TOOTH DISCOLORATION AND BLEACHING

Ilan Rotstein

Tooth discoloration usually occurs owing to patient- or dentist-related causes[1,2] (Table 16-1).

PATIENT-RELATED CAUSES

Pulp Necrosis

Bacterial, mechanical, or chemical irritation to the pulp may result in tissue necrosis and release of disintegration by-products that may penetrate tubules and discolor the surrounding dentin. The degree of discoloration is directly related to how long the pulp has been necrotic. The longer the discoloration compounds are present in the pulp chamber, the greater the discoloration. Such discoloration can usually be bleached intracoronally (Figures 16-1 and 16-2).

Intrapulpal Hemorrhage

Intrapulpal hemorrhage and lysis of erythrocytes are a common result of traumatic injury to a tooth. Blood disintegration products, presumably iron sulfides, flow into the tubules and discolor the surrounding dentin. If the pulp becomes necrotic, the discoloration persists and usually becomes more severe with time. If the pulp recovers, the discoloration may be reversed, with the tooth regaining its original shade. The severity of such

Table 16-1   Causes of Tooth Discoloration

Patient-Related Causes	Dentist-Related causes
Pulp necrosis	Endodontically related
Intrapulpal hemorrhage	Pulp tissue remnants
Dentin hypercalcification	Intracanal medicaments
Age	Obturating materials
Tooth formation defects	Restoration related
Developmental defects	Amalgams
Drug-related defects	Pins and posts
	Composites

discoloration is time dependent; intracoronal bleaching is usually quite effective in this type of discoloration.[3–5]

Dentin Hypercalcification

Excessive formation of irregular dentin in the pulp chamber and along canal walls may occur following certain traumatic injuries. In such cases, a temporary disruption of blood supply occurs, followed by destruction of odontoblasts. These are replaced by undifferentiated mesenchymal cells that rapidly form irregular dentin on the walls of the pulp lumen. As a result, the translucency of the crowns of such teeth gradually decreases, giving rise to a yellowish or yellow-brown discoloration.

Extracoronal bleaching may be attempted first. However, sometimes root canal therapy is required followed by intracoronal bleaching.

Age

In elderly patients, color changes in the crown occur physiologically, a result of excessive dentin apposition, thinning of the enamel, and optical changes. Food and beverages also have a cumulative discoloration effect. These become more pronounced in the elderly, owing to the inevitable cracking, crazing, and incisal wear of the enamel and underlying dentin. In addition, amalgam and other coronal restorations that degrade over time cause further discoloration. When indicated, bleaching can be successfully done for many types of discolorations in elderly patients.

TOOTH FORMATION DEFECTS

Developmental Defects

Discoloration may result from developmental defects during enamel and dentin formation, either hypocalcific or hypoplastic. Enamel *hypocalcification* is a distinct brownish or whitish area, commonly found on
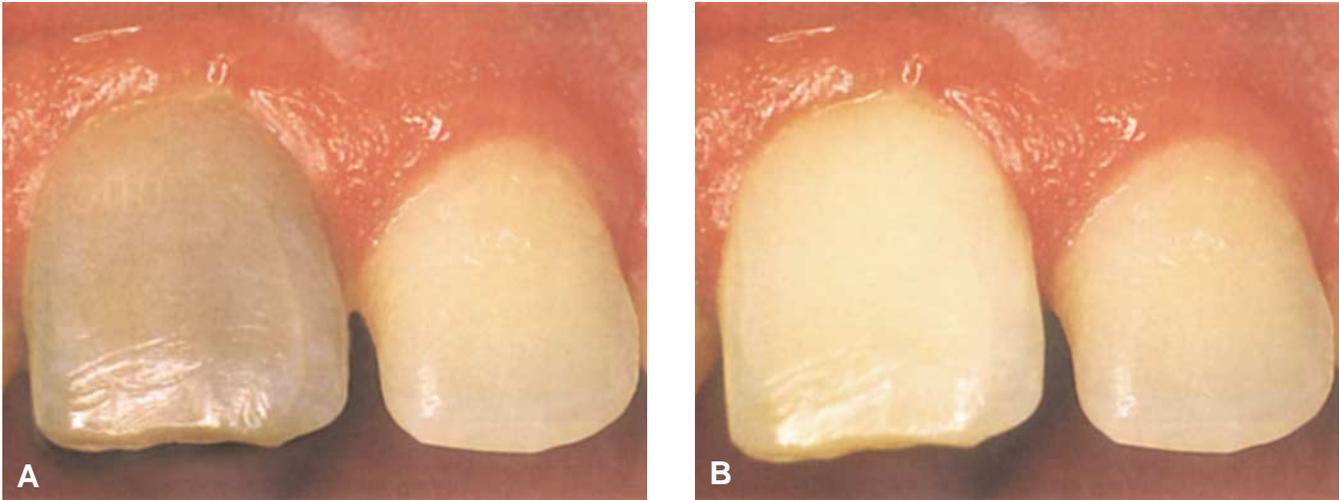
Figure 16-1 **A,** Post-traumatic discoloration of a maxillary left central incisor. **B,** A mixture of perborate and distilled water, placed in the chamber 2 times over 3 weeks, achieved the lightening of the tooth to its natural color. Reproduced with permission from Docteur Anne Claisse-Crinquette. L'eclaireissement des dyschromies: une longue histoire. Paris, France, Endo Contact 1999;2:16.

the facial aspect of affected crowns. The enamel is well formed with an intact surface.

Enamel *hypoplasia* differs from hypocalcification in that the enamel is defective and porous. This condition may be hereditary, as in amelogenesis imperfecta, or a result of environmental factors such as infections, tumors, or trauma. Presumably, during enamel formation, the matrix is altered and does not mineralize properly. The defective enamel is porous and readily discolored by materials in the oral cavity. In such cases, bleaching effect may not be permanent depending on the severity and extent of hypoplasia and the nature of the discoloration.

Several other systemic conditions may cause tooth discoloration. Erythroblastosis fetalis, for example, may occur in the fetus or newborn because of Rh incompatibility factors, with resulting massive systemic lysis of erythrocytes. Large amounts of hemosiderin pigment are released, which subsequently penetrate and discolor the forming dentin. This condition may also present a variety of systemic complications.[6] However, such discoloration is now uncommon and is not amenable to bleaching. High fever during tooth formation may also result in chronologic hypoplasia, a temporary disruption in enamel formation that gives rise to banding-type surface discoloration. Porphyria, a
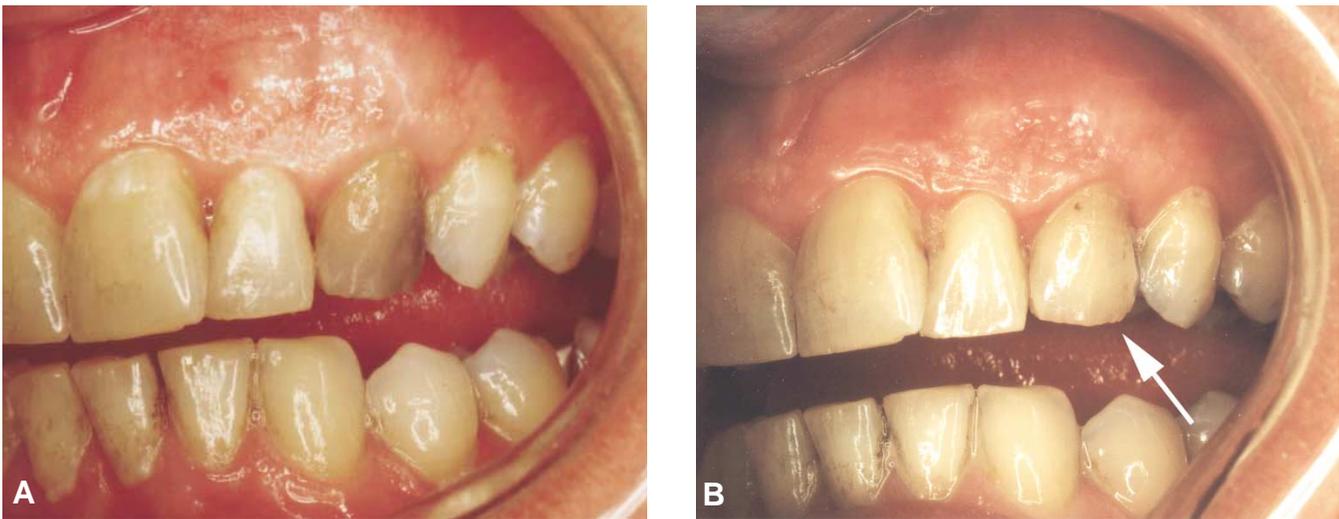

Figure 16-2 Traumatic pulp necrosis and dentin discoloration of a maxillary central incisor. **A,** Before treatment. **B,** Esthetic results following one treatment of "walking bleach" with sodium perborate mixed with Superoxol. Reproduced with permission from Nutting EB, Poe GS. Dent Clin North Am 1967;655.

metabolic disease, may also cause red or brownish discoloration of deciduous and permanent teeth. Thalassemia and sickle cell anemia may cause intrinsic blue, brown, or green discolorations. Amelogenesis imperfecta may result in yellow or brown discolorations. Dentinogenesis imperfecta can cause brownish violet, yellowish, or gray discoloration. These conditions are usually not amenable to bleaching and should be corrected by restorative means.

Drug-Related Defects

Administration or ingestion of certain drugs during tooth formation may cause severe discoloration both in enamel and dentin.[7,8]

Tetracycline. This antibiotic was used extensively during the 1950s and 1960s for prophylactic protection and for the treatment of chronic obstructive pulmonary disease, *Mycoplasma*, and rickettsial infections. It was sometimes prescribed for long periods of time, years in some cases, and therefore was a common cause of tooth discoloration in children. Although, today, tetracycline is not usually administrated chronically, dentists still face the residue of damage to the appearance of the teeth of the prior two generations.

Tooth shades can be yellow, yellow-brown, brown, dark gray, or blue, depending on the type of tetracycline, dosage, duration of intake, and patient's age at the time of administration. Discoloration is usually bilateral, affecting multiple teeth in both arches. Deposition of the tetracycline may be continuous or laid down in stripes depending on whether the ingestion was continuous or interrupted (Figure 16-3).

The mechanism of tetracycline discoloration is not fully understood. Tetracycline bound to calcium is thought to be incorporated into the hydroxyapatite crystal of both enamel and dentin. However, most of the tetracycline is found in dentin.

Tetracycline discoloration has been classified into three groups according to severity.[9] First-degree discoloration is light yellow, light brown, or light gray and occurs uniformly throughout the crown, without banding. Second-degree discoloration is more intense and also without banding. Third-degree discoloration is very intense, and the clinical crown exhibits horizontal color banding. This type of discoloration usually predominates in the cervical regions.

Repeated exposure of tetracycline-discolored tooth to ultraviolet radiation can lead to formation of a reddish-purple oxidation by-product that permanently discolors the teeth. In children, the anterior teeth often discolor first, whereas the less exposed posterior teeth are discolored more slowly. In adults, natural photo-bleaching of the anterior teeth is observed, particularly in individuals whose teeth are excessively exposed to sunlight owing to maxillary lip insufficiency.

Two approaches have been used to treat tetracycline discoloration: (1) bleaching the external enamel surface[9–11] (Figure 16-4) and (2) intracoronal bleaching following intentional root canal therapy[12–14] (Figure 16-5).

Endemic Fluorosis. Ingestion of excessive amounts of fluoride during tooth formation may produce a defect in mineralized structures, particularly in the enamel matrix, causing hypoplasia. The severity and degree of subsequent staining generally depend on the degree of hypoplasia and are directly related to the amount of fluoride ingested during odontogenesis.[15] The teeth are not discolored on eruption, but their surface is porous and will gradually absorb colored chemicals present in the oral cavity.

Discoloration is usually bilateral, affecting multiple teeth in both arches. It presents as various degrees of intermittent white spotting, chalky or opaque areas, yellow or brown discoloration, and, in severe cases, surface pitting of the enamel. Since the discoloration is in the porous enamel, such teeth can be bleached externally.

Figure 16-3 Fluorescent photomicrograph of tetracycline-discolored tooth. Tetracycline deposition is seen as stripes caused by start and stop ingestion. (Courtesy of Dr. David L. Myers.)
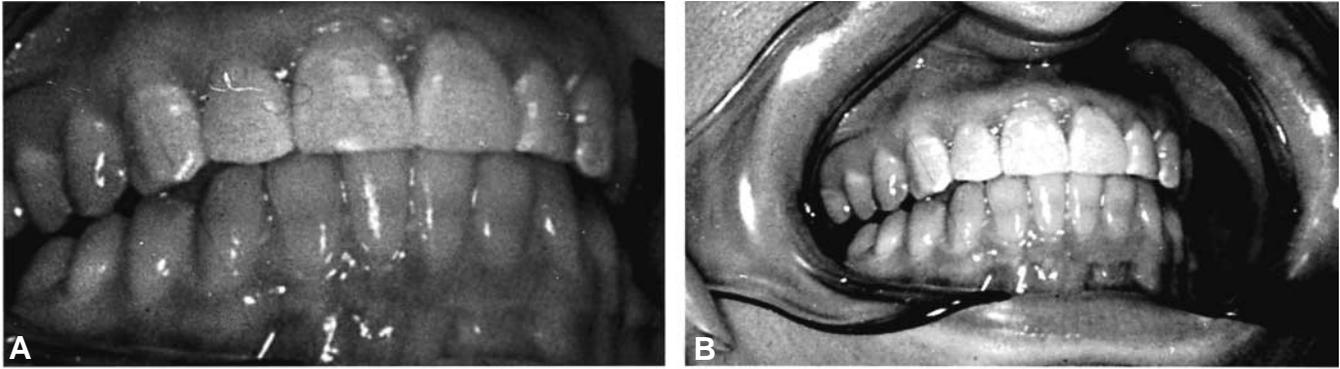
Figure 16-4 Extracoronal bleaching of tetracycline-discolored maxillary incisors. **A**, Before bleaching. **B**, After three sessions of bleaching with *Hi Lite Dual Activated Bleach* as compared to unbleached discolored area of mandibular incisors. (Courtesy of Drs. Fred and Scott Hanosh.)

DENTIST-RELATED CAUSES

Discolorations owing to various dental materials or unsuitable operating techniques do occur; such dentist-related discolorations are usually preventable and should be avoided.

Endodontically Related

Pulp Tissue Remnants. Tissue remaining in the pulp chamber disintegrates gradually and may cause discoloration. Pulp horns must always be included in the access cavity to ensure removal of pulpal remnants and to pre-
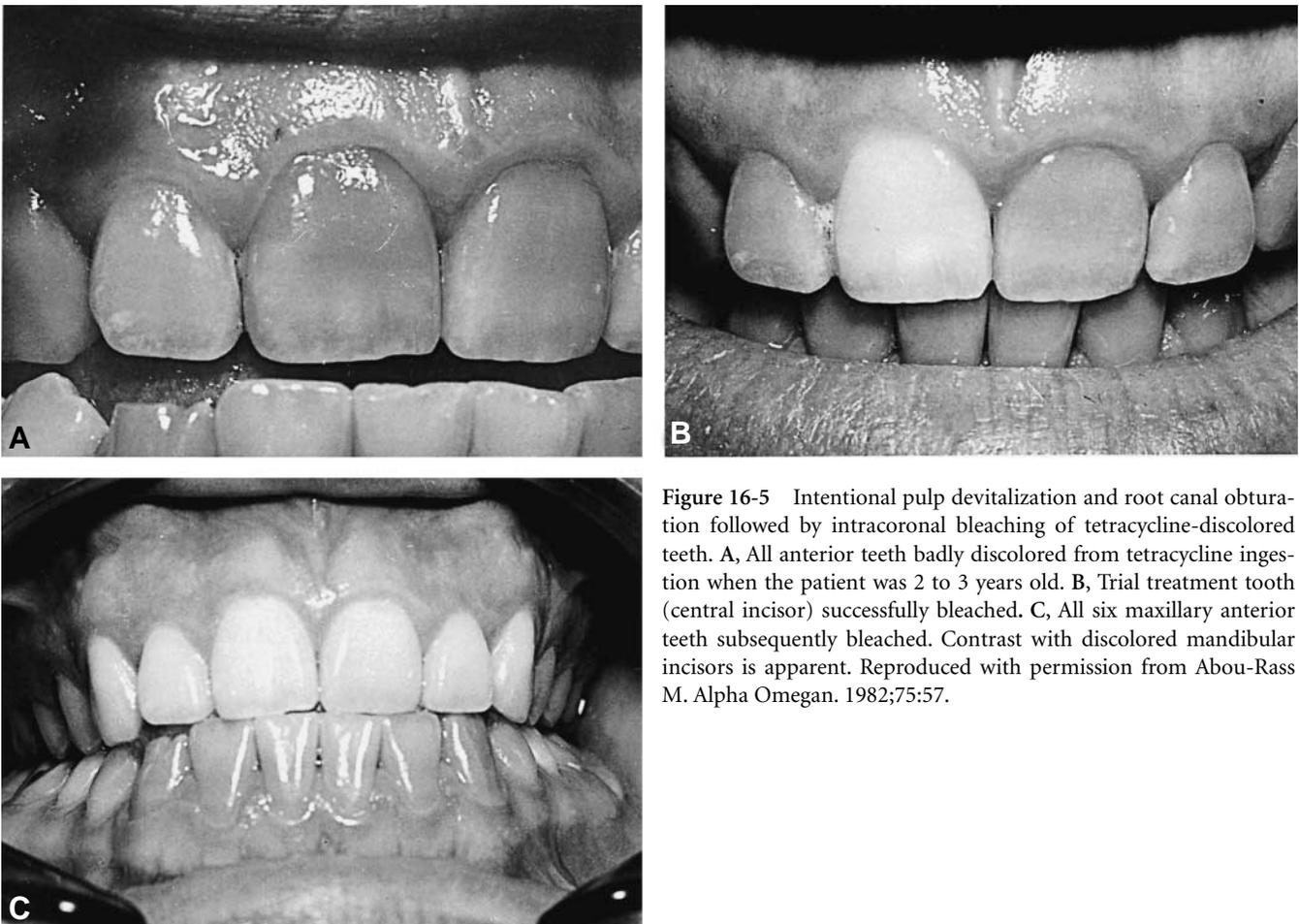

Figure 16-5 Intentional pulp devitalization and root canal obturation followed by intracoronal bleaching of tetracycline-discolored teeth. **A**, All anterior teeth badly discolored from tetracycline ingestion when the patient was 2 to 3 years old. **B**, Trial treatment tooth (central incisor) successfully bleached. **C**, All six maxillary anterior teeth subsequently bleached. Contrast with discolored mandibular incisors is apparent. Reproduced with permission from Abou-Rass M. Alpha Omegan. 1982;75:57.

vent retention of sealer at a later stage. Intracoronal bleaching in these cases is usually successful.

Intracanal Medicaments. Several intracanal medicaments are liable to cause internal staining of the dentin. Phenolics or iodoform-based medicaments sealed in the root canal and chamber are in direct contact with dentin, sometimes for long periods, allowing penetration and oxidization. These compounds have a tendency to discolor the dentin gradually.

Obturating Materials. This is a frequent and severe cause of single tooth discoloration. Incomplete removal of obturating materials and sealer remnants in the pulp chamber, mainly those containing metallic components,[16] often results in dark discoloration. This is easily prevented by removing all materials to a level just below the gingival margin (Figure 16-6).

Intracoronal bleaching is the treatment of choice; prognosis, however, in such cases depends on the type of sealer and duration of discoloration.

Restoration Related

Amalgams. Silver alloys have severe effects on dentin owing to dark-colored metallic components that can turn the dentin dark gray. When used to restore lingual access preparations or a developmental groove in anterior teeth, as well as in premolar teeth, amalgam may discolor the crown. Such discolorations are difficult to bleach and tend to rediscolor with time.
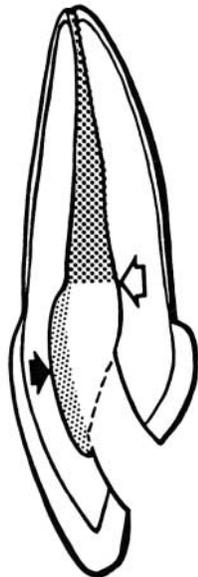

Figure 16-6   Gutta-percha and dentin removal prior to bleaching. **Heavyily dotted area** represents gutta-percha filling, which is removed to a level just below the gingival level (**open arrow**). **Lightly dotted area** represents removal of dentin to eliminate heavy stain concentration and pulp horn material (**black arrow**).

Sometimes the dark appearance of the crown is caused by the amalgam restoration that can be seen through the tooth structure. In such cases, replacing the amalgam with an esthetic restoration usually corrects the problem.

Pins and Posts. Metal pins and prefabricated posts are sometimes used to reinforce a composite restoration in the anterior dentition. Discoloration from inappropriately placed pins and posts is caused by the metal seen through the composite or tooth structure. In such cases, coverage of the pins with a white cement or removal of the metal and replacement of the composite restoration is indicated.

Composites. Microleakage around composite restorations causes staining. Open margins may allow chemicals to enter between the restoration and the tooth structure and discolor the underlying dentin. In addition, composites may become discolored with time, affecting the shade of the crown. These conditions are generally corrected by replacing the old composite restoration with a new, well-sealed one.

BLEACHING MATERIALS

Many different bleaching agents are available today; the ones most commonly used are hydrogen peroxide, sodium perborate, and carbamide peroxide. Hydrogen peroxide and carbamide peroxide are mainly indicated for extracoronal bleaching, whereas sodium perborate is used for intracoronal bleaching.

Hydrogen Peroxide. Various concentrations of this agent are available, but 30 to 35% stabilized aqueous solutions (Superoxol, Perhydrol Merck & Co.; West Point, Pa.) are the most common. Silicone dioxide gel forms containing 35% hydrogen peroxide are also available, some of them activated by a composite curing light (Figure 16-7).

Hydrogen peroxide is caustic and burns tissues on contact, releasing toxic free radicals, perhydroxyl anions, or both. High-concentration solutions of hydrogen peroxide must be handled with care as they are thermodynamically unstable and may explode unless refrigerated and kept in a dark container.

Sodium Perborate. This oxidizing agent is available in a powdered form or as various commercial preparations. When fresh, it contains about 95% perborate, corresponding to 9.9% of the available oxygen. Sodium perborate is stable when dry. In the presence of acid, warm air, or water, however, it decomposes to form sodium metaborate, hydrogen peroxide, and nascent oxygen.

Three types of sodium perborate preparations are available: monohydrate, trihydrate, and tetrahydrate. They differ in oxygen content, which determines their

Figure 16-7   Bleaching gel applied to labial surfaces of maxillary teeth and activated by resin curing light. Note the rubber dam carefully protecting gingiva from bleaching agent. (Courtesy of Shofu Dental Corp.;San Marcos, Calif.)

Figure 16-8   Sodium perborate powder and liquid are mixed to a thick consistency of wet sand.

bleaching efficacy.[17] Commonly used sodium perborate preparations are alkaline, and their pH depends on the amount of hydrogen peroxide released and the residual sodium metaborate.[18]

Sodium perborate is more easily controlled and safer than concentrated hydrogen peroxide solutions. Therefore, it should be the material of choice in most intracoronal bleaching procedures (Figure 16-8).

Carbamide Peroxide.   This agent, also known as urea hydrogen peroxide, is available in the concentration range of 3 to 45%. However, popular commercial preparations contain about 10% carbamide peroxide, with a mean pH of 5 to 6.5. Solutions of 10% carbamide peroxide break down into urea, ammonia, carbon dioxide, and approximately 3.5% hydrogen peroxide.

Bleaching preparations containing carbamide peroxide usually also include glycerine or propylene glycol, sodium stannate, phosphoric or citric acid, and flavor additives. In some preparations, carbopol, a water-soluble polyacrylic acid polymer, is added as a thickening agent. Carbopol also prolongs the release of active peroxide and improves shelf life.

Carbamide peroxide–based preparations have been associated with various degrees of damage to the teeth and surrounding mucosa.[19–23] They also may adversely affect the bond strength of composite resins and their marginal seal.[24,25] Since long-term studies are not yet available, these materials must be used with caution.

BLEACHING MECHANISM

The mechanism of tooth bleaching is unclear. It differs according to the type of discoloration involved and the chemical and physical conditions at the time of the reaction. Bleaching agents, mainly oxidizers, act on the organic structure of the dental hard tissues, slowly degrading them into chemical by-products, such as carbon dioxides, that are lighter in color. Inorganic molecules do not usually break down as well. The oxidation-reduction reaction that occurs during bleaching is known as a redox reaction. Generally, the unstable peroxides convert to unstable free radicals. These free radicals may oxidize (remove electrons from) or reduce (add electrons to) other molecules.

Most bleaching procedures use hydrogen peroxide because it is unstable and decomposes into oxygen and water. The **mouthguard** bleaching technique employs mainly carbamide peroxide as a vehicle for the delivery of lower concentrations of hydrogen peroxide, which require more exposure time. The rate of hydrogen peroxide decomposition during mouthguard bleaching depends on its concentration and the levels of salivary peroxidase. With high levels of hydrogen peroxide, a zero-order reaction occurs, so that the time required to clear the hydrogen peroxide is proportional to its concentration. The longer it takes to clear the hydrogen peroxide, the greater the exposure to reactive oxygen species and their adverse effects.

BLEACHING TECHNIQUES FOR ENDODONTICALLY TREATED TEETH

Intracoronal bleaching of endodontically treated teeth may be successfully carried out many years after root canal therapy and discoloration. A successful outcome depends mainly on the etiology, correct diagnosis, and proper selection of bleaching technique (Table 16-2).

The methods most commonly employed to bleach endodontically treated teeth are the **walking bleach** and the **thermocatalytic** techniques. Walking bleach is preferred since it requires less chair time and is safer and more comfortable for the patient.[26,27]

Walking Bleach

The walking bleach technique should first be attempted in all cases requiring *intracoronal* bleaching. It involves the following steps:

1. Familiarize the patient with the possible causes of discoloration, procedure to be followed, expected outcome, and possibility of future **re**discoloration.
2. Radiographically assess the status of the periapical tissues and the quality of the endodontic obturation. Endodontic failure or questionable obturation should **always be re-treated** prior to bleaching.
3. Assess the quality and shade of any restoration present and replace if defective. Tooth discoloration frequently is the result of leaking or discolored restorations. In such cases, cleaning the pulp chamber and replacing the defective restorations will usually suffice.
4. Evaluate tooth color with a shade guide and, if possible, take clinical photographs at the beginning of and throughout the procedure. These provide a point of reference for future comparison.
5. Isolate the tooth with a rubber dam. The dam must fit tightly at the cervical margin of the tooth to prevent possible leakage of the bleaching agent onto the gingival tissue. Interproximal wedges and ligatures may also be used for better isolation. If

Table 16-2   Indications and Contraindications for Bleaching Endodontically Treated Teeth

Indications	Contraindications
Discolorations of pulp chamber	Superficial enamel discolorations
Dentin discolorations	Defective enamel formation
Discolorations not amenable to extracoronal bleaching	Severe dentin loss
	Presence of caries
	Discolored composites

Superoxol is used, a protective cream, such as Orabase or Vaseline, must be applied to the surrounding gingival tissues prior to dam placement.

6. Remove all restorative material from the access cavity, expose the dentin, and refine the access. Verify that the pulp horns and other areas containing pulp tissue are clean.
7. Remove all materials to a level just below the labial-gingival margin. *Orange solvent*, chloroform, or xylene on a cotton pellet may be used to dissolve sealer remnants. Etching the dentin with phosphoric acid is unnecessary and may not improve the prognosis.
8. Apply a sufficiently thick layer, at least 2 mm, of a protective white cement barrier, such as polycarboxylate cement, zinc phosphate cement, glass ionomer, *IRM* (Dentsply/Caulk; York, Pa.) or *Cavit* (Premier Dental Products, King of Prussia, Pa.), to cover the endodontic obturation. The coronal height of the barrier should protect the dentinal tubules and conform to the external epithelial attachment.[28]
9. Prepare the walking bleach paste by mixing sodium perborate and an inert liquid, such as water, saline, or anesthetic solution, to a thick consistency of wet sand (Figure 16-8). Although a sodium perborate and 30% hydrogen peroxide mixture bleaches faster, in most cases, long-term results are similar to those with sodium perborate and water alone and therefore need not be used routinely.[4,5,29] With a plastic instrument, pack the pulp chamber with the paste. Remove excess liquid by tamping with a cotton pellet. This also compresses and pushes the paste into all areas of the pulp chamber.
10. Remove excess bleaching paste from undercuts in the pulp horn and gingival area and apply a thick well-sealed temporary filling (preferably *IRM*) directly against the paste and into the undercuts. Carefully pack the temporary filling, at least 3 mm thick, to ensure a good seal.
11. Remove the rubber dam and inform the patient that bleaching agents work slowly and significant lightening may not be evident for several days.
12. Evaluate the patient 2 weeks later and, if necessary, repeat the procedure several times.[29] Repeat treatments are similar to the first one.
13. As an optional procedure, if initial bleaching is not satisfactory, strengthen the walking bleach paste by mixing the sodium perborate with gradually increasing concentrations of hydrogen peroxide (3 to 30%) instead of water. The more potent oxidizers may have an enhanced bleaching effect but are not used routinely because of the possibility of permeation into the tubules and damage to the cervical periodontium by these more caustic agents.

Thermocatalytic

This technique involves placement of the oxidizing chemical, generally 30 to 35% hydrogen peroxide *(Superoxol)*, in the pulp chamber followed by heat application either by electric heating devices or specially designed lamps.

Potential damage by the thermocatalytic approach is external **cervical root resorption** caused by irritation to the cementum and periodontal ligament. This is possibly attributable to the oxidizing agent combined with heating.[30,31] Therefore, application of highly concentrated hydrogen peroxide and heat during intracoronal bleaching is questionable and should not be carried out routinely.

Ultraviolet Photo-Oxidation

This technique applies ultraviolet light to the labial surface of the tooth to be bleached. A 30 to 35% hydrogen peroxide solution is placed in the pulp chamber on a cotton pellet followed by a 2-minute exposure to ultraviolet light. Supposedly, this causes oxygen release, like the thermocatalytic bleaching technique.[32]

Intentional Endodontics and Intracoronal Bleaching

Intrinsic tetracycline and other similar stains are incorporated into tooth structure during tooth formation, mostly into the dentin, and are therefore more difficult to treat from the external enamel surface. Intracoronal bleaching of tetracycline-discolored teeth has been shown clinically[12] and experimentally[13,14] to lead to significant lightening.

The technique involves standard endodontic therapy (pulpectomy, cleaning and shaping, and obturation) followed by an intracoronal walking bleach technique. Preferably, only intact teeth without coronal defects, caries, or restorations should be treated. This prevents the need for any additional restoration, thereby reducing the possibility of coronal fractures and failures. The most discolored tooth should be selected for trial treatment (see Figure 16-5).

The procedure should be carefully explained to the patient, including the possible complications and sequelae. A treatment consent form is necessary! Sacrificing pulp vitality should be considered in terms of the overall psychological and social needs of the individual patient as well as the possible complications of other treatment options. The procedure has been shown to be predictable and without significant clinical complications.[12,33]

Complications and Adverse Effects to Bleaching

External Root Resorption.   Clinical reports[34–44] and histologic studies[30,31,45] have shown that intracoronal bleaching may induce external root resorption (Table 16-3 and Figure 16-9). This is probably caused by the oxidizing agent, particulary 30 to 35% hydrogen peroxide. The mechanism of bleaching-induced damage to the periodontium or cementum has not been fully elucidated. Presumably, the irritating chemical diffuses via unprotected dentinal tubules and cementum defects[46,47] and causes necrosis of the cementum, inflammation of the periodontal ligament, and, finally, root resorption. The process may be enhanced if heat is applied[48] or in

Table 16-3   Clinical Reports of External Root Resorption Associated with Hydrogen Peroxide Bleaching

| Authors	No. of Cases	Age of Patients	Previous Trauma		Heat Applied		Barrier Used	
			Yes	No	Yes	No	Yes	No
Harrington and Natkin[34]	7	10–29	7	0	7	0	0	7
Lado et al.[35]	1	50	0	1	1	0	0	1
Montgomery[36]	1	21	1	0	?	?	?	?
Shearer[37]	1	?	0	1	1	0	0	1
Cvek and Lindvall[38]	11	11–26	10	1	11	0	0	11
Latcham[39]	1	8	1	0	0	1	0	1
Goon et al.[40]	1	15	?	?	0	1	0	1
Friedman et al.[41]	4	18–24	0	4	3	1	0	4
Gimlin and Schindler[42]	1	13	1	0	1	0	0	1
Al-Nazhan[43]	1	27	0	1	1	0	0	1
Heithersay et al.[44]	4	10–20	4	0	4	0	0	4
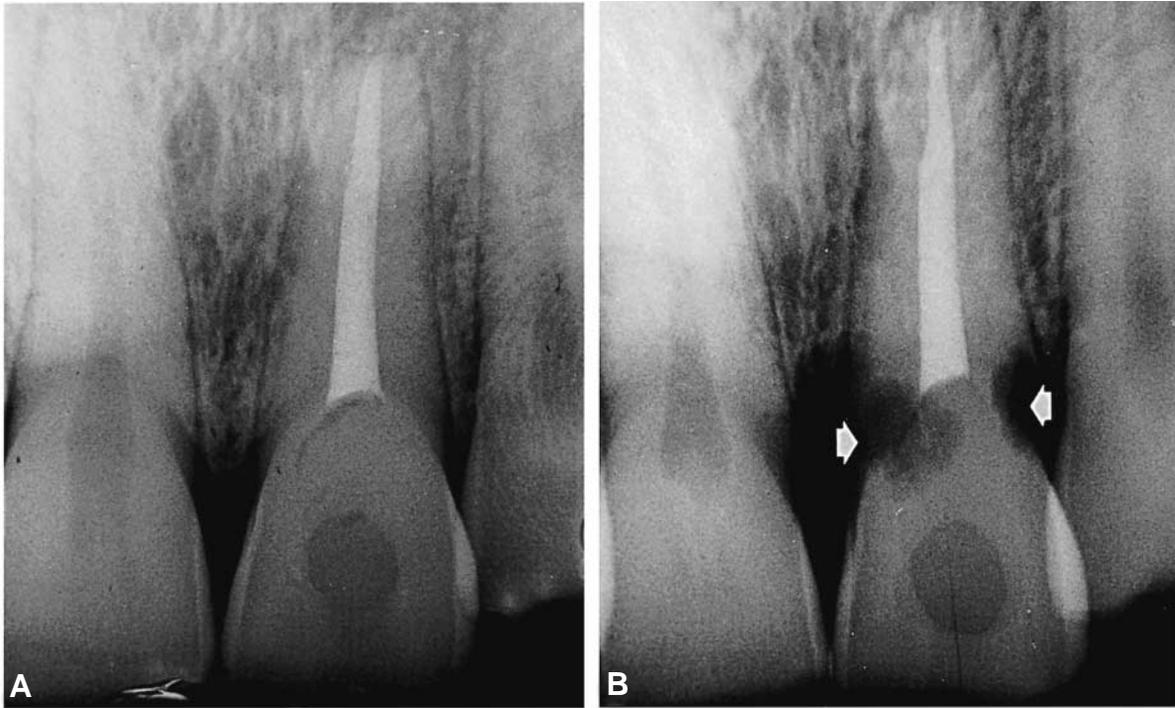
Figure 16-9   Postbleaching external root resorption. **A**, Nine-year recall radiograph of a central incisor devitalized by trauma and treated endodontically shortly thereafter. **B**, Radiograph taken 2 years following bleaching. Superoxol and heat were used first and followed by "walking bleach" with Superoxol and sodium perborate. (Courtesy of Drs. David Steiner and Gerald Harrington.)

the presence of bacteria.[38,49] Previous traumatic injury and age may act as predisposing factors.[34]

Chemical Burns.   Thirty percent hydrogen peroxide is caustic and causes chemical burns and sloughing of the gingiva. When using such solutions, the soft tissues should always be protected with *Vaseline* or *Orabase.*

Damage to Restorations.   Bleaching with hydrogen peroxide may affect bonding of composite resins to dental hard tissues.[50] Scanning electron microscopy suggests a possible interaction between composite resin and residual peroxide, causing inhibition of polymerization and an increase in resin porosity.[51] This presents a clinical problem when immediate esthetic restoration of the bleached tooth is required. It is therefore recommended that residual hydrogen peroxide be totally eliminated prior to composite placement.

It has been shown that immersion of peroxide-treated dental tissues in water at 37°C for 7 days prevents the reduction in bond strength.[52] In another study, the efficacy of **catalase** in removing residual hydrogen peroxide from the pulp chamber of human teeth, as compared to prolonged rinsing in water, was assessed.[53] Three minutes of catalase treatment effectively removed all of the residual hydrogen peroxide from the pulp chamber.

Suggestions for Safer Bleaching of Endodontically Treated Teeth

- **Isolate the tooth effectively.** Intracoronal bleaching should always be carried out with rubber dam isolation. Interproximal wedges and ligatures may also be used for better protection.
- **Protect the oral mucosa.** Protective creams, such as *Orabase* or *Vaseline,* must be applied to the surrounding oral mucosa to prevent chemical burns by caustic oxidizers. Animal studies suggest that catalase applied to oral tissues prior to hydrogen peroxide treatment totally prevents the associated tissue damage.[54]
- **Verify adequate endodontic obturation.** The quality of root canal obturation should always be assessed clinically and radiographically prior to bleaching. Adequate obturation ensures a better overall prognosis of the treated tooth. It also provides an additional barrier against damage by oxidizers to the periodontal ligament and periapical tissues.
- **Use protective barriers.** This is essential to prevent leakage of bleaching agents that may infiltrate between the gutta-percha and root canal walls, reaching the periodontal ligament via dentinal tubules, lateral canals, or the root apex. In none of the clinical reports of post-bleaching root resorption was a protective barrier used.

Various materials can be used for this purpose. Barrier thickness and its relationship to the cementoenamel junction are most important.[28,55] The ideal barrier should protect the dentinal tubules and conform to the external epithelial attachment (Figure 16-10).

- **Avoid acid etching.** It has been suggested that acid etching of dentin in the chamber to remove the smear layer and open the tubules would allow better penetration of the oxidizer. This procedure has not proven beneficial.[56] The use of caustic chemicals in the pulp chamber is undesirable as periodontal ligament irritation may result.

- **Avoid strong oxidizers.** Procedures and techniques applying strong oxidizers should be avoided if they are not essential for bleaching. Solutions of 30 to 35% hydrogen peroxide, either alone or in combination with other agents, should not be used routinely for intracoronal bleaching.

    Sodium perborate is mild and quite safe, and no additional protection of the soft tissues is usually required. Generally, however, oxidizing agents should not be exposed to more of the pulp space and dentin than absolutely necessary to obtain a satisfactory clinical result.

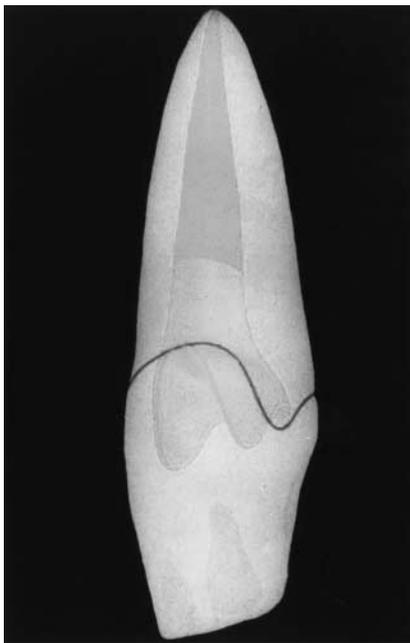- **Avoid heat.** Excessive heat may damage the cementum and periodontal ligament as well as dentin and enamel, especially when combined with strong oxidizers.[30,31] Although no direct correlation has been found between heat applications alone and external cervical root resorption, it should be limited during bleaching procedures.

- **Recall periodically.** Bleached teeth should be frequently examined both clinically and radiographically. Root resorption may occasionally be detected as early as 6 months after bleaching. Early detection improves the prognosis since corrective therapy may still be applied.

Post-Bleaching Tooth Restoration

Proper tooth restoration is essential for long-term successful bleaching results. Coronal microleakage of lingual access restorations is a problem,[57] and a leaky restoration may lead to rediscoloration.

There is no ideal method for filling the chamber after tooth bleaching. The pulp chamber and access cavity should be carefully restored with a light-cured acid-etched composite resin, light in shade. The composite material should be placed at a depth that seals the cavity and provides some incisal support. Light curing from the labial surface, rather than the lingual surface, is recommended since this results in shrinkage of the composite resin toward the axial walls, reducing the rate of microleakage.[58]

Placing white cement beneath the composite access restoration is recommended. Filling the chamber completely with composite may cause loss of translucency and difficulty in distinguishing between composite and tooth structure during rebleaching.[59]

As previously stated, residual peroxides from bleaching agents, mainly hydrogen peroxide and carbamide peroxide, may affect the bonding strength of composites.[25,50] Therefore, waiting for a few days after bleaching prior to restoring the tooth with composite resin has been recommended. Catalase treatment at the final visit may enhance the removal of residual peroxides from the access cavity; however, this requires further clinical investigation.[53]

Packing calcium hydroxide paste in the pulp chamber for a few weeks prior to placement of the final restoration, to counteract acidity caused by bleaching agents and to prevent root resorption, has also been suggested; this procedure, however, is unnecessary with walking bleach.[18]

VITAL BLEACHING TECHNIQUES

Many techniques have been advocated for extracoronal bleaching of vital teeth. In these techniques, oxidizers are applied to the external enamel surface of the teeth.

Figure 16-10 Schematic illustration of the intracoronal protective bleach barrier. The shape of the barrier matches the contour of the external epithelial attachment. Reproduced with permission from Steiner DR, and West JD.[28]

Thermo/Photo Bleaching

This technique basically involves application of 30 to 35% hydrogen peroxide and heat or a combination of heat and light or ultraviolet rays to the enamel surface (Table 16-4). Heat is applied either by electric heating devices or heat lamps. The technique involves the following steps:

1. Familiarize the patient with the probable causes of discoloration, procedure to be followed, expected outcome, and possibility of future rediscoloration.
2. Make radiographs to detect the presence of caries, defective restorations, and proximity to pulp horns. Well-sealed small restorations and minimal amounts of exposed incisal dentin are not usually a contraindication for bleaching.
3. Evaluate tooth color with a shade guide and take clinical photographs before and throughout the procedure.
4. Apply a protective cream to the surrounding gingival tissues and isolate the teeth with a rubber dam and waxed dental floss ligatures. If a heat lamp is used, avoid placing rubber dam metal clamps as they are subjected to heating and may be painful to the patient.
5. **Do not inject a local anesthetic.**
6. Position protective sunglasses over the patient's and the operator's eyes.
7. Clean the enamel surface with pumice and water. Avoid prophylaxis pastes containing glycerine or fluoride.
8. As an optional procedure, acid etch the darkest or most severely stained areas with buffered phosphoric acid for 10 seconds and rinse with water for 60 seconds. A gel form of acid provides optimum control. Enamel etching for extracoronal bleaching is controversial and should not be carried out routinely.
9. Place a small amount of 30 to 35% hydrogen peroxide solution into a dappen dish. Apply the hydrogen peroxide liquid on the labial surface of the teeth using a small cotton pellet or a piece of gauze. A bleaching gel containing hydrogen peroxide may be used instead of the aqueous solution (see Figure 16-4).
10. Apply heat with a heating device or a light source. The temperature should be at a level the patient can comfortably tolerate, usually between 125°F and 140°F (52°C to 60°C). Rewet the enamel surface with hydrogen peroxide as necessary. If the teeth become too sensitive, discontinue the bleaching procedure immediately. Do not exceed 30 minutes of treatment even if the result is not satisfactory.
11. Remove the heat source and allow the teeth to cool down for at least 5 minutes. Then wash with warm water for 1 minute and remove the rubber dam. Do not rinse with cold water since the sudden change in temperature may damage the pulp or can be painful to the patient.
12. Dry the teeth and gently polish them with a composite resin polishing cup. Treat all of the etched and bleached surfaces with a neutral sodium fluoride gel for 3 to 5 minutes.
13. Inform the patient that cold sensitivity is common, especially during the first 24 hours after treatment. Also, instruct the patient to use a fluoride rinse daily for 2 weeks.
14. Re-evaluate the patient approximately 2 weeks later on the effectiveness of bleaching. Take clinical photographs with the same shade guide used in the preoperative photographs for comparison purposes. If necessary, repeat the bleaching procedure.

Complications and Adverse Effects. *Postoperative Pain.* A number of short- and long-term symptoms may occur following extracoronal bleaching of vital teeth. A common immediate postoperative problem is pulpalgia characterized by intermittent shooting pain. It may occur during or after the bleaching session and usually persists for between 24 and 48 hours. The intensity of the pulpalgia is related to duration and the temperature of the bleaching procedure. Shorter bleaching periods are therefore recommended. If long-term sensitivity to cold develops, topical fluoride treatments and desensitizing toothpastes should be used to alleviate these symptoms.

Pulpal Damage. Extracoronal bleaching with hydrogen peroxide and heat has been associated with some pulpal damage. Although investigators have not found significant irreversible effects on the pulp,[10,60,61] these procedures must be approached and carried out with caution[62] and not in the presence of caries, areas of exposed dentin, or in close proximity to pulp horns.

Table 16-4   Indications and Contraindications for Thermo/Photo Extracoronal Bleaching

Indications	Contraindications
Light enamel discolorations	Severe dark discolorations
Mild tetracycline discolorations	Severe enamel loss
	Proximity of pulp horns
Endemic fluorosis discolorations	Hypersensitive teeth
	Presence of caries
Age-related discolorations	Large/poor coronal restorations

Defective restorations must be replaced prior to bleaching. Teeth with large coronal restorations should not be bleached.

Dental Hard Tissue Damage.   Hydrogen peroxide has been shown to cause morphologic and structural changes in enamel, dentin, and cementum in vitro.[20–23,63] A reduction in microhardness has been observed.[64] These changes may cause dental hard tissues to be more susceptible to degradation and to secondary caries formation.

Mucosal Damage.   Caustic bleaching agents in contact with the oral mucosa may cause peroxide-induced tissue damage.[54] Ulceration and sloughing of the mucosa are caused by oxygen gas bubbles in the tissue. Generally, the mucosa appears white but does not become necrotic or leave scar tissue. The associated burning sensation is extremely uncomfortable for the patient. Treatment is by extensive water rinses until the whiteness is reduced. In more severe cases, a topical anesthetic, limited movements, and good oral hygiene aid healing. Application of protective cream or catalase can prevent most of these complications.

Laser-Activated Bleaching

Recently, a technique has been introduced using lasers for extracoronal bleaching. Two types of lasers can be employed: the argon laser that emits a visible blue light and a carbon-dioxide laser that emits invisible infrared light. These lasers can be targeted to stain molecules and, with the use of a catalyst, rapidly decompose hydrogen peroxide to oxygen and water. The catalyst/peroxide combination may be damaging; therefore, exposed soft tissues, eyes, and clothing should be protected.

Combination of both lasers can effectively reduce intrinsic stains in the dentin. An argon laser can be targeted at stain molecules without overheating the pulp. It is easy to use and is best for removal of initial dark stains, such as those caused by tetracycline. However, visible blue light becomes less effective as the tooth whitens, and there are fewer stain molecules. On the other hand, the carbon-dioxide laser interacts directly with the catalyst/peroxide combination and removes the stain regardless of the tooth color.

Some techniques involve high-concentration hydrogen peroxide formulations as active ingredients (35 to 50%). It was reported that such laser bleaching techniques lightened teeth faster. However, short-term postoperative sensitivity can be profound.

Laser bleaching is a relatively new technique, and there are currently no long-term studies regarding its benefits or adverse effects.[65]

Mouthguard Bleaching

This technique has been widely advocated as a home bleaching technique, with a wide variety of materials, bleaching agents, frequency, and duration of treatment. Numerous products are available, mostly containing either 1.5 to 10% hydrogen peroxide or 10 to 15% carbamide peroxide, that degrade slowly to release hydrogen peroxide. The carbamide peroxide products are more commonly used. Higher concentrations of the active ingredient are also available and may reach up to 50% (Table 16-5).

Treatment techniques may vary according to manufacturers' instructions. The following step-by-step instructions should be used only as a general guideline. The procedure is as follows:

1. Familiarize the patient with the probable causes of discoloration, procedure to be followed, and expected outcome.
2. Carry out prophylaxis and assess tooth color with a shade guide. Take clinical photographs before and throughout the procedure.
3. Make an alginate impression of the arch to be treated. Cast the impression and outline the guard on the model. It should completely cover the teeth, although second molars need not be covered unless required for retention. Place two layers of die relief on the buccal aspects of the cast teeth to form a small reservoir for the bleaching agent. Fabricate a vacuum-form soft plastic matrix, approximately 2 mm thick, trim with crown and bridge scissors to 1 mm past the gingival margins, and adjust with an acrylic trimming bur.
4. Insert the mouthguard to ensure a proper fit. Remove the guard and apply the bleaching agent in the space of each tooth to be bleached. Reinsert the mouthguard over the teeth and remove excess bleaching agent.
5. Familiarize the patient with the use of the bleaching agent and wearing of the guard. The procedure is usually performed 3 to 4 hours a day, and the bleaching agent is replenished every 30 to 60 minutes. Some clinicians recommend wearing the guard during sleep for better long-term esthetic results.
6. Instruct patients to brush and rinse their teeth after meals. The guard should not be worn while eating. Inform the patient about thermal sensitivity and minor irritation of soft tissues and to discontinue use of the guard if uncomfortable.
7. Treatment should be for between 4 and 24 weeks. Recall the patient every 2 weeks to monitor stain lightening. Check for tissue irritation, oral lesions,

Table 16-5   Common Commercial Brands of Home-Use Bleaching Products

Brand	Company	Active Ingredient	Concentrations Available, %
BriteSmile	BriteSmile	10% HP	6, 8, 10
Contrast P.M.	Interdent	10% CP	10, 15, 20
Denta-Lite	Challenge	10% CP	10
Karisma	Confi-Dental	10% CP	10
Natural White	Aesthete	6% HP	6
Nite White	Discus Dental	10% CP	5, 10, 16, 22
Nupro Gold	Dentsply/Ash	10% CP	10
NuSmile	M&M	15% CP	15
Opalescence	Ultradent	10% CP	10, 35
Perfecta	Am Dent Hygienics	11% CP	11, 13, 16
Rembrandt	Den-Mat	10% CP	10, 15, 22
Spring White	Spring Health	10% CP	10
White & Brite	Omnii	10% CP	10, 16
Platinum	Colgate	10% CP	10
Zaris System	3-M	10% CP	10, 16

HP = hydrogen peroxide; CP = carbamide peroxide.

enamel etching, and leaky restorations. If complications occur, stop treatment and re-evaluate the feasibility of continuation at a later date. Note that frequently the incisal edges are bleached more readily than the remainder of the crown (Table 16-6).

The long-term esthetic results of this method are unknown. However, it appears that rediscoloration is not more frequent than with the other techniques. To date, no conclusive experimental or clinical studies on the safety of long-term use of these bleaching agents are available. Therefore, caution should be exercised in their prescription and application.[66] Of major concern

Table 16-6   Indications and Contraindications for Mouthguard Vital Bleaching

Indications	Contraindications
Superficial enamel discolorations	Severe enamel loss
Mild yellow discolorations	Hypersensitive teeth
	Presence of caries
Brown fluorosis discolorations	Defective coronal restorations
Age-related discolorations	Allergy to bleaching gels
	Bruxism

is the products marketed to the public over the counter, often without professional control. Their use should be discouraged.

Complications and Adverse Effects. *Systemic Effects.* Controlled mouthguard bleaching procedures are considered relatively safe.[19] However, some concern has been raised over bleaching gels inadvertently swallowed by the patient. Accidental ingestion of large amounts of these gels may be toxic and cause irritation to the gastric and respiratory mucosa.[67] Bleaching gels containing carbopol, which retards the rate of oxygen release from peroxide, are usually more toxic. Therefore, it is advisable to pay specific attention to any adverse systemic effects and to discontinue treatment immediately if they occur.

Dental Hard Tissue Damage. *In vitro* studies indicate morphologic and chemical changes in enamel, dentin, and cementum associated with some agents used for **mouthguard** bleaching.[20–23] Long-term in vivo studies are still required to determine the clinical significance of these changes.

Tooth Sensitivity. Transient tooth sensitivity to cold may occur during or after mouthguard bleaching. In most cases, it is mild and ceases on termination of treatment. Treatment for sensitivity consists of removal of the mouthguard for 2 to 3 days, reduction of wearing time, and re-adjustment of the guard.

Pulpal Damage.   Long-term effects of mouthguard bleaching on the pulp are still unknown. To date, no correlation has been found between carbamide peroxide bleaching and permanent pulpal damage.[19] The pulpalgia associated with tooth hypersensitivity is usually transient and uneventful.

Mucosal Damage.   Minor irritations or ulcerations of the oral mucosa have been reported to occur during the initial course of treatment. This infrequent occurrence is usually mild and transient. Possible causes are mechanical interference by the mouthguard, chemical irritation by the bleaching active agent, and allergic reaction to gel components. In most cases, readjustment and smoothing the borders of the guard will suffice. However, if tissue irritation persists, treatment should be discontinued.

Damage to Restorations.   Some *in vitro* studies suggest that damage of bleaching gels to composite resins might be caused by softening and cracking of the resin matrix.[24,25] It has been suggested that patients are informed that previously placed composites may require replacement following bleaching. Others have reported no significant adverse effects on either surface texture or color of restorations.[19] Generally, however, if composite restorations are present in esthetically critical areas, they may need replacement to improve color matching following successful bleaching.

It has also been reported that both 10% carbamide peroxide and 10% hydrogen peroxide may enhance the liberation of mercury and silver from amalgam restorations and may increase exposure of patients to toxic by-products.[68] Although bleaching gels are mainly applied to the anterior dentition, excessive gel may inadvertently make contact with posterior teeth. Coverage of posterior amalgam restorations with a protective layer of dental varnish prior to gel application may prevent such hazards.

Occlusal Disturbances.   Typically, occlusal problems related to the mouthguard may be mechanical or physiologic. From a mechanical point of view, the patient may occlude only on the posterior teeth rather than on all teeth simultaneously. Removing posterior teeth from the guard until all of the teeth are in contact rectifies this problem. From a physiologic point of view, if the patient experiences temporomandibular joint pain, the posterior teeth can be removed from the guard until only the anterior guidance remains. In such cases, wearing time should be reduced.

For further information on extracoronal, home-bleaching methods, the reader is referred to the writings of Haywood and colleagues.[69–74]

REFERENCES

1. Walton RE, Rotstein I. Bleaching discolored teeth: Internal and external. In: Walton RE, Torabinejad M, editors. Principles and practice of endodontics. 2nd ed. Philadelphia: WB Saunders; 1996. p. 385.
2. Rotstein I. Bleaching nonvital and vital discolored teeth. In: Cohen S, Burns RC, editors. Pathways of the pulp. 7th ed St. Louis: CV Mosby; 1998. p. 674.
3. Freccia WF, et al. An in vitro comparison of non-vital bleaching techniques in the discolored tooth. JOE 1982;8:70.
4. Rotstein I, et al. *In vitro* efficacy of sodium perborate preparations used for intracoronal bleaching of discolored non-vital teeth. Endod Dent Traumatol 1991;*7*:177.
5. Rotstein I, Mor C, Friedman S. Prognosis of intracoronal bleaching with sodium perborate preparations *in vitro*: 1 year study. JOE 1993;19:10.
6. Atasu M, Genc A, Ercalik S. Enamel hypoplasia and essential staining of teeth from erythroblastosis fetalis. J Clin Pediatr Dent 1998;22:249.
7. Lochary ME, Lockhart PB, Williams WT Jr. Doxycycline and staining of permanent teeth. Pediatr Infect Dis J 1998;17:429.
8. Livingston HM, Dellinger TM. Intrinsic staining of teeth secondary to tetracycline. Ann Pharmacother 1998;32:607.
9. Jordan RE, Boskman L. Conservative vital bleaching treatment of discolored dentition. Compend Contin Educ Dent 1984;5:803.
10. Arens DE, Rich JJ, Healey HJ. A practical method of bleaching tetracycline-stained teeth. Oral Surg 1972;34:812.
11. Cohen S, Parkins FM. Bleaching tetracycline-stained vital teeth. Oral Surg 1970;29:465.
12. Abou-Rass M. The elimination of tetracycline discoloration by intentional endodontics and internal bleaching. JOE 1982;8:101.
13. Lake F, O'Dell N, Walton R. The effect of internal bleaching on tetracycline in dentin. JOE 1985;11:415.
14. Walton RE, O'Dell NL, Lake FT. Internal bleaching of tetracycline stained teeth in dogs. JOE 1983;9:416.
15. Driscoll WS, et al. Prevalence of dental caries and dental fluorosis in areas with optimal and above-optimal water fluoride concentrations. J Am Dent Assoc 1983;107:42.
16. Van der Burgt TP, Plasschaert AJM. Bleaching of tooth discoloration caused by endodontic sealers. JOE 1986;12:231.
17. Weiger R, Kuhn A, Löst C. *In vitro* comparison of various types of sodium perborate used for intracoronal bleaching. JOE 1994;20:338.
18. Rotstein I, Friedman S. pH variation among materials used for intracoronal bleaching. JOE 1991;17:376.
19. Haywood VB, Heymann HO. Nightguard vital bleaching: how safe is it? Quintessence Int 1991;22:515.
20. Rotstein I, et al. Histochemical analysis of dental hard tissues following bleaching. JOE 1996;22:23.
21. Seghi RR, Denry I. Effects of external bleaching on indentation and abrasion characteristics of human enamel *in vitro*. J Dent Res 1992;71:1340.
22. Zalkind M, Arwaz JR, Goldman A, Rotstein I. Surface morphology changes in human enamel, dentin and cementum following bleaching: a scanning electron microscopy study. Endod Dent Traumatol 1996;12:82.
23. Bitter NC. A scanning electron microscope study of the long-term effect of bleaching agents on the enamel surface *in vivo*. Gen Dent 1998;46:84.

24. Crim GA. Post-operative bleaching: effect on microleakage. Am J Dent 1992;5:109.

25. Titley KC, Torneck CD, Ruse ND. The effect of carbamide-peroxide gel on the shear bond strength of a microfil resin to bovine enamel. J Dent Res 1992;71:20.

26. Spasser HF. A simple bleaching technique using sodium perborate. N Y State Dent J 1961;27:332.

27. Nutting EB, Poe GS. A new combination for bleaching teeth. J South Calif Dent Assoc 1963;31:289.

28. Steiner DR, West JD. A method to determine the location and shape of an intracoronal bleach barrier. JOE 1994;20:304.

29. Holmstrup G, Palm AM, Lambjerg-Hansen H. Bleaching of discoloured root-filled teeth. Endod Dent Traumatol 1988;4:197.

30. Madison S, Walton RE. Cervical root resorption following bleaching of endodontically treated teeth. JOE 1990;16:570.

31. Rotstein I, et al. Histological characterization of bleaching-induced external root resorption in dogs. JOE 1991;17:436.

32. Lin LC, Pitts DL, Burgess LW. An investigation into the feasibility of photobleaching tetracycline-stained teeth. JOE 1988;14:293.

33. Anitua E, Zabalegui B, Gil J, Gascon F. Internal bleaching of severe tetracycline discolorations: four-year clinical evaluation. Quintessence Int 1990;21:783.

34. Harrington GW, Natkin E. External resorption associated with bleaching of pulpless teeth. JOE 1979;5:344.

35. Lado EA, Stanley HR, Weisman MI. Cervical resorption in bleached teeth. Oral Surg 1983;55:78.

36. Montgomery S. External cervical resorption after bleaching a pulpless tooth. Oral Surg 1984;57:203.

37. Shearer GJ. External resorption associated with bleaching of a non-vital tooth. Aust Endod Newslett 1984;10:16.

38. Cvek M, Lindvall AM. External root resorption following bleaching of pulpless teeth with oxygen peroxide. Endod Dent Traumatol 1985;1:56.

39. Latcham NL. Postbleaching cervical resorption. JOE 1986;12:262.

40. Goon WWY, Cohen S, Borer RF. External cervical root resorption following bleaching. JOE 1986;12:414.

41. Friedman S, et al. Incidence of external root resorption and esthetic results in 58 bleached pulpless teeth. Endod Dent Traumatol 1988;4:23.

42. Gimlin DR, Schindler WG. The management of postbleaching cervical resorption. JOE 1990;16:292.

43. Al-Nazhan S. External root resorption after bleaching: a case report. Oral Surg 1991;72:607.

44. Heithersay GS, Dahlstrom SW, Marin PD. Incidence of invasive cervical resorption in bleached root-filled teeth. Aust Dent J 1994;39:82.

45. Heller D, Skriber J, Lin LM. Effect of intracoronal bleaching on external cervical root resorption. JOE 1992;18:145.

46. Rotstein I, Torek Y, Misgav R. Effect of cementum defects on radicular penetration of 30% H_2O_2 during intracoronal bleaching. JOE 1991;17:230.

47. Koulaouzidou E, et al. Role of cementoenamel junction on the radicular penetration of 30% hydrogen peroxide during intracoronal bleaching in vitro. Endod Dent Traumatol 1996;12:146.

48. Rotstein I, Torek Y, Lewinstein I. Effect of bleaching time and temperature on the radicular penetration of hydrogen peroxide. Endod Dent Traumatol 1991;7:196.

49. Heling I, Parson A, Rotstein I. Effect of bleaching agents on dentin permeability to Streptococcus faecalis. JOE 1995;21:540.

50. Titley KC, Torneck CD, Ruse ND, Krmec D. Adhesion of a resin composite to bleached and unbleached human enamel. JOE 1993;19:112.

51. Titley KC, et al. Scanning electron microscopy observations on the penetration and structure of resin tags in bleached and unbleached bovine enamel. JOE 1991;17:71.

52. Torneck CD, Titley KC, Smith DC, Adibfar A. Effect of water leaching on the adhesion of composite resin to bleached and unbleached bovine enamel. JOE 1991;17:156.

53. Rotstein I. Role of catalase in the elimination of residual hydrogen peroxide following tooth bleaching. JOE 1993;19:567.

54. Rotstein I, Wesselink PR, Bab I. Catalase protection against hydrogen peroxide-induced injury in rat oral mucosa. Oral Surg 1993;75:744.

55. Rotstein I, Zyskind D, Lewinstein I, Bamberger N. Effect of different protective base materials on hydrogen peroxide leakage during intracoronal bleaching in vitro. JOE 1992;18:114.

56. Casey LJ, Schindler WG, Murata SM, Burgess JO. The use of dentinal etching with endodontic bleaching procedures. JOE 1989;15:535.

57. Wilcox LR, Diaz-Arnold A. Coronal microleakage of permanent lingual access restorations in endodontically treated anterior teeth. JOE 1989;15:584.

58. Lemon R. Bleaching and restoring endodontcally treated teeth. Curr Opin Dent 1991;1:754.

59. Freccia WF, Peters DD, Lorton L. An evaluation of various permanent restorative materials' effect on the shade of bleached teeth. JOE 1982;8:265.

60. Cohen SC. Human pulpal response to bleaching procedures on vital teeth. JOE 1979;5:134.

61. Robertson WD, Melfi RC. Pulpal response to vital bleaching procedures. JOE 1980;6:645.

62. Nathanson D. Vital tooth bleaching: sensitivity and pulpal considerations. J Am Dent Assoc 1998;128 Suppl:41S.

63. Rotstein I, Lehr T, Gedalia I. Effect of bleaching agents on inorganic components of human dentin and cementum. JOE 1992;18:290.

64. Lewinstein I, Hirschfeld Z, Stabholz A, Rotstein I. Effect of hydrogen peroxide and sodium perborate on the microhardness of human enamel and dentin. JOE 1994;20:61.

65. Garber DA. Dentist-monitored bleaching: a discussion of combination and laser bleaching. J Am Dent Assoc 1997;128 Suppl:26S.

66. Li Y. Toxicological considerations of tooth bleaching using peroxide-containing agents. J Am Dent Assoc 1997;128 Suppl:31S.

67. Redmond AF, Cherry DV, Bowers DE Jr. Acute illness and recovery in adult female rats following ingestion of a tooth whitener containing 6% hydrogen peroxide. Am J Dent 1997;10:268.

68. Rotstein I, Mor C, Arwaz JR. Changes in surface levels of mercury, silver, tin and copper of dental amalgam treated with carbamide peroxide and hydrogen peroxide. Oral Surg 1997;83:506.

69. Haywood VB, Heymann HO. Response of normal and tetracycline-stained teeth with pulp-size variation to nightguard vital bleaching. J Esthet Dent 1994;6:109.

70. Haywood VB. Nightguard vital bleaching: current concepts and research. J Am Dent Assoc 1997;128 Suppl:19S.

71. Haywood VB. Bleaching vital teeth: current concepts. Quintessence Int 1997;28:424.

72. Leonard RH, Haywood VB, Phillips C. Risk factors for developing tooth sensitivity and gingival irritation in nightguard vital bleaching. Quintessence Int 1997;28:527.

73. Haywood VB. Extended bleaching of tetracycline stained teeth: a case report. Contemp Esthet Restor Pract 1997;1:14.

74. Haywood VB. Critical appraisal: at home bleaching. J Esthet Dent 1998;10:94.