



### **Therapeutic Endpoint**

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We have historically defined the endpoint of periodontal treatment as clinical resolution, primarily manifesting as reduction in several parameters including pocket depth, bleeding, swelling, redness, and increases in others such as bone and gingival attachment levels. When we achieve favorable results we conclude that the endpoint of therapy has been achieved and place the patient on a 3 month maintenance interval because periodontal disease is a chronic, non-curable bacterial infection, requiring on-going therapy.

We have not; however, looked at the pre and post-op levels of the bacteria that started the disease process in the first place. How do we know if we have achieved reduction in bacteria levels and distribution? Clinical improvement is not necessarily accompanied by bacterial reduction or increases in the level of other pathogens.

Periodontal disease is the result of the interplay between bacteria and the body's immuno-inflammatory response to the bacteria. Bacteria are necessary but not sufficient on their own to cause the tissue destruction typically seen in perio disease. This phenomenon is not unique to the oral cavity. If we have an unlit cigarette in our hand, that unlit cigarette will never contribute to lung cancer or heart disease. It has to be burned and smoked. The body's response to the toxins in the tobacco, paper and additives can initiate disease processes.

Using currently available DNA testing of the specific bacteria in an individual patient's mouth can enable us to monitor pre and post-op levels of bacteria. If favorable clinical disease resolution is accompanied by significant reduction in bacterial levels we can then conclude that we have reached the endpoint of therapy and the patient's likelihood of recurrence is low, given that the patient's home care is frequent and effective and 3 month maintenance appointments are strictly adhered to. If; however, our favorable clinical outcomes are accompanied by a slight reduction in the pre-op levels of bacteria, and/or an increase in other species of bacteria that were low pre-operatively, we have a very different outcome and prognosis. Patients with this outcome have not achieved the same therapeutic endpoint as the previous patient example did, even if the level of clinical resolution is identical. A patient with favorable clinical results and unfavorable bacterial results may require a second round of active treatment with adjunctive therapy including systemic and locally applied antimicrobials such as Arestin. This patient has greater risk of disease recurrence due to the persistence of pathogenic bacteria. More frequent maintenance intervals, along with other bacterial reduction and host modulatory protocols may be indicated for a these patients.



Re-defining the endpoint of therapy, to include comparison of pre and post-operative levels of bacteria, is becoming the standard of care. In the near term other factors will also be included in the therapeutic endpoint, such as monitoring the levels of inflammatory mediators. As the specific mechanisms of periodontal disease are unraveled, our approach to treatment needs to evolve to take research results into account, all aimed at improving the care we provide for our patients. We must never lose sight of that ultimate endpoint of any treatment we undertake.

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