ABSTRACT

Antibiotic therapy is not a substitute for dental or surgical intervention. Infected wisdom teeth, periodontal pockets greater than 5 mm, and diseased dental pulp tissue require mechanical intervention to restore a patient's health and homeostasis.[1] When a patient presents with a bacterial infection, the dentist will prescribe an antibiotic to kill the offending bacteria (bactericidal) or suppress the bacterial metabolic activity and growth (bacteriostatic), thereby allowing the immune system to clear the infection.[2] Antibiotic therapy is frequently necessary as an adjunct to mechanical dental therapy and is rarely utilized as a standalone treatment.[3]Antibiotics have specific pharmacologic effects on bacterial metabolism but, in most cases, will not directly interact with human physiology.[4] There are some notable exceptions, such as the tetracycline class of antibiotics, whereby the inherent anti-inflammatory properties of the molecules are also desired and utilized. Antibiotics, however, are not without adverse effects on human physiology. Most of these effects occur through damage and destruction to the human microbiome, the bacterial multispecies symbiont present in every human being.[5] A healthy microbiome is necessary for human metabolic homeostasis in areas as diverse as the immune system, digestion, neurotransmitter production and utilization, and control of inflammation.[6-8] These issues fall well beyond the customarily understood antibiotic side effects of diarrhea, an upset stomach, and nausea. Dental professionals must consider the patient's immediate health to clear an acute infection and the long-term ramifications of the human microbiome's health with dental antibiotic therapy.[9]

EDUCATIONAL OBJECTIVES

Upon completion of this course, the dental professional should be able to:

1. List and describe the common antibiotics utilized in dentistry
2. Discuss the role of antibiotics in dental infectious disease
3. Describe the safety, dosages, and contraindications for antibiotic use in dentistry
4. Outline how the information in this course can be used to improve patient care outcomes

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Antibiotic therapy and dentistry: Common oral infections and the appropriate antibiotic pharmacology

A PEER-REVIEWED ARTICLE | by Eric Bornstein, DMD

The vast majority of historical prose pertaining to the discovery of antibiotics begins with Sir Alexander Fleming in 1928 and his discovery of the fungus *Penicillium notatum*, producing penicillin.[10,11] This narrative, however, leaves out several significant observations made by careful physicians treating patients before Fleming. For example, in 1871, the father of modern surgical antisepsis, Joseph Lister, observed that the fungus *Penicillium glaucum* had inhibitory effects on bacterial growth in urine. Lister reportedly used an extract of this *Penicillium glaucum* to cure the infected wounds of a nurse at Kings College in London.[12] Eighteen years later, Jean-Paul Vuillemin coined the term "antibiosis."[13] This term defines the ability of one organism to kill another, thus ensuring its own survival.[14] Finally, in 1897, Ernest Duchesne discovered that after

injecting *E. coli* or *Salmonella typhi* into guinea pigs, the animals quickly died. However, the animals lived when the bacteria were premixed with *Penicillium glaucum*. Duchesne postulated that his findings could lead to both prophylactic and therapeutic applications for the fungus in antibiosis.[15]

The narrative that most people learned states that in 1928, Alexander Fleming accidentally discovered (in forgotten colonies of *Staphylococci*) that a molecule secreted from the fungus *Penicillium notatum* was inhibiting the growth of his in vitro bacteria. This molecule was later purified and called penicillin. The first industrial production of penicillin occurred in 1940.[16] For oral infections, the first recorded use of penicillin for Vincent's angina infection (acute necrotizing ulcerative gingivitis) was in 1945,[17] and the antibiotic appears to have entered general use for wisdom teeth infections and oral surgery in approximately 1947.[18] A list of common antibiotics utilized in modern dentistry can be found in Table 1.[19]

Mechanisms of action for antibiotics

Each antibiotic class listed in Table 1 has a unique mechanism of action (MOA) (Figure 1). For an antibiotic to be useful in human therapy, the MOA must selectively target a metabolic pathway unique to bacteria, not human metabolism.[20] Some unique bacterial processes that antibiotics target are peptidoglycan (cell wall) biosynthesis, bacterial protein synthesis, bacterial DNA uncoiling for transcription and replication, and folic acid synthesis. These unique capabilities make antibiotics powerful medicines in the treatment of infectious diseases.[21]

TABLE 1: Common antibiotics used in dentistry

Antibiotic class	Examples
Beta-lactam	Penicillin, amoxicillin
Cephalosporin (beta-lactam)	Cephalexin
Macrolide	Azithromycin, clarithromycin
Lincosamide	Clindamycin
Quinolones	Ciprofloxacin, levofloxacin
Tetracyclines	Doxycycline
1st generation 2-methyl-5-nitroimidazole	Metronidazole
Combination antibacterial	Amoxicillin/clavulanate

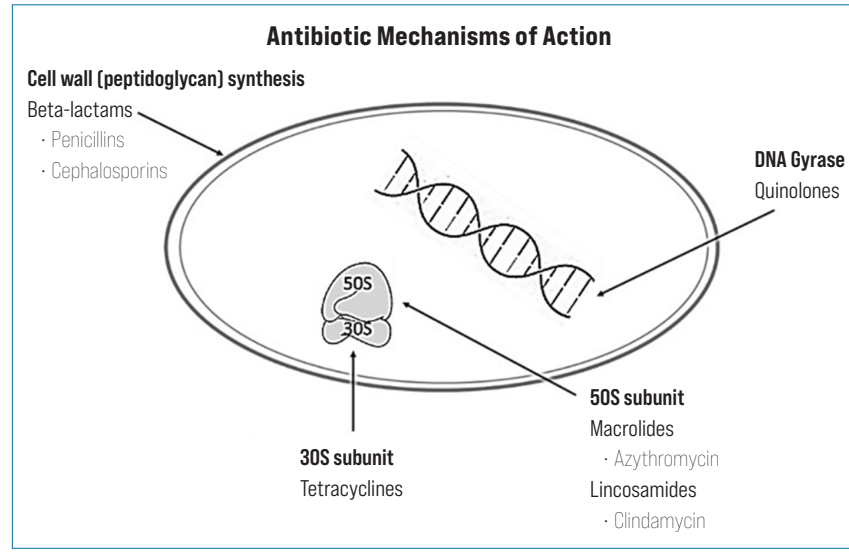


FIGURE 1: Antibiotics and their MOAs

Beta-lactam antibiotics: Beta-lactam antibiotics work by inhibiting the synthesis of the peptidoglycan polysaccharide layer of bacterial cell walls. The beta-lactam ring in an antibiotic molecule binds to enzymes called penicillin-binding proteins (PBPs). PBPs catalyze the final stages of peptidoglycan biosynthesis. By forming stable covalent complexes with PBPs, beta-lactam antibiotics inhibit peptidoglycan crosslinking, leading to bacterial cell death. Cell death occurs from cell lysis in a hypoosmotic environment.[22]

Macrolide antibiotics: Macrolide antibiotics inhibit bacterial protein synthesis in bacteria by binding to the 23S rRNA of the bacterial 50S ribosomal subunit. Protein synthesis is thereby inhibited by preventing the transpeptidation/translocation step in the process. When the advancement of the mRNA-tRNA moiety is halted, protein synthesis is prevented.[23]

Lincosamide antibiotics: Lincosamide antibiotics prevent bacterial replication through a bacteriostatic mechanism that interferes with protein synthesis. Lincosamides bind to the 50S subunit of the bacterial ribosome on the 23S portion of the subunit. This binding disrupts the transpeptidation reaction and inhibits early peptide chain elongation.[24]

Quinolone antibiotics: Quinolone antibiotics inhibit bacterial DNA synthesis. This class targets two bacterial enzymes responsible for DNA replication: DNA gyrase and topoisomerase IV. By inhibiting these enzymes, quinolones cause breaks in the bacterial DNA strands and prevent the DNA breaks from being repaired.[25]

Tetracycline antibiotics: Tetracyclines inhibit bacterial protein synthesis by binding to the 30S and 50S subunits of the bacterial ribosome and blocking the attachment of charged tRNA at the P site in the peptide chain. The inhibition of the formation of a peptide chain inhibits bacterial growth.[26]

2-methyl-5-nitroimidazole antibiotics: 2-methyl-5-nitroimidazole antibiotics exert antibacterial effects in an anaerobic environment against most obligate anaerobes by damaging the organism's DNA. Antibiotics such as metronidazole easily diffuse through the membrane of both aerobic and anaerobic bacteria. The reduced form of 2-methyl-5-nitroimidazole antibiotics and free radicals can interact with DNA, causing inhibition of DNA synthesis and DNA degradation, leading to the death of bacteria.[27]

Combination antibiotics: Augmentin is a combination of amoxicillin (a penicillin-class antibacterial) and the beta-lactamase inhibitor clavulanic acid. Clavulanic acid is a beta-lactam structurally related to the penicillins and can inactivate some beta-lactamases (enzymes that break beta-lactam rings) by blocking the active sites of these enzymes.[28]

Oral infections and common antibiotic regimens

More than 700 different species of bacteria have been identified in the oral cavity.[29] The three most prevalent infections treated with antibiotics by dentists are periodontal disease, periapical abscess (necrotic and infected dental pulp tissue), and pericoronal infection around erupting wisdom teeth.[30]

Periapical abscess: Pulpitis will lead to infection and periapical abscess when bacteria enter the nerve canal through caries, tooth fracture, or the apical foramen. A periapical abscess is an infection of the periodontal tissues around the apical foramen of a tooth that will not resolve without root canal therapy and often leads to acute pain and swelling in the affected patient. Periapical abscess symptoms include tooth sensitivity, hypereruption, lymphadenopathy, and swelling. Such a condition will often require antibiotics to assist in controlling the infection before root canal therapy can be completed (Table 2).[31]

Root canal therapy includes infected tissue extirpation and mechanical curettage to remove infected pulp tissue and bacteria, which is then followed by disinfection and sealing with inert materials such as gutta-percha. If the periapical abscess spreads to adjoining teeth or contiguous tissue, it will cause cellulitis, requiring more aggressive antibiotic treatment. When using adjunctive antibiotics, in addition to adequate debridement and surgical drainage, a dentist should endeavor to prescribe the antibiotics for as short a time as possible to assist the patient's immune system in clearing the pathogens.[32] Without speedy antibiotic treatment, facial swelling, fever, trismus, and cellulitis can follow, leading to fever, sepsis concerns, and potentially Ludwig's angina.[33]

Periodontal disease: When periodontal pathogens penetrate gingival epithelium, they elicit an inflammatory response from the innate immune system.[35-37] If left unchecked, this response will cause constant chronic inflammation and periodontal tissue destruction, including the alveolar bone. Subgingival plaque that harbors periodontopathic bacteria contains upwards of 500 different species of bacteria.[38] A select group of these bacteria is considered causative organisms for periodontitis and periodontal disease. These species include *Porphyromonas gingivalis*, *Fusobacterium sp.*, *Treponema sp.*, *Aggregatibacter actinomycetemcomitans*, *Prevotella sp.*, *Bacteroides sp.*, *Campylobacter sp.*, and *Eikenella*.[39,40]

Almost immediately, the innate immune system recognizes highly antigenic lipopolysaccharide (LPS) and extracellular polymeric secretions (EPS) from the biofilm of the growing colonies.[41,42] These antigenic biofilm components include bacterial polysaccharides, proteins, lipids, and extracellular DNA.[43] The innate immune system will then marshal polymorphonuclear leukocytes and macrophages to migrate into the area and surrounding tissues. The newly activated immune cells will secrete multiple inflammatory cytokines and chemokines into the surrounding periodontal tissues and pocket area that include tumor necrosis factor-alpha (TNF-α), interleukin-6 (Il-6), interleukin-17 (Il-17), and matrix metalloproteinases (MMPs).[44,45]

Once the periodontal pocket is 4 mm deep or greater, the microbial colonies will not be cleared by the immune system without mechanical debridement or surgical intervention, and a chronic infectious/inflammatory condition will become established.[46,47] The result of this chronic condition will be destruction of the alveolar bone and surrounding tissues, bleeding, inflammation, and spreading of the disease.[48] Left untreated, periodontal disease will lead to systemic inflammation that can exacerbate diabetes, cardiovascular disease, and neurological disease.[49] Mechanical

TABLE 2. Commonly prescribed antibiotics in endodontics[34]

Antibiotic	Loading dose	Maintenance dose
Amoxicillin	1000 mg	500 mg every 8 hours
Amoxicillin/clavulanic acid	1000 mg	500 mg every 8 hours
Clindamycin	600 mg	300 mg every 6 hours
Clarithromycin	500 mg	250 mg every 12 hours
Azithromycin	500 mg	250 mg every 24 hours
Metronidazole	1000 mg	500 mg every 6 hours

periodontal therapy is necessary to treat chronic periodontal conditions, and adjunctive antibiotic therapy is frequently warranted and required (Table 3).[50,51]

Pericoronitis and third molar infection: Pericoronitis is an acute inflammation and infection localized around partially impacted third molars. Symptoms include generalized pain, trismus, discomfort with mastication and swallowing, swelling of the gingival flap, facial swelling, fever, lymphadenopathy, and deep space infection.[53] Pericoronitis is exacerbated by food particles trapped under gingival flaps and chronic masticatory trauma on the gingival flaps. In severe cases of pericoronitis, the treatment would include antibiotics and oral surgery (Table 4).[54]

TABLE 3. Commonly prescribed antibiotics for periodontitis[52]

Antibiotic	Dose
Metronidazole	500 mg every 8 hours/8 days
Clindamycin	300 mg every 8 hours/8 days
Doxycycline	100-200 mg every 24 hours/21 days
Ciprofloxacin	500 mg every 12 hours/8 days
Azithromycin	500 mg every 24 hours/4–7 days
Metronidazole/amoxicillin	250 mg every 8 hours/8 days (each drug)
Metronidazole/ciprofloxacin	500 mg every 12 hours/8 days (each drug)

TABLE 4. Commonly prescribed antibiotics for pericoronitis[55]

Antibiotic	Dose
Amoxicillin/metronidazole	500 mg (each) every 8 hours/5 days
Augmentin (for b-lactam-producing strains) with resistance	625 mg every 12 hours/5 days (each drug)
Metronidazole	500 mg (each) every 8 hours/5 days
Azithromycin[56] (with penicillin allergy)	500 mg every 24 hours/3–5 days

Antibiotic resistance

Bacterial resistance to antibiotics is a worldwide phenomenon associated with high rates of human morbidity and mortality. Resistant infections can occur when a person is colonized with a pathogen that has acquired one or multiple resistant traits. Depending on the level and number of resistant traits a pathogen develops (or acquires), it can become virtually untreatable with conventional antimicrobial compounds and methods.

Much of the modern discussion concerning dental misuse of antibiotics as a large driver of antibiotic resistance is misguided. The overwhelming peer-reviewed evidence is contrary to this opinion. A recent study by Tiseo et al. (2022) in the journal *Antibiotics* stated the following:[57]

- Overuse and misuse of antibiotics in both humans and animals are major contributors to antimicrobial resistance.
- Agricultural use of antibiotics is of particular concern.
- It is estimated that 73% of all antimicrobials sold globally are used in animals raised for food.
- Antimicrobial use in animals is primarily for growth promotion and mass prophylaxis and is administered in feed at low doses, conditions ideal for the selection of drug resistance. As a result, food animals may be a larger reservoir of resistance genes than humans.
- Agricultural use of antibiotics has been implicated in the emergence of ESBL-producing *coli*, fluoroquinolone-resistant campylobacter species, and multidrug-resistant Salmonella species.

A second study by Xu et al. (2022) in *Frontiers in Microbiology* stated the following:[58]

- In Africa, the European Union, and the United States, an estimated 50–80% of all antibiotics are applied to animals, primarily to promote the growth of animals and to prevent bacterial infection.
- Antibiotics used in food animals are predicted to increase by 11.5% (up to 200,235 tons) in 2030.
- Approximately 75% of antibiotics are not absorbed by the animals and are excreted from the body via feces and urine, which can directly contaminate and harm the surrounding environment.
- The misuse or overuse of antibiotics in animal production has led to diverse antibiotic-resistant bacteria (ARB) and antibiotic-resistance genes (ARGs), which can be transferred in animals and humans.
- There is increasing evidence that antibiotic resistance in humans is mainly related to the wide application of nontherapeutic antibiotics in animals.

These facts, however, concerning the overuse and misuse of antibiotics in animals, do not absolve dental professionals from their culpability in poor decision-making with antibiotics. There is, unfortunately, little antimicrobial testing and susceptibility analysis that occurs in the dental setting. Therefore, relatively few objective decisions are made when treating a bacterial infection, such as periodontal disease, pericoronal disease, or apical periodontitis.[59] Most dental infections are simply treated with broad-spectrum antibiotics, such as amoxicillin, azithromycin,

and metronidazole. These subjective decisions can lead to improper antibiotic choices and durations of therapy.[60]

There are four primary metabolic changes by which bacteria gain the ability to resist antibiotics. The metabolic changes result from adapted genetic expression or the acquisition of resistance genes. These changes are listed below:

- Conformational change in an antibiotic's binding site[61]
- Structural modification of porins that will no longer allow antibiotics into a bacterial cell[62]
- The creation of efflux pumps to remove antibiotics from the inside of bacterial cells[63]
- Enzymatic inactivation of antibiotics by the bacteria[64]

The general method by which bacteria will develop resistance to an antibiotic is depicted in Figure 2.[65]

Antibiotic resistance is becoming more prevalent in all human infections.[66] Because most physicians frequently prescribe antimicrobial treatment "empirically" for all manner of pulmonary, soft tissue, and UTI infections without antibiotic susceptibility testing,[67] the dentist does not know what potential resistant strains could be causing an infection in a patient with oral symptoms.[68] It is, therefore, imperative that a dentist, during the "past medical history" and "poly-pharmacy" analysis of a patient visit, ask the simple question: "Have you taken any antibiotics for anything in the last three to four months?"[69] Knowledge of prior recent antibiotic usage for bronchitis, skin infection, or UTI will give the dentist a much greater ability to choose a proper antibiotic for an oral infection.[70] Below is a potential scenario:

A 76-year-old female presents to the dentist with facial swelling secondary to an endodontically involved tooth. Upon questioning, the dentist discerns that the patient was prescribed a three-week regimen of amoxicillin within the last 90 days for bronchitis that resolved 30 days ago. With this knowledge, only gained by asking the question, "Have you taken any antibiotics for anything in the last three to four months?" the dentist can now contemplate treating the patient with azithromycin as a first-line therapy, as there is a good chance that the pathogen causing the oral swelling is partially resistant to amoxicillin, as a result of the prior bronchitis treatment.

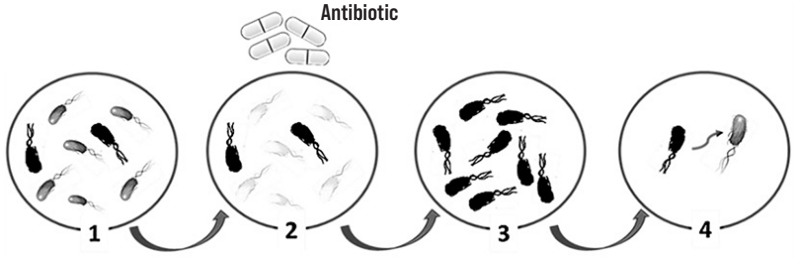
Antibiotic therapy and harm to the microbiome

The human bacterial microbiome comprises the entire set of bacteria on or within the human body.[71] The majority of the microbes are symbiotic in nature, indicating that both the bacteria and host benefit from the relationship.[72] The bacterial microbiome assists human metabolism,[73] immune regulation,[74] and is interconnected with most organs in the human body.[75,76] Maintaining a healthy microbiome and its proper symbiosis is essential to the health and well-being of an individual.[77]

If the human microbiome is disrupted by exposure to antibiotics, dysbiosis will occur and can lead to significant health conditions.[78] Antibiotic-induced microbiome dysbiosis will increase a patient's exposure to inflammatory bowel disease,[79] pseudomembranous colitis,[80] irritable bowel syndrome,[81] diabetes,[82] obesity,[83] cancer,[84] cardiovascular problems,[85] and central nervous system disorders.[86] It is, therefore, incumbent on the dental professional, when adjunctive antibiotic therapy is necessary, to discontinue the antibiotic therapy as quickly as possible to minimize damage to the microbiome.[87]

Changing pathogenic profile of oral bacteria in odontogenic and periodontal infections

As we approached the end of the 20th century, multiple strains of the skin pathogen *Staphylococcus aureus* were found and cultured in the oral microbiome. These *S. aureus* cultures were reported in the peer-reviewed literature as causative agents in oral infections. (1991–2000).[88-91] This trend has continued with increasing frequency through 2022, with multidrug-resistant forms of *S. aureus*, such as MRSA, becoming more frequent in oral microbiology sampling and epidemiology.[92-96] To make matters worse, the *S. aureus* strains found in the oral cavity are highly pathogenic biofilm producers resistant to neutrophil attacks and many antibiotics.[97,98] Highly virulent and pathogenic *S. aureus* is now considered the leading cause of aggressive acute bacterial endocarditis, with 30% mortality rates within



1. Resistant and nonresistant bacteria in an infection
2. Antibiotics kill the susceptible bacteria
3. Antibiotic-resistant bacteria overgrow and dominate the infection
4. Antibiotic-resistant bacteria transfer mutations to other bacteria

FIGURE 2: How antibiotic-resistant infections develop

one year.[99] These microbiological and epidemiologic emerging issues may portend changes to antibiotic prophylaxis and dental infection treatment regimens in the future.[100,101]

Most recently, and representative of the changing profile of the oral microbiome, the American Heart Association in an effort to minimize side effects from antibiotics such as *Clostridium difficile*, replaced bacterial endocarditis prophylaxis with clindamycin to azithromycin, doxycycline, or a cephalosporin.[102] Doxycycline retains a robust ability to inhibit *S. aureus* and MRSA in soft tissue and oral infections.[103]

Conclusion

Dentists prescribe antimicrobial therapy for the treatment and prevention of oral infection and bacterial endocarditis prevention. The majority of oral infectious diseases are managed with mechanical therapy, and adjunctive antibiotics if deemed necessary. Indications for the use of systemic antibiotics in dentistry are specific and limited.

When a dentist prescribes an antibiotic, it is based on clinical symptoms, objective and subjective data, and their own past clinical experience as a guide. The vast majority of dental professionals do not know what microorganism is responsible for the infection they are treating, as microbial testing and antibiotic susceptibility analyses are not frequently done in a dental setting.

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1. The fungus that secretes penicillin is:
   - A. *Penicillium glaucum*
   - B. *Penicillium notatum*
   - C. *Aspergillus*
   - D. *Candida*

2. Who coined the term "antibiosis," which defines the ability of one organism to kill another and ensure its own survival?
   - A. Joseph Lister
   - B. Alexander Fleming
   - C. Jean-Paul Vuillemin
   - D. Louis Pasteur

3. For oral infections, the first recorded use of penicillin was for:
   - A. An infected parotid gland
   - B. Apical surgery
   - C. Pericoronitis
   - D. Acute necrotizing ulcerative gingivitis

4. Beta-lactam antibiotics include:
   - A. Penicillin, amoxicillin, and cephalexin
   - B. Azithromycin and clarithromycin
   - C. Ciprofloxacin and levofloxacin
   - D. None of the above

5. Macrolide antibiotics include:
   - A. Penicillin, amoxicillin, and cephalexin
   - B. Azithromycin and clarithromycin
   - C. Ciprofloxacin and levofloxacin
   - D. None of the above

6. Quinolone antibiotics include:
   - A. Penicillin, amoxicillin, and cephalexin
   - B. Azithromycin and clarithromycin
   - C. Ciprofloxacin and levofloxacin
   - D. None of the above

7. For an antibiotic to be useful in human therapy, the MOA must selectively target a metabolic pathway unique to:
   - A. Fungi
   - B. Bacteria
   - C. Viruses
   - D. Humans

8. Beta-lactam antibiotics work by inhibiting bacterial:
   - A. Protein synthesis
   - B. Cell wall synthesis
   - C. DNA uncoiling
   - D. None of the above

9. Macrolide antibiotics work by inhibiting bacterial:
   - A. Protein synthesis
   - B. Cell wall synthesis
   - C. DNA uncoiling
   - D. None of the above

10. Quinolone antibiotics work by inhibiting bacterial:
   - A. Protein synthesis
   - B. Cell wall synthesis
   - C. DNA uncoiling
   - D. None of the above

11. More than ___ different species of bacteria have been identified in the oral cavity.
   - A. 200
   - B. 400
   - C. 500
   - D. 700

12. Which is the most prevalent infection treated with antibiotics by dentists?
   - A. Periodontal disease
   - B. Periapical abscess
   - C. Peri-coronal infection
   - D. All of the above

13. Periapical abscesses require which of the following mechanical therapies?
   - A. Scaling and root planing
   - B. Operculectomy
   - C. Root canal therapy
   - D. None of the above

14. The first line of adjunctive antibiotic therapy for a periapical abscess is:
   - A. Amoxicillin
   - B. Clindamycin
   - C. Ciprofloxacin
   - D. Doxycycline

15. Once a periodontal pocket is 4 mm deep or greater:
   - A. Antibiotic therapy is necessary
   - B. Combination antibiotic therapy is necessary
   - C. Mechanical debridement or surgical intervention is necessary
   - D. None of the above

16. Two antibiotics frequently added to metronidazole for periodontal infection are:
   - A. Doxycycline and clindamycin
   - B. Azithromycin and clindamycin
   - C. Amoxicillin and ciprofloxacin
   - D. None of the above

17. In severe cases of pericoronitis, the treatment should include:
   - A. Scaling and root planing
   - B. Antibiotics and oral surgery
   - C. Root canal therapy
   - D. None of the above

18. Second-line antibiotic therapy for pericoronitis in patients allergic to beta-lactam antibiotics is:
   - A. Amoxicillin
   - B. Clindamycin
   - C. Ciprofloxacin
   - D. Azithromycin

19. The overwhelming evidence pointing to the growth of antibiotic resistance comes from:
   - A. Dental SBE prophylaxis
   - B. Dental antibiotic therapy
   - C. Antibiotics placed in animal feed
   - D. None of the above

20. The largest reservoir of resistance genes for antibiotics is found in:
   - A. Animals
   - B. Humans
   - C. Birds
   - D. None of the above

21. The metabolic changes by which bacteria gain the ability to resist antibiotics are:
   - A. Conformational change in an antibiotic's binding site
   - B. Structural modification of porins and the creation of efflux pumps
   - C. Enzymatic inactivation of antibiotics by the bacteria
   - D. All of the above

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22. The human microbiome is symbiotic, which means:

A. Neither the bacteria nor the host benefit from the relationship
B. Only the bacteria benefit from the relationship
C. Only the host benefits from the relationship
D. Both the bacteria and the host benefit from the relationship

23. If the microbiome is disrupted by exposure to antibiotics, the following will occur:

A. Extended health
B. Dysbiosis
C. A and B
D. None of the above

24. A disrupted microbiome can cause:

A. Pseudomembranous colitis and irritable bowel syndrome
B. Diabetes and obesity
C. Cancer and cardiovascular problems
D. All of the above

25. For how long should a dental professional continue to prescribe antibiotic therapy?

A. As long as possible
B. One week after the infection has been cleared
C. The shortest possible time to clear an infection
D. Until the patient desires to discontinue the therapy

26. In the last 25 years, there has been a significant increase in which of the following bacterial species found in the oral cavity and oral infections?

A. *Campylobacter*
B. *Staphylococcus aureus*
C. *Streptococcus*
D. *Enterococcus*

27. Antibiotic susceptibility testing in the dental office is:

A. Rarely performed
B. Always performed
C. Not necessary
D. Not recommended

28. *Staphylococcus aureus* is now the most prevalent infecting organism found in which bacterial infection?

A. Onychomycosis
B. Seborrheic dermatitis
C. Acute infective endocarditis
D. None of the above

29. A highly pathogenic strain of *Staphylococcus aureus* is:

A. ESBL B. VRE C. CRE D. MRSA

30. The pathogenic ability of *Staphylococcus aureus* to ___ makes it so dangerous.

A. Control human cells
B. Produce biofilm
C. Destroy antibodies
D. None of the above

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Antibiotic therapy and dentistry: Common oral infections and the appropriate antibiotic pharmacology

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2. Discuss the role of antibiotics in dental infectious disease
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10.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	25.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
11.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	26.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
12.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	27.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
13.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	28.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
14.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	29.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
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