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Review article

Lack of integration of smooth titanium surfaces: a working hypothesis based on strains generated in the surrounding bone

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It has been observed that the polished neck of dental implants does not osseointegrate as do textured surfaces. Similar findings were reported in the orthopedic literature on artificial hip endoprostheses. In Dentistry, lack of osseointegration was attributed to increased pressure on the osseous bed during implant placement, establishment of a physiological "biologic width", stress shielding and lack of adequate biomechanical coupling between the load-bearing implant surface and the surrounding bone. Among the many variables that may affect osseointegration, this analysis proposes to include stress transfer as a significant one. Therefore the present report discusses the relationship between the stresses applied and bone homeostasis. Any viable osseous structure (including the tissue that surrounds the polished implant neck) is subjected to periodic phases of resorption and formation. Clinical and experimental data have shown the detrimental effects of lack of function in that bone mass decreases with time. Due to inadequate mechanical stimuli, bone that is resorbed during normal turnover is redeposited in lesser amounts than previously, a process observed clinically as resorption. The stress ranges which cause bone to resorb, maintain or increase its mass and the level that eventually causes bone to fracture have been delimited in the literature. Applying these values to the situation to dental implants, it follows that if it is to be stable, crestal bone must be subjected to suitable levels of mechanical stimulation. We suggest that smooth surfaces will not provide adequate biomechanical coupling with the bone surrounding the implant neck in that the stress range induced by a polished surface is limited. We propose that the surface texture of threaded, plasma-coated or sandblasted implants generates a heterogeneous stress field around an implant in function. By design, such a stress field includes force levels which are conducive to bone formation. Hence, during the formation phase of bone turnover, osteoblast lineages are much more likely to be stimulated by biomechanical signals of appropriate magnitude.

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Key words: bone – dental implants – stress – strain – bone resorption

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Experimental data have shown that placement of the polished neck of ITI dental implants 1 mm below the bone crest results in an additional 1 mm bone loss as compared to controls where the apical border of the smooth surface was placed level with the bone margin (Hämmerle et al. 1996). This focal study paralleled a series of investigations showing that textured titanium surfaces promoted integration while smooth areas were essentially devoid of bone contact. As to this issue, some interpretations have been presented that did essentially link integration to titanium surface phenomena and cell adhesion. We wish to offer an additional explanation that involves mechanical coupling between implant surface and surrounding bone.

Observations. Available clinical evidence indi-

cates that crestal bone resorption around dental implants tends to be arrested by threads or a porous surface texture. According to data by Brånemark et al., the bone level adjacent to implants decreased by 0.5 to 1 mm during the first year of function. Subsequently, the rate of bone loss was considered either stationary (Brånemark et al. 1969) or significantly reduced (approximately 0.1 mm/year (Cox & Zarb 1987; Malevez et al. 1996)). In a 1992 study, Quirynen et al. reported increased marginal bone loss around smooth vs threaded portions of the implants tested (Quirynen et al. 1992). Comparable findings were published in an experimental study by Pilliar et al. (1991). In the absence of plaque-associated infection, some crestal bone loss occurred for both the threaded and partially porous-coated implants while no significant bone loss was observed with fully porous-coated implants.

Additional evidence can be derived from histological data. In a 1991 study, Buser et al. showed that an increase in surface roughness correlated with an augmentation of the percentage of bone contact around the implant. Similar conclusions were drawn in a 1996 study by Larsson et al.

Cellular activity. It has been observed that certain cell lineages differed in their affinity for various implant surface textures (Rich & Harris 1981). It also appeared that cells have a tendency to align themselves with fine striations and grooves such as those left after machining (Brunette 1988). One standpoint holds that cells might somehow better adhere to rough vs smooth surfaces and thus explain the lack of integration commonly observed on polished titanium (Burchard et al. 1991). Indeed, data from one *in vitro* study suggest that cells are more adherent to sandblasted than smooth surfaces (Bowers et al. 1992). However, according to the results published, 1) the difference between "smoother" and "rougher" surfaces is small (approx 15%) and, 2) time was the major factor in promoting cell adhesion to the test surfaces. In another study (Wennerberg et al. 1996), a minor difference (6%) in bone contact surface was detected between the percentages on titanium surfaces of two different roughnesses. In spite of elaborate claims by the authors, it is doubtful that any significant conclusion can be drawn from the findings presented before further evidence is presented.

Still, most data available to-date tend to show that "rough" textures promote increased cellular activity when compared to "smooth" titanium surfaces (Groessner-Schreiber & Tuan 1992). In this respect, in a recent study by Boyan et al. (1998) on osteosarcoma cells (MG63), rougher surfaces were associated with a significant increase in alkaline phosphatase activity.

These studies, however, do not explain why macroscopic surface features such as threads do also arrest alveolar crest bone resorption.

Stress transfer. Many authors have described mathematical modeling procedures that describe the stress fields surrounding implants in function. Some of these implied that peri-implant stress levels might reach a magnitude that could prove detrimental to the fixture (Rieger et al. 1989b; Clift et al. 1993; van Zyl et al. 1995). Other researchers hypothesized that altering the geometry of the implant and abutment could improve stress transfer to the crestal bone by increasing compression and decreasing shear (Mihalko et al. 1992). Finally, one group has produced finite element models which concluded that crestal bone loss was actually the result of disuse atrophy due to the low stress level acting on the surrounding bone in this area (Vaillancourt et al. 1995).

Scope. The present report discusses the relation between strains and changes in bone mass. Yet, in the context of dental implants, this is not to discount other issues such as material biocompatibility (LeGeroz & Craig 1993), biology of osteoblasts (Wlodarski 1990) and bioactive molecules (O'Neal et al. 1992; Davies 1996) that may have a significant impact on the osseointegration process.

Indeed a host of agents have been linked to osteoblast activity: growth factors (prostaglandins (PGE_1, PGF_2), the transforming growth factor-β superfamily (TGF-β) (Elima 1993) that includes bone morphogenetic proteins (BMP) (Urist et al. 1983), insulin-like growth factors (IGF-1, IGF-2), fibroblast growth factor (FGF), epidermal growth factor (EGF)), hormones (Calcitonin, triiodothyronine, PTH, hydrocortisone, dexamethasone) and vitamins (vit. D metabolites (Finkelman et al. 1991; Atkin et al. 1992)). As other activators of bone formation have been described: retinoid acid (Tabin 1991), high-molecular hyaluronic acid (Sasaki & Watanabe 1995) and fracture hematoma (Mizuno et al. 1990).

Induction (Urist et al. 1967), that is the differentiation of tissue after contact with specific cell types or materials, is another characteristic of bone formation. In this regard, various interactions during embryogenesis have been described (Hall & Van Exan 1982; Melton 1991). Further, bone formation can be induced by implantation of auto- (Kamijou et al. 1994) or allogenic tissues or materials. For instance, when embedded into a muscle, urinary bladder epithelium will cause mineralized deposits to appear (Friedenstein 1968). Various hard tissues have also demonstrated osteoinductive properties (Burwell 1966; Pinholt et al. 1990; Jergensen et al. 1991). Whether synthetic materials are osteoinductive in humans is still a mat-

ter of debate (Damen et al. 1990; Gatti & Zaffe 1991).

The various aspects of bone formation *in vivo* are complex, interrelated and, at times, contradictory. For instance, the importance of strain in bone mass maintenance will be detailed in later sections. Yet it is known that osteoblasts may also form calcifying matrix in a petri dish (i.e. without being subjected to any loading) (Peel 1995). This inconsistency may be related to the age of the animal and/or the stem cells; further information, however, is lacking.

Purpose. Knowing that a comprehensive picture of the processes involved in osteogenesis and bone homeostasis has yet to be drawn (Lavelle 1993), the present project will restrict itself to two aspects: 1) present an overview of present-day knowledge of the effect of force application on bone remodeling whereby special emphasis will be placed on quantifiable force levels and, 2) propose an explanation of the lack of integration that is commonly observed on smooth titanium implant surfaces.

Strains and bone growth

Stresses and strains

Applying force to a bone (stress) deforms (strains) its structural arrangement. While being the primary factor, one must recognize that force is an abstraction that can be quantified only by determining changes in a specimen's geometry. Stress is defined as a force divided by unit area (Mpa) while the resulting strain (ε) is the ratio between the length of an object under stress and its original dimension. Strain is thus a dimensionless entity. Due to the minute variations observed, biomechanicians quantify such alterations in microstrains ($\mu\varepsilon$), a scale in which 10^6 $\mu\varepsilon$ would equal a theoretical deformation of 100%. Thus 1000 $\mu\varepsilon$ in compression equals a shortening of 0.1% (99.9% of the original length) and 20,000 $\mu\varepsilon$ in tension equals a stretching from 100% to 102% of the original length (Frost 1994). One general conversion rule is the equation that relates stresses and strains: $\varepsilon=\sigma/E$ or in layterms: the resulting strain is equal to the applied stress divided by the modulus of elasticity of the material. In later sections of this report, owing to the $\varepsilon=\sigma/E$ relation, stress and strain will be used almost interchangeably. In the context of force application, however, it should be borne in mind that changes in bone mass are dependent on strains (i.e. alterations in shape) and not stresses *per se*.

Two types of applications are shown in Fig. 1a, b. In the compression testing model described by Shelton & El Haj (1992), a cylinder was filled with macroporous gelatin beads, periosteal cells and a culture medium. The medium was loaded via a piston and the strain applied to the cells was calculated from the variation in height of the bead col-

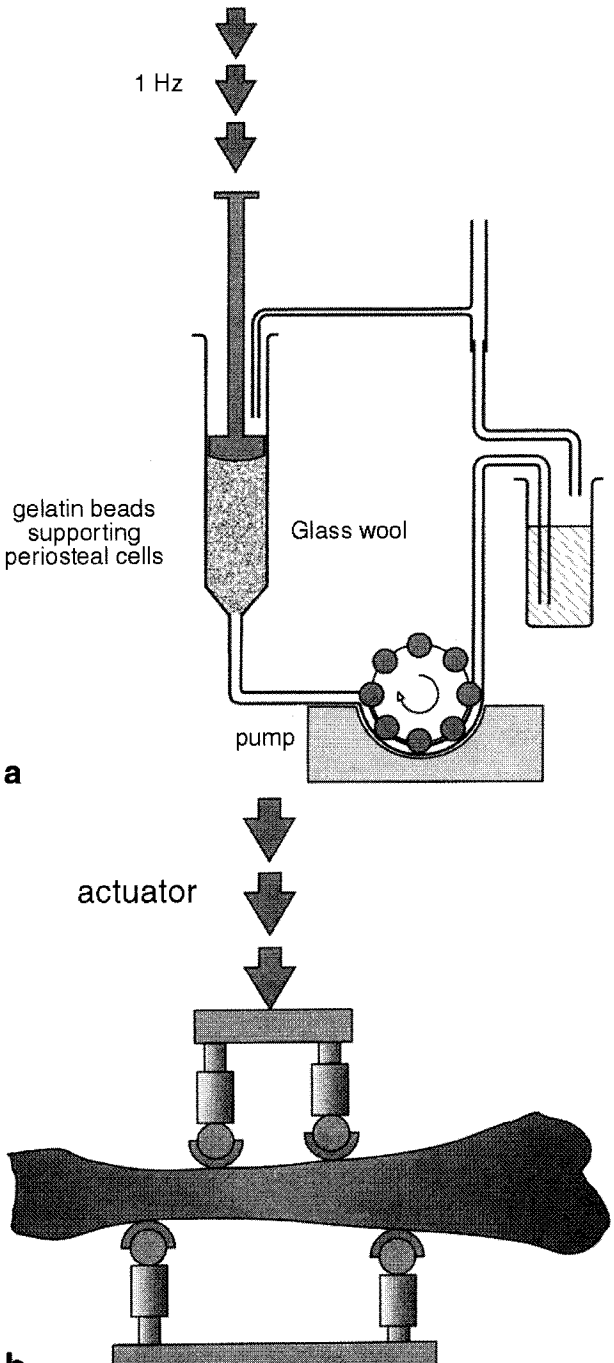

Fig. 1. (a) Bead column testing model. Cells are cultured on macroporous gelatin beads compacted inside a glass column. The system is continuously flushed with culture medium and subjected to cyclic force application. (Adapted from Shelton & El Haj 1992.) (b) Long-bone straining. In this test, the bone is treated as a standard beam and several material characteristics can be determined. The model can be refined by adding strain gauges and other actuators. (Adapted from Creighton University, Center for Hard Tissue Research (Hagino et al. 1993).)

umn: $\varepsilon=(L_o-L_{loaded})/L_o$. (Fig.1a). For solid bodies such as bones, various beam models may be relevant. Fig. 1b depicts the straining of a long bone using four point force application. Compressive and tensile stresses can be calculated using standard beam bending equations: $\varepsilon_c=M/EZ$ with: M: bending moment, E: modulus of elasticity and Z: section modulus. Other physical parameters that may be relevant are the polar moment of inertia $J_p=I_x+I_y$ and torque $T=\tau J_p/r$ with: τ: shear stress and r: radius (Akhter et al. 1992). A third method consists in gluing strain gauges directly onto bone. This approach has been taken, among others, by Lanyon et al. (1975) on an ankle and by Hylander et al. (1977) on the mandible of a primate. More recently Szivek et al. (1997) have presented calcium phosphate ceramic coated strain gauges whose contact with the bone surface was much improved relative to the previously used cyanoacrylate-bonded gauges. It was thus expected to improve sensing accuracy of the measuring device. The advantage of this technique being that physiologic strains ε can be directly measured *in vivo*. The testing setup, however, is complex and requires transcutaneous wire connections to external electronic devices.

In vivo strains

Basic multicellular units. Strain levels have clinical implications in orthopedics and related fields. Indeed, they significantly influence bone turnover in terms of ultimate growth or resorption. This interaction has been demonstrated by Hillam & Skerry (1995). Using fluorochromes and tartrate resistant acid phosphatase as markers, they demonstrated that labeling activity was significantly increased when rat ulnae were subjected to extraneous loading. To facilitate understanding of bone responses to mechanical strains, some physiologists parcel bone into Basic Multicellular Units (BMU) (Parfitt 1994). Each BMU is a functional group of osteoclasts and osteoblasts, precursor and supporting cells, capillaries and special organizations at the microscopic level. One such structure's average life is about 4 months during which it has been estimated to erode and fill approximately 0.003 (Recker 1983) to 0.05 mm^3 (Frost 1987, 1990) bone.

Bone mass. Skeletal bone mass is dependent on many parameters, a number of which can be directly or indirectly linked to hormonal activity (Miller et al. 1996). Bone mass increases by addition of new bone in both cortical and trabecular components thus resulting in periosteal expansion and endocortical contraction (Forwood & Burr 1993). Further, bone is essentially a composite material that comprises compact and trabecular moieties. Both of which are regulated to optimize the bone's strength-to-weight ratio. This latter aspect is of significance when considering the structural resistance of the bony housing that encases a dental implant in function.

Rho fraction and strain ranges. Normal bone physiology implies cycles of resorption and apposition, whereby the difference between the volume of bone that is resorbed and the quantity that is deposited is expressed as the "rho fraction" (ρ) (Frost 1994) (Fig. 2). A positive ρ indicates that bone mass increases whereas a negative ρ denotes bone resorption. Clinical and experimental evidence indicates that the ρ fraction is a function of the strain levels that act on the bone. Accordingly, strains may be subdivided into five ranges, none of which has definite boundaries (Fig. 3):

- *Disuse – bone resorption* – Depending on the author, typical bone strains in this range stay below 10 $\mu\varepsilon$ (Rubin & Lanyon 1985), 50 $\mu\varepsilon$ (Jaworski et al. 1980) or 200 $\mu\varepsilon$ (Martin & Burr 1989). Clinically the detrimental effects of weightlessness or bedrest on bone mass are known (Smith & Gilligan 1996). A similar phenomenon is likely to occur in the remaining alveolar bone after tooth extraction and may explain ridge resorption after tooth loss. In effect, the substantial drop in functional stress levels could be the main cause of the negative ρ fraction observed.
- *Normal load – bone homeostasis* – comprises the range between "disuse" ($\approx$100 $\mu\varepsilon$) and "mild overload" (2000 $\mu\varepsilon$). On the human tibial shaft which was the object of Lanyon et al.'s study

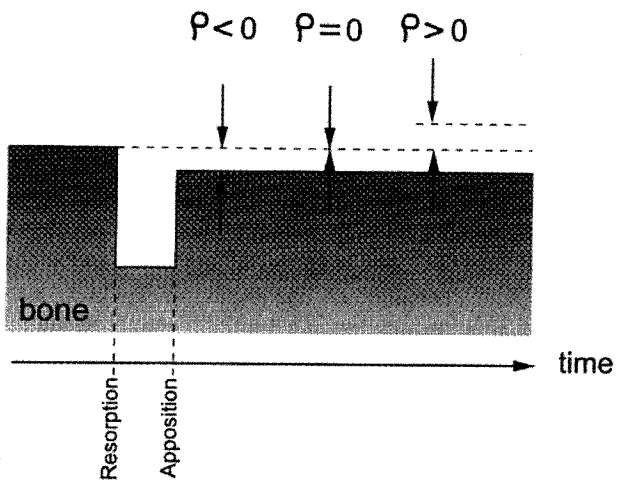

Fig. 2. Rho fraction. Any portion of bone (including the alveolar crest) is periodically resorbed and formed anew. The difference between the quantity that is resorbed and that is deposited is termed rho (ρ) fraction. A negative ρ fraction thus translates clinically into bone resorption.

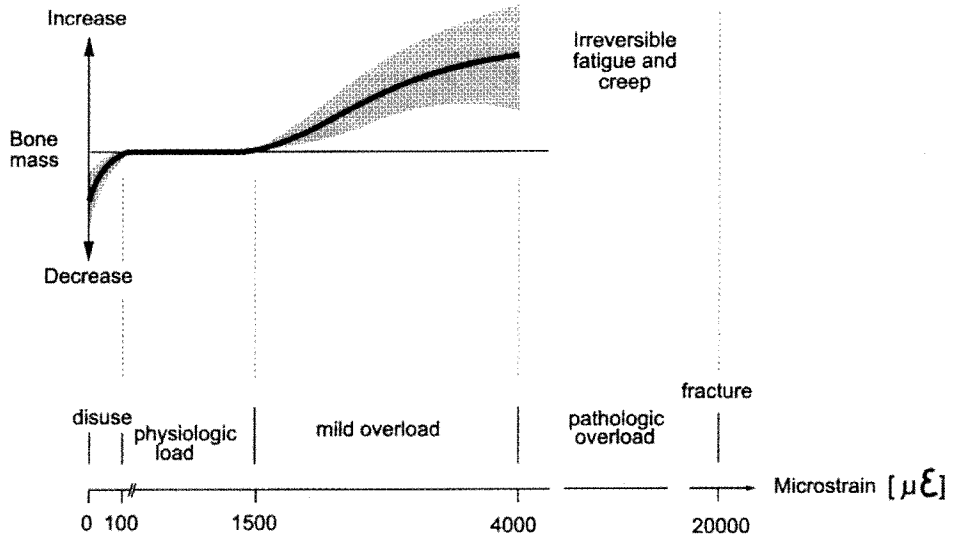

Fig. 3. Physiologic and pathologic strain levels and corresponding ρ fraction expressed as an increase or decrease in bone mass. All ranges defined are estimates and boundaries may vary by as much as 50%.

(1975), strains of (400 με) were recorded during normal walking. By definition, in this range, bone mass is in steady-state and the ρ fraction is zero.

- *Mild overload – bone mass increase* – covers the range between 2000 με and 4000 με. It includes the highest recording ever performed under physiological conditions: 3200 με were measured on the tibia of a galloping horse (Rubin 1984). Frost and others have suggested that strains in this range will cause bone mass to increase in response to the strains that appear inside the osseous structure. According to Frost, 1500–2500 με is the range of "minimum effective strain for mechanically controlled bone remodeling". That is the strain range above which bone mass will increase until peak strains are kept below a 1500–2500 με threshold (Rubin 1984) (mechanostat principle (Frost 1987)). It is known that microcracks do appear in heterogeneous anisotropic living tissue such as bone (Li et al. 1985) and it has been hypothesized that the damage caused by these fissures may act as a stimulus for bone remodeling (Currey 1962, Martin & Burr 1982).
- *Pathologic overload – irreversible bone damage* – A bony structure that is subjected to strains above 4000 με is unable to undergo normal processes of adaptation and repair. Therefore creep phenomena and fatigue flaws will appear. In implantology, such a situation has been created artificially by Isidor (1996). Implant suprastructures were placed on implants in such a way as to exentrically overload the fixtures. This arrangement caused about 60% of the test implants to fail by loss of osseointegration as evidenced by implant mobility and development of a radiolucent zone around the fixtures.
- *Fracture.* Tests performed by Cochran (1982) indicated that bone's flexural resistance is about 120 Mpa which corresponds to a stretching of about 2% (i.e. 20,000 με). According to Frost, the fracture strain is 25,000 με.

The strain ranges (i.e. windows) outlined earlier may not be adequate for all osseous structures and a "variable window" concept may be applicable. For instance Rawlinson et al. (1995) have shown that the mean strain levels on rat calvarial bone (30 με) was low compared to the ulnae (1000 με) of the same animals. They hence suggested that some cell types might be more strain-sensitive so as to adequately operate under small functional loads.

The above scheme is based on strain magnitudes as the main determinant of bone formation. Alternatively, some authors have linked bone mass alterations to strain frequency (Qin et al. 1998), strain history related parameters (Carter 1987), strain rate (O'Connor & Lanyon 1982), strain energy density (Fyhrie & Carter 1986) or strain gradients (Gross et al. 1997; Judex et al. 1997). Still, although conceptual refinements are likely to be forthcoming, strain magnitude will serve as the basis for the present discussion.

Experimental setups

Research in this field has essentially dealt with the osteogenic response in relation to two parameters: stress magnitude and time dependency (frequency,

waveform and resting periods). Experiments were conducted using a variety of models: *in vivo* long bones, cultured organs, wound chambers and "stretchable" plated tissue cultures. Accurate comparison between results are difficult though due to the variety of testing conditions which also include the age of the tissue (embryo, young, adult) and outcome assessment (increased synthesis of PgE_2, cAMP, IGF-1, non-collagenous protein or accumulation of mineral deposits, increased incorporation of ^{3}H-amino acid, etc.). However, in the context of the present analysis, results are pertinent only when data relative to cell deformation (με) are provided. As an example of *in vivo* setups, an externally loadable ulna preparation in adult turkeys similar in its principle to Fig. 1b was designed. In this experiment, a dose response relationship between daily peak strain magnitude and bone cross-sectional area could be demonstrated (Rubin & Lanyon 1985). When subjected to 100 stress cycles at 2000 με/day for 16 weeks, bone sections increased by 12.5% relative to controls (Rubin et al. 1995). In the testing setup described by Guldberg et al. (1997), a hydraulically activated bone chamber was implanted into canine tibial and femoral metaphyses. Intermittent strains ranging from −2000 to +3000 με were applied and significantly enhanced reparative bone formation as compared to unloaded contralateral controls.

The aim of this type of study is to identify the cellular and biochemical components which convert a physical stimulus into a biologic signal. Such a mediator would then control the transduction mechanisms, the initial deposition of mineralized material and the eventual formation of structured bone. Therefore, in contrast to *in vivo* setups, pure *in vitro* models were designed. The one described by Brighton et al. (1991) consisted essentially of a petri dish featuring an elastic base onto which the cells were cultured. When the bottom was inflated (like a balloon), its surface was stretched and the cells were distended to 200, 400 and 1000 με. Unfortunately, due to the heterogeneity of the time parameter, changes relative to controls were observed but no systematic pattern emerged. Other researchers have used a similar setup (peak strain: 4000 με) and found that several fractions of bone cells (i.e. distinct evolutionary stages between osteoblasts and osteocytes) reacted differently when subjected to an identical strain application (Mikuni-Takagaki et al. 1996).

To overcome the drawbacks of *in vivo* models (lack of direct access) and *in vitro* models (questionable force transfer), Zaman et al. (1992) have described an organ culture set-up. In this design, osteocytes were still integrated into a physiologic matrix thus being subjected to normal stress transfer patterns. It has been hypothesized that this model could duplicate phenomena such as a local increase of hydrostatic pressure (Frost 1987) or an alteration of electric potentials as stimuli for bone mass augmentation (Bassett 1984; Reich et al. 1990; Van den Kuij et al. 1985).

Finally, to investigate the effect of movement on bone formation (Cameron et al. 1973), Goodman and co-workers (1994) designed a "micromotion chamber" which was implanted into the tibial metaphysis of rabbits and in which two concentric chambers could be rotated relative to each other. While in this latter model, the relationship between the magnitude of motion and με is as yet undefined, it may quite well approximate the bone–metal interface of freshly placed implants.

As to the effect of micromovements, the overall relationship between bone formation and amplitude of movement could be described as in Fig. 4. Without interfacial motion osteoblasts will form bone. When subjected to minute (magnitude not defined) shear stresses, mineralized material deposition will increase. Beyond an optimum value, bone formation will decline and ultimately cease completely.

To summarize the findings, it appears that mechanical strains will increase production of mineralized material. Intermittent strains are essential (Hert et al. 1971) but the precise influence of frequency relative to peak strains is still unknown.

Finally, since more than 90% of bone cells are osteocytes (Parfitt 1977), a specific role of osteocytes as a mechanosensor is likely. However, although a great many biomolecules have been

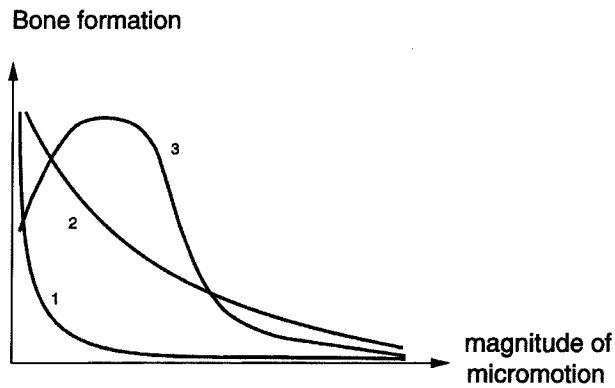

Fig. 4. Theoretical relationships between bone formation and magnitude of interfacial micromovement. (1) (i.e. complete immobilization) appears as unrealistic and disproved by available evidence on osseointegration. Pattern (2) is possible but indications are that some degree of micromovement (magnitude not defined) is actually beneficial to osseointegration (3) (Pilliar et al. 1986). This latter pattern would match the proposed concept of stress that induces strain (i.e. deformation and micromovement) which in turn induces bone formation. The actual magnitude of micromotion is undefined.

Table 1.

Strength (Reilly et al. 1974):	longitudinal	tension:	133 Mpa
		compression:	193 Mpa
		shear:	68 Mpa
	transverse	tension:	51 Mpa
		compression:	133 MPa
Modulus of elasticity:	cancellous bone:		±4.5 Gpa (Choi et al. 1990)
	cortical bone:	longitudinal:	17.0 Gpa (Reilly et al. 1974)
		transverse:	3.3 Gpa (Reilly et al. 1974)
	titanium:		±120 GPa (Ashby & Jones 1986)

linked to increased bone activity (Zaman et al. 1997), a mechano-specific growth factor which may be responsible for integrating the strain response has yet to be identified (El Haj 1990). Alternatively, a mechanism based on extracellular fluid shear stresses has recently been put forward by Weinbaum (1998).

Load transfer on dental implants

Mathematical models

A number of publications have presented – at times extremely elaborate – mathematical models of the load transmission on dental implants. The mechanical properties of the metal and the bone (Table 1) were characterized as to their modulus of elasticity, their Poisson's ratio (the relationship between diametral contraction to elongation when a cylindrical body is stretched) and their yield energy levels under tensile and shear stresses (Von Mises criterion a.o.). Finite element analyses were performed for implants, surrounding bone and the metal–tissue interfaces (Rieger et al. 1989; Van Rossen et al. 1990; Kregzde 1993; Van Zyl et al. 1995). In general terms, these studies tend to promote the idea that the type of stress that is active at the implant–bone interface may be inadequate either quantitatively or qualitatively (Mihalko et al. 1992). Many authors favor a concept of "local overload" to explain the "saucerization" of the bony crest adjacent to implants. The exact mechanism is unclear but a "microfracture" concept is often used (Roberts et al. 1989). Only one research group has investigated the view that there might not be enough stresses generated to maintain bone homeostasis (i.e. negative ρ fraction) (Deporter et al. 1990; Pilliar et al. 1991; Vaillancourt et al. 1995).

Computed strain levels

In spite of the availability of the data reviewed above, few authors have attempted to correlate functional force levels with the degree of straining of the supporting bone. Vaillancourt et al. (1995) have estimated the upper limit of the disuse range to be at 1.6 Mpa. According to the $\varepsilon=\sigma/E$ relation, this would correspond to a microstrain level of 137 με −360 με depending on the value chosen as the bone's modulus of elasticity. A more elaborate albeit still schematic model was proposed by Lum & Osier (1992). In this simulation, the implant was maintained by an upper and a lower layer of cortical bone separated by an intervening cancellous band. Under the assumptions made (implant length: 10 mm, crown length: 8 mm, horizontal force component 6 N) the resulting maximal horizontal pressure at the bottom of the implant was computed as 1.92 Mpa. Therefore, if $E_{\text{cancellous bone}}$ was set to 4500 MPa, the resulting strain was 417 με.

It should be mentioned here that models such as the one published by Lum & Osier should be handled with caution as they are extremely sensitive to the parameters used. For instance available data for horizontal force vectors range from 6 N (Lum & Osier 1992) to 11–21 N (Richter 1998) to 40–50 N (Graf & Geering 1977). Depending on the force value used, the microstrain level in the alveolar bone can be vastly out of range. Besides, these models do not yet account for the stress concentrating effect of the surface texture.

The microstrain ranges described earlier have been obtained by applying forces to long bones or cell cultures. How this translates into force transmission at the bone–implant interface is still unclear. In particular, it is not clear whether localized, intermittent strains above the 4000 με limit will have the same deleterious effects as in other types of osseous structures.

Notwithstanding inadequacies in the design of the mathematical modeling or the physical parameters used, it appears that the stress levels at the bone margin–implant interface are within physiological bounds and the horizontal strain levels generated during function do promote bone homeostasis at the bony crest.

And yet, polished implant collars do not integrate (Hämmerle et al. 1996)!

Stress shielding

One explanation that is prevalent in the orthopedic literature is related to an effect known as "stress shielding" (Pilliar et al. 1979; Weinans et al. 1994). Stress shielding is caused by the difference in elasticity of bone and artificial endoprostheses (Head et al. 1995). In effect, during function, bone will flex more than the much stiffer prosthetic component, thereby creating zones of unloading of the osseous tissue. These zones thus lack adequate stimulation and bone atrophy or osteoporosis (Johnston et al. 1995) results. Hence, some researchers aim at developing materials whose properties would range close to natural structures and thus allow for even stress distribution during implant function (Zhang et al. 1996).

In all likelihood however, stress shielding is limited to the field of orthopedic endoprostheses such as hip stems in which the force vector range is limited and the hip stem flexes in near parallel planes. Conversely, explanations related to differences of bone and implant stiffnesses make little sense in dental implantology. Indeed available evidence suggests that teeth are subjected to loads in varying directions (Wiskott & Belser 1995) and therefore the implant's neck should subject the majority of the cicumferencial bone to adequate strain levels and thus preclude resorption.

Clinical implications

When applied to dental implantology, the concept of physiologic vs non-physiologic strain levels may explain the satisfactory outcome of some immediate loading protocols in which splinting is involved. In effect, at the time of implant placement, bone contact is generally poor. Therefore by distributing forces to the surrounding bone, the immediate post-operative splinting of implants using bars (Chiapasco et al. 1997) or full-arch restorations (Tarnow et al. 1997) may decrease strains generated during function on the few areas of bone contact to the 'mild overload' range and thus ensure adequate integration.

Data have been published as to the high failure rate of implants placed in type IV bone structure (Jaffrin & Berman 1991) implying that the "spongy" structure *per se* was detrimental to implant stability. Yet the mechanostat principle (Frost 1987) states that the bony housing should adapt to the loads applied *via* the implant (Ogiso et al. 1994; Steflik et al. 1995; Celetti et al. 1995) and densify the initially loose osseous structure. Therefore an alternate explanation would state that the implants that failed lacked adequate primary integration because of poor initial coupling due to the looseness of the bone structure. The net effect was that the implants were only partially integrated, that is, these implants had bone–metal contact only on a limited part of the implant housing. Due to inadequate distribution of the contact area, the surrounding bone mass could not adjust to functional and/or parafunctional loading. Indeed under physiological conditions (i.e. without barrier techniques), indications are that the bone–implant interface will only densify in response to functional demands below the most coronal bone layer (Ogiso et al. 1994; Celetti et al. 1995) and that osseous tissue cannot "grow" coronally to optimize stress transfer to the alveolar bone. The failing implants described by Jaffrin & Berman, after having integrated in some portions of the implant's surface, may thus subsequently have loosened either by fracture or progressive bone degeneration due to strains above the "mild overload" range.

Coupling

Conceptually, "coupling" is the opposite of "stress shielding" (i.e. "uncoupling"). Osseointegration can be regarded as the healing phenomenon by which bone regains its original shape (i.e. before implant placement) and includes the embedded titanium body as if it were osseous tissue. Biomechanical implant–bone coupling exists when appropriate force transfer takes place between both media. The implant surface must thus establish a mechanical continuum with the surrounding bone. For this to occur, intimate tissue adaptation is essential.

Coupling and bone stimulation are thus closely interrelated. Bone stimulation will occur during any facial muscular activity that induces some degree of flexing inside the maxillae. Comminution of food is a prime example but lower stress levels such as those generated during swallowing, sucking or yawning (Gates & Nicholls 1981) will also to some extent strain the upper and lower jaw bones.

Biomechanical coupling is the basis for the alternative hypothesis we wish to offer to explain the initial resorption phenomena observed at the alveolar crest surrounding dental implants.

The importance of implant surface texture

Premises

At this point, several premises should be established. Hence it is postulated that:

1. There is a relation between the arrest of crestal bone resorption and implant surface profile characteristics. To ensure bone containment, the implant's surface must be either machined

as threads or given a rough texture. Indications are that surface structure will influence cell positioning (Eisenbarth et al. 1996). It is unclear, however, how contact guidance translates into synthesis of mineralized material given the absence of biomechanical strains in these experimental designs. Whether textured and smooth surfaces are different in terms of their surface properties at the molecular level (Kasemo & Lausmaa, 1986) is unclear. One 1993 study by Larsson et al. (1996) failed to demonstrate systematic differences in bone responses between machined, electropolished and surfaces with two different thicknesses of TiO_2 layers.

2. Horizontal overload cannot be the cause for the limited crestal bone resorption observed. Indeed "saucer-shaped" bone loss appears clinically as a self-limiting phenomenon. Yet in the "overload" concept, the supracrestal lever would continually increase while the resisting (intrabony) lever would be reduced concomitantly, thereby initiating a cycle of events which could not stop in any other fashion than by overloading the implant (i.e. "pathologic range") and/or fracturing the implant out of its bony housing (Fig. 5).
3. For mature osseous structures (i.e. not during developmental stages), strains of adequate magnitude are essential for bone homeostasis. Since any viable bone (including the alveolar crest) is periodically resorbed and formed anew, a zero or positive ρ fraction can only be achieved if strain levels stay above the "disuse range". Bone will thus neither predictably form nor stably remain in areas that are located outside fields of appropriate stress levels. In implantology, this statement translates clinically into the observation that leaving a space for the ingrowth of bone-derived cells using barrier techniques (Dahlin et al. 1990) will not *per se* ensure bone formation. Indeed, according to the mechanostat principle, the various parts of the skeleton are at an optimum strain value and, even if an adequate void is provided, there may be "no incentive" for bone to grow into it if strain levels are too low. This is in contrast with the application of barrier techniques in a periodontal context where PDL regeneration appears as being essentially related to competitive colonization of the root surface (Aukhil et al. 1985).
4. Macroscopically, bone responds to mechanical stress in a remarkable fashion that is best expressed in Wolff's law (Wolff 1892; Cowin 1986) as "form follows function". This law (actually a *principle* rather than a *law*) states that the overall shape of bones adapts to stress application. Therefore localized defects in the contour of the maxillae that have only a minor influence on the distribution of force flow lines (Wiskott et al. 1995) may never be subjected to remodeling so as to integrate themselves into a homogenous bone shape.
5. It follows from items #3 and #4 that global and local factors are active in bone remodeling. Global factors will determine the overall shape of bones so as to enable them to respond to functional demands as expressed in Wolff's law. Conversely, local factors are active on-site only (within a range of tenths of a millimeter) and will, for instance, determine the quality of implant integration. In this respect, besides a host of cellular and molecular aspects, it is likely that adequate force transfer (i.e. coupling) between an implant and its bony housing is essential.

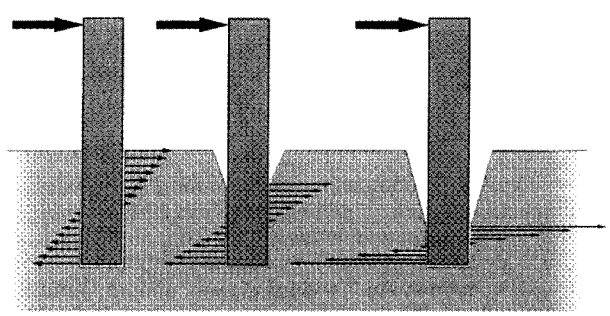

Fig. 5. Strains on implants with varying bone heights. When bone height is lost, the resulting lever system becomes more unfavorable and stresses applied at the bone crest may increase dramatically.

Surface texture as stress distributor

We suggest that textured surfaces provide the type of mechanical link between metal and bone that is required for appropriate stimulation of osseous tissue. Not primarily in terms of a mechanical interlock but because, by virtue of their irregular shape, threads, grooves, pits, notches etc. will create a heterogeneous field of force vectors inside the surrounding bone during muscular activity. It is proposed that a discrete level of the differential stress magnitudes thus generated will match the strain level (με) required to trigger specific lineages of bone cells to start bone production (Fig. 6). Once this occurs, bioactive molecules are synthesized and activate the surrounding cell population to form bone as well. Strains that range outside the "physiologic" and "mild overload" range serve no "useful" purpose in terms of bone mass homeostasis or augmentation.

Heterogeneous stress fields typically develop in the material that surrounds threaded devices.

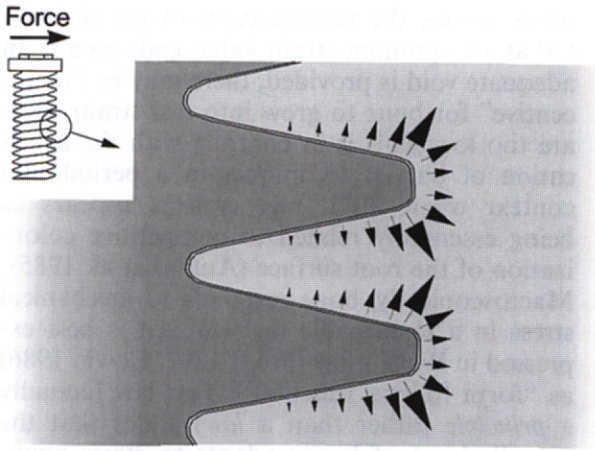

Fig. 6. Threads as stress distributors. Due to their uneven contour, threads (or plasma coatings or sandblasted surfaces) will generate a heterogeneous stress field. It is proposed that discrete levels of the stresses thus created will match the "physiologic load" or "mild overload" range thus prompting osteocytes to form new bone.

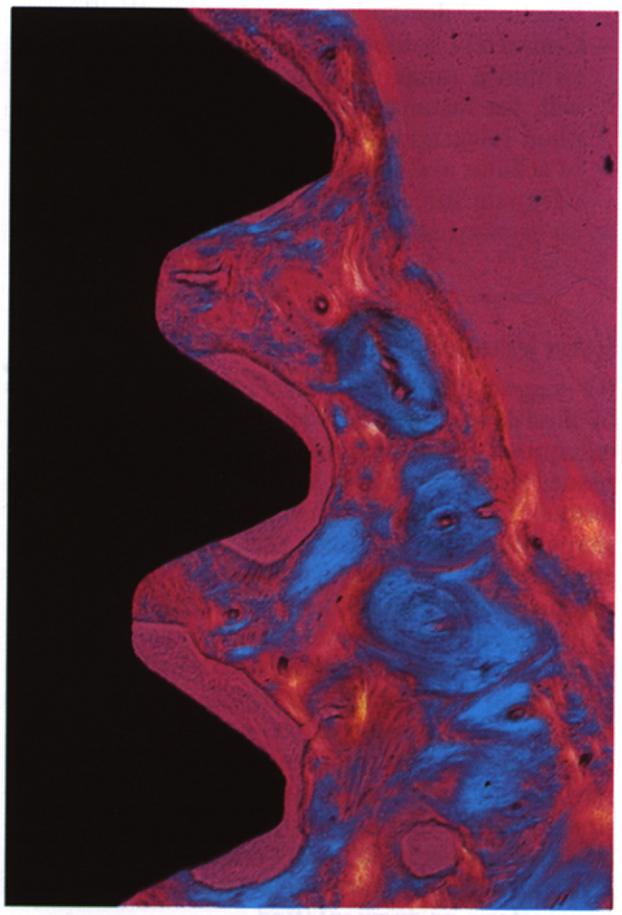

Fig. 7. Cuplike bone formation on implant thread depicted in Fig. 6. (From Wehrbein & Diedrich (1992), reprinted with permission.)

Using photoelasticity techniques, they have been demonstrated for endodontic posts (Standlee et al. 1972) as well as in bolt–nut type connectors (O'Hara 1997). The same effect was evidenced in the bony housing surrounding dental implants. Kohn et al. (1992) concluded "For all characteristic loading conditions, the local strain field within the interfacial zone is inhomogeneous. [...] strain is concentrated at the point where bone contacts the outer edge of the thread and strain decreases from the exterior to the interior regions of the thread." More recently Hansson & Werke (1997) also showed that the shape of the thread profile affected the magnitude of the stresses in the bone.

When strains decrease below a specific level, bone formation will cease, bone production thus being an "all or nothing" phenomenon. Indeed histologic slides invariably show definite islands of osseous tissues and no zones of diffuse calcification (Frost 1997). One remarkable illustration is presented in Fig. 7. The implant shown was used as anchorage for orthodontic tooth movement using a spring. That is a rather constant force level to which functional chewing strains were superimposed. One explanation for the cuplike bone tissue on top of the threads would be that the leverage systems that were active during tooth movement

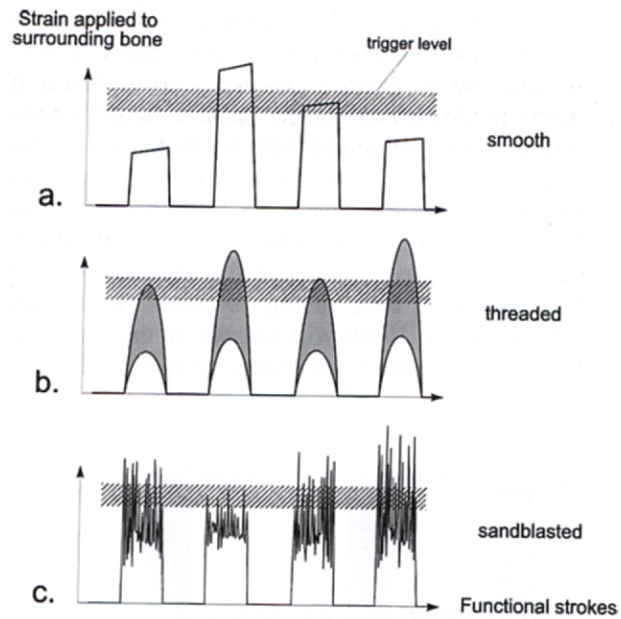

Fig. 8. Explanatory diagram of differential stress production. Two dimensional view of three dimensional phenomenon. (a) Smooth surface stress generation. Strain levels might reach appropriate levels but are limited in time and distribution on the load bearing surface. (b) Threaded surface. Due to the profile of the thread, an envelope of strains is generated during each muscular stroke. (c) Ragged, sandblasted surface. The strains generated are distributed erratically but span a wide range both in time and space. ▨: strain range appropriate for triggering osteocytes to form bone.

matched precisely the strain level needed for the osteocytes to start bone production in this area. Alternatively, we suggest that the order of magnitude was adequate, but that it were the implant threads that have provided the heterogeneous stress field needed to induce strains of appropriate magnitude and thus trigger bone formation.

Referring to the statement made in the previous section "smooth surfaces don't integrate", it is likely that bone stimulation by smooth surfaces can only be of adequate magnitude by chance since it is a linear function of a unique variable, that is the load applied to the implant during functional strains on the maxillae. This situation would occur, say, one out of 10, 50 or 100 cycles and may be insufficient to induce bone formation. Conversely, a rough surface *per se* will generate "trigger-ready" levels during each straining cycle of the jaw bones (Fig. 8).

The stress distribution principle developed herein is not linked to a specific force transducing mechanism. Whether bone formation depends on strain magnitude, strain rate, other time- or surface-dependent interactions or microcrack growth (Taylor 1997) has no bearing on the pertinence of our proposal since all are basically dependent on local stress fields.

Moreover, rather than immobilization (Ivanoff et al. 1996), a concept that integrates adequate bone stimulation via biomechanical coupling may well be the explanatory key to successful osseointegration. Indeed, with reference to cell straining as trigger for bone production, biomechanical coupling would account for 1) the lack of osseointegration observed in the absence of congruent implant bed and implant (Carlsson et al. 1988), 2) the positive effect of micromovements according to Fig. 4 since, perioral and masticatory muscular activity will flex the mandibular and maxillary bones.

Final remarks

The present discussion addresses only the elementary processes that are related to strains and bone formation. A number of issues were purposely omitted:

- The influence of infection and plaque-associated disease;
- Bone repair processes after fracture;
- The significance of implant surface characteristics at the molecular level;
- The role of bioactive molecules;
- Processes that are active during growth or repair and that are inactivated in later stages;
- Differences in bone structure in terms of cortical thickness and trabeculation.

The concept proposed herein of surface texture as stress levels distributor does by no means suggest that adequate strain levels are the only requirement for successful integration of load bearing surfaces. We merely suggest that this aspect be included into future research. In particular we advocate that future experimental models of bioactive surfaces include mechanical strains as a parameter (Cooper et al. 1998).

Résumé

Le col poli des implants dentaires ne s'intègre pas à l'os alors que les surfaces rugueuses le font. Des effets semblables ont été rapportés dans la littérature orthopédique sur les prothèses de hanches. En dentisterie, l'absence d'ostéointégration a été attribuée à une pression augmentée sur le lit osseux durant le placement de l'implant, l'établissement d'une largeur biologique physiologique, le stress de protection et le manque d'association biomécanique adéquat entre la surface implantaire supportant la charge et l'os alvéolaire avoisinant. Parmi les nombreuses variables pouvant influencer l'ostéointégration, cette analyse propose d'inclure le transfert de stress comme une variable significative. C'est pourquoi, ce présent rapport discute la relation entre les stress appliqués et l'homéostase osseuse. Toute structure osseuse viable (incluant le tissu entourant le col de l'implant poli) est sujete à des phases périodiques de résorption et de formation. Des données cliniques et expérimentales ont montré les effets néfastes du manque de fonction en montrant que la masse osseuse diminuait avec le temps. Vu des stimuli mécaniques inadéquats l'os est résorbé durant son renouvellement normal et de moindres quantités d'os que précédement sont déposées, un procédé qui est observé cliniquement en tant que résorption. Le stress qui provoque cette résorption osseuse maintient ou augmente sa masse, et le niveau qui en fin de compte amène l'os à une fracture osseuse a été décrit dans la littérature. En plaçant ces valeurs dans la situation des implants dentaires, il s'ensuit que si l'ensemble doit être stable, l'os crestal doit être soumis à des niveaux adéquats de stimulation mécanique. Les surfaces lisses n'apporteront pas d'association biomécanique adéquate avec l'os avoisinant le col de l'implant vu que les limites de stress induites par une surface polie sont faibles. La texture de surface des implants à filetage, recouverts de plasma, ou soufflés par du sable provoquerait un champ de stress héterogène autour d'un implant en fonction. Un tel stress inclus des niveaux de force qui provoqueront donc la néoformation osseuse. Durant la formation du renouvellement osseux les ostéoblastes seront donc davantage stimulés par des signaux biomécaniques d'une amplitude appropriée.

Zusammenfassung

Man hat beobachten können, dass die polierten Implantathälse nicht so osseointegrieren, wie wir das von strukturierten Oberflächen gewohnt sind. Ähnliche Beobachtungen wurden in der orthopädischen Literatur über künstliche Hüftgelenksprothesen beschrieben. In der Zahnmedizin wurde die fehlende Osseointegration dem übermässigen Druck auf das Knochenbett während dem Eindrehen des Implantates, dem Einstellen einer physiologischen "Biologischen Breite", einem Selbstschutz des Knochens und einer ungenügenden biomechanischen Wechselwirkung zwischen der lastaufnehmenden Implantatoberfäche und dem umgebenden Knochen zugeschreiben. Neben den vielen verschiedenen Variablen, welche die Osseointegration beeinflussen können, kommt diese Analyse zum Schluss, dass die Kraftübertragung ein signifikanter Faktor sein muss. Deshalb diskutiert diese Arbeit auch die Wechselwirkung zwischen Kraft-

einwirkung und Knochenhomöostase. Jede lebende knöcherne Struktur (darin eingeschlossen sind auch die den polierten Implantathals umgebenden Gewebe) durchläuft periodisch Phasen der Resorption und der Neubildung. Klinische und experimentelle Daten haben die schädlichen Einflüsse fehlender Funktion gezeigt, nämlich dass die Knochenmasse mit der Zeit abnimmt. Infolge ungenügender mechanischer Reize wird Knochen, der während den normalen Umbauvorgängen resorbiert wird, in geringeren Mengen wiederangelagert als vorher, ein Vorgang, der klinisch als Resorption beschrieben wird. Die verschiedenen Kräfte, welche Resorption oder Vermehrung der Knochenmasse verursachen und auch der Grenzwert, der unter Umständen den Knochen zur Fraktur bringen kann, wurden in der Literatur festgelegt. Wenn wir diese Werte auf die Situation der Zahnimplantate übertragen folgt daraus, dass der Knochenkamm, sofern er stabil bleiben soll, einem angepassten Ausmass an mechanischen Reizen ausgesetzt werden muss. Wir vermuten, dass die glatten Oberflächen eine ungenügende biomechanische Verbindung mit dem den Implantathals umgebenden Knochen eingeht, so dass das Ausmass der durch eine polierte Oberfläche induzierten Kraft beschränkt ist. Wir glauben auch, dass die Oberflächenstruktur von geschraubten, plasmabeschichteten oder sandgestrahlten Implantaten ein heterogenes Kräftemuster um in Funktion stehende Implantate bewirkt. Ein solches Kraftfeld, bestimmt durch das Implantatdesign, schliesst Kraftspitzen ein, die konduktiv für die Knochenbildung sind. Somit werden die Osteoblastensäume durch Reize von adäquater Grösse während der Bildungsphase des Knochenumbauprozesses bedeutend mehr stimuliert.

Resumen

Se ha observado que el cuello pulido de los implantes dentales no se osteointegra como lo hacen las superficies rugosas. Se han reportado hallazgos similares en la literatura ortopédica en endoprótesis de cadera artificial. En dentistería, la ausencia de osteointegración se atribuyó a un incremento de la presión en el lecho óseo durante la colocación del implante, a un establecimiento de una "anchura biológica" fisiológica, a una protección de estrés y a una ausencia de un acoplamiento biomecánico adecuado entre la superficie del implante orientada a la carga y el hueso circundante. Entre las muchas variables que pueden afectar la osteointegración, este análisis propone incluir la transferencia de estrés como una significativa. Por lo tanto, el presente artículo discute la relación entre el estrés aplicado y la homeostasis ósea. Cualquier estructura ósea viable (incluyendo el tejido que rodea el cuello pulido del implante) es sometido a fases periódicas de reabsorción y formación. Los datos clínicos y experimentales han mostrado los efectos perjudiciales de la ausencia de función en la que la masa ósea disminuye con el tiempo. Debido a un estímulo mecánico inadecuado, el hueso que se reabsorbe durante el recambio normal se redeposita en menores cantidades que con anterioridad, un proceso observado clínicamente como reabsorción. Los rangos de estrés que causan la reabsorción ósea, mantienen o disminuyen su masa y el nivel que eventualmente causa la fractura del hueso ha sido delimitado en la literatura. Aplicando estos valores a los implantes dentales, se considera que para ser estable, el hueso crestal debe ser sometido a niveles adecuados de estimulación mecánica. Nosotros sugerimos que las superficies lisas no van a suministrar un acoplamiento biomecánico adecuado con el hueso que rodea al cuello del implante en el cual el rango de estrés inducido por una superficie pulida es limitado. Nosotros proponemos que la textura de la superficie de implantes roscados, cubiertos de plasma o espolvoreados genera un campo de estrés heterogéneo alrededor de un implante en función. Por diseño, tal campo de estrés incluye niveles de fuerza que son conductivos para la formación ósea. Por lo tanto, durante la fase de formación de recambio óseo, los linajes de osteoblastos parecen ser más estimulados por señales biomecánicas de magnitud apropiada.

要旨

歯科インプラント頚部の研磨表面は、粗い表面構造とは異なり、骨性統合が起きないことが観察されてきた。類似の所見は整形外科の人工股関節においても報告されている。歯科において骨性統合の欠如は、インプラント埋入時の骨床に加わる過度の圧力、生理的な"生物学的幅径"の確立、応力の遮蔽と、荷重インプラント表面と周囲の骨の間の適切な生体力学的な結合の欠如によるものと考えられてきた。

本報告は、骨性統合に影響を及ぼしうる多くの変数の中に、重要なものの一つとして、応力伝達を含むことを提案している。従って本報告は加えられる応力と骨ホメオスターシスの間の関係を考察している。

全ての生きた骨構造は（インプラント頚部の研磨面の周囲の骨組織も含めて）定期的に吸収と生成の段階を繰り返している。臨床的及び実験的データは、機能の欠如は有害な作用を及ぼし、経時的に骨塊が減少することを示している。正常な代謝中に吸収される骨は、器械的刺激が不足する場合には再沈着の量が減少するという過程は、臨床的には吸収として観察される。骨の吸収、維持あるいは増加をもたらす応力の領域及び骨の破折を起こす応力のレベルは、文献で明らかにされている。これらの値を歯科インプラントの状況にあてはめると、インプラントが安定するには歯槽頂骨は適切なレベルの器械的刺激にさらされなくてはならないと言える。

われわれは、研磨表面で生じる応力の領域は限られているため、滑沢な表面はインプラント頚部の周囲の骨に適切な生体力学的な結合をもたらさないと考える。ねじ式、プラズマ溶射あるいはサンドブラストを施したインプラントの表面構造は機能中のインプラント周囲に均質な応力の場を生み出すと提案する。デザイン上、このような応力の場は、骨伝導性のある力のレベルを含むものである。従って骨代謝の生成の段階で、骨芽細胞は適度の生体力学的信号によって刺激を受ける可能性が極めて高いと考えられる。

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