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Getting to the root of the problem with dental implants

A peer-reviewed article written by Nicole Fortune, MBA, RDH

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Getting to the root of the problem with dental implants

Abstract

There have been many changes concerning dental implants through the years, including the amount of information available. There are many studies, textbooks, magazine articles, case presentations, and opinion pieces readily accessible to dental clinicians. It is easy to become confused about best practices and concerned that the implant may be harmed during preventive maintenance visits. It seems that these concerns and hesitations lead to inaction in assessment, diagnosis, and treatment. In addition, it is not often clear what has caused peri-implant disease. This course aims to identify the differences in diagnostic criteria when examining natural dentition versus dental implants, outline a comprehensive flow of diagnostics for dental implants, explain potential etiologies, and explore emerging research.

Educational objectives

1. Describe how dental implants compare to natural dentition when evaluating health and identifying disease
2. Create a workflow that clinicians are confident in following to be certain their dental implant exams are complete
3. Identify potential etiologies beyond the common etiology of residual cement on the dental implant
4. Explore new research and future findings

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According to Grand View Research, "the global dental implant market was valued at USD 3.6 billion in 2020 and is expected to expand at a compound annual growth rate (CAGR) of 11.0% from 2021 to 2028."[1] As the US population ages and more people become aware of the importance of good oral health, including tooth replacement, dental professionals will encounter patients with implants more frequently. Dental implants are highly successful, with a failure rate of 5%-10%.[2] Although that number seems low, with the growing demand for dental implants, there will be more opportunities for failure. It is important that we feel confident in our knowledge and ability to not only recognize the signs of dental implant failure but also identify possible etiologies that could result in future problems.

Causes of implant failure

The majority of dental implant failures occur in the short term during or shortly after the surgical healing phase, but several studies have suggested a significant number of implants develop peri-implant infections with resulting bone destruction after several years postplacement.[3,4] In one study and literature review, 1% to 47% of dental implants were diagnosed with peri-implantitis.[5]

The American Academy of Periodontology (AAP) has created categories to define dental implant conditions and parameters for diagnosis. Peri-implant mucositis is diagnosed when there is inflammation of the soft tissues but no evidence of bone loss. Peri-implantitis is diagnosed when there is a progressive loss of bone and inflammation is present in the soft tissues. The AAP states these conditions are a result of poor plaque control, and peri-implantitis is assumed to follow untreated mucositis.[6]

Peri-implantitis is associated with large soft tissue ulcerations, an advanced rate of bone destruction, and is histologically different compared to natural dentition.[7] In addition, treatments for advanced peri-implantitis have been shown to be unpredictable.[8] Research has shown that nonsurgical treatment of peri-implant mucositis prevents the disease progression to peri-implantitis.[7,9] It is important to identify implants at the mucositis phase and appropriately treat these areas to prevent disease progression and avoid an increasingly unpredictable treatment result.

A third category considers the etiologies of implant failure not related to poor plaque control.[3] These include but are not limited to bone loss due to extraction trauma, thin buccal plates, sinus complications, and iatrogenic causes such as poor implant or restoration position.[3]

When diagnosing periodontal conditions around natural dentition, it is generally accepted that the most important indication of health is a lack of bleeding on probing (BOP).[4] In addition, probing depths greater than 4 mm are considered outside the parameters of health.[4] Periodontal charting and the clinical relevance of the measurements around dental implants have been subjects of debate within the dental profession for many years.[10-12] One question is, if peri-implant tissues are not histologically similar to natural dentition, should they be treated the same way with periodontal probing, and should the results be based upon the criteria used for natural dentition? The 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions supports probing implants to note any bleeding on probing that would indicate inflammation and to determine any changes in depth from previous measurements.[13]

Probing

The confusion concerning how to assess implant health may lead to unidentified and untreated disease. Whether to probe around implants has been questioned based on concerns that the probe will damage the soft tissue or the implant body and that BOP results may not be an indicator of inflammation. The concern that probing will damage the soft tissue due to the lack of fibrous attachment of the peri-implant mucosa to the implant body has been examined, and long-term damage has not been identified.[14] Studies show that any damage to the mucosal seal using a probing force of 0.25 N healed within five days with a complete repair of the mucosal seal.[15] If there is damage to the seal, there may be some BOP that could be interpreted as a sign of inflammation and disease.

A study in the *Brazilian Journal of Oral Sciences* compared BOP across three subject groups: peri-implant health, peri-implant mucositis, and peri-implantitis. They concluded that all of the implants in the mucositis and peri-implantitis groups showed BOP, but BOP was also noted in some of the healthy implants.[16] This implies that periodontal probing yielded a false positive result in some healthy implants, and the concern is that this will lead to overtreatment of the implant. This study concluded that BOP alone may not be a definitive indicator of disease but fell short of advising against periodontal probing.[13] Considering the potential for rapid and nonlinear tissue destruction brought on by peri-implant inflammation, and that this study found 100% of the diseased implants had BOP, it cannot be eliminated as a sign of inflammation. If BOP is present, the clinician must be able to discern if BOP is due to disease or irritation to the mucosal seal.

Another concern with probing implants is potential damage to the implant body, especially when a metal probe is used. There are potential risks of scratching or altering the abutment surface, allowing biofilm to develop, or creating a galvanic reaction, leading to peri-implant inflammation. Research has shown that metal periodontal probes do not have significant effects on the abutment surface.[17] When the clinician probes carefully, with adequate force, there should be no concerns of probing leading to implant damage and inflammation or implant failure.

Pocket depth

The measurement of periodontal probing depths may not imply disease. One report in *Perio-Implant Advisory* states that probing depths are expected to be greater around dental implants due to the increased biologic width, and healthy pocket depths can measure more than 4-5 mm.[18] When analyzing long-term survival rates, Winitsky et al. found that periodontal probing depth was not correlated with dental implant health, and this did not change over the course of 14-20 years.[19]

Several researchers also argue that changes in probing depth and the presence of BOP together are not accurate indices to determine implant health.[6,7] In one study, 127 implants were followed for 7.6 years. It revealed that 60% of the periodontal depths measured greater than 4 mm and 80% of the sites had BOP. These sites had limited average bone loss of 0.07 mm annually and thus these implants were not diagnosed with peri-implantitis.[20] These results were confirmed in a study following single implants over 16-22 years.[21] In contrast, a 2017 study of 130 implants, with a mean implant age of 6.5 years, found increasing pocket depths associated with peri-implantitis.[22]

Considering that a single measurement of BOP or pocket depth may not be an accurate indicator of disease, another diagnostic tool needs to be added to the evaluation. The World Workshop consensus is if pocket depths are increasing and there is BOP present, suspected peri-implantitis should be further confirmed with radiographs to determine if there has been bone destruction beyond expected post-healing remodeling.[3] The radiographic criterion for peri-implantitis using a periapical radiograph is bone loss of at least 3 mm when compared to a previous radiograph.[14] If there is no earlier comparison radiograph, the criteria for peri-implantitis diagnosis are bone loss of at least 3 mm in combination with probing depths of at least 6 mm with BOP.[14]

Exudate

According to the AAP, another measure of health is the absence of exudate.[14] Exudate can be noted upon probing but can also be detected by palpating the buccal and lingual aspects of the dental implant site.[23] **Figure 1** shows exudate present after palpating the buccal aspect of the dental implant site. A study in the *Journal of Periodontology* used submucosal samples to perform gene sequencing comparing dental implants with suppuration and those without. The researchers found that the group with suppuration had higher pathogenic microbes and concluded that the presence of suppuration was an indicator for greater risk for further peri-implant bone destruction.[24]

FIGURE 1

Early intervention

A study in the *Journal of Periodontology* concluded, "The diagnosis of peri-implant diseases cannot rely solely upon individual clinical parameters but rather require a combination of criteria."[25] Considered together, an increase in pocket depth, BOP, and the presence of suppuration give the clinician strong indicators of a dental implant's health. Ramanauskaite et al. found that the severity of these three clinical parameters correlated with the severity of the disease, concluding that, like periodontal disease, peri-implantitis is a progressive disease.[26] However, bone destruction can progress more quickly around implants. One reason for this is that implants lack a self-limiting process with a connective tissue capsule that protects the bone from a progressing periodontal lesion that occurs around teeth.[27] According to Roncati, this allows the inflammatory lesions around dental implants to progress more quickly and deeply into the soft tissue and bone, resulting in a faster and nonlinear periodontal breakdown.[27]

This further strengthens the rationale that early intervention is highly important to prevent continued bone loss. Khammissa et al. state that inflammation associated with dental implants responds best to early intervention and that "regular assessment will permit timely treatment."[28] This study highlights that most peri-implantitis cases are treated with surgical intervention for which the outcomes are not predictable, and it is best to intervene nonsurgically at the first sign of inflammation. Therefore, it is imperative the clinician have a framework to evaluate dental implants in a meticulous fashion. Clinicians in the same office should be calibrated in their measurements and follow the same step-by-step framework for evaluation. Below is a suggested workflow with notable details:

1. Ask patient if they have noticed any pain, sensation, or odor in the implant area.
2. Palpate buccal and lingual aspects of the implant area.
3. Note any exudate present with detail.
   a. Location (buccal, lingual, mesial, distal, line angle, direct buccal, direct lingual)
   b. Amount (light, moderate, heavy)
   c. Color (transparent, white, yellow)
   d. Consistency (watery, thin, thick)
4. Gently probe all aspects of the implant.
   a. Can the subgingival area be accessed easily for probing? (Note any crown or prosthetic design preventing accurate probing.)
   b. Are there any depths greater than 4 mm?
   c. Have these depths changed since the last measurement?
5. Is there BOP?
   a. Location (buccal, lingual, mesial, distal, line angle, direct buccal, direct lingual)
   b. Immediate or delayed
   c. Amount (light, moderate, heavy)
   Is this a change from last measurement?
6. Expose radiographs.
   a. Compare to previous radiographs for changes in bone levels.
   b. If there is no previous radiograph, evaluate if there appears to be bone loss of at least 3 mm that is evident on the film and a periodontal probing depth of at least 6 mm in that area.

Utilizing a consistent workflow and evaluation system to assess dental implant health is a starting point for the clinician, but it does not indicate the etiology of disease. There are several factors that could cause peri-implant breakdown, and identifying the etiology of the inflammation may help to prevent further tissue destruction.

Etiology

Poor oral hygiene: The AAP states that the majority of peri-implantitis cases are caused by poor plaque control.[4] The clinician should closely investigate the area in an attempt to determine why there is poor plaque control. A logical first step is to disclose the patient. Disclosing solution shows the clinician the amount of plaque in the patient's mouth and gives the patient an opportunity to evaluate their home care. Some disclosing agents will distinguish between early and mature or acidic biofilm. This gives the clinician a deeper understanding of the problem areas for patients.[29,30] If the patient has poor oral hygiene practices, evident from the diffuse, heavy plaque accumulations noted by the disclosing agent, it may be logical to assume any inflammation is a result of these poor habits. In this case, the best course of action is patient education in overall home-care techniques. If there is poor plaque control only in the area of the dental implant, have the patient demonstrate their technique. The tools and techniques used for natural teeth may not be ideal around implants. In fact, our traditional tools may pose a risk to the implant. Remnants of dental floss have been identified as the etiology of some cases of peri-implantitis.[31] Floss can catch on sharp crown margins or on the roughened implant body. One key question to ask your patient is if they feel the floss catch or if the floss is difficult to remove around the dental implant. Monje et al. recommend patients be instructed to use interproximal brushes instead of floss.[32]

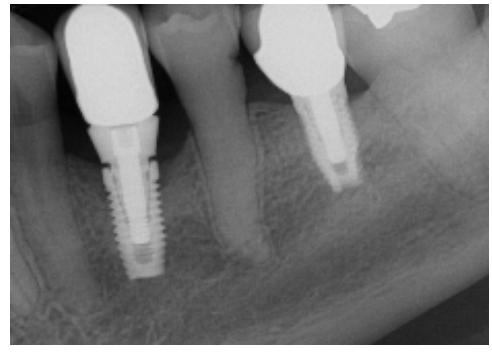

FIGURE 2

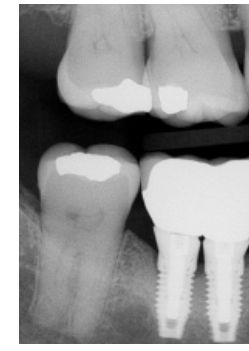

FIGURE 3

Restorative clues: Radiographs may give clues to other etiologies. For example, in **figure 2**, the radiograph reveals the abutment was not seated completely into the implant body. This is an area that may lend itself to more biofilm accumulation. **Figure 3** shows two implants in close proximity to each other. The space between these implants may not be wide enough for the patient to access during home care. Also, it's important to note the crowns are fused, making interdental care more difficult.

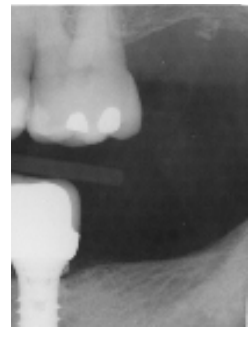

FIGURE 4

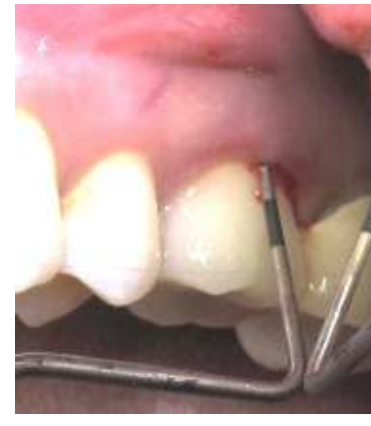

FIGURE 5

FIGURE 6

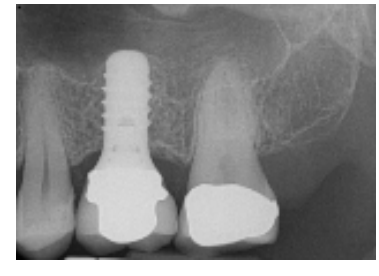

FIGURE 7

Excess cement: Excess cement has been identified as the cause of some cases of peri-implantitis due to the cement harboring biofilm, including destructive microbes.[33,34] In one study, Dr. Thomas Wilson, Jr. used a dental endoscope to inspect the subgingival environment for evidence of cement on both healthy and diseased implants. He found that no cement was seen on any of the healthy implants but was found on 81% of the diseased implants. After the cement was nonsurgically removed, the majority of the diseased implants showed no signs of inflammation.[35] A periapical radiograph will show if there is residual cement on the mesial or buccal. The radiograph in **figure 4** shows residual cement on the distal. This radiograph will not show any residual cement on the buccal or lingual aspects.

Understanding this, if an implant is overall healthy with the exception of a pocket on the buccal or lingual, it is reasonable to consider that residual cement is the etiology. **Figure 5** shows an example of an isolated pocket on the buccal aspect of an implant, and **figure 6** is the same implant with a flap raised, revealing residual cement. The radiograph showed no signs of bone loss (**figure 7**).

Phenotype deficiency: Another possible etiology of inflammation is a deficiency in the peri-implant phenotype. The phenotype is defined as the characteristics of the supporting tissues around an implant. This includes the width of the keratinized mucosa, thickness, and tissue

height.[36] Avila-Ortiz et al. state, "the evidence is equivocal regarding the effect that the presence or absence of keratinized mucosa has on the long-term health of the peri-implant tissues."[30] The importance of the peri-implant phenotype is not well known and there are many studies and expert opinions debating this topic.[32] A 2018 study of 54 patients over nine months, with either a wide (at least 2 mm) or a narrow (less than 2 mm) phenotype, found that the wide group had less discomfort brushing, which led to less plaque accumulation and less inflammation than the narrow group.[37] Also, a 2019 cross-sectional study suggested that the width of keratinized tissue is a risk indicator for dental implant inflammation, and a thick phenotype may reduce the risk for peri-implant diseases.[38] Patients who do not have diligent home care, do not follow an individualized recall schedule, and have less than 2 mm of keratinized mucosa may be more susceptible to inflammation.[39]

It stands to reason if there is pain or bleeding present during home care, many patients may avoid the area, leading to more biofilm accumulation and resulting in soft tissue inflammation. The researchers of an article in the *Journal of Clinical Medicine* state, "a lack of keratinized mucosa in patients with inadequate oral hygiene could be regarded as a predisposing factor for peri-implant diseases, since it is associated with more recession, less vestibular depth, and more plaque accumulation, which, in turn, may be predisposing to inflammation."[40] Soft-tissue grafting can be completed around dental implants to increase the width of keratinized tissue. **Figure 8** shows a postoperative photo of gingival grafting around dental implants.

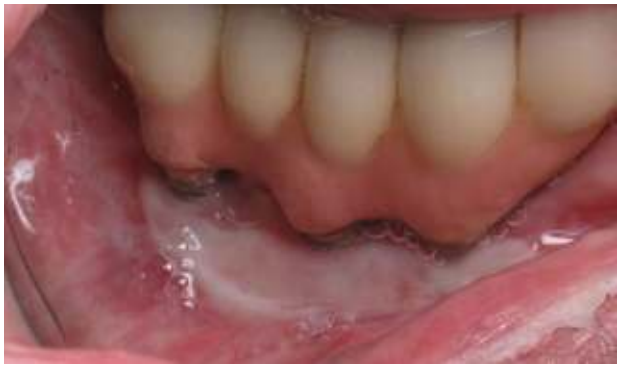

FIGURE 8

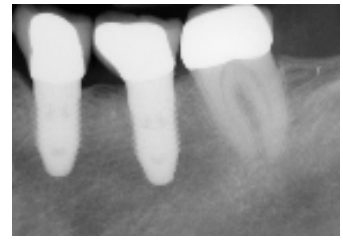

FIGURE 9

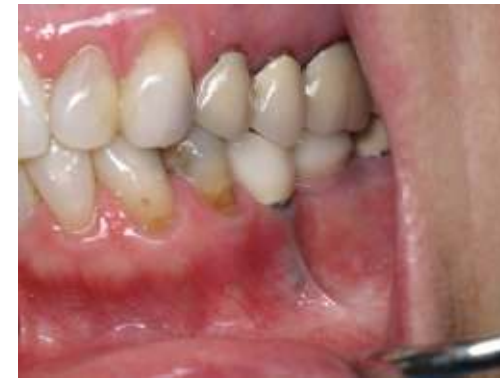

FIGURE 10

Surgical factors: There may be surgical factors that result in peri-implant bone loss. For example, in one retrospective study, over 40% of diseased implants were placed too close to the buccal plate.[41] **Figure 9** shows a dental implant in site no. 20 that was placed outside of the bony envelope and too close to the buccal plate, which led to destruction of the buccal plate. Due to this bone destruction, the gray implant body is easily noted in the photo. **Figure 10** is the corresponding radiograph. Although the implant in site no. 20 does not exhibit signs of inflammation and does not have increasing probing depths, it should be closely monitored as it has already lost bone. Interestingly the implant in site no. 19 was not placed too close to the buccal plate and appears to maintain health.

Dental implants that were placed in a site that had apical surgeries are at risk of retrograde peri-implantitis where bone destruction begins at the apical aspect of the implant. A paper by Mohamed et al. reviewed cases of retrograde peri-implantitis. They emphasize the importance of careful monitoring of implants placed in sites with previous apical pathologies.[42] However, a study in the *Journal of Periodontology* found that dental implants placed in sites with previous apical surgeries did not have an increase in failure.[43] Dental implant mobility should be evaluated. A systematic review in the *Journal of Oral & Maxillofacial Research* found that all of the articles stated an implant becomes mobile in the final stages of disease and is irreversible.[44]

Implant design: The dental implant prosthetic may contribute to peri-implant disease. One study found that the design of the prosthetic prevented the patients from performing adequate oral hygiene, even if they displayed good oral hygiene on natural dentition. Some patients had no signs of bone loss around natural dentition but did have areas of bone loss around the implant.[45] The emergence profile of the restoration appears to be an important factor in long-term implant health. A retrospective study in the *Journal of Periodontology* noted that a restoration emergence angle greater than 30 degrees correlated to marginal bone loss.[46]

Medical etiologies: Patients' medical histories can impact implant health. One study found that patients who take antidepressants—specifically selective serotonin reuptake inhibitors or tricyclic antidepressants—were more at risk for implant failure.[47] Researchers suggest this is because these medications may prevent full osseointegration.[43]

Smoking is detrimental to oral health and can be a factor in implant diseases. This makes sense considering smoking is an independent risk factor for periodontal disease, and smokers have twice the risk for periodontal disease.[48] One review and meta-analysis found that the level of risk for implant failure was related to how much the patient smokes. An increase in the number of cigarettes showed an increase in implant failure.[49] When

compared to patients with IL-1 genotype, smokers had a 2.5 increased risk factor for implant failure. Nonsmokers with the IL-1 genotype did not show an increased risk.[50]

Patients who take proton pump inhibitors (PPI) may be at higher risk for implant failure. Froum explains that PPIs reduce the absorption of multiple vitamins and minerals that may affect jawbone density and ultimately put implants at risk for failure.[51] Froum also acknowledges there are some cases of implant failure with patients suffering from rheumatoid arthritis (RA), but there is no scientific data supporting RA as a risk factor.

Diabetes is a well-known risk factor for periodontal disease, with three times the risk.[52] As with periodontal disease, uncontrolled diabetics are at a greater risk for peri-implantitis and dental implant failure. Well-controlled diabetics do not appear to have an elevated risk.[53-55]

Metallosis: There is some new and evolving research suggesting titanium particles that become embedded in the adjacent soft tissue contribute to the peri-implant disease process. The theory developed from research showing titanium joint replacements developed a secondary inflammatory response due to the titanium particles released from the joint and embedded in the neighboring soft tissue. It has been suggested this was the cause of failure of some joint replacements.[56,57]

It has been found that biofilm, specifically *S. mutans* and *P. gingivalis*, affect the oxidized layer of the dental implant by corrosion.[58] Corrosion of the implant surface allows for more biofilm adhering to the implant surface, which leads to more corrosion.[59] When corrosion occurs, titanium ions are released. Mastication can frequently cause micromovements of the dental implant, resulting in the release of ions off the dental implant surface. This also will result in corrosion.[60]

Wilson explains that titanium ions cause the release of cytokines, which stimulates inflammation.[57] Clinicians cannot identify this etiology chairside; however, future research may reveal unique traits of this response that can be identified clinically. Also, continued support for the metallosis theory may influence future dental implant materials.

The subject of a recent case report was found to be hypersensitive to titanium.[61] This, combined with possible metallosis, was hypothesized as the reason for continued peri-implantitis around multiple titanium implants. These titanium implants were replaced with zirconia, and the researchers found no signs of disease during the first 18 months post-placement.

Conclusion

Consistent implant evaluation at each recall will alert the clinician to changes and potential peri-implant breakdown. Identification and understanding of potential etiologies allow the clinician to determine what treatments to recommend and any actions to prevent future inflammation. Clinical assessment combined with critical thinking may be the best tools available to help patients maintain their dental implants for life.

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QUESTIONS

1. The dental implant business is expected to have a compounding growth rate of ___ over the next seven years.
   - A. 5%
   - B. 50%
   - C. 11%
   - D. 75%

2. Dental implants have a failure rate of:
   - A. 10-20%
   - B. 20-15%
   - C. 10-35%
   - D. 5-10%

3. Dental implants fail because of:
   - A. Peri-implant infection
   - B. Failed osseointegration
   - C. Both A and B
   - D. Neither A nor B

4. Why are there inconsistent case reports of peri-implantitis in the literature?
   - A. There are no universally accepted diagnostic criteria.
   - B. Case reports do not include peri-implantitis.
   - C. There is no perceived reason.
   - D. Both A and B

5. According to AAP guidelines, peri-implant mucositis is diagnosed when:
   - A. There is an increase in pocket depths
   - B. There is bone loss evident
   - C. There is BOP
   - D. All of the above

6. Peri-implantitis:
   - A. Has no known etiology
   - B. Is not treatable
   - C. Is histologically different than periodontal infection around natural dentition
   - D. Does not follow peri-implant mucositis

7. Probing dental implants:
   - A. Has been shown to damage the mucosal seal
   - B. Is not the only indicator of disease
   - C. Is the only diagnostic method for peri-implantitis
   - D. Will damage the implant body

8. The reason(s) to probe dental implants is (are):
   - A. To note any BOP
   - B. To check for increasing pocket depths
   - C. Both A and B
   - D. Neither A nor B

9. A study in the *Brazilian Journal of Oral Sciences* found:
   - A. BOP only happens around diseased implants.
   - B. BOP can be present in some healthy implants but is always present in diseased implants.
   - C. BOP never happens around diseased implants.
   - D. None of the above

10. When there is increased pocketing and BOP present, the best step to take is:
    - A. Use an ultrasonic scaler to debride the implant
    - B. Take a radiograph to confirm bone loss beyond normal healing
    - C. Reschedule the patient
    - D. Do nothing

11. Which is the criteria for diagnosing peri-implantitis using a radiograph?
    - A. Bone loss of at least 3 mm when compared to a previous radiograph
    - B. Bone loss of less than 4 mm when compared to a previous radiograph
    - C. No evident bone loss
    - D. Bone loss of at least 7 mm when compared to a previous radiograph

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QUESTIONS

12. Exudate around dental implants:
    A. Can be detected when probing
    B. Can be detected from palpating the buccal and lingual
    C. Is an indicator of possible infection
    D. All of the above

13. Patients with the IL-1 genotype:
    A. Cannot get peri-implantitis
    B. Always get peri-implantitis
    C. Have the same rate of peri-implantitis as those without
    D. Have a greater risk of getting peri-implantitis

14. Metallosis:
    A. Is a possible cause of secondary inflammatory response
    B. Has contributed to failure of knee and other major joint replacements
    C. Is a new area of research in cases of peri-implantitis
    D. All of the above

15. Patients with rheumatoid arthritis:
    A. May be at increased risk for implant failure
    B. Cannot have dental implants
    C. Are less susceptible to peri-implantitis
    D. Always have implant failure

16. Smokers:
    A. Are at no risk for peri-implantitis
    B. Have twice the risk for periodontal disease
    C. Have a risk of peri-implantitis that is proportional to the number of cigarettes smoked daily
    D. Both B and C

17. Medical histories:
    A. Should be reviewed carefully as several conditions or medications can affect dental implant health
    B. Are irrelevant to dental implant health
    C. Are not needed at dental visits
    D. Will show all the reasons for implant failure

18. The following can affect implant health:
    A. Highly contoured emergence profile
    B. Floss remnants on the implant
    C. Neither A nor B
    D. Both A and B

19. Retrograde peri-implantitis:
    A. Doesn't exist
    B. Is when bone loss occurs at the apical end of the implant
    C. Is never a factor in implant health
    D. None of the above

20. Which of the following may affect dental implant health?
    A. Surgical placement of the implant too close to the buccal plate
    B. The brand of toothpaste used for home care
    C. The brand of implant placed
    D. The sex of the patient

21. Why should there be a standard workflow for assessing dental implants at recall visits?
    A. So all the clinicians are evaluating the same things
    B. So the clinician knows what should be checked
    C. To have all the information to compare to previous visits
    D. All of the above

22. Dental cement:
    A. Is always visible on a radiograph
    B. Harbors biofilm that may cause inflammation
    C. Does not contribute to peri-implant disease
    D. Was not found around any diseased implants in a study by Wilson

23. What was found to be the minimum amount of keratinized tissue that will help reduce plaque accumulation?
    A. 6 mm
    B. 2 mm
    C. 1 mm
    D. 8 mm

24. Phenotype is defined as:
    A. A type of skin disease
    B. The size of the implant
    C. The characteristics of the supporting tissues around the implant
    D. A type of bone loss seen around dental implants

25. The best way to know how well a patient is removing biofilm with their home care is:
    A. To disclose
    B. To ask them
    C. By having them rinse with mouthwash
    D. By looking at the back of the mouth

26. What should be noted if there is BOP?
    A. Location
    B. Amount
    C. Immediate or delayed
    D. All of the above

27. What is one reason peri-implantitis progresses more quickly than periodontal disease around natural dentition?
    A. Aggressive toothbrushing
    B. The lack of connective tissue
    C. It doesn't progress more quickly
    D. The patient's age

28. A study in the *Journal of Periodontology* says:
    A. No maintenance visits are needed after restoring the implant
    B. BOP always indicates peri-implant infection
    C. The diagnosis of peri-implant disease requires a combination of criteria
    D. Probing significantly damages the dental implant

29. BOP, exudate, and pocket depth:
    A. Become more severe as peri-implantitis progresses
    B. Are not good measurements of dental implant health
    C. Should only be evaluated every five years
    D. None of the above

30. What will give the dental implant a better long-term prognosis?
    A. Uncontrolled diabetes
    B. Early detection and treatment of inflammation
    C. Poor home care
    D. Smoking

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Getting to the root of the problem with dental implants

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1. Describe how dental implants compare to natural dentition when evaluating health and identifying disease
2. Create a workflow that clinicians are confident in following to be certain their dental implant exams are complete
3. Identify potential etiologies beyond the common etiology of residual cement on the dental implant
4. Explore new research and future findings

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4.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	19.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
5.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	20.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
6.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	21.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
7.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	22.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
8.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	23.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
9.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	24.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
10.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	25.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
11.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	26.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
12.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	27.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
13.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	28.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
14.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	29.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
15.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	30.	Ⓐ	Ⓑ	Ⓒ	Ⓓ

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