2 Teeth and implants

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Clinicians who use dental implants in the treatment of their patients require an understanding of the nature of osseointegration and the important fundamental differences between dental implants and natural teeth. The main comparisons are summarised in Table 1 and illustrated in figure 1 which shows a single tooth implant and the adjacent natural teeth. The tooth originally formed within the jaws and erupted through the overlying mucosa in a complex series of biological events that are by no means fully understood. The implant on the other hand was surgically placed within the jaw bone, and is one of the few prosthetic devices that has been shown to successfully and permanently breach the surface epithelium with minimal or no complications.

An osseointegrated implant restoration may closely resemble a natural tooth. However, the absence of a periodontal ligament and connective tissue attachment via cementum, results in fundamental differences in the adaptation of the implant to occlusal forces, and the structure of the gingival cuff.

Gingiva versus periimplant soft tissues

In healthy teeth the gingival margin is located on enamel. The gingival margin is scalloped and forms a shallow sulcus at the tooth surface. The gingiva rises between the teeth to form the interdental papillae, which are complex structures. Between the anterior teeth the papillae are pyramidal structures with the attachment of the gingivae following the contour of the cement enamel junction (fig. 2). In the molar regions, the buccal and lingual papillae at natural tooth embrasures are separated by the 'col', an area of gingivae which forms a slight dip beneath the contact point. A complex array of gingival connective tissue fibres form well defined bundle groups:

- Interdental fibres
- Dento-gingival fibres
- Circular fibres
- Alveolar crest fibres.

Many of these fibres are inserted into the root cementum between the alveolar crest and cement enamel junction, and are therefore dependent upon the presence of natural teeth.

In the case of an implant, a transmucosal element (an abutment, neck of the implant or the restoration) protrudes through the overlying mucosa which heals and adapts around it without a cementum attachment. The collagen fibres within the periimplant mucosa run parallel to the abutment with no insertion into the abutment surface. There have been descriptions of more ordered fibre arrangements in relation to transmucosal implant surfaces which have a rougher surface (such as plasma spraying). In this situation some fibres appear to run at right angles to the implant surface, but there is no good evidence of an attachment mechanism. However a rough abutment surface does have potential negative properties,

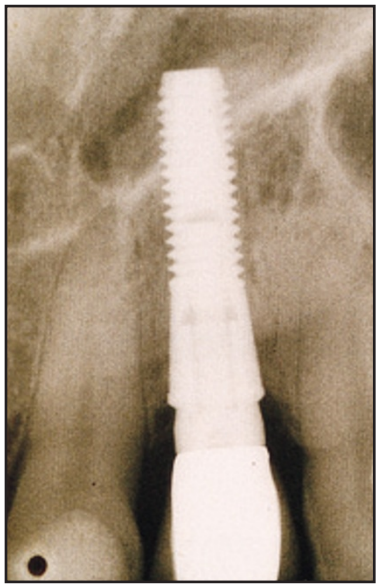

Fig. 1b Radiograph of the single tooth implant and adjacent teeth. The bone contacts the implant surface with no intervening radiolucent space which would be observed if there were fibrous tissue encapsulation. The bone margin is coincident with the implant/abutment junction. The adjacent teeth have a normal periodontal ligament space

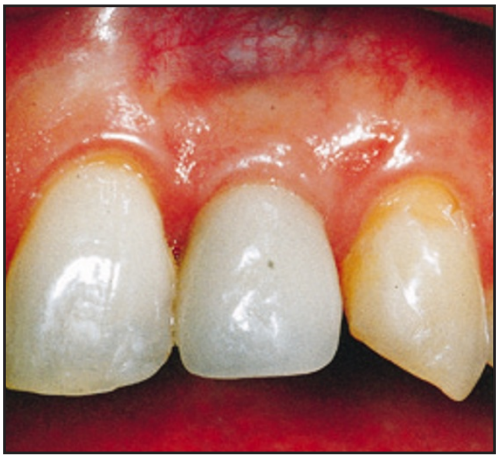

Fig. 1a. Clinical photograph of a single tooth implant replacing the upper left lateral incisor. The porcelain fused to metal crown appears to emerge from the gingiva with interdental tissue which appears very similar to normal papillae

In this part, we will discuss:

- Gingiva versus periimplant soft tissues
- Periodontal ligament versus osseointegration
- Periodontitis and peri-implantitis

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Gingiva versus periimplant soft tissues
- Junctional epithelium
- Biological width
- Probing depth examination

Table 1 Healthy teeth versus healthy implants

	Healthy teeth	Healthy implants
Gingival sulcus depth	Shallow in health	Dependent upon abutment length and restoration margin
Junctional epithelium	On enamel	On titanium
Gingival fibres	Complex array inserted into cementum above crestal bone	No organised collagen fibre attachment – parallel fibres
Crest of bone	1 to 2 mm apical to CEJ	According to implant design eg at or about first thread in threaded implants or at the level of change in surface morphology
Connective tissue attachment	Well organised collagen fibre bundles inserted as Sharpey's fibres into alveolar bone and cementum	Bone growing into close contact with implant surface: oxide layer/ bone proteoglycan and collagen
Physical characteristics	Physiologic mobility caused by viscoelastic properties of the ligament	Rigid connection to bone - as if ankylosed
Adaptive characteristics	Width of ligament can alter to allow more mobility with increased occlusal forces	No adaptive capacity to allow mobility. Orthodontic movement impossible
Proprioception	Highly sensitive receptors present within the periodontal ligament	No ligament receptors

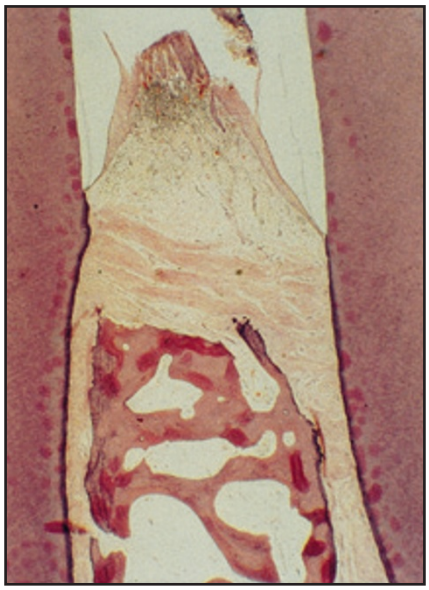

Fig. 2 A histological section of an interdental space between two teeth. The enamel has been removed by the demineralisation process. The junctional epithelium outlines the enamel space and terminates at the level of the root cementum. The interdental bone septum is situated just below the cement enamel junction (in health 1–2 mm) and there is a well developed transeptal fibre arrangement. There is a small inflammatory infiltrate in the gingival connective tissue at the top of the papilla

such as increased corrosion potential and microbial contamination if it becomes exposed within the oral cavity.

The papillae which form around a single tooth implant may be supported by collagen fibres attached to the adjacent natural teeth. However, in cases where there are adjacent implants rather than teeth, the formation of soft tissue papillae is less predictable and their form is dependent upon the presence of an adequate thickness of soft tissue, bone height, implant spacing and careful contouring of the crown profiles to encourage the appearance and maintenance of a papillary form (fig. 3). The soft tissue between multiple posterior unit implants is more likely to have a flat contour but again may be influenced by soft tissue thickness and crown morphology.

Junctional epithelium

In healthy teeth the junctional epithelium (fig. 4) is attached to enamel by hemidesmosomal contacts and a basal lamina-like structure formed by the epithelial cells. The biological attachment mechanism is now thought to be mediated through particular adhesins or integrins, which are fundamental in cell to cell adhesion as well as cell to matrix adhesion. It is well established that a junctional epithelium

will also form on root surface cementum, dentine and various dental materials including implant components (fig. 5). A normal junctional epithelium can be regenerated from adjacent oral mucosa/gingiva following excision, and the new junctional epithelium is indistinguishable from that which previously existed. It is thought that the properties of the junctional epithelium are dictated by the influence of the underlying connective tissue, the presence of an inflammatory infiltrate and the presence of a tooth/implant surface to which it adheres (rather than the inherent properties of the epithelial cells). The junctional epithelium has a particularly high turnover and is permeable to both the ingress of substances and to components of the immune and inflammatory system. It is therefore well equipped to deal with the problems of a breach in the epithelial integrity caused by an emerging tooth or implant. The junctional epithelium may be found on the implant itself or on the abutment. This will be because of differences in the designs of implants, the biological requirements of the attachment of the soft tissue cuff and the level of the junction between abutment and implants.

Biological width

In teeth, the concept of the biological width is well established, in that a zone of attached connective tissue separates the underlying alveolar bone from the apical termination of the junctional epithelium (fig. 6a). The connective tissue zone is about 2 mm wide and the length of the junctional epithelium about 1.5 mm. Figures 6b and c show two different designs of implants and the corresponding biological width. In the first case the implant design is typical of a submerged (two stage) system such as the Branemark. After 1 year of function the bone margin is usually located at the first thread. The junctional epithelium (1.5 mm to 2 mm apicocoronal width) is located on the abutment, and a zone of non-arranged connective tissue of about 1mm to 2 mm in width intervenes. The join between abutment and implant head is located within this zone. In contrast the non-submerged (single stage) implant (typical of the ITI Straumann type) is placed so that its roughened surface is placed within bone, but the smooth neck which is an integral part of the implant performs the function of the transmucosal element. The junctional epithelium is therefore routinely located on the implant, and the implant/abutment join is located coronal to this level. It has been postulated that the join within the submerged (two stage) system may influence the level of soft tissue attachment and biological width. This may be caused by micromovement between the two components or by allowing microbial penetration of the microgap between

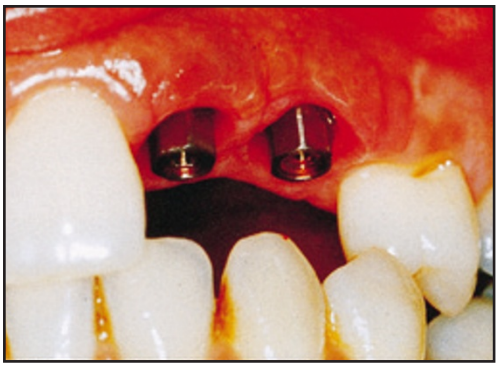

Figure 3a. Two hexagonal abutments used to support single implant crowns emerging through a cuff of gingiva. The space around them has been created by a larger healing abutment which has been replaced by the hexagonal abutment. The gingival tissue between the abutments has a form which resembles a normal papilla but is flatter and is not supported by a normal gingival fibre arrangement

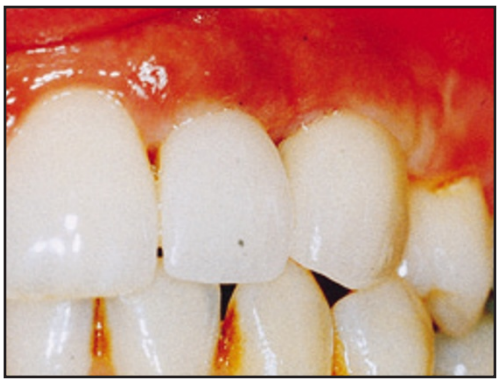

Fig. 3b The porcelain fused to metal crowns have been cemented onto the abutments. The emergence of the crowns from the soft tissue produces a natural looking appearance

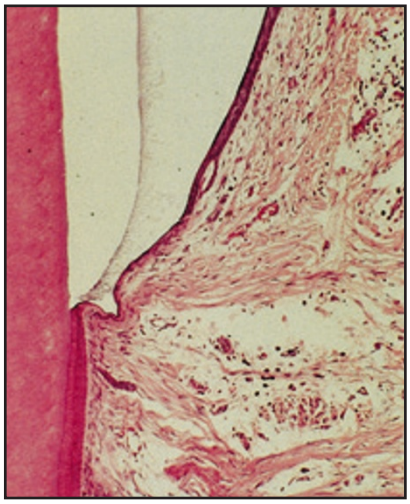

Fig. 4. A histological section of junctional epithelium at a natural tooth. It terminates at the cement enamel junction and was attached to the enamel by hemidesmosomes and a basal lamina-like structure. Collagen fibres are inserted into the cementum and radiate into the gingival connective tissue

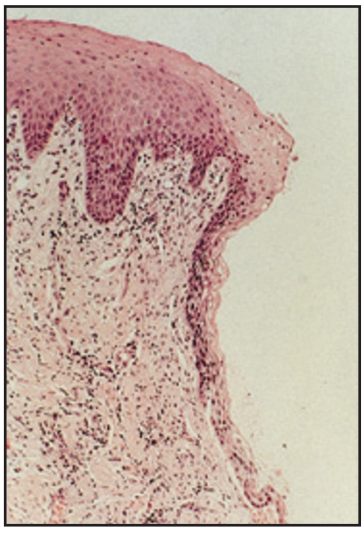

Fig. 5 A histological section of the soft tissue cuff excised from around an implant. A non-keratinised sulcular and junctional epithelium is present and is very similar to that which exists around teeth. The collagen fibre bundles are not so well organised as there is no attachment to the abutment/implant surface

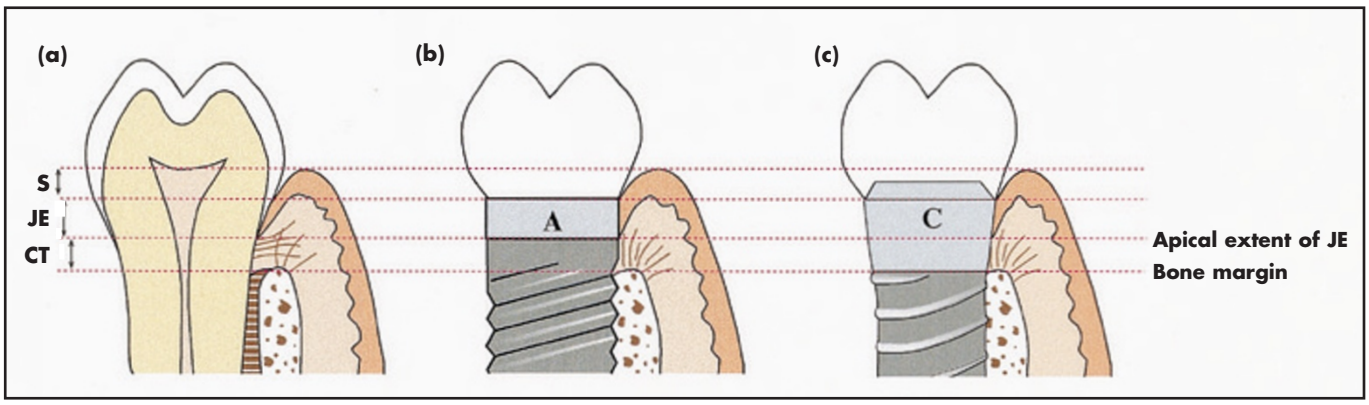

Fig. 6 a,b,c The biological width of the dentogingival junction in (a) teeth and (b) around implants typical of the Branemark system, and (c) the non-submerged ITI implant system. S= sulcus which is approximately 0.5 to 1 mm deep; JE = junctional epithelium which is about 1.5 to 2 mm in apicocoronal width; CT = Connective tissue zone (1 to 2 mm in width) in which the fibres are attached to root cementum in teeth but run parallel to the implant surface; A = abutment — The abutment to implant junction is situated beneath the soft tissue in the Branemark system; C = smooth transmucosal collar of the IT system

Periodontal ligament versus osseointegration
- Periodontal ligament
- Osseointegration

implant and abutment. At present the theoretical differences between the two types do not reveal any major differences at the histological level or in their clinical performance.

Probing depth examination

Periodontal probing of natural teeth is an important part of any dental examination. It is well established that the probe penetrates the junctional epithelium to some degree in health, and that this penetration increases in the presence of inflammation. Under these latter circumstances the probe is stopped by the most coronal intact gingival connective tissue fibres, about 2 mm from the bone. The situation around the dental implant is different and the sulcus depth is very much dependent upon the thickness of the soft tissue cuff. Probing depths around implants are generally deeper than around teeth, but penetration of the soft tissue at the base of the sulcus occurs to a similar degree with the probe tip finishing short of the bone margin by about 2 mm. The information gained from probing around implants is of questionable value and many clinicians do not recommend probing, preferring to rely on radiographic assessment of bone levels. In addition, digital pressure on the external surface of the periimplant soft tissue may elicit signs of inflammation such as bleeding or suppuration.

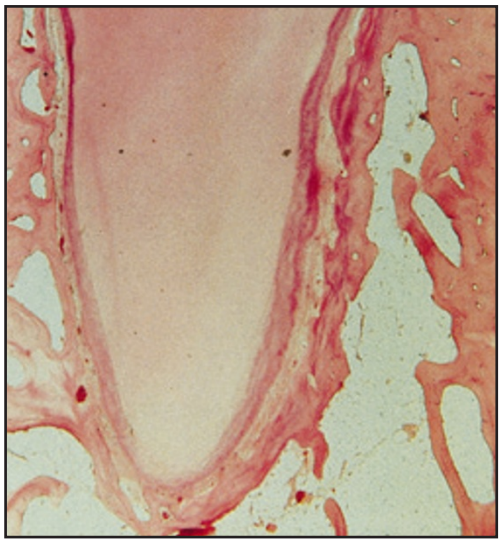

Fig. 7 A histological section of a tooth root, periodontal ligament and alveolar bone. The periodontal ligament is inserted into the cementum and the lamina dura as Sharpey's fibres. The viscoelastic properties of the ligament give the tooth a degree of mobility and the ligament is able to respond to increased forces by remodelling processes

Periodontal ligament versus osseointegration

Periodontal ligament

The periodontal ligament is a complex structure, about 0.1 to 0.2 mm in width, providing support to the teeth in a viscoelastic manner (fig. 7). The ligament comprises collagen fibres which are embedded as Sharpey's fibres in the root cementum and the alveolar bone, together with the blood supply and connective tissue ground substance which provide the other key elements to the supporting mechanism. The periodontal ligament has a sensitive proprioceptive mechanism which can detect minute changes in forces applied to the teeth. Forces applied to the teeth are dissipated through compression and redistribution of the fluid elements as well as through the fibre system. Forces transmitted through the periodontal ligament can result in remodelling and tooth movement as seen in orthodontics or in the widening of the ligament and an increase in tooth mobility in response to excessive forces (eg occlusal trauma). The periodontal ligament is therefore capable of detecting and responding to a wide range of forces.

Osseointegration

The precise nature of osseointegration at a molecular level is not fully understood. At the light microscopical level there is a very close adaptation of the bone to the implant surface (fig. 8). At the higher magnifications possible with electron microscopy, there is a gap (about 100 NM in width) between the implant surface and bone. This is occupied by an intervening collagen rich zone adjacent to the bone and a more amor-

phous zone adjacent to the implant surface. Bone proteoglycans may be important in the initial attachment of the tissues to the implant surface, which in the case of titanium implants consists of a titanium oxide layer, which has the properties of a ceramic. Osseointegration is not an absolute phenomenon and can be measured as the proportion of the total implant surface that is in contact with bone. Greater levels of bone contact occur in cortical bone than in cancellous bone, where marrow spaces are often adjacent to the implant surface. The degree of bone contact may increase with time and function. When an implant is first placed in the bone there should be a close fit to ensure stability. The space between implant and bone is initially filled with blood clot and serum/bone proteins. Although great care is taken to avoid damaging the bone, the initial response to the surgical trauma is resorption, which is then followed by bone deposition. There is a critical period in the healing process at around 2 weeks post implant insertion when bone resorption will result in a lower degree of implant stability than that achieved initially. Subsequent bone formation will result in an increase in the level of bone contact and stability. This has been demonstrated in unloaded implants in the early healing period and over longer time periods following loading of the implant. Thus osseointegration should be viewed as a dynamic process in which bone turnover occurs, but not as the same adaptive process that occurs within the ligament of natural teeth. Osseointegration is more akin to an ankylosis, where the absence of mobility and no intervening fibrous tissue capsule is the sign of successful integration. Under these circumstances there is no viscoelastic damping system although proprioceptive mechanisms may operate within bone and associated oral structures. Forces are distributed to the bone and may be concentrated in certain areas, particularly around the neck of the implant. Some designs, particularly those with threads, may dissipate the forces more effectively. Excessive forces applied to the implant may result in remodelling of the marginal bone ie apical movement of the bone margin with loss of osseointegration. The exact mechanism of how this occurs is not entirely clear but it has been suggested that microfractures may propogate within the adjacent bone. This type of bone loss caused by excessive loading may be slowly progressive to a point where there is catastrophic failure of the remaining osseointegration or fracture of the implant. Fortunately, either eventuality is rare. Excessive forces are usually detected prior to this stage through radiographic marginal bone loss or mechanical failure of the superstructure and/or abutments (See Part 10).

It has been shown however, that well controlled forces result in an increase in the degree of bone to implant contact and remodelling of adjacent trabecular structures to dissipate the forces. Adaptation is therefore possible, though osseointegration does not permit movement of the implant in the way that a tooth may be orthodontically repositioned. Therefore the osseointegrated implant has proved itself to be a very effective anchorage system for difficult orthodontic cases, and may be used as an alternative anchorage system to head gear. The fact that the implant behaves as an ankylosed unit also restricts its use to individuals who have completed their jaw growth (fig. 9). Placement of an osseointegrated implant in a child will result in relative submergence with growth of the surrounding alveolar process during normal development. It is therefore advisable to delay implant placement until after growth is complete.

Fig. 8a A histological section through an osseointegrated screw shaped implant which has been in place for 6 months. Bone is in close apposition over a large proportion of the surface

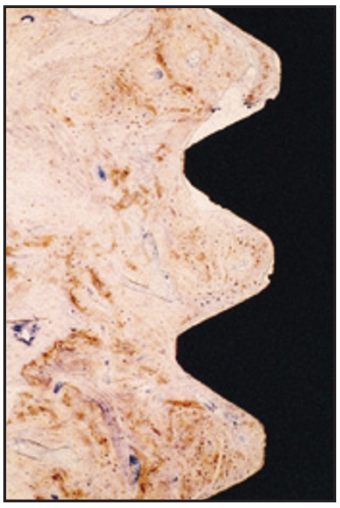

Fig. 8b A higher power view of an area of figure 8a showing bone filling the thread profiles and contacting the implant surface without a visible gap (at this magnification), except for a small area of marrow space

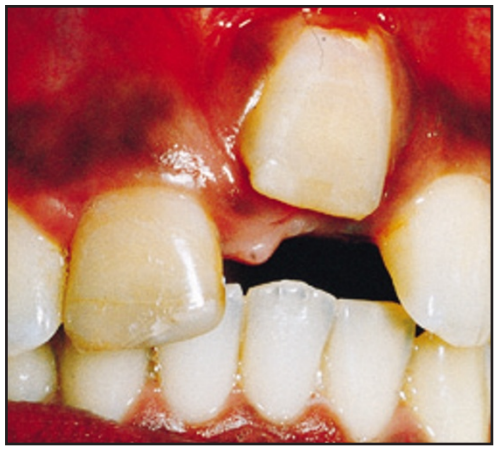

Fig. 9 An ankylosed tooth following trauma. Damage to the periodontal ligament has led to a boney ankylosis and resorption. The tooth has no detectable mobility and has not developed into a normal vertical position with the adjacent teeth. In this respect it is behaving like an osseointegrated implant. An osseointegrated implant should not be placed in a child until growth is complete

Periodontitis and peri-implantitis

It is quite possible that bacteria which are implicated in periodontitis, such as *Porphyromonas gingivalis,* are also the major pathogens in destructive inflammatory lesions around implants (peri-implantitis). There is

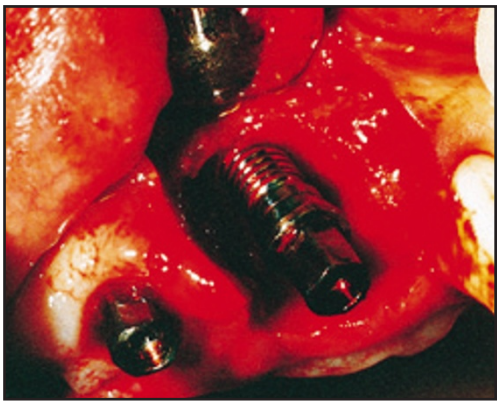

Fig. 10 An exposed implant following destruction of the most coronal bone by an inflammatory infiltrate. There was a plaque induced inflammation caused by retention of cement at the crown abutment junction which was situated subgingivally

therefore a possibility of colonisation or infection of the implant surfaces from pre-existing periodontopathic bacteria. The destruction of the supporting tissues of teeth and implants have many similarities but there are important differences caused by the nature of the supporting tissues (see earlier). This is particularly noticeable with the different patterns of tissue destruction observed. Peri-implantitis affects the entire circumference of the implant resulting in a 'gutter' of bone loss filled with inflammatory tissue extending to the bone surface (fig. 10). In contrast, periodontitis-affected teeth commonly have irregular loss of supporting tissues, often confined to proximal surfaces and resulting in complex infrabony defects. In addition, for the most part the periodontal tissues are capable of 'walling off' the inflammatory lesion from the alveolar bone and periodontal ligament with a zone of fibrous tissue. It would seem probable that destructive inflammatory lesions affecting both teeth and implants have stages in which the disease process is more rapid (burst phenomenon) followed by periods of relative quiescence. The incidence of peri-implantitis would appear to be low, but can result in rapid destruction of the marginal bone and is difficult to differentiate from bone loss because of excessive forces. This problem is dealt with in Part 10.

Conclusion

Modern osseointegrated implants are a useful alternative to natural teeth. There are fundamental differences between them, and an understanding of the attachment mechanisms of hard and soft tissues and their responses to the harsh environment of the oral cavity is essential to the dental surgeon who is involved in providing this form of treatment.