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A Brief Note on Dental Hypersensitivity

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Commentary

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DESCRIPTION

Dentin hypersensitivity is a type of short-term dental discomfort that arises from exposed dentin surfaces in reaction to stimuli that are typically thermal, evaporative, tactile, osmotic, chemical, or electrical, and that cannot be attributed to any other dental illness. Dentin sensitivity is common, but pain is rarely felt in regular activities like as sipping a cold beverage. Although the phrases dentin sensitivity and sensitive dentin are interchangeable when referring to dental hypersensitivity, the latter is the more correct term.

INTRODUCTION

The true cause of dentine hypersensitivity is a subject of debate. The odontoblastic transduction theory,' the 'neural theory,' and the 'hydrodynamic theory' have all been proposed in an attempt to explain the reason of dentine hypersensitivity. Brannstrom proposed the hydrodynamic or fluid movement hypothesis in 1964, and it is the most widely recognized model. The flow of fluid within the tubules is increased when the exposed dentine surface is exposed to temperature, chemical, tactile, or evaporative stimuli, according to this notion.

Fluid can flow away from or towards the pulp in the dentinal tubules. Dentine is made up of millions of microscopic tubular structures that radiate outwards from the pulp and are typically 0.5-2 micrometers in diameter. Changes in the flow of the plasma-like biological fluid present in the dentinal tubules can elicit a pain response by triggering mechanoreceptors on nerves located at the pulpal aspect. Cold (air pressure), drying, sugar, sour (dehydrating chemicals), or pressures operating on the teeth can all increase hydrodynamic flow. Teeth sensitivity is commonly triggered by hot or cold foods or drinks, as well as physical pressure. Triggers such as cold and drying can drive

dentinal fluid to flow away from the pulp, while heat can lead it to move towards the pulp. According to studies, stimuli that cause dentinal fluid to shift away from the pulp evoke a greater painful response.

Rapp et al. proposed the odontoblastic transduction theory, in which odontoblasts operate as receptor cells that conduct impulses *via* synaptic connections to the nerve ends, causing pain. However, there isn't a lot of evidence to back this claim. Thermal or mechanical stimuli can directly alter nerve terminals within the dentinal tubules *via* direct communication with pulp nerve endings, according to the neural theory.

Gingival recession and tooth wear are two frequent ways for dentine to become exposed. Gingival recession (receding gums), which exposes root surfaces and results in the loss of the cemented layer and smear layer, is the most common cause of DH. Receding gums can be a sign of chronic periodontitis or a sign of long-term harm from excessive or aggressive tooth brushing or abrasive toothpaste (dental abrasion) (gum disease). Acid erosion, which is the loss of hard tooth tissues owing to acids, is a less prevalent cause. It can be caused by gastric reflux syndrome, bulimia, or excessive ingestion of acidic foods and beverages.

When teeth are exposed to a low pH on a regular basis, the mineral content on the outer layer of enamel dissolves, exposing the dentine and causing hypersensitivity. Other factors include dental whitening, tobacco usage (which can induce recession and thus sensitivity), fractured teeth, and abfraction (grinding). Abfraction lesions, which are wedge-shaped abnormalities that occur at the cervical region of the teeth, can exhibit evidence of abfraction. Abfraction lesions do not have a direct link to nutrition, periodontal disease, or abrasion.

Dentin hypersensitivity is not a true form of allodynia or hyperalgesia, according to most specialists on the subject. It is a normal, physiologic reaction of the nerves in a healthy, non-inflamed dental pulp in a scenario where the insulating layers of gingiva and cementum have been destroyed. Contrary to popular belief, not all exposed dentin surfaces result in DH. Others believe that the existence of patent dentinal tubules in hypersensitive dentin areas may cause higher pulp irritation, resulting in reversible inflammation.