

100 DAYS OF IMPLANT: A REVIEW

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Abstract:

Purpose: The purpose of this paper is to analyse the current understanding of clinical assessments and factors that determine the success and failure of osseointegrated dental implants.

Study Selection: This is a narrative review performed through scientific articles published between the year 2000 and 2017, indexed in MEDLINE and pubmed databases. The study selected articles that focused on osseointegration around the implant body.

Results: The Implant –Tissue interface is an extremely dynamic region of interaction. The process of osseointegration involves an initial interlocking between alveolar bone and the implant body and later biological fixation through continuous bone apposition and remodelling towards the implant. The process itself is quite complex and there are many factors that influence the formation and maintenance of bone at the implant surface.

Conclusions: This narrative review explores the extent of current knowledge of osseointegration and aims to consolidate this knowledge and highlight the most pertinent questions relating to this area.

Introduction

Osseointegration is a phenomenon where intimate contact between bone and biomaterials occurs at the optical microscopy level, enabling dental implants to replace load bearing tooth organs restore their form and intraoral function⁴. A thorough and complete understanding of what happens at the bone and implant surface is important and thereby enabling a treatment plan which conforms to the standards and gives a better clinical predictability².

The term osseointegration was first used by prof I.P Branemark, in 1950, during an examination of microcirculation of bone and wound healing through means of vital microscopy, and accidentally discovered the process of osseointegration and a new dimension in the field of implantology.

Mechanism of Osseointegration

The healing of an osseous wound around a dental implant is a coordinated and sequentially organised repair mechanism. The process takes place by the communication between different types of cells. This sequence of cell communication is explained as the four phases of osseointegration.

Phase 1- Hemostasis

Phase 2- Inflammatory phase

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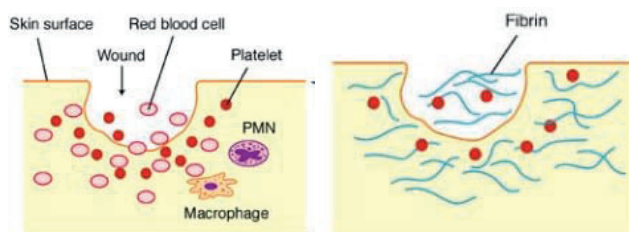
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Phase 3- Proliferative phase

Phase 4-Remodelling phase

Hemostasis

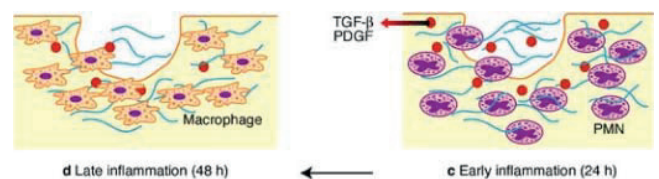
Hemostasis or exudative phase begins with the surgical drilling by the dental implant drill followed by the placement of implant. The duration of this phase is minutes to hours. The mechanical crushing of the bone matrix liberates bone matrix protein, growth factors and differentiation factors into the implant site. Injured blood vessels create a polymerization of fibrinogen with the help of thrombin. Bioactive molecules such as thrombin, ADP, collagen, fibrinogen and thrombospondin are generated. Thromboxane released from platelets plays a critical role initial vasoconstriction. The release of cytokines from the degranulated platelets is the beginning of inflammatory phase.



Inflammatory Phase

Inflammatory phase begins approximately after 10 minutes and lasts for first few days after surgery. This phase begins with degranulation of platelets and release of growth factors like transforming growth factor (TGF-B), platelet derived growth factor (PDGF), Fibroblast growth factor (FGF), Bradykinin etc¹. Bradykinin increases the vascular permeability and endothelial cells in the blood vessel wall promote the attachment of pmns/leucocytes. The pmns invade the blood clot by amoeboid migration, squeezing through little gaps in the walls of blood vessels with the help of proteases and finally enter the wound site. Leucocytes/pmns then chemotactically navigate through

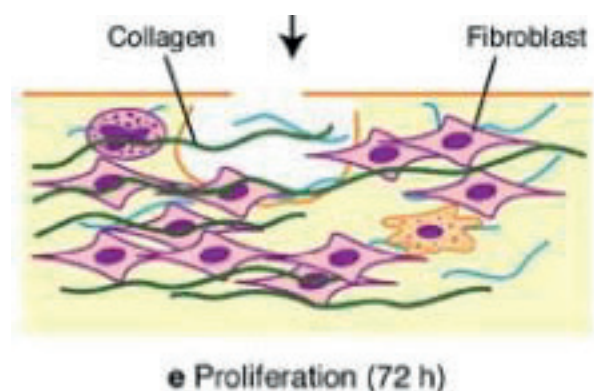
the wound along the molecular concentration gradient. The molecules include bacterial proteins, fibrinopeptides and proinflammatory interleukins. On arrival, they kill the bacteria through the release of reactive oxygen species. Pmns also release highly digestive enzymes such as collagenase and elastase.



The wound then proceeds to a stage of healing process. The tissue debris are biochemically degraded, and underwent phagocytosis by macrophages. The macrophages synthesis proinflammatory cytokines and proteases using antigenic inhibitors for digesting proteases, macrophages stops the tissue degradation started by pmns. This will in turn produce growth factors by matrix proteins and proteoglycans.

Proliferative phase

The transition into proliferative phase is characterized by the formation of new extra cellular matrix and angiogenesis. Messenger substance like VEGF, PEGF, FGF stimulates fibroblasts and helps angiogenesis. Fibroblasts appear on the 3rd or 4th day. They migrate into the wound by amoeboid movements. They synthesis the protective and stabilising components of extracellular matrix such as collagen, elastin and proteoglycans.



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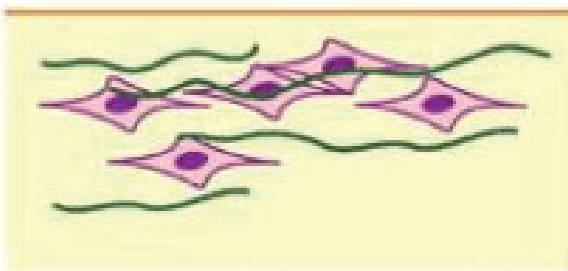
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The low oxygen concentration in the wound affects both macrophages and endothelial cells and stimulates them to create the intercellular transcription factor, hypoxia inducible factor. VEGF influence perivascular cells. They migrate into VEGF gradient, into areas of low partial oxygen pressure. Here they form new blood vessels, which integrate into existing network.

Angiogenesis restores the oxygen supply, which is a prerequisite for osteogenesis. Activated osteoclasts attach themselves to the fracture areas of residual bone, resorbing it and creating space for bone healing¹. However, it will influence the primary stability of implants. Osteoclasts dissolve the bone by the action of hcl and proteases and release BMP, TGF-B, PDGF from bone matrix, which in turn initiates the formation of new bone. Perivascular cells migrate into implant surface and differentiate into new osteoblasts under influence of bmps. Osteoblasts then form an organic matrix, along with calcium phosphate. Towards the end of 2 weeks woven bone is formed at the implant surfaces, this will create a secondary stabilisation. The formation of woven bone concludes the proliferative stages of wound healing.

Remodelling phase

The initial bone formed, i.e.; woven bone is grown parallel to the implant surface. On remodelling phase, parallel bone formed is structured perpendicular to the peak of implant threads. Organisation of the bone becomes more trabecular. This process happens by the coupling action of osteoclasts and osteoblasts.



f Remodelling (weeks to months)

Osteoclasts activated by osteoblast messenger RANKL, resorb the woven bone and the osteoblasts lay down highly organised lamellar bone. This simultaneous action of osteoblasts and osteoclasts are coordinated by osteocyte and its messenger sclerostin. In the final state, the newly formed unit, containing a central blood vessel called osteone or Haversian system.

Discussion

Successful osseointegration from the clinical stand point is a measure of implant stability, which occurs after implant integration. Alberktsson et al referred to six factors that determine the success of osseointegration, implant biocompatibility, design characteristics, implant surface, state of host bed, surgical technique, implant loading condition.

One of the most important aspects about achieving osseointegration clinically is implant primary stability. Initial or primary stability is the mechanical interlocking between the bone and the implant where there exists no biologic interplay¹³. The mechanical interlocking is influenced by the implant geometry and topography at different levels, as well as the implant osteotomy protocols, which all regulate the strain applied to the hard tissue¹⁸. The higher insertion torque of the implant is intuitively and fallaciously perceived as higher primary stability, which has been clinically regarded as an indication for procedures such as immediate loading¹⁵. However, in reality, the stability of the implant would decrease beyond the yield strain of the bone due to excessive micro crack formation and compression necrosis, which both phenomena trigger bone remodelling¹². Compression necrosis occurs when the hard tissue around the implant is faced with excessive strain, where the circulation of the capillaries and nerves is severely damaged¹⁹.

The surgical drilling speed proportionally influences heat generation to the surrounding bone¹⁴. Research has indicated that an overheat exceeding 47° C

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for 1 min would provoke an irreversible thermal injury to the bone with osteoclasts activation due to local surgical instrumentation damage or osteocyte death²⁰. Yeniyol et al have demonstrated that higher degree of osseointegration for implants placed in sites prepared under low-speed drilling. A recent experimental study has shown that higher osseointegration levels and lower degree of bone dieback for drilling less than 400 rpm¹⁷. Bone cell survival is very susceptible to heat. Eriksson has demonstrated that in rabbit, bone temperature as low as 3° above normal (40°C), can cause bone cell necrosis. Therefore, a conscious effort is made to control temperature elevation every time a rotary instrument is placed in contact with bone. At least 50ml/min of cooled irrigation.

The original Branemark surgical approach should be used to maintain aseptic surgical protocol. Normal skin flora, including staphylococcus epidermis and staphylococcus aureus are able to adhere to implant biomaterials. A bacterial smear layer may develop, which will be difficult for the body to remove by phagocytosis or bone cell activity. Antimicrobial mouth rinses significantly reduce bacterial count in the saliva for more than 4 hours. Povidone iodine alcohol solution have antibacterial properties used in preoperative site disinfection. Although a sterile technique is not possible within the mouth, scrubbing around and within the mouth with these agents reduce the risk of a bacterial smear layer by inadvertent contamination by direct contact with the implant or indirectly via a glove or instrument²¹.

Conclusion

Attempted to concise the process of osseointegration through the four phases of tissue healing. Factors affecting the process of osseointegration provides the clinicians, information regarding the importance of surgical protocol. Experiencing implant osseointegration as a biological process may provide the clinician new targets to improve the therapy with dental implants

Reference

1. Terheydon H, Lang NP, Bierbaum S, Stadlinger B, Osseointegration-communication of cells. Clin Oral Implants Res. 2011;12(1):1-9.
2. Mavrogenis A, Dimitriou R, Parvizi J, biology of implant osseointegration. J Musculoskelet Neuronal Interact. 2009;9(1):61-71
3. Parithimarkalaignan S. Osseointegration: An update. J Indian Prosthodontic Soc. 2013;57(2):2-6
4. Carrillo C, Aliaga M et al; Tooth implant connection: A bibliographic review. Med Oral Patol Oral Cir. 2010;15(1):5
5. The glossary of prosthetic terms, 6th ed. J Prosthet Dent. 1994;71:89
6. Meng et al. Dental implant bioactive surface modifications and their effects on osseointegration: a review. Biomaterials. 2016;107:1-12
7. Yona KS, Rittel D. An overview of the mechanical integrity of dental implants. Biomed Res International. 2015;1(1):1-5
8. Swamiv, Vijayaraghavan V, Swami V. Current trends to measure implant stability. J Indian Prosthodontic Soc. 2016;60(1):24-30
9. Javed F et al; Role of primary stability for successful osseointegration of dental implants: Factors of influence and evaluation. Interventional Medicine & Applied Science. 2013;5(4):162-167
10. Khan et al; Osseointegration and more: A review of literature. IJD. 2012;3(3):12-17
11. A Chug et al; Osseointegration-molecular events at the bone-implant interface. A review. 2012;1(1):1-10. Contemporary Implant Dentistry 3rd edition-Carl E Misch
12. Chamay A, Tschantz P (1972) Mechanical influences in bone remodelling. Experimental research on Wolff's law. J Biomech. 1972;5:173-180
13. Halldin A, Jimbo R, Johansson CB, Wennerberg A, Jacobsson M, Albrektsson T, Hansson S (2011) The effect of static bone strain on implant stability and bone remodelling. Bone. 2011;49(1):1-10
14. Iyer S, Weiss C, Mehta A (1997) Effects of drill speed on heat production and the rate and quality of bone formation in dental implant osteotomies. Part II Relationship between drill speed and healing. Int J Prosthodont. 1997;10(1):1-6
15. Javed F, Romanos GE (2010) The role of primary stability for successful immediate loading of dental implants. A literature review. J Dent. 2010;38(1):1-10
16. Norton M (2013) Primary stability versus viable constraint - a need to redefine. Int J Oral Maxillofacial Implants. 2013;28(1):1-10
17. Yeniyol S, Jimbo R, Martin C, Tovar N, Janal MN, Coelho PG (2013) The effect of drilling speed on early bone

PROSTHETIC AND IMPLANT DENTISTRY

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- healing to oral implants. *Oral surg pral med oral pathol oral Radiol* 116
18. Gottlow J, Barkarmo S, Sennerby L (2012) An experimental comparison of two different clinically used implant designs and surfaces. *Clin Implant Dent Relat Res* 14:e204-212
 19. Zizic TM, Marcoux C, Hungerford DS, Dansereau JV, Stevens MB (1985) Corticosteroid therapy associated with ischemic necrosis of bone in systemic lupus erythematosus. *Am J Med* 79:596-604
 20. Eriksson R A, Albrektsson T, Magnusson B (1984) Assessment of bone viability after heat trauma. A histological, histochemical and vital microscopic study in the rabbit. *Scand J Plast Recontstr Surg* 18:261-268
 21. Contemporary Implant Dentistry-Carl E Misch 3rd Edition