Jorge Perdigão
Editor

Tooth Whitening

An Evidence-Based Perspective

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Preface

Our team started working on this project immediately after I finished editing and writing the book *Restoration of Root Canal-Treated Teeth: An Adhesive Dentistry Perspective* (Springer, 2016). As with the previous book, this new book project made me feel truly blessed to have known so many talented colleagues from different parts of the world. The countries represented in this book include Brazil, Germany, Portugal, Spain, and the United States of America.

More interestingly, the coauthors of this book represent different generations of dental professionals. We will not mention here how old the oldest authors are, but the two youngest authors were born in 1987 and 1989. Dentistry is indeed an outstanding global and beautiful vocation.

The driving force behind the current book was the need for a compilation of independent evidence-based information on dental whitening. We have all fielded questions from patients inquiring about different whitening methods, including over-the-counter bleaching, *as-seen-on-TV* laser bleaching, shopping-mall bleaching, and jump-start bleaching, just to mention a few. As a dental professional, I have been asked about bleaching techniques that I had never heard before, mostly anecdotal, yet the patients had read all the details about these supposedly *cutting-edge* methods online.

My ultimate goal is to contribute to a better understanding of dental whitening and how we can improve its outcome based on the available evidence.

Thank you for reading.

Minneapolis, MN, USA — Jorge Perdigão

Acknowledgments

My gratitude extends to *all* my current and former students, mentors, and teachers. I am also fortunate to have worked in clinical and research projects with so many gifted coworkers in so many countries. And I am extremely appreciative of my family for their patience and support. We never quit.

Jorge Perdigão

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Part I

Tooth Whitening with Peroxides

Introduction to Tooth Whitening

1

So Ran Kwon

Abstract

Few dental treatments have been more successful and conservative in nature than tooth whitening. Therefore, it is noteworthy to mention the efforts of pioneers in our dental profession who continuously attempted to search for the most effective and safest whitening agent. This quest has extended to determine the best whitening technique to meet our patients' desires and expectations about the aesthetic outcome. Here, a short history of tooth whitening agents developed and employed based on the type of discoloration is summarized, as is our current knowledge on the relative efficacy and safety of various types of tooth whitening regimens available. The information on proper diagnosis and treatment planning will guide the clinician in establishing a step-by-step protocol for determining the etiology of the discoloration, selecting the best whitening technique, and monitoring tooth color until the desired outcome has been achieved.

1.1 History of Tooth Whitening

Tooth whitening is a conservative and effective method to lighten discolored teeth and has been practiced in dentistry for many centuries. During the course of development, careful observation and research on various materials and techniques enabled the dental profession to introduce effective, safe, and predictable methods of whitening.

In the mid-1800s, crowns were commonly used for the treatment of discolored teeth (Kirk 1906). However, early pioneers were concerned with the aggressive

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removal of tooth structure using this technique and found tooth whitening to be a promising alternative (Kirk 1906). Despite the great plea for preservation of tooth structure and less invasive dentistry, the majority of practitioners opposed tooth whitening and argued that it was technique sensitive, the duration of treatment was too long, and the relapse of color to the original shade was too frequent (Kirk 1889). Nevertheless, the quest for the ideal whitening material continued and resulted in numerous experimental whitening agents, which were either direct or indirect oxidizers employed mostly for the treatment of nonvital teeth (Kirk 1889). A variety of whitening agents were used, reflecting the diverse nature of discoloration. For example, oxalic acid removed iron stains associated with pulp necrosis and hemorrhage (Atkinson 1862), chlorine was indicated for silver and copper stains encountered with amalgam restorations (Kirk 1889), and ammonia readily removed iodine stains caused by root canal therapy (Stellwagen 1870). The most resistant stain, originating from metallic salts of metallic restorations, was removed using cyanide of potassium, although due to toxicity, its use was not recommended.

While most of the early dental literature focused on nonvital bleaching, as early as 1868, whitening of vital teeth was being attempted with oxalic acid (Latimer 1868). Hydrogen peroxide, currently the most widely used whitening material, was reportedly used in 1884, and was called hydrogen dioxide by Harlan (Harlan 1884). At the time, chemical manufacturing companies were relatively unrestricted, as were dentists, who were at liberty to mix their own solutions in their office (Haywood 1992). Early work also experimented with a variety of ways to speed the bleaching process, including electric current (Westlake 1895), ultraviolet rays (Rosenthal 1911), and other heating instruments and lights (Abbot 1918; Fisher 1911). Manufacturing companies introduced bleaching products in the early 1900s, a transition that limited the choice of materials available to the dental profession (Haywood 1992). The product Superoxol was introduced by a manufacturing company, and developed into the bleaching agent used by the majority of dentists because of its efficacy and safety (Haywood 1992).

The introduction of easy and safe to use bleaching agents eventually gave rise to over-the-counter (OTC) products that could be used at home.

The innovative technique of home whitening can be traced back to the orthodontist, Bill Klusmier, in the late 1960s in Fort Smith, Arkansas (Haywood 1991). While treating a patient during the orthodontic retention phase, he recommended placing Gly-oxide, an oral antiseptic containing 10 % carbamide peroxide (Marion Merrel Dow, Inc.) into the orthodontic positioner at night to facilitate tissue healing (Haywood 1991). He noticed a significant improvement in tissue health and an additional benefit of lightening of tooth color. Further investigation using 10 % carbamide peroxide in a custom-made tray worn at night led to the first publication on "Nightguard Vital Bleaching" in 1989 (Haywood and Heymann 1989). This technique caused a major shift from the in-office use of highly concentrated hydrogen peroxide with activating lights, to home whitening using lower concentrations of carbamide peroxide. In addition, this technique had fewer side effects and could be offered to a larger section of the general patient population at a lower cost (Haywood 1992).

Since the introduction of Nightguard Vital home whitening, the formula has been continually improved. Carbopol was added to increase the gel viscosity, so it would stay in the tray longer. This also enabled slow release of the active ingredient, increasing the duration of its effectiveness (Matis et al. 1999). Since some were concerned that whitening gels might cause enamel erosion, various forms of fluoride were added to the formulation. Tooth sensitivity was one of the most common reason patients gave for stopping the whitening process before the desired endpoint, so desensitizers were added, such as potassium nitrate, sodium fluoride, and amorphous calcium phosphate. These additions effectively reduced the incidence and severity of tooth sensitivity (Browning et al. 2008; Gallo et al. 2009; Maghaireh et al. 2014; Navarra et al. 2014; Wang et al. 2015).

As demand for white teeth increased, manufacturers began supplying over-the-counter products (OTC). The early OTC products were introduced in 1990, and involved a three-step system: an acid prerinse, a lower strength peroxide material, and a final toothpaste. Most often, these systems were inappropriately used, causing damage to the enamel (Cubbon and Ore 1991). Strip technology, which involved placing a clear strip of tape with 6.5 % hydrogen peroxide onto the tooth (Crest White Strips, Proctor and Gamble), was an innovative advance for home-whitening systems (Gerlach 2000).

The evolution of techniques for tooth whitening is summarized in Table 1.1 and reflects the efforts of the dental profession's efforts to preserve tooth structure and simultaneously enhance the restoration and aesthetics of smiles. The future will likely bring about even more innovations.

1.2 Current Tooth Whitening Techniques

Tooth whitening is now the most common elective dental procedure (Dutra et al. 2004), and has proven to be safe and effective when supervised by the dentist (American Dental Association Council on Scientific Affairs 2009). More than 1 million Americans whiten their teeth annually, resulting in nearly $600 million in revenues for dental offices (Dutra et al. 2004). Considering the numerous over-the-counter whitening products available and the heightened consumer interest in whiter teeth, it is the responsibility of the dental profession to educate the public about the efficacy and adverse effects of different tooth whitening modalities, suggest or provide appropriate options based on patient's needs and preference, and establish reliable and valid monitoring tools for the whitening process.

The variety of methods and products available reflects the high demand for whiter teeth. Traditionally tooth whitening could be classified into three categories: (1) professionally applied in-office whitening with high-concentration materials (Fig. 1.1a); (2) dentist-dispensed patient-applied home-whitening with custom fabricated trays (Fig. 1.1b); and (3) over-the-counter products (Fig. 1.1c) like strips, paint-on gels, or brush-on adhesive liquids (Kwon and Li 2013). With the increased demand and a quest for less expensive options, protocols for do-it-yourself (DIY) whitening (Fig. 1.1d) are now found on the Internet, using natural ingredients such

Table 1.1 History of tooth whitening

Date	Name	Material used	Discoloration
1799	Macintosh (Dwinelle 1850)	Invented chloride of lime (called bleaching powder)	
1848	Dwinelle (1850)	Chloride of lime	Nonvital teeth
1860	Truman (1889)	Chloride and acetic acid, Labarraque's solution (liquid chloride of soda)	Nonvital teeth
1861	Woodnut (1861)	Advised placing the bleaching medicament and changing it at subsequent appointments	
1868	Latimer (1868)	Oxalic acid	Vital teeth
1877	Chapple (1877)	Hydrochloric acid, oxalic acid	All discolorations
1878	Taft (Haywood 1992)	Oxalic acid and calcium hypochlorite	
1884	Harlan (1884)	Used the first hydrogen peroxide (called hydrogen dioxide)	All discolorations
1893	Atkinson (1862)	3–25 % pyrozone used as a mouthwash, which also lightened teeth	
1895	Garretson (Haywood 1992)	Applied chlorine to the tooth surface	Nonvital teeth
1910	Prins (Haywood 1992)	Applied 30 % hydrogen peroxide to teeth	Nonvital and vital
1916	Kaine (Haywood 1992)	18 % hydrochloric acid (muriatic acid) and heat lamp	Fluorosed teeth
1911	Fisher (1911)	Reported on the use of hydrogen peroxide with a heating instrument or a light source	Vital teeth
1924	Prinz (1924)	First recorded use of a solution of perborate in hydrogen peroxide activated by a light source	Vital teeth
1942	Younger (Haywood 1992)	5 parts of 30 % hydrogen peroxide heat lamp, anesthetic	
1958	Pearson (1958)	Used 35 % hydrogen peroxide inside tooth and also suggested 25 % hydrogen peroxide and 75 % ether, which was activated by a lamp producing light and heat to release solvent qualities of ether	Nonvital teeth
1961	Spasser (1961)	Walking bleach technique; sodium perborate and water is sealed into the pulp chamber	Nonvital teeth
1965	Bouschor (1965)	5 parts 30 % hydrogen peroxide, 5 parts 36 % hydrochloric acid, 1 part diethyl ether	Orange colored fluorosis stains
1965	Stewart (1965)	Thermocatalytic technique; pellet saturated with superoxol is inserted into the pulp chamber and heated with a hot instrument	Nonvital teeth
1966	Colon and McInnes (1980)	Repeats Bouschor's technique using controlled hydrochloric acid-pumice abrasion	
1967	Nutting and Poe (1967)	Combination walking bleach technique, Superoxol in pulp chamber (30 % hydrogen peroxide)	Nonvital teeth
1968	Klusmier (Haywood 1991)	Home bleaching concept started as an incidental finding; Gly-oxide which contains 10 % carbamide peroxide is placed in custom-fitted orthodontic positioner	Vital teeth

1970	Cohen and Parkins (1970)	35% hydrogen peroxide and a heating instrument	Tetracycline stains
1972	Klusmier (Haywood 1991)	Used the same technique with Proxigel as it was thicker and stayed in the tray longer	Vital teeth
1975	Chandra and Chawla (1975)	30% hydrogen peroxide 18% hydrochloric acid flour of Paris	Fluorosis stains
1977	Falkenstein (Haywood 1992)	1-min etch with 30% hydrogen peroxide 10% hydrochloric acid 100 W (104 °F) light gun	Tetracycline stains
1979	Compton (Haywood 1992)	30% hydrogen peroxide heat element (130–145 °F)	Tetracycline stains
1979	Harrington and Natkin (1979)	Reported on external resorption associated with bleaching pulpless teeth	Nonvital teeth
1982	Abou-Rass (1982)	Recommended intentional endodontic treatment with internal bleaching	Tetracycline stains
1984	Zaragoza (1984)	70% hydrogen peroxide and heat for both arches	Vital teeth
1986	Munro (Haywood 1992)	Used Gly-oxide to control bacterial growth after periodontal root planning. Noticed tooth lightening	Vital teeth
1987	Feinman (1987)	In-office bleaching using 30% hydrogen peroxide and heat from bleaching light	Vital teeth
1988	Coastal Dental Study Club (Haywood 1992)	Mouth guard bleaching technique	Vital teeth
1988	Munro (Darnell and Moore 1990)	Presented findings to manufacturer, resulting in first commercial bleaching product: White + Brite (Omnii Int)	Vital teeth
1989	Croll (1989)	Microabrasion technique 10% hydrochloric acid and pumice in a paste	Vital teeth, superficial discoloration, hypocalcification
1989	Haywood and Heymann (1989)	Nightguard vital bleaching, 10% carbamide peroxide in a tray	All stains, vital and nonvital teeth
1990		Introduction of commercial, over-the-counter bleaching vital teeth products	Vital teeth
1991		Bleaching materials were investigated while the FDA called for all safety studies and data. After 6 months the ban was lifted	
1991	Numerous authors	Power bleaching, 30% hydrogen peroxide using a light to activate bleach	All stains, vital teeth
1991	Garber and Goldstein (1991)	Combination of bleaching power and home bleaching	Vital teeth
1991	Hall (1991)	Recommended no etching teeth before vital bleaching procedure	Vital teeth
1994	American Dental Association (Engel 2011)	Safety and efficacy established for tooth bleaching agents under the ADA seal of approval	

(continued)

Table 1.1 (continued)

Date	Name	Material used	Discoloration
1996	FDA (http://google2.fda.gov/search?q=ion+laser+technology+for+bleaching+teeth&client=FDAgov&site=FDAgov&lr=&proxystylesheet=FDAgov&requiredfields=-archive%3AYes&output=xml:no_dtd&getfields=*)	FDA approved ion laser technology: argon and CO_2 laser for tooth whitening with patented chemicals	
1996	Reyto (1998)	Laser tooth whitening	Vital teeth
1997	Settembrini et al (1997)	Inside/outside bleaching	Nonvital and vital teeth
1998	Carrillo et al (1998)	Open pulp chamber, 10% carbamide peroxide in custom tray	Vital
2000	Miara (2000)	Compressed bleaching technique in patient's own bleaching tray	Vital teeth
2000	Gerlach (2000)	5–10% hydrogen peroxide OTC tooth whitening strips	Vital teeth
2001	Kurthy (2001)	Deep bleaching technique	Vital teeth
2005	Lynch (2004)	Ozone whitening using ozone machine	Vital teeth
2006	Kwon (2007)	Sealed bleaching: prevents evaporation of active agent by placing a wrap onto the power whitening gel	Vital teeth
2006		Various whitening applications; use of brush applications, pens, and varnish	Vital teeth
2011	ISO 28399 (2011)	International Standard Organization: Dentistry: Products for External Tooth Bleaching	Vital Teeth
Present		Plasma arc, halogen, UV, LED and light-activated bleaching techniques; reduction in time with power gels for in-office bleaching; Laser-activated bleaching; home bleaching available in different concentrations and with new desensitizers	Vital teeth

Adapted and updated from data in Haywood (1992), with permission from Taylor & Francis Group, LLC

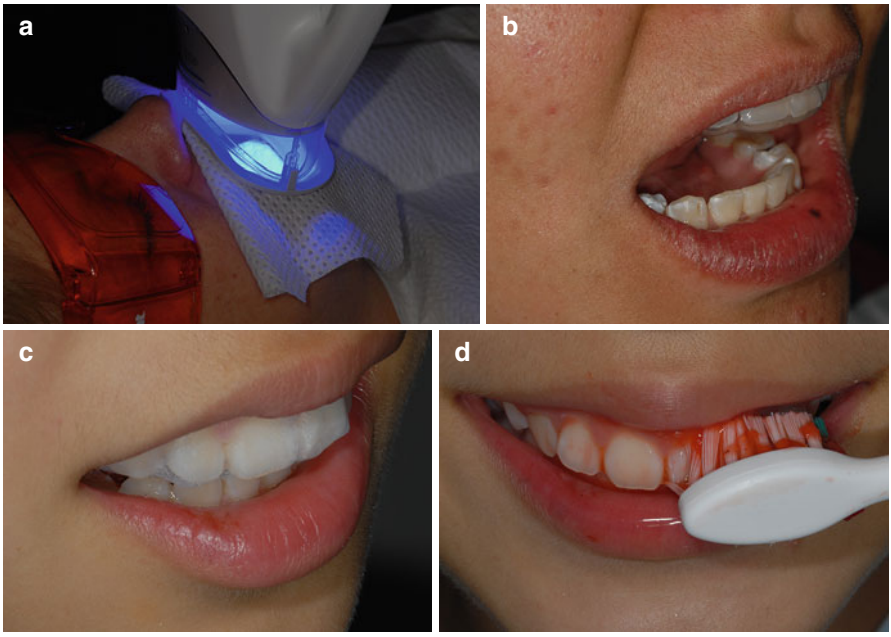

Fig. 1.1 (**a**) In-office whitening procedure with light activation. (**b**) At-home whitening with custom fabricated trays. (**c**) Over-the-counter whitening with strips. (d) Do-it-yourself whitening with strawberry puree

as lemons, apples, and strawberries (Kwon and Li 2013; Natural Teeth Whitening Solutions). The availability of OTC products and various DIY methods has provided the general population better access to whitening, but use without the supervision of a dentist has raised several potential concerns. Tooth discoloration can be the secondary effect of an undiagnosed illness, overuse of whitening materials can damage the enamel surface, and the at-home process might go unmonitored (Hammel 1998; Kwon and Li 2013; Natural Teeth Whitening Solutions). Therefore, the supervision of a dentist or use of custom fabricated trays should be the treatment modality of choice. The patient's final decision, however, will most likely depend on preference. Although at-home whitening with 10 % carbamide peroxide is safe and effective under a dentist's supervision (American Dental Association Council on Scientific Affairs 2009), in-office whitening has its merits, especially in elderly patients who may prefer the convenience and in young children who may require full supervision during the entire procedure. Also, patients who cannot tolerate wearing trays and those who desire an immediate effect might also prefer an in-office treatment.

Several studies have compared the efficacy, side effects, and patient acceptance of in-office, at-home, or over-the-counter whitening. Patient opinion was found to depend on the whitening product, study design, application time, and methods of color assessment. One study evaluated the time required to achieve a six-tab difference on a Vita Classical shade guide, and found this occurred the fastest with

Table 1.2 Summary of current vital tooth whitening techniques

	In-office whitening	At-home whitening	OTC	DIY
Supervision	Yes	Yes	No	No
Active ingredient	HP	HP/CP	HP/misc	Natural ingredients
Concentration	~up to 40 %	~7–35 % CP	~up to 12 % HP	N/A
Activators	Chemical, LED, Laser	Chemical	Chemical, light	N/A
Efficacy	Good	Good	Mod-good	Questionable
Safety	Good	Good	Mod-good	Questionable
Costs	High	Mod	Low	Lowest

Mod Moderate, *HP* Hydrogen peroxide, *CP* Carbamide peroxide

in-office whitening, followed by at-home whitening, with over-the-counter whitening requiring the most time (Auschill et al. 2005). The various techniques caused similar levels of gingival or tooth sensitivity, and patients tended to prefer at-home whitening, as previously reported (Bizhang et al. 2009; Da Costa et al. 2010; Giachetti et al. 2010; Serraglio et al. 2016). In vitro studies comparing all four whitening techniques showed in-office, at-home, and over-the-counter whitening produced good results, whereas do-it-yourself whitening with strawberry puree was ineffective (Kwon et al. 2015a, b). Despite the equivalencies in endpoint whiteness, a concern remains that DIY whitening could reduce tooth microhardness values (Kwon et al. 2015a). A complete summary, including a comparison of the characteristics of current vital tooth whitening technologies, is listed in Table 1.2. It must be noted that this presents an overall comparison and may vary based on the specific material employed.

1.3 Diagnosis and Treatment Planning

If a patient desires whiter teeth or would benefit from tooth whitening in conjunction with restorative or orthodontic treatment, their prognosis depends on the nature of the discoloration and the expectations of the patient. Discoloration due to extrinsic origins respond better to whitening but even discoloration due to intrinsic origin (e.g., tetracycline staining) can respond to whitening if the treatment time is sufficient (Haywood 1991). The absolute contraindications to tooth whitening are few, but unrealistic expectations, an unwillingness to comply with treatment, pregnancy, allergy to components in the whitening material, and severe sensitivity should be carefully considered before starting treatment.

1.3.1 Check List During Examination

Like any dental examination, the proper steps for diagnosis include obtaining medical and dental history and radiographs and conducting a thorough clinical examination.

Active dental caries that may be close to the pulp should be given special attention. Carious lesions can be temporarily treated prior to the whitening treatment and finalized once the color is stabilized.

A single dark tooth is a red flag and might be associated with a previous traumatic injury or even a periapical pathosis (Kwon 2011). Radiographs and pulp vitality testing can guide the treatment (Chap. 6).

Crack lines are not an absolute contraindication but the patient should be aware they may exacerbate sensitivity or become even more visible after tooth whitening (Kwon et al. 2009).

Localized decalcification areas and white spots should be carefully examined as they might blend in with the lighter tooth color or could become more noticeable (AlShehri and Kwon 2016). In these instances, other conjunctive treatments such as microabrasion or resin infiltration and restorative treatment may be indicated (Chaps. 6, 9, 10, 12, 13, and 15).

Translucent areas often observed on incisal edges will remain translucent upon whitening treatment and may end up looking grayish, continuing to be a concern for some patients. In severe cases, a resin composite restoration to mask the translucency may be needed.

Existing tooth-colored restorations in the aesthetic zone should be carefully examined since there may be a need for retreatment that should be explained in advance, to allow the patient to make the necessary financial commitment.

The symmetry in gingival contour should be observed and possibly resolved prior to whitening, in order to enhance the aesthetic outcome.

Severe abrasion, attrition, and recessions should also be observed and explained to the patient, as root exposures will not respond to whitening (Hilton et al. 2013; Pashley 1989).

Preexisting tooth sensitivity needs to be addressed prior to the treatment, since it may become severe upon treatment compromising the outcome of the treatment.

1.3.2 New Challenges in Tooth Whitening

1.3.2.1 Failed Attempts of Tooth Whitening

With the increased interest in tooth whitening, patients currently consult a dental professional about this technique after several failed attempts of trying it on their own (Fig. 1.2). Many have used over-the-counter products in various forms with unsatisfactory results, yet exhibit teeth that are already quite light, making the treatment more challenging. Therefore, it is prudent to establish the expectation of the patient and discuss the feasibility of reaching this goal. A very realistic and natural outcome is to reference the white of the eye (Mrazek 2004). However, patients often want teeth that are even whiter, at which point the dentist should carefully discuss the patient's treatment goal, in detail.

1.3.2.2 Erosion

As lifestyles have changed throughout the decades, the consumption of soft drinks has increased in the United States by 300 % in 20 years (Calvadini et al. 2000; Lussi

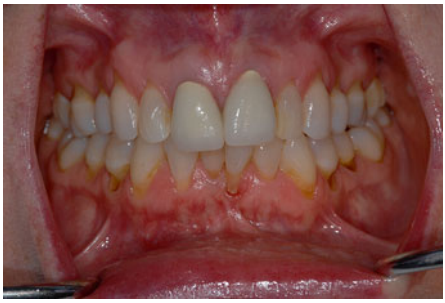

Fig. 1.2 Patient complained about previous failed attempts of tooth whitening. A thorough examination of existing restorations, recessions, abfractions, and gingival asymmetry was followed by a comprehensive treatment plan to satisfy patient's desire for an aesthetic outcome

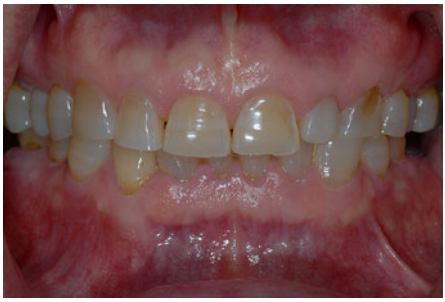

Fig. 1.3 Generalized erosion of teeth can contribute to a more chromatic appearance of teeth

et al. 2006). At the same time, the incidence of dental erosion is growing steadily (Lussi et al. 2006). Initially, erosion is limited to the enamel, but in advanced cases dentin becomes exposed and causes functional and aesthetic concerns that require treatment. Generally, the tooth becomes more chromatic with the loss of enamel, and one of the first distinct visual changes patients complain about is tooth color (Fig. 1.3). The treatment plan may vary depending on the severity and location of dental erosion. Restorative options, including direct resin composite and indirect porcelain restorations, are suggested for the rehabilitation of a severe loss of tooth structure. While dental erosion is considered to be a contraindication to tooth whitening (Lussi et al. 2006), it may be beneficial in the early stages if the patient desires a whiter smile. Indeed, since the prevalence of dental erosion is steadily increasing, the topic merits continued research.

1.3.2.3 Tooth Whitening in Children

Another emerging topic is the age deemed appropriate for tooth whitening (Fig. 1.4a, b). The American Academy on Pediatric Dentistry Council on Clinical Affairs recognized the increased desire for whiter teeth in pediatric and adolescent patients and advised the judicious use of whitening for vital and nonvital teeth, as well as consultation with the dentist to determine the appropriate method and timing for treatment (American Academy on Pediatric Dentistry Council on Clinical Affairs 2015). A single clinical study is currently registered to evaluate the efficacy and tooth sensitivity in an adolescent population (patients ranging from 12 to 20 years) (Pinto et al. 2014).

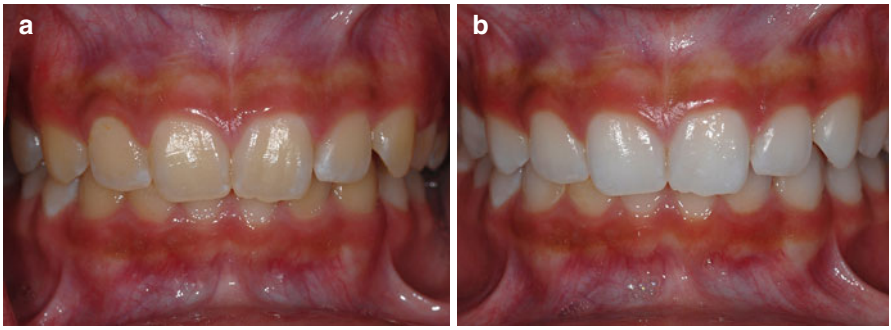

Fig. 1.4 (**a**) The best time for initiating whitening in children should be carefully discussed with the parents. This 12-year-old child complained about his dark teeth as well as the localized white areas on the upper anterior teeth. (**b**) Treatment options include at-home whitening with custom fabricated trays when the child is compliant or in-office whitening where the whole procedure is performed in the clinic

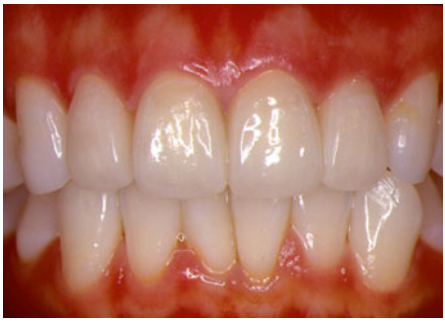

Fig. 1.5 Patient complained about her upper four anterior teeth which had been restored with porcelain laminate veneers. Over time, she noticed a slight darkening of her restored teeth. In this case, whitening from the lingual may reduce the chromacity of her restored teeth

1.3.2.4 Tooth Whitening on Teeth with Veneers and Orthodontic Braces

Lastly, with the increased interest in cosmetic dentistry more patients have existing anterior composite resin or porcelain veneers. Over time, teeth become more chromatic, which can shine through existing veneer restorations (Fig. 1.5). To brighten teeth, yet preserve the existing restoration, 10 % carbamide peroxide on the lingual surface can be applied with custom trays (Barghi and Morgan 1997; Haywood and Parker 1999). However, the efficacy of whitening through the lingual surface is mainly based on a few clinical cases and evidence is limited. With the increased awareness for a brighter smile, we face new situations. For example, increasingly patients are requesting tooth whitening while orthodontic braces are in place. A few studies showed tooth whitening with custom fabricated trays over brackets could whiten teeth evenly (Jadad et al. 2011). Nevertheless, more research is needed to address these special and challenging situations to help clinicians in the decision-making process.

1.3.3 Monitoring the Progress of Tooth Whitening

The success of tooth whitening is mainly determined by changes in tooth color and is subjective to each patient; however, evaluating tooth color is extremely difficult because of the complex optical characteristics of the tooth, which include gloss, opacity, transparency, translucency, and optical phenomena such as metamerism, opalescence, and fluorescence (Hunter 1987). Patients commonly inquire about the expected final shade after tooth whitening. So first recording the baseline tooth color will help determine the prognosis, and is invaluable in monitoring progress. The prognosis of whitening is significantly enhanced with shades in the yellow-orange range, whereas gray and bluish discolorations are more stubborn (Leonard 2003). Additionally, rather than promising a specific shade, it is prudent to suggest a reliable reference point, such as the white of an eye, so the patient can perceive the difference (Mrazek 2004). Commonly the white of the eye is whiter than the baseline tooth color, providing a good reference point for the progress being made during treatment. One of the best ways to demonstrate the efficacy and progress of whitening is to compare the color difference of the upper, treated arch versus the lower, untreated arch. This difference is very helpful in encouraging compliance and also for some who cannot discern color changes well. Many times it is also important to have color change validated by friends or family, and photographs can be an essential monitoring tool (Kwon and Li 2013).

The Vitapan Classical (VITA Zahnfabrik, Bad Sackingen, Germany) shade guide, with values oriented according from the lightest to the darkest tab, is commonly used for visual shade matching. Nevertheless, the lack of logical order, uniform color distribution, and light shade tabs has been pointed out as drawbacks of the Vitapan Classical (Ontiveros and Paravina 2009). To facilitate the monitoring of tooth whitening, a shade guide was developed, the VITA Bleachedguide 3D Master (VITA Zahnfabrik, Bad Sackingen, Germany), composed of 15 tabs that exhibit a wider color range and more consistent color distribution, compared to the Vitapan Classical. The VITA Bleachedguide 3D Master was also evaluated to be the easiest to arrange, the most harmoniously arranged and most preferred for the monitoring of tooth whitening (Paravina 2008). The initial color tab, selected during baseline color measurements, can be easily placed along whitened teeth, leading to the anticipation of the whitening progress. The effect of tooth whitening can be easily monitored by selecting the closest shade tab before and after whitening and counting the difference in tab numbers, expressed as a difference in shade guide units (ΔSGU) (Kwon et al. 2015b).

Methods using specialized instruments to determine tooth shade have become available with advancements in technology. These methods have the advantage of being uninfluenced by the human eye, environment, and light source, and generate reproducible results (Chu 2003). Additionally, methods using instruments provide objective shade data and allow different image-analysis options, such as basic shade analysis, smile analysis, and synchronization, to produce a split image of pre- vs. postwhitening. These images (Fig. 1.6a, b) can be printed immediately and are

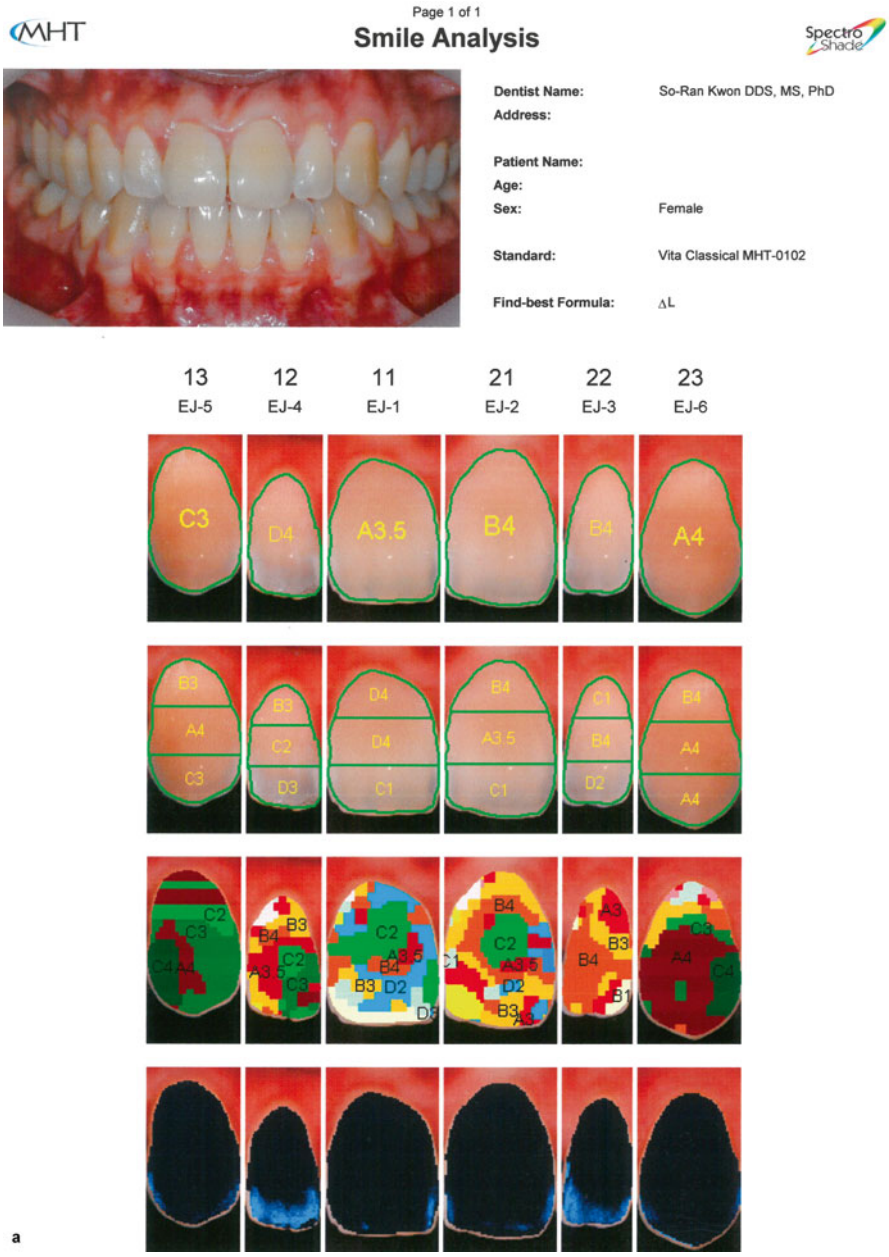

Fig. 1.6 (**a**) Smile analysis before whitening. (**b**) Smile analysis after whitening

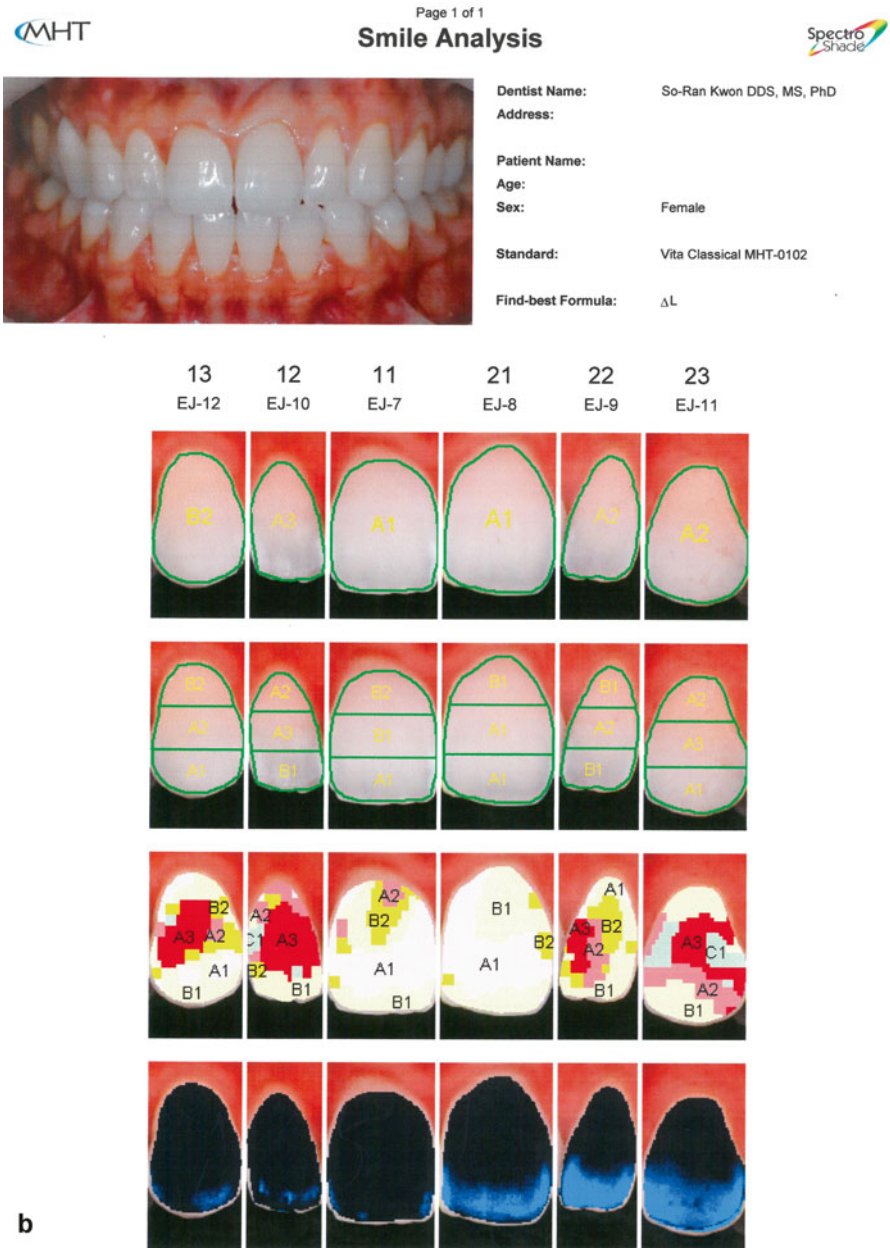

Fig. 1.6 continued

effective tools to show objective results on the progress of tooth whitening. They also provide motivation to initiate and continue treatment. This technique is currently used more often in research, because it is as yet expensive and time consuming for use in clinical dentistry.

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Tooth Whitening: How Does It Work

2

So Ran Kwon

Abstract

Tooth discoloration is classified as extrinsic or intrinsic, with extrinsic stains arising from accumulation of residue on the surface of the tooth, and intrinsic discoloration from stains within the enamel or dentin. For both types of stains, tooth whitening with hydrogen peroxide is a common treatment. Hydrogen peroxide likely exerts its effects by interacting with chromophores within the tooth structure, acting via what is known as the "chromophore effect." Despite having the desired cosmetic effect, however, hydrogen peroxide treatment also may likely affect sound tooth tissue; and the unknowns surrounding unwanted side effects remain a concern. Here, the etiology of extrinsic and intrinsic stains is summarized, as is our current understanding of hydrogen peroxide treatment and mechanisms of action. This information might guide further research and development efforts to create new technology for the treatment of tooth discoloration.

2.1 Etiology of Discolorations

The human tooth is composed of three dental hard tissues: enamel, dentin, and the cementum, which are each distinct in their mineral composition and function. Of the three tissues, dental enamel is the most mineralized and is the hardest tissue of the body. Enamel is ~96 % mineral, 3 % water, and 1 % organic matter by weight, whereas dentin is 70 % mineral, 20 % organic matrix, and 10 % water by weight (Nanci 2013). Unlike bone tissue, which remodels itself continuously through

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balanced bone resorption and formation, the dental hard tissue does not turn over. Nonetheless, the enamel interface undergoes continuous, dynamic ion exchange with the oral biofilm, with calcium phosphate apatite crystals moving in both directions to maintain proper mineral balance (Peters 2010). Indeed, enamel and dentin form a semipermeable membrane that allows small molecules to pass into the tooth structure. This property largely accounts for tooth discoloration from extrinsic sources. The key properties that cause enamel and dentin to bind to and retain staining molecules, however, are still not well understood.

2.1.1 Extrinsic Stains

Tooth discoloration is broadly classified as extrinsic or intrinsic, depending on the origin of the stain (Watts and Addy 2001). Extrinsic stains arise due to the accumulation of residue on the enamel surface and can be accentuated by pitting or irregularities of the enamel, salivary composition, salivary flow rates, and poor oral hygiene (Hattab et al. 1999). The affinity of material to the tooth surface plays a critical role in the deposition of extrinsic stains (Plotino et al. 2008). Stains might adhere to the tooth via several types of attractive forces including long-range interactions such as electrostatic and van der Waals forces, and short-range interactions such as hydration forces, hydrophobic interactions, dipole-dipole forces, and hydrogen bonds (Nathoo 1997). Extrinsic stains take on a variety of colors, which reflect the nature of the stain.

Brown stain is a thin and bacteria-free pellicle commonly found on the buccal surface of the maxillary molars and on the lingual surface of the mandibular incisors (Leung 1950). It is commonly associated with poor oral hygiene and likely results from the deposition of tannins found in tea, coffee, and other beverages (Hattab et al. 1999). Tobacco stains present as a tenacious dark-brown discoloration primarily on the cervical one-third to one-half of teeth. The severity of discoloration is influenced by characteristics of the tooth surface rather than the amount of tobacco consumed (Manly 1973).

Black stain is a continuous narrow black line along the gingival margin of the enamel and encircles the tooth (Faunce 1983). It is usually associated with a mucinous plaque that is infiltrated with chromogenic bacteria, primarily *Actinomyces* (Slots 1974). The black stain is ferric sulfide and is formed by the reaction between hydrogen sulfide, a metabolic by-product of bacteria, and iron from the saliva and gingival exudate (Reid et al. 1977). It is more common in females and may occur in individuals with excellent oral hygiene (Goldstein and Garber 1995).

Green stains on the labial surface at the gingival third of the maxillary anterior teeth are attributed to the infiltration and growth of fluorescent bacteria and fungi, such as *Penicillum* and *Aspergillus* (Bartels 1939). It is common in children, affecting boys more frequently than girls (Leung 1950).

Orange stain appears as a yellow, orange, or a reddish-orange line in the cervical third of the incisors (Faunce 1983). The stain is associated with chromogenic bacteria, such as *Serratia marcescens* and *Flavobacterium lutescens* (Carranza and Newman 1996).

Metallic stains are common in industrial workers exposed to metal-containing dust or in individuals who have received certain orally administered drugs or locally applied therapeutic agents (Hattab et al. 1999). The metal that combines with the acquired pellicle at the tooth surface varies the stain color: dust composed of mercury and lead are gray, copper and nickel stains are green to blue-green, and chromic acid fumes produce a deep orange color in the enamel. The use of iodine solution and stannous fluoride produces a brown discoloration, while silver nitrate results in a black discoloration (Hattab et al. 1999). Antiseptic stain has been observed after prolonged use of chlorhexidine mouth rinses and is characterized by a brown, diffuse discoloration of the teeth (Linden et al. 1986). Despite the fact that extrinsic discolorations may vary in color, severity, and location, all stains can be easily removed by dental prophylaxis.

2.1.2 Intrinsic Stains

In contrast to the superficial nature of extrinsic stains, intrinsic discolorations are incorporated during tooth formation or after eruption, and are attributable to the presence of stain molecules within the enamel and dentin (Dahl and Pallesen 2003). Preeruptive stains arise due to dental fluorosis, tetracycline staining, hematologic disorders, and inherited developmental defects of enamel or dentin without systemic features (Hattab et al. 1999).

2.1.2.1 Dental Fluorosis

Because it is widely available from multiple sources, dental fluorosis is the most common cause of intrinsic discoloration and manifests as a subsurface hypomineralization of tooth enamel caused by chronic ingestion of fluoride during odontogenesis (Burt 1992). The nature and severity of dental fluorosis depends on the dosage, duration of exposure, stage of ameloblast activity, and susceptibility of the individual (Driscoll et al. 1983). Initially, tooth discoloration may not be evident upon eruption, but becomes more evident as the exposed porous surface gradually absorbs chromogenic substances in the oral cavity (Steinberg et al. 1999). Clinically, signs of mild fluorosis range from delicate accentuation of the perikymata pattern to white opaque spots or lines. In severe cases, brown pitting patches or localized loss of external enamel may occur (Hattab et al. 1999).

2.1.2.2 Tetracycline Staining

Tetracycline staining was first reported in the late 1950s after its introduction and widespread use as a broad-spectrum bacteriostatic antibiotic (Shwachman et al. 1958–1959). Tetracycline exposure between the second trimester in utero to approximately 8 years of age can affect the teeth, skeleton, and fingernails (Bevelander 1964). The tetracycline molecule chelates with calcium in hydroxyapatite crystals, predominantly in dentin, forming a tetracycline-calcium orthophosphate complex (Mello 1967). The color of tetracycline-stained teeth becomes more intense upon chronic exposure to sun and artificial light because of the photooxidation of this complex (McEvoy 1989). The severity of stains depends on the type of tetracycline

consumed and the time, duration, and amount of drug intake (Dayan et al. 1983). Proper diagnosis is imperative as tetracycline staining is considered one of the most difficult stains to remove. Diagnosis is established by acquiring patient history, and assessing clinical appearance and fluorescence under ultraviolet light (Hattab et al. 1999).

2.1.2.3 Developmental Defects and Others

Developmental defects of enamel or dentin are associated with amelogenesis imperfecta, dentinogenesis imperfecta, and enamel hypoplasia. Discolorations due to developmental defects often become worse over time as the rough surfaces allow stains to accumulate more easily. Numerous other factors can also adversely affect the ameloblast and cause enamel hypoplasia, including nutritional deficiencies, viral exanthematous diseases, trauma to developing teeth, birth trauma, metabolic diseases, hemolytic diseases in newborns, local infection, ingestion of chemicals, and other genetic factors (Hattab et al. 1999).

Intrinsic stains can also be acquired after eruption resulting in local discoloration. The severity of the discoloration varies depending on the cause, and may appear mild yellow-orange to very dark brown or black. Dental caries are a major cause of local discoloration and often start as an opaque, white halo, ultimately developing into an unsightly brown or black area due to the reaction of bacterial by-products with decalcified dentin (Eriksen and Nordbo 1978). Restorative materials that leak around the margins may allow debris or chemicals to enter, resulting in discoloration of the underlying dentin (Steinberg et al. 1999). Amalgam pigmentation is very common and can result in greenish-black pigmentation caused by the products of tin oxidation (Goldstein and Garber 1995). Endodontic materials and sealers also have various staining potentials that, over time, cause intrinsic discoloration of a tooth with a filled root canal (van der Burgt and Plasschaert 1985).

The success of tooth whitening depends upon proper diagnosis of the initial cause of discoloration. Although stains are categorized as extrinsic or intrinsic, in most cases the etiology is multifactorial. Further, extrinsic stains often become internalized over time as the enamel and dentin are permeable to organic and inorganic substances. Therefore, careful examination and history taking is required to properly determine all components that contributed to the discoloration.

2.2 Mechanism of Peroxide Action

The importance of appearance in our society has increased the demand for instant tooth whitening, resulting in a prolific and diverse array of products and techniques. To keep pace with this trend, dental professionals have invested a great amount of effort to elucidate the mechanisms through which peroxides promote tooth whitening. The “chromophore theory” is based on the interaction of hydrogen peroxide (H_2O_2) with organic chromophores within the tooth structure and has traditionally been accepted as the mechanism by which peroxide exerts its whitening effects. Organic chromophores have electron-rich areas, and when reactive

oxygen species such as hydrogen peroxide encounter stain molecules, they convert the chromophore chains into simpler structures or alter their optical properties and diminish the appearance of the stain (Albers 1991). Although the chromophore theory is widely adopted, it is not fully understood and many fundamental questions remain: How do organic chromophores diffuse into the tooth? How do they interact with tooth structure? Where do they accumulate? What is the composition and fate of the breakdown products of any oxidation process? These questions merit further investigation.

To facilitate understanding of tooth whitening, the process must be evaluated in three distinct phases: (1) the movement of the whitening agent applied from the outer surface into the enamel and dentin; (2) the interaction of stain molecules with hydrogen peroxide upon its penetration into the tooth structure; and (3) the micromorphologic changes induced by peroxide-based materials on tooth surface and structure that may lead to optical changes. The outcome of these three phases results in the final color change of the tooth after whitening. Ideally, whitening agents will maximize lightening while minimizing concurrent damage to the tooth structure (Kwon and Wertz 2016). Investigating the effects of peroxide during each phase will increase our understanding of the whitening process and optimize whitening approaches.

2.2.1 Phase One: Diffusion

Extrinsic stains that are limited to the external surface of the tooth can be readily removed with tooth brushing or dental prophylaxis. However, once the stain becomes internalized within the enamel and dentin, hydrogen peroxide must penetrate these layers in order to interact with organic chromophores. Although peroxide-based tooth whitening was introduced in the 1800s, the quantification of hydrogen peroxide penetration into the pulp cavity was not quantified until 1987 by Bowles and Ugwuneri (Bowles and Ugwuneri 1987). This in vitro study used leucocrystal violet and horseradish peroxidase to spectrophotometrically measure hydrogen peroxide at submicrogram levels and is a well-established, accurate, selective, and sensitive analytical method that is still used today (Mottola et al. 1970).

Studies evaluating the diffusion phase found that peroxide penetration was enhanced by the following: higher hydrogen peroxide concentrations (Bowles and Ugwuneri 1987; Gökay et al. 2004; Hanks et al. 1993; Palo et al. 2010); prolonged application (Hanks et al. 1993; Kwon et al. 2012b; Rotstein et al. 1991); increased temperature (Bowles and Ugwuneri 1987; Rotstein et al. 1991); the large size of dentinal tubules in young teeth (Camps et al. 2007); variations in tooth structure due to location, acid etching or restorations (Benetti et al. 2004; Camargo et al. 2007; Camps et al. 2010; Palo et al. 2012; Patri et al. 2013); and light activation (Camargo et al. 2009). Penetration was also improved by specific formulations and delivery systems (Bharti and Wadhwani 2013; Cooper et al. 1992; Gökay et al. 2005; Park et al. 2016; Pignoly et al. 2012; Thitinanthapan et al. 1999). The results of all reviewed studies are in accordance with Fick's second law of diffusion, which states

that the diffusion of a molecule is proportional to the surface area, diffusion coefficient, and concentration, and that it is inversely proportional to the diffusion distance (Brotherton Boron 1994).

Additionally, studies were performed to determine the path of diffusion into the tooth by whitening agents. Since peroxide-based materials are water soluble, it was speculated that the diffusion of these molecules was similar to the flow of fluids that occur in the enamel interprismatic spaces and dentinal tubules (Ake-Linden 1968; Kwon et al. 2012a; Pashley 1996). In a study utilizing confocal laser scanning microscopy, the diffusion pathway of hydrogen peroxide was correlated to Rhodamine B. This demonstrated diffusion of the dye into interprismatic spaces and accumulation along the dentin-enamel junction, followed by uptake into the terminal branch of the dentinal tubules where it could directly access the predentin and pulp cavity (Kwon et al. 2012a). However, this is not simply passive diffusion of these molecules, but requires a concentration gradient that is determined by the chemical affinity for each dental tissue (Ubaldini et al. 2013). Thus, as chemical composition can affect the outcome of treatment, it is important to identify the optimal whitening concentration and application times so that concurrent tooth structure damage may be minimized without compromising whitening efficacy.

2.2.2 Phase Two: Interaction

Although a wide variety of whitening products are available, most contain hydrogen peroxide as the active agent (Dahl and Pallesen 2003). Hydrogen peroxide may be applied directly, or produced in a chemical reaction from sodium perborate or carbamide peroxide (Budavari et al. 1989). Hydrogen peroxide (H_2O_2) is slightly more viscous than water with a molar mass of 34.0147 g/mol and acts as a strong oxidizing agent (Hess 1995). The rate of decomposition and the type of active oxygen formed depends on the temperature and concentration of peroxide, as well as the pH and presence of cocatalysts and metallic reaction partners (Goldstein and Garber 1995).

Homolytic cleavage, the splitting of shared, bonding electrons resulting in an unshared electron:

$$\mathrm{HOOH} \rightarrow \mathrm{H}\cdot + \cdot\mathrm{OOH}\ \text{or}\ \mathrm{HO}\cdot + \cdot\mathrm{OH}$$

This type of cleavage is favored by light and heat and forms free radicals.

Heterolytic cleavage, which is a deprotonation reaction leaving an electron pair:

$$\mathrm{HOOH} \rightarrow \mathrm{H}^{+} + \cdot\mathrm{OOH}^{-}$$

This deprotonation occurs at increased pH and generates perhydroxyl anions (Feinman et al. 1991).

A third pathway is derived by a *combination of homolytic and heterolytic cleavage* and generates active oxygen that is both an anion and a free radical:

$$\mathrm{HOO}\cdot + \mathrm{OH}^{-} \rightarrow \mathrm{O}\cdot^{-}_{2} + \mathrm{H_2O}\ (\text{basic condition})\ \text{and}\ \mathrm{HOO}\cdot \rightarrow \mathrm{O}\cdot^{-}_{2} + \mathrm{H}^{-}\ (\text{acidic condition})$$

Active oxygen is attracted to electron-rich areas of stain molecules and cleaves double bond to reduce color or remove the compound (Albers 1991).

Despite the well-known chemistry of hydrogen peroxide and its application to establish the chromophore theory, many issues remain unsolved. Studies using Fourier Transform Infra-Red (FTIR) and Raman spectroscopies failed to detect chromophores or their breakdown products in the enamel, and are inconsistent with the chromophore theory (Darchuk et al. 2008; Eimar et al. 2012; Fattibene et al. 2005). Thus, continued investigation is required to fully understand the mechanisms of hydrogen peroxide in eliminating stains.

Ideally, as hydrogen peroxide moves from the external tooth surface into the enamel and dentin, its oxidizing action should be limited to organic chromophores until it reaches a certain saturation point, or whitening threshold. Oxidizing action beyond the whitening threshold—characterized by the depletion of chromophores—has been cautioned against as it might compromise tooth structure. Indeed, review of the literature suggests hydrogen peroxide has significant interactions with the organic and inorganic components of enamel and dentin well before the saturation point. This may account for alterations in the physical properties of the tooth substrate after the whitening treatment (Attin et al. 2005).

Extensive studies using ion-selective electrode probes, FT-Raman spectroscopy, and a combination of scanning electron microscopy (SEM) and energy-dispersive X-ray spectrometer and microcomputerized tomography suggest that hydrogen peroxide interacts with the tooth structure, and changes the chemical composition of enamel and dentin (Kwon and Wertz 2016). While evidence exists that peroxide-based materials do not irreversibly influence the chemistry of enamel and dentin beyond clinical relevance (Arcari et al. 2005; Cavalli et al. 2011; Goo et al. 2004; Lee et al. 2006; Mc Cracken and Haywood 1996; Rodrigues et al. 2007), several studies have demonstrated significant changes in the calcium/phosphate ratio, indicative of alterations in the inorganic components of hydroxyapatite (Al-Saleni et al. 2007; Berger et al. 2010; Bizhang et al. 2006; de Freitas et al. 2004; Efeoglu et al. 2005; Efeoglu et al. 2007; Rotstein et al. 1996; Rotstein et al. 1992). Microcomputerized tomography studies of enamel treated with 10 % or 35 % carbamide peroxide demonstrated a demineralization depth of 50 μm and 250 μm, respectively (Efeoglu et al. 2005; Efeoglu et al. 2007). Furthermore, infrared spectroscopic analysis showed changes in the enamel that was both concentration and time dependent.

It is worth noting that changes in the organic component of enamel and dentin are likely due to the oxidizing ability of hydrogen peroxide, while changes in the mineral component are mainly attributed to its acidity (Jiang et al. 2007). Several studies provide evidence supporting that the organic matrix of enamel and dentin is oxidized by hydrogen peroxide. X-ray diffraction analysis of hydroxyapatite suggests hydrogen peroxide influences the organic tissue, and Nuclear Magnetic Resonance-based measurements indicate that proline and alanine may be more susceptible to an attack by the hydroxyl radical (Kawamoto and Tsujimoto 2004; Sato et al. 2013; Toledano et al. 2011). Other studies assessing morphological changes in the enamel and dentin used atomic force microscopy (AFM) and FTIR to show that the tooth enamel matrix protein or organic matrix of dentin had partially lysed,

causing these effects (Abouassi et al. 2011; Chng et al. 2005; Hegedüs et al. 1999; Mahringer et al. 2009; Sato et al. 2013; Ubaldini et al. 2013). Moreover, other studies have implicated that proteolysis by dentin metalloproteinases and cathepsin B might also compromise the organic component of dentin (Sato et al. 2013; Toledano et al. 2011).

Collectively, these studies demonstrate that hydrogen peroxide indeed interacts with all components of dentin and enamel. Thus, it may not only target chromophore stains, but also whiten by modifying the organic substances within the tooth. Future studies must identify the clinical significance of interactions between hydrogen peroxide and each tooth layer.

2.2.3 Phase Three: Surface Change and Color

The anticipated final outcome of tooth whitening is to increase color lightness and reduce chroma in the yellow-blue and red-green spectrum based on the CIE Lab system (Commission Internationale de l'Eclairage 1995). The separate contributions of enamel and dentin on tooth color have been evaluated, with some studies placing more emphasis on the role of dentin (Kwon et al. 2013; Wiegand et al. 2005; Kugel et al. 2007). Nevertheless, enamel characteristics also play a key role in the optical properties of the tooth. Enamel contributes to the overall tooth color by decreasing the translucency of the tooth, masking the color of the underlying dentin (Kawamoto and Tsujimoto 2004; Ma et al. 2009, 2011). Changes in the enamel have been attributed to micromorphological alterations through deproteinization, demineralization, and oxidation of the most superficial enamel layer (Eimar et al. 2011; Ma et al. 2009, 2011). This changes the density of enamel making the distribution of enamel crystals less compact and potentially increasing its refractive index (Li et al. 2010; Ma et al. 2011).

Determining how subtle enamel surface changes affect the tooth has been an area of interest. Studies have found that rough surfaces create a more diffuse reflection, turning the object brighter, whereas a smooth surface leads to more specular reflection. Additionally, an increase in back scattering of short wavelengths, reflected as bluish-white, plays a considerable role in the light scattering of teeth (Joiner 2004). This is most easily demonstrated by the whitish color change in early caries lesions due to the increased opacity of the tooth enamel (Ma et al. 2009, 2011; Vieira et al. 2008). Further, some studies suggest tooth color change that is associated with tooth whitening is mainly due to mineral loss rather than the breakdown of chromophores (Jiang et al. 2007; Kwon et al. 2002; Lee et al. 2006; Mc Cracken and Haywood 1996). The subsequent uptake of minerals after tooth whitening and the reversal of the treatment substantially support this suggestion (Li et al. 2010).

Because of the impact of surface changes on the appearance of tooth color, changes in surface topography have been extensively investigated. SEM and AFM studies showed increased roughness and surface irregularities upon whitening treatment (Ben-Amar et al. 1995; Bitter and Sanders 1993; Hosoya et al. 2003; McGuckin et al. 1992; Pedreira De Freitas et al. 2010; Pinto et al. 2004; Shannon et al. 1993;

Yeh et al. 2005; Zalkind et al. 1996). Notably, most of these changes have not been seen in studies where a remineralizing agent or saliva was used as a storage medium (Duschner et al. 2006; Haywood et al. 1991; Joiner et al. 2004; Scherer et al. 1991; Turkun et al. 2002; White et al. 2003). Thus, continued research on the effects of whitening treatments on surface and tooth color changes are necessary to order to prescribe treatments that will have long-lasting effects with minimal changes to the overall structure of the tooth.

This up-to-date review of the literature illustrates that tooth whitening occurs in three distinct phases, challenging the validity of the widely accepted "chromophore effect" as the dominant mechanism of hydrogen peroxide. As such, this theory must be modified to reflect the true complexity of the mechanisms that drive whitening. Indeed, stains are not determined by the properties of the organic staining molecules alone but are also affected by micromorphologic alterations on the tooth surface and within the tooth structure; thus, whitening likely affects intact enamel and dentin microstructures, an underrecognized concern (Kwon and Wertz 2016). In future studies, an appreciation of the complexity of the tooth whitening process will spearhead innovation toward materials and techniques that meet the ever-growing interest in safely obtaining a brighter smile.

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Overall Safety of Peroxides

3

Yiming Li

Abstract

Current tooth whiteners contain peroxides as active ingredients, which release hydrogen peroxide (H_2O_2) in the process of application. The primary source of safety concerns with the peroxide-based tooth whiteners is the capability of H_2O_2 to produce oxidative free radicals or reactive oxygen species (ROS), which have been associated with various pathological consequences including carcinogenesis and degenerative diseases. This chapter will review and discuss toxicology of H_2O_2, its presence in the human body, and its potential systemic effects, genotoxicity, and carcinogenicity on the basis of evidence available in the literature.

3.1 Background

Safety concerns with peroxide-based tooth whiteners are primarily originated from their content of peroxide compounds (Li 1996, 1997, 2011; Li and Greewall 2013). Carbamide peroxide ($CH_6N_2O_3$) and hydrogen peroxide (H_2O_2) are the most commonly used peroxide compounds as the active ingredient in current extracoronal tooth-whitening products, while sodium perborate ($NaBO_3$) is primary for intracoronal bleaching procedures (Rotstein and Li 2008). Carbamide peroxide, or urea hydrogen peroxide, is a white crystal or a crystallized powder. Chemically, carbamide peroxide is composed of approximately 3.5 parts of H_2O_2 and 6.5 parts of urea; a tooth whitener of 10% carbamide peroxide thus contains approximately

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3.5 % H_2O_2. Sodium perborate is also a white powder available either as monohydrate, trihydrate, or tetrahydrate. The monohydrate and tetrahydrate forms are commonly used for intracoronal bleaching, with H_2O_2 content theoretically around 34 % and 22 %, respectively. In an aqueous medium, both carbamide peroxide and sodium perborate decompose to release H_2O_2, which, therefore, is the true active ingredient of the peroxide-based tooth-whitening products.

3.2 Toxicology of Hydrogen Peroxide

H_2O_2 as a chemical was first identified in 1818, and the well-known Fenton reaction was proposed in 1894. Two enzymes, peroxidase and catalase, found in 1898 and 1901, respectively, were quickly recognized to play important roles in H_2O_2 metabolism in humans. Shortly after the discovery of another important enzyme, superoxide dismutase (SOD), in 1969, research efforts on biological properties of H_2O_2 have significantly increased (Li 1996).

The toxicology of H_2O_2 has been investigated extensively, and there are a number of comprehensive reviews on the topic available in the literature (IARC 1985; ECETOX 1993; Li 1996; SCCP 2005; CEU 2011). A key characteristic of H_2O_2 is its capability of producing reactive oxygen species (ROS), which are known to induce various toxicities, including hydroxyl free radicals that have been implicated in various stages of carcinogenesis (Floyd 1990; Li 1996). Oxidative reactions of ROS with proteins, lipids, and nucleic acids are believed to be involved in a number of potential pathological consequences; the damage by oxidative free radicals may be associated with aging, stroke, and other degenerative diseases (Harman 1981; Floyd et al. 1988; Lutz 1990; Li 1996).

The major mechanism responsible for the observed toxicity of H_2O_2 is believed to be the oxidative reactions and subsequent damage in cells by ROS. In cell culture studies, H_2O_2 is highly cytotoxic at concentrations ranging from 1.7 to 19.7 μg/mL or 0.05 to 0.58 mmol/L (Rubin and Farber 1984; Bates et al. 1985; Ramp et al. 1987; Tse et al. 1991; Hanks et al. 1993; Li 1996, 2003). Hepatocytes were less sensitive to the cytotoxicity of H_2O_2 than fibroblasts and endothelial cells (Sacks et al. 1978; Simon et al. 1981; Rubin and Farber 1984), while human gingival fibroblasts derived from primary cultures and L929 mouse fibroblasts (ATCC CCL 1; Manassas, VA) were found to respond similarly to the cytotoxicity of H_2O_2 (Li 1996).

On the other hand, the human body is equipped with various defensive mechanisms available at cellular and tissue levels to prevent potential damage of H_2O_2 to cells during oxidative reactions and to repair any damages sustained. A number of enzymes, such as catalase, SOD, peroxidase, and selenium-dependent glutathione peroxidase, exist widely in body fluids, tissues, and organs, to effectively metabolize H_2O_2 (Floyd 1990; Li 1996). Simply adding iron chelators and antioxidants or increasing serum concentration in culture media effectively reduces or eliminates the cytotoxicity of H_2O_2 (Sacks et al. 1978; Rubin and Farber 1984). In a cell culture study, 20 mM H_2O_2 was undetectable after 30 min in the culture media alone and after 15 min in the media with bone tissues, indicating decomposition and inactivation of

hydrogen peroxide in cell culture systems (Ramp et al. 1987). These enzymes also exist in human saliva; in fact, salivary peroxidase has been suggested to be the body's most important and effective defense against the potential adverse effects of H_2O_2 (Carlsson 1987). Marshall and coworkers (2001) found that the human oral cavity, including that of adults, juveniles, infants, and adults with impaired salivary flow, was capable of eliminating 30 mg H_2O_2 in less than one and a half minutes.

3.3 Peroxides in the Human Body

The detection of H_2O_2 in human respiration was first reported in 1880; however, it was not until 1969 when SOD was discovered H_2O_2 was recognized as an important by-product in oxygen metabolism of humans (Li 1996, 2011). H_2O_2 is now known as a normal intermediate metabolite in humans. It exists in human serum, and it is present in human breath at levels ranging from 0.34 to 1.0 μg/L (Sies 1981; Williams et al. 1982). The daily production of H_2O_2 in human liver is approximately 6.48 g in a period of 24 h (FDA 1983). An important source of endogenous H_2O_2 is from phagocytic cells, such as neutrophils and macrophages, which play an essential role in defense against various pathological microorganisms.

3.4 Systemic Effects

Systemic effects of H_2O_2 have been investigated for both acute and chronic exposures. A unique characteristic of H_2O_2 in inducing systemic toxicity is its concentration in addition to the dosage.

The reported acute systemic toxic effects of H_2O_2 in animals vary widely, according to the H_2O_2 concentration as well as the application mode. In rats, the intravenous 50 % lethal dose (LD_{50}) of H_2O_2 was found to be 21 mg/kg (Spector 1956). Using the up-and-down method, in which the dosing is adjusted up or down according to the outcome (death or survival) of the animal that received the previous dosage, the oral LD_{50} of 4 % H_2O_2 solution in male and female rats was estimated at 780 and 600 mg/kg, respectively (Li 1996). The LD_{50} for percutaneous application of H_2O_2 is much higher at >7,500 mg/kg (FDA 1983). The values of LD_{50} are inversely related to the concentrations of H_2O_2, and they vary markedly between different animal species and strains (IARC 1985; FDA 1983; ECETOX 1993; Li 1996). Tissue responses to topical application of H_2O_2 are also related to the H_2O_2 concentration, but they are usually minimal at low concentrations of ≤3 %.

Acute toxicity, including fatalities, has been reported in humans who accidentally ingested large amounts of concentrated H_2O_2 solutions (Spector 1956; Giusti 1973; Giberson et al. 1989; Humberston et al. 1990; Rackoff and Merton 1990; Christensen et al. 1992; Cina et al. 1994; Sherman et al. 1994; Asanza et al. 1995; Ijichi et al. 1997; Rider et al. 2008; Byrne et al. 2014). A retrospective survey of a regional poison control center found that over a 36-month period, 325 cases were caused by H_2O_2 poisoning, which accounted for 0.34 % of all the reported causes (Dickson and Caravati 1994); however, the majority of the 325 cases (71 %) was

pediatric population (age <18 years), with ingestion of H_2O_2 solution being the most common route of exposure (83 % of cases). One major factor associated with the toxicity of H_2O_2 is its concentration. Ingestion of H_2O_2 solutions of less than 10 % usually produces no significant adverse effects, although it may cause mild irritation to mucous membranes, which results in spontaneous emesis or mild abdominal bloating (Humberston et al. 1990; Dickson and Caravati 1994). Exposure to H_2O_2 concentrations higher than 10 %, however, can result in severe tissue burns and significant systemic toxicity. In addition to the tissue damage caused by oxidative reactions, gas embolism is responsible for various pathological consequences of H_2O_2 ingestion (Rackoff and Merton 1990). Each milliliter of 1 % H_2O_2 releases 3.3 mL oxygen; therefore, 10 mL of 30 % H_2O_2 can produce 1 liter oxygen (Giberson et al. 1989; Humberston et al. 1990). Common symptoms observed in acute toxicity of H_2O_2 include stomach and chest pain, retention of breath, foaming at the mouth, loss of consciousness, motor and sensory disorders, fever, gastric hemorrhage, and liver damage. Although rare, death can occur.

Several animal studies have been conducted on acute systemic toxicity of tooth whiteners containing carbamide peroxide. Oral gavage of 5 g/kg tooth whiteners containing 10 and 22 % carbamide peroxide produced no evidence of acute systemic toxicity in rats (Cherry et al. 1993; Adam-Rodwell et al. 1994). One study reported unusually low LD_{50} (87.18–143.83 mg/kg) of two products containing 10 % carbamide peroxide in female Swiss mice (Woolverton et al. 1993). The reasons for the low LD_{50} values are unclear but may be attributed to differences in animal species, materials, and method. Using the up-and-down method, the LD_{50} of a tooth-whitening gel with 10 % carbamide peroxide was estimated at 23.02 g/kg in female rats (Li et al. 1996).

Chronic systemic toxicity of H_2O_2 has been investigated using animal models. No visible abnormalities were detected in mice drinking 0.15 % H_2O_2 (about 150 mg/kg/day) *ad libitum* for 35 weeks, and their growth was also normal (FDA 1983). Necropsy results, however, showed changes in the liver, kidney, stomach, and small intestine. Solutions of >1 % H_2O_2 (>1 g/kg/day) caused pronounced weight loss and death of mice within two weeks. A rat study by Ito et al. (1976) found that when administered by an oral gastric catheter 6 days weekly for 90 days, the dose of 506 mg/kg suppressed body weight gain, decreased food consumption, and caused changes in hematology, blood chemistry, and organ weights. The principal tissue affected was gastric mucosa, and the effects were local. The no-observed-effect level (NOEL) of H_2O_2 was 56.2 mg/kg/day. Another rat study found that the NOEL of H_2O_2 was 30 mg/kg/day when animals were treated by oral gastric catheter daily for 100 days (Kawasaki et al. 1969). The same study showed no adverse effects in rats receiving the diet containing 6 mg H_2O_2 in 20 g of food.

3.5 Genotoxicity

The genotoxic potential of H_2O_2 has been investigated extensively using microbes, plants, insects, cultured mammalian cells, and animals (IARC 1985; ECETOX 1993; Li 1996; SCCP 2005). In a number of bacterial systems, H_2O_2 induced

point mutations or single-strand breaks in DNA. Positive mutagenicity of H_2O_2 has also been detected in some newer tester strains of the Ames *Salmonella* mutagenicity test; however, effects are eliminated when tested with S9 activation. S9 is a rat liver microsomal preparation that contains various enzymes. It has been found to increase the sensitivity and overall performance of the Ames *Salmonella* mutagenicity test, and therefore, experiments both with and without S9 are required for the Ames *Salmonella* mutagenicity test (Maron and Ames 1983). The results obtained from mammalian cells are similar to those from the Ames *Salmonella* mutagenicity test; that is, the genotoxic effects of H_2O_2 are detected only in test systems without S9 activation. The effect of S9 on H_2O_2-induced DNA or chromosomal changes in mammalian cells in vitro is believed to originate from the H_2O_2-degrading enzymes in the S9, which is the same as that observed in the Ames *Salmonella* mutagenicity test.

The genotoxicity of H_2O_2 has also been examined using in vivo systems, and results indicate that H_2O_2 is not genotoxic in various animal models (IARC 1985; ECETOX 1993; Li 1996; SCCP 2005). The overall data available so far show that H_2O_2 is genotoxic only in in vitro systems without enzymatic activation. When enzymatic activation is incorporated into in vitro systems or when tested in animals, H_2O_2 is nongenotoxic.

3.6 Carcinogenicity

The carcinogenicity of H_2O_2 was the subject of a number of critical reviews (IARC 1985; ECETOX 1993; Li 1996, 1998, 2000, 2011). Several investigators found no evidence of carcinogenicity of H_2O_2 or carbamide peroxide. Repeated subcutaneous injections of 0.5 % H_2O_2 for up to 332 days did not induce tumors in a mouse study (Nakahara and Fukuoka 1959). Another 56-week study showed that 5 % carbamide peroxide and 3 % H_2O_2 were inactive as tumor promoters (Bock et al. 1975). Klein-Szanto and Slaga in 1982 reported that twice-weekly application of 15 and 30 % H_2O_2 on mouse dorsal skin for 50 weeks did not induce any squamous cell carcinomas, and they thus concluded that H_2O_2 at 15 and 30 % was not a complete carcinogen. The same study also found that at 15 and 30 % concentrations, H_2O_2 was not a tumor initiator but exhibited extremely weak tumor-promoting activity after 25 weeks of twice-weekly application following previous application of the carcinogen DMBA as the initiator. At concentrations <15 %, H_2O_2 did not cause tumor promotion. In contrast, Nagata and coworkers (1973) reported that a single subcutaneous injection of 0.6 % H_2O_2 was not carcinogenic, and in fact, repeated applications of 0.6 % H_2O_2 on mouse skin significantly inhibited tumor development induced by the potent carcinogen benzo(α)pyrene.

The studies that reported carcinogenicity of H_2O_2 and subsequently generated safety concerns about the use of H_2O_2 or peroxide-containing tooth whiteners were conducted by Ito's group (1981, 1982, 1984) and Weitzman and coworkers (1986). In the 1981 study by Ito and coworkers, male and female C57Bl/6 J mice received 0.1 % or 0.4 % H_2O_2 in drinking water for up to 100 weeks, with distilled water as the negative control. An increased incidence of duodenal carcinoma was observed

in females only in the 0.4 % H_2O_2 group (4 of 50 mice), and one carcinoma was observed in one male mouse in each of the 0.1 and 0.4 % groups. However, results showed no dose-related incidence of duodenal adenomas. Using standard methods for data analysis in which sexes are analyzed separately, no significant increase in carcinoma incidence was noted in males or females. Statistical significance was achieved only when the data from males and females were combined.

In the second study by Ito's group (1982), three strains of mice, including the C57Bl/6 N strain that was used in the initial study, received 0.1 % or 0.4 % H_2O_2 solution in drinking water for up to 740 days. Duodenal cancer (pathologically not defined as benign, malignant, carcinoma, or adenoma) was observed only in C57Bl/6 J mice between 420 and 740 days, with an incidence of 1 and 5 % for the 0.1 and 0.4 % H_2O_2 groups, respectively. However, temporary cessation of H_2O_2 and replacement with distilled water for 10, 20, or 30 days decreased the incidence of lesions in both the stomach and duodenum.

The third study by Ito's group (1984) investigated four strains of mice that received 0.4 % H_2O_2 solution in drinking water for 7 months (C57Bl/6 N mice) and 6 months (other three strains). The incidence of duodenal lesions was highly strain-dependent and inversely related to duodenal, liver, and blood catalase activity. C57Bl/6 N mice had low catalase activity, and the number of tumors was 41 times that observed in mice with high catalase activity, and about ten times higher than that for the strain with normal catalase activity. Of particular interest is the observation that another strain of catalase-deficient mice had a lower duodenal tumor incidence, both in total number of tumors and number of tumors per mouse, than that of the C57Bl/6 N mice.

Because of the potential significance of the results reported by Ito's team, these studies were reviewed and carefully evaluated for study design, experimental conduct, and data presentation (FDA 1983; IARC 1985; FDA 1988; ECETOX 1993). Major limitations of the research include unverified H_2O_2 concentration and stability in drinking water, inadequate control and documentation of tumor pathology, and lack of information on food consumption and survival. In addition, these studies did not measure individual animal water intake, which is relevant because reduced water intake may contribute to the development of lesions. When water consumption is decreased, the texture of the stomach contents changes, which may increase the likelihood of tissue injury when coarse materials transverse the duodenum, resulting in an increased rate of cell proliferation, or regenerative hyperplasia (Bertram 1991). From a 14-day study in C57Bl/6 N mice, water consumption was found to decrease with increasing H_2O_2 content (Weiner et al. 2000). Therefore, in the same strain of mice it is appropriate to assume that the decrease in water intake also occurred during H_2O_2 exposure in Ito's studies. As a consequence, gastrointestinal irritation occurred. As observed in Ito studies, changes to the epithelia were primarily localized to the duodenum, indicating that the lesions are not chemically induced but indicative of mechanical irritation. On the other hand, as demonstrated by Ito and coworkers, the C57Bl/6 N mouse strain used in their studies has a low level of duodenal catalase activity and a high spontaneous incidence of premalignant duodenal lesions. The difference in catalase activity among animal strains

likely is one of the reasons that other studies that used a similar experimental design to the study of Ito's group, have not found carcinogenicity of H_2O_2. As such, after evaluating the Ito studies, the Cancer Assessment Committee (CAC) of the US Food and Drug Administration (FDA) concluded that Ito's studies did not provide sufficient evidence that H_2O_2 was a duodenal carcinogen.

The study by Weitzman et al. (1986) examined effects of topical application of H_2O_2 on the cheek porch mucosa of male Syrian golden hamsters. Animals were treated twice weekly with DMBA, a carcinogen, in combination with 3 or 30 % H_2O_2 for 19 or 22 weeks. Groups receiving DMBA or 30 % H_2O_2 alone were also included. Results showed that 30 % H_2O_2 alone did not induce any tumors at either of the two time periods. At 19 weeks, no tumors were observed in animals receiving the DMBA and 3 % H_2O_2, and 30 % H_2O_2 had no tumor-enhancing effect. After 22 weeks, there was no tumor-enhancing effect with 3 % H_2O_2. The incidence of carcinomas was higher in animals receiving a combination of 30 % H_2O_2 and DMBA (5/5 animals) compared to those treated with DMBA alone (3/7 animals), but the significance level was marginal ($p = 0.054$). The significance of the observed increase in incidence of carcinoma associated with 30 % H_2O_2 at 22 weeks has been questioned because of the small number of animals used and the marginal statistical significance observed (Li 1996; Marshall et al. 1996). It is also difficult to explain the marked differences in results between the two time periods, an interval of only 3 weeks. In addition, repetitive treatment with H_2O_2 solutions greater than 15 % was considered too irritating to tissues to enable detection of tumor-promoting activity, because cells would not survive the toxic effects of high concentrations of H_2O_2 (Klein-Szanto and Slaga 1982). Marshall and colleagues (1996), using the similar experiment design to the Weitzman study, found that H_2O_2 up to 3 % was not carcinogenic or cocarcinogenic. The studies by Weitzman et al. (1986) and Marshall et al. (1996) are particularly significant in that they do not demonstrate a synergistic effect between H_2O_2 and the polycyclic aromatic hydrocarbon DMBA during coadministration. Tumor promotion studies (Bock et al. 1975; Klein-Szanto and Slaga 1982) provide additional evidence for a lack of interaction between chemical carcinogens and H_2O_2. The study by Marshall et al. (1996) found a reduction in tumor incidence following H_2O_2 administration, and such an effect was observed with 3 % H_2O_2 and baking soda in the hamster cheek pouch model.

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Complications from the Use of Peroxides

4

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Abstract

While dental whitening or dental bleaching is one of the most popular aesthetic procedures, dentists should base their decision to prescribe peroxide-based whitening agents on evidence-based techniques and regimens. The contact of bleaching products with the mucosa, dental tissues, and preexisting restorations may trigger a series of adverse effects both on soft and hard tissues. This chapter details the precautions that must be taken prior to prescribing different bleaching therapies with the goal of improving the patient's smile, without damaging oral tissues. Additionally, this chapter will discuss tooth sensitivity, which is the most common side effect of any bleaching treatment. The prevention of tooth sensitivity is of upmost importance to avoid discomfort to patients and increase patient's compliance.

4.1 Introduction

Tooth bleaching (or tooth whitening) is a cosmetic procedure widely used owing to its technical simplicity, proven clinical efficacy, and noninvasiveness, as it does not remove tooth structure. Tooth bleaching is based on the oxidative potential of hydrogen peroxide (HP), the main active component of bleaching agents, as this molecule is able to diffuse through the tooth enamel and promote the breakdown of organic

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pigments in dentin. The two bleaching techniques traditionally used under the supervision of a dental professional are the at-home (Chap. 6) and the in-office (Chap. 7) bleaching techniques. Dentist-supervised at-home whitening is considered the safest for the patient, as a result of the low concentration of carbamide peroxide (CP) or hydrogen peroxide (HP) used. Although the dental professional supervises the at-home bleaching technique, the patient applies the product at home. This bleaching procedure has raised questions concerning the risk of systemic toxicity in addition to the indiscriminate and/or inappropriate use of bleaching products by patients, which may increase the risk of adverse effects on oral tissues.

In-office techniques traditionally involve the use of HP-based gels in high concentrations (30–40 %). The procedure is performed in the dental office under full control of the dentist. However, the use of HP in high concentrations results in the diffusion of the molecule into the pulp chamber in toxic levels, resulting in important changes in the pulp connective tissue that have been linked to high levels of postbleaching sensitivity (Costa et al. 2010; de Almeida et al. 2015b; Simões et al. 2015). Additionally, extreme caution must be taken to protect oral soft tissues and prevent swallowing of bleaching product, as contact of this product with oral mucosa can cause chemical burns resulting in severe discomfort for the patient.

In clinical practice, tooth sensitivity and gingival tissue irritation are the most frequently reported adverse effects by patients who undergo different bleaching treatments (Almeida et al. 2012; Simões et al. 2015). However, in addition to its adverse effects on soft tissues, tooth bleaching has been scientifically proven to cause changes in mineralized tissues and in existing restorations, the extent of which depends on the technique used (Hayacibara et al. 2004; Cavalli et al. 2004; Sasaki et al. 2009; Mourouzis et al. 2013; AlQahtani 2013; Soares et al. 2013b; Torabi et al. 2014b). Thus, in this chapter, we will discuss changes in soft tissues, mineralized tissues, and adhesive restorations caused by the different bleaching techniques currently available and the clinical aspects related to postbleaching tooth sensitivity.

4.2 Effect on Oral Soft Tissues

The literature has shown that HP causes major changes in various cell types (Zhu et al. 2012). Therefore, the contact of the molecule with biological tissues such as gingival tissue, periodontal ligament, and pulp tissue during bleaching is not desirable. As bleaching products with different HP concentrations, presentation forms, and application protocols are available for clinical or home use, distinct cellular responses are expected depending on the regimen used. Thus, the dentist should be aware of the possible adverse effects of each therapy in order to use the best clinical alternative for each particular clinical situation. In this chapter, greater emphasis will be given to the effects of the different bleaching modalities on gingival and periodontal tissues. The effect of these techniques on the pulp tissue will be described in detail in Chap. 5.

Direct contact of the bleaching gel with oral soft tissues may cause chemical burns due to the caustic potential of HP, resulting in the development of gingival ulcers and erosions (Powell and Bales 1991; Haywood et al. 1997; Oda et al. 2001). HP may also cause changes in the periodontal tissue that can lead to gingival recession. The magnitude of these effects is proportional to the contact time and concentration of HP in the bleaching product (Powell and Bales 1991; Haywood et al. 1997; da Costa Filho et al. 2002). These negative effects can be prevented in the in-office technique by carefully applying a gingival barrier, which effectively precludes the contact of the whitening gel with gingival tissue and periodontal ligament. For the unsupervised at-home technique, the fabrication of a suitable tray with clear instructions for use is indispensable to prevent irritation of oral tissues. However, many over-the-counter (OTC) products (Chap. 6) are available for use without dentist supervision and are applied in trays with poor adaptation to dental arches, which can increase the risk of product contact with the periodontal tissues, especially in patients with dental misalignment. In addition, bleaching strips with various HP concentrations, which do not require the use of trays, have gained popularity mainly because of their low cost compared to that of dental professional-supervised bleaching. However, these OTC products result in direct contact of the gingival papilla with the bleaching strip, as they are not customized to fit the individual dental arches of patients. With this in mind, the biological effects of different types of bleaching techniques on soft oral tissues are discussed in the following sections based on scientific evidence available in the current literature.

4.2.1 At-Home Bleaching

The supervised at-home bleaching technique is based on the use of bleaching gels containing 10–22 % CP or 4–10 % HP. However, only 10 % CP has received the American Dental Association (ADA) seal of acceptance, as seen in Chap. 6. Therefore, at-home bleaching that involves the use of a 10 % CP gel has been considered the safest treatment modality. In this regard, the recent literature provides long-term reports on the aesthetic and biological effects up to 17 years post treatment (Boushell et al. 2012).

CP is a product of the weak link between HP and urea, which is easily broken in the presence of water, releasing about 3.3–3.5 % HP in the process (Kwon et al. 2002; Sulieman 2008). Thus, the mechanism of CP home bleaching gels is the slow and gradual release of low HP concentrations onto the tooth structure. For this reason, the product should be applied daily for 1–8 h, over relatively long periods (1–4 weeks) in order to achieve the desired aesthetic result.

Gingival irritation associated with supervised home bleaching is related to two key factors, namely (1) mechanical trauma due to the tray and (2) the toxic effect of the gel on the oral mucosa. The first step to prevent trauma to gingival tissues during at-home bleaching is to use custom-fitted trays by dentists in a patient's stone model. Prefabricated trays do not provide good adaptation and may expose the oral mucosa to contact with the peroxide bleaching product. Still, the tray may cause trauma due

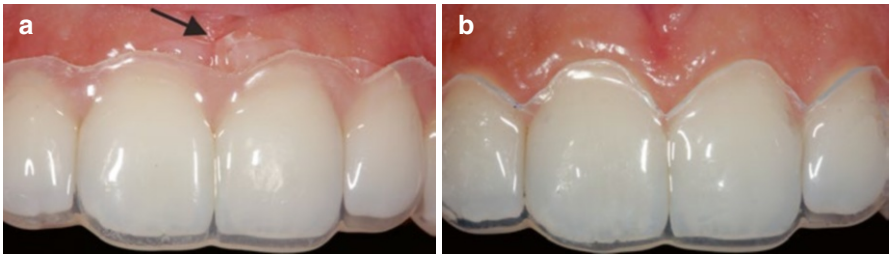


Fig. 4.1 Traditional tray showing (**a**) the evident possibility of direct contact of the bleaching gel (*arrow*) with soft tissue and (**b**) the scalloped tray, 0.5 mm short of the gingival tissue line, that allows keeping the bleaching gel in contact exclusively with enamel

to defects in the stone model or improper trimming of the tray. To prevent this adverse event, during tray try-in the practitioner must identify potential compression areas that can produce traumatic ulcerative lesions, which may result in severe discomfort for the patients (Matis 2003).

Once the possibility of mechanical trauma to gingival tissue caused by the tray is ruled out, efforts should be directed to retain the gel inside the tray throughout its use, reducing the possibility of leakage of the product and its consequent contact with adjacent soft tissues. In vitro studies have determined that low HP concentrations (3 %) exert a cytotoxic effect on gingival fibroblasts and negatively affect proliferative capacity, fibronectin expression, and Type I collagen (Tipton et al. 1995; Oda et al. 2001). Animal studies have shown that the topical application of 10 % CP in rat tongue for 20 min once a week, for 3 weeks, promoted epithelial changes characterized by increased cell proliferation of the basal layer, which was transient and reversible 10 days after the procedure (De Castro Albuquerque et al. 2002). This demonstrates that even low HP concentrations can have a toxic potential when in direct contact with oral mucosa cells.

Several alternatives have been discussed in relation to the design of the tray that can prevent leakage of the bleaching gel to soft tissue regions. Scalloping the tray at the gingival level has proven to be an effective measure to prevent the outflow of product to regions beyond the cervical tooth region (Matis 2003). It is, therefore, suggested that the tray does not extend to the gingival tissue or should be ≈0.5 mm short of the gingival tissue line, preventing its compression while minimizing the possibility of direct contact of the bleaching product with soft tissue. After the appropriate volume of the bleaching gel is applied into the tray, the excess gel must be removed using a toothbrush or a cotton swab immediately after the patient adapts the tray in the mouth. As discussed in Chap. 6, the use of tray reservoirs does not improve bleaching effectiveness (Matis 2003). Additionally, the inclusion of reservoirs results in a greater amount of HP detectable in saliva (Matis et al. 2002). Thus, reservoirs are not indicated. This will result in better adaptation of the tray to the teeth to be bleached and less leakage of the bleaching gel, preventing tissue damage. Figure 4.1a shows the overlapping of extended scalloped trays with the gingival tissue in comparison trays that are trimmed to avoid overlapping with the tissue (Fig. 4.1b).

In a clinical study conducted by Leonard et al. (2001), a 10% CP gel applied daily (6–8 h) for 14 days using scalloped trays resulted in minimal adverse effects on oral soft tissues. The authors conducted an analysis of the marginal (gingival index) and nonmarginal gingiva (nonmarginal gingival index), and the possible changes in non-gingival soft tissues (non-gingival oral mucosal index) after 7 and 14 days of treatment. They did not observe any significant differences in all the parameters tested between the bleached and nonbleached groups. Only 8% of the patients reported gingival irritation during the treatment course, and none of them reported gingival irritation during 3, 6, and 47 months posttreatment. Similar results were found in the study by Almeida et al. (2015) where the application (2 h daily) of bleaching gels containing 10 and 16% CP in scalloped trays for 21 days did not cause genotoxicity in the adjacent gingival tissue to the teeth subjected to bleaching. In pre- and postbleaching analyses, Firat et al. (2011) observed that the application of 35% CP gel at home in trays with gingival trimming for 15 days (30 min/day) caused no changes in clinical parameters for the gingival tissue and periodontium, but increased levels of proinflammatory cytokines in crevicular fluid. A number of clinical studies reported that contact of at-home bleaching gels with oral tissues resulted in inflammation and erosion of the marginal gingiva, as well as cervical resorption and gingival recession, with the contact of the products with the marginal gingiva for long periods being contraindicated (Powell and Bales 1991; Haywood et al. 1997). Costa-Filho et al. (2002) performed a gingival tissue biopsy for smokers and nonsmokers who underwent treatment with a gel containing 10% CP, applied for 8 h/day for a period of 5 weeks. Biopsies performed immediately after the bleaching treatment revealed an increase in epithelial thickness and cell proliferation to basal and parabasal layers, resulting in morphometric changes of the gingival tissue, when compared with biopsies performed 15 days before the bleaching treatment. The results were similar between the smokers and nonsmokers. In the abovementioned clinical studies, bleaching trays without gingival trimming and reservoir in the vestibular region were used.

More recent studies reported that the immediate efficacy of bleaching does not appear to be affected by the smoking habit (de Geus et al. 2015a, b, c). Additionally, tray whitening did not induce DNA damage to soft tissues during the treatment (de Geus et al. 2015c) or even increase the risk of tooth sensitivity (de Geus et al. 2015a).

The possibility of daily contact of bleaching agents rich in HP with oral tissues remains controversial. Several studies showed that HP in high concentrations might promote cancer in the duodenum and jejunum of rats treated concomitantly with carcinogens. However, HP administration alone did not lead to the development of lesions in these tissues (Naik et al. 2006; Minoux and Serfaty 2008; Paula et al. 2015). However, according to the results reported by Hannig et al. (2003), only 1.25% HP present in the gel with 10% CP was detected in saliva from patients who underwent bleaching in conventional individualized trays with a 1.5-mm reservoir, which was the highest release observed within the first 5 min. Thus, the systemic effects of HP derived from at-home bleaching are still considered quite controversial (Naik et al. 2006; Minoux and Serfaty 2008). It is noteworthy that a

cocarcinogen agent does not start mutations alone but requires a neoplastic initiator in the oral cavity (Naik et al. 2006). The possibility of the correlation between a cocarcinogen agent and an initiator is a contraindication for carrying out aesthetic treatment.

Treatment supervision by a qualified dental professional is extremely important to diagnose early changes in hard and soft tissues. The indiscriminate online access to home bleaching materials and the use of OTC products from online retailers and conventional drugstores may increase the safety risks of tooth whitening.

While there are an abundant number of studies on CP-based gels for at-home application, few studies are available related to the effects of HP-based home gels on soft tissues. Several studies showed that the amount of HP in saliva is proportional to the HP concentration in bleaching gels and that the use of CP-based gels results in lesser amount of HP in saliva than products containing pure HP (Hannig et al. 2003, 2005). The decomposition of 10 % CP into HP has been demonstrated to occur primarily in the first hour after bleaching, and this degradation occurs primarily in the region of product contact with the tooth surface (Matis et al. 1999). However, current clinical evidence shows that a better bleaching outcome is obtained when 10 % CP is applied for 8–10 h (Matis et al. 2009; Cardoso et al. 2010), as seen in Chap. 6.

Thus, the application of a sufficient amount of bleaching gel (enough to cover the tooth surface) in a custom-fitted scalloped tray without reservoirs promotes effective tooth bleaching with minimal damage to oral soft and/or pulp tissue. Furthermore, in order to prevent inadvertent swallowing of bleaching product residues, home bleaching with the use of gels with 10 % CP applied using a scalloped tray model slightly short of the free gingival margin (≈0.5 mm) has been recommended.

The direct contact of bleaching gels with gingival and periodontal tissues should be avoided in order to eliminate the possibility of tissue damage mediated by HP.

4.2.2 OTC Products

Currently, another type of tooth bleaching treatment, which involves the use of OTC products, has become popular (see Chap. 6). OTC products can be purchased in supermarkets, drugstores, or even on the Internet, and are used without dentist supervision. These products emerged in the United States about 15 years ago as an alternative treatment of stained teeth with lower cost than traditional supervised treatment (Demarco et al. 2009). The active component is the same as that in

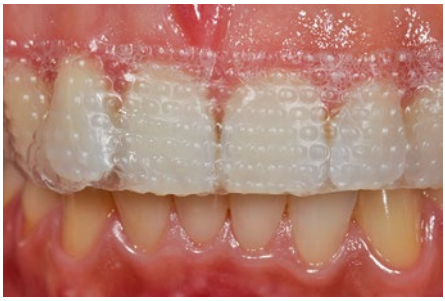


Fig. 4.2 Gingival tissue in contact with bleaching strips

traditional bleaching agents, that is, either CP (10–18 %) or HP (1.5–14 %), which is available in various forms such as bleaching strips, varnishes, gels, paint-on liquids, mouthwashes, and toothpastes. However, these products offer no protection to soft tissue adjacent to the teeth subjected to bleaching. As such, their indiscriminate use without professional guidance raises concerns about the possible adverse effects (Demarco et al. 2009).

Also available online are bleaching gels to use with universal trays, even those administered with lights and electrodes. Poor adaptation of the tray results in the flow of the material to the oral cavity, causing contact of a large amount of product with the oral mucosa and possible swallowing of high concentrations of toxic components. An OTC product was applied in areas of gingival recession (Ghalili et al. 2014), a procedure contraindicated especially when using prefabricated trays. Studies that used varnishes and paint-on liquids showed that these products do not promote effective bleaching of the tooth surface (Kishta-Derani et al. 2007; Lo et al. 2007). Furthermore, clinical evidence suggests that both at-home bleaching and in-office bleaching are equally efficient for bleaching teeth and are found to be superior to Whitestrips (Bizhang et al. 2009). We therefore consider that these materials, apart from having poor aesthetic effectiveness, may also cause some risk to the health of consumers (Fig. 4.2).

Among the OTC bleaching products, bleaching strips are the most popular products owing to their aesthetic result, making them superior to other products of the same category available in the market (Xu et al. 2007; Yudhira et al. 2007; Kwon et al. 2013). These products were created to use without trays, with a thin layer of HP added to the adhesive surface that is released in relatively short periods (5–60 min). A systematic literature review demonstrated that there are differences in aesthetic effectiveness between the products due to the levels of active ingredients and that the whitening strips and products with high concentrations of HP caused more adverse effects (Hasson et al. 2006).

Bleaching gels with 10 % CP for at-home use contain 3.3–3.5 % HP in its composition, about half of the HP concentration found in the less-concentrated strips. Clinical studies have demonstrated that the HP concentration in the saliva of patients who underwent bleaching with strips (5.3 % HP) is about two to four times higher than the concentration in saliva observed for 10 % and 15 % CP gels applied in custom trays (Hannig et al. 2003, 2005).

As the bleaching strips have a predefined shape, contact of the HP-rich surface with the gingival papilla occurs during treatment. As discussed previously, adverse effects on gingival and periodontal tissues are expected, and the extent thereof may lead to the development of problems in patients, especially because the products are applied without dentist supervision, which may lead to its indiscriminate use.

The main concern regarding the safety of these bleaching strips is related to the absence of protection of the gingival tissues.

In an interesting study by Auschill et al. (2012), the bleaching efficacy and biological effects on soft tissues provided by bleaching agents with similar HP concentrations were evaluated. However, the products were applied according to the supervised at-home technique or by using a bleaching strip. Patients were instructed to use a home bleaching gel containing 5% HP in a scalloped tray with a 1.0-mm reservoir or to apply a bleaching strip containing 5.3% HP, without any method of protection of soft tissues, as recommended by the manufacturers. Both products were applied twice daily for 30 min during the 14-day period. The bleaching efficacy during, at the end, and 18 months after the treatment was statistically similar for both products. However, 40% of the patients who used the bleaching strip reported soft tissue irritation, while only 20% of patients who underwent bleaching with a tray reported the development of this adverse effect. Tooth sensitivity was also more prevalent in the patients who used a bleaching strip (60%) than in those who used a gel in the tray (47%). In both groups of patients, the adverse effects were considered mild and transient. Taking into account both biological factors (soft tissue irritation and tooth sensitivity), the incidence of discomfort during bleaching was higher for the patients who underwent bleaching with bleaching strips.

Other clinical studies showed that the percentage of tooth sensitivity and soft tissue irritation are proportional to the concentration of HP in bleaching strips, the contact time with tooth and soft tissues, and the total treatment time (Kugel et al. 2011; Donly et al. 2010). According to the data reported by Swift et al. (2009), about 80% of soft tissue irritation cases occurred after long treatment periods in patients who used bleaching strips. Lucier et al. (2013) evaluated the toxic effect of bleaching strips containing 6–14% HP on the gingival epithelium in vitro in a three-dimensional culture model. The authors observed that the application of the bleaching strips for 30 min resulted in changes in tissue morphology that were associated with the apoptotic death of cells in all epithelial layers, inducing keratinocyte proliferation and increased expression levels of proinflammatory cytokines. These effects were proportional to the HP concentration.

The presence of cracks, enamel craze lines, exposed dentin, caries lesions, wear facets, noncarious cervical lesions, restorations with marginal gaps, gingival recession, gingivitis, and periodontal disease can influence the extent of the harmful effects of bleaching products on oral and pulpal tissues.

However, the negative effects caused by bleaching agents used in these specific clinical situations have been rarely studied. By contrast, a number of studies have evaluated the aesthetic effectiveness of bleaching products and techniques. Thus, the dentist should carry out a careful clinical exam prior to starting the bleaching treatment in order to determine the ideal treatment for each particular case. As discussed above, the use of products containing HP for tooth bleaching without professional supervision represents a health risk to the population generally unaware of the factors involved with the use of such products and the conditions of their oral health. Regulatory bodies in Brazil (ANVISA 2015) and in the European Union (European Commission Scientific Committee on Consumer Products 2007; European Commission Scientific Committee on Consumer Safety 2010) have already restricted the commercialization of sodium perborate and hydrogen peroxide-based bleaching products in order to protect the population from the risks of using bleaching agents without professional supervision. However, in several countries, including the United States, such products are still accessible over-the-counter to the general population and have great economic impact owing to the strong aesthetic appeal involved (Demarco et al. 2009). Nevertheless, according to the Food and Drug Administration (2003), hydrogen peroxide is safe at concentrations of up to 3%, but there are insufficient data available to permit final classification of its effectiveness at 1.5–3 % concentrations for long-term OTC use in the mouth.

4.2.3 In-Office Bleaching

It is well established that once the in-office technique is decided upon, all oral soft tissues, as well as the face and eyes of patients, must be protected from accidental contact with the bleaching products. Clinical studies in which qualified practitioners performed the entire bleaching procedure reported a percentage of 0–4 % of patients with mild to moderate soft tissue irritation, irrespective of the HP concentration used (Marson et al. 2008; Ward and Felix 2012). This result is expected, as the placement of a suitable gingival barrier with flowable light-cured resin effectively prevents the contact of the bleaching gel with the gingival and periodontal tissues. However, the gingival barrier must be created carefully, and it must extend not only to the cervical region of the teeth to be bleached but also to the adjacent soft tissues in order to prevent inadvertent contact with the bleaching product (please refer to video “In-Office Whitening”). Lip retractors and labial and lingual protectors should also be used with constant suction. Figure 4.3 demonstrates the correct use of gingival barriers and intraoral protective equipment in the in-office bleaching. This equipment will prevent the contact of the bleaching agent with other oral tissues caused by inadvertent movements of the patient. At the end of the application period, the gel must be carefully aspirated from the tooth surface, followed by washing with simultaneous suction, in order to prevent the flow of highly concentrated product to the oral cavity and the swallowing of product residue by the patient (Fig. 4.3).

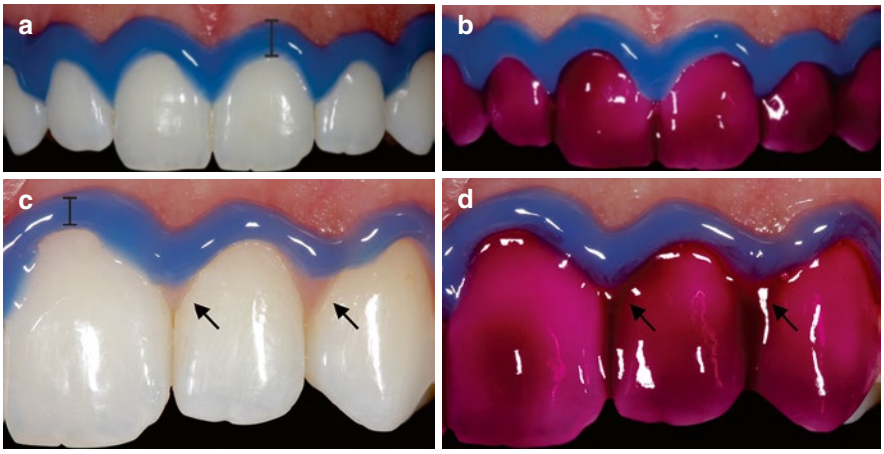


Fig. 4.3 (**a**) Correct use of the gingival barrier, carefully positioned in the cervical region of the enamel, covering interdental regions and a considerable portion of the marginal gingiva. (**b**) Application of bleaching gel in ideal conditions, minimizing the possibility of accidents with the product. (**c**) Incorrect application of the gingival barrier, which does not adequately extend to the tooth surface, only extending slightly into the soft tissue (*arrow*) and gingival papillae. (**d**) A typical accident during the bleaching procedure – the bleaching gel contacts the gingival tissue (*arrow*). This is a placebo gel without HP, just for illustration purposes

However, quite often, gingival barriers are positioned inappropriately or are moved during the procedure, allowing direct contact of highly concentrated and thus toxic peroxides with the adjacent gingival tissue. When such accidents occur, the mucosa becomes temporarily whitened and is likely to return to clinical normality after the application of a neutralizing agent and rehydration. These effects may be observed in Figs. 4.4 and 4.5.

Figure 4.6 depicts a clinical case in which the gingival papillae retracted and became erythematous after the at-home part of a "jump start" technique procedure (described in Chap. 6). During prolonged contact with the oral mucosa, significant epithelial alteration associated with acute inflammation of the underlying connective tissue may occur. These pathological changes generated by incorrectly performing the bleaching procedure may cause discomfort of the patient. The severity of the damage to the buccal mucosa can be directly related to the

Fig. 4.5 (**a**) Mandibular incisors submitted to in-office bleaching treatment with 38% HP. (**b**) Either the application of the gingival barrier in an excessively humid operative field or the extended time that the gel was in contact with the teeth may have caused alterations in the gel's thixotropic characteristics and lead to a leakage of gel, causing extensive damage to the soft tissue. (**c**) Application of neutralizing product based on sodium bicarbonate. (**d**) Clinical aspect 45 min after the incident

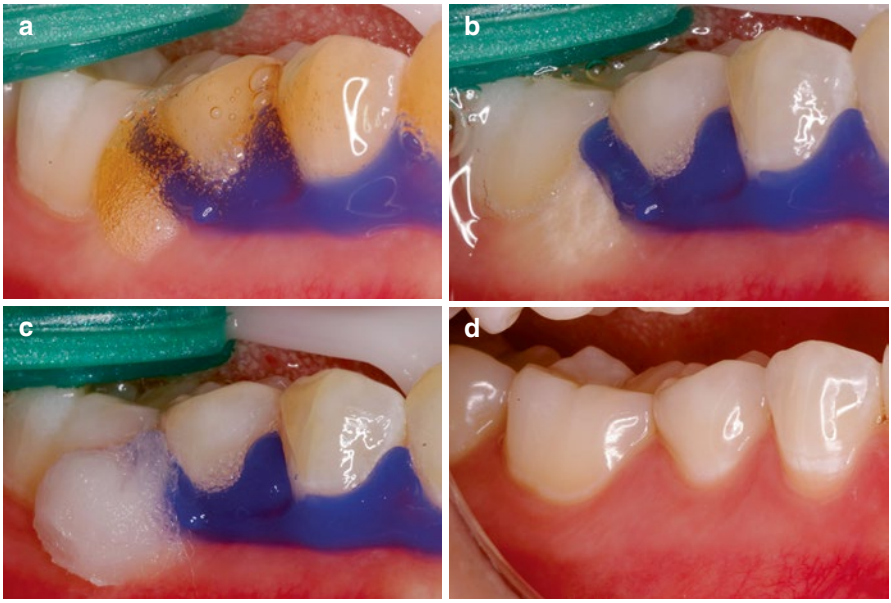
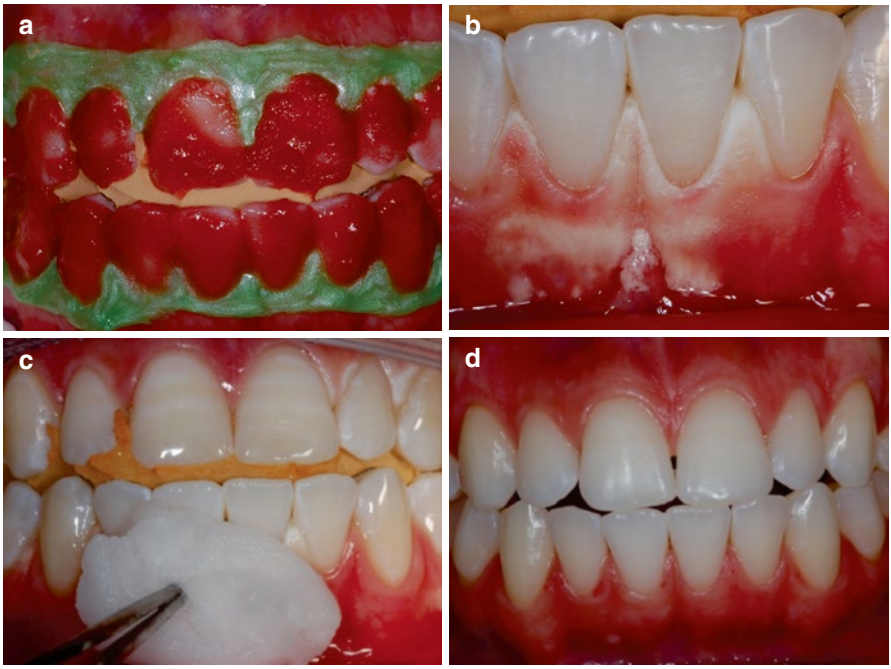


Fig. 4.4 (**a**) Seepage of 35 % HP bleaching gel to the region that is not protected with the gingival barrier. (**b**) Clinical characteristic of the gingival tissue immediately after contact with the bleaching gel. (**c**) Application of a 10 % sodium bicarbonate neutralizing agent. (**d**) Clinical characteristic of the gingival tissue 7 days after the incident



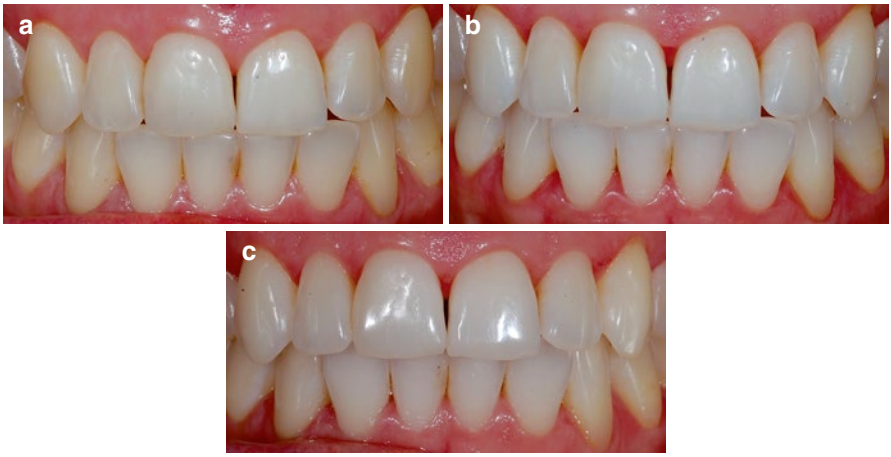


Fig. 4.6 Effect of 10 % CP on the gingival papilla region after the at-home component of a "jump start" technique procedure. An allergic reaction to a component of the bleaching product may have occurred since no tray compression was observed in the papilla region. (**a**) Pretreatment view. (**b**) After removing the bleaching agent and gingival barrier, retraction in the gingival papilla associated with an erythematous surface is observed in the region between teeth #6 (13) and #9 (21). (**c**) Clinical aspect after 7 days

concentration of HP present and/or released by the bleaching product, its pH, and the time of contact of the gel with the tissue. In an ongoing in vivo study by our research group, the oral mucosa of rats is exposed to the application of different bleaching gels for 30 min. Then, biopsy samples of damaged tissues treated or untreated with a neutralizing agent (such as sodium bicarbonate) are obtained and processed for microscopic analysis of tissue response. Preliminary analysis of histological sections stained with hematoxylin and eosin revealed that the extent of tissue changes varied according to the bleaching product and that treating the damaged mucosa with a neutralizing agent reduces the extent of damage caused by the bleaching gels, particularly those with HP concentrations greater than 15 % (Fig. 4.7).

4.3 Effect on Oral Hard Tissues

4.3.1 Change in Color of Tooth Structure

Tooth bleaching has been the first choice of treatment of intrinsic pigmentation of tooth structure (Williams et al. 1992; Perdigão 2010). The bleaching process is believed to occur via the action of the low-molecular-weight HP, which easily diffuses through the enamel and dentin, releasing reactive oxygen species that effectively promote the oxidation of the organic substrate present in the tooth structure. As a result,

dental pigmentation molecules become simpler or are eliminated. Although the traditional in-office bleaching technique (30–40 % HP, applied for 30–60 min) provides highly satisfactory cosmetic results in a short period, the biological effects are currently controversial because of the scientific evidence proving that this therapy can cause irreversible damage to the pulp-dentin complex.

The intense tooth sensitivity in patients treated with in-office bleaching causes great discomfort to patients, which has led researchers to reassess the concepts used in the last decades.

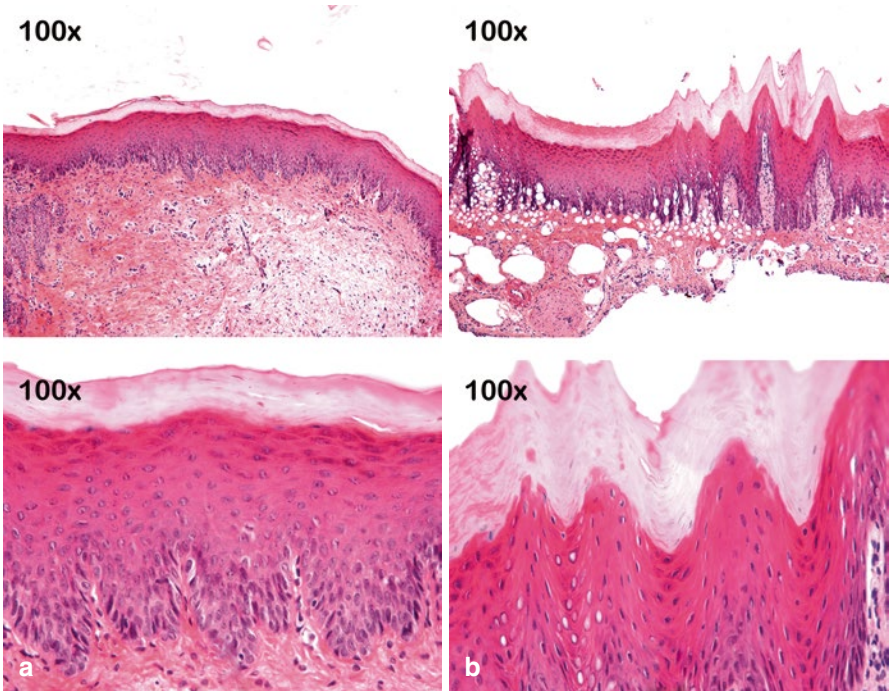


Fig. 4.7 Bleaching gels are applied to the buccal mucosa of rats for 30 min, and then the injured tissue is treated or untreated with a neutralizing agent (sodium bicarbonate). The mucosa exposed to 10 % CP gel does not show any noticeable change in the epithelium and underlying connective tissue (**a**, **d**). However, the epithelium treated with gels containing 15% (**b**, **e**) or 35 % (**c**, **f**) HP shows numerous fingerlike papillae, acanthosis, and large areas of cell vacuolation. Intense inflammation associated with cell hydropic degeneration and extensive areas of edema can be observed in the underlying connective tissue. However, these tissue changes appeared less intense when the mucosa of the animals exposed to these gels with high HP concentrations were subsequently treated with a neutralizing agent

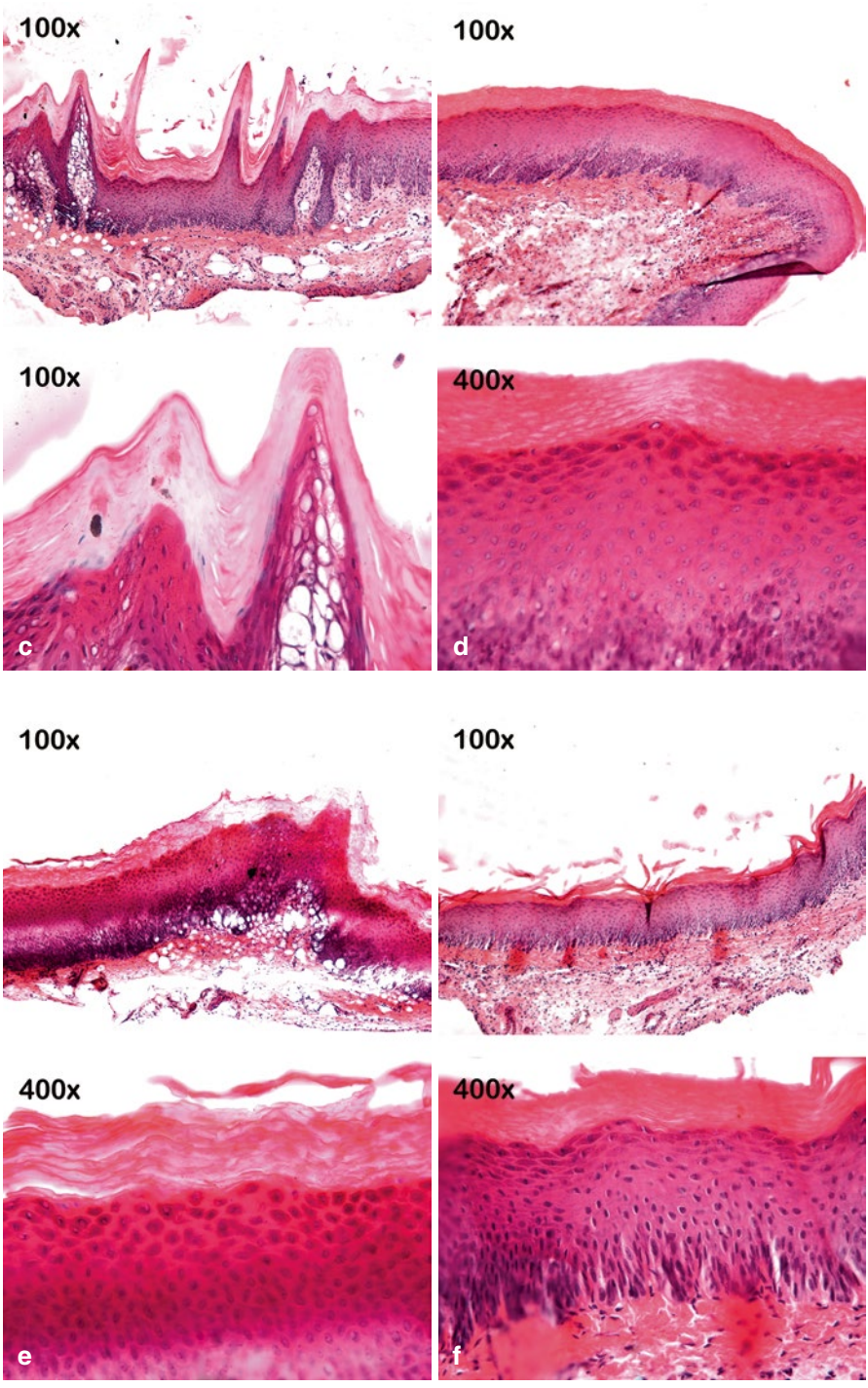


Fig. 4.7 (continued)

Our research group has evaluated some parameters for the application of at-home and in-office bleaching techniques, with the aim of finding more effective and more biocompatible bleaching techniques. These parameters include the following: (1) the need to irradiate the in-office bleaching product with a light source; (2) HP concentration in bleaching gels; (3) the contact time of the product with the dental surface; (4) the need for reapplication of the product on the tooth surface during the same clinical session; (5) the composition of the bleaching gel (CP or HP); (6) combined use of at-home and in-office bleaching techniques; and (7) acid etching of the enamel prior to bleaching.

The irradiation of in-office bleaching agents with light sources has had a strong commercial appeal in recent decades. It has been widely used in dental offices to (supposedly) accelerate the bleaching procedure, a technique known as power bleaching. The action mechanism proposed for irradiation with light is based on thermocatalysis, resulting in a twofold increase in HP decomposition with a temperature increase of 10 °C (Buchalla and Attin 2007). However, the real benefits of light-activating bleaching products remain controversial in the literature. According to in vivo and in vitro studies conducted by our group, irradiation of the 35 % HP bleaching gel with halogen lamps (20–40 s/application), and LED (60 s/application) or LED/laser sources (3 min/application), did not promote a significant increase in the bleaching effect after the first bleaching session to 1–6 months after bleaching. Patients who underwent bleaching with light irradiation reported a longer duration and greater intensity of tooth sensitivity (Almeida et al. 2012; Simões et al. 2015).

Based on these findings, the use of traditional in-office gels in combination with light sources should be eliminated from everyday practice.

In relation to the HP concentration used in the in-office technique, in vitro studies that used ultraviolet reflection spectrophotometers showed that the color change was saturated after three or four sessions when 35 % HP gels were used, with about 50–60 % of the total color change obtained after the first bleaching session (Briso et al. 2010b; Soares et al. 2014a; de Almeida et al. 2015b). By using nonstained specimens with external pigments, previous authors have observed that a 20 % HP gel resulted in the same bleaching outcome as a gel with 35 % HP; this means that about 60 % of the color change occurred after the first bleaching session, based on the similar color change pattern observed after the second and third sessions (de Almeida et al. 2015b). When specimens stained with black tea (yellow pigment) were used, a 17.5 % HP gel promoted a gradual color change in the tooth structure. While a more concentrated gel (35 % HP) caused 50 % of the color change after the first session, a 17.5 % HP gel promoted about 36.5 % color change, with the bleaching effect being intensified gradually throughout the sessions so that no difference with the traditional 35 % HP protocol was observed at the end of four sessions (Soares et al. 2014a). It is noteworthy that these results obtained for both 35 % HP

gel and for 17.5 % HP gel were measured for the same duration of contact with the tooth structure (45 min).

The advantage of using in-office bleaching materials of lower concentration is based on the fact that these products minimize HP diffusion over the tooth structure by about 60 %, which has a positive biological effect on pulp cells and confers less risk to the oral mucosa.

Our data is in agreement with the results of Sulieman et al. (2004). These authors observed that the bleaching efficacy was proportional to the concentration of the bleaching agent applied on teeth discolored with black tea. It took 2, 4, 7, and 12 applications for the gels containing 25 %, 15 %, 10 %, and 5 % HP, respectively, to reach the same bleaching effect observed after a single application of the gel containing 35 % HP. Thus, gels with reduced HP concentration can reach the same bleaching standard attained with more commonly used higher HP concentrations. However, the effects of lower HP concentrations are more gradual and depend on intensity of the stains.

Similar results were obtained for the at-home bleaching technique. After treatment, gels with 16 % and 20 % CP were observed to have the same bleaching potential as a 10 % CP gel, with the latter resulting in lower tooth sensitivity and lower incidence of soft tissue irritation (Meireles et al. 2010; Basting et al. 2012). Regarding the presentation form, we observed that home bleaching gels with 10 % CP have the same bleaching potential as gels with 6 % HP when applied using the same treatment regimen (1.5 h daily for 3 weeks). However, the HP-based gel resulted in HP diffusion into the tooth structure at about 50 % greater intensity. Furthermore, the application of the product with 10 % CP for 1.5–3 h resulted in the same aesthetic effect after 7, 14, and 21 days of treatment; and the shorter the contact time, the lower the HP trans-amelo-dentinal diffusion (de Almeida et al. 2015a). Home treatment with gels containing 10 % CP, applied for 3–4 h a day for 3 weeks in a custom-fitted tray, had the same bleaching potential as traditional in-office bleaching (Briso et al. 2010b; Almeida et al. 2012; Basting et al. 2012).

Figure 4.8 is a graph developed from data observed in our laboratory and clinical trials. The at-home bleaching technique using either 10–16 % CP or 3–7 % HP in a custom-fitted tray versus the in-office technique (20–40 % HP) most often provides similar results at the end of the third week of treatment, reaching the chromatic saturation in most of the cases within this period. We have also found that the combination of at-home and in-office techniques (jump start) provides a faster color change at the beginning of treatment, which makes this an interesting option to accelerate the aesthetic result. The association of in-office bleaching sessions with low HP concentrations (15–20 %), with daily applications of bleaching gels with 10 % CP over a short period (1.5 h daily), following the supervised home bleaching technique (scalloped custom-fitted tray with no reservoirs), presents itself as an

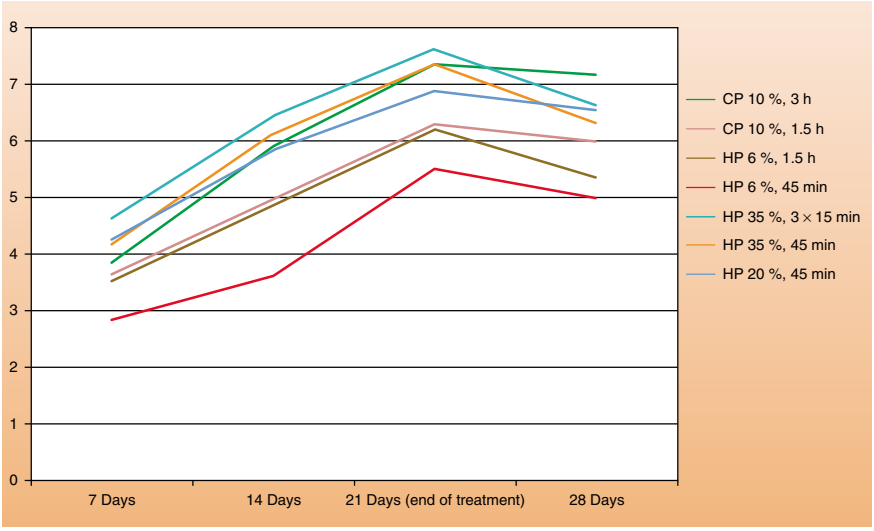


Fig. 4.8 Color change (Delta E), according to bleaching posology employed and treatment time

attractive alternative to accelerate the aesthetic result using more biologically compatible bleaching techniques. However, practitioners should be aware that the indication of the at-home technique should be based on a detailed initial clinical examination to avoid the application of the material in areas that may increase the toxic potential of this therapy to the oral tissues. These precautions will be discussed later in this chapter.

The need for successive reapplication of the bleaching product during the same clinical session has also been questioned. In a recent study conducted by our group, we observed that bleaching gels with 35–38 % HP retain about 86 % of the initial concentration of HP after 45 min of contact with the tooth structure (Marson et al. 2015).

These results demonstrate that reapplication of the bleaching product during in-office bleaching is not necessary.

In the study of de Almeida et al. (2015b), a 35 % HP gel resulted in the same bleaching outcome when applied once for 45 min or three times for 15 min each. Similarly, Soares et al. (2014a) found that the continuous application of a 17.5 % HP gel for 15 min or reapplication of the product three times for 5 min each resulted in the same aesthetic effects after six whitening sessions. The application of the in-office bleaching product on the tooth structure over reduced periods of time promotes gradual and effective bleaching when 35 % HP bleaching gels were tested (Soares et al. 2014a). However, application of gels with low HP concentrations on

the tooth structure over short periods (5–15 min) resulted in a limited lightening effect, even after six bleaching sessions (Soares et al. 2014a).

Finally, acid etching of the tooth structure prior to in-office bleaching has been indicated in order to increase the effectiveness of this clinical procedure. In an experiment conducted recently by our research group, etching the enamel with 37 % phosphoric acid for 20 s immediately prior to application of 35 % HP bleaching gel (three applications of 15 min each) did not result in a significant increase in bleaching effectiveness and did not interfere with HP diffusion over the tooth structure. Thus, as enamel acid etching with phosphoric acid changes the mineral structure of the enamel and removes hydroxyapatite without benefiting the bleaching outcome, it should not be used with in-office whitening.

4.3.2 Microabrasion and Tooth Bleaching

In some cases, stains can still be observed on the enamel after completion of the bleaching treatment. While there may be several types of stains, we found that these usually have well-defined contours and are whitish. Some of these stains can be transient and become imperceptible with the color stabilization and rehydration of the dental element after bleaching. Often, these white spots already existed but only became apparent after the color change produced by the treatment. On the other hand, yellow teeth with enamel whitish stains may be candidates for tooth whitening to have the stains attenuated.

Considering the texture or color changes of the surface layers of enamel, enamel microabrasion using acidic and/or abrasive agents has been suggested as an excellent alternative to improve the appearance of the teeth (Croll 1997) by camouflaging the white spots when they are not very deep (Chap. 9). Although some studies (Paic et al. 2008; Rodrigues et al. 2013) showed that the wear of the tooth surface after this procedure is minimal, it is necessary to consider that these changes can reach different depths. Additionally, the aprismatic enamel layer may also be affected during removal, in addition to the removal by enamel etching by hydrochloric acid in the microabrasive material. These changes may substantially alter the permeability of dental hard tissues. This issue has concerned some researchers, especially when bleaching with 35 % HP is carried out immediately after the microabrasion procedure, as the HP diffusion over the tooth structure in these conditions is about 20 % higher (Briso et al. 2014a). This increase in HP diffusion may decrease the safety of the procedure considerably.

Thus, in most cases, the microabrasive treatment is complemented with bleaching because of the yellowish color of the teeth after erosion of the enamel. However, for convenience, aesthetic rehabilitation is initiated by performing the bleaching treatment, which may be sufficient to make the intrinsic enamel stains partially or totally imperceptible, as previously described. If the enamel microabrasion is still deemed necessary, teeth will invariably present a more yellowish appearance due to enamel removal and consequent proximity to dentin. In these cases, a waiting period of 7 days is recommended before at-home bleaching is initiated with low-concentration bleaching products.

4.3.3 Change in Hardness/Susceptibility to Caries/ Demineralization/Importance of Saliva

The effect of bleaching agents on dental enamel has been extensively studied in the literature (Kwon et al. 2002; Spalding et al. 2003; Cavalli et al. 2004; Faraoni-Romano et al. 2008; Forner et al. 2009). Morphological changes, increased surface porosity, exposure of prisms, reduced organic content, change in the calcium/phosphate proportion, and reduced microhardness, are the main changes that occur in enamel upon bleaching. These changes depend on the contact time of the gel with the dental substrate, the CP/HP concentration in the product, and the pH of the product during its use.

Soares et al. (2013b) showed that 16 % CP gel resulted in the formation of deeper pores on the enamel surface compared to those formed with 10 % CP, along with a more pronounced loss of calcium and phosphorus. Taking into account that the only variable was the CP concentration, being all the other parameters standardized (pH of the bleaching gel, the interval between applications, and the total treatment time), the concentration of the bleaching gel was responsible for the more pronounced changes observed when a 16 % CP gel was used. Current literature also demonstrates that the use of high HP concentrations induces more pronounced alterations in the ultimate tensile strength of human enamel accompanied by changes in the enamel's internal micromorphology and some intraprismatic material loss (da Silva et al. 2005) (more information in Chap. 6).

As the pH of at-home CP bleaching gels ranges from 5.6 to 7.3 and the urea released during the degradation of CP increases the pH within 15 min, the original pH of these gels is unlikely to have any association with structural changes in the enamel, even with prolonged contact time with the tooth surface. Thus, the pores are formed on the enamel surface after bleaching as a result of the disruption of enamel protein matrix and subsequent loss of the crystalline material surrounded by this matrix. This hypothesis derives from the observation in several studies that enamel dissolution occurs heterogeneously, with areas of erosion interleaved with areas of intact enamel (Kwon et al. 2002; Spalding et al. 2003; Cavalli et al. 2004). As the distribution of proteins and other organic materials are uneven on the enamel surface, the defects observed after bleaching occur heterogeneously (Kwon et al. 2002). Other studies have demonstrated that the dissolution occurs primarily in the interprismatic regions and in the enamel hypomineralization areas, which are the regions with the greatest amount of organic material (Spalding et al. 2003; Cavalli et al. 2004).

When gels with high HP concentration were used for in-office whitening, the morphological changes on the enamel surface were significant enough to be observed even after a single application of the product on enamel, increasing the density of pits and different degrees of porosity (Kwon et al. 2002; Spalding et al. 2003). These changes may have been caused synergistically by the oxidative effect of HP and its acidic pH. Although the average pH of in-office bleaching products is around 6.5, many gels have a pH between 3.6 and 5.0 (Price et al. 2000), which are pH values below the critical pH for enamel dissolution (5.5). Recent studies have shown that the pH of bleaching product has a direct relationship with the

roughness of tooth enamel after bleaching and that the pH of bleaching agents tends to decrease with increased contact time with the tooth structure (Trentino et al. 2015; Abe et al. 2016).

Despite the various morphological changes observed on the enamel surface, studies have shown that these changes are mild to moderate. However, the contact of bleaching products with dentin can cause more severe changes. Reduction in wear resistance (Faraoni-Romano et al. 2009), decreased hardness (Faraoni-Romano et al. 2008; Forner et al. 2009), and increased surface roughness (Faraoni-Romano et al. 2008) have been described to be more pronounced in enamel. These findings may be explained by the specific composition of dentin, which has a greater organic content than enamel. Additionally, dentin has increased susceptibility to the HP oxidative action and acidic pH of the bleaching gels, as the critical pH value for the dentinal dissolution is between 6.2 and 6.7 (Faraoni-Romano et al. 2009). Therefore, the contact of bleaching agents with exposed dentin areas is highly contraindicated.

As the changes in the enamel are considered subtle, it remains a challenge how to extrapolate these results to the in vivo situation, where factors such as saliva and the presence of fluoride may act to remineralize tooth structure (Kwon et al. 2002). Studies that performed bleaching in situ (Rodrigues et al. 2005; Faraoni-Romano et al. 2009) or applied human or artificial saliva to specimens in between the bleaching steps (Spalding et al. 2003; Faraoni-Romano et al. 2008; Sasaki et al. 2009) showed insignificant changes in the enamel, which is attributable to the remineralizing action of saliva. Sasaki et al. (2009) also demonstrated that the storage in artificial saliva of specimens bleached for 14 days resulted in a significant increase in microhardness. In their study, Spalding et al. (2003) observed under scanning electron microscopy that bleaching with 35 % HP followed by immersion in human saliva for 1 week resulted in the formation of a granular blanket on the enamel surface, which was probably due to remineralization process by saliva. Soares et al. (2013a) observed that the use of solutions with 0.2 and 0.05 % sodium fluoride for 1 min after each application of the bleaching gel prevented the structural changes observed in the enamel when gels containing 10 and 16 % CP were used. Kemaloğlu et al. (2014) also demonstrated that fluoridated solutions (2.1 % sodium fluoride) significantly prevent mineral loss in enamel subjected to 38 % HP bleaching gels.

Although these changes tend to reverse when in contact with saliva and fluoride, the use of peroxides in demineralized areas may worsen existing conditions. In this context, during clinical examination, the practitioner must pay attention to the presence of incipient carious lesions that may have become wider due to the bleaching treatment. In a recent study conducted by our group, we found that the application of a 35 % HP gel (three times for 15 min) on specimens with demineralized enamel to simulate incipient lesion caries resulted in a more intense HP diffusion over the tooth structure than that observed in healthy and bleached specimens. In the same study, we found a greater reducing effect on enamel microhardness when demineralized specimens were bleached, wherein the bleaching increased the depth of demineralization of incipient caries lesions. The surface and subsurface morphology were also more heavily affected in the previously demineralized enamel subjected to bleaching (Briso et al. 2015).

Thus, at the end of the bleaching treatment, the presence of saliva and use of fluorides to promote mineral saturation in the tooth structure are important to promote a reduction in the demineralization process and an increase in the remineralization of tooth structures. Prior to tooth bleaching, the dental professional should perform a careful examination in order to detect the presence of exposed dentin areas, enamel hypomineralization, and incipient carious lesions, considering that application of bleaching gel is contraindicated in these regions.

4.3.4 Effects on Restorations

Patients who undergo bleaching treatment may have various types of restorations. Successful tooth bleaching relies on the direct contact of the bleaching gel with the teeth and, hence, with the restorations, which may affect the characteristics of the restorative material (Türker and Biskin 2003). The main changes are related to the surface roughness (Türker and Biskin 2002, 2003), microhardness (Türker and Biskin 2002), color (Gurbuz et al. 2013), and the marginal integrity of the restorations (Ulukapi et al. 2003).

4.3.4.1 Roughness

Surface roughness is an important characteristic of restorative materials. Adequate surface polishing of restorations results in lower risk of plaque retention and better aesthetics, which ultimately increase the longevity of restorations (Steinberg et al. 1999). The effect of bleaching agents on the roughness of restorative materials is controversial in the literature. Slight changes in surface roughness of resin hybrid materials after in-office bleaching (Hayacibara et al. 2004) and formation of microscopic cracks on the surface of the composite (Mourouzis et al. 2013) have been reported, as well as minimal effects on dental amalgam, composite resin, glass ionomer, and porcelain exposed to bleaching products (de A Silva et al. 2006).

In any case, new polishing should be considered in restorations subjected to bleaching treatment, as no matter how mild, roughening of the restorative materials might occur. Research studies that use the same bleaching products and methodologies are rare, making a direct comparison of results impossible. In actual clinical practice, restorations are simultaneously subjected to the formation of biofilm, tooth brushing, and mastication, besides the chemical challenges in the oral cavity – conditions that are hardly simulated in laboratory studies. Meanwhile, saliva could dilute the bleaching gel, often reducing its concentration and its effect on the surface of the restorative materials (Wattanapayungkul et al. 1999; Steinberg et al. 1999; de A Silva et al. 2006).

The roughness of indirect restorative materials, such as fiber-reinforced composites and porcelain, increases after exposure to bleaching agents.

The Bis-GMA and UDMA matrix of indirect resins is greatly affected by the action of bleaching products, causing the erosion of the resin matrix and the consequent displacement of filler particles. In turn, porcelains may also exhibit changes in surface roughness (Türker and Biskin 2003; Schemehorn et al. 2004; Torabi et al. 2014b) that are attributed to the reduction in SiO_2 and K_2O_2 molecules (Moraes et al. 2006). These findings, however, are opposed to those of a previous study that observed polished porcelains had a higher resistance to bleaching products (Butler et al. 2004). These controversial results reported in the literature can be explained by the different methodologies and bleaching products used. While some studies use actual dosages, others subject their specimens to long periods of exposure to the bleaching product.

4.3.4.2 Hardness

The hardness of a material essentially relates to its properties, which in turn interferes with its durability (Atash and Van den Abbeele 2005; AlQahtani 2013). Reports showed reduced Vickers and Knoop hardness of resin materials when exposed to bleaching agents. Reactive oxygen species are believed to promote the cleavage of polymer chains, degrading the organic matrix that leads to the chemical softening of resin (Taher 2005; de Alexandre et al. 2006; Briso et al. 2010a; AlQahtani 2013). For the same reason, the hardness of the pit and fissure sealants subjected to bleaching with low concentrations of CP was reduced. In this case, the materials that showed the lowest microhardness values were those without filler particles because of the higher percentage of organic matrix in their composition (de Alexandre et al. 2006).

An in vitro study (Torabi et al. 2014a) also demonstrated changes in porcelain microhardness. Although these values were significant, the release of SiO_2 was not clinically observed. An important factor to be emphasized is that glazed surfaces seemed less susceptible to hardness changes, while the opposite was observed in polished surfaces (Torabi et al. 2014a). Thus, finishing porcelain surfaces prior to the bleaching treatment is recommended.

It is noteworthy that the bleaching products are highly unstable and that their pH can affect the Knoop hardness of the restorative materials. For this reason, some bleaching agents may cause more changes than others. Therefore, the selection of bleaching agents that keep the pH around 7 throughout the complete bleaching procedure is recommended (Briso et al. 2010a).

4.3.4.3 Change in Color, Brightness, and Fluorescence

These properties have great importance to the aesthetic restorations of composite resin and porcelain. The color, brightness, and fluorescence of direct and indirect restorative materials are known to undergo changes during the bleaching treatment (Canay and Cehreli 2003; Hubbezoglu et al. 2008; Li et al. 2009; Kara et al. 2013). However, the color change that occurs in the dental tissue is much more intense, making it imperative to replace the aesthetic restorations once the bleaching treatment is completed. Therefore, it is recommended to wait until the dental tissue is rehydrated and reaches color stability, which occurs approximately 7–15 days after completion of the bleaching treatment.

Significant changes in brightness and fluorescence of restorative materials were also found after bleaching, reinforcing the need to replace aesthetic restorations as part of treatment plans (Yalcin and Gurgan 2005; Gurbuz et al. 2013; Klukowska et al. 2013; Bueno et al. 2013). In porcelain subjected to low-concentration bleaching agents, these changes were observed and attributed to the type and structure of the crystals present in the porcelain studied. Composite resins for indirect restorations are more susceptible to color changes during bleaching than ceramics, showing less color stability in chemical challenges (Kara et al. 2013). We must, however, take into account that none of these studies were conducted in a situation identical to the oral environment, where the presence of saliva could change the outcome.

4.3.4.4 Microleakage and Effects on Bond Strength

Currently, restorative techniques are based on the adhesive bonding of resin materials to tooth structure. Some studies showed changes in the marginal sealing of restorations subjected to bleaching treatment (Owens et al. 1998), causing a decrease in the bond strength (Cavalli et al. 2005).

Moreover, class V restorations subjected to bleaching treatment have been reported to present the greatest changes in the adhesive interface with dentin, making these regions more prone to the occurrence of microleakage (Bektas et al. 2013). The difference between the substrates suggests that the deleterious action of peroxides is more pronounced in tissues with higher organic content (Carrasco-Guerisoli et al. 2009). This fact was also confirmed by White et al. (2008), who showed that the occurrence of microleakage at cavity margins of class I restorations was not influenced by treatment with different bleaching products.

Taking into account the findings reported in the recent literature, a thorough evaluation of preexisting restorations is crucial, and, in the case of defective margins, protection of tooth-restoration interface with materials such as pit and fissure sealants or adhesive systems is recommended.

As mentioned previously, the restorations of teeth with great aesthetic involvement should be replaced after bleaching. In such cases, residual oxygen from the decomposition of bleaching agents may be present within the dental tissues, negatively interfering with the interaction of the adhesive system with the tooth structure as well as the degree of conversion of restorative materials (Cavalli et al. 2005; Briso et al. 2014b). This requires an interval of 7–15 days between the end of the bleaching treatment and the replacement of the restorations to eliminate all the excess oxygen (Briso et al. 2014b).

Previous studies suggested the use of antioxidants in order to reduce this interval, aiming to counteract the negative effects of the presence of oxygen in the tooth structure (Freire et al. 2009; Garcia et al. 2012; Briso et al. 2014b; Arumugam et al. 2014). Although many antioxidants have been studied, such as lycopene, proanthocyanidin, and α-tocopherol (Arumugam et al. 2014), sodium ascorbate at the concentration of 10 % is the most widely studied (Briso et al. 2014b). Its application is recommended for 5–10 min prior to performing the restorative procedures (Freire et al. 2009; Briso et al. 2012, 2014b). Its use has been associated to a significant

improvement in the marginal sealing of the restorations performed, increased bond strength, and the micromechanical interactions that occur between the adhesive system and the tooth substrate (Abraham et al. 2013).

The clinician must exert some caution when performing restorations immediately after the bleaching treatment, as at this time the teeth can appear dehydrated and with unstable color, but a color rebound may occur after a few weeks.

4.4 Tooth Sensitivity

4.4.1 Symptoms

Reactive oxygen species from bleaching products quickly reach the pulp-dentin complex, triggering a series of biological reactions that may change the pulp condition, causing pain to patients (Markowitz 2010). Tooth sensitivity is the most frequent clinically detectable side effect of the bleaching treatment, and its occurrence raises concerns for practitioners and causes disturbance to patients, causing them to drop out of the treatment (Rahal et al. 2014).

The penetration of HP in dental pulp results in the release of biochemical mediators involved in the inflammatory process, which sensitize the pulp nociceptors and play a role in pain modulation by causing an increase in vascular permeability and vasodilation, changing the sensitivity threshold of nerve fibers (Markowitz 2010). Moreover, when the peroxide from bleaching agents comes in contact with MDPC-23 odontoblast cells during in-office dental bleaching, significant changes in their morphology may occur and the mitochondrial respiration rate decreases (Costa et al. 2010; Soares et al. 2014b). Despite the obvious differences between the experimental models, tests conducted in Wistar rats also confirm the aggressive potential of the in-office bleaching treatment. Such damages were proportional to the intensity of the therapy used (Cintra et al. 2013). In turn, a recent study on human teeth showed that excessive exposure to peroxides may lead to a slight disturbance in the odontoblast layer on premolars and coagulation necrosis areas in lower incisors subjected to the in-office bleaching treatment (Costa et al. 2010).

All of these occurrences have encouraged authors to carry out studies with the aim to minimize these side effects (Giniger et al. 2005; Armênio et al. 2008; Tay et al. 2009). Among clinical analyses, neurosensory investigations through quantitative sensory tests have led to the conclusion that patients who undergo bleaching treatments experience different levels of discomfort, a fact observed when the painful tooth sensitivity threshold was altered (Rahal et al. 2014).

In clinical practice, the methods used to minimize patient discomfort rely on the administration of analgesics and/or the use of topical desensitizing agents,

which may be added into the composition of bleaching agents (Jorgensen and Carroll 2002; Croll 2003; Giniger et al. 2005; Haywood 2005; Armênio et al. 2008; Tay et al. 2009; Basting et al. 2012). Several types of desensitizing agents with different action mechanisms have been used, some having physical action that seals the dentinal tubules and others having neural action that blocks nerve stimulation (Tay et al. 2009; Basting et al. 2012; Palé et al. 2014).

In fact, some desensitizers are effective and reduce the discomfort caused by the bleaching treatment. A recent report (Rahal et al. 2014) showed a reduction in the neurosensorial response of teeth treated with a desensitizer after bleaching. In this split-mouth design study, the bleaching treatment was performed in the upper arch. Only one hemiarch received the topical desensitizer containing 5 % potassium nitrate and 2 % sodium fluoride. The results obtained after the use of the desensitizer showed a clear reduction in sensitivity.

Despite all these studies on tooth sensitivity, products or techniques that restrict the action of peroxides on dental pigments or that effectively modulate the penetration of peroxides in the pulp tissue have not yet been developed. Dentists have very limited information on the potential for tooth sensitivity when considering tooth bleaching as a therapy in which a HP-based drug is topically applied to tooth enamel, thus causing undesirable side effects. Therefore, a patient-specific individualized treatment is indispensable for delivering a controlled peroxide dosage in function of each individual patient's conditions.

In case of teenage patients who have wide pulp chambers, they should be treated in a highly conservative manner, using low-concentration bleaching materials and restricting their use to a few hours per day or simply discontinue the use for a few days at a time.

In general, special attention should be given to the dosage used. Sometimes, the practitioner may intuitively consider that the greater the amount of peroxide that penetrates into dentin, the greater the color changes obtained. However, all bleaching therapies (at-home and in-office) may provide similar results, taking an average of 3 weeks for chromatic saturation (Bernardon et al. 2010). In the case of the in-office technique with 35 % HP, the 30-min application time has been proven to provide the same results as the 45-min exposure to bleaching. Additionally, the continuous renewal of the bleaching product every 15 min has proven to be unnecessary to achieve bleaching results, as the product retains its activity throughout the clinical session (Marson et al. 2015). While the dosage most often adopted for in-office bleaching (35 % HP from 45 to 60 min with multiple changes) denotes great effectiveness in promoting peroxide penetration into the pulp tissue, it does not result in effective bleaching of the teeth (Costa et al. 2010; Soares et al. 2014b).

Another factor to be considered is the condition of the oral environment, which should also be carefully analyzed. Besides recommending various bleaching techniques, the practitioner must pay attention to the alternative routes of peroxide diffusion, which can be neglected by the operator, greatly favoring the penetration of the peroxide into the pulp chamber.

4.4.2 Protection Protocols

The treatment options currently adopted by clinicians can result in all the unwanted biological effects previously mentioned. This is especially important when no concern is given to the suitability of the oral cavity for receiving a HP-based product and when dosage is not adjusted for each type of patient. Thus, in addition to the care in the indication of a suitable dosage for the patient, we must highlight some specific conditions in which the penetration of peroxide into the pulp tissue is increased, thereby causing great possibility of undesirable side effects to patients.

4.4.2.1 Incipient Carious Lesions

Owing to the difficulty of diagnosis or negligence, incipient carious lesions often do not receive adequate attention prior to tooth bleaching. Several studies showed that both at-home and in-office bleaching treatment provides transitional histological changes in the enamel (Akal et al. 2001; Bistey et al. 2007). However, performing in-office bleaching treatment on enamel with incipient carious lesions accelerates the evolution of the caries lesion to deeper enamel areas (De Arruda et al. 2012; Briso et al. 2015).

The presence of demineralized areas has also been recently associated to the amount of peroxide that reaches the dentin-pulp complex. Demineralized substrate has been found to offer lower resistance to the penetration of HP (Briso et al. 2015), resulting in higher posttreatment sensitivity. Thus, bleaching is totally contraindicated for teeth presenting demineralized areas. Once the demineralized areas are detected and the contributing factors are identified, the practitioner must establish the most appropriate therapeutic approach for each specific case (Hicks et al. 1984; Hunt 1990; Willmot 2004). Usually the treatment is initiated with daily low-concentration fluoride rinses (0.05 %). Depending on the case, the practitioner may increase the fluoride dosage with weekly application of 5 % fluoride varnish or 1.23 % acidulated fluoride-phosphate gel (Pinto 2001) in the weeks prior to the bleaching treatment until the remineralization of the region is observed.

The application time varies according to the product used, the first being applied to the tooth surface for 4 min according to the American Dental Association Council on Scientific Affairs (Pinto 2001; Braxton et al. 2014), while the varnish is maintained on the treated surface for 24 h. After remineralization of lesions is detected, the bleaching treatment can be performed. In cases where the stains show again signs of activity during the bleaching treatment, discontinuation of treatment and remineralization of new lesions are recommended.

4.4.2.2 Presence of Cracks in the Enamel

The presence of cracks in the enamel is common in buccal surfaces, though rarely valued by practitioners. These changes appear as fissures within the enamel, extending mainly to the cervical-incisal axis, even reaching the dentin-enamel junction and causing fractures in the tooth structure (Abbott and Leow 2009).

These defects on the enamel surface represent a rapid penetration route of the peroxides used in the in-office bleaching treatment (Briso et al. 2014a) and may be harmful to pulp health and increase transoperative and postoperative pain. Thus, regardless of the bleaching technique used, sealing these cracks with adhesive materials is recommended.

4.4.2.3 Exposed Dentin Areas

Exposed dentin areas, especially in the cervical and incisal regions, are common in the oral cavity.

> Exposed dentin facilitates the diffusion of peroxide, causing sensitivity during the course of treatment. For this reason, it is essential to protect these areas with appropriate resin materials.

In the cervical region, two distinct conditions determine the type of treatment, namely the presence and absence of cavitation. When cavitation is observed, this area should be protected with resin-modified glass ionomer, with the possibility of leaving the ionomer material underneath the final composite restorations with composite resin after the bleaching treatment.

On the other hand, when the cervical dentin tissue is exposed without cavitation, the insertion of the restorative material will invariably result in an anatomical overcontour in the region. For this reason, we indicate the application of an adhesive system as a means of sealing the dentinal tubules in the region. The material of choice for these cases is a self-etching adhesive, as its acidic monomers slightly etch dentin, followed by immediate penetration of the fluid resin, while minimizing the incidence of etching in areas not protected by the adhesive (Yousaf et al. 2014). However, because of its high solubility in the oral environment, in case of longer bleaching treatments, the sensitivity may relapse. For this reason, reapplications of the adhesive are often necessary (Baracco et al. 2012). Similarly, exposed dentin in incisors, especially lower incisors and canines, is common. These regions must also be protected before the bleaching treatment, although in this case, a pit and fissure sealant is recommended (Fig. 4.9).

4.4.2.4 Presence of Restorations with Marginal Gaps

The presence of restorations on teeth that will receive bleaching treatment should be carefully evaluated, as the presence of fractures and marginal gaps represent places of easy diffusion of peroxides. These areas should be protected by sealing the tooth/restoration interface to reduce or prevent the penetration of the bleaching material into the dental tissue (Patri et al. 2013).

Although no material has the ability to hermetically seal the tooth/restoration interface (Gokay et al. 2000; De Munck et al. 2005; Cenci et al. 2008), the movement of dentinal fluid toward the tooth surface (Vongsavan and Matthews 1991), associated with the use of sealing materials, is believed to be a deciding factor to implement

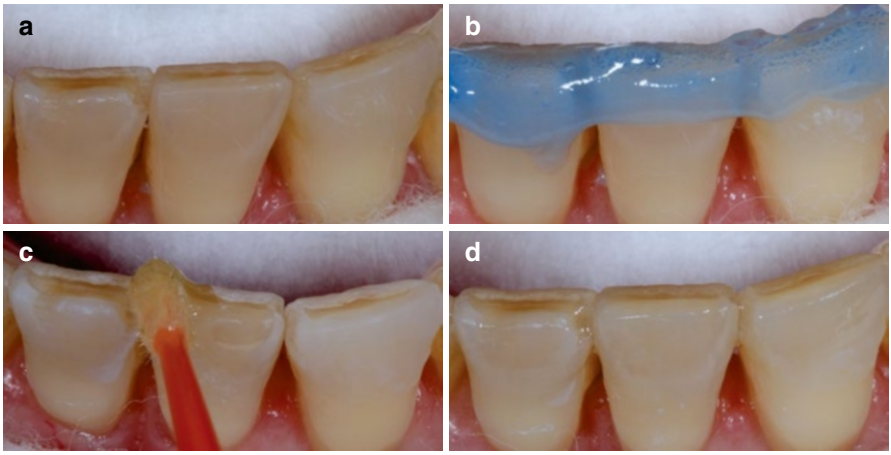


Fig. 4.9 (**a**) Incisal region with dentin exposure. (**b**) Acid etch with 35 % phosphoric acid (total-etch technique). (**c**) Application of dentin/enamel adhesive. (**d**) Exposed dentin area is sealed and teeth are ready to undergo bleaching treatment

an adequate protective measure for dental tissues. The adhesive systems and the pit and fissure sealant are materials of choice for marginal sealing of these restorations because they have good penetration and flow, forming an effective physical action in these regions, at least during the course of the bleaching treatment.

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Human Pulpal Responses to Peroxides

5

Diana Gabriela Soares, Josimeri Hebling, and Carlos Alberto de Souza Costa

Abstract

It is known that most patients subjected to professional tooth bleaching report posttreatment hypersensitivity that varies from slight to intolerable. The pathway for tooth bleaching-induced sensitivity has been correlated with the capability of hydrogen peroxide (H_2O_2), the main active component of bleaching gels, to diffuse through enamel and dentin to reach the pulp tissue. Since H_2O_2 is a toxic reactive oxygen species with a high oxidative power, it is expected that the contact of this molecule with the pulp tissue promotes oxidative cells damage leading to local connective inflammation that trigger nociceptive stimulus. However, as this clinical symptom is transient, discussion about how relevant is this adverse collateral effect to the pulp-dentin complex remains. Then, in this chapter, the authors describe some relevant clinical and laboratorial data currently provided by a number of in vitro and in vivo studies in which traditional in-office (professional) and at-home bleaching therapies were tested, as well as discuss alternative tooth bleaching protocols that may prevent or at least minimize the negative effects of these esthetic treatments to the pulp tissue vitality.

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5.1 Introduction

Despite the well-known role played by hydrogen peroxide (H_2O_2) on oxidative cells stress and inflammatory tissue reactions induction, this molecule has been widely used as the main active principle for bleaching therapies performed in vital teeth. The results of randomized clinical trials for a wide array of bleaching products have shown that a number of patients undergoing H_2O_2-based bleaching therapies complain about tooth sensitivity (Reis et al. 2011, 2013; He et al. 2012; Tay et al. 2012; Santana et al. 2014; de Paula et al. 2015).

Overall, to be regarded as an esthetically effective therapy, the H_2O_2 present in the bleaching gels has to diffuse through enamel to reach dentin substrate in which the intrinsic pigments are mainly located. This effect is possible since H_2O_2 features high stability in comparison to other reactive oxygen species (ROS) due to its low oxidation potential. After reaching dentin, the H_2O_2 molecule must give rise to other free radicals with higher oxidation potential and capable of promoting effective chromogen decomposition. However, dentin is a highly permeable tubular substrate that provides an easy pathway for inward H_2O_2 diffusion to reach the pulp chamber. Indeed, several laboratory studies (Gökay et al. 2000, 2004, 2005; Camargo et al. 2007; Ubaldini et al. 2013) have demonstrated that high concentrations of residual H_2O_2 remain undissociated within hard tooth structures, and these nonreacted molecules are able to contact pulp cells. According to these studies, the higher the concentration of H_2O_2 on the bleaching gel and the contact time with tooth structure, the higher the detection of residual H_2O_2 inside pulp chamber.

The presence of high concentrations of H_2O_2 in the extracellular environment is considered a dangerous condition, since this molecule can diffuse through cellular membranes. Once in the cell cytoplasm, H_2O_2 may be dissociated into several toxic free radicals, leading to an oxidative stress condition, which is correlated with a plethora of cellular alterations, ultimately with cell death. The sensitivity of cells to undergo oxidative stress during periods of high ROS exposure appears to be cell type specific (Ceçarini et al. 2007). It has been shown that human dental pulp cells feature a high sensitivity to the oxidative stress mediated by H_2O_2 in vitro. Therefore, the same amount of H_2O_2 capable of causing complete depletion on human dental pulp cells viability has no significant toxic effect on primary culture of human gingival fibroblasts and other cell lineages (Zhu et al. 2012). Consequently, to understand the biological effects of residual H_2O_2 released from bleaching gels on pulp tissue, our research group has carried out innovative laboratory studies using artificial pulp chambers, as well as in vivo histopathological investigations in human teeth. An overview of the scientific and clinical data obtained in such studies is presented in this chapter.

5.2 In-Office Bleaching Mediating Toxicity on Dental Pulp Cells

About 86–90 % of patients subjected to professional in-office bleaching therapy (i.e., 30–40 % H_2O_2 bleaching gels applied onto dental structure for 30–60 min at each bleaching session) report posttreatment tooth sensitivity (Tay et al. 2012;

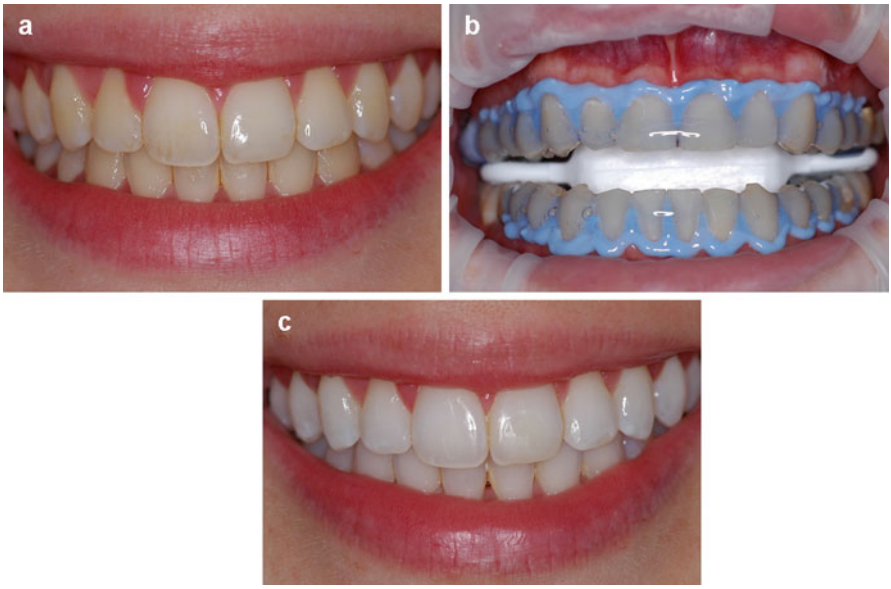

Fig. 5.1 Patient subjected to a professional in-office tooth bleaching therapy, which can be esthetically regarded as a success. However, the patient reported postbleaching sensitivity in the anterior teeth (Images provided by Dr. Heraldo Riehl) (**a**) Clinical condition before bleaching therapy, (**b**) In-office bleaching therapy with a 35% H_2O_2 gel, (**c**) Post-bleaching clinical condition.

Santana et al. 2014), with intensity varying from moderate to severe in around 60 % of cases (Fig. 5.1) (Tay et al. 2012). The prevalence of tooth sensitivity may reach 100 % when traditional in-office therapy is associated with light sources (Reis et al. 2011).

Recent clinical studies have demonstrated that the prevalence and intensity of tooth sensitivity is restricted to anterior teeth and proportional to the tooth size, with incisors being the most susceptible teeth (de Almeida et al. 2012; Bonafé et al. 2013). Therefore, the toxic potential of traditional in-office therapy on pulp cells has been the focus of a number of preliminary studies carried out by our research group. When a 35 % H_2O_2 gel was applied for three consecutive periods of 15 min onto 3.5-mm thick enamel/dentin discs (simulating upper central incisors) adapted to artificial pulp chambers (APC), gel components that diffused through dental structure (extract) caused toxic effects to pulp cells. In such laboratorial studies the extracts were applied to the cells for 1 or 24 h. After a 1-h contact time, the odontoblast-like cells, an immortalized cell lineage from rat dental papillae, featured 50–60 % of cell viability reduction (Soares et al. 2013a, b; Duque et al. 2014; de Almeida et al. 2015; Soares et al. 2015a). Almost 100 % of cell viability reduction was observed after 24-h exposure of pulp cells to these extracts (Coldebella et al. 2009; Trindade et al. 2009). Cell morphology was completely disturbed, with cells showing apoptotic body-like structures, as shown on Fig. 5.2. Human dental pulp cells exposed to the bleaching gel extracts were even more vulnerable, corroborating data previously reported in the literature. Around 97 % of cell viability reduction was observed in human pulp cells after only 1-h contact time. The

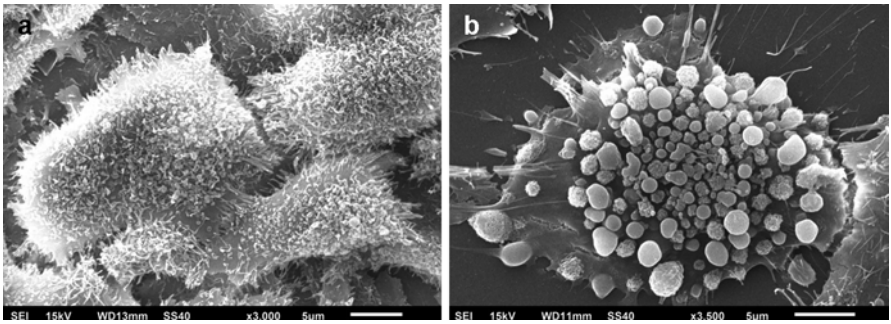

Fig. 5.2 (**a**) Scanning electron micrograph showing the morphology of normal odontoblast-like MDPC-23 cells. Original magnification = ×3,000. (**b**) Scanning electron micrograph showing a MDPC-23 cell exposed for 1 h to the extract obtained after simulated traditional in-office bleaching therapy. Original magnification = ×3,500

intensity of the negative effects caused by the 35 % H_2O_2 bleaching gel on pulp cells was proportional to their contact time with dental structure, which was directly related to the amount of H_2O_2 capable of diffusing through enamel/dentin discs (Soares et al. 2013a, b, 2015a; Duque et al. 2014). Also, association of this in-office bleaching technique with light sources increased significantly the in vitro cytotoxicity (Dias Ribeiro et al. 2009), since this procedure leads to more intense H_2O_2 diffusion through tooth structure, as previously demonstrated (Camargo et al. 2009).

According to our data, pulp cell viability reduction after tooth bleaching seems to be associated with cell membrane rupture and oxidative stress generation, in a time/concentration dependent fashion. In fact, these effects are proportional to the H_2O_2 dosage capable of reaching the cells, with high concentrations leading to cell death by necrosis following two pathways: (1) direct contact with free radicals from extracellular H_2O_2 decomposition, leading to cell membrane rupture; (2) the onset of a pathologic oxidative stress condition after H_2O_2 diffusion through cell membrane and further decomposition into free radicals on cytoplasm, culminating with lipid peroxidation (Soares et al. 2014a, 2015c).

Since the amount of H_2O_2 capable of reaching cells after trans-enamel and transdentinal diffusion is the main pathway for bleaching-induced cell toxicity, reducing this phenomenon is highly required for minimizing the oxidative damage on pulp cells, turning bleaching into a safe procedure compatible with pulp health. More recent data collected by our research group determined that decreasing the H_2O_2 concentration in the bleaching gel by 50 %, and applying the gel from 5 to 45 min on enamel, may decrease 11.3- to 4.5-fold, respectively, the toxicity of in-office tooth bleaching to pulp cells compared with simulated traditional therapy (35 %-H_2O_2 3 × 15 min) (Soares et al. 2014a). These alternative protocols using a 17.5 % H_2O_2 gel allowed such cells to overcome the low initial oxidative damage and feature a regenerative phenotype through time (Soares et al. 2014a, 2015b).

According to our data, the degree of disturbance on the expression of odontoblastic markers (ALP, DSPP, DMP-1, and mineralized nodule deposition) after tooth bleaching, was related to the oxidative stress intensity and to the overexpression of

IL-1β, TNF-α, IL-6, and COX-2 on remaining cells (Soares et al. 2015b, 2016a). It has been demonstrated that both oxidative stress intensity and pro-inflammatory mediator dosage have direct relationship on pulp tissue regeneration capability. Exposure of human dental pulp cells to high doses of pro-inflammatory mediators, such as TNF-α and IL1-β, may negatively interfere with their odontoblastic markers expression and mineralization rate, whereas low dosages seem to have an opposite effect (Min et al. 2006; Paula-Silva et al. 2009; Alongi et al. 2010). Nevertheless, it was demonstrated that human dental pulp cells under intense oxidative stress triggered by long-term exposure to H_2O_2 had the expression of DSPP and DMP-1 mRNA downregulated associated with no mineralized matrix deposition. Conversely, the treatment of these cells with nontoxic concentrations of H_2O_2 enhanced ALP activity and calcified nodule deposition as well as increased DSPP, OPN, and OCN expression (Lee et al. 2006, 2013; Min et al. 2008; Matsui et al. 2009).

Therefore, the lower the concentration of H_2O_2 in contact with pulp cells after tooth bleaching, the higher the regenerative capability of human pulp tissue to overcome the oxidative damage.

With this in mind, future studies should focus on strategies for reducing the amount of residual H_2O_2 and other free radicals capable of diffusing through the dental structure to reach the pulp chamber, since these toxic molecules play a central role on pulp tissue damage.

Recent data obtained in our lab demonstrated that bleaching gels containing 8–10 % H_2O_2 minimize significantly the initial toxicity on human dental pulp cells (Soares et al. 2015a). Also, we observed that these bleaching gels are capable of promoting the same color alteration on thin enamel/dentin discs as high concentrated gels applied on thick discs (unpublished data). Therefore, tailoring the bleaching regimen to the tooth size appears to be an interesting alternative to achieve an effective, safe, and biocompatible in-office bleaching technique.

Therefore, tailoring the bleaching regimen to the tooth size appears to be an interesting alternative to achieve an effective, safe, and biocompatible in-office bleaching technique.

5.3 Effects of At-Home Bleaching on Pulp Cells In Vitro

The at-home bleaching therapy with 10 % carbamide peroxide (CP) agents has been considered the safest method for tooth bleaching since minimal clinical adverse effects have been reported in literature (Boushell et al. 2012). The scientific data obtained in recent years by our research group has demonstrated that the application of 10 % CP gel from 1.5 to 8 h onto dental structure does not result in significant

toxic effects on both odontoblast-like cells and human dental pulp cells (Soares et al. 2011; Lima et al. 2013; Duque et al. 2014; Almeida et al. 2015). According to our data, application of 10 % CP gel for 4 h onto 3.5 mm enamel/dentin discs results in 16 times less intense H_2O_2 diffusion in comparison to the traditional in-office bleaching protocol, i.e., 35 % H_2O_2 gel 3 × 15 min (Duque et al. 2014). Nevertheless, higher CP concentrations (15–22 %) are available for at-home tooth bleaching, with the appeal of promoting a faster bleaching outcome.

Data collected from our group showed that increasing CP concentration from 10 to 16 % increases the toxicity of this treatment to pulp cells (Soares et al. 2011). Taking into consideration that clinical studies have demonstrated that higher concentrations of CP gels cause the same bleaching outcome as that achieved by using 10 % CP gel (Matis 2003; Meireles et al. 2010, Basting et al. 2012), the use of higher concentration products has no advantage and may be more toxic to pulp tissue.

H_2O_2-based at-home bleaching products are also available. We have observed that the application of 6 % H_2O_2 gels from 45 min to 1.5 h have no toxic effect on odontoblast-like cells, instead the amount of H_2O_2 capable of diffusing through dental structure and interact with the cells was twice as high as that of the 10 % CP gel (Almeida et al. 2015). Also, a 10 % H_2O_2 whitening strip (WS) applied for 1 h on tooth structure did not cause trans-enamel and trans-dentinal cytotoxicity to this same pulp cell lineage. The WS resulted in about 13 times lower H_2O_2 diffusion than that observed for traditional in-office bleaching therapy with the 35 % H_2O_2 gel (Soares et al. 2013b). Despite the interesting results obtained with WS, this treatment is currently carried out without direct professional supervision. Therefore, there is a risk of overuse associated with this over-the-counter at-home bleaching technique, which, in turn, may cause higher toxic effects to pulp cells. According to our data, the toxic effect of the WS and CP was time dependent; the toxicity of these products increases significantly after 1-h daily application during 5 consecutive days (Lima et al. 2013; Soares et al. 2013b).

The main concern regarding at-home bleaching relies on the fact that it is a patient-applied therapy; therefore, there is a risk of gel application on exposed dentin areas, craze lines, and restoration interfaces that may increase H_2O_2 diffusion through hard tooth structures. Also, the inappropriate use of the tray may result in gel overflow, with extended soft-tissue exposure and likely material ingestion. Therefore, it is imperative that this treatment is performed under professional supervision. Also, the professional should perform a detailed clinical evaluation before prescribing this therapy.

5.4 Histopathological Analysis of Human Dental Pulp After In-Office Bleaching

Based on data collected in in vitro studies, it is believed that the presence of high concentrations of H_2O_2 within the pulp chamber is responsible for the intense oxidative stress condition that causes cell membrane disruption associated with pulp cell

death by necrosis (Soares et al. 2014a, 2015b, c, 2016a). This kind of cytotoxic effect also promotes extensive damage on the neighboring tissue, since lysosome enzymes and other toxic substances are leached from dying cells, causing a ripple effect. Consequently, acute inflammatory reaction is expected with expression of a plethora of pro-inflammatory cytokines and chemokines, followed by expression of hyperalgesia mediators, such as prostaglandins (Cooper et al. 2010; Markowitz 2010; Pashley 2013; Cooper et al. 2014). These cellular events have been correlated with the clinical symptoms of tooth sensitivity. In order to detect these events on human pulp tissue, our group associated with other research groups around the world has conducted histopathological analysis on human teeth subjected to professional in-office bleaching therapies. In many of these studies, sound premolars and mandibular incisors scheduled to be extracted for orthodontic reasons were selected, after respective approval by Ethical Committees. Therefore, the volunteers (or legal guardians for patients below 18 years of age) received all necessary explanations including the experiment rationale, the clinical procedures to be performed and possible risks, and signed a consent form explaining the research protocol.

When a bleaching gel with high concentration of H_2O_2 (38 %) was applied for three times of 10 min each in sound mandibular incisors from young patients (age mean 16.2 years), intense pulpal damage occurred in about 75 % of samples. Pulp necrosis was observed in a wide area of coronal pulp tissue. Deposition of reactionary dentin in part of the coronal and root pulp tissue associated to mild local inflammatory response was also detected in bleached incisors (Fig. 5.3).

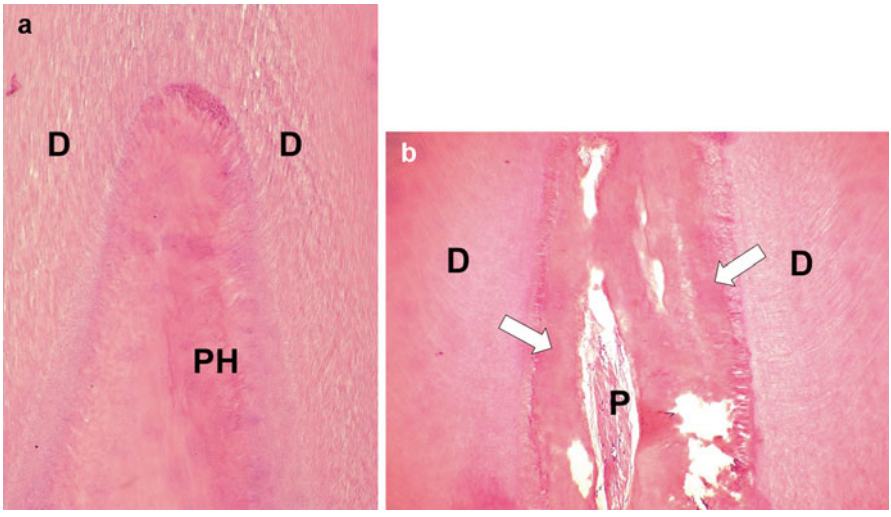

Fig. 5.3 (**a**) Pulp horn (*PH*) of a human lower incisor subjected to a professional in-office bleaching with 38 % H_2O_2. Note the large area of necrosis. H/E, ×64. (**b**) Intense deposition of tertiary dentin is observed (*arrows*) in the radicular pulp chamber, in which a small area of residual pulp (*P*) tissue with inflammation can be seen. H/E, ×64. (*D* dentin)

On the other hand, pulps of premolars subjected to the same bleaching protocol with the 38 % H_2O_2 gel showed the following histopathological features: tubular dentin and predentin, intact odontoblastic layer, and cell-free zone and cell-rich zone, such as observed in control groups (nonbleached incisors and premolars) (control teeth – Fig. 5.4).

In this experiment, the mean of dentin thickness was 1.82 ± 0.08 mm for bleached incisors and 3.10 ± 0.11 mm for bleached premolars. Only patients who had their incisors bleached reported tooth sensitivity (de Souza Costa et al. 2010). Similar results were found when sound mandibular incisors (mean age 18.2 years) were bleached with a 35 %-H_2O_2 gel applied on enamel for 3 consecutive periods of 15 min each or 1 period of 45 min. Similar pulp tissue responses were found for both tested protocols with the 35 %-H_2O_2 gel. The majority of bleached incisors (80 %) exhibited large zone of coagulation necrosis in the coronal pulp tissue associated to mild/moderate inflammatory response on surrounding tissue. Tertiary dentin adjacent to the necrotic tissue was observed in 25 % of those teeth, associated with reactionary dentin in the lateral walls of the coronal and root pulp chambers (Fig. 5.5). Moderate deposition of reactionary dentin with no coronal pulp necrosis occurred in only 20 % of samples, which exhibited mild inflammatory pulp reaction. All patients subjected to bleaching protocol reported tooth sensitivity (Roderjan et al. 2015).

These data corroborate those obtained from clinical investigations, in which the authors demonstrated that tooth sensitivity is restricted to anterior teeth subjected to in-office bleaching (de Almeida et al. 2012; Bonafé et al. 2013). According to these authors, teeth bleached with 35 %-H_2O_2 gel applied on enamel for 3 consecutive periods of 15 min each had tooth sensitivity in 76.6 % of lateral incisors, 53.3 % of central incisors, and 30 % of canines, with no discomfort reports for premolars.

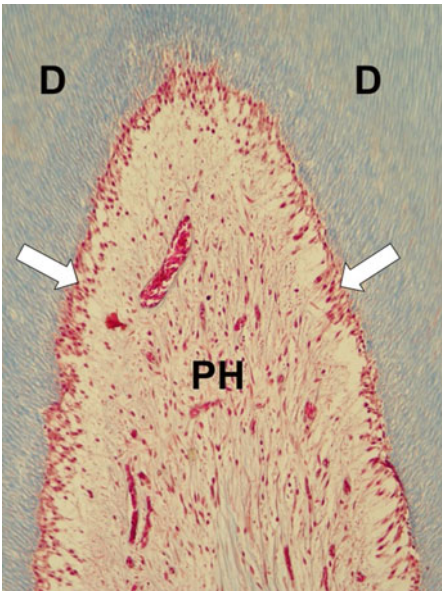

Fig. 5.4 Pulp horn (*PH*) of a human tooth exhibiting normal histological features. Note the odontoblasts (*arrows*) underlying the tubular dentin (*D*) and the subjacent cell-free zone. A number of small blood vessels among collagen fibers and fibroblasts can be observed. Masson's Trichrome, ×125

Thus, one can conclude that enamel/dentin thickness plays an important role in H_2O_2 diffusion across hard tooth structures to reach pulp chamber, causing tissue damage and postbleaching sensitivity, which are more intense in anterior than in posterior teeth.

Another investigation showed that partial pulp necrosis occurred in 60 % of mandibular incisors of elderly patients (54–62 years old; mean 58.2 ± 4.3) in comparison with 100 % of young patients (18–30 years old; mean 20.1 ± 4.3) that were bleached using the same in-office bleaching therapy. Additionally, areas of pulp necrosis were larger in young teeth. The main dentin thickness on young and elderly patients was 1.77 ± 0.08 mm and 1.99 ± 0.10 mm, respectively. As observed in the previous study, the histological sections evidenced a number of differentiated odontoblast-like cells that deposited a layer of reparative dentin below the necrotic area. These pulp responses against bleaching products are similar to those observed after calcium hydroxide application on pulps of sound teeth mechanically exposed (Roderjan et al. 2014). Therefore, it seems that even in human pulps strongly damaged by in-office tooth bleaching procedures, the pulp cells subjacent to the necrotic tissue are capable of maintaining their phenotype, confirming the data collected in previous in vitro studies carried out by our research group.

Based on these data, tooth sensitivity experienced by patients subjected to in-office bleaching with 35–38 % H_2O_2 may be associated with inflammatory reaction caused by oxidizing toxic compounds from bleaching gels capable of reaching the pulp chamber, producing an intense chemical irritation of pulp cells. Because of the fact that in-office tooth bleaching causes pulp damage, the release of cell-derived factors, such as prostaglandins, would excite or sensitize pulpal nociceptors. Additionally, we believe that the fluid shifts that occur in dentinal tubules due to vasodilation and increased pulp pressure during local tissue inflammation may trigger impulses in the intradentinal pulpal nerve fiber endings, causing the intense tooth sensitivity that has been claimed by patients subjected to this professional in-office therapy. However, further studies are needed to assess in detail this topic.

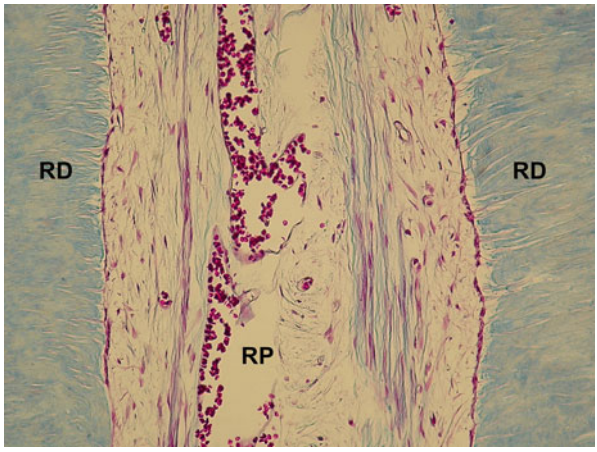

Fig. 5.5 Radicular pulp (*RP*) tissue of a bleached human lower incisor. Note the lateral deposition of reactionary dentin (*RD*), which exhibits a reduced number of tubules with cytoplasm processes within them. Masson's Trichrome, ×180

5.5 Tooth/Restoration Interface as a Pathway for Bleaching Inducing Toxicity

Recent clinical trials showed that the intensity of tooth sensitivity is increased when traditional in-office tooth bleaching is performed in anterior teeth that have adhesive restorations with no clinical sign of margins degradation (Bonafé et al. 2013). It seems that the presence of restorations in tooth to be bleached may enhance H_2O_2 diffusion into the pulp chamber. In addition, dental materials used to restore dental cavities interfere significantly with this H_2O_2 diffusion through enamel and dentin (Gökay et al. 2000, 2004; Benetti et al. 2004; Camargo et al. 2007). Therefore, as several restorative materials and bleaching protocols are available in clinical practice, the question related to the safety of tooth bleaching performed in restored teeth remains a concern (Fig. 5.6).

Previous studies demonstrated that different adhesive systems have variable degrees of susceptibility to H_2O_2, as follows: one-step self-etch > two-step self-etch > etch-and-rinse systems (Van Landuyt et al. 2009; Didier et al. 2013; Dudek et al. 2013; Roubickova et al. 2013). According to the results collected in our laboratory, self-etch adhesive interfaces act as a pathway for H_2O_2 diffusion from tooth surface into pulp chamber, increasing significantly the toxicity of a 35 %-H_2O_2 gel on pulp cells (Soares et al. 2015d). Nevertheless, no significant difference concerning cytotoxicity and trans-enamel and trans-dentinal diffusion of H_2O_2 was observed when etch-and-rinse adhesive restorations where bleached with a 20 or 35 % H_2O_2 gels for 45 min (Soares et al. 2014c). One can conclude that the compromised bond performance of some self-etch adhesives to enamel and dentin creates a more permeable tooth/restoration interface, facilitating H_2O_2 diffusion through dental structure. Resin-modified glass-ionomer cements (RMGIC) interface seem to present the same susceptibility to H_2O_2 as self-etch adhesive systems. The shear bond strength of RMGIC to tooth structure is significantly lower than those observed for etch-and-rinse adhesive systems, due to the low cohesive strength of GIC (Marquezan et al. 2011; Bonifácio et al. 2012; Nujella et al. 2012). According to our results, applying a 35 %-H_2O_2 gel onto enamel/dentin discs containing RMGIC interfaces subjected

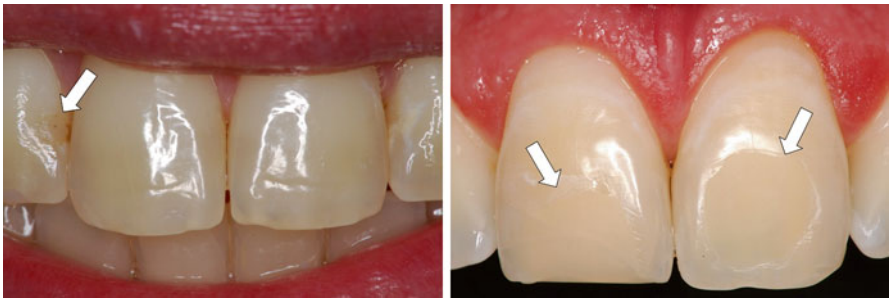

Fig. 5.6 Anterior teeth with resin composite restorations (*arrows*) that were selected by a clinician for professional bleaching (Images provided by Dr. Heraldo Riehl)

to hydrolytic degradation allowed for a more intense H_2O_2 diffusion through hard dental structures, as well as increased the in vitro cytotoxicity to odontoblast-like cells, leading to increased oxidative stress and IL-1β overexpression as well as disturbing the odontoblastic markers expression (Soares et al. 2016b).

Therefore, clinicians should pay attention when selecting the bleaching protocol to be applied in patients that have a number of restored teeth, especially incisors. The materials used for restoring carious and noncarious lesions, as well as the integrity of restoration margins, should be also analyzed in detail in order to prevent, at least in part, damage to pulp tissue and possible postbleaching pain.

5.6 Strategies to Prevent Tooth-Bleaching Mediating Pulp Cells Oxidation

The primary strategy for minimizing pulp tissue damage is based on reducing the amount of residual H_2O_2 capable of reaching the pulp tissue.

As discussed above, reducing the H_2O_2 concentration on bleaching gels, as well as the contact time of these toxic products with tooth structure, seems to have a positive biologic effect. According to our results, a 17.5 % H_2O_2 bleaching gel reduced significantly the trans-enamel and trans-dentinal toxic effects of the therapy to pulp cells, which is commonly observed after professional in-office tooth bleaching using 35 % H_2O_2 gels (Soares et al. 2014a). It was demonstrated that even a concentration of 17.5 % H_2O_2 decreased the human pulp cell viability by 86 %, 77 %, and 65 % after applying the product on enamel/dentin discs for 45, 15, or 5 min, respectively (Soares et al. 2014a). These bleaching protocols also induced intense overexpression of pro-inflammatory mediators and caused reduction on odontoblastic markers expression, demonstrating that the remaining cells were under pathologic oxidative stress (Soares et al. 2015b). Regarding the esthetic bleaching effectiveness, similar color alteration as traditional protocol (with 35 % H_2O_2 gels) was achieved after four sessions when the 17.5 % H_2O_2 gel was applied for 45 min on enamel. However, application of this gel for 15 or 5 min on enamel did not result in the same bleaching outcome as that of high concentrations, even after six sessions (Soares et al. 2014b). A more positive biological effect on human dental pulp cells was achieved when less concentrated bleaching gels were used (8–10 % H_2O_2); but the bleaching effectiveness remained as the main drawback for these alternative tooth bleaching therapies (Soares et al. 2015a).

Strategies based on increasing H_2O_2 decomposition into hydroxyl radicals (OH•) in order to eliminate residual H_2O_2 that can diffuse deeply into enamel and dentin to reach the pulp tissue in concentrations high enough to cause intense tissue damage and postbleaching sensitivity has been evaluated currently. It is known that OH• has higher oxidation potential ($E^0 = 2.8$ V) in comparison to H_2O_2 ($E^0 = 1.8$ V), which in turn make its interaction with chromogens more effective. Also, since OH• is

considered as a transient molecule (half-life of 10^{-9} s), diffusion of high concentrations of this free radical into the pulp chamber is not expected. Indeed, some researchers have demonstrated that incorporation of chemical substances containing iron or manganese as active principles on bleaching gels enhanced the bleaching effectiveness and minimized H_2O_2 diffusion into the pulp chamber (Torres et al. 2010, 2013). These molecules accelerate H_2O_2 decomposition into OH• by means of a Fenton reaction. Results obtained by our research group demonstrated that incorporation of ferrous sulfate on the thickening agent of a 35 %-H_2O_2 gel reduced in about 15 % the negative effect of the bleaching product on odontoblast-like cells (Duque et al. 2014). We also have determined that manganese chloride and hemic peroxidase have a positive effect on reducing the toxicity and oxidative stress on pulp cells mediated by a 35 %-H_2O_2 gel in vitro (unpublished data). All these molecules also increased the esthetic bleaching effectiveness and decreased significantly the H_2O_2 diffusion through hard tooth structures. Therefore, we believe that the chemical activation of low-concentrated in-office bleaching gels will open new perspectives for obtaining effective and biocompatible products and therapies that will turn this esthetic procedure more safe and painless.

Other research groups have proposed the prebleaching administration of anti-inflammatory drugs (Paula et al. 2013) and/or topic application of sodium fluoride/potassium nitrate desensitizing agents (Reis et al. 2011) as palliative alternatives to minimize postbleaching tooth sensitivity. However, both anti-inflammatory drugs and desensitizing agents do not prevent the pulpal damage caused by the ROS-mediated oxidative stress. Administration of antioxidant agents seems to have a more rational appeal, since these agents may act limiting the extension of oxidative damage by donating an electron to the arriving free radicals (Moores 2013). Previous studies performed by our group demonstrated that antioxidant molecules, such as alpha-tocopherol (vitamin E) and ascorbic acid (vitamin C), were capable of preventing the H_2O_2-mediated pulp cells damage, since they inactivate extracellular ROS arising from bleaching gels, as well as the intracellular ones released by cells that undergo oxidative stress (Lima et al. 2010a, b; Vargas et al. 2014a, b). We also observed that oral administration of ascorbic acid (200 mg/kg) 90 min prior application of a 35 %-H_2O_2 bleaching gel during 5 min on rat molars protected the pulp tissue against the toxic effects of this therapy (Lima et al. 2016). All bleached teeth from animals with no ascorbic acid pretreatment featured large necrosis areas on coronal pulp 6 and 24 h after bleaching (Fig. 5.7).

On the other hand, the smaller necrotic area, mainly located at the upper zone of the pulp horn, was observed in 80 % of the animals that received oral administration of ascorbic acid before tooth bleaching. Also, 24 h after bleaching, only 40 % of teeth exhibited punctual areas of necrosis associated with discrete disorganization of reminiscent pulp tissue (Fig. 5.8) (Lima et al. 2016).

Therefore, one can conclude that antioxidant therapy may prevent the strong immediate pulp damage caused by in-office tooth bleaching procedure and enhance the pulpal healing with time. However, more studies are necessary in this research field to determine the ideal and safe doses of antioxidant agents to be administrated for human beings.

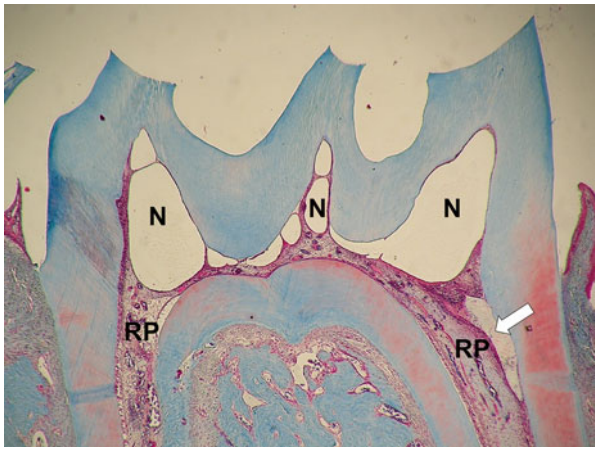

Fig. 5.7 First molar of rat subjected to bleaching treatment. Note the wide necrosis (*N*) of the coronal pulp tissue. The radicular pulp (*RP*) is well maintained in spite of the notable inflammatory reaction and a small local area of edema (*arrow*). Masson's Trichrome, 32×

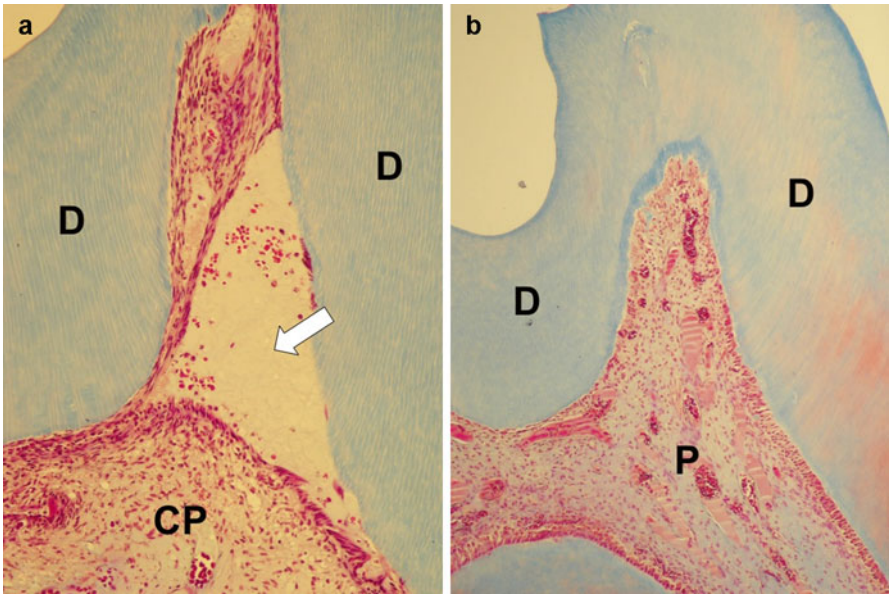

Fig. 5.8 (**a**) Coronal pulp (*CP*) of a molar of rat that received oral administration of ascorbic acid (200 mg/kg) 90 min prior application of bleaching agent. Despite the intense damage in part of the pulp horn (*arrow*), a large area of the subjacent coronal pulp is preserved. Masson's Trichrome, 125×. (**b**) Seven days after tooth bleaching, complete pulpal healing (*P*) was observed in teeth of rats treated with ascorbic acid. Masson's Trichrome, 96× (*D* dentin)

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Part II

Current Techniques for Dental Whitening with Peroxides: Evidence Supporting Their Clinical Use

At-Home Tooth Whitening

6

Jorge Perdigão, Alessandro D. Loguércio, Alessandra Reis, and Edson Araújo

Abstract

Peroxides have been used to whiten teeth for over a hundred years. The popularity of dental whitening has increased with the introduction of nightguard vital whitening in 1989, as the appearance of the dentition and the color of the teeth have increasingly become a concern for a large number of people. Current methods for at-home whitening include materials prescribed by dental professionals, bleaching products available over the counter to use without the involvement of a dental professional, and "do-it-yourself" methods widely advertised on the Internet. This chapter compares the efficacy of the most popular at-home whitening techniques, including dental professional-prescribed at-home tray whitening with carbamide peroxide (a precursor of hydrogen peroxide), over-the-counter whitening, and combined in-office with at-home tray whitening. At-home tooth whitening with a custom-fitted tray is the safest and most effective technique if carried out under the supervision of a dental professional. This chapter reviews the advantages and disadvantages of different at-home whitening techniques, respective side effects, and treatment recommendations based on current scientific information. Clinical cases will illustrate clinically relevant at-home whitening techniques.

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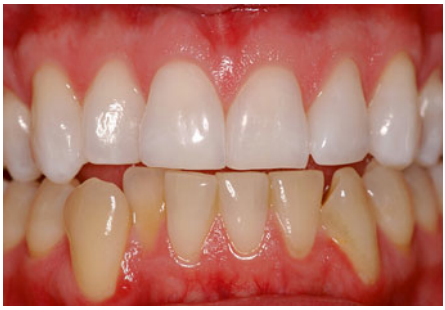

Fig. 6.1 Retracted view of a 28-year-old patient who bleached his maxillary teeth with 10 % carbamide peroxide gel (Opalescence 10 %, Ultradent Products, Inc.) in a custom-fitted tray overnight for 3 weeks. In the first appointment patient had stated that he did not want to whiten the mandibular teeth. He later decided to have the lower teeth bleached with 10 % carbamide peroxide in a custom-fitted tray

6.1 Introduction

In an independent survey conducted on behalf of the American Academy of Cosmetic Dentistry (2004), 99.7 % of adults in the USA believed that a smile is an important social asset. When respondents were asked, "What would you most like to improve about your smile," the most common response was "whiter and brighter teeth." More recently, Hendrie and Brewer (2012) reported that deviation from normal tooth spacing and/or the presence of yellowed teeth have negative effects on ratings of attractiveness and these effects are markedly stronger in females.

In 1989, Haywood and Heymann introduced the *nightguard vital bleaching* (NGVB) technique using a vacuum-formed custom-fitted "soft plastic nightguard, approximately 2-mm thick (similar to an athletic mouthguard)" filled with Proxigel (Reed & Carnrick Pharmaceuticals), a 10 % carbamide peroxide gel available over the counter as a Food and Drug Administration (FDA)-approved antiseptic (Haywood and Heymann 1989). Teeth were treated for 2–6 weeks and evaluated at 2 and 5 weeks to assess color change. This at-home whitening technique with carbamide peroxide in a custom-fitted tray, which is commonly referred to as "at-home whitening," "at-home bleaching," or "tray whitening,"[1] has become very popular worldwide. Numerous clinical studies and reports have described the effectiveness (Fig. 6.1) and safety of *tray whitening* (Reinhardt et al. 1993; Kihn et al. 2000; Cibirka et al. 1999; Matis et al. 1998, 2006; Meireles et al. 2008b; Li 2000; ADA Council on Scientific Affairs 2010, 2012). According to the American Dental Association Council on Scientific Affairs (ADA) (2010), "data accumulated over the last 20 years indicate no significant, long-term oral or systemic health risks associated with professional at-home tooth bleaching materials containing 10 % carbamide peroxide." Carbamide peroxide is a precursor of hydrogen peroxide, as described in Chap. 3. Besides at-home whitening with carbamide peroxide in a custom-fitted tray, there are currently other techniques that involve at-home whitening:

[1] The terms "whitening" and "bleaching" are used interchangeably in the literature.

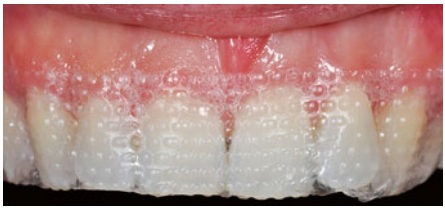

Fig. 6.2 Frontal view of a 38-year-old patient 5 min after she applied a bleaching strip to her maxillary teeth

1. Dentist-administered in-office whitening followed by at-home whitening with a peroxide-based gel in a custom-fitted tray, also known as *jump-start technique*. In-office bleaching is performed first by a dental professional to provide an initial jump-start bleaching effect. Then, the patient is prescribed at-home whitening (Kugel et al. 1997), usually 10–20 % carbamide peroxide gel for daily application, which is to be used until the desired shade is obtained (Deliperi et al. 2004).
2. Dentist-administered, custom-fitted bleaching tray containing a higher concentration of carbamide peroxide (usually ≈ 35 %), also known as *waiting-room* whitening. Patient wears the tray filled with carbamide peroxide gel for periods of 30 min to 1 h while waiting in the dental office.
3. Over-the-counter (OTC) tooth-whitening products for home use without professional supervision, such as gels, rinses, paint-on films, strips (Fig. 6.2), and kits with prefabricated "thermoforming" or "thermofitting" trays that can be molded in hot water by the user at home (please see Chaps. 1 and 4 for more information on OTC whitening products). Dual-arch trays prefilled with a silicone material, from which the patient can make a custom-fitted prosthesis, are available online from auction sites.

OTC tooth-whitening products were not included in the list of accepted whitening agents by the now extinct American Dental Association Seal of Acceptance Program. Some toothpastes are also marketed as having a whitening effect. They typically contain an abrasive to remove and/or prevent surface stains, such as hydrated silica, calcium carbonate, dicalcium phosphate, dihydrate, calcium pyrophosphate, alumina, perlite, and sodium bicarbonate (Joiner 2010). The inclusion of peroxides in toothpaste is much more challenging in terms of formulation and the short exposure time. A toothpaste with 0.5 % calcium peroxide has been shown to reduce natural extrinsic stain after 6 weeks (Ayad et al. 1999). In the European Union, the maximum concentration of peroxide allowed in toothpastes and mouth rinses is 0.1 % (European Commission Scientific Committee on Consumer Products 2007). According to the same document, most clinical studies with peroxide-containing toothpastes are sponsored by the respective manufacturers and rarely published.

Indications for at-home whitening with carbamide peroxide gel in a custom-fitted tray:

- Yellow teeth caused by aging (Fig. 6.1), which is discussed in Sect. 6.4.4.1.
- Tetracycline staining, especially degrees I and II (Jordan and Boksman 1984) (Fig. 6.3), which is discussed in Sect. 6.4.4.2.

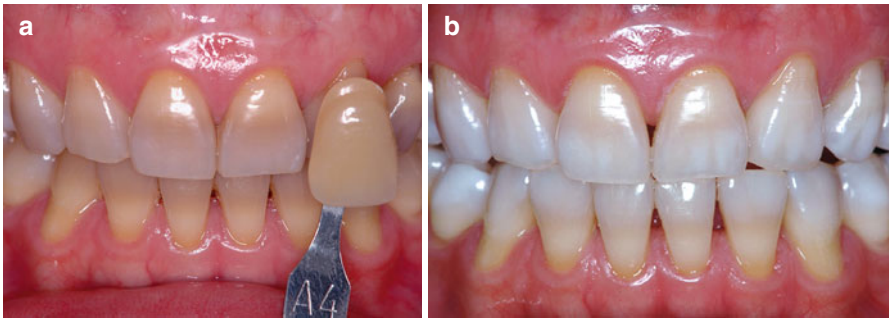

Fig. 6.3 (**a**) A 23-year-old patient with a history of antibiotic intake when she was a child. Although patient did not recall which type of antibiotic she had been prescribed, the clinical exam suggested that the discoloration was compatible with tetracycline staining. (**b**) Aspect after 4 months of at-home whitening with 10% carbamide peroxide gel with potassium nitrate and sodium fluoride (Opalescence 10% PF, Ultradent Products, Inc.) overnight in a custom-fitted tray. Patient returned to clinic for monthly recalls. She did not experience any sensitivity or any other side effects

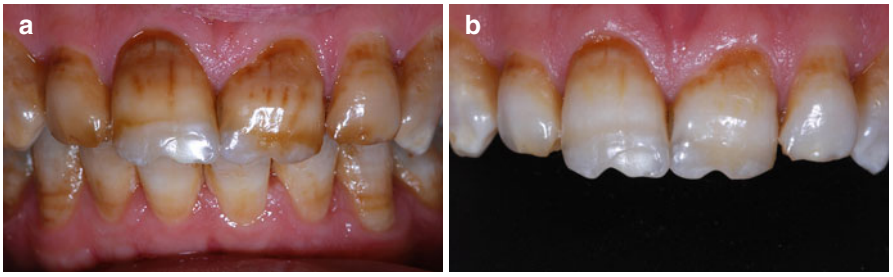

Fig. 6.4 (**a**) This 38-year-old patient was born and raised overseas in an "area where everybody had brown teeth". He used to drink water from a water well when he was a child in his home country. According to the patient's account, all his siblings who lived in the same area had "brown teeth." The medical history revealed no significant findings. After an intra-oral exam, patient was informed that at-home whitening might improve the appearance of his teeth as long as he understood that the treatment could span over a few months. Patient agreed to have his teeth whitened with 10% carbamide peroxide gel with potassium nitrate and sodium fluoride (Opalescence 10% PF, Ultradent Products, Inc.) at-home in a custom-fitted tray. Patient was scheduled for monthly recalls. (**b**) After 3 months of treatment, the appearance of the teeth improved considerably. Patient was very happy, in spite of confessing that he did not wear the tray on a daily basis. Patient chose to stop the treatment for a few months and then restart (Reprinted with Permission from Perdigao J (2010) Dental whitening – revisiting the myths. Northwest Dent 89:19–21, 23–6. (Northwest Dentistry, *The Journal of the Minnesota Dental Association*))

- Yellow/brown stains from enamel fluorosis (Fig. 6.4) or from idiopathic causes, which are discussed in more detail in Sect. 6.4.4.3. Clinical solutions related to this topic can be found in Chaps. 12, 13, and 15.
- Discolored tooth caused by calcific metamorphosis (Fig. 6.5), which is discussed in more detail in Sect. 6.4.4.4.
- Whitening of anterior teeth prior to esthetic rehabilitation with veneers (Fig. 6.6) or direct resin-based composite restorations (Chap. 14).
- Dietary stains

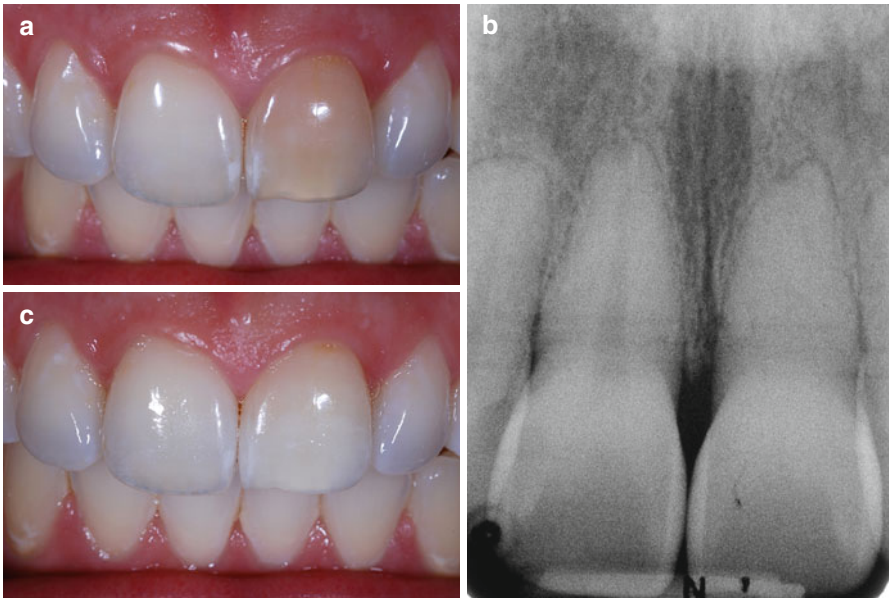

Fig. 6.5 (**a**) Frontal view of maxillary incisors in a 26-year-old patient whose chief complaint was her discolored tooth #9 (FDI 2.1). Patient had had a traumatic injury to this tooth when she was 12 years old. No other signs or symptoms were associated with this tooth. Response to percussion was identical in all her maxillary anterior teeth. Although the response to cold was negative, the pulp responded to the electric pulp tester. (**b**) Periapical radiograph showing a calcified pulp space in tooth #9 (FDI 2.1). (**c**) Clinical aspect after 2 weeks of at-home whitening with 10% carbamide peroxide gel with potassium nitrate and sodium fluoride (Opalescence 10% PF, Ultradent Products, Inc.) in a custom-fitted tray. Patient decided to bleach for another period of 2 weeks, but she did not return for the recall appointment

Contraindications for at-home whitening with carbamide peroxide gel in a custom-fitted tray:

- Patient's unrealistic expectations.
- Patient unsure if he/she is willing to carry out the treatment on a daily basis.
- Smoking. As a precautionary measure, patients are informed to refrain from smoking between 2 h before inserting the tray and 2 h after removing the tray. This relative contraindication derives from the findings of the "hamster study" (Weitzman et al. 1986), as discussed in detail in Chap. 3. More recently, it has been reported that at-home bleaching does not induce DNA damage to the gingival tissue of smokers during a 3-week treatment (de Geus et al. 2015c). However, smokers had slightly darker teeth after 1 month of at-home bleaching. This may be an important detail of information to disclose to patients prior to starting the bleaching treatment (de Geus et al. 2015a). A clinical study also concluded that bleaching with 10% carbamide peroxide during 3 h daily for 3 weeks is effective in smokers even after 1 year, but dental prophylaxis may be necessary to remove extrinsic stains caused by diet and smoking (de Geus et al. 2015b).

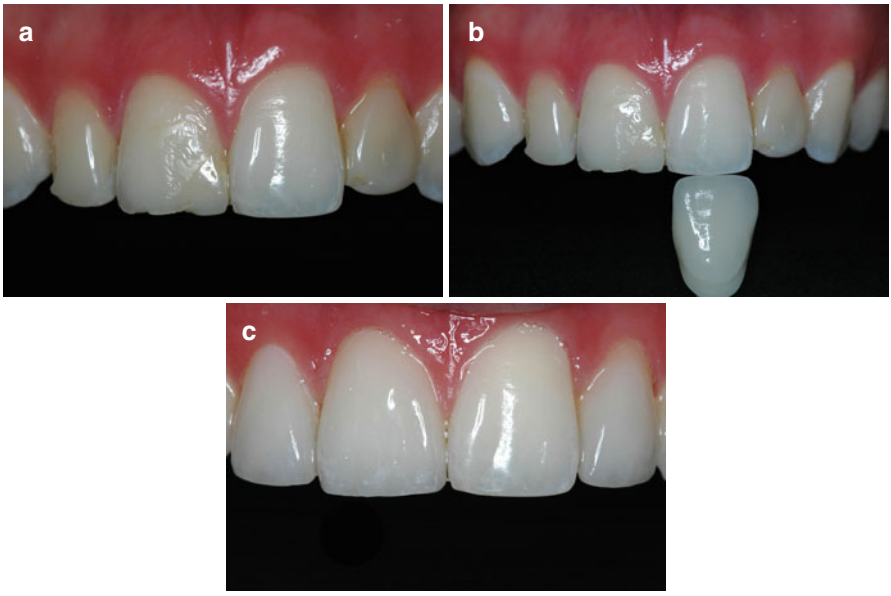

Fig. 6.6 (**a**) A 22-year-old patient with several resin-based composite restorations in his discolored maxillary incisors. Tooth #9 (FDI 2.1) was not restored. (**b**) Clinical aspect after 2 weeks of overnight at-home whitening with 10% carbamide peroxide gel with potassium nitrate and sodium fluoride (Opalescence 10% PF, Ultradent Products, Inc.) in a custom-fitted tray. (**c**) Porcelain veneers were bonded with a etch-and-rinse adhesive and a light-cure resin-based luting cement on teeth #7 (FDI 1.2), #8 (FDI 1.1), and #10 (FDI 2.2) 5 weeks after patient completed the bleaching treatment

- Pregnancy and breastfeeding. There is not enough available evidence concerning either the teratogenicity or the safety for breastfeeding infants of the ingredients of whitening agents.
- Root (dentin) hypersensitivity. Preexisting tooth sensitivity must be treated prior to starting the whitening treatment to block any patent dentinal tubules.
- Gingival recession with discolored roots, especially in elderly patients. Radicular dentin does not respond to whitening as well as coronal dentin does (Haywood 2003).
- Possible allergy to inactive components of the bleaching gel.
- Acatalasemia. A hereditary disorder in which the blood catalase activity level is below normal (European Commission Scientific Committee on Consumer Products 2007).
- G6PD deficiency. A genetic disorder of erythrocytes that lack the enzyme G6PD, causing them to destroy prematurely and preventing patient with this disorder to break down hydrogen peroxide (European Commission Scientific Committee on Consumer Products 2007).
- Some patients with xerostomia, as dry mouth may affect the degradation of hydrogen peroxide (European Commission Scientific Committee on Consumer Products 2007).

Table 6.1 Advantages and disadvantages of at-home whitening with a custom-fitted tray supervised by a dental professional

Advantages	Disadvantages
Very effective, durable whitening	Tooth sensitivity
Backed with clinical and laboratory research	Patient compliance
Safe	Relatively long treatment time
Low cost compared to in-office procedures	Over-the-counter whitening methods are less expensive

- Discolored endodontically treated teeth *do not typically respond to external tray whitening* as well as vital teeth do. However, some cases of light staining in endodontically treated teeth that have darkened recently can be successfully bleached externally using ≈ 20 % carbamide peroxide gel in a specially designed bleaching tray (Fig. 6.7). The prognosis of the at-home whitening treatment in these cases depends on the nature and duration of the discoloration.
- Deep white spot lesions. White spots deeper than 0.5 mm may be better camouflaged with other treatment modalities, including microabrasion, enamel etching followed by resin infiltration (Chaps. 9, 10, 12, and 13), or removal of the discolored area and restoration with a dentin adhesive and a resin-based composite material (Chap. 15, Sect. 15.3).

6.2 Advantages and Disadvantages of At-Home Whitening

Two major advantages of at-home whitening are its efficacy (Fig. 6.1) and stability of posttreatment color (Table 6.1) (Swift et al. 1999; Ritter et al. 2002). After 10 years, 43 % of patients that whitened their teeth with 10 % carbamide peroxide for 6 weeks deemed the color to be stable (Ritter et al. 2002).

A major disadvantage of at-home whitening is patient's compliance (Table 6.1), as the dental professional is unable to monitor the daily treatment. Meireles et al. (2008a) asked subjects to return all used and unused bleaching gel syringes to ensure compliance based on the amount of gel used. This method, however, may be difficult to implement on a regular basis.

Another disadvantage of at-home whitening compared to in-office whitening is the longer treatment time for the former. However, one session of in-office whitening is not usually sufficient to achieve optimal results (Al Shethri et al. 2003), resulting in a similar overall treatment time for the two techniques.

6.3 Efficacy and Durability

There are several variables that influence the treatment outcome, including the technique, the type of bleaching agent, the concentration, and the application time (Joiner 2006; Buchalla and Attin 2007; Meireles et al. 2008b; Matis et al. 2009a).

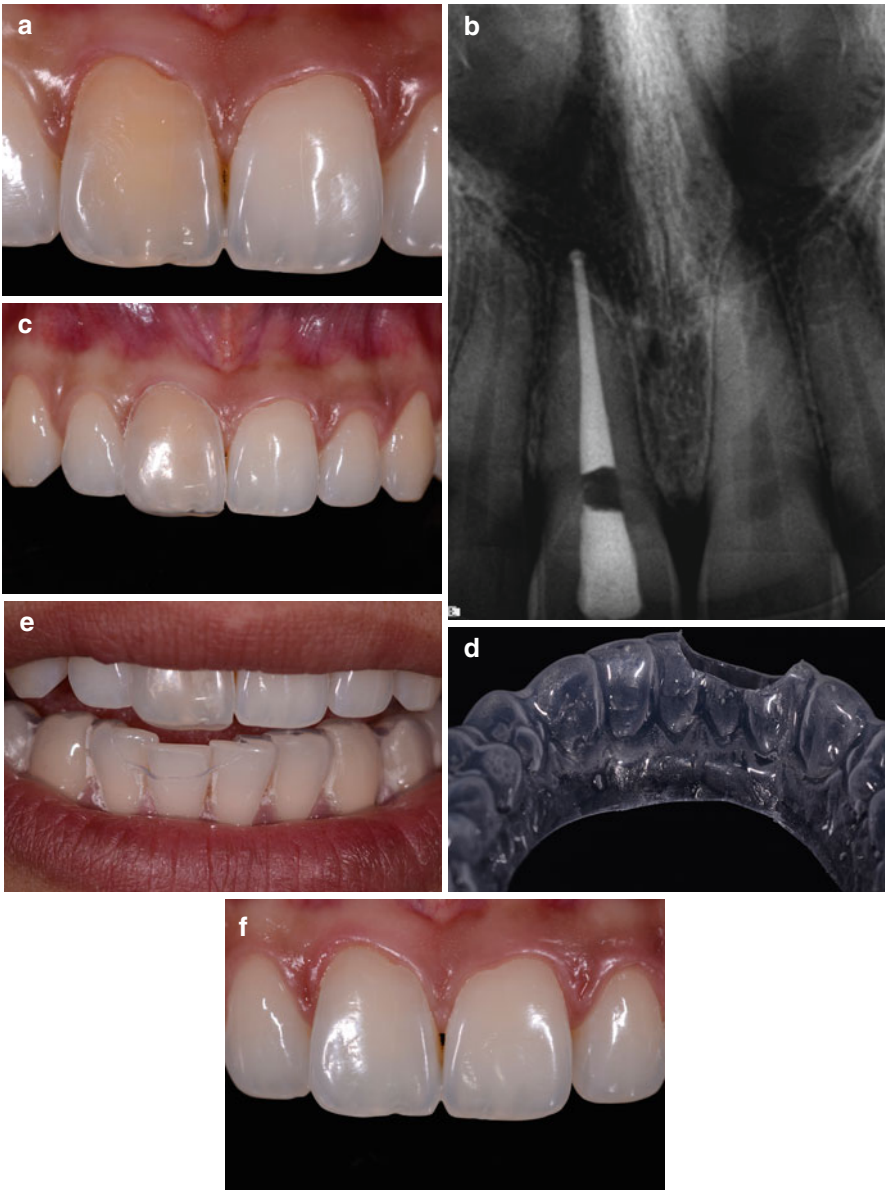

Fig. 6.7 (**a**) A 24-year-old patient suffered a traumatic injury to her tooth #8 (FDI 2.1). After 4 years, her tooth became darker without any symptoms. The patient immediately visited her dentist who diagnosed pulpal necrosis. (**b**) A root canal treatment was performed and the lingual access preparation restored with a resin-based composite material. (**c**) A special bleaching tray was fabricated to bleach the discolored tooth following an at-home regimen. (**d**) A lower tray was fabricated and customized to serve as stabilizer for the upper one-tooth tray. The lower tray was not used as a bleaching tray. (**e**) Patient wearing the upper bleaching tray and the lower stabilizing tray. (**f**) After 5 days of at-home whitening with 22% carbamide peroxide gel (Whiteness Perfect 22%, FGM) for 2 h twice daily

6.3.1 At-Home Whitening in a Custom-Fitted Tray

Dentist-prescribed overnight bleaching with carbamide peroxide in a custom-fitted tray has been shown to be the safest, most effective method of tooth whitening (Haywood 2003; Matis 2004; Matis et al. 2009a).

Two clinical studies evaluated the 2-year effectiveness of tray whitening with carbamide peroxide (Swift et al. 1999; Meireles et al. 2010). In the first study, 29 patients had their maxillary teeth treated with a 10% carbamide peroxide gel nightly for 2 weeks. Teeth became eight shades lighter after 2 weeks of treatment when color was measured with the Vita Classical A1-D4 shade guide (VITA Zahnfabrik H. Rauter GmbH & Co. KG) organized by value (lighter to darker). Twenty-four patients were recalled after 2 years. Teeth in 20 patients (83.3%) had darkened an average of two shades, which occurred during the first 6 weeks posttreatment. The lightening effect remained statistically significant at 2 years. Overall, patients were satisfied with the shade. In the second study (Meireles et al. 2010), 92 patients whitened their maxillary anterior teeth with 10% carbamide peroxide or with 16% carbamide peroxide in a custom-fitted tray for 2 h/day during 3 weeks. Shade evaluations were carried out at baseline, 1 month, 6 months (Meireles et al. 2008b), 1 year (Meireles et al. 2009), and 2 year post-bleaching (Meireles et al. 2010). Although the 16% carbamide peroxide group showed some reversal of the whitening effect at 1 year (Meireles et al. 2009), both treatment groups had the same median tooth shade 1 year after bleaching, which was lighter than at baseline. At 2 years, the median tooth shade remained lighter than at baseline for both carbamide peroxide concentrations tested.

Boushell et al. (2012) evaluated patients' satisfaction and reported side effects of at-home whitening with 10% carbamide peroxide in a custom-fitted tray up to 17 years posttreatment. Thirty-one participants who had completed clinical studies using 10% carbamide peroxide were contacted at least 10 years posttreatment. Patient satisfaction with tray whitening was determined to last an average of 12.3 years posttreatment.

In case the patient perceives that there has been a color regression and is willing to touch-up the teeth color, the original bleaching tray may be applied for 2 or 3 nights using the same 10% carbamide peroxide gel. In case the tray no longer fits the patient's teeth as a result of recent restorations or extracted teeth, disposable trays prefilled with 6%, 10%, or 15% hydrogen peroxide gel (Opalescence Go, Ultradent Products, Inc.) may be used 2 or 3 days. The respective manufacturer recommends decreasing contact times with increasing hydrogen peroxide concentration. For the lowest concentration, the manufacturer suggests that patients wear the disposable tray for 60–90 min daily, whereas for the highest hydrogen peroxide concentration the recommended contact time is 15–20 min daily.

Patients often inquire if they need to refrain from a potentially staining diet during and after at-home whitening. Current evidence from controlled clinical trials suggests that coffee does not interfere with the outcome of whitening nor does it affect tooth sensitivity (Rezende et al. 2013). Peroxide-based whitening agents are effective in preventing any staining from coffee or red wine during the treatment (Cortes et al. 2013). As a result, the need for a white diet during the bleaching treatment has been challenged. A study determined whether a white

diet is necessary by evaluating the effects of coffee, tea, wine, and dark fruits on tooth whitening during the bleaching process (Matis et al. 2015). From five published studies, the authors concluded that a nonwhite diet was not significantly associated with less tooth whitening, and there was only a weak positive association between tooth whitening and diet for subjects who consumed large amounts of coffee/tea.

After the whitening regimen is completed, both coffee and red wine cause enamel color change. Red wine, however, stains enamel more intensely than coffee (Cortes et al. 2013).

6.3.2 Jump-Start Whitening

The objective of the *jump-start* technique is to boost the bleaching effect with the in-office treatment, then improve color stability with the at-home component to reach a more esthetic result compared to in-office bleaching alone (Deliperi et al. 2004; Matis et al. 2009b). Clinical evidence, however, does not support this assumption. Two recent clinical studies reported that the results of the combined in-office/at-home technique were similar to those obtained only with the at-home technique (Bernardon et al. 2010; Dawson et al. 2011). Therefore, the in-office component of the combined jump-start technique does not improve the treatment outcome and may be considered redundant. Nevertheless, this technique may motivate some patients, as the whitening effect is visible immediately.

A more recent version of the *jump-start* technique is known as *deep whitening technique* (Kör Whitening, Evolve Dental Technologies, Inc) (Sulieman 2008). This technique currently includes three different modalities, according to the severity of the discoloration: (A) 2 weeks of at-home overnight whitening with 16 % carbamide peroxide, followed by one in-office whitening session with 34 % Tri-Barrel Hydremide™ peroxide[2]; (B) in-office "conditioning" visit with 13 % Tri-Barrel Hydremide™ peroxide in whitening trays followed by 3–4 weeks of at-home overnight whitening, and a final in-office whitening session with 34 % Tri-Barrel Hydremide™ peroxide; (C) in-office "conditioning" visit with 13 % Tri-Barrel Hydremide™ peroxide in whitening trays, followed by 6–8 weeks of at-home overnight whitening, and a final in-office whitening session with 34 % Tri-Barrel Hydremide™ peroxide. All three methods require periodic at-home maintenance after the treatment.

> Currently, there is no *independent* scientific evidence to back the use of the so-called deep whitening technique.

[2] Hydremide is a trademark by Evolve Dental Technologies, Inc. Tri-barrel hydremide peroxide is 1 barrel of hydrogen peroxide gel, 1 barrel of carbamide peroxide gel, and the third barrel contains an activator. The term hydremide derives from combining *HYDROgen* (peroxide) with *carbaMIDE* (peroxide).

The trays used in the *deep bleaching technique* are specially made trays (Kurthy 2001). As per the respective manufacturer, these trays provide better sealing than conventional bleaching trays, enabling the whitening agent to be active all night as opposed to other tray whitening methods (Kör Whitening 2015). The manufacturer's website also states: "Clinical Research associates as well as other researchers have found that whitening gel in conventional whitening trays is only strongly active for 25–35 minutes. This is due to rapid contamination of the whitening gel by saliva." However, this statement is not supported by independent research. It has been shown that hydrogen peroxide releases all of its peroxide in 30–60 min, with a quick decline, while carbamide peroxide releases about 50 % of its peroxide in 4 h, then experiences a slow decline (Haywood 2005).

More than 50 % of the carbamide peroxide active agent is available after 2 h. The percentage of carbamide peroxide recovered from tray and teeth is 10 % at 10 h (Matis et al. 1999).

The use of light sources to allegedly activate the peroxide during the in-office component of the jump-start technique has been used in many dental offices. According to Christensen (2003), "all whitening methods are successful to some degree" but "the use of lights with bleaching has been mainly a marketing tool."

6.3.3 Over-the-Counter (OTC) Whitening

Sales of OTC bleaching products have increased dramatically in recent years (Chap. 1), driven not only by their lower cost compared to professional tooth-whitening techniques but also by strong consumer demand for esthetic dental care and easy access through online auctions and e-commerce sites. Additionally, OTC bleaching products are easy to use and convenient for the patient (Kugel 2003). Concentrations as high as 44 % carbamide peroxide are available from online auctions sites and retailers. Non-dental options are the latest trend, including mall kiosks, salons, and spas. More recently, whitening has been performed in passenger ship cruises (ADA Council on Scientific Affairs 2010).

How does the efficacy of OTC whitening products compare to that of the dentist-prescribed at-home whitening? While there are many studies comparing OTC whitening with dentist-prescribed whitening, there are only a few *independent* clinical studies (Serraglio et al. 2016). A study (Bizhang et al. 2009) measured tooth shade with spectrophotometry and concluded that 6 % hydrogen peroxide whitening strips applied twice a day for 30 min each for 2 weeks are not as effective as at-home whitening with 10 % carbamide peroxide overnight for 2 weeks. Kishta-Derani et al. (2007) evaluated four paint-on films self-adhering solutions that are brushed on the tooth surface. These paint-on films contained hydrogen peroxide, sodium percarbonate or carbamide peroxide. Two of the paint-on films did not result in any

significant whitening effect after 2 weeks of daily application. The results obtained with OTC whitening are not as pleasant and the procedure is not as safe as those methods prescribed by a dental professional (Haywood 2003). For similar concentrations of hydrogen peroxide, OTC bleaching strips cause more gingival irritation and tooth sensitivity than at-home tray whitening, as discussed in Chap. 4.

Given that OTC whitening products are not custom-fitted to the patient's mouth, they are not the ideal vehicle for the application of peroxide-based gels. Ill-fitting trays may result in soft tissue injury, poor patient compliance, and malocclusion problems (Kugel 2003).

6.4 At-Home Whitening with a Custom-Fitted Tray Supervised by a Dental Professional

6.4.1 Treatment Plan

The correct diagnosis of the origin of the discoloration is critical, as different treatment options lead to different clinical outcomes. It is, therefore, imperative that the dental professional understands the etiology of each specific tooth discoloration case to be able to diagnose and prescribe the proper treatment for each patient. Please refer to Chap. 1 for further details.

A full-mouth exam and recent periapical radiographs of the anterior teeth are essential during the diagnostic appointment. Intra-oral photographs are extremely valuable to document the pretreatment tooth color for future comparisons and to include in the patient's record. Pulp testing is always necessary for single-tooth discolorations. Patient must be informed that existing anterior esthetic restorations, including porcelain and resin-based composites, will not lighten with bleaching agents, except for superficial extrinsic stains (Fig. 6.8). *These restorations must be replaced after the whitening treatment is completed to ensure an acceptable esthetic outcome.* Additionally, patient must also be informed that amalgam restorations that come in contact with the bleaching gel may generate a "greening effect" of the tooth structure in areas immediately adjacent to the amalgam material (Haywood 2002).

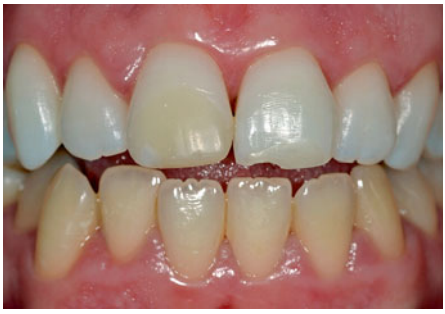

Fig. 6.8 Existing resin-based composite restorations on teeth #8 (FDI 1.1) and #9 (FDI 2.1) after at-home whitening of the maxillary arch with 10% carbamide peroxide with potassium nitrate and sodium fluoride (Opalescence 10% PF, Ultradent Products, Inc.) in a custom-fitted tray for 3 weeks

Haywood (2003) defined nightguard vital bleaching as a three-step technique:

1. Whitening material, which is usually a thick peroxide-based gel
2. Application prosthesis, currently known as the bleaching tray
3. Treatment regimen

Haywood (2003) suggested that wearing a tray on only one arch might improve patient's compliance, as patient can directly observe the color change in one arch compared to the arch that is not undergoing treatment. Additionally, the interocclusal thickness of both maxillary and mandibular trays may exacerbate TMJ disorder symptoms (Robinson and Haywood 2000).

6.4.2 Whitening Material

Carbamide peroxide in concentrations between 10 and 22 %,[3] and hydrogen peroxide in concentrations from 4 to 8 % have been used for at-home bleaching for different periods of time (Joiner 2006; Meireles et al. 2008b; Matis et al. 2009a). A recent systematic review and meta-analysis of at-home whitening concluded that carbamide peroxide results in a slightly better whitening efficacy than hydrogen peroxide when applied in a custom-fitted tray (Luque-Martinez et al. 2016).

The bleaching agent for at-home application that has been more frequently scrutinized in the dental literature is 10 % carbamide peroxide (Matis 2004). Chemically, carbamide peroxide is a crystalline material containing a molecule of urea complexed with a single molecule of hydrogen peroxide – 10 % carbamide peroxide contains approximately 3.3–3.5 % hydrogen peroxide (Cooper et al. 1992; Sulieman 2008; ADA Council on Scientific Affairs 2010). Carbamide peroxide is preferred over hydrogen peroxide because it is more stable than hydrogen peroxide, providing a nonaqueous formula of available hydrogen peroxide (Fischer 1995).

Current carbamide peroxide bleaching gels contain glycerin as a humectant and flavor enhancer; and a thickener, usually a polymer (Carbopol,[4] The Lubrizol Corporation). Carbopol polymers are cross-linked high molecular weight homo- and copolymers of acrylic acid, therefore containing active carboxyl groups. These polymers are slightly acidic, which lowers the pH of the bleaching gel. Accordingly, bases such as sodium hydroxide may be used to make the gel less acidic. Similar thickeners and bases are also used in the composition of hydrogen-peroxide-containing OTC bleaching strips.

[3] The chance of a mismatch between the advertised concentration and the actual concentration is very high (Matis et al. 2013).

[4] Carbomer 934P or Carbopol 934P (The Lubrizol Corporation) is primarily used in commercially available oral formulations, including bleaching gels for tray whitening.

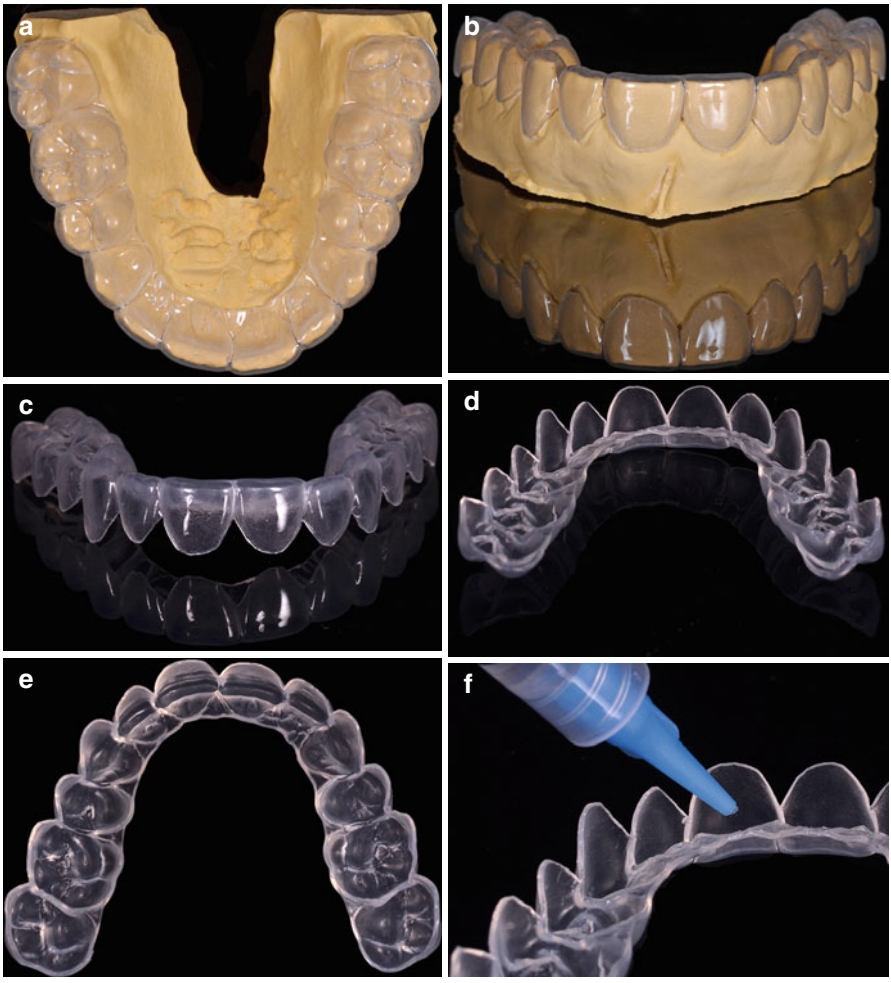

Fig. 6.9 Custom-made bleaching tray for at-home whitening. (**a**) Occlusal view of the tray inserted onto the stone model. The model has been trimmed to remove the palatal area to enhance the vacuum over the teeth and obtain a tighter adaptation of the heated tray material to the teeth. (**b**) Frontal view of the model after the tray was scalloped around the gingival margins. (**c**) Frontal view of the scalloped tray. (**d**) Lingual view of the scalloped tray. (**e**) Incisal view of the scalloped tray. (**f**) Demonstrating to patient how to load the bleaching gel into the tray

6.4.3 Bleaching Tray Design

Several brands of thermoplastic materials are available to fabricate bleaching trays. We currently use a 0.035″-thick ethylene vinyl acetate material that is heated prior to forming the tray around the stone model in a vacuum or pressure device, as shown in the video *Fabrication of a Whitening Tray*. After cooling, the tray is then trimmed in a horseshoe shape (Fig. 6.9). The fine trimming must follow the scalloped contour of the free gingival margin (Haywood 1997b). The design has evolved

to a scalloped tray slightly short of the free gingival margin (0.5–1.0 mm) to prevent possible irritation caused by the contact of the gel with the soft tissues (Chap. 4). The scalloped design is contraindicated with low-viscosity bleaching gels, as the gel is more likely to leak to the mouth and irritate the tongue and lips (Haywood 2003). In specific situations, including clinical cases of one-tooth whitening, the tray may be slightly extended gingivally. In case of inadvertent fabrication of shortened trays, successful whitening still occurs beyond the borders of the short tray without demarcation lines on the teeth (Oliver and Haywood 1999), as peroxides diffuse easily through enamel.

The use of tray reservoirs to make space to retain the bleaching gel has been patented (Fischer 1992). It remains, nevertheless, a controversial issue. Light-cured block-out resin spacers are recommended by some manufacturers, but the use of spacers to create reservoirs for the bleaching gel does not seem to increase the success of home bleaching (Javaheri and Janis 2000; Matis et al. 2002). The bleaching gel remains active for longer periods when reservoirs are used (Matis et al. 2002), which may be the reason why tray reservoirs result in higher rates and higher intensity of gingival inflammation during at-home bleaching (Kirsten et al. 2009).

The tray is then tried-in after fine trimming to check for a tight fit, making sure that the patient does not feel any sharp edges. The dental professional must examine the soft tissues very carefully at this stage to identify areas of compression that may cause discomfort to the patient. It is crucial to demonstrate how to dispense the right amount of gel into the tray, usually one drop (Fig. 6.9f). To verify the gel covers the buccal aspect of the tooth the patient is instructed to ensure that a very slight amount of gel has extruded from the tray at its gingival border. Then, the excess gel is wiped out with a toothbrush or a cotton swab to prevent the contact of the gel with the mucosa. The bleaching gel may also be applied from the lingual in case the buccal enamel is covered with restorative material (Fig. 6.10). Haywood and Parker (1999) described a case of porcelain veneers bonded to tetracycline-stained teeth that resulted in a graying of the veneers. A custom-fitted tray with no reservoirs and no gingival scalloping was used to bleach the teeth with 10 % carbamide peroxide applied nightly for 9 months.

- The use of spacers for the bleaching gel does not improve the success of home bleaching.
- The bleaching gel remains active for longer periods of time when spacers are used.
- The use of a reservoir in the tray may result in higher intensity of gingival inflammation.

6.4.4 Treatment Regimen

6.4.4.1 Physiological Discoloration

The recommended duration of the treatment for the *original* nightguard vital whitening technique with 10 % carbamide peroxide was 2–6 weeks (Haywood and

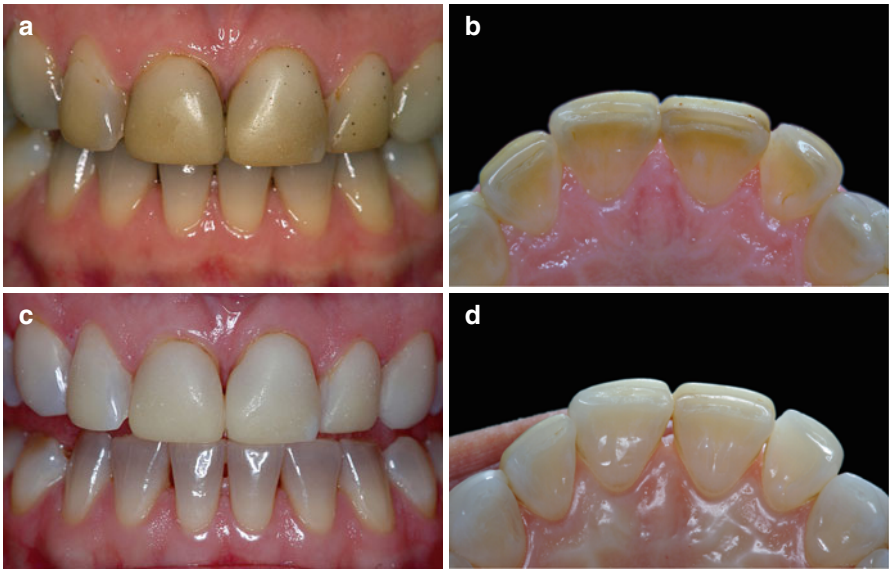

Fig. 6.10 (**a**) A 43-year-old patient visited the University of Minnesota School of Dentistry Comprehensive Care Clinic to ask for a second opinion about her front maxillary teeth. She had direct resin-based composite veneers placed approximately 20 years back, but the restorative material had become discolored with "black spots all over." The patient was not sure if porcelain veneers were indicated for her clinical situation. (**b**) The lingual view of the maxillary incisors depicted a slight grayish dentin discoloration. Although the medical history was negative for antibiotic ingestion, the patient vaguely recalled having some fever episodes and possibly taking antibiotics in her childhood. We then informed the patient that we might be able to whiten her teeth if she agreed to wear a tray with 10% carbamide peroxide gel with potassium nitrate and sodium fluoride (Opalescence 10% PF, Ultradent Products, Inc.) for 2–6 months at night. After the patient agreed and signed the respective consent form, a custom-fitted tray was fabricated and the patient instructed to apply the whitening gel into the lingual aspect of the tray to whiten the teeth from the lingual surface. Patient was also instructed to return to the clinic every month. (**c**) Retracted frontal view after three months of at-home whitening. Note that the composite stains were removed by the peroxide oxidative action. Patient did not experience any sensitivity or any alterations of the soft tissues at each periodical recall. (**d**) Lingual view after 3 months. Compare the shade with that of **b**. Old resin-based composite restorations were removed at a subsequent appointment and enamel polished with diamond pastes. After observing the final result, patient was unsure whether or not she wanted veneers. She decided that she did not want any other treatment (Reprinted with Permission from Perdigao J (2010) Dental whitening – revisiting the myths. Northwest Dent 89:19–21, 23–6. (Northwest Dentistry, *The Journal of the Minnesota Dental Association*)

Heymann 1989). Currently, the typical treatment time for teeth that are inherently discolored by aging or discolored by diet and chromogenic diet is from 2 to 4 weeks, especially if the treatment is carried out overnight.

Although higher concentrations of peroxides result in a faster rate of whitening than 10% carbamide peroxide, they reach a similar final result (Matis et al. 2000; Meireles et al. 2009; Basting et al. 2012). Higher concentrations, however, increase the incidence of tooth sensitivity (Matis et al. 2000). We have only prescribed 10%

carbamide peroxide for at-home whitening of physiological discoloration in the last 10 years. This concentration is the only one that has been approved by the American Dental Association (ADA Seal Product Category 2015).

A clinical study tested four different application times of 10 % carbamide peroxide – 15 min, 30 min, 1 h or 8 h. After 16 days, 15 out of 15 (100 %) subjects that had bleached for 8 h/day were satisfied with the results, while only 5/15 subjects that had bleached 1 h per day were satisfied with the results (Cardoso et al. 2010). Matis et al. (2009a) pooled data from nine clinical studies from the same research center, which included in-office and tray whitening. These authors concluded (1) whitening is most effective when bleaching gel is placed in trays and the trays are used overnight; and (2) tray whitening during the daytime for shorter periods of time was the second most effective whitening method.

Current clinical evidence suggests that 10 % carbamide peroxide is as effective as higher concentrations but results in lower incidence of sensitivity than higher concentrations (Matis et al. 2000). Overnight tray whitening with 10 % carbamide peroxide results in whiter teeth and more durable results than whitening for a few hours during the daytime (Matis et al. 2009a; Cardoso et al. 2010).

Should the recommended treatment be 2–4 weeks overnight for all patients? This treatment regimen is usually adequate for shades A and B (reddish-brownish and reddish-yellowish, respectively) in the Vita Classical A1-D4 shade guide (VITA Zahnfabrik H. Rauter GmbH & Co. KG). When the tooth color has a gray component (C and D shades, Vita Classical A1-D4 shade guide) or when teeth are discolored by the accumulation of tetracycline stains in dentin, teeth do not respond to whitening as well, especially when the stain accumulates in the cervical third.

The prescription of at-home bleaching treatments to child and teenage patients has become a pertinent issue, as parents often ask their family dentists about the possibility of whitening young patients' teeth. Croll (1994) described a protocol for "at-home" tooth bleaching in young patients. According to Croll and Donly (2014), tray whitening of the permanent dentition in children and teenagers is safe and can be performed in a similar manner as for adults. The American Academy of Pediatric Dentistry has published a policy since 2009 on the use of dental bleaching for child and adolescent patients (American Academy of Pediatric Dentistry Council on Clinical Affairs 2015). However, this policy does not address the recommended contact time of the gel with the dentition of young patients. While there is an abundant amount of information on the safety of at-home bleaching gels for adults, studies focused on the tolerable carbamide peroxide concentration and respective contact time with the tooth surface for young patients in terms of pulpal health are lacking. With this in mind, *stronger* evidence may be needed to start recommending tray whitening in child and teenage patients on a regular basis.

6.4.4.2 Tetracycline-Stained Teeth

Tetracyclines and their derivatives are broad-spectrum antibiotics active against both Gram-positive and Gram-negative bacteria as well as infections caused by Mycoplasma, Rickettsia, and Chlamydia. They are also used in rheumatoid arthritis, chronic respiratory diseases, and in the management of periodontal disease (Seymour and Heasman 1995; Tilley et al. 1995; Sánchez et al. 2004; Tredwin et al. 2005). Tetracyclines are contraindicated during pregnancy because they cross the placenta and are toxic to the developing fetus (Sánchez et al. 2004), causing tooth discoloration and enamel hypoplasia if administered during the period of tooth development.

The affinity of tetracycline for dental tissues was first described by Shwachman et al. (1958–1959) in pediatric patients with cystic fibrosis of the pancreas treated with long-term antibiotic therapy. Soon thereafter, Zegarelli et al. (1960) reported similar findings in 38 of 52 children with cystic fibrosis of the pancreas treated with tetracycline. In 1962, Davies and coworkers suggested that tetracycline is deposited on the organic matrix of bones and teeth prior to calcification (Davies et al. 1962). The fluorescence of the pigment and the histological findings confirmed the clinical observation that the pigmentation was due to tetracycline (Wallman and Hilton 1962). When 50 out of 64 newborns that had been given tetracycline in the neonatal period were followed up, 46 of them were found to have yellow or brown discoloration of the teeth, with or without enamel hypoplasia. The greater the total dose of tetracycline per birth weight, the greater was the change. The severity of the stain and its pattern depended on the tetracycline type, dosage, and duration of therapy (Wallman and Hilton 1962).

The affinity of tetracycline for mineralizing tissue is the result of binding to calcium to form a tetracycline-calcium orthophosphate insoluble complex (Gassner and Sayegh 1968; Eisenberg 1975). Chelation with iron has also been reported for tetracycline-induced tooth discoloration (Salman et al. 1985; Bowles and Bokmeyer 1997). Teeth with tetracycline deposits emit yellow fluorescence when observed under ultraviolet light in a darkened room as opposed to the bluish fluorescence characteristic of nonpigmented teeth. The tetracycline stain undergoes degradation by exposure to light, which results in darker staining with age (Abou-Rass 1988).

In 1978, it was reported that minocycline was a viable alternative to treat cases of acne that did not respond to treatment with other tetracyclines (Cullen 1978). Minocycline is a semisynthetic tetracycline derivative used for the treatment of acne for those suffering from rheumatoid arthritis, and chronic respiratory infections (Tilley et al. 1995; Tredwin et al. 2005). In 1980, in a letter to the editor of the *Journal of the American Academy of Dermatology*, a dermatologist described a 42-year-old patient who had been on minocycline, 100 mg two to three times a day, for approximately 4–5 years (Caro 1980). The patient's dental hygienist had noted the development of a gray discoloration of the patient's teeth. Additionally, the patient also stated that she retained a tan for longer than normal and that the skin and fingernails had a gray appearance. The patient's dental crowns had to be restained to match the gray color of the natural dentition. In 1985, a retrospective cohort study found that 4 of 72 patients who had minocycline therapy during adolescence had

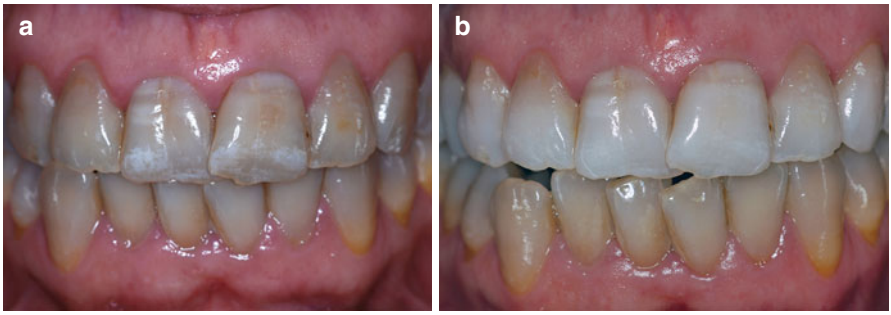

Fig. 6.11 (**a**) 38 year old patient with history of tetracycline ingestion. She was diagnosed with mild tetracycline staining. Additionally, the maxillary central incisors had white spot areas in the incisal third. (**b**) After 3 months of at-home whitening with 10% carbamide peroxide with potassium nitrate and sodium fluoride (Opalescence 10% PF, Ultradent Products, Inc.) in a custom-fitted tray with monthly recalls. Both the tetracycline stains and the white spot areas were successfully camouflaged, in spite of a residual gray band in the cervical third

minocycline-associated tooth discoloration, which occurred after only 4 weeks of treatment in one case (Poliak et al. 1985). Other cases of post-eruptive tooth staining with minocycline have been described (Salman et al. 1985; Bowles and Bokmeyer 1997; Cheek and Heymann 1999). Discoloration caused by minocycline is usually green-gray/blue-gray (Tredwin et al. 2005). Besides discoloration of teeth, minocycline may also cause a blue staining of the sclera, ears, and oral mucosa, which may be irreversible (Dodd et al. 1998; LaPorta et al. 2005; Johnston 2013). Minocycline also causes discoloration of nonvital teeth (Dabbagh et al. 2002; Kim et al. 2010) as discussed in Chap. 8.

The esthetic management of patients with tetracycline-stained teeth is a challenge since the degree of staining varies from mild to severe (Jordan and Boksman 1984).

1. Mild tetracycline staining (Fig. 6.11) is usually very receptive to whitening. This staining is yellow to gray with minimal or no banding and is uniformly spread throughout the tooth, but more confined to the incisal three-quarters of the crown.
2. Moderate tetracycline staining (Fig. 6.12) may vary from a uniform deep yellow discoloration, which is responsive to bleaching, to a dark-gray discoloration band located between the cervical fifth of the crown and the tooth surface located incisally to the band.
3. Severe tetracycline staining (Fig. 6.13) appearing blue-gray or dark gray, accompanied by significant banding across the tooth. Although whitening will somehow lighten these teeth, they may not become esthetically acceptable without bonded restorations.

Clinical studies have demonstrated that mild-to-moderate tetracycline stains can be removed relatively well (Figs. 6.3, 6.11, and 6.12) using the at-home whitening technique with carbamide peroxide in a custom-fitted tray, even though an extended

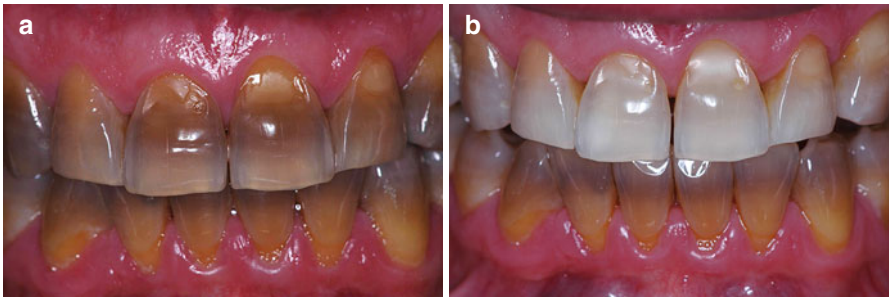

Fig. 6.12 (**a**) A 39-year-old patient with a history of tetracycline ingestion during infancy. He was informed that long-term whitening (2–6 months) might lighten his teeth. However, there was no assurance given of the final whitening result. Patient agreed to carry out the treatment by wearing a custom-fitted tray with 10% carbamide peroxide gel (Opalescence 10%, Ultradent Products, Inc.) every night. Instructions were carefully given to the patient, and a new appointment set up for within 1 month (and every month thereafter). (**b**) Final result after 6 months. No sensitivity was reported at any recall period; no alterations of soft tissues were observed. Patient started whitening the lower arch immediately after the completion of the treatment in the upper arch (Reprinted with Permission from Perdigao J (2010) Dental whitening – revisiting the myths. Northwest Dent 89:19–21, 23–6. (Northwest Dentistry, *The Journal of the Minnesota Dental Association*))

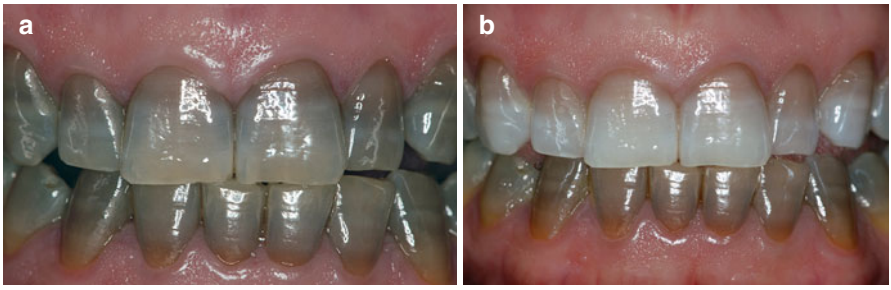

Fig. 6.13 (**a**) Severe tetracycline staining in a 42-year-old patient. (**b**) Final aspect after 6 months of at-home whitening with 10% carbamide peroxide gel with potassium nitrate and sodium fluoride (Opalescence 10% PF, Ultradent Products, Inc.) in a custom-fitted tray with monthly recalls. As expected, and as the patient had been informed, the cervical third was the most resistant area to whitening

treatment time may be required to achieve satisfactory results (Leonard et al. 2003). For mild-to-moderate tetracycline-stained teeth, the recommended treatment is 2–6 months with monthly recalls to evaluate the tooth color and potential side effects (irritation of soft issues, exacerbation of symptoms from TMJ disorders, and tooth sensitivity). The stains that are most difficult to remove are those located at the cervical third. If no improvement in tooth color is observed within the first 3 months, it is unlikely that any improvement will occur (Deliperi et al. 2006). In fact, the maximum lightening effect occurs during the first month (Matis et al. 2006). Therefore, patients with tetracycline-stained teeth must be informed that a residual gray stain may still be perceptible at the end of the treatment at the cervical third. These clinical cases may need a longer bleaching regimen (Matis et al. 2006).

Patients with tetracycline-stained teeth participated in a clinical trial of tray whitening with 10 % carbamide peroxide for 6 months. The 90-month follow-up determined the stability, posttreatment side effects, and patient satisfaction (Leonard et al. 2003). *Shade was stable at least 90 months after treatment*. Patients in this study were overwhelmingly positive about the procedure in terms of shade retention and lack of posttreatment side effects, as 60 % of the subjects reported no obvious shade change or only a slight darkening not noticed by others.

A total of 44 subjects bleached their tetracycline-stained teeth overnight for 6 months using trays with reservoirs, and then followed for 5 years. This was a split-mouth design study that used two of three different concentrations of carbamide peroxide – 10 %, 15 %, or 20 %. More than 65 % of the maximum tooth whitening remained for all carbamide peroxide concentrations. However, 15 and 20 % carbamide peroxide caused significantly more sensitivity than 10 % carbamide peroxide (Matis et al. 2006). In this study, there was a reversal of color change in tetracycline-stained teeth at 5 years.

Although at-home whitening with 10 % carbamide peroxide for up to 6 months remains the first choice for whitening tetracycline-stained teeth, these patients may need to rebleach or touch-up the tooth color approximately 5 years after the original treatment.

6.4.4.3 Fluororis and Fluorosis-Like Enamel Hypocalcifications

Excessive fluoride intake may result is dental fluorosis, which is a hypomineralization of enamel characterized by opaque white areas or discolorations ranging from yellow to dark brown (Horowitz et al. 1984). The severity of fluorosis is correlated with the amount and duration of fluoride ingestion during tooth development (Robinson and Kirkham 1990). In more severe cases the enamel surface becomes pitted, displaying porosities on the surface (Chap. 15). The degree of enamel hypomineralization may vary on different parts of the tooth surface due to the variation in enamel thickness (Fejerskov et al. 1990). Not all white or brown demineralized enamel areas are caused by fluorosis; therefore, they may be considered idiopathic (Cutress and Suckling 1990; Croll 2009) (Fig. 6.14). The term enamel "dysmineralization" has been used when referring to fluorosis-like enamel discolorations (Croll 1990).

Dental fluorosis was referred to as *mottled teeth* in 1916 by McKay and Black because fluoride had not yet been recognized as the cause for this discoloration. McKay and Black summarized very precisely the major characteristics of *mottled teeth* including "the suspicion which is thrown on the water supply in the causative relation" and "localization in definite geographical areas, and its occurrence in the native children thereof".

A precursor of the in-office whitening technique for mottled teeth (e.g., enamel fluorosis) was published by Smith and McInnes in 1942. The successful bleaching technique consisted of direct application of a bleaching mixture of 5 ml of Superoxol

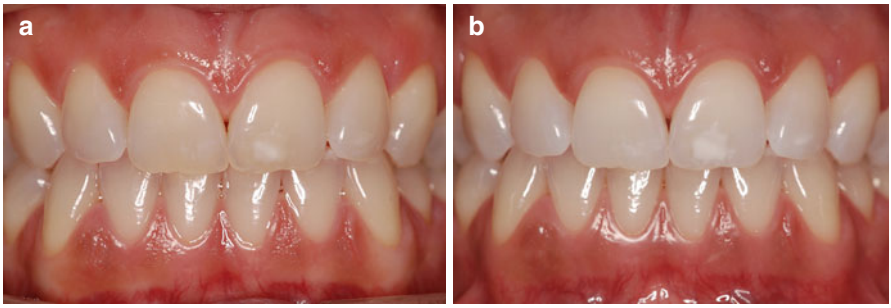

Fig. 6.14 (**a**) A 22-year-old patient whose chief complaint was "yellow teeth." She also had white spots on tooth #9 (FDI 2.1) and tooth #10 (FDI 2.2). (**b**) The at-home whitening treatment with 10 % carbamide peroxide gel with potassium nitrate and sodium fluoride (Whiteness Perfect 10 %, FGM) in a custom-fitted tray highlighted the white spot areas

and 1 ml of ether. Heat was then applied by the patient according to his/her particular tolerance, using a modified soldering iron.

The efficacy of at-home whitening to treat discolorations caused by fluorosis or by idiopathic causes depends on the stain (Haywood 2003). At-home whitening usually lightens enamel brown stains (Fig. 6.4), but it may not work so well for some white areas (Haywood and Leonard 1998; Bodden and Haywood 2003; Perdigao 2010). In case the enamel demineralization is superficial (<0.5 mm), tray whitening may camouflage the white spots without removing them (Fig. 6.11). Conversely, at-home whitening may highlight the whitish areas in cases of deeper white spots (Fig. 6.14). A few applications of a microabrasion suspension (Croll and Cavanaugh 1986a, b; Croll 1997), which contains hydrochloric acid (HCl) and silicon carbide, may be used to disguise the white spots (for more information on enamel microabrasion, please refer to Chaps. 9 and 12). The microabrasion compound is applied by rubbing onto the enamel surface, removing a thin layer of enamel (Donly et al. 1992; Paic et al. 2008). However, it is difficult to predict when enamel microabrasion will remove a stain completely from a tooth (Celik et al. 2013), as the defect may be deeper than microabrasion can reach.

Resin-infiltration after enamel etching with HCl (Chaps. 10 and 13) may be the current treatment modality best suitable for white spots (Senestraro et al. 2013). Robinson et al. (1976) introduced a combination of HCl enamel etching with the application of a low-viscosity resorcinol-formaldehyde resin as a potential cariostatic treatment. Among several research papers on the topic of enamel etching with HCl followed by resin infiltration published in the 2000s, it is worth highlighting two from the same research group. Paris et al. (2007) used confocal microscopy to study resin infiltration of carious lesions using 15 % HCl to etch enamel, followed by immersion in ethanol for 30 s and the application of a commercial dentin adhesive, ExciTE (Ivoclar Vivadent). In 2009, Paris and Meyer-Lueckel described the masking of white spots with resin infiltration using 15 % HCl etching followed by a drying step with ethanol, and a very low viscosity light-cured resin (tetraethylene glycol dimethacrylate). Please refer to Chap. 10 for details.

Haywood and Leonard (1998) reported the use of nightguard vital bleaching with 10% carbamide peroxide to remove a brown stain from the maxillary central incisor of a 13-year-old patient. Without any further treatment, the discoloration had not returned after 7 years.

6.4.4.4 Single-Tooth Whitening

Traumatic injury of the pulp of vital teeth may result in calcific metamorphosis or dystrophic calcification. The pulp produces reparative dentin that may obliterate partially or completely the entire pulp space (Holcomb and Gregory 1967; Stroner and Van Cura 1984; Amir et al. 2001). Teeth with pulpal calcific metamorphosis are often more opaque and darker than adjacent teeth (Fig. 6.5a) and usually respond positively to vitality tests. The presence of reparative dentin does not usually result in delayed responses to the electric pulp tester (Seltzer et al. 1963).

Denehy and Swift (1992) described a method for lightening vital teeth with calcified pulpal spaces. The tray coverage of the adjacent teeth is trimmed to prevent their contact with the bleaching gel (Fig. 6.15). In case the patient desires to lighten the other teeth in addition to the tooth with calcific metamorphosis, these authors recommended bleaching the single discolored tooth first and then making a new full-coverage tray to lighten the entire arch (Denehy and Swift 1992). Another technique being currently used for whitening individual teeth with discoloration caused by calcific metamorphosis uses an especially designed one-tooth tray (Fig. 6.16).

Teeth discolored from trauma usually bleach well, especially vital teeth without radiographic evidence of internal or periapical pathology. One-tooth whitening of teeth with calcific metamorphosis is a very conservative treatment without the need to remove tooth structure.

6.5 Side Effects

Chapters 3, 4 and 5 include a comprehensive description of adverse effects caused by peroxide-based whitening agents.

Although the safety and efficacy of 10% carbamide peroxide is well documented (Matis et al. 1998, 2000, 2009a; Swift et al. 1999; Ritter et al. 2002; Leonard et al. 2003; Zekonis et al. 2003; Meireles et al. 2009), vital tooth whitening, irrespective of method, causes side effects (Li 2011). One study concluded that there were minimal clinical side effects up to 17 years post nightguard vital bleaching with 10% carbamide peroxide (Boushell et al. 2012). In this study, the Löe's gingival index and external cervical resorption findings were considered within normal expectations.

Two treatment-related predictors for side effects are *bleaching gel concentration* and *contact time* (Bruzell et al. 2013).

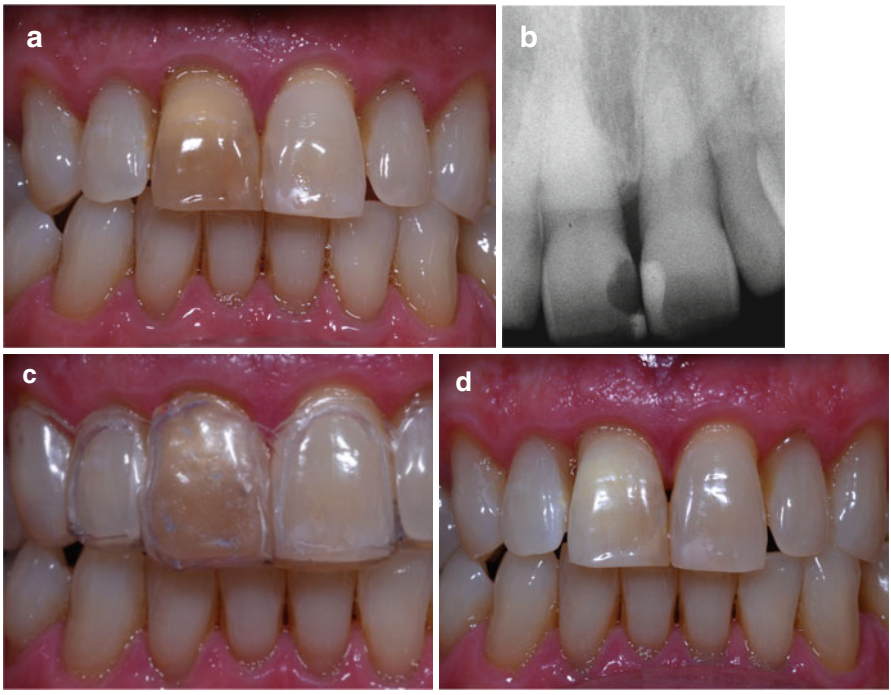

Fig. 6.15 (**a**) A 51-year-old patient with discolored tooth #8 (FDI 1.1) who came to our clinic asking for a second opinion. His dentist had recommended a full-coverage tooth-colored restoration for esthetic reasons. Patient had an old resin-based composite restoration on the mesial-lingual aspect, which he did not want to have removed. Tooth responded positively to cold applied from the lingual surface, although less intensely compare to the other maxillary front teeth. (**b**) Periapical radiograph showing a calcified pulpal space on tooth #8 (FDI 1.1) and a coronal radiolucent image corresponding to the existing resin-based composite restoration. The diagnosis was calcific metamorphosis. (**c**) Patient whitened the discolored tooth with 10 % carbamide peroxide gel with potassium nitrate and sodium fluoride (Opalescence 10 % PF, Ultradent Products, Inc.) in a custom fitted tray using the technique described by Denehy and Swift (1992). The tray coverage of the adjacent teeth was trimmed to prevent their contact with the bleaching gel. Only tooth #8 (FDI 1.1) was bleached. (**d**) After 3 weeks of tray whitening of tooth #8 (FDI 1.1). Patient was extremely satisfied with the result. We recommended the replacement of the discolored restoration, which was still visible after the tooth whitened considerably. Patient decided that he did not want to have the restoration replaced

6.5.1 Changes in Physical Properties and Ultra-morphology of Enamel and Dentin

Most studies indicate that whitening agents containing peroxides have no *permanent* significant deleterious effects on enamel and dentin surface morphology, surface microhardness, and chemical composition (Sulieman et al. 2004; Joiner 2007), while other studies show enamel erosions and changes in the enamel structure, as discussed in Chap. 4 (Haywood et al. 1991; Shannon et al. 1993;

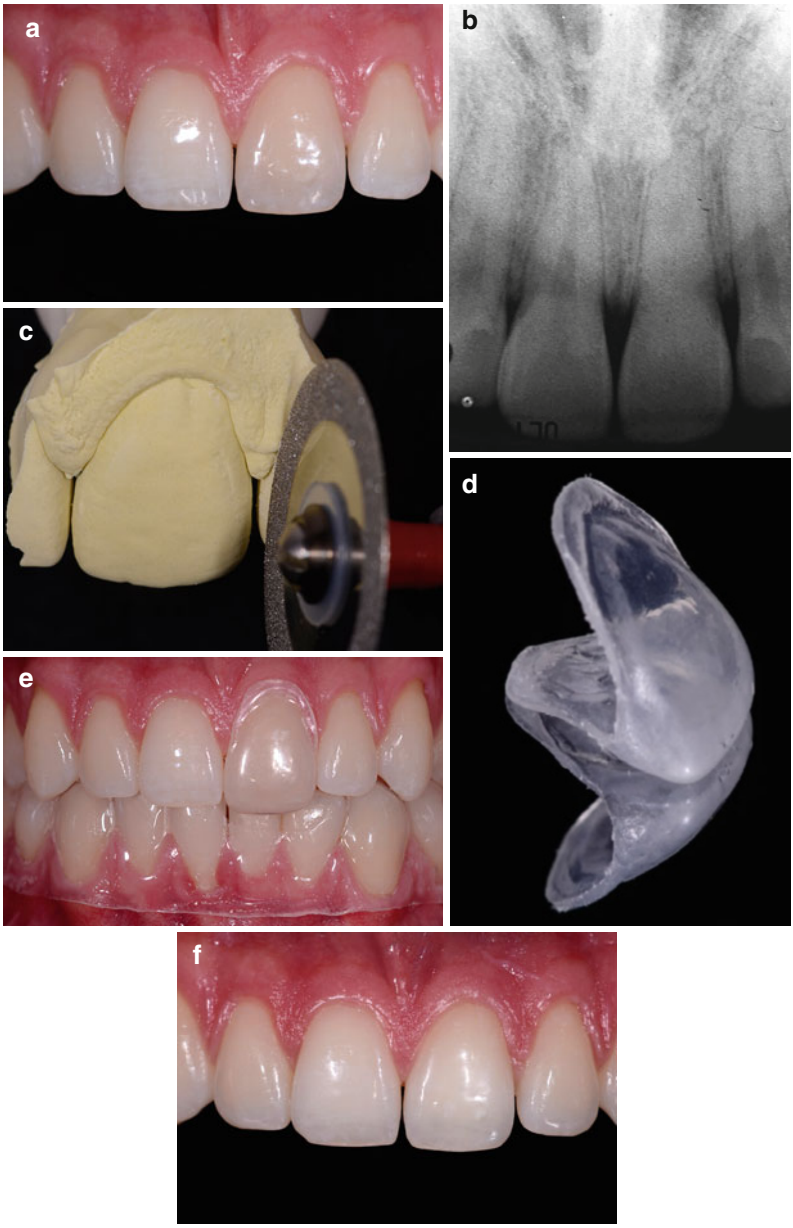

Fig. 6.16 (**a**) A 25-year-old patient with discolored #9 (FDI 2.1). Patient had an accident when she was 21 years old. (**b**) After radiographic exam and clinical exam, the diagnosis was calcific metamorphosis. The tooth responded to cold stimulus applied form the lingual aspect. (**c**) and (**d**) A tray was fabricated to cover only the discolored tooth. (**e**) Single-tooth tray inserted onto tooth #9 (FDI 2.1) and the lower stabilizing tray to prevent the upper single tooth tray from dislodging. The lower tray was not used as a bleaching tray. (**f**) Clinical aspect after 3 weeks of daily whitening with 10 % carbamide peroxide gel with potassium nitrate and sodium fluoride (Whiteness Perfect 10 %, FGM) for 4 h daily. This patient had decided that she did not want to perform the treatment overnight

Bitter 1998). Microhardness has been the most frequently used method for evaluating the effects of peroxides on enamel and dentin (Joiner 2007). Lopes et al. (2002) found no adverse effects on enamel microhardness and surface morphology with 10 % carbamide peroxide, but bleaching with 3 % hydrogen peroxide affected negatively the enamel hardness and surface morphology, which may have been caused by the lower pH of the hydrogen peroxide material. Other authors (Efeoglu et al. 2007) measured significant reduction in the mineral content of the enamel surface (but not dentin) after bleaching with 35 % hydrogen peroxide for 2 h.

It has been reported that peroxides increase the porosity of enamel (Ben-Amar et al. 1995), alter the chemical composition of dentin (Rotstein et al. 1992), reduce the ultimate strength of enamel (Cavalli et al. 2004; da Silva et al. 2005), reduce the flexural strength and flexural modulus of dentin (Tam et al. 2005), and induce morphological alterations in the hydroxyapatite crystallites (Perdigao et al. 1998; Perdigao and Lopes 2006).

A study tested the effects of the Carbopol polymer and glycerin (separately and in association) on the physical properties of enamel and dentin in addition to the effects of 10 % carbamide peroxide (Basting et al. 2005). The baseline microhardness was not recovered during the 14-day posttreatment phase. All the materials tested and their associations changed the microhardness of dental tissues, even in the presence of artificial saliva. There was a tendency toward lower microhardness after treatment with the Carbopol polymer.

The change in chemical composition of dentin post-bleaching may be due to a reduction of the organic component in dentin (Rotstein et al. 1992). Treatment with 30 % hydrogen peroxide causes changes in the chemical structure of the dentin and cementum, making them more susceptible to degradation. Exposure to 30 % hydrogen peroxide for 24 h also causes a significant decrease in the hardness and Young's modulus of intertubular dentin, as measured with AFM and nanoindentation (Chng et al. 2005). The effect of hydrogen peroxide on dentin may be a result of both its strong oxidizing action and its low pH. Peritubular dentin is more resistant to the effects of hydrogen peroxide than intertubular dentin, which may be a result of the higher mineral content of peritubular dentin. Enamel treatment with carbamide peroxide for 6 h per day during 14 days, followed by storage in artificial saliva in between each application, resulted in significant reduction of the ultimate tensile strength of enamel, regardless of the concentration of carbamide peroxide (10 %, 15 %, 16 % and 20 %) (Cavalli et al. 2004).

Another study (da Silva et al. 2005) evaluated the effects of peroxide bleaching regimens on the ultimate tensile strength of human enamel. All different bleaching procedures (7.5 % hydrogen peroxide, 30 min per day for 14 days; 10 % carbamide peroxide 6 h per day for 5 days; 10 % carbamide peroxide 6 h per day for 14 days; 35 % carbamide peroxide, two applications of 30 min with a 5-day interval between applications; 37 % carbamide peroxide, two applications of 30 min with a 5-day interval between applications; 35 % hydrogen peroxide, two applications of 15 min with a 7-day interval between applications) significantly reduced enamel ultimate tensile strength. This reduction in the ultimate tensile strength was accompanied by changes

in the enamel internal micromorphology, with a possible loss of interprismatic matrix and presence of porosities at the prism-fractured ends in the bleached fractured enamel might indicate that some intraprismatic material was also lost. These features were more pronounced in the group treated with 35 % hydrogen peroxide.

Different at-home whitening regimens with several concentrations of carbamide peroxide have also been shown to reduce both the flexural strength and flexural modulus of dentin (Tam et al. 2005). This reduction was not observed for in-office treatment with hydrogen peroxide possible due to the shorter treatment time compared to that of carbamide peroxide.

Ultra-morphological studies with Transmission Electron Microscopy (Perdigao and Lopes 2006) revealed that the treatment of enamel with 10 % carbamide peroxide did not result in changes in the morphology of the crystallites immediately below the enamel surface (Fig. 6.17), but this alteration was pronounced when enamel was treated with 38 % hydrogen peroxide following the recommendations for in-office whitening (Perdigao and Lopes 2006). This is in agreement with other authors, who found that the most severe changes in enamel topography occurred with products of lower pH (Shannon et al. 1993). The periphery of the crystals was not well defined as in the control group denoting a mottled etched pattern and loss of structural arrangement.

When teeth are exposed to a 10 % carbamide peroxide gel for 6 h they lose 1 μg/mm^2 of calcium. If teeth are exposed to a cola beverage for 2.5 min (the time equivalent to drinking a 16 oz. beverage) the amount of calcium lost from these teeth is also 1 μg/mm^2 (McCracken and Haywood 1996). Orange juice causes more harm to enamel surfaces than whitening materials (Sulieman et al. 2004). Therefore, *adverse effects on enamel and or dentin caused by whitening materials may reflect the pH of the formulation used rather than the bleaching agent itself* (Sulieman et al. 2004). Some of these alterations caused by peroxide bleaching materials on the dental hard tissues may be transient and reversed when treated with a fluoride solution.

The data obtained in all the in vitro studies mentioned in this section may have to be analyzed with caution. The clinical relevance of some of the methods used to assess the effects of peroxides on dental hard tissues may not reflect the clinical conditions accurately. Studies have used whitening agents of low pH, which may have caused microscopic erosion and other alterations of the substrate that were not accounted for in the research methodology (Joiner 2007).

6.5.2 Decrease in Bonding Effectiveness

As discussed in Chap. 4, peroxide bleaching materials significantly decrease the bond strength of resin-based composite to bleached enamel and dentin, both for at-home vital whitening and for intra-coronal nonvital whitening (Cavalli et al. 2001; Shinohara et al. 2005). For enamel specifically, 10 % carbamide peroxide reduces the bond strengths of resin-based composite to etched enamel (Cvitko et al. 1991; Barghi and Godwin 1994; Ben-Amar et al. 1995; Spyrides et al. 2000), and increases

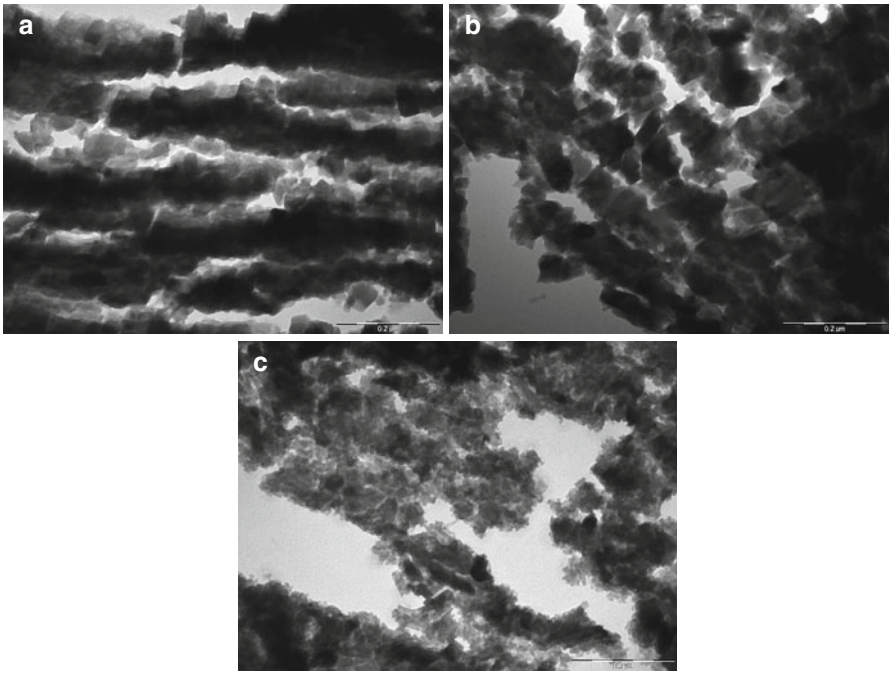

Fig. 6.17 (**a**) Transmission electron micrograph of untreated human enamel. Original magnification = ×150,000. (**b**) Transmission electron micrograph of human enamel bleached with 10 % carbamide peroxide gel (Opalescence 10 %, Ultradent Products, Inc.) for 2 weeks, 8 h daily. Specimens were stored in artificial saliva at 37 °C between bleaching sessions. Original magnification = ×150,000. (**c**) Transmission electron micrograph of human enamel bleached with 38 % hydrogen peroxide (Opalescence Xtra Boost, Ultradent Products, Inc.), four consecutive applications of 15 min each. The ultra-morphology of the enamel crystallites is substantially different from those shown in **a** and **b**. Original magnification = ×150,000

enamel surface porosity (Ben-Amar et al. 1995). This reduction may be as high as 76 % of the bond strengths to unbleached enamel (Spyrides et al. 2000). Removal of surface enamel prior to bonding restores bond strengths to normal level (Cvitko et al. 1991). The use of acetone-based adhesives or drying agents, such as 70 % alcohol and acetone, may also restore bond strength of resin-based composite to enamel immediately after bleaching (Barghi and Godwin 1994; Niat et al. 2012).

The enamel and dentin bond strengths remain low for the first 2 weeks post-bleaching. After a lapse of 2 weeks, the bond strengths return to the level at of untreated substrates (Cavalli et al. 2001; Shinohara et al. 2005). Increased concentration of carbamide peroxide did not extend the time needed prior to bonding.

Dentists must wait for at least 2 weeks after the patient completes the whitening treatment prior to performing any adhesive restorative procedure.

Resin-enamel interfaces in enamel bleached with 10% carbamide peroxide exhibit extensive nanoleakage compared to those of the unbleached control group (Lai et al. 2002). Reduction of resin-enamel bond strength in bleached etched enamel may be caused by a delayed release of oxygen that affects the polymerization of resin components (Lai et al. 2002). Although enamel bond strengths to beached enamel are reduced after bleaching, this reduction can be reversed with treatment with an antioxidant prior to bonding, such as 10% sodium ascorbate (Lai et al. 2002; Türkün and Kaya 2004).

6.5.3 Pulp Injury

Because this topic is extensively and elegantly discussed in Chap. 5, we will only cite two studies that suggest the relatively innocuous nature of 10% carbamide peroxide to the human pulp. To study the pulp injury potential of 10% carbamide peroxide, an ex vivo study included 16 patients who had four premolars scheduled to be extracted for orthodontic reasons (Fugaro et al. 2004). Tooth #5 was bleached for 4 days, tooth #12 was treated for 2 weeks, tooth #21 was bleached for 2 weeks followed by 2 weeks without treatment and tooth #28 was not treated, serving as the control. All whitening treatments were performed overnight with 10% carbamide peroxide. All teeth were extracted at the same time and prepared for histological evaluation at two different research centers. Slight pulpal changes caused by 10% carbamide peroxide were detected in 16 of the 45 bleached teeth. Neither moderate nor severe reactions were observed. The slight histological changes sometimes observed after bleaching tend to resolve within 2 weeks posttreatment (Fugaro et al. 2004).

The apparent safety of nightguard vital bleaching may be due to the response by odontoblasts with increased heme oxygenase-1 (HO-1) production. Seventeen intact first premolars scheduled for orthodontic extraction were bleached with 10% carbamide peroxide for 4 h immediately preceding extraction. Fourteen additional premolars from the same individuals were not bleached. Upon extraction and histological evaluation, no significant differences were found in the concentration of the enzyme HO-1 in the pulp, which is normally increased in cells subjected to oxidative stress (Anderson et al. 1999). Please refer to Chap. 5 for in-depth information on the topic of pulp injury with whitening agents.

6.5.4 Tooth Sensitivity

Since this topic is discussed in more detail in Chaps. 4 and 5, we will include here additional information on tooth sensitivity related to at-home whitening with a custom-fitted tray.

Tooth sensitivity seems to be the most common lateral effect of whitening with carbamide peroxide, which results from the penetration of peroxides into the pulp space, but sensitivity usually relapses with termination of treatment (Haywood

Table 6.2 Predictors for tooth sensitivity during at-home whitening treatment

Gingival recession
History of sensitive teeth
Contact time of the bleaching gel with teeth
Changing the bleaching gel more than once/day
Concentration of carbamide peroxide > 10 %

1997a). A slightly higher incidence of sensitivity is associated with higher concentrations of carbamide peroxide (Matis et al. 2000). Another factor that could play a role in sensitivity is the pH of the bleaching gel as the pH of whiteners used with the at-home technique can be as low as 5.6 (Price et al. 2000).

In a clinical study, while 71 % of subjects who used 20 % carbamide peroxide reported tooth sensitivity, only 37 % of subjects who used 10 % carbamide peroxide experienced tooth sensitivity (Basting et al. 2012). The chance of *moderate* tooth sensitivity is 10 %, while there is a very slight chance of *severe* sensitivity (4 %). Patients can be advised that there is a 40–50 % chance that they will experience *mild* sensitivity with 15 % or 20 % carbamide peroxide (Jorgensen and Carroll 2002). Sensitivity tends to occur early in treatment and diminishes with time. As reported in some studies, sensitivity rates are similar for the bleaching gel and placebo, indicating that the active peroxide ingredient is not always responsible for causing tooth sensitivity.

There are a few predictors for tooth sensitivity associated with at-home whitening (Table 6.2). Patients with gingival recession are more likely to experience tooth sensitivity during home whitening treatment (Jorgensen and Carroll 2002). Other predictors are a history of sensitive teeth, enamel craze lines, and patients who changed the whitening solution more than once a day, contact time of the bleaching agent with the teeth, or the utilization of higher concentrations of carbamide peroxide such as 20 % (Haywood 1997a; Leonard et al. 1997; Gerlach et al. 2000; Cardoso et al. 2010; Basting et al. 2012; Özcan et al. 2014). In a clinical study in which patients used a 10 % carbamide peroxide gel, subjects whose bleaching regimen was 8 h daily experienced significantly more sensitivity than those who bleached for 1 h per day (Cardoso et al. 2010).

A multicenter, questionnaire-based prospective study reported tooth sensitivity after 14 days and after 1-year posttreatment (Bruzell et al. 2013). The prevalence of experienced tooth sensitivity at the first recall was independent of bleaching procedure (at-home vs. in-office), whereas prevalence of gingival irritation was higher after in-office treatment. At the second follow-up (1 year), two and three patients reported side effects attributed to the bleaching treatment in the at-home and in-office groups, respectively.

Peroxides diffuse very quickly into dentin reaching the pulp chamber, but the rate of penetration depends on the concentration and composition of the whitening agent and the thickness of the hard tissue (Hanks et al. 1993; Thitinanthapan et al 1999; Gokay et al. 2000). Some specific brands of 10 % carbamide peroxide gels result in greater concentration of hydrogen peroxide within the pulp chamber than other similar concentrations, regardless of the diffusion time (Hanks et al. 1993). In extracted teeth, significantly less peroxide reaches the pulp from a 15 % carbamide peroxide gel (equivalent to 5.25 % hydrogen peroxide) than from a 5 % hydrogen

peroxide gel within a 15-min period (Cooper et al. 1992), which attests the lower rate of decomposition of the carbamide peroxide compared to hydrogen peroxide. When hydrogen peroxide is applied for periods longer than 15 min, it is capable of diffusing through 0.5 mm of patent dentin, damaging pulpal tissue (Hanks et al. 1993). Clinically, 0.5 mm corresponds to areas of deep noncarious cervical lesions. In case teeth are already sensitive prior to starting the treatment, this might indicate that the dentin tubules are patent, which would make bleaching strongly contraindicated. Pulpal tissue has limited volume to expand from injury; therefore, it may have a compromised response to inflammatory stimuli because of pressure associated with edema, causing sensitivity (Heyeraas and Kvinnsland 1992). In case heat is used, such as in the jump-start technique, pulpal enzymes may be significantly inhibited (Bowles and Thompson 1986).

Manufacturers have added potassium nitrate and sodium fluoride to the composition of their whitening gels to prevent sensitivity during at-home bleaching treatment. Carbamide peroxide gels for the at-home technique typically contain 0.11 % (w/w) fluoride and 3 % (w/w) potassium nitrate (Chen et al. 2008).

The use of sodium fluoride daily after bleaching does not affect the bleaching efficacy of carbamide peroxide but reduces the intensity of tooth sensitivity (Armênio et al. 2008). The application of a 5 % potassium nitrate and fluoride gel in the bleaching tray has been shown to reduce the incidence of tooth sensitivity in patients undergoing at-home whitening with 10 % carbamide peroxide in a custom-fitted tray (Haywood et al. 2001).

Clinical studies have reported that potassium nitrate and sodium fluoride added to a 10 % carbamide peroxide gel reduced sensitivity over a 2-week at-home treatment when compared to a 10 % carbamide peroxide gel without desensitizer (Tam 2001; Browning et al. 2008; Navarra et al. 2014). The application of 3 % potassium nitrate and 0.11 % fluoride desensitizing agent for 30 min prior to at-home whitening with 10 % carbamide peroxide decreases tooth sensitivity when compared with a placebo in a population at risk for tooth sensitivity (Leonard et al. 2004). Another clinical trial reported that the use of 5 % potassium nitrate and 2 % sodium fluoride prior to at-home vital bleaching with 16 % carbamide peroxide did not affect the bleaching efficacy but reduced the number of days during which patients experienced tooth sensitivity (Kose et al. 2011). To corroborate the effectiveness of potassium nitrate and sodium fluoride, their use prior to in-office bleaching with 35 % hydrogen peroxide also reduces tooth sensitivity significantly (Tay et al. 2009; Wang et al. 2015).

Some clinicians recommend brushing with potassium-nitrate-containing toothpaste for 2 weeks before initiating the at-home whitening treatment with a custom-fitted tray. This pretreatment has been shown to be beneficial to patients by reducing tooth sensitivity during whitening (Haywood et al. 2005). Dentists have also recommended the application of the desensitizing toothpaste in the bleaching tray for up to 30 min (Haywood 2003). This recommendation may not be safe, as the toothpaste may contain ingredients that cause soft tissue reactions when the toothpaste is left in contact with the soft tissue for that period of time.

Other desensitizers have been used. A clinical study compared the efficacy of two desensitizers included in the composition of carbamide peroxide gels. Potassium

nitrate was found to be as effective as amorphous calcium phosphate (ACP) to prevent sensitivity (Matis et al. 2007). However, 15 % carbamide peroxide gel with potassium nitrate and sodium fluoride achieved significantly greater whitening results than a 16 % carbamide peroxide gel with amorphous calcium phosphate (ACP) (Matis et al. 2007). When comparing potassium nitrate with potassium oxalate as desensitizers included in the composition of 10 % carbamide peroxide gels, it was reported that the gel with potassium oxalate caused significantly more sensitivity at 1 week than the 10 % carbamide peroxide gel with potassium nitrate or 10 % carbamide peroxide gel without any desensitizer (Perdigao et al. 2013).

- Brushing with potassium-nitrate-containing toothpaste for 2 weeks prior to the bleaching treatment reduces tooth sensitivity during the treatment.
- Potassium nitrate is the recommended desensitizer to use with at-home whitening treatments (Tam 2001; Matis et al. 2007; Wang et al. 2015), as it reduces postoperative sensitivity without reducing efficacy when added to 10 % carbamide peroxide (Browning et al. 2008).
- The penetration of peroxides into dentin can be reduced by application of a desensitizing agent or a fluoride varnish (Hannig et al. 2011).

6.6 Recommendations for Dental Professionals

- Refrain from being too optimistic when predicting the post-treatment tooth color.
- If the patient's shade falls in the C or D range in the Vita Classical A1-D4 shade guide, the final result will not be as pleasant as that for patients in the A or B shade range.
- Prescribe 10 % carbamide peroxide with desensitizer included in the bleaching gel.
- Inform patient that sensitivity is likely to occur; prescribe potassium-nitrate-containing toothpaste to replace patient's regular toothpaste. This toothpaste may be more effective if used during 2 weeks prior to starting the bleaching regimen.
- The application of a gel of potassium nitrate and sodium fluoride prior to at-home bleaching decreases tooth sensitivity and reduces the number of days during which patients experience tooth sensitivity.
- Recommend that patient wears the bleaching tray preferably overnight.
- Give patient written instructions:
  - Brush and floss before placing the tray.
  - Insert the tray snugly over the teeth; gently tap the tray to move the gel into place and adapt tray to the teeth
  - Watch for excess gel along the tray border; remove it with a toothbrush or with a cotton swab. Rinse mouth with water and do not swallow the gel.
  - Wear tray for the time recommended.
  - Brush and floss after removing the tray; wash tray thoroughly with water to remove the residual gel.

- Smokers should refrain from smoking from 2 h prior to inserting the tray in the mouth to 2 h after removing the tray

6.7 Frequently Asked Questions

To summarize the contents of this chapter we present below a series of questions and answers on the topic of at-home whitening.

6.7.1 Patients

Do I need to refrain from a potentially staining diet during and after tray whitening?

Current evidence from controlled clinical trials suggests that coffee, tea, red wine, do not interfere considerably with the outcome of whitening.

Is it OK that I wear my tray filled with 10 % carbamide peroxide for 2 h per day?

That would be perfectly fine, but it will take longer to achieve the desired lightening effect compared to bleaching overnight. Current clinical evidence suggests that overnight tray whitening results in whiter teeth and more durable results than whitening for a few hours during the daytime.

My teenage daughter wants to have her teeth bleached. Is it safe?

As discussed in Sect. 6.4.4.1 our opinion is that we may need *stronger* evidence to recommend tray whitening in child and teenage patients.

I have some yellowish tooth-colored fillings in my front teeth. Will they get whiter if I bleach my teeth?

They may look slightly lighter as a result of the oxidation of the surface pigment (Fig. 6.10c), but in most cases tooth-color filling materials will not bleach (Fig. 6.8). Existing tooth color fillings (resin-based composite restorations) may need to be replaced after the whitening treatment is completed to make your smile look uniform.

How does the efficacy of OTC bleaching products compare to that of the dentist-prescribed at-home whitening?

One of the very few *independent* studies, mentioned earlier in this chapter, concluded that 6 % hydrogen peroxide whitening strips applied twice a day for 30 min each for 2 weeks are not as effective as at-home whitening with 10 % carbamide peroxide overnight for 2 weeks. Another study concluded that some paint-on

products are ineffective. A recent systematic review reported that OTC products should be assessed independently.

OTC bleaching strips also cause more tooth sensitivity and gingival irritation than at-home tray whitening prescribed by a dental professional. In summary, at-home whitening with 10 % carbamide peroxide in a custom-fitted tray is safer and more effective than OTC bleaching treatments. The results obtained with OTC whitening are not as pleasant and the procedure is not as safe as those methods currently prescribed by a dental professional.

6.7.2 Dental Professionals

You usually prescribe 10 % carbamide peroxide for your clinical cases of at-home whitening in a custom-fitted tray. What is the evidence?

Dentist-prescribed overnight bleaching with carbamide peroxide in a custom-fitted tray is the safest and most effective method of tooth whitening. Although higher concentrations of peroxides result in a faster rate of whitening than lower concentrations they reach a similar final result. Additionally, lower concentrations of carbamide peroxide result in lower incidence of sensitivity than higher concentrations. The durability of the whitening effect is another factor that needs to be taken into account. Patients who had whitened their teeth using 10 % carbamide peroxide in a custom-fitted tray were contacted at least 10 years posttreatment. Patient satisfaction with tray whitening was determined to last an average of 12.3 years posttreatment.

Do you also perform in-office whitening? Or do you only prescribe at-home whitening?

Some patients choose not to be treated with at-home tray whitening for several reasons. We always inform these patients that we also perform in-office whitening, but that several sessions may be needed to obtain an acceptable final tooth color. Patients must also be informed that in-office whitening is more likely to cause tooth sensitivity than at-home whitening with a custom-fitted tray. Other potential adverse effects of in-office whitening with >30 % hydrogen peroxide are described in Chaps. 4, 5, and 7.

Does fluoride help in preventing sensitivity during my treatment?

The use of sodium fluoride daily after bleaching does not affect the bleaching efficacy of carbamide peroxide and may reduce the intensity of tooth sensitivity.

Do higher concentrations of carbamide peroxide result in whiter teeth?

Current evidence from controlled clinical trials suggests that higher concentrations result in a faster rate of whitening than lower concentrations with a similar final result. At 1 year, 16 % carbamide peroxide concentration does not increase the

longevity of the whitening effect compared to 10 % carbamide peroxide. A slightly higher incidence of sensitivity is associated with higher concentrations.

What concentration of peroxide is best for our patients?

From the previous question/answer, the efficacy of 10 % carbamide peroxide is similar to that of higher concentrations; therefore, we only recommend 10 % carbamide peroxide, except for cases of external whitening of lightly discolored endodontically treated teeth in which the stain is recent.

What is the recommended duration of at-home whitening treatments?

We currently inform our patients that discolorations caused by aging and chromogenic foodstuff usually achieve a satisfactory lighter color after 2–4 weeks of overnight tray whitening with 10 % carbamide peroxide. However, this result depends on the patient's compliance. The ideal final color is not reached if patient does not wear the tray daily. For gray tooth shades, C's and D's in the Vita Classical A1-D4 shade guide, we recommend 4–6 weeks of overnight tray whitening with 10 % carbamide peroxide, with the prospect of a residual darker area at the cervical third of the teeth.

What is the best desensitizing agent to use with peroxides?

There is strong evidence that the only desensitizer that has been shown to reduce sensitivity during whitening treatment is potassium nitrate. The evidence also shows that the combination of potassium nitrate with sodium fluoride in toothpaste is capable of preventing tooth sensitivity when used for 2 weeks prior to starting the whitening treatment, or as a gel when applied 30 min prior to the inserting of the bleaching tray. The inclusion of potassium nitrate in the composition of the carbamide peroxide whitening gel may also help in the prevention of tooth sensitivity.

Is the jump-start technique more efficient than the at-home technique alone?

Current evidence from controlled clinical trials suggests that tray whitening alone results in similar outcomes compared to the *jump-start* technique. However, some clinicians claim that the jump-start technique motivates patients, as the initial results are visible immediately after the initial in-office whitening component.

Can tetracycline-stained teeth be bleached with at-home whitening regimen using a carbamide peroxide gel?

Yes, but it depends on the intensity of the stain. Please refer to Sect. 6.4.4.2.

Do dentists have to wait for 2 weeks after the conclusion of the whitening treatment before they can place bonded restorations on recently whitened teeth? Does this apply to internal and to external whitening?

Yes. This principle applies to all peroxide-based whitening regimens and techniques.

How do we know that the concentration written on the syringe corresponds to the concentration of peroxide inside the syringe?

The chance of a mismatch between the advertised concentration and the actual concentration is very high. For bleaching some gels, the actual concentration is between 16 % and 36 % lower than the label indicates.

I heard in a dental convention and recently read in a manufacturer's site that gel in conventional whitening trays is only strongly active for 25–35 min…

As discussed in Sect. 6.3.2, more than 50 % of the active agent is available after 2 h. The percentage of carbamide peroxide recovered from tray and teeth is 10 % at 10 h.

Does at-home whitening affect the vitality of the pulp?

For in-depth information on pulpal inflammation triggered by peroxide-based bleaching agents, please refer to Chap. 5. In case heat is used, such as in the jump-start technique, pulpal enzymes may be significantly inhibited.

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In-Office Whitening

7

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Abstract

In this chapter, the step-by-step procedure of in-office whitening (or in-office bleaching) and the efficacy and side effects of this bleaching modality will be presented. Other characteristics of this protocol such as the number of clinical appointments required to achieve effective whitening, concentration of the bleaching products, the effects of dentin dehydration and demineralization on the final outcome, as well as bleaching-induced tooth sensitivity will be addressed. At the end, some frequently asked questions will be answered.

7.1 Introduction

In-office whitening is a treatment option in the dental bleaching armamentarium. Not every patient can wear tray delivery products. Some patients do not adapt well to the at-home protocol due to the need of the daily usage of a bleaching tray as well as the need to wait for some weeks to see the results of the treatment. In some cases, in-office bleaching is performed to motivate patients before starting an at-home protocol in the combined or jump-start technique.

This is the reason why in-office bleaching should be considered an alternative option to the more traditional and safer at-home bleaching procedures. Several aspects of in-office bleaching modality will be discussed in this chapter to provide

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clinicians with a better understanding of the protocol and particularities of the technique and facilitate its incorporation into the daily practice with confidence.

7.2 Efficacy

In-office whitening is performed with high concentrations of hydrogen peroxide (HP), usually ranging from 15 to 40 %. Regardless the concentration of the bleaching gel, HP is the active molecule that acts as a strong oxidizing agent through the formation of free radicals, reactive oxygen molecules, and HP anions (Bowles and Ugwuneri 1987).

Previous studies have claimed that tooth shade is affected by the intrinsic organic chromophores present in the dental structure (Fuss et al. 1989; Watts and Addy 2001; Sulieman et al. 2003; Joiner 2006). Organic chromophores are colorful chemical molecules, which consist of complex molecules such as aromatic compounds or bioinorganic metallic complexes such as chelates (Eimar et al. 2012b). These chemical compounds can be easily identified with Fourier Transform Infra-Red (FTIR) and Raman spectroscopies (Eimar et al. 2012b). However, studies using these techniques researchers were never able to detect any of these potential chromophores (Fattibene et al. 2005; Eimar et al. 2011, 2012a). More recently, researchers suggested that HP whitens teeth by mere oxidation of the transparent organic matrices. This process turns them whiter and more opaque, which results in whiter dental appearance (Kawamoto and Tsujimoto 2004; Eimar et al. 2012b).

Some particularities of in-office bleaching should, however, be discussed. The lighter appearance of teeth immediately after an in-office bleaching session cannot be only attributed to the oxidizing action of the HP into the dental organic substrate. Apart from oxidization, dental dehydration and enamel demineralization are expected to occur. As in-office bleaching is usually performed under isolation (rubber dam or light-cured gingival barrier plus lip and cheek retractors), dental dehydration will be always associated with the procedure. This effect is demonstrated when a rubber dam is used to isolate the teeth even for short periods of time (Fig. 7.1). A recent research paper (Burki et al. 2013) demonstrated that the application of a rubber dam alone, even for a short period of 10 min, would cause a lightening of the tooth for a ΔE of 7.3, without any actual bleaching having occurred. Dehydration of teeth can make them appear whiter by increasing enamel opacity. Light can no longer scatter from hydroxyapatite crystal to crystal (Fondriest 2003; Burki et al. 2013). Loss of translucency on dehydration causes more reflection, masking the underlying color of dentin, and thus appears lighter. This "lightened" teeth (by dehydration) return to a normal color after a period of hours or days (Fig. 7.1).

Apart from dehydration, enamel demineralization results from the low pH of most bleaching products currently available. Most in-office bleaching gels are delivered in low pH because they are more stable in acid solutions than in basic solutions. When HP is to be stored, a weak acid is usually added to the solution to prevent it from decomposing (Chen et al. 1993), which makes the bleaching product acidic

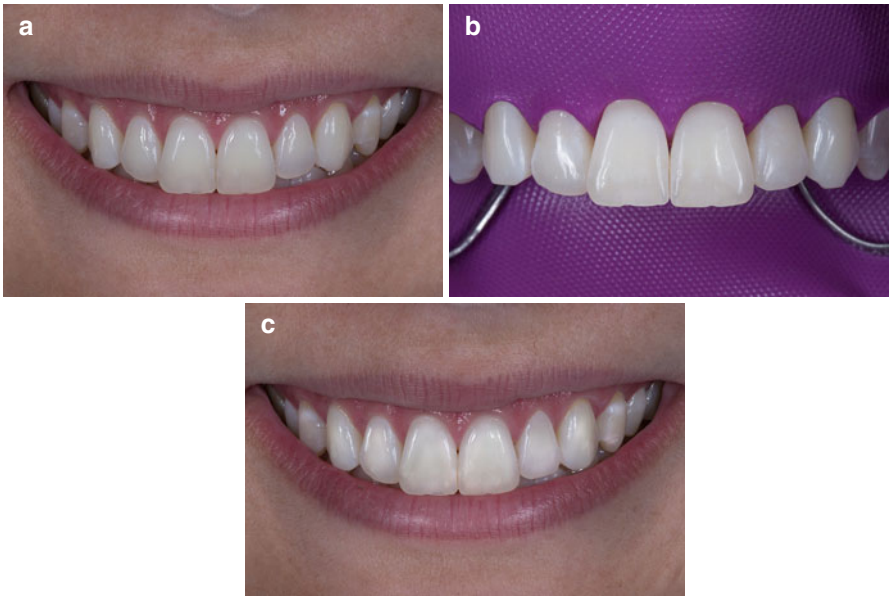

Fig. 7.1 The effect of rubber dam on lightness can be observed in these three photographs. (**a**) Patient's smile before rubber dam isolation. (**b**) The rubber dam was placed in the upper dental arch and left undisturbed for 10 min. (**c**) The effect of dehydration is observed immediately after rubber dam removal (Images provided by Camilo Andrés Pulido Mora, DDS, MS)

enough to produce enamel demineralization. The pH of in-office bleaching gels may vary from 2.0 to 9.0 (Price et al. 2000; Freire et al. 2009; Majeed et al. 2011).

Therefore, taking into consideration that transient dental dehydration and demineralization occurs concomitantly to the permanent effect of dental bleaching during in-office bleaching, the result of in-office bleaching cannot be assessed immediately after the in-office bleaching session. The reliability of color measurements is questionable if carried out immediately after treatment, leading to the conclusion that in-office whitening is as efficient as at-home bleaching.

The effect of color change when evaluated before complete dental rehydration occurred in the study of Matis et al. (2007). The graphic below shows the changes in L^* (Fig. 7.2) and b^* (Fig. 7.3) parameters after a single in-office bleaching session. In the "x" axis, time 0 means the color taken immediately after in-office bleaching and the other times represent weekly measures up to 6 weeks. In general, color change (L^* and b^* parameters) seems much more pronounced when they were measured immediately after the procedure (time 0), with significant reductions of L^* and increases of b^* after 1–2 weeks due to the dental rehydration and remineralization. Therefore, the "real" bleaching effect (produced by oxidization) can only be measured 1–2 weeks after the end of the in-office bleaching.

The difference between the "whitening outcome" observed immediately after bleaching and that measured 1 week later has been erroneously interpreted as color rebound, with some researchers concluding that in-office bleaching is not as efficient

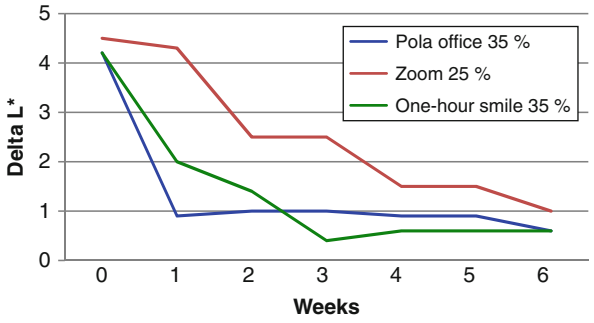

Fig. 7.2 Variation in the ΔL^* in a 6-week period after a single in-office bleaching with different products (Adapted from Matis et al. 2007)

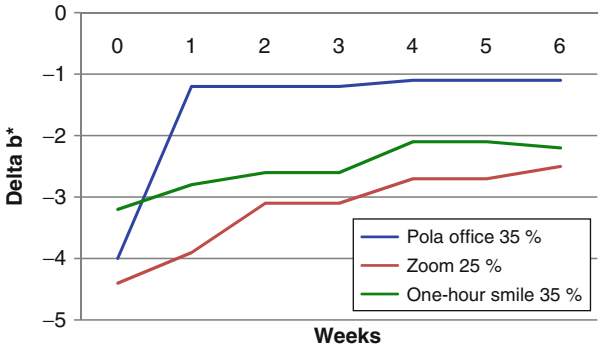

Fig. 7.3 Variation in the Δb^* in a 6-week period after a single in-office bleaching with different products (Adapted from Matis et al. 2007)

as at-home bleaching (Matis et al. 2007). Several studies have demonstrated that 1 week of at-home bleaching with 10 or 16% carbamide peroxide gel usually results in a change of two to four shade guide units in the value-oriented Vita Classical A1-D4™ shade guide (VITA Zahnfabrik H. Rauter GmbH & Co.KG, Bad Säckingen, Germany) (Zekonis et al. 2003; Bernardon et al. 2010; da Costa et al. 2010; Rezende et al. 2013). This is approximately equivalent to the change reported after a single in-office bleaching session with 35% HP gel when used for 45–60 min (Zekonis et al. 2003; Bernardon et al. 2010; Kossatz et al. 2011; Reis et al. 2011b).

There are also other factors that may explain the general belief that in-office bleaching is not effective. It is known that the whitening effect is related to the concentration, application time, and the number of changes of the in-office bleaching gel (Dietschi et al. 2006; Joiner 2006; Matis et al. 2007). In an ongoing systematic review of the literature (Luque-Martinez et al. 2016) we observed a high heterogeneity among studies in many issues, such as type of materials, concentration of the products, and significant variations in in-office bleaching protocols.

Inefficient bleaching protocols will not lead to a satisfactory bleaching outcome. For instance, some studies performed only a single in-office bleaching session (da Costa et al. 2010; Giachetti et al. 2010; Moghadam et al. 2013; Pintado-Palomino et al. 2015), which is not enough to reach patient's satisfaction (de Silva Gottardi et al. 2006; Salem and Osman 2011). At least two or three bleaching sessions may need to be performed to obtain a similar whitening degree of a 2 or 3-week at-home

bleaching (Marson et al. 2008b; Tay et al. 2009; Bernardon et al. 2010; Basting et al. 2012; Reis et al. 2011b, 2013).

Similar variation occurs in regard to product application time. While a 40–50-min application is related with a significant whitening outcome, there are reports of shorter application times, such as 10–20 min (Auschill et al. 2005; Giachetti et al. 2010; Mehta et al. 2013). A very recent clinical study reported that a single 15-min application of the 35 % HP does not achieve the same degree of whitening produced by two and three 15-min applications of the same product (Kose et al. 2015).

Some manufacturers advocate the application of their products with light activation (quartz–tungsten halogen light curing units, LEDs, and lasers) to optimize the bleaching outcome (Ziemba et al. 2005; Kishi et al. 2011; Bortolatto et al. 2014). The benefits of this association are rather controversial (Buchalla and Attin 2007; He et al. 2012), but it seems to be useless for high-concentrated HP gels (Marson et al. 2008b; Alomari and El Daraa 2010; Kossatz et al. 2011; He et al. 2012). For low-concentrated HP gels this light association may have some benefits, but this still requires further evaluations (Ziemba et al. 2005; Ontiveros and Paravina 2009; Bortolatto et al. 2014). This will be discussed in more detail in the section of frequently asked questions in this chapter.

This variation makes the comparison of the in-office bleaching protocols very difficult. However, efficient whitening has been observed in studies that employed 35 % HP, with reports of overall color change of five to eight shade guide units after two in-office bleaching sessions (Marson et al. 2008b; Tay et al. 2009; Bernardon et al. 2010; Strobl et al. 2010; Reis et al. 2011a). This wide range of color change probably is the result of the small variations in the HP concentration, number of bleaching sessions, and baseline color of the participants in the clinical trials (Rezende et al. 2015b).

7.3 Adverse Effects

As in-office bleaching is used with higher HP concentrations, there are more concerns about adverse effects in comparison with at-home bleaching. The two most frequent adverse effect of in-office bleaching is bleaching-induced tooth sensitivity (TS) and gingival tissue burning.

7.3.1 Bleaching-Induced Tooth Sensitivity (TS)

While effective bleaching is reported to occur with in-office bleaching, several publications have reported that patients undergoing bleaching procedures frequently complain of painful and uncomfortable sensations arising in the treated teeth. Although pain in bleached teeth can be evoked by cold or other stimuli, most patients complain of tingling or shooting pain (zingers) of very short duration but variable frequency (Haywood 2005) without provoking stimuli (Markowitz 2010).

Unfortunately, this side effect is very frequent. The reported risk of bleaching-induced TS in clinical trials of dental bleaching is quite variable but easily exceeds 50 %. A recent study that evaluated the individual patient data of 11 clinical trials regarding bleaching produced a more accurate estimate of these risks. For in-office bleaching in higher concentration (35 %), the risk of TS was reported to be 62.9 % (95 % CI 56.9–67.3), which was not too different from that reported for 10–16 % carbamide peroxide for at-home bleaching (51 % with a 95 % CI 41.4–60.6) (Rezende et al. 2015b). Although the risk of TS was reported to be similar, the intensity of TS was very different between bleaching protocols. In a 0–4 pain scale, the overall mean intensity of bleaching-induced TS for in-office bleaching was 2.8 ± 2.9, while for at-home bleaching was 0.5 ± 0.9 (Rezende et al. 2015b).

The etiology of bleaching-induced TS is not fully understood. Since the hydrodynamic theory of dentin sensitivity has been widely accepted as the explanation of dentinal sensation, some authors have used this theory to explain bleaching-induced TS (Swift 2005). However, pain during and following bleaching treatment can affect intact teeth lacking dentin exposure and this is in sharp contrast with the hydrodynamic theory (Markowitz 2010).

In face of that, other investigators have hypothesized that bleaching-induced TS may result from some degree of pulpal inflammation due to the higher amount of HP that reaches the pulp. It is widely known that HP can pass easily through the enamel and dentin to the pulp (Cooper et al. 1992) and can cause damage to the pulp cells as seen in Chap. 5 (Costa et al. 2010). Further proof of this passage of HP is the fact that color changes in dentin next to the pulp occur as fast they do at the dentin–enamel junction (McCaslin et al. 1999; Haywood 2005). Pulp tissue damage is likely to lead to the release of cell-derived factors, such as adenosine triphosphate (Cook and McCleskey 2002) and prostaglandins, which excite or sensitize pulpal nociceptors (Huynh and Yagiela 2003) causing the bleaching-induced TS (please refer to Chap. 5).

Several factors may affect the ability of HP to permeate the dental structures and consequently the damage produced by the bleaching gels. For instance, the amount of HP that permeates dental pulps is higher in teeth with restorations (Gokay et al. 2000; Patri et al. 2013; Parreiras et al. 2014). In restored teeth, the depth and size of the restorations (Parreiras et al. 2014), as well as the type of adhesive and restorative material (Gokay et al. 2000), may also play a significant role on the amount of HP penetration.

The tooth type is another important factor. Literature findings report that for the upper dental arch (Bonafe et al. 2013), the tooth that was reported to give most complaints of bleaching-induced TS was the upper lateral incisor. The thinner enamel and dentin layers of incisors compared to other teeth may allow the fast passage of HP to the pulp, allowing less time for the production and release of protective enzymes against damage by HP. This was also in agreement with recent histological studies of human pulps after in-office bleaching (Costa et al. 2010; Roderjan et al. 2015). In one study (Costa et al. 2010), the authors observed notable damage to the pulp tissue of lower incisors but not to premolars (Chap. 5).

Baseline color was strongly associated with TS in a recent study that pooled the data from 11 studies from the same research group (Rezende et al. 2015b). In other words, the darker the teeth, the lower the intensity and risk of TS. Darker teeth

probably have higher organic content to retain the HP in the enamel and dentin substrates, allowing less surplus HP to travel to the pulp tissue. Under these circumstances, it is possible that less HP comes in contact with the pulp tissue, which generates lower TS. This, however, is a hypothesis yet not supported by basic research.

Several approaches have been tested to minimize the adverse side effect of TS. The administration of some drugs perioperatively during in-office bleaching, such as selective anti-inflammatory drugs (etoricoxibe) (de Paula et al. 2013), nonsteroid anti-inflammatory drugs (ibuprofen) (Charakorn et al. 2009; Paula et al. 2013), antioxidants (ascorbic acid) (de Paula et al. 2014), and corticoids (dexamethasone) (Rezende et al. 2015a), was not effective to prevent the risk as well as the intensity of TS as confirmed by a recent systematic review of the literature (Faria et al. 2015). Under per-oral administration, several factors such as the immune system, lymphatic drainage, urinary excretion, and morphological characteristics of the dentin substrate may modulate the amount of the medicine that reaches the plasma and extracellular fluid around pulp cells, making these approaches not effective.

The most effective measures to minimize this side effect were through the application of topical desensitizers (Wang et al. 2015). The preoperative application of a gel composed of 5 % potassium nitrate and 2 % sodium fluoride for 10 min was capable to reduce the risk of TS by half, as well as the intensity of TS (Tay et al. 2009). The effect of fluoride in this process is not clear and the desensitizing effect of the association of sodium fluoride and potassium nitrate seems to be more related to the presence of potassium nitrate. This substance penetrates the enamel and dentin to travel to the pulp where it creates a calming effect on the nerve by affecting the transmission of nerve impulses (Ajcharanukul et al. 2007). After the nerve depolarizes in the pain stimulus response, it cannot repolarize, so the excitability of the nerve is reduced. Potassium nitrate has almost an anesthetic effect on the nerve (Haywood 2005).

In regard to the action of fluorides, it is hypothesized that the precipitation of calcium fluoride crystals in dentin can reduce the functional radius of the dentinal tubules and also the permeability of this tissue to the hydrogen peroxide. By doing so, less hydrogen peroxide reaches the pulp chamber, reducing the tooth sensitivity. This, however, is yet to be confirmed, as this process seems to occur only when there are exposed dentin surfaces.

Another study showed that previous desensitization with Gluma desensitizer (Heraeus Kulzer), composed of 5 wt% glutaraldehyde and 35 % weight% HEMA (Baba et al. 2002; Qin et al. 2006), for 1 min significantly reduced sensitivity of the anterior teeth during and after whitening compared with a placebo pretreatment (Mehta et al. 2013). The authors of this study hypothesized that glutaraldehyde (molar mass 100 g/moL) and HEMA (molar mass 130 g/moL) might penetrate through enamel and dentin along the same pathway as the peroxide radicals. On the way to the pulp, glutaraldehyde might react by cross-linking with enamel matrix proteins and with proteins in the dentin tubular liquid, thus reducing easy passage of the HP radicals to the pulp.

Although some opinion leaders claim that application of desensitizer gels prior to in-office bleaching affects the bleaching efficacy, this was not confirmed by recent meta-analyses of the literature (Wang et al. 2015), probably because the desensitizer gels used are transparent.

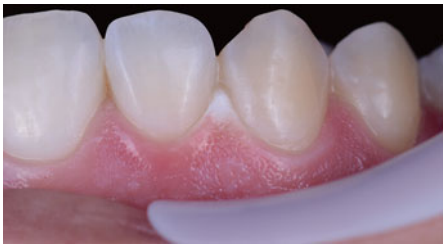

Fig. 7.4 Chemical burning of the cervical gingiva of several teeth after an in-office bleaching application with a high-concentration hydrogen peroxide

7.3.2 Gingival Tissue Irritation

As long as adequate protection of the gingival tissues is performed with a light-cured gingival barrier or rubber dam isolation, gingival burning (Fig. 7.4) is not expected to occur. This is usually not reported in clinical trials of in-office bleaching studies and reflects the clinical experience of the study authors. As seen in Chap. 4, in case this occurs the dentist should apply a drop of catalase and/or sodium bicarbonate (usually provided by the manufacturer) on the ulcerated lesion to arrest the burning effect. No other measure is usually required; but in case the patient feels any discomfort, a corticoid ointment may be prescribed to relieve pain.

7.4 Treatment Regimen with Step-By-Step Procedures

As mentioned earlier, in-office bleaching protocols vary significantly in the clinical reports. The application time of the bleaching gel, the number of bleaching sessions, whether or not the protocol is associated with light and the number of product refreshment on the dental surface are some examples.

In this chapter, we will describe all steps involved in an effective bleaching protocol and also report some of the variations of each step as long as they can still result in an effective whitening outcome. For didactic reasons, this section will be described in steps.

7.4.1 Making a Decision About the In-Office Bleaching Gel

There are many in-office bleaching products in the dental market, which makes their choice quite difficult. They vary in the active concentration of HP, which ranges usually from 15 to 40 % and in terms of pH (Freire et al. 2009; Price et al. 2000; Majeed et al. 2011). There are some products that contain other additives such as calcium gluconate and calcium phosphates and desensitizing agents (sodium fluoride, potassium nitrate). These systems also vary in their mode of application: most of them require product refreshment during a single in-office session, while for some products; a single 40–50-min application is required.

The literature is scarce regarding the comparison of these systems both in terms of effectiveness and side effects, and, therefore, the choice of these products is

usually based on empirical evidence. Comparison of in-office bleaching gels with different HP concentrations is also scarce. A single study that compared the color change and bleaching-induced TS of 20 % versus 35 % HP with 2 % calcium gluconate reported no significant difference in the risk of TS and a significant lower degree of whitening for the 20 % HP gel (Reis et al. 2013).

As previously mentioned, whitening products should have a relatively alkaline pH to minimize potential damage, but there is a wide pH variation among in-office bleaching gels (Price et al. 2000; Freire et al. 2009; Majeed et al. 2011). This variation could be the result of the different formulations used by each manufacturer, because bleaching agents contain stabilizers and other inorganic components that allow them to be stored for prolonged periods. In-office bleaching gels are delivered in low pH because they are more stable in acidic solutions than in basic solutions. When the HP is manufactured, a weak acid is usually added to the solution to prevent it from decomposing (Chen et al. 1993).

Some investigators have reported that the HP delivered in an alkaline medium increases the effectiveness of bleaching in the wool industry. This effectiveness is explained by the fact that the dissociation constant of the HP is about 11.5. In fact, the findings of one study showed that in a pH = 9.0, the dissociation rate of the HP was 2.7 times higher than that in an acidic solution (pH = 4.4) (Frysh et al. 1995) and this was recently confirmed by Torres et al. (Torres et al. 2014). They observed in vitro that the efficacy of hydrogen peroxide bleaching is directly proportional to the increase of the pH of the bleaching gel. These variations, however, did not seem to produce differences in tooth-bleaching effectiveness when products with acidic and alkaline pH were compared, although a significant decrease of tooth sensitivity has been shown for alkaline gels (Kossatz et al. 2012).

Additionally, it is worth mentioning that alkaline gels usually show more stable pH during application than acidic gels (Marson et al. 2008a), which allows them to be applied in a single application without the need of several product replenishments (Reis et al. 2011a, b; Kossatz et al. 2012).

Although there is biological plausibility to choose bleaching products containing desensitizing agents such as potassium nitrate, to the best of the authors' knowledge no randomized clinical trials have compared the TS levels produced by in-office gels with and without desensitizer agents. Only at-home clinical studies evaluated this hypothesis (Navarra et al. 2014; Gallo et al. 2009) as previously mentioned in the Chap. 6.

In summary, we recommend the use of 35 % alkaline gels, containing desensitizing agents. As mentioned in the section on frequently asked questions, reduced HP concentration can be used in the combined or jumped-started technique. In regard to the presence of desensitizing agents, we still recommend products containing it. The absence of evidence that desensitizing containing gels can reduce TS cannot be interpreted as evidence of absence of an effect. These studies are usually low powered and we cannot rule out the fact that desensitizing-containing gels can provide some beneficial effect. Until high-powered studies are published, we should work in the conservative way and use such type of products, as they do not have any known detrimental effects. Finally, products should be applied according to the respective manufacturer's instructions.

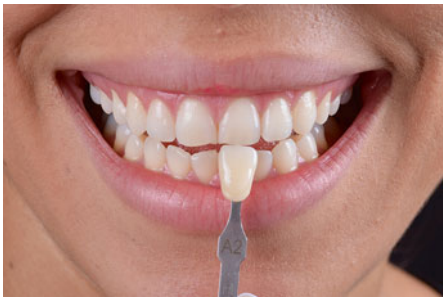

Fig. 7.5 The baseline tooth color being recorded with a value-oriented shade guide after performing a dental prophylaxis

7.4.2 Determination of the Baseline Tooth Color

This procedure allows dentist and also the patient to monitor color change during the bleaching protocol (Fig. 7.5). Patients usually very quickly get used to the new tooth color and may not remember what color their teeth were before protocol. This is even more important when both dental arches are bleached simultaneously. Shade recording can be a procedure with a value-oriented or bleach shade guide (Fig. 7.5), spectrophotometer, or by means of dental photographs.

Some authors encourage whitening one dental arch at a time (Haywood 2005), because it minimizes TS, allows the patient to monitor the opposing arch to compare progress, and it also encourages compliance. However, this procedure increases significantly the cost of the bleaching protocol, as it requires more dental visits.

Another advantage of color recording is that baseline dental color can predict the whitening degree obtained after dental bleaching. A recent multivariable regression analysis (Rezende et al. 2015b) identified a significant relationship between baseline color and age in relation to color change estimates. After adjustment for the other variables, every increase of one shade guide unit (in the value-oriented Vita Classical A1-D4™ shade guide) in the baseline color resulted in an increase of approximate 0.66 in the final color change in ΔSGU and 2.48 for the ΔE, meaning that the darker the baseline tooth color, the higher the degree of whitening. In an opposite trend, the degree of whitening is negatively affected by the participant's age (Rezende et al. 2015b).

This allows for the dentist to manage the patient's expectations in regard to the bleaching outcomes. Older patients with lighter baseline color may request more than the two bleaching sessions to achieve the same whitening degree than younger patients with darker baseline dental color.

It is important to perform a dental prophylaxis recording the baseline tooth color. A recent published paper showed a significant difference (average of two ΔE units of change) on tooth color when measured before and after dental prophylaxis. This may reach the threshold for clinical detection ($\Delta E = 3.0$) for some patients (de Geus et al. 2015).

7.4.3 Application of a Desensitizing Agent

As reported earlier, one of the main side effects of in-office dental bleaching is TS. Although this side effect cannot be completely eliminated, the number of patients that experience TS and the intensity of TS can be reduced by previous application of a desensitizing gel composed of 5 % potassium nitrate (Tay et al. 2009; Wang et al. 2015). Desensitizers composed of glutaraldehyde and HEMA was also reported to be effective to reduce the bleaching-induced TS, and can be an alternative to the potassium nitrate gel (Mehta et al. 2013).

This procedure can be performed before or after isolation of the dental arch, as the material is not aggressive to the gingival tissue. However, as the gel is usually agitated with the aid of a rotating brush it is recommended to apply the desensitizer before the protection of the soft tissues. The buccal surface of all teeth to be bleached should be covered with a 1-mm thick layer of the desensitizer and left in place for at least 10 min (Fig. 7.6). At the end of this period, the product should be agitated in each dental surface for 20 s with a rotating brush before removal. The inclusion of this step into the in-office bleaching protocol does not jeopardize the whitening efficacy of the hydrogen peroxide (Tay et al. 2009). After this period, the product should be removed with gauze (Fig. 7.7) or with a saliva ejector before application of the in-office bleaching gel. Rinsing can be performed as a final step for complete removal of the product.

7.4.4 Protection of the Soft Tissues

Hydrogen peroxide in high concentrations, such as those used for in-office bleaching, may cause burning of the dental tissues (Fig. 7.4). Several attempts should be made to avoid contact with the soft tissues.

The use of lip and cheek retractors associated with a light-cured gingival barrier (Fig. 7.8) is quite common. The former can maintain lips, cheeks, and even tongue away from the bleaching gel while the latter prevents the contact of the bleaching gel with the gingival tissue. An increased frequency of micronuclei of cells from the gingival tissue (which is an evidence of genotoxicity) was observed in patients

Fig. 7.6 Application of a desensitizing gel composed of 5 % potassium nitrate for 10 min (Dessensibilize KF 2 %, FGM, Joinville, SC, Brazil). After this period, the product should be agitated in each dental surface for 20 s with a rotating brush before removal

Fig. 7.7 Removal of the desensitizing gel with dental gauze or high-speed suction. After removal of the excesses, water rinsing was performed

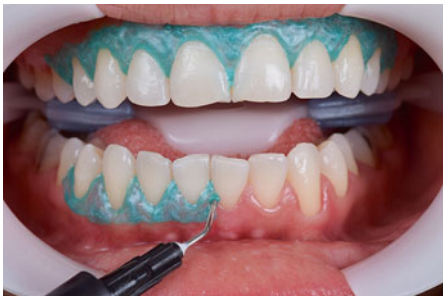

Fig. 7.8 A lip and cheek retractor (ArcFlex, FGM, Joinville, SC, Brazil) is applied, followed by the application of a light-cured gingival barrier to protect the marginal gingival tissue

submitted to in-office bleaching (Klaric et al. 2013), which may be the result of soft burning or even the contact with the gingival barrier. To avoid this, the light-curing gingival barrier should be adequately light cured (Fig. 7.8), according to the respective manufacturer's recommendations, and clinicians should look at the teeth from an incisal aspect to detect any sealing failure of the gingival tissue.

Rubber dam isolation can also be used for protection of the soft tissues. However, before rubber dam installation, a thick layer of petroleum jelly should be applied on the gingival tissue of the teeth to be bleached. Due to the hydrophobic nature of the petroleum jelly, the bleaching gel will be prevented from contacting the gingival tissue even if eventual isolation failure occurs.

7.4.5 Application of the In-Office Bleaching Gel

After choosing the in-office bleaching product, the manufacturer's instructions should be followed (Fig. 7.9 and 7.10). Variations to what is advocated by manufacturers may lead to either whitening at reduced speed or increased TS rates (Reis et al. 2011b; Kose et al. 2015). By increasing the number and/or time of application, one may increase the degree of whitening obtained but at the time the risk of TS is also increased. In an opposite trend, reducing the number and/or time of application reduces the probability of TS but also limits the degree of whitening.

Most in-office bleaching gels require replenishing the product during a period that varies from 40 to 50 min. Some products require two, three, or four product replenishments in each clinical session. There are some products, however, that are indicated for a single 40–50-min application without replenishment. These products

usually possess a basic pH that allows them to be used for longer application times without increasing the risk of TS (Kossatz et al. 2012; Reis et al. 2013). The product should be firstly removed with a cotton pellet, gauze, or high-speed suction (Fig. 7.11) before rinsing the dental surfaces with water. This procedure prevents any kind of soft tissue burning.

A recent clinical trial evaluated the impact of changing the bleaching protocol of a high-concentration (35 %) in-office bleaching product. Instead of performing three 15-min applications as suggested by the manufacturer, the product was kept for 45 min without replenishment. A reduction of the bleaching speed and increase in the TS intensity was observed, probably as a result of the slow but significant reduction of the pH of the product throughout the 45-min application (Reis et al. 2011b).

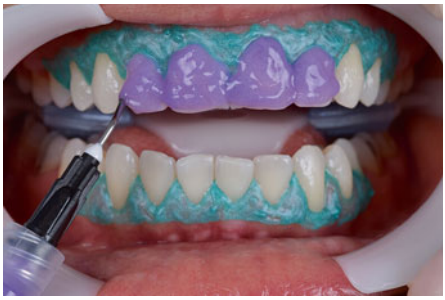

Fig. 7.9 The 35 % hydrogen peroxide in-office bleaching gel (Whiteness HP Blue 35 %, FGM, Joinville, SC, Brazil) is mixed and applied in all teeth to be bleached

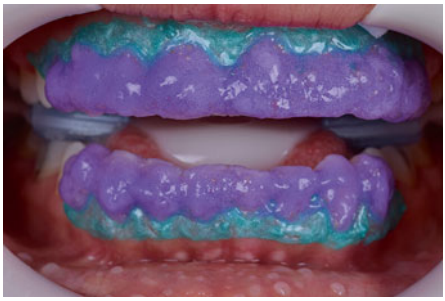

Fig. 7.10 After some time in place, bubbles are visible in the gel, which result from the decomposition of the hydrogen peroxide

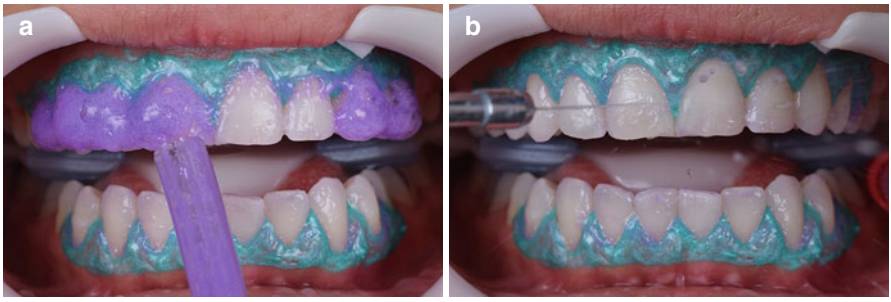

Fig. 7.11 (**a**) A suction tip was first used to remove the gel prior to (**b**) water rinsing of the tooth surfaces

As discussed in more detail in the section on frequently asked questions, some manufactures advocate the application of their products with light activation (quartz–tungsten halogen light curing units, LEDs or lasers) to optimize the bleaching outcome (Ziemba et al. 2005; Bortolatto et al. 2014). A recent systematic review of the literature concluded that light increases the risk of TS during in-office bleaching, and light may not improve the bleaching effect when high concentrations of HP (25–35 %) are employed. Therefore, dentists should use the light-activated system with great caution or avoid its use altogether (He et al. 2012). However, for low-concentrated HP gels the benefits of such association is yet to be determined.

Some manufacturers advocate the application of their products with light activation (quartz–tungsten halogen light curing units, LEDs, and lasers) to optimize the bleaching outcome (Ziemba et al. 2005; Kishi et al. 2011; Bortolatto et al. 2014). The benefits of this association are rather controversial (Buchalla and Attin 2007; He et al. 2012), but it seems to be useless for high-concentrated HP gels (Marson et al. 2008b; Alomari and El Daraa 2010; Kossatz et al. 2011; He et al. 2012). For low-concentrated HP gels, this light association may have some benefits; but this still requires further evaluations (Ziemba et al. 2005; Ontiveros and Paravina 2009; Bortolatto et al. 2014). This is discussed in more detail in the section on frequently asked questions in this chapter.

A single in-office bleaching session is usually not enough to achieve patient's satisfaction (de Silva Gottardi et al. 2006; Salem and Osman 2011). Studies that demonstrate that in-office bleaching is as effective as at-home bleaching usually performed two to three in-office bleaching sessions. Because in-office whitening often takes more than one appointment to achieve the adequate whitening, appointments generally are scheduled at least 1 week apart to allow the discomfort to dissipate. However, this procedure is purely based on empirical evidence.

Several clinical studies from our research group indicated that the TS induced by in-office only cause complaints during the initial 48 h post bleaching. Also, a recent randomized clinical trial revealed that a 2-day interval between two in-office bleaching sessions did not increase the risk and intensity of bleaching-induced TS (de Paula et al. 2015). However, in this paper, a calcium-containing alkaline gel applied for a single 40-min application without replenishment was used (de Paula et al. 2015), which prevent us from generalizing this protocol to all in-office bleaching gels present in the market.

In the clinical case, two clinical appointments were required to achieve patient satisfaction. The color achieved after the end of the bleaching procedure should be recorded with the same instrument used to record the baseline color. This measurement, however, should be done 4–7 days after the last in-office bleaching session to avoid the effects of dehydration and demineralization on the final outcomes (Fig. 7.12).

7.5 Durability of Color Change and Need for Touch-Up

As explained earlier in this chapter, the very short color reversal that occurs within some days after the in-office bleaching session cannot be interpreted as lack of effectiveness of the in-office bleaching protocol. In a way to avoid patient's

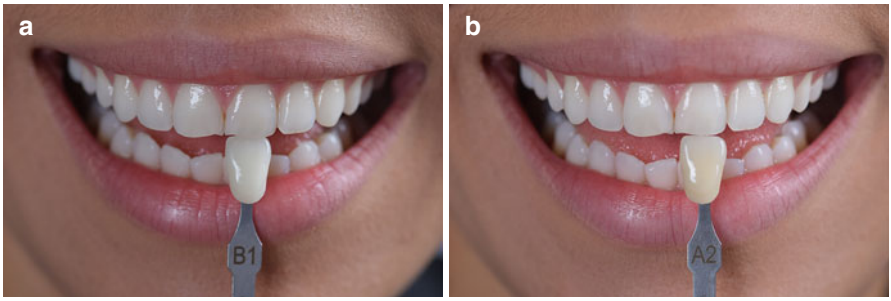

Fig. 7.12 One week after the second in-office bleaching session, the color of the patient's teeth was checked. (**a**) Teeth reached B1 color (the lightest color in the value-oriented Vita Classical shade guide), which is five tabs lighter than the baseline patient's teeth (A2) at the beginning of the treatment (**b**)

frustration, they should be instructed that a slight darkening is expected to occur in the following days as a result of dental rehydration and remineralization, and this does not necessarily mean that the bleaching was not efficient. An adequate measurement of the baseline tooth color will allow dentist to monitor the degree of color change that was due to the oxidizing nature of the hydrogen peroxide gel.

Although there are many randomized clinical trials reporting the immediate effects of several bleaching techniques, few of them evaluated the long-term efficacy of in-office bleaching (Giachetti et al. 2010; Mondelli et al. 2012; Tay et al. 2012). The few studies reported in the literature showed that in-office bleaching has stable results in periods ranging from 9 months to 2 years (Giachetti et al. 2010; Tay et al. 2012).

On the other hand, we may expect darkening of the dental structure in longer periods of time. As teeth grow older, there is a continuous deposition of secondary dentin by the pulp and higher enamel wear. Both factors together increase the yellowish appearance of the teeth. Additionally, we cannot rule out the effect of the staining produced by beverages and food (Meireles et al. 2010). Although this is usually an extrinsic staining and therefore may be easily removed by prophylaxis, it may affect the patient's overall perception of whiter teeth.

Based on the aforementioned explanations, touch-up bleaching may be performed whenever color rebound is detected. Specific protocols and products were discussed in the Chap. 6. Other option is to apply a new single in-office bleaching session that may achieve satisfactory results. It may be emphasized, however, that the literature lacks randomized clinical trials on this topic.

7.6 Frequently Asked Questions

7.6.1 Do We Need Lights to Activate Peroxidases?

As heat and light can accelerate the dissociation of hydrogen peroxide (Ontiveros 2011), both methods have been associated with in-office bleaching as early as 1918 (Abbot 1918). However, as we already mentioned earlier in this chapter, the

literature findings point out that there is no advantage of associating it with high concentrations of HP gels (Marson et al. 2008b; Alomari and El Daraa 2010; Kossatz et al. 2011; He et al. 2012).

At first glance, this seems to be contradictory. In fact, from chemical theories, one knows that in the simplest chemical reactions, the highest concentration of reactants raises collisions per unit time, and hence increases the reaction rate. However, if the reaction is complex and involves a series of consecutive steps, there might be a limit to which the increased concentration leads to faster reaction rates. We hypothesize that 35 % HP alone already produces enough free radicals for oxidizing organic component of dentin, and, thus, the increase in free radicals produced by the light activation might be useless. Consequently, the further increases in HP radicals produced by light activation do not lead to faster bleaching due to the presence of unknown rate-determining steps in the oxidizing mechanism of tooth bleaching.

On the other hand, this may not be the case when using low HP gels. Randomized clinical trials that evaluated the effect of light associated with low HP concentration seemed to show a faster whitening degree (Tavares et al. 2003; Ontiveros and Paravina 2009). This may not be the case when using low HP concentrated gels. For these gels it seems that the limiting factor of the oxidizing reaction rate was the amount of free radicals, and thus the association with light, which likely increases the amount of free radicals, may produce a faster reaction rate and a whitening degree similar to that of the 35 % HP gel associated or not with light (He et al. 2012; Bortolatto et al. 2014). However, these findings are still preliminary and require further evaluations.

7.6.2 Are Light-Activated Peroxides Available?

Some manufacturers indicated that their products contain orange-red color of carotene as colorants and these compounds can be considered as activators because they absorb primarily at wavelengths of blue lights. If the bleaching agent absorbs the light energy of this wavelength, it heats and thus decomposes (Ontiveros 2011). Unfortunately, a literature review indicated that although the temperature of the carotene-containing bleaching gel can increase considerably, this increase was not high enough to accelerate HP decomposition significantly (Buchalla and Attin 2007).

Another option is the addition of some metals to enhance the oxidizing power of the HP, as ferrous compounds or titanium dioxide. The photolysis of HP associated with these compounds needs to be activated by a very specific wavelength, which depends on the metals included (Ziemba et al. 2005; Kishi et al. 2011; Ontiveros 2011; Bortolatto et al. 2014).

For instance, one manufacturer combined iron with a low-concentrated HP formulation. With ferrous compounds, HP can be combined with iron known as Fenton reagent. Fenton reagents result in disproportion in which the iron is simultaneously reduced and oxidized to form both hydroxyl and peroxide radicals by the same HP. When Fe reacts (with or without UV radiation), the process is renewed and the redox reaction is further fueled (Ontiveros 2011). This is the reason why products that contain ferrous components recommend light activation by ultraviolet lights

(Kugel et al. 2009; Ontiveros and Paravina 2009). The use of UV lamps requires care. Patients, dentist, and auxiliaries should be protected because of the known damage the ultraviolet radiation can cause on the skin. It should be mentioned that Fenton reaction occurs with or without ultraviolet light activation. Perhaps, futures studies should focus on the evaluation of the bleaching efficacy of these bleaching systems without the use of UV lights.

Some low-concentrated HP gels (6–15 %) containing semiconductors of titanium oxide nanoparticles doped with nitrogen have shown good bleaching efficacy comparable to 35 % HP gels (Bortolatto et al. 2014; Martin et al. 2015). When exposed to blue light (LED/Laser device), these nanoparticles catalyze the formation of hydroxyl radicals from HP (Sakai et al. 2007). As these titanium oxide bleaching formulations can be used with visible lights they are safer than the previous formulations that recommend UV light activation.

7.6.3 Manufacturers Recommend Several Consecutive Applications of the In-Office Whitening Gel? How Many Applications Are Needed? For How Long?

Clinicians should follow the manufacturer's instructions for application of the in-office bleaching gels. There are some products that are necessary to be refreshed two to four times in a 40–50-min clinical session, while other products require that the product be left undisturbed for the whole period it stands on the dental surface. It is suggested that acidic gels and those that do show reduction of the pH over time should be refreshed; alkaline gels that keep the pH alkaline during application can be left on the surface for the whole application period.

However, this can be changed based on the patient's profile. In case the professionals are dealing with a very sensitive patient, the number of product refreshments as well as its application time can be reduced. This will probably reduce the risk and intensity of TS (Kose et al. 2015) but will also require more applications to achieve patient's satisfaction.

Usually, two to three in-office bleaching sessions using 35 % HP are required to show a significant color change (Marson et al. 2008b; Tay et al. 2009; Bernardon et al. 2010; Strobl et al. 2010; Reis et al. 2011a), but unfortunately this can vary depending on the baseline color of the participants in the clinical trials (Rezende et al. 2015b).

7.6.4 Are Calcium Phosphate and Fluoride Containing Gels Effective to Decrease Tooth Sensitivity Caused by In-Office Bleaching?

As previously mentioned, there are numerous studies that have exhibited microstructural changes of enamel surface induced by in-office bleaching agents (Dahl and Pallesen 2003) and it results from the low pH of most bleaching products

available in the market. Also, clinicians believe that these superficial alterations to the enamel surface increase TS induced by in-office gels, mainly because the surface becomes more porous to passage of HP.

This led different clinicians to evaluate whether the preoperative application of remineralizing agents (Loguercio et al. 2015) or the addition of different remineralizing products to in-office bleaching formulations (fluoride, calcium phosphate compounds, etc.) (Basting et al. 2012; Kossatz et al. 2012) might have an impact on the reduction of bleaching-induced TS. These studies failed to find a reduction of the bleaching-induced TS; however, no detrimental effect on the whitening efficiency was detected (Basting et al. 2012; Kossatz et al. 2012; Loguercio et al. 2015).

A recent literature review that investigated the impact of bleaching procedures on enamel surface indicated that these adverse on enamel effects are minimal. Laboratory studies that simulated the intraoral conditions as closely as possible reported that as soon as bleached enamel comes in contact with saliva, remineralization occurs and within a few days no adverse effects can be measured (Attin et al. 2009). This was also confirmed by in vivo studies when in-office gels were used after prolonged and repeated applications (Spalding et al. 2003; Cadenaro et al. 2008, 2010).

7.6.5 Why Are Some In-Office Whitening Products Referred to as "Chemically Activated"?

As previously described, the in-office gels are more stable in acid solutions than in alkaline solutions (Chen et al. 1993). This is why the majority of bleaching gels commercially available are presented in two syringes/bottles, one containing the HP product and other containing the colorants, thickening agent, etc.

When clinicians mix both syringes/bottles, a "chemical activation" occurs by mixing two components of the respective bleaching gels, which can indeed increase HP decomposition and the in-office gels are ready to use. This has led to erroneous interpretation of in-office gels being "chemically activated." Actually, the main function of the activating gel component (synonymously referred to as "catalyst" or "booster") is to increase the pH of the mixed gel to achieve a alkaline pH close to the pKa of the hydrogen peroxide (pka = 11.0), thereby increasing the decomposition rate of peroxide and the formation of oxidative radicals (Buchalla and Attin 2007).

7.6.6 Does the "Jump-Start" Technique Improve the Final Result of a Whitening Treatment?

As indicated in the Sect. 7.2, there are several factors that may explain the clinician's belief that in-office bleaching is not efficient when compared to at-home bleaching. Thus, the combination of in-office and at-home bleaching ("combined bleaching technique") has been suggested for some clinicians as a way to potentiate the bleaching effect and improve color stability (Kugel et al. 1997; Deliperi et al. 2004; Matis et al. 2009; Bernardon et al. 2010).

However, considering that both bleaching techniques (in-office and at-home techniques) are effective, the main advantage of the "jump-start" technique is for some patients who demand faster ways of bleaching (Matis et al. 2009; Bernardon et al. 2010). In this way, the "jump-start" technique, as the name suggests, is commonly used to motivate the patients to comply with the at-home bleaching protocol.

Usually, the in-office bleaching is applied before starting the at-home protocol; however, the in-office bleaching can be incorporated in any moment, mainly when there is a low response to the at-home bleaching. The number of in-office bleaching sessions associated with the at-home procedure will be dictated by the patients' demand and the whitening response to the procedure.

Usually, clinical studies that performed the combined or jump-start bleaching technique have used high hydrogen peroxide concentrations for the in-office phase (Kugel et al. 1997; Deliperi et al. 2004; Matis et al. 2009; Bernardon et al. 2010). This means that high levels of bleaching-induced TS were reported (Kugel et al. 1997; Deliperi et al. 2004; Matis et al. 2009; Bernardon et al. 2010).

More recently, a clinical study that compared a low and high concentration of HP combined with 10 % carbamide peroxide for at-home bleaching showed that both protocols yielded the same whitening effect. The constant delivery of the at-home bleaching gel for the 2 weeks following the in-office bleaching might have compensated for the lower HP concentration of the in-office gel. However, the use of the low HP concentration for the in-office phase of the bleaching protocol reduced the risk and intensity of bleaching-induced TS (Rezende et al. 2016).

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Intracoronal Whitening of Endodontically Treated Teeth

8

Jorge Perdigão, Andressa Ballarin, George Gomes, António Ginjeira, Filipa Oliveira, and Guilherme C. Lopes

Abstract

Several techniques have been used within the last 170 years to lighten discolored endodontically treated teeth. Internal whitening or intracoronal whitening offers some advantages over more invasive treatments, as: (1) it is relatively easy to carry out; (2) it involves the removal of minimal tooth structure; and (3) the cost of treatment is low compared to that of other restorative options including full- and partial-coverage restorations.

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The whitening techniques currently used to lighten darkened endodontically treated teeth are based on the release of active oxygen inside the pulp chamber and subsequent diffusion into the dentinal tubules. Hydrogen peroxide in a concentration of approximately 30–35 % and/or sodium perborate have been the chemicals most often used as oxygen sources. The prognosis of intracoronal whitening is good for discolorations of endodontically treated teeth caused by necrotic pulp tissue or blood components, with a short-term success rate of 50–90 %. However, the long-term success rate is considerably lower, as some color regression may occur after the initial bleaching effect.

Undesirable side effects of intracoronal whitening include occasional external cervical resorption and, more rarely, ankylosis. External cervical resorption has been often associated with anterior teeth that had been endodontically treated as a result of traumatic injury in young patients. Other side effects, such as alteration of enamel morphology and decrease in physical characteristics of enamel and dentin, are minimal and transient according to some authors, or inexistent according to other authors. This chapter will review the clinical techniques and provide step-by-step guidelines for the dental professional to carry out internal whitening of endodontically treated teeth.

8.1 Introduction

Root canal therapy often results in tooth discoloration (Kingsbury 1861). When the clinician believes that preserving the patient's own tooth structure is the most appropriate treatment, intracoronal whitening (or internal whitening or internal bleaching[1]) may be indicated for esthetic reasons. While internal bleaching is a conservative technique compared to more invasive procedures such as veneers or full-coverage esthetic restorations, it results in a relatively low long-term success rate (Brown 1965; Howell 1981).

Ancient Egyptians and Romans used various methods to whiten teeth. Romans used urine because it contains ammonia, a cleaning agent now found in many household products. Egyptians used a paste of vinegar and pumice, combining the eroding effect of acetic acid with the abrasive effect of pumice. Intracoronal whitening of discolored endodontically treated teeth has been described in the dental literature since the nineteenth century (Dwinelle 1850; Fitch 1861). Dwinelle (1850) used *chloride of lime* or *bleaching powder* (currently known as calcium hypochlorite) and soda to successfully lighten nonvital teeth. The procedure was known as "bleaching" as consequence of the use of *bleaching powder*. In May 1884, in a lecture to the Section of Oral and Dental Surgery of the American Medical Association, Harlan described for the first time the use of hydrogen peroxide to bleach root canal-treated teeth (Harlan 1884):

> In order to bleach a pulpless tooth the operator must first fill the root at least one third its length… Discolored dentine if hard need not be cut away. With the rubber dam adjusted over the adjacent teeth, including the one to be operated upon, the cavity is thoroughly washed with H_2O_2 repeatedly and then carefully dried….

[1] The terms "whitening" and "bleaching" are used interchangeably in the literature.

Other bleaching agents were more popular among dentists until the 1900s (Chap. 1), including sodium peroxide, oxalic acid, potassium cyanide, chlorine- and sulfur-based materials, and pyrozone (a mixture of ether and hydrogen peroxide) (Kingsbury 1861; Truman 1881; Kirk 1893). By 1903, the allusion to the use of hydrogen peroxide to bleach nonvital teeth had become more usual in the dental literature (Buckley 1903; Miller 1903).

More recently, sodium perborate, hydrogen peroxide, and carbamide peroxide have been the materials of choice for intracoronal whitening (Brown 1965; Boksman et al. 1983; Carrillo et al. 1988; Vachon et al. 1998). Two materials have been favored, namely, 30–35 % hydrogen peroxide (Superoxol) and powdered sodium perborate, which have been used either alone or in combination (Weisman 1963; Freccia et al. 1982). Hydrogen peroxide generates free radicals and active oxygen (Kashima-Tanaka et al. 2003).

8.2 Etiology of Discoloration in Endodontically Treated Teeth

Discoloration of nonvital teeth can be classified as intrinsic and/or extrinsic (Hattab et al. 1999; Plotino et al. 2008). Extrinsic stains (Chap. 1), by definition, are caused by extrinsic agents and located on the outer surface of the teeth. *Intrinsic stains* result from the incorporation of chromogenic materials into dentin either during odontogenesis or after eruption (Plotino et al. 2008). Intrinsic stains can be caused by local or by systemic causes (Fig. 8.1). This chapter will focus on stains of *intrinsic* origin.

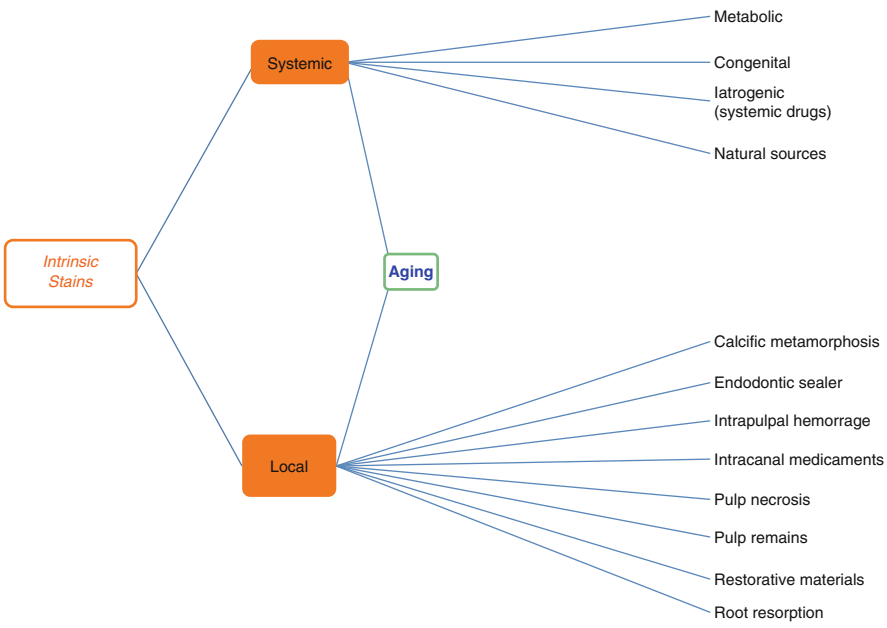

Fig. 8.1 Causes of intrinsic stains

8.2.1 Systemic Causes of Posteruptive Intrinsic Stains

The developing dentition can be affected by a number of systemic factors, including metabolic and genetic diseases that cause intrinsic tooth discoloration (Plotino et al. 2008). Systemic factors also include drug-induced discolorations, including those from the intake of antibiotics and excess fluoride. In the case of fluoride its source can be either iatrogenic or from natural sources, such as drinking water.

Although most publications refer to aging as a local cause of intrinsic discoloration, the physiological continuous deposition of secondary dentin throughout the life of an individual may be considered both a systemic and a local cause of intrinsic discoloration, as reactionary tertiary dentin forms in response to external stimuli, such as tooth attrition and bacteria from caries lesions. The deposition of both secondary dentin and tertiary dentin results is a narrower pulp space with time. Thicker dentin affects the light-transmitting properties of teeth, resulting in a gradual darkening of the tooth and increased opacity. In addition, the chemical structure and physical properties of dental hard tissues change over time.

8.2.2 Local Causes of Posteruptive Intrinsic Stains

Local causes of posteruptive intrinsic stains include intrapulpal hemorrhage, residual pulpal tissue after endodontic therapy, calcific metamorphosis, endodontic materials, restorative materials such as amalgam, and root resorption. Other less frequent causes are trauma to developing teeth and periapical infection of primary teeth (Hattab et al. 1999). Besides being responsible for posteruptive pigmentation of vital teeth (Chap. 6), the antibiotic minocycline also causes discoloration of nonvital teeth. In one case, minocycline was utilized in the root canal as a component of antibiotic paste resulting in blue discoloration of immature necrotic permanent teeth in children (Dabbagh et al. 2002). In another case, minocycline was applied as a component of a triple antibiotic mixture inside a root canal of a tooth with a necrotic pulp, as an attempt to disinfect the root canal system for revascularization. Minocycline resulted in tooth discoloration 6 weeks after the triple antibiotic paste had been applied (Kim et al. 2010).

Necrotic pulps may cause tooth discoloration as a result of the decomposition of the pulpal tissues producing colored byproducts that infiltrate the dentinal tubules. Discoloration related to improper endodontic treatment may be caused by trauma inflicted during pulp extirpation or obturation materials left in the pulp chamber. Another cause of discoloration in endodontically treated teeth is the residual pulp tissue left in the pulp horns when the access to the pulp chamber is under-prepared (Brown 1965; Faunce 1983). Evidence suggests that stains in endodontically treated teeth are not just confined to the pulp chamber, but they penetrate into the dentin substrate showing through dentin and enamel (van der Burgt and Plasschaert 1986).

Fitch (1861) stated, “the disintegration of the red corpuscles of the blood is another source of discoloration. The hematine, or iron, which is supposed to constitute the pigment of these globules, passes readily into the tubules of the dentine from the pulp chamber of the tooth, whenever the red disks are disintegrated, and

the discoloration becomes more or less permanent. These red globules may be dissolved or broken up." Spasser (1961) reported that "in many cases, the hemolysis of red blood cells with the release of hemoglobin, especially when associated with hemorrhage following pulp extirpation or trauma" was responsible for the discoloration of endodontically treated teeth. More recently other authors have corroborated this theory. Diffusion of blood components into the dentinal tubules caused by pulp extirpation or traumatically induced internal pulp bleeding is one of the causes for discoloration of nonvital teeth (Arens 1989). Products of blood (erythrocytes) decomposition, such as hemosiderin, release iron. The iron can be transformed to ferric sulfide with hydrogen sulfide produced by bacteria, which causes a dark brownish discoloration of the tooth (Brown 1965; Glockner et al. 1999; Attin et al. 2003). Other end products of hemoglobin decomposition, biliverdin and bilirubin, are themselves known causes of color alteration in the skin and mucosae, being responsible for jaundice in some systemic diseases (Shibahara et al. 2002; Leuschner 2003).

Coronal discoloration of endodontically treated teeth may also be caused by endodontic sealers (Fig. 8.2), such as Grossman's cement and AH26 silverfree (Dentsply Caulk) (van der Burgt and Plasschaert 1985; van der Burgt et al. 1986). Temporary restorative materials, such as classical ZOE, Cavit (3M ESPE), and I.R.M. (Dentsply Caulk), also result in discoloration of endodontically treated teeth (van der Burgt et al. 1986). Two mineral trioxide aggregate (MTA)-based endodontic materials, ProRoot MTA (Dentsply Tulsa Dental Specialties) and MTA Angelus (Angelus Indústria de Produtos Odontológicos), have also been reported to cause tooth

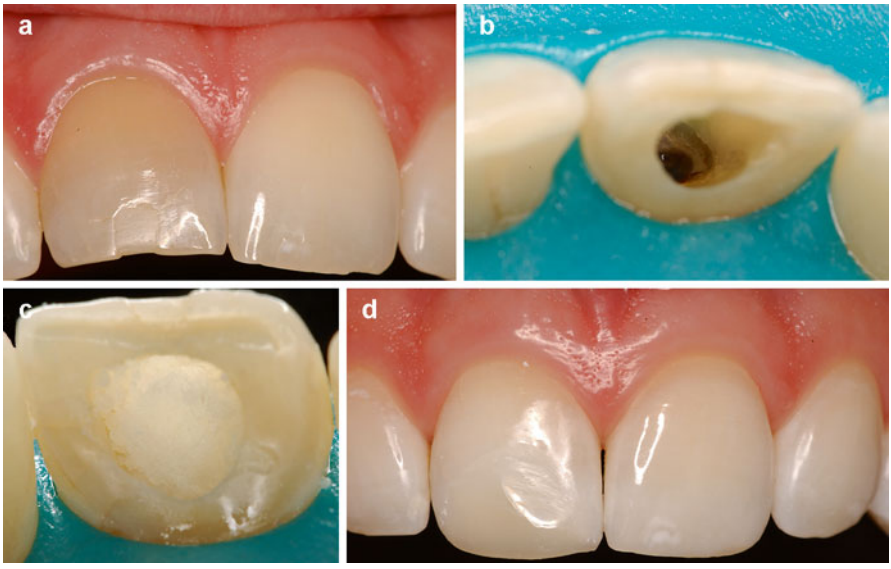

Fig. 8.2 Clinical case of a recent discoloration (**a**) caused by endodontic sealer (**b**) in the pulp chamber. This tooth lighten with the walk bleach technique using sodium perborate mixed with distilled water after only one session (**c**, **d**)

discoloration when used over a period of 12 weeks (Jang et al. 2013). The discoloration was observed at the MTA-dentin interface and intracoronal dentin surface. Removal of the discolored MTA resolved the discoloration.

8.3 Treatment Plan Considerations

When referring to whitening of nonvital teeth, Salvas (1938) wrote, "bleaching teeth is, at best, more or less of an unsatisfactory operation. Although we may succeed in restoring the color of a tooth, it is seldom permanent." The unpredictability of intracoronal whitening has not changed considerably since Salvas wrote these statements in 1938.

Taking into consideration that the treatment outcome as well as the durability of the treatment varies for each clinical situation, the patient must be informed of these inherent limitations of intracoronal whitening. Another detail that the patient must be aware of is that root dentin does not respond well to bleaching, either external or internal (Kwon 2011). This is especially significant in case there is gingival recession that has resulted in exposed root surface.

The indications and contraindications for intracoronal whitening are displayed in Table 8.1. Prior to starting an internal whitening procedure to lighten single or multiple teeth, it is crucial to understand all treatment options available for each specific clinical case. The prognosis of internal whitening depends on several factors, including the etiology and the duration of the discoloration. Additionally, a history of traumatic injury to the tooth or teeth may be associated with subsequent external cervical resorption. It is also important to understand that the intrinsic discoloration in endodontically treated teeth affects dentin but not enamel (van der Burgt et al. 1986). A precise diagnosis relies on accurate clinical and radiographic exams. The presence of craze lines on the enamel structure and marginal microgaps in existing restorations may result in seepage of the intracoronal bleaching agents to the surrounding tissues. For this reason, the use of transillumination to diagnose microgaps and craze lines is

Table 8.1 Indications and contraindications for internal whitening of endodontically treated teeth

Indications	Contraindications
Discolorations from pulpal trauma or pulp remains	Inadequate root canal treatment
Discolorations that do not respond to external bleaching techniques	Untreated caries lesions and abfraction lesions
Dentin discolorations of various origins, including endodontic sealers	Loss of coronal tooth structure that prevents sealing of the bleaching material inside the pulp chamber
	Defective restorations or enamel craze lines that may result in seepage of the bleaching material to the periodontal tissues
	Patient's high expectations
	Pregnancy
	Discoloration caused by oxidation of metals (silver, amalgam)
	Discolorations restricted to enamel

extremely important. The treatment plan must be based on existing evidence to improve the chance of clinical success.

Intraoral photographs are essential to document the baseline color. Although the color of darkened endodontically treated teeth is not usually within the range of universal shade guides, the clinician may still take a photograph with a tooth tab from the Vita Classical A1-D4 shade guide (VITA Zahnfabrik H. Rauter GmbH & Co. KG) next to the darkened tooth. This will provide an excellent comparative reference for the postoperative versus the preoperative color.

8.4 Whitening Techniques for Endodontically Treated Teeth

Superoxol used to be the most commonly used intracoronal bleaching material. More recently, water has been used (instead of hydrogen peroxide) mixed with sodium perborate (Ari and Ungör 2002; Plotino et al. 2008). Sodium perborate ($NaBO_3$) is a white crystalline powder that contains about 95 % of the perborate corresponding to 9.9 % available oxygen (Rotstein and Friedman 1991). In contact with water, sodium perborate decomposes with the liberation of hydrogen peroxide and later of oxygen. Sodium perborate is, therefore, a hydrogen peroxide precursor (Ari and Ungör 2002). Warm air and acidic solutions also initiate the decomposition of sodium perborate. A thick creamy paste of sodium perborate with water is currently recommended, left in the pulp chamber for varying periods of time.

Two techniques for bleaching endodontically treated teeth have been used through the years, namely, the *thermocatalytic technique* and the *walking bleach technique*. Other bleaching techniques include *combined* thermocatalytic and walking bleach techniques (Freccia et al. 1982).

8.4.1 Thermocatalytic Technique

In the *thermocatalytic technique*, heat is used to activate the release of nascent oxygen from the oxidizing agent or agents, often hydrogen peroxide and sodium perborate (Ari and Ungör 2002). Several sources of heat have been utilized as adjunct activation methods, including ultraviolet (UV) lights, infrared lights, flamed instruments, and electrical sources of light and heat. Fischer (1911) reported heating hydrogen peroxide with a special mercury arc light with a quartz lens, or Kromeyer's lamp, to irradiate the teeth with ultraviolet (UV) rays to mimic the sunlight. Prinz (1924) recommended using heated solutions consisting of sodium perborate and Superoxol for cleaning the pulp cavity. Dietz (1957) recommended a 20-in. infrared light for the thermocatalytic technique in case only one appointment was feasible. In 1963, the use of 30 % hydrogen peroxide with a source of light and heat from a distance of 5 cm was reported (Weisman 1963).

In light of the risk of side effects from heating hydrogen peroxide in the pulp chamber space (Madison and Walton 1990), including external cervical resorption, the *thermocatalytic technique is no longer advocated*.

8.4.2 Walking Bleach Technique

For the walking bleach technique, hydrogen peroxide or water is mixed with sodium perborate, but *heat is not used* to trigger the release of nascent oxygen. The use of an intracoronal mixture of sodium perborate mixed with Superoxol or water has been described as a successful technique (Kirk 1893; Nutting and Poe 1967; Rotstein et al. 1993a). Saline and anesthetic have also been used instead of water (Madison and Walton 1990). Propylene glycol, which is largely used as in the pharmaceutical and food processing industry as a humectant and preservative, has also been suggested in some countries as a vehicle for the intracoronal bleaching paste in lieu of water. There is, however, no evidence that propylene glycol is more effective than water to mix with sodium perborate for internal whitening.

The first description of the walking bleach technique using a mixture of sodium perborate and *distilled water* was published in 1938 (Salvas 1938). The mixture was left in the pulp space for a few days, while the access cavity was sealed with provisional cement. This same technique was revived in 1961 (Spasser 1961) and modified in 1967 (Nutting and Poe 1967), when 30 % hydrogen peroxide was used instead of water to improve the bleaching effectiveness of the mixture. Although 30 % hydrogen peroxide may boost the efficacy of internal whitening, the procedure can be successfully accomplished without hydrogen peroxide (Salvas 1938; Spasser 1961).

Clinically, the walking bleach technique of sodium perborate mixed with water has been reported to be effective. In 57 out of 95 teeth (60 %), a good or acceptable result was obtained after one or two visits (Holmstrup et al. 1988). The remaining 38 teeth (40 %) were treated over 3–9 visits. The method used sodium perborate moistened with water and the access cavity was temporarily restored with Cavit (3M ESPE) between visits. While 55/69 (79.7 %) of the teeth examined at 3 years still had a good or acceptable result, 14 teeth (20.3 %) showed recurrence of discoloration that was considered unacceptable. The total number of teeth with an unacceptable result either initially or after 3 years was 25 % of the treated teeth. Despite recurrence, all examined teeth showed less discoloration than before bleaching. Interestingly, it has been reported that patients treated with the walking bleach technique brush more often than other patients, developing a superior dental hygiene regimen (Abou-Rass 1988).

> In vitro and clinical studies have shown that three applications of *sodium perborate* mixed with *water* are equally effective as applying sodium perborate and the 30 % hydrogen peroxide solution (Rotstein et al. 1991c; Rotstein et al. 1993a; Holmstrup et al. 1988).

With the advent of the at-home vital whitening technique (Chap. 6), carbamide peroxide (also known as urea peroxide) has been advocated for the walking bleach technique alone or in combination with sodium perborate (de Souza-Zaroni et al. 2009). Aldecoa and Mayordomo (1992) suggested a 4-week treatment with 10 % carbamide peroxide mixed with sodium perborate as a "second phase" of internal

whitening once the first phase of bleaching with hydrogen peroxide mixed with sodium perborate was completed. The association of carbamide peroxide with sodium perborate is more effective than that of water with sodium perborate (Yui et al. 2008). Another study reported contradictory findings, as the sodium perborate mixed with 37 % carbamide peroxide association proved to be as effective as sodium perborate mixed with distilled water for intracoronal bleaching (de Souza-Zaroni et al. 2009).

As several concentrations of carbamide peroxide gels are available for the at-home whitening technique, the use of 10, 17, or 37 % carbamide peroxide has been studied regarding the ensuing infiltration of peroxide into radicular dentin. All three concentrations resulted in significantly lower penetration of peroxide into radicular dentin than a combination of Superoxol with sodium perborate. However, 37 % carbamide peroxide resulted in significantly more penetration of peroxide that 10 or 17 % carbamide peroxide (Gokay et al. 2008).

Although there are different materials currently available for the walking bleach technique, the combination of *water* with *sodium perborate* is still preferred, as it may reduce the risks of side effects.

8.4.3 Combined Techniques

Freccia et al. (1982) stained extracted teeth with blood and then compared the results of three nonvital bleaching techniques: thermocatalytic technique, walking bleach, and a combined technique. Although the techniques were equally effective in bleaching blood stained teeth, the walking bleach technique consumed the least operator time.

8.5 Factors That Influence the Prognosis of Intracoronal Whitening

8.5.1 Duration of Discoloration

The prognosis may depend on the duration of the discoloration (Brown 1965; van der Burgt and Plasschaert 1986). Brown (1965) reported that the shorter the time the tooth has been discolored, the more successful bleaching is. Teeth that had been discolored for less than 1 year had an 87.5 % success rate, while for teeth that had been discolored over 5 years, the success rate dropped to 66 %.

8.5.2 Intensity of Discoloration

Severely discolored teeth have less chance of successful bleaching (75 %) than teeth with moderate or slight discolorations (90–100 %) (Brown 1965). Gradual discolorations tend to bleach more efficiently than rapid discolorations.

8.5.3 Potential for Color Regression

Some color relapse may occur in about 50 % of bleached teeth after 1 year, and even more after a longer period (Brown 1965; Howell 1981). Teeth that are more difficult to bleach are more likely to discolor again (Howell 1981). Specific endodontic sealers result is higher risk of color relapse than others (van der Burgt and Plasschaert 1986). The probability of color reversal is much higher when the discoloration is due to metallic stains or silver-containing medicaments (Freccia et al. 1982).

Brown (1965) performed a survey on 80 teeth that had been bleached using 30 % hydrogen peroxide, and compared standardized photographic records of the teeth taken before and after bleaching, and at intervals of l–5 years. The author found that 20 (25 %) teeth failed, of which 14 teeth failed to respond to treatment at all, while in 6 teeth the color relapsed after initially successful bleaching. Of the 60 (75 %) successfully bleached teeth, 23 showed no postoperative change, but in 37 teeth there was some color regression.

Stability of nonvital discolored teeth subjected to *combined* thermocatalytic and walking bleach intracoronal techniques was evaluated at 16 years (1989–2005) (Amato et al. 2006). The series comprised 50 patients (age range 7–30). After 16 years, 35 cases were evaluated. In 22 of these cases (62.9 %) the color had remained stable and was similar to that of adjacent teeth, indicating a successful outcome of the combined bleaching technique. There were 13 cases (37.1 %) classified as failures because of marked color relapse. Radiographically none of the cases reexamined underwent internal or external root resorption. However, there is insufficient evidence in terms of efficacy and safety to substantiate the use of the *combined* technique over the *walking bleach* technique.

8.5.4 Patient's Age

The success rate of the internal whitening technique is 50–90% without a direct relationship between success and patient's age (Brown 1965; Howell 1981). However, other authors have stated that young teeth bleach faster than old teeth because the dentinal tubules are wider in younger teeth (van der Burgt and Plasschaert 1986).

Dietz (1957) reported a direct relation between the age of a tooth and its resistance to bleaching, suggesting that more permanent results were obtained with teeth of the older age groups. Camps et al. (2007) evaluated the diffusion of hydrogen peroxide through human dentin in patients under 20 years old and in patients between 40 and 60 years old. The teeth were endodontically treated, and a defect was created at the CEJ. The access cavities were filled with 20 % hydrogen peroxide gel. The amount of diffusing hydrogen peroxide was assessed at 1, 24, 48, and 120 h. Diffusive flux and maximal diffusion were higher through young teeth than through old teeth.

8.5.5 Type of Discoloration

The prognosis for success of any bleaching technique depends on the cause of the discoloration (Freccia et al. 1982). When the discoloration is a result of products of pulpal decomposition within the dentinal tubules, the prognosis is usually very good. Recent discolorations from endodontic sealers are also easy to whiten (van der Burgt and Plasschaert 1986) (Fig. 8.2). When the discoloration is due to metallic stains or silver-containing medicaments, bleaching is more difficult and it is sometimes not possible to achieve satisfactory results. In fact, internal discoloration caused by oxidation of metals (silver, amalgam) cannot be removed by whitening treatments (Attin et al. 2003).

8.6 Adverse Effects

Hydrogen peroxide is the main toxic substance that has been used for internal bleaching (Chaps. 4 and 5). Hydrogen peroxide is a reactive oxygen species (ROS) (Bax et al. 1992) with oxidative ability (Kashima-Tanaka et al. 2003), as seen in Chap. 3. It is well known that free radicals and ROS exert biological actions such as inflammation, carcinogenesis, aging, and mutation (Valko et al. 2007). ROS also play an important role in tissue injury at sites of inflammation in various diseases (Valko et al. 2007).

8.6.1 External Cervical Resorption (ECR)

Table 8.2 displays a summary of the findings in clinical studies of internal whitening and the reported number of external cervical resorption (ECR) cases. ECR is the loss of tooth hard tissue as a result of odontoclastic activity (Patel et al. 2009). Figure 8.3 shows cone-beam computed tomography images of ECR in endodontically treated teeth that had been unsuccessfully whitened internally prior to the restorative procedures.

It has been reported that hydrogen peroxide is the cause of dentin and cementum alterations leading to complications such as ECR (Harrington and Natkin 1979; Lado et al. 1983; Montgomery 1984). Hydrogen peroxide seeps through the dentinal tubules into the surrounding tissues causing destruction of cells, which triggers an inflammatory process that may lead to ECR. A study suggested that the combination of heat and hydrogen peroxide is responsible for ECR (Madison and Walton 1990). *However*, *other studies reported clinical cases of ECR in which heat was not used* (Goon et al. 1986; Friedman et al. 1988). Even though the walking bleach technique with sodium perborate and hydrogen peroxide is less harmful than the thermocatalytic bleaching technique, ECR may also occur with the walking bleach technique (Goon et al. 1986; Latcham 1986; Friedman et al. 1988). As hydrogen peroxide has been used in most studies that reported ECR, several authors have

Table 8.2 Clinical studies of internal whitening

	Number of teeth	ECR	History of trauma	Outcome
Harrington and Natkin (1979)	4 central incisors were root canal treated in 4 patients whose age ranged from 11 to 15 years of age. In 3 of the 4 cases, the thermocatalytic bleaching technique was carried out from 6 to 15 years after the trauma and completion of root canal therapy Same authors reported 3 extra cases that developed ECR	All with ECR; all resorptive lesions occurred in the cervical third of the root	All 4 teeth	No reported outcome
Cvek and Lindvall (1985)	11 teeth with ECR after bleaching with 30 % H_2O_2	2 teeth – superficial ECR that did not progress; 5 teeth – ECR followed by ankylosis. 4 teeth – ECR was progressive and associated with radiolucency in the adjacent alveolar bone	10/11, when patients were 11–16 years old	
Abou-Rass (1988)	112 severely tetracycline-stained teeth in 20 patients were root canal treated and internally bleached with a thick paste of sodium perborate in 30 % H_2O_2. Procedure was repeated after 1 week if needed.	No report of ECR after 3–15 years	No history of trauma	7 % failure – 8/112 teeth were noticeably dark at the cervical zone Intracoronal restorative failures were relatively high (7 %), endodontic failure was only 2 % and there was no evidence of external root resorption

	Number of teeth	ECR	History of trauma	Outcome
Friedman et al. (1988)	58 bleached teeth were re-examined after 1–8 years	4/58 (6.9 %); resorption started apically; 2 teeth had advanced ECR; 2 teeth had arrested ECR, one of them had been bleached with the walking bleach technique	No trauma for the 4 teeth with ECR	43 teeth (74 %) were bleached once, 15 teeth (26 %) were more than once; all teeth bleached with 30 % H_2O_2; 29/58 teeth (50 %) were found esthetically satisfactory; 17/58 teeth (29 %) were clinically acceptable; 12/58 teeth (21 %) were unacceptable, of which 4 had received full-coverage restorations
Holmstrup et al. (1988)	95 teeth, walking bleach technique with sodium perborate moistened with water	No report of ECR at 3 years	91/95 teeth had history of trauma	In 57 teeth (60 %), a good or acceptable result after 1 or 2 visits. The remaining 38 teeth were treated over 3–9 visits. Satisfactory initial result in 90 % of the cases After 3 years, recurrence of discoloration was observed in 20 % of the teeth
Anitua et al. (1990), Aldecoa and Mayordomo (1992)	258 intact tetracycline-stained teeth underwent elective root-canal treatment; GIC cervical barrier 1 mm below CEJ, walking bleach technique with 30 % H_2O_2 and sodium perborate; repeated 2–3 times every 4 weeks. A mix of 10 % carbamide peroxide + sodium perborate was then applied for 4–6 weeks	No report of ECR at 4 and 6 years	No history of trauma	Color relapse for 6/258 teeth (2.3 %) at the 4-year recall; 10 % of teeth after 6 years
Heithersay et al. (1994)	204 teeth were re-examined after 1–19 years; all teeth had been treated with a combination of thermocatalytic and walking bleach procedures using 30 % H_2O_2	4 teeth (1.96 %) developed invasive ECR. All of these teeth had a history of traumatic injury and the level of gutta-percha was at the CEJ without a barrier	151/204 (77.9 %) had history of traumatic injury	Not reported, as this study was based on radiographic evaluations

(continued)

Table 8.2 (continued)

	Number of teeth	ECR	History of trauma	Outcome
Glockner et al. (1999)	5-year clinical follow-up of teeth bleached with 30 % H_2O_2 with sodium perborate, walking bleach technique for 1 week; procedure repeated until satisfactory results obtained	Not reported	Not reported	Treatment was successful in 68 patients (79 %) after 5 years
Amato et al. (2006)	Thermocatalytic technique used with 35 % H_2O_2 and sodium perborate heated with light source; 50 teeth initially selected, 35 were evaluated at 16 years	None of the 13 failures had radiographic signs of ECR. However, authors stated that for the 9 teeth for which the root canal that had been re-treated, 2 of them *showed fistula, pain and a peri-radicular and/or latero-radicular bone lysis area that had failed to disappear or had reappeared*	42 of the initial 50 teeth has a history of trauma	22 teeth (62.9 %) the color had remained stable and was similar to that of adjacent teeth; 13 cases (37.1 %) classified as failures because of marked color relapse

Excluding case reports of one tooth

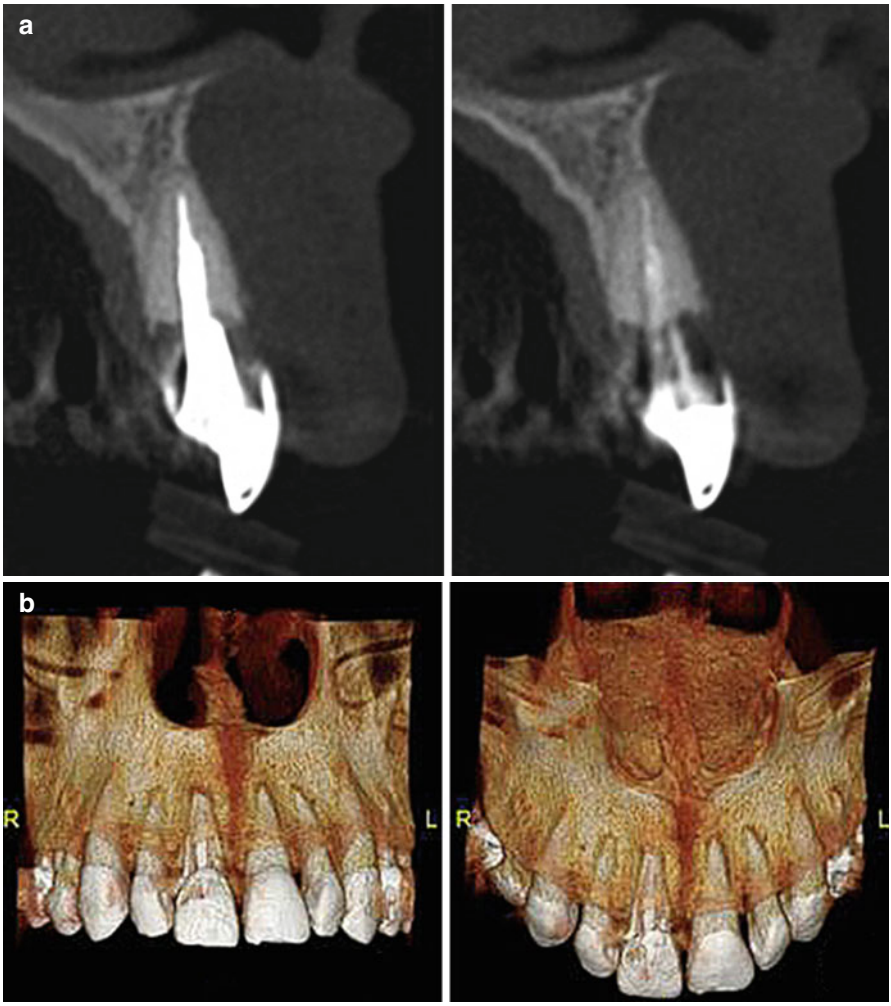

Fig. 8.3 Cone-beam computed tomography (**a**) with reconstructed three-dimensional (3D) image (**b**) showing an ECR lesion on tooth #8 (FDI 1.1). This tooth had been treated with intracoronal whitening in two different occasions, but did not respond to the treatment. The ECR lesion was diagnosed after the tooth was restored with a cast post-and-core and a full-coverage restoration (Images courtesy of Prof. Eduardo Vilain de Melo, Florianópolis, Brazil)

opposed the use of hydrogen peroxide for internal whitening to prevent ECR (Montgomery 1984; Cvek and Lindvall 1985).

The first cases of ECR associated with intracoronal bleaching were published in 1979 (Harrington and Natkin 1979). This paper involved four cases of post-trauma pulpal necrosis in permanent teeth of young patients, ranging from 11 to 15 years of age. The four cases developed ECR lesions in the cervical third of the root. In three of the cases, intracoronal bleaching was performed 6–15 years after the trauma and

subsequent endodontic treatment. Superoxol and a heat source (bleaching tool and a heat lamp) were used for the thermocatalytic bleaching technique. Although traumatic injury to the teeth was a common factor in the patients' history, other studies reported cases of root resorption in teeth without history of trauma (Lado et al. 1983; Goon et al. 1986; Friedman et al. 1988).

The same clinical report (Harrington and Natkin 1979) described three additional similar cases in which ECR also occurred. The authors hypothesized that ECR may have been caused by (1) the bleaching materials passing through the wide dentinal tubules into the periodontal ligament, which triggered an inflammatory resorption; or (2) an injury to the periodontal tissues by heat from the heat lamp or bleaching tool used in these cases.

Contrary to suggestions that ECR is more frequent in traumatized teeth of young patients some authors have reported that bleaching-related ECR is not more likely to occur in young teeth with wide dentinal tubules (Friedman et al. 1988). In this study resorption occurred in 3 of 34 teeth of patients older than 20 years, but only in 1 of 24 teeth of patients younger than 20 years.

Bleaching with sodium perborate and water has been advocated as a low risk alternative to sodium perborate and hydrogen peroxide to prevent ECR lesions (Holmstrup et al. 1988; Madison and Walton 1990). As discussed in Sect. 8.4.2, the walking bleach technique with a paste of sodium perborate and water is currently the standard intracoronal bleaching technique.

Several factors associated with ECR have been described in the literature:

(a) A causal relationship between a specific bleaching technique and ECR is still not clear. Friedman et al. (1988) reported that ECR was not dependent on the bleaching method. Authors reported that 7 % of the teeth displayed ECR in 58 cases monitored during a period of 1–8 years. Contrary to previous reports, the lesions initiated apical to and not at the cementum-enamel junction (CEJ).

(b) A factor that may increase the risk of ECR associated with hydrogen peroxide used in internal whitening is the anatomy of the CEJ. *The penetration of hydrogen peroxide is significantly higher in teeth with cementum defects at the CEJ than in those without defects* (Rotstein et al. 1991b). To prevent the seepage of hydrogen peroxide to the periodontal tissues, Madison and Walton (1990) suggested that bleaching procedures should be confined to the supragingival part of the pulp chamber to prevent chemicals from contacting tubules that communicate with the cervical periodontal tissues.

The placement of a cervical barrier between the pulp chamber and the endodontic filling material has been advocated (He and Goerig 1989; Anitua et al. 1990). Originally the location of the barrier was 1 mm apical to the CEJ (He and Goerig 1989). Other authors suggested that the barrier should be placed from the CEJ level to 2 mm below the CEJ with a minimum thickness of 1 mm (Costas and Wong 1991; Rotstein et al. 1992b). When the barrier was placed 2 mm below the CEJ the esthetic bleaching result from a walking bleach technique with sodium perborate and Superoxol was more acceptable than when the barrier was placed at the CEJ level (Costas and Wong 1991). A 1 mm-thick

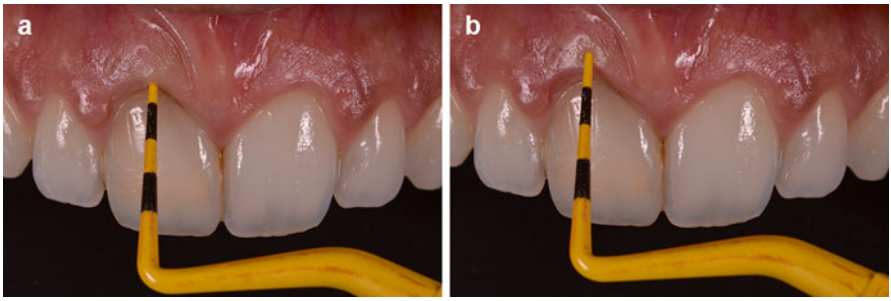

Fig. 8.4 (**a**, **b**) A periodontal probe is used to determine the level of the epithelial attachment from the incisal edge of the tooth. This will serve as guide for placement of the root canal barrier

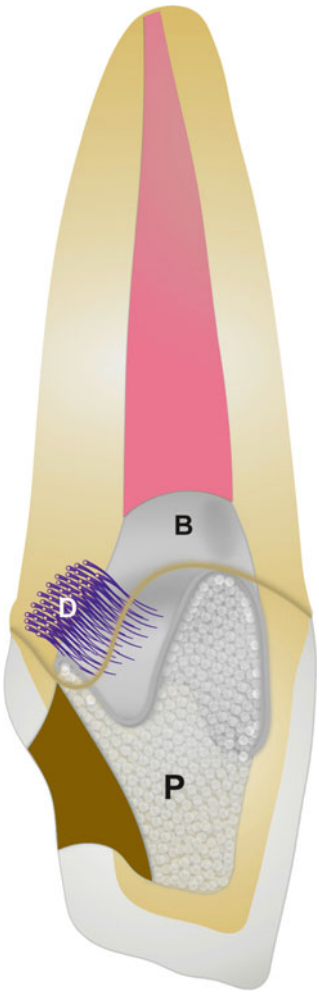

Fig. 8.5 Diagram depicting a coronal extension of the barrier that matches the contour of the epithelial and increases the safety of the bleaching procedure by sealing a wider area against the leakage of peroxides to the periodontal tissues (Adapted from Steiner and West 1994). *D* dentin tubules, *B* cervical barrier, *P* paste of sodium perborate and distilled water

I.R.M. (Dentsply Caulk) barrier resulted in an increased penetration of hydrogen peroxide compared to a 2 mm-thick barrier (Rotstein et al. 1992b).

A mesial, distal, and labial periodontal probing is used to determine the level of the epithelial attachment from the incisal edge of the tooth (Fig. 8.4). This will guide the dental professional to decide the location of the barrier. A coronal extension of the barrier to match the contour of the epithelial attachment (Fig. 8.5) has been proposed to increase the safety of the bleaching procedure by sealing a wider area against the leakage of peroxides to the periodontal tissues (Steiner and West 1994).

ZOE-based materials, such as I.R.M. (Dentsply Caulk), and classical glass-ionomer cement (GIC) materials are not currently recommended for the cervical barrier, as they do not completely prevent leakage of bleaching agents into the coronal part of the root canal (Rotstein et al. 1992b; Brighton et al. 1994).

(c) The pH of the paste left inside the pulp chamber is more acidic when the consistency is too liquid (Rotstein and Friedman 1991). Kehoe (1987) had reported that the pH of dentin and cementum became more acidic after sealing the walking bleach material in the pulp space of endodontically treated teeth, creating the ideal environment for osteoclastic activity. Rotstein and Friedman (1991) reported that *thick pastes* used with the walking bleach technique are alkaline and their alkalinity increases with time. Initially, the pH of sodium perborate mixed with distilled water without dilution was 9.87, but it increased gradually to 10.70 during the following 14 days. The initial pH of sodium perborate mixed with Superoxol was 7.40 and it reached a value of 10.58 after 14 days. In both cases, a marked increase in pH occurred during the first 48 h. Accordingly, it is unlikely that ECR is caused by the acidity of current materials used for intracoronal bleaching.

(d) The type of sodium perborate used in the walking bleach paste is another factor to consider, as the amount of hydrogen peroxide leakage to the periodontal tissues may depend on the form of sodium perborate used. The use of sodium-tetrahydrate mixed with water is recommended as a bleaching agent to reduce the risk of potential development of bleaching-related ECR (Weiger et al. 1994).

(e) Superoxol mixed with sodium perborate increase the solubility of dentin and cementum. It was concluded that 30 % hydrogen peroxide might cause alteration in the chemical structure of the dentin and cementum, such as reduction of the organic component, making them more susceptible to degradation (Rotstein et al. 1992a).

(f) The removal of the smear layer from the dentin walls of the access cavity may increase the diffusion of the whitening agent through the dentinal tubules. Since there is a higher hydrogen peroxide diffusion when the access cavity is etched with phosphoric acid or rinsed with EDTA followed by NaOCl (Surapipongpuntr et al. 2008; Camps et al. 2010), it may be prudent to preserve the smear layer to decrease the risk of ECR (Camps et al. 2010). Hydrogen peroxide is a potent stimulator of osteoclastic bone resorption. A significant increase in bone resorption was noted when rat osteoclasts, cultured on bovine cortical bone, were exposed to hydrogen peroxide (Bax et al. 1992).

(g) Some authors have suggested that bacteria in the gingival sulcus or in the pulp chamber may play a role in the root resorption process (Cvek and Lindvall

1985; Rotstein et al. 1991a; Heling et al. 1995). Hydrogen peroxide in high concentrations may increase bacterial penetration through dentinal tubules (Heling et al. 1995). This pathway for bacterial invasion may be a consequence to structural defects or pathological alterations of the cementum (Rotstein et al. 1991b).

(h) $Ca(OH)_2$ (calcium hydroxide) is effective in arresting external inflammatory root resorption (Heithersay 1975). Several cases of bleaching-induced root resorption have been treated successfully by intracoronal application of $Ca(OH)_2$ (Montgomery 1984; Gimlin and Schindler 1990). The pH increases in dental tissues after endodontic treatment with $Ca(OH)_2$, which has a positive influence on the local environment of the resorption areas by preventing osteoclastic activity and by stimulating the repair processes of the tissue (Tronstad et al. 1981). As dentinal tubules become wide open when resorption occurs, a communication between the pulp cavity and the periodontal tissues is formed. It has been proposed that $Ca(OH)_2$ placed in the pulp cavity penetrates the dentinal tubules to increase the pH in the root periphery and to promote repair (Tronstad et al. 1981). However, both $Ca(OH)_2$ paste and the resulting hydroxyl ions have been shown to diffuse poorly through dentin (Wang and Hume 1988; Fuss et al. 1989). Additionally, the therapy with $Ca(OH)_2$ was not capable of stopping the resorptive process in clinical cases of young patients treated with the walking bleach technique (Goon et al. 1986; Latcham 1986; Friedman 1989). Bleaching-induced resorption in dogs was observed regardless of the presence of $Ca(OH)_2$, suggesting that *Ca(OH)2 is unable to always prevent root resorption* associated with internal bleaching (Rotstein et al. 1991a).

(i) The resorption process does not seem to involve macrophages (Jimmenez Rubio and Segura 1998), as no correlation was found between the mechanism of action of sodium perborate and the adhesive properties of macrophages.

8.6.2 Ankylosis

Cvek and Lindvall (1985) followed up 11 maxillary incisors in 9 patients with radiographic evidence of post-intracoronal bleaching ECR. Ten of the teeth had been endodontically treated as a consequence of traumatic injury at ages 11–16 years. The bleaching treatment was performed in the traumatized teeth 12–90 months (mean of 48 months) after the accident. In five teeth, the resorption was associated with ankylosis, which may have been caused by loss of vitality of periodontal tissue attached to the root surface as a result of hydrogen peroxide seepage from the pup chamber.

8.6.3 Alterations in the Physical Properties of the Residual Tooth Structure

Intracoronal bleaching affects the ultimate tensile strength and ultrastructure morphology of bovine dentin. Cavalli et al. (2009) used sodium perborate, 35 % carbamide peroxide, 25 % hydrogen peroxide, and 35 % hydrogen peroxide. Bleaching

was performed four times within a 72 h interval. Dentin ultimate strength was significantly higher for the control group, in which dentin was not bleached. More details on this topic are discussed in Chap. 4.

Different intracoronal whitening agents affect dentin fracture strength (Carrasco-Guerisoli et al. 2009). Bovine teeth were subjected to several intracoronal bleaching techniques. Controls were treated with either sodium perborate mixed with 10 % hydrogen peroxide or no bleaching agent. Whitening systems with higher pH did not result in perceptible changes of dentin ultrastructure. Apparently, both low pH and hydrogen peroxide oxidation play a role in altering the ultrastructure of dentin during internal dental bleaching. The use of alkaline products with reduced time of application, as those used for in-office whitening techniques, may prevent such morphological alterations (Carrasco-Guerisoli et al. 2009).

Restorative procedures using composite resin have been described to successfully restore the fracture resistance of bleached endodontically treated teeth (Roberto et al. 2012).

8.6.4 Decrease in Enamel and Dentin Bond Strengths Immediately After Whitening

As described in Chap. 6, dentin and enamel bond strengths are significantly reduced in recently bleached teeth. A delay in adhesive restorative procedures is recommended for endodontically treated teeth that are bleached internally, even in cases in which a paste of sodium perborate with water is used without added hydrogen peroxide or carbamide peroxide. A waiting period of at least 2 weeks after bleaching is recommended prior to performing adhesive restorations with resin-based composite in both enamel and dentin (Shinohara et al. 2005). Nonetheless, some patients may not be able to return for a subsequent appointment to have the access cavity restored definitely with an adhesive technique. Since the access preparation will have to be restored immediately, the application of catalase may be indicated in these cases. Rotstein (1993) reported that one application of catalase for 3 min following intracoronal whitening of nonvital teeth totally eliminated the residual hydrogen peroxide from the pulp chamber and from surrounding periodontal tissues.

8.6.5 Chemical Burns of Soft Tissues

The application of catalase on the mucosa has a protective effect against the hydrogen peroxide-induced injury in animals (Rotstein et al. 1993b). Tripton et al. (1995) reported the in vitro ability of a concentration of catalase >20 U/ml to supress the toxic effects of peroxide on mucosal fibroblasts. As described in Chap. 4, sodium bicarbonate may also be used to treat these chemical burns caused by hydrogen peroxide.

8.7 The Walking Bleach Technique

Figure 8.6 shows a diagram of an endodontically treated tooth before removal of the access cavity restoration (A) and after the bleaching paste is sealed in the pulp chamber (B). Figure 8.7 is a step-by-step clinical sequence of the walking bleach technique.

1. Clinical and radiographic exam (Fig. 8.7a–c).
   (a) It is important to determine the likely source of the discoloration, as some stains are more difficult to remove as discussed in Sect. 8.5.5.
   (b) Check for defective coronal restorations that might have caused microleakage and subsequent internal discoloration. Defective restorations must be replaced temporarily to prevent the bleaching material to leak into the mouth.
   (c) In case of active periodontitis, internal whitening is deferred and the patient referred to a specialist.
   (d) Evaluate the quality of the root canal treatment and status of periodontal/periapical tissues.
   (e) Do not perform internal bleaching if a periapical radiolucency is observed in the respective radiograph.
2. It is of utmost importance to inquire the patient about his/her expectations. Inform patient of the number of sessions that may be needed, how slow the process may be initially, and the respective limitations, including the potential for color regression and the possibility that the color of intact teeth is not attained.
3. Document the pretreatment tooth color with dental photography placing a shade guide (Vita Classical A1-D4 shade guide (VITA Zahnfabrik H. Rauter GmbH & Co. KG) adjacent to the discolored tooth. It is important to document the preoperative color for future reference.

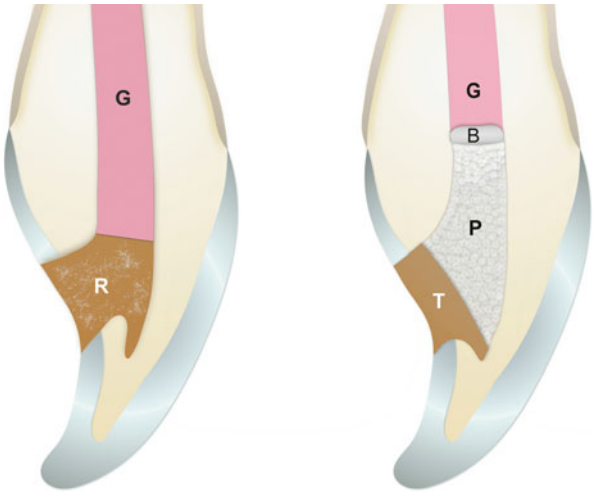

Fig. 8.6 Diagram of an endodontically treated tooth before removal of the access cavity restoration (**a**) and after the bleaching paste is sealed in the pulp chamber (**b**). *G* Gutta-percha, *R* restoration of the access cavity, *B* cervical barrier, *P* paste of sodium perborate and distilled water, *T* temporary restoration between walking bleach sessions

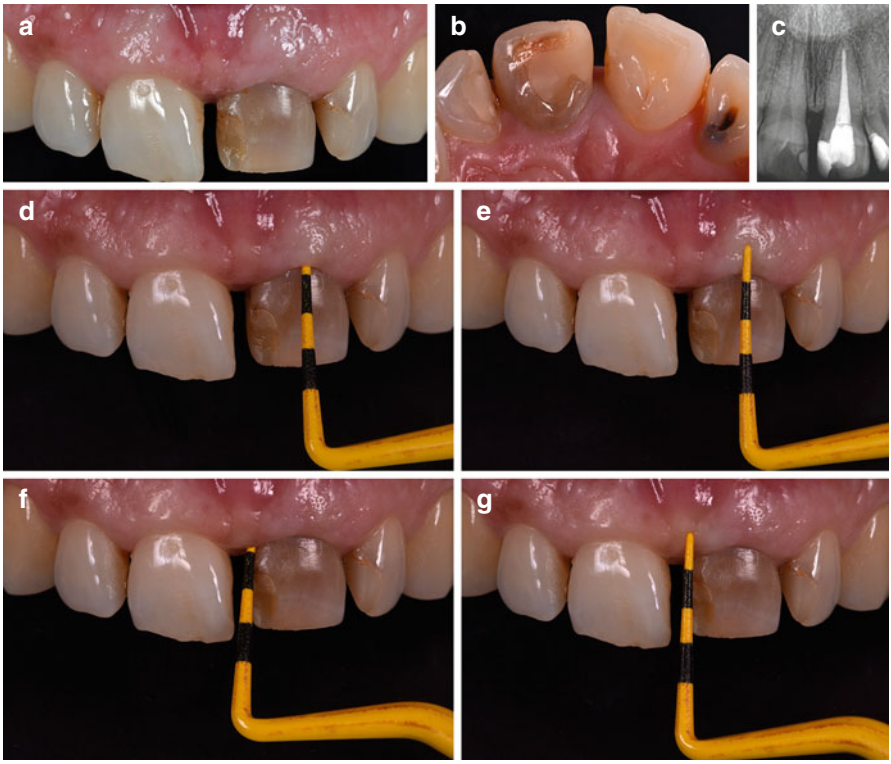

Fig. 8.7 Clinical sequence of the walking beach technique. (**a**) Preoperative frontal view showing a gray/brown discoloration of tooth #9 (FDI 2.1). Patient mentioned that this tooth had been darker than the other teeth for at least 7 years. (**b**) Preoperative palatal (lingual) view. (**c**) Periapical radiograph. (**d**–**i**) Probing sequence to determine the level of epithelial attachment. (**j**) Isolation with rubber dam. (**k**) Removal of coronal restoration and any residual pulp tissue left in the pulp horns. (**l**) A periodontal probe is used to transfer the exact depth level for placement of the root canal barrier. (**m**) Lingual view of gutta-percha removal from the root canal cervical area using a System B Heat Plugger (Kerr Endodontics). (**n**) Frontal view of the System B Heat Plugger (Kerr Endodontics) showing the rubber stop reference corresponding to the same length shown in the periodontal probe of (**l**). (**o**) A resin-modified glass-ionomer material (Vitrebond Plus, 3M ESPE) is applied into the root canal as a 2 mm-thick cervical barrier between the pulp chamber and the endodontic filling material. (**p**) The RMGIC material is light cured from the cavity access for 40 s. Any excess material must be removed with carbide bur in slow speed. (**q**) Sodium perborate is mixed with distilled water to a consistency of slightly moist sand and inserted into the pulp chamber with a plastic instrument or other suitable instrument. (**r**) The pulp chamber is filled with the bleaching paste short of the access cavo-surface margin. (**s**) Residual liquid is removed with dry cotton pellets by gently compressing the paste. A clearance of 1.5 mm is needed to insert a temporary restoration. (**t**) Reinforced zinc oxide eugenol (ZOE) cement (I.R.M., Dentsply Caulk) is used as temporary restorative material. (**u**) Frontal view of tooth #9 (FDI 2.1) immediately after removing the rubber dam. The tooth is apparently lighter as a result of dehydration. (**v**) Frontal view of tooth #9 (FDI 2.1) at the beginning of the second walking bleach session. (**w**) Frontal view of tooth #9 (FDI 2.1) after three walking bleach sessions

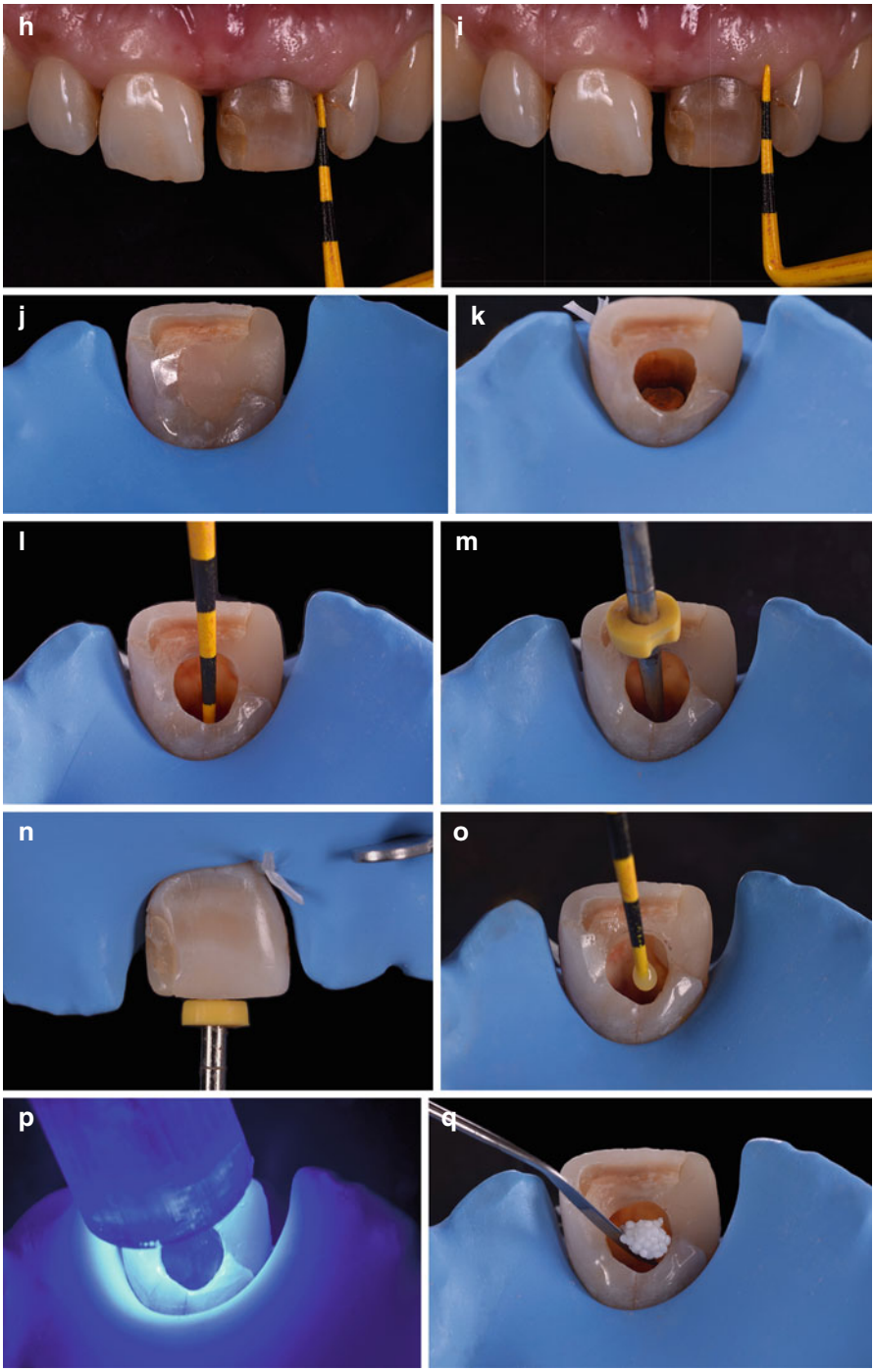

Fig. 8.7 (continued)

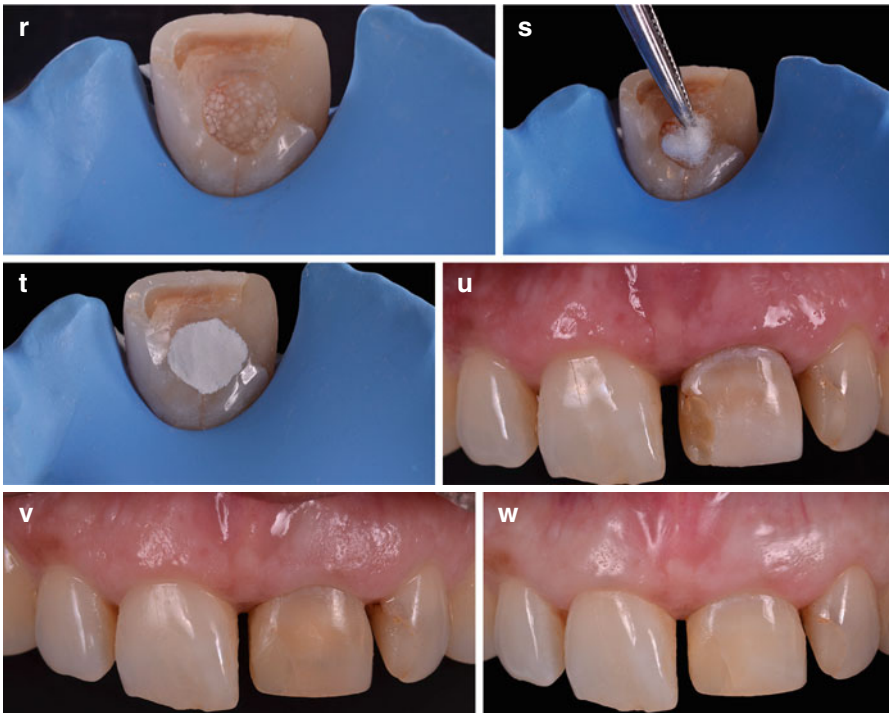

Fig. 8.7 (continued)

4. Periodontal probing determines the level of the epithelial attachment from the incisal edge of the tooth (Fig. 8.7d–i). This will serve as guide for placement of the root canal barrier.
5. Rubber dam isolation is required. The use of a light-cured resin to seal the gingival margin and the interdental papillae may be needed, such as Opaldam (Ultradent Products Inc.).
6. The restorative material is removed from the access cavity and from the pulp chamber (Fig. 8.7j, k). The angulation of the bur must be parallel to the long axis of the anterior tooth being bleached. Often inexperienced clinicians remove the materials from the access preparation with the bur angled facially (Fig. 8.8), which results in inadvertent removal of sound dentin. In rare cases, the dentin structure on the buccal aspect is heavily discolored; therefore, it must be carefully removed in slow speed to avoid facial perforation.
7. At this time, check for residual discolored pulp tissue in the pulp horns and remove it if necessary.
8. A periodontal probe is used to transfer the exact depth level (Fig. 8.7l) for placement of the root canal barrier using the reference measured in Fig. 8.7d–i.

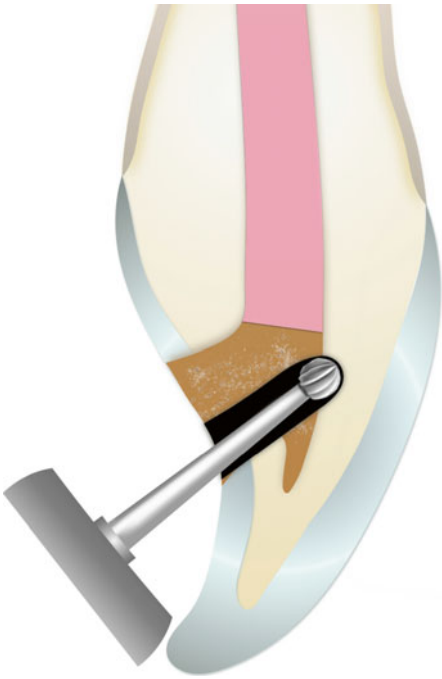

Fig. 8.8 Clinicians must be very careful when removing the restorative materials from the access preparation to avoid removing too much sound tooth structure from the buccal aspect, causing enamel perforation

9. The root canal obturation material is removed from the cervical area of the root (Fig. 8.7m, n), using a System B Heat Plugger (Kerr Endodontics) or a Peeso reamer. A 2-mm thick layer of a resin-modified glass ionomer material, also known as RMGIC (Fig. 8.7o) is applied into the root canal as a cervical barrier between the pulp chamber and the endodontic filling material (Fig. 8.6) to prevent the escape of hydrogen peroxide apically into the root canal and to the periodontal tissues. A polycarboxylate cement may also be used in lieu of the RMGIC, as both materials bond chemically to dentin. Ideally the cervical barrier should match the contour of the epithelial attachment, as proposed by Steiner and West (1994) (Fig. 8.5). The RMGIC material is light cured from the cavity access for at least 40 s (Fig. 8.7p). Any excess material in the pulp chamber must be removed with a carbide but in slow speed.
10. Sodium perborate is mixed with distilled water (anesthetic solution and saline have also been used) to a consistency of slightly moist sand. This mix must possess enough consistency to be inserted into the pulp chamber with a plastic instrument (Fig. 8.8q) or plugged with an amalgam carrier. In case the mix is too runny, it is extremely difficult to insert it adequately into the pulp chamber. Once the paste is inside the pulp chamber (Fig. 8.8r), the residual liquid is removed with dry cotton pellets by gently compressing the paste (Fig. 8.7s).

11. A clearance of at least 1.5 mm is needed to insert a temporary restoration into the access cavity. The area must be gently air-dried, otherwise the restorative material will dislodge easily. Some clinicians use Cavit (3M ESPE) as the temporary restorative material but it may dissolve in the mouth and result in an open access cavity. Reinforced zinc oxide eugenol (ZOE) cement I.R.M. (Dentsply Caulk) (Fig. 8.7t) is recommended. Another temporary restorative technique involves the use of a temporary low-viscosity resin-based material without an intermediate adhesive after acid etching enamel. This will provide a short-term efficient seal and micromechanical retention. After removing the rubber dam, the tooth is often dehydrated (Fig. 8.7u).
12. Patient is informed that he/she needs to return for a subsequent appointment in 1 week to possibly repeat the procedure, and 2 weeks thereafter (Fig. 8.7v, w). Alternative options may have to be considered when results of intracoronal whitening are not acceptable after the third internal bleaching session.
13. A waiting period of at least 2 weeks after the conclusion of the bleaching treatment is recommended prior to performing the definitive adhesive restorations with composite.

8.8 Summary

Internal whitening of endodontically treated teeth is still a good esthetic resource especially when a full-coverage restoration is not indicated or the patient prefers not to have the tooth restored with a full-coverage restoration. Patient must be informed that the chance of color relapse is greater than 25 %. Although ECR and ankylosis seem to occur more frequently in teeth that were exposed to traumatic injury and were bleached intracoronally with hydrogen peroxide, the absence of trauma and whitening with water/perborate mixture may still pose a slight risk of root ECR and ankylosis.

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Part III

Enamel Etching Techniques for Improvement of Tooth Color

Enamel Microabrasion for Removal of Superficial Coloration and Surface Texture Defects

9

Kevin J. Donly and Theodore P. Croll

Abstract

Enamel microabrasion is a method of removing superficial enamel coloration defects. Brown, white, and multicolored spots and streaks in tooth surfaces have been called "dysmineralization," and in some cases, such as fluorosis, their cause is known. In other cases in which etiology of the defect is unknown, the proper diagnosis is "idiopathic enamel dysmineralization." Decalcification defects, which are white, result from acid dissolution of the outer layer of enamel and are the first stage in the dental caries process. Analogous to dermabrasion on skin surfaces, enamel microabrasion permanently removes coloration defects with a compound made of dilute hydrochloric acid and a fine abrasive powder, in a water-soluble silica gel. The enamel surface is reduced microscopically by simultaneous abrasion and erosion, and enamel loss is insignificant and unrecognizable. This chapter reviews the procedure of microbrasion of enamel surfaces and gives examples of treatment for decalcification, dysmineralization, and surface texture improvement. A fourth case shows 27-year postoperative results.

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9.1 Introduction

In the early 1900s, Dr. Walter Kane used muriatic (hydrochloric) acid to erode endemic brown and white fluorosis discoloration from the anterior teeth of people in Colorado. Forty-four-year postoperative results of one of those patients (E. E.G.) were pictured in an article by Dr. Robert McCloskey, published in the *Journal of the American Dental Association* (McCloskey 1984). She had been treated in 1926 as a 16-year-old. Sixty years after Dr. Kane's treatment (1986), the same patient was photographed, then 76 years old (Fig. 9.1) (Croll 1987). A stark difference was noted in the appearance of her incisors and canine teeth, compared to the untreated posterior teeth. Some considered Dr. Kane's method a type of dental bleaching, but actually, the hydrochloric acid was dissolving the outermost layer of enamel that contained the unsightly brown and white discoloration. As evidenced by the 60-year postoperative view, the amount of enamel lost in treatment was unrecognizable and of no long-term consequence (Fig. 9.1). E.E.G. reported that her "upper and lower front teeth" (anterior) were treated by Dr. Kane, and "the teeth in back" (premolars and molars) were not. She also remembered that Dr. Kane had warmed the teeth with careful application of a flame, using a small torch. She did not recall any discomfort from that. The results, as seen in the photograph, are dramatic and the treated teeth give a much better appearance than those not "color-corrected" with Dr. Kane's muriatic acid/heat method.

Croll used a combination of 18 % hydrochloric acid mixed with fine grit laboratory pumice with the idea that combining chemical erosion with mechanical abrasion would also reduce the enamel surface microscopically, but more rapidly, and with the clinician in control. In 1986, Croll and Cavanaugh first reported this type of approach and termed the treatment "enamel microabrasion" (EM) (Croll and Cavanaugh 1986a, b). After much more experience, a textbook followed (Croll 1991).

Enamel microabrasion is analogous to dermabrasion on skin surfaces. It represents a most conservative method of removing intrinsic, yet superficial, enamel dysmineralization (*ibid*, *pg 22*), decalcification, and texture defects avoiding the need for restorative masking with artificial materials such as bonded resin-based composite or bonded porcelain veneers. Successful microabrasion removes an insignificant and unrecognizable amount of surface enamel and with it, the offending discolored or

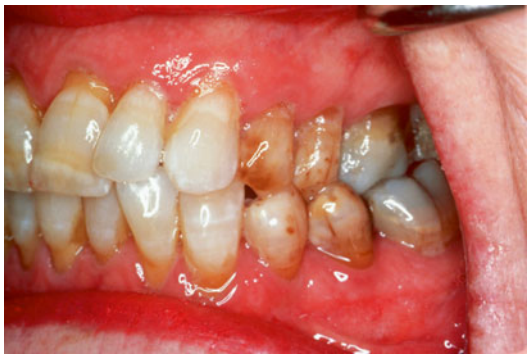

Fig. 9.1 Sixty-year results of eliminating brown fluorosis stain using hydrochloric acid. Anterior teeth treated, posterior teeth untreated

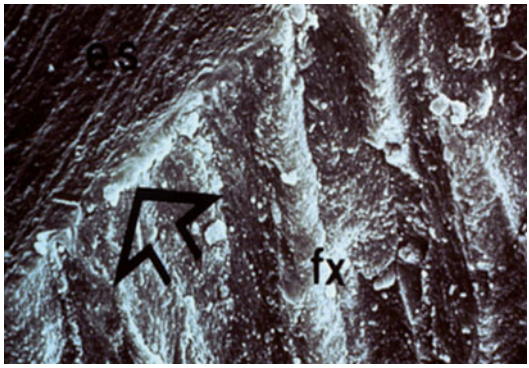

Fig. 9.2 A scanning electron microscopic view of the polished enamel surface following microabrasion and the natural appearance of enamel below the polished surface in this fractured (*fx*) enamel specimen

"maltextured" layer. Oftentimes, enamel microabrasion can be combined with dental bleaching (Haywood and Heymann 1989) for optimum enhancement of tooth appearance (Cvitko et al. 1992; Killian 1993; Croll 1992, 1998).

Enamel microabrasion has become a routine clinical procedure in dentistry (ADA treatment code D9970) and commercial microabrasion products (PREMA®, Premier Dental Products, Plymouth Meeting, PA) (OPALUSTRE®, Ultradent Products, South Jordan, UT) are available to facilitate treatment. These products contain a low concentration of hydrochloric acid (6–9 %) and silicon carbide abrasive powder in a silica gel, for rotary application. Research has shown that use of these products, followed by natural remineralization, creates a lustrous enamel surface that not only is more resistant to acid challenge, but also accumulates less dental plaque than untreated surfaces (Segura et al. 1997a, b). Polarized light microscope and scanning electron microscope studies have revealed that microabrasion results in an enamel surface with a superficial layer of compacted, aprismatic mineral that gives a glass-like clinical appearance. The microabrasion technique removes some surface enamel but also packs some of the abraded calcium and phosphate into the interprismatic spaces to create a highly dense and polished surface (Fig. 9.2) (Berg and Donly 1991; Donly et al. 1992). Combining mechanical abrasion and chemical erosion inspired the terms "abrosion effect" (Donly et al. 1992) and its result, the "enamel glaze."

9.2 Enamel Microabrasion Procedure

The enamel microabrasion procedure can be outlined as follows:

1. Determination is made whether the tooth discoloration or texture defect is relatively superficial. Enamel microabrasion is not indicated for tooth discolorations such as seen with tetracycline dentinal stain, dentinogenesis imperfecta, or deep enamel hypoplastic or hypocalcification defects. EM treatment is indicated for enamel dysmineralization discolorations such as seen with brown and white fluorosis, idiopathic brown or white stain, or superficial enamel texture anomalies.
2. Pretreatment photographs are always advised, both for medico-legal considerations, and for education of the patient and parents.

3. Local anesthesia is used only if needed to facilitate rubber dam placement.
4. Rubber dam application or isolation with OpalDam® or OpalDam Green® (Ultradent Products, South Jordan, UT) is recommended. Protective eyewear for the patient is mandatory.
5. To hasten treatment results, a cylindrical diamond bur can be used at slow speed, to initiate enamel surface microreduction (Croll 1993).
6. Using either the PREMA® Enamel Microabrasion Compound with rotary polishing cups or OPALUSTRE® with OpalCups™, a small portion of slurry is applied to the tooth surface. A high-torque gear-reduction hand piece can be used, but with careful application to avoid splattering, treatment can be achieved safely with a standard slow-speed angle. After 5–10 s, using moderate pressure, the microabrasion compound is rinsed with water, and results observed.
7. Five 10-s applications are repeated until the coloration defect is eliminated. Determination of tooth appearance should be made while the tooth is wet.
8. After completion of microabrasion, the treated teeth should be saturated for several minutes with a fluoride-containing gel.

9.3 Representative Cases

Enamel microabrasion treatment of three patients is documented below. One had decalcification (Croll and Bullock 1994), another, enamel dysmineralization, and the third, congenital enamel texture malformation (Killian and Croll 1990). A fourth case is shown with 27-year postoperative enamel microabrasion results combined with tooth bleaching in the adult years.

9.3.1 Case One

A 16-year-old boy had white decalcification markings in the gingival half of his maxillary anterior teeth, related to inadequate oral hygiene during his orthodontic therapy (Figs. 9.3a–d). There was a caries lesion associated with the decalcification on the maxillary left canine tooth (Fig. 9.3b). The maxillary premolars also had facial decalcification spots. A small shear fracture was noted on the maxillary left first premolar, perhaps occurring during orthodontic bracket removal (Fig. 9.3b). The fractured region was smoothed with a fine-tipped diamond bur. White decalcification areas were seen on some mandibular teeth also, but none were noticeable when the patient spoke or smiled, and none had associated caries.

Treatment is shown in Fig. 9.3a–d.

9.3.2 Case Two

A 10-year-old boy had white and brown idiopathic white and brown dysmineralization, chiefly of the labial surfaces of the maxillary central incisors (Fig. 9.4a). The cause of the white and brown dysmineralization could have been too much systemic

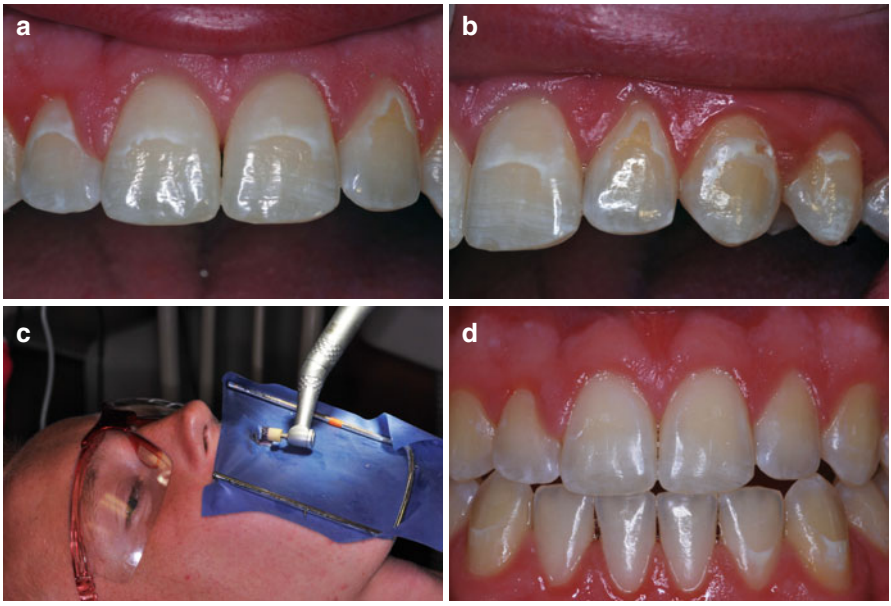

Fig. 9.3 (**a**) Decalcification from inadequate hygiene during orthodontics. (**b**) Left lateral view. Note canine Class V caries lesion and small shear fracture of first premolar. (**c**) Operative field and eye protection. PREMA® applied. (**d**) Three months after enamel microabrasion and resin-based composite restoration of the maxillary left canine tooth

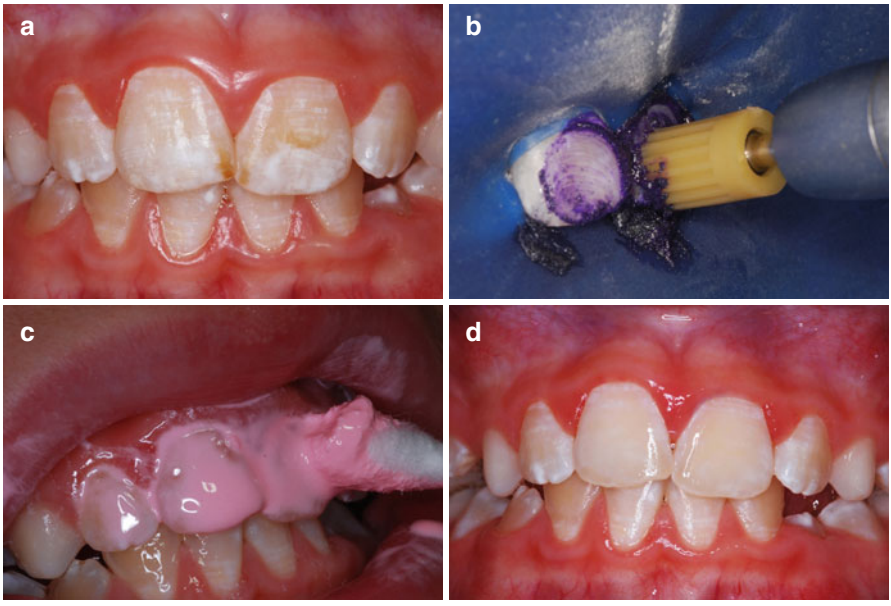

Fig. 9.4 (**a**) A 10-year-old with white and brown idiopathic enamel white and brown dysmineralization. (**b**) PREMA® applied with rubber applicator tip. (**c**) Fluoride gel applied after microabrasion completed. (**d**) Immediately after enamel microabrasion treatment

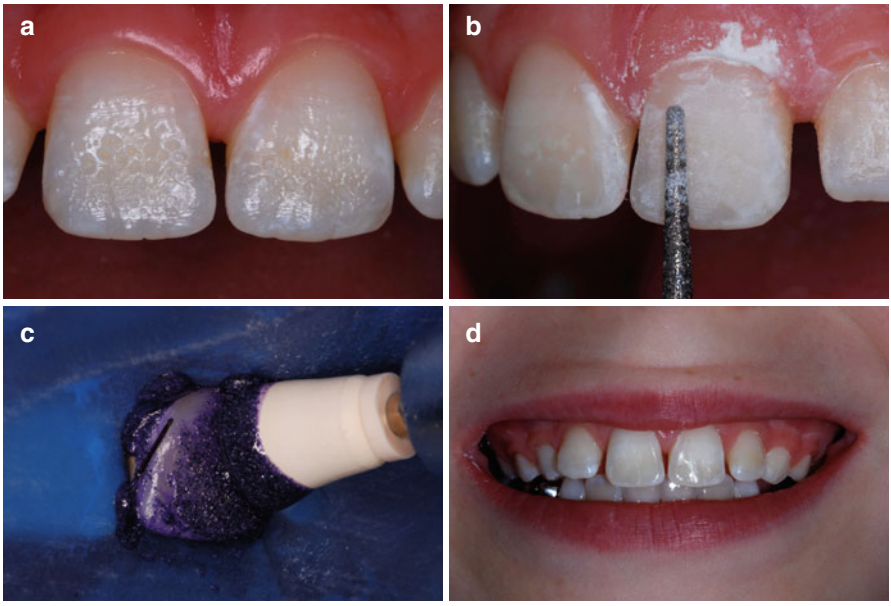

Fig. 9.5 (**a**) An 8-year-old with pitting type amelogenesis imperfecta. (**b**) Slow-speed diamond initiates enamel microreducation. (**c**) OPALUSTRE® slurry applied with rubber cup/brush assembly (OpalCup™). (**d**) Three months after enamel microabrasion, enamel texture is normal

fluoride in the years of amelogenesis, but the permanent first molars where not affected, and the parents could not identify a source of excess fluoride in the child's first decade. In addition, the family's water source was not fluoridated. The diagnosis was recorded as idiopathic white and brown enamel dysmineralization, possibly related to excess fluoride consumption in the early years. The parents were concerned only with the maxillary central incisors at the time of treatment. Additional enamel microabrasion for other teeth would be considered later. PREMA® Compound was used to treat these teeth (Fig. 9.4a–d).

9.3.3 Case Three

An 8-year-old girl had a pitting type of amelogenesis imperfecta (Fig. 9.5a). The labial surface of her maxillary incisors had multiple round notches that did not penetrate deeply into the surface. The maxillary central incisors were affected much more than the lateral incisors. Although the appearance of these incisors could have been substantially improved with bonded resin-based composite, it was felt that the enamel defects were superficial enough to be eliminated, rather than covered up (Killian and Croll 1990).

To hasten the procedure, an initial portion of the enamel removal was achieved with a slow-speed diamond bur prior to placement of the rubber dam (Fig. 9.5b) (Croll 1993). OPALUSTRE® Enamel Microabrasion Slurry was used in the same manner as PREMA® Compound in the other cases shown here. Ultradent's OpalCup™ with

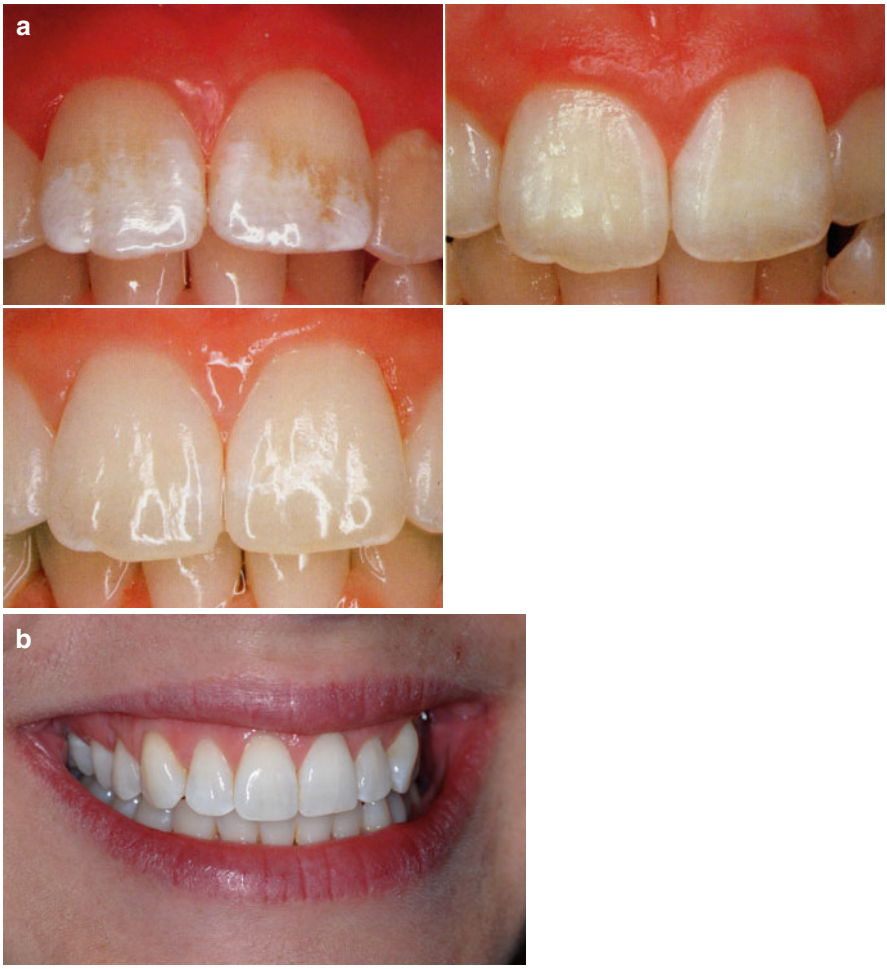

Fig. 9.6 (**a**) Enamel microabrasion of a 10-year-old in 1985, documented (Croll 1991). (**b**) Tooth appearance 27 years after enamel microabrasion and 2 years after custom-tray carbamide peroxide "home bleaching."

internal brush bristles was used to apply the slurry to all four incisors (Fig. 9.5c). Three months later, the incisors gave a much improved appearance (Fig. 9.5d).

9.3.4 Case Four

In 1985, a 10-year-old girl had white and brown idiopathic enamel dysmineralization discoloration of her maxillary central incisors. Enamel microabrasion was completed and treatment was documented, before, immediately after, and 5 years later, (Fig. 9.6a) (Croll 1991). Twenty-five years after treatment, the patient had custom-tray carbamide peroxide tooth bleaching, for additional tooth color improvement (Fig. 9.6b).

9.4 Discussion

After more than a quarter of a century of experience, clinical observations, and research, the following is known about enamel microabrasion:

- Enamel microabrasion is more conservative of tooth structure than treatments such as porcelain or resin-based composite veneering. It also is significantly less expensive.
- Enamel microabrasion results are permanent. The discoloration or texture abnormalities are removed not masked with artificial material that eventually will require additional intervention.
- The glass-like surface resulting from the abrasion/erosion combination resists acid dissolution and bacteria accumulation better than untreated enamel smooth surfaces (Segura et al. 1997a, b).
- Many intrinsic enamel surface defects are superficial enough to be eliminated without need to replace the lost enamel. Slight and moderate white and brown fluorosis discolorations are good examples of this type of dysmineralization. Teeth with deeper enamel defects are to be repaired with traditional restorative methods. In cases for which the clinician is unsure how deep the defect penetrates the surface, there is nothing to lose except clinical time, by attempting microabrasion initially.
- White enamel dysmineralization sometimes does not need to be completely removed during the microabrasion procedure. Even though residual white streaks or spots may show when the tooth is completely dry, such defects are often camouflaged when the tooth surface is saturated with body temperature saliva. Appearance of microabraded teeth should be assessed when wet with saliva, which is their usual state.
- Mechanical stripping of enamel with burs or disks can also remove superficial defects, but not in such a controlled manner and with the ensuing "enamel glaze" formation. However, time can be saved by beginning with mechanical enamel microreduction (diamond burs or disks), followed by completion of treatment with rotary application of the microabrasion compound.
- In many cases, the most striking results occur when enamel microabrasion is combined with dental bleaching. Microabrasion removes unsightly superficial defects and creates a lustrous smooth surface while tooth bleaching whitens the deep intrinsic yellow colorations of dentin that shine through the translucent enamel surface. Microabraded teeth that also undergo dental bleaching with hydrogen peroxide products, give a bright, white, and healthy appearance of long duration.

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Authors' Disclaimer The authors have no financial interest in any product or company mentioned in this chapter. The second author (TPC) formerly had financial interest in PREMA® and OPALUSTRE® by virtue of patent licensing agreements, which ended with expiration of the patents.

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Resin Infiltration After Enamel Etching

10

Sebastian Paris and Hendrik Meyer-Lueckel

Abstract

Buccal white spot lesions are an unpleasant but frequent side effect of orthodontic treatment with fixed appliances but also developmental defects such as fluorosis appear as "white spots." Resin infiltration was originally developed to arrest the progression of noncavitated caries lesions. The technique uses low-viscosity resins that penetrate the porous lesion body of white spot lesions. After infiltration, the material is light cured and thus blocks the diffusion pathways for acids and dissolved minerals. A positive side effect of resin infiltration is that infiltrated lesions lose their whitish appearance and look more similar to sound enamel. This effect can be used to camouflage aesthetically impairing white spot lesions. Using resin infiltration much less enamel is removed compared to micro-abrasion or restorative approaches. Nonetheless, significantly better aesthetic results compared with noninvasive approaches such as fluoridation can be achieved.

10.1 White Spot Lesions

White spots are among the most frequent dental aesthetic impairments. While most whitish discolorations are early, noncavitated caries lesions, they may also represent developmental defects with various etiologies such as fluorosis,

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posttraumatic lesions, or molar incisor hypomineralization (MIH). Common for all these defects is an increased porosity of the enamel that causes the whitish and chalky appearance.

Sound enamel is relatively translucent for the visible light. Due the homogeneous nature of enamel, which mostly consists of apatite crystals, light passes the enamel with only minor scattering (Fig. 10.1a). In white spot lesions, of whatever origin, the enamel structure is interspersed with porosities and voids. Apatite or enamel has a refractive index of 1.62, whereas the content of the porosities has a refractive index of approximately 1.33 when the lesion is wet or 1.0 when the lesions is dry (Kidd and Fejerskov 2004). Due to the different refractive indices of the different media, light is broken at the millions of interfaces between the pores and the enamel and thus the light is scattered (Fig. 10.1b). Because the difference in refractive index is even higher between dry pores and enamel compared to wet pores, dried lesions appear more whitish compared with wet lesions.

10.1.1 Initial Caries

Caries is formed after tooth eruption by dissolution of enamel induced by cariogenic acids being produced by cariogenic bacteria during fermentation of carbohydrates. In the past, caries was often regarded as a transmittable disease caused by an infection with bacteria like *Streptococcus mutans* (Keyes 1960). Today, caries is rather seen as a process triggered by the frequent intake of fermentable carbohydrates (sugars) and characterized by an ecological shift in the oral microbiology toward acidogenic and aciduric bacteria (Marsh 2003). Moreover, it is emphasized that multiple (mostly protective) factors such as oral hygiene, access to fluorides, or amount and composition of saliva influence the caries process (Paris and Meyer-Lückel 2012).

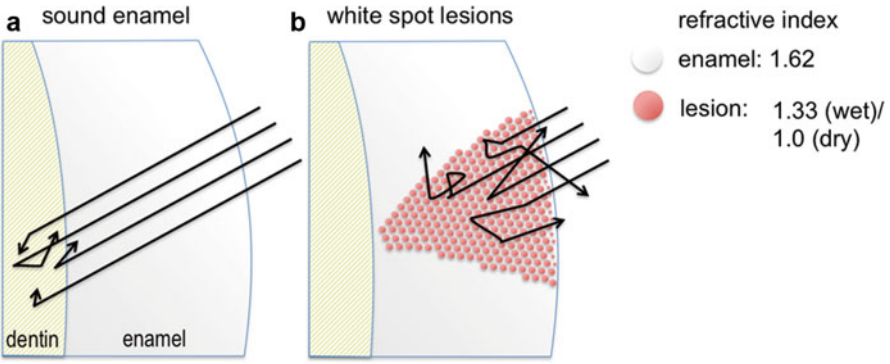

Fig. 10.1 Schematic illustration of the origin of the whitish opaque appearance of initial caries and developmental defects. (**a**) Sound enamel is relatively translucent for visible light. Light beams (*arrows*) are primarily broken within the dentin. (**b**) In white spot lesions, pores contain media with a lower refractive index compared to enamel. Therefore, the light is scattered between the pores and the surrounding apatite crystals. Thus, the lesion appears whitish

The cariogenic bacteria produce organic acids (e.g., lactic acid), which diffuse into the tiny pores of enamel and there dissolve the enamel apatite. This causes an increasing porosity within the enamel. Thus, in the initial stages of the disease the caries lesion is characterized by porous enamel. The word "initial" may be somewhat misleading because lesions that appear as white spots are often several hundred micrometer deep, often extending up the enamel dentin junction or even deeper. It may take weeks to months and years until a caries lesion reaches a stage where so much mineral is lost that the lesion collapses and a cavity forms. Nevertheless, caries is a dynamic process. If the interplay of multiple etiological factors shifts toward less cariogenic conditions, lesions can take up mineral from the saliva again and start to remineralize. Usually de- and remineralization cycles alternate several times a day, but if over time remineralization overweighs demineralization, the lesion does not progress but arrests. This remineralization is usually confined to the outer surface of the lesion where over time a so-called pseudo-intact surface layer is formed. This surface layer has usually a thickness of approximately 20–40 μm (but sometimes even more then 100 μm) and is considerably less porous than the underlying lesion body (Meyer-Lueckel et al. 2007). Thus, arrested or remineralized lesions look very similar to active progressing caries lesions except the higher mineralized surface. For this reason, it is a challenge to differentiate inactive lesions (enamel scars) from active progressing caries.

When white spot lesions arrest, along with the minerals also organic components such as food pigments can be incorporated into the surface layer. This is why some lesions may get a brownish discoloration and thus are called "brown spots" (Fig. 10.2).

Caries lesions usually develop in tooth sites where plaque formation is fostered such as in the fissure system or interproximal. On oral or buccal smooth surfaces plaque formation is usually increased at the cervical margin. Quite frequently, buccal caries lesions are observed after treatment with fixed orthodontic brackets. These appliances hamper the natural cleaning as well as oral hygiene and thus promote plaque formation. After removal of the brackets, these surfaces may be easily cleaned again. This improvement in oral hygiene leads to an arrest of many buccal white spot lesions. These lesions do not need any further treatment, as they are not signs of active disease, but enamel scars. From an aesthetic point of view, however, both active and inactive caries lesions are unattractive and may need intervention.

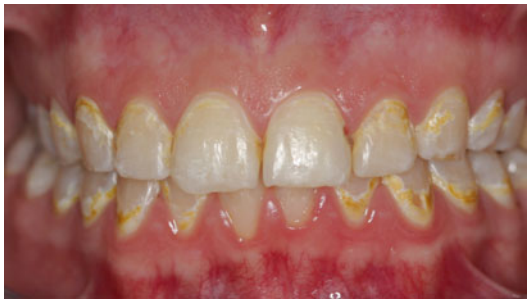

Fig. 10.2 Buccal caries lesions. A 21-year-old patient with high caries risk several months after debonding of orthodontic brackets. Non- and partly cavitated caries lesions are located in the cervical part of the buccal surfaces. The partly yellowish-brownish discoloration is a sign of inactivation. Thus, the lesions currently do not progress any more but are considered as an aesthetic impairment

Buccal early caries lesions can be easily differentiated from developmental defects (see below) as caries only forms in plaque stagnation areas and thus caries on buccal surfaces is confined to cervical areas or the areas around the bracket base (Lovrov et al. 2007; Mitchell 1992).

10.1.2 Developmental Defects

Developmental defects are caused by a disturbance during enamel formation. Multiple factors such as toxic agents, infections, trauma, or irradiation have been elucidated. The resulting defects can be quantitative (with a lack of enamel volume) or qualitative (with a deficiency of enamel structure) or a combination of both. Here we concentrate on qualitative defects, which are characterized by increased enamel porosity.

10.1.2.1 Fluorosis

Today, enamel fluorosis is probably the most prevalent developmental defect of human teeth. It is caused by chronic intoxication with fluorides during the phase of enamel formation. It is believed that daily fluoride doses of 40 µg/kg bodyweight may increase the risk of fluorosis in children and doses above 100 µg/kg almost certainly cause dental fluorosis (Twetman and Ekstrand 2013).

Histologically, enamel fluorosis is characterized by an increased porosity in the enamel (Thylstrup and Fejerskov 1978). In mild forms only the superficial enamel is affected. In severe forms nearly the complete enamel is porous and the surface may show pits and breakdowns. Clinically mild fluorosis appears as cloudy whitish discolorations following the perikymata lines. This is the reason for their wavy appearance. In more severe forms, the white lines converge to white spots that affect from up to the complete cusp (Fig. 10.3). Very severe forms show complete opaque and chalky enamel with pitting and often brownish discolorations. Fluorosis can be distinguished from caries, since fluorosis usually affects multiple homologous teeth, which were formed during the same period. Therefore, fluorosis is often seen on homologous teeth.

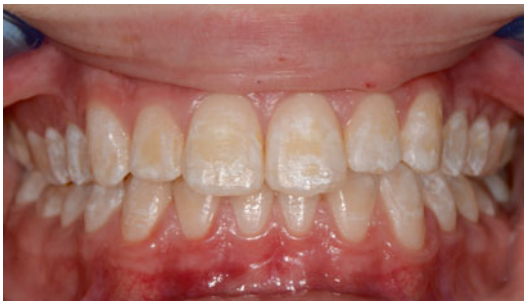

Fig. 10.3 Dental fluorosis: note the cloudy appearance

10.1.2.2 Posttraumatic Defects

Another quite prevalent class of developmental defects is caused by traumatic injuries of primary incisors that cause a disturbance of enamel formation in the permanent successors (Diab and elBadrawy 2000). These defects can comprise qualitative defects (white or yellow-brown discolorations), quantitative defects (enamel hypoplasia), or combinations of both (Diab and elBadrawy 2000). Posttraumatic defects are mostly unilateral and just affect one or two teeth.

10.1.2.3 Molar Incisor Hypomineralization (MIH)

MIH is a developmental defect that affects the first permanent molars and incisors. In some cases, also second permanent molars and primary teeth show alterations. MIH lesions may appear as distinct and clearly demarcated whitish to yellowish discolorations (Willmott et al. 2008; Takahashi et al. 2009). As in other "white spots," the whitish opaque discoloration is caused by an increased porosity of the enamel (Fig. 10.4). In contrast to fluorosis, the opacity is always distinct as the porosity affects the complete enamel depth from the surface up the enamel dentin junction. Yellowish, brownish discolorations are caused by organic components, which fill the porosities. In severe forms the increased porosity may also lead to enamel breakdown (Weerheijm 2003).

The etiology of MIH is still unclear. It is commonly believed that the disease is multifactorial and that causing factors must be present during mineralization of the first molars and incisors, which means up to the first 4 years of life. As possible etiological factors, problems during pregnancy, infectious diseases, or intoxication with antibiotics or dioxins have been discussed (Takahashi et al. 2009).

10.2 Resin Infiltration

The technique of resin infiltration was originally developed to arrest noncavitated caries lesions. The mineral loss of early caries lesions results in the formation and growth of porosities in the enamel matrix. These pores act as diffusion pathways for acids into the enamel and dissolved minerals out of the enamel. Caries infiltration aims to penetrate these pores with low viscosity light curing resins – so called infiltrants – that infiltrate the porous enamel driven by

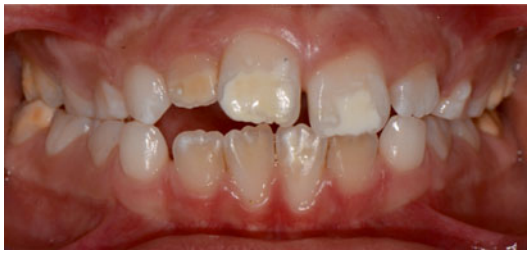

Fig. 10.4 Molar incisor hypomineralization (MIH). The disease is characterized by whitish opaque to yellowish (cheesy), distinct and clearly demarcated discolorations on incisors and first permanent molars (Image by M. Petrou)

capillary forces (Paris et al. 2010). When the resin is light cured, it blocks the diffusion pathways and protects the remaining apatite crystals (Meyer-Lueckel and Paris 2008).

One positive side effect of resin infiltration is an immediate color change of the whitish lesion once it is infiltrated. The infiltrant resin has a refractive index of 1.52, which is close to that of enamel (1.62). Therefore, light scattering between the enamel pores and the surrounding enamel is significantly reduced when the pores are filled with resin instead of water or air (Fig. 10.5). Under best conditions, the resin-infiltrated lesions are optically masked and "disappear" (Paris and Meyer-Lueckel 2009; Eckstein et al. 2015).

To achieve resin infiltration of porous enamel, the lesions need to be conditioned. As the pseudo-intact surface layer has a much lower porosity compared with the underlying lesion body, it hampers penetration of the infiltrant resin and thus must be removed. This can be achieved by etching the lesion with 15 % hydrochloric acid (HCl) gel for 2 min.

In the next step, the lesion must be properly desiccated. As the penetration of the infiltrant resin infiltration is based on capillary action, any other liquid within the porous enamel network would inhibit any infiltration. Usually, the desiccation is achieved by a combination of air-blowing, an application of ethanol and another air-blowing subsequently. Finally, the infiltrant resin is applied for 3 min. Although infiltrants are chemically similar to many commercial bonding agents, it is strongly recommended not to misemploy adhesives or bonding agents for caries infiltration but to use the commercially available infiltrant kit. Infiltrant resins show a much higher penetrability compared with bonding agents and thus ensure a deeper infiltration. But

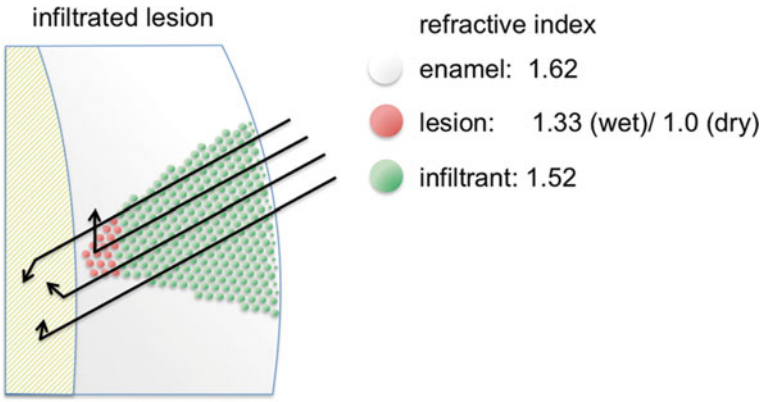

Fig. 10.5 Masking of white spot lesions by resin infiltration. As the infiltrant resin has a similar refractive index as enamel, light scattering within the white spot lesion is significantly reduced (compare with Fig. 10.1)

even with an infiltrant the relatively long application time of 3 min is recommended to ensure a preferably complete infiltration of the lesion body (Fig. 10.6).

Currently, there is only one commercial kit for caries infiltration on the market: Icon (DMG, Hamburg, Germany) is available for proximal or vestibular lesions. For infiltration of aesthetically relevant white spot lesions, the vestibular kit is recommended.

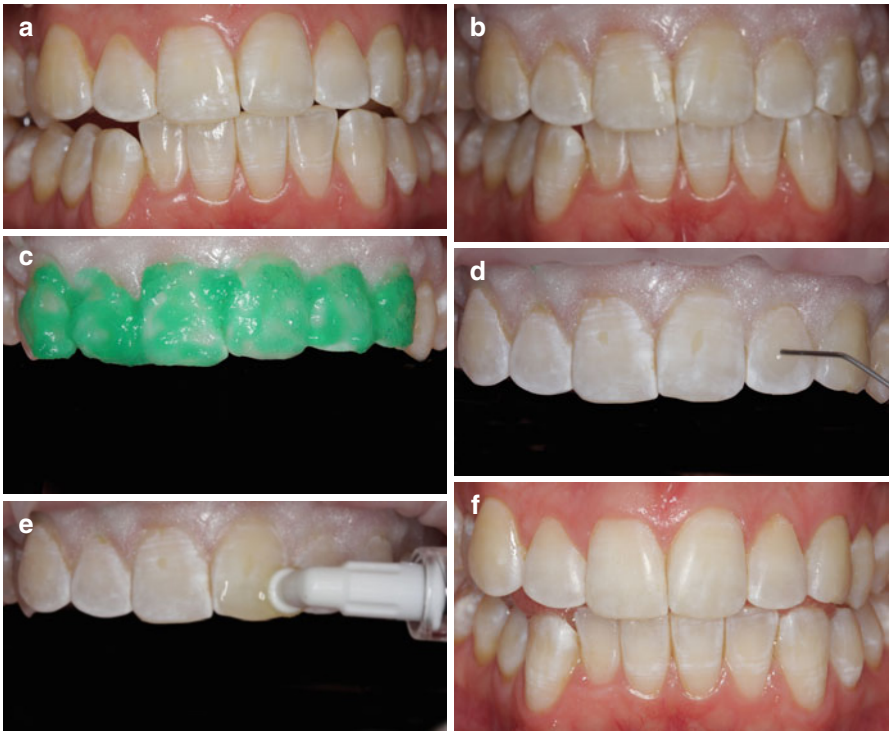

Fig. 10.6 Masking of fluorosis by resin infiltration. (**a**) Baseline. The patient complained about whitish discolorations (dental fluorosis) in the upper front. (**b**) After cleaning and application of light curing dam, the fluorosis becomes more distinct due to desiccation of the teeth. (**c**) The lesions surface layer is eroded by etching with 15 % HCl for 2 min. (**d**) After rinsing the etchant and drying, the teeth look frosty due to the etching pattern. Notice that the lesions are still visible as only the surface layer was eroded. Now ethanol is applied to check the completeness of surface layer erosion (for explanation, please see text). The ethanol is subsequently evaporated to desiccate the lesion as much as possible. (**e**) During application of the infiltrant resin, an immediate color change can be observed. After 3 min, resin surplus is removed and the material is light cured. A second infiltration step compensates possible polymerization shrinkage. (**f**) After polishing the lesions, the lesions in the upper front are nearly completely masked. After some days the aesthetic result is usually even better due to rehydration of teeth

10.3 Case Selection

10.3.1 Infiltration of Buccal Caries

Many patients in need for an aesthetic rehabilitation of buccal white spots had a treatment with fixed orthodontic appliances. It should be ensured that the pathogenic factors causing the caries are under control before aesthetic rehabilitation is planned. Thus, orthodontic brackets should be removed and patients should be able to maintain a proper oral hygiene. To improve the aesthetics of teeth with white spot lesions, several treatment options should be considered:

- Enhancing remineralization
- Bleaching
- Resin Infiltration
- Micro-abrasion
- Composite restorations
- Veneers

In this list of treatment options, the invasiveness increases from remineralization methods toward the aesthetic rehabilitation using veneers. At the same time, the predictability of the aesthetic outcome improves. Thus, the choice of treatment is influenced by the predictable aesthetic outcome of a technique being expected by an individual patient as well as the loss of enamel that has to be removed in order to achieve the respective aesthetic result. In this context, it should be considered that especially for young patients in doubt, the less invasive methods should always be preferred and restorations should be postponed as long as possible (Qvist 2008).

Caries infiltration is "located" in the middle of the various treatment options and thus may be an interesting micro-invasive approach to achieve good aesthetic results with a minimum loss of enamel. To predict the aesthetic outcome of caries infiltration two factors should be considered:

Lesion depth: The deeper the lesions are, the harder it is to achieve a complete camouflage by resin infiltration. Thus, the more whitish a lesion appears even before drying, the harder it is to reach a complete masking.

Lesion activity: Active lesions are easier to infiltrate compared with inactive lesions. As mentioned above the pseudo-intact surface layer inhibits the penetration of the underlying lesion body and active caries lesions usually exhibit shallower and lower mineralized surface layers compared with inactive lesions. Therefore, for active lesions less surface erosion is required and often better aesthetic results are observed in most cases. For this reason, it might be considered to treat buccal lesions shortly after removal of orthodontic brackets.

10.3.2 Infiltration of Developmental Defects

Due to their histological similarity to caries lesions also developmental hypomineralized defects can be resin infiltrated. It could be shown that good aesthetic results can be expected for cases of mild to moderate fluorosis (Auschill et al. 2015; Attal

et al. 2014; Gugnani et al. 2014). Similarly, mild posttraumatic lesions (Munoz et al. 2013) may also be camouflaged by resin infiltration. For MIH lesions, however, both in vitro results (Crombie et al. 2014) as well as clinical reports (Kim et al. 2011) showed rather unreliable aesthetic results. It seems that yellowish or brownish discolored MIH lesions show inferior aesthetic outcomes compared with whitish lesions. This might explain why MIH lesions are obviously harder to be infiltrated as these discolorations are associated with a high content of organic material. Thus, in these lesions infiltration is possibly hampered by the organic material that fills the enamel pores and thus blocks resin penetration. Nonetheless, an infiltration of the underlying lesion and a "superficial" composite restoration of ca. 100–200 µm in depth might be a very minimal invasive technique to be used in MIH teeth where infiltration alone did not work.

Aesthetic indications for caries infiltration:
- Active noncavitated vestibular caries lesions
- Mild fluorosis (TSIF 1–3)
- Mild developmental defects caused by trauma or infection

Limitations of caries infiltration:
- Inactive lesions
  - Here longer etching or micro-abrasion might be required.
- Enamel breakdown
  - A combination of infiltration and subsequent restoration with composites is possible.
- MIH lesions
  - Studies showed that MIH lesions are in 50 % of the cases aesthetically insufficiently camouflaged due to only partial infiltration.

Contraindications for caries infiltration:
- Dentin caries or cavities into dentin
  - The infiltrating resin is too hydrophobic to infiltrate the relatively wet dentin.

10.4 Treatment

Before treatment, teeth should be cleaned using prophylaxis paste. To isolate the gingiva and protect it from the applied chemicals, rubber dam is strongly recommended. As for conventional rubber dam usually ligatures are necessary, light curing rubber dam has been shown to be easier applicable and more convenient for the patient. During application of light curing dam, it should be ensured that on the one hand the plastic dam seals the gingiva tightly but on the other hand does not cover cervical lesions. For lesions that are located very close to the cervical gingival margin, retraction cords help to displace the gingiva before application of the liquid dam.

In the next step, the surface layer is eroded by application of 15 % hydrochloric acid gel (Icon etch) for 2 min. In most cases, it is advisable to cover all buccal

aspects with the etching gel to achieve a homogeneous appearance. Separation of the proximal contact points is usually not necessary. Before rinsing the etching gel, it is advisable to remove the bulk of the gel with a suctor in order to avoid flushing the strong acid into the oral cavity.

The etching step not only removes the outer 30–40 μm of the surface layer but also most brownish discolorations of caries lesions as most of these discolorations are located in the outer lesion surface. Thus, after etching most lesions show a chalky, brightly whitish appearance. In the next step, the lesion needs to be properly desiccated. This is achieved by air-blowing followed by application of ethanol (Icon etch), which is also evaporated by air-blowing subsequently.

This step should also be used to check sufficient erosion of the surface layer. Buccal lesions, especially more inactive ones may have thick and highly mineralized surface layers. In this case, 2 min etching with 15 % HCl may not be sufficient to erode the complete surface layer and infiltration of the lesion body might be impaired.

During the penetration of the ethanol into the air-dried lesion, a similar color change as during the later resin infiltration should be observed within the first 3–4 s after application. The color change from whitish chalky to nearly enamel colored during this "re-wetting test" is usually not as distinct as during application of the resin, but gives a good estimate of the later aesthetic outcome. If this color change during application of ethanol on the dried lesion fails to appear and the lesion remains whitish, it is quite likely that the surface layer is not completely eroded yet. In this case, a second application of the etching gel for another 2 min is recommended. In some cases – especially in "older" inactive lesions – even a third application or micro-abrasion of 100–150 μm is necessary. For that purpose the 15 % HCl etching gel can be mixed with some pumice and then rubbed on the lesion surface using rubber cup polishers. Care should be taken not to abrade too much enamel. In contrast to classical micro-abrasion, not the complete lesion but only the surface layer needs to be removed.

To check the completeness of erosion, after each etching step the "re-wetting test" should be repeated. If the result is positive, the lesion can be finally desiccated. The drying of the lesion is an absolutely essential step, as resin infiltration is based on capillary forces that only work when the pores within the lesion body are filled with air but not water.

Subsequently, teeth are properly dried and the infiltrant (Icon infiltrant) is applied. Again an immediate color change is usually observed. The resin is now allowed to infiltrate the lesion body for approximately 3 min. Before light curing all excess resin is removed from the lesion surface using foam pellets or cotton rolls so that only the resin inside the lesion remains and is light-hardened. Due to the polymerization shrinkage, the resin now should be applied a second time for 1 min, the excess removed, and the material hardened.

To polish the surface, which is still roughened from the etching step and to remove the oxygen-inhibited resin layer, the lesions now should be polished. This step is quite important as the acid-roughened surface that contains a thin oxygen-inhibited resin layer otherwise attracts food stains, which may lead to swift discoloration of the treated surfaces.

The whole procedure for one jaw usually takes about 20–30 min. The immediate masking of the lesions is quite satisfactory for the patient in most cases. As during

treatment the teeth are desiccated, the final aesthetic result after some days is usually even better compared to directly after treatment.

Treatment procedure for caries infiltration to camouflage white spot lesions:

1. Cleaning of teeth
2. Isolation by rubber dam or liquid dam
3. Etching with 15 % HCl for 2 min
4. Rinsing and drying
5. Re-wetting with ethanol to check completeness of surface layer erosion: if negative repeat steps 3–5; if positive move on with step 6
6. Desiccation of ethanol
7. Application of infiltrant for 3 min
8. Removing of resin surplus
9. Light curing
10. Polishing

10.5 Conclusion and Outlook

Although a complete masking cannot be achieved by resin infiltration in every case, a significant improvement of aesthetics is usually observed in the majority of lesions. While shallower caries lesions can often be completely masked, in deep caries lesions at least an improvement is observed. Currently, there are only few long-term data available on the color stability of resin-infiltrated lesions. However, it seems that infiltrated lesions show similar staining as sound enamel (Eckstein et al. 2015; Paris et al. 2013). Fluorotic white spots and those caused by traumatic injury can be masked quite well. For MIH lesions, aesthetic results might not be as good.

In conclusion, resin infiltration is a micro-invasive approach that adds to the existing armamentarium of treatment options to improve the aesthetics of buccal white spot lesions. Due to its relatively easy application, low costs, and satisfying results it may be considered as the first in line treatment option in many cases of vestibular white spot lesions.

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Part IV

Clinical Application of Combined Techniques

Internal Whitening and At-Home Tray Whitening

11

Andressa Ballarin, Guilherme C. Lopes, and Jorge Perdigão

Abstract

Tooth whitening outcomes may be optimized when different bleaching techniques are combined. For patients with generalized physiological tooth discoloration and darkened endodontically treated teeth, internal tooth whitening of these nonvital teeth followed by at-home whitening of both arches is the recommended treatment. Dentists must inform their patients about the expected outcomes of each procedure and provide an evidence-based choice.

11.1 Introduction

As discussed in Chap. 1, there are currently several tooth bleaching techniques available to clinicians. One of the advantages of at-home bleaching is its efficacy, which is readily noticed and viewed very favorably by patients (Swift et al. 1999).

The dental professional often treats patients who have generalized discoloration of vital teeth as a result of physiological aging or extrinsic factors and one (or several) darkened endodontically treated tooth. However, discolored nonvital teeth do not usually respond to external tray whitening as well as vital teeth do, with the exception of

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recent discolorations, as seen in Chap. 6, Fig. 6.7. This chapter illustrates a combined technique in which a darkened nonvital tooth is first treated with internal whitening to reach a tooth color similar to that of the remaining vital teeth, followed by at-home whitening of both arches with 10 % carbamide peroxide in a custom-fitted tray.

11.2 Clinical Technique

This clinical case involved a 31-year-old patient whose chief complaints were the discoloration of a maxillary right canine and the "yellow look" of the other teeth. The medical history was not contributory to dental treatment. Upon clinical and radiographic exam, the patient was informed that the discoloration on tooth #6 (FDI 1.3) was related to the respective root canal treatment. Patient was also informed that internal whitening might result in color relapse after some time. The small risk of external root resorption associated with this procedure, as described in Chap. 8, was also mentioned to the patient. The treatment plan included intra-coronal whitening of tooth #6 (FDI 1.3) followed by restoration with an adhesively luted glass fiber post and a resin-based composite direct restoration. The next step in the treatment plan would be at-home whitening of both arches with 10 % carbamide peroxide in a custom-fitted tray. Tooth #6 (FDI 1.3) would be evaluated 6 months after the restoration to decide if a full-coverage ceramic restoration might be needed. The patient accepted this treatment plan.

Figures 11.1, 11.2, 11.3, 11.4, 11.5, 11.6, 11.7, 11.8, 11.9, 11.10, 11.11, 11.12, 11.13, and 11.14 illustrate the treatment sequence.

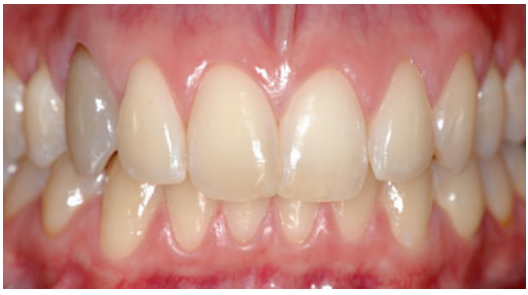

Fig. 11.1 Preoperative view of patient's smile showing severe discoloration of the maxillary right canine (tooth #6, FDI 1.3) and generalized discoloration of the remaining dentition (A3, Vita Classical shade guide)

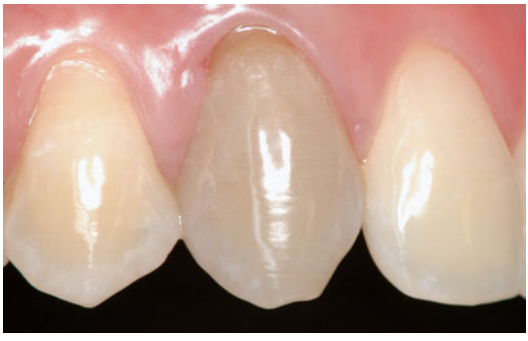

Fig. 11.2 Close-up magnification of tooth #6 (FDI 1.3)

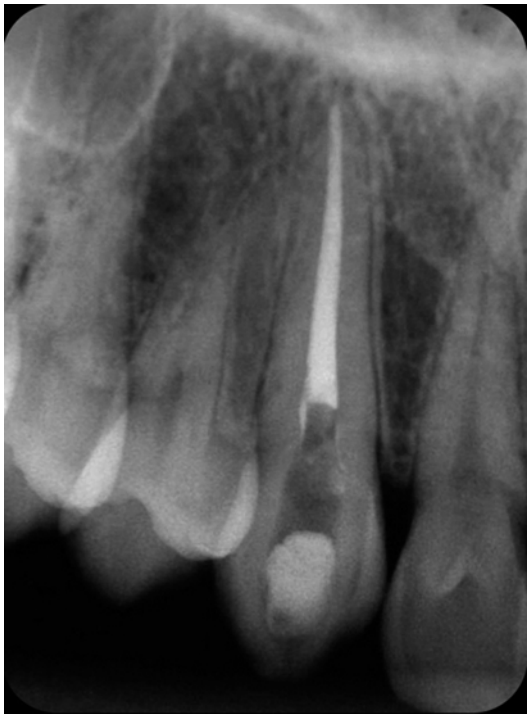

Fig. 11.3 Periapical radiograph of root-canal-treated tooth #6 (FDI 1.3)

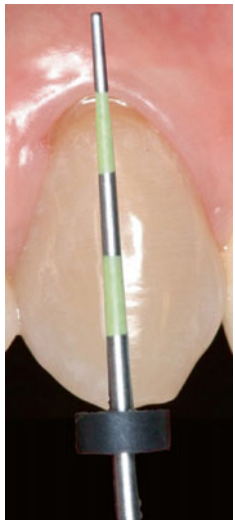

Fig. 11.4 A periodontal probe is used to determine the level of the epithelial attachment from the incisal edge of the tooth. This will serve as guide for placement of the root canal barrier (Chap. 8)

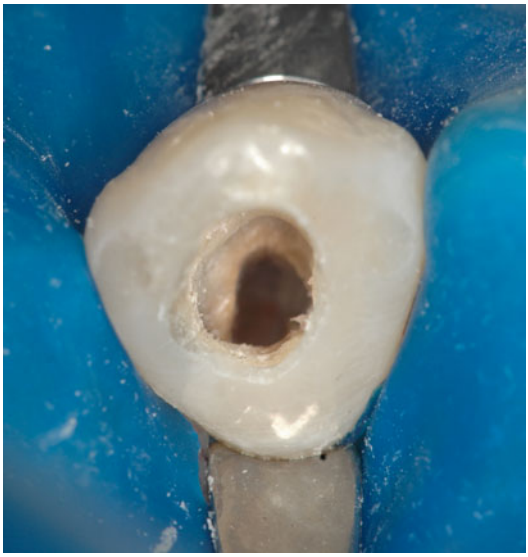

Fig. 11.5 After isolation with rubber dam and a #212 clamp, the restorative material was removed from endodontic access preparation. The root canal obturation material was removed from the cervical area

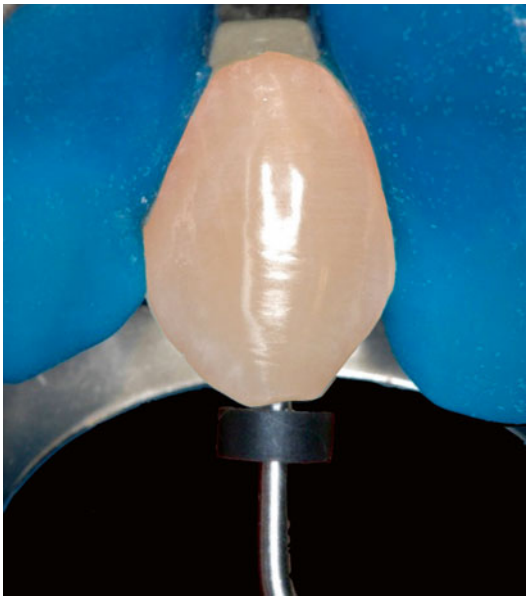

Fig. 11.6 The same periodontal probe is used to check if the root canal obturation material is removed to a level 2 mm apical to the CEJ, calibrated to the first measurement obtained with the periodontal probe as in Fig. 11.4

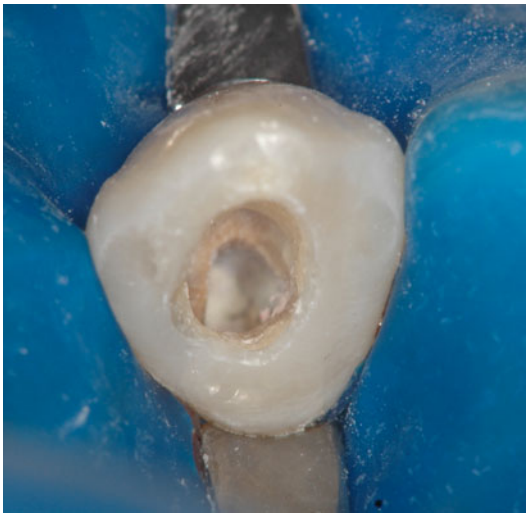

Fig. 11.7 A 2-mm thick layer of a resin-modified glass ionomer (RMGIC) material is applied as a cervical barrier between the pulp chamber and the endodontic filling material to prevent leakage of bleaching agent apically into the root canal and to the periodontal tissues. Any excess of RMGIC must be removed after light curing

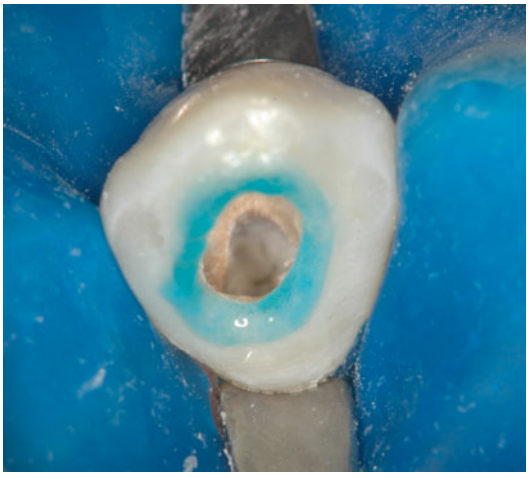

Fig. 11.8 Enamel was acid-etched for 15 s with 35% phosphoric acid to provide micromechanical retention of the resin-based temporary filing material

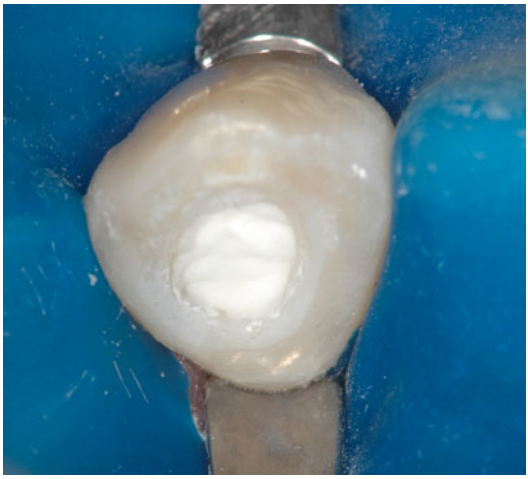

Fig. 11.9 A thick paste of sodium perborate and distilled water was inserted into the pulp chamber space. The residual liquid was removed with dry cotton pellets by gently compressing the paste. A dry cotton pellet was inserted, leaving a clearance of 1.5 mm for the provisional restoration of the access preparation

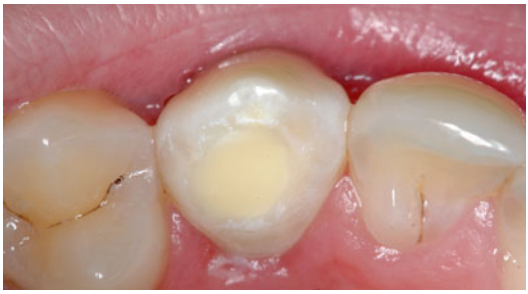

Fig. 11.10 After carefully air-drying the cavo-surface margin, a temporary low-viscosity resin-based material without an intermediate adhesive is inserted and light-cured

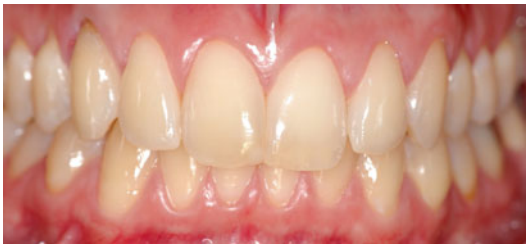

Fig. 11.11 Postoperative view after 3 weekly sessions of intra-coronal whitening with sodium perborate and distilled water

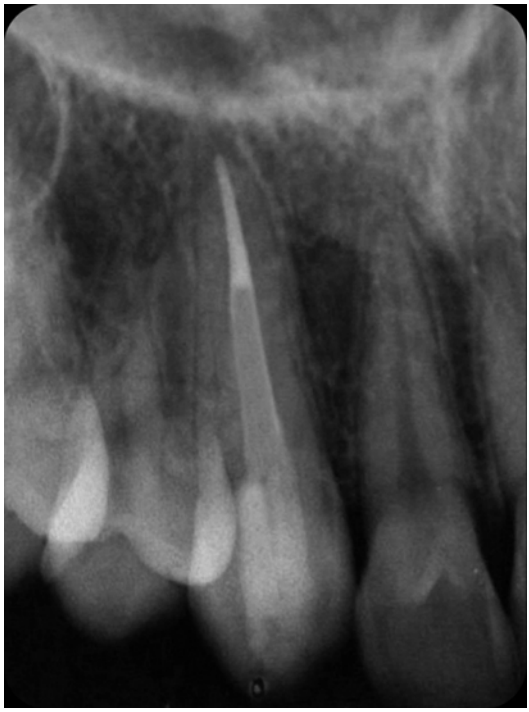

Fig. 11.12 Postrestorative periapical radiograph depicting a glass fiber post bonded to root dentin and a coronal resin-based composite restoration

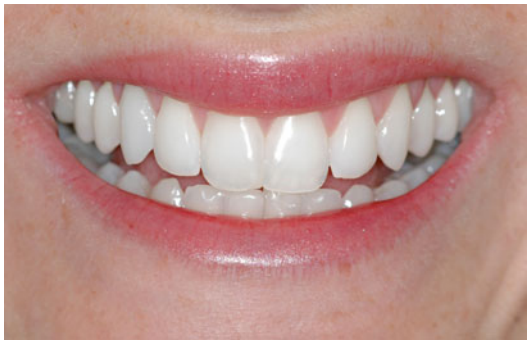

Fig. 11.13 Once the intra-coronal whitening treatment was completed and tooth #6 (FDI 1.3) restored, patient was prescribed at-home whitening with 10% carbamide peroxide with potassium nitrate and sodium fluoride (Whiteness Perfect 10%, FGM) in a custom-fitted tray for 2 weeks

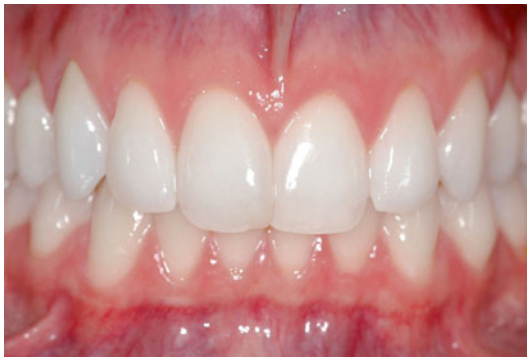

Fig. 11.14 Postoperative view of patient's smile after at-home whitening of both arches with 10% carbamide peroxide gel

Reference

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At-Home Tray Whitening and Enamel Microabrasion

12

Jorge Perdigão, Jennifer M. Homer[#], and Carmen Real

Abstract

Enamel microabrasion has been used to remove hypomineralized areas caused by enamel fluorosis or those caused by an idiopathic condition. The combination of at-home whitening with 10 % carbamide peroxide in a custom-fitted tray and enamel microabrasion is discussed and illustrated in this chapter as a successful treatment combination in cases of brown fluorosis stains that are not completely eliminated with at-home whitening.

12.1 Introduction

In 1986, Croll and Cavanaugh described the "enamel microabrasion" technique (Croll and Cavanaugh 1986a, b), which combines chemical erosion with mechanical abrasion (Chap. 9). The microabrasion paste, which contains hydrochloric acid (HCl) and silicon carbide, is applied by rubbing onto the enamel surface, removing

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a microscopic layer of enamel (Donly et al. 1992). Patients usually report to be satisfied with the aesthetic results upon completion of the enamel microabrasion treatment (Loguercio et al. 2007).

The combination of in-office whitening with enamel microabrasion has been reported in the literature. Bertassoni et al. (2008) used in-office whitening followed by enamel microabrasion to camouflage white spot areas caused by fluorosis. The effect of the in-office whitening component of the treatment was not clear in this article, as no preoperative image was published. Celik et al. (2013a) reported that combining enamel microabrasion and in-office bleaching was more effective in the aesthetic management of fluorosed teeth than enamel microabrasion without whitening. However, authors did not include a control group with in-office bleaching without microabrasion. Whitening might have lightened the fluorotic brown stains without enamel microabrasion. As described in Chap. 4, bleaching with 35 % hydrogen peroxide immediately after the enamel microabrasion procedure increases the diffusion of hydrogen peroxide through enamel by 20 % (Briso et al. 2014), which makes the use of in-office whitening contraindicated as a first step in the management of enamel fluorosis cases.

Other authors have combined enamel microabrasion with at-home whitening. Ardu et al. (2007) treated enamel fluorosis stains using microabrasion to eliminate the superficial enamel layer, followed by at-home bleaching and direct resin-based composite restorations. These authors started with enamel microabrasion and then prescribed a home bleaching technique "to better harmonize tooth color and produce whiter teeth." Celik et al. (2013b) also advocated the use of enamel microabrasion as a first option in the management of fluorosis stains. Nonetheless, they concluded that enamel microabrasion was not very effective in removing fluorosis brown stains. The authors attributed this difficulty in removing the brown stains to the discoloration of the hypocalcified subsurface areas from external sources, probably associated with the penetration of staining agents.

Currently, there are two commercial enamel microabrasion materials available (e.g., PREMA, Premier Dental Products; Opalustre, Ultradent Products Inc.). These products contain a low concentration of hydrochloric acid and silicon carbide abrasive powder in a silica gel. Sundfeld and colleagues (2007) reported that 1–10 applications of 60 s on each tooth of Opalustre (6.6 % HCl) removed 25–200 μm of enamel. Given that enamel microabrasion technique is less conservative than at-home whitening in a custom-fitted tray, *our recommendation is to start with the least invasive technique (whitening) and, in case it does not result in an acceptable aesthetic result, perform enamel microabrasion after whitening*.

12.2 Clinical Case

The chief complaint of this 29-old-female patient was related with the brown stains of her maxillary central incisors (Fig. 12.1a, b). The medical history and the clinical exam led to the diagnosis of enamel fluorosis. After radiographic exam, the treatment plan presented to the patient consisted of long-term at-home whitening with

10 % carbamide peroxide with potassium nitrate and sodium fluoride in a custom-fitted tray overnight with monthly recalls. The patient was informed that whitening is not always successful in removing fluorosis-related stains and that enamel microabrasion and/or direct resin-based composite restorations might be indicated after the conclusion of the whitening treatment. Side effects of at-home whitening were also explained in detail. We also informed the patient that enamel microabrasion removes a microscopic layer of enamel.

The patient agreed with the treatment plan. After preliminary impressions and fabrication of custom-fitted soft bleaching trays, we prescribed Opalescence 10%PF (Ultradent Products Inc.) and demonstrated how to dispense the bleaching gel into the tray. Due to scheduling conflicts, the patient was only able to return after 7 weeks (Fig. 12.2a, b). The brown stains were significantly lighter at this first recall. The patient had no permanent side effects such as tooth sensitivity or soreness of the gingival tissues, but she did report mild tooth sensitivity during the first few days of the treatment. At this time, we suggested one extra month of at-home whitening following the same regimen.

The second recall took place 4 weeks later (Fig. 12.3a, b). There had been a slight improvement in the color of the maxillary incisors. At this time, in agreement with the patient, we decided to perform the enamel microabrasion technique with Opalustre (Ultradent Products Inc.) on the four maxillary incisors (Fig. 12.4a, b).

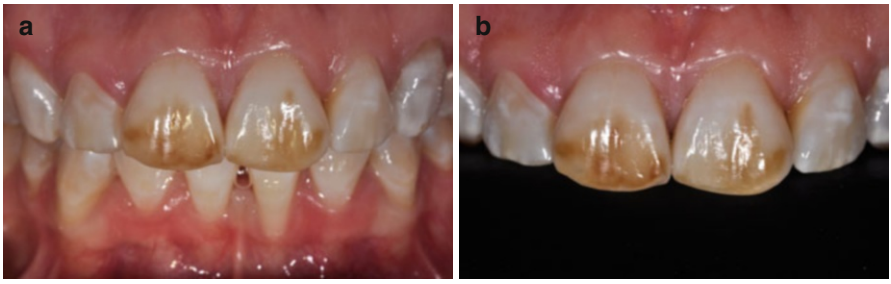

Fig. 12.1 (**a**, **b**) Pre-operative view of the discolored maxillary central incisors displaying brown stains caused by fluorosis

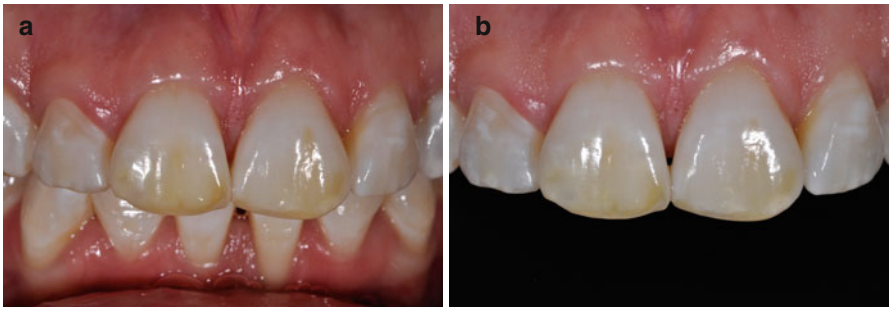

Fig. 12.2 (**a**, **b**) Clinical aspect after 7 weeks of overnight at-home whitening with 10 % carbamide peroxide with potassium nitrate and sodium fluoride in a custom-fitted tray

Six consecutive applications of 60 s each were carried out, with water rinsing between each application. Figure 12.5 depicts the intense dehydration immediately after removing the rubber dam.

The patient returned to clinic 2.5 months later (Fig. 12.6). Although the patient was extremely happy with the color improvement, we suggested a few more weeks of at-home whitening with 10 % carbamide peroxide gel. Figure 12.7a, b represents the final result after 3 weeks of at-home whitening. At this time, the patient decided that she did not want to pursue further treatment.

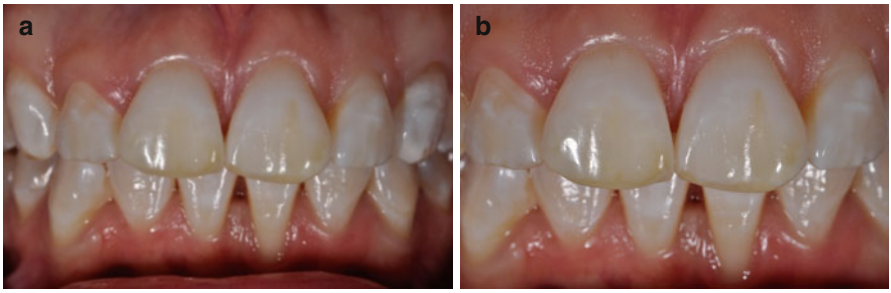

Fig. 12.3 (**a**, **b**) Patient retuned to clinic 4 weeks after the first recall

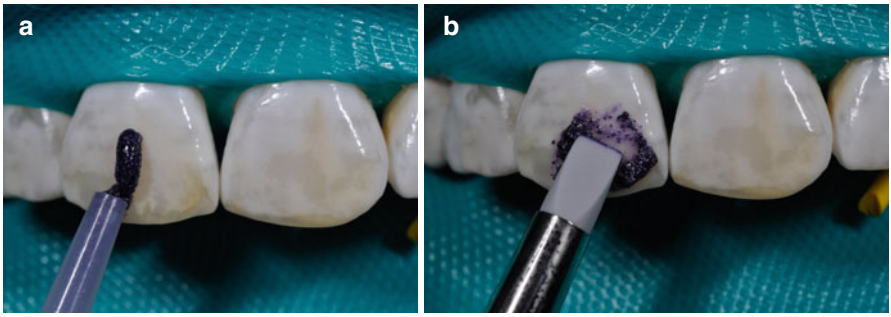

Fig. 12.4 (**a**, **b**) The four maxillary incisors were treated with an enamel microabrasion compound (Opalustre, Ultradent Products Inc.). Six consecutive applications of 60 s each were carried out with water rinsing between each application

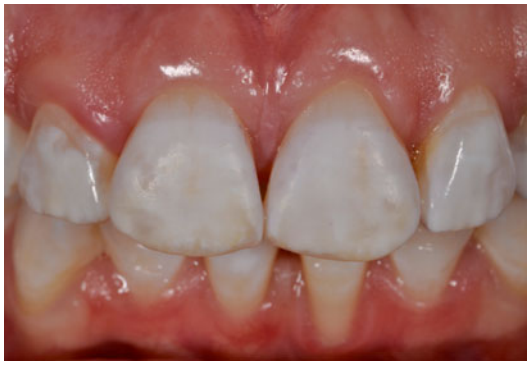

Fig. 12.5 Intense dehydration of the teeth immediately after removing the rubber dam

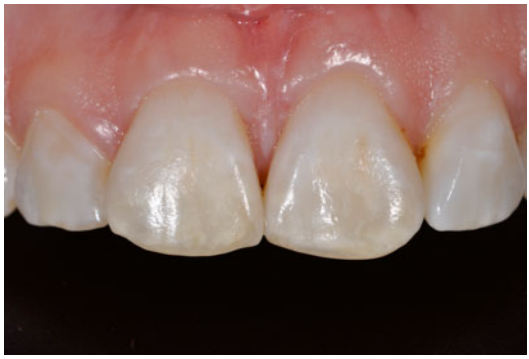

Fig. 12.6 The patient returned to clinic 2.5 months after the enamel microabrasion procedure. The patient was extremely satisfied with the color improvement of her anterior teeth. We suggested a few more weeks of at-home whitening with the same 10 % carbamide peroxide gel and the same bleaching tray, following the same regimen that she had been prescribed in the beginning of the treatment

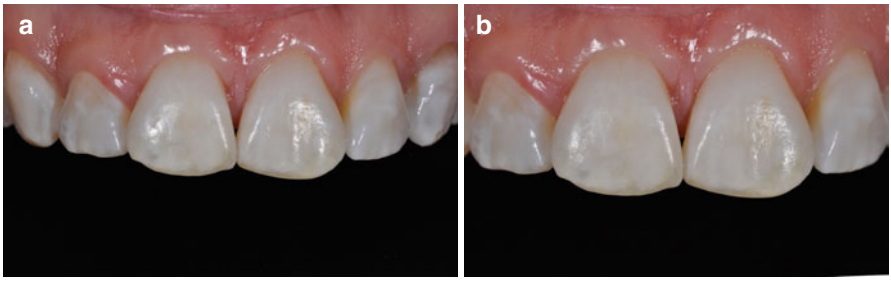

Fig. 12.7 (**a**, **b**) Clinical aspect after 3 extra weeks of at-home whitening with 10 % carbamide peroxide with potassium nitrate and sodium fluoride in a custom-fitted tray. At this time the patient decided that she did not want to pursue any further treatment, as she was extremely happy with the esthetic outcome

Acknowledgments We would like to express our gratitude to Dr. Patricia Nguyen, formerly a University of Minnesota dental student, and to Dr. David W. Klein for their help with the clinical case. Special thanks to all University of Minnesota Comprehensive Care Clinic Orange Group students.

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At-Home Tray Whitening and Resin Infiltration After Acid Etching with HCl

13

George Gomes, Filipa Oliveira, and Jorge Perdigão

Abstract

The treatment of discolored anterior teeth may involve combined techniques. This clinical sequence illustrates the successful bleaching of "yellow" teeth with at-home whitening using 10 % carbamide peroxide in a custom-fitted tray, followed by masking idiopathic white spots with enamel etching with HCl followed by resin infiltration.

13.1 Introduction

As discussed in Chap. 6, not all white or brown spots are caused by fluorosis. Some of these stains may be considered idiopathic (Cutress and Suckling 1990). Enamel "dysmineralization" has been used to refer to fluorosis-like discolorations (Croll 1990).

The concept of resin infiltration after enamel etching with hydrochloric acid (HCL) was introduced by Robinson et al. (1976) as a potential cariostatic treatment. Croll (1987) used a clear resin sealant on phosphoric-acid-etched enamel to saturate the surfaces with resin for smooth surface enamel defects. The research group of

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Paris and Meyer-Lueckel (2009) described the masking of white spots with resin infiltration using 15 % hydrochloric acid etching followed by a drying step with ethanol, and a very low viscosity light-cured resin (tetraethylene glycol dimethacrylate). Please refer to Chap. 10 for details.

13.2 Clinical Technique

The major concern that brought this 28-year-old male patient to seek dental care was the "white areas" in his maxillary anterior teeth. Additionally, the patient was not happy with the overall "yellow" color of his teeth. The patient's medical history did not shed any light into the cause of the white spots. The patient recollection was that these whitish areas were visible since his teenage years. The diagnosis was idiopathic discoloration.

The clinical exam revealed that the periodontal condition was excellent with no probing depths >3 mm. There were a few areas of gingival recession without root sensitivity. Periapical radiographs of the anterior teeth did not disclose any pathology.

The treatment plan presented to the patient included at-home whitening with 10 % carbamide peroxide gel (with potassium nitrate and sodium fluoride) in a custom-fitted tray overnight for 3 weeks, followed by enamel etching with 15 % hydrochloric acid (HCl) and resin infiltration. Figures 13.1, 13.2, 13.3, 13.4, 13.5, 13.6, 13.7, 13.8, 13.9, 13.10, 13.11, 13.12, 13.13, 13.14, 13.15, 13.16, 13.17, 13.18, 13.19, 13.20, 13.21, and 13.22 describe the sequential steps that resulted in a successful outcome.

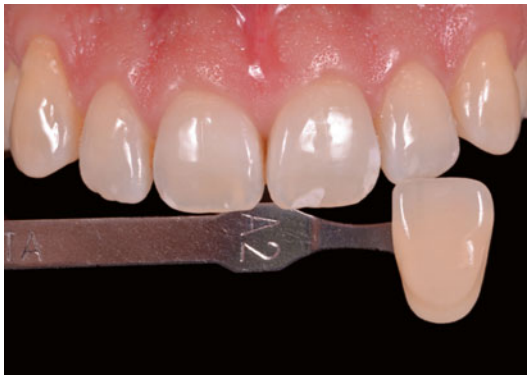

Fig. 13.1 Preoperative view of the patient's maxillary anterior teeth. An A2 Vita Classical A1-D4 shade tab (VITA Zahnfabrik H. Rauter GmbH & Co. KG) is shown as reference

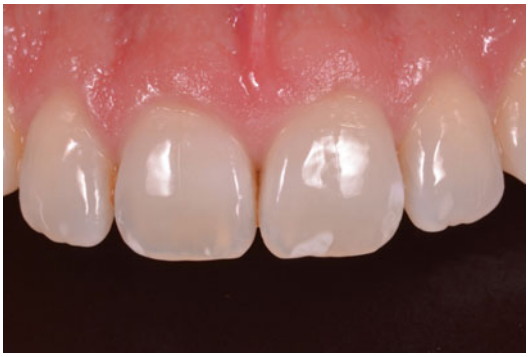

Fig. 13.2 Higher magnification view of maxillary incisors with white spots. Tooth #9 (FDI 2.1) had the widest white spot areas

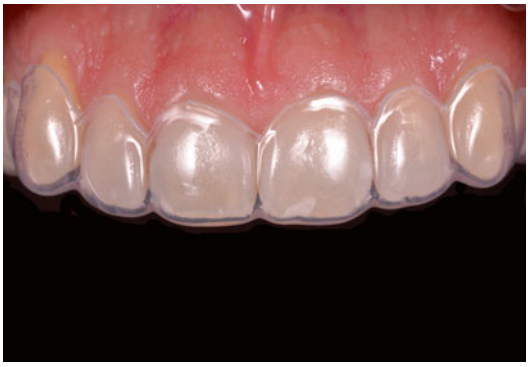

Fig. 13.3 Scalloped custom-fitted bleaching tray short of the gingival tissues to prevent tissue irritation

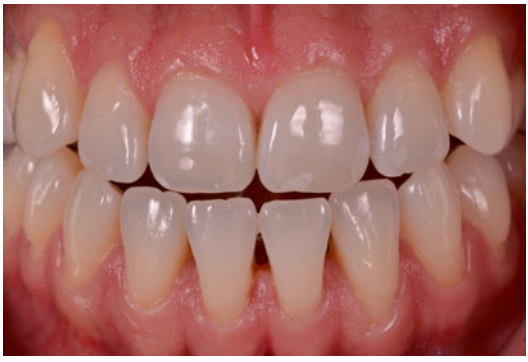

Fig. 13.4 Aspect of anterior teeth after 9 days of at-home whitening with 10 % carbamide peroxide gel with potassium nitrate and sodium fluoride (Whiteness Perfect 10 %, FGM) in a custom-fitted tray overnight

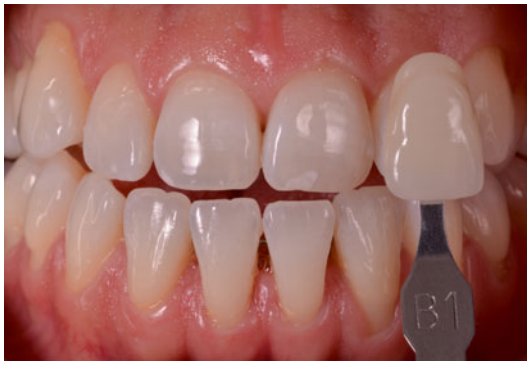

Fig. 13.5 Aspect of anterior teeth after 21 days of at-home whitening. The patient was extremely happy with the lighter color of his teeth

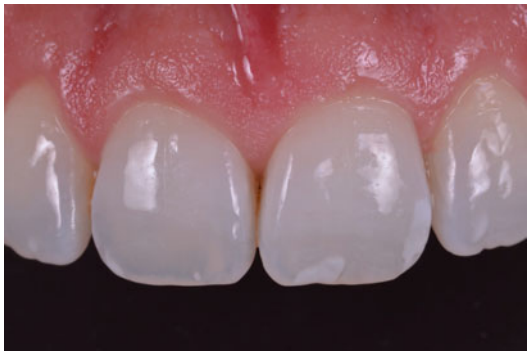

Fig. 13.6 The patient was scheduled for a resin infiltration procedure 2 weeks after finishing the at-home whitening treatment

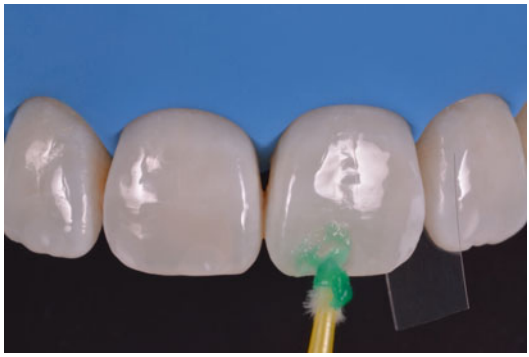

Fig. 13.7 Teeth were cleaned with a suspension of pumice and water and thoroughly washed with water. Area was isolated with rubber dam. A 15 % HCl gel (Icon-Etch, DMG) was applied to the white spot areas of tooth #9 (FDI 2.1). The respective manufacturer provides an application tip in the respective kit; however, we prefer a small round brush to ensure application accuracy

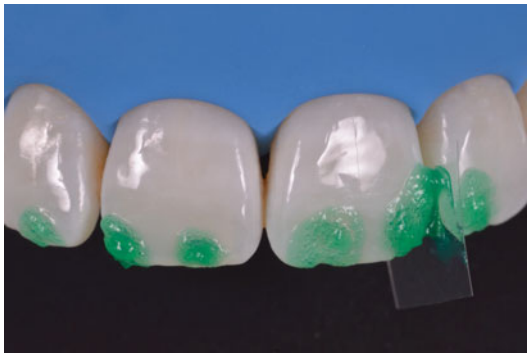

Fig. 13.8 The 15 % HCl gel was extended to the other white spot areas and left undisturbed for 2 min

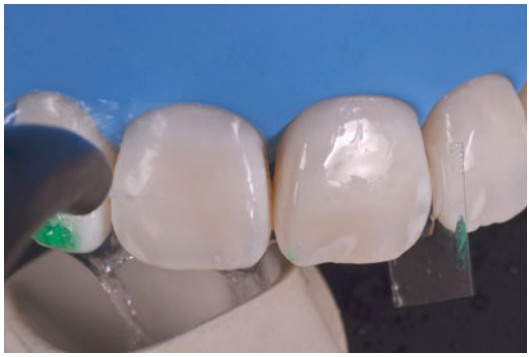

Fig. 13.9 The gel was thoroughly rinsed with water for 30 s with the high-speed suction tip positioned as close as possible to the area being washed. The white spots on tooth #9 (FDI 2.1) were etched a second time with 15 % HCl gel for 2 min, as recommended by the manufacturer

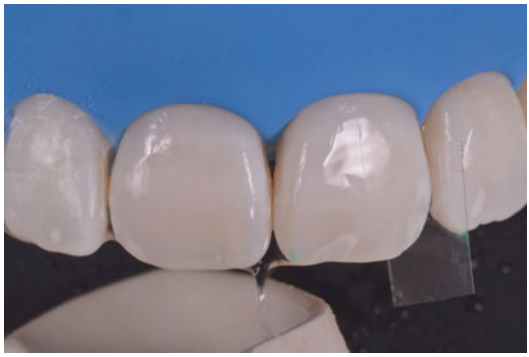

Fig. 13.10 The teeth were thoroughly rinsed with water and inspected to make sure that all residual gel had been removed

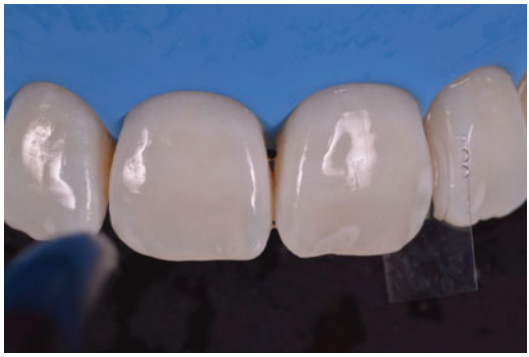

Fig. 13.11 The teeth were air-dried with water- and oil-free air for 15 s

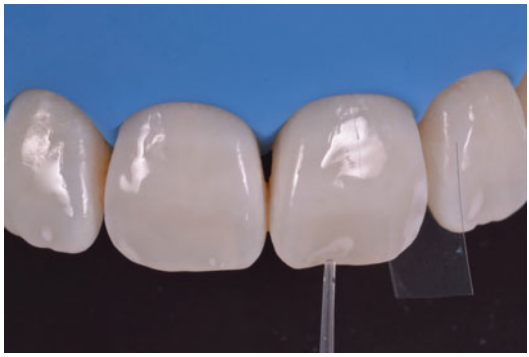

Fig. 13.12 A generous amount of Icon-Dry (DMG), which is composed of ethanol, was applied to the white spot areas and left undisturbed for 30 s. The teeth were air-dried with water- and oil-free air for 15 s

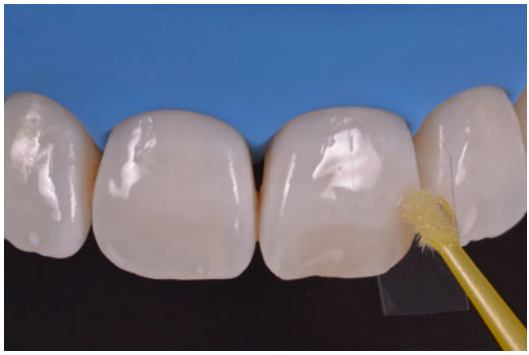

Fig. 13.13 An abundant amount of Icon-Infiltrant (DMG), which contains TEGDMA (triethyleneglycol dimethacrylate), initiators and stabilizers, was applied to the areas that had been etched with 15 % HCl and left undisturbed for 3 min

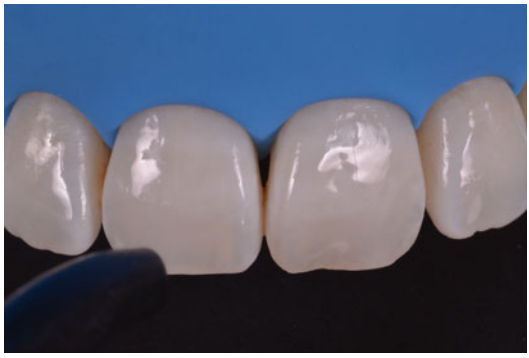

Fig. 13.14 Excess material was gently air-blown for 5 s to prevent pooling around the incisal edge

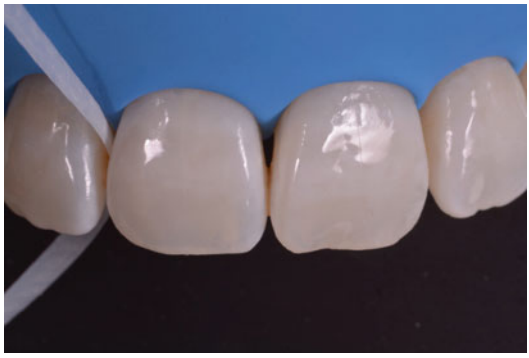

Fig. 13.15 Excess resin was removed with cotton pellets and dental floss

Fig. 13.16 The resin was light-cured for 40 s in each tooth

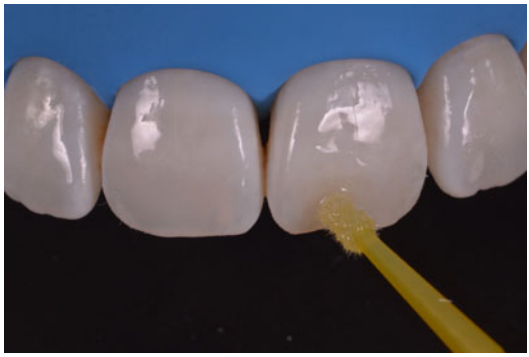

Fig. 13.17 Icon-Infiltrant (DMG) was reapplied and left undisturbed for 1 min. Excess was removed as described above, followed by light curing for 40 s in each tooth

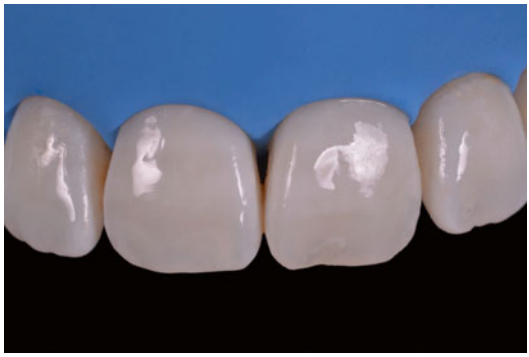

Fig. 13.18 Aspect of the anterior teeth immediately after light curing

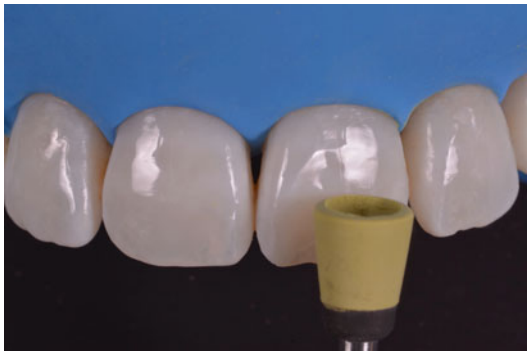

Fig. 13.19 The treated areas were polished with rubber points in slow speed

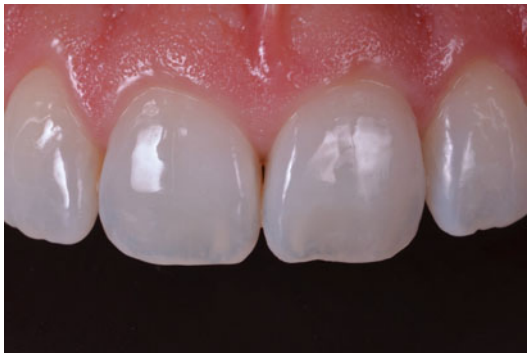

Fig. 13.20 Aspect of the treated teeth immediately after removing the rubber dam

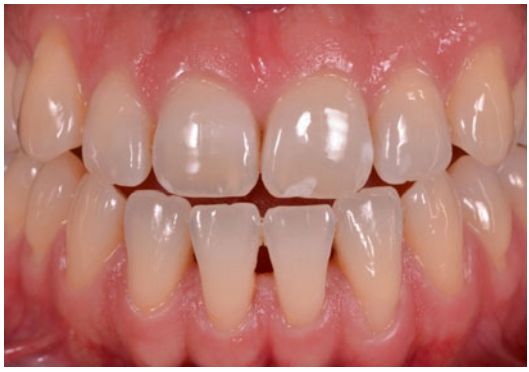

Fig. 13.21 Preoperative view

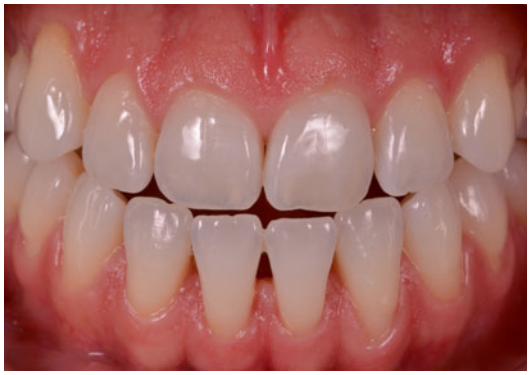

Fig. 13.22 Postoperative view

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At-Home Tray Whitening and Direct Resin-Based Composite Restorations

14

George Gomes, Filipa Oliveira, and Jorge Perdigão

Abstract

As discussed in Chap. 6, the esthetic appeal of anterior teeth may benefit from at-home whitening prior to esthetic rehabilitation with veneers or prior to recontouring clinical crowns with direct resin-based composite restorations. This clinical case illustrates the treatment details of a patient who wanted to change the color and the length of her anterior teeth.

14.1 Introduction

Carbamide peroxide in a custom-fitted tray is a very effective method to change the intrinsic color of teeth and to remove external stains from the tooth surface. When the treatment plan involves dental whitening followed by direct resin composite bonding procedures (recontouring anterior teeth, direct resin composite veneers, bonding orthodontic brackets, among others), the dental professional must take into consideration (as discussed in Chaps. 4 and 6) that enamel and dentin bond strengths remain low for the first two weeks post-bleaching. For enamel specifically, 10 % carbamide peroxide reduces the bond strengths of resin-based composite to etched

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enamel (Cvitko et al. 1991; Barghi and Godwin 1994; Ben-Amar et al. 1995; Spyrides et al. 2000), and increases enamel surface porosity (Ben-Amar et al. 1995). This reduction may be as high as 76 % of the bond strengths to unbleached enamel (Spyrides et al. 2000).

After a lapse of 2 weeks, the bond strengths return to the level of untreated substrates (Cavalli et al. 2001; Shinohara et al. 2005). Therefore, a minimum waiting period of 2 weeks is recommended after the patient completes the whitening treatment prior to performing any adhesive restorative procedure. In case the patient is unable to wait for 2 weeks, it has been shown that removal of surface enamel prior to bonding restores enamel bond strengths to normal level (Cvitko et al. 1991).

This chapter illustrates a combined technique in which at-home whitening of both arches with 10 % carbamide peroxide in a custom-fitted tray was first completed, followed by direct resin composite recontouring of the patient's anterior teeth.

14.2 Clinical Technique

A 34-year-old female patient called the dental office to set up an appointment primarily because she was concerned about the 'how short her upper front teeth were'. Additionally, the patient 'wanted to have whiter teeth'.

During her appointment, the patient did not mention any other complaints. She just wanted to improve the esthetics of her maxillary anterior teeth as well change the appearance of her smile (Fig. 14.1a, b). The clinical examination revealed no discomfort during function. The temporomandibular joint was asymptomatic. Upon clinical and radiographic examination, the treatment plan presented to the patient to improve the esthetics of her smile included at-home whitening with 10 % carbamide peroxide with potassium nitrate and sodium fluoride in a custom-fitted tray overnight for 2 weeks, followed by recontouring the incisal edges of the maxillary incisors with direct resin-based composite to increase the length of the clinical crowns. We also suggested the correction of the mesio-buccal line angle of tooth # 9 (FDI 2.1) and adjustment of the length of the maxillary canines with direct resin-based composite. The patient agreed with the proposed treatment plan. Figures 14.2, 14.3, 14.4, 14.5, 14.6, 14.7, 14.8, 14.9, 14.10, 14.11, 14.12, 14.13, 14.14, 14.15, 14.16, 14.17, 14.18, and 14.19 show the treatment sequence step by step.

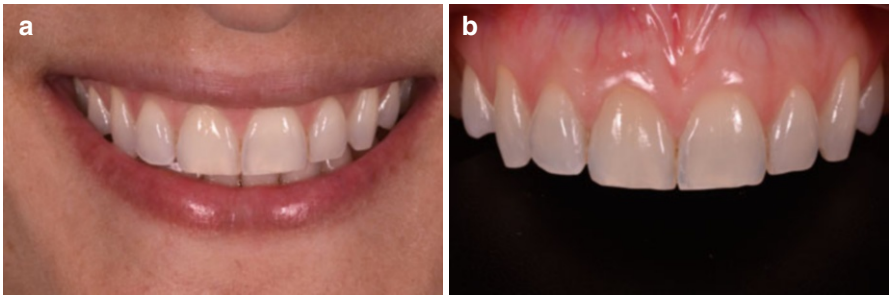

Fig. 14.1 (**a**) Preoperative view of the patient's smile. (**b**) Preoperative view of the patient's maxillary anterior teeth. Note the reduced length of the anterior teeth

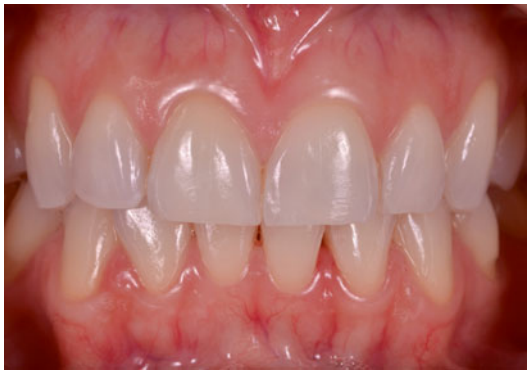

Fig. 14.2 Aspect of anterior teeth after 14 days of at-home whitening. The patient was satisfied with the lighter color of her teeth

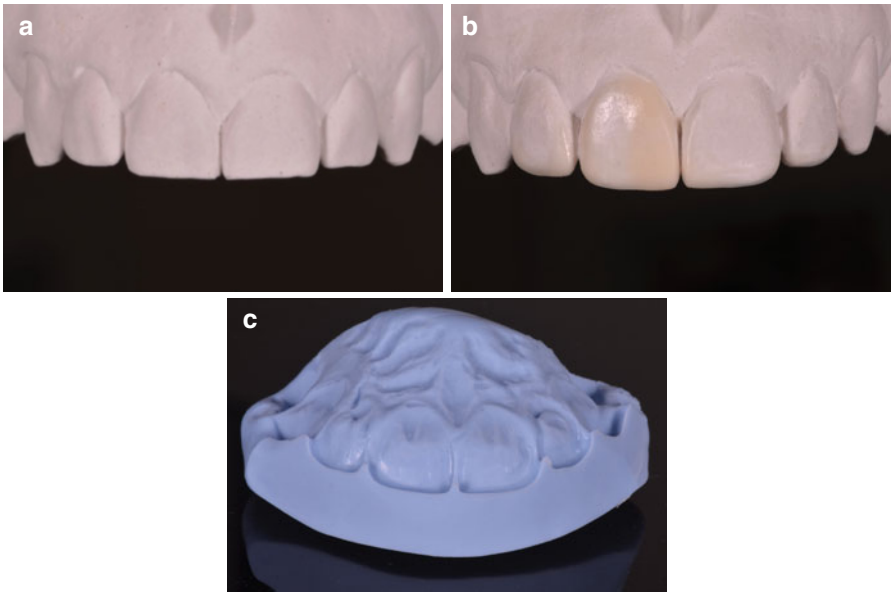

Fig. 14.3 (**a**) Stone model of maxillary anterior teeth. (**b**) The stone model was waxed-up to establish a harmonious length and shape of the anterior teeth. The patient was very happy with this blueprint of her teeth. (**c**) A matrix made of putty-consistency VPS (vinylpolysiloxane) impression material was prepared from the waxed-up model. This silicone index was used as guidance for the lingual contour and to establish the new incisal edge position

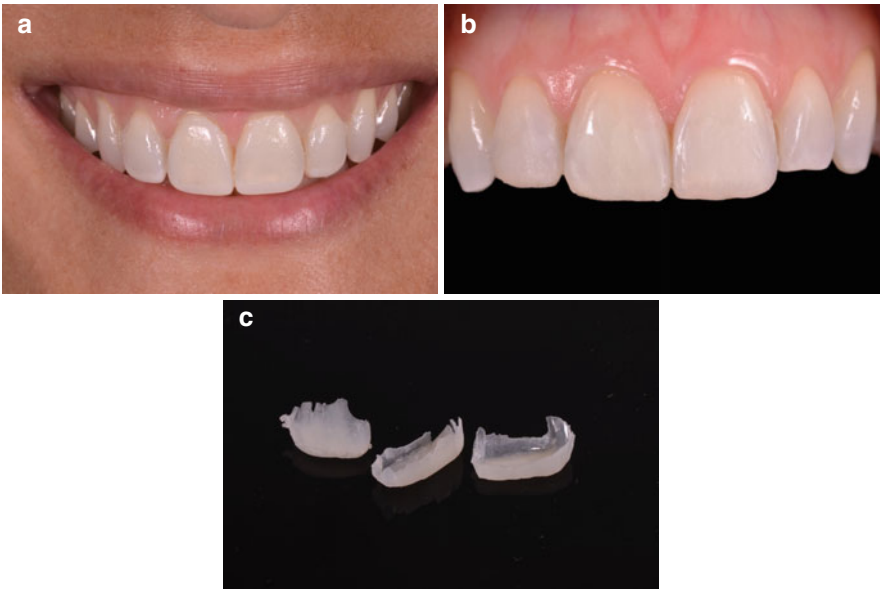

Fig. 14.4 (**a**) A mock-up was made with bis-acryl composite (Protemp 4 (also known as Protemp Plus), 3M ESPE) to allow the patient to foresee the esthetic outcome of the new restorations. (**b**) Diagnostic resin-based composite restorations were made prior to the adhesive procedure, to verify the stratification and thickness of each composite layer and the color. (**c**) Diagnostic restoration upon removal

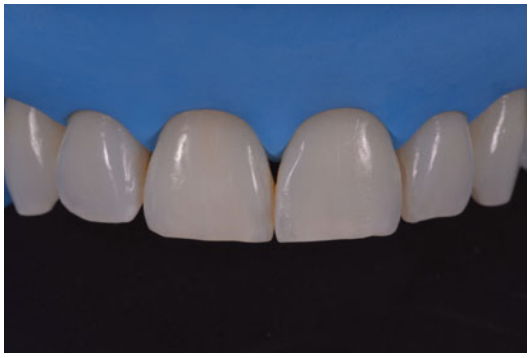

Fig. 14.5 Teeth were cleaned with a suspension of pumice and water and thoroughly washed with water. Area was isolated with rubber dam

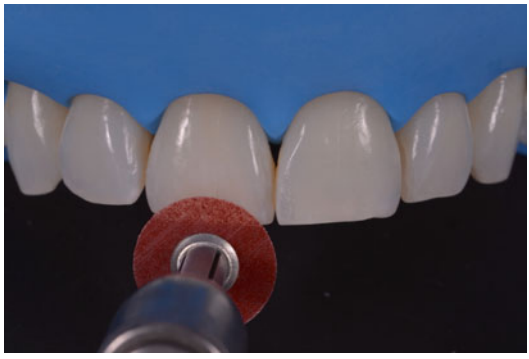

Fig. 14.6 Enamel was roughened with Sof-Lex disks (3M ESPE)

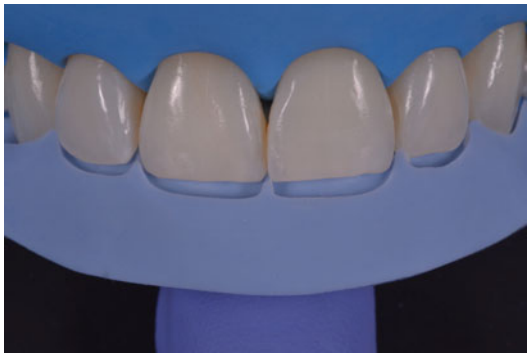

Fig. 14.7 The VPS matrix was tried-in

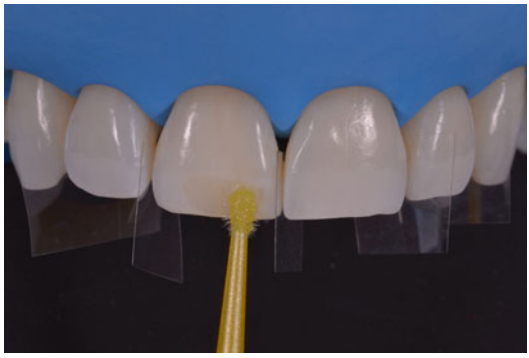

Fig. 14.8 The teeth were etched with 32% phosphoric acid (Scotchbond Universal Etchant, 3M ESPE) for 15 s. The etchant was thoroughly rinsed for 20 s. The two-step etch-and-rinse adhesive (Adper Single Bond Plus (also known as Adper Scotchbond 1 XT and Adper Single Bond 2), 3M ESPE) was vigorously applied for 15 s

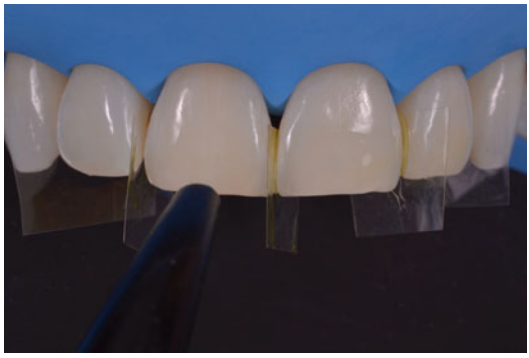

Fig. 14.9 The adhesive was gently air-dried to evaporate the solvent

Fig. 14.10 The adhesive was light-cured for 15 s

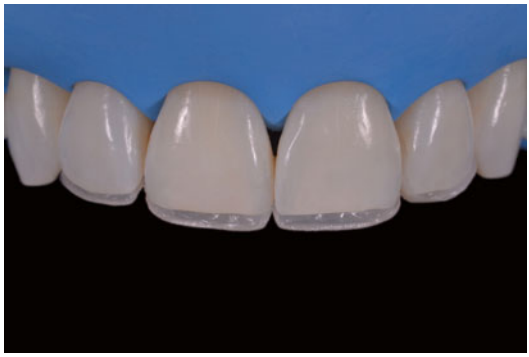

Fig. 14.11 A thin palatal (or lingual) layer of resin-based composite (Filtek Supreme Ultra (also known as Filtek Supreme XTE and Filtek Z350 XT), 3M ESPE) was applied with the VPS matrix using shade B1B for the central incisors and shade B1E for the lateral incisors

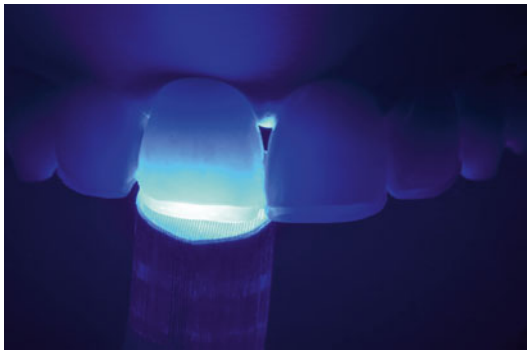

Fig. 14.12 This palatal enamel layer was light-cured from buccal and from lingual for 40 s each

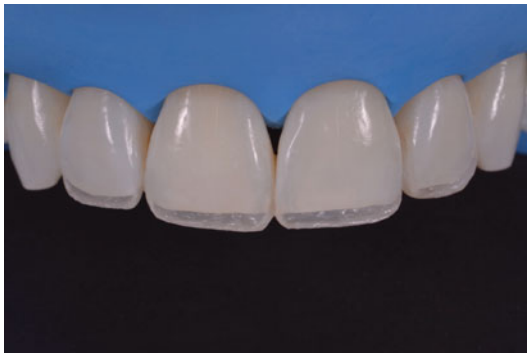

Fig. 14.13 The incisal opaque halo was replicated using shade A2D (central incisors) and shade B1B (lateral incisors), the same shades that had been used for the diagnostic restoration (Fig. 14.4b) as the dentin replacement shades

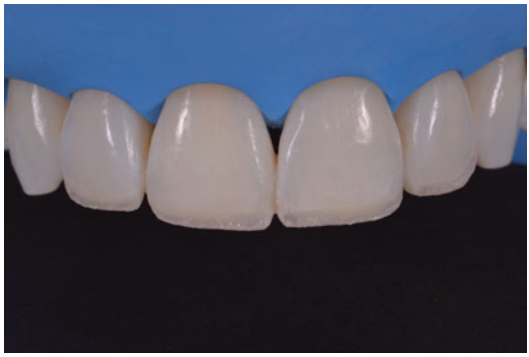

Fig. 14.14 After light curing the incisal opaque halo, the dentin layer was reproduced leaving space for the opalescent layer. The dentin layer was light-cured for 40 s

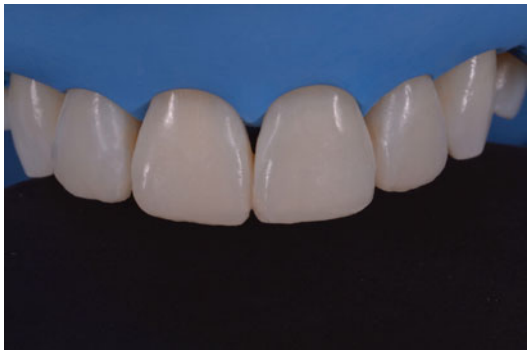

Fig. 14.15 An opalescent layer (shade AT or Amber Translucent) was applied between the A2D dentin replacement composite layer and the A2D incisal opaque halo. The enamel layer was reproduced using shade XWE for the central incisors and shade B1E for the lateral incisors. The restorations were light-cured for 40 s each from buccal and lingual aspects

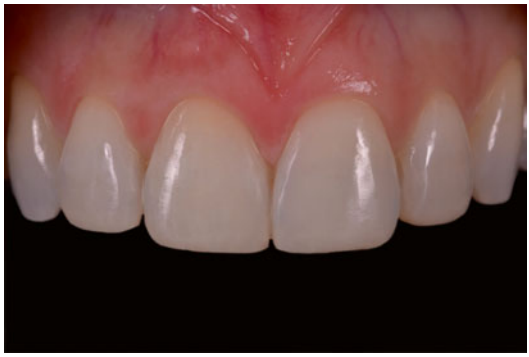

Fig. 14.16 Aspect of the restored teeth after polishing and removing the rubber dam

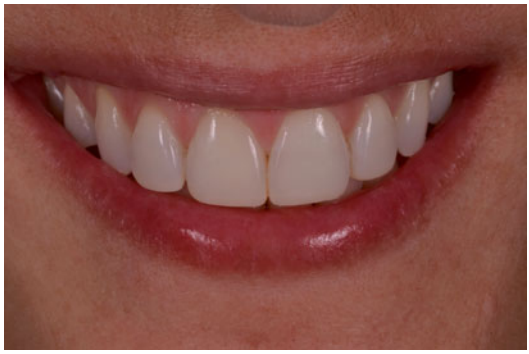

Fig. 14.17 Postoperative view of the patient's smile

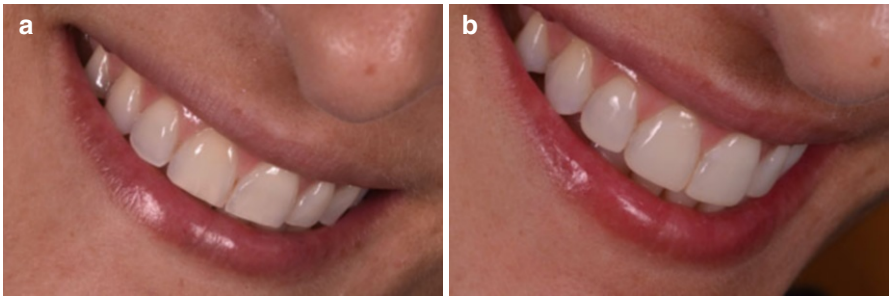

Fig. 14.18 (**a**) Preoperative right side view of the patient's smile. (**b**) Postoperative right side view of the patient's smile

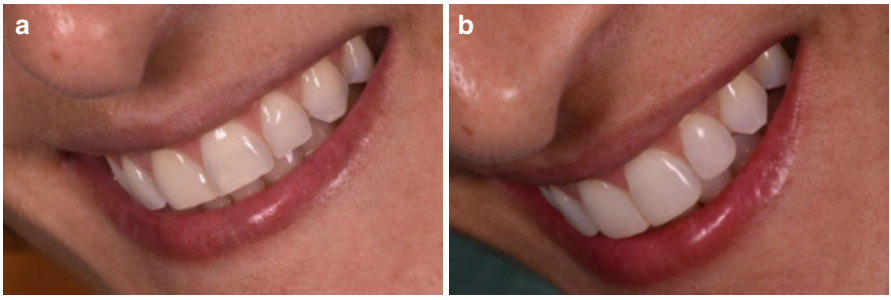

Fig. 14.19 (**a**) Preoperative left side view of the patient's smile. (**b**) Postoperative left side view of the patient's smile

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Restorative Options for Discolored Teeth

15

Edson Araújo and Jorge Perdigão

Abstract

Although dental professionals have an armamentarium of techniques for disguising or removing tooth discolorations, these techniques are not always successful. This chapter will focus on restorative options for discolored vital and nonvital teeth.

15.1 Dental Fluorosis Treated with Porcelain Crowns

As described in Chap. 6, excessive fluoride intake may result in dental fluorosis, characterized by opaque white areas or discolorations ranging from yellow to dark brown (Horowitz et al. 1984). In more severe cases, the enamel surface becomes pitted with porosities. One of the problems when restoring fluorosed teeth is the low bond strength obtained between resin-based composites and unground fluorotic enamel when using self-etch adhesives (Ermis et al. 2007). A two-step etch-and-rinse adhesive, on the other hand, results in statistically similar enamel bond strengths when applied to fluorotic enamel compared to normal enamel (Ermis et al. 2007).

The clinical case depicted in Fig. 15.1 is that of a 20-year-old female patient who decided to seek dental treatment because her self-esteem was extremely low as a result of the appearance of her teeth. This patient had lived in a rural area during

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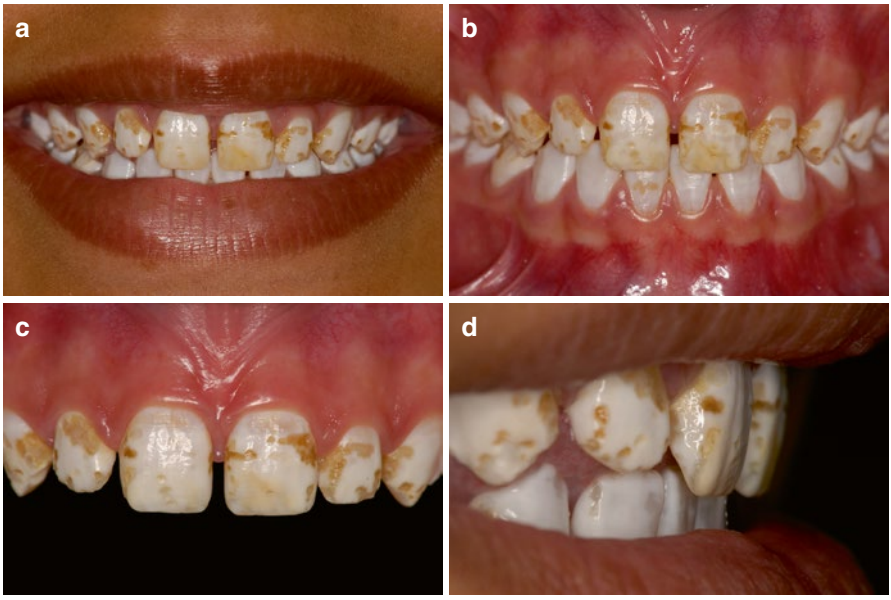

Fig. 15.1 (**a**) Nonretracted frontal view of patient's anterior teeth. (**b**) Retracted view showing enamel fluorosis in all teeth. (**c**) Close-up view of maxillary incisors. (**d**) Lateral close-up view of anterior teeth

childhood. According to the information given to her mother by the patient's local dentist, the drinking water contained excessive fluoride. Other children in the same community had discolored white or brown teeth, according to the patient's mother recollection. Consequently, the diagnosis for this clinical case was dental fluorosis.

Out of the six maxillary anterior teeth, only tooth #8 (FDI 1.1) would fall into TSIF score 6 as per Horowitz et al. (1984), and TF index score 5, as per Thylstrup and Fejerskov (1978). The other five maxillary anterior teeth would fall into TSIF score 7 (Horowitz et al. 1984) and TF index score 6 (Thylstrup and Fejerskov (1978).

Clinical and radiographic exams did not disclose any problem with soft tissues, pulp vitality, and periodontal health. Esthetically, the shape of the anterior teeth was not harmonious. The lateral incisors looked too short while the central incisors were square shaped. A waxed-up model was prepared to reflect longer clinical crowns with a more pleasant proportion. This model was used to explain to patient how a change in the size of the clinical crowns might enhance her smile.

The following treatment plan was agreed with the patient:

1. Direct resin-based composite veneers to mask the discolorations as an immediate solution to improve the patient's self-confidence (Fig. 15.2). Patient was informed that the aspect of her smile would improve considerably but she might still notice a few areas of discoloration.
2. A second phase of the treatment included a gingivoplasty procedure to recontour the tissue (Fig. 15.3) and lengthen the clinical crowns. After soft tissue healing, minimally invasive preparations for porcelain crowns were carried out, followed

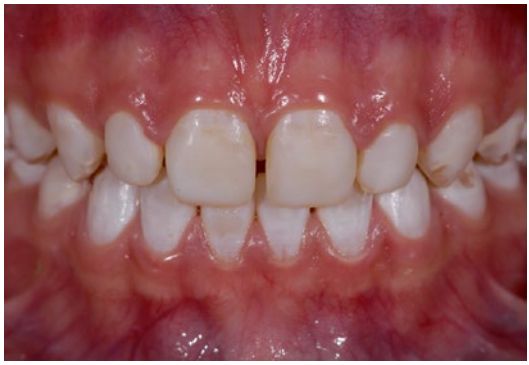

Fig. 15.2 Clinical aspect after direct resin-based composite was bonded to the slightly roughened enamel, using a two-step etch-and-rinse adhesive

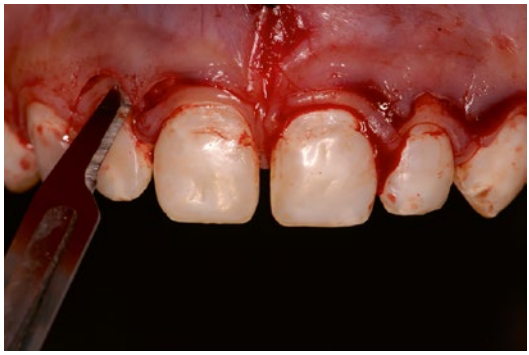

Fig. 15.3 Gingivoplasty to recontour the gingival tissue

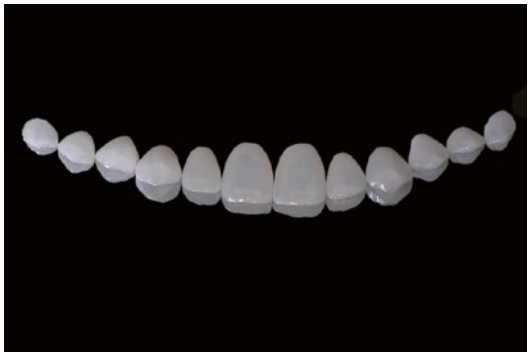

Fig. 15.4 IPS e.max Press (Ivoclar Vivadent) lithium disilicate restorations as received from the dental laboratory

by thin porcelain restorations bonded to enamel to restore function and esthetics (Figs. 15.4 and 15.5).

15.2 Dental Fluorosis Treated with At-Home Whitening and Porcelain Veneers

The major complaint of this 19-year-old female patient was related to the color of her front teeth (Figs. 15.6, 15.7, 15.8, 15.9, and 15.10). She described major social difficulties at school because of her "unpleasant smile." She had no medical

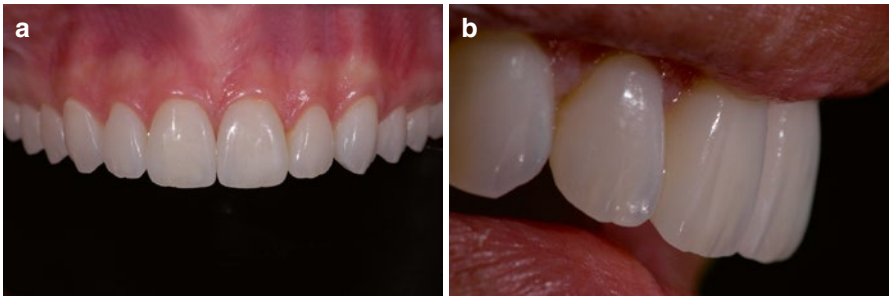

Fig. 15.5 (**a**) Clinical aspect immediately after bonding the ceramic restorations with a two-step etch-and-rinse adhesive (Adper Single Bond Plus, 3M ESPE) and a dual-cured resin-based luting composite material (RelyX ARC, 3M ESPE). (**b**) Patient's smile 2 days after the restorations were bonded

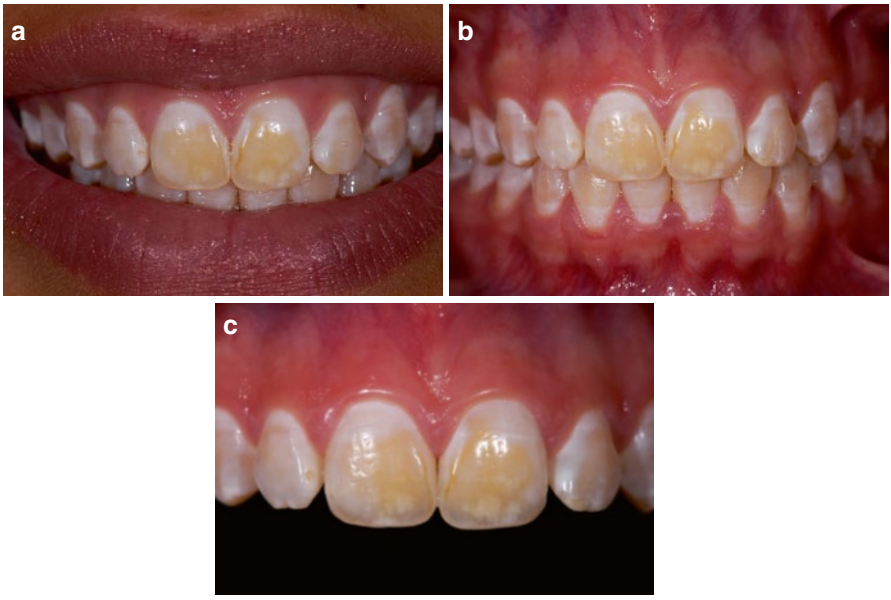

Fig. 15.6 (**a**) Nonretracted frontal view showing the wide yellowish discoloration on teeth #8 (FDI 1.1) and #9 (FDI 2.1) and white spot areas in other teeth. Some teeth display single pitted enamel areas. (**b**) Retracted view. The lower incisors also have yellowish discolorations along the perikymata. (**c**) Close-up view of the maxillary incisors

conditions and no history in her family related to alterations in the appearance of teeth. The clinical exam revealed that the periodontal condition was excellent. Radiographically, there were no structural areas of concern in the periodontal and periapical areas.

Similar to the patient shown in Sect. 15.1, this patient had lived in the same rural area. Other children in the same community had discolored teeth, including some of her family members.

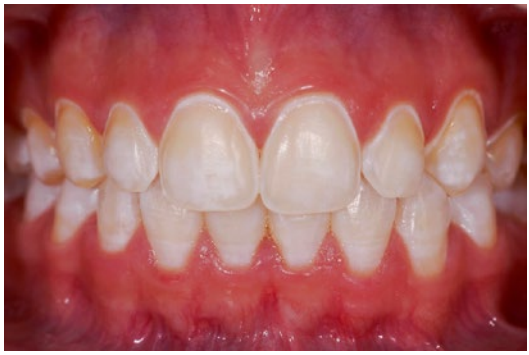

Fig. 15.7 Preparation of maxillary teeth for porcelain veneers. Patient was not anesthetized because the preparation was limited to enamel

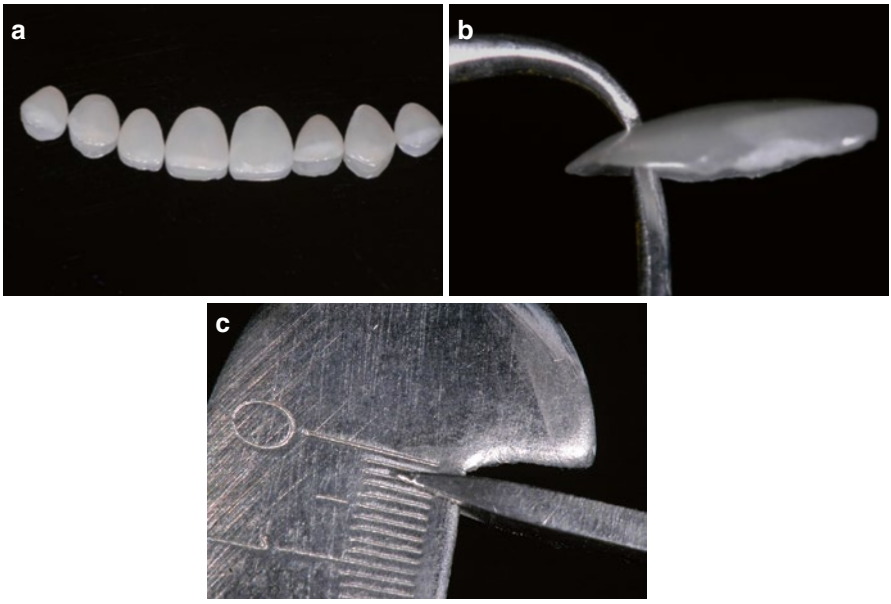

Fig. 15.8 (**a**) Porcelain veneers fabricated with IPS e.max Press (Ivoclar Vivadent) lithium disilicate. (**b**, **c**) The thickness of the veneers ranged from 0.2 mm to 0.3 mm

The diagnosis for this clinical case was dental fluorosis. This patient's fluorosis level would fall into TSIF score of 4 (Horowitz et al. 1984) and a TF index score of 4 (Thylstrup and Fejerskov 1978). The treatment plan proposed to the patient was at-home whitening with 10% carbamide peroxide gel in a custom-fitted tray for one month and possibly every month thereafter up to 5–6 months, depending on the outcome after the first month. In case at-home whitening did not result in "whiter" teeth, we would try enamel microbrasion (Chap. 9) or a more invasive restorative procedure, such as direct or indirect veneers. Patient was informed that microabrasion is usually less conservative than at-home whitening. Patient accepted this initial treatment plan.

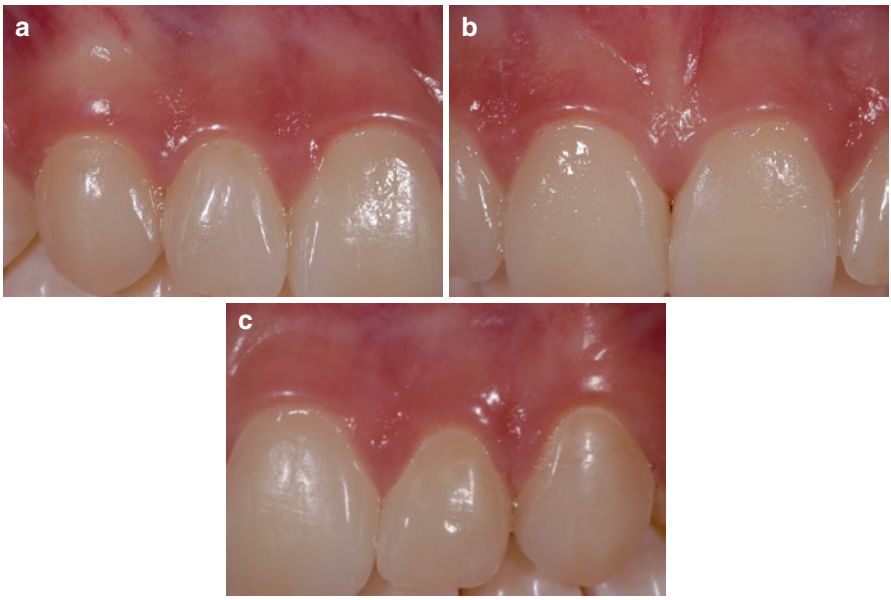

Fig. 15.9 The veneers were cemented with a two-step etch-and-rinse adhesive system (Adper Single Bond Plus, 3M ESPE) and a light-cure resin-based luting composite material (RelyX Veneer, 3M ESPE) (**a**–**c**). One week after the luting procedure, the integration of the porcelain restorations with the gingival tissue was excellent

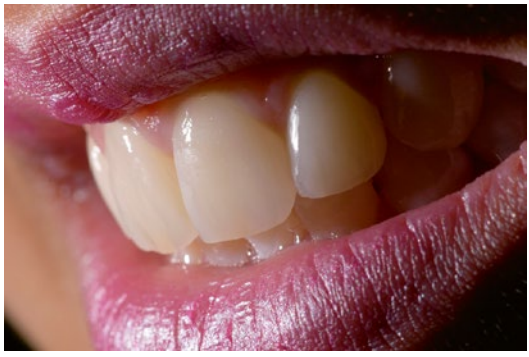

Fig. 15.10 Patient's smile 1 week after the veneers were bonded

Patient returned to the dental office after 5 weeks. No visible changes had occurred with the color of her teeth. Patient's compliance might have been responsible for the apparently unsuccessful whitening regimen, as patient mentioned that she forgot to wear the trays for a few days. At this point we asked patient if she wanted to start the enamel microabrasion procedure, which she declined. She wanted a "more permanent solution."

After presenting the restorative treatment options to the patient, which included direct resin-based composite veneers or porcelain veneers, she returned 2 weeks later to start the clinical procedure for 8 thin porcelain veneers in her maxillary teeth.

15.3 Enamel Idiopathic Hypomineralization Treated with Direct Resin-based Composite

As mentioned in Chap. 6, at-home whitening may highlight the enamel hypomineralized areas and make them more pronounced in case they are located deep in the enamel. The photograph shown in Fig. 15.11a is that of a 19-year-old female patient who had bleached her teeth a few months earlier with 10% carbamide peroxide in a custom-fitted tray for 3 weeks. All the patients' anterior teeth were vital without any clinical or radiographic signs of pathology, except for the enamel hypomineralized area on tooth #10 (FDI 2.2).

According to the patient's description, the white enamel area of tooth #10 (FDI 2.2) became wider and more opaque with the whitening treatment (as shown in Chap. 6, Figs. 6.14a, b). Clinically, the white opaque enamel area exhibited a concavity in the central area of the lesion, denoting loss of enamel (Fig. 15.11). Transillumination confirmed that the center of the lesion had a thinner area of tooth structure compared to the periphery (Fig. 15.12). The increased opacity of the tooth structure surrounding the more translucent zone suggested that the defect was deep inside the tooth and therefore not amenable to enamel microabrasion.

The treatment plan was removal of the hypomineralized area (Fig. 15.13), etching with 35% phosphoric acid for 15 s, and restoration with a two-step etch-and-rinse adhesive (Adper Single Bond Plus, 3M ESPE) followed by a resin-based composite (Filtek Supreme Ultra, 3M ESPE). After inserting and light curing the resin-based composite, the restoration was finished with Sof-Lex XT disks (3M

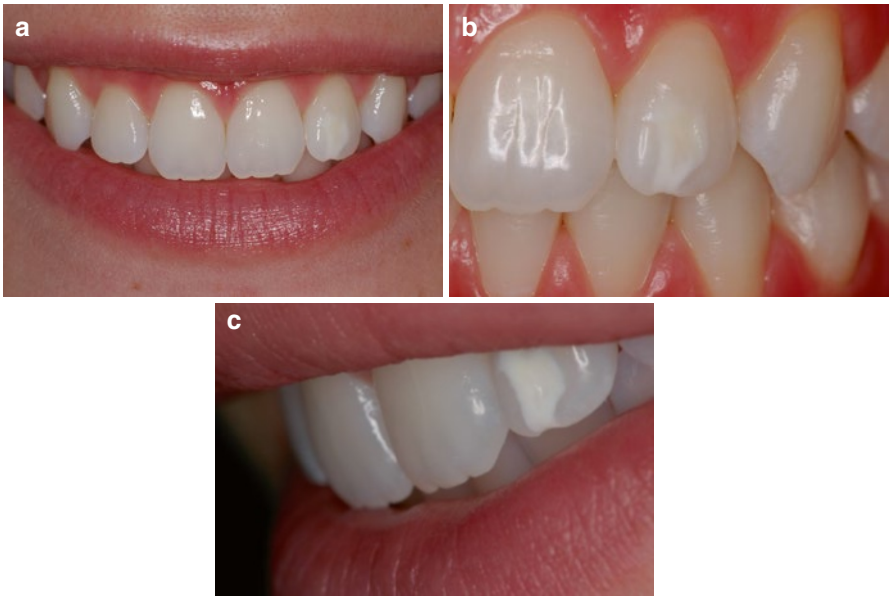

Fig. 15.11 (**a**) Smile of a 19-year-old female patient who had bleached her teeth a few months earlier with 10% carbamide peroxide in a custom-fitted tray for 3 weeks. (**b**, **c**) Tooth #10 (FDI 2.2) displayed an enamel hypomineralized area with a concavity in the central area of the lesion denoting loss of enamel

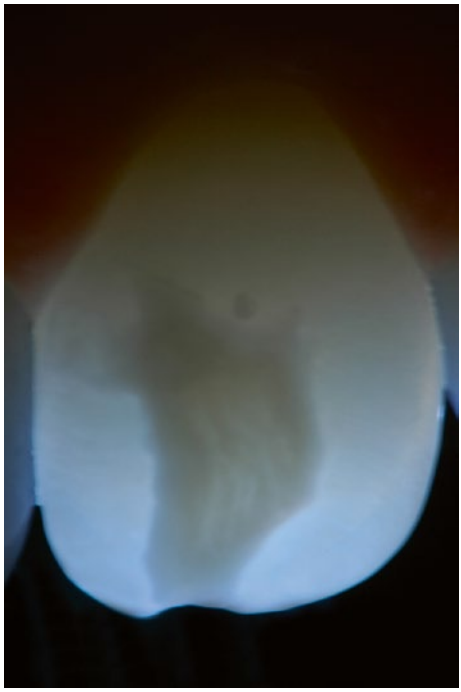

Fig. 15.12 Transillumination confirmed that the center of the hypomineralized area had a thinner area of tooth structure compared to the periphery

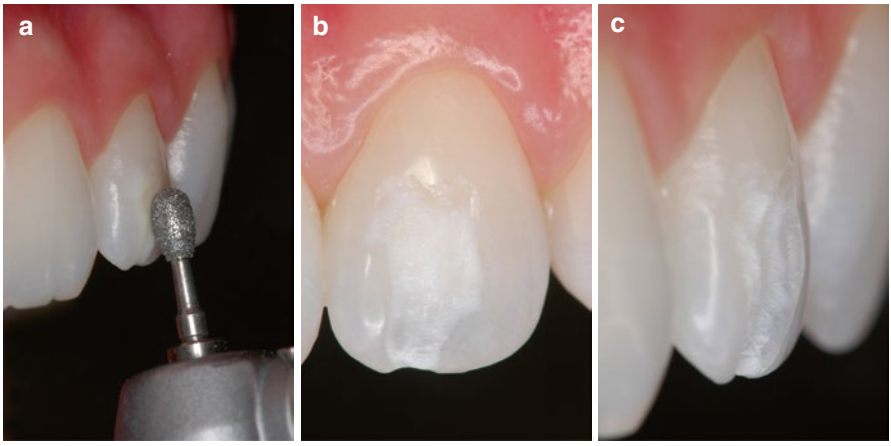

Fig. 15.13 The porous enamel was removed with a diamond bur

ESPE) (Fig. 15.14a) followed by characterization of the secondary anatomy (Fig. 15.14b) with a composite finishing bur. A felt disk (Diamond Flex, FGM) and a fine diamond paste (Diamond Excel, FGM) and used for the final polishing step (Fig. 15.14c). Figure 15.15 is a close-up photograph of the final aspect of restored tooth #10 (FDI 2.2) showing an optimal esthetic integration with the other anterior teeth. Figure 15.16 portrays the patient's new smile.

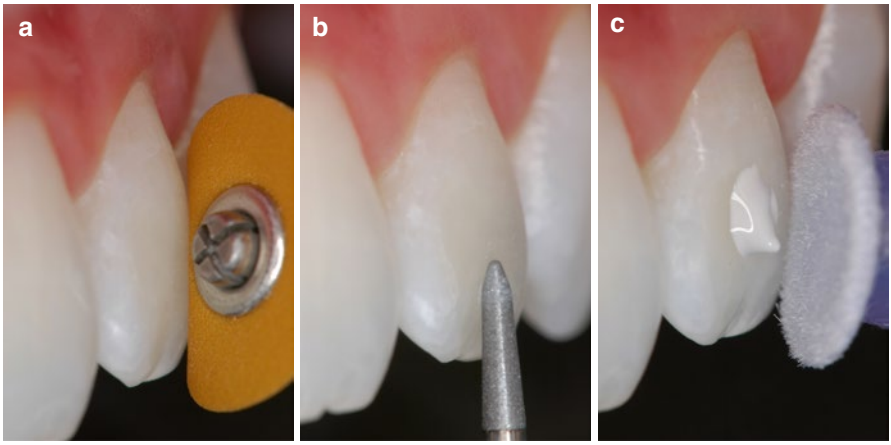

Fig. 15.14 After etching with 35% phosphoric acid for 15 s, a two-step etch-and-rinse adhesive was applied, gently air dried and light cured, followed by a nanofilled resin-based composite; (**a**) Finishing with aluminum oxide disks; (**b**) Placing secondary anatomy with a fine diamond finishing bur; (**c**) The final polishing step was carried out with a felt disk impregnated with a fine diamond paste

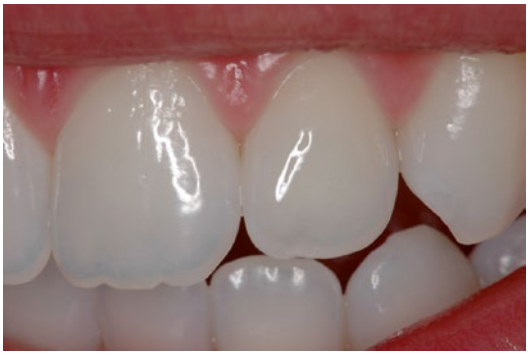

Fig. 15.15 Close-up photograph of the restored tooth

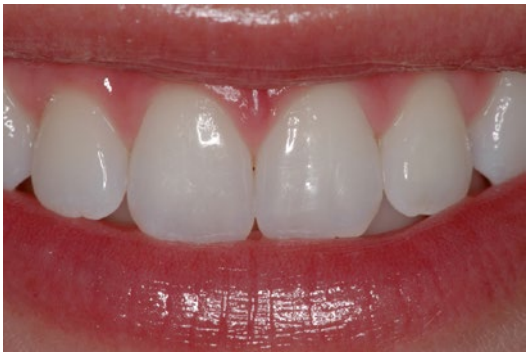

Fig. 15.16 The patient's new smile

15.4 Restorative Solution for a Case of Unsuccessful Intra-coronal Whitening

As described in Chap. 8, intra-coronal whitening of endodontically treated teeth has a fairly good prognosis for discolorations caused by necrotic pulp tissue or blood components, with a short-term success rate of 50–90 %. However, the long-term success rate is considerably lower as some color regression may occur after the initial bleaching effect.

This 35-year-old female patient was concerned about the discoloration of her right maxillary central incisor (Figs. 15.17, 15.18, 15.19, 15.20, 15.21, and 15.22). According to the patient this tooth had been endodontically treated 6 years ago and immediately became darker. Her dentist performed three intra-coronal whitening treatments three years later, followed by two sessions of external in-office whitening. In spite of a slight improvement in the tooth color patient never felt that the tooth was esthetically acceptable.

Medical history was not contributory. Radiographically tooth #8 (FDI 1.1) had no evidence of root resorption or periapical pathology. The radiographic aspect of the root canal treatment was considered excellent. The clinical exam revealed a few areas of incipient caries lesions on the posterior area, besides defective resin-based

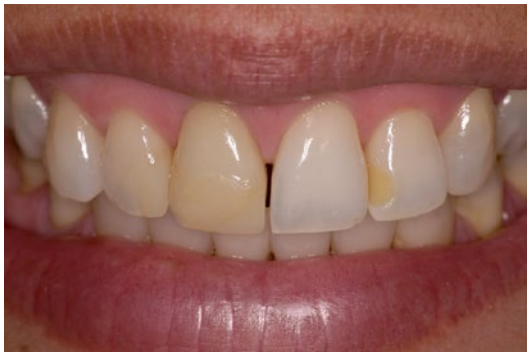

Fig. 15.17 Preoperative view showing the discoloration of tooth #8 (FDI 1.1)

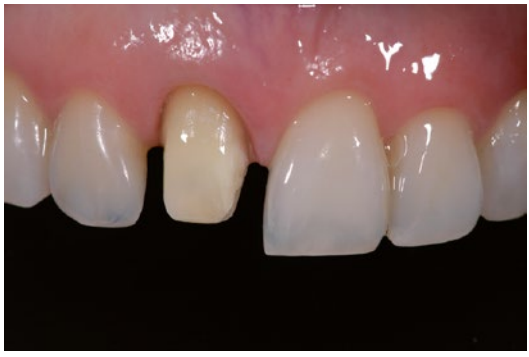

Fig. 15.18 After replacement of resin-based composite restorations and preparation of tooth # 8 (FDI 1.1) for a porcelain crown

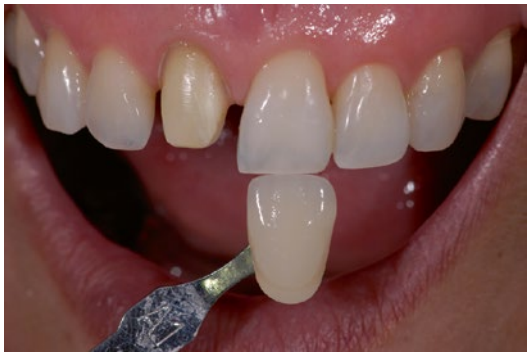

Fig. 15.19 Shade matching

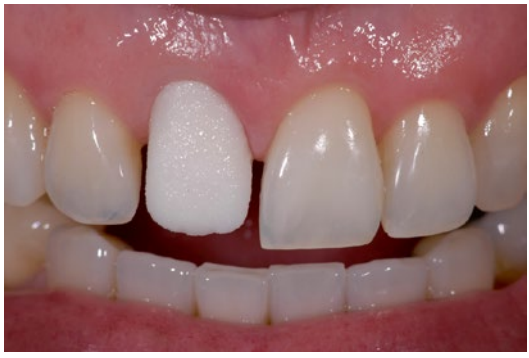

Fig. 15.20 High-opacity lithium disilicate coping try-in (IPS e.max Press HO, Ivoclar Vivadent)

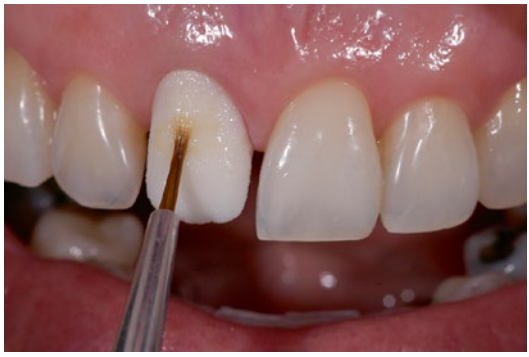

Fig. 15.21 Characterization of the opaque ceramic coping

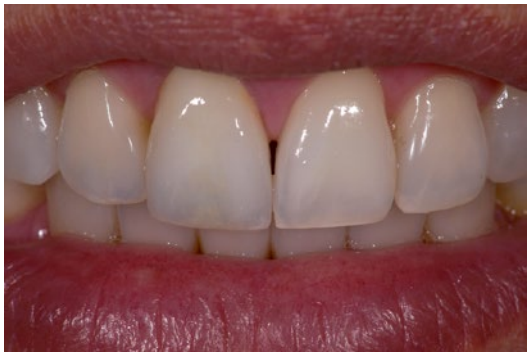

Fig. 15.22 Final aspect after adhesive cementation of the lithium disilicate ceramic crown

composite restorations in the anterior segment. The treatment plan proposed to the patient to solve the compromised esthetics included replacement of the resin-based composite restorations on teeth #7 (FDI 1.2), #9 (FDI 2.1) and #10 (FDI 2.2), followed by a bonded porcelain crown to correct the discoloration of tooth #8 (FDI 1.1).

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