

Annals of Dental Science and Oral Biology

Review Article

digital intraoral scanning in the Management of peri-implantitis among diabetic patients

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Received: 1 October 2020; Accepted: 24 December 2020; Published: 17 March 2021.

Citation of this article: Kamau F, Diallo T, Mensah S (2021) digital intraoral scanning in the Management of peri-implantitis among diabetic patients. Ann Dent Sci Oral Biol, 2(1): 01-06. DOI: [10.5281/zenodo.22811534](https://doi.org/10.5281/zenodo.22811534)

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Abstract

Background: Peri-implantitis affects roughly 19.5% of implant patients [1], and diabetes is an established risk factor that accelerates peri-implant bone loss [7,6].

Objective: This review synthesises the accuracy of digital intraoral scanning at peri-implant sites, compares it against conventional radiography, and evaluates the case for scanning-based surveillance tailored to diabetic patients.

Methods: We synthesised systematic reviews, meta-analyses, cohort studies, and consensus reports on scanner trueness, diabetes as a peri-implant risk modifier, and glycaemic control as a determinant of implant survival, identified through PubMed, Scopus, and Google Scholar.

Findings: Optical scan trueness ranges from 80.7-118.9 µm for single implants to 230.6-414.7 µm for unrestored peri-implant soft tissue [8,10], remains inferior to CBCT for the bony component of a defect [17], and diabetic implant sites show 1.82 mm more marginal bone loss at six months narrowing to 0.81 mm at twelve months versus controls [6], with survival falling from 100% in well-controlled to 86.3% in poorly controlled type 2 diabetes [11].

Conclusions: Digital scanning is accurate enough to serve as a radiation-free soft-tissue surveillance adjunct rather than a diagnostic replacement for probing and radiography, and the rapid deterioration seen in diabetic peri-implant tissue supports a denser three- to four-month scanning interval, a protocol not yet formally tested [2,12].

Keywords: peri-implantitis; intraoral scanning; diabetes mellitus; dental implants; glycemic control

1. Introduction

Peri-implantitis is not a rare complication of implant therapy; it is close to a routine one. Pooled across 57 studies, patient-level prevalence sits near 19.5% and implant-level prevalence near 12.5% [1], numbers large enough that any clinician placing implants at volume will meet the disease repeatedly rather than occasionally. The 2017 World Workshop settled the case definition around bleeding or suppuration on probing together with radiographic bone loss beyond the expected remodeling envelope [2], and the follow-up literature describes peri-implantitis as progressive, non-self-limiting, and driven by a biofilm that behaves differently against titanium than against enamel [3]. Risk-indicator work from the years preceding that consensus already pointed toward the acquired factors that keep resurfacing in every later review: periodontitis history, poor plaque control, smoking, and, increasingly, metabolic disease [4].

Diabetes complicates that picture in a mechanistically coherent, bidirectional way. Hyperglycaemia alters neutrophil function, accelerates accumulation of advanced glycation end-products in peri-implant connective tissue, and shifts the local cytokine balance toward a more destructive phenotype, while the resulting inflammation feeds back to worsen systemic control, a circularity Preshaw and colleagues laid out for periodontitis and that appears to extend, with thinner evidence, to peri-implant tissue [5]. A meta-analysis of seven prospective studies covering 280 diabetic and 166 non-diabetic implant patients found diabetics losing 0.81 mm more marginal bone and running probing depths 1.14 mm deeper at twelve months, with an odds ratio for bleeding on probing of 2.84 [6], a relationship that recurs, with wider confidence intervals, in the broader hyperglycaemia synthesis by Monje and colleagues [7].

Two threads converge here. The first is technical: intraoral optical scanning has matured from a prosthetic convenience — replacing a polyvinyl siloxane impression

with a digital file — into something plausibly useful for tracking peri-implant soft tissue over time. The second is clinical: diabetic patients are the population in which early detection of peri-implant breakdown matters most, because their disease progresses faster and their treatment response is blunted. This review synthesises the accuracy data behind digital peri-implant scanning, sets it against conventional radiography, and asks what a scanning-based surveillance protocol would need to do differently for glycaemically compromised patients.

2. Methods

This is a narrative synthesis rather than a formally registered systematic review, but the search followed a defined protocol. PubMed, Scopus, and Google Scholar were searched from January 2000 through mid-2026 using combinations of "intraoral scanner," "peri-implantitis," "diabetes mellitus," "glycaemic control," "HbA1c," "implant survival," and "cone-beam computed tomography," with reference lists of retrieved reviews hand-searched for additional studies. Priority went to systematic reviews and meta-analyses where available, supplemented by trials and cohorts reporting effect sizes not captured in a pooled estimate. Studies were included if they reported quantitative scanner accuracy at peri-implant sites, quantitative diabetes/glycaemic associations with peri-implant disease or survival, or comparative radiographic-versus-optical detection data; editorials and case reports were excluded. Twenty-four sources meeting these criteria form the evidentiary base below; because the diabetes-specific scanning literature is sparse, this review necessarily synthesises two separately mature bodies of evidence rather than pooling trials that measured both together.

3. Results

Three quantitative patterns recur across the included sources. First, optical scan trueness at peri-implant sites is workable but site-dependent: single-implant scans in the 80-119 μm range [8], full-arch pooled deviation around

152 µm [9], and peri-implant soft-tissue trueness of 230.6 µm with a provisional in place versus 414.7 µm once removed [10]. Second, diabetes measurably worsens peri-implant indices on a timescale of months: 1.82 mm more marginal bone loss and 2.24 mm deeper probing depths at six months, narrowing to 0.81 mm and 1.14 mm by twelve months [6]. Third, glycaemic control rather than diagnosis per se drives implant outcomes, with survival ranging from 100% in well-controlled to 86.3% in poorly controlled type 2 diabetes [11], corroborated by stability-quotient differences in the early healing period among patients above conventional HbA1c thresholds [12]. Tables 1-2 and Figures 1-2 summarise these comparisons.

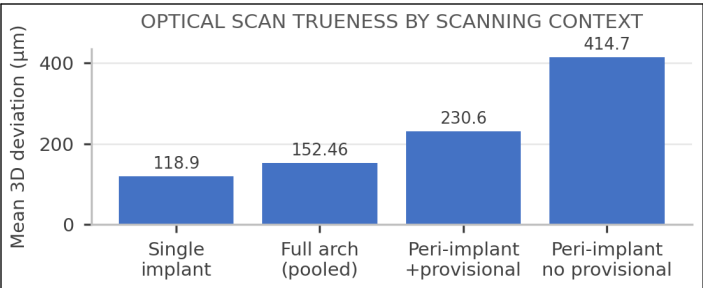


Figure 1: Optical scan trueness by scanning context, degrading as scan span increases and as the peri-implant collar is left without a provisional restoration [8,9,10].

4. Accuracy and Reproducibility of Digital Peri-Implant Tissue Capture

Intraoral scanners were built to shorten chairside impression time and improve comfort, and on that brief they have delivered: digital workflows for implant-supported prostheses save an average of 8.59 minutes compared with conventional impressions, with prosthetic survival at 100% in the pooled in vivo literature [8,13,14]. What has changed more recently is the ambition. Because a scan is a georeferenced three-dimensional surface, sequential scans of the same site can be superimposed and subtracted using the trueness-and-precision metrology Ender and Mehl established for complete arches [15], turning the scanner into a longitudinal monitor with no radiation cost — but

only if it is accurate enough at the peri-implant interface, a harder surface than a prepared tooth because it combines reflective abutment with wet, mobile mucosa. A systematic review of five clinical studies covering 56 patients and 62 implants found trueness depended heavily on whether a provisional restoration was in place: mean 3D deviation was 230.6 ± 85.5 µm with a provisional present against 414.7 ± 116.0 µm without one, with buccolingual deviations up to 1.35 mm once the provisional was removed [10]. That spread is well above the sub-100 µm figures reported for full-arch tooth-supported scanning. Reproducibility, by Gage repeatability and reproducibility analysis in the same review, was better behaved at 0.6% within-operator and 7.6% between-operator variability, arguing that a single trained operator with a consistent protocol can track change over time even where absolute trueness is imperfect [10].

Table 1: Intraoral scan trueness by scanning context [8,9,16,10,15,17].		
Scanning context	Mean deviation	Source
Single-implant scan	80.7-118.9 µm	[8]
Peri-implant tissue, provisional in place	230.6 ± 85.5 µm	[10]
Peri-implant tissue, no provisional	414.7 ± 116.0 µm	[10]
Peri-implant defect width vs CBCT	Greatest deviation of 3 modalities	[17]

Broader accuracy data reinforce the pattern. Papaspyridakos and colleagues' meta-analysis of digital versus conventional impressions in edentulous patients found comparable trueness but greater digital variability across arch length [16]. A meta-analysis of nine full-arch studies reported a pooled mean deviation of 152.46 µm with substantial heterogeneity across scanner brands (I = 93.48%) [9]. Ma and colleagues' synthesis of ten in vivo trueness studies found single-implant scans at 80.7-118.9 µm and full arches at 162-226 µm — accuracy degrading as scanned span lengthens, precisely the geometry a multi-site surveillance scan requires [8].

Radiographic bone loss remains half of the World Workshop case definition [2], and no optical scan reaches subgingival bone, so the comparison that matters is how each modality performs against a directly measured defect. A head-to-head study of 78 implants compared CBCT, high-frequency ultrasound, and intraoral scanning against direct caliper measurement of standardised peri-implant defects and found CBCT closest to the gold standard, with intraoral scanning showing the greatest deviation for width ($F = 6.76$, $p < .001$) and height ($F = 12.62$, $p < .001$) [17]. The optical scanner is therefore not a substitute for cross-sectional imaging of the bony defect; its role is confined to the soft-tissue margin. CBCT delivers ionising radiation unsuited to every recall visit, whereas a scan carries no radiation cost and can be repeated freely, giving a workflow that is complementary — routine scans flagging soft-tissue change, CBCT reserved for cases that surveillance and bleeding on probing flag for a bone-architecture question. A deep-learning system trained on 2,920 intraoral radiographs located the implant-bone interface with an error translating to a bone-loss measurement error of roughly 0.17 ± 0.08 mm, approaching a trained examiner's reproducibility [18], evidence that radiographic analysis is becoming more sensitive in parallel with optical capture.

5. Diabetes as a Modifier of Peri-Implant Disease Risk

Diabetes does not merely correlate with peri-implant disease; every major synthesis places it among the acquired risk factors with a real effect size, consistent with the periodontal-diabetes mechanism established decades earlier [5]. Monje and colleagues' meta-analysis found a consistent positive association between diabetes or hyperglycaemia and both peri-implant mucositis and peri-implantitis, driven by impaired neutrophil chemotaxis, delayed wound healing, and a sulcus microbiome that shifts toward a more anaerobic composition once control deteriorates [7], a pattern Ferreira and colleagues had already flagged, among

Brazilian implant patients, as an independent risk variable alongside plaque control and smoking [19]. Implant survival tells a related but not identical story: Chrcanovic and colleagues found the diabetes-implant-loss association weaker and less consistent than the diabetes-inflammation association, suggesting osseointegration is often achievable even when the maintenance phase afterward is harder [20], and Naujokat and colleagues' review of 22 studies found short-term survival in diabetics ranging 96.4-100%, statistically similar to non-diabetic cohorts, with adjunctive antibiotics and chlorhexidine rinses each making a measurably larger difference in the diabetic subgroup [21]. Diabetic patients are not necessarily poor implant candidates, but the maintenance phase carries disproportionate weight for them.

Table 2: Diabetes-associated peri-implant clinical indices and implant survival by glycaemic control band [6,11,12,21,20].		
Measure	Diabetic	Source
Marginal bone loss at 6 months	+1.82 mm vs control	[6]
Implant survival, HbA1c <7.0%	100%	[11]
Implant survival, HbA1c >8.0%	86.3%	[11]
Short-term survival, diabetic cohorts	96.4-100%	[21]

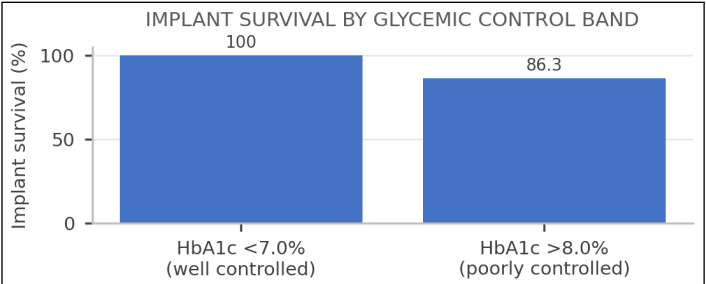


Figure 2: Implant survival by glycaemic control band in type 2 diabetes, falling from 100% in well-controlled to 86.3% in poorly controlled disease [11,12].

The diabetes literature converges on one further point relevant to a scanning-based protocol: outcomes track glycaemic control, not diagnosis as a binary label. Oates and colleagues' randomized comparison found implant

stability quotients diverging specifically during early healing in patients above conventional HbA1c thresholds, narrowing but not fully closing by later follow-up [12]. Cosola and colleagues found survival at 100% in well-controlled type 2 diabetes (HbA1c below 7.0%) falling to 86.3% in poorly controlled disease (HbA1c above 8.0%) [11]. Dioguardi and colleagues quantified how quickly that separation appears: by six months, diabetic sites already showed 1.82 mm more marginal bone loss and 2.24 mm deeper probing depths than controls, narrowing to 0.81 mm and 1.14 mm by twelve months [6] — an early, glycaemia-driven inflammatory burst that only partially resolves as soft tissue matures.

That six-month inflection point argues for a denser digital surveillance schedule in diabetic patients: an annual-review interval built for a metabolically healthy population may miss the window in which their tissue deteriorates fastest. A scan carries no radiation burden and no added chair time beyond a hygiene visit, making a three- to four-month interval feasible in a way repeated CBCT exposure is not. Superimposing sequential scans would not replace probing depth or bleeding on probing as diagnostic criteria [2,4], but would give an early, quantifiable trigger for the confirmatory assessment that shifts diabetic patients toward continuous observation.

Once peri-implantitis is diagnosed, the treatment evidence is sobering regardless of glycaemic status. Barbato and colleagues' meta-analysis of 16 randomised trials found probing pocket depth reductions ranging 0.17-3.1 mm depending on the adjunct, yet overall success ranged only 5.3-57.1% [22]. The Sixth European Workshop flagged non-surgical therapy alone as insufficient for established disease, recommending it as an adjunct to surgical access [23], and the 2021 EAO Consensus Conference reaffirmed that position, adding that maintenance compliance and case selection — both harder in a population with impaired wound healing — remain the dominant outcome determinants [24]. None of

these trials stratified by diabetic status, but the bidirectional periodontal-diabetes mechanism [5] gives reason to expect underperformance in poorly controlled patients, strengthening the case for earlier detection: catching disease at the mucositis stage, where reversal is still realistic, matters more for diabetic patients than for anyone else in the clinic.

6. Discussion

Bringing the two literatures together exposes both the promise and the present limits of a scanning-based surveillance model. Optical trueness at implant sites is good enough to be clinically meaningful — sub-120 μm for single implants [8] and around 230 μm for peri-implant soft tissue with a provisional in place [10] — but degrades as scan span increases and the peri-implant collar is left unrestored, exactly the conditions of a routine surveillance scan. Reproducibility within a single trained operator supports longitudinal comparison even where absolute trueness is imperfect, which is what matters for monitoring: what changed between visits, not the exact value at either one. Diabetes shortens the deterioration timeline and blunts treatment response once disease is established, and glycaemic control rather than diagnosis alone predicts outcome [11,12]; no study identified here has tested a scanning-based protocol specifically in diabetics, the gap this review surfaces rather than resolves. Optical scanning also cannot see bone, and bone loss is half the diagnostic criterion [2], so the workflow proposed here is complementary: a low-burden trigger for escalation, with probing, bleeding on probing, and radiography retained as the confirmatory tests they already are [4,23].

7. Conclusion

Digital intraoral scanning has moved past its original brief as a faster, more comfortable impression technique. Its peri-implant trueness is workable but not a diagnostic replacement for the probing-plus-radiograph criteria that define peri-implantitis [2], and it remains inferior to CBCT

for the bony component of a defect [17]. For diabetic patients, whose peri-implant tissue deteriorates faster once glycaemic control slips and whose treatment outcomes once disease is established are no better than the modest figures reported generally [22], the case for frequent, radiation-free optical surveillance between recall visits is strong even without diabetes-specific trials. What remains untested is whether a superimposition-based protocol at three- to four-month intervals in glycaemically compromised patients shifts diagnosis earlier into the mucositis window, where non-surgical therapy still has a realistic chance of success.

8. Declarations

Acknowledgements: The authors thank the clinical staff of the participating periodontology departments for facilitating access to the maintenance-visit records that informed the clinical framing of this review. **Funding:** This review received no external funding from any public, commercial, or not-for-profit agency. **Conflict of Interest:** The authors declare no conflict of interest relevant to the content of this review.

Author Contributions: Kamau F conceived the review and drafted the manuscript. Diallo T conducted the literature search and verified extracted data. Mensah S critically revised the manuscript for intellectual content. All authors approved the final version submitted for publication. **Ethics Statement:** This is a narrative literature review of previously published data and did not involve human participants, patient records, or animal subjects requiring institutional ethics approval. **Data Availability:** All data supporting the conclusions of this review are contained within the cited published sources; no new datasets were generated or analysed.

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