Clindamycin in dentistry: More than just effective prophylaxis for endocarditis?

Itzhak Brook, MD, MSc, a Mike A. O. Lewis, PhD, BDS, FDSRCS, FRCPat.h George K. B. Sándor, DDS, PhD, MD, FRCD(C), FRCS(C), FACS.c Marjorie Jeffcoat, DMD,d L. P. Samaranayake, BDS, DDS, FRCPat, FCDSHK,e and Jorge Vera Rojas, DDS,f Washington, DC, Cardiff, Wales, Toronto, Canada, Philadelphia, Pa, Hong Kong, and Tlaxcala, Mexico
GEORGETOWN UNIVERSITY, CARDIFF UNIVERSITY, HOSPITAL FOR SICK CHILDREN, UNIVERSITY OF PENNSYLVANIA, UNIVERSITY OF HONG KONG, AND UNIVERSITY OF TLAXCALA

Clindamycin is a broad-spectrum antibiotic with activity against aerobic, anaerobic, and \(\beta\)-lactamase–producing pathogens. This antibiotic has been used for many years as prophylactic treatment during dental procedures to prevent endocarditis. However, the spectrum and susceptibility of the bacteria species involved in dental infections indicate that clindamycin would also be an effective treatment option for these conditions. In addition to its antiinfective properties, clindamycin has high oral absorption, significant tissue penetration, including penetration into bone, and stimulatory effects on the host immune system. This review discusses the microbiologic and clinical evidence supporting the efficacy and safety of clindamycin for the successful management of dental infections. (Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2005;100:550-8)

Clindamycin, an antimicrobial agent that has been in use worldwide for more than 3 decades, has been consistently effective in the treatment of infections involving a wide spectrum of facultative and strictly anaerobic bacteria.\(^1,2\) The clinical value of clindamycin in a number of medical settings is well known, but it has been under-recognized as an antimicrobial agent for use in dentistry.

During the past decade, both the British Society for Antimicrobial Chemotherapy\(^3\) and the American Heart Association\(^4\) revised their recommendations for prophylaxis of infective endocarditis following dental procedures, which has resulted in the routine use of clindamycin by general dental practitioners in the United Kingdom and the United States. In addition, clindamycin is prescribed in other countries not only for prophylaxis of endocarditis but also for the treatment of acute dental infections\(^5\) and for prophylaxis of local infections following orthognathic surgery.\(^6\)

Historically, the principle reason for the restricted use of clindamycin in dentistry has been concern regarding potential adverse events, in particular, the development of \textit{Clostridium difficile} diarrhea or pseudomembranous colitis. However, a comprehensive review of the risk factors for \textit{C difficile} infection associated with antibiotic therapy revealed that this concern is likely to be unfounded.\(^7\) Although this therapeutic complication has been reported for many antibiotics, and the potential risk with most agents is stated in the literature, it is becoming increasingly clear that the true incidence of the problem is low for all antimicrobials. It is now recognized that \textit{C difficile}–associated problems are particularly rare when clindamycin is used in outpatient ambulatory care settings.\(^8\) Indeed, there is ample evidence accumulated over more than 2 decades indicating that clindamycin is not a frequent cause of antibiotic-associated colitis. From a dental perspective, it would appear that the incidence of \textit{C difficile} infection with clindamycin is no greater than that with amoxicillin or amoxicillin/clavulanate, 2 antimicrobial agents frequently used in the management of acute dental infection.\(^9\) In view of this unwarranted preconception about clindamycin, this review covers the microbiology of dental infections and positions clindamycin in relation to other antimicrobial agents often prescribed in dentistry.

**MICROBIOLOGY OF DENTAL INFECTIONS**

The commensal oral microflora comprise more than 350 morphologically and biochemically distinct microbial species. Analysis through molecular detection methods indicates a myriad presence of previously
unknown and uncultivable organisms in the oral cavity. Such a microbial diversity not only has prevented clear identification but also has hindered the etiologic association of these microbes with specific oral infections. It is generally recognized that strict anaerobes comprise a majority of the healthy commensal oral microflora, and they outnumber facultative organisms by a ratio of 100:1 within the gingival crevice. Contemporary microbiologic studies have revealed that the bacteria recovered from dental infections reflect this mixed population of facultative and strictly anaerobic bacteria that also are regarded as indigenous constituents of host oral microflora.

**Dentoalveolar and endodontic infections**

Microbiologic investigations of acute dental infections undertaken during the past 15 years have confirmed the polymicrobial nature of dentoalveolar and endodontic infections. The major facultative isolates have been streptococci, in particular members of the *Streptococcus milleri* group, while the predominant strictly anaerobic isolates have been *Peptostreptococcus, Veillonella, Prevotella*, and *Actinomyces* species (Table I). Quantitative studies have revealed the viable microbial flora of acute dental abscesses to be approximately 10⁷ colony-forming units/mL. As stated, not only are strict anaerobes isolated more frequently than facultative species, but the organisms occupy nearly 90% of the overall viable mixed flora.

Pathogenicity experiments have revealed that strictly anaerobic species, in particular gram-negative bacilli, are the likely pathogens in periapical infections. In addition, abscess models have shown that combinations of bacterial species, such as *S milleri*, anaerobic gram-positive collis, and anaerobic gram-negative bacilli, are more pathogenic when inoculated subcutaneously in pairs into animals than when inoculated separately.

In addition to experimental investigation of the potential pathogenicity of isolates, attempts have been made to relate the association of specific bacterial species and the severity of clinical symptoms. For instance, *Fusobacterium nucleatum* has been recovered more often from patients with severe odontogenic infections.

**Periodontal infections**

Gingivitis and periodontitis, the most frequently occurring diseases in humans, are essentially initiated by dental plaque, which is recognized as a complex biofilm comprised of an orderly community of bacteria encased in a polymeric matrix derived both from their metabolic products and from the host nutrients. In addition to subgingival plaque bacteria, the specific and nonspecific defense mechanisms of the host play a critical role in periodontal diseases. At present, the major bacterial pathogens implicated in periodontal disease, so-called periodontopathogens, include *Actinobacillus actinomycetemcomitans*, *Porphyromonas gingivalis*, *Bacteroides forsythus*, *Prevotella nigrescens*, *Fusobacterium* species, and *Treponema* species (Table I). Periodontal disease is an archetypal example of a polymicrobial infection, and the synergistic reactions between the microorganisms appear to play a key role in the pathogenic process. As stated, both the specific and nonspecific immune responses of the host to subgingival plaque are considered to play critical roles in the initiation, progression, and recovery from periodontal diseases. One of the most

### Table I. Predominant bacterial species encountered in acute dental infections

<table>
<thead>
<tr>
<th>Anaerobic bacteria</th>
<th>Dentoalveolar and endodontic infections</th>
<th>Periodontal infections</th>
<th>Peri-implant infections</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gram-positive cocci</td>
<td>Peptostreptococcus species</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td></td>
<td>Peptostreptococcus micros</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Gram-positive bacilli</td>
<td>Actinomyces species</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td></td>
<td>Eubacterium species</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td></td>
<td>Propionibacterium species</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td></td>
<td>Lactobacillus species</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Gram-negative bacilli</td>
<td>Veillonella species</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td></td>
<td>Prevotella species</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td></td>
<td>Prevotella nigrescens</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td></td>
<td>Prevotella intermedia</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td></td>
<td>Porphyromonas species</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td></td>
<td>Porphyromonas gingivalis</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Bacteroides species</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td></td>
<td>Bacteroides forsythus</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td></td>
<td>Fusobacterium species</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td></td>
<td>Fusobacterium nucleatum</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Spirochetes</td>
<td>Treponema denticola</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td></td>
<td>Treponema socranski</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Facultative anaerobic bacteria</td>
<td>Gram-positive cocci</td>
<td>Streptococcus species</td>
<td>X</td>
</tr>
<tr>
<td></td>
<td></td>
<td>β-hemolytic streptococci</td>
<td>X</td>
</tr>
<tr>
<td></td>
<td></td>
<td><em>Streptococcus milleri</em> group</td>
<td>X</td>
</tr>
<tr>
<td></td>
<td>Gram-positive bacilli</td>
<td>Lactobacillus species*</td>
<td>X</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Actinobacillus actinomycetemcomitans</td>
<td>X</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Capnocytophaga species</td>
<td>X</td>
</tr>
</tbody>
</table>

*Species encountered in this infection.*
*Microorganisms associated with dental caries.*
important components of the host response is the gingival crevicular fluid (GCF), which contains both specific and nonspecific defense factors. These include polymorphonuclear leukocytes that migrate into the gingival crevice, as well as the recently described antimicrobial factors present on epithelia (such as α-defensins, β-defensins, and cathelicidins). The host immune response is essential for maintaining a healthy periodontium. The damage and destruction of periodontal tissues associated with gingivitis and periodontitis are unlikely to occur in the absence of periodontopathic bacteria.

Peri-implantitis

Peri-implantitis is an emerging form of periodontitis that is a direct consequence of the increasing use of dental implants. Peri-implant disease refers to the general category of pathologic changes that can occur in the hard and soft tissues surrounding an implant. The integration of an implant can be jeopardized by the presence of bacteria in the tissues and the concomitant inflammatory reactions. However, most peri-implant disease is plaque-induced, and the spectrum of bacterial species recovered is comparable to that encountered in periodontitis. Bacterial species that have received particular attention in this context include A. actinomyctemcomitans, P. gingivalis, Prevotella intermedia, and F. nucleatum (Table I).

In summary, many studies have proven that a wide range of bacterial species that are in essence constituents of the commensal oral microflora cause dental infections. However, it is likely that strict anaerobes, particularly gram-negative bacilli, play a key role in the etiopathology of dental infections, although interactions with other species are a common feature.

ANTIMICROBIAL SUSCEPTIBILITY OF DENTAL INFECTIONS

The global emergence of antibiotic-resistant bacteria is a phenomenon of much concern. Classic examples are the wide prevalence of medically important bacteria, such as Staphylococcus aureus and Streptococcus pneumoniae, that are resistant to both older and newer generations of penicillin and many other antibiotics. Similarly, some bacterial species recovered from acute dental infections have demonstrated reduced in vitro antimicrobial susceptibility. Although isolates from acute dental infection rarely demonstrated resistance to penicillin 15 years ago, this is no longer the case. Resistance to penicillin has increased significantly in gram-negative anaerobic bacilli, in particular Prevotella, Porphyromonas, Bacteroides, and Fusobacterium species, owing to their production of β-lactamas. Studies in the United Kingdom indicated that penicillin resistance in acute dentalveolar infections rose from 3% in 1986 to 23% of isolates in 1995. Similar incidences of increased penicillin resistance have been reported in the United States (33%), Sweden (38%), and Japan (39%).

The presence of penicillin-resistant bacteria has been reported to be the cause of treatment failures in head and neck infections of dental origin. The production of β-lactamases not only plays a direct pathogenic role by destroying the drug, but also indirectly “shields” non-β-lactamase-producing, penicillin-sensitive bacteria within the infection. The widespread use of penicillin has contributed to this problem, because it has been shown that the administration of penicillin leads to the emergence of β-lactamase-producing bacteria, especially gram-negative bacilli, in sites such as the oropharynx. Similar patterns of increased resistance to other antibiotics prescribed for dental infections appear not to have been reported. Of specific interest is the extremely low incidence of resistance to clindamycin, even in countries such as Germany and Japan, where this agent is used frequently to treat acute dental infections.

USE OF ANTIBIOTICS IN DENTAL INFECTIONS

The majority of acute dental infections can be managed successfully through surgical drainage alone. However, occasions do arise when adequate drainage cannot be achieved or the patient has clinical signs of systemic upset. In these circumstances, it may be necessary to provide antimicrobial therapy. Identification of the causative organisms and determination of antimicrobial susceptibility in a specific patient are not practical in a general practice setting, because the specimen cannot be processed fast enough to be of value in making treatment decisions. Even in a hospital setting, microbiologic findings are rarely available to the clinician within the first 48 hours because of the prolonged incubation times required to isolate strict anaerobes. More recently, molecular-based techniques have been developed to provide rapid identification of particular bacterial species, especially penicillin-resistant Prevotella species. These techniques may prove helpful for bacterial identification if they become widely available in the future.

At present, recommendations of choice antimicrobial agents for particular forms of dental infections are based on published data. Agents in the penicillin group, principally phenoxymethylpenicillin and amoxicillin, traditionally have been regarded as the antimicrobials of choice in the treatment of acute dental infections in the United Kingdom, while erythromycin traditionally has been used as an alternative agent for patients with
hypersensitivity to penicillins. However, resistance to these antimicrobial agents has been demonstrated within the oral flora and this observation must be taken into consideration when prescribing these drugs. Other antimicrobial agents that have been suggested for the treatment of acute dental infections include metronidazole, ornidazole, cephalosporin, azithromycin, spiramycin, amoxicillin-clavulanate, and clindamycin. Standard dosages usually are used for dental outpatients, although increased dosages and combination therapy have been recommended for severe infections or when surgical drainage cannot be achieved. In the treatment of acute dental infections, a high-dosage, short-course (3-g amoxicillin twice in 8 hours) treatment regimen has been found to be as effective as a conventional 5-day course of penicillin (250 mg every 6 hours). Combination therapy most frequently involves the use of metronidazole with either penicillin or amoxicillin.

Referral to a specialist is required when a patient presents with symptoms of cellulitis, difficulty in swallowing, tachycardia, hypotension, raised temperature, lethargy, or dehydration. Specialist care also may be necessary when standard outpatient treatment fails to result in symptomatic improvement. Hospitalization and provision of intravenous antibiotics is necessary when a patient has marked tissue swelling (particularly in the neck), inadequate hydration, and fever. Management of such infections should be aggressive, because rapid systemic involvement can ensue.

Dental infection remains a significant threat to the patient. Untreated or inadequately treated infections can progress to cause widespread swelling of the tissue spaces in the head and neck (Ludwig’s angina), with the spread of infection to the mediastinum. Death from dental infections still occurs, due to late intervention and/or treatment failure. Airway management is the sine qua non of therapy, often necessitating intubation or a surgical airway (such as a tracheostomy) as a life-saving measure. In children, these infections occur most frequently in the maxilla and can spread rapidly into the periorbital tissues with generalized systemic symptoms. These observations emphasize the importance of timely surgical treatment of dental infection, supported by appropriate antimicrobial therapy.

**Periodontal disease**

Numerous studies have shown that the clinical outcome of periodontal treatment is improved if specific microbial pathogens are eradicated from the tissues. Because local scaling and root planing alone cannot achieve this predictably, mechanical treatment has been combined with the delivery of either topical or systemic antimicrobial therapy. Agents used in this situation include amoxicillin, clindamycin, metronidazole, tetracycline, and doxycycline. Interestingly, results of recent trials indicate that some of the therapeutic effects are due, in part, to properties other than the antimicrobial activity of these drugs. Some antibiotics have been shown to have antiinflammatory properties that are independent of their antimicrobial activities.

**Peri-implantitis**

Peri-implantitis has been shown to respond to local mechanical and chemical means of reducing the microbial flora in the immediate vicinity of the implant. However, consideration has to be given to the possibility of causing physical damage to the surface of the implant. Irrigation with an antiseptic, such as chlorhexidine, has been found to be beneficial. Alternatively, the efficacy of systemic antimicrobial therapy, such as amoxicillin, metronidazole, or tetracycline, has also been investigated.

**ANTIMICROBIAL ACTIVITY OF CLINDAMYCIN**

Clindamycin works by inhibiting protein synthesis at the bacterial 50S ribosomal subunit, thus interfering with the process of peptide-chain formation in bacteria. It also may inhibit the binding of aminoacyl—transfer ribonucleic acid (tRNA) or the translocation of messenger ribonucleic acid (mRNA) following amino acid binding on the ribosome, further disrupting protein synthesis.

Clindamycin has a high level of in vitro activity against a variety of facultative and strictly anaerobic bacteria. The spectrum of susceptible gram-positive organisms includes *Actinomyces*, *Eubacterium*, *Lactobacillus*, *Peptostreptococcus*, *Propionibacterium*, and *Staphylococcus* species, including penicillin-resistant strains. Clindamycin also is effective against β-hemolytic streptococci (groups A, B, C, and G), *Streptococcus viridans* group, and members of the *S milleri* group. With regard to strict anaerobes, clindamycin has activity against a range of gram-negative species, including *Porphyromonas* species, *Prevotella* species, *Bacteroides fragilis* group, *Veillonella* species, and *Fusobacterium* species, including β-lactamase—producing strains. Although this drug’s activity is poor against gram-negative facultative organisms, such as *Eikenella corrodens*, *Haemophilus* species, *Moraxella* species, and *Escherichia coli*, these organisms have not been implicated as major pathogens in dental infections.

In addition to a direct antibacterial effect on ribo¬somal units, clindamycin has a number of unique pharmacologic features that enhance its clinical efficacy (Table II). Clindamycin is the only proven antibiotic that reduces the adherence of bacteria to the epithelial...
Table II. Pharmacologic features of clindamycin

<table>
<thead>
<tr>
<th>Feature</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wide spectrum of in vitro antimicrobial activity that includes</td>
<td>those species implicated as pathogens in dental infections</td>
</tr>
<tr>
<td>Achievement of high levels in saliva, gingival crevicular fluid, and</td>
<td>bone</td>
</tr>
<tr>
<td>Reduction of the expression of virulence factors (M protein, capsule,</td>
<td>and toxins)</td>
</tr>
<tr>
<td>Increased bacterial phagocytosis and killing</td>
<td></td>
</tr>
<tr>
<td>Intracellular activity</td>
<td></td>
</tr>
<tr>
<td>Activity in conjunction with the host defense system</td>
<td></td>
</tr>
<tr>
<td>Suppression of the adherence of bacteria to the mucosal epithelial cells</td>
<td></td>
</tr>
<tr>
<td>and the expression of virulence factors (such as production of M protein</td>
<td>by β-hemolytic streptococci, capsule formation</td>
</tr>
<tr>
<td>by facultative gram-positive Streptococcus species)</td>
<td></td>
</tr>
<tr>
<td>Postantibiotic effect</td>
<td></td>
</tr>
</tbody>
</table>

Cells of mucosal surfaces and inhibits the expression of virulence factors. The antimicrobial inhibits the production of M protein by group A β-hemolytic streptococci, thereby inhibiting capsule formation by facultative gram-positive Streptococcus species. It also inhibits toxins produced by Clostridium species and S aureus. Furthermore, clindamycin can inhibit bacterial proteins, toxins, enzymes, and cytokines once bacteria are inside target tissue. Clindamycin induces morphologic changes on the surface of bacteria that render them more susceptible to being killed and also stimulates chemotaxis, thus promoting mobilization of polymorphonuclear leukocytes to the site of infection and resulting in ingestion of bacteria.

TISSUE CONCENTRATIONS OF CLINDAMYCIN

Clindamycin reaches high concentrations in saliva, GCF, and bone. Several studies have shown that the concentration of clindamycin in these tissues is approximately 40% to 50% of the concentration in serum. In a recent animal study, Zetner and colleagues found that the mean concentration of clindamycin in mandibular bone in dogs and cats was approximately 3 times the serum concentration after intravenous infusion of clindamycin. In another study, the concentration of clindamycin in bone and other tissue was above the minimal concentration at which 90% of the isolates are inhibited (MIC90) for pathogens that are likely to be introduced into the tissues in patients undergoing oral and maxillofacial surgery. The ability of this antimicrobial to reach high intracellular concentrations in tissues facilitates activity against intracellular pathogens by enhancing opsonization, phagocytosis, and intracellular killing by macrophages. The high intracellular concentration of clindamycin and extended activity inside the bacterium yield a postantibiotic effect by which the antimicrobial remains active although serum concentration levels are subinhibitory. This postantibiotic effect may vary with the type of bacteria.
play a useful role in the treatment of a number of dental 
and maxillofacial infections.

CLINDAMYCIN IN THE TREATMENT OF 
DENTAL AND MAXILLOFACIAL INFECTIONS

Acute dental infections

The efficacy of clindamycin in dental infections 
has been investigated in 5 prospective, randomized-
controlled, clinical trials, and 1 retrospective study, 
mostly involving management of periapical abscesses 
(Table III). The clinical trials effectiveness was high, with 
success rates of 97%-100%. The dosage of clindamycin 
used in these studies was 600 mg/day (150 mg 4 times 
daily), except for the retrospective study, in which 
patients received 1200 to 2400 mg/day. In addition, the 
clinical improvement in pain, fever, and swelling 
occurred more rapidly in patients receiving clindamycin 
than in those given the control therapy (usually penicil-
lin). In practice, the recommended dosing for clinda-
mycin ranges from 150 mg 4 times daily to 300 mg 
4 times daily.

In vitro susceptibility tests included in the aforemen-
tioned studies demonstrated that the majority of iso-
lates were inhibited by a clindamycin concentration of 
1.2 μg/mL or less. Clindamycin inhibited 94%-100% 
of anaerobic clinical isolates with MIC < 1.2 μg/mL 
and 90%-100% of aerobic isolates with MIC < 1.0 
μg/mL.5,31,81 A dose of 300 mg produces a 1.1 μg/mL 
concentration in GCF after 6 hours, well above serum 
concentrations (0.1 μg/mL).

Periodontal disease

Four trials involving patients with periodontal disease 
have shown long-term benefits in the treatment of 
refractory disease for clindamycin at a dosage of 600 
mg/day for 7 days. The endpoints used were clinical 
features combined with microbiologic outcomes at 
1 and 2 years.82-84 One study revealed that clindamycin 
was superior to tetracycline at follow-up after 1 year.85

Peri-implantitis

There are no clinical trials assessing the efficacy of 
clindamycin either at the time of implant placement or 
at subsequent development of peri-implantitis. The drug’s 
spectrum of antimicrobial activity along with the high 
concentrations achieved in bone indicate that clinda-
mycin could be an effective agent in the management of 
implant-related infections, an issue deserving further 
investigation.

CONCLUSIONS

Clindamycin has been used clinically for more 
than 30 years and has demonstrated a good record of 
efficacy and safety in a variety of infections, including 
odontogenic infections. Nevertheless, the use of this 
agent has been limited, generally owing to concern 
about the development of antibiotic-associated pseudo-
membranous colitis. However, data indicate that the 
association between clindamycin and pseudomembra-
nous colitis is unfounded, because it rarely occurs in 
outpatients treated with clindamycin. The occurrence of 
pseudomembranous colitis is no greater with clindamy-
lin than with many other commonly used antibiotics.

Clindamycin is a unique antimicrobial that achieves 
high tissue (including bone) concentrations, penetrates 
intracellularly, increases phagocytosis, inhibits toxin 
production, and has a postantibiotic effect. A review of 
the microbiologic and clinical literature relating to 
maxillofacial infections has revealed this antimicrobial 
to be a highly effective agent in the field of dentistry. 
On this basis, clindamycin should be considered as a 
first-line antimicrobial for all dental infections.

REFERENCES

1. Wilson WR, Cockerill FR III. Tetracyclines, chloramphenicol, 
erithromycin, and clindamycin. Mayo Clin Proc 1987;62: 
906-15.
2. Van Landuyt HW. Spectrum of clindamycin in vitro effects 

Table III. Clinical trials assessing efficacy of clinda-
mycin in the treatment of odontogenic infections

<table>
<thead>
<tr>
<th>Clinical trial</th>
<th>n</th>
<th>Dosage and regimen</th>
<th>Success rate, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schuen 19745,31,86-88</td>
<td>36</td>
<td>Clindamycin 150 mg PO q 6 h for 6 days</td>
<td>100</td>
</tr>
<tr>
<td></td>
<td>32</td>
<td>Penicillin V 250 mg q 6 h for 6 days</td>
<td>97</td>
</tr>
<tr>
<td>Kannangara 198088</td>
<td>10</td>
<td>Clindamycin phosphate 300-600 mg IV or IM q 6 h for 5 days</td>
<td>100</td>
</tr>
<tr>
<td></td>
<td>25</td>
<td>Penicillin 4-20 million units q 24 h for 5 days</td>
<td>80</td>
</tr>
<tr>
<td>Gilmore 198881</td>
<td>28</td>
<td>Clindamycin 150 mg PO q 6 h for 7 days</td>
<td>100</td>
</tr>
<tr>
<td></td>
<td>27</td>
<td>Phenoxymethyl penicillin 250 mg q 6 h for 7 days</td>
<td>100</td>
</tr>
<tr>
<td>Mangundjaja 199687</td>
<td>74</td>
<td>Clindamycin 150 mg PO q 6 h for 7 days</td>
<td>100</td>
</tr>
<tr>
<td></td>
<td>73</td>
<td>Clindamycin 150 mg PO + ibuprofen 600 mg PO q 6 h for 7 days</td>
<td>99</td>
</tr>
<tr>
<td>Mangundjaja 19905</td>
<td>52</td>
<td>Clindamycin 150 mg PO q 6 h for 7 days</td>
<td>100</td>
</tr>
<tr>
<td></td>
<td>54</td>
<td>Ampicillin 250 mg PO q 6 h for 7 days</td>
<td>98</td>
</tr>
<tr>
<td>von Konow 199231</td>
<td>30</td>
<td>Clindamycin 150 mg PO q 6 h for 7 days</td>
<td>97</td>
</tr>
<tr>
<td></td>
<td>30</td>
<td>Phenoxymethyl penicillin 1 g q 12 h for 7 days</td>
<td>97</td>
</tr>
</tbody>
</table>

PO, by mouth; IV, intravenous; IM, intramuscular.
Adapted from Ehrenfeld et al.89

Reprint requests:
Mike A. O. Lewis, PhD, BDS, FDSRCS, FRCPath
Wales College of Medicine
University of Cardiff
Heath Park
Cardiff CF14 4XN
United Kingdom
lewismao@cardiff.ac.uk

ON THE MOVE?
Send us your new address at least six weeks ahead

Don’t miss a single issue of the journal! To ensure prompt service when you change your address, please photocopy and complete the form below.

Please send your change of address notification at least six weeks before your move to ensure continued service. We regret we cannot guarantee replacement of issues missed due to late notification.

JOURNAL TITLE: ____________________________________________

OLD ADDRESS: ____________________________________________
Affix the address label from a recent issue of the journal here.

NEW ADDRESS: ____________________________________________
Clearly print your new address here.
Name ___________________________________________________
Address ________________________________________________
City/State/ZIP __________________________________________

COPY AND MAIL THIS FORM TO: Subscription Customer Services
Elsevier Inc.
6277 Sea Harbor Dr
Orlando, FL 32887

OR FAX TO: 407-363-9661
OR E-MAIL: elspcs@elsevier.com

OR PHONE: 800-654-2452
Outside the U.S., call 407-345-4000