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Neuroplasticity & Rehabilitation of Edentulous Patient: A Review

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Abstract: ***A vast and complex array of orofacial muscles and movements necessitate sophisticated and adaptive neural circuits providing for their control and integration with other motor functions. It is apparent from anatomical, electrophysiological, imaging and behavioural studies of the face sensorimotor cortex in humans or laboratory animals, that the face primary motor cortex and the face primary somatosensory cortex, contribute not only to the control of elemental and learned orofacial movements, but also to mastication, swallowing; these in the past have been mostly attributed to brainstem regulatory mechanisms. However, sensorimotor capabilities of edentulous patients rehabilitated with complete dentures, or even implant-supported prostheses, do not match those of dentate subjects. It is unclear why some patients adapt quickly to losing their teeth or prosthetic replacements and relearn and regain the lost sensorimotor skills or acquire new ones; whereas others adapt slowly, or sometimes do not, retaining their sensorimotor deficits. Extensive neuroscience research has provided insights into the remarkable neuroplastic ability of the nervous system in general, plus its crucial role in the adaptive processes during development through adulthood, and following any rehabilitative attempt.***

Keywords: Neuroplasticity; motor control; somatosensory; muscles; orofacial

1. Introduction

Over the past three decades, there have been considerable advances in understanding the peripheral and central neural mechanisms underlying the initiation and regulation of motor functions.[1] Many orofacial movements necessitate exquisite motor control processes to coordinate the activity of the vast array of muscles in the orofacial region. The sensorimotor region of the cerebral cortex is a vital element in the regulation and coordination of the orofacial muscles. This review focuses on the neuroplasticity of sensorimotor cortex in relation to the aquisition of motor skills and adaptation to alterations in the oral environment.

What is Neuroplasticity?

It is the ability of the sensorimotor cortex to undergo structural and functional changes throughout life. These neuroplastic changes represent crucial processes for numerous brain functions. These functions include cognition, memory, motor skill aquisition, perceptual learning, and adaptation following damage to CNS structures (e.g., by stroke), or alterations to peripheral tissues or nerves that modify sensory inputs to the CNS.

Neuroplasticity and related behavioural changes may not only reflect adaptive modifications that are beneficial, but could instead represent maladaptive modifications, that result in impaired function. Neuroplastic changes are also evident during development, when an infant encounters novel sensory and motor experiences as he/she learns and acquires new motor skills (e.g., walking). These changes also occur in adulthood, when a person learns to play a musical instrument or a sport or develop a particular surgical skill.

Mechanism

Such neuroplastic changes involve several intracortical neurochemical changes (e.g., in Ach, GABA, norepinephrine, glutamate). Rapid neuroplastic changes can occur within minutes, and may be explained by mechanisms such as altered synaptic efficiency or unmasking (e.g., through disinhibition) of existing intracortical excitatory synaptic connections, which are usually ineffective because of inter- and intra-hemispheric lateral inhibition.

Long-Lasting Neuroplastic Changes

These may be attributed to several mechanisms like:

- Enhanced gene expression
- Synaptogenesis
- Cortical volume changes
- Axonal sprouting
- Dendritic branching

Long-term potentiation may play a role at early as well as late phases of the neuroplastic process.

2. Importance

Tooth loss may induce neurodegenerative cognitive decline[2,3], which has been speculated to be a major risk factor for

- Cognitive impairment[4], dementia, and Alzheimer's disease[5]

Chewing disability has also been reported to have effects on

- Cognitive decline, activities of daily living, quality of life, physical functions, and mortality rate.6

Oral reconstruction using a denture prosthesis may prevent cognitive decline due to tooth loss, as use of a denture positively activates chewing-related cortices for prefrontal and parietal sensorimotor cognitive control, as well as parietal and temporal sensory associations

- Prefrontal Cortex: It is involved in both cognitive function and regulation of behaviour
- Previous investigations have found that wearing a denture activates the prefrontal cortex during the chewing act, as

compared to a tooth loss condition, which is accompanied by improved masticatory activity and occlusal contact status in partially edentulous individuals

- Recently, Takeuchi et al reported that loss of posterior teeth occlusion was independently associated with cognitive decline, and that maintenance and restoration of posterior teeth occlusion may be a preventive factor against cognitive decline in aged individuals

A pilot study by Perumal et al showed functional improvement in brain function of edentulous patients with complete denture rehabilitation.[7] The patients selected were edentulous for a year, and they were to be rehabilitated with complete dentures for the first time. The study group had Class I residual ridge relationship with adequate interarch space. Patients with symptoms of temporomandibular disorders, xerostomia, orofacial motor disorders, severe manifestations of systemic diseases, and psychological or psychiatric conditions, were not included as a part of the study group, as it could influence their response to treatment giving false results.

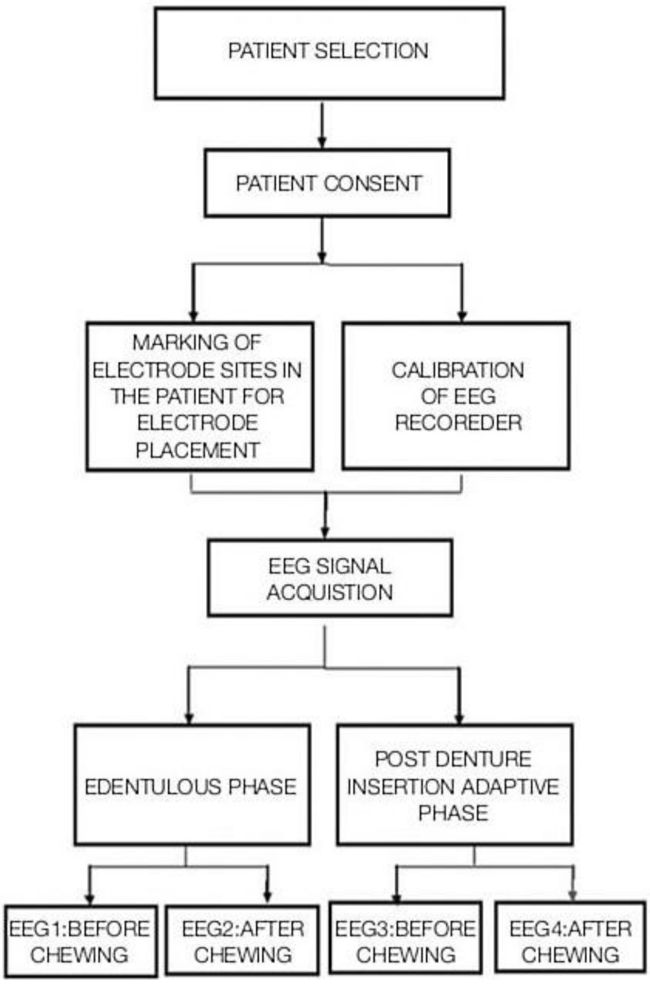

Figure 1: Study procedure Flow chart

The EEG signals were used to check the brain activity. Patients were instructed to take adequate sleep the night before, and were restricted from consuming caffeine at least eight hours before the EEG analysis. The recordings were obtained in two sets: before chewing paraffin gum, and after three minutes of chewing gum, with one-minute resting intervals between each EEG recording (Fig 1). The total duration of recordings was fifteen minutes. The data was saved to be analysed and compared with the EEG records two months after denture placement.

Complete dentures were fabricated with balanced occlusion for all the patients with maximized comfort. After two months of denture usage, the EEG recordings made before and after chewing were made, similar to pre-treatment EEG results (Fig 2).

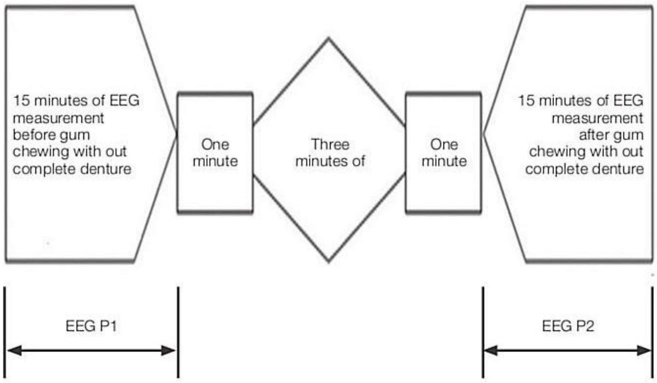

Figure 2: Flow chart for EEG measurement

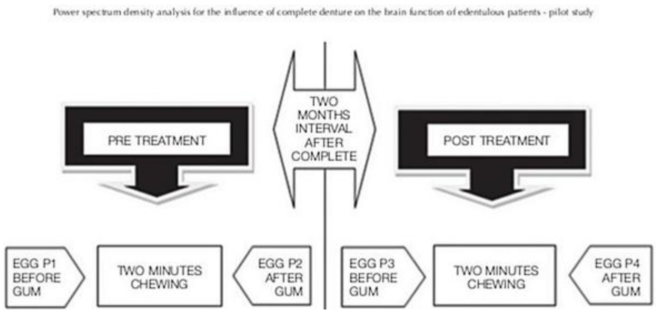

Figure 3: Data acquisition of EEG

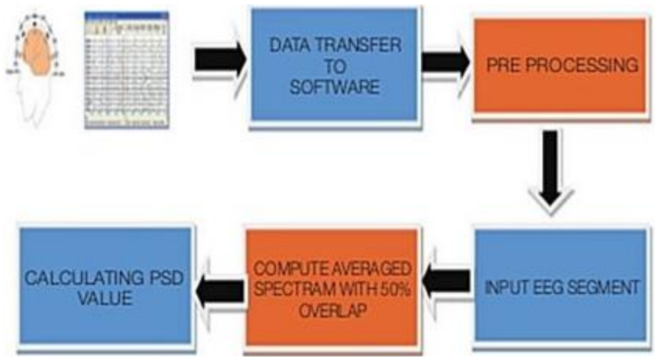

Figure 4: Flow chart for EEG Analysis

The pre-treatment and post-treatment EEG data was analyzed for differences in power spectral density (PSD) with gum-chewing (Fig 4). The alpha waves of the greatest amplitude were recorded.

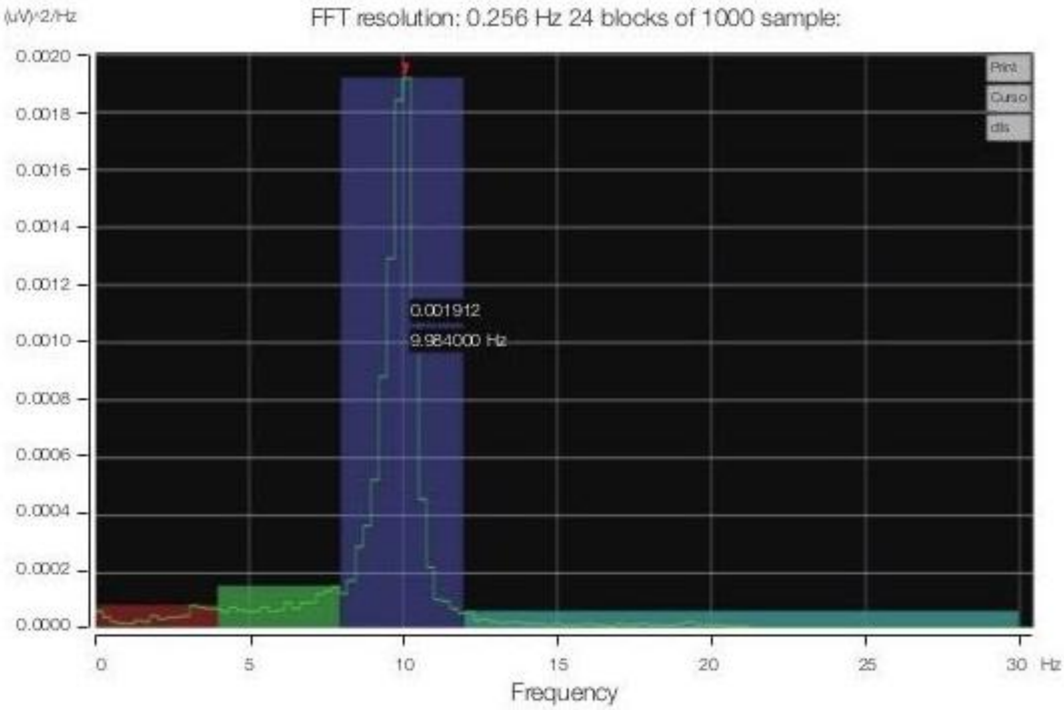

Figure 5: Power spectral density value

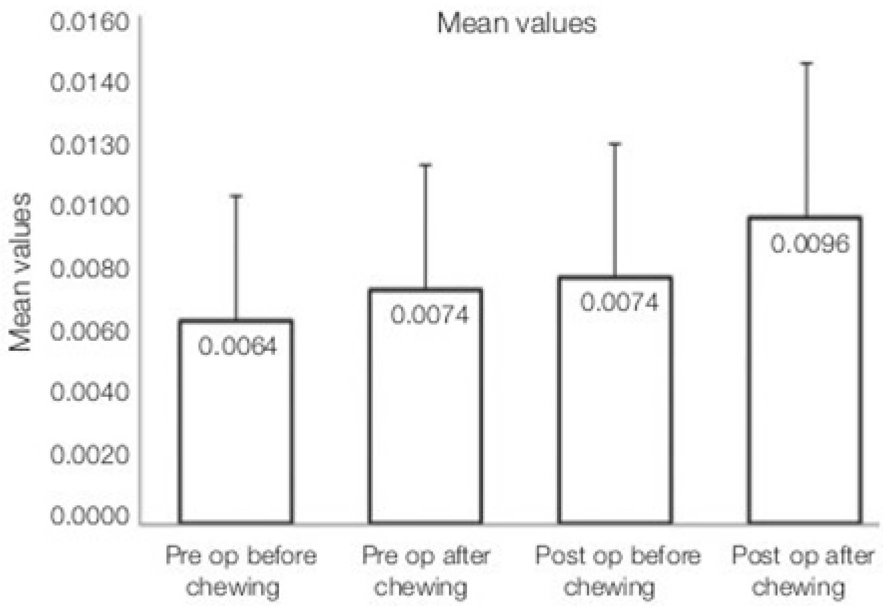

Figure 6: Mean values of edentulous phase and post denture insertion adaptive phase before and after chewing

Table 1: Power spectral density (relative units)

S. NO:	EEG P1	EEG P2	EEG P3	EEG P4
1	0.0025	0.0034	0.0038	0.0053
2	0.0039	0.0042	0.0051	0.0074
3	0.0095	0.0107	0.0131	0.0188
4	0.0039	0.0042	0.0057	0.0061
5	0.0133	0.0141	0.0133	0.0154
6	0.0062	0.0078	0.0099	0.0121
7	0.0021	0.0023	0.0039	0.0042
8	0.0058	0.0119	0.0085	0.0123
9	0.0012	0.0067	0.0042	0.0080
10	0.0016	0.0025	0.0026	0.0054
11	0.0121	0.0131	0.0167	0.0170
12	0.0049	0.0049	0.0032	0.0046
13	0.0026	0.0013	0.0018	0.0032
14	0.0133	0.0143	0.0133	0.0178
15	0.0039	0.0045	0.0042	0.0055
16	0.0082	0.0093	0.0095	0.0096
17	0.0056	0.0058	0.0067	0.0069
18	0.0124	0.0135	0.0114	0.0144
19	0.0067	0.0074	0.0087	0.0089
20	0.0075	0.0076	0.0078	0.0083

EEG p1: Edentulous phase before chewing, EEG p2: Edentulous phase after chewing, EEG p3: Post denture insertion adaptive phase before chewing, EEG p4: Post denture insertion adaptive phase after chewing

Results showed that the alpha waves PSD related to before-chewing values increased in post-denture adaptive phase (Table 1, Fig 6)

3. Clinical Implications

A low number of teeth increased the risk of higher prevalence and incidence of dementia. Stein et al conducted a longitudinal study and concluded that edentulism or very few teeth (=<9) may be predictors of dementia in late life.[4]

The neuroplastic capabilities of the face sensorimotor cortex may reflect or allow for functional adaptation (or maladaptation) of the masticatory system to an altered oral state or altered oral motor behavior . Some patients adapt quickly to losing their teeth, or prosthetic replacements, and relearn and regain lost sensory-motor skills or aquire new ones, whereas other patients adapt slowly, or not at all, and retain their sensory-motor deficits.

There was another study by Kamiya et al [8], the purpose of which was to present the effects of wearing a denture on prefrontal activity during chewing performance. They specifically examined the activity of 12 elderly edentulous subjects and 12 young healthy controls, using functional near-infrared spectroscopy (fNIRS) to evaluate the quality of prefrontal functionality during chewing performance, under the conditions of wearing a denture and tooth loss. The findings were then compared with those of young healthy controls. fNIRS and EMG were used simultaneously to detect prefrontal and masticatory muscle activities during chewing, while occlusal force and masticatory score were also studied using a food intake questionnaire (Table 2,3,4).

Table 2: Masticatory score for Tooth Loss, Wearing Denture and Young

Tooth Loss [mean (SD)]	Wearing Denture [mean (SD)]	Young [mean (SD)]
48.0 (22.1) **a	81.1 (11.7) b	98.5 (2.4)

The score for Tooth Loss was significantly decreased as compared with Wearing Denture ($p<0.01$, **paired t-test). Significant differences were found among Tooth Loss, Wearing Denture and Young [$p<0.05$, Kruskal-Wallis one-way ANOVA on ranks and multiple comparisons versus control group (Young), Dunn's method, a: Tooth Loss vs. Young, b: Wearing Denture vs. Young].
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Table 3: Occlusal force (N) for Tooth Loss, Wearing Denture and Young

Tooth Loss [mean (SD)]	Wearing Denture [mean (SD)]	Young [mean (SD)]
304.8 (104.2) **a	638.7 (127.0)	1200.7 (642.3)

Occlusal force for Tooth Loss was significantly decreased as compared with Wearing Denture ($p<0.01$, **paired t-test). Significant differences were found between Tooth Loss and Young [$p<0.05$, Kruskal-Wallis one-way ANOVA on ranks and multiple comparisons versus control group (Young), Dunn's method, a: Tooth Loss vs. Young, b: Wearing Denture vs. Young].
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Table 4: Masticatory muscle EMG activities for Tooth Loss, Wearing Denture and Young

Table 4. Masticatory muscle EMG activities for Tooth Loss, Wearing Denture, and Young.

Electromyography	Tooth Loss [mean (SD)]	Wearing Denture [mean (SD)]	Young [mean (SD)]
Cycle duration (msec)			
AD	857.0 (153.2) a	834.8 (161.3) b	669.2 (71.5)
Burst duration (msec)			
Mm	212.1 (60.3) **	327.9 (75.2)	268.4 (24.7)
Ta	222.1 (83.8) **	305.4 (67.8)	245.6 (46.5)
AD	338.8 (69.9)	313.4 (41.3)	300.1 (53.0)
Area (mV·sec)			
Mm	0.011 (0.006) †† c	0.026 (0.017)	0.036 (0.018)
Ta	0.009 (0.004) †† c	0.017 (0.010)	0.025 (0.012)
AD	0.010 (0.004)	0.013 (0.003)	0.013 (0.007)
Peak amplitude (mV)			
Mm	0.12 (0.13) b	0.15 (0.09)	0.28 (0.15)
Ta	0.09 (0.09) b	0.15 (0.09)	0.19 (0.09)
AD	0.19 (0.07)	0.23 (0.07)	0.24 (0.19)

Masticatory muscle EMG activities for Tooth Loss were significantly decreased as compared with Wearing Denture ($p<0.01$, **paired t-test, ††Wilcoxon signed Rank Test). Furthermore, significant differences were found among Tooth Loss, Wearing Denture, and Young [$p<0.01$, Kruskal-Wallis one-way ANOVA on ranks and multiple comparisons versus control group (Young), Dunn's method, a: Tooth Loss vs. Young, b: Wearing Denture vs. Young; $p<0.05$, one-way ANOVA and multiple comparisons versus control group (Young), Dunnett's method, c: Tooth Loss vs. Young].

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An individual's inherent neuroplastic capability may contribute to mechanisms whereby patients undergoing oral rehabilitation can (or cannot) restore the lost orofacial sensorimotor functions. This information is important since pain, injuries to the oral tissues, and modifications to the dental occlusion induced by tooth loss or attrition, are common occurrences in humans that may sometimes be accompanied by impaired oral sensorimotor functions.

Impaired oral motor functions are common in many neurological disorders (e.g., brain injury, stroke, Parkinson's disease). Such impairment can sometimes make the most vital functions like eating, swallowing and speaking difficult , thereby reducing the patient's quality of life. Therefore, understanding the mechanisms and cortical neuroplastic processes underlying orofacial sensorimotor functions and adaptation, is important for the development of new treatment strategies to facilitate recovery of such patients suffering from orofacial pain conditions, and sensorimotor deficits, and improve their quality of life. In recent years, principles of sensorimotor cortex neuroplasticity have been translated in to novel evidence-based practices. These include inducing cortical neuroplasticity or reversing undesirable neuroplastic changes in order to enhance the effectiveness of rehabilitative approaches in patients suffering from chronic pain or sensorimotor disorders.

These principles need to be thoroughly investigated as they represent a fruitful research avenue for providing insights into

- Cortical neuroplastic changes that have positive beneficial effects, to allow the patient to adapt to the altered state
- Those changes that may be associated with maladaptive behaviors
- Such insights are crucial in order to bring forth innovative therapeutic strategies that will exploit the positive neuroplastic changes

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