



Osseointegration- A Bond between Bone and Implant: A Review

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Abstract

The formation of a direct interface between an implant and bone, with no intervening soft tissue, is referred to as osseointegration. An implant is currently considered osseointegrated when there is no progressive relative movement between the implant and the bone with which it has direct contact. A histologically observed direct bone contact may indicate a lack of a local or systemic biological response to that surface. As a result, it is proposed that osseointegration is caused by the absence of a negative biological tissue response rather than the presence of an advantageous biological tissue response. This article evaluates the mechanism, concepts, phases, bone-tissue response, and osseointegration related factors.

Keywords: osseointegration, implants, implant tissue interface, osseointegration factors, fibro osseous integration.

Introduction

The goal of modern dentistry is to return patients to oral health in a predictable fashion.^[1] The range of treatment options for patients with missing teeth has expanded, thanks to the science of osseointegration. Both patients and dentists are familiar with the effects of partial or complete edentulism. The interdisciplinary approach of periodontists and prosthodontists in particular has provided impressive evidence of various techniques and methods used to make up for tooth loss. To restore edentulous spaces the patient preference towards a fixed and permanent alternative is favored, where implants are the closest possible way, amongst the restorative options, to achieve both.

The phenomenon of osseointegration (OI) was first observed by Bothe, Beaton and Davenport in 1940 in an animal model, where they observe the titanium had fused with the bone and concluded titanium had great potential as a prosthetic material in future. In

1951, Gottlieb Leventhal placed titanium screws in rat femur and found them to be "slightly tighter" at the end of 6 weeks as compared to when they were placed. In fact, in one specimen the femur fractured when he attempted to remove the screw. Per-Ingvar Brånemark in 1952 conducted an experiment where he utilized a titanium implant chamber to study blood flow in rabbit bone. At the end of the research, when he had to remove the titanium chambers from the bone, he discovered that the bone had integrated so completely with the implant that the chamber could not be removed. Brånemark called this "osseointegration".^[2]

Brånemark first placed an implant in 1965, in a cleft palate patient for the retention of the palatal obturator which was stable for over 40 years. In 1977, (OI) implants became acceptable in Sweden.

Definitions:

Acc. to Brånemark: A direct structural and functional connection between the ordered living bone and surface of load carrying implant.

Acc. to Zarb and Albrektsson: Osseointegration is a process whereby clinically asymptomatic rigid fixation of alloplastic materials is achieved and maintained in bone during functional loading.

Acc. to American Academy of Implant Dentistry: contact established without interposition of non-bone tissue between normal remodeled bone and on implant entailing a sustained transfer and distribution of load from the implant to and within bone tissue.

Mechanism of (OI):

The mechanism of osseointegration can be further explained under 3 phases;

- Inflammatory phase
- Proliferative phase
- Maturation phase

Inflammatory phase:

- Cellular events:

It is initially nonspecific, consisting primarily of neutrophils, and peaks after 3-4 days of surgery. However, by the end of the week, the inflammatory response becomes more specific focusing on B and T cells, monocytes, macrophages, and natural killer cells.

- Vascular events:

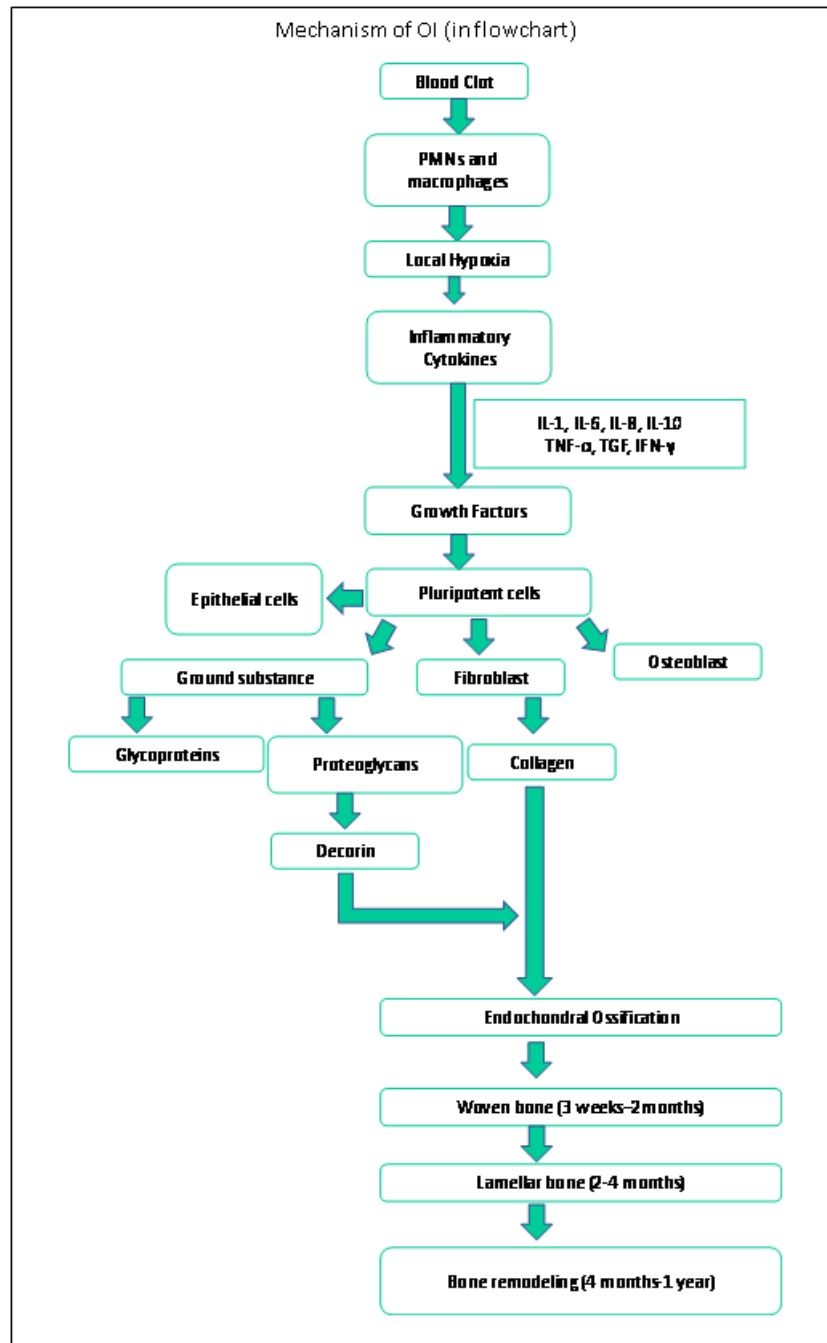
When platelets come into contact with synthetic surfaces, histamines and serotonin are released, causing platelet aggregation and thrombosis.

Proliferative phase:

During this phase, the surrounding vital tissues initiate vascular ingrowth, a process known as neovascularization. Metabolism of local inflammatory cells, fibroblasts, progenitor cells, and other local cells causes hypoxia in the wound area, causing mesenchymal cells to differentiate into fibroblasts, osteoblasts, and chondroblasts. The cells deposit an extracellular matrix, and eventually a fibro cartilaginous callus forms, which transforms into a bone callus. Woven bone (immature bone) is formed.

Maturation/remodeling phase:

Undifferentiated mesenchymal cells, in the advancing granulation tissue mass, lay down woven bone on the scaffold of necrotic bone in the peri implant space caused by operated trauma. This process occurs concurrently with the previously mentioned fibrocartilaginous callus ossification. The simultaneous resorption of these composite trabeculae and formation of new bone, combined with the deposition of mature concentric lamellae, results in complete bone remodeling, leaving a zone of living lamellar bone that is continuous with the surrounding basal bone.



Concepts of Osseointegration

1. Concept of Soft Tissue Anchorage (FIBRO-OSSEOUS INTEGRATION)
2. Concept of Bony Anchorage (OSSEOINTEGRATION)

Fibro- Osseous Integration

- Supported by Linkow (1970), James (1975), Wiess (1986)

- In 1986, the American Academy of Implant Dentistry defined fibrous integration as “tissue-to-implant contact with healthy dense collagenous tissue between the implant and bone.”
- According to this theory, collagen fibers function similarly to Sharpey’s fibers in natural dentition. The fibers affect bone remodeling where tension is created under optimal loading conditions

- It is not accepted widely. as Sharpey's fibers are absent between the bone and implant, to dissipate the load. Therefore, bone remodeling cannot be expected to occur in fibro-osseous integration.^[3]

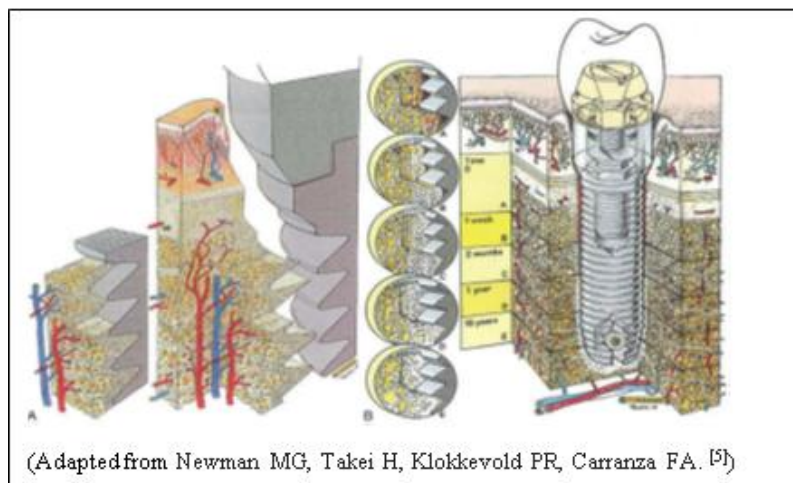
Osseointegration

- Supported by Brånemark in 1985
- This was first described by Strock as early as 1939 and more recently by Brånemark *et al.*,^[4] in 1952
- Brånemark theorizes that the implant must be protected and completely out of function, as

he envisions a period of healing of at least 1 year, in which new bone is formed close to the immobile resting implant

- Meffert, *et al.*, (1987) redefined and subdivided osseointegration into
- Adaptive osseointegration: has osseous tissue approximating the surface of the implant without apparent soft tissue interface at the light microscopic level
- Biointegration: is a direct biochemical bone surface attachment confirmed at the electron microscopic level.

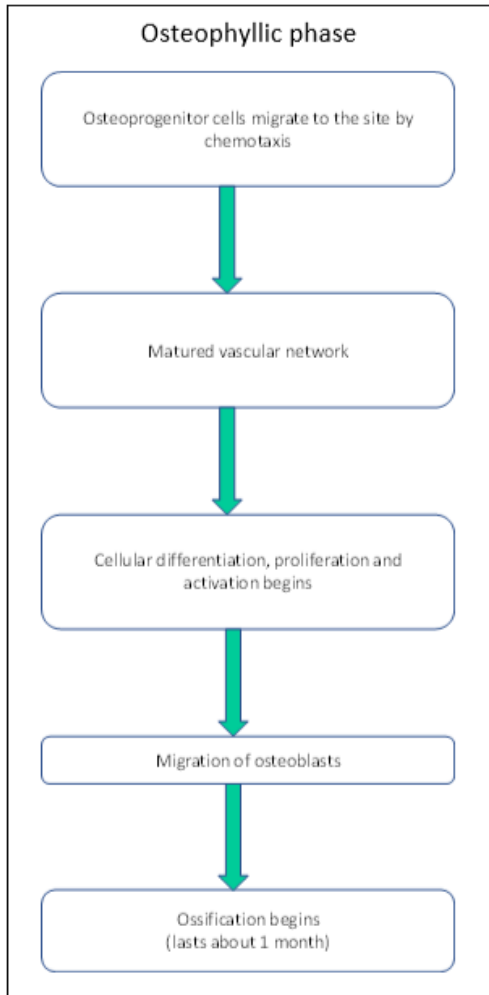
Process of osseointegration



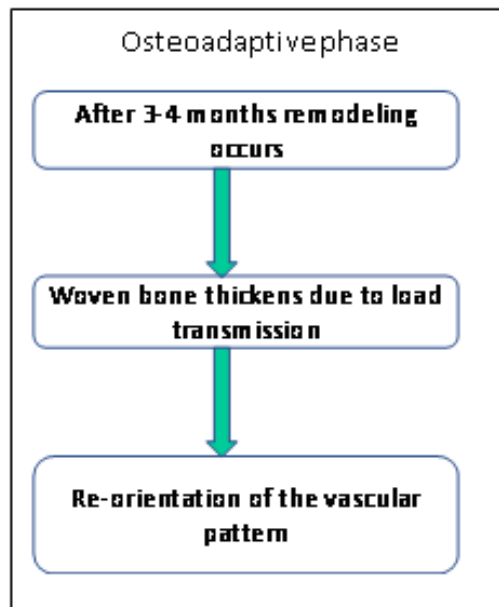
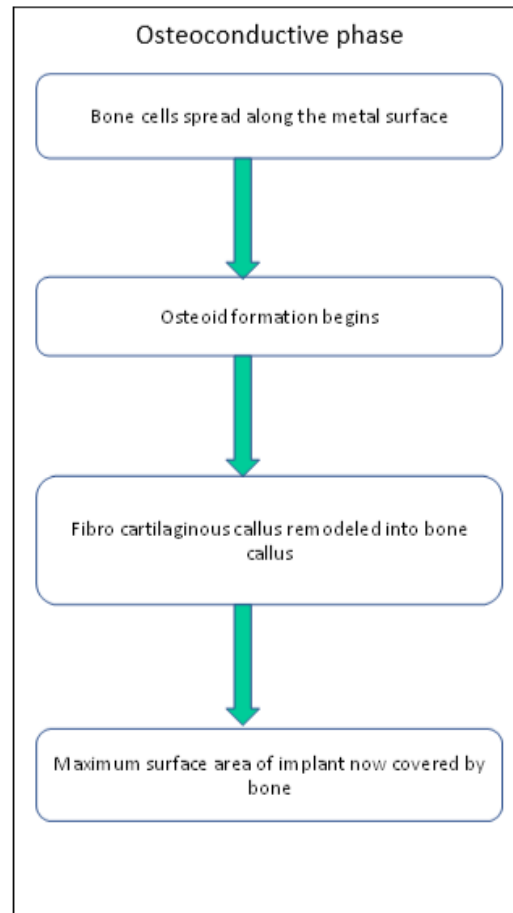
Osseointegration occurs in 3 phases;

1. Osteophyllic phase
2. Osteoconductive phase
3. Osteoadaptive phase

1. OSTEOPHYLLIC PHASE



2. OSTEOCONDUCTIVE PHASE



Bone tissue response (Osborn and Newesley, 1980)

1. CONTACT OSTEOGENESIS
2. DISTANT OSTEOGENESIS

Contact Osteogenesis;

1. Osteogenic cells are first recruited at the implant surface and new (de novo) bone forms first at the implant site
2. Blood supply is between cells and the old bone

Distant Osteogenesis;

1. Osteogenic cells are recruited at the old bone surface which provides a site for the osteogenic cells and lay down new matrix that impinges the implant
2. Blood supply is between the cells and implant
3. Hence, new bone is laid over the old bone surface at the peri implant site

Soft tissue around implants

Soft tissue factors crucial for predictable long-term peri-implant tissue stability, include the biologic width; the papilla height and the mucosal soft-tissue level; the amounts of soft-tissue volume and keratinized tissue; and the biotype of the mucosa (Thoma *et al.*, 2014)^[6]. In a recent systematic review and meta-analysis by Longoni *et al.*, in 2019 concluded that adequate width of keratinized tissue around implants is important.

Factors affecting osseointegration^[7] (Albrektsson, 1983)

1. Implant material biocompatibility
2. Implant design
3. Implant surface characteristics
4. Surgical technique
5. Status of the host bed
6. Loading conditions

Implant Material Biocompatibility:

1. Metals such as commercially pure (c.p.) titanium, niobium, and possibly tantalum are well accepted in bone because they are coated with a highly adherent, self-repairing, and corrosion-resistant oxide layer

2. Bone is less tolerant of metals such as cobalt-chrome-molybdenum alloys, stainless steels, and titanium alloys
3. Ceramics such as calcium phosphate hydroxyapatite (HA) and various types of aluminum oxides have been shown to be biocompatible, but they are less commonly used due to insufficient documentation and very few clinical trials

Implant Design:

1. Threaded implants have a larger functional area for stress distribution and provide better primary anchorage than cylindrical implants
2. Because V-shaped threads transfer vertical forces in an angulated path, they may not be as effective as square-shaped threads in stress distribution
3. The greater the length, the greater the primary stability. Shorter implants (10 mm or less) have been linked to increased bone loss
4. When compared to narrow implants, wide diameter implants place less stress on the crestal bone
5. Including micro threads in the implant neck aids in the preservation of marginal bone because these threads anchor in the bone. In contrast, a smooth machined neck is associated with greater bone loss
6. The platform-switching concept also preserves the bone and prevents crestal bone loss. This technique employs a narrow diameter abutment over a large diameter implant
7. Advantages of one-piece implants over two-piece implants include the elimination of the implant-abutment junction, which maximizes strength, eliminates micro movement, and prevents bacterial penetration at the implant-abutment junction in two-piece implants
8. By including a Morse taper in 2-piece implant systems, bacterial penetration at the junction has been reduced

Implant Surface Characteristics:

1. Surface topography is concerned with the degree of roughness of the surface as well as the orientation of surface irregularities
2. Benefits of increasing surface roughness
 - i. Increased implant surface area to bone, resulting in increased bone at implant surface
 - ii. Improved biomechanical interaction between the implant and bone
3. Smooth surfaces do not result in acceptable bone cell adhesion, and clinical failure is likely
4. Liu *et al.*, in 2019 in a systematic review and discussed the superiority of current surface modification programs and provided a comprehensive reference information and an extensive overview for better fabrication and design of orthopedic implants ^[8]

Surgical Technique:

1. Optimal surgical technique to promote regenerative bone healing rather than reparative bone healing (Erickson R.A)
2. Use of sharpened and graded drills in a series
3. Enough cooling. For bone tissue necrosis, the critical time / temperature relationship is around 47⁰ C applied for one minute
4. Slow drill speed (less than 2000 rpm with irrigation and tapping at 15 rpm)
5. Implant insertion with a moderate power

Status Of The Host Bed:

1. Poor bone bed due to irradiation: not an absolute contraindication to implants. However, some time before implant placement is preferable
2. Low ridge height and resorption, as well as osteoporosis, are indications for ridge augmentation with bone grafts prior to or during implant placement
3. Infection
4. Poor bone quality: According to Brånemark *et al.*, and Misch, D1 and D2 bone densities

show good initial stability and better osseointegration, whereas D3 and D4 bone densities show poor prognosis

Loading Conditions:

Premature loading results in soft tissue anchorage and poor long-term function, whereas delaying loading with a two-stage surgery results in bone healing and positive long-term function. Del Fabbro *et al.*, in 2019 in a systematic review that evaluated the survival rates of immediately loaded implants after at least five years, concluded that immediate loading of implants appears to have long-term predictability and success rate under well-defined circumstances. ^[9]

Implant success criteria (Albrektsson and Zarb, 1986)

1. No mobility
2. Absence of periapical involvement
3. Asymptomatic site: No persistent pain, discomfort or infection
4. Stable crest levels of the bone
5. Crestal bone loss <0.25mm after 1 year of loading
6. Healthy periodontal tissues
7. A success rate of 85% at the end of a 5-year observation period and 80% at the end of a 10-year period are minimum levels of success

Methods to evaluate osseointegration;

1. Percussion test
2. Radiographs
3. Reverse torque test
4. Resonance Frequency Analysis (RFA)
5. PERIOTEST

Percussion Test:

An osseointegrated implant makes a ringing sound on percussion whereas an implant that has undergone fibrous integration produces a dull sound.

Reverse Torque Test:

A reverse or unscrewing torque is applied to assess the implant stability at the time of abutment connection. Implants that rotate under the applied torque are considered failures and are then removed.

Resonance Frequency Analysis (RFA);

1. Proposed by Meredith *et al.*, 1996
2. RFA is a method to determine the stability (level of osseointegration) in implants
3. The stability is presented as Implant Stability Quotient (ISQ) value
4. Higher the ISQ, higher the stability
5. RFA includes sending magnetic impulses to a transducer that is temporarily attached to the implant. As the rod vibrates, the probe reads the resonance frequency and translate it into an ISQ value
6. This technique uses handheld frequency response analyzer
7. ISQ>70 = high stability, ISQ 60-69= Medium stability, ISQ<60= low stability

Periotest:

Noninvasive device to monitor implant stability.

1. Quantifies the mobility of the implant—measures the reaction of peri implant tissues to a defined impact load
2. Handpiece: electronically controlled translational hammer bearing an 8gm rod with a sensor at its tips
3. When activated rod taps implant abutment up to 16 times in 4 seconds
4. Rod decelerate – touches the implant and accelerate – Rebounds of the implant
5. 0 mobility:0.4-0.5 sec, -8-+4=0.65 (palpable movement), +4 - +9= high failure rate
6. Greater the implant stability—shorter the time elapsed

Conclusion

Osseointegration is a complex process. During the inflammatory and remodeling phases of bone healing- cell types, implant and bone tissues, growth factors, and cytokines all work together. This means that osseointegration should be viewed as an expression of bone's endogenous basic regenerative potential, rather than an exclusive reaction to a specific implant material. Improved biologically-driven design strategies for endosseous implants will

result in a better understanding of the complex biological events that occur at the bone-implant interface.

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