Colours, Forms and Structures of Teeth: Restoring the Eroded Sparkle

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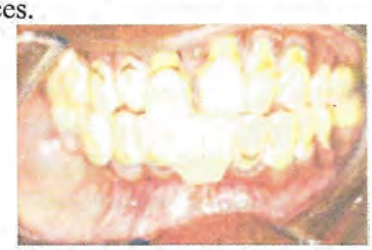
INTRODUCTION

In modern society, aesthetics form an important part of dental treatment, with the ultimate objective of creating a beautiful smile, with teeth of pleasing inherent proportion to one another, and a pleasing tooth arrangement in harmony with the gingiva, lips and face of the patient [1].

Colours are the smiles of nature (Leigh Hunt) and according to Phyllis Diller; a smile is a curve that sets everything straight. Little wonder then, why patients and clients now demand not only a healthy mouth, but also a perfect smile.

Among the significant factors that affect the overall dental appearance are tooth colour, shape/form and position, quality of restoration; and the general arrangement of the dentition, especially of the anterior teeth [2]. The teeth are an integral part of facial aesthetics and it has been said that the most common associations with facial attraction are the eyes and the mouth [3]. The oral region plays an important role when an individual speaks or approaches another person; for example poor oral hygiene and ugly teeth are noticed easily by other people [4]. On the contrary, a set of well-formed teeth, bright and creamy white, pleasing in forms, sizes and arrangement is usually seen in television commercials, promoting various goods and services.



Dirty and ugly looking teeth

sparkling smile

The Human Dentition

Humans, like other mammals are diphyodont, meaning they develop two sets of teeth (the primary/deciduous set and the permanent teeth). Humans usually have 20 primary (incisors, canines and molars) and 32 permanent teeth (incisors, canines, premolars and molars). Primary teeth begin to form between the sixth and eighth week in-utero and permanent teeth start developing in the twentieth week in-utero. The different tooth types have identifiable features that distinguish them from one another [5].

A tooth is composed of a crown (i.e. the portion exposed to the oral cavity) and one or more roots (i.e. the portion enveloped in bone). It is made up of three distinct hard tissues, enamel, dentine and cementum, and one soft tissue, the pulp, which provides the blood and the nerve supply to the crown and root.

Terms of orientation for teeth are mesial (toward the anatomical midline, or the point between the two central incisors), distal (away from the midline); buccal (toward the cheek), labial (toward the lip), lingual

(toward the tongue), and occlusal (the chewing surface of a tooth)[6].

Tooth Structure

The enamel is the hardest and most highly mineralized substance of the body. It is normally visible and must be supported by underlying dentine. Ninety five percent of enamel consists of mineral (hydroxyl apatite); water is 4% and organic material 1% by weight[7]. In the primary dentition, enamel is white because it is not translucent. The normal colour of the enamel in the permanent teeth varies from light yellow to greyish white. At the edges of teeth where there is no dentine underlying the enamel, the colour sometimes has a slightly blue tone. Since enamel is semi-translucent, the colour of dentine and any restorative material underneath the enamel strongly affects the appearance of a tooth.

The dentine which is a porous yellow-hued material is made up of 70% inorganic materials, 20% organic and 10% water by weight [7]. Dentine is softer than the enamel but acts as a protective layer and supports the crown of the tooth. The cementum on the other hand is a specialized bone-like substance covering the root of a tooth. It is approximately 45% inorganic material (mainly hydroxyl apatite), 33% organic material (mainly collagen) and 22% water[8]. The dental pulp is a vital, reactive, dynamic tissue located in the Centre of the tooth and it communicates with the periodontium through the apical and accessory foramina. It provides the functions of induction, formation, nutrition, defense and innervation[5].

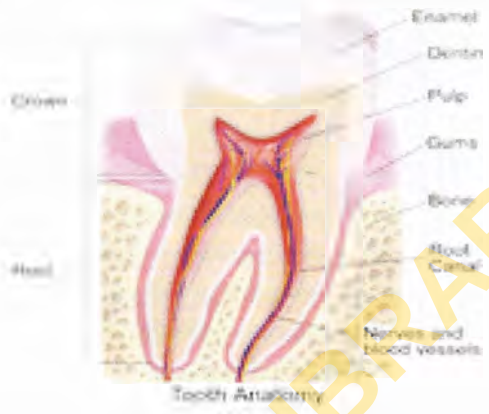
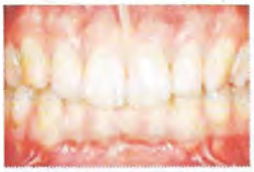
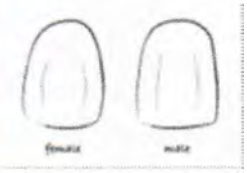


Fig 1

Tooth Form

Williams [9] in 1914 categorized tooth form into tapered, oval and square. Gender-specific differences in tooth form seem natural as gender specific anatomic differences are ubiquitous throughout the human body. According to Frush and Fisher [10] in 1955, feminity is characterized by roundness, smoothness and softness leading to an oval tooth form with round edges. In contrast, a masculine tooth form expresses vigour, boldness and hardness; therefore, it should have a cubical shape.





Female tooth form

Male tooth form

Tooth Colour

A variety of colours can typically be seen in a tooth and from the gingival margin to the Incisal edge of the tooth, a gradation of the colour occurs [11]. Basically, the colour of the

dentine is mainly responsible for the colour of the tooth [12] especially where it consists of thick layers and where the enamel layer is thin.

It has been reported that the first evidence of abnormality in human dentition is often an observable difference in the colour of teeth [13]. The composition of tooth structures will also affect the outward appearance of a tooth. If structures like the enamel, the dentine or the pulp change, the light transmitting and light reflecting properties of a tooth are likely to be altered [11]. When alterations in the colour or structures occur, the form may be affected and the resultant effect could be very distressing, depending on the age of the patient and the severity of tooth discolouration. The effect is profound, especially when there is discolouration of anterior teeth which are usually exposed when someone smiles.

A smile has a great impact in a beauty conscious society, and when it is diminished by dental diseases, it often results in loss of self-esteem and damage to physical and mental health [14, 15]. According to Bryan and Welbury [16], abnormalities of shape, size, colour and structure of the whole or part of the anterior dentition in children could have significant effect on psychosocial development and interaction with peers.

In recent years, the desire by patients to improve their dental appearance has grown tremendously. Rising to this demand, a number of methods and approaches to successfully improve the colour of teeth and restoring their forms and structures have been developed.

Colour and Colour Perception

Physicists describe colour as being derived from varying wavelengths of particles of light in an electromagnetic field. Red has the lowest frequency, violet the highest frequency and yellow lies in between. Furthermore, neurophysiologists tell us that the photosensitive cells of the retina can determine varying degree of three Colours i.e. red, yellow and blue.

The phenomenon of colour is a psychophysical response to the physical interaction of light energy with an object, and the subjective experience of an individual observer [17]. Colour can be described according to Munsell Colour Space in terms of hue, value and chroma [18] Hue is the attribute of a colour that enables one to distinguish between different families of colour, for example, reds, blues and greens. Value indicates the lightness of a colour ranging from pure black to pure white. Chroma is the degree of colour saturation and describes the strength, intensity and vividness of a colour. In addition to these three, other more subtle secondary optical properties of the tooth exist which can affect the overall appearance of the tooth. These include translucency, opacity, iridescence, surface gloss and fluorescence [19].

The colour of a tooth is determined by a combination of its optical properties. Tooth colour has been shown to result from the volume scattering of light i.e. illuminating light follows highly irregular light paths through the tooth before it emerges at the surface of incidence and reaches the eye of the observer [20, 21]. The perception of tooth colour is therefore a complex phenomenon, being influenced by a number of factors, including the type of incident light, the reflection and absorption of light by the tooth, the adaptation state of the observer and the context in which the tooth is viewed.

Generally speaking, the colour of the teeth is determined by the combined effects of intrinsic and extrinsic colourations [6]. Intrinsic tooth colour is associated with the light scattering and absorption properties of the enamel and dentine [22] while extrinsic colour is associated with absorption of materials onto the surface of enamel, and in particular the pellicle coating, ultimately resulting in extrinsic stain [23].

CLASSIFICATION OF TOOTH DISCOLOURATION

Historically, tooth discolouration or staining has been classified into extrinsic and intrinsic based on the location of the stain on the tooth. A third category known as internalized discolouration has, however, been considered [24].

Extrinsic Discolouration:

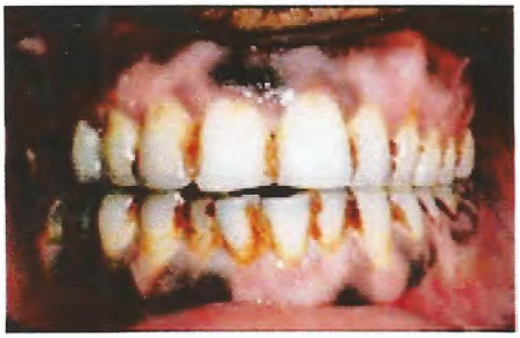
This can be defined as discolouration located on the outer surface of the tooth structure and is caused by topical or extrinsic agents [25]. It can be divided into two groups, direct and indirect, or based on its origin, into metallic and non-metallic. Direct staining is caused by compounds incorporated into the pellicle layer and the stain is as a result of the basic colour of the chromogen. Indirect staining on the other hand is caused by a chemical interaction at the tooth surface. It is usually associated with cationic antiseptics and metal salts. These agents are without colour or produce a different colour from the stain on the tooth surface as shown in the classification below, by Nathoo [26]

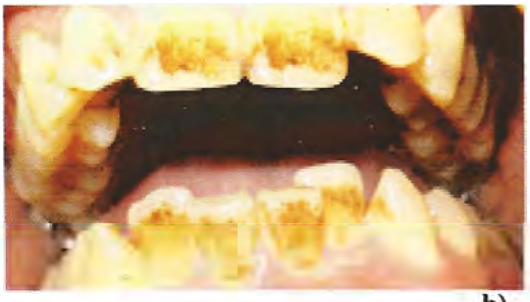
- Nathoo type 1 (N1): N1-type coloured material (chromogen) binds to the tooth surface. The colour of the chromogen is similar to that of dental stains. It's caused by tea, coffee, wine, chromogenic bacteria, and metals.
- Nathoo type 2 (N2): N2-type coloured material changes colour after binding to the tooth. The stains actually are N1-type food stains that darken with time.
- Nathoo type 3 (N3): N3-type colourless material or prechromogen binds to the tooth and undergoes a chemical reaction to cause a stain. N3-type stains are caused by carbohydrate-rich foods (e.g. apples, potatoes), stannous fluoride, and chlorhexidine.

Table 1: Extrinsic causes of tooth discolouration

Classification Non-Metallic stains		Factors responsible	Examples	Colour
	DIRECT STAINS	**Diet**	Tea, Coffee & other foods	Brown to black
		Oral hygiene	Dental plaque, calculus & food particles	Yellow/brown
			Chromogenic bacteria	Brown/black/green Orange
		Habits	Tobacco smoking/chewing Pan chewing	Dark brown/black Red-black
		Medications	Cationic antiseptics e.g.: chlorhexidine	Yellow brown
			Essential oils/ Phenolic mouth rinse	Yellow
			Minocycline Doxycycline	Green-grey Yellow-brown
Metallic stains	INDIRECT STAINS	**Medications**	Iron containing oral solutions	Black
			Copper salt in mouth rinse	Green
			Potassium permanganate in mouth rinse	Violet to black
			Stannous fluoride Silver nitrate	Golden brown Grey
		Occupation and environment	Exposure to iron, manganese, silver	Black
			Exposure to mercury and lead dust	Blue green
			Copper & nickel	Green
			Chromic acid fumes	Deep orange

a)





b)

a) Extrinsic stains on facial surfaces of teeth as a result of calculus stained with kolanut chewing. b) Coffee & Tea stains

Intrinsic Discolouration:

This occurs following a change to the structural composition or thickness of the dental hard tissues [6]. During odontogenesis, teeth may become discoloured from the changes in the quality or quantity of enamel or dentine or from the incorporation of discolouration agents into the dental hard tissues. A number of metabolic, systemic and local factors may affect the developing dentition, giving rise to intrinsic discolouration. Examples of these are alkaptonuria, congenital erythropoietic porphyria, congental hyperbilirubinaemia, Amelogenesis Imperfecta (AI), Dentinogenesis Imperfecta (DI), tetracycline staining, dental fluorosis, enamel hypoplasia etc.

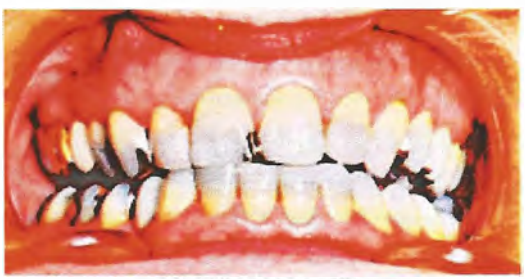
Internalized Discolouration:

This is the incorporation of extrinsic stain within the tooth substance following dental development. It occurs in enamel defects and in the porous surface of exposed dentine [6]. The routes by which pigments may become internalized are developmental defects such as those found in dental fluorosis and acquired defects resulting from tooth wears, gingival recession, dental caries and restorative materials.

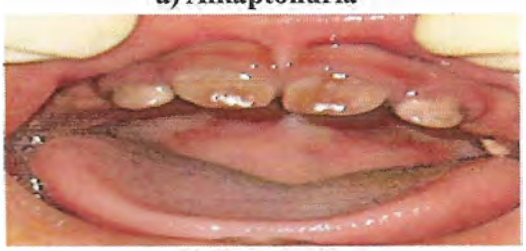
MECHANISM OF TOOTH DISCOLOURATION

Intrinsic Discolouration: The mechanism of formation of intrinsic discolouration differs depending on the specific disorder.

Alkaptonuria: Also known as phenylketonuria or ochronosis. It is an in-born error of metabolism, inherited as an autosomal recessive disorder, resulting in incomplete metabolism of tyrosine and phenylalanine. Deficiency of a hepatic enzyme, homogentisate 1, 2 dioxygenase (HGD) which breaks down homogentisic acid (HGA) results in accumulation of the acid and its oxidized product [28]. The build-up of this acid results in bluish discolouration of the dentition [29].



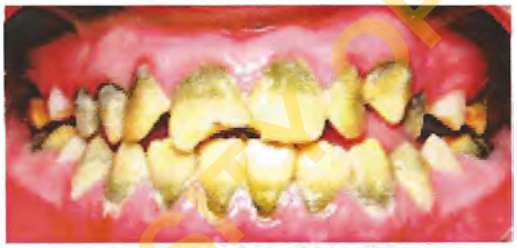
a) Alkaptonuria



b) Porphyria

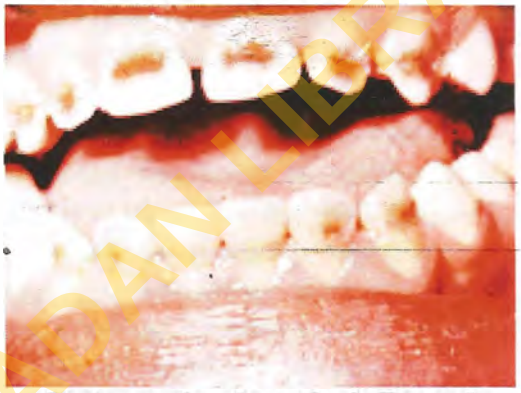
Congenital Erythropoietic Porphyria (Gunther's disease): This is a rare autosomal recessive disorder of porphyrin metabolism, resulting in an increase in the formation and excretion of porphyrins. There is accumulation of porphyrins in bone marrow, red blood cells, urine, faeces and teeth. A characteristic reddish-brown discolouration of the affected teeth (Erythrodontia) results. These teeth show a red fluorescence under ultraviolet light [30,31,32]

Congenital hyperbilirubinaemia: Diseases that can cause hyperbilirubinaemia and intrinsic discolouration include sickle cell anaemia, thalassaemia, haemolytic disease of the newborn (HDN) due to Rhesus factor, ABO, or other erythrocyte antigen incompatibility and biliary atresia [33]. These diseases have the potential to cause haemolysis and the subsequent dose-dependent incorporation of biliverdin into developing teeth, producing yellow-green tinge on the tooth surface [34]



Hyperbilirubinaemia

Enamel Hypoplasia (EH): Disturbance of the developing tooth germ following trauma, infection or nutritional deficiency gives rise to localized or generalized enamel defects [35, 36]. Turner's hypoplasia results from trauma to the developing, yet unerupted tooth, with the localized opacity becoming visible following tooth eruption. Generalized EH may occur in a large number of foetal or maternal conditions such as maternal Vitamin D deficiency, rubella infection, drug intake during pregnancy and in paediatric hypocalcaemic conditions [37]. Such defects will be chronologically laid down in the teeth depending on the stage of development at the time of interference, and the effect is directly related to the degree of systemic upset. Systemic post- natal infections such as measles, chicken pox, streptococcal infections, and scarlet fever can also cause enamel hypoplasia.



Post- measles chronologic Enamel hypoplasia in a 15-year old

Molar-Incisor hypomineralization: An idiopathic condition characterized by severe hypomineralized enamel affecting incisors and permanent first molars. The associated discolouration may vary from white to yellow to brownish areas with sharp demarcation between sound and affected enamel. The possible aetiologies include environmental changes, infections during the early childhood, dioxin in breast milk and genetic factors [38,39].

Genetic Defects and Hereditary Disorders

These include amelogenesis imperfecta (AI), Dentinogenesis imperfecta (DI) and dentinal dysplasia.

Amelogenesis Imperfecta (AI): This is a group of hereditary disorders characterized by abnormal enamel formation, affecting both primary and permanent dentitions [40]. It can be transmitted as an autosomal

dominant, autosomal recessive, X linked or sporadic cases. It may be associated with morphologic or biochemical changes elsewhere in the body. AI is caused by mutations in the genes that control enamel formation. Five different genes have been identified namely ENAM, AMELX, KLK4, MMP20 and DLX3 [41]. Because of the numerous clinical manifestations of this condition, many classifications have been designed. The first classification was introduced by Weinmann et al. in 1945 [40], but the most widely used classification was presented by Witkop in 1988 [40] with the different clinical descriptions given by Ng and Messer in 2009 [41]. This classification which is based on phenotypes and inheritance patterns categorized AI into four main types and then 15 subtypes [41]. Types I -IV amelogenesis imperfecta are the hypoplastic, hypomaturation, hypocalcified and hypomaturation-hypoplastic with taurodontism respectively. All these present various degrees of quantitative or qualitative enamel defects with associated intrinsic tooth discolorations varying from white opacities to yellow or brown discolourations.

Amelogenesis imperfecta

Dentinogenesis Imperfecta (DI): This genetically determined dentine defects may be in isolation or be associated with a systemic disorder. Based on this, three types of DI have been described [42].

Type I - Dentinogenesis Imperfecta (Associated with Osteogenesis Imperfecta, OI)

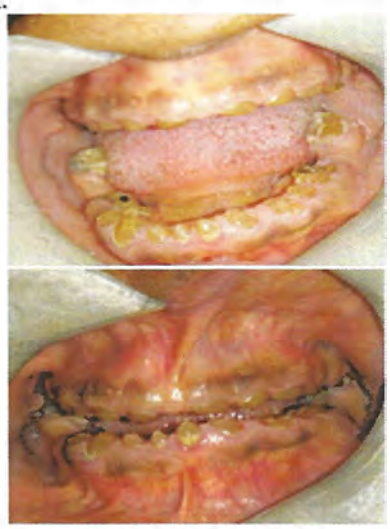
Type II - Dentinogenesis Imperfecta (Hereditary opalescent dentine)

Type III-Dentinogenesis Imperfecta (Brandywine isolate hereditary opalescent dentine)

Type I is caused by mutations in either of the two genes (COLIA1 or COLIA2 genes) involved in the synthesis of collagen type I [43]. The inheritance may be dominant or recessive, the latter being more severe and often fatal in early life. Opalescent teeth are more common in the dominant inheritance pattern.

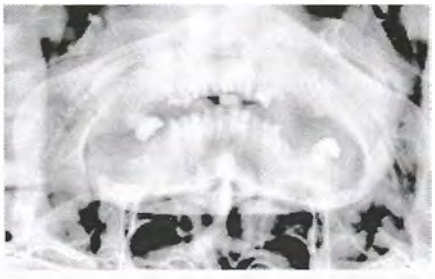
Types II and III are associated with mutations of the dentine sialophosphoprotein (DSPP) gene located among a cluster of four other genes involved in bone and/or dentine formation on chromosome 4 [44].

Teeth with DI show a variety of colours from yellow to brown to blue grey. The colours change depending on whether they are absorbed by transmitted or reflected light. They show a high degree of amber-like translucency. The enamel fractures easily, exposing the dentine which becomes stained readily; thus, the condition affects the colours, forms and the structures of teeth.



34year-old female patient with Dentinogenesis Imperfecta

Radiographically the crown has a characteristic bulbous appearance due to constriction at the cervical portion of the tooth. The roots are short and slender with partial or complete obliteration of the pulp [44].



Orthopantomogram of the 34 year old patient with Dentinogenesis Imperfecta

Dentinal dysplasia (DD): This occurs in two types, DD type I and DD type II. There is a significant overlap between different types of DD and DI. DD type I involves sharp conical short roots or rootless teeth and is associated with premature loss of teeth while type II involves normal tooth roots but abnormal amber colour primary dentition, permanent teeth of normal morphology and colour, with pulp chamber shape anomalies and multiple intra-pulpal calcification.

DD is caused by mutations in the DSPP gene (4q21.3) coding for dentine sialoprotein and dentine phosphoprotein [45].

Tetracycline Discolouration

Tetracycline was first introduced in 1947 [46] as a group of broad spectrum antibiotics for treatment of various infections, but the first cases of tetracycline-induced discolourations were reported by Schwachman and Schuster in 1956 [47]. According to Urist and Ibsen [48], tetracycline and its homologous derivatives have the ability to form complexes with calcium ions on the surface of hydroxylapatite crystals within bone and dental tissues. Dentine has been found to be more susceptible to staining than enamel [49]. The severity of staining is related to the time of the administration and the stage of odontogenesis. The primary teeth begin calcification at about the end of the fourth month of gestation and end at 11 – 14 months of age while calcification of the permanent dentition begins after birth and continues until 8 years of age, except for the third molars [47]. Since tetracycline is able to cross the placental barrier, its administration should be avoided in expectant mothers from 29 weeks in utero, in infants and young children until 12 years of age, as well as in breast feeding mothers [50].

The tetracycline-induced discolouration is permanent and varies from yellow or grey to brown with or without banding, depending on the type of antibiotic used [49]. The affected teeth tend to be yellow on eruption and after exposure to light, the fluorescent yellow staining changes to non-fluorescent brown colour with time, with the anterior teeth being more particularly susceptible to light induced colour changes. The various analogous derivatives of tetracycline produce different colour changes, for instance chlortetracycline produces a slate grey colour and oxy-tetracycline causes a creamy discolouration [51] [52]. Tetracycline stained teeth fluoresce under ultraviolet light, giving off a bright yellow colour.

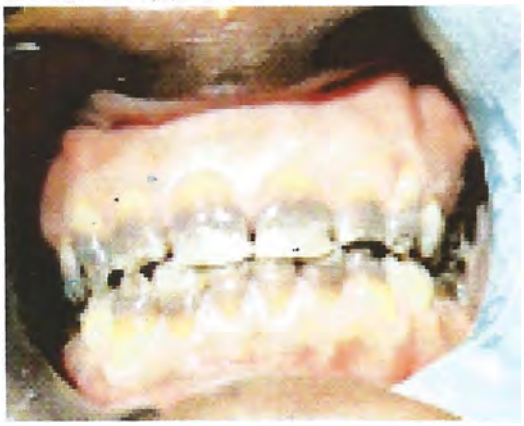
Tetracycline discolouration is classified according to extent, degree and location of the staining [53].

First degree: Mild staining, varies from yellow to grey discoloration with no banding.

Second degree: Moderate tetracycline staining, yellow to dark grey banded staining.

Third degree: Severe tetracycline staining, blue grey or black with significant banding across the tooth.

Fourth degree: Extended and more severe staining.



Severe tetracycline discolouration in a 35year-old male

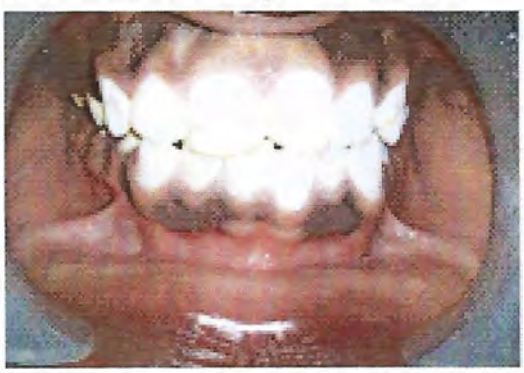
There have been reports of adults experiencing tooth colour change with the use of long term tetracycline therapy [54]. Prolonged ingestion of minocycline - a second generation derivative of tetracycline, used for the treatment of acne vulgaris, respiratory diseases and rheumatoid arthritis can lead to green grey to blue grey intrinsic staining of the teeth.

Minocycline causes staining, not only during tooth development but also after complete formation and eruption of teeth [10]. Minocycline is well absorbed from the gastro intestinal tract and chelates with iron ions to form insoluble salts that are either exuded from gingival crevicular fluid to extrinsically stain or incorporated into the secondary dentine [55].

Ciprofloxacin: a quinolone used in the treatment of Klebsiella infections in infants has been associated with greenish discolouration of the teeth [56].

Dental Fluorosis: This is characterized by enamel discolouration resulting from sub-surface hypomineralization due to excessive ingestion of fluoride during the early maturation stage of enamel formation [57]. The association between fluoride intake and its effect on enamel was noted by Dean HT as far back as 1932 [58]. Dental fluorosis may occur endemically from naturally occurring water supplies or from fluoride from other sources such as mouth rinses, tablets, toothpaste, salt, milk, gels, varnishes, fresh water fish and tea. In a survey by Ajayi and Solanke (2015)[59] among selected 12 – 14 year old students in Ibadan, early tooth brushing with fluoride toothpaste and inappropriate use of fluoride supplement were the two factors found to be significantly associated with the condition. The ingestion of fluoride is particularly important in infants as dental fluorosis can only originate during tooth development with the critical stage translating to a period from birth to age 8 in a child[60]. The mechanism of formation of fluorosis has been suggested to be due to delayed withdrawal of amelogenins- the enamel proteins which cause inhibition in the growth of enamel crystals, so that when the tooth erupts, the enamel remains incompletely mineralized [61]. The proteolytic activity of serine proteases which are responsible for the hydrolysis and removal of these enamel proteins was found to be reduced in fluorosed compared to control enamel [62].

The clinical appearance may vary based on the severity, from areas of enamel flecking to diffuse opaque mottling, while the colour of the enamel ranges from chalky white to a dark brown or black appearance. The black/brown discolouration is post eruptive and probably caused by internalization of extrinsic stain into the porous enamel [63].



Dental fluorosis in a 14 year- old patient

Trauma: Acute trauma to an erupted tooth can cause intra-pulpal haemorrhage giving the tooth a reddish tinge. This discolouration can change to grey – brown in a matter of days as the pulp becomes necrotic [27]. Haemolysis of the red blood cells would follow and release the haem group to combine with the putrefying pulp tissue to form black iron sulphide, giving rise to very dark brown discolouration which is deposited along the dentinal tubules producing a bluish black cast [6]. The discolouration of a necrotic tooth caused by trauma will become more severe over time. In our environment, many of traumatized anterior teeth go untreated, thus resulting in progressively darker discolourations. A study conducted by Ajayi and colleagues [64] among 1532 secondary school children, aged 12 -19 years revealed that out of 165 children that had traumatized their anterior teeth, only nine (5.5%) consulted the dentist following trauma. The average time lapse between trauma and dental consultation was 3.5 years.

Luxation injuries following trauma may also lead to excessive dentine formation and narrowing of the pulpal space. This is called calcific metamorphosis which results in yellowish or yellow brown discolouration [65]. Root resorption following trauma is often clinically asymptomatic, however, a pink spot lesion may be present at the cement-enamel junction (CEJ) giving rise to what is known as "pink tooth of mummery" [27].

Ageing: A general darkening of tooth colour naturally occurs with age, as a result of deposition of secondary dentine. It has been hypothesized that pigments and ions of an amorphous organic and inorganic nature permeate through the enamel, depositing at the dentine-enamel junction and within the dentine structure [66]. Combined with an ever-decreasing enamel thickness as a result of normal wear, the dentine colour begins to dominate anterior tooth shade [66].

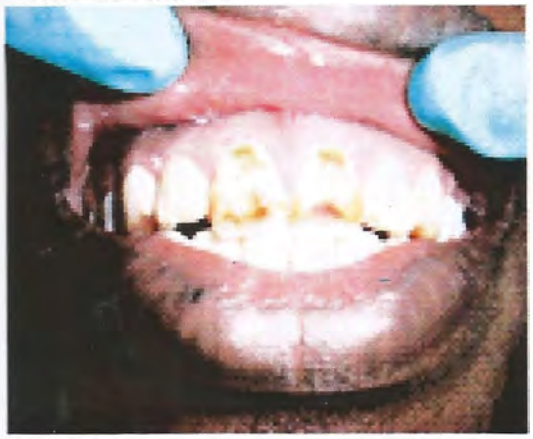
MECHANISM OF EXTRINSIC DISCOLOURATION

The interest in the mechanisms of extrinsic tooth staining was rekindled in 1971 with the observation by Flotra et al [67] that tooth discolouration increases with the use of chlorhexidine. Prolonged use of chlorhexidine, Listerine and delmopinol mouth rinses gives rise to characteristic staining of the tongue and teeth. Though different theories for extrinsic staining have been proposed, most evidence indicates that the likely cause of staining is the precipitation of anionic chromogens unto adsorped cations. Thus, polyphenols found in dietary substances being anionic are able to react with cations adsorped to surfaces such as the cationic antiseptic or polyvalent metal ions to produce staining [24]. Tannins found in tea, coffee and other beverages bind to pellicle and form brown discolouration, even if the teeth are relatively plaque free [2].

INTERNALIZED DISCOLOURATION

Dental defects (developmental or acquired) may permit the entry of chromogenic materials into the body of enamel or dentine.

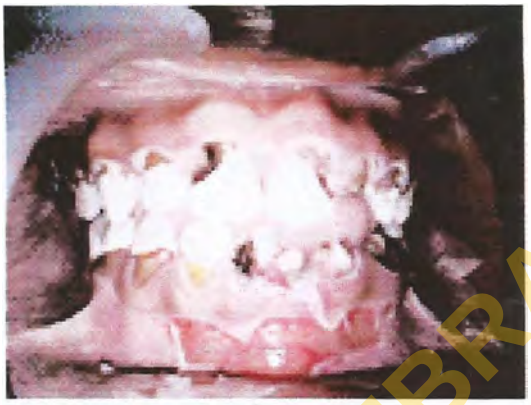
Developmental defects: These defects create their own colour change in the tooth, caused by influences on light transmission through the enamel and dentine. However, extrinsic stains can be taken up by these defects post eruptively either due to increased porosity as found in fluorosis or hypoplasia, or the defects may expose dentine either directly or later caused by early loss of enamel as in dentinogenesis imperfecta [6]



Dental fluorosis with internalized stains

Acquired defects: Dental conditions can directly or indirectly cause tooth discolouration. These defects include:

Dental caries: Dental caries is a bacterial infection of multi-factorial aetiology that damages the structures of a tooth. The pathogenesis of dental caries begins with an incipient lesion confined to the enamel layer. Incipient carious lesions are associated with plaque accumulation and manifest as chalky white areas of discolouration secondary to demineralization. As caries progresses into the dentine, the overlying translucent enamel reveals the colour of the underlying caries and appears yellowish brown. Extensive caries that destroys both enamel and dentine produces a colour that ranges from light dark brown or almost black. The hard arrested lesion is black, having picked up stain from exogenous sources [68]. The brown discolouration of dental caries has been attributed to the formation of Maillard pigments (reaction between proteins and small aldehydes produced by cariogenic bacteria), melanins, lipofuscins and uptake of various food colours and bacterial pigments [69,70]



Rampant caries in a child patient

Tooth wear and gingival recession: Tooth wear is a progressive loss of outer layers of enamel and dentine caused by erosion, attrition and abrasion and thus revealing the yellow colour underlying dentine while gingival recession is a condition characterized by retraction of the gingival margin, thereby exposing the more yellowish root surface of the tooth.

Restorative materials/Iatrogenic causes: Some materials used in tooth restoration may have an effect on the colour of teeth. Amalgam restorations can generate corrosion products (e.g. silver sulphide), leaving a grey black colour in the tooth, especially in large cavity preparations with undermined enamel. Electron microscopic studies have shown that this discolouration is caused by the migration of tin into the dentinal tubules. Eugenol and phenolic compounds used during root canal therapy (RCT) contain pigments which may stain the dentine. Some of the poly antibiotic pastes used as intra canal medicaments may cause a darkening of the root dentine [71].

Table 2: Causes of intrinsic and internalized discolouration

Intrinsic Discolouration		Internalized Discolouration	
Condition	**Colour**	**Condition**	**Colour**
Alkaptonuria	Bluish	**Dental Caries**	
Porphyria	Reddish brown	**Incipient**	Chalky white
Hyperbilirubinaemia	Yellow – green	**Active**	Yellowish brown
Turner's Hypoplasia	Localized opacity	**Arrested**	Dark brown
Molar Incisor Hypomineralization	Yellow to Brown	**Restorative Materials**	
Amelogenesis Imperfecta	White opacity to yellow – brown	**Amalgam**	Greyish black
		Composite/GIC	Yellowish brown
Dentinogenesis Imperfecta	Blue Brown	**Intra-canal Medicament e.g. Iodoform, ledermix**	Brownish grey
Dentine Dysplasia	Yellow	**Obturating Materials and sealers**	Greyish
Tetracycline	Yellow, brown, dark grey, bluish grey	**Tooth wear**	Yellowish
Minocycline	Green grey to blue grey		
Ciprofloxacin	Green		
Dental fluorosis	Chalky white to Dark brown/black		
Trauma			
Pulpal Necrosis	Greenish Brown		
Internal Resorption	Pinkish		
Pulpal Calcification	Yellowish		

PREVALENCE OF TOOTH DISCOLOURATION

Some studies have documented the prevalence of tooth discolouration generally while several others reported on specific aetiologic factors. These studies are either community or hospital based. Many of the community based studies on general prevalence were on participants' self-perception of tooth discolouration. For example Xiao et al. (2007) [73] reported that 48.9% of adult and teenage Chinese population perceived that they had tooth discolouration while the figure was 50% among the general public in the United Kingdom [74] and 24.5% among adolescents in a Nigerian rural setting [75]. However, no epidemiologic data is available on prevalence of tooth discolouration in the USA, though an increase in the prevalence of mild to moderate fluorosis has been observed in the United States of America even in areas with non-fluoridated public water supplies [76].

Ajayi et al found a prevalence of dental fluorosis to be 11.4% among school children aged 12 – 14 years [77] in Ibadan. Ibadan is a non-fluoridated city with fluoride

concentration of 0.02 – 0.03mg/L in different drinking water sources (Ajayi, Denloye and Dosumu) [78]. Other epidemiologic prevalent studies on specific aetiological factors are shown in the table 3 below.

Table 3: Tooth Discolouration Prevalence as reported by Different Authors

	Umesi-Koleoso[79] 2004	Koleoso el al[80] 2004	Faezeh et al[81] 2008	Ibiyemi and Taiwo[75] 2011
Dental Fluorosis	36.5%	-	0.3%	7.0%
Tetracycline Stain	21.5%	9.2%	-	3.6%
Enamel Opacities	16.0%	23.0%	-	-
Enamel Hypoplasia	7.0%	-	19.8%	1.8%
Trauma	2.5%	-	0.8%	-
Pulp Necrosis	-	0.8%	-	2.1%
Dental Caries	-	-	71.1%	-
Extrinsic	-	-	24.7%	16.7%

A clinic based study by Gbadebo and Ajayi [82] analyzed the different causes of tooth discolouration in a 3 year review of patients with discoloured teeth. It was observed that pulpal necrosis accounted for 87%, (a finding further buttressing the delayed presentation in our environment) followed by enamel hypoplasia7% and tetracycline stain 2%. Tetracycline stain was also diagnosed in 2.2% of the 3750 patients seen **Table 3: Tooth Discolouration Prevalence as reported by Different Authors** in Benin City, Nigeria [83].

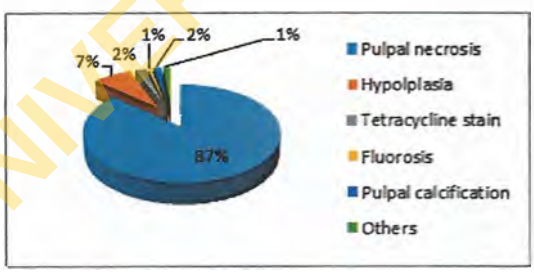


Fig 2: Distribution of types of tooth discolouration seen over 3 years at the Conservation Clinic UCH.

EFFECTS OF TOOTH DISCOLOURATION

Dental appearance is an important factor in facial attractiveness and may therefore contribute greatly to human social interactions. Many people desire to have teeth without spots or blemishes. Tooth colour has been found to be one of the most important factors determining satisfaction with dental appearance[2]; consequently tooth discolouration can have negative, psychological, social and clinical implications especially when it affects the anterior teeth. It has been reported, depending on age, that 12.1 – 15.5% of UK adult population were dissatisfied with the appearance of their teeth and 17.9 – 21.3% were dissatisfied with their tooth colour [84]. In USA [85] and China [73], 34% and 52.6% respectively were found to be dissatisfied with their tooth colour. However, in a preliminary study on tooth colour perception and general dental appearance among dental patients in UCH, Ibadan, 63.6% were satisfied with their tooth colour and 54.5% with their dental appearance [86]. Dissatisfaction with colour may be a

primary reason for dissatisfaction with dental appearance.

Tooth discolouration produces profound embarrassment and more psychological distress [87]. Negative emotions such as fear, anxiety, depression and timidity are sometimes exhibited in cases of anterior tooth discoloration. This can affect the general health and ability of an individual to participant fully in society, fulfilling roles as family members, friends, coworkers and other roles requiring interaction with others [73]. Furthermore, Shaw (1981) [88] found that children with normal dental appearance are judged to be better looking, more desirable as friends, more intelligent and less likely to behave aggressively.

Tooth sensitivity and pain could result from discolouration caused by toothwear lesions, hypoplasia and dental caries.

ARRIVING AT A DIAGNOSIS

The patient's chief complaint could be dissatisfaction with tooth colour, surface texture, tooth contour/form or a combination of all these. Some patients may present with pain and discolouration resulting dental caries, trauma that leads to pulpal necrosis, severe developmental or acquired defects. In all these, a thorough history[89] is very essential and this includes:

- **Medical History** (Serious illnesses, fevers, periods of hospitalization, use of tetracycline and other antibiotics during the period of tooth development)
- **Residence History** (Systemic fluoride, trace elements in the water supply, period spent at the present residence)
- **Family History** (Genetic disorders, affected siblings, parents or offspring)
- **Dental History** (Previous dental treatments, episode of trauma, endodontic pathology or treatment)
- **Chronology** (When first noticed, whether primary and permanent dentitions are affected)
- **Oral Hygiene habits** (Type of dentifrice, frequency of toothbrush, use of mouth rinses)
- **Diet History** (Nutritional deficiencies or ingestion of foods that can stain teeth e.g. coffee, tea)
- **Social History** (Smoking, occupational exposure to metals)

It is important to assess patient's expectation as regards treatment outcome in terms of tooth colour and contour. It will be necessary to find out:

- How important is dental appearance to the patient's occupation?
- What is patient's level of awareness of cosmetic dental procedures?
- If the patient is prepared to make all the necessary efforts to keep the required number of visits.

The clinical examination should also include the following:

Scratch Test: To distinguish between extrinsic and intrinsic discolouration. Light scratching with dental explorer or scaler will remove adherent plaque causing extrinsic discoloration.

Ultraviolet Light: Under UV light, teeth with tetracycline staining and congenital porphyria will fluoresce yellow and red respectively.

Clinical photograph: To record baseline information to allow later comparison or for medico-legal reasons.

RESTORING THE COLOUR, FORM AND STRUCTURE

The colour, form and structure of the affected teeth can be successfully restored if the aetiology is correctly identified, mechanism of formation clearly understood and the required therapy professionally implemented. Various methods and

approaches that can be used include whitening toothpaste, professional cleaning to remove stain and tartar, micro abrasion of enamel with abrasives and acids, placement of crowns and veneers and finally tooth extractions and replacement with dentures or implant-retained prostheses in severe cases with defective form and structure.
Extrinsic staining caused by foods, (e.g. carotene in oranges and carrots), beverages, or habits such as tobacco smoking or chewing is treated through scaling and polishing either manually or ultrasonically. Also, cessation of dietary or other contributory habits should be encouraged to prevent further staining.

Tooth brushing: Effective tooth brushing with dentifrice especially those containing tooth whitening agents (1% or less hydrogen peroxide) helps to prevent further staining.

Micro abrasion: This technique involves multiple applications of a mixture of a weak acid (e.g. 18% hydrochloric acid) and abrasive pumice slurry[90] using a rubber cup on a slow hand-piece. The resultant surface is smooth and has a glazed appearance. It is indicated in the treatment of superficial discolourations limited only to enamel e.g. mild fluorosis. It is simple, quick to perform, cost effective and well tolerated by patients. Micro abrasion technique using either a fine diamond or carbide-finishing bur at relatively low speeds and very light pressure is equally effective.

Tooth Bleaching/ Whitening:
Just as many people and races believe that lighter skin colour is more attractive and beautiful than a dark skin, so it is for tooth colour, the whiter, the better. It should therefore not surprise anyone that some individuals with acceptable shades of skin and tooth colour can go to any length to make their skin fairer and their teeth whiter still! Tooth bleaching is based upon the use of hydrogen peroxide (H_2O_2) as an active agent, applied directly or produced in a chemical reaction from sodium perborate or carbamide peroxide. H_2O_2 acts as a strong oxidizing agent through the formation of free radicals, reactive oxygen molecules and hydrogen peroxide anions. These reactive molecules attack the long chained, dark coloured chromophore molecules and split them into smaller less coloured and more diffusible molecules. In essence, hydrogen peroxide causes decolourization or bleaching of the coloured materials found within the tooth structures giving rise to whiter teeth[91]. H_2O_2 or carbamide peroxide can be applied using paint on, mouth guard or strip product formats.

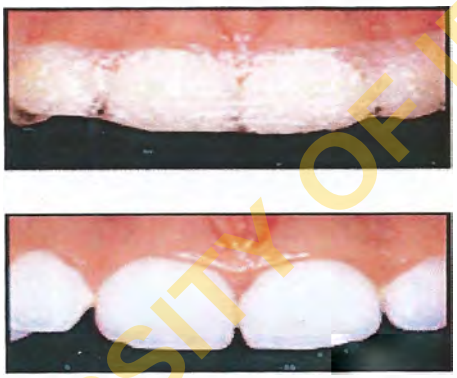
Tooth bleaching procedures are categorised into two:

Vital Teeth Bleaching Techniques[92]
Vital tooth bleaching is indicated primarily for patients with generalized yellow, orange or light brown extrinsic discolouration including chlorhexidine. For intrinsic discolourations, tetracycline stained teeth are the slowest to respond to bleaching, brown fluorosed teeth are moderately responsive and teeth discoloured by age, genetics, smoking or coffee are the fastest to respond[93,94]. The procedure can be done at home by the patient or professionally applied by dentists.

Home Bleaching—bleaching agent is applied using night guard/ matrix or polyethylene strips.
Matrix—Patient uses 10-22% carbamide peroxide in a custom-made tray.
Polyethylene whitening strips—these are 5.3% hydrogen peroxide impregnated polyethylene strips.
Many patients find home bleaching to be very convenient and simple to apply with satisfactory outcome especially if the

patient is compliant, following the dentist's and manufacturer's instructions. However, patients should be made to understand the side effects of bleaching which include tooth sensitivity and cervical external root resorption.

Vital bleaching matrix

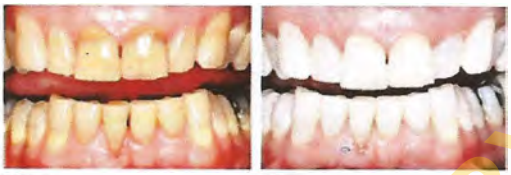


Polyethylene Whitening Strip

In- office Bleaching is sub-divided into:

– **Thermocatalytic**—uses 35-40% H_2O_2 professionally applied under rubber dam isolation while the agent is activated by heat or light such as Tungsten halogen curing light, Xenon plasma arc light, CO_2 laser, etc.

– **Non Thermocatalytic**—Heat source is not used. The bleaching agent is modified McI'nnes solution[95].



Before and after vital teeth bleaching

Non Vital Teeth Bleaching Techniques[96]

Non- Vital bleaching is indicated for the treatment of teeth with discolouration secondary to pulpal degeneration.

The procedure can be done through:

-Thermo catalytic in- office bleaching—30% H_2O_2 + Heat.

-Walking bleaching/Intra-coronal (uses a mixture of 30% H_2O_2 + Sodium Perborate).

- Inside/Outside Bleaching Technique (Modified Walking bleaching Technique). This involves intra-coronal bleaching of an endodontically treated tooth along with home bleaching of the whole arch.

-Closed Chamber/Extra-coronal bleaching. It is indicated for totally calcified tooth, maintenance bleaching and in adolescent with incomplete gingival third calcification. It is done via a bleaching tray.

Pretreatment and after 2nd visit Non Vital teeth bleaching

Laser Assisted Bleaching: This technique achieves power bleaching with the help of efficient energy source with minimum side effects. Laser bleaching gel contains thermally absorbed crystals, fumed silica and 35% hydrogen peroxide. Lasers used

include Argon laser, CO_2 and GaAlAs (Galium Aluminium Arsenics) diode laser.

Bonded Restorations:

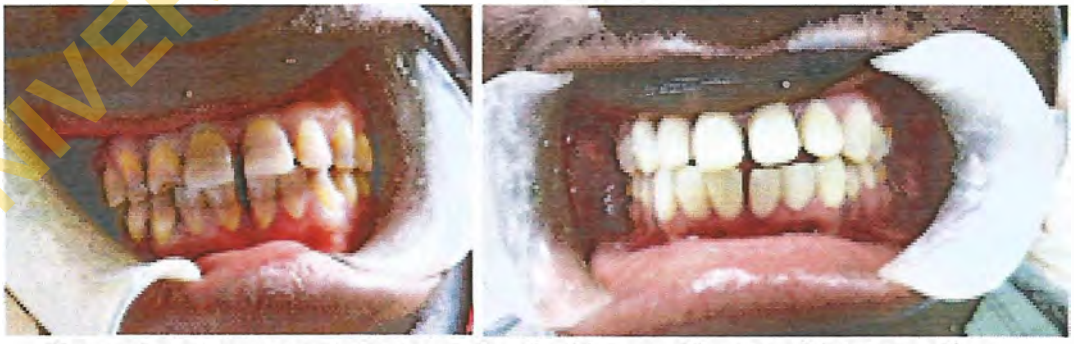
Discoloured teeth may be treated with bonded restorations to cover or disguise underlying stain. Such restorations include direct composite resin veneers, preformed acrylic laminates, laboratory fabricated acrylic or composite resins veneers, porcelain and glass ceramic veneers[97] to the more invasive all ceramic or porcelain fused to metal crowns.

Veneers are sheets/shells of aesthetic restorative materials bonded to the labial surface of a tooth while crowns are cemented extra-coronal restorations that cover the outer surface of a clinical crown. They may be used to treat generalized intrinsic discolouration in which bleaching is not indicated or in which aesthetic results of bleaching fail to meet the patient's expectations.

The discoloured teeth can first be treated with micro abrasion or macro abrasion to remove discoloured areas, aesthetic composite resin or porcelain veneers are then bonded to provide good colour and form. However, direct composite restorations have limited longevity because of increased susceptibility to staining, wear and marginal fractures.

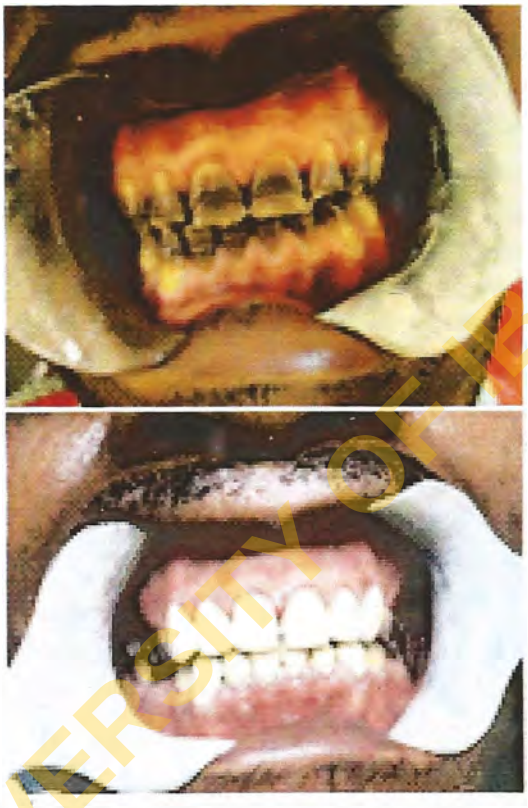
Severe dental fluorosis treated with micro-abrasion and direct composite facing in a 13 year old male patient

On the other hand, porcelain or other all-ceramic veneers have been rated the best [98] because they are resistant to wear, provide excellent shade match with natural teeth, are not susceptible to discolouration and the highly glazed surface does not irritate the gingiva [98].



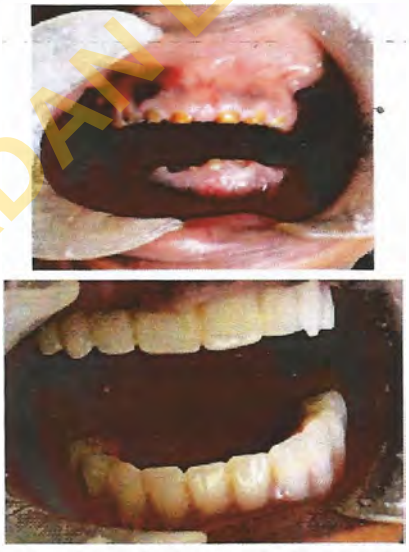
Pre and post treatment photographs of a 32 year old man with tetracycline stain restored with veneers

With the increase in patient's demand for treatment of unaesthetic discoloured teeth, full crowns are the most predictable and durable bonded restorations. They are, however, the most invasive with substantial removal of sound tooth substance during tooth preparation. They are used mainly for very severe intrinsic tooth discolouration.

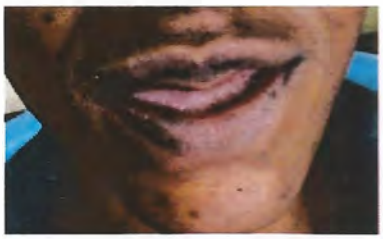


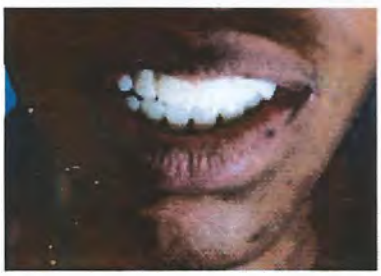
Pre and post treatment photographs of a 35 year old man with tetracycline stain restored with full crowns.

Dental Extractions: Though a restorative dentist usually does all that is possible to make sure a tooth remains functional in the dental arch, a time comes when this is no longer feasible, such teeth are then sent for extractions. Such situations arise in patients with severe AI or DI or some cases of rampant dental caries. All the same, all hope is not lost, since the replacement of such extracted teeth is still possible with dentures or implant-retained prostheses.



Pre treatment and post treatment photographs of the patient with Dentinogenesis imperfecta with a removable partial denture





The 34 year old patient pre and post treatment Smiles.

Dental Implant: Dental implantology is one of the defining advances in clinical Dentistry. It is a device inserted into or on the jaw bone to anchor artificial tooth or prosthesis. It provides a predictable way of restoring function and aesthetics. The most recent commonly used implant in modern implantology is the osseointegrated implant introduced by Branemark.

Advantages of implant retained prosthesis when compared to the other forms of tooth replacement are preservation of adjacent teeth, bone stabilization and maintenance, retrievability, high predictability, psychological and functional improvement and longevity. Implant may actually last for a life time. However, the major problems with implant are the cost and the involvement of surgery.

CONCLUSION AND RECOMMENDATIONS

My observation and that of several others in dental profession was that most people visit the dentists only when they are in pain. Many people go around with unsightly appearance of their discoloured teeth and consciously try to hide them while talking. Many go around with poor oral hygiene even among the elites. Though some may not be aware that help can be sought, others are not bothered, while many that seek dental treatment do not come back once they know the financial implication of the procedure and worse still, few believe that it is their identity.

Sir, let me point it out again that almost, if not all the causes of extrinsic tooth discolouration are acquired and several of the intrinsic aetiologies are preventable. It is interesting to know that as far back as 2004, it was reported[99] in the USA that about 30% of the patients were already using bleaching and tooth whitening at home; you can imagine what the figure is now? For us in Nigeria, what then may be the way forward?

I. The professional body should intensify efforts on oral health education and awareness programme. These should include measures at preventing tooth discoloration as discussed in this lecture.
II. The enlightenment programme should also include information on the available safe methods of enhancing the dental aesthetic in order to boost the people's self-confidence, self-esteem and social functioning.
III. Those in the medical and dental professions should take the lead and pay more attention to our oral health care.
IV. The treatments should be made more affordable and the procedures simple, noninvasive and non-stressful.
V. Consumables should be available for proper training of our undergraduate students who will take over the management of these cases as awareness improves.

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