*Position Paper*

# Systemic Antibiotics in Periodontics*

THIS POSITION PAPER ADDRESSES the role of systemic antibiotics in the treatment of periodontal disease. The document consists of three sections: 1) concept of antibiotic periodontal therapy; 2) efficacy of antibiotic periodontal therapy; and 3) practical aspects of antibiotic periodontal therapy. The conclusions drawn in this paper represent the position of The American Academy of Periodontology and are intended for the information of the dental profession. *J Periodontol 1996;67:831-838.*

## INTRODUCTION

The microbial etiology of inflammatory periodontal diseases provides the rationale for use of antibiotics in periodontal therapy.

Antibiotics and chemotherapeutics have been prescribed for periodontal patients who do not respond to conventional mechanical therapy, for patients with acute periodontal infections associated with systemic manifestations, for prophylaxis in medically-compromised patients, and as an adjunct to surgical and non-surgical periodontal therapy. A recent review addresses antibiotic therapy for medical indications.[1] This position paper is concerned with the use of antibiotics in treatment of periodontitis lesions.

The concept of antibiotic periodontal therapy centers upon the pathogenic microbiota, the patient, and the drug. These issues are each addressed separately.

## PERIODONTAL PATHOGENS

The rational use of antibiotics for the treatment of periodontitis presupposes knowledge of the pathogenic microbiota. More than 500 bacterial taxa have been identified within periodontal pockets.[2] However, at present relatively few species have been strongly associated with periodontitis lesions (Table 1). Most putative pathogens are indigenous to the human oral cavity, and in some situations superinfecting (enteric Gram-negative rods, pseudomonads, staphylococci, yeasts) organisms may be present. Periodontitis lesions usually harbor a constellation of putative pathogens rather than a single pathogenic species.

Most putative periodontal pathogens are Gram-negative anaerobic rods. However, some pathogens are Gram-positive facultative and anaerobic cocci and rods and others are Gram-negative facultative rods. The putative periodontal pathogens vary considerably in sensitivity to several antibiotics, making simplistic antimicrobial chemotherapy problematic.[4]

## PATIENTS

Prime candidates for systemic antimicrobial therapy are patients who exhibit continuing loss of periodontal attachment despite diligent conventional mechanical periodontal therapy. Refractory periodontitis is often related to persistent subgingival pathogens and perhaps to impaired host resistance.[5] Patients with localized juvenile periodontitis and other types of early-onset periodontitis,[6] or with medical conditions predisposing to periodontitis[7] may need antibiotic therapy. Patients with acute or severe periodontal infections (periodontal abscess, acute necrotizing gingivitis/periodontitis) may also benefit from antibiotic therapy.[8] Patients with gingivitis or stable adult periodontitis usually respond well to mechanical periodontal therapy and derive little or no additional benefit from antibiotic therapy.[9]

## THE DRUG

The efficacy of periodontal antibiotic therapy is determined by the antimicrobial spectrum and the pharmacokinetic characteristics of the drug[10] and by local environmental factors[11] including 1) drug binding to tissues; 2) protection of pathogens through binding, consumption, or degradation of the drug by non-target microorganisms; 3) biofilm phenomena in plaque protecting the pathogens; 4) total bacterial load relative to the maximum achievable antibiotic concentration; 5) effectiveness of the host defenses; and 6) pathogens in periodontal tissues, root surfaces, and extra-dental oral sites not affected by the therapy.

Systemic antibiotic therapy has certain advantages over topical application of antimicrobial agents. Systemic antibiotics via serum can reach microorganisms at the base of deep periodontal pockets, in furcation areas, and within gingival epithelial and connective tissues. Also, systemic antibiotics may be capable of affecting periodontal pathogens colonizing oral mucosa and other extra-dental sites.[12-14] The possibility of markedly suppressing or eliminating periodontal pathogens from the entire mouth may reduce the risk for future recolonization of the periodontal pocket and recurrent disease pro-

*This position paper was developed under the direction of the Committee on Research, Science and Therapy and approved by the Board of Trustees of The American Academy of Periodontology in May 1996.*

**Table 1. Association Between Putative Periodontal Pathogens and Progressive Periodontal Disease***

| Very Strong | Strong | Moderate | Early Stage of Investigation |
|---|---|---|---|
| *Actinobacillus actinomycetemcomitans* | *Bacteroides forsythus* | *Streptococcus intermedius* | *Selenomonas* species |
| Spirochetes of acrute necrotizing gingivitis | *Prevotella intermedia* | *Prevotella nigrescens* | Gram-negative enteric rods |
| *Porphyromonas gingivalis* | *Eubacterium nodatum* | *Peptostreptococcus micros* | *Pseudomonas* species |
| | *Treponema denticola* | *Fusobacterium nucleatum* | *Staphylococcus* species |
| | | *Campylobacter rectus* | |
| | | *Eikenella corrodens* | |

*Modified from Socransky and Haffajee.[3]

gression. Moreover, systemic antibiotic therapy is carried out with relatively low financial cost to the patient.

Disadvantages of systemic antibiotic therapy when compared to locally applied antimicrobial agents include inability to achieve high gingival crevicular fluid concentration,[15] adverse drug reactions,[16] selection of multipleantibiotic resistant microorganisms,[4] and uncertain compliance.[17]

Combination drug therapy may be useful in periodontitis that involves a variety of periodontopathic species with differing antimicrobial susceptibilities. Combination therapy should include drugs that exhibit synergy or additive effects in vitro. For example, metronidazole-amoxicillin and metronidazole-ciprofloxacin act synergistically against *Actinobacillus actinomycetemcomitans* and other major periodontal pathogens.[18] However, some antibiotics through combination antagonism can lead to a reduction, rather than an increase, in antimicrobial activity. Antagonism occurs between bacteriostatic tetracyclines and bactericidal ß-lactam antibiotics.[19]

## CLINICAL STUDIES

Controlled clinical trials are necessary to determine the effectiveness of antibiotic therapy in periodontics.[20] Many existing studies are difficult to interpret because of open study designs, small sample size, short-term evaluation periods, clinically different patient groups, undetermined periodontitis disease activity, unknown baseline microbiota, varying antimicrobial regimens, and insufficient supragingival plaque control.[9] Since many clinical trials on antibiotic periodontal therapy have been performed in patients with little or unknown disease activity, they may have underestimated the potential value of systemic antibiotics in the treatment of progressive periodontitis.

Studies of several forms of periodontitis have evaluated systemic antibiotics as an adjunct to periodontal scaling and root planing. This is in agreement with good medical practice which dictates that the bacterial load should be reduced as much as possible prior to administration of antibiotic therapy. In addition, this study design aims to investigate possible additional clinical and/or microbiological effects of the antimicrobial drug in comparison to a placebo medication or no medication.

Table 2 lists investigations which evaluated the effect of antimicrobial therapy in adults with clinical evidence of recent disease-active periodontitis. Most studies concerned with patients with disease progression suggest that systemic antibiotic therapy may provide additional clinical benefits to conventional mechanical periodontal therapy, particularly in refractory patients.

Table 3 suggests a substantial benefit from using systemic antibiotics in treatment of localized juvenile periodontitis (LJP). However, open study designs are used in studies of LJP, mainly because of a limited number of study patients. Although the suppression of subgingival *A. actinomycetemcomitans* to below detectable levels is a major goal in the treatment of localized juvenile periodontitis,*52* other putative pathogens associated with LJP should be suppressed as well.

## PRACTICAL ASPECTS OF PERIODONTAL ANTIBIOTIC THERAPY

### Patient Selection

Antibiotic therapy is usually reserved for patients having continued periodontal breakdown after conventional mechanical treatment, although some patient categories with recognized increased risk for periodontal breakdown, such as juvenile periodontitis and other types of early onset periodontitis, may be treated with antibiotics during initial mechanical therapy.

### Microbiological Analysis

Microbiological analysis of the subgingival microbiota may be carried out after completion of conventional mechanical therapy to help assess the need for additional therapy, such as antibiotic treatment. Reevaluation with microbiological testing at 1 to 3 months after antimicrobial therapy may be desirable to verify the elimination or marked suppression of the putative pathogen(s) and to screen for possible superinfecting organisms, including Gram-negative enteric rods, pseudomonads, and yeasts. With the use of antibiotics for treatment of periodontal disease, the dentist is encouraged to know the specific antimicrobial susceptibility pattern of suspected pathogens in order to avoid prescribing antibiotics against undetected resistant pathogens, Treatment with inappropriate antimicrobial agents may lead to overgrowth of pathogens and poor clinical response.[53]

**Table 2. Clinical Studies of Systemic Antibiotic Therapies in Adult Periodontitis Paffents with Recent Disease Activity Prior to Antibiotic Therapy**

| Study | N Patients/ Disease | Antibiotic/Dose | Concurrent Treatment | Control | Follow-up (months) | Outcome |
|---|---|---|---|---|---|---|
| **Tetracyclines** | | | | | | |
| Rams and Keyes[21] | 21 RAP | TET 250 mg QID, 14 d | — | P | 11 | TET reduced PD, motile organisms, and crevicular leukocytes in refractory sites; DB study. |
| Lundström et al[22] | 9 RAP | DOX 100 mg/d, 14 do or MET 200 mg TID, 7 d | Sc | — | 18 | DOX or MET+Sc equally reduced clinical and microbiological parameters for at least 18 months. |
| Rams et al[23] | 10 RAP | TET 250 mg QID, 14 d | Sc | — | 61-72 | ETE+Sc reduced PD, AL, BOP, mobile organisms, and crecivular leukocytes. |
| Haffajee et al[24] | 33 RAP | TET 250 mg QUD, 21 d | S | — | 6 | Reduced *Aa* and *Cr* after therapy. No change in *Pg* and *Pi*. Reduction in AL. |
| Papil and Lewis[25] | 16 RAP | TET 250 mg QID, 6 d | Sc | — | 6-24 | Reduced PD maintained for up to 24 months. |
| Kulkarni et al[26] | 27 RAP | DOX 200 mg LD DOX 100 mg/d, 21 d | Sc | P | 7 | 43% reduction in disease activity risk with DOX vs. P; 45% of DOX group developed abcesses within 7 months. DOX reduced *Pg*, *Pi*, *Fuso*, and spiro, but not *Aa*; DB study. |
| **Metronidazole** | | | | | | |
| Gusberti et al[28] | 5 RAP | MET 250 mg TID, 10 d | Sc | — | 9 | MET+Sc reduced PD, AL, BOP, SPiro, *Pg*, and *Fuso* for at least 9 months. |
| Mombelli et al[29] | 10 RAP | ORN 500 mg TID, 10 d | Sc | — | 11 | ORN reduced PD, AL, BOP, spiro, *Pg*, and *Fuso* for at least 9 months. |
| **Penicillins** | | | | | | |
| Magnusson et al[30] | 10 RAP | AMOX/CLA 250 mg TID, 14 d | Sc | — | 12 | AMOX/CLA reduced AL, PD, and disease active sites from 14% initially to 7% at 9 months. One patient had 10% active sites with additional AL at 9 months and 12 months. |
| Magnusson et al[31] | 21 RAP | AMOX/CLA 250 mg TID, 14 d or CLIN QID, 10 d | Sc | P | 24 | No difference in % sites with AL in both groups. More % sites gained attachment in antiobiotic group than in P group. Increase of PD and tendency of renewed AL after 12 to 15 months. |
| Haffajee et al[27] | 40 RAP | AMOX/CLA 250 mg TID, 30 d | Sc + S | P | 10 | AMOX/CLA improved PD and AL compound to P or ibuprofen. More decrease in *Aa*, *Pg*, *Pi*, and *Cr* with *AMOX/CLA* than with controls. |
| **Clindamycin** | | | | | | |
| Gorden et al[32] Gorden et al[34] | 13 RAP | CLIN 150 mg QID, 7d | Sc | | 12 | CLIN+Sc improved clinical parameters, reduced motile organisms, and decreased annual rate of sites with disease activity from 1.0 to 0.5%. |
| Walker and Gordon[34] | 24 RAP | CLIN 150 mg QID, 7 d | Sc | — | 24 | CLIN improved clinical parameters, reduced annual rate of disease activity rate from 8% to 0.5% sites/patient, and increased time of episodes from 5 to 17 months, *Pg*, *Pi*, and *Pm* reduced or absent from 12 months. |
| **Combination Therapy** | | | | | | |
| Chin Quee et al[35] | 50 | ROD 3 tabs (1 tab MET 24 mg + SPIRA 750,000 IU) BID, 14 d | Sc | Sc, P | 6 | ROD+Sc reduced AL and spiro better than Sc alone; DB study. |
| van Winkelhoff et al[36] | 11 LJP 11 RPP | MET 250 mg TID + AMOX 375 mg TID, 7 d | Sc | — | 9-11 | Eliminated *Aa* in all patients; improved PD and BI. |
| Goene et al[37] | 4 RAP | MET 250 mg TID + AMOX 375 mg, TID, 7 d | Sc, S | — | 2-9 | Eliminated *Aa* in all patients; improved clinical parameters. |

**Table 2. Continued**

| Study | N Patients/ Disease | Antibiotic/Dose | Concurrent Treatment | Control | Follow-up (months) | Outcome |
|---|---|---|---|---|---|---|
| van Winkelhoff et al[38] | 28 LJP<br>50 AP<br>40 RAP | MET 250 mg TID + AMOX 375 mg TID, 7d | Sc | — | 3-9 | Eliminated *As* in 97% and *Pg* in 88% of patients; improved clinical status; additional AL in 6 patients. |
| Pavicic et al[18] | 3 RAP | MET 500 mg BID + CEP 200 mg BID, 7 d | Sc | — | 3 | Eliminated *Aa*; improvement of clinical status. |
| Pavicic et al[39] | 48 AP | MET 250 mg TID + AMOX 375 mg TID, 7 d | Sc | — | 36 | Eliminated *Aa* in 47/48 and *Pg* in 16/18 patients; no further AL. |

*Aa: Actinobacillus actinomycetemcomitan*
AL: Attachment loss
AMOX: Amoxiccilin
AMOX/CLA: Amoxicillin-clavulanic acid
AP: Adult periodontitis
BI: Bleeding index
BID: Twice a day
BOP: Bleeding on probing
CEF: Cefixime
CLIN: Clindamycin
*Cr: Campylobacter rectus*
d: Days
DB: Double blind
DOX: Doxycycline
*Fuso: Fusobacterium* species
LD: Loading dose
LJP: Localized juvenile periodontitis
MET: Metronidazole
ORN: Ornidazole
P: Placebo
PD: Probing depth
*Pg: Porphyromonas gingivalis*
*Pi: Prevotella intermedia*
QID: Four times a day
RAP: Refractory adult periodontitis
RPP: Rapidly progressive periodontitis
ROD: Rodogyl
S: Periodontal surgery
Sc: Scaling and root planing
SPIRA: Spiramycin
Spiro: Spirochetes
TET: Tetracycline
TID: Three times a day

**Table 3. Clinical Studies of Systemic Antiobiotic Therapies in Localized Juvenile Periodontitis Patients**

| Study | N Patients | Antibiotic/Dose | Concurrent Treatment | Control | Follow-up (months) | Outcome |
|---|---|---|---|---|---|---|
| Lindhe[40] | 16 | TET 250 mg QID, 14 d | S | — | 24 | 25% of patients had recurrent diesease activity in 24 months. |
| Slots and Rosling[41] | 6 | TET 250 mg QID, 14 d | Sc | — | 9 | TET reduced AL and suppressed *Aa* better than Sc alone. |
| Kornman and Robertson[42] | 8 | TET 250 mg QID, 28 d | Sc, S | — | 2 | TET+Sc better than Sc alone on sites with *Aa*. TET+Sc better than TET+Sc and Sc alone on sites with both *Aa* and *Pg/Pi*. |
| Kunhira et al[43] | 16 | PEN 250 mg QID, 10 d 3 mo for 9 mo | S | S,P | 9 | No advantage to adjuctive PEN; DB study. |
| Mandell et al[44] | 4 | DOX 100 mg BID LD, 100 mg/d, 14 d | — | S | 10 | DOX+S reduced *Aa* better than DOX alone. |
| Mandell and Socransky[45] | 8 | DOX 100 mg BID, LD 100 mg/d, 14 d | S | — | 12 | DOX+S suppressed *Aa* and arrested disease activity for 12 mo in 6/8 patients. |
| Novak et al[46] | 4 | TET 250 mg QID, 21-42 d | — | — | 3 | TET alone improved clinical and radiographic parameters. |
| Asikainen et al[47] | 16 | DOX 200 mg LD, then 100 mg daily, 13 d | Sc | P | 2 | No clinical or microbiological benefits of DOX; DB study. |
| Saxén et al[48] | 14 | DOX 200 mg LD, then 100 mg daily, 13 d | Sc | P | 20 | DOX reduced *Aa* better than P; DB study. |
| Novak et al[49] | 4 | TET 250 mg QID, 42 d | — | — | 2-48 | TET without Sc reduced PD, AL, and BOP for up to 4 years. |
| Saxén and Asikainen[50] | 27 | MET 200 mg TID 10 d or TET 250 mg QID, 12 d | Sc | Sc | 18 | Best clinical results with MET+Sc. *Aa* eliminated in 100% of MET group, in 44% of TET group, and in 67% of Sc group. |
| Christersson and Zambon[51] | 6 | TET 250 mg QID until 1 wk after *Aa* became undetectable or for a maximum of 8 weeks. | — | Sc | 24 | Improvement of clinical parameters, 4/6 patients were *Aa* positive after 12 months. |

*Aa: Actinobacillus actinomycetemcomitan*
AL: Attachment loss
BID: Twice a day
BOP: Bleeding on Probing
CLIN: Clindamycin
*Cr: Campylobacter rectus*
d: Days
DB: Double blind
DOX: Doxycycline
LD: Loading dose
MET: Metronidazole
P: Placebo
PD: Probing depth
PEN: Pencillin
*Pg. Porphyromonas gingivalis*
*Pi: Prevotella intermedia*
QID: Four times a day
S: Periodontal surgery
Sc: Scaling and root planing
TET: Tetracycline
TID: Three times a day

## Sampling Procedure

Microbial samples may be obtained from individual pockets with recent disease activity or from pooled subgingival sites. A pooled subgingival sample may provide a good representation of the range of periodontal pathogens to be targeted for antibiotic therapy.[54] A representative subgingival sample can be obtained from pooling one deep bleeding or suppurating periodontal pocket in each quadrant of the mouth. Subgingival samples can be collected with sterile paper points or a curet. For culturing, the specimens must be placed in a transport medium which has been formulated for reducing conditions to sustain viability of the sampled microorganisms.

## Culture Methods

Microbial anaerobic culturing provides the most comprehensive assessment of the periodontal microbiota. Total cell counts, the relative proportions of putative periodontal pathogens, and the occurrence of unusual subgingival organisms can be determined. Culturing is presently a prerequisite for in vitro antimicrobial sensitivity testing. Disadvantages with culturing are a time-consuming analysis, requirement of skilled technical personnel, relatively high financial costs, inability to grow some organisms, and limited survival time of sampled organisms.

## Non-Culture Techniques

Molecular genetic techniques to detect periodontal bacteria include species-specific DNA probes and polymerase chain reaction-based assays.[55] These techniques do not require viable cells and can show high sensitivity and specificity. They also have the potential to discriminate between high and low virulent strains of a given species. Molecular genetic detection techniques are relatively easy, fast and inexpensive to perform. Disadvantages are the limited number of species that can be detected, the inability to quantitate target organisms, and the inability to perform antimicrobial sensitivity testing of target microorganisms.

Polyclonal or monoclonal antibodies can be used to detect putative periodontal pathogens in direct plaque smears.[55] Most immuno-serodiagnostic assays are based on immunofluorescent and enzyme-linked immunosorbent detection. Disadvantages of immuno-serodiagnostic techniques are the limited number of species that can be detected and the inability to determine antimicrobial sensitivity of suspected pathogens.

## Antimicrobial Sensitivity Testing

Resistance of putative periodontal pathogens to antimicrobial agents has increased.[4] Therefore, antimicrobial sensitivity testing is important if the clinician contemplates using antibiotics in the management of patients with refractory periodontitis. The American Academy of Periodontology 1995 Parameters of Care[56] state: Once the diagnosis of refractory periodontitis has been made, the following steps may be taken:

1. Collection of subgingival microbial samples from selected sites for analyses, possibly including antibiotic-sensitivity testing;
2. Selection and administration of an appropriate antibiotic regimen;
3. In conjunction with the administration of an antimicrobial regimen, conventional periodontal therapies may be used;
4. Reevaluation with microbiological testing as indicated.

There are some clinical situations in which sensitivity testing may not add information that significantly alters the way the disease is treated. For example, patients with classical clinical characteristics of localized juvenile periodontitis are ordinarily heavily colonized with *A. actinomycetemcomitans* which is nearly always sensitive to tetracyclines in vitro.[16] Also, some periodontal putative pathogens (i.e., *Porphyromonas gingivalis* and *Campylobacter rectus*) are usually susceptible to a wide range of antimicrobial agents and the antimicrobial profiles are quite predictable.[16]

**Table 4. Common Antibiotic Therapies in Treatment of Periodontitis***

| Drug | Adult dosage |
|---|---|
| Tetracycline-HCI | 500 mg/TID/21days |
| Metronidazole | 500 mg/TID/8 days |
| Ciprofloxacin | 500 mg/BID/8 days |
| Metronidazole & amoxicillin | 250 mg/TID/8 days of each drug |
| Metronidazole & ciprofloxacin | 500 mg/BID/8 days of each drug |

From Slots and van Winkelhoff.[57]

## Selection of Antibiotics

Relatively few studies have been performed regarding which antibiotics should be selected for refractory patients in whom the subgingival biota have been characterized through microbial testing, In addition, the optimal dose of antibiotics remains unclear[10] since most current antibiotic regimens are empirically developed rather than through systematic research.[20] Tables 2 and 3 may be of some value in making decisions on appropriate prescription regimens. Table 4 lists frequently prescribed antibiotic regimens for treatment of periodontitis.[57]

*Tetracyclines* may be indicated in periodontal infections in which *A. actinomycetemcomitans* is the prominent pathogen; however, in mixed infections these antibiotics may not provide sufficient suppression of subgingival pathogens to arrest disease progression.[11]

*Metronidazole* may arrest disease progression in refractory periodontitis patients with Porphyromonas gingivalis and/or Prevotella intermedia infections with fewor no other potential pathogens.[58]

**Table 5. Antibiotic Regimens With Acute Periodontal Abcesses***

| |
|---|
| Amoxicillin: loading dose of 1.0 g followed by a maintenance dose of 500 mg/TID for 3 days, followed by a patient evaluation to determine whether further antiobiotic therapy or dage adjustment is required. |
| **With Allergy to ß-lactam Drugs:** |
| Azithromycin: loading dose 1.0 g on day 1, followed by 500 mg once daily on days 2 and 3; or |
| Clindamycin: loading dose of 600 mg, followed by 300 mg/QID/3 days. |

*From Pallasch.[10]

*Clindamycin* has demonstrated efficacy in refractory periodontitis and may be considered with periodontal infections of *Peptostreptococcus*, ß-hemolytic streptococci, and various oral Gram-negative anaerobic rods.[34] Clindamycin should be prescribed with caution because of the potential for pseudomembranous colitis as a result of intestinal overgrowth with *Clostridium difficile*. Other antibiotics can occasionally also cause pseudomembranous colitis.

*Amoxicillin-clavulanic* acid may represent an alternative to clindamycin.[30] In guided tissue regeneration, systemic amoxicillin-clavulanic acid therapy has been used to suppress periodontal pathogens and increase the gain of clinical attachment.[59]

*Ciprofoxacin* is effective against enteric rods, pseudomonads, staphylococci, *A. actinomycetemcomitans*, and other periodontal organisms.[60] Ciprofloxacin may be combined with metronidazole or a ß-lactam drug for treatment of mixed anaerobic periodontal infections.[57]

*Metronidazole plus amoxicillin* provides a relatively predictable eradication of periodontal *A. actinomycetemcomitans* and *P. gingilalis* in early-onset forms of periodontitis and in refractory adult periodontitis.[18,38,61] Ciproflaxin may substitute for amoxicillin in individuals who are allergic to ß-lactam drugs and are at least 18 years of age.

Antibiotic therapy in conjunction with primary surgical incision and drainage may be necessary for acute periodontal abscesses with systemic manifestations (fever, malaise, lymphadenopathy). Table 5 describes adult regimens that may be used with acute periodontal abscesses.

## Sequencing of Antibiotic Therapy

Antibiotics should be considered as an adjunct to mechanical periodontal debridement. Use of potent antibiotics presupposes adequate clinical diagnosis, thorough mechanical debridement, and microbiological analysis where indicated. The following schedule represents apractical approach to periodontal antibiotic therapy.

1. Initial periodontal therapy should include thorough mechanical root debridement combined with surgical access if needed.

2. Antibiotics may be prescribed on the basis of the clinical need for further treatment, the findings of microbiological testing, and the medical status and current medications of the patient.

3. The clinical response should be evaluated 1 to 3 months after completion of the mechanical therapy. If periodontal disease appears to progress or if inflammation does not resolve, a microbiological examination of the subgingival microbiota may help determine the presence and amount of remaining putative periodontal pathogens.

4. If the clinical examination warrants it, 1 to 3 months after systemic antimicrobial therapy, another microbiological test may be needed to verify the subgingival elimination of target pathogen(s) and screen for possible superinfecting organisms. High levels of subgingival *Streptococcus sangui.v/mitis*, *Actinomyces*, and *Veillonella* species are suggestive of periodontal health or minimal disease.[62]

5. After resolution of the periodontal infection, the patient should be placed on an individually tailored maintenance program. Supragingival plaque control in the supportive periodontal therapy phase may help prevent recolonization by putative periodontal pathogens.[63] Recurrence of progressive disease may prompt repeated microbiological testing and subsequent antibiotic therapy targeted against the specific microorganisms.

## Summary

Systemic antibiotic therapy in periodontics is based on the premise that specific microorganisms initiate destructive periodontal disease and that the antibiotic agent in vivo can exceed concentrations necessary to kill or inhibit the pathogen(s).

Systemic periodontal antibiotic therapy aims to reinforce mechanical periodontal treatment and to support host defenses in overcoming the infection by killing subgingival microorganisms that remain after conventional mechanical periodontal therapy. Pathogens may escape the effect of mechanical treatment because of their ability to invade periodontal tissues, to reside in anatomical tooth structures inaccessible to periodontal instruments, or as a result of poor host defense mechanisms.

Systemic antibiotic therapy may benefit periodontitis patients who are refractory to conventional mechanical periodontal treatment. The patient's medical status and possible drug-interactions with concurrent medications should be considered and antimicrobial sensitivity testing in some patients may be valuable. Single antimicrobial drug therapies are able to suppress some subgingival bacteria for a prolonged period of time depending on the host defense and oral hygiene efforts. Combination drug therapies which aim at enlarging the antimicrobial spectrum and exploiting synergy between antibiotics may be indicated with complex mixed periodontal infections.

The emerging resistance among oral and medical pathogens to common antibiotics dictates a restrictive and conservative use of systemic antibiotic therapy.

## Acknowledgments

The primary author for this paper was Dr. Jørgen Slots. Members of the 1995-1996 Committee on Research, Science and Therapy included Drs. Gary C. Armitage, Chair; David Cochran; Robert E. Cohen; Gary Greenstein; Angelo Mariotti; Michael P. Rethman; Martha J. Somerman; Thomas E. Van Dyke; Terry Rees, Consultant; W. Lee Young, Jr., Consultant; Roy C. Page, Consultant, Philip J. Hanes, Consultant; David Dennison, Consultant; and Robert J. Genco, Consultant.

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