceptive measures to stabilize their problem.

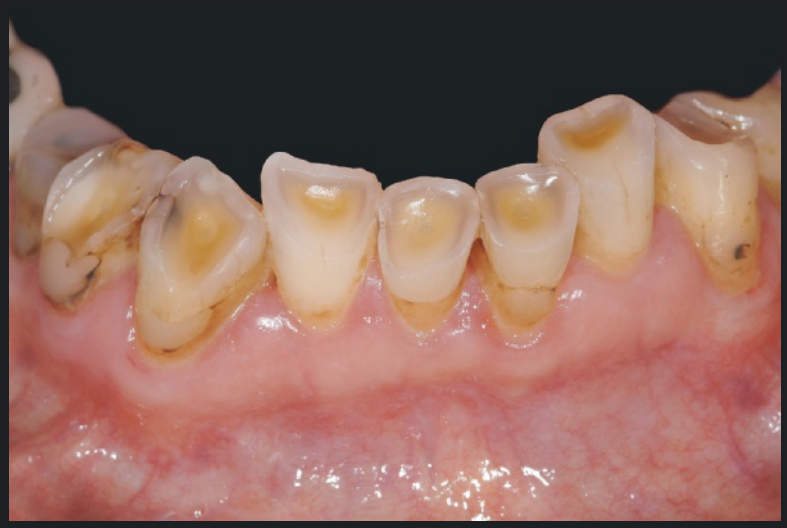
Age-related tooth wear





Time is a crucial factor to assess tooth wear severity and prognosis as evidenced in these intraoral images of elderly patients. Without any preventive measures, attrition and erosion will lead to significant, damaging effect on natural or restored dentition, even without exceeding "physiologic" levels of erosion, attrition, and abrasion. This deleterious effect on tooth structure is unavoidable without preventive measures (see following chapters).





Tooth wear is a common term to describe a **multifaceted** pathology; despite the fact a few patients present lesions linked clearly to a predominant wear mechanism, there is only a low prevalence of this condition. On the contrary, the vast majority of patients affected by tooth wear suffer from a combination and effects of multiple risk factors and co-factors, leading to various localizations, severity levels, and dynamics of hard tissue loss.

A detailed **oberservation** of existing wear lesions and monitoring, if appropriate (mild and moderate severity), with the help of the patient's medical history, allows us to comprehend or at least approach more closely the globality of risk factors. Again, one should not forget the time dynamics and interplay of identified risk factors; with analogy to financial investment strategies, former and current results (clinical findings) are not always predictive of future outcomes (progression and mechanisms of tooth wear)!

Recommended Readings

1. Bartlett DW, Fares J, Shirodaria S, Chiu K, Ahmad N, Sherriff M. The association of tooth wear, diet and dietary habits in adults aged 18–30 years old. J Dent 2011;39:811–816.
2. Bartlett DW, Lussi A, West NX, Bouchard P, Sanz M, Bourgeois D. Prevalence of tooth wear on buccal and lingual surfaces and possible risk factors in young European adults. J Dent 2013;41:1007–1013.
3. Hellstrom I. Oral complications in anorexia nervosa. Scand J Dent Res 1977;85:71– 86.
4. Huysmans MC, Chew HP, Ellwood RP. Clinical studies of dental erosion and erosive wear. Caries Res 2011;45(suppl 1): 60– 68.
5. Imfeld T. Dental erosion. Definition, classification and links. Eur J Oral Sci 1996;104:151–155.
6. Kitasako Y, Yoshiyuki Sasaki Y, Takagaki T, Sadr A, Tagami J. Age-specific prevalence of erosive tooth wear by acidic diet and gastroesophageal reflux in Japan. J Dent 2015;43:418–423.
7. Kreulen M, Van 't Spijker A, Rodriguez JM, Bronkhorst EM, N.H.J. Creugers NHJ, Bartlett DW. Systematic review of the prevalence of tooth wear in children and adolescents. Caries Res 2010;44:151–159.
8. Lavigne GJ, Kato T, Kolta A, Sessle BJ. Neurobiological mechanisms involved in sleep bruxism. Crit Rev Oral Biol Med 2003;14:30–46.
9. Lee AL, Goldstein RS. Gastroesophageal reflux disease in COPD: Links and risks. Int J Chron Obstruct Pulmon Dis 2015;10:1935–1949.
10. Linkosalo E, Markkanen H. Dental erosions in relation to lactovegetarian diet. Scand J Dent Res 1985;93:436–441.
11. Litonjua LA, Andreana S, Bush PJ, Cohen RE. Tooth wear: Attrition, erosion and abrasion. Quintessence Int 2003;34:435–446.
12. Loomans B, Opdam N, Attin T, et al. Severe Tooth Wear: European Consensus Statement on Management Guidelines. J Adhes Dent 2017;19:111–119.
13. Lussi A, Schlueter N, Schmalz G, et al. Consensus Report of the European Federation of Conservative Dentistry. Clin Oral Investig 2015;19:1557–1561.
14. Lussi A, Ganss C (eds). Erosive Tooth Wear: From Diagnosis to Therapy, vol 25, Monographs in Oral Science. Basel: Karger, 2014.

15. McGuire J, Szabo A, Jackson S, Bradley TG, Okunseri C. Erosive tooth wear among children in the United States: Relationship to race/ethnicity and obesity. Int J Paediatr Dent 2009;19:91–98.
16. Meurman JH, Vesterinen M. Wine, alcohol, and oral health, with special emphasis on dental erosion. Quintessence Int 2000;31:729–733.
17. Okunseri C, Wong MC, Yau DT, McGrath C, Szabo A. The relationship between consumption of beverages and tooth wear among adults in the United States. J Public Health Dent 2015;75:274–281.
18. Reddy A, Norris DF, Momeni SS, Waldo B, Ruby JD. The pH of beverages in the United States. J Am Dent Assoc 2016;147:255–263.
19. Salas MM, Nascimento GG, Vargas-Ferreira F, et al. Diet influenced tooth erosion prevalence in children and adolescents: Results of a meta-analysis and meta-regression. J Dent 2015;43:865–875.
20. Salas MMS, Nascimento GG, Huysmans MC, Demarco FF. Estimated prevalence of erosive tooth wear in permanent teeth of children and adolescents: An epidemiological systematic review and meta-regression analysis. J Dent 2015;43:42–50.
21. Schlossman M, Montana M. Preventing damage to oral hard and soft tissues. In: Spolarich AE, Panagakos FS (eds). Prevention Across the Lifespan: A Review of Evidence-Based Interventions for Common Oral Conditions. Charlotte, NC: Professional Audience Communications, 2017:97–120.
22. Schlueter N, Amaechi BT, Bartlett D, et al. Terminology of Erosive Tooth Wear: Consensus Report of a Workshop Organized by the ORCA and the Cariology Research Group of the IADR. Caries Res 2020;54:2–6.
23. Sovik JB, Skudutyte-Rysstad R, Tveit AB, Sandvik L, Mulic A. Sour sweets and acidic beverage consumption are risk indicators for dental erosion. Caries Res 2015;49:243–250.
24. Wetselaar P, Manfredini D, Ahlberg J, et al. Associations between tooth wear and dental sleep disorders: A narrative overview. J Oral Rehabil 2019;46:765–775.

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