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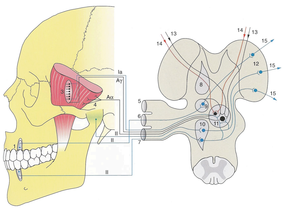
A NOVEL CONCEPT IN IMPLANT UTILITY

Those patients, who performed total implant rehabilitation, will experiment a significant amount of masticator overload. Some of the ceramic bridges and crowns will fracture.

The bone sensibility stimulated by dental implants is almost equal to that deep visceral sensitivity; a very broad dull and undefined one, with no spatial orientation.

The lack of pulpo-dental central nervous system information, almost stop the brain capacity to build a cerebral model of mastication designed and conditioned on the physical properties of the food. Based on current investigation, the author propose a bionic model of dental-pulp sensibility in order to regain the lost brain capacity.



1 Periodontal receptors
2 Joint capsule receptors
3 Muscle spindles (stretching)
4 Motor end plates
5 Trigeminal nerve (semilunar ganglion)
6 Trigeminal nerve (semilunar ganglion)
7 Trigeminal nerve (semilunar ganglion)
8 Mesencephalic tract nucleus
9 Superior sensory nucleus (n.V)
10 Inferior sensory nucleus (n. V)
11 Motor nucleus (n. V)
12 Thalamus
13 Descending paths from cortex (α-motor = "voluntary motor")
14 Descending paths from limbic system (γ-motor = "psychovegetative impulses"
15 Ascending paths to cortex ("consciousness")

The dental perception is one of the most accurate and refined sensibility presented on the sensitive homunculus, the PTP (Phantom Tooth Pain) is a clinical proof about the great disturbance that fall over sensitive cortex. (**2**) When it happens some sort of tooth amputation like extraction, root canal, infection, or trauma on the terminal endings of trigeminal nerve. It's almost probable that it will affect the anatomical network of synapses between sensitive neurons and from these to another level of the conscious and sub conscious of pain.

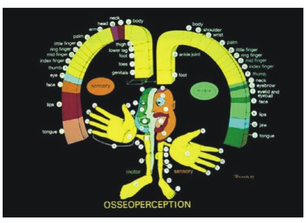
We don't know yet what's the quality and quantity of those sensitive disturbances, but what we do know, is the cut relation between peripheral nerve lesion and central nerve system disturbance. (**3**, **4**)

When a person loose all the teeth, we can replace their dentures with a pair of structures anchored on implants. All implantologist took the experience of overload upon the implant with broken structure, lost of prosthesis teeth, and chewing decoordination. The lost of peripheral sensitive nerve stimuli, drag the mastication sensation upon other lost sensations of the individual, with no reference on the muscular strength for the chewing movements. This lake of feed-back will turn the mastication effort into a repeated overload movement over the prosthesis and in consequence, over the implants. (**5**)

Pöllmann found PTP in 1.7% of an unselected population, in which, however, 2.620 out of 3.126 subjects had edentulous areas. (**5**) The above characteristics, combined with the fact that the dental pulp with nerve endings has usually been amputated, suggest that a central mechanism plays a role in PTP (phantom tooth pain).

Central modifications could be triggered by abnormal impulses generated by an amputation neuroma at the root apex (**2**); experimentally, functional alterations in the properties of neurons in the trigeminal spinal tract have been described following tooth pulp deafferention in the cat (**3**, **4**).

Pain is not a privilege of migraineurs, actually everyone reading this paper, has experienced pain, it varies in intensity, and quality, and has been defined as an "unpleasant sensory and emotional experience associated with actual or potential tissue damage"(Mersky, 1986). Pain serves a critical protective function by alerting the organism to the existence of tissue damage. (**6**) Without it, an organism would continue an activity that could potentially worsen the damage. Pain fibers initiate withdrawal reflexes within the spinal cord, to provide protection prior to the conscious perception of pain that helps identify the location and extent of tissue damage. Collateral pahtways of this system, may also disrupt ongoing neuronal activity and alter behaviour such that it interferes with the healing process. Thus, just as pain serves a protective purpose, the act of alleviating it with behavioural strategies of mastication, can also serve a purpose beyond simple relief. (**7**)



Three types of pain exist: somatic, visceral and deafferentation. (**1**) Somatic pain is the traditional form of pain associated with tissue injury. It results from the stimulation of nociceptive pain fibbers, is well localized, and can be readily described by the patient as an aching or sharp sensation. After the initial sharp, pain sensation (fast pain), a dull, aching pain (slow pain) often follows. Visceral pain is qualitatively similar to somatic pain and has the same kind of source but is experienced in the thoracic or abdominal cavity and is often poorly localized or perceived in a location other than the actual site of tissue damage (referred pain) That type of pain, often occur when patient has an abscess inner bone(8) maxilla or mandible. Deafferentation pain is often an extremely intense pain that is unfamiliar to the patient nociceptive system.

A peripheral nerve lesion led to a loss of epicritic and spinal cord mechanisms for pain leading to uncontrolled pain. Epicritic sensibility came to be associated with A-fiber function, whereas protopathic sensibility came to be associated with C-fiber function. (**9**)

Melzack and Wall contended that the presence of pain depended on the balance of inputs of A- and C-fibers. Predominant inputs of A-fibers blocked pain and predominant inputs of C-fibers favored pain. Melzack and Wall (**6**) argued that this "gating" occurred in the dorsal horn. Some psychophysical studies in normal human volunteers have lent direct support to this conceptualization. Svensson et al. (**7**) recently showed that tonic painful stimulation of jaw muscles induces mechanical hyperesthesia of human facial skin, and considered such hyperexcitability changes as part of the pathophysiological mechanism involved in painful temporomandibular disorders.

We think that reverting the amputation state of the implant treatment and dental rehabilitation, by means of a electrical device (resembling that who's employed on cardiac pacemaker), may took back the perfect mastication and chewing sharp movements.

Dental Implants, they are not teeth

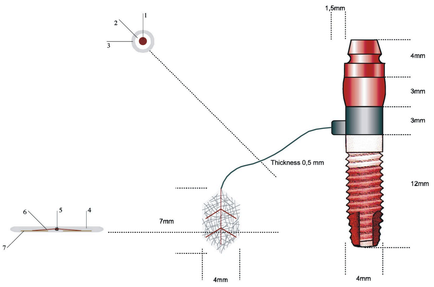
A natural tooth consists of a crown (the part you see above the gum), and the root (the part hidden under the gum, within the jawbone). It is the root that actually holds the natural tooth in place.

When a person is missing a tooth, the dentist must decide how to re-create the crown portion, and he must choose the best method to hold it in place. Dental implants were created for this purpose.

Dental implants are metallic cylinders that are placed into the jawbone where original teeth once existed. These root-like cylinders are used to secure a replacement tooth in place in a spot where a tooth is missing. Dental implants can also be used to secure loose teeth in place by being installed alongside the loose teeth and anchoring to them with splinted crowns. This will allow the loose teeth to function better and Implants;

From it's same beginning the dental implants progress on the field of optimizing the surface and the osteoconduction. Recently, some advancement was got from the immediate loading and relative benefits on getting the definite denture in place.

One of most frequent complaints on patients with complete denture fixed on implants, is the loss of three-dimensional perception of what are they biting when perform mastication. The reason is the loss of feed-back nociceptive perception of the neuronal input coming from the teeth pulp. last longer in the mouth.



The new concept

The new concept of sensitive implants, anchor on the anatomy of the face and the nearest endings of the different roots of the trigeminal nerve, to the implants located either on the maxilla or the mandible. When observing the front view of the face and skull, we may point a straight-line joining together the opening of the frontal nerve, (V1 ophtalmic branch of trigeminal nerve) the opening of sub orbital nerve, (V2 maxillary branch of trigeminal nerve) and the lower opening of the mental nerve (V3 mandible branch of trigeminal nerve). All of those nerve endings have the possibility to generate nociceptive sensation because all the three branches have afferent mielinized fibbers that can conduct the stimuli over the trigeminal SNC nodes. Instead of that, the bone perception is a deep dull sensation, poorly localized (visceral type sensation).

When treating patients with temporo-mandibular disorders (ex: Algo-disfunctional syndrome) we perceive the importance of bringing the brain good three dimensional information about the mandibular movements, the necessary strength of mastication muscles, and the correct oblique mandible movements over pre-molar or canine guidance.

We propose developing a sensitive device:

1. That can produce and modulate a nervous stimulation at the end or beyond the endings of the trigeminal nerve;
2. This device could be applied onto a dental implant or a dental bridge and convert the chewing forces into electrical current that could be transformed into nerve stimulation and or neuromodulation.

How to generate a nerve impulse from an Implant?
As we say yet, the perception of pain depends on the stimulation of the nerve endings of A-delta fibbers. (**6**)
We can generate an electrical stimuli starting on a small piezo-electrical unit, located in a special abutment. The piezo - electrical unit must be miniaturized and be able to generate an electrical impulse from the chewing forces of the mouth. Then, the electrical impulse can be carried out by means of a delicate bounded wire of inner nucleus of cupper and outer side of titanium. The end of this wire can be made of a biological membrane with metallic patches; which can can transport the electrical stimuli into the neighbor zone of nerve ending.