

## Etiology of Discolourations of Tooth

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**Abstract:** Differentiation of the quality and cause of the stain is of more than academic interest, as knowledge of the etiology helps the dentist to better plan the technique for whitening and also to accurately predict the outcome of the treatment. This article reveals etiology of discoloration.

Date of Submission: 26-04-2019

Date of acceptance: 11-05-2019

### I. Introduction

The pellicle is easily stained and displays many colours ranging from white to green and can become extremely opaque depending on the source of the pigmentation.

#### Classification:

Staining and discoloration are divided into two main categories Intrinsic and Extrinsic staining<sup>1,2,3</sup>.

#### Intrinsic:

These are the result of color changes of internal structures of teeth caused by **i)** Systemic factors, **ii)** Local factors. Intrinsic stains are distributed throughout the tooth and are again divided into:

- A) Those caused during odontogenesis.
- B) Those occurring post-eruptively.

During odontogenesis causes are further classified into Hereditary And Non-Hereditary. Hereditary condition includes Amelogenesis Imperfecta, Dentinogenesis Imperfecta, Alkaptonuria, Sickle cell anemia, Thalassemia and Porphyria while Non-Hereditary condition includes Drug Toxicity-Tetracycline, Fluoride Toxicity -Fluorosis, Erythroblastosis Fetalis.

Post-eruptively reasons are further divided into Physiological and Pathological. Physiological condition like Habits (diet, chewing and smoking) and Ageing. Pathological conditions are divided into Pulpal and Non-Pulpal. Pulpal causes are Traumatic and Non-Traumatic. Non-pulpal causes are mostly Iatrogenic restorations and root canal treatment.

#### Hereditary Disorders:

##### i) AMELOGENESIS IMPERFECTA;

This condition affects both the primary and permanent dentition and can be divided into three groups<sup>4</sup>

- a) HYPOMATURATION; enamel chips away from underlying dentin. Enamel exhibits normal thickness but is soft.
- b) HYPOCALCIFIC; Enamel completely abrades away after eruption leaving the crown color ranging from a dull opaque white to a dark brown. They are rough and pitted.
- c) HYPOPLASTIC; Enamel is thin to the point of eliminating interproximal contacts. They have a smooth hard yellow appearance showing occasional pitting.



**Clinical Relevance;**

Vital bleaching is contra-indicated. Full coverage is mandatory for teeth with insufficient, weak or abraded enamel.

**Dentinogenesis Imperfecta;**

It is the most common hereditary dystrophy that affects primary more seriously than permanent dentition. Clinical crown appear reddish brown to gray opalescence. Enamel is friable and break soon after eruption. Exposed softened dentin abrades away.



**Clinical Relevance;**

Vital bleaching is contra-indicated due to thin non-existent enamel.

**Alkaptonuria;**

It is a rare condition causing dark brown pigmentation of permanent teeth.

**Clinical Relevance;**

Vital bleaching decreases or eliminates discoloration. In severe cases additional veneering may be needed.

**Sickle Cell Anemia and Thalessemia;**

Both are inherited blood dyscrasis with discoloration ranging from brown to greenish-blue.

**Clinical Relevance;**

Condition is not a self-treating and stains do not improve with time unlike erythroblastosis fetalis. Bleaching with the addition of veneer for more severe cases is indicated.

**Porphyria;**

It is Genetically transmitted disorder of porphyrin metabolism. Hematoporphyrin pigment creates a reddish brown discoloration of the teeth also known as “erythrodontia”. More common in primary than in permanent dentition. Coloration distributed throughout enamel, dentin and cementum and fluoresces red UV light.

**Clinical Relevance;**

Responds to bleaching in combination with veneering.

**Non-Hereditary Disorders;**

**Erythroblastosis fetalis;**

It is a hematological disorder of the neonate results in discoloration of the teeth ranging in color from brown to greenish blue.

**Clinical Relevance;**

No treatment is necessary as condition resolves with time.

**Endemic Fluorosis;**

It is a condition caused by excessive intake of fluoride. Appearance ranges from slight wisps and flecks of opaque white to mottled or pitted darkened section to normal looking areas<sup>6</sup>. It is considered to be a form of enamel hypoplasia due to ametabolic alteration of ametoblasts. Degree of severity of staining is directly

proportional to the amount of fluoride absorbed and teeth can be affected from second trimester in utero through age nine.



Clinical Relevance;

Simple Fluorosis;

Appears as brown pigmentation on smooth enamel surface. Responds well to bleaching.

OPAQUE FLUOROSIS;

Appears as flat gray or white flecks on the enamel surface. Responds to poor bleaching as teeth cannot be brought to the shade of lightness in the affected area.

#### **Drug Toxicity - Tetracycline;**

First reported by Schwasman and Schuster in 1956. Tetracycline crosses placental barrier and affects both dentition. It binds calcium phosphate complex. Greatest concentration is found in Dentino-Enamel Junction. Teeth exposed to light show greater discoloration of facial surface of anterior teeth. Fluorescence necessary for precise diagnosis and description. Three categories were first proposed by Jordan and Booksman<sup>7</sup>.

#### **First Degree Tetracycline-Staining**

CLINICAL RELEVANCE;

Light yellow brown or gray staining. Uniformly distributed throughout the crown and no evident banding or localization. Responds well to bleaching in 3 to 4 sessions.

#### **Second Degree Tetracycline-Staining**

CLINICAL RELEVANCE;

It has a dark or grey staining. Although stains are more extensive, they are still uniform and have no banding.

#### **Third Degree Tetracycline-Staining**

CLINICAL RELEVANCE;

It has a dark gray or a blue staining usually with marked banding. Responds to bleaching but bands evident even after extensive treatment.

#### **Intrinsic Discoloration Caused Post Eruptively**

**Ageing;**

Excellent example in which extrinsic discoloration combines with more intrinsic physiologic changes. Natural process of gradual pulp withdrawal with the formation of secondary dentin causes the development of yellowish brown color.

Clinical Relevance;

Strongest indication for tooth whitening. Results are most rapid and predictable. Standard vital bleaching used for these cases.

#### **Foods and Beverages;**

Some of the best known causes of staining are smoking, chewing tobacco, tea and coffee. Degree and quality of the staining directly related to the type, frequency, length and quality of exposure to the staining agents.

Clinical Relevance;  
Standard vital bleaching produce excellent dramatic results.

### **Pathological Causes**

#### **Pulpal causes- Traumatic<sup>8</sup>**

##### **Necrosis;**

Pulp degeneration without hemorrhage also results in necrotic tissue that contains various protein degradation products. These products create a grayish brown discoloration of the crown though the discoloration is not as pronounced as with hemorrhage.

Clinical Relevance;  
Responds well to non- vital bleaching techniques.

##### **Hemorrhage;**

Severe trauma causes hemorrhage as blood vessels rupture in the pulp chamber and this blood is driven hydraulically into the pulp chamber. Blood will undergo hemolysis emitting hemoglobin(Hb). The released Hb, is further degraded releasing iron that forms a black compound by combining with hydrogen sulfide to form iron sulfide.

##### **Clinical Relevance;**

Immediately after injury the crown remains pink. As blood progressively breaks down the tooth becomes orange then brown, blue or black. It responds well to non-vital bleaching techniques. The degree of discoloration in these teeth is directly related to the length of time between pulp death and the treatment whether or not hemorrhage is involved. The longer the discoloring compounds in the pulp chamber, the deeper the penetration into the dentinal tubules and greater the discoloration, consequently more difficult the bleaching task.

#### **Non Traumatic**

##### **Idiopathic Pulpal Recession;**

A tooth can occasionally display idiopathic pulpal recession. They remain vital but display a yellow to brown darkening. Appearance is of a non-vital tooth, but actual condition of the tooth can be differentiated by vitality testing. Such teeth show greatly diminished pulp chamber size radio-graphically.

Clinical Relevance;

Standard vital bleaching techniques done if overall whitening is desired. This removes discoloration of the tooth due to idiopathic pulpal recession as well as whitening the neighbouring teeth. In case of multiple restorations heat/ peroxide bleaching systems are effectively used. Veneering is also an alternative method.

##### **Non-Pulpal Causes;**

Dental Metals;

Silver amalgams cause grey to black stains. Pins cause blue grayish stains. Metal amalgams can reflect discoloration through the enamel

##### **Clinical Relevance**

Bleaching may be unnecessary if the amalgams are changed Otherwise veneering is preferred.

Non alloy materials and Medications;

Materials and Medications: used in dental restorations can themselves cause staining if they leak or otherwise reach the dentinal tubules. Break down of restorations such as acrylic, silicate cements or composite resin can cause the tooth to look grayish and discolored. Volatile oils cause yellowish brown stains.

Clinical Relevance;

It responds well to bleaching, following replacement of degraded restorations.

Root Canal Treatment Practices;

Trauma caused during pulp extirpation in the course of endodontic treatment, in turn causes breakdown of hemoglobin. Failure to remove all pulp remnants or residual tissue in the pulp during root canal treatment is a frequent cause of discoloration

Root Canal Materials.

Sealers containing silver, volatile oils causes black, yellowish and brown stains respectively. Medicaments and materials used can also cause discoloration.

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Prof.Dr.K.Vinayagavel M.D.S" Etiology of Discolourations of Tooth" IOSR Journal of Dental and Medical Sciences (IOSR-JDMS), vol. 18, no. 5, 2019, pp 89-93.