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An Analysis of Factors Affecting Bone Loss Around Osseointegrated Implants in the Edentulous Maxilla.

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A treatise submitted in partial fulfillment for the degree of

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Abstract

Introduction

Osseointegrated implants are considered a reliable treatment for edentulous patients with few failures reported. Several theories and risk factors have been offered to account for failures with none being reliable predictors of failure in isolation of other factors. Continuous peri-implant bone loss across time is an indicator of implant failure although its relationship to patient and local factors and the clustering of implant failures in individual patients is poorly understood. In addition, consensus reports have called for patient-centred outcomes to be evaluated.

Aims

To analyse both the factors affecting peri-implant bone loss and patients' perception of maxillary implant reconstructions.

Materials and methods

This study was undertaken to review bone loss around machined surface Bränemark implants placed in the maxillae of elderly patients in relation to age, gender, health status, smoking, mucosal health, oral hygiene, length of implants, prosthetic type and opposing occlusion. A retrospective analysis of maxillary implant reconstructions at Sydney Dental Hospital (n=36, ages 32-73 at implant placement, 20M: 15F) was performed 11-19 years after implant placement. Initial clinicians were 3 oral surgeons and a prosthodontist. Bone loss relative to implant threads was assessed from radiographs at stage two surgery and differing intervals thereafter. Correlations and interactions between recorded factors and bone loss were assessed. Patients' perception of their treatment satisfaction was assessed by their responses to 12
statements in a questionnaire and verified by marking of a visual analogue scale (VAS).

Results
Significant correlations (p < .05) were detected between peri-implant bone loss and mucosal health, oral hygiene and implant length. Patient satisfaction with implant based reconstructions of the edentulous maxilla was high, with insignificant difference between prosthetic types. Failure to meet patient expectation was the most frequent negative report.

Discussion
Implant and oral health factors have a greater effect on peri-implant bone loss than general patient factors. Studies with greater sample size are indicated to clarify possible associations and interactions between other factors that may individually have undetectable effects. Patient satisfaction with implant reconstruction is high and appears to be more strongly related to an adequate solution for the patients’ presenting complaint than to the treatment modality selected.

Conclusion
Long term success of implant treatment may depend on the patient’s ability to maintain oral hygiene and peri-implant mucosal health. Adequate clinical instruction and reinforcement of implant-specific cleaning techniques is warranted.
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Introduction

Following the presentation of Bränemark et al of the concept of osseointegration in 1977, confirmative studies such as that of Adell et al. (1981) opened the way for the more routine use of oral implants to replace teeth in edentulous patients. Further studies confirmed high levels of success, additionally identifying risk factors for failure, such as smoking. Progressive peri-implant bone loss is a recognised indicator of failure, though the factors that affect this bone loss are not fully understood.

Before examining current theories of this bone loss and possible influencing factors, the nature of the implant's interface with the soft and hard tissues will be reviewed.

1.1 Osseointegration and the Mucosal Interface

Osseointegration has been defined by Albrektsson et al. (1981) as a “direct functional and structural connection between living bone and the surface of a load-bearing implant”. The definition refers to a physical integration of the machined implant and the bone, macroscopically at the level of the implant threads and microscopically at the level of the machining irregularities (Puleo and Thomas 2006). The physical contact is possibly due to the inert nature of the titanium oxide layer that forms on commercially pure titanium when exposed to the atmosphere. It is in contrast with implants that are prepared or coated to have a biologically “active” surface.
In their review, Esposito et al. (1998) discussed the definitions of osseointegration presented by Brånemark in 1996 (patient-based, macro and microscopic medical and histologic classifications), noting the need for a definition that clarifies that there is an absence of a soft-tissue interface between the implant and bone.

A more recent definition that perhaps more accurately reflects the dynamic nature of the implant/bone interface was proposed by Cochran (2006), as: “Osseointegration: Stability of an implant in bone that represents a dynamic equilibrium between existing native bone (primary contact) and remodelling and new bone formation (secondary contact) and its maintenance at the bone-implant interface”

Raghavendra and colleagues (2005) reviewed the evidence relating integration and adaptation processes. Brånemark’s surgical protocol, as described by Adell et al. (1981), required that the vitality of the bone be respected at all times; drills being used with isotonic coolant and the drilling sequence being such that the chance of overheating is minimised. The difference in diameter between each successive drill decreases so that the final drill removes the least bone. The implant may then be screwed into the bone, such that the surface closely approximates the prepared bone ensuring that the implant achieves primary stability, without trauma to bone.
Implant stability is essential during the healing period, being achieved with the Brånemark protocol by mucosal closure and relief of the overlying prostheses. In a review of the literature, Szmukler-Moncler and colleagues (1998) estimated that the maximum micro-motion that the osseointegrated interface could accommodate was 50-150μm. Fibrous encapsulation of the implant may occur with greater movement.

1.1.1 **Endosseous Healing and Initiation of Remodelling**

Endosseous healing follows implant placement in a similar manner to other forms of bone injury. The sequence of events can be divided into haematoma...
formation, clot resolution, osteogenic cell migration, formation of new bone and then remodelling. A summary of these processes is as follows

1) Haematoma formation - the implant surface becomes coated with plasma proteins, rapidly followed by platelet adhesion, activation and degranulation. A fibrin clot rapidly forms, providing a physical barrier to prevent further bleeding; additionally it serves as a reservoir of growth factors and cytokines (Gemmel and Park 2000).

2) Intense cellular activity follows the infiltration of the clot by neutrophils, monocytes and fibroblasts. This intensity leads to some anaerobic metabolism, creating lactic acid, reducing the pH and creating an oxygen gradient that is chemotactic for endothelial and mesenchymal cells.

3) Fibrinolysis occurs with the formation of a loose connective tissue stroma that supports angiogenesis. From about 4 days up to approximately 3 weeks granulation tissue forms, the initial matrix gradually being replaced by a collagen-rich matrix, which precedes bone formation.

4) Osteoblasts secrete non-collagenous bone proteins, primarily osteopontin and sialoprotein, onto the implant surface. Calcium binding occurs at the binding sites on the proteins, followed by the crystal growth phase. The collagenous matrix becomes mineralised in a rapid and asynchronous manner, leading to the formation of woven bone. This process predominates within the first 4-6 weeks after implant placement. During these initial healing stages necrosis and resorption of traumatised bone from both the osteotomy preparation and implant placement occurs. Roberts (1988) estimated this to be a zone of approximately 1mm in compact bone, despite optimal surgical technique. Osborn and Newesley
in Raghavendra et al. (2005) proposed that machined surface implants integrate by "distance osteogenesis", that is, formation of new bone on the surface of the existing peri-implant bone until integration is complete. This is in contrast with "de novo" bone formation on the surface of the implant, which occurs on implants with some surface preparations (Davies 2000).

5) After about 4 weeks, the woven bone is gradually replaced by lamellar bone with haversian architecture. Having collagen fibrils arranged in parallel layers with alternating courses, the lamellar bone has high strength, which by virtue of being in close contact with the implant surface gives the implant the necessary rigid fixation to accommodate loading.

From about one month after surgical placement the bone begins to remodel as a process that occurs throughout life. Remodelling reflects functional adaptation of the bone structure to load, and occurs by osteoclastic resorption and lamellar bone deposition that are coupled both in space and time (Stanford and Brand 1999). Roberts (1988) estimated this to involve about 30% of the implant surface per annum. The changes in peripheral bone density around implants over a one-year period and under different loading protocols have been demonstrated by subtraction radiography as described by Appleton and colleagues (2005). Though this measurement technique lacks validation, the results are in keeping with current theories on the anabolic effects of physiological overload, implying that at the time of placement of a prosthesis, an implant may not have reached its optimal ability to withstand load.
Remodelling process occurs in the maintenance of bone around loaded implants over time and may be affected by systemic or local changes in bone metabolism, which may influence implant success or failure. The mechanisms involved and possible role in pathology of peri-implant bone loss are discussed further in Section 2.4.1.

1.1.2 The Soft Tissue Interface

The placement of a transmucosal abutment onto the implant induces the mucosa to form an attachment-complex analogous to the dento-gingival complex. With natural teeth, this provides a functional seal to prevent ingress of bacteria and their toxic products.

Whilst the dimensions of both attachment complexes are similar, the connective tissue around implants is significantly different (Avery 1994). With natural teeth, the presence of cementum on the root surface allows the attachment of dento-alveolar and dento-gingival collagen fibre bundles, extending laterally, apically, coronally and interdentally. In contrast, with implants the fibres extend from the periosteum of the bone crest parallel to the abutment surface to insert into the mucosal margin (Berglundh et al., 1991).
Inflammatory lesions around teeth and implants
(Adapted from O’Neal and Edge 1994)

Key: GM – gingival margin, aJE – apical junctional epithelium, BC – bone crest
aICT – apical termination of connective tissue,

Schematic drawing demonstrating the landmarks on the tooth and implant that separate the area of the sulcus and the junctional epithelium, the connective tissue and the alveolar bone. The sulcus and junctional epithelium extend from the GM to the aJE, the connective tissue extending

Schematic drawing of 21 days of plaque formation, demonstrating the area of connective tissue inflammatory infiltrate that extends from the gingival margin to the apical termination of the connective tissue. The area of the inflammation at 21 days is similar adjacent to both the tooth and

Schematic drawing of 3 months of plaque formation, demonstrating the area of inflammatory connective tissue infiltrate that extends from the gingival margin to just short of the apical termination of the junctional epithelium with the tooth, and from the GM to the aJE with the implant.

Schematic drawing of destructive periodontitis demonstrating the area of inflammatory connective tissue infiltrate that extends from the gingival margin to just below the apical termination of the junctional epithelium with the tooth, and from the gingival margin to the bone crest with the implant.

Figure 2. Inflammatory lesions around teeth and implants
The connective tissue is less encapsulated and less organised with fewer fibroblasts with a greater resemblance to scar tissue than gingival tissue. As a result, the healing process during bacterial affront shows a reduced ability to produce adequate collagen to limit the spread of inflammatory cells and may lead to further and more rapid breakdown (Berglundh et al., 1992). Refer to Figure 2.

This structural difference appears to have important clinical consequences. The initial microbial colonisation on titanium implants appears to follow the same pattern as on natural teeth (Zitzmann et al., 2001) and induces a similar inflammatory response. However, a study with beagle dogs has shown differing patterns of spread of inflammatory cells in response to plaque induced lesions. With teeth, the inflammatory response is prevented from reaching the crestal bone by connective tissue, whilst with implants the inflammatory cells extend into the bone marrow spaces of the alveolar bone (Lindhe et al., 1992). The study showed wider progression of lesions and greater bone loss around implants than teeth.

In a review, Esposito et al. (1998a) suggest that periodontal probing depth around implants is not a reliable indicator of pathology due to differing tissue thicknesses and abutment lengths. Another clinical consequence of the differing structures is that a periodontal probe is more likely to displace the junctional epithelium and penetrate to the bone crest around implants than around teeth. Bleeding on probing cannot be used to discriminate between healthy or diseased peri-implant states (Esposito et al., 1998a)
1.1.3 Bone Loss in the First Year

| a) Brånemark implant showing the implant collar and its junction to the abutment | b) Radiograph depicting classic first year pattern of bone loss to the first thread |

Figure 3. a) Brånemark implant showing the junction between the collar and the abutment and the implant threads. b) Radiograph indicating a healthy bone-implant interface one year after loading.

The Brånemark surgical protocol describes placement of the implant such that the collar is level with the bone crest. After second stage surgery, it is usual for some marginal bone loss to occur and usually to the level of the first implant thread (about 1.5mm) from the marginal bone level, as illustrated in Figure 3. A variety of explanations have been proposed, including cratering to improve stress distribution (Kitamura et al., 2004), trauma from elevating the periosteum (Adell et al., 1981), establishment of a biologic width of the
implant-mucosal unit, and presence of a microgap between the implant and abutment (Isidor 2006). A number of factors suggest that a microgap is the most likely explanation:

1) The marginal bone loss occurs with unloaded trans-mucosal healing abutments;
2) The microgap has been shown to be colonised by bacteria (Quirynen et al., 1993, 1994);
3) A persistent acute inflammatory reaction can be observed histologically at the level of implant-abutment interface (Broggini et al., 2003). This is not observed with one-piece implants (Hermann et al., 2000); and
4) The dimension of the bone to bacterial source is very similar to that reported for plaque in periodontal pockets and the bone crest (Waerhaug 1979, Jones and Cochran 2006).

Whatever explanation one accepts, this initial marginal bone loss appears to be a normal aspect of 2-part implant systems and does not appear to adversely affect outcomes. The microgap does not appear to prevent attachment of the mucosa to the abutment. Therefore, from the point of view of monitoring marginal bone levels, it is appropriate to record the bone level at one year and to monitor bone loss from that time.

The proposal that a minimum distance between bone and bacteria of the microgap is supported by Tarnow and colleagues (2000). In their study measurements of the inter-implant crestal bone height and inter-implant distances at the implant/abutment interface were made. Standardised,
digitised scanned radiographs from 36 patients with adjacent machined titanium implants were used. A significant trend towards inter-implant crestal bone loss was found when the implants were less than 3mm apart. (See figure 2). This supports the hypothesis that bone resorption will occur to a distance of approximately 1.5mm from bacterial challenge and gives additional support to microgap bacteria being a cause of bone loss to the first thread.
The effect of inter-implant distance on the height of inter-implant bone crest
(Adapted from Tarnow, Cho and Wallace 2000)

Key: A and B represent the lateral distance (bone loss) from the implant to the bone crest, C is the vertical crestal bone loss, D is the distance between implants at the implant-abutment interface.

**Measurements recorded**

- A and B represent the lateral distance (bone loss) from the implant to the bone crest, C is the vertical crestal bone loss, D is the distance between implants at the implant-abutment interface.

**Inter-implant distance >3mm**

- Lateral bone loss from adjacent implants (A and B) does not overlap, with minimal resultant crestal bone loss (C).

**Inter-implant distance <3mm**

- Lateral bone loss from adjacent implants (A and B) overlap, with resultant increase in crestal bone loss (C).

**Distribution of measurements D and C**

- Relation ship between crestal bone loss and inter-implant distance.

Figure 4. The effect of inter-implant distance on the height of inter-implant bone crest
1.2 Principal Theories of Peri-implant Bone Loss

1.2.1 Overloading

'Overload' has been described as occurring when occlusal forces exceed the mechanical or biological load-bearing capacity of the osseointegrated oral implant-bone interface, or the prosthesis, resulting in either mechanical failure or failure in osseointegration (Isidor 1999, Isidor 2006).

In a histological study of failed Brånemark implants in humans it was concluded that for 8 out of 10 implants the reason for failure was overload (Esposito et al., 2000). Calculating from existing studies of machined implants, Esposito et al. (1998a, 1998b) suggested that late failures (after more than one year of loading) were due to overload in 90% of cases, 10% being attributed to peri-implantitis.

The significance of overloading is controversial with conflicting results from animal studies. A study using primates (Isidor 1996, 1997b) involved placement of implants in the posterior maxilla bilaterally, with significant unilateral overload and ligatures promoting peri-implant plaque accumulation on the contralateral side. On the loaded side 5 out of 8 implants lost integration without significant inflammation, whilst on the contralateral side inflammation and loss of peri-implant bone was noted. In contrast, a dog study by Heitz-Mayfield et al. (2004) reported that implants with unilateral contacts in hyper-occlusion resulted in no clinical or radiographic evidence of loss of integration.
In the primate study (Isidor 1997b), the unloaded implants showed poor implant to bone contact (38%), whilst in the study of Heitz-Mayfield et al. (2004), the bone to implant contact was between 69% and 79%. Lateral loading concentrates forces on the peri-implant crestal bone (Kitamura et al., 2004) such as in primates that have a more elliptical masticatory pattern, whereas dogs have a vertical masticatory pattern. In another implant loading study in primates, where implants were placed in hyper-occlusion against natural teeth, the effect of load was remodelling and thickening of the peri-implant bone and intrusion of the opposing teeth (Ogiso et al., 1994). These studies do not establish that loading alone is the causative factor of implant failure due to (a) the studies being animals and (b) the studies having used different study designs. However they do provide evidence of the biological consequences of loading of bone, that is important in understanding peri-implant bone changes.

Occlusal forces affect bone surrounding implants. Deformation of bone is described as strain, which is the relative change in the length (deformation) of the bone and often expressed in microstrain, where 1000 microstrain units equate to a deformation of 0.1%. Being related to actual dimensional change, strain depends on the mechanical properties of bone, which will differ in different anatomical locations and inter-individually. The effect of this variability will depend on the reaction of the bone to applied strain.

Stanford and Brand (1999) reviewed the historical concepts of loading and bone remodelling from which Frost (1992) developed his mechanostat theory, which is most easily understood from the following diagram:
Figure 5. Frost's mechanostat theory. Minimal effective strain (MES) of 50 to 250 μ-strain units is necessary to prevent net loss in bone mass (disuse atrophy), whereas steady state levels of normal remodeling exists from 50 to 250 and 2500 to 3500 μ-strain. The shaded area represents the range of response related to bone mass. Peak load magnitudes creating strains above 2500 to 3500 μ-strain MES, lead to new bone formation (modelling) that continues until increased bone mass decreases the strain values below modelling MES. Peak load levels >25,000 μ-strain lead to rapid catastrophic fracture. Adapted from Stanford and Brand (1999).

The mechanostat theory suggests that bone with a lower density and therefore lower modulus of elasticity will be deformed more per unit force than denser bone. The result would be greater microstrain per unit force and an increased likelihood of the force reaching the pathological overload zone. This appears to offer an explanation for the higher failure rates of implants in the maxilla, as noted in published data.

Clinically it is difficult to quantify both the magnitude and direction of occlusal forces. As a result, only a small number of experimental studies have been
performed and it is difficult to draw definitive conclusions. A biologically plausible theory suggested by Isidor (2006) would be that overload would be a causative factor of peri-implant bone loss for those patients in whom the bone repair mechanism was overwhelmed by the loading-induced micro fractures. This theory suggests that either load changes or bone metabolism changes could result in late peri-implant bone loss in a previously satisfactory situation.

The importance of possible effects of modulators on the ability of bone to withstand load on oral implants is not fully understood. A diagrammatic representation of possible sites of effect is illustrated in Figure 5 (from Schoenau, 2005).

![Diagram]

Figure 6. Diagrammatic representation of modulation of bone turnover. Reproduced from Schoenau (2005)

There is a lack evidence for the possible role of modulators on peri-implant bone loss.
1.2.2 Peri-implantitis

Peri-implantitis has been defined as an inflammatory process affecting the surrounding tissues of an osseointegrated implant during function, resulting in loss of supporting bone (Albrektsson and Isidor (1994)). The loss of supporting bone distinguishes peri-implantitis from peri-implant mucositis, which is a reversible inflammatory change.

The similarities between the gingivitis model for teeth and peri-implant mucositis model were demonstrated by Pontoreiro et al. (1994). The cause-effect relationship between bacterial accumulation and inflammatory change was established. Peri-implant mucositis may progress to peri-implantitis with progressive destruction of the implant supporting tissues until failure (Esposito et al., 1998b). Strategies for treatment of peri-implant inflammation have been established e.g. the Cumulative Interceptive Supportive Therapy (CIST) protocol (Lang et al., 2004).

The colonisation of peri-implant pockets with similar subgingival microbiota as surrounding teeth was shown by Quirynen et al. (2006) to occur within two weeks. In a similar study, De Boever and De Boever (2006) analysed the peri-implant pocket microbial composition of well controlled (less than 20% plaque and bleeding on probing scores) advanced aggressive periodontitis patients. For the 68 implants in 22 patients placed with a one-stage protocol, they found that by day 10 post-surgery, the pocket microflora was very similar to the natural teeth, with the presence of five known periodontal pathogens at
most sites. Over the following 6 months, this peri-implant microbiota remained almost unchanged, but was not accompanied by peri-implant mucositis, peri-implantitis, loss of bone or failure to integrate. This suggests that secondary factors are involved in destructive peri-implantitis, not only the presence of pathogenic bacteria.

The explanation is supported by Esposito et al. (1998b) who, on the basis of available evidence at that time, suggested that implants may be the ideal treatment for patients who had been affected by aggressive periodontal disease. Van Steenberghe et al. (1999) in their discussion of factors affecting marginal bone loss suggested that implants from the Brånemark system appeared to be more resistant to loss of attachment than teeth. However, a 10-year prospective study by Karoussis et al. (2003), compared success and complication rates between a group of patients who lost their teeth due to periodontal disease and a second group without a history of periodontitis. The researchers found a significantly higher incidence of peri-implantitis in the patients with a history of periodontal disease (28.6% vs 5.8%) and a significantly lower success rate. A recent review of 218 patients who had received 1057 implants in the preceding 9-14 years found a similar association (Roos-Jansaker et al., 2006). In a review, Schou et al. (2006) stated that available evidence does not allow firm conclusions to be drawn about the outcome of implant treatment in patients with a history of periodontitis.

A link between poor oral hygiene and bone loss is difficult to establish despite evidence of a correlation between plaque and peri-implant inflammatory
change. The difficulty is in part due to the averaging of bone loss values that is frequently reported in clinical studies, combined with the removal of high bone loss implants from the calculations if the implants are lost (Esposito et al., 1998b). Only a weak correlation has been established for machined surface implants in one 15-year study (Lindquist et al., 1996).

Salcetti and co-workers (1997) explored the suggestion of a patient-based risk factor in peri-implantitis. The researchers analysed the peri-implant sulcus fluid in two groups of patients for the presence of anabolic transforming growth factor β (TGF β) and platelet-derived growth factor (PDGF) and catabolic bone factors (interleukin-1β (IL-1β) and prostaglandin E2 (PGE2). The first group of 21 had a failing implant site, 15 of these also had a healthy implant site. The second group consisted of 8 patients with only healthy implant sites. Plaque samples were also analysed using a DNA hybridisation technique to identify 40 organisms. The analysis found no significant differences between microbial, inflammatory or growth factors between healthy and diseased implants in group one, but greater levels of inflammatory mediators and PDGF in patients with diseased implants than in the healthy controls. The study concluded that ‘risk appears to be primarily at a patient level and secondarily at a site or implant level from a clinical, microbial and biochemical perspective’ (Salcetti et al., 1997).

1.2.3 Combined theory

Establishing whether peri-implant bone loss is due to inflammation or loading when both are present is difficult. The difficulty has been discussed in reviews
(Esposito et al., 1998b, van Steenberghe et al., 1999, Isidor 2006), where the consensus is that there is only weak evidence for a combined aetiology but that there is reason to speculate that both factors may act synergistically.

1.3 Risk Factors for Endosseous Implants

1.3.1 Recognised risk factors

a) Poor bone quality and quantity/grafting
b) Smoking
c) History of radiotherapy
d) Bruxism
e) Opposing dentition
f) Implant number and cantilever length
g) Operator experience

a) Poor bone quality and quantity/grafting

Poor bone quality and volume are recognised relative contraindications to implant therapy (Molly 2006, Kronström et al 2001, Esposito et al., 1998a, Engquist et al., 1988, Bryant 1998, Jemt and Häger 2006). Despite the lower success rates, Jemt and Lekholm (1995) concluded that a survival rate of 70% for these patients was acceptable given that failures tend to occur early and, in most cases grafting was not a viable option. Onlay bone grafting for severely resorbed ridges gave comparable success rates to severely resorbed ridges but significantly worse than only moderately resorbed ridges.
There is a lack of quality data on the relative success rates of different grafting techniques and the continuing resorption of autogenous onlay grafts should be acknowledged (Chiapasco et al., 2006). From a treatment planning perspective, the possible lower success rates of shorter implants should be weighed up against the increased morbidity associated with more surgical procedures (Renouard and Nisand 2006).

b) Smoking

Bain and Moy (1993) described the negative effect of smoking on implant success, whilst the benefit of a smoking cessation protocol at the time of surgical placement was shown by Bain (1996). In their review on the aetiopathogenesis of implant failures, Esposito et al. (1998b) agreed that smoking increases the risk of early implant failure. The effect of components of cigarette smoke on tissue function, particularly microcirculatory changes, has been documented (DeLuca et al., 2006).

After assessing for publication bias, Hinode and colleagues (2006) completed a meta-analysis of 19 selected studies reporting on this issue, although none of the included studies were longitudinal - results reproduced in Figure 6. These results would suggest smoking affects short-term implant success, that is, the osseointegration process. To what extent smoking affects the long-term success of implants is less clear.
Figure 7. Random effects of the odds ratios of osseointegrated implant failure on smoking for individual studies and overall. The diamond symbol shows the odds ratio and the horizontal line represents the 95% confidence intervals.

Reproduced from Hinode et al. (2006).

In a recent 20-year retrospective study, DeLuca et al. (2006) reported on the outcome of 1852 implants placed in 464 patients. Early failures were significantly associated with smoking (odds ratio 1.76) which is in agreement with most other studies (Hinode et al., 2006). DeLuca et al. (2006) also reported on the relationship of a smoking history and late implant failure. The criterion for smoking history was defined as greater than 25 cigarette years (cy), that is, the product of the number of cigarettes per day and the number of years of smoking, before Stage II surgery. The study found a significantly
greater chance of late implant failure with a smoking history (odds ratio 2.01). This interesting finding is suggestive of a correspondence with the reduction in bone density associated with smoking (DeLuca et al., 2006). More rapid resorption of less dense bone could occur in association with bacterially induced inflammatory challenge. Lindquist et al. (1997) showed only a slight increase (0.6mm at 10 years) in bone loss around mandibular implants when smoking was associated with poor oral hygiene. The greater bone density found in the anterior mandible may have minimised potential bone loss.

c) History of Radiotherapy

Radiotherapy is commonly used in conjunction with chemotherapy and surgery for oral malignant tumours and has a profound effect on all cells within the exposed field. The radiation affects bone by initiating cell death and impairing the healing response. The result is a hypovascular, hypocellular and hypoxic tissue that is intolerant to traumatic and surgical insults in a dose-dependant manner (Esposito et al., 1998b). The complex interrelationship of dose-dependancy, time, hyperbaric oxygen therapy and the reduction in implant integration rates has been discussed by Granström (2003).

d) Bruxism

The increased forces associated with bruxism (sleep and daytime) are believed to adversely affect implant outcomes (Esposito et al., 1998b) and may be a factor in the “clustering” of implant failures in individual patients (Ekfeldt et al., 2001). Analysing data from 17 patients with clustering of
failures in the maxilla, Jemt and Häger (2006) reported that failures were often initiated in one quadrant, leading to unfavourable stress distributions that may have precipitated further overload-related implant losses. However, when comparing matched controls, no difference in occlusal wear, or signs and symptoms of bruxism could be observed. The authors concluded that there did not appear to be an association.

One problem with studies relating to sleep bruxism is the subjective nature of its clinical assessment. Now recognised to be a movement disorder during sleep, an accurate diagnosis can only be accurately made with sleep studies and video monitoring (Kato et al., 2001).

e) Opposing dentition

Becktor et al. (2002) reported that unilateral opposing occlusions increased the failure rate of osseointegrated implants in the grafted edentulous maxilla. In a review of occlusal considerations in implant therapy, Kim et al. (2005) speculated that overload would be more likely with an opposing occlusion supported on implants. This has been proposed to arise as a result of the decreased tactile perception of implants compared with teeth, the altered neurophysiological feedback from the periodontal tissue receptors to the jaw muscles, and the loss of load-sharing from tooth periodontal ligament compliance. There is little evidence of association with different occlusal support in opposing dentitions and peri-implant bone loss (Kim et al., 2005).
f) Implant number and cantilever length

Brunski (1999) discussed the increased likelihood of overloading with insufficient implants to support a prosthesis and with increased cantilever lengths. Several studies have reported greater loss of the distal implant when a cantilever is present (for review see Kim et al., 2005), which supports the hypothesis of loading-related clustering of implant failures as proposed by Jemt (2006). When the distal implant is lost, unless it is replaced or the cantilever reduced, increased forces will then apply to the next most distal implant with the increased possibility of sequential implant loss. Also important is the proposal that with greater numbers of implants there is an increased risk of prosthetic misfit leading to increased bacterial accumulation, inflammatory response and bone loss (Hermann et al., 2005).

g) Operator experience

Surgical experience has been reported to be important in the success of implant therapy, with greater failure rates attributed to surgeons with fewer than 50 implant surgeries (Esposito et al., 1998b).

1.3.2 Systemic Factors

Systemic factors affecting the success and failure rates of implant placement include,

a) age
b) systemic disease (current and previous medical conditions)
c) medications
d) genetics
e) smoking (discussed in section 2.5.1).

a) Age

In a review of biological factors in implant failure, Esposito and colleagues (1998b) discussed the changes in mineral composition, structure, amount of bone morphogenetic proteins (BMPs) and decreased healing rate of bone with increasing age, although clinical studies do not support an increased failure rate for implants.

In a well-controlled study examining peri-implant bone loss with age, Bryant and Zarb (2003) closely matched implants and prostheses of an older age group (60-74 years, n=32) with a younger group (29-49 years, n=34). Exclusion criteria included absence of systemic illness that precluded minor oral surgery. In the elderly group, the incidence of medications and systemic illness was greater although this factor was not analysed further. The conclusion was that elderly patients should expect similar success rates and similarly low rates of peri-implant bone resorption.

In contrast, a single operator study (Moy et al., 2005) reviewed 4680 implants placed in 1140 patients over a 21-year period and concluded that patients in the age range 60-79 years were more than twice as likely to experience failures than those in the under 40 years group. Despite being a retrospective
study, the results provide valuable analysis of outcomes from routine practice using a standardised surgical protocol.

Factors affecting both the short and long-term prognosis of implants are likely to be cumulative (Mombelli and Cionca (2006)). Although there is little evidence to suggest that age is an outright risk factor, a patient's age may act synergistically with other risk factors to negatively influence outcomes.

b) Systemic disease

In recent years, five non-systematic reviews have been published on the topic of associations between implant success and systemic diseases (Sugerman and Barber (2002), Beikler and Flemmig (2003), van Steenberghe et al. (2003), Wood and Vermilyea (2004), Mombelli and Cionca (2006)).

Except for acute contraindications to surgery, all the above reviews attest to the resiliency of bone healing and osseointegration, citing short case series or case reports of successfully integrated implants with a diverse range of medical conditions (for example, Human Immunodeficiency Virus - Rajnay and Hochstetter (1998)) and medications (for example, cytotoxic chemotherapy, - McDonald et al.(1998)).

Mombelli and Cionca (2006) suggest that whilst a single factor may appear not to alter the risk to a measurable extent, combinations of factors may have a noticeable effect. Case studies have provided additional knowledge;
however, the results should be interpreted with caution. The high prevalence of diabetes and osteoporosis in the community require appraisal of these diseases.

Diabetes

There is strong evidence that diabetes is a risk factor for gingivitis and periodontitis, with the level of glycaemic control being an important determinant in the association (Mealey and Oates (2006)).

There are two types of diabetes: Type I (previously termed 'insulin-dependent') caused by autoimmune destruction of the β cells of the pancreas that leads to insufficient or no insulin; and Type 2 (previously termed 'non-insulin dependent') which is insulin resistant combined with an inability to produce sufficient additional insulin to compensate. Diabetes is thought to affect 120-140 million people worldwide and is becoming increasingly prevalent, with Type 2 diabetes accounting for 90-95% of cases (Sugerman and Barber (2002)).

Diagnostic testing for diabetes include fasting plasma glucose (normal ≤ 100 mg/dl) and a 2-hour post-glucose load test which indicate short-term control of blood glucose levels. Glycosylated haemoglobin (HbA1c) levels indicate the average blood glucose levels over the preceding 30-90 days, allowing assessment of longer-term control. The normal value is < 6% (Mealey and Oates (2006)). HbA1c levels correlate well with the development of diabetic complications.
Table 1. Correlation between HbA1c levels and mean plasma glucose levels (healthy normal = < 100mg/dl)

<table>
<thead>
<tr>
<th>HbA1c (%)</th>
<th>Mean Plasma Glucose (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>6</td>
<td>135</td>
</tr>
<tr>
<td>7</td>
<td>170</td>
</tr>
<tr>
<td>8</td>
<td>205</td>
</tr>
<tr>
<td>9</td>
<td>240</td>
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<tr>
<td>10</td>
<td>275</td>
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<tr>
<td>11</td>
<td>310</td>
</tr>
<tr>
<td>12</td>
<td>345</td>
</tr>
</tbody>
</table>

Adapted from Mealey and Oates (2006)

In a review of the literature on diabetes and periodontal disease, Mealey and Oates (2006) suggested that the following factors may be of relevance to peri-implant bone loss:

- Impaired osseous healing and bone turnover in association with hyperglycaemia has been demonstrated in several studies.
- The level of inflammatory mediators in gingival crevicular fluid is related to the level of glycaemic control; for example, the levels of IL-1β have been shown to be twice as high in subjects with HbA1c levels above 8% compared with those below this level. The net effect of diabetes is increased inflammation, attachment loss and bone loss around natural teeth.
- Hyperglycaemia leads to glycosylation of proteins and an accumulation of advanced glycation end-products (AGEs). These stabilised proteins produce vascular damage and disturb healing. The association
between AGEs and diabetic complications would suggest that the level of glycaemic control and duration of diabetes are important.

Similar structures and environments of periodontium and peri-implant mucosa (section 2.5) suggest that similar findings could be found around implants as with teeth. An increase in both short-term (failure of osseointegration) and long-term (loss of integration) failures would be expected. Peri-implant bone loss would be expected to increase.

The expectation is supported by additional information from animal studies regarding implants and diabetes which suggest that bone-to-implant contact is decreased in uncontrolled diabetic animals compared with controls, but similar in controlled diabetes (Beikler and Flemmig (2003)).

A frequently cited report of 213 successfully restored implants from 227 placed (6.7% failure) in 34 Type 2 diabetic patients did not report on either short- or long-term levels of diabetic control (Balshi and Wolfinger (1999)). Olsen et al (2000) reported a 10% failure rate in a 5-year follow-up on implants placed in the anterior mandible of 89 patients. Hba1c and fasting glucose levels were assessed before and after treatment, although interestingly, no correlation was found between these parameters and implant success. However, a correlation was found between duration of diabetes and implant success.

In reviewing the literature on diabetes and implant failure, Mombelli and Cionca (2006) concluded that the evidence was equivocal. The largest cohort study including 48 diabetic patients (Moy et al., 2005), discussed in the
context of age) found diabetes to be a significant risk factor (relative risk 2.75%). An unpublished observation (Acursi (2000)) quoted by Mombelli and Cionca (2006) provided the highest level of evidence, with matched controls for 15 diabetic patients and found no increased risk of implant failure.

In summary, although the pathophysiology of periodontal and microvascular complications of diabetes are well documented, there is lack of evidence with regard to peri-implant bone loss and equivocal evidence on implant survival.

Osteoporosis

Osteoporosis is a progressive systemic disease characterised by low bone mass and deterioration of bone tissue, leading to bone fragility and fracture. Current diagnostic criteria are based on dual-energy X-ray absorptiometry, with a diagnosis of osteoporosis if the bone mineral density is 2.5 standard deviations below that of a mean young population (Glaser and Kaplan (1997)).

Osteoporosis is more common in the elderly and post-menopausal females and with an ageing population the incidence is likely to increase. The relationship with implants is that the jawbones may be affected similarly to the rest of the skeleton, though the evidence for this is controversial (Beikler and Flemmig (2003)). There is lack of evidence to suggest that impaired bone metabolism associated with osteoporosis is sufficient to affect implant integration.
A study comparing mineral content in mandibular bone in 7 osteoporotic and 11 non-osteoporotic women, 5 years after implant loading showed significantly greater marginal bone loss in the osteoporotic group, although no implant failures (Von Wowern and Gottfredsen (2001)). The authors stated that pre-existing osteoporosis may be a risk factor for peri-implant bone loss, but should not contraindicate implant therapy.

c) Medications

Whilst there is a theoretical basis for many medications to affect bone homeostasis, there is a lack of clinical evidence relating prescription medications to either peri-implant bone loss or implant failure:

- Concurrent chemotherapy and implant placement greatly increase failure rates. However, it does not appear to adversely affect integrated implants (Wolfaardt et al., 1996).

- Nifedipine, a cardiovascular regulator which induces gingival enlargement around teeth, may cause peri-implant mucosal hyperplasia, but does not adversely affect osseointegration (Silverstein et al., 1995).

The commencement of etindronate therapy in a 76-year-old woman with pre-existing osteoporosis was suggested to have caused the failure of her 5 mandibular implants (Starck and Epker (1995)). Whilst analysis of the case suggests the loss may have been multifactorial (short implants, long cantilevers, a poorly fitting upper denture and bruxism), the case is relevant as it is known that the bisphosphonate group of medications (to which etidronate
belongs) disturb bone homeostasis. There is increasing concern in recent years due to reports of osteonecrotic lesions of the jaws following bisphosphonate therapy (Ruggiero et al., 2004, Melo and Obeid (2005)). Bisphosphonates are commonly prescribed as their efficacy in preventing osteoporotic fractures is well documented. For this purpose, bisphosphonates are best prescribed in low dosages of the weaker variants (Cheng et al 2005). The osteonecrotic complications described have almost always involved stronger formulations that are used for oncological purposes. Bisphosphonates are thought to act by decreasing osteoclastic activity and hence bone resorption, in addition to disrupting blood flow and angiogenesis (Cheng et al 2005). A recent randomised-controlled trial comparing implant treatment in 335 patients was undertaken comparing outcomes with alendronate therapy and a placebo (Jeffcoat (2006)). Implant success was greater than 99% for both groups at 2 years. Although the bisphosphonate therapy did not appear to adversely affect implant outcomes, the long-term cumulative effects of these medications, which have a long half-life (over 10 years) is unknown. They may have relevance in the maintenance of peri-implant bone levels as bisphosphonates have been shown to inhibit peri-implant bone resorption in an experimental peri-implantitis model in dogs (Shibutani et al., 2001).

In comparison with the gross disruption that radiotherapy induces in bone, which does not necessarily inhibit osseointegration, medications have only mild effects. However, the long-term effect of medications on implants have not been investigated and, if the suggestion of synergistic effects of risk
factors (Mombelli and Cionca (2006)) is valid, further study of this area is merited.

d) Genetics

The publication of the Minnesota Twin Periodontal Study in 1994 established the importance of genetics in susceptibility to periodontal disease (Michalowicz (1994)). Michalowicz and colleagues reviewed the evidence for a genetic component of periodontal disease and concluded that approximately half the variance in periodontal disease in a population may be attributed to genetic variance (Michalowicz et al., 2000).

The similarity between periodontitis and peri-implantitis has led to a number of studies assessing the genetic component of peri-implant pathology. Knowledge of the human genome and advances in amplification of sections of DNA, such as polymerase chain reaction (PCR), has enabled assessment of the genetics of individuals’ inflammatory mediators. Polymorphism of the interleukin 1 gene has been associated with peri-implantitis when combined with smoking, which is a recognised risk factor (Feloutzis et al., 2003, Grucia et al., 2004). The importance of the genetics of both this and other inflammatory mediators and growth factors is not fully understood.
### 1.4 Characteristics of Implant Success and Failure

Table 2. Results from major studies for fixed implant supported prostheses in edentulous patients:

<table>
<thead>
<tr>
<th>Author/year</th>
<th>Study type</th>
<th>Total Implants</th>
<th>Observation Period (y)</th>
<th>Md I</th>
<th>Md L</th>
<th>Md S (%)</th>
<th>Mx I</th>
<th>Mx L</th>
<th>Mx S (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adell et al, 1981</td>
<td>R</td>
<td>2768</td>
<td>3-8</td>
<td>423</td>
<td>38</td>
<td>91</td>
<td>472</td>
<td>89</td>
<td>82</td>
</tr>
<tr>
<td>Albrektsson, 1988</td>
<td>MC</td>
<td>3643</td>
<td>3</td>
<td>1029</td>
<td>41</td>
<td>96</td>
<td>164</td>
<td>18</td>
<td>89</td>
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<td></td>
<td></td>
<td>5</td>
<td>29</td>
<td>14</td>
<td>93</td>
<td>11</td>
<td>0</td>
<td>100</td>
</tr>
<tr>
<td>Adell et al 1990</td>
<td>MC</td>
<td>4636</td>
<td>5</td>
<td>480</td>
<td>43</td>
<td>91</td>
<td>524</td>
<td>84</td>
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<td>67</td>
<td>86</td>
<td>86</td>
<td>115</td>
<td>115</td>
<td>78</td>
</tr>
<tr>
<td>Brånemark et al 1995</td>
<td>R</td>
<td>882</td>
<td>10</td>
<td>406</td>
<td>30</td>
<td>93</td>
<td>476</td>
<td>102</td>
<td>79</td>
</tr>
</tbody>
</table>

Table 3. Results from major studies for removable full arch prostheses in edentulous patients:

<table>
<thead>
<tr>
<th>Author/year</th>
<th>Study type</th>
<th>Total Implants</th>
<th>Observation Period (y)</th>
<th>Md I</th>
<th>Md L</th>
<th>Md S (%)</th>
<th>Mx I</th>
<th>Mx L</th>
<th>Mx S (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Engquist et al, 1988</td>
<td>RMC</td>
<td>339</td>
<td>0-3</td>
<td>148</td>
<td>9</td>
<td>94</td>
<td>191</td>
<td>58</td>
<td>70</td>
</tr>
<tr>
<td>Jemt et al, 1996</td>
<td>PMC</td>
<td>510</td>
<td>0-5</td>
<td>393</td>
<td>14</td>
<td>96</td>
<td>117</td>
<td>30</td>
<td>74</td>
</tr>
<tr>
<td>Eklund et al, 1997</td>
<td>R</td>
<td>221</td>
<td>0-8</td>
<td>43</td>
<td>13</td>
<td>70</td>
<td>178</td>
<td>69</td>
<td>61</td>
</tr>
<tr>
<td>Bergendahl and Engquist, 1998</td>
<td>P</td>
<td>122</td>
<td>3-10</td>
<td>68</td>
<td>1</td>
<td>98</td>
<td>54</td>
<td>16</td>
<td>70</td>
</tr>
</tbody>
</table>

Key:
- Study type: P = prospective, R = retrospective, MC = multi-centre, RMC = retrospective multi-centre, PMC = prospective multi-centre
- I = implants, L = implants lost, S = reported success rate
- Md = mandibular, Mx = maxillary
The following observations may be made from these studies:

- Implant treatments appear to have a relatively high success rate
- Implant treatment in the maxilla is less successful than in the mandible
- Implant supported overdentures in the maxilla appear to have a significantly worse prognosis.

Each of these observations bears further comment. The low failure rates reported require that large studies are necessary to show statistically significant associations with potential contributing factors (Listgarten (1997)). The apparently greater vulnerability of implants in the maxilla suggests that there is a higher chance of finding correlations between risk factors and failure when studying implants placed there.

Considerable variation in bone quality and quantity has been observed between individuals, with age variation, between the jaws, and at different sites within the same jaw. In general, the mandible has a thicker cortical plate and denser bone trabeculae than the maxilla, with both jaws having a less dense trabecular bone structure posteriorly. The consensus is that mandibular flexion from function and parafunction helps to maintain the bone in the anterior region, which forms the basis of most mandibular implant reconstructions. In contrast, it is not uncommon for there to be severe resorption of the anterior maxilla. Lekholm and Zarb (1985) classified bone quality on a 1 to 4 scale and bone quantity from A to E.
Fig. 1A. Morphological parameters of bone quality according to Lekholm and Zarb (1985) Type 1: mostly homogenous cortical bone, Type 2: a thick layer of cortical bone surrounding dense trabecular bone, Type 3: a thin layer of cortical bone surrounding dense trabecular bone, and Type 4: a thin layer of cortical bone surrounding trabecular bone of low density.

Fig. 1B. Classification of alveolar bone with regard to quantitative criteria. While class A bone shows no signs of atrophy, class C and D in the maxilla, and class B and C in the mandible have reduced alveolar bone volume that is unfavorable to implant placement.

Figure 8. Reproduced from Watzek and Gruber (2002)

This classification is simple and the assignment to categories arbitrary; however, the classification has gained widespread acceptance and provides a useful guide. Poor bone quality and severe resorption (categories D and E) have been associated with poor implant outcomes (Kronström et al., 2001, Esposito et al., 1998a, Engquist et al., 1998, Bryant (1998)). Higher failure rates have been attributed to low bone-to-implant contact and hence primary stability following surgery. The hypothesis that the lower mineral density leads to more rapid progression of osteoclastic resorption from overload or inflammation has also been suggested (Watzek and Gruber (2002)).
In studies on maxillary implant reconstructions, for example Ekfeldt et al (1997), a fixed prosthesis is the treatment of choice. The default treatment of an overdenture may be preferred in one or more of the following scenarios:

- when too few implants integrate,
- when bone volume precludes placement of sufficient implants,
- too severe tissue loss and/or compromised maxillo-mandibular relationship.

As loss of bone density often accompanies a loss of bone volume, this results in a preference for overdentures where implant numbers and their lengths are compromised. Force distribution between rigid implants and the resilient mucosa, when insufficient implants are available to fully support the overdenture may be a cause of problems. This is predictive of higher failure given that an overdenture will be subject to the same forces as a fixed prosthesis (Zitzmann and Marinello (2000b)). A study of maxillary prostheses in patients with different degrees of resorption concluded that for matched bone quality and quantity the implant and prosthetic success rates for fixed and removable prostheses were similar (Jemt and Lekholm (1995)). Correspondingly, the evidence suggests that it is not necessarily short implants but the clinical situations that preclude longer implants that are associated with higher failure rates (Renouard and Nisand (2006)).

Implant failures can be divided into early and late categories. Implants lost within the first year after Stage II surgery have been regarded as failures of
osseointegration (Listgarten (1997)). From a systematic review of the incidence of complications in prospective longitudinal studies of at least 5 years duration, Berglundh and colleagues (2002) completed a meta-analysis from their included studies; implant loss before function could be expected to occur in 2.5% of implants placed, with a following loss rate of 2-3%. Weyant and Burt (1993) support the finding that the rate of implant loss decreases rapidly after the second year.

Failures of osseointegration may result from a number of causes (Esposito et al., 1998b):

- Poor surgical technique
  - trauma from bone overheating (greater than 47°C for 1 minute),
  - misshapen or oversized osteotomy, resulting in poor initial implant stability,
  - bacterial contamination,
  - operator inexperience leading to one or more of the above.
- Compromised healing as a result of local or systemic factors
- Premature or inadvertent loading creating excessive micro-motion during the healing phase

Fracture of implants is a rare complication, occurring in 0.08 to 0.74% of implants placed (Berglundh et al., 2002) and is more likely to occur after loss of supporting bone.
1.4.1 Success criteria

Esposito et al., (1998a) suggested that to be considered successful an implant must fulfill the following criteria:

1) Functional success – ability to chew
2) Tissue health – presence and maintenance of osseointegration
3) Absence of pain and other pathological processes
4) User satisfaction – aesthetics and absence of discomfort.

To be considered less than successful, an implant must have: a) fallen below some performance characteristic that can be measured, or b) that it is not possible to assess the implant adequately. Albrektsson and Zarb (1993) suggested that implants should be evaluated in four categories:

1) Successful
2) Surviving
3) Unaccounted for
4) Failed

Albrektsson and Zarb (1993) suggest that for an implant to be in the successful category, it should be assessed for mobility and show radiographic evidence that marginal bone loss falls within the criteria set out by Albrektsson et al. in 1986. The authors suggest that bone loss should be less than 1.5mm in the first year and less than 0.2mm per annum thereafter. Also
acknowledged were the problems with bone height measurements from radiographs with likely over- and under-estimations, in addition to the two-dimensional nature of the assessment. In reviewing the evidence on radiographic assessment, Esposito et al. (1998) concluded that bone loss measurements below 0.2mm were very difficult to achieve. Despite limitations, the criteria set out by Albrektsson et al. in 1986 are widely accepted. This is supported by the average values recorded over years which may be used to estimate the rate of bone loss and which may be useful for individual implants. Radiographic evidence of bone loss has been shown to correlate well with histometric measurements (Hermann et al., 2001).

The requirements of measurement for accurate reporting and fair representation in clinical studies differ from those in clinical practice. In their consensus statement for the Third International Team for Implantology (ITI), Lang et al. (2004) stated that radiographs should be used to give a baseline measurement to a known landmark for comparison with subsequent radiographs. Similarly, significant change rather than absolute values have been proposed with resonance frequency analysis (RFA), which is a relatively new modality for testing the bone to implant interface (Meredith et al., 1996, Aparicio et al., 2006).

From a research viewpoint, averaging bone levels for numerous implants may give a misleading impression (Esposito et al., 1998). Marginal bone loss has been poorly reported in the literature. In a review of all available studies in English from 1981 to 1997, Goodacre et al. (1999) were only able to identify 3 studies reporting marginal bone loss.
The acceptance that progressive marginal bone loss is a negative prognostic indicator for implants has prompted the search to understand the possible aetiology. The principal explanations are overloading of the implant, inflammatory bone loss or a combination of both of these factors (Van Steenberghe et al., 1999, Esposito et al., 1998b, Isidor (2006)). These principal causes of bone loss may be subject to many modifying influences, the understanding of which is of benefit in treatment planning for the individual patient.

There is a lack of consistency in reporting on implant outcomes, with differing data collection methods and presentation and analysis of results, which could lead to a lack of clarity in establishing success rates. Presented results tend to focus on implant and prosthesis survival and are often presented as mean values. Similarly, success rates for individual patients are not explored which may disguise the fact that individual patients suffer above average complications or losses. Clustering of implant failures within individual patients suggests that patient-specific factors are important in the outcome of implant therapy (Weyant and Burt (1993)).

Marginal bone loss is accepted to be a pathological sign leading to implant failure (Esposito et al., 1998). A recent paper by Fransson et al. (2005) reviewed the radiographs of 662 patients who had fixed prostheses on a total of 3413 Brånemark implants for between 5 and 20 years. The authors found that of the 28% of patients with progressive bone loss at one implant, 33% had progressive bone loss at three or more implants. They concluded that
there is a higher incidence of bone loss detected with subject-based data rather than implant-based data.

In the averaging of bone loss values amongst a study group, failing implants in individual patients may not be identified if the average value meets the study's success criteria. As a result, such studies do not help in elucidating patient-specific factors which result in unsuccessful implant treatment for an individual patient and which provides information that would be beneficial in selecting the optimum treatment plan.

Additionally, successful implant rehabilitation requires that patients adapt functionally and psychosocially to their prosthesis and not simply that they remain in-situ. Consensus reports from the European Workshops on Periodontology (Albrektsson and Isidor (1994), Lang et al., 2002) have called for patient-based outcomes to be part of the evaluation of implant therapy.

1.5 Oral Health-Related Quality of Life

The optimum treatment for replacing missing natural teeth is sought by patients and prosthodontists. Patients seek satisfactory comfort, function and aesthetics which are subjective parameters that are difficult for a clinician to assess.

Slade and Spencer introduced the Oral Health Impact Profile (OHIP) in 1994. This 49-item questionnaire provides a validated assessment of the impact of
oral health on quality of life from a general and non-specific clinical viewpoint. The OHIP is the most frequently used tool for assessing OHRQL (Strassburger et al., 2006), however, it is less than ideal for assessing implant specific treatment modalities.

Pjetursson et al. (2005) used a questionnaire of 13 qualified statements concerning aspects of function and chewing comfort, phonetics, aesthetics, oral hygiene practices, general satisfaction and cost. The statements were evaluated using a six-grade categorisation from 'yes definitely' to 'definitely not' and confirmed using a visual analogue scale (VAS). The researchers found greater than 90% satisfaction on all parameters with implant therapy and good correlation between the qualified statements and VAS.

Options for implant-based prostheses in the edentulous maxilla comprise either overdentures (with or without palatal coverage) or fixed bridges. Two randomised controlled trials to assess patients’ perspective on these treatments have been reported. De Albuquerque et al. (2000) evaluated general satisfaction, physical function and psychosocial function for 16 patients, who initially had new conventional complete dentures. Thirteen patients progressed to the second part of the study, which involved fabrication of a mandibular implant-supported bridge, and a long-bar-overdenture on four implants in the maxilla. Group 1 (n=7) initially wore an overdenture with a palate and Group 2 (n=6) one without. After two months, the prosthetic designs were reversed. No difference was found between dentures with and without a palate and interestingly the ratings of the implant overdentures were not significantly higher than for the new complete dentures. The authors
suggest that for patients with good support for a conventional prosthesis, implant overdentures in the maxilla should not be the treatment of choice.

Heydecke et al. (2003) performed a similarly designed study comparing a palateless overdenture on a long-bar with fixed bridges. The overdenture resulted in statistically significantly better ratings for speech, ease of cleansing and general satisfaction, with 9 of the 13 patients choosing to keep the overdenture. Surprisingly the fixed bridge received lower ratings both for stability and for chewing test foods.

In contrast, a study by Zitzmann and Marinello (2000a) also comparing overdentures with fixed bridges found both groups similarly satisfied, with slightly higher ratings for speech with overdentures. The treatment plans were successfully completed, such that patients expecting fixed bridges received fixed bridges and vice-versa. Ekfeldt et al. (1997) reported on overdentures that were provided as a result of implant failures (“rescue” group) and planned overdentures (“planned” group). Patients from the rescue group reported lower levels of satisfaction, which the authors attributed to disappointment with not receiving their treatment of choice.

Karabuda et al. (2002) reported on patient satisfaction with two different retentive elements for mandibular overdentures, finding no difference between ball attachments and bar and clips. Information is required regarding resilient attachments, which are sometimes indicated in compromised implant situations.
1.6 Summary of Introduction

Rehabilitation of the edentulous jaws with osseointegrated implants is a well-documented treatment modality with predictably high success rates overall. Whilst risk factors have been identified, understanding the aetiology of clusters of failures in individual patients is still poor, denying the clinician the ability to predict such patients pre-operatively. Further research both from patient and implant perspectives is required.

Oral Health-Related Quality of Life is an important outcome measure of implant rehabilitation. Generally, high levels of satisfaction with this treatment have been reported and the correlation with treatment in accordance with the patients' wishes identified. In certain cases the compromise of using a resilient attachment system will be necessary. There is a lack of data on whether the use of resilient attachments significantly compromises OHRQL. Such information may be useful to assist in establishing realistic patient expectations.

1.7 Aims

To provide additional information to guide treatment planning by analysing the effect of clinical and patient variables on peri-implant bone levels, implant success, and patients' perception of their treatment.
1.8 Hypothesis

Patient factors have greater influence on the success of implant treatment than clinical factors. Factors that will increase peri-implant bone loss are systemic disease, smoking (both before and after implant surgery), poor mucosal health, poor oral hygiene, shorter implants, prosthesis type - overdentures greater than fixed bridges, and implant supported opposing dentitions rather than natural teeth. Age and gender do not affect peri-implant bone loss.

Resilient attachment of maxillary implant overdentures compromises patients' oral health-related quality of life.
Method

2.1 Patients

The patient group for this retrospective study comprised all 36 patients who had implants placed in their edentulous maxillae between 1986 and 1995 at the Sydney Dental Hospital, Sydney, Australia. Patients with maxillary implant reconstructions were selected to maximise the chances of identifying influencing factors, due to the reported higher failure rate in the maxilla. There were 20 males and 15 females with ages ranging from 32 to 73 at implant placement (Figure 1). All had been referred to the Prosthodontic Department for consideration of implant treatment options, to address their difficulty in managing a conventional complete denture. The subjects of this study were those for whom optimised conventional dentures were unsuccessful and who were subsequently restored with full-arch prostheses, supported either entirely by implants or by a combination of implants and mucosa.

![Bar chart showing age and gender distribution](image-url)

Figure 9. Age and gender distribution
2.2 Implant Treatment

From the clinical records and discussion with the treating prosthodontist, the following information regarding the implant treatment was obtained:

- Prosthodontic assessment was made by clinical examination and followed by provision of a maxillary denture, optimised for each patient. Radiographic assessment was by an orthopantomogram. For those patients with persistent adaptation difficulties, the clinical preparation continued with a CT scan of the maxilla incorporating a radiographic guide fabricated from a duplicate of the new maxillary denture. Implant position was determined from this data in acknowledgement of the tooth arrangement.

- Surgical assessments were made and optimal implant lengths determined. Bone volume to accommodate implants at least 7mm long and 3.75mm wide was required. The longest implants that could be placed at each site were planned, such that implant length is an indicator of available bone volume. Onlay grafting was required for 2 cases and was performed as a separate procedure by harvesting iliac crest bone. Local grafting was required at 4 implant sites for 2 patients. Exclusion criteria for the patient group were unsuitability to undergo the surgical procedures or unreasonable treatment expectations.

One hundred and seventy four Brånemark Mk I machined surface implants were placed by 3 oral surgeons following the accepted standardised surgical protocol (Adell et al., 1981). This involved the gentle preparation of the host bone sites with copious sterile saline irrigation, guided by a surgical template and placement of the implants under torque to achieve primary stability.
Implants were between 7 and 20mm in length (see table 1 for distribution). The implants were surgically uncovered following a healing time of between 6 to 8 months and temporised with healing abutments, before attachment of a definitive screw-retained trans-mucosal abutment. Further healing of between 2 to 4 weeks was observed before beginning prosthodontic procedures.

Table 4. Implant length distribution

<table>
<thead>
<tr>
<th>Implant length (mm)</th>
<th>7</th>
<th>10</th>
<th>13</th>
<th>15</th>
<th>18</th>
<th>20</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number placed</td>
<td>24</td>
<td>57</td>
<td>47</td>
<td>31</td>
<td>11</td>
<td>4</td>
<td>174</td>
</tr>
<tr>
<td>Early loss, up to 1 yr</td>
<td>3</td>
<td>9</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>17</td>
</tr>
<tr>
<td>Late loss, after 1 yr</td>
<td>5</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>10</td>
</tr>
<tr>
<td>Total losses</td>
<td>8</td>
<td>12</td>
<td>4</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>29</td>
</tr>
<tr>
<td>Failure %</td>
<td>33.3</td>
<td>21</td>
<td>8.5</td>
<td>6.5</td>
<td>9</td>
<td>0</td>
<td>16.6</td>
</tr>
</tbody>
</table>

The final prostheses were either overdentures (30) on bars retained by clips or attachments, or fixed bridges (5). All were provided by the same prosthodontist. Bars and clips were obtained from Cendres et Metaux (Biel-Bienne, Switzerland), and attachments from Ceka (Antwerp, Belgium). In 10 cases where peri-implant bone loss was noted at review, the overdenture attachment was modified by using a resilient material - Molloplast B (Buffalo Dental Manufacturing Co, Syosset, NY), with the intention that this would increase the distribution of occlusal forces and improve stability and comfort.

2.3 Patient Reviews and Examination

Attempts were made to contact all patients and invite them to attend a clinical and radiographic review (by the author). Of the 36 recorded patients, 23 were
reviewed over 7 months. Fifteen of the 23 patients were continuing to attend the Sydney Dental Hospital clinic, eight were attending private clinics, two had moved interstate, five had lost all implants, four were deceased, and one declined. Permission to review the patients was sought from all treating clinicians and reports written.

2.3.1 Medical Status

Medical histories were obtained, including dosage and duration of all medications. The patients’ health status in relation to prior clinical records was confirmed. Medical status was analysed as follows:

| 1. No recorded systemic illness requiring medication | 0 |
| 2. Systemic illness requiring regular prescription medication | 1 |

2.3.2 Smoking History Since Stage II Surgery

Smoking was categorised under the number of cigarette years (cy); see Deluca et al. (2006) since stage II surgery. Cigarette years were calculated as a product of the number of cigarettes per day and the number of years. Smoking histories were analysed as follows:

| 1. Zero years smoking with implants (non-smoker) | 0 |
| 2. History of light smoking with implants (< 50cy) | 1 |
| 3. History of heavy smoking with implants (> 200cy) | 2 |
2.3.3 Smoking History Before Surgery

This was assessed as the number of cigarette years prior to surgery and scored as follows:

| 1. Never smoked regularly or < 50 cy | 0 |
| 2. Smoking history > 50 cy | 1 |

Clinical examination involved recording of the current prosthesis type, opposing dentition, oral hygiene status and gingival index;

2.3.4 Current Prosthesis Type

This was analysed as follows:

| 1. One or two bars supporting an overdenture with a resilient attachment | 0 |
| 2. Bar supporting an overdenture with rigid attachments | 1 |
| 3. Fixed bridge or fully implant supported overdenture | 2 |

This groups the prostheses into combined implant and mucosal support, with rigid or resilient attachments and those utilising solely implant support. These groups were selected based on possible variations in transfer of occlusal force with each design. Details of the opposing dentition were recorded from the clinical records and radiographic assessment.
2.3.5 Opposing Dentition

This was analysed as follows:

| Natural teeth or natural teeth and removable partial denture with bounded saddle (Kennedy Class III) | 0 |
| Natural teeth and bilateral distal extension removable partial denture (Kennedy Class I) | 1 |
| Natural teeth and distal segment implant bridge | 2 |
| Fully implant supported bridge/Implant overdenture | 3 |

These groups were selected to differentiate the opposing occlusal loading, with support from periodontal ligaments only, periodontal ligaments and mucosa, periodontal ligament and implants and predominantly implants. All subjects presented bilateral mandibular contacts in the posterior segments.

2.3.6 Oral Hygiene Status

Oral hygiene status was recorded following a three point scale described by Lindquist et al. (1988):

| No visible plaque | 1 |
| Local plaque accumulation | 2 |
| General plaque accumulation greater than 25% | 3 |

Consideration was given to recording the oral hygiene status of the opposing dentition. Preliminary data indicated the opposing dentition only involved natural teeth in 19 cases and that the oral hygiene index indicated scores that were similar to those around the maxillary implants. As a result, further oral hygiene data relating to the opposing dentition were not collected.
2.3.7 Modified Gingival Index

The condition of the implant marginal mucosa was assessed according to the modified gingival index (GI) described by Mombelli et al (1987):

<table>
<thead>
<tr>
<th>Condition</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>No bleeding when a periodontal probe is passed</td>
<td>0</td>
</tr>
<tr>
<td>along the mucosal margin adjacent to the implant</td>
<td></td>
</tr>
<tr>
<td>Isolated bleeding spots visible</td>
<td>1</td>
</tr>
<tr>
<td>Blood forms a confluent red line on the mucosal margin</td>
<td>2</td>
</tr>
<tr>
<td>Heavy or profuse bleeding</td>
<td>3</td>
</tr>
</tbody>
</table>

The reasons for tooth loss were not clear from the clinical records, and patient recall was poor. As a result it was not possible to establish whether periodontal disease had been a factor in tooth loss and further, whether there was any association with peri-implant bone loss.

2.4 Radiographs

The radiographic assessment protocol involved recording from available radiographs at baseline (time related to second stage surgery), and at differing intervals thereafter. Time since second stage surgery was calculated in months to identify differences in time between radiographs for each patient. A combination of orthopantomogram and peri-apical radiographs was used. Almost all orthopantomograms were taken on programme 11 of a Siemens Orthophos Plus (Sirona, Bensheim, Germany) machine that has been in use at the Sydney Dental Hospital for over fifteen years. Programme 11 gives a constant 25 percent vertical magnification and is designed for implant assessment. The difficulties both in obtaining reliably clear orthopantographic images of the anterior maxilla and the disadvantage of non-standardised peri-apical radiographs are acknowledged. Whenever possible, peri-apical
radiographs were used. When orthopantomograms were unclear this was noted and additional data from chronologically similar radiographs sought. The magnification factor was not an issue as the relative position of the bone to the implant threads was assessed.

The form of a Brånemark Mk I implant and an attached standard abutment is illustrated below in Figure 2.

Figure 10. The implant is placed such that the implant/abutment junction is at the level of the bone crest. Both this and the threads provide radiographic landmarks.

No record of categorisation of bone quality or quantity was found in the clinical records. Bone density was not assessed from the computerised tomograms as these early films provided only bone density measurements (in Houndsfeld units) for single points and the resultant readings showed an unreasonable amount of scatter (from zero in cortical bone to one thousand in cancellous bone).
2.4.1 Radiographic Measurement

The mesial and distal bone levels were assessed by a single operator from radiographs, with a light box using initial visual assessment and then 8x magnification. The most coronal bone levels were assessed to the nearest half thread, with rounding up to the next half thread where appropriate. For this, a bone level apical to the inter-thread space would be scored to the next most apical thread and a level apical to a thread would be scored to the next inter-thread space. The mesial and distal measurements were averaged.

![Typical peri-apical radiograph](image)

Bone level apical to thread 4, coronal to inter-thread space. Score 4.5

Figure 11. Example radiograph depicting assessment of bone loss.

Due to the difficulty in reading some radiographs and the possibility of operator fatigue, the reliability of the measurements was assessed. This was achieved by re-assessing every 5th radiograph selected after a time interval of greater than two weeks. The time interval was chosen to reduce the possibility of operator recall biasing the result. This method confirmed reader reliability.
Discrepancies were dealt with using the following protocols:

1) Where differences of half a thread (0.3mm) were noted the greater value was recorded.

2) Where differences were greater than half a thread a further separate assessment was made. If this was within half a thread of one of the previous scoring, the greater value was scored. If the difference between the third scoring was greater than half a thread from both the previous assessments, the implant site reading or that radiograph would have been considered too unreliable and discarded. No occurrences of this discrepancy were identified.

2.4.2 Bone Loss Calculations

The selection of a radiograph at one year following stage two surgery, would provide a bone level reading after initial bone healing and remodelling, as described by Adell et al. (1981). The ensuing loss of bone over time could then be calculated from this baseline, using the knowledge that the Brånemark implant thread pitch measures 0.6mm (+/- 0.005mm), as illustrated in Figure 2. Due to the variation in the timing of radiographic assessment, it was apparent that data would be lost should the first radiograph after Stage II surgery be used as a baseline. In the worst case this was 83 months. As Stage II surgery was a common factor between all subjects, this time was taken as the baseline from which all radiographic calculations were determined.

The present study was designed to calculate bone loss around implants after initial stabilisation and to relate the data to actual bone loss - adjustments
were made for the first year bone loss. Adell et al (1981) reported on 981 implants placed in the maxilla, divided into 3 time intervals with all implants in place in excess of one year. The reported healing and remodelling mean bone loss was 1.3mm, 1.5mm and 1.3mm. As the first thread represents 1.5mm bone loss from the implant shoulder, adjustments were made by subtracting 1.5mm, (as a criterion for success - Albrektsson et al., 1986), from the total bone loss over time and subsequently subtracting 12 months from the total number of months since the baseline date of Stage II surgery.

It was acknowledged that due to the pitch of the implant thread and the unknown rotational position of the implant, some inaccuracy in measurements may have occurred. However, due to the number of radiographs taken over the extensive time period from Stage II surgery to final data (minimum 11 years for assessed patients), it was considered unlikely that this would effect any significant changes in bone loss that may have occurred.

For failed implants (all of which were subsequently removed), the time elapsed before removal was calculated from the date of initial surgical placement of the implant. Failures were divided as follows:

<table>
<thead>
<tr>
<th>Implant lost before or within first year of loading</th>
<th>0</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subsequent losses</td>
<td>1</td>
</tr>
<tr>
<td>Iatrogenic failures (unusable due to positioning)</td>
<td>2</td>
</tr>
</tbody>
</table>

### 2.5 Patient Satisfaction Questionnaire (PSQ)

The PSQ was adapted from Pjeturrson et al. (2005) and is included in the Appendix. Each patient’s perception of their implant treatment was assessed by evaluation of 12 statements concerning aspects of function and chewing.
comfort, phonetics, aesthetics, oral hygiene practices, general satisfaction and cost (Table 5). The statements were evaluated using a four-point Likert scale, that is, ‘Yes, definitely’, ‘Yes, possibly’, ‘No’, ‘I don’t know’. When comparing implants with natural teeth in questions 6, 7, and 8, a three-grade categorisation was used, that is, ‘teeth’, ‘the same’ and ‘implants’. Additionally, the patients were asked to mark the respective visual analogue scale (VAS - a 100mm straight line with the left end indicating ‘no satisfaction’ and the right end ‘total satisfaction). When comparing implants with teeth, the left end indicated ‘teeth’, the middle 20mm ‘the same’ and the right end ‘implants’.

Table 5. Patient Satisfaction Questionnaire statements

1. ‘My denture/bridge functions very well, and I can chew on it very well’
2. ‘I feel more secure biting firmly on my denture/bridge now that it is secured to implants’
3. ‘I can speak well with my denture/bridge’
4. ‘I am please with how my teeth look’
5. ‘I cannot clean my implant very well’
6. ‘It is easier for me to clean my implants than to clean my natural teeth’
7. ‘I need more time to clean my implants/bridge that it took to clean my teeth’
8. ‘The tissues around the implants bleed more than they did around my teeth’
9. ‘With my treatment I got exactly what I expected’
10. ‘I would go through such treatment again, if needed’
11. ‘I would recommend this treatment to a relative or friend, if needed’
12. ‘I found the cost of the treatment was justified’

The PSQ was given to the patients at the start of the appointment, with an example question on the cover page to permit explanation. Confidentiality and
the absence of bearing on future treatment were assured. Patients were left uninterrupted to complete the questionnaires, which were marked only with the subject number. In two cases where the patients did not attend for review, the questionnaires were posted (with return stamped addressed envelopes), after a telephone call to establish willingness to participate.

2.6 Data Analysis

Patients were assigned a number to assure their anonymity during data analysis. Patient data varied in the quantity per variable due to missing data which is a feature of retrospective studies. A low number of data points and the actuality that most of the data collected was categorical, restricted the type of data analysis that could be applied.

Statistical analyses to determine associations between bone loss and patient or clinical characteristics of implant treatment, and between these characteristics, were mostly correlations. Calculated bone loss, as described above, was used as the dependent variable in univariate and one-way analysis of variance (ANOVA) to determine differences between groups in relation to smoking variables and prosthetic type.

The PSQ was scored according to the analysis described by Pjetursson et al. (2005) analyses to allow for comparisons between studies. Percentages were calculated for each four-point statement and, mean (standard deviation) and median were calculated for each visual analogue scale. Correlational analysis was undertaken to determine associations.
3.1 Study Population

Of the thirty-six patients who received implant-based rehabilitation of their edentulous maxillae, thirty patients had radiographic data for seven or more years, and details of the opposing dentition and prosthesis type. Twenty-three of these thirty patients were available for clinical assessment by the author and twenty-two patients completed the Patient Satisfaction Questionnaire (PSQ). The loss to follow-up of patients is discussed in the next section. A summary of the study characteristics is presented in Table 6:

Table 6. Characteristics of Study Population

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total number of patients with maxillary dental implant rehabilitation at SDH from 1987 to 2006</td>
<td>36 (100%)</td>
</tr>
<tr>
<td>Number of patients with 7 or more years of radiographic data</td>
<td>30 (83%)</td>
</tr>
<tr>
<td>Number of patients assessed clinically</td>
<td>23 (64%)</td>
</tr>
<tr>
<td>Number of patients with complete data - clinical examination, radiographs and PSQ</td>
<td>22 (61%)</td>
</tr>
<tr>
<td>Number of patients with total implant loss</td>
<td>4 (11%)</td>
</tr>
<tr>
<td>Number of patients lost to follow-up</td>
<td>4 (11%)</td>
</tr>
<tr>
<td>Number of patients deceased</td>
<td>4 (11%)</td>
</tr>
<tr>
<td>Number of patients declining assessment</td>
<td>1 (3%)</td>
</tr>
<tr>
<td>Years to assessment from Stage 2 surgery for patients with complete data</td>
<td>11-18</td>
</tr>
<tr>
<td>Age-range at placement</td>
<td>32-73</td>
</tr>
<tr>
<td>Age-range at examination</td>
<td>46-91</td>
</tr>
<tr>
<td>Number of males</td>
<td>20</td>
</tr>
<tr>
<td>Number of females</td>
<td>15</td>
</tr>
</tbody>
</table>

SDH – Sydney Dental Hospital

3.2 Implants Placed

One hundred and seventy-four implants were placed in the thirty-six patients. Radiographic data for a minimum of seven years was available for one
hundred and fifty five implants. At the time of assessment, thirty implants had
failed, thirty-three were lost to follow-up, resulting in one hundred and eight
implants available for clinical assessment.

A summary of this parameter is presented in Table 7:

<table>
<thead>
<tr>
<th>Table 7. Summary of implant group.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total number of implants</td>
</tr>
<tr>
<td>Number of implants with 7 or more years of available radiographic data</td>
</tr>
<tr>
<td>Number of implants assessed clinically</td>
</tr>
<tr>
<td>Number of failed implants</td>
</tr>
<tr>
<td>Number of implants in the four patients with total implant loss ('clustered failures')</td>
</tr>
<tr>
<td>Number of implant lost within 1 year of loading</td>
</tr>
<tr>
<td>Number of implants lost after 1 year loading</td>
</tr>
<tr>
<td>Number of implants fractured</td>
</tr>
<tr>
<td>Number of non-functional implants (buried)</td>
</tr>
<tr>
<td>Number of implants lost to follow-up</td>
</tr>
</tbody>
</table>

For distribution of implant lengths/failures, refer to Table 8.

The following results have been reported in two sections:

a) Variables which were recorded on a patient-specific basis - medical
status, concurrent smoking and smoking history, prosthesis type,
opposing occlusion, age and gender, were analysed for each patient.

b) Variables which were recorded for each implant - plaque scores,
modified gingival index.

3.3 Patient Related Analyses

3.3.1 Complete implant failure cases

Four patients lost implants (13) within the first two years of loading. Six of
these implants were lost within one year of stage II surgery, whilst a further
seven implants were lost within the following year. A summary of these findings is shown in Table 8:

Table 8. Summary of cases with complete implant failure

<table>
<thead>
<tr>
<th>Patient</th>
<th>Implant length (mm)</th>
<th>Comments (LL – late loss; EL – early loss; EAS – threads exposed at surgery)</th>
</tr>
</thead>
<tbody>
<tr>
<td>8</td>
<td>7</td>
<td>LL</td>
</tr>
<tr>
<td></td>
<td>7</td>
<td>LL</td>
</tr>
<tr>
<td></td>
<td></td>
<td>bruxism noted</td>
</tr>
<tr>
<td>11</td>
<td>13</td>
<td>LL</td>
</tr>
<tr>
<td></td>
<td>7</td>
<td>LL</td>
</tr>
<tr>
<td></td>
<td>13</td>
<td>EL</td>
</tr>
<tr>
<td></td>
<td></td>
<td>combination syndrome (noted below)</td>
</tr>
<tr>
<td>29</td>
<td>10</td>
<td>LL</td>
</tr>
<tr>
<td></td>
<td>7</td>
<td>LL</td>
</tr>
<tr>
<td></td>
<td>7</td>
<td>EL</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>LL</td>
</tr>
<tr>
<td>33</td>
<td>10</td>
<td>5 threads EAS – EL</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>1 thread EAS - EL</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>4 threads EAS - EL</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>1 thread EAS – EL (all lost before prosthetic reconstruction)</td>
</tr>
</tbody>
</table>

Records for two of the patients indicated clinical signs of possible contributing factors in these complete implant-loss patients. In the case of patient 8, a clinical diagnosis of bruxism had been made; whilst in patient 11, the presence of ‘combination syndrome’ had been recorded pre-operatively. ‘Combination syndrome’ was originally described by Kelly (1972) and based on a group of six patients with maxillary complete dentures opposing mandibular anterior teeth. The characteristics were a reduction in maxillary anterior bone and an enlargement of the maxillary tuberosities (see page 86).

Another patient’s surgical records indicated that the implants were not placed with the implant collar at the level of the bone crest and threads were exposed at surgery - patient 33 in Table 8.
The clinical records did not permit further assessment such as smoking status for example.

3.4 Characteristics of Bone Loss

The average bone loss per implant was calculated for the one hundred and fifty-five implants with radiographic data, after adjustment for bone loss in the first year. Four groups were created with bone loss values above and below the 0.2mm per annum level suggested as a criterion for success (Albrektsson and Zarb, (1993)). The analysis showed that no bone loss (36.81%) and bone loss less than 0.1mm (34.72%) accounted for the majority of the implants assessed. The percentage of implants within each grouping is indicated in Figure 12:

![Average Bone Loss Per Annum](image)

Figure 12. Absolute bone loss per annum as a percentage of 155 implants assessed radiographically, after adjustment for year-one bone loss
In general, bone loss was greatest during the first year of function and was followed by reduced bone loss in subsequent years. Bone loss in the first year was generally to the level of the first thread and although some cases had greater loss, implant stability was maintained. Examples of both of these scenarios are illustrated in Figure 13 (patient 34) and Figure 14 (patient 27).

**Patient 34**

![Graph showing bone loss over time for patient 34](image)

*Figure 13. Bone loss over time for patient 34, indicating rapid bone loss to the first or second thread in the first year and relative stability thereafter.*

**Patient 27**

![Graph showing bone loss over time for patient 27](image)

*Figure 14. Bone loss over time for patient 27, indicating rapid bone loss in the first year of up to the fourth thread, but relative stability thereafter.*
In other cases, as illustrated in Figure 15, a similar pattern of bone loss occurred in the first year of function but with continuing bone loss with some implants, and stable bone levels with other implants in the same patient. This indicates that implant-specific factors may have a role in bone loss. Graphs of bone loss over time for all other patients are located in Appendix 2.

![Patient 10 Graph](image)

Figure 15. Bone loss over time for patient 10, indicating continuing bone loss for some implants and stable bone levels for others.

3.5 Summary of Implant Outcomes

Over the duration of loading, 87.5% of the implants lost on average less than 0.2mm of bone per annum. This corresponds to 73% of the total implants that fulfilled the success criteria described by Albrektsson and Zarb (1993), of the remaining implants, those with greater bone loss but still supporting a prosthesis were given a survival status - 17%, with the remaining 10% considered as failures. The allocation of success categories according to Albrektsson and Zarb (1993), is indicated in Figure 16:
3.6 Factors Potentially Affecting Bone Loss

3.6.1 Health Status

All twenty-three patients who attended for clinical assessment were ambulatory and sufficiently healthy to allow routine dental treatment. The presence of systemic illness or not is indicated in Table 9:

<table>
<thead>
<tr>
<th>Patient health characteristics</th>
<th>No. of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients with no recorded systemic illness requiring medication</td>
<td>11</td>
</tr>
<tr>
<td>Patients with systemic illness requiring regular prescription medication</td>
<td>12</td>
</tr>
</tbody>
</table>

The medical status of two patients for whom radiographic data was available only, had been recorded by a registered nurse in the preceding year. The data
from these two patients were included with those of the clinically assessed group.

A range of systemic conditions and associated medications was recorded.

These findings are summarised in Table 10:

<table>
<thead>
<tr>
<th>Patient</th>
<th>Cardiac</th>
<th>IDDM</th>
<th>OI (4)</th>
<th>PKS</th>
<th>Sed</th>
<th>NSAID</th>
<th>Hypo-Thyroid</th>
<th>A/Dep</th>
<th>No. of meds</th>
</tr>
</thead>
<tbody>
<tr>
<td>3</td>
<td>✓</td>
<td>✓</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>5</td>
</tr>
<tr>
<td>5</td>
<td>✓</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>✓</td>
<td></td>
<td></td>
<td>4</td>
</tr>
<tr>
<td>6</td>
<td>✓</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>5</td>
</tr>
<tr>
<td>7</td>
<td>✓</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>✓</td>
<td></td>
<td></td>
<td>3</td>
</tr>
<tr>
<td>10</td>
<td>✓</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>✓</td>
<td></td>
<td></td>
<td>4</td>
</tr>
<tr>
<td>16</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>✓</td>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>25</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td></td>
<td></td>
<td>7</td>
</tr>
<tr>
<td>28</td>
<td>✓</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>29</td>
<td>✓</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>✓</td>
<td></td>
<td></td>
<td>4</td>
</tr>
<tr>
<td>32</td>
<td></td>
<td>✓</td>
<td></td>
<td>✓</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>34</td>
<td></td>
<td>✓</td>
<td></td>
<td></td>
<td>✓</td>
<td></td>
<td></td>
<td></td>
<td>4</td>
</tr>
<tr>
<td>36</td>
<td>✓</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2</td>
</tr>
</tbody>
</table>

Key: IDDM = Type 1 Diabetes (insulin dependent), OI (4) = Osteogenesis imperfecta type 4, PKS = Parkinson's disease, Sed = Sedative, NSAID = Non-steroidal anti-inflammatory drug, A/Dep = Antidepressant or anti-psychotic

For the purpose of these data, low dose aspirin (a non-steroidal anti-inflammatory drug) formulations were recorded as cardiac medications because of their anti-platelet action, dosages being substantially less than those indicated for analgaesia. The association of implants to medical status was similar as indicated in Figure 17:
Figure 17. Distribution of implant numbers in patients with and without systemic conditions.

Further subdivision of the systemic illness patient group was not made as sample size was small. No statistically significant correlation could be found between patient medical status and bone loss, n (122), r = -0.041, p > .05.

3.6.2 Smoking with Implants

The effect of low and high levels of smoking and not smoking whilst using an implant-supported prosthesis was assessed. The distribution of patients into groups by level of cigarette use is indicated in Table 11:

<table>
<thead>
<tr>
<th>Concurrent smoking and implants</th>
<th>No. of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zero years smoking with implants (non-smoker)</td>
<td>16</td>
</tr>
<tr>
<td>History of light smoking with implants (&lt; 50cy)</td>
<td>2</td>
</tr>
<tr>
<td>History of heavy smoking with implants (&gt; 200cy)</td>
<td>5</td>
</tr>
</tbody>
</table>

Smoking was categorised under the number of cigarette years (cy) that a patient had since stage II surgery. Cigarette years were calculated as a product of the number of cigarettes per day and the number of years.

The number of implants within each smoking group is indicated in Figure 17:
No significant difference in bone loss with and without smoking was found by Univariate ANOVA, $F(2, 24) = 0.535$, $p > .05$.

A weak but statistically significant correlation was found between smoking with implants and the modified gingival index score, an indicator of mucosal health, $n(108)$, $r = .256$, $p < .05$. Figure 19 below shows that no smoking is strongly related to a low index score, whilst heavy smoking correlated with heavy gingival bleeding compared with patients who did not smoke.
3.6.3 Smoking History

The possible effect on bone loss of a history of smoking before implant placement on bone loss was assessed; the distribution of patients and their smoking history is illustrated in Table 12:

Table 12. Smoking history prior to implant surgery

<table>
<thead>
<tr>
<th>Smoking prior to implant surgery</th>
<th>No. of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Never smoked regularly or &lt; 50 cy</td>
<td>11</td>
</tr>
<tr>
<td>2. Smoking history &gt; 50 cy</td>
<td>12</td>
</tr>
</tbody>
</table>

Smoking was categorised under the number of cigarette years (cy) that a patient had smoked prior to stage I surgery. Cigarette years were calculated as a product of the number of cigarettes per day and the number of years.
Figure 20. Distribution of implant numbers with and without a history of smoking prior to implant placement.

No significant association in the amount of bone loss around implants in patients with and without a history of pre-treatment smoking was found, $n$ (108), $r = .083$, $p > .05$.

A statistically significant correlation was found between patients with a history of smoking and both the modified gingival index, $n$ (108), $r = .333$, $p < .05$, and the plaque index, $n$ (108), $r = .342$, $p < .05$. Both indices show that patients with greater smoking histories tend to have less clean implants and greater peri-implant mucosal inflammation.
Figure 21. Association of Smoking History and Modified Gingival Index scores.

Figure 22. Association of Smoking History and Plaque Index

3.6.4 Type of Prostheses

The type of prosthesis being worn at the time of patient assessment was recorded. For non-attending patients with radiographic data, the prosthesis in use at the time of the last radiograph was recorded. The prosthesis type and numbers of patients are indicated in Table 13.
Table 13. Prosthesis type at time of assessment

<table>
<thead>
<tr>
<th>Type of maxillary prosthesis at time of assessment</th>
<th>No. of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>One or two bars supporting an overdenture with a resilient attachment</td>
<td>12</td>
</tr>
<tr>
<td>Bar supporting an overdenture with rigid attachments</td>
<td>6</td>
</tr>
<tr>
<td>Fixed bridge or fully implant-supported overdenture</td>
<td>9</td>
</tr>
</tbody>
</table>

The average bone loss in microns per month for each prosthetic type was calculated and the results are illustrated in Figure 22.

A statistically significant difference was found between overdentures with resilient attachment and fixed bridges with regard to bone loss around the supporting implants, based on analysis with one-way ANOVA, multiple comparisons with LSD corrections (F (2, 24) = 4.55, p < .05). Statistical analysis of differences between resilient and rigid attachments was not possible due to the small sample size.

![Average Ratio Bone Loss by Prosthetic Type](image)

Figure 23. Average bone loss per month by prosthetic type, as an average of values for the supporting implants
3.6.5 Opposing Occlusal Surfaces

The nature of the opposing dentition at the time of patient assessment was recorded. For non-attending patients with radiographic data, the opposing dentition at the time of the last radiograph was recorded. The grouping of the opposing occlusal surfaces and numbers of patients within each group are indicated in Table 14:

<table>
<thead>
<tr>
<th>Nature of opposing occlusal contacts</th>
<th>No. of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Natural teeth or natural teeth and removable partial denture with bounded saddle (Kennedy Class III)</td>
<td>11</td>
</tr>
<tr>
<td>Natural teeth and bilateral distal extension removable partial denture (Kennedy Class I)</td>
<td>6</td>
</tr>
<tr>
<td>Natural teeth and distal segment implant bridges</td>
<td>2</td>
</tr>
<tr>
<td>Fully implant supported bridge/ Implant overdenture</td>
<td>17</td>
</tr>
</tbody>
</table>

There was no statistically significant correlation between the opposing dentition and bone loss n (30), r = -.116, p > .05.

3.6.6 Age

No statistically significant correlation was found between peri-implant bone loss and patient age, either individually n (30), r = -.262, p > .05, or in age groups (see Figure 9), n (30), r = -161, p > .05.

3.6.7 Gender

There was no statistically significant correlation between patient gender and bone loss n (30), r = -.025, p > .05.
3.6.8 Oral hygiene

The amount of plaque around the implants was scored according to Lindquist et al. (1988). The majority of implants (84) were plaque-free by visual assessment and the allocation of plaque scores is illustrated in Table 15.

Table 15. Plaque scores in relation to individual implants

<table>
<thead>
<tr>
<th>Characteristic of plaque score</th>
<th>No. of implants</th>
</tr>
</thead>
<tbody>
<tr>
<td>Implants with no visible plaque</td>
<td>84</td>
</tr>
<tr>
<td>Implant with local plaque accumulation</td>
<td>17</td>
</tr>
<tr>
<td>Implants with general plaque accumulation greater than 25% of abutment surface.</td>
<td>7</td>
</tr>
</tbody>
</table>

Mucosal status

![Bar chart showing mucosal status](image)

Figure 24. Oral hygiene scoring (as described by Lindquist et al., 1988) by number of implants.

Key: 1 = no visible plaque, 2 = local plaque accumulation, 3 = general plaque accumulation greater than 25%.

A statistically significant association was found between plaque scores and bone loss, n (108), r = .189, p = .05. The results signified greater bone loss to be associated with a higher plaque score. Figure 25 illustrates the association.
The plaque score was highly statistically significantly correlated with the modified gingival index, n (108), r = .678, p < .001. This indicated validity in the clinical recording of these parameters.

Univariate ANOVA was used to analyse the main effects and interactions between smoking data and measures of oral health, that is the plaque score and mucosal gingival index (MGI). Significant main effects were found for both plaque index and the MGI, but no significant interaction was found between smoking with implants or smoking history with either index.

### 3.6.9 Mucosal Status

The health of the peri-implant mucosa at each implant was assessed according to the index described by Mombelli et al. (1987). Table 25 shows that most of the implants examined (70) were surrounded by healthy mucosa.
Figure 26. Modified Gingival Index (Mombelli et al., 1987) by number of implants
Key: 0 = No bleeding when a periodontal probe is passed along the mucosal margin adjacent
to the implant, 1 = isolated bleeding spots visible, 2 = blood forms a confluent line along the
mucosal margin, 3 = heavy or profuse bleeding

Figure 27. Average Bone Loss in relation to Modified Gingival Index Score

A statistically significant association was found between the Modified Gingival
Index (MGI) score and bone loss n (108), r = .232, p < .05. The direction of
the relationship showed greater bone loss to be associated with a greater
modified gingival index score, that is, poorer peri-implant mucosal health.
3.6.10 Implant Length

The degree of bone loss in microns per month, after adjustment for year-one bone loss, was plotted for each implant as illustrated in Figure 28. Many individual implants scored zero bone loss, although it is evident that there is a wide variation.

A weak although statistically significant correlation was found between implant length and the amount of bone loss, n (146), \( r = -.183, p < .05 \) with the direction of the relationship indicating that as implant length increased, bone loss decreased.

![Implant Length by Bone Loss](image)

Figure 28. Bone loss in microns per month plotted for each implant. Each dot (or cross, triangle, square, diamond) represents one implant and the colours indicate the implant length.
3.7 Quality of Life Assessment

Twenty-two patients completed the quality of life assessment. The data from one patient were excluded as evaluation of the treatment history revealed both post-surgical neuropathy and an opinion that conventional dentures would have been more appropriate treatment. The questionnaire scores were extremely low and would have confounded the overall result.

The 12-question quality of life questionnaire, adapted from Pjeturrson et al. (2005), addresses functional, aesthetic, cleaning, patient expectation and cost parameters. Questions 1-6 and 9-12 provide absolute scores, whilst questions 6, 7 and 8 make comparisons between teeth and implants. The questions are included in appendix 1.

A highly statistically significant correlation was found between the average Likert 4-point scale scores and the average visual analogue scale (VAS) scores for questions 1-6, 9-12, n (21), r = .860, p < .001. This suggests a high level of internal validity for the patients' responses to the statements. The percentage of each response from the Likert 4-point scale for questions 1-6, 9-12 is illustrated in Figure 29:
Responses from both the Likert 4-point and VAS generally indicate high levels of patient satisfaction, although with a few patients giving the response of “no” to some statements. The mean VAS for all 9 questions was 85.00 (SD 2.66). A failure to match expectation was the most frequent negative report, with issues relating to cleaning also recording less positive reports.

Analysis of the questions comparing teeth and implants in relation to cleaning and mucosal bleeding, (6, 7 and 8) show significant variation in patient opinion on these factors (Figure 30). Question six - if implants are easier to clean than teeth, with more responses favouring teeth as easier to clean, but an even distribution over the four responses. Question seven - if implants take longer to clean, with more agreement than disagreement. Question eight - whether the tissues around implants bleed more than teeth, with most responses in
agreement, slightly fewer in total disagreement and relatively few unsure responses.

![PSQ 4-point Scale](image)

Figure 30. Percentage of each response to comparative questions.

Analysis of the patient satisfaction questionnaire by prosthesis type revealed little difference in patient satisfaction between overdentures and fixed prostheses. Statistical analysis of these differences was not possible due to the small number of patients. Slightly greater satisfaction with chewing was evident with fixed bridges, and for speaking with overdentures.
Figure 31. Patient satisfaction (VAS) by prosthesis type.

Analysis of the questions comparing teeth and implants in relation to cleaning and mucosal bleeding, (6, 7 and 8) are illustrated in Figure 32.

Question six - are implants easier to clean than teeth, with more agreement for overdenture patients who need only to clean under a bar than for fixed bridge patients, who generally found cleaning at least as difficult as with natural teeth. Question seven - whether implants take longer to clean, with a greater proportion of the overdenture patients reporting that cleaning around the bars supporting their prostheses took longer than cleaning natural teeth. Question eight - whether the tissues around implants bleed more than teeth, with little difference in response between the two prosthetic types and most not reporting any difference.
Figure 32. Analysis of the comparative questions, A = Q 6, B = Q 7, and C = Q 8, by prosthesis type.
Although correlations were calculated between the average PSQ 4-point scale, the average VAS and the clinical parameter of medical status, modified gingival index, plaque score and smoking data, no statistically significant associations were detected.
Discussion

The aim of the study was to gain a greater understanding of the factors that affect peri-implant bone loss, a recognised predictor of a poor long-term prognosis. Factors assessed were age, gender, health status, mucosal health, oral hygiene, smoking, length of implants, prosthetic type and opposing occlusion. Although some associations were evident, such as mucosal health and implant cleanliness, most factors had no direct relationship with peri-implant bone loss as assessed by radiographic measurement. The results are discussed under the relevant headings.

4.1 Study Population

The loss of patient data in follow-up examinations in this study is slightly greater than some earlier reports, for example Adell et al. (1981), but at twenty five percent is lower than others such as the fifty-six percent loss reported (Jemt and Johansson, (2006)) in a recent fifteen year follow-up study on implants in the edentulous maxilla. Six of the patients (16.6%) were deceased or had moved residence, leaving a relatively low rate (8%) of patients who could not be contacted. The low rate of inaccessible patients may be explained by the fact that this patient group represented pioneering implant treatment in Australia and interest has been maintained in following up the outcomes. For example, copies of the radiographs of patients transferred to private practice prosthodontists were often sent to the initially treating prosthodontist and were maintained in the hospital records.
4.2 Implant Treatment Outcomes

The outcome of implants in the current study, that is success or survival, is also in keeping with published reports for the edentulous maxilla (Engquist et al., 1988, Ekfeldt et al., 1997). Jemt and Johansson (2006) analysed loss to follow-up of patient data and concluded that whilst many of the lost patient group were accounted for, that is deceased, moved residence or too ill to participate, the group showed a trend for lower implant survival. In the current study the bone loss of the lost to follow-up group was not significantly greater than the reviewed group (p > .05). The results indicated that the adjusted bone loss rate from the first year onwards is in keeping with the results of Adell et al. (1981) in reporting that the majority of implants (71.5%) had less than or equal to 0.1mm of bone loss per annum.

Whilst acknowledging the possible inaccuracies associated with the first year bone loss adjustment and radiographic measurement (refer to page 56), the similarity between the findings of the current study and earlier reports validates further analysis of data from this patient group.

Whilst longitudinal bone loss calculations could not be made for the four patients with total implant loss, analysis of these patients is relevant to ascertain whether these early losses could bias the remaining data. A cause of rapid bone loss was found to be unlikely from the radiographic data available as none of the implants in this total failure group were preceded by continuous bone loss. This would suggest that the losses were either related to failure of osseointegration (early losses) or to traumatic or acute overload.
For patients 8 and 11, a clinical diagnosis of bruxism and combination syndrome respectively had been recorded. Combination syndrome, as described by Kelly (1972), implies an absence of mandibular posterior teeth, both this and bruxism could be expected to create high and localised forces on the implants in the anterior maxilla, potentially causing loss of integration.

Patient 33 had four implants placed superficially, where implant threads were above the bone crest after stage one surgery. This may have reflected the relative inexperience with implant surgery of the surgeon at that time. All of the implants for this patient failed before loading, which implies failure of osseointegration. Superficial implants would be more likely to be loaded by the overlying prosthesis, which could result in a level of micromotion incompatible with osseointegration (Szmukler-Moncler et al., 1998). However, superficially placed implants in another patient, (Appendix 2, no.1) did not lead to adverse consequences.

Further data relating to this failure group were not available, for example with regard to smoking or bone quality and quantity, although the generally shorter implants used for these four cases could indicate that a lower volume of bone was available at time of surgery. The relatively high rate of early loss relative to later loss (17% compared to 8%) is in keeping with previous reports (Engquist et al., 1988, Adell et al., 1981). Similarly, the greater failure rate of the shorter implants (Table 4) is also in agreement with previously reported data.
Chuang et al. (2002) commented on some of the problems associated with analysis of data in implant studies. The authors suggest that binary analysis of implant survival, such as “yes/no”, is inappropriate as this assumes that implants are independent of one another. The authors note recommendations that only one implant should be selected from each patient, but suggest that as this could result in the loss of significant data, a statistical method to allow for interrelationships should be used. The data from the current study suggest that selecting one implant from each patient would be a flawed as differences within a patient were evident. Reviewing the data from patient 10 (Figure 16) indicates how a patient with eight implants showed considerable variation in bone loss between implants. Averaging of bone loss between the implants for this patient would lose the integrity of the data. Similar observations can be made for other patients (see Appendix 2). For this study, patient factors were analysed on a patient basis (medical status, concurrent smoking and smoking history, prosthesis type, opposing occlusion, age and gender) and implant factors on an implant basis, (plaque scores, modified gingival index) with acceptance of the grouping of implants in individual patients.

4.3 Factors Effecting Bone Loss

4.3.1 Age

The absence of a detectable association between peri-implant bone loss and patient age supports the hypothesis that age does not negatively influence implant success. This supports the findings of Bryant and Zarb (2003), but is in contrast to the findings of Moy et al. (2005). This may be a result of the
controlled nature of Bryant and Zarb's study, whereas many interactions may have been possible with the retrospective analysis of Moy et al. (2005). In particular, an interaction with medical conditions may have occurred, given that Moy et al. (2005) reported that diabetes (type 2 diabetes, also known as maturity onset diabetes, is age-related) presented the greatest risk factor for implant treatment.

4.3.2 Gender

In agreement with many other reviews (Sugerman and Barber (2002), Beikler and Flemmig (2003), van Steenberghe et al. (2003), Wood and Vermilyea (2004)), the data showed a lack of detectable association between peri-implant bone loss and patient gender, and support the hypothesis that gender is not a predictor in implant success.

4.3.3 Health Status

Numerically the distribution of both patients and implants with and without systemic medical conditions was similar. No statistically significant correlation between patient medical status and bone loss was detected. An absence of correlation contrasts with reports of greater implant failure with systemic disease, for example the study by Moy et al. (2005), predicts greater bone loss.

Older age groups are more likely to be edentulous, to require implant treatment and are more likely to have systemic medical conditions. The high
success rates of implants reported in edentulous patients suggests that medical conditions in general do not adversely affect implant treatment success. Therefore large numbers of patients may be required for studies to detect any statistically significant health effects on implants. This problem is also evident with medications and was commented on by Thomson et al. (2000) when investigating associations between medications and xerostomia. The problem of polypharmacy represents a ‘formidable methodological challenge’ and the authors’ opinion was that for such studies to detect meaningful associations, the study group was required to be ‘prohibitively large’ (Thomson et al., 2000).

Forty one medications were prescribed to the twelve medicated patients in this group, with only seven of these prescriptions being for the same drug. However, some of the medications belonged to similar pharmacological groups. Although the current study does not allow determination of an association between medical status and peri-implant bone loss, given the increasing numbers of implant treatments, collection of sufficient data would be more likely in the future.

Patient 28 suffered from Osteogenesis imperfecta (Type 4) and had been taking 30mg of pamidronate every three months for over ten years. This represents long-term usage of a low-dose of a relatively potent bisphosphonate medication. Osteogenesis imperfecta (OI) is caused by a genetic defect in the production of type 1 collagen, resulting in brittle bones that fracture easily, with healing occurring over the usual timeframe. Type 4 OI has an intermediate level of severity, type 1 being mild and type 2 lethal, with
categorisation being established on clinical characteristics such as pigmentation of the sclera. Treatment with low doses of pamidronate is an established prophylactic treatment for OI, although the optimum bisphosphonate treatment regimen and long-term outcomes are still unknown (Rauch and Glorieux (2004)). Considering the recent concern regarding bisphosphonate medications and possible links to osteonecrotic lesions of the jaws (Ruggiero et al. (2004), Melo and Obeid (2005)) and the case report of implant failures and a possible association with a bisphosphonate medication (Starck and Epker (1995)), the successful treatment of patient 28 (patient 28 in Appendix 2) is interesting. The combination of the long half-life (estimated at over ten years, Cheng et al., 2005) and the long duration of use increases the chance of complications. The patient in the current study did not suffer from undue bone loss or other implant complications in either jaw, which were restored with full-arch prostheses.

4.3.4 Smoking with Implants

Approximately twice as many of the clinically assessed implants were in non-smokers than smokers, with thirty-seven percent being in heavy smokers (> 200 cy). No association was found between smoking with implants and bone loss. Additionally no interaction between smoking and plaque scores was detected which is in contrast to the report of Lindquist et al (1997), where a difference in bone loss was detected after ten years only when both factors were present. The data from the current study do not support the hypothesis that smoking increases peri-implant bone loss. In contrast, a treatise (Har 2006, p.59, 67) on implant survival at an associated Sydney hospital
(Westmead Centre for Oral Health) reported a strong association between smoking and implant failure, with data indicating an increase in losses after five years. This suggests that smoking would increase bone loss. The contrasting results suggest that an interaction between factors is of importance in bone loss.

An interesting finding was the association between smoking and poorer mucosal health. The association between mucosal health and bone loss would suggest that further research into the nature of the smoking effect on health/inflammatory response, or health behaviour of smokers is warranted.

4.3.5 Smoking History

The distribution of patients and implants with and without a history of smoking during the pre-surgical period was almost equal. No association was detected between a history of smoking and peri-implant bone loss. The finding of DeLuca et al. (2006) that a history of smoking increases the risk of late implant failures by a factor of two suggests that a history of smoking would increase bone loss. The results of the current study do not support this hypothesis.

The association between a history of smoking and increased plaque scores and decreased mucosal health suggests that either the oral health behaviour of smokers differs, or the effects of smoking on systemic health are detectable in the mucosa in the long-term. The increased plaque scores in those with a smoking history would suggest poorer dental health behaviour. This contrasts
the analysis of dental health behaviour that suggested that the alteration of a patient’s health consciousness would be unlikely to alter dental health behaviour (Toneatto and Binik (1990)). Further research is indicated to clarify these associations.

4.3.6 Type of Prosthesis

Twice as many patients were restored with overdentures than with fixed bridges with two-thirds having resilient attachment. Statistically, the data revealed that greater peri-implant bone loss was associated with the resilient attached overdentures than with the fixed bridge group. Greater bone loss was found between the rigidly attached overdentures than the fixed bridges, though this result did not reach statistical significance. As peri-implant bone loss is a predictor of poor prognosis, the association is in keeping with the literature reporting lower success rates with overdentures (Engquist et al., 1988, Ekfeldt et al., 1997). The current results support the hypothesis that greater peri-implant bone loss would occur with overdentures. The results also support the assertion of Zitzmann and Marinello (2000) that overdentures are subject to the same forces as fixed bridges, although generally the number of supporting implants is lower.

Analysis of the cases revealed that patients 10 and 24 had resilient attached overdentures and significant bone loss. As clinical maintenance is necessary with resilient attachment and one of these patients did not attend for consultation for eight years (failure to meet financial obligations for treatment), there is a possibility that the result may have been biased by factors other
than prosthesis type. Greater numbers of subjects in a controlled study would be required to clarify these associations.

4.3.7 Opposing Occlusal Surfaces

Although almost equal numbers of patients presented with some opposing natural teeth or a fixed full-arch implant bridge, no association was detected between the opposing dentition and peri-implant bone loss. The role of periodontal proprioceptors in detecting and giving feedback to masticatory muscles has been discussed (Kim et al., 2005). The data from the current study do not support the hypothesis that loss of proprioception would increase peri-implant bone loss. This loss of proprioception may be too small an effect to be detected, which together with the small numbers of patients may not allow this reflex effect to be identified.

4.3.8 Oral Hygiene

A statistically significant association between plaque scores and peri-implant bone loss was detected, and the difference in bone loss with different scores is illustrated in Figure 25 (page 77). The results support the hypothesis that poorer oral hygiene would result in greater bone loss. The finding is also in keeping with the predicted low resistance of the peri-implant mucosa to inflammatory challenge shown in animal studies (Berglundh et al., 1992), but contrasts with the longitudinal clinical study of Lindquist et al. (1996). A possible explanation could be that osseous destruction is more rapid in maxillary bone as a result of its less dense trabecular structure. The study of
Lindquist et al. involved the anterior mandible, characterised by a thick cortical plate and dense trabeculae. Detection of the association of bone loss and oral hygiene in the current study may have been possible as a result of both measures (plaque index and bone loss) being recorded at each implant rather than a patient oral hygiene score and averaged bone loss value. However, it is also possible that in this study the presence of a few poorly maintained resilient attached overdentures could have biased the results.

4.3.9 Mucosal Status

A statistically significant association between increasing modified gingival index (MGI) score and peri-implant bone loss supports the hypothesis that poorer mucosal health is associated with greater bone loss. The strong association between increasing plaque score and MGI validates the clinical recording, in keeping with establishment of plaque on implants and the associated inflammatory response (Zitzmann et al., 2001). The increased bone loss with increased inflammatory response, as indicated by the MGI, correlates with both plaque index and the association between plaque index and bone loss. However, Salvi and Lang's (2004) review reported that the evidence for an association between mucosal health and peri-implant bone loss was weak. It is possible that with the small number of patients in the present study, and the nature of the resilient attachment that the results may be confounded. The seven implants with an MGI score of 3 would have met the diagnostic criteria of peri-implantitis described by Lang et al (2004), providing an explanation for the severe bone loss (Figure 27 – page 78).
4.3.10 Implant Length

A statistically significant association was detected between implant length and peri-implant bone loss, such that the greater bone loss was observed with shorter implants. The hypothesis that greater bone loss would occur with shorter implants is supported.

If peri-implant bone loss is taken to be a negative prognostic sign for implant success, the current finding is in keeping with clinical reports of lower success rates with shorter implants (Jemt and Johansson (2006), Engquist et al., 1988, Ekdeldt et al., 1997). Finite element analysis of stress distribution around loaded implants, for example Kitamura et al. (2004), suggests that the implant surface area would not be closely related to implant success for the lengths commonly used. The reasoning being due to the concentration of stress in the coronal peri-implant cortical bone, with rapidly decreasing stress values apically. Dispersion of the majority of stress in the coronal area of bone predicts that with all other factors being equal, (bone quality/quantity, force magnitude and direction), differences would only be apparent with implants not long enough to extend beyond the principal stress zone.

The data from the current study neither support nor reject the suggestion of Jemt and Lekholm (1995), that it is not short implants per se, but the clinical indications for short implants that are associated with greater failure. Peri-implant bone quality was not assessable in the current study and the available data were insufficient to analyse statistically the effect of implant position due to the low numbers of implants placed in the posterior maxilla.
The occurrence of relatively severe bone loss in individual implants of all lengths (Figure 29 – page 79) and the correlation between bone loss and mucosal health, suggests that implants with severe bone loss may have suffered peri-implantitis. The evidence suggests that at a mucosal level the implant length is immaterial.

4.4 Quality of Life Assessment

Both the Likert 4-point scale and visual analogue scale (VAS) of the patient satisfaction questionnaire (PSQ) showed generally high levels of satisfaction with treatment. This finding is reinforced by the high correlation between the Likert 4-point scale and the VAS.

The extremely low scores reported by one patient indicate a failure of treatment despite the apparent clinical success. Less information was available regarding the psychological aspects of patient adaptation and satisfaction at the initiation of this patient’s treatment. A recent report using a standardised psychometric analysis (Symptom Checklist-90-R) suggested that it is possible to detect a psychological basis for prosthesis rejection before treatment (Eitner et al., 2006). The failure of this patient’s treatment is acknowledged and the exclusion of the data from the PSQ was necessary to avoid it biasing the overall data.

The low numbers precluded statistical analysis of the differences between responses for individual questions. However, it appears noteworthy that the
most common negative report was a failure to meet each patient’s expectation. A characteristic of the patient group was a failure to adapt to a conventional complete denture, a fact that by Eitner’s report (2006) would suggest a likelihood of psychological incompatibility. However, poor patient adaptation to conventional complete dentures is characteristic of most implant studies, contrasting with reports of patient satisfaction with implant prostheses that are generally very positive (Strassburger et al., 2006).

Difficulty in cleaning is expected, especially for elderly patients in contrast to cleaning of a complete denture, although the majority (68%) presented the subjective report of cleaning the prosthesis well. The questions relating to cleaning that compared implants with teeth revealed trends for patients’ perceptions of requiring greater time requirement for cleaning implants, and that teeth were easier to clean. It is possible that the reasons for loss of teeth were related to a lack of oral hygiene practices. The reinforcement of oral hygiene at regular reviews may have changed patient attitudes and time investment in dental health, with the result that the majority of implants (78%) were found to be plaque-free at assessment.

Comparison of patient satisfaction by prosthesis type detected minimal differences. Considering the high levels of satisfaction reported, this would suggest that patient satisfaction is related to treatment that succeeds in addressing each patient’s specific needs, rather than to a specific treatment modality.
Slightly greater satisfaction with speech and cleaning with overdentures was reported. The fixed bridge group reported a slightly greater satisfaction with chewing. These differences are quantitatively much smaller than those reported in the crossover trial of Heydecke et al. (2003). Additionally, in the study of Heydecke and colleagues, patient satisfaction with chewing was greater for the overdenture group. A possible explanation for the small differences between prosthesis type reported in the current study is the restoring prosthodontist’s use of provisional restorations and multiple adjustments to optimise speech and cleaning, before the definitive restoration.

Although confidentiality was assured and assurance that the treating clinician would not view the questionnaire responses, the questionnaire was administered by a clinician at the treating hospital. Therefore, the possibility of bias and unrealistically positive reports is acknowledged.

No statistically significant associations between patient satisfaction and clinical parameters were found. This would suggest that although some patients were aware of clinical problems at an implant level, this did not significantly decrease treatment satisfaction. There is also a possibility that the number of patients was insufficient to detect an association.

4.5 Limitations of the Study

The quality of data available for a retrospective study is dependent upon protocols followed at the time of treatment and the quality of the documentation. Data may be absent or in the case of radiographic data the
available data may be unusable. Studies with retrospective design are at risk from potential recall errors and examiner bias (Jokstad et al., 2004). A prospective study can avoid many of these problems and for a study such as the current research, the radiographs and investigators need to be standardised. Controlling the loss of patient data in follow-up consultations is more likely in a prospective study. However, it should also be acknowledged that agreeing to participate in a clinical study may affect patients' oral health behaviour; additionally those refusing to participate may limit the study population's representiveness. Both factors may produce confounded results.

In the current study, the lack of radiographs in the first five years after stage two surgery necessitated adjustment for the first year bone loss to avoid considerable loss of data. The adjustment to all implants not placed superficially minimises the chance of errors in the relative rates of bone loss for implants and should not affect the correlations detected. However, the adjustment may affect the application of success criteria to a particular implant group.

Errors in data collection are more likely to affect the correlations between investigated factors and bone loss, and may occur due to non-standardised or indistinct (lacking contrast) radiographs, combined with the small distances under assessment.

Sample size is a significant limitation of implant studies attempting to detect small differences in a generally successful treatment protocol. The limitation is particularly evident with aspects such as health and medications, where great
difficulty is encountered in recruiting an adequately homogeneous study group with sufficient numbers of specific conditions to detect differences. The current study could be regarded as a pilot study to indicate what data should be regularly recorded for all implant patients and for future prospective studies. With adequate data collection over time, detection of interactions of risk factors to bone loss and thus implant failure may be possible. The hypothesis of interaction of risk factors to create clusters of implant failures was recently described by Mombelli and Cionca (2006). Greater patient numbers can decrease the possibility of a few patients with adverse outcomes biasing the overall result.

Possible bias may have been introduced by the patient satisfaction questionnaire being presented by the assessing clinician. A method of avoiding this problem would be for a separate clinician to conduct interviews and possibly at a location distant from the treating hospital. Additionally, the use of a Likert 4-point scale may not allow answers that accurately represent the patients' opinion. Greater breadth would have been possible with a scale of more categories which may have made relationships with other variables more evident.
Conclusion

Acknowledging the limitations, the current research has demonstrated that clinical implant studies should account for and report on every individual implant, to avoid loss of integrity of data and false positive outcomes. The associations detected in the current study between bone loss, implant hygiene and mucosal health support the weak associations reported in the literature. Further studies with large patient populations are necessary to provide stronger outcome data. The results suggest that implant and oral health factors have a greater effect on peri-implant bone loss than general patient factors, such as systemic disease, smoking, opposing dentition, age and gender. Additionally, studies with greater sample size are indicated to clarify possible associations and interactions between other factors that may individually have undetectable effects.

Patient satisfaction with implant-based rehabilitation of the edentulous maxilla is generally high, with the most frequent complaint being a failure to match patients' expectations with treatment outcomes. Research targeting pre-treatment patient education and patients' psychometric profiles may clarify this disparity. Patient satisfaction appears to be more strongly related to an adequate solution for patients' presenting complaints than to the treatment modality selected.

In conclusion, continuing research based on sound scientific methodology into parameters affecting implant survival and patient satisfaction would continue to enhance treatment protocols for optimal patient care.
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Appendix 1

Patient Satisfaction Questionnaire
(Adapted from Pjetursson et al., (2005))

EXAMPLE:
Shown below is an example of how to answer the questions in this questionnaire:

Please answer all the questions as accurately as possible. For each question, please:

Example Question

1) Tick the box next to the most appropriate answer

My denture/bridge functions very well, and I can chew on it very well

☐ Yes, definitely
☐ Yes, possibly
☐ I don't know
☐ No
☐

2) Mark a cross on the line below to indicate your degree of satisfaction with your ability to chew

---

No satisfaction

Total satisfaction
Question 1

Tick the box next to the most appropriate answer

My denture/bridge functions very well, and I can chew on it very well

☐ Yes, definitely
☐ Yes, possibly
☐ I don’t know
☐ No

Mark a cross on the line below to indicate your degree of satisfaction with your ability to chew

<table>
<thead>
<tr>
<th>No satisfaction</th>
<th>Total satisfaction</th>
</tr>
</thead>
</table>

Question 2

1) Tick the box next to the most appropriate answer

I feel more secure biting firmly on my denture/bridge now that it is secured to implants

☐ Yes, definitely
☐ Yes, possibly
☐ I don’t know
☐ No

2) Mark a cross on the line below to indicate your degree of satisfaction with biting firmly.

<table>
<thead>
<tr>
<th>No satisfaction</th>
<th>Total satisfaction</th>
</tr>
</thead>
</table>
Question 3

1) Tick the box next to the most appropriate answer

I can speak well with my denture/bridge

☐ Yes, definitely
☐ Yes, possibly
☐ I don’t know
☐ No

2) Mark a cross on the line below to indicate your degree of satisfaction with *speaking* with your denture/bridge.

________________________________________________________________________
No satisfaction                      Total satisfaction

Question 4

1) Tick the box next to the most appropriate answer

I am pleased with how my teeth look

☐ Yes, definitely
☐ Yes, possibly
☐ I don’t know
☐ No

2) Mark a cross on the line below to indicate your degree of satisfaction with *how your teeth look*

________________________________________________________________________
No satisfaction                      Total satisfaction
Question 5

1) Tick the box next to the most appropriate answer

I cannot clean my implants very well

☐ Yes, definitely
☐ Yes, possibly
☐ I don’t know
☐ No

2) Mark a cross on the line below to indicate how well you can clean around your implants.

________________________________________
Can’t clean

Clean easily

Question 6

1) Tick the box next to the most appropriate answer and,

It is easier for me to clean my implants than to clean my natural teeth

☐ Yes, definitely
☐ Yes, possibly
☐ I don’t know
☐ No

2) Mark a cross on the line below to indicate which is easier to clean

________________________________________
Easier with teeth

Easier with implants
Question 7

1) Tick the box next to the most appropriate answer

I need more time to clean my implants /bridge than it took to clean my teeth

☐ Yes, definitely
☐ Yes, possibly
☐ I don’t know
☐ No

2) Mark a cross on the line below to indicate which (teeth or implants) took relatively longer to clean.

________________________________________
More time with teeth                          More time with Implants

Question 8

1) Tick the box next to the most appropriate answer

The tissues around the implants bleed more than they did around my teeth

☐ Yes, definitely
☐ Yes, possibly
☐ I don’t know
☐ No

2) Mark a cross on the line below to indicate around which (teeth or implants) bleed more

________________________________________
More with teeth                               More with Implants
Question 9

1) Tick the box next to the most appropriate answer

With my treatment, I got exactly what I expected

☐ Yes, definitely
☐ Yes, possibly
☐ I don’t know
☐ No

2) Mark a cross on the line below to indicate your degree of satisfaction with your treatment relative to what you expected.

No satisfaction

Total satisfaction

Question 10

1) Tick the box next to the most appropriate answer

I would go through such treatment again, if needed

☐ Yes, definitely
☐ Yes, possibly
☐ I don’t know
☐ No

2) Mark a cross on the line below to indicate your degree of willingness to undergo such treatment if needed.

No willingness

Total willingness
Question 11

1) Tick the box next to the most appropriate answer

I would recommend this treatment to a relative or friend, if needed

☐ Yes, definitely
☐ Yes, possibly
☐ I don’t know
☐ No

2) Mark a cross on the line below to indicate your degree of recommendation

__________________________________________________________________________
No recommendation    Total recommendation

Question 12

1) Tick the box next to the most appropriate answer

I found the cost of the treatment was justified

☐ Yes, definitely
☐ Yes, possibly
☐ I don’t know
☐ No

2) Mark a cross on the line below to indicate your degree of satisfaction with the cost

__________________________________________________________________________
No satisfaction    Total satisfaction

END OF QUESTIONNAIRE

THANK YOU FOR ITS COMPLETION
Bone loss (implant threads) against time (months) for all patients
Bone loss (implant threads) against time (months) for all patients
Bone loss (implant threads) against time (months) for all patients