Tooth whitening: concepts and controversies

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Abstract

Today's society dictates that it is the norm for people to have straight, white teeth. The demand therefore for tooth whitening in dental practice has increased exponentially over the last decade. A common approach to achieving this goal is by bleaching. This article discusses clinical aspects of dental bleaching by providing an evidence-based review of current literature. Topics covered include aetiology of tooth discolouration, indications for bleaching, its mode of action, and different types of bleaching regimes, indications and potential side effects.

Introduction

The cosmetic impairment of tooth discolouration, especially in the anterior region, can be treated by a number of invasive therapies such as indirect crowns and veneers, microabrasion, or by the placement of direct composite. In certain clinical situations, the procedure of tooth whitening or bleaching can be employed as a less invasive alternative to restoration with either ceramic or composite. Bleaching of teeth can be achieved either by an external – or vital – approach (nightguard vital bleaching) (Heywood 1991), where vital teeth are bleached by direct contact with an agent such as carbamine peroxide, or by an internal – or non-vital – approach, where non-vital teeth are bleached with an agent such as sodium perborate in a walking bleach technique (Attin et al 2003). A third approach, which is a modification of both techniques, can be employed when bleaching vital and non-vital teeth in the same arch. This is called inside/outside bleaching (Settembrini et al 1997). The aim of this review is to discuss the concepts involved in both the vital and non-vital bleaching of teeth, and to provide advice, based on the evidence from current literature, to reduce the risks of complications and to ensure successful bleaching therapy.

Causes of tooth discolouration

Tooth discolouration may be described as intrinsic, extrinsic or a combination of both (Hattab et al 1999). It varies in appearance, aetiology, severity, localisation and adherence to tooth structure (Dahl and Pallesen 2003). The causes of intrinsic tooth discolouration can be attributed to changes to the structure of dentine or enamel (Figure 1), or by incorporation of chromatogenic material into tooth tissue, either during odontogenesis or post eruption. The main cellular changes observed in intrinsically stained teeth often provide a clue to the aetiology of the pathologic process involved. Discolouration can manifest as either a red, brown, grey or yellow appearance. Internal pulp bleeding caused by trauma or pulp extirpation can cause a temporary red colour change to the crown.

Then, as blood degenerates and breaks down, products such as haemosiderin, haemin, haematin and haematoidin release iron (Dahl and Pallesen 2003). The iron can be converted into black ferric sulphide with hydrogen sulphide produced by bacteria, which causes a grey staining of the tooth. In addition to blood degradation, degrading proteins of necrotic pulp tissue may also cause discolouration. If pulp tissue is not completely extirpated and remains in the pulp horns, discolouration may result from the break up of the proteins of the necrotic pulp tissue (Guldener and Langeland 1993), causing a grey or brown hue to the crown (Figure 2). Yellow discolouration is often due to the reactionary laying down of tertiary dentine sclerosing the root canal and pulp chamber. Because enamel is relatively translucent, the additional volume of dentine obliterating the pulp chamber produces a yellow hue to the crown (Figure 3) (Faunce 1983). Intrinsic discolouration is also caused by exposure to high levels of fluoride, tetracycline administration during childhood, inherited developmental disorders, jaundice in childhood, porphyria, caries, restorations and trauma to the developing tooth germ. After eruption, ageing, pulp necrosis

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Figure 1: Intrinsic tooth colour change due to tetracycline staining.

Figure 2: Brown/grey appearance of a nonvital central incisor.

Figure 3: Yellow intrinsic discolouration of the upper right central incisor due to sclerosis of the pulp chamber.

Figure 4: Yellow discolouration of the maxillary anterior dentition due to extrinsic agents such as food colouring and tobacco use.

and iatrogenesis are the main causes of intrinsic discolouration (Olgart and Bergenholtz 2003). Extrinsic staining results mainly from dietary factors and smoking (Figure 4). Foods containing tannins such as red wine, coffee and tea can give rise to extrinsic stain. Carotenes in oranges and carrots, and tobacco use, whether it is smoking or chewing, also give rise to extrinsic stain (Watts and Addy 2001). Wear of tooth structure, deposition of secondary dentine due to ageing or as a consequence of pulp inflammation, and dentine sclerosis affect the light-transmitting properties of enamel and dentine, resulting in a gradual discolouration. For example, tetracycline staining is persistent, whereas discolouration of ageing responds quickly in most instances (Heywood 1995).

History

The first publications describing techniques and chemicals for bleaching non-vital teeth appeared in the latter half of the 19th Century. The bleaching agent of choice was chloride of lime (Dwinelle 1850). Other agents described for the bleaching of pulpless teeth included aluminium chloride and hydrogen peroxide, used either alone or in combination with heat. The active ingredient common to all the early medicaments was an oxidising agent, which acted either directly or indirectly with the organic component of the tooth. Concern about the side effects of some of these agents was justified however, because some chemicals used were very poisonous, such as cyanide of potassium (Barker 1861). The walking bleach technique that was introduced in 1961 involved placement of a mixture of sodium perborate and water into the pulp chamber, which was sealed into place between dental visits (Spasser 1961). This method was later modified by replacing water with 30-35% hydrogen peroxide to improve the whitening effect (Nutting 1963). Although most of the early publications described non-vital bleaching, a 3% solution of Pyrozone was used safely as a mouthwash as early as 1890, which not only

Figure 5: A white silicone barrier material is used to protect the gingival tissues during power bleaching.

reduced caries, but also whitened teeth (Atkinson 1893). The observation that carbamine peroxide caused lightening of teeth was made in the late 1960s by an orthodontist (Klusmier), who had prescribed an antiseptic containing 10% carbamine peroxide to be used in a tray for the treatment of gingivitis. This technique, which is the method of home bleaching today, was not widely accepted by the dental profession until 20 years later when it was described in a 1989 publication (Haywood and Heymann 1989).

Mechanism

Hydrogen peroxide is a colourless liquid with a bitter taste and is highly soluble in water to give an acid solution. It has a wide number of industrial applications, for example bleaching or deodorising textiles, wood pulp, fur and hair, and in the treatment of water and sewage. Hydrogen peroxide is a reactive oxygen species and acts as a strong oxidising agent through the formation of free radicals Tredwin et al 2006), which attack the organic molecules responsible for tooth discolouration. When complex, pigmented organic molecules (chromaphores) are broken down by the action of free radicals, simpler molecules are produced, which reflect less light (Frysh 1995). During tooth bleaching, more highly pigmented carbon ring compounds are converted to carbon chains, which are lighter in colour. The carbon double bond chains (yellow in colour) are converted into hydroxyl groups, which are essentially colourless. The radicals also reduce coloured metallic oxides like Fe2O3 (Fe3+) to colourless FeO (Fe2+). The bleaching process continues until all of the original pigment is rendered colourless (Albers 1991). The chemistry of carbamine peroxide, used for nightguard vital bleaching, is slightly different from hydrogen peroxide as it also contains urea, which permits the peroxide to remain in contact with the tooth for longer. Although the action of carbamine peroxide also causes the breakdown of pigmented carbon compounds as described above, the degradation is slower than with hydrogen peroxide alone.

External (vital) bleaching

The bleaching of vital teeth can occur inside the surgery (power bleaching) or outside the surgery (nightguard vital bleaching). Power bleaching accomplishes complete lightening during treatment in the surgery, whereas nightguard vital bleaching involves the application of a peroxide gel to the tooth surface via some means of carrier, usually a custom fitting bleaching tray.

Power bleaching

Power bleaching of vital teeth generally uses a high concentration of peroxide solution (35- 50% hydrogen peroxide) placed directly on the teeth, often supplemented by a heat or light source to activate or enhance peroxide release (Feinman et al 1987). Because the hydrogen peroxide concentration is so high, soft tissues must be very well protected to prevent injury (Figure 5). Definite indications for its use include treatment of generalised gross staining such as tetracycline staining and perhaps dentine sclerosis, which take a long time using the nightguard vital bleaching technique, and for patients who may have difficulty in compliance with the nightguard vital bleaching technique.

Power bleaching has several potential disadvantages:

1. Neither the patient nor the dentist can exactly control the amount of lightening (compared to the nightguard vital bleaching technique). The technique runs the risk of both over- and under-bleaching.
2. The fee is usually higher as a greater amount of chair time is required.
3. There is a possibility of soft tissue damage due to the caustic nature of the high concentrations of peroxide.
4. There is a greater risk of post-operative sensitivity (Goldstein 1988). A higher incidence of tooth sensitivity (67-78%) was reported after power bleaching (Heywood and Berry 2001, Cohen and Chase 1979) compared with the nightguard vital bleaching method, using 10% carbamine peroxide (15-65%) (Nathanson and Parra 1987, Heywood 1996, Leonard 1998, Schulte et al 1994).

Nightguard vital bleaching

Nightguard vital bleaching, or 'take home' bleaching, is the more commonly used bleaching technique because it is easy to perform and is generally less expensive for the

Figure 6: Take-home bleaching tray, extended as far as the gingival margin.

Figure 7: Take-home bleaching tray in situ.

patient. It involves the use of a 10-20% solution of carbamine peroxide in a gel form (approximately equal to 3.4-7% hydrogen peroxide) delivered to the tooth surface by a custom-made, vacuum formed, plastic bleaching tray (Figures 6 and 7). Manufacturers have offered carbamine peroxide in a variety of different concentrations, ranging from 10% to over 20%, but the best combination of safety, limited side effects and speed of action is obtained with a 10% solution of carbamine peroxide approved by the ADA (American Dental Association). Products carrying the ADA accepted label have passed a rigorous set of safety and efficacy standards (Tam 1999). A survey by Christensen (1989) indicated that 90% of dentists surveyed used a 10% concentration of carbamine peroxide for take home bleaching (Christensen 1991). Although the evidence base in the dental literature on the efficacy of nightguard vital bleaching is mostly limited to case reports, it is generally advocated that most teeth are susceptible to bleaching (Tam 1999). The process requires longer contact time compared to power bleaching, but it is safe and the results are generally excellent (Figure 2). The first subjective change in tooth colour is generally observed after two to four sessions of bleaching. In a clinical study of nightguard vital bleaching with 10% carbamine peroxide, 92% of subjects experienced some lightening of teeth after a six-week period (Haywood et al 1994). Another clinical trial by Swift et al (1999) examined the efficacy of 10% carbamine peroxide nightly for two weeks. They reported that the lightness of the crown of the tooth increased by, on average, eight shade units on the Vita¨ shade guide, calibrated according to a lightness value.

Internal (non-vital) bleaching

The whitening of endodontically treated teeth can be carried out by an internal whitening treatment known as non-vital bleaching or the 'walking bleach technique'. This therapy involves placement of a bleaching agent into the empty pulp chamber of a non-vital, discoloured tooth, and is a more conservative option compared to restoration with veneers or crowns. The two most common bleaching agents used for this technique are hydrogen peroxide and sodium perborate, and various sources have been applied to speed up the reaction and improve the bleaching effect. The decomposition of hydrogen peroxide into active oxygen is accelerated by application of heat or light (Howell 1980). The thermocatalytic breakdown of hydrogen peroxide was proposed for many years as the best technique for the whitening of non-vital, discoloured teeth because of the high reactivity of hydrogen peroxide upon application of heat (Hardman et al 1985). In this procedure, heat from a special lamp or hot instrument was applied to a well of 30-35% hydrogen peroxide in an empty pulp chamber. Temporary restorations impregnated with 30-35% hydrogen peroxide were often used between visits. Although there is little doubt regarding the clinical efficacy of non-vital bleaching using 30-35% hydrogen peroxide (Chen et al 1993) (either thermoactivated or not), serious concerns regarding the safety of this technique, in particular the risk of producing external cervical root resorption, which is discussed later, have rendered this technique unadvisable, and the application of sodium perborate instead of hydrogen peroxide is now recommended. Sodium perborate is a hydrogen peroxide releasing agent, and since 1907 it has been employed as an oxidiser and bleaching agent, especially in washing powders and other detergents. It comes in powder form and can be mixed into a paste or putty with either pure water or hydrogen peroxide. Several studies have reported bleaching effectiveness by comparing mixtures of sodium perborate with distilled water or hydrogen peroxide in

Figure 8: The distance between the CEJ and the incisal edge is measured with a periodontal probe on the facial.

Figure 9: Having recorded the measurement between the CEJ and the incisal edge, the periodontal probe now assists in accurate.

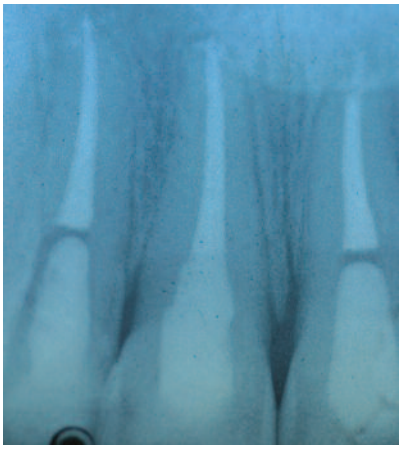

Figure 10: Good quality root canal treatments, showing thorough obturation and access cavities prepared for internal bleaching.

different concentrations. Rotstein et al (1991, 1993) and Weiger et al (1994) did not report any significant difference in effectiveness between sodium perborate mixed with 3-30% hydrogen peroxide, and distilled water, except for the time taken to achieve a clinically acceptable result. However, mixing sodium perborate with hydrogen peroxide was shown to accelerate the rate of colour change. In the case of severe discolouration, it is safe to mix sodium perborate with a 3% solution of hydrogen peroxide; however it is not appropriate to use 30% hydrogen peroxide because of the possible risk of inducing cervical root resorption (Friedman et al 1988). This is discussed in more detail below.

Clinical stages for internal bleaching

1. Radiographic examination A recent pre-operative radiograph is necessary prior to treatment to assess the quality of the root canal treatment. The root canal should be thoroughly condensed along its whole length to prevent the apico-coronal migration of micro-organisms or bleaching agents, which may have a detrimental effect on the surrounding tissues. Should the quality of the root canal treatment be suboptimal, the tooth should undergo corrective endodontic therapy prior to the commencement of bleaching (Figure 8).

2. Preparation of the access cavity - The pulp space should be completely debrided of any necrotic material, pulp tissues, or restorative or root canal materials. The smear layer on the dentinal surface of the pulp chamber is removed by applying 37% phosphoric acid gel and irrigated with 2.5-5% sodium hypochlorite.

3. Cervical seal - Gutta-percha (GP) is removed with a round ended, long shank bur to a level of 1-2mm below the CEJ (cementoenamel junction). It is helpful to measure this distance pre-operatively by recording the distance from the incisal tip to the CEJ on the facial aspect with a graduated probe (Figures 9 and 10). The coronal access is then sealed with a glass ionomer cement (GIC) or accelerated zinc oxide (ZOE) plug to prevent the diffusion of bleaching agents from the pulp chamber throughout the root filling, as root fillings do not provide an effective barrier on their own (Figure 11) (Attin et al 2003). Rotstein et al (1992) demonstrated that a 2mm layer of GIC or composite is essential. Alternatively, Bergenholtz et al (1982) showed histologically that ZOE cement also provides a hermetic seal.

4. Application of bleaching agent - A small drop of distilled water is mixed with sodium perborate powder (Amosan¨ Oral-B) until a putty consistency is achieved (Figure 12). The sodium perborate putty is applied to the empty pulp chamber with an amalgam plugger or similar instrument, covered with cotton pellet and sealed with an adhesive provisional restoration. It is often difficult to place the provisional restoration directly over the cotton pellet without displacing it. To immobilise the pellet, it is helpful to first wet the pellet with a bonding agent and then light cure the bond once the pellet is in place. A provisional restoration must then be placed, as a sound seal is required around the access cavity to prevent leakage of the bleaching agent into the oral cavity. A light cured GIC or an accelerated ZOE material can be employed for this purpose. This procedure is repeated every three to four days until successful bleaching becomes apparent. This normally occurs after one to four visits (Figure 13).

5. Permanent restoration - Once the desired colour change has been achieved, a sound restoration with sealed dentinal tubules is a prerequisite to a successful bleaching therapy (Abou Raas 1998). The access cavity should be restored with a composite, which is adhesively attached to

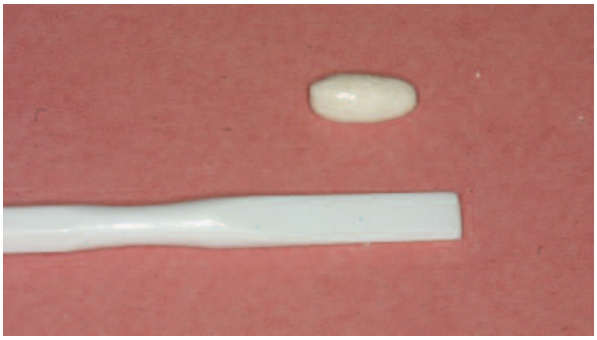

Figure 11: Sodium perborate (Amosan ®, Oral-B) mixed with distilled water to a puttylike consistency.

Figure 12: The pre-operative appearance of the maxillary right lateral and central incisors.

both enamel and dentine. It is recommended to choose a composite with a high value (light colour) to help compensate if the bleaching therapy alone does not provide the full extent of desired lightness. The timing of placement of the final restoration is also important, as it has been shown that the bond strengths of composite to bleached enamel and dentine is temporarily reduced. It is recommended to wait for at least seven days post bleaching prior to bonding composite as a definitive restoration (Nathanson and Parra 1987).

Inside/outside bleaching

Another bleaching technique has been described for clinical situations where an endodontically treated tooth is present within the arch and the arch as a whole is to be bleached. This technique, called 'inside/outside bleaching' allows the endodontically treated tooth to be bleached both from within the sealed pulp chamber (inside) and from the facial enamel (outside) simultaneously. The technique for inside/outside bleaching involves the fabrication of a vacuum-processed plastic mouthguard, trimmed to the facial and lingual margins as previously described for nightguard vital bleaching. Coronal access to the endodontically treated tooth (or teeth) is achieved and the coronal GP is sealed with a light cured GIC or accelerated ZOE, as previously described for non-vital bleaching. The patient is instructed how to inject 10% carbamine peroxide gel into the coronal orifice and into the nightguard. The bleach tray is worn for a minimum of two hours, up to a maximum of an overnight period, as described above. The patient is then instructed to insert a cotton wool plug into the coronal access to prevent the ingress of food particles. Once the non-vital tooth has been bleached to an acceptable match with the adjacent teeth, coronal access can be definitively restored with a high-value shade composite resin, and further nightguard vital bleaching can be continued if desired (Settembrini et al 1997).

Controversies

Tooth sensitivity

Unfortunately the aetiology of bleaching-related tooth sensitivity is neither well understood nor easily measured; however the hydrodynamic theory is a mechanism frequently cited to explain it (Brannstrom 1986). According to this model, peroxide solutions introduced into the oral environment contact available dentinal surfaces and cause retraction of odontoblastic processes, resulting in rapid fluid movement inside the dentinal tubules. This ultimately manifests in stimulation of mechanoreceptors at the pulp periphery, with the resultant feeling of pain when such teeth are exposed to cold or pressure, or even when they are at rest. Tooth sensitivity, if present, normally persists for up to four days after the cessation of bleaching (Frysh et al 1993, Jacobsen and Bruce 2001, Blong et al 1985).

Patient selection must be carefully considered prior to prescribing bleaching, as some patients are more susceptible to tooth sensitivity than others. In particular, it is wise not to recommend bleaching for patients with generalised gingival recession (Figure 14). Age may also have an effect on tooth sensitivity as the dentinal tubules in younger dentine are wider and enamel is more porous. Also the presence of old, leaking restorations provides a more rapid portal of entry into the pulp for irritating chemicals. Several agent-related factors can also affect tooth sensitivity. Increasing the concentration of peroxide provides a more rapid bleaching effect; however it also increases the risk of tooth sensitivity. When prescribing a bleaching regime, it is important to differentiate between the concentrations of hydrogen peroxide and carbamine peroxide. A 10% solution of carbamine peroxide is approximately 3% hydrogen peroxide and 7% urea. Concentrations higher than 10% carbamine peroxide may cause increased tooth sensitivity (Giniger et al 2005). Increasing the temperature can also enhance the effect of bleaching while also having an adverse effect on sensitivity.

Figure 13: The whitening effect of sodium perborate on the maxillary right lateral and central incisors after two applications.

Figure 14: Generalised gingival recession. This patient presented with severe pain after four days of external bleaching with 10% carbamine peroxide.

A 10% increase in temperature doubles the rate of chemical reaction; however temperatures elevated to a clinically uncomfortable level may result in latent tooth sensitivity or even irreversible pulpal inflammation. In addition to concentration and temperature, the degree of bleaching is also related to the amount of time that the bleaching agent is in contact with the tooth surface. The longer the time, the greater the lightening effect and the greater the likelihood of sensitivity (Baratieri et al 1995).

Tooth sensitivity can, however, be reduced by reducing the amount of time spent bleaching per day, bleaching on alternative days or by the substitution of a desensitising agent, such as KNO3 gel, into the bleach tray between periods of bleaching. Another approach to reducing sensitivity during bleaching is by the addition of desensitising agents such as potassium nitrate (KNO3) or fluoride, in the form of SnF2, to carbamine peroxide to produce 'sensitive-formula' gels. Fluoride acts as a tubule blocker to limit the fluid flow to the pulp. KNO3 penetrates the tooth to the pulp and has a numbing or calming effect on nerve transmission. Unfortunately, neither agent has proven to be particularly effective. KNO3 has a limited capacity to achieve antihypersensitivity unless used for long periods, and fluoride formulations are also slow acting and can cause significant tooth discolouration. In a recent double blind clinical trial by Giniger et al (2005) the effect of addition of amorphous calcium phosphate (ACP) to a 16% carbamine peroxide gel on the degree of hypersensitivity was studied. The results reported significantly reduced hypersensitivity compared to carbamine peroxide bleaching alone after 19 days, both in terms of intensity and duration. There was no associated reduction in the degree of tooth lightening with the ACP solution. This is the first study to show that ACP added to carbamine peroxide may reduce hypersensitivity and, although the results appear promising, further research is required before making a clinical recommendation for the use of ACPcontaining products.

External cervical root resorption

Cervical root resorption is a painless, inflammatory-mediated external resorption of the root, which can be seen after trauma and following internal bleaching. It is usually detected only through routine radiographs; however papillary swelling or tenderness to percussion can sometimes be observed. While the causes of resorption are not fully known, a review of the literature indicates a number of possible causes (Lado et al 1983). Patients tend to be younger than 25 years and most report a history of trauma. From a clinical viewpoint, what does appear to be an important factor is the regime of internal bleaching employed. It has been proven that formulations using either 30% hydrogen peroxide alone, or in combination with sodium perborate, are more toxic for periodontal ligament cells than sodium perborate mixed with water (Harrington and Natkin 1979). Heating the peroxide with a hot instrument also appears to promote resorption. Application of heat leads to a widening of the dentinal tubules and facilitates diffusion of molecules in the dentine (Pasley et al 1983). Moreover, application of heat results in generation of hydroxyl radicals from hydrogen peroxide, which are extremely reactive and have been shown to degenerate components of connective tissue (Dahlstrom et al 1997). Unsurprisingly, therefore, several authors have demonstrated that a high concentration of hydrogen peroxide, in combination with heating, seems to promote cervical root resorption (Baratieri et al 1995).

Table 1 provides an overview of clinical studies in which the occurrence of cervical root resorption was observed in association with the technique used. When interpreting the data in Table 1 it is important to note that a large number of cases had suffered known trauma. Perhaps the observation of greatest clinical significance is that there

Table 1
Overview of cervical root resorption observed in clinical studies

Reference	Sample Number	Treatment	Heat	Cervical Seal	Trauma	Resorption
Abou-Raas (1998)[4]	112	Wbt: sodium perborate +30% H_2O_2	No	-	-	0
Anitua et al (1990)[70]	258	Wbt: sodium perborate +30% H_2O_2	No	-	-	0
Friedman et al (1988)[30]	58	a) Thermocatalytic - 30%H_2O_2 b) Wbt: 30% H_2O_2 c) Thermocatalytic + 30%H_2O_2		No No No	No No No	1 1 2
Heithersay et al (1994)[71]	204	Wbt: sodium perborate +30% H_2O_2 Thermocatalytic	Yes	Yes	Yes	4
Holmstrup et al (1988)[72]	69	Wbt: sodium perborate + H_2O_2	No	Yes	Yes	0

have been no reported cases of cervical root resorption following internal bleaching using a combination of sodium perborate and water, or sodium perborate and a low concentration, i.e. 3% solution, of hydrogen peroxide. The author was unable to find published data on the incidence of cervical root resorption using a 10% carbamine peroxide solution in the inside/outside technique.

Stability

Advice regarding the long-term stability of bleaching is perhaps the most uncertain aspect of the therapy, as many factors must be considered when attempting to predict the outcome, including the aetiology and original degree of discolouration, dietary and smoking factors, patient age, etc. Data on the duration of both external and internal bleaching are mostly related to case reports, and only a few clinical trials are available for review. Tam et al (1999) reviewed 23 patients 1.5 and three years post external bleaching, and reported that 62% reported slight or no reversal in tooth colour. Another study by Ritter et al (2002) reported that 43% of patients perceived their tooth colour as stable 10 years after a six-week course of external bleaching. Swift et al (1998) reported that two years after external bleaching, regression of two shade tabs on the Vita® shade guide occurred; however the regression occurred during the first six months after bleaching. Amato et al (Amato et al 2006) evaluated the chromatic stability of internal bleaching from a population of 50 patients after 16 years. They reported colour stability in 62.9% of cases.

Effects on enamel

Questions have been raised about the effect of bleaching on the structure of the tooth itself. Surface alterations in enamel topography have been reported in several studies. Shannon et al (1993) evaluated the surface topography of enamel tabs exposed to 15% carbamine peroxide for 15 hours a day, using scanning electron microscopy, and detected significant alterations compared to a control group. This is due to a detectable loss of calcium from the surface enamel along with a loss in surface hardness depth of approximately 25µm. Bitter (1998) demonstrated that teeth bleached in vivo with 35% carbamine peroxide (35 min/day for 14 days) lost their aprismatic layer and the damage was not repaired after 90 days. However, the concentration of peroxide and amount of exposure may influence the amount of alteration to the enamel. Using infrared spectroscopy, Oltu and Gŭrgan (2000) compared the mineral composition of enamel exposed to 35% carbamine peroxide, to 10% and 16% carbamine peroxide, and detected change at 35% but no detectable change at 10% and 16%. A clinical implication of these findings may be that teeth are more susceptible to extrinsic discolouration after bleaching due to increased surface roughness.

Effects on restorations

Bleaching has little or no effect on most of the common restorative materials (Dishmann et al 1994). Bleaching may increase the solubility of glass ionomer and other cements (McGukin et al 1991) and reduce the bond strength between enamel and resin composites, at least for a short time. Because bleaching releases oxygen into the tooth, the oxygen released inhibits the polymerisation of the resin (Titley et al 1989). A delay of a week or more following the bleaching process is advised, prior to the placement of a new composite, to allow for this effect to be dissipated (Haywood 1992). Bleaching may cause a slight increase in surface roughness of some types of resin composite, and the hardness may be very slightly increased, but neither is clinically significant (Christensen 1989, Friend et al 1991). Bleaching has no effect on porcelain and, although it does encourage the release of mercury from some types of amalgam, the clinical relevance of this is not known (Hummert et al 1993).

Toxicity

It has been reported that the safety of bleaching using carbamine peroxide should not be an issue since both hydrogen peroxide and urea are found in every human cell; however it must be remembered that the dose makes the poison. Controversy still does exist regarding the safety issues of peroxide-containing products. Heymann (2005) has stated that: 'Literally hundreds of millions of teeth in the US have been bleached over the past 15-20 years without one credible account of any significant untoward effect appearing in the literature. Dozens upon dozens of clinical trials over this same time period have also affirmed the safety of vital tooth whitening when used in a shortterm treatment duration according to manufacturers'
instructions'. However, Heymann accepts concerns regarding the safety of toothwhitening products, if not used correctly, by stating that: 'Valid concerns still exist regarding individuals who may ignore manufacturer or dentist instructions and overuse whiteners for months or years. Long-term adverse effects on soft or hard tissues cannot be totally ruled out when these products are badly abused or overused'. (Heymann 2005).

Concerns have been expressed over the potential adverse effects of the use of hydrogen peroxide as a bleaching agent. Effects such as localised tissue irritation and external cervical root resorption have already been discussed. However, clinical studies addressing other adverse effects, in particular carcinogenesis, are lacking (Haywood 2006). Reactive oxygen radicals are a potential source of cell damage, causing DNA strand breaks, genotoxicity and cytotoxicity. Although these radicals tend neither to cross biological membranes nor travel large distances within a cell, numerous animal studies have demonstrated precancerous cellular changes, and indeed carcinoma, when hydrogen peroxide has been in direct contact with tissues, indicating that hydrogen peroxide might possibly act as a promoter (da Costa Filho et al 2002). It is therefore prudent to recommend that until clinical research to address the question of possible mutagenicity is concluded, bleaching therapies utilising high concentrations of hydrogen peroxide should not be used without gingival protection, and that hydrogen peroxide-containing products should not be used in patients with damaged oral mucosa (Kinomoto et al 2001).

Conclusion

- Whitening of teeth can be achieved either by an external – or vital – approach, where vital teeth are bleached by direct contact with an agent such as carbamine peroxide, or by an internal – or non-vital – approach, where nonvital teeth are bleached with an agent such as sodium perborate, in a walking bleach technique;
- most teeth are susceptible to bleaching;
- during tooth bleaching, reactive oxygen produced by the breakdown of peroxide causes more highly pigmented carbon ring compounds to be converted to carbon chains, which are lighter in colour;
- increasing the concentration of peroxide provides a more rapid bleaching effect; however it also increases the risk of tooth sensitivity;
- tooth sensitivity, if present, normally persists for up to four days after the cessation of bleaching and can be reduced by reducing the amount of time spent bleaching per day, bleaching on alternative days or by the substitution of a desensitising agent, such as KNO3 gel, into the bleach tray between periods of bleaching;
- there is a greater risk of post-operative sensitivity following power bleaching than with take-home bleaching;
- there have been no reported cases of cervical root resorption following internal bleaching using a combination of sodium perborate and water, or sodium perborate and a low concentration of hydrogen peroxide;
- the stability of bleaching is multi-factorial and variable. Only a few clinical trials are available for review;
- enamel may become more susceptible to extrinsic discolouration after bleaching due to increased surface roughness;
- bleaching has little or no effect on most of the common restorative materials; and
- controversy still exists regarding the safety issues of peroxide-containing products.

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