Kaohsiung Journal of Medical Sciences (2018) xx, 1–7

Available online at www.sciencedirect.com

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journal homepage: http://www.kjms-online.com

Review Article

Dental deafferentation and brain damage: A review and a hypothesis

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Received 25 October 2017; accepted 12 January 2018

KEYWORDS
Dental deafferentation;
Alzheimer's disease;
Brain damage;
Temporal-mandibular joint

Abstract In the last few decades, neurobiological and human brain imaging research have greatly advanced our understanding of brain mechanisms that support perception and memory, as well as their function in daily activities. Knowledge of the neurobiological mechanisms behind the deafferentation of stomatognathic systems has also expanded greatly in recent decades. In particular, current studies reveal that the peripheral deafferentations of stomatognathic systems may be projected globally into the central nervous system (CNS) and become an associated critical factor in triggering and aggravating neurodegenerative diseases.

This review explores basic neurobiological mechanisms associated with the deafferentation of stomatognathic systems. Further included is a discussion on tooth loss and other dental deafferentation (DD) mechanisms, with a focus on dental and masticatory apparatuses associated with brain functions and which may underlie the changes observed in the aging brain. A new hypothesis is presented where DD and changes in the functionality of teeth and the masticatory apparatus may cause brain damage as a result of altered cerebral circulation and dysfunctional homeostasis. Furthermore, multiple recurrent reorganizations of the brain may be a triggering or contributing risk factor in the onset and progression of neurodegenerative conditions such as Alzheimer's disease (AD). A growing understanding of the association between DD and brain aging may lead to solutions in treating and preventing cognitive decline and neurodegenerative diseases.

Conflicts of interest: The author declares no conflicts of interests.

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https://doi.org/10.1016/j.kjms.2018.01.013

Introduction

Dental deafferentation (DD) is defined as the elimination or reduction of peripheral afferent neural inputs related to dental and masticatory apparatuses. Examples of DD include: Tooth loss, local and/or generalized periodontal detachment, improper operative or prosthetic restorations, and orthodontic managements and the unavoidable consequence of impaired mastication. These DDs may cause sensory and motor cortical reorganization [1—4], affect cerebral functional streams toward multisensory hubs [5,6], impair gustatory, auditory and olfactory perceptions [7—11], and result in memory and cognition impairment [12—30].

One of the greatest health threats of the twenty-first century has been cognitive frailty and cognitive decline in old age [31,32]: Nearly 50% of adults over the age of 85 are predicted to become afflicted with Alzheimer's disease (AD) [31]. Clinicians may find it necessary to develop new concepts and strategies in coping with these new and challenging circumstances. Recent advances in the biology of stomatognathic systems, together with human and animal experimental studies of the brain, have begun to shed light on the neural mechanisms that may be involved, as well as their potential roles in cognitive decline and memory impairment. Ultimately, the development of therapeutic interventions and preventive strategies for neurodegenerative conditions will demand a greater understanding of the processes underlying both normal and pathological brain aging processes, as related to stomatognathic systems.

The greatest risk factor for cognitive decline and Alzheimer's disease in older adults is age itself [31,32]. Accumulating scientific evidence has indicated a correlation between DD and impaired brain functions such as memory loss, cognitive impairment, and even prodromes of AD and dementia [12—30]. A recent review of both animal trials and clinical research [33] has highlighted the causal relationship of mastication on cognitive functions. Inflammatory mediators, bacteria, and toxins associated with periodontitis have been hypothesized to aggravate brain inflammation [13,34,35] although, to date, no conclusive study has been completed.

The correlation between the DD with memory loss, cognitive impairment, and prodrome of AD and dementia is not yet fully explored. A new hypothesis emerges, however, when combining the latest studies: DDs and related changes in the functionality of teeth and masticatory apparatus may cause significant damage to the functions of the brain, and may be a triggering or aggravating factor in the onset and progression of AD and dementia. Since many DDs are potentially modifiable conditions, the management of any type of DD may prove beneficial to the prevention of neurodegenerative diseases.

Dentition and mastication: the correlation between tooth loss and dysfunctional mastication

Dentition is a group-functional sensory organ. During eating and mastication, various textures and consistencies of food can elicit different levels of enjoyment. These effects can be amplified when certain textures are accompanied by pleasant flavors to stimulate our gustatory, olfactory, and even auditory senses. Such experiences can elicit vivid memories and intense nostalgia as well. It is like an eating-mastication "symphony orchestra," where each tooth is a "musician" performing its indelible, intricate, and indispensable function in creating the overall harmony of eating-mastication [36]. Should one or more "musicians" (teeth) change their function or become absent, the orchestra's overall harmony will be affected, creating temporary or long-term dissonances and increasing mental and physical stress [36]. To adapt to alterations, cope with stress, and maintain the effectiveness of the eating-mastication function when facing such changes, it is necessary have related sensory cortical and motor reorganization [1—4]. This "symphony" can also be orchestrated by different organ systems such as mastication-related muscles, different sensory organs, the temporal-mandibular joint (TMJ), and the autonomic nervous system. Each of these systems coordinates with one another and integrates into a "natural melody." Any deficiencies caused by DD will mistune this "natural melody," causing remapping and reorganizations of both sensory and motor cortices, and damaging the developed functions of cortical networks, especially in the elderly who may have weakened reorganization capacity [31,32].

The uniqueness of DDs: how structural DDs augment functional DDs

DDs involve dysfunctional changes of the dental and masticatory apparatuses. The typical DD is a tooth loss. Many clinicians thought that the neural elements of dental pulp and the periodontal apparatus would be the deafferentated components (Table 1). However, the functionally deafferentated components after tooth loss are far more complicated than previously thought. Because each tooth is physically connected to one another by contact areas and transeptal fibers, the loss of one tooth may affect the coordination of the dentition of that same arch as well as the dentition on the opposite arch during mastication, impacting particularly the teeth adjacent to the site of the lost tooth. Due to interdental connections of the transeptal fibers and contact points of adjacent teeth, the dentition of the same arch may work as connected groups. During mastication, the opposite arch contributes to completing the masticatory function. Any single structural or dental deafferentation can thus cause multiple functional deafferentations of adjacent teeth or opposite teeth. Furthermore, these deafferentations can be aggravated by periodontal detachment and/or orthodontic treatment. Consequently, functional deafferentations may have several times the influence centrally than structural deafferentations.

The unique characteristics of multiple recurrent DDs

Unlike most other types of peripheral deafferentations, DDs may develop as a frequently recurring event of significant

Please cite this article in press as: Jou Y-T, Dental deafferentation and brain damage: A review and a hypothesis, Kaohsiung Journal of Medical Sciences (2018), https://doi.org/10.1016/j.kjms.2018.01.013