Tooth Discolouration and Its Treatment Using KTP Laser-assisted Tooth Whitening

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Summary: When confronted clinically with the problem of tooth discolouration, the basic strategy is to first identify the aetiology of the discolouration, and then to apply the selected treatment, using the philosophy "as much as required, but as little as necessary." For internal discolourations, green laser light from a KTP or argon laser is able to elicit photochemical reactions both within tooth structure and within a bleaching gel applied onto the labial surface. The unique optical and chemical interactions makes photochemical bleaching possible through promoting oxidation events in a controlled manner. This paper outlines a clinical approach to the diagnosis and management of patients with tooth discolouration, and summarises the technical and clinical aspects of the Smartbleach system for laser whitening. While extremely useful for routine in-surgery whitening treatments, the system is also able to treat severe tetracycline staining, to achieve pleasing improvements in appearance. A case is presented to illustrate the application of the Smartbleach system in these more challenging situations. A simple objective method for assessing tooth shade changes during laser whitening is also described.

Key words: discolouration, whitening, KTP laser, argon laser, tetracycline.

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The attractiveness of a smile is an important component of oral health. The colour and arrangement of the teeth, and their relationship to the soft tissues and bones of the face, give the overall aesthetic value of the human face. Of these components, the shade of the teeth is the easiest to alter to gain a cosmetic improvement for the dental patient. Having said this, it must be remembered that the health of the gingival tissues can greatly affect the appearance of the smile. Thus, periodontal care is an important part of cosmetic dentistry, and should be undertaken before laser-assisted tooth whitening.

DIAGNOSTIC WORK-UP OF THE PATIENT

Dentists are asked frequently to give an expert opinion regarding the likely causes for tooth discolouration. It is therefore essential to understand the interactions between tooth development and the various agents that can influence enamel and dentine formation.

When assessing the patient with discoloured teeth, it is essential to determine the nature of the problem, specifically whether the discolouration is due to internal or external factors. With internal discolouration, intrinsic stains such as pigments of various types have become incorporated into the enamel or dentine, either during its formation, or after eruption. In contrast, with external staining, a superficial layer forms

on the surface of the tooth through the action of dietary or lifestyle factors, such as tobacco, tea or coffee.

Intrinsic Discolouration

Intrinsic discolouration of the pre-eruptive type presents a diagnostic challenge to the dentist. Developmental defects may be defined as disturbances in hard tissue matrices and in their mineralisation arising during odontogenesis.

Discoloured enamel is defined as an obvious abnormal appearance of the enamel which, because of its colour and distribution, cannot be considered within the normal range of variation in colour and shade of tooth enamel. Unlike bone, enamel and dentine are not labile tissues. Because of this, developmental defects provide a permanent marker of events during tooth development. As many as 50 different conditions operating either locally or systemically have been causally linked to developmental disturbances in tooth formation. Altered tooth structure may not only stain, but may also be more prone to increased penetration of chromogens from the diet.

Developmental defects in enamel may be caused by a range of factors, including: birth trauma, premature birth and low birth weight, neonatal hyper-bilirubinaemia (jaundice), febrile bacterial infections such as otitis media, viral diseases in childhood (such as measles and chickenpox), anti-neoplastic drugs used to treat malignancies in young children, and major nutritional deficiencies during the early years of life (particularly Vitamins A, C and D). In icterus gravis neonatorum, high levels of circulating bilirubin in the infant cause clinical jaundice. Bilirubins from the peripheral circulation can become incorporated into tooth structure which is still forming, causing a yellow-green discolouration and hypoplasia of the primary teeth. The permanent dentition, however, is not affected as the state of hyperbilirubinaemia is short lived.

Enamel malformation and hypoplasia can be caused by poor maternal nutrition, particularly a high alcohol intake and deficiencies in vitamins and major nutrients during pregnancy. Other medical problems affecting the pregnant mother that can cause enamel hypoplasia in the infant include poorly controlled diabetes mellitus, toxaemia of pregnancy, hypocalcaemia, hypothyroidism, hypoparathyroidism, gastrointestinal malabsorption, liver malfunction, and chronic renal failure.

Several uncommon medical and inherited conditions have also been associated with alterations in tooth structure, including internal discolourations. Such medical conditions include congenital erythropoietic porphyria, phenylketonuria (PKU), trichodento-osseous syndrome, incontinentia pigmenti, Ehlers-Danlos syndrome, and cleidocranial dysplasia. In congenital erythropoietic porphyria, there is increased formation and excretion of porphyrins, which can result in purple-red or red-brown discolouration of the dentine in both the primary and permanent dentitions. Under ultraviolet light, the porphyrins in the affected dentine emit a red fluorescence.

In alkaptonuria and phenylketonuria, affected individuals are unable to oxidise homogentisic acid, a metabolite in the oxidation pathway of the amino acids tyrosine and phenylalanine. With the passage of time, abnormal pigmentation may occur in cartilage, connective tissues and teeth. Within the teeth, the homogentisic acid oxidises to a coloured quinone compound within the alkaline environment of mineralisation, re-suiting in a dark brown discolouration. The same events can occur in patients with phenylketonuria.

Inherited disorders affecting hard tissues are relatively uncommon. In amelogenesis imperfecta, the tooth colour ranges from an opaque white to yellow, and tends to darken with age. Because of increased porosity, newly erupted teeth may become stained from dietary and other chromogens. In dentinogenesis imperfecta, the teeth are opalescent and discoloured, with a colour ranging from bluish-grey to brown to yellow.

Disturbances which affect single teeth suggest a local aetiology, such as damage to the tooth germ of a permanent incisor from infection or trauma involving the deciduous precursor which is located immediately adjacent. In contrast, conditions that involve many teeth suggest a systemic disturbance or a genetic aetiology. Thus, trauma to the primary maxillary incisors can result in an area of chronic periapical infection that will be juxtaposed on the labial aspect of the developing permanent central incisor. Cariously exposed deciduous molars can exert similar effects on the premolars. In both situations, damage to the ameloblasts of the permanent tooth germ alters the maturation of enamel at the site of injury. This explains why these defects are usually very well defined.

Dental Fluorosis

Fluorosis has been identified as a significant cosmetic concern. Dental fluorosis is a specific disturbance of tooth formation caused by excessive intake of fluoride during the formative period of the dentition.[22] Exces-

sive intake of fluoride can cause developmental enamel defects ranging from mild opacity through to hypoplasia (with pitting, grooves, or missing areas of enamel) together with yellow-brown discolouration (mottling). The formation of subsurface voids which contain water causes a qualitative defect of the enamel, which is identified visually as an abnormality in the translucency of enamel, with a dominant white colour. With the more severe forms of fluorosis, combinations of hypoplasia, discolourations and opacities can occur on the same tooth.

Mild enamel fluorosis is typified by horizontal white flecking, generalised white opacity, and lack of lustre of the teeth, particularly the incisal edges of the maxillary incisors. With increasing severity, the white opacity of the teeth is accompanied by surface pitting (hypoplasia) and yellow-brown discolourations that are demarcated poorly. In the most severe cases, there is marked surface pitting, and areas of enamel may be structurally defective and may have chipped off during normal function. All clinical variants of fluorosis are symmetrical in appearance. Horizontal banding may be seen where the exposure levels have varied dramatically over time. Opaque white patches in the enamel may become stained yellow to dark brown over time. Affected teeth may also show a pronounced accentuation of the perikymata.

The manifestations of dental fluorosis depend upon the peak concentrations achieved in the blood following exposure to fluoride (usually by ingestion), the duration of exposure, and the age of the subject. The critical period is during the maturation period of tooth enamel, which for the cosmetically important maxillary anterior teeth is the second and third year of life.[9] Fluorosis is considered probable following intakes of 0.1 mg F/kg body weight during infancy.[23] Other reports have suggested a lower threshold of 0.03 to 0.10 mg F/kg body weight for European children.[2,21]

There are indications that susceptibility to dental fluorosis is increased by malnutrition.[36,57] In the absence of malnutrition, significant fluorosis affecting permanent teeth is rare unless drinking water contains in excess of 2 ppm fluoride. There have been many reports of moderate and severe fluorosis in geographic areas of the world such as India, Sri Lanka, China, Kenya and Senegal, which have natural levels of fluoride in ground water well above this level.

Various sources of systemic fluoride exposure can be identified, including drinking water, ingested toothpastes, beverages, and foods. Dentists, physicians, and pharmacists who prescribe fluoride supplements for use in nonfluoridated communities must base their decisions on the fluoride concentration of the domestic water supply, fluoride exposure from foods and beverages, and the child's weight/height/age. They must also provide advice on proper use of dentifrice (toothpaste), including avoidance of ingestion, use of small amounts, and need for supervised use by preschool children. Current information suggests that 0.05 to 0.07 mg F/kg body weight/day is a useful upper limit for fluoride intake in children, and this forms the basis for the recommendations of many national dental associations and health bodies. This figure includes fluoride intake from both dietary sources (foods, particularly seafoods; drinking water and beverages, such as tea) and nondietary sources (ingested toothpaste, fluoride tablets, mouthrinses and gels). It should be remembered the total ingested quantity may not be bio-available because of the effects of food and binding of fluoride to calcium.

Mild and moderate fluoroses are generally limited to situations where toothpaste is swallowed, tablets ingested, or water levels contain high naturally occurring levels of fluoride. Both conditions can be rectified cosmetically by enamel microabrasion, a simple procedure which can be undertaken in the dental surgery in approximately 10 minutes.[51]

Even in more severe cases of enamel fluorosis, rapid regeneration of the subsurface water voids can be obtained when Recaldent (GC Asia Dental, Singapore) – casein phosphopeptides complexed to amorphous calcium phosphate - is applied after enamel microabrasion.[31,32,38,47] Recaldent delivers calcium and phosphate in a unique soluble form to help remineralise enamel. Calcium phosphate is normally insoluble, ie, it forms a crystalline structure at neutral pH. However, the casein phosphopeptides keep the calcium and phosphate in an amorphous, noncrystalline state. The neutral ion species CaHPO4 formed from these ions can enter areas of porosity in tooth enamel, and replace water voids to form new hydroxyapatite, restoring a normal appearance to the enamel.

Tetracycline Staining

Tetracycline compounds bind to dentine and enamel by calcium chelation, and become incorporated into teeth and bones during calcification. They cause a range of discolourations ranging from yellow to brown, and grey. The most cosmetically important teeth, the permanent anterior teeth, are susceptible to staining from tetracycline during their formation, ie, between the ages of 3 months and seven years. During this "window

of susceptibility", the severity of staining is related to the dose and duration of use of the medication. The exact colour depends on the particular tetracycline analogue and its dose, for example, Aureomycin gives a grey-brown colour, while Achromycin gives a more yellow colour. There is strong evidence that dentine mineralisation is affected by systemic tetracycline therapy, and that tetracycline can be incorporated into peritubular dentine after mineralisation of the primary dentine matrix.[10,17,28]

While further chemical changes in the tetracycline complexes occur with age and exposure to sunlight, a degree of "natural photobleaching" of the incisal aspects of the anterior teeth can occur in individuals whose teeth are excessively exposed to sunlight because of a short upper lip and protruding incisors.[15,27]

While medical practitioners are now well aware that tetracycline antibiotics must be avoided from the second trimester in utero to 8 years of age, there are some clinical situations where their use is medically justified. The most common of these is the control of secondary respiratory infections in young children with chronic bronchitis, brochiectasis or cystic fibrosis. They are particularly valuable in the treatment of atypical pneumonia syndromes, chlamydial genital infections, rickettsial infection (Rocky Mountain spotted fever, typhus, Q fever), Lyme disease, and Ehrlichiosis.[4,39]

The chelate formed between tetracyclines and hydroxyapatite or calcium orthophosphate is the red quinone product 4-., 12-. anhydro -4-oxo-4-dedimethylaminotetracycline (AODTC). This coloured product is relatively resistant to oxidation from peroxide, but can be broken down (photo-oxidised) by green light in a particular narrow spectral range of visible green light (512 to 540 nm).[15] AODTC has a maximum absorption at 530 nm in the visible green spectrum and can be broken down (photo-oxidised) by light at this wavelength. Lin and others27 showed that tetracycline staining could be removed from teeth that were intensely stained with tetracycline by using only light, provided it was in the specific wavelength of absorption of the AODTC complex, ie, the 512- to 534-nm band. Under these conditions, the molecule breaks down to a simpler quinone which can then be oxidised using conventional bleaching materials such as hydrogen peroxide.

Laser light of the appropriate wavelengths can be generated using an argon or KTP laser (514.5 and 532 nm, respectively). Patients with intense tetracycline staining typically show a banded pattern, with less marked staining of the incisal edges of the maxillary incisor teeth. This effect occurs because the incisal edges of these anterior teeth may lighten with time, according to the extent of their exposure to sunlight. The posterior teeth do not change colour as they do not get significant exposure to sunlight.

The presence of tetracycline in teeth can be confirmed by using an ultraviolet lamp to illuminate the teeth while in a darkened environment. Under ultraviolet light, tetracycline compounds emit a characteristic yellow-green fluorescence emission that is easily detected by the naked eye.[3,24,58] Third molar teeth (especially their root apices) removed from young adults will demonstrate this fluorescence if these individuals received tetracyclines for the treatment of severe acne during their teenage years.

As will be discussed further below, KTP or argon laser whitening is an important advance in the treatment of patients with tetracycline staining. Conventional treatments for tetracycline teeth such as veneers or crowns, depending on the severity of the discolouration, are expensive, and must be repeated at some point during the patient's adult life. Laser whitening can photo-oxidize the stain and thus provide a cosmetic benefit without destruction of the teeth.

Other Intrinsic Discolourations

With advanced age, the enamel becomes thinner because of attrition, allowing the yellow colour of the underlying more sclerotic dentine to be seen more clearly. As secondary dentine is deposited within the dentinal tubules, this accentuates the normal yellow colouration of the dentine and causes optical isotropy, that is, less bending and distortion of light as it moves across the dentine.

Blood products can cause both pre- and posteruptive discolourations in teeth. When erythrocytes break down, their haemoglobin is released, and iron from this can combine with hydrogen sulphide to form iron sulphide, which is coloured bluish black. A common posteruptive cause of such discolouration is trauma to an incisor tooth. Prior to eruption, haemoglobin can be released from lysis of erythrocytes in the peripheral circulation, such as occurs in erythoblastosis foetalis (Rhesus incompatibility). The same process is involved in the intrinsic discolouration that occurs in patients with sickle cell anaemia, and thalassaemia. In the latter two conditions, the discolouration is more severe and does not improve with time.

Endodontic pathology and endodontic treatment are one of the most common posteruptive causes of intrinsic discolouration of single teeth. Internal haemorrhage within the pulp chamber can result in an appearance

much like a bruise, while necrosis of the pulp, with the associated degradation of the dental pulp by ischaemia and bacterial infection, typically results in greyish-brown discolouration. Following trauma to a tooth that retains its vitality but undergoes sclerosis or dystrophic calcification, a marked yellowing can occur. Moreover, internal resorption can lead to a pink discolouration as the resorbing tissue destroys the normal dentine and enamel which overly the dental pulp.

In teeth that have undergone endodontic therapy, colour changes can occur because of the medicaments, filling material, or sealer used within the root canal space. Components of some materials used in endodontic therapy can cause discolouration, for example epoxy resin sealers, antibacterial pastes which contain phenolic compounds or tetracycline analogues, formocresol, and iodine.

Other posteruptive causes of intrinsic discolouration include corrosion products from amalgam restorations, which can react with sulphur to form silver and mercuric sulphides, with grey and black colourations as a result.

Extrinsic Discolouration

Staining of teeth or restorations from external sources can be due to smoking, tannin-containing drinks such as tea, coffee and red wine, mouthrinses (such as chlorhexidine, cetylpyridinium chloride, benzalkonium chloride, sanguinaria, and essential oils), stannous fluoride, silver nitrate, and highly coloured foods or condiments such as soy sauce and berry juices. Pellicle, plaque and calculus can all take up stains on their surface, while active or arrested lesions of dental caries or dental erosion can take up stains from the oral fluid environment. Tannins found in tea, coffee and other beverages bind to pellicle and can form an even brown-coloured coating on teeth even if the teeth are relatively plaque free. A common site for tannin staining is the lingual aspect of the mandibular anterior teeth.

Extrinsic staining of the teeth can also occur from materials that are placed in the mouth and chewed habitually, for example tobacco, cola nut, areaca, betel, and khat.

Forming a Diagnosis

In coming to a final diagnosis of the causes of dental discolouration, it is essential to take a thorough history that includes:

- Medical history during the period of tooth development (serious illnesses, fevers, periods of hospitalisation, use of tetracyclines and other antibiotics)
- Residence history (systemic fluoride, trace elements in the water supply, periods spent living at extremely high altitudes)
- Family history (affected siblings, ancestors or offspring)
- Dental history (previous dental treatment, episodes of trauma, endodontic pathology)
- Chronology (when first noted, whether primary and permanent dentitions are affected)
- Oral hygiene habits (type of dentifrice, frequency of tooth brushing, use of mouthrinses)
- Lifestyle patterns and occupation (smoking, coffee and tea intake)

Siblings and parents should be examined whenever a genetic dental disorder is suspected.

The key to managing discoloured teeth is to follow a systematic and critical approach in diagnosis and treatment planning. The following 20 questions should be considered as part of this decision making process:[51]

- Is the patient's complaint tooth colour, surface texture, tooth contour, or a combination of these?
- Is the discolouration developmental or acquired in nature?
- Is there a genetic or familial component?
- What is the contribution of the patient's medical status?
- What is the contribution from the patient's lifestyle?
- If lifestyle factors are operating, will these habits be difficult for the patient to change?
- What are the risks associated with treatment?
- Will the discolouration worsen if nothing is done?
- What is the patient's level of awareness of cosmetic dental procedures?
- How important is dental appearance to the patient's occupation?
- Is the patient's cosmetic demand driven by his/her own perception or by the comments made by others?
- Has the patient come with an expectation that a particular treatment strategy will be followed?
- What are the indirect consequences of treatment, eg, the need to replace existing restorations?
- What is the expected result in terms of tooth colour and contour?
- What is the number and duration of appointments required?
- Is the patient prepared to make the necessary effort

in terms of time and expense in gaining and maintaining a worthwhile result?
- Have the relevant treatment options been explored with the patient, to allow him/her to make an informed decision?
- Is it essential to photographically record the baseline situation, to allow later comparison, or for medicolegal reasons?
- What treatment options are available if the suggested treatment fails to achieve the desired result?
- Should the patient be assessed or treated by another dentist or dental specialist?

TREATMENT PLANNING

The basic strategy is to first identify the aetiology of the discolouration, and to apply the selected treatments using the philosophy "as much as required, but as little as necessary."

For extrinsic discolouration, the tooth surface can be treated, eg, removal of stains and surface deposits by professional prophylaxis. Enamel micro-abrasion is also of great utility, since by smoothing the labial enamel surface, this treatment also increases light reflectance from the surface.

For internal discolouration, options include:

- Chemical bleaching with peroxide compounds, either passively or assisted by heating
- Photochemical bleaching (eg, green laser light for tetracycline staining)
- Masking the discolouration with an opaque composite resin veneer, a porcelain laminate veneer, or a full crown.

For combined pathology on the teeth, it is sensible to treat the surface using a minimal-intervention approach (eg, micro-abrasion or power bleaching) and to delay placement of permanent restorations until the effectiveness of treatment can be assessed.

PHOTOCHEMICAL LASER WHITENING

Since its introduction in the late 1990s, photochemical laser whitening has proven to be both safe and effec tive. The KTP or argon laser Smartbleach (HTL, Herzele, Belgium) method used for one appointment gives a greater shade improvement than diode laser bleaching or home bleaching with trays.[54] Dramatic results can be achieved with one treatment. [29,45,51,55]

Contra-indications for argon or KTP laser whitening can be listed as follows:

- Severe untreated tooth sensitivity of exposed cervical dentine from dental erosion, gingival recession, or gingival pathology (such as lichen planus). Such patients should have their sensitivity problem addressed first. Laser activation of topical fluoride with the argon or KTP laser is an effective strategy for desensitizing, as is the topical application of Recaldent. Because patients with gingival pathology such as lichen planus have delicate, friable tissues which respond badly to trauma,46 particular care is required when placing the flowable composite blockout to prevent contact of the soft tissues with the bleaching gel and to minimize trauma to the tissues.
- Where the patient has unrealistic preconceptions of the treatment result
- Where the patient is unable to sit still in the dental chair, and tolerate the required intraoral isolation and retraction devices
- Where the patient is not compliant with the lifestyle and dietary changes needed to prevent reformation of extrinsic stains (eg, a heavy smoker)
- Where the patient is unwilling to have the restorations in the teeth to be bleached changed, after a necessary delay of 2 weeks after the whitening treatment.

The patient should be informed before the treatment is commenced that the colour of restorations is not changed by laser whitening. In addition, there must be a delay of 2 weeks to allow oxygen levels in the enamel to return to normal before attempting to bond to the tooth, otherwise oxygen inhibition of resin polymerisation will compromise the replacement restorations. Exposure of enamel to 35% hydrogen peroxide for 5 to 30 minutes has been shown to cause a significant reduction in the adhesive bond strength of composite resin to enamel. Scanning electron microscopic examination of fractured peroxide-treated specimens has revealed that the bond failures occur primarily at the bonding resin-enamel interface, and that they are associated with areas of resin nonattachment and altered resin quality. These changes are caused by the presence of residual peroxide or reactive oxygen species at or near the enamel surface.[43] Lower shear bond strengths to enamel are the direct result of oxygen reactive species in the enamel surface changes.[13,41] The decrease in the adhesive bond strength of resin to 35% hydrogen peroxide-treated enamel is not caused

by a change in the elemental composition of treated enamel surfaces. No major changes occur in the elemental composition of enamel exposed to 35% hydrogen peroxide, other than an increase in nitrogen content.[37]

Often the patients can state their expectations clearly, and may come armed with information from acquaintances regarding the types of treatment that can be offered. In general, photochemical bleaching routinely allows worthwhile changes in tooth shade to be achieved. Other issues such as tooth surface texture can be addressed by enamel microabrasion.

Patients may have unrealistic expectations of the longevity of the result that is obtained. While the chemistry of bleaching is irreversible, the shade of a patient's teeth over the passage of years will alter and increase in value because of external stains and internal optical changes. Attrition and deposition of secondary dentine conspire to alter the shade of all vital teeth with advancing age, unless the dentine has been obscured by an opaque full coverage restoration.

Laser Whitening for Tetracycline Stains

Since the clinical aspects of the Smartbleach process have been presented in earlier patents,[44] reports,[45] and case studies,[29,51,54,55] this discussion will focus on more technical aspects. As mentioned earlier, when tetracycline compounds become incorporated into hydroxyapatite, the resulting red quinone product AODTC can undergo continued photo-oxidation. For this to occur, the molecule must absorb light with high photon energy at the correct wavelength: either 290 nm and 365 rim in the ultraviolet spectrum, or 512 to 540 rim in the visible green spectrum. [14,15,27] Because the maximum absorption aligns extremely well with the wavelength of the KTP laser (532 rim), energy from this laser can cause terminal photo-oxidation of the quionone molecule, which renders it colourless. The use of the KTP laser in combination with a hydrogen peroxide based gel ensures that complete and irreversible bleaching of the red quinone occurs. The feasibility of photobleaching has been demonstrated experimentally. Extracted tetracycline-stained teeth from rats, dogs and humans have been shown to lighten from photo-oxidation with exposure to sunlight.[27,40,56]

In addition to driving photo-oxidation reactions within the tooth, some of the visible green laser energy applied to the site is absorbed in Rhodamine B red dye, which is present within the bleaching gel (0.5 % w/w).

This molecule has its maximal absorption at 539 nm. When this dye is exposed to 532 nm light, it absorbs photons of energy with subsequent electronic transition to the singlet excited state. From here, the electrons may the fall back to the ground state and release the gained energy via electronic or physical processes (fluorescence or heat), or they may jump to the next excited state (intersystem crossover), which is the triplet excited state. The path is dictated by environmental variables, such as the temperature and pH. Once in the triplet excited state, the molecule may fall back to the ground state or undergo reactions with molecular oxygen, resulting in production of hydroxyl radicals, superoxide ions, peroxides, labile singlet oxygen, or reactive oxygen species. In this way, the interaction between the KTP laser energy and the dye is a photochemical process that results in the production of oxygen free radicals, and is not merely photothermal in nature. The fluorescence light emitted by the Rhodamine dye, when exposed to the KTP laser, has a wavelength of 602 nm, and falls within the visible orange/red spectrum. It can be seen by the clinical operator during the Smartbleach procedure, since it passes through the protective glasses.

A portion of the KTP laser energy absorbed into the Rhodamine B dye is also transferred from the excited molecule into the bleaching gel in the form of thermal energy. This transfer results in controlled heating of the gel and not the tooth, minimizing the possibility of thermal insult to the dental pulp.54,55 This superficial heating of the gel accelerates the breakdown of hydrogen peroxide, which further boosts the overall yield of oxygen free radicals over a given time.

Addressing Patient and Clinician Concerns

The Smartbleach system employs photochemical reactions at an alkaline pH to both break down tetracycline stains and to produce reactive oxygen molecules. The primary action of Smartbleach is photochemical, not photothermal. The gel has a high pH (approximately 9.5). Under these alkaline conditions, the perhydroxyl radical is produced from hydrogen peroxide.[49] This radical is more reactive than superoxide and other radicals. In addition, under alkaline conditions, etching of the tooth surface does not occur. The photobleaching effect can be obtained equally well with the argon laser and the KTP laser.

KTP and argon laser whitening using the Smartbleach system has been shown by electron probe mi-croanalysis to give rapid and deep penetration of

oxygen molecules into intact tooth structure. In contrast, with home bleaching treatments, the penetration is superficial. Some patients experience tingling when the peroxide reaches the level of the dental pulp and is broken down there by the enzyme catalase into oxygen and water. The risk of eliciting transient inflammation of the pulp because of percolation of hydrogen peroxide into the pulp via areas of exposed dentine or enamel fractures has been discussed in the literature.[20,30] Several studies have determined that 30% hydrogen peroxide can penetrate to the dental pulp within 15 min of application to the enamel surface.[12]

Home-based methods use gels with either carbamide peroxide or hydrogen peroxide as the active ingredients. These gels typically are acidic preparations with hygroscopic (water absorbing or desiccating) properties. Thus, tooth sensitivity and rebound of colour change often occur due to remineralisation and rehydration at the end of a home bleaching treatment.[51] Such problems do not occur with the Smartbleach system, since the pH is strongly alkaline and the teeth are not etched or dehydrated during the treatment process.

As with all bleaching treatments, excessive exposure of a tooth to strong oxidizing agents can lead to "overbleaching". In this situation, total oxidation of enamel proteins occurs, and the volume which was occupied by these (approximately 7% of the enamel) is now replaced by water, since the organic (protein) molecules have been oxidized fully to carbon dioxide gas. This protein alteration and destruction can be shown experimentally by exposing powdered dentine to 30% hydrogen peroxide. This treatment alters the chemical structure of the dentine, making it more susceptible to degradation via loss of organic components.[35] The explanation for this exaggerated over-bleaching effect is that the bleaching process has transformed the organic substances (ie, proteins) into simpler chemical intermediates, and eventually into carbon dioxide and water.

This over-bleaching effect can only occur if the dentist repeats an in-surgery whitening treatment at high frequency, or the patient over-uses a home bleaching treatment (in terms of duration or frequency of exposure). The subsurface defects caused by over-bleaching result in a diffuse opacity of the enamel. As with other types of subsurface defects, this can be rectified by rebuilding subsurface mineral using topically applied microcomplexes of casein phosphopeptides and amorphous calcium phosphate (eg, Recaldent chewing gum or GC Tooth Mousse [GC Asia Dental] applied topically each night before bed).[47,48,50]

Safety Issues with Hydrogen Peroxide

Hydrogen peroxide has been used in dentistry for more than 70 years to bleach teeth.[59] It can exert toxic effects on soft tissues in high concentrations and with exposures of prolonged duration.49 Thus, when hydrogen peroxide-based gels are used for laser-enhanced whitening, vitamin E neutraliser gel or cream must be kept at hand for rapid application in the event of leakage of or accidental soft tissue contact with the gel.

When concentrated hydrogen peroxide solutions and gels have been used for in-office bleaching treatments, significant adverse soft and hard tissue effects have not been observed clinically, other than a relatively common but self-limiting post-treatment sensitivity.11 This can be attributed to the natively acidic pH of commercial hydrogen peroxide preparations. In contrast, the pH with the Smartbleach method is approximately 9.5, which prevents erosion of the tooth surface.

Hydrogen peroxide is a designated hazardous substance at concentrations above 5%. Direct contact between 30% hydrogen peroxide preparations and the skin or eyes may cause severe irritation or burns. Soft-tissue damage can occur following inadvertent exposure of oral soft tissues to concentrated solutions of hydrogen peroxide[1,6] if a neutraliser is not applied, with epithelial damage, leading to vesicle formation and ulceration if left in place for extended periods.

Hydrogen peroxide can diffuse through lipid membranes, and once inside the cell, it is able to react with transition metals to produce the highly reactive hydroxyl radical which can initiate chain reactions of lipid peroxidation leading to cell rupture.[7]

Superoxide can reduce a cellular source of ferric to ferrous iron, and the latter then reacts with hydrogen peroxide to produce a more potent oxidizing species, such as the hydroxyl radical or an equivalently reactive species. This in turn initiates the peroxidative decomposition of the phospholipids of cellular membranes, which results in damage to lysosomal membranes and leakage of their destructive contents. The hydroxyl radical also damages the inner mitochondrial membrane, which can lead to the loss of viability of the cell.[49]

Humans are well endowed with anti-oxidant defences against reactive oxygen species. These anti-oxidants, or free radical scavengers, include ascorbic acid (vitamin C), alpha-tocopherol (vitamin E), beta-carotene, coenzyme Q10, and trace elements including selenium and zinc.[19] While these substances are effective

at reducing the potential for cellular damage from free radicals, the major anti-oxidant defensive mechanisms are enzymes. Superoxide dismutase converts superoxide radicals to hydrogen peroxide, while catalase and glutathione peroxidase detoxify hydrogen peroxide. Superoxide dismutases control intracellular levels of the superoxide anion, and thereby regulate the secretion of hydrogen peroxide from cells.

As would be expected from this, reactions elicited by hydrogen peroxide in a given tissue are influenced by the presence of superoxide dismutase and catalase, and the local concentration of metals such as iron and selenium.18 Normal oral soft tissues, including dental pulp, contain superoxide dismutase, and the level of activity is typically increased in inflamed tissues.[16]

Catalase is highly effective at preventing cellular damage from hydrogen peroxide. Catalase applied following intra-coronal application of concentrated hydrogen peroxide during bleaching procedures has been shown to eliminate residual hydrogen peroxide within 3 minutes.[33] At the microscopic level, normal human serum contains catalase, and this provides protection from adverse effects of hydrogen peroxide.[25] In contrast, the catalase activity of normal pulp appears to be low relative to other soft tissues.[8]

Direct application of 30% hydrogen peroxide to dental enamel has been used for many years as part of the clinical management of noncarious enamel defects. Lightly discoloured or stained defects of the enamel have been treated by bleaching the affected area without adverse effects on the dental pulp.[42]

Hydrogen peroxide has the potential to affect dental enamel because of the acidic pH of the solution in its native form. The pH of concentrated solutions of hydrogen peroxide as supplied by the various manufacturers is 5.0 to 6.0.[34] Concentrated (30%) solutions of hydrogen peroxide can transiently reduce the microhardness of enamel and dentine. This reduction can be noted with exposure times as short as 5 min for the dentine and 15 min for enamel.[26] This effect does not occur with the Smartbleach system because of its high pH.

THE SMARTBLEACH PROCEDURE

The appointment for laser whitening is approximately 75 min in duration, including the initial preparation and photographs. The patients must remain still with isolation devices in their mouth a sizeable portion of this time. Isolation is undertaken using a self-holding retractor and a combination bite block and saliva ejector device. To ensure that the lips and tongue are fully protected from contact with the bleaching materials, the isolation devices must remain in position at all times. The patient is not permitted to rinse.

Before the bleaching gel is placed on the teeth, extrinsic stain and deposits of pellicle is removed by prophylaxis using pumice. Any remaining organic proteinaceous material on the tooth surface will interact chemically with the bleaching agent, and also reduce its penetration into the enamel, reducing its effectiveness. Conventional prophylaxis and polishing pastes must not be used, since the oils which they contain can coat the tooth surface and impair the generation of oxygen free radicals.

The exposed cervical root surfaces of the teeth and the cervical gingival tissues are then protected from the hydrogen peroxide gel and from dehydration, using a flowable composite material (Smartblock). The soft tissues need to be protected as these would normally absorb the visible green laser light, resulting in thermal damages. Once the isolation is in place, flowable composite is applied directly to the teeth at the level of the gingival sulcus, using a syringe with a blunt tip which is placed directly into the opening of the gingival crevice. The gingival sulcus must be dried carefully to remove moisture before applying the flowable composite material. The first application should cover the cervical aspect of the teeth, for approximately 1 mm.

Any areas of exposed dentine on the crown or root surface must also be protected, by applying the flowable composite. If an area of exposed dentine is missed, the patient may experience discomfort from accelerated penetration of the bleaching gel at that site. The same problem can occur with occult defects such as enamel lamellae and cracks in the enamel, resulting in transient pulpal hyperaemia. Once the first increment has been polymerised, the material is then built up towards the sulcus. Rubber-dam or wax strips cannot be used as these do not provide adequate protection from the laser light, bleaching gel, or dehydration. If gel leaks from around the flowable composite, the tissue effects can be neutralised instantly using Vitamin E, applied as a liquid or cream.

The patient, the assistant, and the dentist performing the laser whitening procedure must wear protective glasses, because the human eye is extremely sensitive to visible green light. Appropriate protective glasses, goggles or face shields will have an attenuating power of log 4 (OD 4).

To mix the Smartbleach gel, the hydrogen peroxide liquid is titrated from a syringe into the gel powder, and mixed with the powder to give the desired gel con-

sistency. Once mixed, the gel must be allowed to stand in its closed container for 5 min to allow the carbonate buffer system within the gel to elevate the pH to approximately 9.5. Because the liquid is corrosive, protective gloves and glasses must be worn when handling the bleaching materials. The Smartbleach gel is coloured using Rhodamine B dye, which absorbs visible green laser light, and breaks down to release oxygen free radicals. The absorption process also results in an increased temperature of the gel on its outer surface,[54,55] which accelerates the decomposition of the hydrogen peroxide. The bleaching gel is applied in a predetermined sequence, which ensures that each tooth has a similar time of exposure to the gel and laser.

Once gel has been applied to the teeth, laser activation can then take place, using the KTP or argon lasers (with wavelengths of 532 or 514.5 rim, respectively). These laser wavelengths have a very low absorption in (and thus a high transmission through) dental enamel, which means that they do not cause direct heating of enamel, unlike infrared lasers. The laser energy is delivered through a glass optical fiber that can be held in a stilette handpiece through which the bare fiber extrudes. Bleaching does not in any way damage the fiber. A visible red aiming beam is used to target the gel and areas of discolouration during the procedure. If the aiming spot is not a circle, the end of the fiber must be cleaved to make the end surface perpendicular to the axis of the fiber.

The laser is applied with a spot size of 6 to 8 mm, using continuous mode. The beam is used in a sweeping action across each tooth for 30 s at a power of 0.8 W. This spot size can be obtained with a bare fiber by holding it approximately 12 mm away from the tooth. At no point should the fiber ever touch the bleaching gel.

After completion of one pass of all teeth to be treated, the gel is removed by suction and then by triple spray. The teeth are then washed thoroughly to maintain hydration. After inspection of the effect, the teeth can be dried and fresh gel applied.

The combination of gel and laser can be repeated for three passes in the one session. In the case of banded areas of staining, gel and laser can be targeted to the banded areas.

After the third pass is complete, neutral sodium fluoride gel can be applied to the teeth, and the teeth irradiated using 0.4 W for 15 s per tooth. This laser fluoride treatment is able to reduce the acid solubility of the enamel. This helps to protect the teeth from erosion, particularly from acidic drinks. The fluoride gel is rinsed off, and the flowable composite block-out removed.

The final shade can then be checked, using shade tabs. Alternatively, two calibrator shades, such as C4 and B1, can be included in the image to act as reference points.

Many bleaching systems cause either etching or dehydration of the teeth, or both. Neither occur with the Smartbleach system, since the teeth are kept in a hydrated state, and the gel is alkaline and so cannot etch the teeth. Therefore, the shade changes that occur are not simply the effect of the teeth being etched, which in itself could cause some rebound within a short time period. The treated teeth should have normal lustre, and the pattern of light reflection from them should be unchanged.

After treatment, a patient should not consume pigmented foods for 72 h, because of the unstable nature of the freshly bleached enamel surface. Coffee, tea, red wine, and smoking should be avoided. The patient must maintain a high standard of oral hygiene, and attend for follow up care as required.

Clinical Cases

The KTP laser can be used for gingival surgery without bleeding. In some cases, such as when the maxillary incisors already have crowns, just the maxillary canines and all the mandibular anterior teeth can be treated with Smartbleach (Figs 1 to 7).

For patients with severe tetracycline staining, excellent results can be achieved, although typically 2 or 3 full treatments (spaced 2 weeks apart) may be needed. We and others using this method have seen cases of shade changes from C4 to B1.

Under ultraviolet light, a characteristic green-yellow fluorescence occurs with tetracycline.[4,24,58] Disappearance of this fluorescence after treatment confirms that true photochemical breakdown of this has been achieved. In severe cases, a dramatic reduction in staining from tetracycline can be achieved.

MEASUREMENT OF SHADE CHANGES DURING LASER WHITENING

Several techniques have been developed for objective measurements of changes in tooth colour. The reproducibility of shade measurement and comparison using single shade guides is less than perfect. When pre- and post-treatment photographs are taken, numerous fac-

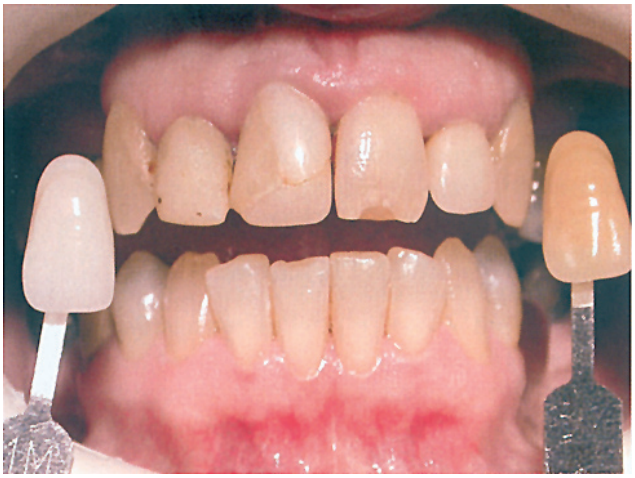

Fig 1 Case 1 prior to treatment, with 1 M1 and 5M3 shade guides included in the image. The intended treatment plan was KTP laser-assisted periodontal therapy, gingivoplasty, laser whitening (completed in one visit), followed by placement of porcelain veneered to metal crowns on the maxillary incisors.

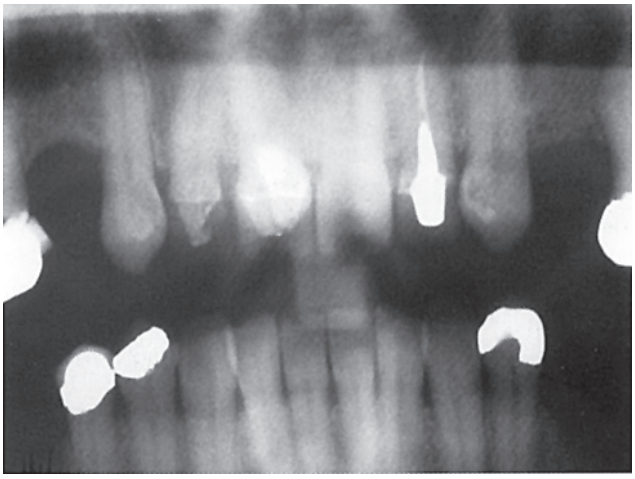

Fig 2 Portion of a panoramic radiograph, showing the anterior teeth. Mild horizontal bone loss has occurred.

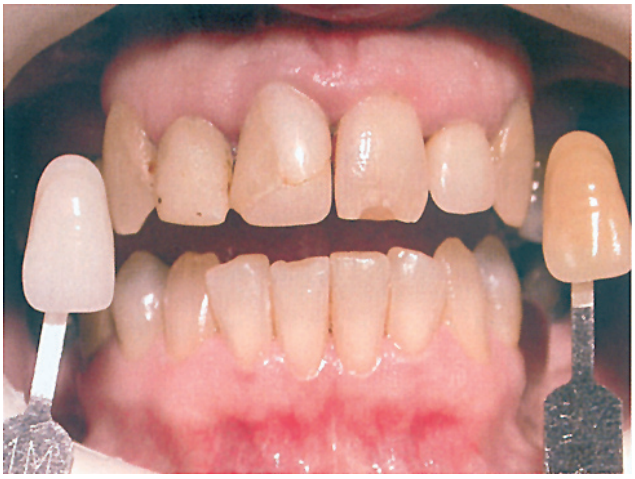

Fig 3 Clinical situation immediately after periodontal debridement, followed by pocket disinfection and gingivoplasty with the KTP laser. Note the absence of bleeding at the surgical site.

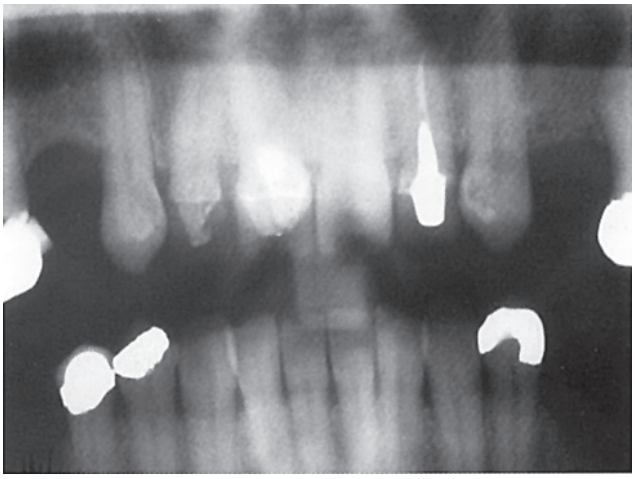

Fig 4 Flowable composite resin has been plated to protect the surgical site and the mandibular gingiva, and then Smartbleach gel has been placed on the maxillary canines and eight of the mandibular teeth.

tors conspire to make the images difficult to compare, including differences in lighting and exposure. For conventional film-based cameras, film type, film temperature, and processing conditions introduce additional variations.

The authors employ a method termed DOTCAM (digital objective tooth colour analysis method) for simple and objective digital analysis of changes in tooth shade from digital clinical images.[53] In this method, two shade standards serve as calibration points for the interpolation of tooth shade, using the mean blue pixel intensity from histogram analysis.

The DOTCAM technique is based on the principles originally described by Bentley and colleagues.[5] The digital representation of a colour image uses red, green and blue values to describe the colour of each picture element (pixel). The blue colour value has been shown to provide the best correlation to the overall impression of lightness of a tooth. A low blue value gives an increase in the yellow component of colour, because of

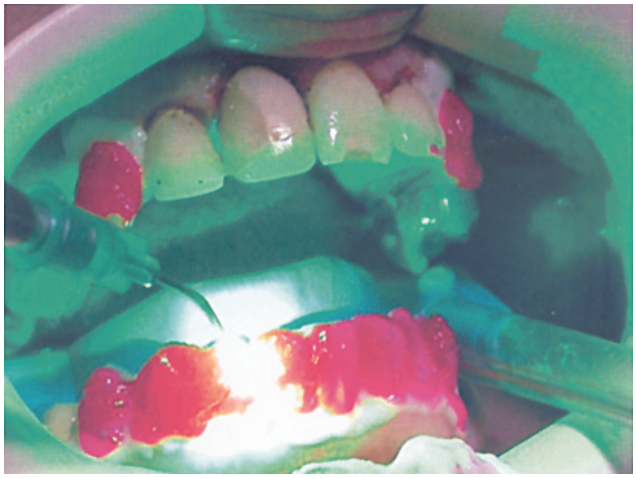

Fig 5 The Smartbleach gel is activated with the KTP laser.

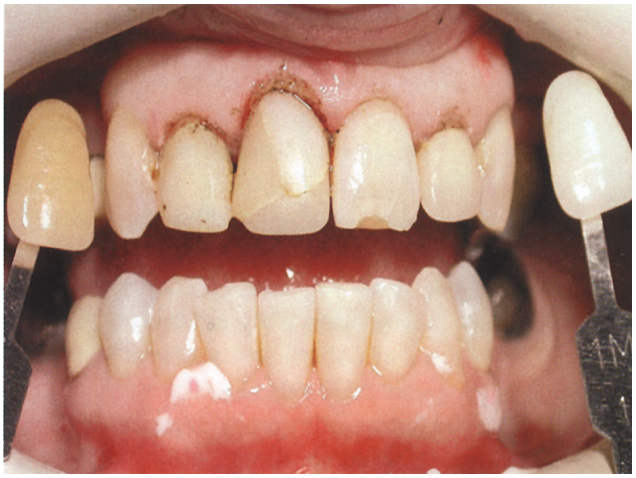

Fig 6 The situation at the end of the first visit. Some residues of Vitamin E neutralizer gel are present on the mandibular gingiva. The laser whitening effect can be seen clearly.

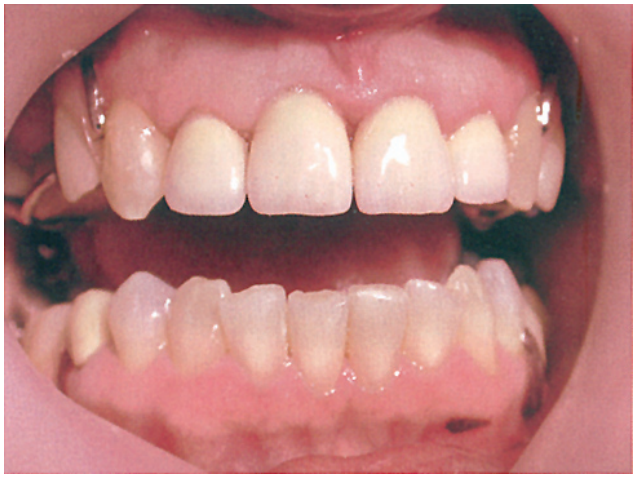

Fig 7 Final result of treatment, immediately after cementation of crowns on the maxillary incisors.

Table 1 Effect of camera image resolution on pixels per tooth

Image size	Pixels per image	Pixels per tooth
160 X 120	19,200	2,054
320 X 240	76,800	8,218
352 X 288	101,376	10,847
640 X 480	307,200	32,870
696 X 532	370,272	39,619
2048 X 1536	3,145,728	336,592

The data were determined using a standardised field of view, with a shade tab occupying the same area in each image and six different consumer-level digital cameras. The shade tab was the same size as a typical permanent maxillary central incisor tooth.

the reciprocal arrangement between blue and yellow in the additive colour system. The data for blue pixel values for the labial surface of a tooth can be aggregated in software, and the mean value determined.

For the mean blue value to have meaning, a scale of measurement must be generated which is specific to the camera equipment used. To create this internal scale, two standards must be included in each view (slide, negative, or digital image) which is recorded. These standards are the visibly lightest and darkest shade guides on hand (eg, Vita B1 and C4). Having these internal standards overcomes problems with exposure, film processing, and other variables that affect the final image. An alternative exists where only a single tooth is to be treated, and nearby teeth have porcelain facings or full veneer crowns, where one such tooth could serve as the "light" standard. If this is done, care must be taken that all extrinsic stain is removed from the porcelain before photographs are taken.

The full labial surface of both standards must be present in every image, and this requires some planning beforehand in terms of composing the photograph and placing the retractors. Self-holding retractors are useful as the patient can then hold in place one standard in each hand.

The DOTCAM method requires basic computer equipment to perform the image editing and pixel data

calculations. For ease of use, the colour resolution ("colour depth") used on the computer monitor should be 16 bit (high colour) or 24 bit (true colour). Clinicians who use digital still cameras or have combined a conventional film-based camera with a computer and a scanner will be able to adapt this method to their own practice. Film scanners (used with slides or negatives) will provide more discrimination than flatbed scanners used with photographic prints. The higher the image resolution (in pixels), the greater is the number of pixels per tooth, and thus the greater is the ability to discriminate subtle differences in lightness between images (Table 1).

The software necessary for image editing and pixel calculations can be obtained commercially, for example PaintShop Pro or Adobe Photoshop. The software must have a selection tool and histogram analysis capabilities.

The step-by-step procedure for DOTCAM can be summarized as follows:

Step 1. The baseline image is taken with a digital camera, ideally with a resolution of at least 2.5 megapixels. Alternatively, the image could be acquired by scanning a colour transparency, a negative or a print. The baseline image must have the two shade guides present in the same focal plane as the teeth under treatment. Normally, the central incisors are a useful reference point.

The baseline image is then opened in PaintShop Pro or Photoshop. The original image is immediately backed up to hard disk using a lossless image format (such as TIFF) before any selections or modifications are made. Images which have intense highlights from the flash or other lighting, or in which the entire labial surface is not present are not well suited to analysis. In these instances the image should be re-taken. This is clearly a situation where a digital camera offers an advantage by providing immediate feedback.

Step 2. The paintbrush or erase tool is used to outline the labial surface of the individual teeth and the shade tab standards with black. This step may be facilitated by cropping the image, to remove lips and structures other than the teeth and the shade standards.

Step 3. The "magic wand" selection tool is then used at a tolerance of 5 to 20 pixels to select areas of strong reflection, or obvious highlights from the flash. These areas of almost pure white will distort the histogram analysis, and must be are removed by filling these with black using the paintbrush tool set to black.

Step 4. The magic wand tool is then used to select the labial surfaces of the "light" shade standard, employing a pixel value tolerance of approximately 100. This will select only the areas of shade and will ignore the black background and the highlight areas removed in step 3.

Step 5. A histogram analysis of the selected area is obtained using the "toggle histogram window" button command in PaintShop Pro or the "Image-histogram" menu command in Photoshop. The information is selected for the blue channel, and the mean blue value recorded. The histogram shape will change from tooth to tooth according to the distribution of pixel values, but this is not important.

Steps 4 and 5 are then repeated for the "dark" shade standard, and then for each of the treated teeth, to obtain a mean blue value for each.

The ratio for each tooth is then calculated as follows:

Ratio = (tooth - dark standard) / (light standard - dark standard)

A convenient means of expressing the ratio is as a percentage, ie, by multiplying the value obtained above by 100. The higher the blue value, the lighter the tooth, and thus the greater the ratio. An extremely white tooth may exceed the value of the "light" standard and give a ratio greater than 1, or a percentage greater than 100. Similarly, it is possible to obtain negative values if the tooth in question is darker than the "dark" standard.

Comparing data from before- and after-treatment images allows the actual effect of treatment to be evaluated. The ratios for each tooth can be recorded on a simple odontogram for later reference.

Results using DOTCAM

Using this objective method, greater whitening effects have been shown for photochemical laser bleaching using the Smartbleach system (with the KTP laser) compared with photothermal diode laser bleaching and home nightguard vital bleaching. In a series of patients with a baseline shade of A3, the change in ratio for blue channel intensity on their maxillary incisor teeth with the KTP laser Smartbleach method was 33.71 to 37.56. In contrast, with the diode laser OpusWhite method (Lumenis, Yokneam, Israel), the improvement was 8.10 to 11.58, which was comparable to one week of home treatment with PolaNite bleaching gel (SDI, Bayswater, Victoria, Australia) in custom trays, which gave a change of 13.83 to 15.77. It must be remembered that these results are independent of the effect

of the bleaching treatment on the reflective properties of the tooth surface, since saturated areas of highlights were digitally filtered from the individual tooth data before analysis.

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