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REVIEW

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Dental implants: Maintenance, care and treatment of peri-implant infection

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Abstract

Osseointegration is becoming increasingly routine in the rehabilitation of partially or fully edentulous patients. However, the surrounding tissues may be subject to inflammatory conditions similar to periodontal disease and so require maintenance. This article discusses the background, aetiology, diagnosis of peri-implant diseases, and the maintenance, care and treatment of peri-implant infection in osseointegrated implants. Three case studies are presented to illustrate points in the care of implants.

Key words: Osseointegration, peri-implant mucositis, peri-implantitis.

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INTRODUCTION

The provision of implant retained prostheses is becoming more and more common. In the last 30 years over 700 000 patients have been treated using the Brånemark system alone (Nobel Biocare, personal communication, 2003). The high survival rate of osseointegrated dental implants is well documented, but it is becoming increasingly clear that successfully integrated implants are susceptible to disease conditions that may lead to the loss of the implant.[1] Although placement and restoration are usually the field of the periodontal, oral and maxillofacial surgery or prosthetic specialist, given the increasing numbers of patients treated with osseointegrated fixtures it is increasingly likely that maintenance of these implants will become much more common by the general dentist. The aim of this paper is to discuss the background to implant failure, types of implant-associated diseases, diagnosis, maintenance and treatment of osseointegrated fixtures.

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Implant failure

Implant complications can be due to a number of causes including prosthesis instability, implant mobility, occlusal trauma, fractured components, pain, inflammation, infection and neuropathy.[2] In this paper, we are primarily interested in the failure of the supporting tissue of implants and will not discuss in any great detail prosthetic, material or surgical complications.

Failure of implants may be described as early or late. Early failure follows shortly after placement and osseointegration is never really achieved. Late failure occurs in a successfully integrated implant some time after placement and subsequent restoration. The causes of late failure may be marginal infection/disease or biomechanical overload. However, an analysis of the clinical trials of the ITI system reveals that a very small proportion of failures seem to be associated with occlusal overload.[3] From this analysis the major cause of late failures could be attributed to peri-implant infections. It was noted that patients with good oral hygiene tended to keep implants longer.

Aetiology

Peri-implant infections are generally classified as peri-implant mucositis and peri-implantitis depending on the severity. Peri-implant mucositis is defined as a reversible inflammatory reaction in the soft tissues surrounding an implant. Peri-implantitis is an inflammatory reaction with loss of supporting bone in the tissues surrounding an implant.[4]

An increasing number of studies suggest that anaerobic plaque bacteria may have an adverse effect on peri-implant tissue health.[3] As soon as an implant is exposed to the oral cavity plaque will form on its surface. The process may be identical to that seen on teeth, with the formation of pellicle and subsequent microbial colonization. In edentulous patients colonization of the peri-implant sulcus originates from the microflora found in saliva.[5] There are relatively few studies that have investigated colonization in partially edentulous patients. However, it is not unreasonable to suggest that implant sulci may be colonized from

bacteria in saliva or neighbouring periodontal pockets. A comparison of residual periodontal pockets and peri-implant sulci found that the same bacteria colonized both.[6] If periodontal pathogens were identified in pockets prior to implant placement they were also detected at implant sites three months after exposure to the oral environment. Quirynen *et al*.[7] demonstrated bacterial penetration between the components of the Brånemark implant system. The inner spaces were easily colonized and bacteria may leak out from these spaces through the implant-abutment interface into the peri-implant area.

The microbiota associated with healthy peri-implant tissues closely resembles that of the flora associated with gingival health. The organisms associated with mucositis are very similar to that of gingivitis and, unsurprisingly, that of peri-implantitis is very similar to that seen in periodontitis.

Experimentally induced peri-implantitis was induced in patients six months after abutment connection in two stage implants by asking these subjects to refrain from oral hygiene procedures for three weeks.[8] As a result of plaque accumulation, gingival indices and probing depths increased around the implants and also the teeth in a similar way. Hence, the accumulation of plaque around implants can lead to peri-implant mucositis. Using dark-field microscopy to analyze plaque samples collected, the percentage of coccoid cells, motile rods and spirochaetes from the peri-implant mucositis sites was very similar to that from the gingivitis sites.[8] Interestingly, the inflammatory infiltrate was of equal size to adjacent control teeth and to implants when *de novo* plaque formation was studied in a beagle dog model.[9]

Comparisons of successful and failing implants have shown differences in the composition of the associated flora. Successful implants were sparsely colonized by Gram positive cocci, compared to failing implants sites, which contained large numbers of Gram negative anaerobes.[10,11] *Actinobacillus actinomycetemcomitans*, *Prevotella intermedia*, *Porphyromonas gingivalis*, Fusobacterium species, *Campylobacter rectus* have all been associated with failing implants.[12-14] These organisms and *Treponema denticola* have been found at peri-implantitis sites.[15]

Peri-implantitis can be experimentally induced in dogs and monkeys by the placement of ligatures to enhance plaque accumulation at the mucosal margin around implants.[16,17] Lang *et al*.[17] showed very similar increases in plaque and gingival indices, pocket depth and loss of attachment, histological changes and shifts in composition of the microflora around implants and teeth with experimentally induced peri-implantitis and periodontitis.

In summary, it appears that the peri-implant tissue is colonized by the same flora as the periodontium and that disease of this tissue is very similar to gingivitis or periodontitis.

Features and frequency

The following signs and symptoms are typical for peri-implantitis lesions: radiological evidence for vertical destruction of the crestal bone. The defect is usually saucer shaped and there is osseointegration of the apical part of the fixture; vertical bone destruction associated with the formation of a peri-implant pocket; bleeding and suppuration on probing; possible swelling of the peri-implant tissues and hyperplasia; and pain is an unusual feature, which, if present, is usually associated with an acute infection.

An estimation of the prevalence of peri-implantitis is difficult and depends upon the criteria used to separate health from disease. A mean crestal bone loss of 0.9-1.6mm is expected during the first post-surgical year and then a yearly loss of 0.02-0.15mm subsequently.[18-21] However, Buser *et al*.[22] showed that in 62 patients with 97 implants, the majority had very little change in bone level (±0.7mm) over an eight year period. Earlier, Buser *et al*.[23] reported on the follow-up of 2359 non-submerged ITI implants for up to eight years. They showed that the highest number of failures (16) were in the first 12 months of which five were due to infection and 11 due to mobility. Over the next seven years no more than five implants per year failed. Of the 16 implants that failed during this period, six failed due to infection, five to mobility, two to progressive bone loss and three to implant fracture. Overall the incidence of infection per year was around 0.6 per cent.

Diagnosis

The diagnosis of peri-implantitis needs careful differentiation from peri-implant mucositis, primary failures to achieve tissue integration and problems lacking an inflammatory component. This includes ruling out unusual anatomical features, unusual tissue morphology, hyperplastic responses and exposure of parts of the implant due to recession or surgical trauma. Given the similarity between periodontal and implant diseases the diagnostic parameters used for assessing peri-implantitis are the same as one would use for assessing periodontitis.[24] The parameters include clinical indices, peri-implant probing, bleeding on probing (BOP), suppuration, mobility, peri-implant radiography and microbiology.

Clinical indices

Swelling and redness of the peri-implant mucosa have been reported from peri-implant infections in addition to the other signs discussed below. There are difficulties in using indices developed for periodontal disease, perhaps due to the different structure of the tissues around implants. The soft tissue layer immediately adjacent to an implant is a less vascular, less cellular, highly collagenous scar tissue compared to normal gingival tissue.[25] In addition, texture and colour may depend on appearance before implantation and properties of the implant surface. The amount of plaque around an implant should always be evaluated.

Peri-implant probing

The soft tissue cuff around an implant in a canine model has been shown to be about 3-3.5mm regardless of system and the connective tissue attachment of 1-1.5mm.[26] Therefore, generally successful implants allow the probe to penetrate approximately 3mm.[27] The exception here is deeply submerged implants. However, when placing implants one should, ideally, try not to create deep pockets as those over 5mm are ideal niches for putative periodontopathogens and may be confused for peri-implantitis.[12] There is no scientific evidence to suggest that periodontal probing affects the integrity of an implant, but it should be noted that a metal probe may damage the implant surface.[28] A rigid plastic probe is ideal. Probing the peri-implant sulcus with a blunt, straight periodontal probe allows for assessment of peri-implant probing depth, distance between the soft tissue margin and a reference point on the implant for measuring hyperplasia or recession, bleeding and suppuration. Lang *et al*.[29] investigating the effect of different mucosal conditions around implants confirmed the excellent sealing effect of the soft tissue collar in health and peri-implant mucositis and reported relatively uninhibited penetration to the alveolar crest in peri-implantitis lesions. Probing around oral implants should be considered a reliable and sensitive parameter for the long term monitoring of peri-implant mucosal tissues.

Bleeding on probing

Bleeding on probing indicates inflammation in the pocket or sulcus. It has been shown that it is not a reliable predictor for progression of periodontal disease.[30] Instead its absence is a much better predictor for stability. In the absence of any evidence to the contrary, it would seem reasonable to extend this concept to implants.[27]

Suppuration

Neutrophils are present whenever disease is present. High numbers have been linked with inflammation of the peri-implant tissues,[31] suggesting that suppuration maybe a sign of peri-implantitis.[27]

Mobility

Implant mobility is an indication of lack of osseointegration, but it is of no use in diagnosing early implant disease, rather it shows the final stages of de-integration. Initially the bone loss associated with peri-implantitis is observed to be marginal and results in the formation of infrabony defects. The apical portion of the implant will be fully integrated, so an increase in mobility will not be evident. Complete loss of osseointegration would be reflected in a sudden increase in implant mobility.

Peri-implant radiography

Conventional radiography has been widely applied to evaluate the bony structures adjacent to implants over long periods of time. However, minor changes in bone morphology may not be noticed until they reach a significant size. Nevertheless, the distance from implant shoulder to the alveolar bone crest is a reliable parameter providing the radiographs are properly standardized. The implant shoulder in one-stage systems can be easily used, but two-stage systems require a clearly defined landmark. In most cases this is the apical termination of the cylindrical part.[3] Radiographic evidence of bone to implant contact does not indicate osseointegration. Digital subtraction radiography can increase the sensitivity significantly and has been successfully applied.[24]

Table 1. Main diagnostic differences between peri-implant mucositis and peri-implantitis

Clinical parameter	Peri-implant mucositis	Peri-implantitis
Increased probing depth	+/-	+
BOP	+	+
Suppuration	+/-	+
Mobility	-	+/-
Radiographic bone loss	-	+

Microbiology

As mentioned above there are differences in healthy and disease peri-implant sulcus microflora. Mombelli and Lang[26] suggest that too little is known to make a definitive statement regarding the use of microbiological testing in determining the risk of peri-implantitis. It will probably remain a research tool for the next few years.

Table 1 briefly outlines the main differences between peri-implant mucositis and peri-implantitis.

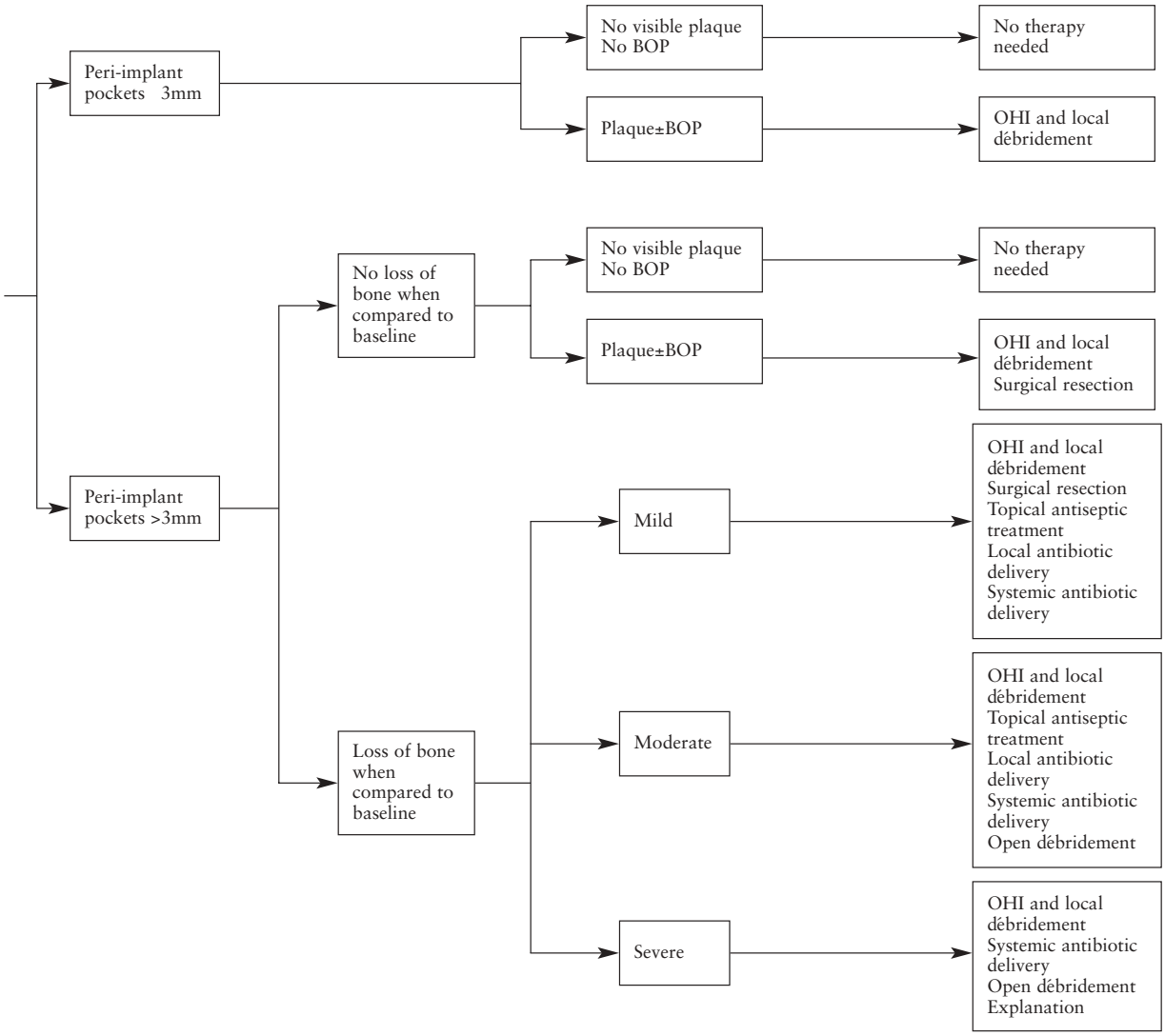
Maintenance, care and treatment

There are a number of steps at time of placement and restoration that can improve the long-term prognosis of fixtures. Patient motivation and oral hygiene are paramount. Periodontal health should be achieved prior to proceeding with implant therapy. Restorations should be cleansable with well fitting margins. In addition, as much of the mucosal tissue as possible should be preserved in its original position.

A maintenance programme should be undertaken after successful implant therapy. This should be tailored to the individual and include regular recalls to provide optimal disease prevention. The recall visit is similar to that for a periodontal patient in maintenance in that each visit includes examination, re-evaluation, diagnosis, motivation, and treatment of infected sites. Before a patient is enrolled in a maintenance programme one should ensure that baseline data has been established. Probing pocket depths and mucosal margins position are both noted and radiographic crestal bone levels are established.

The decision process for peri-implantitis maintenance and treatment should be a rational and evidence–based approach.[26] As such these authors have

Table 2. Treatment of peri-implant infection (adapted from Mombelli & Lang[27])



developed a decision process which we have adapted (Table 2).

The first question is 'Are there peri-implant pockets greater than 3mm?' One should also assess presence/absence of plaque and bleeding. If the answer is in the negative to all three, then no therapy is required, the length of recall appointment may be increased and radiographs taken every other year. The presence of plaque or bleeding indicates insufficient oral hygiene. The patient's oral hygiene should be checked and proper plaque control measures introduced/re-inforced. The implant should be cleaned by instruments softer than titanium, such as polishing with a rubber cup and paste, floss (Fig 1A, B, C), interdental brushes or using plastic scaling instruments. These have been shown not to roughen the implant surface unlike metal and ultrasonic scalers.[32]

If there are pockets over 3mm the next question is 'Is there bone loss?' Where there is bone loss there may be peri-implantitis. No bone loss may reflect a primary failure of the implant to integrate, submerged placement of the fixture, or unfavourable tissue morphology. If there is no bone loss, one should assess plaque and bleeding. An absence of both indicates no therapy is required. The presence of one or both indicates a need for oral hygiene instruction, local débridement and perhaps surgical resection to reduce the depth of the peri-implant pocket. Surgical resection is generally confined to implants placed in non-aesthetic sites. Probing depths of 4 or 5mm may be caused by tissue swelling and can often be corrected by improvement of peri-implant plaque control. The presence of pus or pockets greater than 5mm indicates that additional measures may be required, including application of antiseptics, such as 2 per cent chlorhexidine or 3 per cent w/v hydrogen peroxide. In addition, local or systemic antibiotics may be considered. The decision for local or systemic

Fig 1A. A length of floss has been threaded through the mesial contact point between the implant crown and the adjacent natural tooth, and looped back through the distal contact point (Ultradent; Oral-B Laboratories, Sydney, Australia).

Fig 1B. The ends of the floss are crossed over on the labial aspect to encircle the implant crown.

Fig 1C. By apically directing the ends of the floss, and with a slight 'sawing' action, the floss penetrates the sulcus of the peri-implant mucosa to effect submucosal débridement.

antibiotics depends on the distribution patterns of these pathogens, the periodontal conditions of the rest of the teeth and whether the implant problem is localized. Obviously a localized implant problem can be treated by local drug therapy. Lang *et al*.[3] suggest the following antibiotic regimes: systemic ornidazole 500mg bds for 10 days or metronidazole 250mg tds for 10 days or a once daily combination of metronidazole 500mg and amoxicillin 375mg for 10 days. Local application of antibiotics consisted of the insertion of 25 per cent tetracycline fibres for 10 days.[3] Provided that mechanical and antiseptic protocols are followed prior to administering antibiotic therapy, it appears that peri-implant infection may be successfully controlled using antibiotics.[27]

When there is bone loss, the next question is 'How extensive is it?' and can be divided into mild, moderate or severe. Mild bone loss may be treated by cleaning the implants, surgical resection, topical antiseptic treatment, local or systemic antibiotics. Moderate bone loss indicates the same treatment for mild, but open débridement should be considered. This surgical approach is associated with recession with possible exposure of the neck of the implant fixture and consequent aesthetic problems. Bone grafting may be considered to fill the infrabony component of the peri-implant bone defect. Lastly, advanced bone loss may indicate cleaning the implant, oral hygiene instruction, local and/or systemic antibiotic delivery, open débridement or explantation. If a decision has been made to remove the implant, explantation trephines are available to suit the implant system concerned. It should be noted that these trephines have an external diameter of up to 1.5mm greater than the diameter of the implant to be removed. Thus explantation may be associated with significant bone removal including buccal or lingual bone cortices, and damage to adjacent natural teeth where the inter-radicular space is limited. An alternative approach is to allow progressive bone loss from peri-implantitis to occur, resulting in sufficient bone loss to allow removal of the implant with extraction forceps. In the experience of the authors, implants may be removed by forceps when there is less than 3 to 4mm of residual bone support.

Incomplete surface decontamination seems to be a major problem in implant maintenance. The screw thread makes scaling difficult and the presence of the periopathogenic bacteria is associated with a poor response to guided tissue or bone regeneration.[33] As a result, there is little evidence of true re-osseointegration in humans. However, there is early experimental evidence to suggest that re-osseointegration may be possible following appropriate decontamination procedures of sand-blasted and acid-etched implant surfaces.[34] If an implant does not respond to treatment, the evidence suggests that rather than trying to save the failing implant, it would be better to remove it and place another fixture once the site has healed.

Case study 1

A 34-year-old male patient presented with an implant replacing the upper right central incisor tooth. The implant had been restored with a crown 12 months previously. On presentation, slight oedematous swelling and loss of stippling of the marginal peri-implant mucosa was noted (Fig 2A). A probing pocket depth of 3mm was found on gentle probing of the peri-implant

Fig 2A. Slight oedematous swelling and loss of stippling of marginal peri-implant mucosa of an implant replacing 11.

Fig 2B. Probing pocket depth of 3mm.

Fig 2C. Bleeding after gentle probing.

pocket (Fig 2B). Within a minute of probing the pocket, bleeding was noted (Fig 2C). Radiographic examination confirmed that there was no loss of crestal bone. On this basis, a diagnosis of peri-implant mucositis was made. Treatment carried out included submucosal débridement with floss and re-inforcement of home-care methods.

Case study 2

A 55-year-old female presented with a fluctuant swelling on the buccal aspect of an implant at the 15 site. The swelling had been present for one month. Initial discomfort and slight swelling were first noticed by the patient about a week after the crown had been cemented into place three months previously. Due to an extended trip overseas, she was unable to have the problem attended to until her return. Clinical

Fig 3A. Fluctuant swelling and 6mm pocket associated with implant at 15 site.

Fig 3B. Radiograph showing slight loss of crestal bone on the mesial aspect.

Fig 3C. Post-therapy probing depths were reduced to 3mm with no bleeding.

Fig 4A. Purulent discharge and swelling of mucosa around implant at 11 site.

Fig 4B. Radiograph showing circumferential bone loss to the fourth implant thread.

Fig 4C. Radiograph of same implant at the time of second stage surgery confirming that bone levels were initially at the height of the implant collar.

Fig 4D. Appearance of peri-implant mucosa following five days of antibiotic therapy.

examination confirmed the presence of a pointing abscess and a pocket of 6mm on the buccal aspect of the implant (Fig 3A). Radiographic examination showed that there was slight loss of crestal bone on the mesial aspect (Fig 3B). A diagnosis of an acute abscess associated with peri-implantitis was made. Likely aetiological factors included food impaction, floss impaction or excess cement. The pocket was débrided with plastic curettes, and the abscess drained. A course of antibiotics was prescribed (amoxycillin 500mg tds for five days). On reviewing the implant six weeks later, complete resolution of the abscess was noted. Peri-implant probing pockets were 3mm with absence of bleeding after probing (Fig 3C). No further treatment was required apart from routine recall and maintenance care.

Case study 3

A 42-year-old male patient presented with swelling of the mucosa around an implant at the 11 site. The implant had been placed three years previously and restored with a porcelain crown cemented on to a ceramic abutment. Clinical examination confirmed the

Fig 4E. Appearance of implant and surrounding bone upon elevation of buccal flap. Implant is exposed to the fourth thread.

Fig 4F. Appearance of implant and surrounding bone on elevation of a palatal flap.

Fig 4G. Closure of flap following débridement and cleaning. Note the position of the gingival margin.

Fig 4H. Appearance of implant and mucosal tissus at 18 months following surgery. Note the position of the gingival margin, two to three millimetres of recession is apparent.

presence of pockets of 5 to 9mm circumferentially. A purulent discharge from the peri-implant crevice was seen on palpation (Fig 4A). Circumferential bone loss up to the fifth implant thread was noted (Fig 4B). A comparison with a baseline radiograph taken soon after second-stage surgery (Fig 4C) confirmed that bone loss had taken place, thus confirming the diagnosis of peri-implantitis. After discussing treatment options with the patient, it was decided that an attempt be made to treat the infection by open flap débridement, bearing in mind that marginal tissue recession would be likely to occur. A combination of antibiotics was prescribed for 10 days, commencing five days pre-operatively (200mg metronidazole tds and 500mg amoxylcillin tds). On the fifth day of systemic antibiotic therapy, the condition of the peri-implant mucosa appeared much improved (Fig 4D). Buccal and palatal flaps were raised to expose the contaminated implant surface (Fig 4E and 4F). The implant surface was carefully débrided to remove granulation tissue, and disinfected with alternating applications of 3 per cent hydrogen peroxide and 2 per cent chlorhexidine gel (Periogard, Colgate Oral Care, Sydney, NSW) with each application flushed away with copious saline irrigation. The chemical agents were rubbed over the titanium surface with fine cotton tip applicators. Flaps were closed with 5/0 chromic gut suture (Fig 4G). After 18 months, the peri-implant tissues were healthy with no evidence of inflammation, bleeding or suppuration. However, significant recession of the marginal mucosa had occurred (Fig 4H).

CONCLUSION

In this article we have discussed the background, aetiology, diagnosis and management of osseointegrated implants. The three cases illustrate a number of points that should be considered in the treatment of peri-implant mucositis and implantitis, and are those that one may see in practice. The increasing acceptance of implant placement as a standard treatment option for patients will mean that more and more dentists will be involved in the long term care and maintenance of these implants.

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