

Do long-term followed-up Brånemark™ implants commonly show evidence of pathological bone breakdown? A review based on recently published data

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The first patient provided with osseointegrated oral implants was treated in 1965, but implant treatment first became a routine procedure during the 1980s. Since then, thousands of patients have undergone restoration with implants in the partially and fully edentulous jaw and encouraging reports on the treatment results have been published (1, 3, 11, 19, 20, 24, 27, 36, 37, 46). However, during the past 10 years an increasing number of scientific papers have been published on implants associated with peri-implant bone loss, in some cases referred to as peri-implantitis (12, 18, 21, 25, 26, 44). Radiographic data on bone loss provide essential information for classification of implant outcome and the present authors are critical of any criteria for success that omit the reporting of marginal bone loss (18, 21). Studies that present detailed radiographic data on both group and individual levels in their analyses of the prevalence of continuous bone loss generally describe different criteria for the inclusion of affected implants, resulting in prevalences from 7.7% to 39.7% in long-term follow-up studies (12, 25, 26, 44). Whether or not these prevalences are comparable among studies is an important question for proper risk analyses of implant patients. The purpose of the present review is to analyze the impact of different radiographic inclusion criteria in the same patient group and to analyze the concept of

continuous bone loss and its potential relation to peri-implantitis.

Long-term papers by Bryant (19) and Jemt & Johansson (36) reporting marginal bone loss around Brånemark™ oral implants

Bryant (19) compared a matched series of 140 Brånemark™ implants (Nobel Biocare AB, Göteborg, Sweden) placed in an older cohort (60–74 years of age at surgery) with 133 implants placed in a younger cohort (26–49 years of age at surgery). There was no difference in mean crestal bone loss between the two groups at follow-up times of up to 10 years. Furthermore, it was noted that average crestal bone level at loading was lower (more apical) in maxillary fixed prostheses than in mandibular cases. During the first year of loading, mean annual bone loss was 0.17 mm for the old group and 0.24 mm for the young group. Bryant (19) observed that an annual bone loss of 0.2 mm would have meant that 2.0 mm of bone was lost at 10 years and 3.4 mm of bone at 17 years, the maximal follow-up length of the study. However, in reality the mean annual bone loss following the first

year was 0.043 mm for the older and 0.041 mm for the younger patient group, resulting in accumulated averages around 0.5 mm at 10 years and, only in the young group, slightly more than 1 mm at 17 years. The older patient group had a cumulative bone loss pattern approaching 0 mm per year in the mandible after 4 years. Including both jaws, the older group demonstrated an annual average bone loss of 0.022 mm and the young group 0.016 mm per year after the 4-year period.

The Jemt and Johansson material has been presented in detail in 5- and 15-year follow-up publications (36, 37). In the present review, we have analyzed the same material, but limited the follow-up period to 10 years. The present 10-year group covers all patients consecutively provided with fixed prostheses supported by implants in the edentulous maxilla and treated at the Brånemark Clinic, Gothenburg, Sweden from 1 January 1986 to 31 December 1987 (36, 37).

In total, 76 patients were included, 48 of them male. Mean patient age at the time of implant surgery was 60.1 years (SD 11.6 years), with a range of 32 to 75 years. In total, 450 turned, titanium Brånemark implants (Nobel Biocare) were placed

according to a two-stage surgical protocol (2, 36, 37). Abutment connection surgery was performed after a healing period of 6–8 months. Thereafter, all patients were treated with fixed prostheses, designed with a cast Type II gold-alloy framework supporting conventional acrylic resin teeth (34, 37, 49). After insertion and final tightening of the bridge locking screws 2–6 weeks later, the patients were scheduled for check-up visit after 1, 5, and 10 years (Table 1), but were encouraged to contact the treating clinic if they experienced problems with their prostheses (34, 36). Intra-oral apical radiographs were obtained at the oral and maxillofacial radiological specialist clinic (Public Dental Health Service, Göteborg, Sweden) at the time of prosthesis insertion and after 1, 5, and 10 years of use. Bone loss was measured in relation to the threads of the implants to the closest 0.3 mm on the mesial and distal sides of the implant. A mean value between the mesial and distal side was used for each implant. The reference for these measurements was the fixture–abutment junction, placed 0.8 mm coronal to the implant reference point, as used in previous studies (29, 46). Bone level and bone loss were measured at different time intervals.

Table 1. Mean marginal bone level in relation to fixture–abutment junction (FAJ) during the follow-up period

	Follow-up periods		
	0–1 year	0–5 years	0–10 years
Patients	71	62	41
Implants	402	350	238
Bone level in relation to FAJ (mm)			
Mean	1.8	2.0	2.0
SD	0.60	0.58	0.61
Number of implants (%)			
Bone level to FAJ			
<0.9 mm	65 (16.2)	47 (13.4)	36 (15.1)
0.9–1.9 mm	208 (51.7)	163 (46.6)	100 (42.0)
2.0–3.0 mm	94 (23.4)	109 (31.1)	70 (29.4)
3.1–3.8 mm	25 (6.1)	24 (6.9)	25 (10.5)
> 3.8 mm	10 (2.5)	7 (2.0)	7 (2.9)
Number of patients with 0/1/2/3/ >3 implants with bone level three or more threads apical to FAJ			
	54/8/3/4/2	43/10/6/3/–	23/11/3/1/3

Percentage of implants is given in parenthesis.
Data from Jemt & Johansson (36).

Forty-one patients had radiographs available for consecutive analyses from prosthesis placement to the 1-year, 5-year, and 10-year follow-up times. Implants with a marginal bone level of more than 3 mm below the fixture–abutment junction (corresponding to the third thread of the implant) accounted for 8.7% at the 1-year and 13.4% at the 10-year follow-up visit (Table 1). The corresponding number of patients with at least one implant with a bone level of more than 3 mm below the fixture–abutment junction increased from 23.9% at 1 year to 43.9% at 10 years. Average bone loss was 0.4 mm (SD 0.31 mm) at 1 year and 0.5 mm (SD 0.47 mm) at 5 years of loading (Table 2). Thereafter, only small changes of average bone loss were observed at 10 years follow-up (Table 2). The proportion of implants showing more than 2.0 mm of bone loss was 2.0% during the first 5 years and 4.7% during the 10 years of follow-up (Table 2). The corresponding proportions of patients with at least one implant with more than 2.0 mm of bone loss were 4.9% and 24.3% at 5 and 10 year follow-up, respectively. A bone loss of more than 3.0 mm was detected for 1.3% of the implants during the 10-year study period.

Long-term follow-up studies by Fransson et al. (26) and Roos-Jansåker et al. (44) reporting marginal bone loss around Brånemark implants

Fransson et al. (26) analyzed radiographs from 1,346 patients who had attended follow-up visits at the Brånemark clinic, Gothenburg, Sweden during 1999. All patients had been treated with turned, Brånemark™ implants (Nobel Biocare). Only patients with a minimal follow-up of 5 years were included, and patients with overdentures, bone grafts, or barrier membranes were excluded. A total of 684 patients were excluded. Bone level was analyzed for the remaining 662 subjects with follow-up periods ranging from 5 to 20 years. Sites that demonstrated a bone level corresponding to the position at, or apical to, the third marginal thread unit of an implant (similar to >3 mm apical to the fixture–abutment junction) were identified. The authors defined *progressive bone loss* as bone-level alterations occurring between the first and the last annual radiographic check-ups, 5 to 20 years after prosthesis placement.

Table 2. Mean marginal bone loss and distribution of implants and patients with regard to bone resorption during different periods of follow-up

	Follow-up periods					
	0–1 year	0–5 years	1–5 years	0–10 years	1–10 years	5–10 years
Patients	70	61	61	41	41	41
Implants	398	346	346	238	238	238
Bone loss (mm)						
Mean	0.4	0.5	0.1	0.6	0.2	0.0
SD	0.31	0.47	0.39	0.59	0.57	0.44
Number of implants (%)						
Bone loss						
<0.1 mm	148 (37.2)	113 (32.7)	196 (56.6)	70 (29.4)	64 (26.9)	134 (56.3)
0.1–1.1 mm	218 (54.8)	179 (51.7)	135 (39.0)	112 (47.1)	126 (52.9)	88 (37.0)
1.2–2.0 mm	27 (6.8)	47 (13.6)	13 (3.8)	45 (18.9)	33 (13.9)	14 (5.9)
2.1–3.0 mm	5 (1.3)	5 (1.4)	2 (0.6)	8 (3.4)	11 (4.6)	1 (0.4)
>3.0 mm	0	2 (0.6)	0	3 (1.3)	4 (1.7)	1 (0.4)
Number of patients with: 0 / 1 / 2 / 3 implants with >2 mm bone loss during the follow-up period						
	66 / 3 / 1 / –	58 / – / 2 / 1	58 / 3 / – / –	31 / 9 / 1 / –	31 / 9 / 1 / –	39 / 2 / – / –

Percentage of implants is given in parenthesis.
Data from Jemt & Johansson (36).

Implants were included in the study irrespective of the magnitude of bone loss.

A total of 184 patients (27.8%) were found to have one or more implants with at least some additional bone loss according to the criteria for inclusion. The remaining 478 patients displayed no implants with bone level below 3 mm in relation to the fixture–abutment junction at final examination, despite presenting measurable bone loss during the first year after implant installment. Forty of the latter patients had one or more implants with a marginal bone loss of at least three threads or more at 1 year, but showed no detectable bone loss thereafter. The follow-up period of the 184 patients varied between 5 and 20 years, with a mean of 9.1 years. Implants placed in the mandible accounted for 62.5% and in the maxilla for 37.5%. One-third of the patients revealed three or more implants with progressive bone loss according to the definition, the proportion of which made up 12.4% of the 3,413 investigated fixtures. The paper did not describe the magnitude of bone loss or whether bone loss increased with increasing times between 5 and 20 years.

Roos-Jansåker et al. (44) analyzed 999 implants clinically and radiographically with a separately reported failure rate of 4.3% at 9–14 years of follow-up. The same clinical material had been analyzed after 1 and 5 years of follow-up. The authors evaluated probing depth and bleeding on probing separate from bone loss. The long-cone radiographic technique was used, with calculations of bone level mesially and distally, with the site showing the most bone loss taken as representing the critical bone level. Initially there was a drop-out of 22 patients who died and 54 patients who left the original study for other reasons. In the final radiographic analysis another two patients with 12 implants refused participation in the radiographic investigations, giving a final number of 987 test implants. In 21% of the implants, the bone level was located at or apical to the third thread, compared to 12% at the 1-year examination. Forty-eight percent of the implants demonstrated probing depths of 4 mm or more with bleeding on probing, with 7% of the implants displaying probing depths of 6 mm or more. With the definition of peri-implantitis used in this study [which included a minimal bone loss of 1.8 mm after the implant's first year in service (21)], 6.6% of implants were identified as having peri-implantitis after 9–14 years. In fact, 7.7% of the implants exhibited bone loss according to the definition, but the criteria for peri-implantitis also included bleeding and/or pus at probing, and the latter finding was absent in 1.1%.

Furthermore, it is noteworthy that 10.7% of the implants showed a radiographic peri-implant bone gain from the first year to final examination with simultaneous bleeding in 8.4% of the sites. The authors observed that with the simple definition of peri-implantitis suggested by Albrektsson & Isidor (5), as many as 43.3% of implants had peri-implantitis, perhaps not surprisingly because the Albrektsson & Isidor (5) criteria did not identify a minimal amount of bone loss to qualify for peri-implantitis.

Are the data of Bryant (19) and Jemt & Johansson (36) in conflict with those of Fransson et al. (26) and Roos-Jansåker et al. (44)?

It is interesting to observe that if using the material of Jemt & Johansson (36), but applying the criteria used by Fransson et al. (26) the study implants ($n = 27$) displayed between 0.3 mm to 4.2 mm of bone loss. If applying the bone loss criteria used by Roos-Jansåker et al. (44) the study implants ($n = 19$) showed from 1.9 mm to 4.2 mm of bone loss in the same time period. Accordingly, the same implants are not included when using inclusion criteria from different studies (Fig. 1). The mean bone loss at 10 years in the study of Jemt & Johansson (36) was 1.7 mm (SD 0.94), if applying the criteria by Fransson et al. (26), but 2.6 mm (SD 0.67) if using the criteria of Roos-Jansåker et al. (44). This difference in mean bone loss was statistically significant ($P < 0.05$), showing results will differ significantly dependent on the inclusion criteria used. Obviously, to tackle the problem of whether or not peri-implantitis is a common phenomenon with long-term implants, it is necessary to assess separately normal bone remodeling in cases without implants, what is acceptable bone loss around implants in quoted criteria for success, and what are the reasons for unacceptable bone loss around implants.

What is acceptable bone loss around teeth and after extraction of teeth?

Hugoson & Laurell (30) recorded a slow, continuous bone loss around teeth, which reached an annual average of 0.1 mm in a longitudinal study population. Since oral maintenance was considered 'very high' in

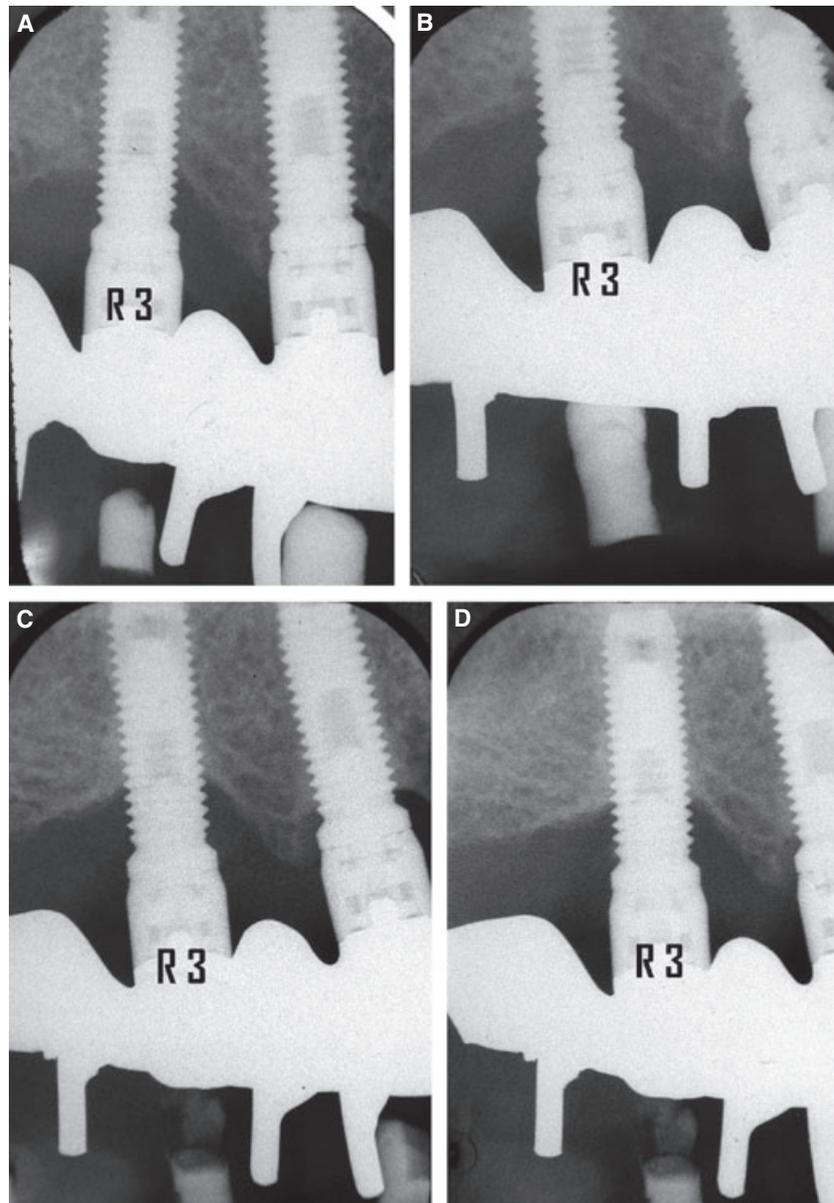


Fig. 1. Fifty-year-old male patient provided with six implants in the edentulous maxilla. Right terminal implant (R3) at the time of prosthesis placement (A), after 1 (B), 10 (C), and 15 (D) years of follow-up. Notice early bone loss at placement of prosthesis (A) with bone level at the fourth (distal) and fifth (mesial) threads of the implants. Same bone level at first annual check-up (B) but with a bone

level at the fifth (distal) and fifth to sixth (mesial) thread at 10th and 15th annual check-ups. Average bone loss of <0.5 mm during 9–14 years would lead to inclusion according to Fransson et al. (26), but not according to Roos-Jansåker et al. (44). Notice also the stable bone level at the adjacent implant during the follow-up period.

the entire patient group, and only a few individuals presented obvious bone loss, they observed that 'these few individuals could hardly explain the gradual, although small, decrease in bone height with increasing age found in all age groups'. Thus they proposed 'it is reasonable to suggest that there is a natural, biological, continuous alveolar bone resorption taking place after the age of 30 years' (30). This slow continuous biological change has been extensively discussed in the orthodontic literature,

implicating obvious skeletal growth and tooth migration as well as tooth eruption (4, 14), and has more recently been addressed in the prosthetic literature in relation to growth and implant infra-position (16, 35, 41). For comparison, Papanou et al. (42), who examined periodontal disease progression, observed a mean crestal bone loss around diseased teeth of 0.3 mm per year among subjects who were at least 70 years of age at the onset of a 10-year follow-up study.

Tooth extraction and subsequent treatment with dentures have shown significant level of alveolar crest resorption, which was most pronounced during the early phases in long-term follow-up studies (15, 22, 45).

Accordingly, continuous skeletal growth/remodeling and tooth migration as well as tooth eruption must be taken into consideration when evaluating bone loss around teeth in long-term follow-up studies. As a consequence, even small continuous changes of bone levels around teeth may after several decades reach clinically significant levels in a few healthy subjects.

'Acceptable' bone loss around osseointegrated implants and definition of peri-implantitis

The evidence from the studies of Jemt & Johansson (36) and Bryant (19) is, in fact, that bone is not, on average, lost around the osseointegrated oral implants at all or only in small proportions of a millimeter each year, provided follow-up periods of 10 years or more are considered. In fact, the evidence points to a steady-state bone situation at least in the mandible (19), which of course does not exclude the possibility that individual patients with implants may display a pattern of more substantial bone loss. The most cited criteria for success of oral implants in literature define 'normal' bone loss as follows:

Albrektsson et al. (7), supplemented with Albrektsson & Zarb (6) and supported by Roos et al. (43), accepted a vertical bone loss of 1 mm during the first year of function, followed by an annual bone loss <0.2 mm after the first year in service. This implies that for a successful implant, bone loss should be limited to <1.8 mm at 5 years and to <2.8 mm at 10 years follow-up visits.

Albrektsson and Isidor (5) accepted a first year bone loss of 1.5 mm, followed by the same <0.2 mm annual bone loss suggested by Albrektsson et al. (7). This implies that bone loss for a successful implant should be <2.3 mm at 5 years and <3.2 mm at 10 years follow-up visits.

Ellegaard et al. (25) and Baellum & Ellegaard (12) identified ailing implants as those with a bone loss of >1.5 mm at 5 years and >3.5 mm at 10 years of follow-up. They measured bone level at the mesial and distal sites of the implants and then rounded off to the nearest 0.5 mm. However, so far, there are no indications in the literature that bone loss above

these levels would ultimately lead to implant failures within a given time period.

How has peri-implantitis been defined?

Albrektsson & Isidor (5) defined peri-implantitis as inflammation 'with loss of supporting bone in the tissues surrounding a functioning implant'. According to this definition, any sign of bone loss (even <0.2 mm annually) with inflammation may be interpreted as indicative of peri-implantitis, but it is noteworthy that the same authors in the same paper (5) accepted a bone loss of 1.5 mm during the first year and >0.2 mm annually thereafter as acceptable for implant success.

Roos-Jansåker et al. (44) defined peri-implantitis as implants demonstrating bleeding on probing and/or pus combined with a total bone loss of 1.8 mm or more during 8–13 years following the first annual check-up. This definition is different from that of Fransson et al. (26), who defined *any* bone resorption after the first year as being indicative of progressive bone loss.

What are the reasons for secondary loss of anchoring bone?

Woelfel et al. (47) described more than 55 different factors that can be considered in relation to bone resorption underneath a complete denture. Secondary bone loss around initially osseointegrated implants has been suggested to depend on anything from normal remodeling to overloading, peri-implantitis, and microgap leakage (40). In fact, there seems to be a difference in opinion that is dependent on the affiliation of the researchers. At a consensus meeting arranged by the prosthodontic department in Toronto, secondary pathological bone loss around implants was deemed dependent on overloading with rare instances of peri-implantitis as a possible side phenomenon. Another consensus meeting arranged the very same year by periodontists came to the opposite conclusion – that peri-implantitis was the major cause of secondary implant failure and only rarely if at all, was overloading the incriminating factor (38). A potentially clarifying study in monkeys claimed that overloading as well as peri-implantitis may cause implant failures (32, 33). However, again without reaching a perfect consensus, it has been suggested that only severe

over-occlusion resulted in bone loss. Having said this, however, the ligature used in the same monkey study for establishing peri-implantitis is, likewise, everything but a normal finding around implants. In a rabbit experiment, Duyck (23) found unaffected bone volume as a response to static loading, but impaired bone volume in response to dynamic loading. Duyck (23) discussed comparable studies which failed to identify this connection (31, 39), but attributed the conflicting data to too small load applications in the previous studies. Fig. 2 describes a patient who may have lost bone due to overload rather than to peri-implantitis.

Retrieved implants have been used to determine whether peri-implantitis is a major cause of implant failure. As failing implants commonly demonstrate bone loss as well as inflammation/infection (17), the finding of peri-implantitis around failing implants is, in fact, poor evidence of what originally initiated the bony destruction.

The present authors wish to stress that overloading is not the only potential biomechanical complication around implants. Stress shielding, i.e. underloading, and inflammation may also cause implant failure. Orthopedic surgeons recognize that bone that is not physically loaded tends to resorb.

Discussion with our own opinions

In 1991, Zarb and Albrektsson wrote an editorial entitled 'Osseointegration – the requiem for the periodontal ligament' and were bold enough to publish the editorial in a periodontally oriented journal (48). Our colleagues in the discipline of periodontology did not agree then any more than they do today. At the time, periodontists emphasized that the prevailing opinion of the early 1990s of minute long-term bone resorption around Brånemark implants was entirely based on the early evidence of osseointegration in totally edentulous individuals; once more experience from single units emerged, it would be obvious that implants became infected from nearby teeth, and hence would show a high incidence of peri-implantitis. However, time has revealed that this was a misconceived concept, as single implants show clinical results as good as those previously reported for fully edentulous patients (8, 28). What is interesting today, is that the old notion that Brånemark turned implants were only rarely struck by progressive bone loss/peri-implantitis is now being challenged in papers published in the past few years (26, 44).

We agree that there are some documented problems with unacceptable bone resorption, which is possibly a result of peri-implantitis and which occurs at a high rate with rough, plasma-sprayed surfaces (10, 13). It is not surprising that plasma-sprayed implants have by and large been removed from clinical usage. Naturally, we recognize the possibility that peri-implantitis may occur around other types of implant as well, even if we do not agree about the abundance of this problem around minimally rough, Branemark implants. Obviously, the definition of peri-implantitis (5), as claiming any bone loss together with inflammation, is not acceptable to fulfill this diagnosis. In fact, the paper by Albrektsson and Isidor (5) accepted as a normal finding 1.5 mm of bone loss during the first year of function and, thereafter, an annual bone loss of <0.2 mm, which makes it clear that we actually meant (but unfortunately did not write) that bone loss *beyond* the 'acceptable level' was evidence of peri-implantitis if combined with inflammation. Hence, it is our opinion that Fransson et al. (26) had no strict criteria for their definition of 'progressive bone loss' in that they only used the implant status at 1 year. In contrast, Roos-Janåker et al. (44), in their 9- to 14-year study, accepted additional bone loss of up 1.8 mm *after the passage* of the first year. It is, therefore not surprising that Fransson et al. (26) diagnosed 12.6% of implants with progressive bone loss, whereas Roos-Janåker et al. (44) only found 6.6% of their long-term followed-up implants to suffer from peri-implantitis. Our opinion is that even the figures by Roos-Janåker et al. are unrealistically high, because peri-implantitis in 6.6% of implants at 9–14 years in all probability would result in a much higher incidence of obvious patient problems, as actually indicated in long-term investigations by Bryant (19), Jemt & Johansson (36), Ekelund et al. (24), and Attard & Zarb (11). Furthermore, the progressive bone loss (26) and peri-implantitis (44) described in the publications cited may in many cases represent normal bone remodeling, instead of a pathological condition. This is supported by the fact that the maximal follow-up period of 14 years (44) and 20 years (26), respectively, would allow for a total of 4.1 and 5.3 mm of bone loss to be within the normal range according to published criteria (5, 7) for successfully functioning oral implants.

Accordingly, a single-minded explanatory model for peri-implantitis as the only reason for bone loss is, according to our understanding, confusing because an *increase* of bone levels has also been observed at implants that have shown bleeding at

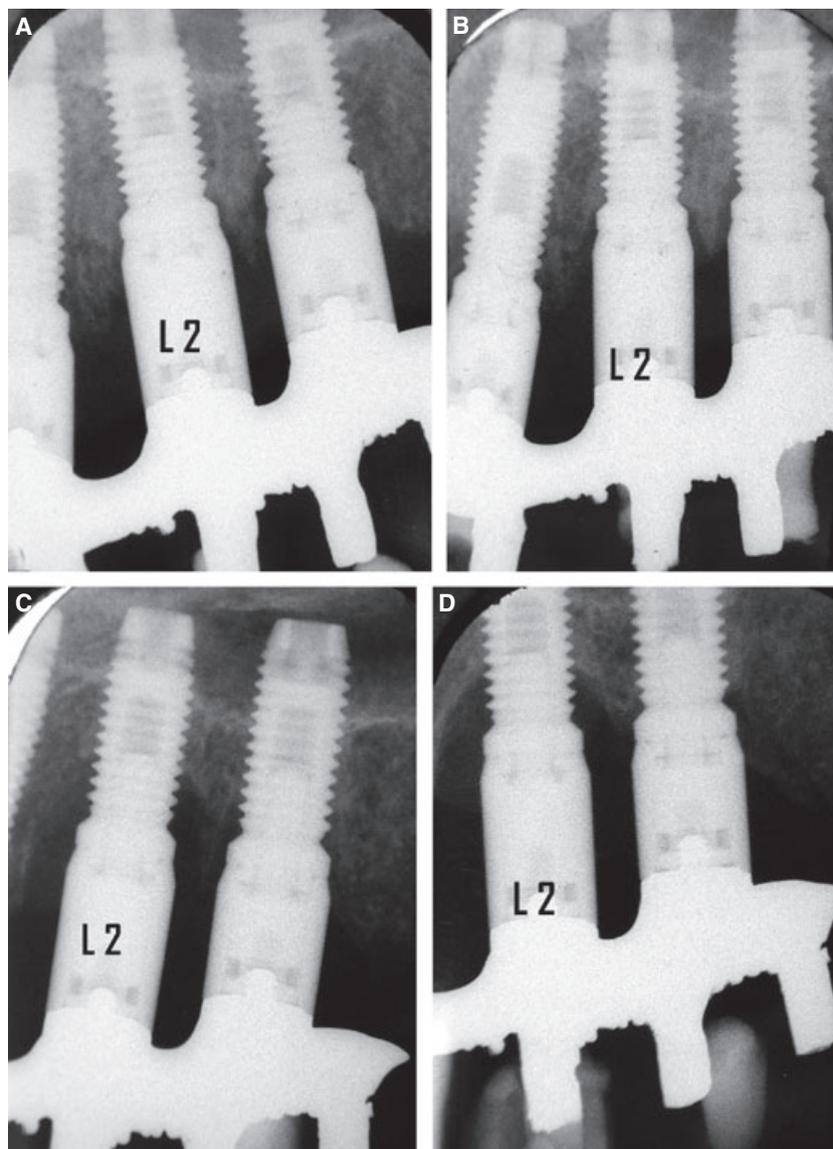


Fig. 2. Forty-six-year-old female patient provided with six implants in the edentulous maxilla. Short (10 mm) left intermediate implant (L2), placed in the canine region, at the time of prosthesis placement (A), after 1 (B), 10 (C), and 15 (D) years of follow-up. Notice stable bone level at the implant (L2) from placement of prosthesis (A) to the

first annual check-up (B). Thereafter, notice obvious bone loss at the intermediate implant (L2) after 10 (C) and 15 (D) years of follow-up. Stable bone levels at the adjacent implants could be taken as an indication of load-induced bone loss at the short implant, placed in the canine region (L2).

probing combined with pus (44). Furthermore, it can also be questioned whether the inclusion criteria of bone loss in some articles (12, 25, 26, 44), are clinically relevant because the implants studied did not seem to exhibit higher risk for late complete failure. Instead, most implants lost after the early phase of loading do not show any continuous progressive bone loss before they are removed, indicating reasons other than peri-implantitis for the failures. The observation that peri-implantitis is a relatively rare phenomenon around long-term followed-up oral implants, has been further confirmed in the most

recent paper by Åstrand et al. (9) which reported a prevalence of peri-implantitis of 2.4% during a period of 20 years.

Conclusion

We consider that marginal bone loss around implants is a complex phenomenon, caused by several different factors that are not yet fully understood. A single-minded explanatory model for bone loss around implants is not acceptable.

In analogy with teeth, we consider that a slow continuous bone loss can be accepted around implants. More bone loss can occur during the early phase after implant placement.

We consider that it is important to maintain mucosal health around implants, and that signs of inflammation in the mucosa with pus at probing should be treated.

We consider that the inclusion criteria for bone loss around implants used in present follow-up studies vary so much that comparison between studies may not be possible.

We consider that inclusion criteria for bone loss used in follow-up studies cannot be used to identify implants with an elevated risk for late implant failure.

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