

Review

# Biological factors contributing to failures of osseointegrated oral implants

## (I). Success criteria and epidemiology

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The aim of this review was to offer a critical evaluation of the literature and to provide the clinician with scientifically-based diagnostic criteria for monitoring the implant condition. The review presents the current opinions on definitions of osseointegration and implant failure. Further, distinctions between failed and failing implants are discussed together with the presently used parameters to assess the implant status. Radiographic examinations together with implant mobility tests seem to be the most reliable parameters in the assessment of the prognosis for osseointegrated implants. On the basis of 73 published articles, the rates of early and late failures of Brånemark implants, used in various anatomical locations and clinical situations, were analyzed using a metanalytic approach. Biologically related implant failures calculated on a sample of 2,812 implants were relatively rare: 7.7% over a 5-year period (bone graft excluded). The predictability of implant treatment was remarkable, particularly for partially edentulous patients, who showed failure rates about half those of totally edentulous subjects. Our analysis also confirmed (for both early and late failures) the general trend of maxillas, having almost 3 times more implant losses than mandibles, with the exception of the partially edentulous situation which displayed similar failure rates both in upper and lower jaws. Surgical trauma together with anatomical conditions are believed to be the most important etiological factors for early implant losses (3.6% of 16,935 implants). The low prevalence of failures attributable to peri-implantitis found in the literature together with the fact that, in general, partially edentulous patients have less resorbed jaws, speak in favour of jaw volume, bone quality, and overload as the three major determinants for late implant failures in the Brånemark system. Conversely, the ITI system seemed to be characterized by a higher prevalence of losses due to peri-implantitis. These differences may be attributed to the different implant designs and surface characteristics. On the basis of the published literature, there appears to be a number of scientific issues which are yet not fully understood. Therefore, it is concluded that further clinical follow-up and retrieval studies are required in order to achieve a better understanding of the mechanisms for failure of osseointegrated implants.

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Oral implants have revolutionized the practice of dentistry. Over the years, a large number of different implant systems have been introduced. As shown in Table 1, a classification of oral implants can be based on placement modality and implant

design. Reviews on currently used oral implants have been presented (1, 2). Due to the long-term clinical use of osseointegrated oral implants, the scientific literature is extensive and continuously increasing. The present review will exclusively focus

Table 1  
*Classification of oral implants*

Subperiosteal implants	
Ramus frame implants	a "hybrid" between subperiosteal and endosseous implants
Endosseous implants	{ pins and needles blades and disks root-formed analogues (screws, cylinders, hollow baskets, truncated cones and combination forms)
Transosteal implants	mandibular staple bone plate and transmandibular implants

on osseointegrated systems (root-formed analogues; Table 1). Relevant articles dealing with various osseointegrated implant systems are reviewed. However, the epidemiological data on implant failures are essentially based on the Brånemark system, due to the fact that too little scientific information is available regarding the long-term performances of other implant systems (3, 4).

Many experimental and clinical studies have focused on the mechanisms of tissue integration and the possibilities to secure long-term success. Closely related to these issues are, however, the less well-known biological processes which may contribute to the failure of an implant.

The aim of the present article was to identify, the biological factors contributing to failures of osseointegrated oral implants. The first part of this review includes: definitions of implant failures, parameters for success/failure evaluation, reliable diagnostic tools for the evaluation of implant conditions and the epidemiology of implant losses. In the second part of the review, which will be published separately, the etiology and the pathophysiology of failing implants will be discussed. Only the English language literature was evaluated.

### Definitions of osseointegration, success and failure

The concept of *osseointegration* was developed by BRÅNEMARK (5) in the middle of the 1960s and led to the predictable long-term success of oral implants. Osseointegration has been defined from various viewpoints, including the description of long-term clinical results, a numeric evaluation of interfacial mechanical capacity, and the morphological appearance of the tissue-implant interface (6). One of the first definitions of osseointegration, given by ALBREKTSSON *et al.* (7), was a "direct functional and structural connection between living bone and the surface of a load-bearing implant". Subsequently, several other definitions of osseointegration were presented (8, 9). According to ZARB & ALBREKTSSON (10), the only acceptable definition of osseointegration is based on a clinical examina-

tion. These authors described osseointegration as "a process in which a clinically asymptomatic rigid fixation of alloplastic material is achieved and maintained in bone during functional loading". This is in contrast to implants surrounded by fibrous connective tissue (fibrointegration), which have shown a clinically discernible mobility when loaded (11).

Obviously, it is of great interest to be able to define osseointegration. According to the Dorland Illustrated Dictionary (1994, p. 1198), osseointegration is the direct anchorage of an implant by the formation of bony tissue around the implants without the growth of fibrous tissue at the bone-implant interface. On the basis of previous suggested definitions, a provision of definitions from various viewpoints was recently given by BRÅNEMARK (12) (Table 2).

Success, in general terms, can be defined as the gaining of what is aimed at. Therefore, to be considered successful, an osseointegrated oral implant has to meet certain criteria in terms of function (ability to chew), tissue physiology (presence and maintenance of osseointegration, absence of pain and other pathological processes) and user satisfaction (aesthetics and absence of discomfort). Obviously, every single implant has to fulfill and be tested for all the defined success criteria, otherwise it should be considered as *surviving*. This term applies to those implants which are still in function, but which have not been tested with respect to success criteria, or where neither the criteria for success or failure are met (13, 14).

Consequently, a failure may be defined as the first instance at which the performance of the implant, measured in some quantitative way, falls below a specified, acceptable level (15). This definition of implant failure includes a great deal of arbitrariness and encompasses a large variety of clinical situations, ranging from all symptomatic mobile implants to implants showing more than 0.2 mm of peri-implant bone loss after the first year of loading (14, 16), or bleeding pockets exceeding 5 mm of probing depth (17).

As shown in Table 3, failures can be divided into

Table 2  
Definitions of osseointegration (12)

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(a) *From the viewpoint of the patient*  
An implant fixture is osseointegrated if it provides a stable and apparently immobile support of a prosthesis under functional loads, without pain, inflammation or loosening over the lifetime of the patient.

(b) *From a viewpoint of macro- and microscopic biology and medicine*  
Osseointegration of a fixture in bone is defined as the close apposition of new and reformed bone in congruence with the fixture, including surface irregularities, so that at light microscopic level, there is no interpositioned connective or fibrous tissue and that a direct structural and functional connection is established, capable of carrying normal physiological loads without excessive deformation and without initiating rejecting mechanisms.

(c) *From a macroscopic biomechanical point of view*  
A fixture is osseointegrated if there is no progressive relative motion between the fixture and surrounding living bone and marrow under functional levels and types of loading for the entire life of the patient and exhibits deformations of the same order of magnitude as when the same loads are applied directly to the bone.

(d) *From a microscopic biophysical point of view*  
Osseointegration implies that at light microscopic and electron microscopic levels, the identifiable components of tissue within a thin zone of a fixture surface are identified as normal bone and marrow constituents which continuously grade into a normal bone structure surrounding the fixture: that mineralized tissue is found to be in contact with the fixture surface over most of the surface within nanometers so that no functionally significant intervening material exists at the interface.

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*biological failures* (related to biological processes) and *mechanical failures* of the components (including fractures of implants, coatings, connecting screws and prostheses). An *iatrogenic failure* can be defined as one characterized by a stable and osseointegrated implant, but due to malpositioning, it is prevented from being used as part of the anchorage unit. This group also includes implants which have to be removed due to violation of anatomical structures such as the inferior alveolar nerve. Another group of failures can be related to *inadequate* or *insufficient patient adaptation* (psychological, aesthetical and phonetical problems). In the following, the review will only deal with biological failures.

A biological failure can be defined as the inadequacy of the host tissue to establish or to maintain osseointegration. Such failures can further be divided according to chronological criteria in *early* (or *primary*) failures (failure to establish osseointegration, i.e., an interference with the healing process) and *late* (or *secondary*) failures (failure to maintain the established osseointegration, i.e., processes involving a breakdown of osseointegration). One way to chronologically discriminate early from late failures may be to confine all implants removed before bridge insertion (or after an “appropriate” healing period) to the group of early failures. Similarly, all failures occurring after the prosthetic rehabilitation may belong to the group of late failures. Obviously, this subdivision is not sufficient because there is not yet any non-invasive method which may accurately determine if and to what extent an implant is osseointegrated. Recently, new techniques for a non-invasive evaluation of the bone-implant interface were described (18, 19), however, being still at experimental and clinical trial stage levels.

The division of biological failures into early and late failures is an attempt to provide a simple and practical sub-classification. In fact, this classification has been employed in most clinical studies and will also be used in the present review.

The loss of osseointegration is clinically manifested by a *peri-implant radiolucency* and *implant mobility*. These signs are considered to arise from the replacement of the highly specialized bone tissue with a fibrous connective tissue capsule, unable to contribute to the functional capacity of the bone-implant unit.

From both theoretical and practical points of view, the distinction between successful and failed implants on the one hand, and failing and failed implants on the other appears crucial, as it is possible that implants might fail in a slow and gradual way. Therefore, if reliable symptoms or signs distinguishing the early changes preceding the loss of osseointegration could be identified together with etiologic factors, a treatment modality might be provided.

In the literature, terms currently used to indicate

Table 3  
Classification of oral implant failures according to the osseointegration concept

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Biological:	
Early or primary (before loading):	failure to establish osseointegration
Late or secondary (after loading):	failure to maintain the achieved osseointegration
Mechanical:	fracture of implants, connecting screws, bridge frameworks, coatings, etc.
Iatrogenic:	nerve damages, wrong alignment of the implants, etc.
Inadequate patient adaptation:	phonetical, aesthetical, psychological problems, etc.

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failing implants or complications are: peri-implant disease, peri-mucositis and peri-implantitis (14). *Peri-implant* disease is a collective term for inflammatory reactions in the soft tissues surrounding functioning implants. *Peri-implant mucositis* is a term describing reversible inflammatory reactions in the soft tissues surrounding a functioning implant, whereas *peri-implantitis* refers to inflammatory reactions with loss of supporting bone in the tissue surrounding a functioning implant (14). These definitions may be regarded as working definitions. It is likely that, if the causes and processes which lead to implant failure are unravelled, the various definitions of failure may be more precise.

Finally, a clear distinction should be made between failures and complications. Complications might indicate an increased risk for a failure, but are either of temporary significance or amenable to treatment. In this paper, only established failures and failing implants will be discussed.

### **Evolution of parameters used for success/failure evaluation**

One of the first attempts to evaluate in an objective way the clinical performance of osseointegrated oral implants was made by the Swedish National Board of Health and Welfare in 1975 (20). Periodontal (gingival index, plaque index and pocket depths), prosthetic (type of occlusion) and radiographic parameters (absence of peri-implant radiolucency), together with the patient's opinion on the treatment, were used. At the Harvard Consensus Conference in 1978 (21), success/failure criteria for all types of oral implants were established. Subsequently, ALBREKTSSON *et al.* (16) proposed stricter criteria which have been generally accepted. At that time conventional periodontal indices were not included, because they were not considered to be correlated to implant success, and this opinion was supported by SMITH & ZARB (22) who did not consider it necessary to include any measure of mucosal health in their proposed criteria for implant success. In 1988, at the second Consensus Conference (23), it was concluded that different success criteria should be applied to different implant systems. Unfortunately, no agreement was reached, and the success criteria of the first conference in 1978 were maintained. Periodontal indices (lack of redness, bleeding on probing, probing from a fixed reference point) were again proposed as clinical criteria to be used in evaluating implant conditions (24). Further, specific criteria for different implant designs were suggested. Consensus was expressed on excluding probing depths, as that might be related to the

thickness of the mucoperiosteum and consequently might not be correlated to implant success or failure. Recently, at the 1st European Workshop on Periodontology (14), it was agreed that an absence of mobility, an average radiographic marginal bone loss of less than 1.5 mm during the first year of function and less than 0.2 mm annually thereafter, and absence of pain and/or paresthesia, were to be considered success criteria for osseointegrated implants. It was also suggested that probing depths related to a fixed reference point and bleeding on probing should be measured.

Periodontal parameters were introduced to better describe the state of peri-implant tissues in order to provide clinical tools to identify failing implants. Obviously, the discrimination between failing and failed implants can be relevant not only for scientific purposes (e.g., mechanisms of failure), but also because a cause-related therapy could thereby be attempted, if a failing implant and its causative etiology can be identified.

### **Parameters used for evaluating failed implants**

The evolution from anecdotal clinical observations to scientific evidence requires quantification based on the availability of parameters able to convert subjective impressions into objective data (17). The parameters, which have been employed clinically to evaluate implant conditions, will be discussed with the attempt to identify the most reliable ones. Ideally, it would be of great interest to differentiate between parameters indicating failed and failing implants, respectively. Some variations in the assessment methodology have to be considered, since implants are characterized by different designs and subjected to different surgical procedures and loading regimens.

The most common diagnostic criteria employed for the evaluation of established implant failures are the following:

#### **Clinical signs of early infection**

During the healing period (3 to 9 months), complications such as swelling, fistulas, suppuration, early/late mucosal dehiscences, and osteomyelitis, can occasionally be present and may indicate implant failure. The most rational and common explanation for this is infection. However, early wound dehiscences of submerged implants can also be seen in relation to poorly retained sutures, inadequate flap adaptation, or premature wearing of a denture (25). Early signs may represent a much more critical finding than if the same complication occurs later, because the healing process,

leading to the integration of the implant in the surrounding bone, can be disturbed. Late superficial postoperative infection of soft tissues can sometimes be attributed to a retained piece of suture material (25) or to insufficient tightening of the cover screw, and are generally uncomplicated, while late deep infections are more serious. Late exposures of the implants, occurring after some months of healing, are in most cases attributed to the pressure of dentures (25) in combination with a thin alveolar crest, superficially buccally or lingually installed fixtures, and a thin mucosa.

In a study monitoring 509 implants, a total of 36 soft tissue perforations over implants were recorded (26). 4 of these implants (11%) were not integrated at abutment connection. Other signs of infections were present around about 20 implants, of which 6 (30%) turned to be early failures. These percentages were much higher than those based on implants showing uneventful healing and calculated within the same study (2.8%).

Consequently, clinical signs of infections observed during the postoperative submerged period may lead to an increased risk for implant failure, which does not seem to be as high as could be feared. Therefore, signs of infection cannot be used alone to determine the fate of an implant, but should be evaluated in conjunction with other parameters such as radiolucency and mobility. In the absence of these signs of implant failure, clinical signs of infection represent a complication, which, if left untreated, might lead to an implant failure.

#### **Pain or sensitivity**

Pain or discomfort is often associated with mobility and could be one of the first signs which indicates an implant failure (27–29). Pain or sensitivity when chewing, tightening the abutment screw, or at percussion can be experienced, even under local anaesthesia. No studies have identified the structural correlation to pain around implants. Interestingly, failed implants can also be completely asymptomatic (30). Further, pain may reflect adverse tissue reactions not primarily related to implant mobility. For instance, in cases of implants placed over the mandibular canal, after nerve transposition or lateralization procedures, pain can reflect intraosseous oedema and pressure on the inferior alveolar nerve and may therefore be associated with perfectly stable implants. It has been suggested that persistent discomfort may be evident long before any radiographic changes (25). However, scientific evidence is still lacking.

#### **Clinically discernible mobility**

Once the clinician has distinguished between the mobility of a poorly connected abutment and the

mobility of the underlying implant, the implant must be suspected to be surrounded by a fibrous tissue capsule. Mobility is always a clear sign of failure.

Several different kinds of mobility have been recognized: (1) rotation mobility; (2) lateral or horizontal mobility; (3) axial or vertical mobility; as well as different degrees of mobility (31). Further, it has been suggested that slight changes of horizontal mobility can be better evaluated by means of electronic devices such as the Periotest (32–39). This procedure provides an objective method to measure the degree of mobility. It has been shown that implant and abutment length, the type of jaw treated, as well as the bone density have a major influence on Periotest values (40–42). However, despite some claims (35, 43), it remains to be demonstrated if changes in Periotest scores are an early sign of implant failure, preceding radiographic changes.

It has been suggested that implants should also be evaluated for possible rotational movements (44, 45). Recently, it was, e.g., proposed to apply a reverse-torque test, with forces not exceeding 10 Ncm, to every single implant at abutment connection to discover mobile implants (28). With this procedure, an incidence of 4.7% of early failures was reported. Interestingly, as will be described later, this figure slightly exceeded the average rate of 3.6% of early failures calculated with a meta-analytic approach (Table 8). Further, the incidence of failures diagnosed by the reverse torque test was 3 × higher than that observed by FRIBERG *et al.* (46). In the report by SULLIVAN (28), an increase of the reverse-torque test to 20 Ncm was shown to reduce the number of late failures. However, the figures for early failures were not reported. It is yet unclear if this method is advisable or not due to the risk of inducing an iatrogenic fracture at the bone-implant interface. In addition, clinical observations (26, 47) indicate that an increase in bone apposition may occur over time. Therefore, it is not recommendable to apply a 20 Ncm reverse torque to screen implants for rotational mobility.

Rotational mobility, in absence of vertical and horizontal mobility, may not necessarily be associated with the presence of a soft tissue capsule, but might reflect a weak or immature bone/implant interface, particularly for implants placed in sites with limited bone volume or bone of low mineral content. In contrast, horizontal and vertical mobility invariably reflects bone loss and the presence of a peri-implant soft capsule (30). Consequently, even though a simplified dichotomous (yes/no) scale for mobility assessment might be preferable because it eliminates subjective interpretations,

some weakly osseointegrated implants might yet be iatrogenically rotated and removed.

Occasionally, clinically discernible mobility can be present without distinct radiographic bone changes (29). Therefore, mobility is the cardinal sign of implant failure.

#### **Radiographic signs of failure**

In general, intraoral radiographs are taken after the abutment connection, in order to control that abutments are properly seated. On the basis of measurements on these radiographs, the baseline value for future marginal bone changes can be established. Standardized periapical radiographs should be taken at regular follow-up intervals to detect peri-implant radiolucency and/or progressive marginal bone loss or "saucerization" (48–52).

Although panoramic radiographs have been used (35, 53–56), this technique is of limited value in monitoring implant conditions due to inferior image resolution and the inability to modify the angulation of the X-ray beam (51). In fact, while panoramic films are able of resolving about 5 line pairs per mm, periapical films can clearly delineate at least 10 line pairs per mm (57).

There can be 2 well-distinct radiographic pictures: a thin peri-implant radiolucency surrounding the entire implant, suggesting the absence of a direct bone-implant contact and possibly a loss of stability (29, 47, 58, 59), and an increased marginal bone loss. In the first case, the implant is usually found mobile when tested, whereas in the latter, the fixture can be stable. It should be considered that an abnormal rate of marginal bone loss can also be a sign of a mechanical failure (fracture of the implant) (26, 47, 60). Since the distinction between these 2 radiographic pictures is not always clear, when a suspected peri-implant radiolucency or excessive marginal bone loss is observed, it is recommendable to remove the prosthetic construction and check the implants for stability. Clinically discernible mobility after bridge removal can confirm the presumptive radiographic diagnosis of implant failure.

The resolution of the radiographic technique together with the projection of anatomical structures could limit the detection of a thin soft tissue layer surrounding the fixture. This could explain why, occasionally, barely perceptible clinical mobility does not correspond to appreciable radiographic changes. Conversely, due to the Mach band effect (a visual phenomenon, where the borders of adjacent areas of different photographic densities appear to have larger density differences than really exist, i.e., the presence of soft tissue around the implant is simulated) (49, 52, 61), peri-implant

radiolucency can, occasionally, be noted even in cases of successful implants. In an evaluation of the accuracy and precision in the radiographic diagnosis of clinical mobility, it was concluded that despite the relatively good diagnostic accuracy, the probability of predicting fixture instability can be low in a population showing a low prevalence of mobile fixtures (62). However, in this latter study, implant losses were analyzed over a 5-year period, and the positive predictive values (17%) were calculated with a far too optimistic prevalence of early fixture loss of 1.5% according to FRIBERG *et al.* (46). If recalculating positive predictive values at a prevalence of implant failure of 10%, the predictability of standardized intraoral radiographs definitely improves (about 61%). The above mentioned conclusions were modified by recent findings from another radiographic investigation (29). In fact, the positive predictive value (i.e., the probability that an implant was clinically mobile, when the radiographic evaluation had demonstrated signs of loss of osseointegration) was found to be much higher (83%). From a selected population of 482 implants, 114 of the 138 implants which were radiographically reported as suspicious for loss of osseointegration, were found to be clinically mobile. Interestingly, the remaining 24 were regarded as stable. Unfortunately, as the authors pointed out, the study design did not permit evaluation of the true negative predictive value (the number of implants which were mobile despite the absence of radiographic signs of implant failure), because patients without radiographic signs of lack of osseointegration were not tested for implant stability.

Therefore, the radiographic examination remains one of the primary tools for detection of failed implants in clinical routine, even though it is not as accurate as the mobility test.

The most important factors for making a proper radiographic evaluation of the implant conditions are the quality of the radiographs (29, 62–65) together with the examiner's experience (29, 62).

#### **Dull sound at percussion**

It has been suggested (22, 44) that a subdued sound upon percussion is indicative of soft tissue encapsulation, whereas a clear crystalline sound indicates successful osseointegration. Once the clinician has verified that the abutment is properly attached to the implant, the test is conducted by hitting the abutment with a loosely held metallic instrument. Although it is a rather subjective test without a solid scientific background, it can provide a useful indication to the examiner. It has also been suggested that a dull tone on percussion might be present long before radiographic signs of implant

failure (25). Unfortunately, no scientific evidence has been presented yet to substantiate this hypothesis.

In conclusion, the cardinal sign of implant failure is mobility. However, the necessity of removing the fixed prosthesis, which can be a cumbersome and time-consuming procedure, has made intraoral conventional radiography a valuable aid in determining the success of oral implants in clinical routine.

### Parameters used for evaluating failing implants

The clinical signs previously discussed emerge only when the failure process reached an irreversible state. However, the ideal parameter for monitoring implant conditions should be sensitive enough to distinguish early signs of implant failure. The following parameters have thus been proposed.

#### Radiographic observed progressive marginal bone loss

There seems to be unanimous consensus that progressive marginal bone loss is a pathological sign which can lead to implant failure. However, to what extent the marginal bone resorption should progress in order to advocate treatment, and which is the most appropriate treatment procedure, remains to be decided.

One of the most commonly used success criteria for the evaluation of marginal bone loss was proposed by ALBREKTSSON *et al.* (16, 22). These authors suggested using less than 1.5 mm of marginal bone loss during the 1st year of loading and thereafter less than 0.2 mm yearly as success criteria. This concept was probably developed from the radiographic findings on the mean marginal bone loss around Brånemark implants (60). However, this parameter was meant to be employed to evaluate individual implants (13). Recently, this statement has been slightly modified (14), adding the word "average". It is interesting to observe that despite the majority of clinical follow-up studies referring to the ALBREKTSSON *et al.* success criteria, few authors actually apply the bone loss criterion to individual implants.

Some authors doubt that a firm limit for an acceptable annual bone loss can be established (26, 66). This doubt was based on reported higher amounts of bone loss, which stabilized after 2 or 3 years without leading to loss of the implant. It has therefore been proposed that an implant should be considered failed when the marginal bone loss has reached the apical 1/3 of the implant (27). Moreover, from a technical point of view, it is not

possible to verify an annual progression in the range of 0.1 mm on radiographs (50).

Some *in vitro* studies based on the Brånemark implant design provided information on the accuracy of periapical radiographs in the assessment of marginal bone levels around implants. It was found that stereoscopic radiographs were superior to single radiographs, which could not detect a gap of 0.2 mm between the bone and implant, and that the vertical angulation of the X-ray beam influenced, to a great extent, the accuracy of bone level assessments (63). In particular, the deviation from a perpendicular projection relative to the long axis of the implant should not exceed 9°. The importance of a strict parallelism was stressed in another investigation, and it was concluded that single radiographs were unreliable due to the great difficulties of obtaining serially identical radiographs in clinical cases (64). However, the threads of the Brånemark implants were found useful in controlling the identity of serial radiographs (67), but again it was concluded that the accuracy obtained from the ideal *in vitro* situation might be difficult to reproduce in the clinical situation. Another study confirmed that it is extremely difficult to achieve valid bone loss measurements below 0.2 mm (65).

Several techniques for taking optimal intraoral radiographs have been described (48, 49, 52, 68). However, the satisfaction of the requirements for identical exposure geometry are very difficult to meet in clinical practice, particularly when compared to the *in vitro* situation.

Another important limitation of the radiographic examination is that only the interproximal aspects of the implant can be evaluated.

Bone loss around the neck portion of Brånemark implants has been subjected to numerous radiographic investigations (26, 27, 60, 66, 69–87). Marginal bone loss has been observed around other implant systems as well (88–101), although with some differences. In fact, more marginal bone loss was described among some other implant systems, when compared to Brånemark implants (53–55, 92, 102).

It can be argued that marginal bone loss around the neck of osseointegrated implants is probably influenced by the implant design, both in the short- (96, 103) and long-term period (54, 55, 92). However, due to the complex interactions between surgically-induced trauma, stress distribution, microbiota and host response on the marginal bone loss, the exact rôle played by the various implant designs and surface characteristics remains to be understood. In this context, it should also be observed that even the differences in radiographic image characteristics of different implant designs

can affect the possibility to confirm osseointegration and to control image identity (104).

It has been suggested that digital subtraction radiography might be useful to detect more subtle changes in bone density adjacent to the implant, improving both accuracy and precision (49, 50, 105–107). However, few data to validate this technique have been presented (96). It should be also observed that intraoral periapical radiographs have at least  $2 \times$  the resolving power of digital intraoral imaging (49, 51).

Even though it is extremely difficult to obtain accurate and reproducible bone-level measurements over time using a radiographic technique, this method seems more reliable than probing, particularly in the presence of inflamed peri-implant tissues and bony defects, in monitoring implant conditions.

#### **Clinical signs of late infection**

A progressive marginal infection can lead to implant failure (108, 109). However, clinical signs of infection such as hyperplastic soft tissues, supuration (spontaneous, on probing, or under pressure), swelling, fistulation, color changes of the marginal peri-implant tissues, etc., are signs which call for intervention. In the absence of mobility and radiographic changes, these signs indicate more a complication (amenable to treatment) than a failure.

#### **Bleeding on probing (BOP)**

BOP (probing in the depth of the pocket until a slight resistance is met) is one of the periodontal parameters used to evaluate the presence of an inflammatory process at the base of a periodontal pocket. It should not be confused with the sulcus bleeding index, which is meant to evaluate the condition of the superficial periodontium.

BOP has been employed to measure peri-implant tissue conditions around Brånemark implants (110). No correlations were observed between bleeding and histological, microbiological or radiographic changes typical for gingivitis and/or periodontitis. It was hypothesized that bleeding could have been produced by undue force onto the periodontal explorer. These preliminary findings were recently confirmed in an animal study (111). Conversely, experimental findings around the ITI implants yielded completely different results (112). Healthy sites were characterized by complete absence of BOP (0%), whereas both peri-implant mucositis and peri-implantitis sites showed significant bleeding (67 and 91%, respectively). The reason for these differences might be attributed to the different probing forces used (0.5 N versus 0.2

N, respectively). However, recent findings (113) also suggested that BOP cannot be used as discriminator of a healthy or diseased peri-implant state. Therefore, the procedure of recording BOP around implants is not scientifically supported.

#### **Sulcus bleeding index (SBI)**

The SBI can be defined as the bleeding tendency of the alveolar mucosa surrounding the implant abutment observed by running a periodontal probe along the abutment circumference 1 mm into the mucosal pocket and parallel to the margin of the soft tissues (26). A 4-score scale modified SBI has been also proposed (109). Despite its own limitations (e.g., in smokers), SBI can be used instead of BOP for a more objective evaluation of the superficial peri-implant soft tissue conditions. While this parameter might distinguish between healthy and inflamed tissues, it is not able to identify failing implants.

#### **Pocket probing depth (PPD)**

PPD is defined as the linear distance from the free mucosal margin to the base of the pocket (68, 114). The “base” of the pocket is generally defined as the apical termination of the junctional epithelium. However, when referring to the implant situations, some authors (55) consider the level of the marginal bone to be the base of the pocket. This has generated additional confusion on the rôle of probing at implants. The significance of probing around teeth has been extensively evaluated (115). However, before applying a well-characterized periodontal parameter to the implant situation, 2 basic questions should be answered: what is the goal of probing and what does it really measure?

It has been demonstrated, under experimental conditions, that at Brånemark implants, the probe tip penetrated apically of a laterally displaced junctional epithelium ending close to the alveolar crest (111). Conversely, around ITI implants, probing was able to identify the apical termination of the junctional epithelium with a mean error of 0.05 mm in healthy sites and 0.02 mm in inflamed sites, respectively (112). The reason for these differences might again be attributed to the different probing forces employed (0.5 N versus 0.2 N, respectively). However, in a recent review (116), the latter findings were presented in a different way. In fact, it was stated that, “In inflamed tissues around one-stage non-submerged implants, periodontal probes penetrated close to the bone level, whereas the probe tips tended to stop at the histological level of connective tissue adhesion if healthy tissues were present”.

Probing cannot easily be performed around all implant or abutment designs. For example, probing a cylinder is practical when the neck is continuous with the cylinder circumference, but can be difficult around screws, particularly when bone resorption has reached the level of the threads (117, 118). It has therefore been suggested that the sensitivity of PPD might be low at implants compared to natural dentition, due to the possible underestimation of the true PPD values (117).

The superstructure, in most of the cases, has to be removed to permit probing as well. It has also been suggested (17, 116) that implants should be designed in a way to permit probing.

Probing penetrates the peri-implant tissues with resultant damage (110, 111). The magnitude and the clinical significance of such damage has yet to be determined. Another direction pursued has been the application of standardized forces. However, preliminary studies have indicated that the reproducibility was not necessarily increased (119).

It can be concluded that increased pocket depth can be correlated with a higher degree of inflammation of the peri-implant mucosa (110, 113, 119, 120), but not necessarily to bone loss (55). However, absolute PPD alone cannot be used as an indicator of a pathological condition, since additional factors, such as tissue thickness and different abutment lengths (68, 117, 121), may influence the PPD assessments around implants when compared with teeth. Progressive PPD over time might therefore be a better indicator for failing implants than absolute probe measurements. Conversely, other authors consider a deep pocket a protected habitat for putative pathogens, which is a clear indication of peri-implant pathology (15, 116).

It remains also to be established if probing can distinguish failed implants, characterized radiographically by a thin peri-fixtural radiolucency.

#### **Mucosal recession (REC)**

REC can be defined as the linear distance between the location of the free mucosal margin and a fixed landmark (68, 122). When threads or a rough implant surface become exposed, it might be difficult to maintain the area clean from plaque and the prognosis of the implant may become questionable. Otherwise, recession is mainly an aesthetical problem and not an indication of failing implants.

#### **Probing "attachment" levels (PAL)**

PAL are probing depths related to a fixed reference point on the implant in order to monitor "attachment" loss over time (17, 114, 119). In other words,

PAL is the sum of PPD and REC. It has been suggested that increased measures of 2 mm or more should be interpreted as resorption of alveolar bone (68, 114). The same practical limitations related to probe penetration and implant shape, as discussed above, are evident. Even though this is a more accurate method to monitor implant health (123), it still provides less precise information than radiographs, particularly when tissues are inflamed or in the presence of infra-bony craters (119). However, additional information on the tissue conditions at buccal and lingual surfaces can be gained when comparing PAL with radiographs.

#### **Crevicular fluid analysis**

The analysis of crevicular fluids (composition and flow rate), though able to distinguish between healthy and inflamed sites, has not been demonstrated as being capable of differentiation between destructive and non-destructive inflammation. Therefore, this technique cannot yet be used to identify failing implants (113, 124–136). However, recent findings from a preliminary report seem to indicate that increased levels of interleukin-1 $\beta$  might be associated with failing implants (134). These findings were not confirmed by another controlled study (113).

#### **Microbiological sampling**

There is not yet a complete agreement on periodontal pathogens, even though some species (for example *Porphyromonas gingivalis* and *Actinobacillus actinomycetemcomitans*) are heavily associated with periodontal breakdown (137–143). A similar flora as that present in gingival pockets of teeth in health and disease has been observed around implants, though with some differences in partially- and fully-edentulous patients (68, 69, 109, 110, 113, 124, 125, 144–159). However, there is not any convincing evidence yet that laboratory tests or commercially-available kits for identification of suspected periodontal pathogens are of any use in the implant situation. In fact, it is yet unknown whether these suspected pathogens induced bone loss, or whether they simply found an ideal habitat in infrabony pockets which may have been initiated by overloading.

One exception might be the rare condition of osteomyelitis around implants, where the causative bacterial species must be identified, and an effective antibiotic treatment administered. The use of microbiological sampling is, otherwise, yet confined to research situations.

### Absence of keratinized mucosa

A relationship in correlation between implant failure and absence of an adequate band of keratinized mucosa surrounding the abutment has been suggested (160). In particular, authors have directly attributed the reason for some late losses to the lack of keratinized mucosa (35, 161–164). One hypothesis behind this idea is that the keratinized tissue is more resistant to inflammatory destructive processes induced by the oral microbiota. However, there is no scientific evidence supporting this hypothesis (35, 60, 69, 70, 97, 110, 122, 124, 125, 165–167). In conclusion, keratinized mucosa does not appear to be related to implant failure. However, the presence of keratinized tissue might facilitate the patient's hygienic procedures.

In conclusion, while it is possible to clearly differentiate between a successful and a failed implant, it still remains difficult to identify failing implants. In this context, the distinction between a failed implant, characterized by a thin peri-implant radiolucency and mobility, and a failing implant, characterized by progressive marginal bone loss, clinical signs of peri-implant infection and absence of discernible mobility, might disclose a different etiology (overload and peri-implantitis). This distinction seems justified in light of experimental (168, 169) and clinical (30, 170) findings. Obviously, both etiological factors can interact with each other, resulting in a variety of intermediate situations.

So far, there is substantial agreement and scientific evidence that bone level assessments are more reliable with a well-performed intraoral radiographic examination than with all the conventional periodontal indices (22, 31, 55, 111, 117, 121, 171). However, radiographic findings should always be carefully evaluated in conjunction with other clinical parameters.

### Frequencies of early and late failures

In order to provide data on the prevalence and incidence of biological failures in different clinical situations, a large number of clinical follow-up studies were analyzed. In this section, only investigations dealing with the Brånemark system were evaluated. This does not mean that other implants cannot achieve similar or even better results; however, since the Brånemark system can be considered the reference implant for osseointegrated oral implants, more scientific reports are available on this specific system (3, 4). Moreover, it was judged that it was easier to draw conclusions if only one well-characterized implant system was analyzed, thus avoiding confounding factors derived from

different implant characteristics (material, coating, roughness, shape, surgical and prosthetic procedures, etc.). On the other hand, with this approach, the generalization to other implant systems is limited. For a comparative analysis of failure patterns among different osseointegrated oral implant systems, please refer to ESPOSITO *et al.* (4).

Studies which did not include complete data on implant losses were excluded or only partially employed. From 159 follow-up investigations published through February 1997 on the Brånemark system, 73 articles were selected. The excluded articles did not provide detailed information on how many implants were placed in each jaw, did not differ between early and late implant losses, did not specify the follow-up periods properly, or presented the same patient materials already available in other publications. Failure data regarding the “development group” (58, 60, 172) or the “casuistry group” (173) were also excluded, since these investigations were based on prototypes of titanium implants having different designs compared to the Brånemark implants used today. However, failure rates of these studies, with one exception (173), were much higher than those hereby presented. The outcome of implants placed in irradiated patients will be discussed separately in the 2nd part of the review.

One rationale for analyzing failures instead of success is that smaller differences are more promptly recognized. For example, if a 96% success rate of a certain technique is compared with a 98% success rate of another technique, almost no difference may be noticed. Conversely, using a failure rate approach, it is obvious that the former modality fails twice as often than the second.

Fixtures removed for mucosal disorders (suspected peri-implantitis cases), but still clinically stable, were recorded as failures in our analysis. Mechanical and iatrogenic failures, i.e., implants which had fractured, or were not used as abutments (“sleeping implants”), were not counted as biological failures. It should be emphasized that the topic of this analysis is the fate of the individual implant rather than the outcome of prosthetic reconstructions. Therefore, in terms of functioning bridges, failure rates are lower.

In Table 4, a summary of follow-up studies, providing data on implant losses in fully edentulous patients, is shown. Data are subdivided in 2 groups: patients wearing fixed bridges and overdentures, respectively. The higher failure rates in the overdenture group, both for early and total number of losses (5.9 and 12.8%, respectively), particularly in Swedish studies (75, 80, 174–176), may be explained by the fact that this treatment alternative was mainly used in critical situations. Such

Table 4

Studies reporting on failure rates for Brånemark implants in totally edentulous patients wearing fixed prostheses and overdentures

Authors (ref. no.)	Implants inserted/failed*		Implant location maxilla/mandible	Failures early/late**	Loading time (range)
<b>Fixed prostheses</b>					
ADELL <i>et al.</i> , 1981 (60)	230/37	(9.1)	100/130	<b>21</b> (13;8)/ <b>16</b> (9;7)	1–9 yr
LINDQUIST <i>et al.</i> , 1987 (198)	42/2	(4.8)	maxilla	<b>2/0</b>	3 yr
AHLQVIST <i>et al.</i> , 1990 (199)	269/9	(1.9)	82/187	<b>5</b> (5;0)/ <b>4</b> (4;0)	2 yr
JOHANSSON & PALMQVIST, 1990 (200)	286/25	(5.2)	112/174	<b>15</b> (11;4)/ <b>10</b> (8;2)	3–9 yr
ADELL <i>et al.</i> , 1990 (201)	869/18	(1)	mandible	<b>9/9</b>	1–10 yr
TRIPLETT <i>et al.</i> , 1991 (202)	45/2	(0)	atrophic mand.	<b>0/2</b>	15–72 months
JEMT, 1991 (203)	125/4	(3.2)	maxilla	<b>4/0</b>	3 yr
KRUMP & BARNETT, 1991 (204)	154/3	(2)	mandible	<b>3/0</b>	16–71 months
QUIRYNEN <i>et al.</i> , 1991 (66)	589/30	(2.5)	269/320	<b>15</b> (11;4)/ <b>15</b> (8;7)	5–83 months
TOLMAN & LANEY, 1992 (205)	1230/64	(2.2)	292/938	<b>27</b> (16;11)/ <b>37</b> (29;8)	1–75 months
ERICSSON <i>et al.</i> , 1994 (78)	63/2	(3.2)	mandible	<b>2**/0</b>	18 months
JEMT, 1994 (77)	449/31	(3.3)	maxilla	<b>15/16</b>	5 yr
OLSSON <i>et al.</i> , 1995 (206)	563/27	(0.5)	200/363	<b>3</b> (3;0)/ <b>24</b> (23;1)	3 yr
JEMT & LEKHOLM, 1995 (80)	655/64	(3.5)	maxilla	<b>23/41</b>	5 yr
HENRY <i>et al.</i> , (207)	837/0	(0)	mandible	<b>0/0</b>	10 yr
LINDQUIST <i>et al.</i> , 1996 (87)	272/3	(0.7)	mandible	<b>2/1</b>	12–15 yr
ZARB & SCHMITT, 1996 (208)	259/32	(8.1)	26/233	<b>21</b> (0;21)/ <b>11</b> (0;11)	11–15 yr
BALSHI <i>et al.</i> , 1997 (209)****	425/31	(0.9)	370/55	<b>4</b> (4;0)/ <b>27</b> (25;2)	3 yr
Subtotal fixed prostheses (%)	6609/384	(2.6)	2722/3887	<b>171</b> (107;64)/ <b>213</b> (163;50)	1 month–15 yr
	100/5.8		41/59	<b>45</b> (63;37)/ <b>55</b> (77;23)	
<b>Overdentures</b>					
ENGQUIST <i>et al.</i> , 1988 (174)	339/67	(13.6)	191/148	<b>46</b> (38;8)/ <b>21</b> (20;1)	3 months–4 yr
TRIPLETT <i>et al.</i> , 1991 (202)	85/6	(1.2)	atrophic mand.	<b>1/5</b>	15–72 months
SMEDBERG <i>et al.</i> , 1993 (75)	86/12	(8.1)	maxilla	<b>7/5</b>	18–32 months
JEMT, 1993 (175)	211/70	(7.1)	maxilla	<b>15/55</b>	3 yr
HEMMINGS <i>et al.</i> , 1994 (210)	68/6	(5.9)	mandible	<b>4/2</b>	3–9 yr
PALMQVIST <i>et al.</i> , 1994 (176)	89/27	(21.3)	maxilla	<b>19/8</b>	1–5 yr
JEMT & LEKHOLM, 1995 (80)	127/36	(9.4)	maxilla	<b>12/24</b>	5 yr
WRIGHT <i>et al.</i> , 1995 (211)	98□/3	(2)	mandible	<b>2/1</b>	3 yr
JEMT <i>et al.</i> , 1996 (81)	510□□/44	(3.1)	117/393	<b>16</b> (9;7)/ <b>28</b> (21;7)	5 yr
ZARB & SCHMITT, 1996 (212)	132/5	(2.3)	17/115	<b>3</b> (?)/ <b>2</b> (?)	3–13 yr
HOOGHE <i>et al.</i> , 1997 (213)	561□□□/13	(2)	40/521	<b>11</b> (3;8)/ <b>8</b> (5;3)	3–108 months
Subtotal overdentures (%)	2306/295	(5.9)	878/1428	<b>136</b> (103;30)/ <b>159</b> (138;19)	3 months–13 yr
	100/12.8		38/62	<b>46</b> (76;22)/ <b>54</b> (87;12)	
<b>Total full edentulism (%)</b>	<b>8915/679</b>	<b>(3.4)</b>	<b>3600/5313</b>	<b>307</b> (210;94)/ <b>372</b> (301;69)	
	<b>100/7.6</b>		<b>40/60</b>	<b>45</b> (68;31)/ <b>55</b> (81;19)	

\* In parenthesis the percentage of early failures.

\*\* The first figure in parenthesis refers to maxillary and the second to mandibular losses.

\*\*\* Both failed fixtures were in the 1-step procedure group.

\*\*\*\* The majority of the implants were inserted in fully edentulous patients.

□46, □□177 and □□□22 fixtures were originally kept sleeping at initial overdenture placement. Failures occurring in these groups were calculated.

Table 5

Clinical studies reporting on failure rates for Brånemark implants in partially edentulous patients wearing partial fixed prostheses and single crowns

Authors (ref. no.)	Implants inserted/failed*		Implant location maxilla/mandible	Failures early/late**	Loading time (range)
<b>Partial bridges</b>					
ERICSSON <i>et al.</i> , 1986 (214)	41/0	(0)	29/12	<b>0/0</b>	6–30 months
JEMT <i>et al.</i> , 1989 (173)	448/13	(0.9)	238/210	<b>4(4;0)/9(8;1)</b>	1–5 yr
QUIRYNEN <i>et al.</i> , 1991 (66)	509/29	(3.3)	304/205	<b>17(11;6)/12(6;6)</b>	2–77 months
TOLMAN & LANEY, 1992 (205)	302/4	(0)	106/196	<b>0/4(2;2)</b>	1–75 months
JEMT & LEKHOLM, 1993 (76)	259/7	(0.5)	101/158 (p)***	<b>4(3;1)/3(1;2)</b>	5 yr
NEVINS & LANGER, 1993 (215)	1203/56	(2.6)	652/551 (p)	<b>31(21;10)/25(10;15)</b>	2–9 yr
ZARB & SCHMITT, 1993 (216)	94/7	(4.2)	50/44	<b>4(0;4)/3(3;0)</b>	2–8 yr
ZARB & SCHMITT, 1993 (217)	105/2	(1.9)	41/64 (p)	<b>2(0;2)/0(0;0)</b>	32–89 months
LEKHOLM <i>et al.</i> , 1994 (27)	558/36	(3.6)	220/338	<b>20(9;11)/16(8;8)</b>	5 yr
OLSSON <i>et al.</i> , 1995 (79)	69/8	(1.4)	mandible	<b>1/7</b>	5 yr
NEVINS & LANGER, 1995 (218)****	309/7	(2.3)	177/132	<b>7(3;4)/0</b>	1–8 yr
BALSHI <i>et al.</i> , 1996 (180)	50/0	(0)	4/46 (p)	<b>0/0</b>	3 yr
BAHAT & HANDELSMAN, 1996 (181)	446/8	(0.7)	68/378 (p)	<b>3(0;3)/5(0;5)</b>	2–78 months
Subtotal partial bridges (%)	4393/177 100/4.0	(2.1)	1990/2403 45/55	<b>93(51;42)/84(38;46) 53(55;45)/47(45;55)</b>	1 month–9 yr
<b>Single implants</b>					
JEMT <i>et al.</i> , 1990 (145)	23/1	(0)	21/2	<b>0/1(1;0)</b>	3 yr
JEMT & PETERSSON, 1993 (219)	70/1	(0)	59/11	<b>0/1(1;0)</b>	3 yr
EKFELDT <i>et al.</i> , 1994 (220)	93/2	(1.1)	84/9	<b>1(0;1)/1(1;0)</b>	14–55 months
THILANDER <i>et al.</i> , 1994 (221)	27/0	(0)	19/8	<b>0/0</b>	3 yr
ANDERSSON <i>et al.</i> , 1995 (222)	65/1	(0)	62/3	<b>0/1(1;0)</b>	2–3 yr
BECKER & BECKER, 1995 (179)	24/1	(0)	7/17	<b>0/1(1;0)</b>	1–2 yr
ENGQUIST <i>et al.</i> , 1995 (177)	82/2	(2.4)	74/8	<b>2(1;1)/0</b>	1–5 yr
HAAS <i>et al.</i> , 1995 (182)*****	76/2	(1.3)	59/17	<b>1(1;0)/1(0;1)</b>	6 months–5 yr
MALEVEZ <i>et al.</i> , 1996 (83)	84/2	(1.2)	75/9	<b>1(0;1)/1(1;0)</b>	3–62 months
AVIVI-ARBER & ZARB, 1996 (223)	49/1	(2)	35/14	<b>1(1;0)/0</b>	1–8 yr
BALSHI <i>et al.</i> , 1996 (180)	22/1	(4.5)	4/18	<b>1(?)0</b>	3 yr
BAHAT & HANDELSMAN, 1996 (181)	59/2	(0)	21/38	<b>0/2(0;2)</b>	3–26 months
HENRY <i>et al.</i> , 1996 (84)	107/3	(0.9)	88/19	<b>1(1;0)/2(2;0)</b>	5 yr
Subtotal single implants (%)	781/19 100/2.4	(1)	608/173 78/22	<b>8(4;3)/11(8;3) 42(50;38)/58(73;27)</b>	3 months–8 yr
<b>Total partial edentulism (%)</b>	<b>5174/196 100/3.8</b>	<b>(2)</b>	<b>2598/2576 50/50</b>	<b>101(55;45)/95(46;49) 52(54;45)/48(48;52)</b>	

\* In parenthesis the percentage of early failures

\*\* The first figure in parenthesis refers to maxillary and the second to mandibular losses.

\*\*\* (p)=posterior area.

\*\*\*\* Both total and partial edentulous “recalcitrant” periodontal patients are included.

\*\*\*\*\* Immediate implants and guided bone regeneration techniques were also included.

All late failures in single implant investigations, but one (145), occurred during the first year of loading.

In studies (179–182) single implants replaced molars.

Table 6

Studies reporting on failure rates for Brånemark implants used in complicated situations (GBR procedures, nerve transposition, immediate placement in extraction sockets, maxillary tuberosity implants) and bone grafts

Authors (ref. no.)	Implants inserted/failed	Implant location maxilla/mandible	Failures early/late***	Loading time (range)
<b>Complicated situations</b>				
PAREL & TRIPLETT, 1990 (224)	63/1 (i+a)* (1.6)**	mandible	1/0	1–41 months
TOLMAN & KELLER, 1991 (225)	303/2 (i) (0.7)	44/259	2(0;2)/0	1–6 yr
KRUMP & BARNETT, 1991 (204)	41/3 (i+a) (7.3)	mandible	3/0	19–48 months
FRIBERG <i>et al.</i> , 1992 (183)	23/3 (n) (13)	mandible	3/0	1–12 months
BAHAT, 1992 (226)	72/5 (0)	maxillary tuber	0/5	6–37 months
TULASNE, 1992 (187)	43/3 (2.3)	maxillary tuber	1/2	0–4 yr
BECKER <i>et al.</i> , 1994 (227)	49/3 (i+GBR) (6.1)	33/16	3(?) / 0	1 yr
ROSENQUIST, 1994 (184)	250/36 (n) (4.8)	mandible	12/24	0–1 yr
DAHLIN <i>et al.</i> , 1995 (228)	55/6 (GBR) (7.3)	35/20	4(3;1)/2(2;0)	2 yr
BALSHI <i>et al.</i> , 1995 (229)	51/7 (11.8)	maxillary tuber	6/1	1–63 months
HIRSCH & BRÅNEMARK, 1995 (185)	63/5 (n) (7.9)	mandible	5/0	15–63m
Subtotal complicated situations (%)	1013/74 (100/7.3)	278/735 (27/73)	40(10;27)/34(10;24) 54(25;67)/46(29;71)	0–6 yr
<b>Bone grafts</b>				
ADELL <i>et al.</i> , 1990 (188)	140/39 (o) (7.7)	maxilla	11/28	1–10 yr
ISAKSSON & ALBERIUS, 1992 (230)	46/8 (o) (13.4)	maxilla	6/2	2 yr–55 months
TOLMAN & LANEY, 1992 (205)	109/9 (o; s; ng) (8.3)	92/17	9(7;2)/0	1–75 months
NYSTRÖM <i>et al.</i> , 1993 (231)	177/40 (o) (9)	maxilla	16/24	17 months
ISAKSSON <i>et al.</i> , 1993 (232)	59/14 (ig) (24)	maxilla	14/0	0–1 yr
RAGHOEBAR <i>et al.</i> , 1993 (233)	95/5 (s) (5.3)	maxilla	5/0	0–30 months
KELLER <i>et al.</i> , 1994 (234)	83/6 (s; ng) (1.2)	maxilla	1/5	1–6 yr
JENSEN <i>et al.</i> , 1994 (235)	291/19 (s; ng; o) (4.1)	maxilla	12/7	6–52 months
SCHLIEPHAKE <i>et al.</i> , 1994 (236)	55/2 (o) (3.6)	42/13	2(2;0)/0	2–80 months
JEMT & LEKHOLM, 1995 (80)	83/12 (o) (3.6)	maxilla	3/9	5 yr
KREKMANOV, 1995 (237)	187/29 (ig) (15.5)	maxilla	29/0	1–4 yr
BLOMQVIST <i>et al.</i> , 1996 (238)	182/37 (s) (16.5)	maxilla	30/7	2 months–4 yr
KÖNDELL <i>et al.</i> , 1996 (239)	85/24 (o) (22.3)	maxilla	19/5	4–6 yr
LUNDGREN <i>et al.</i> , 1996 (240)	30/0 (s) (0)	maxilla	0/0	1–3 yr
ÅSTRAND <i>et al.</i> , 1996 (241)	92/22 (o) (7.6)	maxilla	7/15	3 yr
DALELEMANS <i>et al.</i> , 1997 (242)	121/8 (s) (4.1)	maxilla	5/3	3–80 months
Subtotal bone grafts (%)	1833/274 (100/14.9)	1803/30 (98/2)	169(167;2)/105(105;0) 62(99;1)/38(100;0)	0–10 yr

\* (i)=immediate placement; (GBR)=guided bone regeneration; (n)=nerve transposition; (a)=alveolectomy; (o)=onlay bone grafts; (s)=sinus grafts; (ng)=nasal grafts; (ig)=inlay grafts after Le Fort I osteotomy.

\*\* In parenthesis the percentage of early failures.

\*\*\* The first figure in parenthesis refers to maxillary and the second to mandibular losses.

Table 7

Detailed analysis of failure rates for Brånemark implants inserted in the maxillary tuberosity and in bone grafts placed in the atrophic maxilla; data from Table 6

Surgical procedures	Implants inserted/failed*	Failures early/late**	Loading time (range)
maxillary tuber sinus and nasal lifts	166/15 (4.2)	7/8 (9)	0–63 months
onlay grafts	583/53 (7.4)	43/10 (9.1)	0–80 months
inlay grafts (Le Fort I)	741/153 (9.4)	70/83 (20.6)	1 month–10 yr
	246/43 (17.5)	43/0 (17.5)	1 month–4 yr

\* In parenthesis the percentage of early failures.

\*\* In parenthesis the total percentage of implant losses (early + late failures).

situations included the following cases: insufficient bone volume present for implant placement in number and positions necessary for a fixed restoration; the prognosis for a fixed implant-supported bridge was questionable; and when the treatment was an emergency solution for patients planned to receive a fixed restoration, but who had lost one or more implants. If the small group of patients, where a cluster of failures occurred, was not considered, according to QUIRYNEN *et al.* (171), implants used for supporting overdentures did not seem to fail more frequently than those supporting full fixed bridges.

Table 5 presents the outcome in partially edentulous patients. Data are again divided into 2 categories: subjects wearing a partial bridge and single crowns, respectively. It is evident that implants placed in partially dentate patients perform somewhat better than those placed in totally edentulous patients (2% of early and 3.8% of total losses versus 3.4% and 7.6%, respectively). In particular, both the early and total failure rates for single implants were very low (1 and 2.4%, respectively), even in groups representing the development period (145, 177); in clinical centers with little experience (178); or when a single implant was used to replace a molar (179–182). The high success rate might be partly explained by a more accurate patient selection in terms of age, health and anatomical conditions, together with a more favourable load distribution (molar areas excluded). Even though the observation period was still too short to draw final conclusions, this treatment modality seemed to be highly predictable in terms of biological success outcome.

Table 6 presents a rather heterogeneous group of reports on failures of implants used in complicated situations, including bone grafts. The 1st part of the table includes studies dealing with barrier membranes to enhance bone regeneration around implants in the presence of wide bony defects or fenestrations, immediate placement of implants in extraction sockets, technically demanding nerve

transposition, and installation of fixtures in the maxillary tuberosity. Apart from the occurrence of transient or permanent inferior alveolar nerve paresthesia/anesthesia (183–185), the average failure rate of 7.3% can be regarded as an acceptable outcome in relation to the complexity and the risks of these procedures.

From the 2nd part of Table 6, it is evident that the prevalence of failed implants in bone grafts is higher (14.9%) than in the other situations. A comprehensive review on this subject has recently been presented by TOLMAN (186). However, in order to analyze the influence of different surgical procedures on failure rates, Table 7 was created using data already presented in Table 6. It can be observed that implants placed in the maxillary tuberosity seem to behave equally well as those placed in a grafted sinus. Therefore, maxillary tuberosity implants might be a valid alternative to sinus lift procedures (187), local anatomical conditions permitting. Both onlay and inlay grafting procedures resulted in the highest failure rates (20.6 and 17.5%, respectively).

It is generally agreed that implants in maxillas do not perform as well as in the mandibles, and this trend can be noticed in all clinical situations (Tables 4–7) with one exception: the partially edentulous situation (Table 5), where similar failure rates can be noticed for the mandible and the maxilla. Fully edentulous mandibles (Table 4) are slightly over-represented and, obviously, this has influenced the final result, lowering the total failure rate.

Table 8 summarizes the data obtained by adding the total failure rates of the different clinical situations described in Tables 4–6. The average incidence of early failures is approximately 3.6%. The failure rate for late losses (3.6%, Table 8) might actually be an underestimation, as the prosthetic reconstructions were not removed to test implant mobility in all the studies presented. In addition, only a minority of the inserted fixtures were followed up to 5 years or longer. In this respect,

Table 8  
Summary of failure rates for Brånemark implants used in various clinical situations

Clinical situations	Implants inserted/failed*	Implant location maxilla/mandible	Failures early/late**	Loading time (range)
total edentulism (Table 4)	6609/384 (2.6)	2722/3887	171(107;64)/213(163;50)	1 month-15 yr
overdentures (Table 4)	2306/295 (5.9)	878/1438	136(103;30)/159(138;19)	3 months-13 yr
partial edentulism (Table 5)	393/177 (2.1)	1990/2403	93(51;42)/84(38;46)	1 month-9 yr
single implants (Table 5)	781/19 (1.0)	608/173	8(4;3)/11(8;3)	3 months-8 yr
complicated situations (Table 6)	1013/74 (3.9)	278/735	40(10;27)/34(10;24)	0-6 yr
bone grafts (Table 6)	1833/274 (9.2)	1803/30	169(167;2)/105(105;0)	0-10 yr
total	16935/1223 (3.6) 100/7.2	8279/8656 49/51	617(442;168)/606(462;142) 50(72;27)/50(76;23)	

\* In parenthesis the percentage of early failures.

\*\* The first figure refers to maxillary and the second to mandibular losses.

clinical studies with prolonged follow-up periods can provide more precise information.

Table 9 summarizes data on 3013 implants followed for up to 5 years, with adjustments for drop-outs when possible, but without distinction for the different clinical situations. The results show a average prevalence of implant losses over a 5-year period of 8.6% and, when bone grafts are excluded (201 implants), of 7.7%. In this sample, maxillas are slightly over-represented; therefore, this can be considered the worst scenario.

Unfortunately, these data do not provide information on failing implants. In fact, at least from a theoretical point of view, a few additional loaded fixtures, not yet failed, might be failing due to progressive bone loss and mucosal tissue irritation (peri-implantitis group). It is very difficult, at the present time, to provide exact figures for implant losses due to peri-implantitis. However, the total number of implants failed for peri-implantitis seems to be extremely low in the few existing long-term studies providing this information (76, 79, 84, 165, 173, 188, 189). An attempt to calculate the prevalence of implant losses due to peri-implantitis can be found in Table 10. The average prevalence of implant losses attributable to peri-implantitis was 2.8% in relation to the total number of biological failures, and 10.3% in relation to implant losses occurring after the 1st year of loading. Therefore, after approximately 5 years of function, 1 implant out of every 533 inserted seemed to be lost due to recurrent infection (peri-implantitis). However, the data presented must be interpreted with caution, because the distinction between different causes of implant losses could only be found in a minority of the studies analyzed.

Although comparisons with other oral implant systems are beyond the scope of this review, other systems such as the ITI (Table 10) seem to be more often affected by soft-tissue problems and progressive marginal bone loss (peri-implantitis) leading to implant failure (32, 35, 54, 56, 88, 99, 165, 190). With the ITI system, this type of peri-implant infection seemed to be a site-specific infection resembling chronic adult periodontitis (109), which responded favourably to antimicrobial treatment (191, 192). Reports regarding the IMZ system (161, 193) also suggested a higher incidence of implant failure due to peri-implantitis. Unfortunately, these data were not presented in detail to permit a proper evaluation. Different implant designs and surface characteristics, favouring plaque retention (22, 194), might, at least partly, explain these differences. Longer follow-up periods in conjunction with controlled, accurate radiographic studies and monitoring of the soft tissue conditions are needed to confirm this hypothesis (4).

Table 9

Summary of studies reporting on failure incidence for Brånemark implants used in various clinical situations followed up to 5 years after loading

Authors (ref. no.)	Implants inserted/failed	Detection time of failure					
		before loading	1 yr	2 yr	3 yr	4 yr	5 yr
ADELL <i>et al.</i> , 1990 (188)	45/12 (BG)*	4	4	2	0	1	1
ZARB & SCHMITT, 1990 (243)	262/29 (TE)	21	1	5	1	1	0
TOLMAN & KELLER, 1991 (225)	24/0 (II)	0	0	0	0	0	0
JEMT & LEKHOLM, 1993 (76)	259/7 (PE)	4	1	0	0	0	2
KELLER <i>et al.</i> , 1994 (234)	20/2 (BG)	0	0	0	2	0	0
LEKHOLM <i>et al.</i> , 1994 (27)	558/36 (PE)	20	3	8	2	0	3
PALMQVIST <i>et al.</i> , 1994 (176)	8/0 (OV)	0	0	0	0	0	0
JEMT, 1994 (77)	449/31 (TE)	15	7	6	2	0	1
JEMT & LEKHOLM, 1995 (80)	801/111 (TE; BG; OV)	38	40	19	9	3	2
OLSSON <i>et al.</i> , 1995 (79)	69/8 (PE)	1	4	3	0	0	0
HENRY <i>et al.</i> , (207)	83/0 (TE)	0	0	0	0	0	0
HENRY <i>et al.</i> , 1996 (84)	107/3 (SI)	1	2	0	0	0	0
KÖNDELL <i>et al.</i> , 1996 (239)	52/18 (BG)	15	0	2	0	0	1
LINDQUIST <i>et al.</i> , 1996 (87)	272/2 (TE)	2	0	0	0	0	0
total (%)	3013/259 100/8.6	121 47 (early losses)	62	45	16 53 (late losses)	5	10
Subtotal (bone grafts excluded) (%)	2812/216 100/7.7	99 46 (early losses)	52	41	13 54 (late losses)	3	8
Data for maxillas only (bone grafts excluded) (%)	1528/155 100/10.1	63 40 (early losses)	46	29	11 60 (late losses)	2	4

\* PE = partial edentulism; BG = bone grafts; TE = total edentulism; II = immediate implants; OV = overdentures; SI = single implants.

An analysis of the possible causes and their relative importance for implant failure is desirable for several reasons. Such information will draw attention to the areas where diagnostic, preventive and therapeutic tools are needed. Unfortunately, little information is found in the literature on possible mechanisms for failures. The fact that there is not yet agreement on the etiologic rôle of overload and bacterial plaque (peri-implantitis), particularly for late failures, may be one reason.

In Table 11, we attempted to divide failures of Brånemark implants according to chronological distribution and their possible etiologic factors. Out of the total number of failures, early fixture losses accounted for 47% (data from Table 9) which might have been caused by iatrogenic (e.g., infections or bone overheating) and host-related factors, including anatomical factors (e.g., poor bone quality or extremely thin bucco-lingual bone crests). Accordingly, late failures amounted to 53%, of which about 1/2 were detected during the 1st year of loading. It seems reasonable to attribute most of these late failures to overload in relation to poor bone quality and insufficient bone volume. The remaining late failures (after the 1st year of loading) might be attributed to progressive or "dramatic" changes of the loading conditions in relation to

bone quality and volume (90%) and to chronic marginal infections due to bacterial plaque (10%; data from Table 10). Clearly, both these etiologic factors may coexist in the same case.

Another factor which might have influenced the absolute number of late failures is the problem of drop-outs, which is unavoidable in long-term follow-up studies. However, complications in the low compliance group (once excluded, deceased and change of address) seem not likely to have jeopardized bridge function to a level requiring dentist intervention. Considering that implant treatment has mainly been provided by specialists in most of the investigations, one may assume that the patient would return to the center responsible for the treatment in case of subjective problems.

Finally, the reader should be aware that the positive results listed in this section are valid only for experienced specialists and may be influenced by the strict protocol of clinical investigations. The importance of this aspect will be further discussed in the 2nd part of the review.

Another interesting observation was the consistent presence of several failed fixtures in the same patient. In other words, few patients accounted for most of the failures. This has been described in the literature as the "cluster phenomenon" or the "clus-

Table 10

Summary of studies reporting on implant loss prevalence attributable to peri-implantitis regarding Brånemark and ITI systems used in various clinical situations

Authors (ref. no.)	Implants inserted/failed*		Failures early/late**		Removed for peri-implantitis***	Loading time (range)
<b>Brånemark System</b>						
JEMT <i>et al.</i> , 1989 (173)	448/13	(PE)	4/9	(4)	1 (2)	1–5 yr
ADELL <i>et al.</i> , 1990 (188)	140/39	(BG)	11/28	(18)	0	1–10 yr
QUIRYNEN <i>et al.</i> , 1992 (26)	509/29	(PE)	23/6	(2)	0	2–77 months
JEMT & LEKHOLM, 1993 (76)	259/7	(PE)	4/3	(2)	1 (5)	5 yr
MERICSKÉ-STERN & ZARB, 1993 (165)	68/5	(OV mand.)	4/1	(1)	1 (?)	5 yr
OLSSON <i>et al.</i> , 1995 (79)****	69/8	(PE)	1/7	(3)	0	5 yr
HENRY <i>et al.</i> , 1996 (84)	107/3	(SI)	1/2	(0)	0	5 yr
total	1600/104		48/56	(29)	3	2 months–10 yr
(%)	100/6.5		46/54		2.8/10.3*****	
<b>ITI System</b>						
D'HOEDT <i>et al.</i> , 1989 (32)	95/15	(OV; PE; TE)	9/6	(6)	6 (2–4)	2 months–6 yr
BUSER <i>et al.</i> , 1991 (88)	54/2	(PE, SI)	0/2	(2)	2 (2–3)	3 yr
WEDGWOOD <i>et al.</i> , 1992 (190)	461/39	(OV; PE; SI)	30/9	(9?)	9 (?)	6–36 months
SALONEN <i>et al.</i> , 1993 (35)	201/14	(OV, TE, PE)	2/12	(8)	4 (2–4)	21–86 months
MERICSKÉ-STERN & ZARB, 1993 (165)	74/5	(OV mand.)	3/2	(2)	1 (3)	5 yr
LEIMOLA-VIRTANEN <i>et al.</i> , 1995 (56)	153/13	(OV mand.)	4/9	(9)	8 (4–8)	3–10 yr
VERSTEEGH <i>et al.</i> , 1995 (54)	153/33	(OV mand.)	4/29	(29?)	29 (?)	45–109 months
ÅSTRAND <i>et al.</i> , 1996 (99)	216/8	(OV & TE mand.)	4/4	(4)	4 (2)	2 yr
total	1407/129		56/73	(79)	63	2 months–10 yr
(%)	100/9.7		43/57		48.8/91.3*****	

\* PE=partial edentulism; BG=bone grafts; TE=total edentulism treated with fixed bridges; OV=overdentures; SI=single implants; mand.=mandible.

\*\* In parenthesis the number of failures after the first year of loading.

\*\*\* In parenthesis the year of removal.

\*\*\*\* Data presented by ÅSTRAND *et al.* (189)

\*\*\*\*\* The first figure represents the % of implant losses due to peri-implantitis in relation to the total number of failed implants, whereas the second figure refers to the % of losses (suspected peri-implantitis) for implants failed after the first year of loading.

The ITI implants were of different designs: type C, E, F, K, H, TPS, Bonefit, hollow cylinders and hollow screws.

Table 11

Distribution and percentage of failed Brånemark implants in relation to possible etiologic factors over a 5-year period; data from Tables 9 and 10

Early failures (47%)	Late failures (53%)	
iatrogenic/host related factors	before 1 yr of loading (45%) overload/host related factors	after 1 yr of loading (55%) overload (90%)/peri-implantitis (10%)

tering effect" (149, 195, 196), and might indicate that also some genetic or systemic factors can play a rôle in implant failure.

In conclusion, biological implant failures with the Brånemark system are relatively rare: 7.7% over a 5-year period (Table 9, bone grafts excluded). The predictability of implant treatment is remarkable, particularly in partially edentulous situations. In fact, implants in partially edentulous patients (Table 5) perform better than in fully edentulous patients (Table 4) in terms of early and total failure rates (2% and 3.8% versus 3.4% and 7.6%, respectively). Our analysis also confirms the general trend of maxillas to have almost 3 × more implant losses than mandibles (Table 8), both in relation to early and late failures, with the exception of the partially edentulous situation, in which practically no difference could be observed. The low prevalence of peri-implantitis found in the literature together with the fact that, in general, partially edentulous patients have less resorbed jaws, speak in favour of lack of sufficient bone volume, deficient bone quality, and overload as the 3 major determinants for late implant failures for Brånemark implants. These findings are in agreement with a recent immunohistochemical investigation on late implant failures (30). However, the relationships between these negative prognostic factors and infections remain to be established in prospective clinical studies.

## Conclusions

While surgical trauma together with bone volume and quality are generally believed to be the most important etiological factors for early implant failures, the etiology of late failures is more controversial. Loading conditions in relation to jaw volume and bone quality together with chronic infection (peri-implantitis) are considered to play the predominant rôle (197). This review of the literature using a metanalytic approach has shown that late failures caused by peri-implant infection are a rare occurrence, at least in relation to the Brånemark system. Further, our analysis indicates that there might be major differences among oral implant systems in the modality of failure which is in agreement with other preliminary recent findings (4).

A fair comparison between different implant systems will only be possible when a general consensus on the parameters for monitoring implant conditions will be obtained. So far, there is no agreement on the parameters to be employed. Data collected from different trials should be amenable to comparison in order to permit pooling and analysis. Therefore, standardization of clinical trial designs dealing with oral implants is an urgent must. In particular, success criteria must be well described, universally accepted, and should be related to the goal of the treatment. Ideally, in order to avoid possible bias, clinical performances of different oral implants should be evaluated by independent bodies. It is definitively recommended to concentrate more emphasis on the aspects of failure providing adequate figures, so that studies could be more easily compared and precious clinical guidelines retrieved. Moreover, only a limited number of clinical studies have been specifically designed to identify the prognostic factors influencing implant failure. As is evident from the results presented in the reviewed literature, there is also an apparent need for an analysis of the clinical course with new/improved (non-invasive) techniques. Further, in order to understand the mechanisms of failure and their clinical correlates, failed implants and their surrounding tissues have to be collected and analyzed.

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